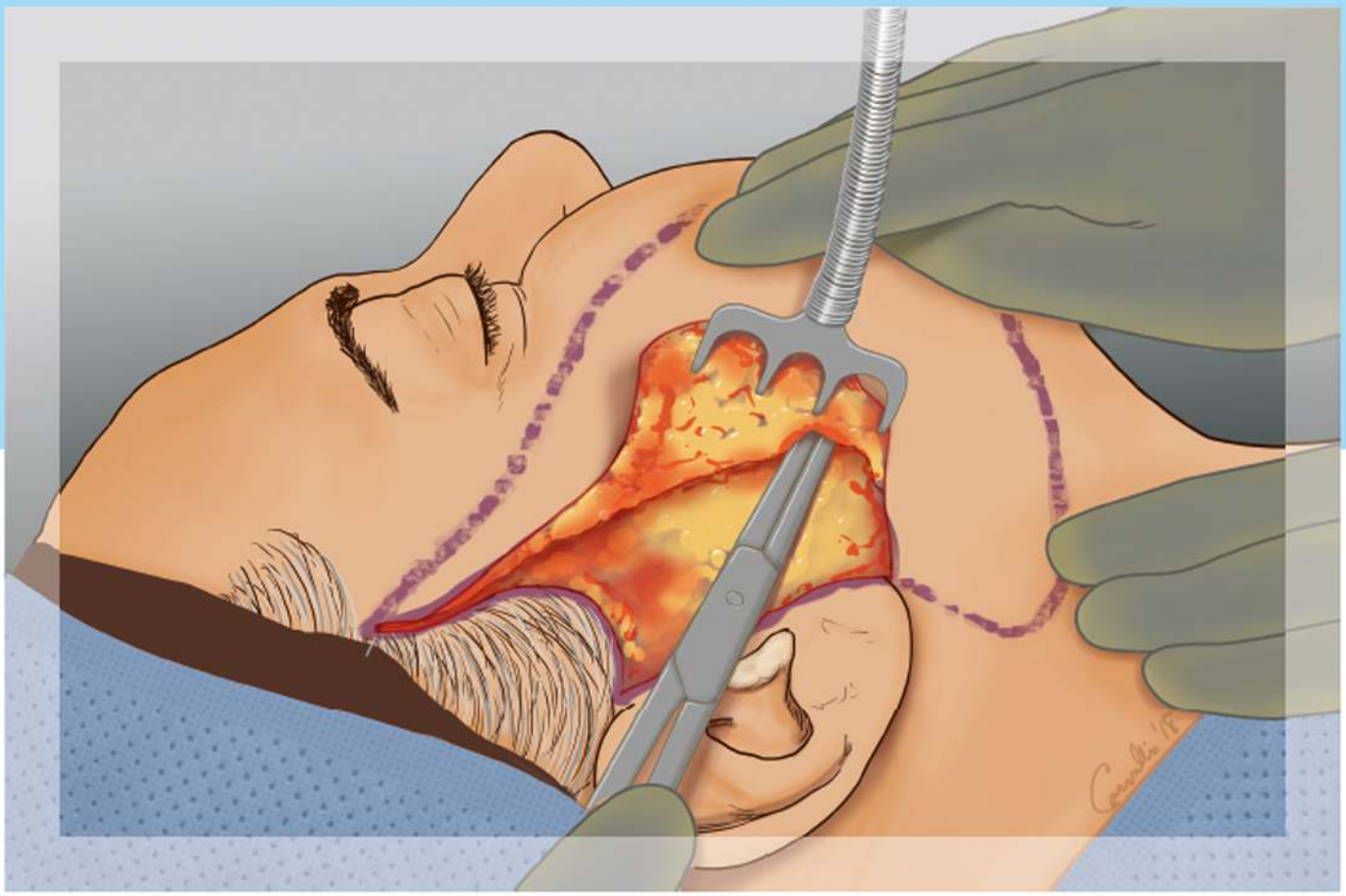
Plastic Surgery

A Practical Guide to Operative Care

Bruce A. Mast



Online at MedOne Videos

Thieme

To access the additional media content available with this e-book via Thieme MedOne, please use the code and follow the instructions provided at the back of the e-book.

Plastic Surgery

A Practical Guide to Operative Care

Bruce A. Mast, MD, FACS
Maurice J. Jurkiewicz Professor
Chief of Plastic and Reconstructive Surgery
Department of Surgery
University of Florida College of Medicine
Gainesville, Florida

782 illustrations

Thieme
New York • Stuttgart • Delhi • Rio de Janeiro

Library of Congress Cataloging-in-Publication Data

Names: Mast, Bruce A., author.
Title: Plastic surgery : a practical guide to operative care / [edited by] Bruce A. Mast.
Other titles: Plastic surgery (Mast)
Description: New York : Thieme, [2021] | Includes bibliographical references and index. |
Identifiers: LCCN 2018041474 (print) | LCCN 2018041934 (ebook) | ISBN 9781626232594 (ebook) | ISBN 9781626232587 (pbk.) | ISBN 9781626232594 (eISBN)
Subjects: | MESH: Reconstructive Surgical Procedures
Classification: LCC RD118 (ebook) | LCC RD118 (print) | NLM WO 600 | DDC 617.9/52--dc23
LC record available at https://lccn.loc.gov/2018041474

Thieme Medical Publishers, Inc.
333 Seventh Avenue, 18th Floor
New York, NY 10001, USA
www.thieme.com
+1 800 782 3488, customerservice@thieme.com

Cover design: Thieme Publishing Group
Typesetting by Thomson Digital

Printed in USA by King Printing Company, Inc. 5 4 3 2 1

ISBN 978-1-62623-258-7

Also available as an e-book:
eISBN 978-1-62623-259-4

Contents

Videos

Video 1: Abdominoplasty. (Courtesy: Bruce A. Mast)

Video 2: Marking superior medical pedicle and infiltrating tumescent solution into the skin to be de-epithelialized and the breast parenchyma. (Courtesy: Rukmini S. Rednam and Bruce A. Mast)

Video 3: Skin overlying the pedicle has been de-epithelialized. Next step is tissue dissection, isolating the pedicled nipple complex and mobilizing the tissue to be excised. (Courtesy: Rukmini S. Rednam and Bruce A. Mast)

Video 4: The mobilized tissue is removed, starting medially and continuing laterally. The pedicle and lateral breast pillar are not undercut. (Courtesy: Rukmini S. Rednam and Bruce A. Mast)

Video 5: Inset of nipple complex and approximation of the vertical incision line. (Courtesy: Rukmini S. Rednam and Bruce A. Mast)

Video 6: Approximation of the medial and lateral breast pillars, followed by inframammary incision line approximation. (Courtesy: Rukmini S. Rednam and Bruce A. Mast)

Video 7: Exposure of pully system in thumb and retrieval of severed flexor pollicis longus tendon. (Courtesy: Brian D. Rinker)

Video 8: Placement of suture in distal tendon segment. (Courtesy: Brian D. Rinker)

Video 9: Further core suture placement in tendon repair. (Courtesy: Brian D. Rinker)

Video 10: Tendon being approximated with tied-down sutures and demonstration of glide within tendon sheath. (Courtesy: Brian D. Rinker)

Video 11: After epitendinous repair. (Courtesy: Brian D. Rinker)

Video 12: Exposure and dissection of gracilis muscle for free tissue transfer. (Courtesy: Mark M. Leyngold)

Video 13: Full dissection of gracilis muscle for free tissue transfer, demonstrating vascular pedicle and surrounding structures. (Courtesy: Mark Leyngold)

Video 14: Random chest flap: soft reconstruction of the hand and upper extremity: Part I. (Courtesy: Wendy Lynne Czerwinski and Robert A. Weber)

Video 15: Random chest flap: soft reconstruction of the hand and upper extremity: Part II. (Courtesy: Wendy Lynne Czerwinski and Robert A. Weber)

Video 16: Rhinoplasty. (Courtesy: Geo N. Tabbal and Alan Matarasso)

Video 17: Animation deformity in breast reconstruction patients. (Courtesy: Bruce A. Mast)

Video 18: Dual plane breast augmentation. (Courtesy: Bruce A. Mast)

Video 19: Endoscopic browlift. (Courtesy: Bruce A. Mast)

Video 20: Breast reconstruction with latissimus dorsi myocutaneous flap. (Courtesy: Bruce A. Mast)

Video 21: CTR: Endoscopic carpal tunnel release. (Courtesy: Harvey Chim)

Video 22: CuTR: Endoscopic cubital tunnel release. (Courtesy: Harvey Chim)

Foreword

Our specialty, plastic surgery, is rapidly expanding and changing. The changes are nowhere more apparent and rapidly evolving than in our operating rooms. The long-established procedures are continually modified to enhance results and increase safety. New procedures are continually described and perfected. New devices, drugs, and instruments are regularly introduced and those already in use are refined.

The growth of the specialty in general and particularly the subspecialties has witnessed the publication of an ever-increasing number of textbooks, some addressing our entire specialty whilst others concentrate on specific subspecialties or even single procedures. So, with all the available texts, not to mention our many journals, why another text, why this book?

A quick and simple answer would be to say its new, it is up to date and contains the latest information. Yes, all of that is correct, but there is more to the book than being current. As the title claims, it is a Practical Guide to Operative Care. It is certainly full of practical information concentrating on operative care and that sets it apart.

Basic principles and regional anatomy are covered first, followed by the extensive, all-inclusive clinical operative care chapters. Each providing a clear description of the operations followed by review questions, their answers, and an extensive bibliography.

Dr. Mast, himself an accomplished educator, has selected other equally well recognized educators, each an expert in their field, to contribute. The result is a comprehensive text aimed not only at trainees and early career surgeons but an up to date refresher for all of us.

Foad Nahai, MD, FACS
Adjunct Professor of Plastic Surgery
The Center for Plastic Surgery at Metroderm
Emory University
Atlanta, Georgia, USA

Preface

Plastic surgery is a broad field, perhaps the broadest of all the surgical disciplines. It is not defined by an organ system, part of the body, or age group. Plastic surgery applies principles of surgical technique and applied anatomy to address conditions of appearance, form, and function that have been caused by disease, trauma, and time. By virtue of this approach, the surgeons who practice plastic surgery are defined by their innovation and creativeness in caring for their patients. The field is broad, almost boundless and takes time and effort to master.

This book is designed to facilitate the mastery of plastic surgery. It is specifically focused at those in the early part of their plastic surgery careers, from fledgling medical students, to eager and absorbing residents, to those in the early years of their practice in need of a refresher for a case they haven't seen since residency. It is not meant to be an all-inclusive encyclopedic treatise of all-things plastic surgery. Rather it is a practical guide to caring for patients with the most common conditions that a plastic surgeon would see in training or in early practice. The chapters are designed in the mode of a teaching conference, beginning with objectives, followed by patient presentations, preparation for surgery, treatment/operative techniques, postoperative care, and outcomes. Each chapter is completed with post-reading questions so that each reader can assure themselves they have absorbed key aspects of the topic.

My hope is for this book to serve as a steady resource that will meaningfully contribute to the education and training of those yearning to be a plastic surgeon, and plastic surgeons yearning to be the best at what they do.

Bruce A. Mast, MD, FACS

Contributors

Nasim Abedi, MD, FRCSC
Plastic Surgeon
Vancouver Coastal Health
University of British Columbia
North Vancouver, British Columbia, Canada

Paul N. Afrooz, MD
Aesthetic Surgery Fellow
Dallas Plastic Surgery
Dallas, Texas

Brendan Alleyne, MD
Resident
Plastic Surgery
Cleveland Clinic
Cleveland, Ohio

Richard Baynosa, MD
Associate Professor and Chairman
Department of Plastic Surgery;
Program Director
Complex Breast, Microsurgical and Robotic Reconstructive Surgery Fellowship;
Associate Program Director Plastic Surgery Residency
School of Medicine
University of Nevada, Las Vegas
Las Vegas, Nevada, USA

Michael L. Bentz, MD, FAAP, FACS
Professor of Surgery
Pediatrics and Neurosurgery
Chairman
Division of Plastic and Reconstructive Surgery
Vice Chairman of Clinical Affairs
Department of Surgery
University of Wisconsin School of Medicine and Public Health
Madison, Wisconsin

Steven L. Bernard, MD
Associate Professor of Surgery
Lerner College of Medicine
Case Western Reserve University
Residency Program Director
Department of Plastic Surgery
Cleveland Clinic
Cleveland, Ohio

Lisa M. Block, MD
Craniofacial Fellowship
University of Pittsburgh
Pittsburgh, Pennsylvania

Cristiano Boneti, MD, FACS
Director
Cancer Institute of America Medical Corporation
Sherman Oaks, California

Christopher J.M. Brooks, MD, FACS
Plastic Surgeon
Joe DiMaggio Children's Hospital
Hollywood, Florida

Patrick J. Buchanan, MD
Fellow
Department of Surgery
Section of Plastic and Reconstructive Surgery
University of Michigan Health System
University of Michigan
Ann Arbor, Michigan

Cagri Cakmakoglu, MD, FEBOPRAS
Clinical Research Fellow
Department of Plastic Surgery
Cleveland Clinic
Cleveland, Ohio

William J. Campbell, MD
Plastic Surgeon
The Georgia Institute for Plastic Surgery
Savannah, Georgia

Jessica A. Ching, MD
Assistant Professor
Division of Plastic and Reconstructive Surgery
University of Florida College of Medicine
Gainesville, Florida

Loretta Coady-Fariborzian, MD
Clinical Associate Professor
Department of Surgery
Division of Plastic and Reconstructive Surgery
University of Florida
Section Chief
Plastic, Reconstructive, and Hand Surgery
Malcom Randall VA Medical Center
Gainesville, Florida

Rachel Cohen-Shohet, MD
Resident
Division of Plastic & Reconstructive Surgery
Department of Surgery
University of Florida College of Medicine
Gainsville, Florida

Wendy Lynne Czerwinski, MD, PhD, FRCSC, FACS
Associate Professor
Division of Plastic Surgery
Baylor Scott & White Medical Center
Temple, Texas

Lynn A. Damitz, MD, FACS
Professor
Chief
UNC Division of Plastic & Reconstructive Surgery
Chapel Hill, North Carolina

Edward H. Davidson
Associate Professor
Department of Plastic and Reconstructive Surgery
Case Western Reserve University
Cleveland, Ohio

Jorge de la Torre, MD, MSHA
Professor and Chief
UAB Plastic Surgery
The University of Alabama at Birmingham
Birmingham, Alabama

Jose L.M. del Yerro, MD
Plastic Surgeon
Martin del Yerro Clinic
Madrid, Spain

Paul Diegidio, MD
Chief Resident
Division of Plastic and Reconstructive Surgery
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina

Richard L. Drake, PhD
Director of Anatomy
Professor of Surgery
Cleveland Clinic Lerner College of Medicine
Case Western Reserve University
Lerner Research Institute
Cleveland, Ohio

Gregory A. Dumanian, MD
Chief of Plastic Surgery
Department of Surgery
Feinberg School of Medicine
Northwestern Medicine
Chicago, Illinois

Eliana F.R. Duraes, MD, MSc PhD
Microsurgery Fellow
Cleveland Clnic
Cleveland, Ohio

Felmont F. Eaves, MD
Professor of Surgery
Division of Plastic and Reconstructive Surgery
Department of Surgery
Emory University School of Medicine
Atlanta, Georgia

Francesco M. Egro, MBChB, MSc, MRCS
Resident Physician
University of Pittsburgh Medical Center
Pittsburgh, Pennsylvania

Gregory R.D. Evans, MD, FACS
Professor
Plastic Surgery and Biomedical Engineering
Founding Chair
Department of Plastic Surgery
Past President American Society of Plastic Surgeons
The University of California, Irvine
Orange, California

Michael Friel, MD
Surgical Director
Ochsner Cleft Palate-Craniofacial Program
Ochsner Health Center for Children
New Orleans, Louisiana

Leonard T. Furlow, Jr., MD
Retired
Gainesville, Florida

Paul A. Ghareeb, MD
Chief Plastic Surgery Resident
Emory University School of Medicine
Atlanta, Georgia

Mark S. Granick, MD, FACS
Professor of Surgery
Chief, Division of Plastic Surgery
Rutgers New Jersey Medical School
Professor of Surgery
Rutgers Robert Wood Johnson Medical School
New Brunswick, New Jersey

Richard Gregory, MD
Private Practice
Celebration, Florida

Varun Gupta, MD
Fellow
Department of Plastic Surgery
Vanderbilt University Medical Center
Nashville, Tennessee

Raffi Gurunluoglu, MD, PhD, FACS
Professor of Plastic Surgery
Lerner College of Medicine
Case Western Reserve University
Department of Plastic Surgery
Cleveland Clinic
Cleveland, Ohio

Jeffrey A. Gusenoff, MD
Associate Professor
Department of Plastic Surgery
Co-Director
Life After Weight Loss Program
University of Pittsburgh
Pittsburgh, Pennsylvania

Alexandra M. Hart, MD
Resident
Division of Plastic and Reconstructive Surgery
Emory University
Atlanta, Georgia

Ahmed M. Hashem, MD
Professor of Plastic Surgery
Department of General Surgery
Faculty of Medicine
Cairo University
Cairo, Egypt

Daniel F. Haynes, MD, BS
Professor of Surgery
Chief, Division of Plastic Surgery
James H. Quillen College of Medicine
East Tennessee State University
Johnson City, Tennessee

Steven J. Hermiz, MD
Resident
Plastic Surgery
Medical University of South Carolina
Charleston, South Carolina

Kent K. Higdon, MD, FACS
Associate Program Director
Assistant Professor
Department of Plastic Surgery
Vanderbilt University Medical Center
Nashville, Tennessee

C. Scott Hultman, MD, MBA, FACS
Ethel and James Valone Distinguished Professor of Surgery
Chief and Program Director
Plastic Surgery Vice Chair for Finance
Department of Surgery Founder and Executive Director
UNC Burn Reconstruction
University of North Carolina
Aesthetic Center Associate Director
NC Jaycee Burn Center
Associate Editor
Annals of Plastic Surgery
Chapel Hill, North Carolina

Sonu A. Jain, MD, FACS
Assistant Professor of Plastic Surgery and Orthopedic Surgery
Departments of Plastic and Reconstructive Surgery and Orthopaedic Surgery
Hand and Upper Extremity Center
Chief
Division of Hand Surgery
Department of Plastic and Reconstructive Surgery
The Ohio State University Wexner Medical Center
Columbus, Ohio

Adam J. Katz, MD, FACS
Professor
Director of Plastic Surgery Research
Laboratory of Bio Innovation and Translational Therapeutics
Director of Clinical Trials
Division of Plastic and Reconstructive Surgery
Department of Surgery
University of Florida College of Medicine
Gainesville, Florida

Jonathan Keith, MD
Clinical Assistant Professor
Assistant Residency Program Director
Division of Plastic Surgery
Rutgers New Jersey Medical School
New Brunswick, New Jersey

Timothy W. King, MD, PHD, MSBE, FAAP, FACS
Associate Professor
Surgery and Biomedical Engineering
Director of Plastic Surgery Research
University of Alabama at Birmingham Children's of Alabama
Birmingham, Alabama

Emil J. Kohan, MD
Private Practice
Beverly Hills, California

Stephanie L. Koonce, MD
Assistant Professor ARCOM
Plastic and Reconstructive Surgery
Mercy Clinic
Fort Smith, Arizona

Nishant Ganesh Kumar, MD
Plastic Surgery Resident
Department of Surgery
Michigan Medicine
University of Michigan
Ann Arbor, Michigan

John Layliev, MD
Resident
Department of Plastic Surgery
Vanderbilt University Medical Center
Nashville, Tennessee

Ashley K. Lentz, MD, MS
Assistant Professor of Surgery
Division of Plastic and Reconstructive Surgery
University of Florida
Gainesville, Florida

Mark M. Leyngold, MD
Assistant Professor
Division of Plastic and Reconstructive Surgery
Department of Surgery
University of Florida College of Medicine
Gainesville, Florida

William C. Lineaweaver, MD
Plastic Surgeon
Joseph M. Still Burn Center
Jackson, Missouri

Albert Losken, MD, FACS
William G Hamm Professor of Plastic Surgery
Program Director
Emory Division of Plastic and Reconstructive Surgery
Atlanta, Georgia

Yee Cheng Low, MD
Plastic Surgeon
LVPG Plastic and Reconstructive
Surgery-1243 Cedar Crest
Allentown, Pennsylvania

Edward A. Luce, MD
Professor of Plastic Surgery
University of Tennessee Health Science Center
Memphis, Tennessee

Maryann E. Martinovic, MD
Plastic Surgeon
Mercy Clinic and Reconstructive Surgery-Coletta
Oklahoma City, Oklahoma

Alan Matarasso, MD
Private Practice
New York, New York

Chris McCarthy, MD
Orthopaedic Hand and Upper Extremity Surgeon
Hand and Microsurgery Associates
Columbus, Ohio

Sushmita Mittal, MD
Resident
Department of Surgery
Division of Plastic Surgery
University of Florida
Gainesville, Florida

R. Brad Nesmith
Plastic Surgery Resident
University of Florida
Gainesville, Florida

Lauren C. Nigro, MD
Resident Physician
Division of Plastic Surgery
Virginia Commonwealth University Health System
Richmond, Virginia

Galen Perdikis, MD, FACS
Chair and Professor of Surgery
Department of Plastic Surgery
Vanderbilt University Medical Center
Brentwood, Tennessee

Luke Pearson, BS
Department of Surgery
Division of Plastic Surgery
University of Florida
Gainesville, Florida

John H. Phillips, MD, MA, FRCS(C)
Associate Professor
Division of Plastic & Reconstructive Surgery
University of Toronto
Toronto, Ontario, Canada

Andrea L. Pozez, MD
Professor and Chair
Division of Plastic and Reconstructive Surgery
Virginia Commonwealth University Medical Center
Richmond, Virginia

Noah H. Prince, MD
Private Practice
Doylestown, Pennsylvania

Rukmini Rednam
Aesthetic Fellow
University of Florida
Gainesville, Florida

Winston Richards
Assistant Professor of Surgery
Department of Surgery
University of South Florida
Tampa, Florida

Brandon K. Richland, MD
Private Practice
Newport Beach, California

Brian D. Rinker, MD
Chair
Division of Plastic Surgery
Mayo Clinic Hospital
Jacksonville, Florida

J. Peter Rubin, MD
Professor and Chair
Department of Plastic Surgery
Professor of Bioengineering
University of Pittsburgh
Pittsburgh, Pennsylvania

Mario Rueda, MD, FACS
Director of Intensive Care Unit
St. Mary's Medical Center
West Palm Beach, Florida
Assistant Professor of Surgery
Florida Atlantic University
Boca Raton, Florida

Steven Rueda, MD
Hand Surgery Fellow
Ohio State University
Columbus, Ohio

Ruston Sanchez, MD
Plastic Surgery Resident
Department of Surgery
University of Wisconsin School of Medicine
and Public Health
Madison, Wisconsin

A. Neil Salyapongse, MD
Professor
Division of Plastic Surgery
Department of Surgery
University of Wisconsin School of Medicine
and Public Health
Madison, Wisconsin

Kenneth C. Shestak, MD
Professor
Department of Plastic Surgery
Head of Faculty Development
Executive Director of the PITT Plastic
Surgery Alumni Association
Department of Plastic Surgery
University of Pittsburgh School of Medicine
Chief of Plastic Surgery
UPMC Magee-Womens Hospital
Pittsburgh, Pennsylvania

Han Shi, BA
Resident
Department of Plastic Surgery
Vanderbilt University Medical Center
Nashville, Tennessee

Andrew G. Silver, MD
Private Practice
Las Vegas, Nevada

Dhruv Singhal, MD
Assistant Professor
Department of Oral and Maxillofacial Surgery
Department of Surgery
Division of Plastic Surgery
University of Florida
Gainesville, Florida

Aditya Sood, MD, MBA
Private Practice Plastic Surgeon
Chicago, Illinois

J. Blair Summitt, MD
Medical Director
Acute Burn Services
Assistant Professor
Department of Plastic Surgery
Vanderbilt University Medical Center
Nashville, Tennessee

Stephanie Suprenant, MD, MPH
Private Practice
Missoula, Montana

Geo N. Tabbal, MD
Private Practice
Dallas, Texas

Henry C. Vasconez, MD, FACS
Director
Burn Clinic
University of Kentucky
Lexington, Kentucky

Manuel R. Vegas, MD, PhD, Fellow of EBOPRAS, Fellow of EBHS
Chief of Microsurgery Section
Plastic Surgery Service
Hospital Universitario Ramon y Cajal
Madrid, Spain

Krishna S. Vyas, MD
Resident
Plastic and Reconstructive Surgery
Mayo Clinic
Rochester, Minnesota

Rachel I. Mason Weber, MD
Private Practice
Hadar, Nebraska

Russell Walther, DDS, MD
Resident
Department of Oral and Maxillofacial Surgery
University of Florida
Gainesville, Florida

Robert A. Weber, MD
Professor and Vice Chair of Surgery
Section of Hand Surgery
Division of Plastic Surgery
Baylor Scott & White Medical Center
Temple, Texas

Julian Winocour, MD
Assistant Professor
Department of Plastic Surgery
Vanderbilt University Medical Center
Nashville, Tennessee

Zachary T. Young, MD
Plastic Surgery Fellow
Medical University of South Carolina
Charleston, South Carolina

Shuhao Zhang, MD
Surgeon
Cosmetic, Plastic & Reconstructive Surgery
Mercy Medical Group
Sacramento, California

James E. Zins, MD
Chairman
Department of Plastic Surgery
Cleveland Clinic
Cleveland, Ohio

Part I

Science and Principles

1 Wound Healing: Science and Clinical Relevance

Aditya Sood, Jonathan Keith, and Mark S. Granick

Abstract

This chapter examines the complicated and intricate process of wound healing, showing that the several phases of healing and wound types require a thorough understanding by plastic surgeons and practitioners in order to yield the most satisfactory outcomes. Wounds may move from an acute phase to the chronic type, requiring the physician to intervene in order to return these wounds back to a favorable phase of the healing cascade. With a multitude of surgical and nonsurgical options for treating wounds, the correct intervention may seem to be an overwhelming or daunting task. Accordingly, the authors stress the importance of education on wound healing pathophysiology and the available surgical and nonsurgical options. Each wound must be carefully evaluated, categorized, and finally treated.

Keywords: cutaneous wound, growth factors, inflammation, proliferation, remodeling, acute, chronic, debridement

1.1 Goals and Objectives

- Understand the wound healing pathophysiology.
- Clearly define the characteristics of acute versus chronic wounds.
- Appreciate the technical aspects of managing different types of wounds.
- Know the evidence-based data on wound healing adjuncts.

1.2 Introduction

1.2.1 Overview

Wound healing is a complicated and intricate process, with several phases of healing and wound types, requiring a thorough understanding by plastic surgeons and practitioners alike to yield the most satisfactory outcomes. Wounds may move from an acute phase to the chronic type, requiring the physician to intervene in order to bring these wounds back into a favorable phase of the healing cascade. With a multitude of surgical and nonsurgical options for treating wounds, the correct intervention may seem to be an overwhelming or daunting task. The authors stress the importance of education on wound healing pathophysiology and the available surgical and nonsurgical options. Each wound must be carefully evaluated, categorized, and finally treated.

1.2.2 Clinical Challenge

Injury to the skin provides a unique challenge to clinicians, as wound healing is a complex and intricate process. With more than 6.5 million chronic skin ulcers caused by pressure, venous stasis, or diabetes mellitus, it is no wonder why cutaneous wound healing has become a topic of ongoing research and debate worldwide.[1] There are a multitude of options when considering adjuncts or primary treatment of these wounds. Cost-effectiveness is an important variable in treating these wounds, as chronic wounds account for an estimated $6 to $15 billion annually in U.S. health care costs.[2] There is tremendous pressure on the medical system and clinicians to develop cost-effective therapies.

There are a multitude of products available for the treatment of chronic wounds beyond the standard surgical debridement. Examples of these products include negative pressure wound therapy, hyperbaric oxygen, biologic dressings, skin substitutes, as well as growth factors and regenerative materials. The overwhelming amount of wound dressings and adjuncts available in the market implies a lack of full understanding of wound care and management.[3] The use of many of these adjuncts or materials is to improve upon specific wound characteristics to bring it as close to "ideal" as possible. It is vital for the clinician to have a thorough understanding of wound healing pathophysiology, wound characteristics, as well as the importance of surgical intervention before applying any of the advanced therapies.

1.2.3 Growth Factors, Cytokines, and Chemokines

Stanley Cohen's experiment in which he noticed that the purification of submaxillary gland extracts led to earlier eyelid separation and eruption of the incisor in mice, eventually leading to the isolation of the first growth factor, epidermal growth factor (EGF), and the 1986 Nobel prize in Medicine.[4] It was after this discovery that knowledge of growth factors has increased rapidly. In only the past few decades, the discovery of growth factors has led to tremendous hope and speculation in regard to their use in the treatment of wounds, particularly difficult-to-heal or chronic wounds. The importance of growth factors, cytokines, and chemokines in regulating the complex process of wound healing has been studied extensively.[5] In vitro experiments have shown that growth factors are very effective in regulating cell proliferation, chemotaxis, and extracellular matrix (ECM) formation. Of particular importance is the EGF family, transforming growth factor-beta (TGF-β) family, fibroblast growth factor (FGF) family, vascular endothelial growth factor (VEGF) family, granulocyte macrophage colony-stimulating factor (GM-CSF), platelet-derived growth factor (PDGF), human growth hormone, and the interleukin (IL) family. It was not until further advances in recombinant technology became available that large amounts of purified growth factors could be obtained and tested in human clinical trials. Since this time, a large number of trials have been performed to evaluate the safety and effectiveness of growth factors in the healing of chronic wounds, including pressure, diabetic, and venous insufficiency–related wounds.

Currently, growth factors have a limited role in clinical practice. In the United States, recombinant human (rh) PDGF-BB (Regranex; Ortho-McNeil-Janssen Pharmaceuticals, Inc., Raritan, NJ) is the only growth factor approved by the U.S. Food and Drug Administration for use in chronic wounds. It has successfully

completed randomized clinical trials in the United States and is available as a commercial product. In this brief discussion, we will review wound-healing mechanics and considerations in treatment, and mention different modalities for treatment. The discussion will be ended with a perspective on the future of growth factors in chronic wounds.

1.3 Wound Healing Pathophysiology

Wound healing involves a complex set of simultaneous processes including activation of the inflammatory cascade, vascular neogenesis, and cell proliferation. It is a set of interactions that is still poorly understood.

With regard to cutaneous wound healing, scarring is the body's attempt at reestablishing homeostasis and preventing infection. The tissue laid down in scarring is inherently different than the skin that it replaces. Therefore, scarring is different from wound healing of other organs, such as the liver or bone, or that which occurs in fetal wound healing, which heals by regeneration and re-creation of the same previously existing tissue. It is critical to understand the differences in these healing processes, as well as the progression of each, so that we can determine where we may intervene clinically to alter the body's response.

There are three phases of wound healing that are generally cited: inflammation, proliferation, and remodeling phases (▶ Fig. 1.1). While devitalized tissue is removed during the inflammatory phase, new tissue is laid down in the proliferative phase, and finally the structure of the scar is optimized during the remodeling phase. The primary goals of the inflammatory phase are ridding the newly created wound of bacteria and dead tissue. At the same time, hemostasis is achieved. When the wound is initially created, there are several processes occurring at the same time including creation of a provisional matrix, release of cells and growth factors from ongoing hemorrhage, and initiation of the clotting cascade. These processes are interactive: as platelets clump to achieve hemostasis, they also release PDGF and TGF-β). Exposed collagen and tissue factor from the initial injury begin the clotting cascade, which ends in the formation of a fibrin-based matrix. Inflammatory cells and growth factors attach to this scaffold to create the proper milieu for subsequent wound healing.

Inflammatory cells are attracted to the wound by TGF-β from platelets, lipopolysaccharide from bacteria, and elements of the clotting cascade.[6] Neutrophils permeate the wound in the first 48 hours to remove dead tissue and destroy bacteria. Monocytes enter the wound at 48 to 72 hours and become phagocytosing macrophages. Interestingly, wound healing cannot progress without the presence of macrophages, but it is able to progress without neutrophils.[7,8] Lymphocytes enter the wound at days 5 to 7. Their role is not well defined, but they may initially play a role in the proliferative phase of wound healing.[9]

During the proliferative phase of wound healing, re-epithelialization and granulation tissue formation occur. This generally occurs from days 4 to 21 after injury. The provisional matrix that was formed in the inflammatory phase is replaced by ECM with type III collagen and blood vessels of new granulation tissue. Macrophages release cytokines that stimulate fibroblasts to lay down this new ECM and endothelial cells form these new blood vessels. Eventually, as the proliferative phase comes to an end, the fibroblasts and blood vessels regress, although we require further research into the regulation of this process.

The remodeling phase is characterized by the replacement of type III collagen with type I collagen, as well as wound contraction by myofibroblasts. The wound strength changes over the course of the remodeling phase. Wounds have only 20% strength of normal skin at 3 weeks after injury, and will reach 70 to 80% strength of normal skin at 1 year.

1.4 Acute versus Chronic Wounds

Wounds that fail to progress through the normal stages of healing become chronic wounds. An acute wound is one that is present up to 3 weeks, while a chronic wound is one that has been present for greater than 2 to 3 weeks. These chronic wounds remain a significant challenge and burden on U.S. healthcare. Chronic wounds including venous stasis ulcers,

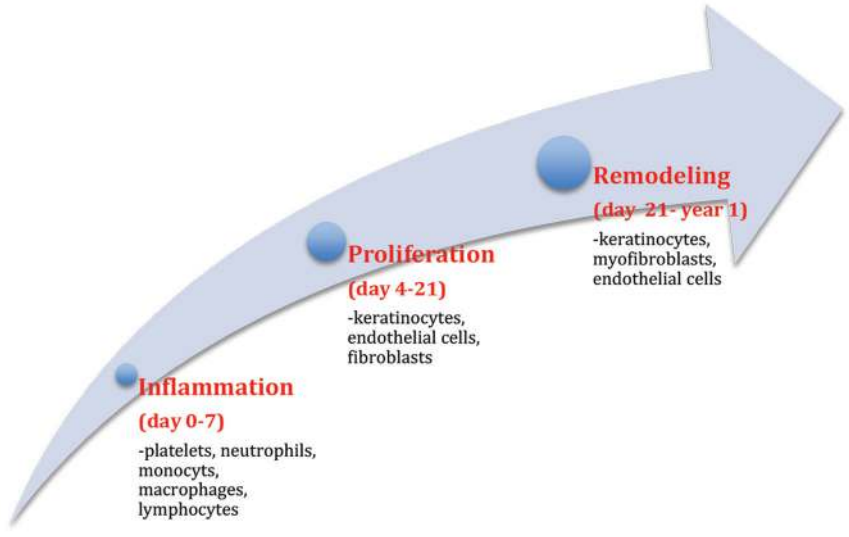


Fig. 1.1 Phases of wound healing. Note the overlapping timeframe of these phases, as well as important cell types.

decubitus ulcers, and diabetic foot ulcers affect over 6 million people in the United States. Despite treatment, the recurrence of these wounds is high and costs the healthcare system billions of dollars each year.[10]

Before determining treatment for a chronic wound, it is important to elucidate the history of the wound and contributing risk factors. Factors that delay wound healing include infection, ischemia, smoking, necrotic tissues, venous stasis, edema, diabetes, and steroids.

1.5 Factors Affecting Wound Healing

Wounds are more susceptible to healing in a moist, clean, and warm environment. A moist wound bed will allow growth factors and various cell types to migrate, facilitating wound edge contraction. In order to create and maintain this environment, appropriate dressings come into play as outlined in the following section.

Bacterial contamination of wounds can prevent the normal phases of wound healing from taking place by virtue of effects on inflammation or by secretion of active proteases. For example, platelet aggregation and hemostasis can be affected by bacteria.[11] Leukocyte function is impaired by bacterial virulence factors and length of inflammation is prolonged.[12] The formation of granulation tissue may be impaired by the presence of bacteria.[13] Not all bacterial species are equal in their ability to retard wound healing: beta-hemolytic streptococcus can produce wound healing complications at lower levels than other organisms.[11]

With regard to ischemia, in vitro studies have demonstrated that neutrophils and fibroblasts do not function appropriately at low oxygen levels.[14,15] Clinically, low subcutaneous oxygen tension of a wound has been shown to be a predictor of surgical wound infections.[16] Therefore, it is critical to optimize bloody supply, especially if the upper or lower extremity is affected. Revascularization should be considered prior to any advanced surgical procedures, although a debridement may be necessary earlier. Similarly, venous stasis ulcers should be accompanied by compression therapy along with debridement to optimize the outcome. Interestingly, although tissue perfusion appears to be critical to wound healing, low hemoglobin levels are not the normal physiologic responses to anemia tend to restore normal oxygen delivery despite low hemoglobin levels.[17,18]

Among the components of cigarettes that can impair wound healing are nicotine, carbon monoxide, and hydrogen cyanide.[19] In addition to being a vasoconstrictor, nicotine increases platelet aggregation, and impairs the proliferation of red blood cells, fibroblasts, and macrophages. Carbon monoxide impairs oxygen transport and metabolism, while cyanide inhibits oxidative metabolism. The complications of wound healing in plastic surgery caused by smoking are well documented in patients having facelifts, breast reduction, breast reconstruction, and abdominoplasty.[20,21,22] Despite the clear benefits of smoking cessation in surgical patients, the time period for cessation is unclear. Some have recommended smoking cessation 4 weeks prior to surgery, and 4 weeks after surgery.[23]

Diabetes can affect wound healing at both the macro and micro levels. Diabetes is known to cause vascular disease impairing blood flow and oxygen delivery to tissues.[24] Hyperglycemia results in protein and enzymatic dysfunction leading to inflammation.[25] In addition, hyperglycemia results in alteration of basement membrane permeability. Again, recommendations for perioperative glycemic control are unclear, but those patients with levels greater than 200 mg/dL generally have worse outcomes.[26]

Steroids have been shown to impair epithelialization, collagen formation, and the inflammatory process. The effect on wound healing is likely clinically apparent only when taken chronically within 30 days of surgery; steroids taken in the acute phase likely have little effect on wound healing.[27] In contrast, although chemotherapeutic drugs would logically impair wound healing based on their ability to impair nucleic acid and protein synthesis, as well as cell division, clinical studies have not borne this out.[28] Radiation causes DNA and protein damage, vascular stasis and thrombosis, impaired fibroblast activity, and in addition may cause clinically significant changes including slower epithelialization, decreased tensile strength, and higher dehiscence rates.[29,30,31]

Examination of chronic wounds should include depth of the ulcer, size, presence of cellulitis, and other signs of infection. Measurements should be performed weekly to determine healing progress. Decrease in wound size of 10 to 15% per week indicates potential for normal healing. Gentle probing may be utilized to determine if bone is present at the base of the wound. If bone is present, there is a high likelihood of osteomyelitis. A thorough neurovascular exam should be documented including pulses, Doppler examination, and sensation using Semmes-Weinstein filaments.[10]

1.6 Surgical Intervention and Debridement

After appropriate assessment of a wound, it may be deemed appropriate for a variety of debridement options: autolytic, enzymatic, mechanical, or even larval therapy. Nonetheless, surgical, or mechanical, debridement remains a mainstay in the treatment of chronic or hard to treat wounds (▶ Fig. 1.2). Through wound bed preparation, even complicated wounds may effectively turn into acute wounds and proper cellular responses induced to allow healing. If surgical intervention is to be performed in an attempt to convert a chronic wound to an acute one, nonviable tissue should be debrided thoroughly until healthy, bleeding tissue is encountered. Multiple debridements may be necessary if the extent of nonviable tissue is not clear initially. The use of hydrosurgery devices has advanced the surgical management of wounds, as it has become an option that can control the depth of tissue removal more precisely, and therefore spare more healthy tissue while still achieving adequate debridement.[32] Debridement of skin should be continued until pinpoint-bleeding tissue that blanches is encountered. Healthy fat will appear soft and shiny, while fascia and tendon that is healthy will appear shiny and taut. Healthy bone will be firm with cortical bleeding.[10] Tissue cultures should be sent for any debrided tissue.

Fig. 1.2 Surgical, or mechanical, debridement of a chronic knee wound in order to remove nonviable tissues (eschar).

Fig. 1.3 **(a)** A chronic open wound on the thigh with excessive exudates. **(b)** The wound is covered with a hydrofiber dressing to absorb exudates and optimize the healing environment.

1.7 Wound Dressings and Healing Adjuncts

As discussed previously, wounds are more prone to healing in a moist, clean, and warm environment. There are four basic principles involved when choosing an optimal dressing type. If a wound is dry or desiccated, it will need hydration. If a wound produces excessive exudates, the fluid needs to be absorbed or removed (▶ Fig. 1.3). If a wound has necrotic tissue or evident debris, it will need a debridement. Lastly, if a wound is infected, it will need to be treated with the appropriate antibacterial agent. Other factors also important in choosing a dressing include providing protection to the peri-wound skin, forming an effective bacterial barrier, conforming to wound shape, producing minimal pain during application and removal, being free of toxic or irritant extractables, not releasing particles or non-biodegradable fibers into the wound, and maintaining the wound at an optimal temperature and pH. Each wound needs a proper assessment of its characteristics and these needs can be met with a corresponding dressing that fits the situation (Box 1.1). It is important to remember that wound environments change and dressings should be changed accordingly.

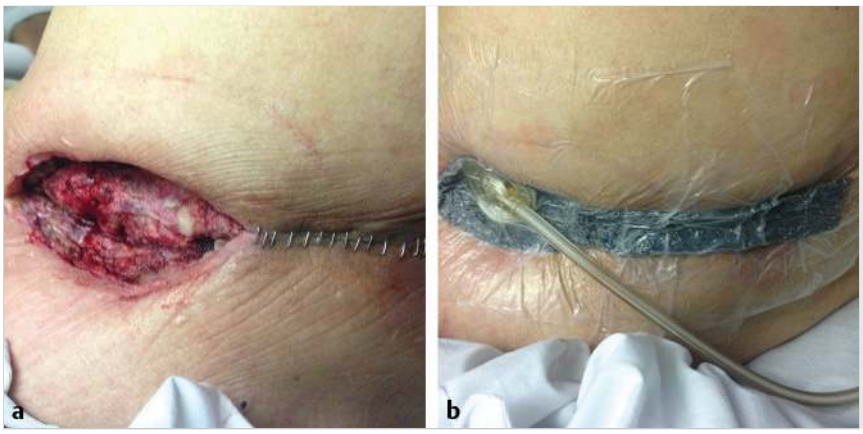


Fig. 1.4 **(a)** A chronic ischial wound after surgical debridement; note exposed periosteum. **(b)** After placement of negative-pressure wound therapy to induce granulation prior to further surgical attempts for coverage.

Box 1.1 Characteristics of an Ideal Dressing

- Creates a moist, clean, warm environment.
- Provides hydration if dry or desiccated.
- Removes excess exudates.
- Prevents desiccation and is nontraumatic.
- Provides protection to the peri-wound area.
- Allows for gaseous exchange.
- Impermeable to microorganisms.
- Free of toxic or irritant particles.
- Can conform to wound shape.
- Minimal pain during application and removal.
- Easy to use.
- Cost-effective.

There are various adjuncts in the form of wound dressings or advanced wound dressings that are available in the market to aid in human healing. Negative pressure wound therapy, for example, has undoubtedly revolutionized wound management and proven beneficial for a variety of wounds (▶ Fig. 1.4). Other modalities, such as hyperbaric oxygen, biologic dressings, skin substitutes, and regenerative materials, have also proven efficacious in advancing the wound healing process through a variety of mechanisms. Due to the sheer number of wound dressings available on the market, it can be implied that there is no easy way to get chronic wounds to heal. Few high-quality, randomized controlled trials evaluating wound dressings exist and do not clearly demonstrate superiority of many materials or categories over one another.[3] Nonetheless, medical and physiologic optimization of patients and surgical means of treating wounds in the form of debridement, revascularization, or a multitude of grafting techniques continues to be the mainstay of chronic wound treatment in the current day and age.

1.8 General Concepts of Growth Factor Therapy

Wound healing has been well described in the literature with regard to its division into three distinct phases: inflammation, proliferation, and maturation. Within these three general phases occurs a series of complex cellular and molecular events with a great degree of overlap and interdependence.[33] Growth factors are fundamental in this process and have been well studied over the past several decades.[5] These factors affect selected target cells or tissues in various ways, including stimulating chemotaxis and cellular proliferation, cellular activation and cytokine production, affecting cellular migration, controlling ECM formation, angiogenesis, regulating wound contraction, and also in reestablishing tissue integrity. Specific details of growth factors, including origin, selected targets, and actions may be found in a multitude of published materials and will not be discussed in this chapter.

Growth factors are involved early in the wound healing process and are released as soon as blood vessels are injured or disrupted. As platelets enter a wound, several growth factors are released including PDGF as well as TGF-β1. These growth factors, among others, are important in chemotaxis for numerous cell types critical for the wound reparative process, including neutrophils, macrophages, fibroblasts, and endothelial cells. In the next phase of wound healing, proliferation, multiple growth factors including VEGF, FGF, PDGF, and certain TGF-β isoforms promote robust angiogenesis and the synthesis of key extracellular components via fibroblasts. These extracellular components include fibronectin, elastin, proteoglycans, and collagen. Growth factors are also involved in the late stages of wound healing, or remodeling, along with various matrix-degrading metalloproteinases (MMPs). It is unlikely that growth factor influence on tissue homeostasis ends after tissue remodeling; they have also been postulated to be important in the maintenance of tissue integrity as well as cell-to-cell communications in the postoperative period.

Growth factors for wounds may be applied locally (or topically). Some come in the form of gels that are applied directly to the wound, such as rhPGDG. Growth factors can also be incorporated into wound dressings or commercially available skin grafts. In certain cases, growth factors may also be injected, an example being palifermin, a type of endothelial/keratinocyte growth factor, indicated for the prevention of oral mucositis in patients receiving radiation therapy.

It is important to note that the current nomenclature of growth factors is not altogether intuitive. The names of various factors do not necessarily have to do with their specific effects but rather the circumstances in which they were identified. For example, FGFs are very potent angiogenic factors, and PDGFs are actually derived from various cell types including platelets, macrophages, neutrophils, as well as smooth muscle cells. The term "growth factor" is also used as an umbrella term to indicate various substances that increase cell proliferation,

mitogenic activity, and ECM formation. The actual category, whether growth factor, IL, or colony-stimulating factor, generally depends on whether it was identified by a biochemist, an immunologist, or a hematologist, respectively.

1.9 The Future of Wound Healing

Substantial progress has been made with regard to the use of growth factors and treatment of chronic wounds. There have been no serious safety issues that have arisen, as systemic absorption appears to be minimal and no negative local effects have been reported. There have been no reports of cancer at the site of application, fibrosis, or worsening of diabetic retinopathy. It is undisputable that growth factors are vital for the normal wound healing process. Although many of them have been proposed for exogenous administration to acute or chronic wounds, only one (PDGF) is currently used to treat both chronic and acute wounds and burns, and that too with limited success. At this point in time, much still remains to be done in regard to the use of growth factors and wound healing. Delivery system of these peptides used in the clinical trials described may not have allowed sufficient peptides to reach the target cells and tissues. It is well known that the microenvironment of chronic wounds can be very hostile, and breakdown of these peptides by residing proteases is very likely. The success of PDGF in diabetic ulcers as shown in several trials may be secondary to the fact that the biologic activity of these peptides may persist in the wound microenvironment.[34] Another important consideration in the use of these topical growth factors is the state of the resident cells in a chronic wound environment. There is evidence that fibroblasts from chronic wounds are not able to respond to certain growth factors.[35] Removal of tissues, via debridement, has been advocated for the use of PDGF in diabetic ulcers, and may help remove these unresponsive cells and allow the peptides to function as they "normally" would.[36]

As the role and delivery of growth factor therapy continues to develop, several other modalities have been described. Gene therapy may prove to be ideal for wounds, as peptides would be viable only for a short period of time in many of these wounds. Another development that has pushed the envelope for delivery is the use of bioengineered skin products and skin substitutes. Some of these materials supply matrix materials alone, while others contain living cells that may adjust to the wound microenvironment and provide growth factors and other substances that may be lacking in the chronic wounds.[37,38] It is unclear how long the transplanted cells survive in the wound, but it is postulated that they may remain there long enough to stimulate and accelerate wound healing. Bioengineered products may help provide growth factors in the right concentration and sequence. This has been difficult to achieve with topical application of recombinant growth factors. In the future, these bioengineered skin products will be engineered to deliver certain growth factors in large quantities and in a timely manner in order to render growth factor therapy more effective.

1.10 Conclusion

Wound healing in any tissue of the human body undergoes a predictable sequence of events: inflammation, proliferation, and wound remodeling. A thorough understanding of wound healing at the cellular level is of vital importance. Acute wounds have the potential to move from the acute wound to chronic wounds, requiring the clinician to be knowledgeable of outside interventions to bring these wounds back into the healing cascade. Wound characteristics, such as edema, exudates, bacterial burden, nonviable tissue, will dictate the intervention and/or debridement. Each wound must be carefully evaluated, categorized, and eventually treated. The importance of identifying the need for debridement, whether surgical or nonsurgical, cannot be overstated. There is an overwhelming number of wound healing adjuncts and interactive dressings on the market, implying a lack of full understanding in treating these wounds. Also, despite the many advances in understanding the science of wound healing and the potential role of supplementing growth factors, there are many more steps that have yet to be discovered or elucidated. It is important for clinicians to have a broad understanding of the sequence of events, cells involved, and important mediators which may allow for healing optimization. Although growth factors may seem basic in concept, advances in molecular science have allowed modern-day clinicians to appreciate the complex interplay between the many cells involved in the different phases of wound healing. As greater understanding of the growth factors in wound healing emerges, including delivery vehicles and optimal time of delivery, future patient care may involve more direct or timely care for wounds and an overall positive impact on healthcare costs associated with this process. Until further data solidify the use of growth factors for treatment of difficult wounds, clinicians must not overlook surgical options as they have and always will remain an important modality for chronic wounds.

1.11 Review Questions

1.11.1 True or False

1. The three phases of wound healing in order are proliferation, inflammation, remodeling.
2. The remodeling phase of wound healing is characterized by the replacement of type III collagen with type I collagen.
3. In addition to being a vasoconstrictor, nicotine increases platelet aggregation, and impairs the proliferation of red blood cells, fibroblasts, and macrophages.

1.11.2 Choose the Best Answer

4. Lymphocytes begin to permeate an acute wound during the normal wound-healing cascade during which period?
   a) Days 1–3.
   b) Days 3–5.
   c) Days 5–7.
   d) Weeks 3–5.
5. After appropriate assessment of a wound, it may be deemed appropriate for a variety of debridement options, including
   a) Autolytic.
   b) Enzymatic.
   c) Mechanical.
   d) Larval.
   e) All of the above.

1.11.3 Answers

1. False.
2. True.
3. True.
4. c. Days 5–7.
5. e. All of the above.

References

[1] Medical Data International, Inc. U.S. Markets for Wound Management Products. Irvine, CA: Medical Data International; 1997

[2] Markova A, Mostow EN. US skin disease assessment: ulcer and wound care. Dermatol Clin. 30(1):107–111, ix

[3] Sood A, Granick MS, Tomaselli NL. Wound dressings and comparative effectiveness data. Adv Wound Care.

[4] Cohen S. Isolation of a mouse submaxillary gland protein accelerating incisor eruption and eyelid opening in the new-born animal. J Biol Chem. 237:1555–1562

[5] Ueno C, Hunt TK, Hopf HW. Using physiology to improve surgical wound outcomes. Plast Reconstr Surg. 117(7) Suppl:59S–71S

[6] Martin P, Leibovich SJ. Inflammatory cells during wound repair: the good, the bad and the ugly. Trends Cell Biol. 15(11):599–607

[7] Leibovich SJ, Ross R. The role of the macrophage in wound repair. A study with hydrocortisone and antimacrophage serum. Am J Pathol. 78(1):71–100

[8] Simpson DM, Ross R. The neutrophilic leukocyte in wound repair a study with antineutrophil serum. J Clin Invest. 51(8):2009–2023

[9] Park JE, Barbul A. Understanding the role of immune regulation in wound healing. Am J Surg. 187 5A:11S–16S

[10] Wei C, Granick MS. Surgical management of chronic wounds T. Wounds. 20 (3):62–66

[11] Robson MC, Stenberg BD, Heggers JP. Wound healing alterations caused by infection. Clin Plast Surg. 17(3):485–492

[12] Janis JE, Harrison B. Wound healing: part I. Basic science. Plast Reconstr Surg. 133(2):199e–207e

[13] Laato M, Lehtonen OP, Niinikoski J. Granulation tissue formation in experimental wounds inoculated with Staphylococcus aureus. Acta Chir Scand. 151 (4):313–318

[14] Hohn DC, MacKay RD, Halliday B, Hunt TK. Effect of O2 tension on microbicidal function of leukocytes in wounds and in vitro. Surg Forum. 27(62):18–20

[15] Juva K, Prockop DJ, Cooper GW, Lash JW. Hydroxylation of proline and the intracellular accumulation of a polypeptide precursor of collagen. Science. 152(3718):92–94

[16] Hopf HW, Hunt TK, West JM, et al. Wound tissue oxygen tension predicts the risk of wound infection in surgical patients. Arch Surg. 132(9):997–1004, discussion 1005

[17] Jonsson K, Jensen JA, Goodson WH , III, et al. Tissue oxygenation, anemia, and perfusion in relation to wound healing in surgical patients. Ann Surg. 214(5): 605–613

[18] Heughan C, Grislis G, Hunt TK. The effect of anemia on wound healing. Ann Surg. 179(2):163–167

[19] Silverstein P. Smoking and wound healing. Am J Med. 93 1A:22S–24S

[20] Bartsch RH, Weiss G, Kästenbauer T, et al. Crucial aspects of smoking in wound healing after breast reduction surgery. J Plast Reconstr Aesthet Surg. 60(9):1045–1049

[21] Knobloch K, Gohritz A, Reuss E, et al. Nicotine in plastic surgery: a review. Der Chirurg; Zeitschrift fur alle Gebiete der operativen Medizen; 2008;79 (10): 956–962

[22] Manassa EH, Hertl CH, Olbrisch RR. Wound healing problems in smokers and nonsmokers after 132 abdominoplasties. Plast Reconstr Surg. 111(6):2082–2087, discussion 2088–2089

[23] Krueger JK, Rohrich RJ. Clearing the smoke: the scientific rationale for tobacco abstention with plastic surgery. Plast Reconstr Surg. 108(4):1063–1073, discussion 1074–1077

[24] Greenhalgh DG. Wound healing and diabetes mellitus. Clin Plast Surg. 30(1): 37–45

[25] Gasser A, Forbes JM. Advanced glycation: implications in tissue damage and disease. Protein Pept Lett. 15(4):385–391

[26] Coursin DB, Connery LE, Ketzler JT. Perioperative diabetic and hyperglycemic management issues. Crit Care Med. 32(4) Suppl:S116–S125

[27] Wang AS, Armstrong EJ, Armstrong AW. Corticosteroids and wound healing: clinical considerations in the perioperative period. Am J Surg. 206(3):410–417

[28] Decker MR, Greenblatt DY, Havlena J, Wilke LG, Greenberg CC, Neuman HB. Impact of neoadjuvant chemotherapy on wound complications after breast surgery. Surgery. 152(3):382–388

[29] Tibbs MK. Wound healing following radiation therapy: a review. Radiother Oncol. 42(2):99–106

[30] Wang Q, Dickson GR, Abram WP, Carr KE. Electron irradiation slows down wound repair in rat skin: a morphological investigation. Br J Dermatol. 130 (5):551–560

[31] Tokarek R, Bernstein EF, Sullivan F, Uitto J, Mitchell JB. Effect of therapeutic radiation on wound healing. Clin Dermatol. 12(1):57–70

[32] Hyland EJ, D'Cruz R, Menon S, et al. Prospective, randomised controlled trial comparing Versajet™ hydrosurgery and conventional debridement of partial thickness paediatric burns. Burns. 41(4):700–707

[33] Lawrence TW. Physiology of the acute wound. In: Granick MS, Long CD, Ramasastry SS, eds. Clinics in Plastic Surgery. Wound Healing: State of the Art. Philadelphia, PA: WB Saunders Co.; 1998:321–340

[34] Castronuovo JJ , Jr, Ghobrial I, Giusti AM, Rudolph S, Smiell JM. Effects of chronic wound fluid on the structure and biological activity of becaplermin (rhPDGF-BB) and becaplermin gel. Am J Surg. 176(2A) Suppl: 61S–67S

[35] Hasan A, Murata H, Falabella A, et al. Dermal fibroblasts from venous ulcers are unresponsive to the action of transforming growth factor-beta 1. J Dermatol Sci. 16(1):59–66

[36] Steed DL, Donohoe D, Webster MW, Lindsley L, Diabetic Ulcer Study Group. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. J Am Coll Surg. 183(1):61–64

[37] Falanga V, Margolis D, Alvarez O, et al. Human Skin Equivalent Investigators Group. Rapid healing of venous ulcers and lack of clinical rejection with an allogeneic cultured human skin equivalent. Arch Dermatol. 134(3):293–300

[38] Gentzkow GD, Iwasaki SD, Hershon KS, et al. Use of dermagraft, a cultured human dermis, to treat diabetic foot ulcers. Diabetes Care. 19(4):350–354

2 Hand and Upper Extremity Anatomy

Loretta Coady-Fariborzian and Winston Richards

Abstract
This chapter discusses in detail the twenty-seven bones, the ligaments, and the tendons that comprise the hand and wrist. Of special importance is the discussion listing the diagnostic requisites for a proper evaluation of wrist injuries, including fractures. In addition to bones, ligaments, and tendons, all other elements of hand and wrist anatomy—blood supply, the fingertip, nerves, skin—are discussed, thus providing a thorough treatment of every component in the anatomical structure.

Keywords: extensor zone, flexor zone, scaphoid, metacarpal, fingertip

2.1 Goals and Objectives

- Name the bones in the hand and wrist and identify them on a plain film.
- Describe the extensor and flexor zones of the hand.
- Describe the blood supply and innervation to the hand.
- Be able to diagnose a structural injury based on known anatomy.

2.2 Pertinent Information

2.2.1 Bones and Ligaments of the Forearm, Wrist, and Hand

The hand and wrist has a total of 27 bones. The wrist is made of the distal ends of the radius and ulna, the eight carpal bones, and the proximal ends of the five metacarpals. The carpal bones consist of two rows of four bones each. The proximal row includes radial to ulnar, the scaphoid, lunate, triquetrum, and pisiform (▶ Fig. 2.1). The distal row includes radial to ulnar, trapezium, trapezoid, capitate, and hamate. The proximal carpal row is considered intercalated because no tendons insert on them and their motion depends on their surrounding articulations. The distal row is connected to each other by short intercarpal ligaments, resulting in negligible motion between them. The extrinsic carpal ligaments connect the distal radius and ulna to the carpal bones. The intrinsic ligaments have their origins and insertions within the carpal bones. When a surgeon wants to enter the wrist joint, he makes a dorsal "ligament sparing" incision, which looks like a radially based "V." This incision splits the dorsal radiotriquetral (radiocarpal) ligament and the dorsal intercarpal ligament. These ligaments are felt to play an important role in stabilizing the proximal carpal row. Palpable landmarks are the sulcus between the scaphoid and trapezoid, the dorsal tubercle of the triquetrum, and the midpoint between Lister's tubercle and the dorsal rim of the sigmoid notch (▶ Fig. 2.2).[1]

The two most important intrinsic ligaments are the scapholunate and lunotriquetral ligaments. The scapholunate ligament is made of a thin volar, a proximal, and a dorsal portion. The dorsal ligament is the thickest, strongest, and most critical scapholunate stabilizer. The radioscaphocapitate and scaphotrapezial ligaments are secondary stabilizers of the scapholunate articulation. The "dart throwing motion" is felt to be the most frequently used wrist motion in activities of daily living. The biomechanics is the radiodorsal/ulnopalmar motion of the midcarpal joint and concerns the scaphocapitate and scaphoid-trapezium-trapezoid (STT) ligament.[2]

When evaluating for bone or ligament injuries to the wrist, four X-ray views of the wrist are needed: posteroanterior (PA), lateral, scaphoid (PA in ulnar deviation), and 45 degree semipronated oblique. A PA view of the wrist should show three smooth radiographic arcs, known as Gilula's lines (▶ Fig. 2.3). Bone articulations normally have apposing parallel surfaces separated by 2 mm or less. A step off or discontinuity of Gilula's lines, carpal overlap, or unusually wide distances between bones suggests an injury. A normal lateral view shows the radius, lunate, capitate, and third metacarpal lined up vertically. Angles frequently used to evaluate carpal stability are the capitolunate, scapholunate, and radiolunate angles, based on the lateral view (▶ Fig. 2.4). The capitolunate angle is measured by drawing a line perpendicular through a line connecting the palmar and dorsal tips of the lunate. This is compared to the capitate axis. The normal capitolunate (CL) axis is 0 ± 15 degrees. The scaphoid is represented by a line that connects the two proximal and distal convexities of the palmar aspect of the bone. The normal scapholunate (SL) angle is 47 degrees (range: 30–60). The axis of the radius is found by drawing a perpendicular through its distal third. The RL should be 0 ± 15 degrees. A dorsal tilt of the lunate "spilled teacup" suggests dorsal intercalated segment instability (DISI), found in scapholunate ligament injuries. The scaphoid shift ("Watson") test is performed by placing pressure over the volar scaphoid while the wrist is moved from the ulnar to radial direction. The unrestrained scaphoid will temporarily shift out of the scaphoid fossa on the radius. A disruption of the scaphoid from the lunate may cause the scaphoid to tilt volarly and the lunate to turn dorsally. This does not always happen, if the secondary stabilizers remain intact. A volar tilt suggests volar intercalated segment instability (VISI), found in lunotriquetral injuries. A disruption of the triquetrum from the scaphoid will cause the triquetrum to turn dorsally and the lunate, still attached to the scaphoid, to turn volarly.[3]

When the scapholunate ligament has been disrupted and the proximal pole of the scaphoid subluxes dorsoradially, the forces across the radiocarpal joint are abnormally distributed, leading to degenerative changes over time. The progression of changes involves first the articulation between the radial styloid and scaphoid, followed by the entire radioscaphoid, then the capitolunate joint. This pattern is known as scapholunate advanced collapse wrist. The radiolunate joint is seldom affected because it remains in contact with normal cartilage due to the restraining effect of the short radiolunate ligament.[3]

2.3 Wrist and Hand Fractures

The scaphoid (navicular) bone is the most commonly fractured carpal bone. The scaphoid has five articulating surfaces and is

Fig. 2.1 Carpal bones.

Fig. 2.3 Gilula's lines.

Fig. 2.2 Palpable landmarks for ligament sparing incision.

nearly entirely covered by cartilage. It is the link between the proximal and distal carpal rows and has multiple ligamentous attachments from where it receives its blood supply. The blood supply enters distally. The proximal pole is dependent on an interosseous blood supply, which makes it vulnerable to avascular necrosis, particularly when fractures involve the proximal third.[3] In the authors' opinion, these are best referred to an experienced wrist surgeon due to the progressive arthritis and dysfunction that can result from nonunions, known as scaphoid nonunion advanced collapse (SNAC).

Fractures of the carpal bones other than the scaphoid are rare and make up 1.1% of all fractures. The more common of those fractures will be discussed. When initial X-rays are negative, but suspicion is high, a computed tomographic (CT) scan is appropriate. Triquetral fractures are the second most common carpal fracture and come in three varieties: dorsal cortical, triquetral body, and volar avulsion. The most common is the dorsal cortical, which is treated with splinting for 3 to 4 weeks.[4]

The trapezium is the next most commonly fractured carpal. The vertical intra-articular fracture is the most common pattern and frequently accompanies the Bennett fracture. Nondisplaced fractures are treated in a thumb spica splint; younger, active patients are best treated with reduction by an open or closed approach to minimize future carpometacarpal (CMC) or STT arthritis.[4]

Hamate fractures may involve the hook or the body. Hook fractures are classically described in golfers, baseball players, and racket sport athletes. This may present with ulnar nerve symptoms. A carpal tunnel view on plain films or CT can secure the diagnosis. The blood supply to the distal hook is poor with a

Fig. 2.4 Lateral wrist view with scaphoid, lunate, capitate, radius lines.

healing rate of 50%. Excision is a reasonable treatment option with no adverse effects to grip or motion. Nondisplaced body fractures can be splinted. A direct blow, repetitive trauma, or sudden contraction of the flexor carpi ulnaris (FCU) can cause a pisiform fracture. Splinting can be successful in nondisplaced fractures. Displaced fractures or nonunions can be treated with excision, with no resulting loss of mobility.[4]

The first CMC joint is unique to humans, allowing opposition and prehension, but hastening the development of arthritic changes. The CMC joint is a biconcave–convex saddle joint, with five articular surfaces and minimal bony constraint. Sixteen ligaments have been described that stabilize the trapezium. Compressive forces magnify greater than 13 times from the fingertip to the trapeziometacarpal joint. Osteoarthritic lesions are first found palmarly on the joint. Degeneration of the posterior oblique ligament has been linked to the development of arthritis when incompetence of the ligament allows abnormal dorsal translation of the metacarpal on the trapezium.[3] The CMC is best viewed by the Roberts technique (thumb AP), where the forearm is maximally pronated and the dorsum of the thumb rests on the X-ray cassette (▶ Fig. 2.5).

The five metacarpal bones each articulate with the carpus. A rigid ligament connection exists between the trapezoid and capitate and the second and third metacarpals that allow for very little mobility. Fracture dislocations are rare and tend to involve significant force (▶ Fig. 2.6). There is greater mobility at the articular surfaces of the fourth and fifth metacarpals, which allows for greater deformity to be accepted when evaluating a fourth or fifth metacarpal fracture. Metacarpal fractures, particularly central ones, are biologically splinted by the intermetacarpal ligaments and interosseous muscles.[5] Patients must be examined for rotational deformity upon digital flexion. If nonoperative treatment is chosen, repeat films must be performed, particularly of spiral or oblique fractures, to verify alignment is maintained. Fractures of multiple adjacent metacarpals are less stable and a lower threshold for surgical fixation is indicated.

Fractures of the metacarpal neck, also known as "boxer's" fractures, are among the most common hand fractures. This is really a "brawler's" fracture since a skilled boxer does not tend to have this fracture pattern. The fifth metacarpal is tolerant of the apex-dorsal deformity and up to 70 degrees of angulation may be compensated for in the fifth metacarpal carpal joint. In comparison, only 10 degrees of angulation is tolerated in the second and third rays. The Jahss reduction maneuver involves flexing the metacarpophalangeal (MCP) joint, proximal interphalangeal (PIP) joint, and distal interphalangeal (DIP) joint and using the proximal phalanx to push the metacarpal head dorsally.[5] This may be difficult to do successfully without sedation. It is the authors' opinion that patients who tend to present with "boxer's" fractures are usually poor surgical candidates for the same reason they sustained the injury and are often unreliable with pin care, splinting, and follow-up.

Intra-articular fractures of the first metacarpal are known as Bennett's and Rolando's fractures. Bennett's fractures are avulsions of the ulnar metacarpal base. The abductor pollicis longus tendon pulls the shaft proximally, displacing the fracture. A Rolando fracture is a comminuted intra-articular fracture of the proximal first metacarpal. Surgical reduction and fixation is often required for both these unstable fracture patterns.[5]

There are 14 phalangeal bones. Nondisplaced distal phalanx and comminuted tuft fractures are most often treated with splinting until tenderness resolves. Minimally displaced, stable, extra-articular fractures of the middle and proximal phalanx may be treated with buddy taping in a compliant patient. Transverse proximal phalanx fractures are unstable and usually best treated with close reduction and internal fixation with a longitudinal pin. We try to minimize open reduction and plate fixation of the middle and proximal phalanx due to the adhesions that can develop between the plate and tendons.

The PIP joint is central to digit function, having up to 120 degrees of mobility. The PIP joint has been described as the "anatomical and functional locus of finger function."[3] It is a hinge, or ginglymus, joint that resists lateral and rotary stress. The joint has a "ligament-box" configuration that resists PIP displacement (▶ Fig. 2.7). The collateral ligaments originate on the lateral aspect of each proximal phalanx condyle. The proper ligament inserts on the volar one-third of the base of the middle

Fig. 2.5 Robert's view of thumb.

Fig. 2.6 Dislocated carpometacarpal joints.

phalanx and the accessory ligament inserts more volarly on the volar plate. The volar plate forms the floor of the joint. Disruption in at least two planes has to take place for displacement of the middle phalanx to occur. Dislocations can usually be treated with reduction and either dorsal splinting or buddy taping. Fracture dislocations or pilon fractures are beyond the scope of this chapter, but can be treated with a variety of options, from extension block splinting to hemi-hamate arthroplasty, depending on the fracture pattern and surgeon experience.[6]

The MCP joint (digits 2–5) forms a condyloid joint. The head is narrow dorsally and wider volary, which creates more contact with the proximal phalanx with greater flexion. The collateral ligaments are more taut in flexion (the "cam effect"), allowing less abduction and adduction, compared with full extension. The MP joints are well protected from injury due to their surrounding support structures (flexor and extensor tendons, volar plate, and intervolar plate ligaments) and location at the base of the fingers.[3] Dislocation of the MP joints is rare, but when they occur, they most frequently are a dorsal dislocation of the index finger due to a forced hyperextension. The volar plate is disrupted proximally, the flexor tendons are found ulnarly, and the lumbricals found radially. The radial digital nerve is superficial and at risk of injury during open reduction.[6]

The mobility of the thumb MCP joint is the most variable of any joint in the body due to the curvature of the metacarpal heads. The joint is supported laterally by the proper collateral ligaments that originate from the lateral condyles of the proximal phalanx and insert on the volar third of the proximal phalanx. The accessory collateral ligaments originate from a more volar position on the metacarpal head and insert on the volar plate and sesamoids. The floor of the joint is formed by the

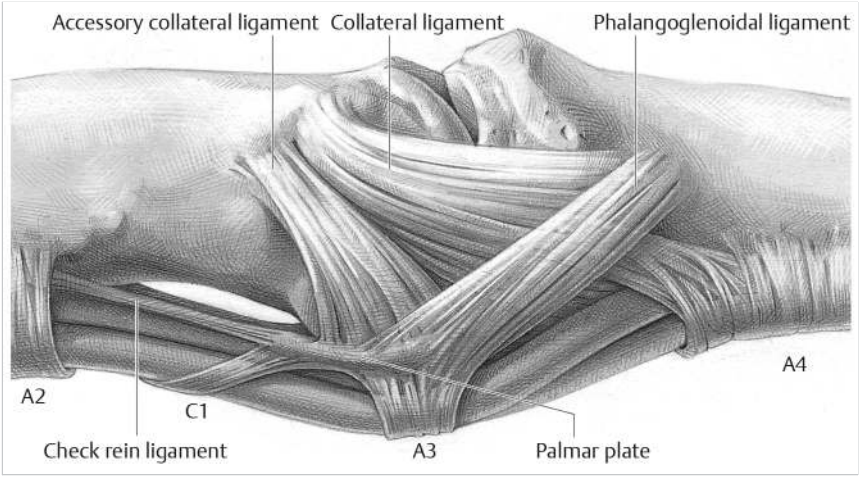


Fig. 2.7 Capsular ligaments of the proximal interphalangeal joint. A2 pulley, C1 pulley, A3 pulley, A4 pulley. From Schmidt HM, Ulrich L. Surgical Anatomy of the Hand. Stuttgart: Thieme; 2004.

volar plate. The proper collaterals are tight in flexion and loose in extension; the accessory collaterals are tight in extension and loose in flexion. Injuries to the ulnar collateral ligament (UCL) are 10 times more common than to the radial collateral ligaments and are a well-known skiing-related injury. The adductor aponeurosis can become interposed between a distally avulsed UCL and its insertion (Stener's lesion), making adequate ligament healing impossible without surgical intervention.[3]

2.3.1 Blood Supply

The hand is supplied by the ulnar artery and the radial artery that originate in the proximal forearm. The ulnar artery continues as the superficial palmar arch and the radial artery continues as the deep palmar arch. An interosseous median artery is a major contributing vessel in 5% of people. An arch is called "complete" when it connects with a branch from another independent artery. The superficial palmar arch is complete in 78.5% of patients; the deep palmar arch is complete in 97% of patients.[7] There are three common digital arteries found at the second, third, and fourth web spaces at the level of the MCP joints. The common digital vessels branch into radial digital and ulnar digital vessels. Up to 98.5 % of extremities have all five digits receiving arterial inflow at the level of the proper digital or common digital artery from both the deep and superficial arches.[3] The ulnar digital artery to the small finger and the radial digital artery to the thumb and index finger tend to be of smaller caliber than the parallel digital artery. This is relevant in a trauma setting when it is more time efficient to concentrate efforts on the larger vessel.

There are consistently four vessels that supply the thumb: the palmar ulnar, palmar radial, dorsal ulnar, and dorsal radial arteries. The palmar blood supply to the thumb arises from the first of four palmar metacarpal arteries, designated as the "princeps pollicis" artery. The princeps pollicis may arise from the first dorsal metacarpal artery, the deep arch, the first palmar metacarpal artery, or the terminal branch of the superficial palmar arch. Several anastomotic connections have been found between the radial and ulnar digital arteries and between the dorsal and palmar systems on cadaveric dissections, which allow for thumb survival after severe vascular injury and provide multiple alternatives in flap designs.[8] Identification of the ulnar artery as the larger artery and originator of the superficial palmar arch led to the conclusion that the ulnar artery is the dominant artery. Subsequent studies found that the radial artery is more important to digital blood flow, with 20% of hands losing pulsatile blood flow to the digits with radial artery compression at the wrist, compared with 5% of hands with ulnar artery compression.[9]

The Allen test was originally described in 1929. The patient closes the hand tightly for 1 minute or until blanching of all digits occurs while the examiner occludes both arteries. The patient extends the fingers and one artery is released. The return of color to the hand indicates an intact circulation from that artery. There have been reports of preoperative Allen's test being poor predictors of postoperative perfusion after radial artery harvest or cannulation.[10] It is the authors' practice to occlude the radial artery with an Acland clamp and listen for perfusion through the common digital vessels prior to proceeding with a radial forearm free or pedicle flap.

Isolated injuries to the radial or ulnar artery do not usually produce critical ischemia or impaired function. Flow will increase through the intact parallel vessel over time. Reconstruction should be considered for improving nerve recovery, diminishing cold intolerance (particularly in colder climates), anticipating future injuries, or to facilitate healing.[3] If ischemia has been ruled out, reconstruction can be performed in a nonemergent setting.

2.3.2 Fingertip

The fingertip is a specialized structure that allows for fine motor activity, sensation, and contributes to aesthetics. The nail complex is divided into the nail bed, nail plate, and eponychium (cuticle), paronychium, and hyponychium (▶ Fig. 2.8). The nail bed consists of the proximal germinal matrix and the distal sterile matrix. The lunula is the white moon-shaped structure that is the junction between the germinal and sterile matrix. The germinal matrix produces ungula keratin. The sterile matrix provides nail plate adherence. Injury or destruction of the germinal matrix will prevent nail growth. An injury or scar to the sterile matrix will create problems with nail plate adherence. The eponychium forms the proximal border and the paronychium forms the lateral soft tissue border of the nail. The

Fig. 2.8 Fingertip anatomy.

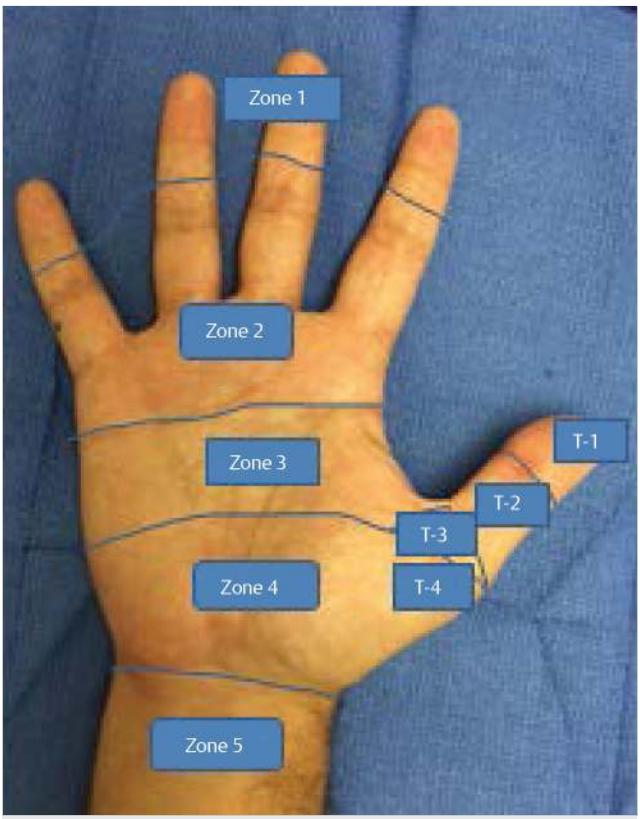


Fig. 2.9 Flexor zones.

dorsal roof, just under the eponychium gives the nail plate its shine. The hyponychium is a plug of keratinous material located just under the distal edge of the nail where the nail bed meets the skin.[11] Rate of nail growth averages 0.1 mm/day, or 2 to 3 mm per month. There is a delay of new growth for 3 to 4 weeks after removal of the nail for nail bed repair.[3]

Deep papillary ridges that produce fingerprints are found on the epidermis of the fingertip. The blood and nerve supply to the fingertip divides near the level of the DIP joint. The proper digital arteries send branches to the nail fold, nail bed, and pulp. The digital nerves send branches to the paronychium, fingertip, and pulp.[11]

2.3.3 Flexor Tendons

The flexor tendon system is divided into five zones (▶ Fig. 2.9). Zone 1 involves only the flexor digitorum profundus (FDP) and extends from the insertion of the flexor digitorum superficialis at the level of the middle phalanx to the FDP insertion on the distal phalanx. Zone 2 is known as "no man's land" and extends from the A1 pulley to the insertion of the FDS tendon. This zone is notorious for being more challenging for both tendon repair and rehabilitation. Zone 3 is at the level of the superficial palmar arch and is where the lumbricals originate from the FDP tendons. Zone 4 is where the flexor tendons are within the carpal tunnel. Zone 5 is the distal forearm, a more desirable location for tendon repair.

The flexor digitorum superficialis muscle belly has two heads of origin: one ulnar and one radial. It lies deep to the wrist flexors (FCU, flexor carpi radialis, and palmaris longus) and the pronator teres. It lies superficial to the FDP and flexor pollicis longus muscle bellies. The FDS divides at the level of the mid forearm into the superficial and deep layers. The superficial layer sends tendons to the middle and ring fingers; the deep layer sends tendons to the index and small fingers. The FDS to the small finger is not consistently present in all individuals. The FDS muscle is innervated by the median nerve and receives its blood supply from the ulnar and radial arteries.[3]

The FDP muscle belly originates from the proximal ulna and interosseous membrane. It lies deep to the FDS muscle belly and adjacent to the FPL muscle belly in the forearm. The tendons usually arise from a common muscle belly, except for the FDP to the index finger. The muscle is supplied predominantly by the ulnar artery. The ulnar nerve innervates the muscle tendon units to the ring and small fingers; the anterior interosseous branch of the median nerve innervates the muscle tendon units to the index and middle fingers.[3]

The FPL tendon originates from the radius and interosseous membrane. It is innervated by the anterior interosseous nerve and supplied predominantly by the radial artery.[3]

All nine flexor tendons are found in the carpal tunnel. The FPL is most radial, the four FDPs are most deep. The FDS tendons to the index and small fingers are superficial to the FDP tendons. FDS tendons to the middle and ring finger are most superficial.[3]

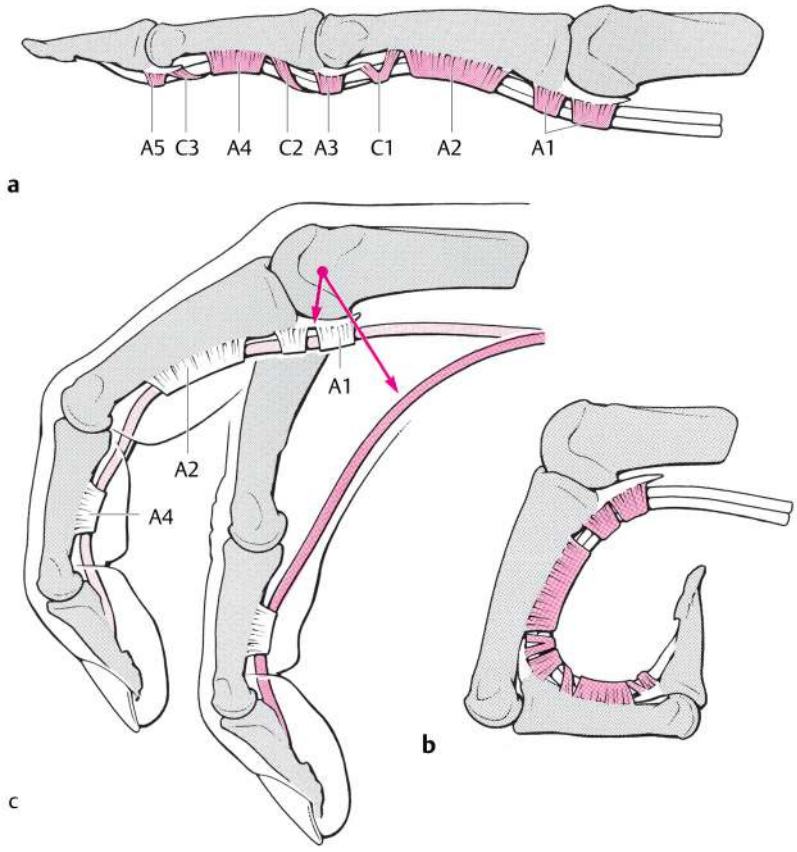


Fig. 2.10 The pulley system of the digital flexor tendon sheaths. **(a)** Lateral view in extension. A1A5, Anular pulleys; C1C3, cruciform pulleys. The undulating course of the flexor tendons corresponds to the palmar contour of the bones of the finger. **(b)** Lateral view in flexion. The flexor tendons describe a harmonious curve, and the pulleys are drawn closer together. **(c)** Bowstringing due to rupture of the A1 and A2 pulleys. The increased distance of the flexor tendons from the pivot point of the metacarpophalangeal joint increases the force of flexion in this joint and exhausts the tendon s available gliding amplitude. The effect of the extensors predominates at the middle interphalangeal joint. From Schmidt HM, Ulrich L. Surgical Anatomy of the Hand. Stuttgart: Thieme; 2004.

After the FDS tendon passes through the A1 pulley, it splits around the FDP tendon and rejoins deep to the FDP at the distal end of the proximal phalanx (known as Camper's chiasm), and then inserts as two separate slips onto the volar middle phalanx.[3]

The flexor sheath is located at flexor zones 1 and 2. The tendons are covered by two thin layers of paratenon (visceral and parietal) within this sheath, which allows for smooth gliding of the tendons. The parietal paratenon allows for passive nutrient delivery to the flexor tendons via diffusion. There is also a direct arterial supply to the flexor tendons through the vincular system at the middle and distal phalanges.[3] The vinculum vessels can be found approaching the tendon dorsally, and may prevent more proximal tendon retraction in the event of a tendon laceration.

The pulleys are fibrous tissue condensations that encircle the flexor tendons, keeping them adjacent to the phalanges, enabling efficient transfer of force from the muscle tendon unit to the phalanges (▶ Fig. 2.10). There are five annular (A1–A5) and three cruciate (C1–C3) pulleys, named in descending order, proximally to distally. The A2 and A4 pulleys insert directly into bone and are found over the proximal and middle phalanges, respectively. The A1, A3, A5 pulleys are located over the MCP, PIP, and DIP joints. The cruciate pulleys lie between the A2–A3, A3–A4, and A4–A5 pulleys. The palmar aponeurotic (PA) pulley lies proximal to the A1 pulley and forms an arch over the flexor tendons. The thumb historically has three pulleys: the A1 lies over the MCP joint, the oblique pulley lies over the proximal phalanx, and the A2 pulley is located over the IP joint. A fourth pulley (annular pulley) has been more recently found in continuity with the A1 pulley.[12]

The A2 and A4 pulleys have traditionally been thought to be most important to prevent bowstringing. Studies have found that 25% of the A2 pulley, up to 75% of the A4, and 25% of combined A2 and A4 pulleys can be excised without significantly affecting motion or work of flexion. Partial A2 and A4 pulley excision may be helpful to facilitate tendon gliding after tendon repair. Isolated removal of the thumb A1 or oblique pulleys did not create significant bowstringing; either one or the other must remain intact.[12]

Closed pulley rupture can happen when a sudden load is placed on the finger flexors, while the DIP joint is hyperextended and the PIP joint is flexed ("crimp" position). This has been classically described in rock climbers, but has been noted in pitchers when throwing a fast ball. This presents as pain, swelling, and ecchymosis over the digit, associated with loss of grip strength and full flexion.[12] The A4 pulley can be completely vented, if necessary, to facilitate repair and rehabilitation.[13]

2.3.4 Extensor Tendons

The extensor tendons are divided into nine zones (▶ Fig. 2.11). Classification allows better communication and treatment planning among surgeons and hand therapists. Injuries at the various zones have different implications for surgery and therapy. Zone 1 is over the DIP joints, Zone 2 is over the middle phalanx, Zone 3 is over the PIP joint, Zone 4 is over the proximal phalanx, Zone 5 is over the MCP joints, Zone 6 is over the metacarpals, Zone 7 is over the carpus, Zone 8 is over the distal forearm, and zone 9 is over the proximal forearm. The thumb is

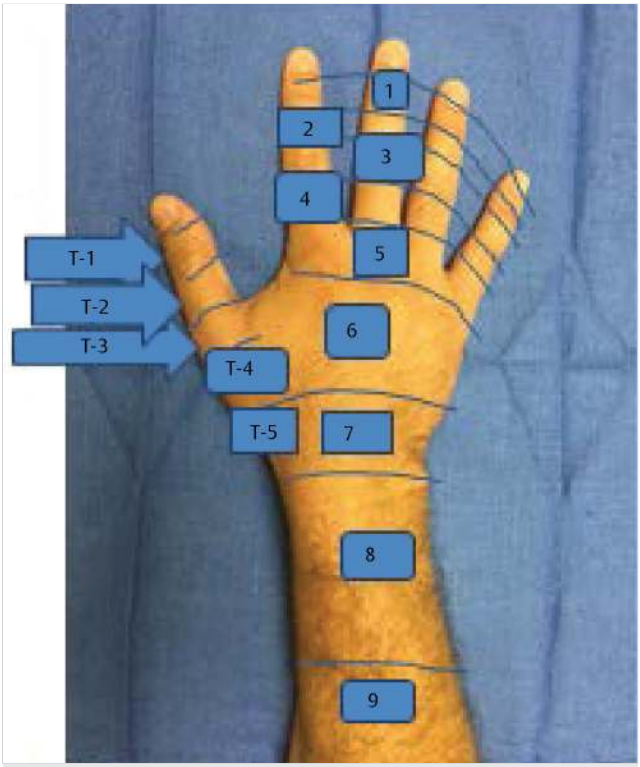


Fig. 2.11 Extensor zones.

classified as T1 over the IP joint, T2 over the proximal phalanx, T3 over the MCP joint, and T4 over the metacarpal.

The wrist extensors originate at the lateral epicondyle and include the extensor carpi radialis brevis (ECRB), extensor carpi radialis longus (ECRL), and the extensor carpi ulnaris (ECU). The finger and thumb extensors include the abductor pollicis longus (APL), extensor pollicis brevis (EPB), extensor pollicis longus (EPL), extensor digitorum communis (EDC), extensor digiti minimi (EDM), and extensor indicis proprius (EIP). They originate at the lateral epicondyle, proximal radius, proximal ulna, and interosseous membrane. The tendons arise 3 to 4 cm proximal to the wrist joint, except for the EIP, whose muscle belly extends to or distal to the wrist joint.[3]

Six extensor compartments are found under the extensor retinaculum at the level of the dorsal wrist, numbered radial to ulnar. The first dorsal compartment has the APL (inserting on the first metacarpal) and EPB (inserting to the first proximal phalanx) tendons. This is the compartment affected by de Quervain's disease. Multiple slips of the APL and/or a separate compartment for the EPB tendon make conservative therapy less likely to be successful. The second dorsal compartment contains the radial wrist flexors, ECRL (inserting on the base of the second MC), and ECRB (inserting to the base of the third MC). This is the compartment affected by intersection syndrome. It is experienced more proximally, but is released here. The third dorsal compartment contains the EPL tendon, which inserts on the base of the thumb distal phalanx. High pressures within this compartment due to a closed distal radius fracture can result in delayed rupture of this tendon. The fourth dorsal compartment contains the EDC and EIP tendons. The posterior interosseous nerve to the wrist joint is found deep to this compartment. The fifth dorsal compartment contains the EIP tendon. The six dorsal compartment contains the ulnar wrist extensor, ECU (inserts on the base of the fifth MC).[3]

Human beings are made with several "spare parts" and redundancy within the extensor system. The EIP (to the index) and EDM (to the small finger) are found ulnar to the index and small finger EDC tendons at the level of the MCP joints. The EDC to the small finger is often missing and the EDM is doubled.[14] Other anatomic variations that have been described include double EIP tendons, double or triple EDC ring, double or triple EDC long, and double EDC small.[15] The juncture tendinum connects the extensor tendons to each other proximal to the MCP joint and can prevent retraction of those tendons after a full-thickness laceration.

The extensor tendons pass over the MCP joints, held midline by the sagittal band, and trifurcates over the proximal phalanx. The central part continues as the central slip and inserts at the dorsal base of the middle phalanx, allowing PIP extension. The lateral slips of the extensor tendon combine with the interosseous and lumbrical muscles to form the lateral bands. These join dorsally to insert at the base of the distal phalanx and form the terminal portion of the extensor tendon, allowing DIP extension.[14]

Several retinacular structures stabilize and coordinate extensor function in the finger. The triangular ligament stabilizes the lateral bands dorsally at the level of the middle phalanx and prevents their volar migration when the PIP joint flexes. The oblique retinacular ligament originates along the volar proximal phalanx and flexor sheath to insert into the terminal tendon to link PIP and DIP joint motion. The transverse retinacular ligaments originate from the volar plate at the PIP joint and insert into the lateral bands, which prevents their dorsal migration during finger extension.[14]

2.3.5 Nerves

The median nerve is found in the forearm between the FDS and FDP muscle bellies. It becomes more superficial in the distal forearm and can be found between the FCR and the palmaris longus tendon below the flexor retinaculum, as it enters the carpal tunnel. The palmar cutaneous nerve branches from the median nerve about 5 cm proximal to the proximal wrist crease, travels ulnar to the FCR tendon, superficial to the flexor retinaculum, and innervates the skin of the thenar eminence. The motor branch of the median nerve comes off as a recurrent, extraligamentous branch just distal to the distal edge of the transverse carpal ligament to innervate the thenar muscles. The proper median nerve continues on to divide into the common and digital nerves that innervate the thumb, index, middle, and radial half of the ring finger. There are variations in the branching pattern of the motor branch of the median nerve that need to be kept in mind when doing carpal tunnel surgery. Less common variations include the subligamentous (dividing within the carpal tunnel), transligamentous (piercing the transverse carpal ligament), originating from the ulnar border of the median nerve, and laying on top of the transverse carpal ligament.[3]

The ulnar nerve enters the forearm between the ulnar and humeral heads of the FCU muscle and travels between the FDP and FCU muscle bellies. It branches dorsally 6 to 8 cm proximal to the proximal wrist crease and innervates the dorsal/ulnar hand. The ulnar nerve proper becomes more superficial

proximal to the proximal wrist crease, where it is located ulnar to the ulnar artery and radial to the FCU tendon. It enters Guyon's canal and divides into the deep and superficial branches. The superficial branch is located ulnarly and innervates the palmaris brevis muscle and supplies sensation as the digital nerves to the ulnar side of the ring finger and either side of the small finger. The deep branch is located radially and innervates the hypothenar muscles, the medial two lumbricals, the interossei muscles, and the adductor pollicis muscle.[3]

Anomalous nerve connections can exist between the median and ulnar nerves that provide a dual nerve supply to muscle bellies. Martin-Gruber connections take place between the median and ulnar nerves in the forearm. Riche–Cannieu connections occur between the motor branch of the ulnar nerve and the recurrent branch of the median nerve.[3]

Radial sensory nerve enters the subcutaneous tissue between the brachioradialis muscle and ECRL tendons about 9 cm proximal to the radial styloid and branches into two main branches 5 cm proximal to the radial styloid. The palmar branch innervates the dorsal radial thumb. The dorsal branch divides into two and innervates the dorsal ulnar thumb and dorsal index and middle fingers.[3]

2.3.6 Skin

Finally, the skin has two distinct layers: the epidermis and dermis. These layers measure 0.05 to 1.5 mm for the epidermis and 0.3 to 3.0 mm for the dermis. Hair follicles are present in varying concentrations; their base is in the deep dermis and they have an epithelial lining. Sebaceous glands and sweat glands lined with epithelium reside in the dermal layer, also. Glabrous skin is naturally hairless and covers the palms and soles.[16]

2.4 Review Questions

2.4.1 True or False

1. The A2 and A4 pulleys must remain completely intact during flexor tendon repair.
2. The flexor tendons get their blood supply from the paratenon, vincular system, and phalanges.
3. The ulnar artery provides the dominant blood flow to the hand.

2.4.2 Choose the Best Answer

4. Which articular surface is *least* prone to degenerative changes after scapholunate ligament disruption?
   a) Radioscaphoid.
   b) Scaphocapitate.
   c) Radiolunate.
   d) Scaphotrapezial.
5. The most common takeoff pattern of the motor branch of the median nerve is
   a) Extraligamentous and recurrent.
   b) Transligamentous.
   c) Subligamentous.
   d) Above the transverse carpal ligament.

2.4.3 Answers

1. False.
2. True.
3. False.
4. c. Radiolunate.
5. a. Extraligamentous and recurrent.

References

[1] Berger RA. A method of defining palpable landmarks for the ligament-splitting dorsal wrist capsulotomy. J Hand Surg Am. 32(8):1291–1295
[2] Kijima Y, Viegas SF. Wrist anatomy and biomechanics. J Hand Surg Am. 34(8): 1555–1563
[3] Green D, Hotchkiss R, Pederson W, Wolfe S. Green's Operative Hand Surgery, 5th edition. San Antonio: Elsevier; 2005
[4] Suh N, Ek ET, Wolfe SW. Carpal fractures. J Hand Surg Am. 39(4):785–791, quiz 791
[5] Meals C, Meals R. Hand fractures: a review of current treatment strategies. J Hand Surg Am. 38(5):1021–1031, quiz 1031
[6] Shah CM, Sommerkamp TG. Fracture dislocation of the finger joints. J Hand Surg Am. 39(4):792–802
[7] Chloros GD, Smerlis NN, Li Z, Smith TL, Smith BP, Koman LA. Noninvasive evaluation of upper-extremity vascular perfusion. J Hand Surg Am. 33(4):591–600
[8] Ramírez AR, Gonzalez SM. Arteries of the thumb: description of anatomical variations and review of the literature. Plast Reconstr Surg. 129(3):468e–476e
[9] Dumanian GA, Segalman K, Buehner JW, Koontz CL, Hendrickson MF, Wilgis EF. Analysis of digital pulse-volume recordings with radial and ulnar artery compression. Plast Reconstr Surg. 102(6):1993–1998
[10] Higgins JP. A reassessment of the role of the radial forearm flap in upper extremity reconstruction. J Hand Surg Am. 36(7):1237–1240
[11] Peterson SL, Peterson EL, Wheatley MJ. Management of fingertip amputations. J Hand Surg Am. 39(10):2093–2101
[12] Zafonte B, Rendulic D, Szabo RM. Flexor pulley system: anatomy, injury, and management. J Hand Surg Am. 39(12):2525–2532, quiz 2533
[13] Tang JB. New developments are improving flexor tendon repair. Plast Reconstr Surg. 2018. Jun;141(6):1427-1437
[14] Amirtharajah M, Lattanza L. Open extensor tendon injuries. J Hand Surg Am. 40(2):391–397, quiz 398
[15] von Schroeder HP, Botte MJ. Anatomy of the extensor tendons of the fingers: variations and multiplicity. J Hand Surg Am. 20(1):27–34
[16] Richards WT, Vergara E, Dalaly DG, Coady-Fariborzian L, Mozingo DW. Acute surgical management of hand burns. J Hand Surg Am. 39(10):2075–2085.e2

3 Anatomy of the Lower Extremity

Mario Rueda, Steven Rueda, Brendan Alleyne, Richard L. Drake, and Raffi Gurunluoglu

Abstract

This chapter addresses the need for a comprehensive knowledge of lower extremity anatomy in the clinical practice of plastic surgery. Reconstructive surgery related to lower extremity trauma incorporates concepts of soft tissue and bone repair and stabilization combined with the use of local muscles and their inherent blood supply for reconstruction. In aesthetic surgery of the lower extremity, success cannot be achieved without an appreciation of the muscular, fascial, and cutaneous interconnections. Also described are the anatomy of the lower extremity with descriptions of the thigh, leg, and foot. Each section begins with the skeletal foundation and progresses into the fascia, muscles, joints, innervations, and vasculature.

Keywords: lower extremity, fascia, skeletal structure, posterior compartment, medial compartment, leg muscles, vasculature, flap surgery, lateral compartment

3.1 Goals and Objectives

- Understand the proper vascular, neural, muscular and bony anatomy of the lower extremity in relation to plastic surgery.
- Appreciate that flap surgery is essentially applied anatomy.
- Know the anatomy well to maximize flap success and patient safety.

3.2 Introduction

A comprehensive knowledge of lower extremity anatomy is necessary for the clinical practice of plastic surgery. Reconstructive surgery related to lower extremity trauma incorporates concepts of soft tissue and bone repair and stabilization combined with the use of local muscles and their inherent blood supply for reconstruction. In aesthetic surgery of the lower extremity, success cannot be achieved without an appreciation of the muscular, fascial, and cutaneous interconnections. This chapter describes the anatomy of the lower extremity with descriptions of the thigh, leg, and foot. Each section begins with the skeletal foundation and progresses into the fascia, muscles, joints, innervations, and vasculature.

3.3 Anatomy of the Lower Extremity

The main functions of the lower extremity are motion and stabilization of the trunk. It consists of a skeletal structure that interacts through several joints: several thick and potent muscles that assist in several movements and a vascular supply. This is covered with skin and subcutaneous tissue. It can be divided into four regions: gluteal, thigh, leg, and foot.

3.4 Fascia

Similar to the trunk, the gluteal region, thigh, and leg have two layers of fascia: the superficial and deep. Superiorly they come together at the level of the inguinal ligament and inferiorly they give rise to retinacular bands.[1]

The superficial fascia encircles the entire lower extremity, including the buttock. It is typically more dense and thicker proximally than in the distal extremity. It is found within the fat of the subcutaneous tissue. It usually gets pierced by nerves and vessels that travel from the deeper structures to the skin.

The deep fascia (also known as fascia lata) is a vascularized fibrous tissue that encircles muscles. It helps separate each region of the lower extremity into different compartments. This does not only serve as pathways for vessel perforators but also improves efficiency by providing attachment areas for muscles. Superiorly, the inguinal ligament serves as the anterior attachment of the deep fascia. Posteriorly, the fascia attaches to the sacrum and coccyx. On the lateral aspect, the fascia joins the iliac crest.

Characteristics of the deep fascia include a lateral band known as the iliotibial band and the saphenous opening. The saphenous opening is an oval defect in the deep fascia that allows the passage of the branches of the femoral artery, the saphenous vein, and lymphatics. It is covered by a portion of superficial fascia that is known as fascia cribrosa.[2]

The fascia of the foot is slightly different from the rest of the extremity. The superficial fascia contains minimal adipose tissue. The deep fascia gives rise to the extensor retinacula, the flexor and peroneal retinaculum, and the plantar fascia. There is superior and inferior extensor retinaculum that supports tendons of dorsiflexion that are located on the dorsum of the foot. The flexor retinaculum is on the medial aspect of the ankle and supports the plantar-flexor tendons. The peroneal retinaculum is on the lateral ankle and supports the tendons of the peroneus longus and peroneus brevis.[3]

3.5 Skeletal Structure

3.5.1 Bones

Femur

The largest and thickest bone in the human body forms the main support for the thigh: the femur.[2] This bone has a posterior concavity that allows it to support the rest of the body. It has special features that articulate with pelvic bones as well as the leg bones.

The superior aspect of the femur contains four irregular structures[1]:

- *Femoral head*: Found on the medial aspect of the bone, it forms two-thirds of a sphere that articulates with the iliac bone at the level of the acetabulum.[1] It is covered with cartilage.

- *Neck of the femur*: Supports the femoral head and connects it to the femoral shaft. It has an inferolateral angulation that projects slightly anteriorly.
- *Greater trochanter*: It is a 1-cm posterior projection found on the lateral aspect of the femur. The gluteus medius, gluteus maximus, obturator internus, and superior and inferior gemellus muscles insert into it.
- *Lesser trochanter*: A medial projection of the femur, it serves as an insertion point to the psoas major and the iliacus.

The inferior portion of the femur bone contains several important features. The medial and lateral condyles are found on the distal portion. They are two projections on the medial and lateral aspects of the distal femur. The medial condyle is longer than the lateral but less prominent. They are separated anteriorly by a depression known as the patellar surface and posteriorly by a notch known as the intercondylar fossa of the femur.[4] The anterior, posterior, and inferior surfaces of the condyles constitute the articular surface of the distal femur. Immediately superior to the condyles, there is the medial and lateral epicondyles. They serve as attachments for ligaments of the knee joint. Superior to the medial epicondyle, there is also the adductor tubercle which serves as the insertion point of the adductor magnus.

Patella

The patella is a sesamoid, flat triangularly shaped bone that is embedded in the quadriceps femoris muscle and makes part of the knee joint. The anterior surface receives the quadriceps femoris tendon.[5] The posterior surface articulates with the femur and contains cartilage. The inferior portion of the posterior surface contains the infrapatellar fat pad. The patella ultimately allows extension force by amplifying the force exerted by the quadriceps.[6]

Tibia

Along with the fibula, the tibia forms the structural base of the leg. It provides support and assists with motion as it serves as the attachment point for several muscles.

The proximal portion of the bone consists of the tibial condyles. They support the articulations to the femur forming the tibiofemoral joint. Similar to the femur, the medial and lateral condyles are separated by the intercondylar area. The cruciate ligaments as well as the menisci attach to this portion of the tibia. The intercondylar area is divided into anterior and posterior parts by the intercondylar eminence; this eminence is formed by projections of the bone called medial and lateral intercondylar tubercles that correspond to a lateral projection of the medial condyle and a medial projection of the lateral condyle.[1] The lateral condyle also contains a facet for the head of the fibula. Immediately below the condyles, there is the tibial tuberosity that serves as attachment for the patellar ligament and continuation of the quadriceps femoris.

The body of the bone is divided into three surfaces: medial, lateral, and posterior. The medial portion can be directly felt and palpated over the skin as it has no attachments (except proximally). The posterior surface has a ridge called "soleal line" that runs from the articular facet for the fibula to the medial border.[2]

The inferior extremity appears smaller than the superior aspect of the bone. It has an inferior articular surface that serves as an articular point with the talus bone. The medial surface is palpable and forms the medial malleolus, and continues inferiorly to have another small surface for articulation with the talus bone. The lateral surface forms the fibular notch, which articulates the fibula.

Fibula

This small and thin bone is part of the leg structure. Its function revolves around providing insertion points for musculature rather than support of the body. It also has two extremities and a body.

The fibular head is separated from the body of the bone by the fibular neck. In its medial aspect, we can find a surface that articulates with the lateral condyle of the tibia. We can also find an interosseous membrane that connects the fibula and the tibia. On the inferior extremity, we find the lateral malleolus. It has a medial surface that articulates with the talus bone.

Foot Bones

The skeletal structure of the foot is unique in that it represents a transition of muscle layout from vertical to horizontal. The ankle itself bridges this transition. In order to achieve this, the human foot has 26 bones.[7]

The tarsal bones are organized into two rows[8]:

- *Proximal*: made up by the talus and calcaneus.
- *Distal*: includes the cuboid, cuneiforms bones (lateral, intermediate, and medial), and the navicular bones. This last bone is found just proximal to the cuneiforms bones.

The first toe consists of a metatarsal bone followed by a proximal and a distal phalange. Second through fifth toes have also a metatarsal bone followed by a proximal phalange; however, it also has a middle phalange that separates the proximal from the distal phalanges. The joints that are found between the metatarsus and the phalanges are called metatarsophalangeal and those found between phalanges are called interphalangeal.

3.5.2 Joints

Hip Joint

The hip joint serves as the area of transfer of support of the trunk into the lower extremities. The pelvis constitutes a stable and complex skeletal structure which is discussed in Chapter 4.

The upper extremity of the femur contains the head of the femur that articulates with the pelvic acetabulum. It forms a ball-socket joint that enables transfer of load as well as motility. The head of the femur as well as the acetabulum is covered with cartilage that minimizes friction and ensures smooth motion. It also contains a capsule that attaches to the bone just outside the acetabular to the base of the femur neck. It permits a wide range of motion.

There are four ligaments that reinforce this important joint and they are the iliofemoral, ischiofemoral, pubofemoral, and the ligamentum teres.[9,10,11] Their main function is to strengthen the capsule and prevent excessive motion of the joint leading to injury or dislocation.

Knee Joint

This joint connects the thigh and the leg. It is made of two articulations: the tibiofemoral and the patellofemoral joints. The first joint is formed by the femoral and tibial condyles and it allows for extension and flexion of the leg. The second joint is formed by the femur and the articular surface of the patella which mainly assists the tibiofemoral joint in its functions.[12]

The joint is reinforced by several ligaments and tendons that provide stability[1]:

- *Anterior*: We can find the patellar ligament, quadriceps tendon, the medial and lateral patellar retinaculum.
- *Lateral*: Fibular collateral ligament (lateral collateral ligament—LCL).
- *Medial*: Tibial collateral ligament (medial collateral ligament—MCL).
- *Posterior*: Oblique popliteal ligament (posterior ligament), arcuate popliteal ligament, anterior cruciate ligament (ACL), posterior cruciate ligament (PCL), meniscofemoral ligaments.

The medial and lateral menisci are also key components in the structure of the knee. They are **C**-shaped fibrocartilaginous structures that minimize friction in the knee joint.[13] They are attached to the tibia and ultimately help transmit the weight of the trunk into the distal structures of the leg.

Ankle Joint

It serves as the connection between the foot and the leg. The ankle is also a transition point where muscles go from a vertical to horizontal arrangement. It is made of three bones: the tibia, the fibula, and the talus.

The ankle includes three joints: the talocrural joint (ankle proper joint) formed by the distal ends of the fibula and tibia that enclose the upper surface of the talus; the subtalar joint where the talus and the calcaneus bones meet; and the inferior tibiofibular joint where the medial and distal end of the fibula articulates with the lateral side of the tibia.[8]

There are four ligaments that reinforce this important joint: the deltoid (medial ankle), calcaneofibular (lateral ankle), anterior, and posterior talofibular ligaments.[1,7,14] Similar to the knee and hip ligaments, these four ligaments allow movement of the foot—dorsiflexion, plantarflexion, rotation, and inversion—by stabilizing the joint and avoiding dislocations or injury.

3.6 Muscles

3.6.1 Gluteal Region

There are three predominant muscles in the buttock area: gluteus maximus, gluteus medius, and gluteus minimus muscles. These are muscles involved with external rotation and the abduction of the hip. We can also find the Tensor fasciae latae muscle that participates in thigh flexion, medial rotation, and abduction. Other muscles that assist in the movement of the pelvis and the hips include the psoas major and iliacus.[1,2]

3.6.2 Tensor Fasciae Latae

- ***General:*** This is a flattened, small, and rectangular muscle that extends from the iliac crest to the knee. The muscle belly is small; however, it has a large aponeurosis.
- ***Insertions and Structure:*** It arises from the outer lip of the iliac crest. The muscle belly descends inferiorly and posteriorly to fuse with the fascia lata, forming the iliopubic tract.[1,2]
- ***Blood supply:*** Derives from the superior gluteal artery and the lateral circumflex femoral artery.
- ***Innervation:*** Superior gluteal nerve from nerve roots L4, L5, and S1.
- ***Classification*** : This muscle flap is a Mathes and Nahai type I class, as it has a single dominant pedicle, the ascending branch of the lateral femoral circumflex artery. It has a wide variety of reconstructive options, both as a local flap (defects of the abdomen, groin, ischium, trochanter, sacrum) and a free flap (defects of the head and neck, upper extremity, lower extremity, foot).[15]

3.6.3 Gluteus Maximus

- ***General:*** This is the largest and most superficial of the gluteal muscles. It plays a significant role in the appearance of the buttock and hip.
- ***Insertions and structure:*** It has a very broad origin, arising from the gluteal zone of the ilium, sacrum, and sacrotuberous ligament and the lumbar fascia. The muscle fibers travel downward and laterally and the fascicles are separated by fibrous septa that join to form an aponeurosis. It inserts into the gluteal tuberosity of the femur and the iliotibial tract.
- ***Blood supply:*** Main arteries that supply this muscle are the superior and inferior gluteal arteries.
- ***Innervation:*** The inferior gluteal nerve is responsible for the innervation of this muscle with fibers from L5, S1, and S2.
- ***Classification:*** This muscle flap is a Mathes and Nahai type III class, as it has two dominant pedicles, the superior and inferior gluteal arteries. It also has two minor pedicles, the first perforator of profunda femoris and two to three intermuscular branches of the lateral femoral circumflex. It has several reconstructive options, both as a local flap (defects of the ischium, trochanter, sacrum) and as a free flap for breast reconstruction.[15]

3.6.4 Gluteus Medius

- ***General:*** It is a triangular-shaped muscle that is deep to the gluteus maximus.
- ***Insertions and structure:*** Originates in the gluteal surface of the ilium, deep to the gluteus maximus. The muscle fibers converge toward the superior extremity of the femur and form a flat tendon at the level of the greater trochanter.
- ***Blood supply:*** The superior gluteal artery is responsible for the blood supply to this muscle.
- ***Innervation:*** Nerve roots from L4, L5, and S1 supply this muscle through the superior gluteal nerve.

3.6.5 Gluteus Minimus

- ***General:*** It is a triangular-shaped muscle that can be found in the deep and anterior portion of the gluteal region.
- ***Insertions and structure:*** It originates from the gluteal surface of the ilium, deep to the gluteus medius. The muscle fibers travel toward the upper extremity of the femur and form a tendon that inserts in the greater trochanter.

- ***Blood supply:*** Similar to the gluteus medius, blood supply derives from the superior gluteal artery.
- ***Innervation:*** The superior gluteal nerve, with roots from L4, L5, and S1 innervate this muscle.

3.6.6 Psoas Major

- ***General:*** It is a long and fusiform muscle that can be found in the external iliac fossa of the pelvis and in the posterior trunk, next to vertebral bodies.
- ***Insertions and structure:*** It originates from vertebral bodies T12 to L5. Muscle fibers travel next to the vertebral column downward. The muscle descends through the internal iliac fossa and it joins the anterior border of the iliac muscle forming the iliopsoas muscle. It ultimately inserts into the lesser trochanter of the femur.
- ***Blood supply:*** Derived from the lumbar branch of the iliolumbar artery.
- ***Innervation:*** Anterior branches of L1–L3 nerves (lumbar plexus).

3.6.7 Iliacus

- ***General:*** It is a triangular-shaped muscle whose apex joins the psoas major in its medial border.
- ***Insertions and structure:*** It originates from the internal iliac fossa and after forming the iliopsoas muscle, it inserts into the lesser trochanter of the femur.
- ***Blood supply:*** It has dual blood supply provided by the iliac branch of the iliolumbar artery and the medial femoral circumflex artery.
- ***Innervation:*** Innervated by the femoral nerve.

Other muscles that are found in the gluteal region but are not described in this book include the obturator internus, pyramidal muscle, gemellus muscles, and obturator externus.

3.6.8 Thigh Muscles

The musculature of the thigh is divided into three distinct compartments that also helps us identify functions for the muscles. The three compartments are as follows:

- *Anterior fascial compartment*: Muscles in this group usually assist in thigh extension. It includes the rectus femoris, vastus lateralis, vastus intermedius, vastus medialis (these four muscles form the quadriceps muscles), sartorius, and pectineus muscles.
- *Posterior fascial compartment*: Involved with flexion of the thigh. It includes the biceps femoris, semimembranosus, and semitendinosus muscles.
- *Medial fascial compartment*: Adduction is the main function of this group of muscles. It includes the gracilis, adductor longus, adductor brevis, and the adductor magnus.

3.7 Anterior Compartment

3.7.1 Rectus Femoris

- ***General:*** It is the most superficial portion of the quadriceps. It is a fusiform-shaped muscle that is immediately over the vastus intermedius.
- ***Insertions and structure:*** It originates from two separate heads—the straight head that arises from the anterior inferior iliac spine and the reflected head that arises just above the acetabulum. The four muscles of the quadriceps (rectus femoris, vastus lateralis, vastus intermedius, and vastus medialis) form on their distal portion the quadriceps tendon, in which the patella is found. Just below the patella, the tendon is known as the patellar tendon and continues and inserts into the tibial tuberosity.
- ***Function:*** Assists in hip flexion as well as knee extension.
- ***Blood supply:*** Derived from the descending branch of the lateral femoral circumflex artery.
- ***Innervation:*** The femoral nerve is responsible for the innervation of all the quadriceps muscles.
- ***Classification:*** This muscle flap is a Mathes and Nahai type II class, as it has a single dominant pedicle, the descending branch of the lateral femoral circumflex artery. It also has two minor pedicles, the ascending branch of the lateral femoral circumflex and the muscular branches of the superficial femoral artery. It has a wide variety of applications, both as a local flap (coverage of the inferior abdomen, groin, ischium, and perineum) and as a functional muscle free flap (facial reanimation and upper extremity reconstruction).[15]

3.7.2 Vastus Lateralis

- ***General:*** It is found just lateral to the vastus intermedius. It is just superior to it and portions of the muscle can cover fibers of the vastus intermedius.
- ***Insertions and structure:*** It originates from a tendinous structure at the external portion of the linea aspera of the femur: this extends proximally into the greater trochanter. The muscle belly descends to the patella and inserts to the tibial tuberosity as the quadriceps tendon.
- ***Function:*** The muscle participates in knee extension.
- ***Blood supply:*** The lateral circumflex femoral artery provides the main blood supply.
- ***Innervation:*** Performed by the femoral nerve.
- ***Classification*:** This muscle flap is a Mathes and Nahai type I class, as it has a single dominant pedicle, the descending branch of the lateral femoral circumflex artery. It also has three minor pedicles—the transverse branch of the lateral femoral circumflex, the posterior branches of the profunda femoris artery, and the superficial branch of the lateral superior genicular. It has a wide variety of options, both as a local flap (coverage of the trochanter, knee, groin, ischium, and perineum) and as a reconstructive option for the abdominal wall and acetabular fossa.[15]

3.7.3 Vastus Intermedius

- ***General:*** It is located around the body of the femur, deep to the rectus femoris muscle.
- ***Insertions and structure:*** It originates from the proximal two-thirds of the anterolateral aspect of the femur. It terminates as a portion of a tendon (quadriceps femoris tendon) that attaches to the tibial tuberosity.
- ***Function:*** Participates in extension of the knee.
- ***Blood supply:*** Derived from the femoral artery.
- ***Innervation:*** Performed by the femoral nerve.

3.7.4 Vastus Medialis

- ***General:*** This muscle is medial to the vastus intermedius. It can be found partially attached to the muscle.
- ***Insertions and structure:*** Originates from the medial portion of the femur and inserts into tibial tuberosity via the quadriceps femoris tendon.
- ***Function:*** It assists in extension of the leg.
- ***Blood supply:*** Femoral artery is the main blood supply.
- ***Innervation:*** Similar to the other vastus muscles, it is innervated by the femoral nerve.
- ***Classification:*** This muscle flap is a Mathes and Nahai type II class, as it has a single dominant pedicle, a branch of the superficial femoral artery. It also has two minor pedicles, branches of the superficial femoral artery and the musculoarticular branches of the descending genicular artery. It can be used as a distally based flap, a functional muscle or as a V-Y advancement flap skin island for coverage of the upper knee.[15]

3.7.5 Sartorius

- ***General:*** It is the longest muscle in the human body and it runs the entire length of the anterior compartment. It is in the superficial portion of the compartment. The proximal portion of the sartorius forms the lateral border of the femoral triangle.
- ***Insertions and structure:*** It originates from the anterior-superior iliac spine. It runs inferiorly and medially. It travels behind the medial condyle of the femur and forms a tendon that inserts into the medial aspect of the tibia's body. The ending of the muscle is called "pes anserinus" and it is formed by the sartorius, gracilis, and semitendinosus tendons.
- ***Function:*** It assists in flexing and lateral rotation of the hip joint.
- ***Blood supply:*** Derived from the femoral artery.
- ***Innervation:*** Done by the femoral nerve.
- ***Classification:*** This muscle flap is a Mathes and Nahai type IV class, as it has a segmental pedicle as the blood supply, six to seven branches of the superficial femoral vessels. It has proven to be useful in covering defects involving the groin, femoral vessels, and the knee.[15]

3.7.6 Pectineus

- ***General:*** This rectangular and flat muscle forms the floor of the femoral triangle. Its origin also forms Cooper's ligament.
- ***Insertions and structure:*** It originates from the pubic bone. The muscle belly proceeds to the pectineal line of the femur where it inserts.
- ***Function:*** This muscle is responsible for flexion and adduction of the thigh.
- ***Blood supply:*** Derived from the obturator artery.
- ***Innervation:*** Femoral nerve supplies this muscle; however, the obturator nerve may provide this function via L2 and L3 roots.

3.8 Posterior Compartment

3.8.1 Biceps Femoris

- ***General:*** Along with the semimembranosus and semitendinosus, the biceps femoris forms the hamstring muscle. It consists of a long and short heads.
- ***Insertions and structure:*** The long head originates from the tuberosity of the ischium. It continues inferiorly toward the lateral posterior aspect of the muscle. The short head arises from the linea aspera of the femur. Its fibers travel inferiorly to join the long head about two-thirds of the way down into the muscle. They then form a tendon that inserts into the lateral portion of the fibular head and the lateral condyle of the tibia.
- ***Function:*** Extends the hip. It also externally rotates and flexes the knee.
- ***Blood supply:*** Popliteal artery supplies the distal portion of the muscle while the inferior gluteal artery supplies the proximal portion.
- ***Innervation:*** The long head is innervated by roots of the tibial part of the sciatic nerve. The short head is innervated by the common fibular nerve.

3.8.2 Semimembranosus

- ***General:*** This muscle is found deep to the semitendinosus muscle. It has a membranous tendon at its origin.
- ***Insertions and structure:*** It originates from the ischial tuberosity. As it travels inferiorly, it forms the pes anserinus. It inserts into the posterior aspect of the medial condyle of the tibia.
- ***Function:*** Assists in the extension of the hip as well as internal rotation and flexion of the knee.
- ***Blood supply:*** Double blood supply from the profunda femoris and the gluteal arteries.
- ***Innervation:*** Innervated by the tibial part of the sciatic nerve (L5, S1, and S2 roots).

3.8.3 Semitendinosus

- ***General:*** A long and fusiform muscle, it is located on the medial aspect of the posterior compartment. It is superior to the semimembranosus muscle.
- ***Insertions and structure:*** It generates from the ischial tuberosity, next to the origin of the long head of the biceps. About a third of the distal portion of this muscle continues as a tendon behind the femur condyle. It inserts into the pes anserinus, after the muscle gives a tendon that is found behind the sartorius and below the gracilis.
- ***Function:*** Similar to the semimembranosus muscle, it assists in flexion and internal rotation of the knee as well as hip extension.
- ***Blood supply:*** Inferior gluteal artery.
- ***Innervation:*** Innervated by roots from L5, S1, and S2 via sciatic nerve.

3.9 Medial Compartment

3.9.1 Gracilis

- ***General:*** It is a long and thin muscle located on the medial aspect of the thigh.
- ***Insertions and structure:*** It originates from the ischiopubic ramus of the pubis. The muscle belly travels inferiorly, medial to the adductor muscles, and becomes continuous with the pes anserinus.
- ***Function:*** Although it assists in thigh adduction, its main function is to assist during internal rotation and flexion of the knee.
- ***Blood supply:*** The medial circumflex femoral artery supplies blood to the gracilis muscle.
- ***Innervation:*** The anterior branch of the obturator nerve, via L2 and L3 fibers, is responsible for its innervation.
- ***Classification*:** This muscle flap is a Mathes and Nahai type II class, as it has a single dominant pedicle, the ascending branch of the medial circumflex femoral artery. It also has a minor pedicle, one or two branches of the superficial femoral artery. It can be used for local defect coverage and reconstruction of the abdomen, groin, perineum, ischium, penis, and vagina. It can also be used as a functional free flap for facial reanimation and upper extremity reconstruction.[15]

3.9.2 Adductor Longus

- ***General:*** It is a triangular-shaped muscle, bigger than the adductor brevis but smaller than the adductor magnus. It is superficial to both these muscles.
- ***Insertions and structure:*** It originates from the body of the pubis. Its fibers travel downward and insert in the linea aspera of the femur. The muscle belly of the adductor longus forms the medial border of the femoral triangle.
- ***Function:*** It assists during thigh adduction.
- ***Blood supply:*** The three adductor muscles are supplied by the profunda femoris artery.
- ***Innervation:*** The anterior branch of the obturator nerve innervates this muscle via L2, L3, and L4 roots.[16]

3.9.3 Adductor Brevis

- ***General:*** Is the smallest of the adductor muscles. It is triangular in shape, with its base directed toward the femur. This muscle is superficial to the adductor magnus and deep to the adductor longus and pectineus muscle.
- ***Insertions and structure:*** It originates from the body of the pubis as well as the inferior ramus. Its fibers travel inferiorly and outward and they insert at the linea aspera of the femur and the lesser trochanter.
- ***Function:*** It adducts the thigh.
- ***Blood supply:*** Supplied by the profunda femoris.
- ***Innervation:*** Innervation is done by the posterior branch of the obturator nerve via L2, L3, and L4 roots.

3.9.4 Adductor Magnus

- ***General:*** It is a triangular muscle, the deepest of the adductor group.
- ***Insertions and structure:*** This muscle originates from the pubis and the tuberosity of the ischium. It continues toward the femur and forms two portions: a lateral (pubofemoral) and medial (ischiocondylar) portion. The lateral muscle that originates from the pubis continues its way to the middle portion of the femur at the level of the linea aspera. The medial portion comes from the ischial tuberosity and travels more distally and inserts into medial condyle of the femur.
- ***Function:*** The pubofemoral portion of the muscle assists in hip flexion, while the ischiocondylar portion assists in hip extension. Both muscles play a role in hip adduction.
- ***Blood supply:*** The profunda femoris artery is responsible.
- ***Innervation:*** The pubofemoral portion is innervated by the obturator nerve via L2 and L3 roots. The ischiocondylar portion is innervated by the tibial part of the sciatic nerve via L4 roots.[17]

3.9.5 Leg Muscles

The leg muscles are generally elongated and thin. Most of them have long and thin tendons at their most distal portion. The muscles are covered by fascia. They are divided into four compartments:

- ***Anterior compartment:*** Made up of the extensor digitorum longus, extensor hallucis longus, tibialis anterior, and the fibularis (peroneus) tertius muscles.
- ***Lateral compartment:*** Is made up of the fibularis brevis and fibularis longus muscles.
- ***Superficial posterior compartment:*** Composed of the gastrocnemius, plantaris, and soleus muscles.
- ***Deep posterior compartment:*** Made up of the flexor digitorum longus, flexor hallucis longus, tibialis posterior, and the popliteus muscles.

3.10 Anterior Compartment

3.10.1 Extensor Hallucis Longus

- ***General:*** This flat and elongated muscle is lateral to the tibialis anterior.
- ***Insertions and structure:*** It originates from the medial aspect of the fibula and the interosseous membrane. Distally it continues as a long tendon that after crossing the extensor retinacula and traveling toward the dorsum of the big toe, it inserts into the distal phalanx of it.
- ***Function:*** It is responsible for extending the big toe. It also assists in dorsiflexion of the foot.
- ***Blood supply:*** Similar to the other anterior compartment muscles, it is supplied by the anterior tibial artery.
- ***Innervation:*** The deep fibular nerve, via roots of L5 and S1, supplies this muscle.
- ***Classification*:** This muscle flap is a Mathes and Nahai type IV class as it has a segmental pedicle, six to eight arterial muscular branches from the anterior tibial artery. It is primarily used for coverage of defects involving the distal third of the tibia.[15]

3.10.2 Extensor Digitorum Longus

- ***General:*** This muscle is also elongated and directed toward the second through fifth toes. It is located on the lateral portion of the anterior compartment and convers a portion of the extensor hallucis longus.

- ***Insertions and structure:*** Being a wide muscle, it originates from the first two-thirds of the anterior surface of the fibula, the superior portion of the interosseous membrane, and the lateral tibial condyle. The muscle belly travels toward the ankle and crosses the extensor retinacula and divides into four tendons that travel toward the dorsum of the lateral four digits. It inserts into the middle and distal phalanges of the second through fifth toes.
- ***Function:*** It assists in the dorsiflexion of the ankle and toe extension.
- ***Blood supply:*** Done by the anterior tibial artery.
- ***Innervation:*** Receives roots L5 and S1 from the deep fibular nerve.

3.10.3 Tibialis Anterior

- ***General:*** A long and fusiform muscle, it is located on the medial portion of the compartment, lateral to the anterior border of the tibia.
- ***Insertions and structure:*** It originates in the lateral aspect of the proximal two-thirds of the tibia. The distal portion of the muscle is composed by a cylindrical tendon that travels on the dorsum of the foot to insert into the first metatarsal and medial cuneiform bones.
- ***Function:*** It is responsible for dorsiflexion and inversion of the foot.
- ***Blood supply:*** Derived from the anterior tibial artery.
- ***Innervation:*** It is performed by the deep fibular nerve through branches L4 and L5.

3.10.4 Fibularis Tertius

- ***General:*** It is a small muscle located on the deep lateral portion of the anterior compartment.
- ***Insertions and structure:*** It originates in the anterior surface of the distal fibula. It continues distally as a tendon that inserts into the dorsal surface of the fifth metatarsal.
- ***Function:*** It assists in dorsiflexion and eversion of the foot.
- ***Blood supply:*** The anterior tibial artery is responsible for its blood supply.
- ***Innervation:*** The deep fibular nerve is responsible for its innervation.

3.11 Lateral Compartment

3.11.1 Fibularis Brevis

- ***General:*** It is covered by the fibularis longus muscle. It is both smaller and shorter than the fibularis longus muscle.
- ***Insertions and structure:*** It originates in the distal two-thirds of the inferolateral portion of the fibular body. It continues distally as a thin muscle that travels posterior to the lateral malleolus, deep to the fibularis longus muscle. It travels lateral to the calcaneus and finally inserts into the base of the fifth metatarsal bone.
- ***Function:*** It is involved in the plantar flexion of the ankle joint. It also assists in foot eversion.
- ***Blood supply:*** Similar to the fibularis longus, it is supplied by the fibular artery.
- ***Innervation:*** Roots of L5 and S1 from the superficial fibular nerve are responsible for innervation.

3.11.2 Fibularis Longus

- ***General:*** It is a fusiform muscle, located on the superficial and lateral aspect of the compartment. Its tendon travels on the external margin of the leg and the plantar surface.
- ***Insertions and structure:*** It originates at the lateral aspect of the lateral tibial condyle and at the head and proximal two-thirds of the lateral aspect of the fibula. In between the two insertions, we can usually identify the fibular nerve. The distal portion of the muscle continues as a tendon that covers the fibularis brevis. It runs behind the lateral malleolus under the peroneal retinaculum. It continues across the lateral aspect of the calcaneus and it crosses the lateral side of the cuboid. Finally, it inserts in the lateral aspect of the medial cuneiform and the lateral portion of the first metatarsal bone.
- ***Function:*** It serves as a plantar flexor at the ankle joint and helps in foot eversion. In conjunction with the tibialis muscles, it maintains support of the arches.
- ***Blood supply:*** Derived from the fibular artery.
- ***Innervation:*** It receives L5 and S1 roots from the superficial fibular nerve.

3.12 Superficial Posterior Compartment

3.12.1 Gastrocnemius

- ***General:*** It is composed of two distinct heads (gemellus muscles): the medial and lateral heads. The muscle establishes the inferior boundaries (medial and lateral) of the popliteal fossa. In some individuals, a sesamoid bone, fabella, can be found on the lateral head of the muscle.
- ***Insertions and structure:*** The lateral head originates from the lateral condyle of the femur and the medial head from the medial condyle. They continue toward the inferior portion of the compartment, and along with the tendon of the soleus and the plantaris, it forms Achilles (calcaneal) tendon. It finally inserts on the posterior surface of the calcaneus bone.
- ***Function:*** It is a potent plantar flexor and it also assists in knee flexion.
- ***Blood supply:*** Derives from sural arteries that come from the popliteal artery.
- ***Innervation:*** Roots S1–S2 from the tibial nerve provide innervation.

3.12.2 Plantaris

- ***General:*** It is the smallest muscle of this compartment. Can be found deep to the lateral head of the gastrocnemius.
- ***Insertions and structure:*** It originates at the lateral supracondylar ridge. Distally it forms a very thin tendon that travels between the soleus and the gastrocnemius. It joins the tendons of the gastrocnemius and the soleus to form Achilles tendon that inserts into the calcaneus.
- ***Function:*** It assists in flexion of the knee and plantarflexion.

- ***Blood supply:*** Sural arteries are responsible for its blood supply.
- ***Innervation:*** The tibial nerve is responsible for its innervation.

3.12.3 Soleus

- ***General:*** It is usually found deep to the gastrocnemius muscle. Its name derives from the appearance of the muscle, which is thought to resemble a sandal.
- ***Insertions and structure:*** It originates from the posterior aspect of the proximal third of the fibula and also from the soleal line on the posterior aspect of the tibia. These two insertions are joined by the tendinous arch of the soleus. The distal portion of the muscle continues as a tendon that, along with the tendons of the two previous muscles, forms Achilles tendon that inserts on the calcaneus.
- ***Function:*** It is involved in plantarflexion.
- ***Blood supply:*** Similar to the other muscles of the superficial posterior compartment, the sural arteries are responsible for its blood supply.
- ***Innervation:*** The tibial nerve with roots from L5 to S2 supplies this muscle.

3.13 Deep Posterior compartment

3.13.1 Flexor Hallucis Longus

- ***General:*** This muscle is the most lateral structure of the deep compartment. It is covered by the soleus muscle.
- ***Insertions and structure:*** It originates from the posterior aspect of the fibula and the interosseous membrane. It continues as a long tendon that travels behind the medial malleolus and the talus. It continues deep to the sustentaculum tali (horizontal eminence in the medial surface of the calcaneus) and finally continues forward to insert at the base of the last phalanx of the first toe.
- ***Function:*** It flexes the great toe. It also assists in plantarflexion.
- ***Blood supply:*** It is derived from the peroneal artery, a branch of the posterior tibial artery.
- ***Innervation:*** S1 and S2 roots from the tibial nerve are responsible for innervation.

3.13.2 Flexor Digitorum Longus

- ***General:*** This long muscle is located on the posterior medial aspect of the deep posterior compartment.
- ***Insertions and structure:*** The muscle originates from the middle third of the posterior aspect of the tibia. It descends initially medial to the tibialis posterior muscle but, in the last third of the leg, it crosses behind this muscle. It forms a tendon lateral to the tibialis posterior that passes behind the medial malleolus and advances forward just medial to the calcaneus. It finally divides into four distinct tendons and inserts at the base of the distal phalanges of the lateral four digits.
- ***Function:*** It participates in flexion of the second through the fifth toes. It also participates in plantarflexion.
- ***Blood supply:*** It is supplied by the posterior tibial artery.
- ***Innervation:*** The tibial nerve (roots L5, S1, and S2) innervates this muscle.

3.13.3 Tibialis Posterior

- ***General:*** This long muscle is located in between the flexor digitorum longus and the flexor hallucis longus.
- ***Insertions and structure:*** Just below the soleal line, in the posterior aspect of the tibia, as well as the posterior aspect of the fibula and the interosseous membrane, we can find the most proximal portion of this muscle. Distally it forms a tendon that travels behind the medial malleolus until it reaches the plantar surface. The tendon then divides into plantar (which inserts into the second, third, and fourth metatarsals, the cuboid, and the second and third cuneiform bones), main (inserts into the navicular and first cuneiform bones), and recurrent portions (inserts into the calcaneus).
- ***Function:*** It participates in plantarflexion and inversion of the foot. It also stabilizes the inferior tibiofibular joint.
- ***Blood supply:*** Derived from the posterior tibial artery.
- ***Innervation:*** It is supplied by the tibial nerve via L4 and L5 roots.

3.13.4 Popliteus

- ***General:*** It is a small triangular muscle that can be found posterior to the knee joint.
- ***Insertions and structure:*** This muscle originates from the lateral condyle of the femur and some of its fibers also arise from the lateral meniscus. It travels downward and toward the medial portion of the extremity, where it inserts into the posterior surface of the superior portion of the tibia.
- ***Function:*** It medially rotates the leg. When we are standing over the extremity, the popliteus unlocks the knee and allows flexion. Also, because of its posterior location, it prevents femur dislocations.
- ***Blood supply:*** Derived from the popliteal artery.
- ***Innervation:*** Branches of L5 fibers innervate this muscle through the tibial nerve.

3.13.5 Foot Muscles

The muscles of the foot can be divided into extrinsic and intrinsic. The extrinsic muscles are those that originate in the leg and were discussed in the previous section. The intrinsic muscles originate in the foot and they are divided into two regions, the dorsal and the plantar region. The dorsal region includes the short extensor digitorum and the short toe extensor muscles. The plantar region includes several muscles that are organized in four layers.[7]

Dorsal region

This region is composed of the extensor hallucis brevis and the extensor digitorum brevis muscles. The former of these muscles extends to the great toe while the latter extends to the lateral four digits. They both help extend the toes. This function is considered accessory and impairment of the muscles does not compromise ambulation. Their blood supply is derived from branches of the dorsalis pedis artery (lateral tarsal artery). They

are innervated by the deep fibular nerve with roots from S1 and S2.

Plantar Region

First layer

It is the closest to the sole and its muscles are deep to the plantar aponeurosis. It includes the abductor hallucis, abductor digiti minimi, and the flexor digitorum brevis. These muscles originate from the calcaneus and insert in the phalanges of the first, fifth, and second through fifth toes, respectively. The tendons from the extrinsic muscles separate the muscles in the first layer from those found in the second layer.

The function, blood supply, and innervation are as follows:

- Abductor hallucis: Abducts the great toe and its blood supply derives from the medial plantar artery. Innervation is by the medial plantar nerve.
- Abductor digit minimi: Flexes and abducts the fifth toe and its blood supply comes from the lateral plantar artery. Innervation is by the lateral plantar nerve.
- Flexor digitorum brevis: Flexes the lateral four toes. Its blood supply comes from medial and lateral plantar arteries. Innervation is by the medial plantar nerve.

Second Layer

It contains two muscle entities: the lumbricals and the flexor digitorum accessorius muscles.

- The lumbricals originate from the medial border of the flexor digitorum longus tendons and insert into the extensor digitorum longus tendons and eventually into the proximal phalanges of the four lateral toes. They flex the metatarsophalangeal joints and extend the interphalangeal joints. Blood supply is derived from the medial and lateral plantar arteries. Innervation is performed by the medial and lateral plantar nerves via S3 roots.
- The flexor digitorum accessorius: This muscle has two heads: the medial head arises from the concave surface of the calcaneus and the lateral head from the inferior surface of the calcaneus. The two heads join and insert into the tendon of the flexor digitorum longus. They assist in plantar flexion of the lateral four toes. It is innervated by the lateral plantar nerve (S1 and S2 roots).

Third Layer

The flexor digiti minimi brevis, flexor hallucis brevis, and adductor hallucis are part of this layer. They all function to maintain the plantar arch.

- The flexor digiti minimi brevis originates in the fifth metatarsal bone and it travels forward to insert into the base of the first phalanx of the first toe. It participates in adduction and flexion of the fifth toe.
- Originating from the medial part of the cuboid bone, the flexor hallucis brevis is responsible for flexion of the great toe. It inserts in the medial and lateral aspect of the first phalanx.
- The adductor hallucis has an oblique head and a transverse head. The oblique head originates from the second through fourth metatarsal bones and inserts into the lateral aspect of the first phalanx of the great toe. The transverse head originates from the metatarsophalangeal ligaments of the third through fifth toes and inserts into the lateral head of the first phalanx of the first toe. It is responsible for the adduction of this toe.

Fourth Layer

Two of the muscles that are found at the level of this layer are the dorsal interossei and the plantar interossei muscles.

- There are four dorsal interossei muscles that are located between the metatarsal bones. Each of them has two heads that originate from the proximal portion of each metatarsal. They continue forward, forming one single tendon that inserts into their respective proximal phalanges (second, third, and fourth phalanges). They are responsible for toe abduction.
- There are only three plantar interossei muscles. They are also located in between the lateral metatarsal bones. Contrary to the dorsal interossei, these muscles have only one head that originates from the medial aspect of their respective metatarsal bone (third through fifth). They continued distally and insert at the level of the third through fifth proximal phalanges. They assist in toe adduction.

3.14 Vasculature

3.14.1 Gluteal Region

Branches of the internal iliac provide most of the blood supply of the gluteal region. The descending aorta bifurcates into the common iliac arteries. These then divide into the external and internal iliac vessels. The former is responsible for supplying the lower extremity, while the latter supplies pelvic structure and muscles in the gluteal region.

The internal iliac initially travels downward and passes to the greater sciatic foramen (▶ Fig. 3.1b). It does this by traveling anterior to the internal iliac vein and posterior to the ureter. It branches into an anterior and posterior trunk. The anterior trunk branches into several smaller vessels including the inferior gluteal artery. The posterior trunk gives off several branches including the superior gluteal and the iliolumbar arteries.

The inferior gluteal artery derives from the anterior trunk of the internal iliac. It exits the pelvis below the piriformis muscle through the greater sciatic foramen. It travels to the posterior aspect of the thigh. It is responsible for supplying the gluteus maximus.

The superior gluteal artery runs initially posteriorly and exits the pelvis above the level of piriformis muscle, through the greater sciatic foramen. It divides into the superior and deep branch. The superior branch is responsible for supplying the gluteus maximus and it does so by piercing through it. Collateral flow between the superior gluteal and inferior gluteal can be found in the muscle. The deep branch also divides into a superior and inferior branch. It is initially just deep to the gluteus medius prior to branching. Both its branches anastomose with the lateral circumflex femoral artery. Its inferior branch, prior to anastomosing, supplies the gluteus medius and minimus.[18]

The iliolumbar artery branches off the posterior trunk of the internal iliac. After its origin, it travels superiorly just medial to

the psoas major and behind the external iliac artery and obturator nerve.[19] As it travels by the psoas, it gives branches that supply this muscle.

3.14.2 Thigh

After its origin from the common iliac artery, the external iliac artery continues to travel inferiorly. Just behind the inguinal ligament, it continues as the common femoral artery. It continues into the thigh by passing through the femoral canal. It travels into the femoral sheath just lateral to the femoral vein and medial to the femoral nerve.

The common femoral artery branches into the superficial femoral (SFA) and the profunda femoris artery (PFA) distal to the inguinal ligament (▶ Fig. 3.1a). The SFA continues to descend through the femoral triangle on the anteromedial portion of the thigh. It travels under the sartorius muscle and enters the adductor canal. It then enters the popliteal fossa up to a level where it becomes the popliteal artery.

The PFA arises from the posterolateral aspect of the common femoral artery. It travels close to the femur in between the adductor longus and the pectineus muscle. It is responsible for supplying several muscles in the thigh via its main branches: the perforating arteries and the medial circumflex femoral and the lateral circumflex femoral artery.

The perforating arteries are four vessels that pierce the adductor muscles and that reach the posterior aspect of the thigh. The branches that are above, in front, and below the adductor brevis are known as first, second and third perforator. The fourth perforator is usually the distal, smaller end of the PFA. The perforators that travel proximally anastomose with the inferior gluteal artery and inferiorly with branches of the popliteal artery.

The medial circumflex femoral artery originates from the proximal PFA. It travels to the posterior portion of the thigh and exits the femoral triangle in between the psoas and the pectineus muscles, next to the neck of the femur and over the external obturator muscle. It then divides into a transverse, deep, ascending, and acetabular branch.[20] The transverse branch continues to travel behind the surgical neck of the femur and it anastomoses with the transverse branch of the lateral circumflex artery. The deep branch is responsible for supplying the proximal muscles. The ascending branch follows the external obturator tendon and along with the ascending branch of the lateral circumflex, it provides collateral blood flow to the neck of the femur. The acetabular branch is responsible for supplying the adipose tissue in the inferior portion of the acetabulum. It usually anastomoses with the obturator artery.

The lateral circumflex femoral artery also originates from the proximal portion of the PFA. It travels between the different

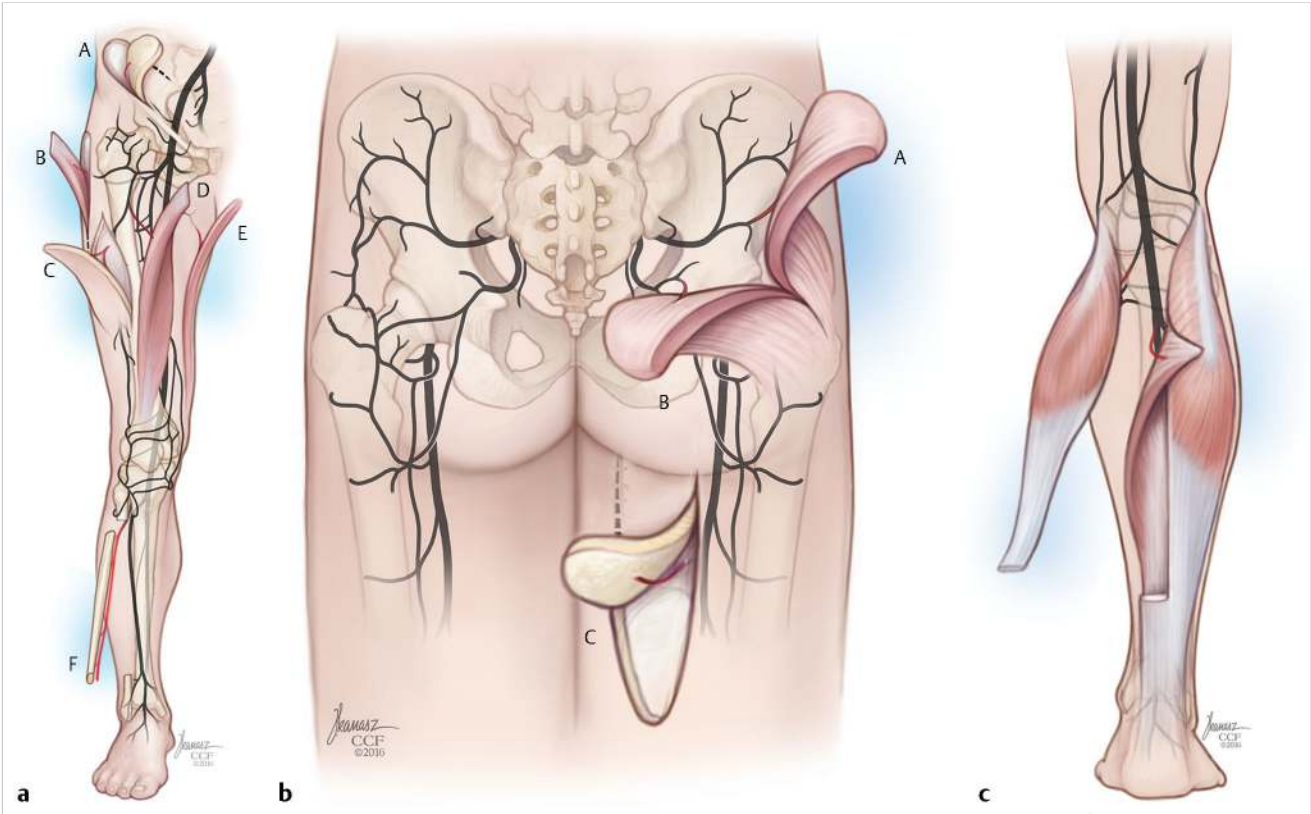


Fig. 3.1 Vasculature supplying local muscle and skin flaps of the lower extremity. **(a)** A, Groin flap (superficial circumflex iliac artery); B, Tensor fascia latae flap (branch off of lateral circumflex femoral artery, LCFA); C, Anterolateral thigh flap (perforator branch (es) of the descending branch of the LCFA); D, Rectus femorsi muscle flap (descending branch of the LCFA); E, Gracilis flap (medial circumflex femoral artery); F, Fibula flap (peroneal artery) **(b)** A, Gluteus maximus flap (superior gluteal artery); B, Gluteus maximus flap (inferior gluteal artery); C, Posterior thigh flap (inferior gluteal artery) **(c)** A, Gastrocnemius flap (medial) (sural artery); B, Soleus flap (posterior tibial, peroneal, sural artery).Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography ©2016. All Rights Reserved.

divisions of the femoral nerve, behind the rectus femoris. It branches into a transverse, ascending, and descending branches. The transverse branch goes across the vastus lateralis and travels anterior to the surgical neck of the femur where it anastomoses with the transverse branch of the medial circumflex femoral artery. The ascending branch travels upward just deep to the rectus femoris, behind the hip join and it anastomoses with the medial circumflex femoral artery just behind the neck of the femur. The descending branch is found deep to the rectus femoris and it distributes through the quadriceps. Following the vastus lateralis, it reaches the external portion of the knee.

3.14.3 Leg

The popliteal artery arises as a direct continuation of the SFA at the level of the popliteal fossa (▶ Fig. 3.1c). It continues to travel distally, and at the level of the popliteus muscle, it produces the anterior tibial artery. The popliteal continues distally and after a few centimeters, it divides into the posterior tibial and the fibular (peroneal) artery. The portion of the popliteal immediately after the anterior tibial artery may be referred as the tibioperoneal or tibial-fibular trunk. The fibular artery travels in the posterior compartment, deep and lateral, closely associated with the fibula. The posterior tibial travels deep, under the transverse fascia; as it descends, it becomes more superficial and its pulse can be felt at the medial malleolus. It branches into the lateral and medial plantar arteries.

The proximal portion of the popliteal artery provides the superior genicular arteries. They arise from either side of the popliteal and travel toward the anterior portion of the knee joint. These vessels anastomose with branches of the lateral circumflex femoral artery and the SFA.

The anterior tibial artery travels downward, just anterior to the popliteus muscle. It continues it course in between the tibia and the fibula and between the extensor digitorum longus and the tibialis anterior. At the anterior aspect of the ankle, between both malleoli, the anterior tibial artery becomes the dorsalis pedis artery.

3.14.4 Foot

The blood supply of the foot is derived from branches of the anterior tibial and posterior tibial. They can be divided into dorsal and plantar arteries. The fibular artery does not usually provide any branches distal to the calcaneus; it does play a significant role in supplying the ankle joint via small terminal branches.

Dorsal Arteries

The anterior tibial artery continues at the level of the foot as the dorsalis pedis artery. It originates at the distal level of the inferior extensor retinaculum and continues up to the space between the first and second metatarsal. The artery passes next to the extensor hallucis longus and extensor digitorum longus tendons. It divides into two terminal branches: the first dorsal metatarsal and the deep plantar artery. It also gives rise to the arcuate artery as its terminal portion.

The first dorsal metatarsal artery travels over the first dorsal interosseous muscle and divides into two dorsal digital arteries that supply the medial aspect of the lateral aspect of the first digit and the medial aspect of the second digit.[21] The deep plantar artery travels forward between the two heads of the first dorsal interosseous muscle, supplying the sole of the foot and contributing to the plantar arch.

The arcuate artery of the foot derives from the terminal portion of the dorsalis pedis. It travels transversally toward the external border of the foot immediately over the metatarsal bases. It anastomoses with the lateral tarsal artery. The arcuate artery gives three metatarsal dorsal arteries that divide into dorsal digital arteries that supply the medial and lateral aspects of the corresponding toes.

Plantar Arteries

These vessels are derived from the posterior tibial artery. It gives rise to the medial and lateral plantar artery.

The medial plantar artery is smaller than its lateral counterpart. It travels to the great toe in the most medial portion of the sole. Initially it can be found superior to the abductor hallucis but later it is situated in between this muscle and the flexor digitorum brevis. At the level of the first metatarsal base, it gives rise to the medial plantar digital artery of the first toe. It anastomoses with the first dorsal metatarsal artery at this level. Near its origin, the medial plantar artery also gives rise to a deep branch that contributes to the plantar arch.

The lateral plantar artery extends forward and laterally in between the flexor digitorum brevis and the flexor accessorius muscles. At the level of the fifth metatarsal base, it changes direction to becomes transverse and completes the plantar arch: it crosses the sole toward the bases of the first and second metatarsal bone and at this level it anastomoses with the deep plantar branch.[1,22]

The Angiosome

Prior to the increased interest in microsurgical free flaps, little importance was assigned to the superficial blood supply of overlying skin. The organization of superficial vasculature territories of the skin and soft tissues has been described by the angiosome model. Perforating vessels give rise to defined islands of blood supply which vary in density according to anatomic location. A total of 376 such vessels of 0.5 mm or greater diameter have been identified in the literature.[23] Just as in the dermatome model, where sensory nerve fiber overlap occurs, the angiosomes have similar watershed areas of overlap. For instance, the head, thorax, and proximal limbs have larger but less dense angiosome pattern as compared to the distal extremities.[23] This network of vessels within the skin assist our clinical guidance in the design of soft-tissue flaps for wound coverage, such as in the field of plastic surgery.

3.15 Review Questions

3.15.1 True or False

1. The boundaries of Hunter's canal, also known as subsartorial or adductor canal are as follows: Sartorius: anterior and medial, vastus medialis: anterior and lateral, adductor longus and adductor magnus: posterior.

3.15.2 Choose the Best Answer

2. A 29 year old male patient complains of inability to dorsiflex his right foot along with sensory loss in the first web space 3 days after undergoing jaw reconstruction for malignant ameloblastoma using a fibula osteocutaneous flap. Which of the following is likely damaged?
   a) Superficial peroneal nerve.
   b) Deep peroneal nerve.
   c) Saphenous nerve.
   d) Tibial nerve.
3. The vascular pedicle for the following muscles originate from the lateral circumflex femoral vessels, except
   a) Vastus Lateralis.
   b) Rectus Femoris.
   c) Tensor fascia Latae.
   d) Gracilis.
4. Neurovascular contents of Hunter's canal include which of the following:
   a) Terminal part of the femoral artery.
   b) Terminal part of the femoral vein.
   c) Nerve to vastus medialis and terminal part of the obturator nerve.
   d) Saphenous nerve.
   e) All of the above.
5. 36 year old morbidly obese woman with a history of failed medial gastrocnemius flap is scheduled for revision knee arthroplasty and requires substantial amount of stable soft tissue coverage:
   a) Distally based rectus femoris flap.
   b) Distally based gracilis flap.
   c) Distally based vastus lateralis flap.
   d) Free tissue transfer.

3.15.3 Answers

1. True.
2. b. Deep peroneal nerve.
3. d. Gracilis.
4. e. All of the above.
5. d. Free tissue transfer.

References

[1] Moore K, Agnur A. Essential Clinical Anatomy. Baltimore, MD: Lippincott Williams & Wilkins; 2002
[2] Standring S. Gray's Anatomy: The Anatomical Basis of Clinical Practice. 40th ed. London: Churchill Livingstone; 2008
[3] Davis WH, Sobel M, Deland J, Bohne WH, Patel MB. The superior peroneal retinaculum: an anatomic study. Foot Ankle Int. 15(5):271–275
[4] Farrow LD, Chen MR, Cooperman DR, Victoroff BN, Goodfellow DB. Morphology of the femoral intercondylar notch. J Bone Joint Surg Am. 89(10):2150–2155
[5] Basso O, Johnson DP, Amis AA. The anatomy of the patellar tendon. Knee Surg Sports Traumatol Arthrosc. 9(1):2–5
[6] Herrington L. The effect of patella taping on quadriceps strength and functional performance in normal subjects. Phys Ther Sport. 5:33–36
[7] Neligan P. Plastic Surgery. 3rd ed. Seattle, WA: Saunders; 2013
[8] Riegger CL. Anatomy of the ankle and foot. Phys Ther. 68(12):1802–1814
[9] Stafford GH, Villar RN. Ischiofemoral impingement. J Bone Joint Surg Br. 93 (10):1300–1302
[10] Nam D, Osbahr DC, Choi D, Ranawat AS, Kelly BT, Coleman SH. Defining the origins of the iliofemoral, ischiofemoral, and pubofemoral ligaments of the hip capsuloligamentous complex utilizing computer navigation. HSS J. 7(3):239–243
[11] Chang CY, Huang AJ. MR imaging of normal hip anatomy. Magn Reson Imaging Clin N Am. 21(1):1–19
[12] Flandry F, Hommel G. Normal anatomy and biomechanics of the knee. Sports Med Arthrosc Rev. 19(2):82–92
[13] Allen AA, Caldwell GL, Fu FH. Anatomy and biomechanics of the meniscus. Oper Tech Orthop. 5:2–9
[14] Siegler S, Block J, Schneck CD. The mechanical characteristics of the collateral ligaments of the human ankle joint. Foot Ankle. 8(5):234–242
[15] Mathes S, Nahai F. Reconstructive Surgery: Principles, Anatomy, & Technique. 1st ed. New York; Churchill Livingstone; St Louis; Quality Medical Publishing; 1997
[16] Tuite DJ, Finegan PJ, Saliaris AP, Renstr, ö, m PA, Donne B, O, ', Brien M. Anatomy of the proximal musculotendinous junction of the adductor longus muscle. Knee Surg Sports Traumatol Arthrosc. 6(2):134–137
[17] Hurwitz ZM, Montilla R, Dunn RM, Patel NV, Akyurek M. Adductor magnus perforator flap revisited: an anatomical review and clinical applications. Ann Plast Surg. 66(5):438–443
[18] Elias Soueid N, Mountcastle TS, Levine JL, Allen RJ, Chiu ES, Vasile J. Superior and inferior gluteal artery perforator flaps. In: Flaps and Reconstructive Surgery. [Edinburgh] : Saunders, 2009:525–539
[19] Rusu MC, Cergan R, Dermengiu D, et al. The iliolumbar artery-anatomic considerations and details on the common iliac artery trifurcation. Clin Anat. 23 (1):93–100
[20] Hallock GG. The gracilis (medial circumflex femoral) perforator flap: a medial groin free flap? Ann Plast Surg. 51(6):623–626
[21] Lee JH, Dauber W. Anatomic study of the dorsalis pedis-first dorsal metatarsal artery. Ann Plast Surg. 38(1):50–55
[22] Orbay H, Kerem M, Unl, ü RE, et al. Vascular anatomy of plantar muscles. Ann Plast Surg. 58(4):420–426
[23] Taylor GI. Grabb and Smith's Plastic Surgery. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014;32–34

4 Anatomy of the Trunk

Mario Rueda, Steven Rueda, Brendan Alleyne, Richard L. Drake, and Raffi Gurunluoglu

Abstract
This chapter shows that successful reconstructive surgery is heavily dependent on a thorough understanding of the components of truncal anatomy. The implications of thorough knowledge for chest wall and breast reconstruction are obvious. Additionally, tissues from the trunk can be used elsewhere as free flaps. The authors describe the anatomy of the trunk, abdomen, pelvis, and posterior trunk. Each section begins with the skeletal foundation and from there progresses into the fascia and muscles and concludes with a discussion of the pertinent vasculature.

Keywords: trunk, vascular anatomy, neural anatomy, muscular anatomy, skeletal anatomy, angiosome, flap surgery

4.1 Goals and Objectives

- Understand the proper vascular, neural, muscular and bony anatomy of the trunk in relation to plastic surgery.
- Appreciate that flap surgery is essentially applied anatomy.
- Know the anatomy well to maximize flap success and patient safety.

4.2 Introduction

Reconstructive plastic surgery is often dependent on a thorough understanding of the components of truncal anatomy. Implications for chest wall and breast reconstruction are obvious. Additionally, tissues from the trunk can be used elsewhere as free flaps. The authors describe the anatomy of the trunk, abdomen, pelvis, and posterior trunk. Each section begins with the skeletal foundation and progresses into the fascia, muscles, and concludes with a discussion of the pertinent vasculature.

4.3 Chest

The superior border of the chest is made up by both clavicles and the manubrium. The anterior axillary line, defined as a line between the middle and lateral end of the clavicle, defines the lateral border. The costal margin, defined by the edge of ribs 7 through 10, and the distal portion of the sternum define the inferior border.

The hallmark of the superficial anatomy of the chest is the breast tissue. It can extend from the second rib to the sixth rib and from the sternum to the latissimus dorsi. The entire chest wall has a superficial fascia that lies just deep to the skin and subcutaneous tissue. Immediately over the breast tissue, this fascia is divided into superficial and deep, surrounding the breast tissue. In between these two layers, Cooper's ligaments run perpendicular, providing support to the parenchyma. Just lateral to the midclavicular line, the nipple-areolar complex can be found at the level of the fourth intercostal space.[1]

The breast has a very rich blood supply that is derived from the internal thoracic perforators, thoracoacromial artery, lateral thoracic artery, and intercostal vessels.[2] The innervation is mainly by the thoracic intercostal nerves branches from T2 to T6. Although the exact nipple location can vary, the innervation and blood supply remains constant at the level of the fourth intercostal space, being provided mainly by T4 lateral cutaneous branch and the internal thoracic artery perforators.[3]

Inspection and examination of the chest reveals a groove on the superior and lateral portion of it, which marks the clavipectoral triangle. It is bordered by the pectoralis major and deltoid muscles as well as the clavicle. It contains the cephalic vein and the clavipectoral fascia, which courses below the pectoralis major. Deep to this triangle, the subclavian vein and subclavian artery can be found.[2]

4.3.1 Skeletal Structure

The skeletal structure of the thorax is formed by the sternum, the ribs, and the vertebral column. Additionally, the clavicles connect the chest with both upper extremities.

The sternum is a convex, flat bone that can be found in the center of the chest. It is divided into the manubrium, body, and xiphoid process; the sternal angle marks the point where the body joins the manubrium. On its lateral surface, it articulates with costal cartilage from the first seven ribs. The clavicles also articulate at the level of the manubrium. The manubrium serves as the insertion of the sternocleidomastoid muscles. The body serves as the origin of the sternocostal head of the pectoralis major.[2]

There are seven pairs of true ribs and five pairs of false ribs. True ribs connect directly to the sternum while false ribs do so indirectly with costal cartilage from ribs above them. The superior border is the insertion point for the intercostal muscles, while the inferior border has the intercostal neurovascular bundle. The inner surface of the ribs is lined with pleura. The head of the ribs articulate, with two facets, with the vertebral column.

The clavicle is a double curved bone that articulates medially to the manubrium and laterally to the acromion forming the sternoclavicular and acromioclavicular joints. The first two-thirds of the ribs have a concave anterior border, while the posterior border is convex. The deltoid, trapezius, subclavius, pectoralis major, sternocleidomastoid, and sternohyoid muscles attach to the clavicle.[4]

The vertebral column will be discussed later in this chapter.

4.3.2 Muscles

The chest has four muscle groups that carry several mobility and breathing functions. They are the pectoralis major, pectoralis minor, serratus anterior, intercostal, and subclavius muscles.

The pectoralis major is the most superficial muscle of the chest. It has a fan-shaped appearance with fibers running from its medial and superior origin to the lateral edge of the bicipital groove of the humerus. The clavicular head originates from the

anterior border of the medial portion of the clavicle superiorly and the sternocostal head from the medial and anterior aspect of the sternum as well as from the costal cartilage of ribs 2 through 6. It assists in the flexion and extension of the humerus as well as in its adduction and medial rotation. The main blood supply is derived from the pectoral branch of the thoracoacromial trunk.[5,6,7,8,9] On the medial aspect of the muscle, there are additional perforator vessels which are derived from the internal mammary system. On the lateral portion, perforators from the intercostal arteries and lateral thoracic artery also supply the muscle.[3] The dominant pedicle, or main blood supply combined with the secondary segmental blood supply, classifies this muscle as a Type V Mathes and Nahai flap.[5] In plastic surgery, this muscle is commonly used to cover local sternal wounds. Innervation is accomplished by the lateral and medial pectoral nerves that contract the clavicular and the sternocostal head, respectively.[6]

The pectoralis minor is a flat and triangular muscle, deep to the pectoralis major. The muscle has three heads that originate from the external border of ribs 3, 4, and 5; the fibers then converge into the internal border of the coracoid process of the scapula. Its main function is to stabilize the scapula against the chest wall. Blood supply is most commonly from the pectoral branch of the thoracoacromial trunk and the lateral thoracic artery. These two dominant pedicles classify this muscle as a type III flap.[5] It is often used to locally cover defects of the shoulder and axilla but, it can also be used for breast implant coverage as well as a functional muscle free flap when combined with microsurgical anastomoses in facial reanimation.[5] It is innervated by the medial pectoral nerve and also receives some innervation from the lateral pectoral nerve that passes directly through it.[10]

The serratus anterior is a flat and thin muscle that lies in the lateral wall of the chest. It originates from the first nine ribs and it inserts on the medial border of the scapula. This muscle holds the scapula against the chest wall and assists in the upward rotation and protraction of the scapula. The superior portion of the muscle derives its blood supply from the lateral thoracic artery, while the lower portion from the thoracodorsal artery.[11] This muscle has two dominant pedicles making it a type III muscle flap. This muscle can be used in plastic surgery to assist in breast reconstruction with coverage of an expander and it also finds use as a functional free flap in facial reanimation.[5] It is innervated by the long thoracic nerve.[12]

The ribs are connected by a series of muscles that play a role in the expansion and active relaxation of the chest cavity during respirations. They accomplish this function in part by the opposing directions of their muscle fibers. The deepest muscle is the innermost intercostal followed by the internal intercostal. The external intercostal muscle is the most superficial. Their blood supply and innervation are derived from the neurovascular bundle that runs on the inferior aspect of each rib.

The subclavius muscle is a small fusiform muscle that is located immediately below the clavicle and above the subclavian artery. It originates just distal to the costal cartilage of the first rib and inserts in the inferior surface of the middle third of the clavicle.[4] Protecting the brachial plexus and subclavian vessels is part of the function of this muscle.[13] It also assists in shoulder depression. Blood supply derives from the thoracoacromial trunk.

4.3.3 The Angiosome

Prior to the increased interest in microsurgical free flaps, little importance was assigned to the superficial blood supply of overlying skin. The organization of superficial vasculature territories of the skin and soft tissues has been described by the angiosome model. Perforating vessels give rise to defined islands of blood supply which vary in density according to anatomic location. A total of 376 such vessels of 0.5 mm or greater diameter have been identified in the literature.[14] Just as in the dermatome model, where sensory nerve fiber overlap occurs, the angiosomes have similar watershed areas of overlap. For instance, the head, thorax, and proximal limbs have a larger but less dense angiosome pattern as compared to the distal extremities.[14] This network of vessels within the skin assists our clinical guidance in the design of local soft-tissue flaps for wound coverage, such as in the field of plastic surgery.

4.3.4 Vasculature

The chest has several networks of blood vessels that supply the skin and soft tissues (▶ Fig. 4.1). There are a series of vessels that connect the subclavian and external iliac arteries and supply the anterior chest wall and the anterior abdominal wall throughout their course. We will discuss the blood supply to the anterior chest structures in this section.

There are two major vessels: the subclavian and axillary arteries that are responsible for perfusion of not only the upper extremities but also of the chest wall.

The subclavian artery originates from the brachiocephalic trunk on the right and the aortic arch on the left. It travels laterally between the anterior and middle scalene muscles and becomes the axillary artery after it crosses the lateral border of the first rib. It gives rise to the internal thoracic artery, dorsal scapular artery, and vessels that supply the neck.

The axillary artery originates on the lateral border of the first rib and becomes the brachial artery after passing the teres major. The pectoralis minor helps in further dividing this vessel into three different parts. The first part is medial to the muscle and gives rise to the superior thoracic artery. The second portion of the vessel is immediately inferior to the muscle and the thoracoacromial and lateral thoracic arteries are derived from it. Finally, the third part is lateral to the pectoralis minor and gives rise to the subscapular artery, anterior humeral circumflex artery, and posterior humeral circumflex artery.[15]

The branch of the subclavian artery that plays a key role in supplying blood to the anterior chest wall is the internal thoracic artery. It travels within the thoracic cavity from its origin toward its bifurcation into the superior epigastric and musculophrenic arteries. It can be found lateral to the sternum, running deep to the internal intercostal muscles but superficial to the innermost muscles. During its course, it gives perforating vessels through the second and sixth intercostal spaces, supplying the pectoralis major and medial chest wall as well as medial breast parenchyma.[16] The internal mammary vessels, also termed “the internal thoracic vessels,” can additionally be used as recipient anastomotic sites for microsurgical free flaps in breast reconstruction.

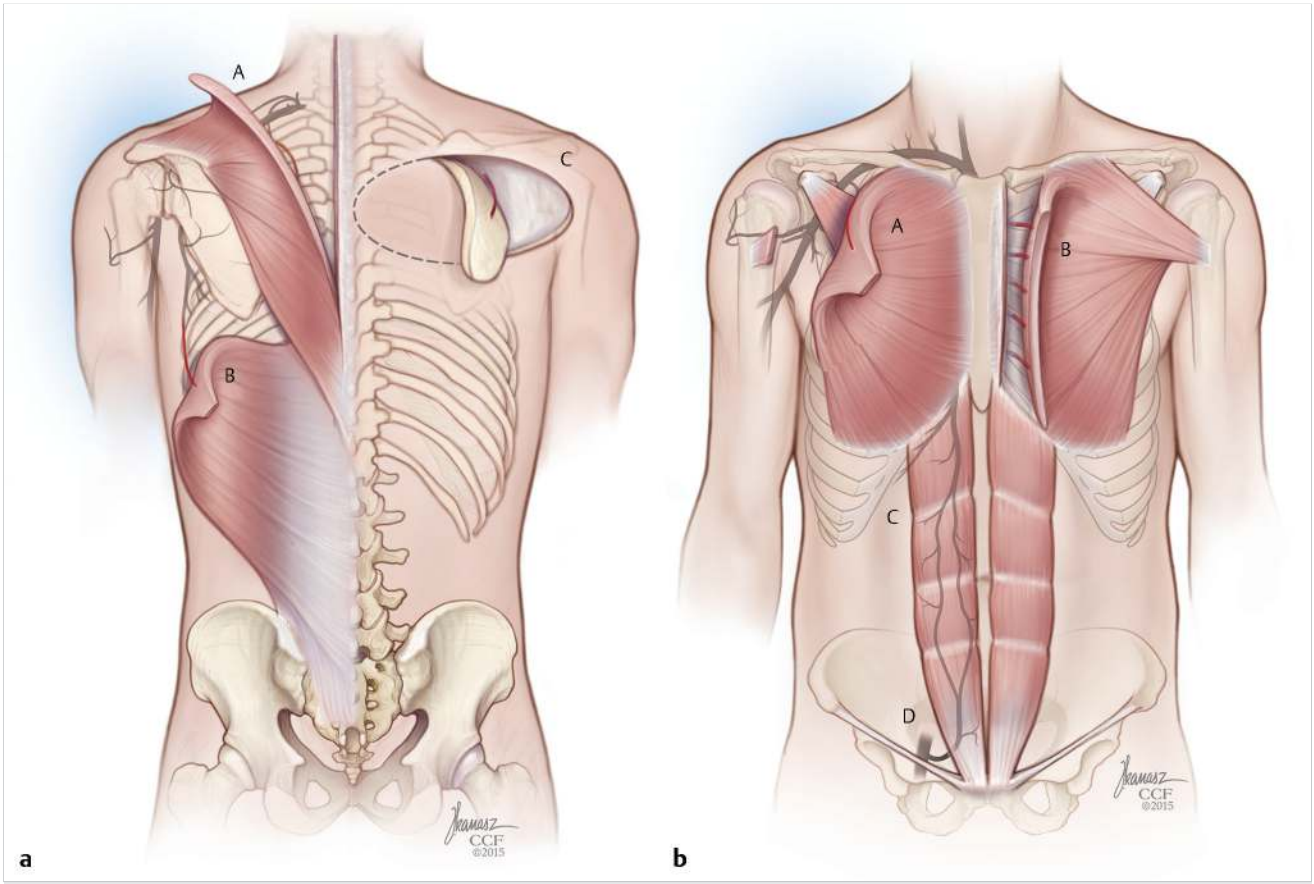


Fig. 4.1 Vasculature supplying local muscle flaps of the thorax and abdomen. **(a)** Dorsal: A, Pectoralis Major flap (Thoracoacromial artery); B, Pectoralis Major flap (Segmental internal mammary perforator vessels); C, Rectus abdominis muscle flap (Superior epigastric artery); D, Rectus abdominis muscle flap (Deep inferior epigastric artery) **(b)** Frontal: A, Trapezius flap (Transverse cervical artery); B, Latissimus dorsi (Thoracodorsal artery); C, Scapular flap (Circumflex scapular artery). Reprinted with permission, Cleveland Clinic Center for Medical Art & Photography ©2016. All Rights Reserved.

The superior thoracic artery arises from the first part of the axillary artery as its only branch and travels along the pectoralis minor border. It supplies the superior portion of the serratus anterior as well as the first and second intercostal spaces.

The thoracoacromial artery is a very short trunk that is deep to the pectoralis minor. It gives rise to four branches: pectoral, acromial, clavicular, and deltoid. The pectoral branch travels in between the pectoralis major and minor with small branches supplying blood to them. Small perforators also supply the skin on the superior and lateral aspect of the chest. The acromial branch goes over the coracoid process and under the deltoid muscles supplying these muscles. The clavicular branch provides blood supply to the subclavius muscle and the deltoid to the pectoralis major and deltoid muscles.[12,13].

The lateral thoracic artery usually travels at the lower border of the pectoralis minor. It supplies the superior portion of the serratus anterior. It has lateral perforators that supply the breast parenchyma as well as the superior and lateral portion of the skin and subcutaneous tissue of the thorax.[17]

The largest branch of the axillary artery is the subscapular artery. It supplies the latissimus dorsi and also provides branches to the inferior portion of the serratus anterior. It begins its course at the lower border of the subscapularis muscle and gives off two branches: the thoracodorsal and circumflex scapular arteries.[16] The branch that supplies the serratus is usually found superficial to the muscle accompanying the long thoracic nerve.

The anterior and posterior circumflex humeral arteries are also branches of the third part of the axillary artery. The anterior circumflex artery runs in front of the neck of the humerus and deep to the coracobrachialis and biceps brachii muscles. It provides blood to the biceps brachii and the deltoid muscle. The posterior humeral circumflex runs posterior to the axillary nerve through the quadrangular space. It also supplies the deltoid muscle as well as provides branches to the teres minor.[18]

Finally, the anterior and posterior intercostal arteries also play a role in the perfusion of the chest wall. The anterior portion arises from the internal thoracic artery, while the posterior portion arises from the thoracic aorta or the costocervical trunk (a branch of the subclavian artery).[19,20]

4.4 Abdomen

The abdominal activity and the abdominal wall make up most of the anterior trunk. It provides flexibility and facilitates mobility. It also provides protection for the abdominal organs.

4.4.1 Fascia

Immediately below the skin, we find Camper's fascia, followed by Scarpa's fascia. Camper's fascia is more prominent below the umbilicus. As it progresses inferiorly, it becomes continuous with the superficial thigh fascia of the thigh. Scarpa's fascia is connected to the external oblique muscle that is immediately posterior to it. It forms the fundiform ligament, which is found at the dorsum of the penis.[15] At the inferolateral portion of the abdominal wall, it is continuous with the fascia lata of the thigh, just below the inguinal ligament. In males, toward midline, Scarpa's continues over the penis and scrotum, forming dartos fascia. In females, it continues into the labia majora and forms the fascia of Colles.[4]

4.4.2 Skeletal Structure

The middle and inferior portion of the trunk lacks the anterior bony structure that is seen in the chest cavity. The anterior abdominal wall lacks bony structures, giving the trunk mobility and flexibility. The posterior skeletal frame and the most inferior aspect of the trunk (vertebral column and the bony pelvis) will be described later in this chapter.

Although not part of the anterolateral portion of the abdomen, the costal margin of the chest is the superior boundary of the abdomen. Inferiorly the pubic tubercle defines the inferior portion of the abdomen.

4.4.3 Muscles

The anterolateral abdominal wall is an extensive association of muscles that extends from the costal margin to the inferior portion of the trunk. The rectus abdominis, external oblique, internal oblique, transverse abdominal muscles, and their respective aponeurosis form this structure. Several anatomical structures are formed at the boundaries of these muscle groups.

The structure that is found in the midline of the abdomen is known as linea alba. This predominantly fibrous white line is formed by the convergence of the aponeurosis of the abdominal muscles. It runs from the xiphoid process to the pubic symphysis. It separates the right and left rectus abdominis muscles. In this line, the umbilicus is situated, which is a scar that arises after the obliteration of the umbilical cord.

The rectus muscles are encased by the aponeurosis of other abdominal muscles that is known as rectus sheath. At approximately one-third of the distance between the umbilicus and the pubic symphysis, we can identify a horizontal line known as the arcuate line. Between the costal margin and this line, the internal oblique aponeurosis divides into an anterior section that passes over the rectus muscle and a posterior section that passes under the rectus. The anterior internal oblique aponeurosis is joined by the external oblique tendinous portion to form the anterior rectus sheath. The posterior rectus sheath is formed by the posterior aspect of the internal oblique aponeurosis and the transversalis aponeurosis. Below the arcuate line, the aponeurosis of the external, internal, and transverse abdominal muscles travel anterior to the rectus muscle. The lack of posterior sheath at this level is what creates the arcuate line.

Just lateral to the rectus muscles, we can find the linea semilunaris. This fibrous line can be found extending from the cartilaginous portion of the ninth rib to the pubic tubercle. It is formed by the internal oblique aponeurosis, as it divides to enclose the rectus muscle.

The rectus abdominis is a long flat muscle that is found on both sides of the abdominal wall. It originates at the pubic crest and inserts to the costal cartilages of ribs 5 through 7 and xiphoid process of the sternum. It is crossed by three bands of fibrous tissues called tendinous intersections. This muscle assists in the flexion of the lumbar spine and forceful exhalation. Its blood supply derives from the superior and inferior epigastric arteries. Due to the presence of these two dominant pedicles, the muscle is considered a type III flap by the classification of Mathes and Nahai. It has been extensively utilized as a flap in breast reconstruction as well as for perineal defects.[5] Innervation is accomplished by the last six thoracoabdominal nerves (T7–T12), which travel through the anterior rectus shealth.[21]

The pyramidalis muscle is also contained in the rectus sheath, anterior to the rectus abdominis. This small triangular muscle originates at the pubic symphysis and pubic crest and inserts at the linea alba.[4] It serves to tense the linea alba. It is supplied by the same arterial vasculature supplying the rectus muscles.

The external oblique muscle is one of three flat muscles that forms the anterolateral abdominal wall. It originates from the fifth through the twelfth ribs and inserts in the iliac crest, pubic tubercle, and the linea alba. It is the largest and most superficial of the three lateral abdominal wall muscles. The medial aspect of the muscle has a tendinous aponeurosis that in the midline makes part of the anterior rectus sheath. The inferior portion of the aponeurosis inserts into the anterior superior iliac spine and the pubic tubercle, forming a defined edge known as inguinal ligament. The external oblique muscle has a segmental blood supply via the lateral cutaneous branches of the inferior eight posterior intercostal arteries. This segmental vascularization classifies this muscle as a type IV flap. This muscle finds uses in coverage of the chest, posterior trunk, and abdomen, as well as in breast reconstruction.[5]

Just deep to the external oblique, we can find the internal oblique muscle. The muscle originates from the iliac crest, thoracolumbar fascia, and the inguinal ligament. It inserts in the 10th, 11th, and 12th ribs, as well as at the linea alba medially. Its aponeurosis is part of the rectus sheath. The inferior aspect of the muscle, corresponding to the muscle fibers that originate from the inguinal ligament, forms an arch over the internal portion of the inguinal ligament and joins the aponeurosis of the transverse abdominal muscle forming the conjoint tendon. This muscle's circulation is derived from a dominant pedicle, a minor pedicle, and a secondary segmental blood supply which classify it as a type V flap. The dominant pedicle is the ascending branch of the deep circumflex iliac artery. The minor pedicles are the lateral branches of the deep inferior epigastric artery and the secondary segmental supplies are derived from the lower thoracic and lumbar arteries. It is used in the coverage of groin, perineal, and greater trochanter wounds. It has also been used as a free flap to the head and neck, upper extremity, and lower extremity.[5]

The deepest of the abdominal wall muscles is the transverse abdominal muscle. It originates from the iliac crest, thoracolumbar fascia, inguinal ligament, and costal cartilage of the 7th through 12th ribs. The muscle inserts into the xiphoid process, linea alba, and pubic crest. Similar to the other two muscles, the aponeurosis forms the rectus sheath as well as the conjoint tendon.

The external oblique, internal oblique, and transverse abdominal muscles assist in the flexion and rotation of the vertebral column. They also help increase intra-abdominal pressure by pulling the chest cavity downward. These muscles derived their blood supply from the lower thoracic intercostal vessels. They are innervated by the thoracoabdominal nerve routes extending from T7 to T12 and L1.[4]

4.4.4 Vasculature

The abdominal wall has an extensive network of vessels that supply the different muscles and soft tissues. There are several anastomoses throughout that ensure adequate collateral flow and can be very helpful for the harvesting of grafts.

As a continuation of the thoracic intercostal vessels, the lumbar arteries supply a portion of the anterolateral abdominal wall. They derive from the posterior portion of the abdominal aorta. They travel horizontally toward midline, crossing behind the sympathetic chain and on the right, posterior to the vena cava. They perforate the transverse abdominal muscle and travel in between this muscle and the internal oblique.

The superior epigastric artery supplies the superior portion of the abdominal wall. It arises from the internal thoracic artery at the levels of the sixth and seventh costal cartilage and anastomoses with the inferior epigastric vessel at the umbilicus. Superiorly, it travels anterior to the transverse abdominal muscle and perforates the rectus muscle.[22]

The inferior epigastric artery supplies the lower portion of the abdominal wall. It is a branch of the external iliac artery and arises just above the inguinal ligament. It pierces the transversalis fascia passing anterior to the arcuate line dividing into a medial and lateral branch. Several perforates arise from these branches supplying the subcutaneous tissue skin overlying the abdominal wall.[22] It continues to ascend in between the rectus muscle and the posterior rectus sheath until it anastomoses with the superior epigastric artery.

Another branch of the external iliac artery, the deep circumflex iliac artery, arises opposite to the inferior epigastric artery. It travels along the iliacus, posterior to the inguinal ligament, toward the anterior superior iliac spine where it gives an ascending branch. It anastomoses with the lateral femoral circumflex artery at this level. The artery then pierces the transversalis fascia and travels along the inner lip of the iliac crest where it gives rise to periosteal branches. It then perforates the transverse abdominal muscle and travels between this muscle and the internal oblique muscle to anastomose into the iliolumbar and lumbar vessels.[22]

The superficial epigastric artery can be found arising from the femoral vessel, below the inguinal ligament. It travels through the femoral sheath and distally it continues anterior to the inguinal ligament.[23] The artery then travels in the subcutaneous tissue in the lower abdomen supplying the overlying skin and adipose tissue. The femoral artery also gives rise to the superficial circumflex iliac artery, which travels over the inguinal ligament in the superficial fascia along iliac crest. It supplies the skin and subcutaneous tissue in the inferior portion of the abdominal wall.[24]

4.5 Pelvis

The most inferior aspect of the trunk is known as the pelvis. As opposed to the superior portion of the trunk, where the differences are mostly related to appearance and the presence of breasts, the pelvis has obvious differences between both men and women. The overall architecture of the pelvis is similar, consisting of a bowl-shaped bony structure with muscles and connection to the lower extremities. However, the perineal anatomy and underlying organs differ between sexes. In the next section, the anatomy of the pelvis will be reviewed.

4.5.1 Skeletal Structure

The pelvis is formed by the iliac bones bilaterally. It is formed by three separate elements known as ilium, ischium, and pubis. In adults, these three are fused into one bone as opposed to infants.

The ilium is divided into two parts: the ala and the body. The arcuate line and the margin of the acetabulum separate the two parts of this bone. The ala is the lateral boundary of the pelvis. Its anterior and posterior borders have two projections: anteriorly they are known as the anterior superior iliac spine and the anterior inferior iliac spine, and posteriorly the posterior superior iliac spine and posterior inferior iliac spine.[4] The external portion of the body of the iliac bone participates in the formation of the acetabulum and it has an articular segment as well as a nonarticular one that forms the acetabular fossa.

The ischium forms the inferior and posterior aspect of the hip bone. It also participates in the formation of the acetabulum. Anterior to the ilium and ischium, we can find the pubic bone. The ischium has an ischial body, an ischial spine, a tuberosity and a ramus. The inferior ramus of the pubic bone units with the iscial ramus to form the obturator foreman.[21]

The sacrum and coccygeal anatomy were described in the back section. These bones articulate with the iliac bones posteriorly to form the sacroiliac joint. As opposed to most of other body joints, its main function is to stabilize the union between the trunk and the lower extremities and not movement. In women, this joint may actually facilitate movement during delivery.

An anterior joint is also formed by the symphysis pubis. It is formed by the pubic area of both iliac bones. Similar to the sacroiliac joint, the symphysis pubis is not mobile and only participates in the stabilization of the pelvis.

4.5.2 Muscles and Genitalia

The bony pelvis provides an open structure; however, there is partial closure that is achieved by a series of muscles known as pelvic floor and the perineum. Because of the structure of the pelvic floor muscles, just inferior to it and lateral to the anal canal, the ischioanal fossa is defined. It is deprived of muscular fibers and filled with adipose tissue.[4,16,21]

Pelvic Floor

There are two important muscles that form the pelvic floor: the levator ani and the coccygeus muscles. They work together to form a concave structure in between the bony pelvis. In the medial portion, there are defects that serve as conduits for the rectum, vagina, and urethra in females and prostate in males.

The levator ani originates in the inner surface of the sidewalls of the lesser pelvis and inserts into the contralateral levator, the inner surface of the coccyx, and the viscera that goes through it. The fibers of the levator that unite with the muscles from the

opposite side as well as those fibers of the external sphincter form the anococcygeal body at midline. It extends from the coccyx to the margin of the anus.

Although the muscle is one single entity, it is divided into four parts that reflect the insertion and direction of its fibers:

1. Puboprostaticus (men): Most medial fibers of the levator, it originates in the pubis and incorporates into the prostatic fascia.
2. Pubovaginalis (women): Originates in the pubis medially and inserts into the vaginal wall.
3. Puborectalis: Lateral to the puboprostaticus/pubovaginalis. Its course is from the pubis to the rectum. It travels posterior to the rectum and along with its longitudinal muscle fibers, it inserts into the fibrous tissue and perianal skin around the anus.
4. Pubococcygeus muscle: Fibers are found lateral to the puborectalis. It extends from the pubis and continues up to the anococcygeal body and coccyx.
5. Iliococcygeus: Is the most lateral portion of the levator. Its fibers course from the obturator fascia and the ischial spine to the anococcygeal body and the coccyx.

Blood supply to the levator ani is provided by the inferior gluteal artery. The innervation to the muscle is provided by the levator ani and inferior rectal nerves as well as the coccygeal plexus. All derive from S3 and S4 branches.

The coccygeus muscle is a triangular muscle that courses from the ischial spine to the lateral border of the sacrum and the anterior face of the last two sacral vertebrae. It is located posterior to the levator ani. It is innervated by S4 nerves.

The pelvic floor muscles are encased by fascia that is thin in the inferior aspect of the muscles but that is thickened in the endopelvic area.

Perineum

The perineum is composed by the fibromuscular structures that close the inferior opening of the pelvis. There are several differences between the men and female perineum; however, they are organized into two planes: a deep urogenital diaphragm and a superficial plane associated with the anus and genitals.

When the pelvis is examined from its inferior opening, it can be approximated by two triangles: the anterior triangle or urogenital triangle and the posterior triangle or anal triangle. Both have vertices corresponding to the ischial tuberosities. The apex of the anterior triangle corresponds to the symphysis pubis and the coccyx corresponds to the posterior triangle.

In the anterior triangle, we find the deep transverse perineal muscle. It is a triangular muscle that originates from the inferior rami of the ischium and inserts into the contralateral muscle forming a dense fibromuscular structure known as the perineal body that is anterior to the anus and posterior to the urogenital organs. It is involved not only in support of the viscera but also in ejaculation and urination. It is innervated by the pudendal nerve from sacral roots 2 to 4.

We can also identify the external sphincter muscle of the urethra in the anterior triangle. It surrounds the membranous urethra. In men, it extends distally and surrounds the prostate. In women, there we can also find the urethrovaginal sphincter muscle, whose fibers surround the female urethra and the vagina. It is involved with continence and urination: upon relaxation, there is flow of urine through the urethra and upon contraction there is interruption of it. It is innervated by the perineal branch of the pudendal nerve.

In the superficial plane of the perineum, we can find muscles with variations depending on patient gender. This is the only muscle found on the posterior aspect of the rectum at this level. Its fibers travel around the anal canal from the perineal body to the anococcygeal raphe. It resembles an arch that surrounds the lateral, anterior, and posterior aspects of the anal canal maintaining fecal continence. It is innervated by the rectal branch of the pudendal nerve.

Male

The penis consists of the root, body, and glands. It is made by three columns: two are located on the dorsal side and are known as corpora cavernosa and the third column is located ventrally and is called corpus spongiosum. They are surrounded by a fibrous layer called tunica albuginea.[25] In the proximal penis, the corpus spongiosum becomes the bulb and the corpora cavernosa the crura.

The erectile tissue is associated with the ischiocavernosus and bulbospongiosus muscles. The ischiocavernous muscle fibers cover the corpora cavernosa ending in the tunica albuginea. It originates from the ischial tuberosity and inserts in the crus of the penis. The bulbospongiosus muscles are located at midline. They travel from the perineal body forward, forming a raphe at their junction in the midline. The fibers at the origin encase the erectile tissue. Posterior fibers form a thin layer that may be absent on the most distal portions of the penis. Anterior fibers travel along the sides of the corpora cavernosa and eventually insert into the corpora themselves. Middle fibers encase the bulb of the penis. Blood supply for these muscles derives from the perineal artery and their innervation is performed by the perineal branch of the pudendal nerve. Their main function is to maintain erection and in the case of the bulbospongiosus, they assist in ejaculation.

Female

The corpora cavernosa are smaller in women and form the clitoris. The corpus spongiosus is substituted for the vestibular bulbs. The ischiocavernous muscle is similar to that found in men as it associates with the corpora cavernosa.

The bulbospongiosus muscles are significantly different from the male counterpart as they are separated by the vagina. They originate from the perineal body and travel on either side of the vagina, covering the bulb of the vestibule, the superficial transverse perineal muscles, and the greater vestibular glands. In addition, women have an additional muscle that originates in the perineal body and goes around the vagina: constrictor muscle of the vulva.

From a functional standpoint, these muscles participate in the erection process as well as sexual intercourse. Innervation and blood supply is also done by the perineal branch of the pudendal nerve and the perineal artery.

In both men and women, there is a fascia that covers the bulbospongiosus, ischiocavernosus, and superficial transverse perineal muscles and is known as Gallaudet fascia or deep perineal fascia.[26] In males, fascia of the penis is continuous with this fascia.

4.5.3 Other Differences between Females and Males

Male

The male genitalia, besides the penis, also are significant for the presence of testicles. They are surrounded by thickened skin with hair follicles known as scrotum. Immediately below the skin we can identify dartos fascia which is responsible for regulating temperature for optimal spermatozoid function.[27] There is also an extension of the internal oblique muscle into the testicles known as the cremaster muscle. It inserts into the fascia and helps retract the testicles.

Colles fascia, a continuation of the abdominal Scarpa's fascia, is continuous with dartos fascia. In addition, there is a thickened extension of Scarpa's from the linea alba to the lateral aspects of the penis known as fundiform ligament.[28] This ligament, in conjunction with the suspensory ligament of the penis that is attached to the pubic symphysis, helps support the penis during erection and sexual intercourse.

Female

The most lateral aspect of female genitalia is called labia majora. It is characterized by the presence of thick skin with multiple hair follicles. Immediately below the skin, we find a continuation of the abdominal Camper's fascia that contains adipose tissue. Deep to it, we find Colles' fascia, which is similar to males, is a continuation of Scarpa's. This fascia is integrated into the fascia lata of the medial thigh and forms the perineal thigh crease at the level of the ischiopubic rami.[28]

Medial to the labia majora, the labia minora can be identified at either side of the vaginal opening. They are covered with squamous cell epithelium and are protected by lubricant produced by its glands. The vaginal opening is found at midline and is also covered with squamous epithelium.

4.5.4 Vasculature

The vascular supply of the pelvic area for both males and females derives from branches of the internal iliac vessels (internal pudendal) and the femoral vessels (superficial and deep external pudendal arteries). A brief summary of the main branches seen are described below.

1. Inferior rectal artery: This artery derives from the internal pudendal. This vessel pierces the obturator internus fascia and its branches supply the gluteus maximus and the anal canal. It anastomoses with the superior and middle rectal arteries and the perineal artery.
2. Superficial external pudendal artery: This artery derives directly from the femoral artery. It travels medially over the spermatic cord/round ligament and supplies branches to the penis and scrotum in men and labia majora in women.[29]
3. Deep external pudendal artery: This artery derives from the femoral artery. As it travels medially along the adductor longus, it is encased by the fascia lata. In men, it gives rise to the external and internal anterior scrotal arteries supplying the scrotum. In women, it gives rise to the anterior labial artery that is responsible for supplying a portion of the labia majora.[16]
4. Perineal artery: This artery derives from the internal pudendal. It runs parallel to the junction of the inferior pubic rami and the pubis, in between the bulbocavernosus and the ischiocavernosus muscles. After supplying these two muscles, in men, it continues toward the base of the penis to give rise to the posterior scrotal branches: the internal and external posterior scrotal arteries that supply the dorsum of the scrotum and the spermatic cord fascia, respectively.[30] In women, it gives rise to the internal and external posterior labial arteries that supply the labia majora and labia minora.[16] The internal pudendal artery is the dominant pedicle in the Singapore flap, also known as the Pudendal-Thigh flap. It is a type A fasciocutaneous flap with important utility in the reconstruction of the vagina and scrotum and defects of the perineum.[5]
5. Dorsal and deep arteries of the penis (men)[4]: They are both derived from the internal pudendal artery. The dorsal artery ascends into the penis between the crus and the symphysis pubis, in between the dorsal nerve and dorsal vein. It supplies the fascia of the corpora cavernosum on the dorsal aspect. The deep artery is situated in between the fascia of the urogenital diaphragm and after piercing it travels into the corpora cavernosum to supply it.
6. Dorsal and deep arteries of the clitoris (women): Both arteries are terminal branches of the internal pudendal artery. The dorsal artery of the clitoris pierces the fascia of the urogenital diaphragm to reach the dorsum of the clitoris, prepuce, and glans. Similarly, the deep artery of the clitoris also pierces through the urogenital diaphragm and supplies the crus of the clitoris.[21]

4.6 Posterior Trunk

The stability and mobility of the trunk is dependent on the skeletal structure and muscles of the back. It provides protection as well as support for motility. Despite having the same underlying muscles, the appearance of the posterior trunk may differ between men and women. The typical male back is triangular in appearance with a broad proximal portion. Females, on the other hand, will have a back resembling an hour-glass.

These soft tissues of the posterior trunk are covered with skin that is characterized by a thicker dermis than that of the rest of the body. The spinous processes are covered by a dense fascia that forms adhesions to the dermis, providing protection from shear trauma.

In addition, we can also find the thoracolumbar fascia. This membrane covers the muscles of posterior trunk and is made up of an anterior, middle, and posterior layer.[31] On the most proximal portion, it can be found superficial to the serratus posterior superior and it is continuous with the nuchal fascia. It can be found medially attached to the spinous processes of thoracic vertebrae and laterally to the angle of the ribs. On the most distal portion, it extends into the posterior iliac spine. The quadratus lumborum is encased in between the anterior and middle layers while the erector spinae is encased in between the posterior and middle layers.

4.6.1 Skeletal Structure

The mainframe of the neck and trunk is the vertebral column. It is located on the posterior aspect of the trunk and it extends

from the cranium to the pelvic bony structure. It comprises 32 to 34 vertebrae divided into 7 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 3 or 5 coccygeal vertebrae. The last two groups are fused to form two solid bones: the sacrum and coccyx.

The functions of the vertebral column include support, motility, and protection. The entire weight of the head and neck, trunk, and upper extremities is sustained by this structure and is transmitted to the lower extremities. The flexibility provided by the articulations between the vertebrae allows for extensive motion. Finally, the column forms a protective cavity in its posterior aspect that encases the spinal cord.

Although part of the upper extremity bony structure, the scapula is associated with several back muscles. This flat and triangular bone lies over the posterior aspect of the first seven ribs. The anterior portion has a concavity that is called the subscapular fossa and faces the thoracic cavity. The posterior aspect faces the back skin and contains a transverse elevation known as the spine of the scapula. Immediately above the spine, we find the supraspinous fossa where the supraspinatus muscle originates immediately below the infraspinous fossa where the infraspinatus muscle originates. On the lateral aspect of the scapula, we find the acromion, a long triangular border that receives insertions of different muscles.

4.6.2 Muscles

Multiple muscles that are present on the posterior aspect of the trunk are intimately associated with upper extremity motion as well as with movement of the spine and body. The groups of muscles can be grouped into three planes: deep, intermediate, and superficial. The deep plane is made by the paraspinous muscles; the intermediate plane is formed by the serratus posterior muscles; the superficial plane is formed by muscles that are closely associated with the upper extremities.

Deep Plane

Deep plane is made by the paraspinous muscles. They extend from the cranium to the sacrum. From a functional point of view, these muscles are responsible for the extension and rotation of the vertebral column. They have been divided into four groups according to their location in the posterior trunk: the deepest layer known as the transversospinal muscles that includes the rotators, semispinalis, and multifidus muscles; the intermediate layer of muscles, named the erector spinae muscles that includes the longissimus, spinalis, and interspinalis muscles; and the most superficial layer known as the spinotransverse muscles that include the capitis and splenius cervicis muscle.[32] The paraspinous muscles have a dual segmental blood supply of the posterior intercostals as well as branches of the lumbar arteries. This is a type C fasciocutaneous flap with uses in sacral and spinal defect coverage.[5]

Intermediate Plane

The serratus posterior superior and inferior lay between the paraspinous muscles and the superficial plane muscles. They are thin and elongated distinct muscles. The superior originates from the spinous processes of C7–T3 and inserts into the upper borders of ribs 2 to 5. The inferior serratus originates from the spinous processes of T11–L2 and inserts into the inferior borders of ribs 9 to 12. The serratus posterior superior elevates the ribs participating in inspiration while the inferior depresses the ribs and participates in expiration. Blood supply is derived from corresponding intercostal vessels. Innervation of both muscles is done by the intercostal nerves, with second to fifth nerves supplying the superior and ninth to eleventh nerves supplying the inferior.

Superficial Plane

The trapezius muscle is a large, flattened, and triangular muscle that when seen with the contralateral side forms a characteristic trapezium. It originates from the nuchal ligament, ligamentum nuchae, and spinal processes of vertebrae C7–T12. It inserts into the clavicle, acromion process, and the scapular spine. The trapezius intervenes in most of the motion that involves the scapula with the different portions of the muscle elevating, retracting, or externally rotating this bone. The dominant blood supply is provided by branches of the transverse cervical artery. The muscle also has three minor pedicles (dorsal scapular vessels, branches of the occipital vessels, as well as posterior intercostal perforators) which make it a type II flap. It is used in coverage of defects involving the skull, posterior trunk, and shoulder.[5] Finally, the trapezius is innervated by the spinal accessory nerve (CN XI) and C3 and C4 cervical nerves.

Another group of muscles involved in the retraction of the scapula are the rhomboid major and minor. They are immediately under the trapezius muscles and are part of the posterior trunk musculature. The muscles originate in the spinous processes of C7 through T5 and insert in the medial border of the scapula. The fibers of the rhomboid minor are superior to those of the rhomboid major. Besides retracting, in combination with the pectoralis minor, contraction of the muscle can help internally rotate the scapula. The blood supply of these muscles is provided by the dorsal scapular artery and the innervation by the dorsal scapular nerve (C4 fibers).[21]

The levator scapula is a flat and elongated muscle that is deep to the scalene muscles and hidden by the sternocleidomastoid and trapezius muscles. It originates from the transverse processes of C1–C4 vertebrae and inserts in the medial border of the scapula. It assists the trapezius in elevation of the scapula and can assist in the internal rotation of the scapula. Blood supply is provided by the dorsal scapular artery. Innervation is carried out by the dorsal scapular nerve (C5 fibers).[4]

The latissimus dorsi is a triangular flat muscle that covers the posterior and inferior aspect of the trunk. The superior portion of the muscle is covered by the trapezius. The muscle originates from the thoracolumbar fascia, posterior aspect of the iliac crest, external aspect of the four last ribs, and the scapula. The muscle participates in the extension, internal rotation, and adduction of the arm. The dominant blood supply is from the thoracodorsal artery. It also receives secondary segmental blood supply from lateral and medial perforating vessels, from the lumbosacral fascia and lumbar arteries. It is classified as a type V flap and has found extensive uses in coverage of chest, abdomen, and head and neck defects. This is a true workhorse flap that is used in reconstruction both as a local flap as well as a free tissue transfer with relatively minor donor-site morbidity and a potential for large defect coverage.[5] In the medial aspects,

the lumbar and intercostals also supply the muscle. The thoracodorsal nerve supplies the latissimus dorsi.

The subscapularis muscle is directly involved with arm motion. This muscle is triangular in shape and originates in the subscapular fossa, occupying the anterior portion of the scapula. It inserts into the lesser tubercle of the humerus, making this muscle a participant in the medial rotation and adduction of the arm. It forms most of the posterior axillary wall. Blood supply to this muscle is provided by the subscapular artery. Innervation is via the superior and inferior subscapular nerves.

A thick and short muscle, the supraspinatus originates in the supraspinous fossa of the scapula and inserts into the greater tubercle of the humerus. The tendon travels superior to the glenohumeral joint and just below the acromion. It participates in the abduction of the arm. Its blood supply derives from the suprascapular artery and its innervation from the suprascapular nerve.[33]

The infraspinatus is a triangular muscle that originates in the infraspinous fossa. Similar to the supraspinatus, it inserts in the greater tubercle of the humerus and is innervated by the suprascapular nerve. It participates in the external rotation of the humerus. The suprascapular and circumflex scapular arteries supply this muscle. Innervation is provided by the suprascapular nerve.

Deep to the infraspinatus, an elongated and flattened muscle can be found: the teres minor. This muscle originates from the external portion of the supraspinous fossa of the scapula, just below the infraspinatus muscle. It covers the posterior aspect of the glenohumeral joint and inserts into the major tuberosity of the humerus. This allows the muscle to externally rotate the humerus. The circumflex scapular artery is responsible for blood supply to this muscle. The axillary nerve innervates this muscle.

The teres major is located deep to the teres minor muscle. It originates from the inferior portion of the infraspinous fossa (near the inferior angle of the scapula) and inserts in the intertubular groove of the humerus. It adducts and internally rotates the humerus. The blood supply of the teres major derives from the thoracodorsal and circumflex scapular artery. It is innervated by the lower subscapular nerve.

4.6.3 Vasculature

The subscapular artery arises from the axillary artery as its largest branch. It closely follows the subscapularis muscle into the inferior portion of the scapula where it anastomoses with the intercostal arteries, the lateral thoracic artery, and the dorsal scapular artery. It gives rise to two branches[34]:

1. *Circumflex scapular artery*: It travels through the triangular space that is defined by the teres minor superiorly, teres major inferiorly, and the triceps laterally.[16] It supplies perforators to the lateral border of the scapula. Covered by the teres minor, the circumflex scapular artery enters the infraspinatus fossa. It anastomoses with the transverse scapular and the dorsal scapular arteries. This is the dominant blood supply for scapular and parascapular flaps. A type B fasciocutaneous flap which can also be raised with as an osteocutaneous (portion of the scapula) flap for mandibular, upper, and lower extremity reconstruction.[5]
2. Thoracodorsal artery: Accompanied by the thoracodorsal nerve, this artery travels inferiorly deep to the latissimus supplying this muscle. It continues to descend and supplies the inferior portion of the serratus anterior. It provides the angular artery that supplies the angle of the scapula and can be found between the serratus anterior and the teres minor.[34]

The suprascapular artery branches off the thyrocervical trunk. It descends deep to the sternocleidomastoid muscles and continues its course by traveling deep and parallel to the subclavius muscle and the clavicle. It then enters the supraspinatus fossa and travels between the supraspinatus muscle and the scapula providing perforators to this muscle. It then descends into the infraspinatus fossa where it supplies the infraspinatus muscle and eventually anastomoses with the circumflex scapular artery and the transverse cervical artery.

The transverse cervical artery is also a branch of the thyrocervical trunk. It courses anterior to the omohyoid and toward the trapezius. Once deep to this muscle, it divides into a superficial and deep branch[16]:

1. Superficial cervical artery: It is found deep to the anterior margin of the trapezius. It courses superiorly and supplies the middle and lateral portions of this muscle.
2. Dorsal scapular artery: This deep branch comes most of the time from the subclavian artery. However, in one-fourth of the population, it can branch from the transverse cervical artery. It passes deep to the levator scapulae into the superior angle of the scapula and then descends under the rhomboid muscles.

The superior gluteal artery is the largest branch of the internal iliac artery. This artery courses posteriorly and passes through the greater sciatic foramen and continues in between the piriformis muscle and the gluteus medius.[35] It finally supplies the gluteus maximus.

The inferior gluteal artery is also a branch of the internal iliac artery. It travels downward through the lesser sciatic foramen, down the piriformis muscle, deep to the pudendal artery, and through the greater sciatic foramen.[35] The superior and inferior gluteal arteries comprise the two dominant pedicles of the gluteal flap. This is a type III flap with uses in coverage of defects of the sacrum, trochanter, and ischium, as well as for the reconstruction of the anus and vagina.[5]

4.7 Review Questions

4.7.1 True or False

1. The transverse abdominis plane (TAP) block is a peripheral nerve block designed to anesthetize the nerves supplying the anterior abdominal wall (T6 to L1). TAP block is performed by infiltration of the local anesthetic in the plane between internal oblique and transversus abdominis.

4.7.2 Choose the Best Answer

2. A 60-year-old male patient with a history of coronary artery bypass grafting using the left internal mammary artery developed a sternal wound that required serial

debridements. The wound is ready for reconstruction. Which of the following flaps cannot be reliably used in this patient?
a) Omental flap.
b) Pectoralis major flap, right sided.
c) Pectoralis major flap, left sided.
d) Rectus abdominis flap, left sided.

3. A 55 year old female patient is considered for unilateral small-to-moderate size autologous breast reconstruction. She has a history of abdominoplasty. All of the following may be an option for reconstruction, except
a) Deep inferior epigastric artery perforator flap.
b) Superior gluteal artery perforator flap.
c) Inferior gluteal artery perforator flap.
d) Transverse upper gracilis (TUG) flap.
4. A 60 year old male patient is preparing to undergo posterior components separation and transverse abdominis release with mesh placement and free tissue transfer for stable soft tissue coverage. Which of the following are the most commonly utilized recipient vessels for microvascular anastomoses?
a) Superior epigastric vessels.
b) Superficial epigastric vessels.
c) Deep inferior epigastric vessels.
d) Internal thoracic vessels.
e) Femoral vessels.
5. Which of the following is true regarding internal thoracic vessels when used as recipient in microsurgical breast reconstruction:
a) The artery has high pressure to allow good inflow.
b) Allow better medial positioning of the flap.
c) Allow earlier physical therapy.
d) Relatively spared after radiotherapy.
e) All of the above.

4.7.3 Answers

1. True.
2. d. Rectus abdominis flap, left sided.
3. a. Deep inferior epigastric artery perforator flap.
4. c. Deep inferior epigastric vessels.
5. e. All of the above.

References

[1] Ellis H. Anatomy of the breast. Surgery. 28:114–116
[2] Naidu BV, Rajesh PB. Relevant surgical anatomy of the chest wall. Thorac Surg Clin. 20(4):453–463
[3] Baek IS, You JP, Rhee SM, et al. A clinical anatomic study of internal mammary perforators as recipient vessels for breast reconstruction. Arch Plast Surg. 40 (6):761–765
[4] Moore K, Agnur A. Essential Clinical Anatomy. Baltimore, MD: Lippincott Williams & Wilkins; 2002
[5] Mathes S, Nahai F. Reconstructive Surgery: Principles, Anatomy, & Technique. New York, NY: Churchill Livingstone; 1997
[6] Ben Hadj Yahia S, Vacher C, Guédon C. [Anatomic study of the location of the thoracoacromial pedicle. Application to the pectoralis major musculocutaneous flap]. Morphologie. 93(300):13–19
[7] Porzionato A, Macchi V, Stecco C, Loukas M, Tubbs RS, De Caro R. Surgical anatomy of the pectoral nerves and the pectoral musculature. Clin Anat. 25 (5):559–575
[8] Steinmann SP, Wood MB. Pectoralis major transfer for serratus anterior paralysis. J Shoulder Elbow Surg. 12(6):555–560
[9] Carlson ER. Pectoralis major myocutaneous flap. Oral Maxillofac Surg Clin North Am. 15(4):565–575, vi
[10] Sanchez ER, Sanchez R, Moliver C. Anatomic relationship of the pectoralis major and minor muscles: a cadaveric study. Aesthet Surg J. 34(2):258–263
[11] Hui KC, Zhang F, Lineaweaver WC, Moon W, Buncke GM, Buncke HJ. Serratus anterior-rib composite flap: anatomic studies and clinical application to hand reconstruction. Ann Plast Surg. 42(2):132–136
[12] Hamada J, Igarashi E, Akita K, Mochizuki T. A cadaveric study of the serratus anterior muscle and the long thoracic nerve. J Shoulder Elbow Surg. 17(5): 790–794
[13] Jorge-Mora MT, Gallego-González S, Corral-Solla EM. Anomalous subclavius muscle insertion as a cause of thoracic outlet syndrome. Rehabilitation. 45 (2):170–172
[14] Taylor I. Grabb and Smith's Plastic Surgery.7th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014;32–34
[15] MacQuillan A, Horlock N, Grobbelaar A, Harrison D. Arterial and venous anatomical features of the pectoralis minor muscle flap pedicle. Plast Reconstr Surg. 113(3):872–876
[16] Neligan P. Plastic Surgery. 3rd ed. Seattle, WA: Saunders; 2013
[17] Kim JT, Ng SW, Naidu S, Kim JD, Kim YH. Lateral thoracic perforator flap: additional perforator flap option from the lateral thoracic region. J Plast Reconstr Aesthet Surg. 64(12):1596–1602
[18] Olinger A, Benninger B. Branching patterns of the lateral thoracic, subscapular, and posterior circumflex humeral arteries and their relationship to the posterior cord of the brachial plexus. Clin Anat. 23(4):407–412
[19] Ellis H. The ribs and intercostal spaces. Anaesth Intensive Care Med. 9:518–519
[20] Hamdi M, Spano A, Van Landuyt K, D'Herde K, Blondeel P, Monstrey S. The lateral intercostal artery perforators: anatomical study and clinical application in breast surgery. Plast Reconstr Surg. 121(2):389–396
[21] Standring S. Gray's Anatomy: The Anatomical Basis of Clinical Practice. 40th ed. London: Churchill Livingstone; 2008
[22] Boyd JB, Taylor GI, Corlett R. The vascular territories of the superior epigastric and the deep inferior epigastric systems. Plast Reconstr Surg. 73(1):1–16
[23] Schaverien M, Saint-Cyr M, Arbique G, Brown SA. Arterial and venous anatomies of the deep inferior epigastric perforator and superficial inferior epigastric artery flaps. Plast Reconstr Surg. 121(6):1909
[24] Koshima I, Nanba Y, Tsutsui T, et al. Superficial circumflex iliac artery perforator flap for reconstruction of limb defects. Plast Reconstr Surg. 113(1):233–240
[25] Brock G, Hsu GL, Nunes L, von Heyden B, Lue TF. The anatomy of the tunica albuginea in the normal penis and Peyronie's disease. J Urol. 157(1): 276–281
[26] Shafik A, Sibai OE, Shafik AA, Shafik IA. A novel concept for the surgical anatomy of the perineal body. Dis Colon Rectum. 50(12):2120–2125
[27] Fisher C, Park M. Penile torsion repair using dorsal dartos flap rotation. J Urol. 171(5):1903–1904
[28] Spirito D. Medial thigh lift and declive: Inner thigh lift without using Colle's fascia. In: Body Contouring: Art, Science, and Clinical Practice. Berlin: Springer Berlin Heidelberg; 2010
[29] La Falce OL, Ambrosio JD, Souza RR. The anatomy of the superficial external pudendal artery: a quantitative study. Clinics (Sao Paulo). 61(5):441–444
[30] Hagerty RC, Vaughn TR, Lutz MH. The perineal artery axial flap. Ann Plast Surg. 31(1):28–31
[31] Bogduk N, Macintosh JE. The applied anatomy of the thoracolumbar fascia. Spine. 9(2):164–170
[32] Manstein ME, Manstein CH, Manstein G. Paraspinous muscle flaps. Ann Plast Surg. 40(5):458–462
[33] Volk AG, Vangsness CT , Jr. An anatomic study of the supraspinatus muscle and tendon. Clin Orthop Relat Res.(384):280–285
[34] Jesus RC, Lopes MCH, Demarchi GTS, Ruiz CR, Wafae N, Wafae GC. The subscapular artery and the thoracodorsal branch: an anatomical study. Folia Morphol (Warsz). 67(1):58–62
[35] Unal C, Yirmibesoglu OA, Ozdemir J, Hasdemir M. Superior and inferior gluteal artery perforator flaps in reconstruction of gluteal and perianal/perineal hidradenitis suppurativa lesions. Microsurgery. 31(7):539–544

5 Surgical Principles: Grafts, Flaps, Microsurgery

Mark Leyngold and Adam J. Katz

Abstract
This chapter examines the different types of grafts and their clinical application in wound reconstruction. Assessment of wounds is discussed, followed by the proper operative techniques for each type of wound. Instructions are also provided involving postoperative care. Receiving special treatment are the various grafts available to the surgeon, including fat, cartilage, bone, tendon, and nerve grafts. The various types of flaps are described, along with the proper technique for flap transfer. Of special significance the attention is given to the principles and techniques of microsurgery paramount, reconstruction of complex defects.

Keywords: skin graft, split-thickness skin graft (STSG), flap, microsurgery, imbibition, Mathes-Nahai musculocutaneous flap classification

5.1 Goals and Objectives

- Describe different types of grafts and their clinical application in wound reconstruction.
- Describe different types of flaps, their clinical application, and relevance.
- Understand the basic concepts of applications of reconstructive microsurgery, microvascular operative techniques, postoperative management, and outcomes.

5.2 Grafts

5.2.1 Anatomy

The skin varies among individuals depending on their age, ethnicity, genetics, diet, sun exposure, and area of the body. In early childhood the skin is thin, but from age 10 until about 35 years it continues to progressively thicken. Sometime in the fourth decade, the dermis starts to thin again, elasticity decreases and sebaceous skin content decreases, and this phenomenon is progressive until the person dies.

The skin also varies according to area of the body, for example, the skin of the eyelids, upper extremity, medial thigh, and postauricular area is thinner than skin on the buttock area, palms, and soles of the feet. Approximately 95% of the skin is dermis, while the other 5% is epidermis.[1] The dermis contains sebaceous glands. Sweat glands and hair follicles are located in the subcutaneous fat just beneath the dermis. The skin vasculature is superficial to the superficial fascia and parallels the skin surface.

5.2.2 Terminology and Properties of Skin Grafts

Split-thickness skin graft (STSG) contains epidermis and variable amount of dermis. It can be used to resurface large surface areas as the donor site does not require closure and generally can be left to heal via secondary intention. STSG has good "take" and vascularizes well. On the minus side, thin STSG tends to shrink considerably, pigment abnormally, and is very susceptible to trauma.[2] Full-thickness skin graft (FTSG) contains epidermis and the entire dermis. The donor site, unlike that of STSG, has to be closed primarily or reconstructed with a STSG or other means making this type of graft limited in supply. FTSGs are thicker and therefore require a well-vascularized wound bed to survive. On the plus side, they have better color match, have less secondary contraction, and tend to look better and feel more natural when compared to STSGs. Common donor sites for FTSG include, but are not limited to, the supraclavicular area, pre- and postauricular skin, the neck region, forearm/arm, abdomen, and the groin region. Common STSG donor sites include inner and outer thigh region, buttock region, and scalp.

5.2.3 Graft Take

Skin graft take occurs in three phases. The first phase consists of serum imbibition and lasts 24 to 48 hours. This is followed by an inosculatory phase and a process of capillary ingrowth that occurs essentially simultaneously, until generalized blood flow has been established by the fifth or sixth postoperative day (▶ Table 5.1).

Retention of the function of skin appendages often depends on graft thickness. Thin grafts often have deficient functions of sebaceous glands and thus have dry and brittle appearance. Only FTSGs retain the full ability to grow hair and have normal sweat and oil secretion.

5.3 Preoperative Considerations

Wounds considered for skin grafting must have a well-vascularized recipient bed. If a wound does not have a vascularized bed, a flap which by definition brings its own blood supply should be considered. Other factors such as cleanliness of the wound, absence of infection, nutritional status, and the presence of healthy granulation tissue should be considered. A value of 10^5 CFU/g of tissue is a good indicator and prognostic factor for graft take; however, nothing can substitute for sound clinical judgment from an experienced surgeon in regard to wound condition. Thus, unhealthy appearance far supersedes the value of microbiologic studies.

Table 5.1 Phases of skin graft healing

Phase	Days	Physiologic event
1. Plasmatic imbibition	0–2	Provides initial support to the graft during post–graft ischemia. Graft gains fluid from edema
2. Inosculation	2–4	Connection of graft and host vessels
3. Revascularization	5–7	Restoration of flow in inosculated vessels and formation of new vascular channels to supply the graft

For wounds amenable to skin grafting, the next important decision is whether to use an FTSG or an STSG. Despite better take of STSG and ability to reconstruct larger surface areas, one needs to consider the degree of graft contracture and cosmetic appearance. The decision should be individualized based on specific patient and anatomic wound location. For instance in areas such as over a joint (i.e., elbows, axilla, and neck) or facial region, FTSG would likely produce a superior outcome due to lower rate of secondary contracture and better color match.[2]

5.4 Operative Technique

5.4.1 Split-Thickness Skin Graft Harvest

Thigh wound should be prepped widely or circumferentially. Any povidone iodine should be completely removed prior to harvest. The size of the graft required should be known and measured out and marked on the thigh. Generally, the graft should be 10 to 15% larger than the size of the wound unless meshing is planned. Lateral or medial thigh regions can be used and it is preferable to harvest the graft higher toward the groin to avoid visible scars. Mineral oil is commonly used to lubricate the skin prior to harvest. The harvest is commonly performed with a dermatome set at appropriate thickness and width. Thickness is often set at 0.014 to 0.015 in and can be checked with a no. 15 blade. Tension on the skin is applied with proximal and distal traction. The dermatome is angled at 45 degrees, and with one smooth motion the graft is harvested and placed in sterile saline. The donor site should be immediately covered with epinephrine solution–soaked sponges to prevent excessive bleeding. If meshed graft is desired, meshers are available with variable templates for meshing from 1.5:1 to 3:1 ratios. The meshed graft is then secured to the recipient wound either with chromic sutures or staples. The graft is dressed with a bolster dressing of choice or a NPWT dressing at 75 mm Hg of suction. The donor site is generally covered with a semiocclusive dressing (i.e., Opsite, Tegaderm, etc.) or xeroform.[3]

5.5 Postoperative Care

A pressure dressing or bolster is left in place for 5 to 7 days and then the graft is examined. If an NPWT dressing is used, it can be left on for a week. For FTSG, this period is varied and could be anywhere from 5 days for eyelids to 2 to 3 weeks for nipple grafts. If an extremity was grafted, early postoperative immobilization, splinting, compression, and extremity elevation are required. If the graft is adherent, Xeroform dressing or Adaptec is applied and reinforced with gauze and bandages that should be changed every day or every other day for the first 3 weeks to prevent desiccation. This can be switched to Vaseline and/or lotion once the graft is healed to prevent drying out. The patient should continue to avoid any shearing to the graft or excessive ambulation or activity to avoid injuring the new skin graft (▶ Fig. 5.1a,b).

The donor-site dressing remains in place for about 10 days while allowing epithelialization of the wound and generally can be removed after this period. When Xeroform is used, it is typically not removed but gradually trimmed as the wound heals and epithelializes (▶ Fig. 5.1c). It is also important to warn patients that they will have a potentially visible patch of discolored skin on their thighs.[3]

5.5.1 Other Types of Grafts

Fat Grafts

Fat grafting has gained popularity recently among plastic surgeons. It can be a useful adjunct to both reconstructive and cosmetic surgeons. Fat is commonly used in breast reconstruction to correct asymmetries and add volume, in facial rejuvenation

Fig. 5.1 **(a)** A 43-year-old woman with a left leg clean fasciotomy wound after compartment syndrome; (**b**) same patient 2 months after split-thickness skin graft (STSG) reconstruction; (**c**) STSG thigh donor site covered with Xeroform.

as a permanent filler to add volume and restore youthful facial appearance, and in many other challenges such as scar management and body contouring procedures. The literature is replete with studies and reports about fat-grafting techniques and outcomes, and many of these present contradictory findings. This makes a choice of methods/practice confusing for one new to the field. In general, the goal of fat grafting is to achieve a predictable and reproducible outcome. Tissue fragments within the graft survive initially by diffusion, just as with a skin graft. Although relatively simple from a technical standpoint, fat grafting is actually a complex process when viewed in its entirety or from a process engineering perspective. It involves a number of steps, including donor-site preparation, tissue/graft harvest, tissue/graft washing and preparation, tissue/graft delivery, and graft/recipient site preparation and care. Each of these steps is associated with a large number of variables and options that may alter the final outcome of the procedure. For this reason, it is difficult to design and carry out well-controlled studies that will yield robust data.

Although the topic of fat grafting could cover an entire chapter itself, a few basic principles will be mentioned. First, logic would dictate that repeatable and predictable volume maintenance requires that the tissue/graft physiology be returned to normal physiological status as much as possible prior to grafting.[4] A large burden of free oil, for instance, can release fatty acids to the surrounding tissues and/or reduce pH, resulting in cell membrane damage. Ideally, the graft should have a known and reproducible aqueous fraction, as this is a volume that will be lost after grafting via resorption. If the graft has a large aqueous component, it will be easier to inject and will be associated with less shear force, but will require relatively more overcorrection to maintain volume. If the graft is particularly concentrated, it will be harder to push through a cannula and result in more cellular damage from shear stress. To minimize cell trauma and damage, it makes sense to use larger bore syringes (i.e., catheter tip syringe vs. Luer lock syringe) and larger bore cannulae for graft delivery. A standard Luer syringe has an internal diameter of 1.8 mm at the tip. The use of syringes, needles, and cannulae with a diameter of 2.5 to 3.0 mm is likely to result in less shear force and cell damage when all other variables are held constant. Further discussion of the many facets and considerations surrounding fat grafting is beyond the scope of this chapter, but the reader is referred to a number of reviews on the subject.[5,6,7,8]

Cartilage Grafts

Cartilage grafts are often helpful during nasal, ear, or eyelid reconstruction or during rhinoplasty. The three most common sites for cartilage harvest include costal rib cartilage, conchal bowl of the ear, and nasal septum. Cartilage can be harvested with or without perichondrium. The characteristics of the cartilage are usually maintained at the recipient site, and will usually maintain the natural shape of the cartilage. Nasal septal and auricular cartilage are most often used in nasal surgery, as spreader grafts, or tip reconstruction/augmentation. Costal cartilage is most often used for nasal dorsum and external ear reconstruction. Cartilage donor sites are generally inconspicuous and the material itself is indispensable for achieving the desired results.

Bone Grafts

Bone grafts are common and are often used for reconstruction of the mandible, long bones of extremities, and the facial skeleton (▶ Fig. 5.2). Autogenous bone grafts or allografts can be used according to surgeon's preference and the type and extent of the defect. For example, in a patient who needs reconstruction of a large mandibular defect with a history of prior radiation, a free fibula graft is a far superior option as compared to an allograft or a nonvascularized bone graft. In general, allografts have less osteoinductive and osteoconductive properties than autogenous bone grafts but have the distinct advantage of absence of donor-site morbidity. Four general types of autologous bone grafts are described: cancellous grafts, cortical grafts, cortico-cancellous nonvascular grafts, and free vascularized grafts. Cancellous grafts are typically used for reconstruction of the alveolar cleft, nasal reconstruction, or in hand reconstruction after removal of a bone cyst. They tend to vascularize better than cortical grafts but require longer time to gain structural stability and strength. The main mechanism for new bone formation is osteoconduction. Cortical bone grafts take longer to vascularize but may provide better initial stability. They have more limited osteoinductive and osteoconductive properties. Fixation of bone grafts has been shown to reduce graft resorption when grafts are placed under mobile tissues. Some of the common donor sites include calvarium, iliac crest, fibula, rib, and distal radius.[3]

Tendon Grafts

Tendon grafting is useful in the management of various upper extremity injuries and defects. It can be done acutely in one stage if the wound is clean with adequate soft-tissue coverage and tendon donor sites are available outside of the zone of injury. If this is not possible, reconstruction can be performed in two stages. In the first stage, silicone rods can be placed to allow for tendon bed preparation, and in the second stage these can be removed and replaced with an appropriate graft. The palmaris longus and plantaris tendons are ideal tendon grafts as they are expendable and easily accessible. It is important to remember that about 20% of patients lack either a palmaris longus or plantaris tendons.

Fig. 5.2 Iliac bone graft designed for nasal reconstruction.

Nerve Grafts

Nerve grafts are commonly used in hand trauma, upper extremity, and facial reconstruction and facial reanimation. Nerve grafts can be allografts or autografts depending on surgeon's preference. When autograft is used, one has to consider denervation somewhere and sensory deficit. Sural nerve is by far the most common nerve utilized for grafting. It courses along the posterolateral ankle and is easily accessible. It is important to inform the patient that grafted nerve is not immediately healed and it may take months until the final result is achieved. Nerve graft essentially serves as a conduit for axonal growth, which typically begins at 2 weeks after grafting and continues 1 mm/day until the target is reached.

5.5.2 Flaps

A flap is defined as a tissue with its own blood supply. This is contradistinction to grafts, which are transplanted without a blood supply. Flaps can include skin, combination of skin and fascia, musculocutaneous flaps, muscle flaps, bone flaps, or composite bone, muscle, and skin flaps (osteocutaneous).

McGregor and Morgan categorized flaps as random or axial. *Random* pattern flaps lack significant bias in their vascular pattern, whereas a*xial* pattern flaps are single-pedicle flaps that encompass an anatomically recognizable arteriovenous system along their long axes. Random cutaneous pattern flaps obtain their blood supply from the subdermal plexus and have limited dimensions as determined primarily by base width and may require surgical delay.[9]

Technique of Flap Transfer

Cutaneous flaps can be classified according to the technique used for flap transfer and the distance between donor and recipient site.

Rotational flaps are semicircular and design and rotate about a pivot axis. The donor site can be closed primarily or skin grafted. To facilitate closure, a burrows triangle or a back cut can be made. If a back cut is made, one has to make sure not to undermine the blood supply to the flap.

Transposition flaps are rotated (laterally) about a pivot point into an immediately adjacent defect. Because the effective length of the flap becomes shorter the farther the flap is rotated, the flap must be designed longer than the defect to be covered, otherwise a back cut may be necessary. The flap donor site can be closed primarily, with a skin graft or another flap such as a bilobed flap.

Rhomboid flap is another type of transposition flap. Limberg flap is commonly used to reconstruct cutaneous defects. It is designed with 60- and 120-degree angles opposing each other. Four different types of flaps can be designed around angles of 60 degrees with recruitment of skin from the area of most laxity (▶ Fig. 5.3).[10]

Musculocutaneous Flaps

Musculocutaneous flaps are composites of skin, subcutaneous tissue, fascia, and muscle supplied by a dominant pedicle. A good example of such wound would be the latissimus dorsi, pectoralis major, or rectus abdominis musculocutaneous flaps. There are advantages to muscle flaps. In addition to superior closure of dead space compared to random pattern flaps, they also are more resistant to infection and show increased blood flow following elevation and transfer. The clinical application of muscle to infected wounds has been successful in osteomyelitis, postthoracotomy mediastinitis, and prosthetic grafts.[11,12,13]

Muscle Flaps

Classification of the blood supply to individual muscles was established by Mathes and Nahai in 1979. The authors described five types of muscle on the basis of their circulatory patterns (▶ Table 5.2) muscle flaps have similar properties to musculocutaneous flap but have the distinct difference in the fact that a skin island is not transferred with the flap. This can be useful when a large area needs to be covered.[14]

5.6 Fasciocutaneous Flaps and Perforator Flaps

As new techniques are being developed there is a trend in plastic surgery toward more and more utilization of fasciocutaneous and perforator flaps. These flaps gained their popularity due to their muscle sparing properties, minimal donor-site morbidity, similar effectiveness in wound coverage as compared to muscle flaps (proven in multiple studies) and versatility in their use and clinical application.[15]

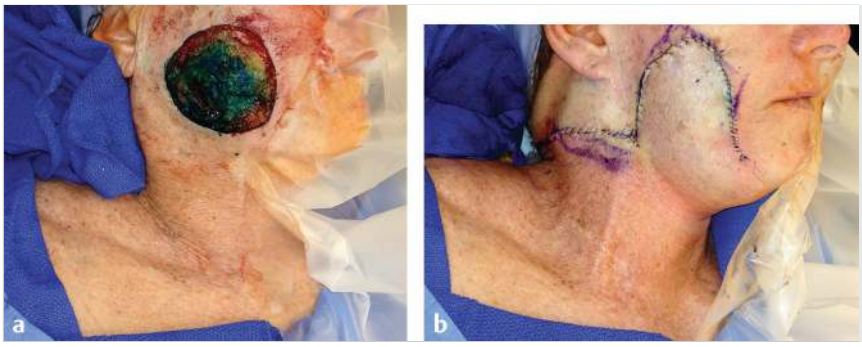


Fig. 5.3 (a) A 55-year-old woman with right cheek defect after wide local excision of invasive melanoma; (**b**) same patient after reconstruction with a Limberg flap.

Table 5.2 Mathes and Nahai Flap Classification

Type	Vascular supply	Examples
I	One vascular pedicle	Gastrocnemius, TFL
II	One dominant pedicle + 1(+) minor pedicle	Soleus, gracilis, biceps/rectus femoris
III	Two dominant pedicles	Gluteus maximus, rectus abdominus, serratus anterior
IV	Segmental pedicles	Tibialis anterior, sartorius
VI	One dominant pedicle + numerous segmental small pedicles	Latissimus dorsi, pectoralis major, internal oblique

Table 5.3 Common indications for free tissue transfer

Indication	Example
Obliteration or reduction of dead space	Reconstruction after extensive soft-tissue resection
Coverage of exposed bone and/or neurovascular tissue	Reconstruction of calvarial, thoracic, or lower extremity defect
Volume and contour reconstruction	Reconstruction of the breast
Vascularized enteral conduits	Reconstruction of pharyngeal and esophageal defects
Composite reconstruction	Combined mandibular and floor-of-the-mouth reconstruction
Functional muscle reconstruction	Facial reanimation for paralysis
Reconstruction and/or replacement of appendages	Digit, penile, and limb reconstructions/replantation

5.7 Principles of Microsurgery

Microsurgery refers to the use of surgical techniques that are beyond the limits of normal human eyesight. In *plastic surgery* specifically, these techniques are used to repair and reconnect blood vessels and nerves for the purposes of replantation of body parts, free tissue transfer, or neurovascular injury. In order for a surgeon to clearly visualize all the vital structures, either loupe magnification or a microscope is commonly used.

5.7.1 History

In the late 1890s and early 1900s, surgeons began suturing blood vessels on animals in the laboratory and humans without any magnification. In 1759, Hallowell performed the first brachial artery repair by hand suture. In 1897 and 1902, J.B. Murphy and Alexis Carrel described the first vascular anastomosis and the triangulation method in end-to-end anastomosis in animals. Jacobson in 1965 anastomosed 1-mm diameter vessels with 100% patency. Further, in 1966, Green used 9–0 nylon suture on rat aortas (1.3 mm) and vena cava (2.7 mm) with patency of 37 out of 40 animals in 21 days. Also, in 1960s, successful arm and hand replantation was performed. Kleinert and Kasdan in 1963 revascularized near-amputated digits under just loupe magnification utilizing vein grafts.[16] Buncke and Schulz in 1965 performed experimental replantation of rabbit ears and monkey digits.[17] The first successful replantation of a completely amputated digit involved the use of a surgical microscope and was performed in 1968 by Tamai and Komatsu.[18] Further developments included foot-operated microscope controls to free up a surgeon's hands; a beam-splitting device to allow the use of a second set of eyepieces for surgical assistance during procedures; optical zoom and independent focus controls; and cooler fiber-optics with reduced likelihood of tissue desiccation and with improved signal transmission.

These technical advances, along with improved knowledge of vascular anatomy, have made available a variety of microsurgical reconstruction options included in the armamentarium of reconstructive surgeons today.

Indications for Free Tissue Transfer

The reconstructive ladder suggests that free tissue transfer should be at the "top" of the pyramid after all other surgical options have been exhausted. Free flaps are generally considered when "less invasive" options such as direct closure, skin grafts, or local/regional flaps are not an option. Some clinical scenarios where free tissue transfer may be considered include wounds and defects with a history of prior radiation, infection, scarring, tumor, or trauma that results in unavailability of local tissue suitable for reconstruction.

A detailed discussion as to which reconstructive option should be chosen is beyond the scope of this chapter. Factors the surgeon should keep in mind when deciding on the flap choice include, but not limited to the size of the defect, its location, shape, and if any prior reconstructive surgeries have been attempted. Other variables to consider include tissue composition (e.g., skin, fascia, muscle, bone, or a combination), aesthetics, pedicle length, and vessel size match (▶ Table 5.3).

5.7.2 Equipment and Surgical Preparation

A microsurgical instrument set should be readily available to the operating team, as well as an extra set in case of instrument contamination or damage during microsurgery. Instrument set should include at least two pairs of jeweler forceps, dilating vessel forceps, straight and curve microsurgical scissors, coupler device, microneedle drivers, venous coupler device, different sizes of single and double Acland clamps, and Waxel spears. Heparin solution generally placed in a 3-mL syringe attached to 27-gauge ophthalmic cannula is commonly used to irrigate the vascular lumen. In the authors' experience, it as always a good idea to set up a separate sterile tray with all the microsurgical instruments on it to facilitate efficiency, and self-sufficient instrument handling without having to ask for instruments during vascular anastomosis (▶ Fig. 5.4).

An operating microscope with two eyepieces or microsurgical loupes anywhere from 3.5× to 8× magnification should be used. It is the authors' preference to use loupe magnification of 3.5× instead of the microscope for routine microvascular cases (exceptions are finger/thumb replants, young

children, or vessel diameter ≤ 1 mm). There are some advantages of loupe magnification when compared to the microscope including visualization of the entire pedicle during microanastomosis ensuring absence of any kinks or twists; avoidance of the complexity and bulkiness of the microscope; ability to perform vascular anastomosis in facilities without appropriate microscope; and cost-efficiency. According to the literature, anastomosis patency rates and flap survival are similar when either a microscope or loupe magnification is used.[19] Choice of magnification highly depends on the surgeon's preference and training.

The free tissue transfer procedure should be outlined with the anesthesia team, the ablative surgeon during immediate reconstructions, as well as the nursing personnel. Also, preoperative and postoperative use of agents such as subcutaneous heparin, Lovenox, aspirin, pressors, as well as antibiotics should be discussed. It is a common practice to paralyze patients intraoperatively, for instance, during an abdominal flap procedure with pedicle intramuscular dissection, and that should be discussed in detail with the anesthesiologist and the ablative team (▶ Fig. 5.5). Patient positioning, length of the procedure, placement of any arterial or central and peripheral venous catheters should be discussed and adequately prepared for in order not to interfere with flap harvest and recipient sites. Warm saline and papaverine or 2% lidocaine should be readily available to counteract any vascular spasm. Functioning pencil Doppler and/or implantable

Fig. 5.4 Microsurgical table setup with instruments.

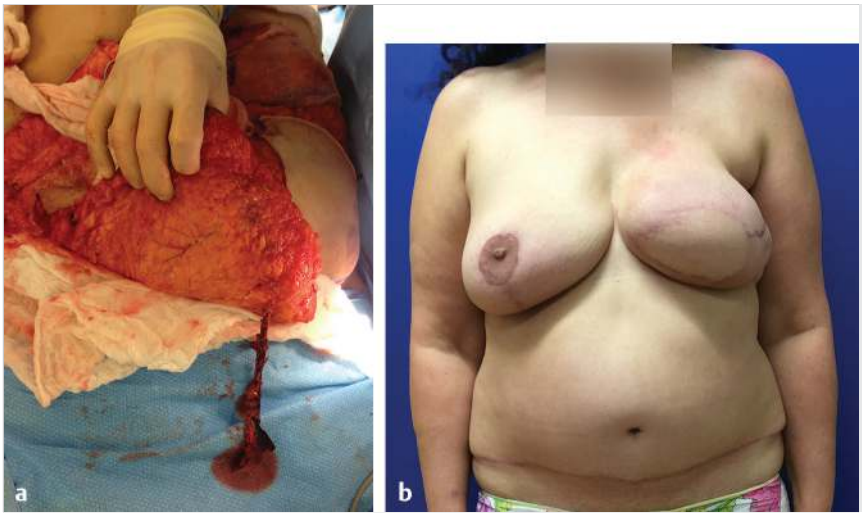


Fig. 5.5 (a) Deep inferior epigastric artery perforator (DIEP) flap after being disconnected from the abdomen; (b) same patient 7 months after left DIEP flap and right balancing breast reduction.

Cook-Swartz Doppler probe for postoperative monitoring in head and neck reconstruction can be very useful. An appropriately warmed room, a Bair Hugger™, and most importantly having a routine team comfortable and prepared for free tissue transfer also may improve flap survival success.

5.8 Technique

Technique for microvascular anastomosis varies from surgeon to surgeon; however, the basic principles remain the same. Meticulous attention to details to ensure flap perfusion and outflow is a must. It is the authors' preference to perform vascular anastomosis under 3.5 × loupe magnification which is sufficient for a majority of flaps and can significantly facilitate the operation without requirement for a microscope. Initially, both the donor and recipient vessels are examined for kinking, twisting, and proper orientation. The anterior aspect of the vessels is marked with a marking pen, as this may expedite anastomosis and decrease confusion in regard to pedicle orientation. Next, lumens are examined for clots, adventitial flaps or tears, valves, and diameter. It is important to ensure a clean, open vascular lumen that is free of debris or platelet clot or any obstruction. Heparinized saline is very useful for luminal irrigation and has become an integral part of microsurgery. Papaverine may be used if vascular spasm is suspected. It is more effective to inject papaverine directly into the adventitia rather than spray it on top of the vessel. If dealing with a smaller caliber vein sometimes, it helps connect the main lumen to a branching vein lumen to create a larger diameter opening for easier anastomosis. In recent years, Synovis microvascular couplers have gained popularity for venous anastomosis. The coupler effectively everts the vessel edges and decreases the anastomotic time when compared to hand-sewn anastomosis. When using loop magnification, 8–0 nylon on a BV130–5 needle is appropriate for the majority of vessels for head and neck and breast reconstruction. The advantage of this caliber suture is that it is more visible. Full-thickness vessel wall bites are a must and the lumen should be clearly visualized before needle entry to prevent partial-thickness bites or suturing the back wall of the vessel. Different suturing techniques can be applied for arterial anastomosis. It is the authors' preference to utilize a 180-degree suture technique, when initially the lumens are lined up appropriately with initial sutures separated by 180 degrees and this is followed by either anterior or posterior wall suturing. Usually only two or three sutures are required for each side and these are not tied until all sutures on each side are placed. It is also vital to examine the back wall of the lumen prior to closing the second side of the vessel to ensure proper placement of all sutures. Prior to tying the last suture, the lumen should be irrigated with heparinized saline solution. Once vascular clamps are released, one should observe for flap perfusion and flow across both arterial and venous anastomosis. Papaverine and warm saline irrigation can be useful at this time. Once the flap perfusion is established, it should be left in place over to percolate and observe for any problems.

5.8.1 Postoperative Care

Postoperatively, careful flap monitoring is imperative. Patients can be admitted either to an intensive care unit, intermediate care unit, or Med-Surg floor depending on the type of microsurgical procedure, adequate and trained nursing support, and surgeon's preference. For the first 24 hours, the flaps are monitored every hour by clinical exam for color change, flap temperature, capillary refill, and skin turgor. External pencil Doppler can also be very useful in detecting problems. One should try to listen and detect both a biphasic arterial Doppler signal and a venous signal over the flap. Buried flaps (i.e., head and neck reconstruction), and/or muscle flaps with STSG can be further monitored by an internal cook's Doppler either over the vein or artery distal to anastomosis. Loss of Doppler signal should warrant immediate flap reexploration. It is the authors' preference to keep patients undergoing breast microsurgical procedure NPO for the first 24 hours postsurgery, keep them at bedrest, and keep the intravenous fluids running at a high rate from 125 to 150 mL/hour of lactate ringers solution. A Foley catheter is kept in for at least 48 hours until patients gain more mobility. Studies have shown that over 80% of vascular thrombosis happen within 24 to 72 hours postoperatively; thus, very close flap and patient monitoring is required within that time frame.[20] Patient's diet is slowly advanced from clear liquids on second postoperative day to regular diet on the third day. With head and neck reconstruction, if patients have an nasogastric tube, feeds can start after 24 hours and a modified barium swallow test is usually done 1 week postoperatively for intraoral flaps to check for aspiration and orocutaneous fistulas.

5.9 Complications

Regardless of the skill and experience of the microsurgeon, complications will happen. Those usually include, but are not limited to, hematoma, seromas, arterial or venous thrombus, infection, partial or complete flap loss, fat necrosis, and delayed healing. When vascular thrombosis is suspected, one should have a very low threshold for return to the operating room for flap exploration. If a vascular thrombus is detected early enough, the flap may still be salvageable. It is imperative to act swiftly when the thrombus occurs which can be facilitated by having experienced and reliable personnel monitoring the flap. Ischemia time until non-reflow phenomenon occurs varies according to the flap with muscle and musculocutaneous flaps having a shorter ischemia time compared to fasciocutaneous flaps. This is obviously more critical during arterial thrombosis as the flap often times will still remain perfused for a prolonged period of time when a venous thrombus occurs. Studies and clinical experiences have shown that hyperbaric oxygen treatments may reverse ischemia reperfusion injury after a prolonged flap ischemia time.[21] The biomolecular mechanism of how hyperbaric oxygen may help reverse or prevent reperfusion injury is still not exactly clear. It is clear, however, that if hyperbaric treatments are to be attempted for flap salvage, it

has to happen ideally within a few hours after reperfusion.[22] Majority of centers may be limited in this regard. To minimize postoperative complications, the surgeon should be fully satisfied with hemostasis, flap perfusion, and inset before leaving the operating room. Meticulous attention to details is imperative for a successful microsurgical practice.

5.10 Review Questions

5.10.1 True or False

1. Split-thickness skin grafts survive by a process called imbibition for the first 24 hours.
2. On areas such as elbow, axilla, or neck, split-thickness skin graft is an acceptable reconstructive option.
3. Fat grafting is commonly used in breast reconstruction to correct asymmetries and add volume, in facial rejuvenation as a permanent filler to add volume and restore youthful facial appearance.

5.10.2 Choose the Best Answer

4. According to Mathes-Nahai musculocutaneous flap classification, what is the classification of the gracilis flap?
   a) Type I.
   b) Type II.
   c) Type III.
   d) Type IV.
   e) Type V.
5. Five hours after immediate left breast reconstruction with a free muscle sparing TRAM flap, the nurse calls you due to change in the flap appearance. On exam, the flap is swollen, and skin paddle has a purplish hue with brisk capillary refill. What is the next appropriate step in management?
   a) Continued close observation by registered nurse and evaluation in the morning.
   b) Releasing the sutures at the bedside.
   c) Leech therapy.
   d) Immediate operative exploration.

5.10.3 Answers

1. True.
2. False.
3. True.
4. b. Type II.
5. d. Immediate operative exploration.

References

[1] Vistnes LM. Grafting of skin. Surg Clin North Am. 57(5):939–960
[2] Rudolph R, Klein L. Healing processes in skin grafts. Surg Gynecol Obstet. 136 (4):641–654
[3] Guyuron B, Ericksson E, Persing J. Plastic Surgery Indications and Practice. 1: 95–104
[4] Dos-Anjos Vilaboa S, Llull R, Mendel TA. Returning fat grafts to physiologic conditions using washing. Plast Reconstr Surg. 132(2):323e–326e
[5] Del Vecchio D, Rohrich RJ. A classification of clinical fat grafting: different problems, different solutions. Plast Reconstr Surg. 130(3):511–522
[6] Eto H, Kato H, Suga H, et al. The fate of adipocytes after nonvascularized fat grafting: evidence of early death and replacement of adipocytes. Plast Reconstr Surg. 129(5):1081–1092
[7] Kirkham JC, Lee JH, Austen WG , Jr. Fat graft survival: physics matters. Ann Plast Surg.
[8] Atashroo D, Raphel J, Chung MT, et al. Studies in fat grafting: Part II. Effects of injection mechanics on material properties of fat. Plast Reconstr Surg. 134(1): 39–46
[9] McGregor IA, Morgan G. Axial and random pattern flaps. Br J Plast Surg. 26 (3):202–213
[10] Lamberty BGH, Healy C. Flaps: physiology, principles of design, and pitfalls. In: Cohen M, ed. Mastery of Plastic and Reconstructive Surgery. Boston, MA: Little Brown & Co; 1995
[11] Ger R. Muscle transposition for treatment and prevention of chronic post-traumatic osteomyelitis of the tibia. J Bone Joint Surg Am. 59(6):784–791
[12] Stark WJ. The use of pedicled muscle flaps in the surgical treatment of chronic osteomyelitis resulting from compound fractures. J Bone Joint Surg Am. 28:343–350
[13] Scully HE, Leclerc Y, Martin RD, et al. Comparison between antibiotic irrigation and mobilization of pectoral muscle flaps in treatment of deep sternal infections. J Thorac Cardiovasc Surg. 90(4):523–531
[14] Mathes SJ, Nahai F. Clinical Applications of Muscle and Musculocutaneous Flaps. St Louis, MO: Mosby; 1982
[15] Pontén B. The fasciocutaneous flap: its use in soft tissue defects of the lower leg. Br J Plast Surg. 34(2):215–220
[16] Atasoy E, Ioakimidis E, Kasdan ML, Kutz JE, Kleinert HE. Reconstruction of the amputated finger tip with a triangular volar flap. A new surgical procedure. J Bone Joint Surg Am. 52(5):921–926
[17] Buncke HJ , Jr, Schulz WP. Total ear reimplantation in the rabbit utilising microminiature vascular anastomoses. Br J Plast Surg. 19(1):15–22
[18] Komatsu S., Tamai S. Successful Replantation of a Completely Cut Off Thumb. Plastic and Reconstructive Surgery Journal.1968;42(4): 374-377
[19] Shenaq SM, Klebuc MJ, Vargo D. Free-tissue transfer with the aid of loupe magnification: experience with 251 procedures. Plast Reconstr Surg. 95(2): 261–269
[20] Hayhurst JW, O'Brien BM. An experimental study of microvascular technique, patency rates and related factors. Br J Plast Surg. 28(2):128–132
[21] Zamboni WA, Roth AC, Russell RC, Graham B, Suchy H, Kucan JO. Morphologic analysis of the microcirculation during reperfusion of ischemic skeletal muscle and the effect of hyperbaric oxygen. Plast Reconstr Surg. 91(6):1110–1123
[22] Baynosa RC, Zamboni WA. The effect of hyperbaric oxygen on compromised grafts and flaps. Undersea Hyperb Med. 39(4):857–865

6 Principles of Facial Aesthetics

Krishna S. Vyas and Henry C. Vasconez

Abstract

This chapter looks at the subjective quality of beauty through a scientific lens. Facial aesthetics are addressed in detail, along with the various factors that affect facial beauty—aging, skin and soft tissue, and skeletal support. Considerations in the preoperative evaluation are covered that will assist the surgeon in the aesthetic assessment of the face. The components of symmetry, height, and width are discussed in relation to frontal and lateral view, midface and lower face analysis and projection. Each section of the face—the upper third, including forehead and eyebrows, the middle third, with the cheeks, nose, ears, and the lower third, the region of the mouth and lips—is thoroughly reviewed with respect to the governing principles, effects of aging, and management of remedial treatments. Finally, the chin and neck are introduced and common problems briefly reviewed.

Keywords: beauty, facial aesthetic units, aging, Fibonacci ratio, dentition

6.1 Goals and Objectives

- Understand principles of facial aesthetics through historical and surgical perspectives.
- Describe anatomic changes associated with facial aging.
- Apply principles of facial aesthetics to the aging face.
- Understand the evaluation of prospective aesthetic patients seeking facial rejuvenation.
- Describe various surgical and nonsurgical options to achieve the goals identified during consultation.

6.2 What Is Beauty?

Beauty has inspired, influenced, and captivated humans throughout time. For thousands of years, we have attempted to understand, define, and depict beauty in art, literature, architecture, nature, and human form. But what is beauty? In the broadest sense, beauty can be defined as the combination of qualities pleasing the aesthetic senses. Attractive faces activate the reward centers in the brain, elicit the development of human relationships, motivate partner selection, and activate positive treatment in many social settings.[1,2] Attractive people are more likely to be hired for jobs, to be promoted, to wait shorter periods of time for services, are less likely to be reported or punished for crimes, are more likely to have dates, and are perceived to possess positive personality attributes such as kindness and honesty.[3,4,5,6]

Although the positive social consequences of facial attractiveness have been well studied, determinants of features that define an attractive face remain poorly understood. If standards for facial beauty are learned and influenced solely by individual preference, culture, and era, then what is perceived as beautiful would vary dramatically across time and space. However, regardless of age, nationality, ethnicity, culture, or era, people share a sense of what is attractive. Several cross-cultural studies support the hypothesis that the perception of beauty is uniform across culture.[7,8,9,10] When presented with attractive and unattractive faces, masks, or dolls, infants gaze at the attractive face for a longer period of time and prefer attractiveness to symmetry.[11,12,13] The emergence of preferences for beauty in early human development and cross-cultural agreement challenges the argument that beauty is defined exclusively by culture and supports the notion that our preferences may be biologically determined by natural selection.[14] But what is ideal for beauty?

Preferences for symmetry are likely to be an evolutionary trait that serves as a surrogate marker for phenotypic and genetic fitness. Asymmetry is a consequence of normal aging and is perceived as less attractive compared to a symmetric face. Averageness, or how closely a face resembles the majority in the population, is also considered attractive.[15,16,17,18] It is important to note, however, that extreme beauty is associated with magnification or diminution of a specific feature. For a male, this could mean an augmented dominant feature, such as a prominent chin. Individual characteristics that deviate from the average can increase attractiveness and should be considered when addressing the patient's aesthetic goals.

It is also important to consider physical characteristics that differ among men and women. In studies involving facial composite photographs, females often preferred men with more masculine features such as deep-set and small eyes, large, wide, and square jaws, prominent chins, thin lips, and facial hair; on the other hand, males preferred females with large foreheads, large eyes, prominent cheekbones, a small nose, small chin, and full lips.[19,20,21] In males, this corresponds to a larger nasofacial angle and smaller nasomental, nasofrontal, and mentocervical angle compared to females. A high eyebrow position and a wide smile were considered attractive "expressive" features of both sexes, suggesting high sociability.

Aesthetic principles must be considered in the context of ethnicity. Fitzpatrick skin type, scarring, and the ability to hide facial incisions vary widely among ethnic groups. Although certain facial proportions are constant throughout ethnicities, westernized principles of facial aesthetics cannot be applied to all since many patients desire to retain certain ethnic features. In contrast, certain well-publicized ethnic features are appealing to other groups, such as the very popular double eyelid blepharoplasty in the Asian population. Our concept of beauty will continue to evolve as we further understand facial harmony and standards of beauty among distinct ethnicities and as the concept of beauty continues to evolve with the increased global heterogeneity of ethnic backgrounds.

6.3 A History of Beauty

While beauty was idealized even in ancient cultures, it was not until the Greek philosophers began to study beauty that it became a discipline. The Greek philosopher and mathematician Plato described the ideal face with symmetry, proportion,

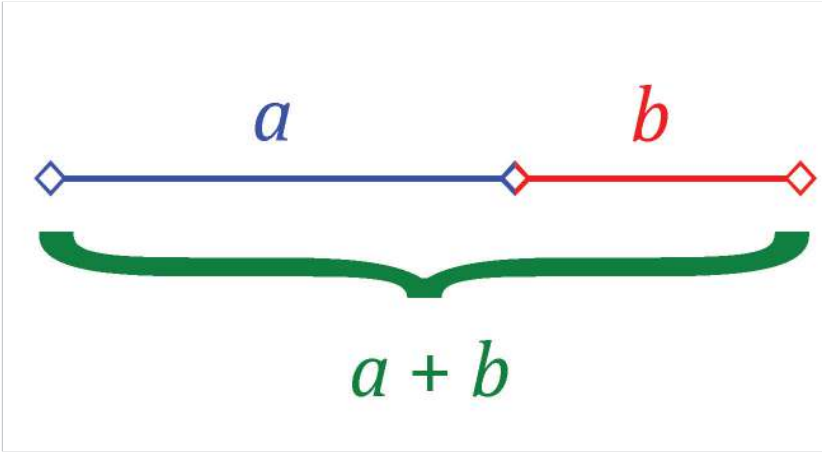


Fig. 6.1 The golden ratio is a visual representation of the number phi, which is the Greek letter ρ or ϕ representing approximately 1.618. Quantities are in the golden ratio if their ratio is the same as the ratio of their sum to the larger of the two quantities. The golden ratio is also called the golden mean, golden section, divine proportion, and golden number. It is present in things considered beautiful in facial aesthetics (Golden Mask), architecture (the Parthenon, Chartres Cathedral), art (the Mona Lisa, Vitruvian Man, Birth of Venus), and nature (arrangement in plants, flowers, shells, spiral galaxies, hurricanes, animals, and the structure of DNA).

harmony, and geometry in his "golden proportions," where the width of the face would be two-thirds its length and the nose would be no longer than the distance between the eyes. In his text the *Elements*, Euclid defined the golden ratio, phi, which is equal to approximately 1.1618. The golden ratio, also called the divine ratio, is the only mathematical relationship consistently found throughout beautiful and harmonic things in nature, art, history, architecture, and the human form and face. This ratio is obtained when a line ABC is cut so that AB/AC = BC/AB (▶ Fig. 6.1). Phi has been studied extensively in facial aesthetics and utilized to derive a golden mask or ideal facial proportions.[22,23] While the mask may be an inexpensive and easy tool for facial analysis, the proportions are thought to be inconsistent and too masculinized for generalization.[24,25] Variations have been proposed, but recent studies utilizing three-dimensional stereophotogrammetry for facial anthropometry suggest that the ratios between facial distances were not related to attractiveness or the golden ratio.[26,27] Smaller faces and uniform facial thirds and fifths are thought to be more ideal ratios for females.[28]

Other Greek figures also helped in defining ideal beauty of the human body, including the sculptors Polykeitos and Praxiteles who developed canons of physical proportions of the ideal body through their art. The Roman architect Vitruvius produced a facial trisection that is still used today by medical professionals. Today, we still utilize the Vitruvian thirds with some modification.[29] During the Renaissance, the ideal representation of facial form was revisited. Leonardo da Vinci exemplified anatomical ideals with geometric principles through the Vitruvian Man and also formulated the ideal facial proportions of vertical fifths. Vitruvian principles also influenced the German printmaker Albrect Durer, who published a four-book series on human proportion in 1528.

Facial aesthetics largely depend on the three-dimensional topography of the central features of the face, namely, the eyes, nose, cheeks, and lips. Cephalometry was introduced in the early 18th century to quantitatively understand the growth, development, and relationship of skeletal structures. Anthropometric methods utilize surface topographic measurements and are preferable to cephalometric methods in determining ideal facial proportions. Anthropometry provides simple, non-invasive, comprehensive three-dimensional population-matched data. Powell and Humphreys provided a detailed analysis of facial contours, angles, and proportions in their 1984 text *Proportions of the Aesthetic Face*.[30]

6.4 Facial Aging

Facial aging reflects the cumulative, dynamic, and interdependent structural changes of the skin, soft tissues (subcutaneous fat, muscle, fascia, and ligamentous structures), and structural support (bone and teeth) of the face, leading to changes in facial volume and topography (▶ Fig. 6.2). [31] There is a general descent of the soft tissues with age, which has been described by an inverted triangle (▶ Fig. 6.3).

The factors contributing to facial aging are multifactorial and can be due to the effects of genetics, gravity, photoaging, medical comorbidities, diet, chronic smoking or alcohol use, large changes in weight, stress, environmental factors, decreased tissue elasticity, craniofacial remodeling, subcutaneous fat redistribution, and other factors. The visible signs of aging can include features such as horizontal and vertical rhytids, temporal atrophy, volume loss in the midface, deep nasolabial folds, and jowls (▶ Fig. 6.4) areas all of which are amenable to treatment (▶ Fig. 6.5).

6.5 Skin

Aging skin results from deterioration of the skin's structure and function and is associated with changes in the epidermis, dermis, and subcutaneous layers. Factors contributing to the appearance of aged skin include dry and thin skin, a dull and rough complexion, enlarged pores, irregular pigmentation, actinic keratosis, loss of firmness and elasticity, broken blood vessels, fine lines, and deep wrinkles. There are three main types of aging responsible for the change in the structure, function, and quality of the skin.

- Biological (intrinsic): chronological age, genetics
- Environmental (extrinsic): sun exposure, pollution, cigarette smoke, or external stress such as dry cold weather
- Mechanical: repeated muscle contraction

In the epidermis, decreased cellular turnover, reduced keratinocytes and epidermal lipids, and decreased moisture result in dry skin. Altered pigmentation results from fewer melanocytes and melanin production. In the dermis, reduced repair and turnover and reduced function of the sebaceous and sweat glands cause dry skin. Histologically, there are fewer fibroblasts and collagen production, reduced dermal matrix, and thicker

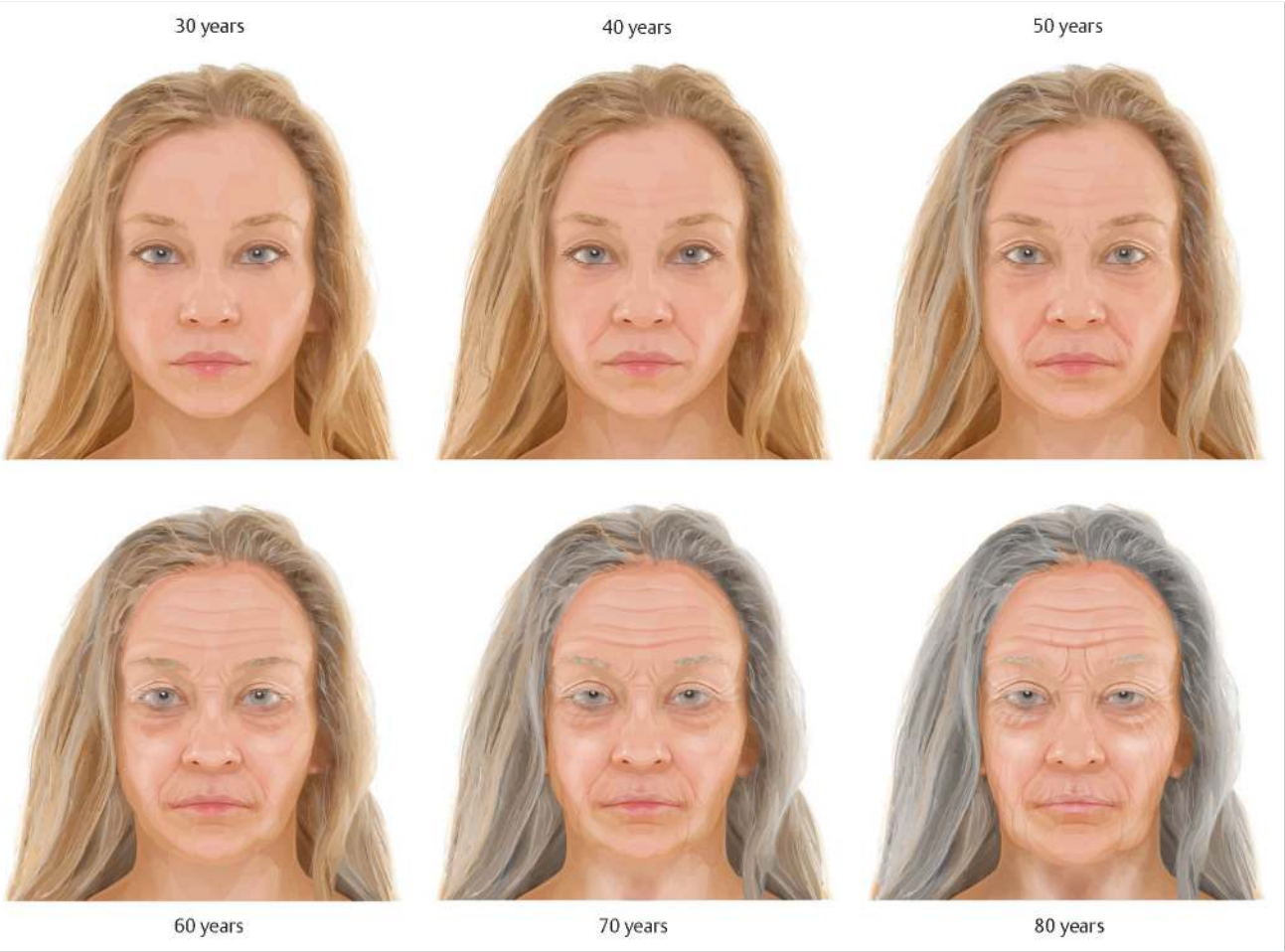


Fig. 6.2 Time lapse of the aging face.

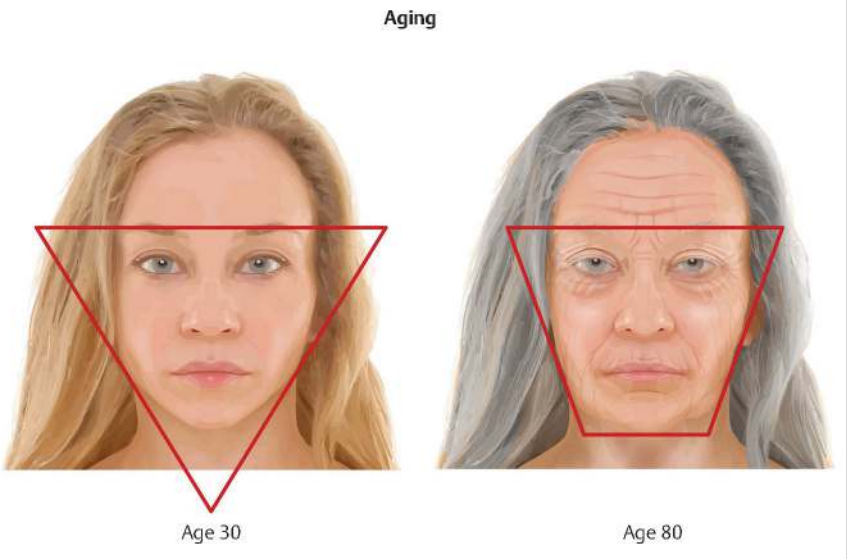


Fig. 6.3 The triangle of youth is lost with aging.

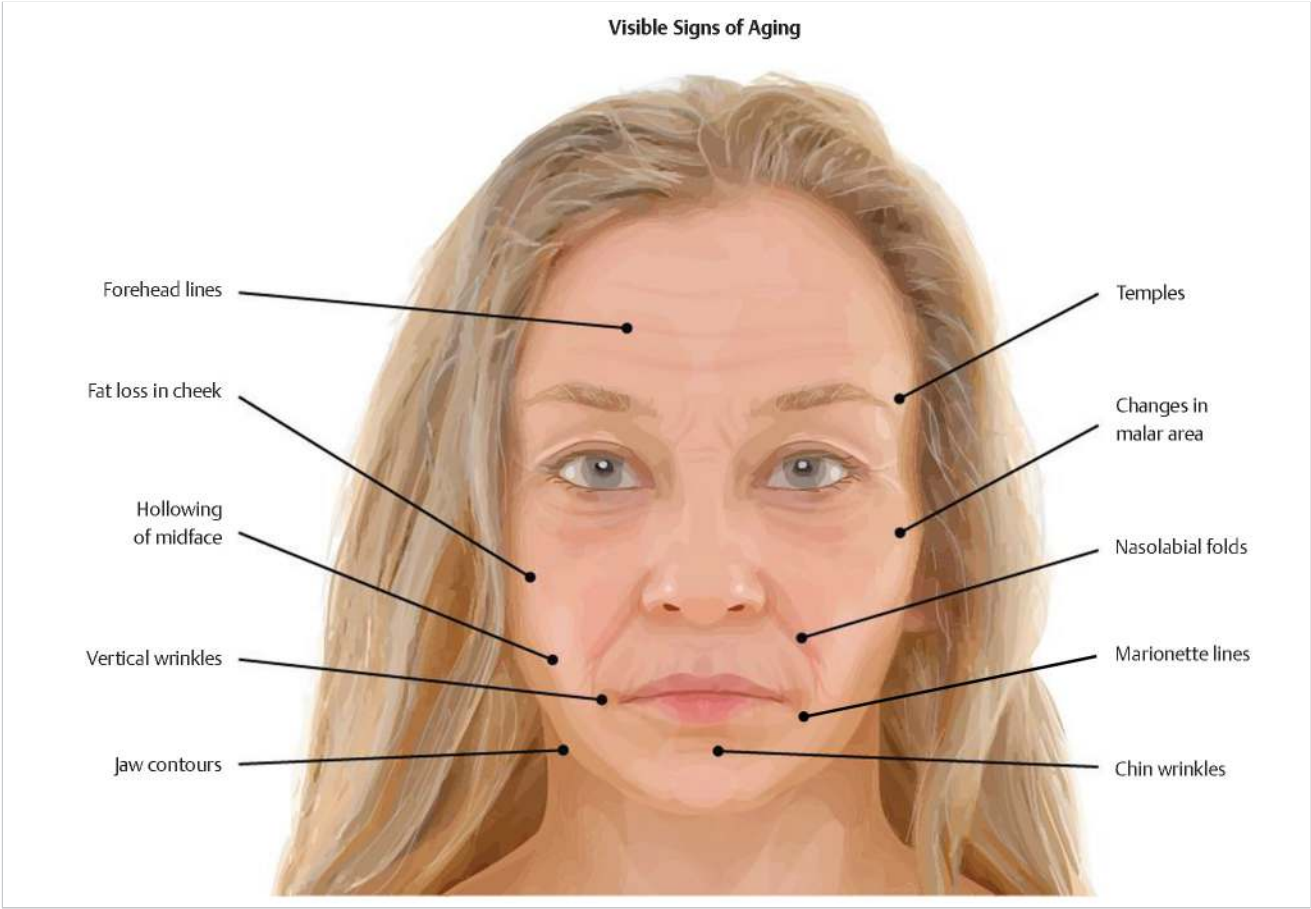


Fig. 6.4 Signs of facial aging include greater visibility of lines and wrinkles (such as in the forehead); nasolabial folds become prominent; there is volume loss in the temples and midface; contours of the jaw change from fat redistribution and changes to the underlying craniofacial skeletal. Many factors contribute to aging of the skin.

elastin fibers with less elasticity. The blood vessels become thin, dilated, and weak. The thinning subcutaneous tissue and weakening retaining ligaments cause the skin to sag.

Management: Treatment of aging skin includes sunscreen, moisturizers, tretinoin, fillers and injectables, lipotransfer/autologous fat grafting, peeling and microdermabrasion, radiofrequency, and laser resurfacing.

6.6 Soft Tissues

The youthful face has an adequate volume and distribution of fat in specific areas. In the aging face, the forehead, temporal, periorbital, malar, glabellar, perioral, and mandibular sites lose fat, while the jowl, submental area, nasolabial folds, and infraorbital areas gain fat (▶ Fig. 6.6).[32] Contour deficiencies become increasingly apparent with the redistribution or atrophy of fat with age, and the defining convexities of the youthful face are lost.[33] Imaging studies reveal that the midfacial fat compartments migrate inferiorly with aging. Retaining ligaments that provide support to the soft tissues and skin of the face, such as the anterior platysma-cutaneous ligaments and mandibular osteocutaneous ligaments, become less supportive.

6.6.1 Management

Procedural rejuvenation techniques include non-invasive, minimally invasive, and invasive procedures, ranging from medical rejuvenation with retinols and alpha-hydroxy acids to procedural rejuvenation with laser resurfacing, botulinum toxin A, fillers, and radiofrequency all the way to surgical interventions such as liposuction or fat grafting, implants, and rhytidectomy.

6.7 Structural Support

Aging of the craniofacial skeleton is due to dynamic changes involving bone expansion and bone loss (▶ Fig. 6.7).[34] Specific changes in the facial skeleton which occur with aging include reduced facial height due to changes in the maxilla and mandible, increased facial width and depth, and increased prominence of the frontal sinus and zygomatic arches. Bony resorption leads to alteration of the structural support of bone and consequently alters overlying soft tissue and skin.

Management: Patients may present for consultation for problems such as midface hypoplasia, prominent zygoma, or square mandibular angle. The size, shape, balance, and proportion of the craniofacial skeleton can be manipulated through

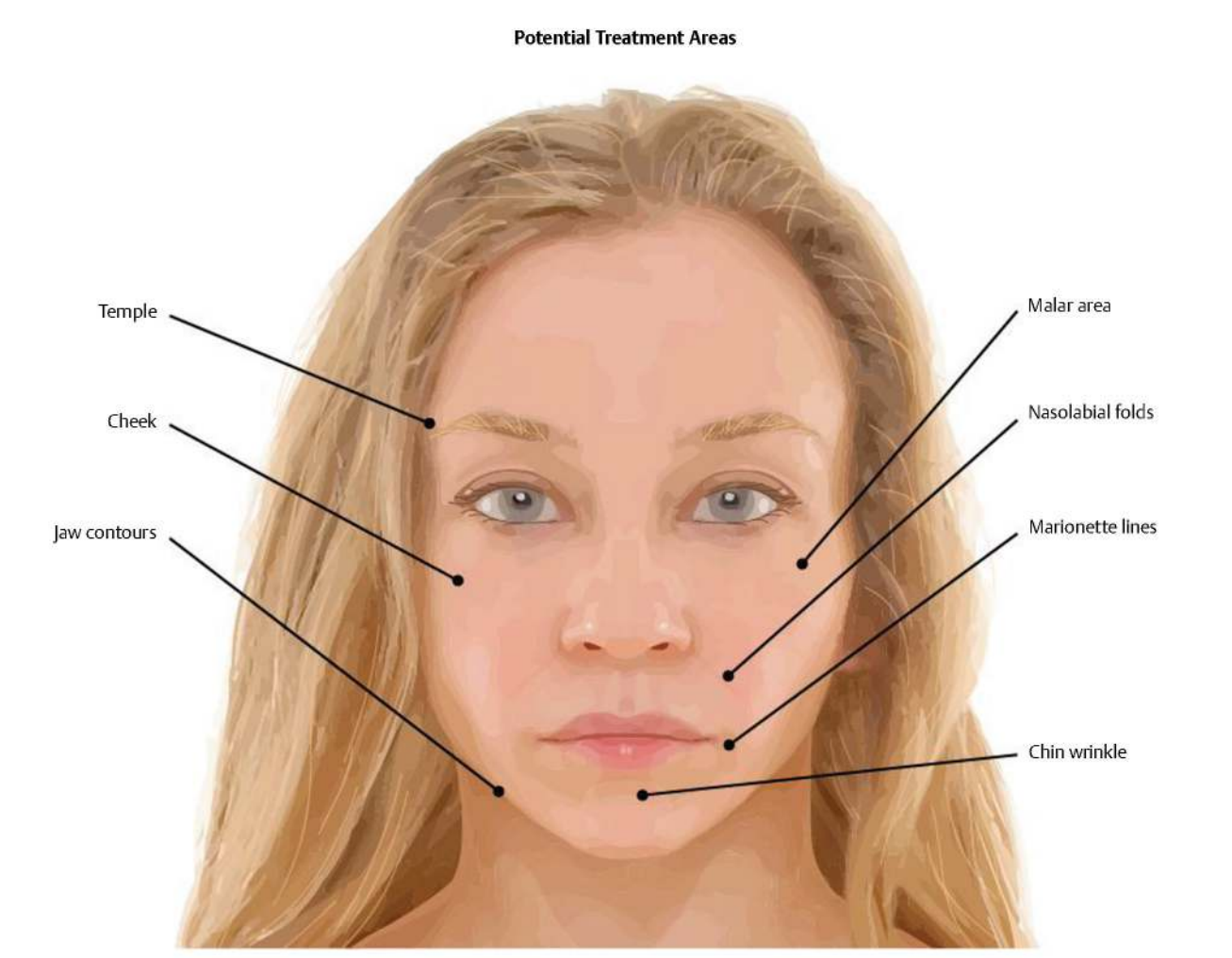


Fig. 6.5 The many potential treatment areas for the aging face.

Fig. 6.6 In youth, the fat compartments of the face are congruent, resulting in a smooth appearance. Redistribution, accumulation, and atrophy of fat compartments with aging leads to facial volume loss. Some areas lose fat (forehead and cheeks), while others gain fat (mouth and jaw), resulting in contour deficiencies.

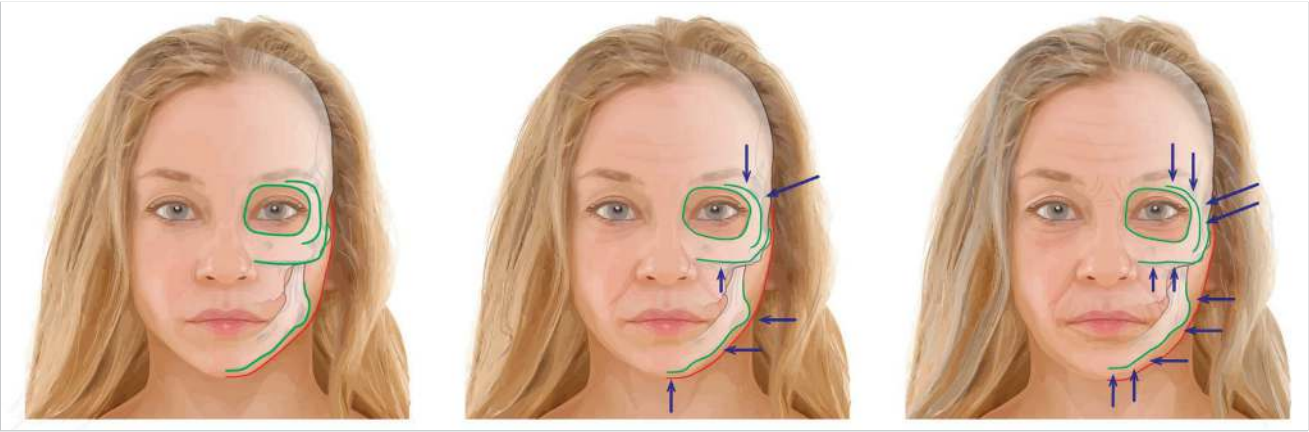


Fig. 6.7 The craniofacial skeleton remodels with aging, with bony resorption leading to volume loss. Consequently, there is a change in the overlying soft tissue and skin. The arrows indicate areas of bony resorption.

segmental osteotomies, genioplasties, mandibular angle reductions, malarplasties, and orthognathic surgeries to alter facial projections and aesthetics.[35]

6.8 Preoperative Evaluation

Goals of the consultation should include an assessment of the patient's objectives, perceptions, and expectations. This includes a consideration of personal preference, gender, cultural differences, and racial background. The goals of facial analysis should be to diagnose and classify deformities or deviations, develop a treatment approach, and predict outcomes. The risks, benefits, costs, side effects, potential adverse events, and anticipated outcomes should be thoroughly reviewed, and any of the patient's questions should be answered.

Preoperative anteroposterior and lateral and oblique photographs assist the surgeon in planning, facilitate patient preoperative and postoperative discussion and review, and should be done for medicolegal documentation.

6.9 Facial Proportions and Aesthetic Assessment

Many methods have been utilized for assessment of the aesthetic units of the face and for determining their relationship and relative proportions in evaluating the face as a whole. Although no algorithm exists for aesthetic assessment of the face, systematic evaluation and preoperative facial measurements may aid in the determination of which facial features need to be altered in order to produce the best harmony.

Hom and Marentette developed an 8-step systematic approach to facial analysis.[36] The initial assessment should begin with an analysis of facial symmetry, height, and width on frontal view. Next, evaluation continues on profile to determine facial projection and the relationship between structures such as the forehead, nose, lip, and chin. Finally, individual subunits are carefully evaluated.

Facial analysis is dependent on the assessment of both soft tissue and the underlying craniofacial framework. Soft tissue reference points (▶ Fig. 6.8), skeletal reference points (▶ Fig. 6.9), and reference planes are defined by cephalometric analysis. Important elements in cephalometric analysis include maxillary, mandibular and incisor positions, and facial proportions. The Frankfort horizontal plane is the standard reference for patient positioning in photographs and cephalometric radiographs. This plane is defined as a line drawn from the superior portion of the external auditory canal (approximately at the level of the superior edge of the tragus) to the inferior border of the infraorbital rim when the patient's gaze is parallel to the floor.

6.10 Frontal View Analysis

6.10.1 Symmetry

Aesthetic facial analysis can begin with a general evaluation for symmetry by bisecting the face with a midsagittal plane and comparing the halves, although symmetry is rarely perfect. The forehead, nose, lips, and chin should lie along this axis.

6.10.2 Height

Facial height is determined by dividing the face into equal horizontal thirds (▶ Fig. 6.10). The boundaries are from the trichion to the glabella (upper face), from the glabella to the subnasale (middle face), and from the subnasale to the menton (lower face). With a receding hairline, the most superior border of the upper third can be determined by identifying the most superior movement of the frontalis muscle.

A second method for assessing facial height takes into consideration only the middle and lower portions of the face. The first measurement is from the nasion to the subnasale and from the subnasale to the menton. With this measurement, the midface is 43% of the total length and the lower face is 57% of the total length. This technique is advantageous since the nasion is a more reproducible landmark than the glabella.

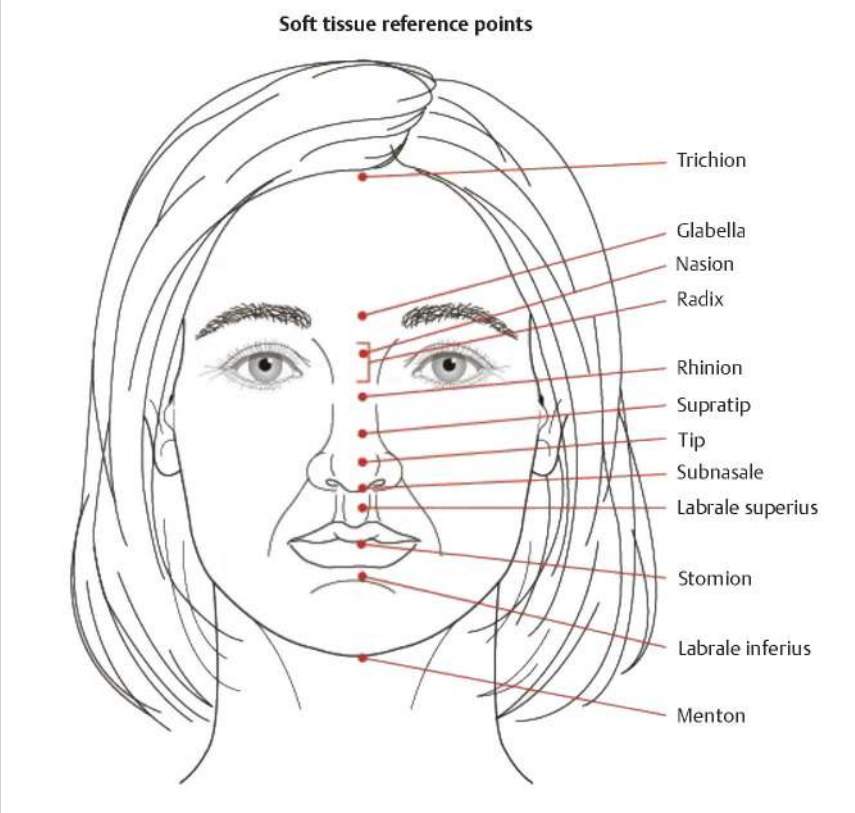


Fig. 6.8 Soft tissue (anthropometric) reference points (frontal view).

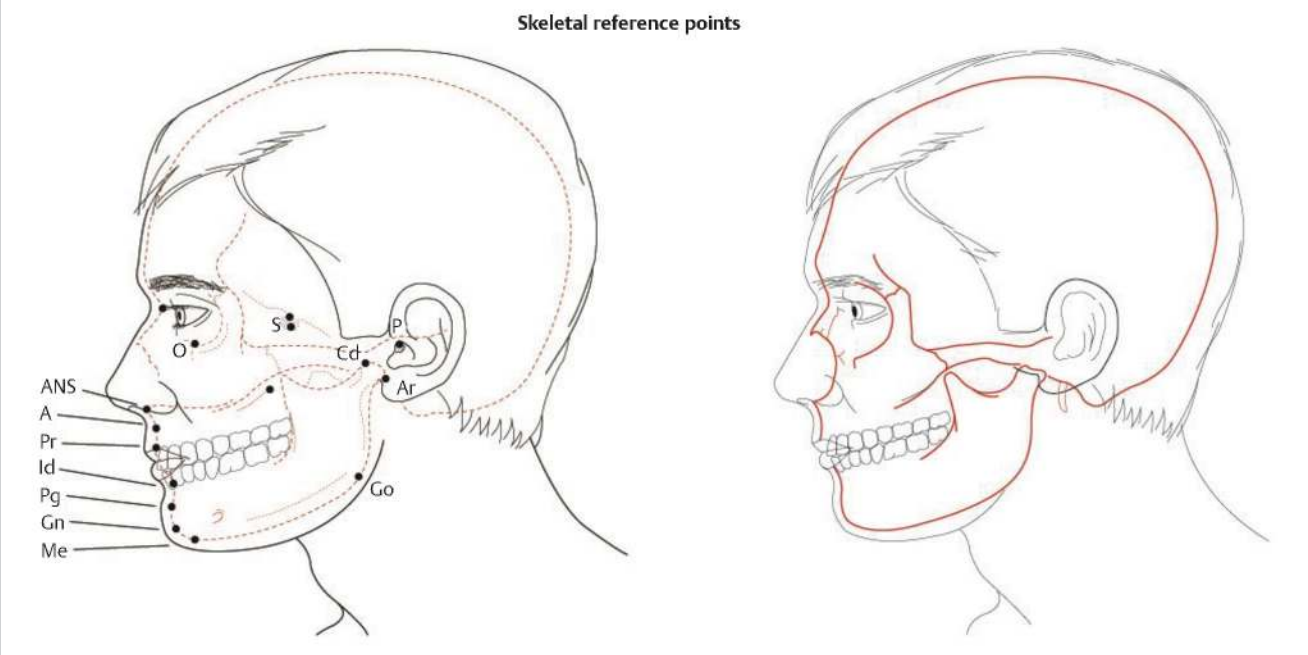


Fig. 6.9 Skeletal (cephalometric) reference points (lateral view).

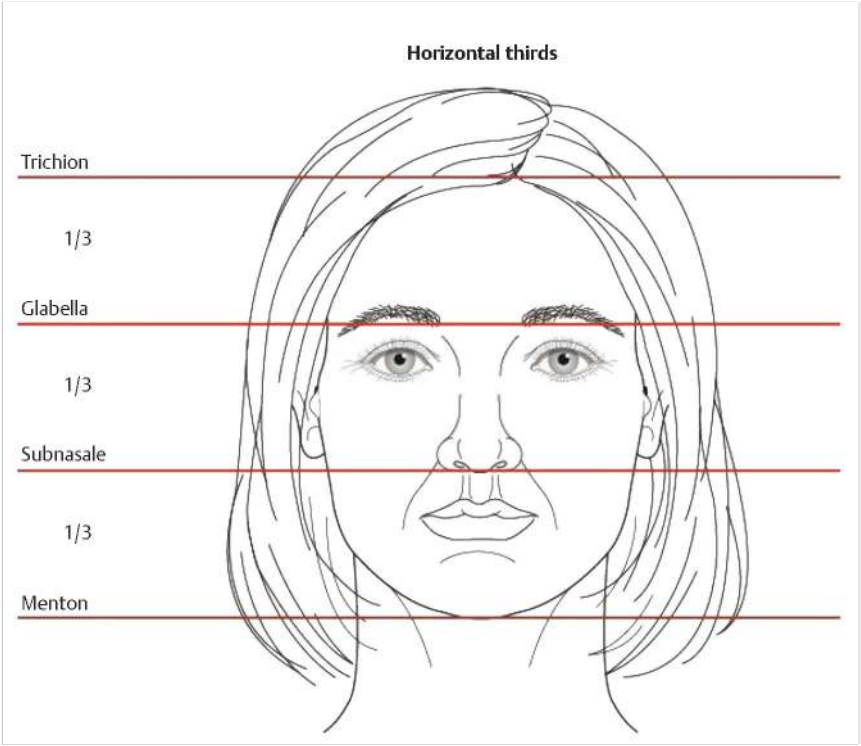


Fig. 6.10 Facial height is divided into equal thirds.

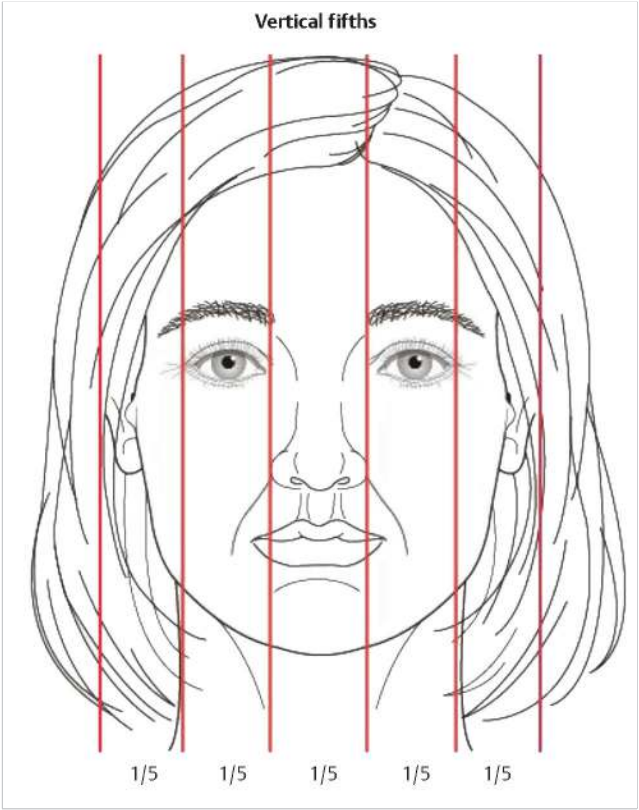


Fig. 6.11 Facial width is divided into equal fifths.

6.10.3 Width

Facial width is then divided into equal fifths to evaluate balance among the parts (▶ Fig. 6.11). The width of one eye opening should equal one fifth of the facial width and this in turn is the width of the intercanthal distance. The lateral fifths on frontal view extend from the lateral canthus to the lateral portion of the helical rim.

6.11 Lateral View Analysis

6.11.1 Height

Facial height is assessed using the same landmarks described previously (▶ Fig. 6.12). In addition, the lower third can be further subdivided into thirds with the stomion separating the upper and middle thirds and the pogonion located in the center of the lower third.

6.11.2 Midface Projection

To assess the relationship of the midface with the upper face, a line is drawn from the nasion to the subnasale. The angle formed with the Frankfort horizontal line should be a right angle on average (▶ Fig. 6.13).

6.11.3 Lower Face Projection

To assess the relationship of the lower face to the midface, a line is drawn from the subnasale to the pogonion perpendicular to the Frankfort horizontal line. If the pogonion is posteriorly positioned, the chin is retruding; if it is anteriorly positioned, the chin is protruding (▶ Fig. 6.13).

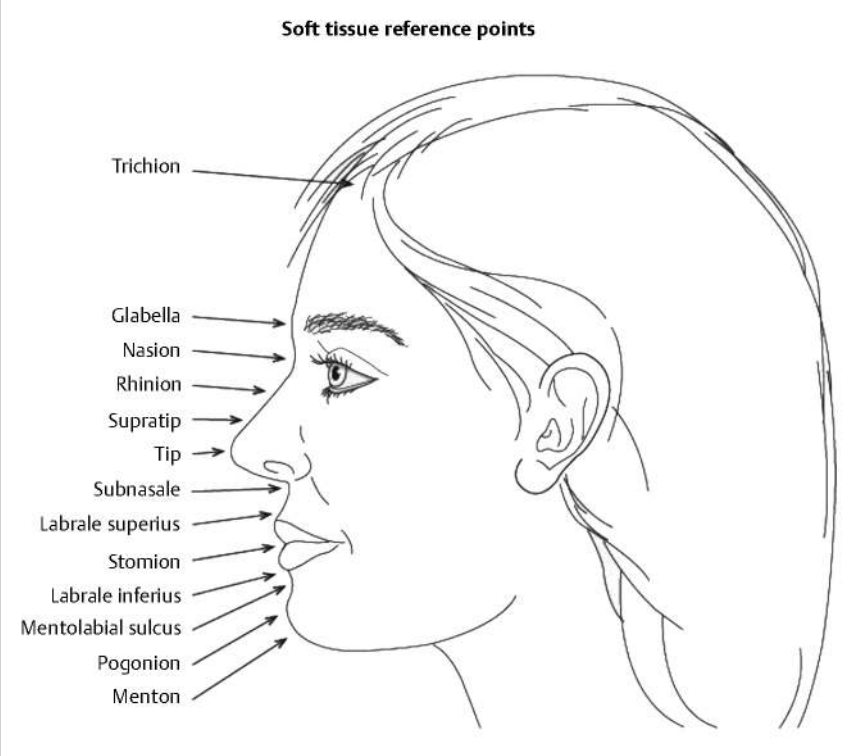


Fig. 6.12 Soft-tissue reference points (lateral view).

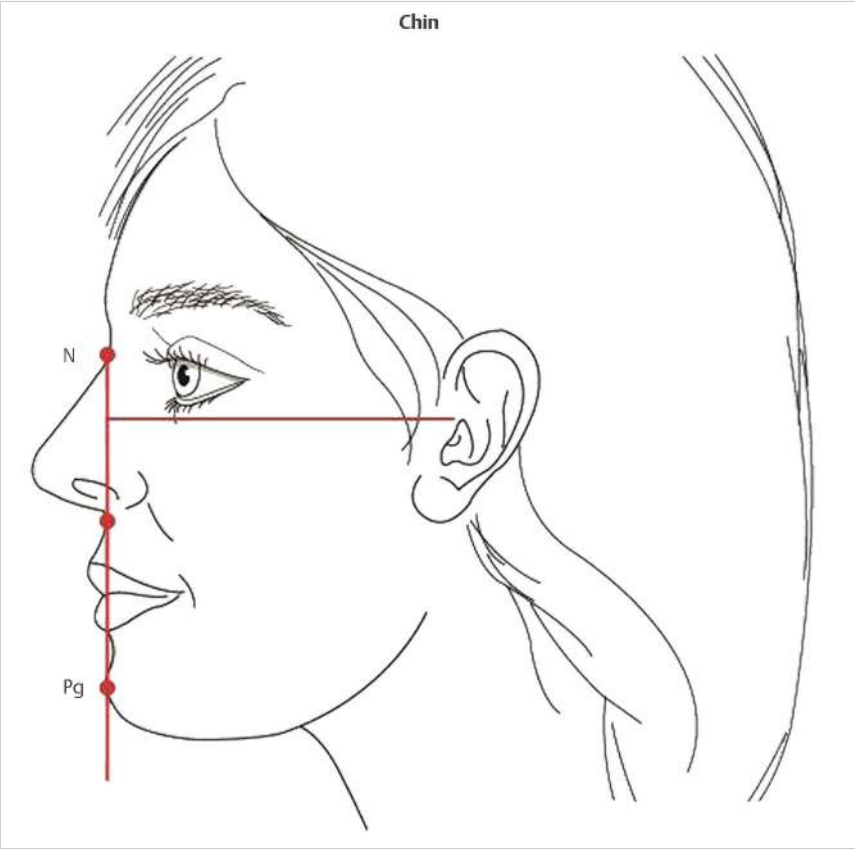


Fig. 6.13 The zero meridian of Gonzalez-Ulloa. The ideal chin position is a vertical line drawn from the nasion to the pogonion, which is perpendicular to the Frankfort horizontal line.

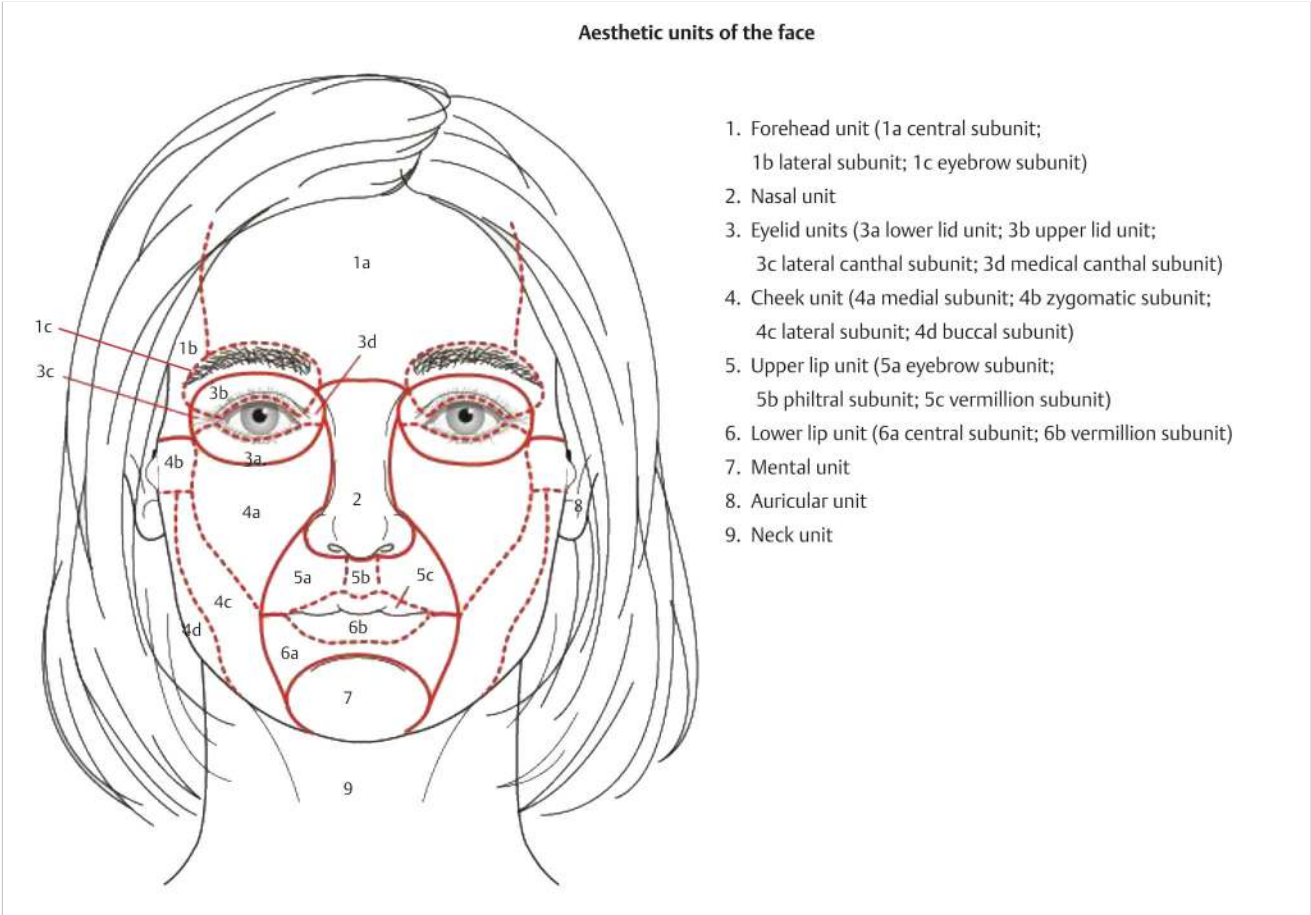


Fig. 6.14 Aesthetic units of the face.

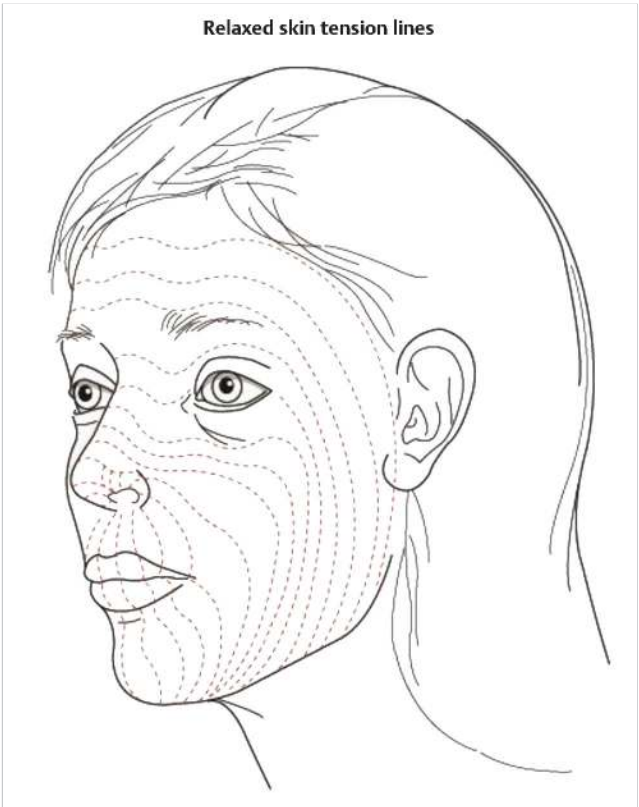


Fig. 6.15 Relaxed skin tension lines of the face.

6.12 Facial Aesthetic Units

The face can be further divided into aesthetic units for further analysis: forehead, nose, eyelid, cheek, lips, chin, ears, and neck (▶ Fig. 6.14). The units of the face are evaluated for contour, texture, skin thickness, and color. Incisions that fall along relaxed skin tension lines and at the intersection of the aesthetic units result in the least noticeable and most favorable scars (▶ Fig. 6.15).

6.13 Upper Third

6.13.1 Forehead and Eyebrow

Principles

The forehead constitutes the entire upper third of the face and is convex on profile with the most anterior point above the nasion at the level of the supraorbital ridge. The boundaries of the forehead are from the frontal hairline to the glabella. In general, men have a more prominent glabellar and supraorbital rim compared to women due to larger, more aerated frontal sinuses. Women tend to have a rounder, more convex forehead. The nasofrontal angle is determined by a tangent passing through the glabella and nasion and a tangent along the nasal dorsum; this angle ranges from 115 to 135 degrees which allows for a

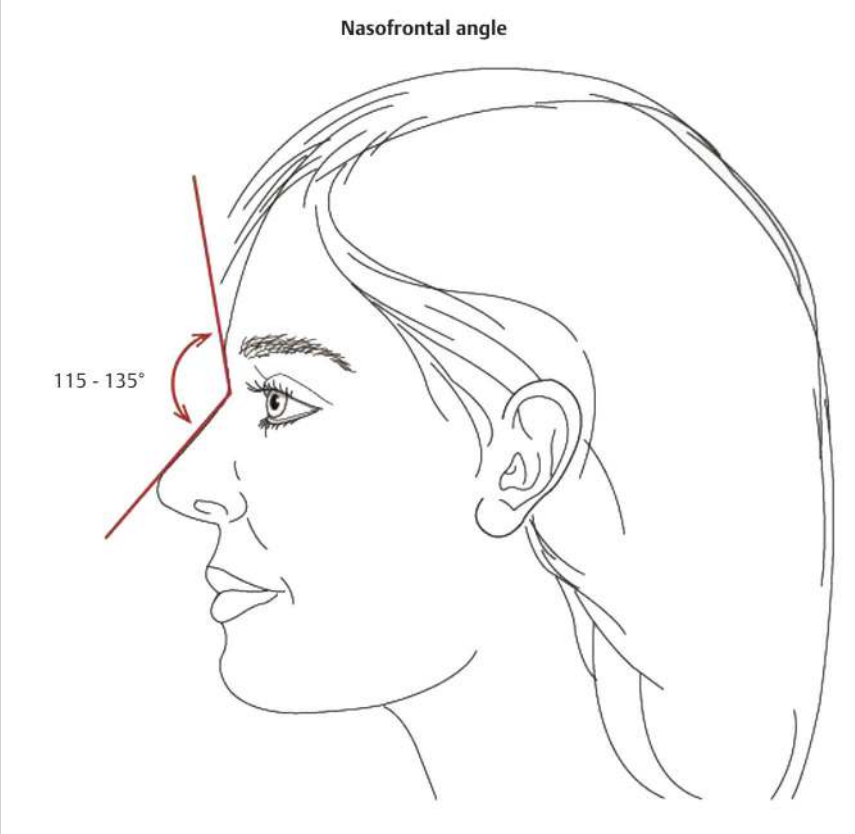


Fig. 6.16 Nasofrontal angle.

wide variation that is gender-, ethnic- and age-related (▶ Fig. 6.16). The layers of the forehead and scalp region are skin, subcutaneous tissues, aponeurosis, loose areolar tissue, and periosteum, which can be remembered by the acronym SCALP. The galea aponeurosis connects the bellies of the frontalis and occipital muscles; laterally, it thins to become incorporated into the superficial temporal fascia.

The female eyebrow is generally located 1 cm above the superior orbital rim and has a more prominent arch (▶ Fig. 6.17). The male eyebrow is located approximately at the level of the rim and is flat. The eyebrow extends medially to a position delineated by a vertical line drawn superiorly perpendicular to the alar base. The lateral aspect of the eyebrow is delineated by an oblique line drawn from the lateral aspect of the alar base through the lateral canthus. The medial and lateral ends of the eyebrow should be located at the same horizontal position. The apex of the eyebrow is delineated by a line drawn vertically from the lateral aspect of the corneal limbus.

6.13.2 Aging

With aging, the upper third of the face becomes elongated as the hairline recedes and the brow descends. In youth, the forehead muscles are covered with full, thick subcutaneous tissue, but as this fullness decreases with age, the mimetic action of the forehead muscles etch fine and deep rhytids into the face, giving rise to dynamic facial lines (▶ Fig. 6.18). The frontalis is the only elevator of the brow and contributes to deep horizontal rhytids. The procerus, corrugator, and orbicularis oculi all depress the brow. The procerus causes transverse glabellar rhytids and the corrugator supercilii causes vertical and oblique glabellar rhytids. Signs of aging in the brow can include brow droop and flattening caused by descent of the forehead inferiorly. Brow ridges may appear more prominent due to resorption of fat and thinning of the skin. Transverse furrows and rhytids, glabellar fullness and glabellar frown lines, temporal hollowing, and a thickened supraorbital rim also contribute to the aged appearance of the forehead.

6.13.3 Management

Hyperdynamic facial lines can be addressed with botulinum toxin, laser resurfacing, chemical peels, or surgery. Brow lifts can address the upper third of the face and are the best way to remove deep forehead rhytids. There are several techniques for browlifts, including direct, mid lift, coronal lift, pre-trichal and bi-planar lift, and the endoscopic brow lift. The benefits of the endoscopic lift are very small, camouflaged scars, less sensory impairment, and faster recovery.

6.13.4 Periorbital

Principles

The eyes and periorbital regions communicate intent, emotion, and alertness. The orbits are located in the lower third of the

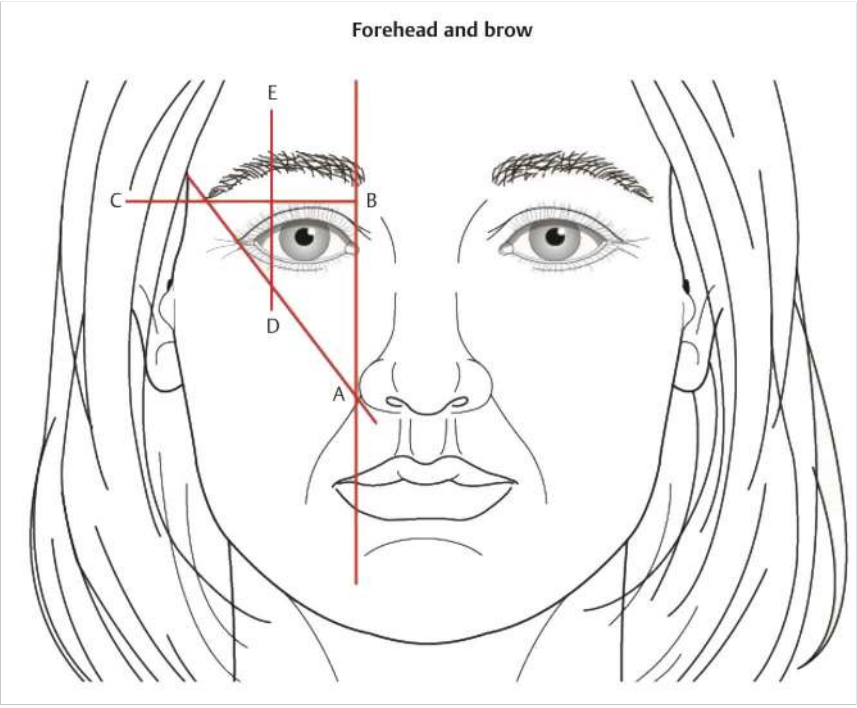


Fig. 6.17 Forehead and eyebrow. Eyebrow position is shown in relation to upper lid sulcus (Line C-B), lateral canthus and ala (diagonal line started at point A), medial canthus (Line B-A) and lateral limbus (Line E-D).

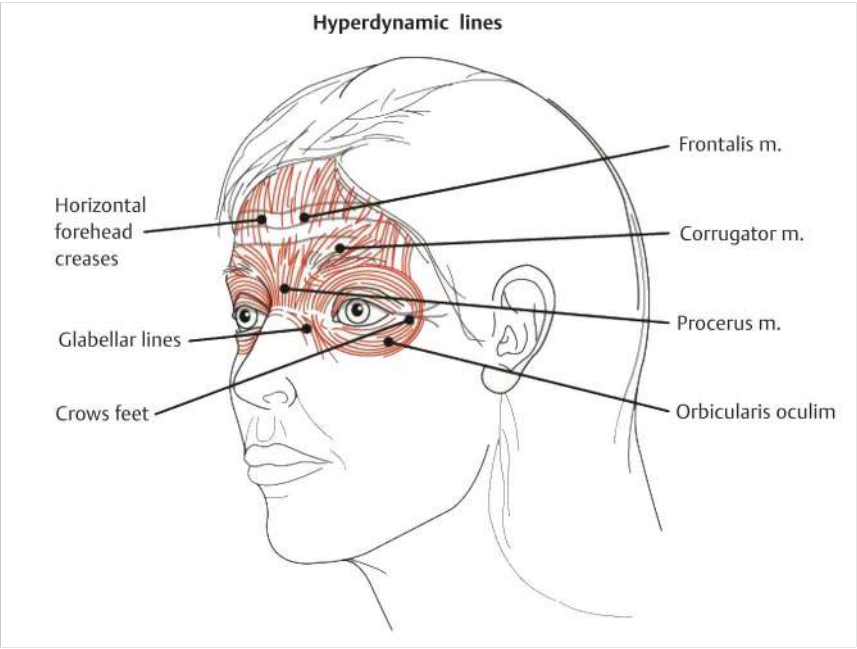


Fig. 6.18 Hyperdynamic lines contribute to fine lines and wrinkles.

upper face and the upper third of the midface. On frontal view, the width of one eye opening from medial to lateral canthus should be one fifth of the total facial width and the intercanthal distance should equal 30 to 35 mm or the width of one eye opening. The superior palebral lid crease is the attachment of the levator aponeurosis into the orbicularis in Caucasians and should be 7 to 10 mm from the eyelash line. The palpebral opening is 10 to 12 mm high and 28 to 30 mm in width. The lateral canthus should lie 2 to 4 mm above the medial canthus. The upper lid should cover 2 to 3 mm of superior iris and the lower lid approximates the inferior iris. The most inferior point of the lower lid margin is along a vertical line passing through the lateral limbus.

Facial analysis of the periorbital region must consider the brow position, dermatochalasia, lid fullness, lid position and symmetry, and lid laxity. Evaluation should include a history and physical examination with testing of visual acuity, extraocular muscle function, corneal sensitivity, cranial nerve VII sensitivity, Bell's phenomenon, lagophthalmos, lid ptosis, lid laxity, and lacrimal function. Lagophthalmos may be identified by

having the patient look downward while tilting the head backward. Horizontal laxity of the lower lid is tested with the "snap back (lid distraction) test" which is performed by pulling the lid away from the globe and releasing it; if it returns slowly, then significant laxity exists and should be noted as it may lead to ectropion post surgery. The strength of the orbicularis oculi is determined by having the patient close his or her eyes tightly while the examiner attempts to manually open them. On the lower eyelid, the presence of herniation of fat, commonly known as "bags," should be evaluated. On the upper eyelid, the presence of ptosis and an inferiorly displaced lacrimal gland should be noted, if present.

The periorbital vector refers to the relationship of the most projecting aspects of the globe and the malar prominence inferiorly, as seen on profile. A negative vector exists when the globe projection is more anterior and this indicates diminished skeletal support to the lower lid. A negative vector increases the risk for lid malposition after lower lid surgery.

6.13.5 Aging

The effects of aging become apparent in the eyes, perhaps sooner than anywhere else in the body. This may be displayed as periorbital hollowing, laxity in the upper and lower lids, narrowing of the horizontal and vertical dimensions of the palpebral fissures, obtuse canthal angles, weakening of the orbital septum with pseudoherniation or at times actual herniation of orbital fat, and hyperdynamic lines, all of which contribute to a tired expression (▶ Fig. 6.10). Bulges appear under the eyelid due to protrusion of the lower eyelid fat pads. The nasojugal groove (tear trough) begins to appear below the medial lower eyelid around 40 years of age and the inferior orbital rim becomes visible at around 50 years of age. Overall, the globe becomes more proptotic with respect to aging due to bone resorption of the inferior orbital rim and decreased fat pads in the cheeks. Repeated contractions of the orbicularis oculi results in wrinkles known as Crow's feet along the lateral orbital rim; these tend to form around the 5th decade.

6.13.6 Management

Signs of aging in the eyelids that can be corrected by various surgical and non-surgical means include increased laxity of the eyelid skin, protrusion of periorbital fat, ptotic brow resulting in hooding, rhytids in the lateral canthal region, ptotic lacrimal glands, xanthelasma, and hypotonicity of the lower lids. Upper blepharoplasty focuses on the superficial structures through a transcutaneous approach: removing excess skin, aesthetic placement of the supratarsal crease, and contouring the deep upper orbital sulcus. Lower blepharoplasty involves the deep structures through a transconjuncival or transcutaneous approach: addressing the orbitomalar sulcus and selective removal and repositioning of periorbital fat.[25] Excessive excision of the lower eyelid skin during blepharoplasty can result in scleral show or even ectropion. Complications of blepharoplasty can include retrobulbar hematoma, hematomas under skin flaps, epiphora, infection, diplopia, lagophthalmos, ectropion, incisional scar thickening, and even blindness.

6.14 Middle Third

6.14.1 Cheeks

Principles

The soft tissue of the cheek projects anteriorly and laterally over the body of the zygoma and the zygomatic arch. The deep cheek fat compartments contribute to the convexity of the cheek and lid-cheek junction. The ovoid cheek axis is angled from the lateral oral commissure to the base of the ear helix. The malar prominence is located eccentrically on the cheek mound over the zygoma, extending up to the inferolateral orbit and is covered with malar fat. An ogee curve has been described at the level that transitions between the lateral orbital wall and the cheek at the lid-cheek junction.

6.14.2 Aging

In youth, the cheeks show smooth round contours. With aging, there is a loss of homogeneity as the nasolabial folds lengthen and deepen and the cheeks lose volume. Overall, the midface descends inferiorly and medially. Around age 50, the skin also becomes more pigmented, sallow, and coarse, and the cheeks lose their projection and appear hollow. The inferior orbital rim resorbs and remodels, losing anterior projection and vertical height. Increased bony resorption may also be secondary to a peri-menopausal state in the mid-50 s of females.

6.14.3 Management

Midface and cheek restoration should involve volume enhancement and contouring. Non-surgical use of fillers such as hyaluronic acid or poly-L-lactic acid can improve the appearance of the aged midface. Autologous fat grafting has become very popular because of its safety profile and effectiveness; it can be done alone or as a combined procedure. Fat grafting provides longer lasting results compared to fillers. Surgical suspension of the midface is the most effective and long-lasting and can be done as part of a facelift or alone as a minimally invasive malar elevation with or without barbed suture. Cheek implants with silicone, Medpor®, or Gore-Tex® have also been used and provide permanent results.

6.14.4 Nose

Principles

As a central aesthetic unit, the nose is a focal point and defining feature for the face and must be analyzed in relationship to other facial structures. Burget and Menick described the topographic subunits of the nose (▶ Fig. 6.19), which are important to consider for surgical planning. On lateral view, the nose begins at the nasion superiorly at the level of the superior palpebral fold of the upper eyelid and extends to the subnasale. From a worm's eye view, the nose is triangular in shape and divided into three units (▶ Fig. 6.20). The columella should be approximately 3–5 mm in width.

The relationships of the nose with other facial structures can be defined by the nasofrontal angle, nasolabial angle, nasofacial angle, and nasomental angle. The nasofrontal angle is the angle

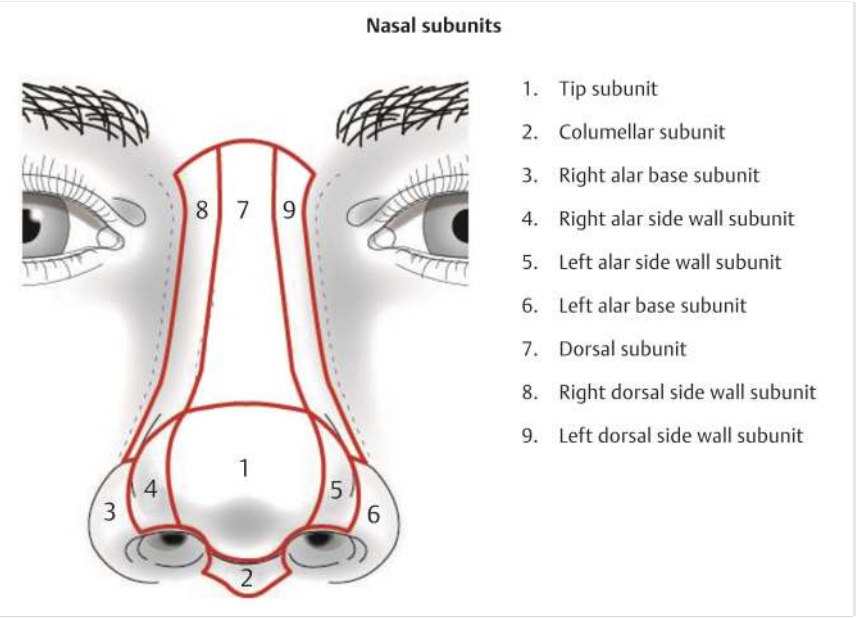


Fig. 6.19 Nasal aesthetic subunits (per Burget and Menick).

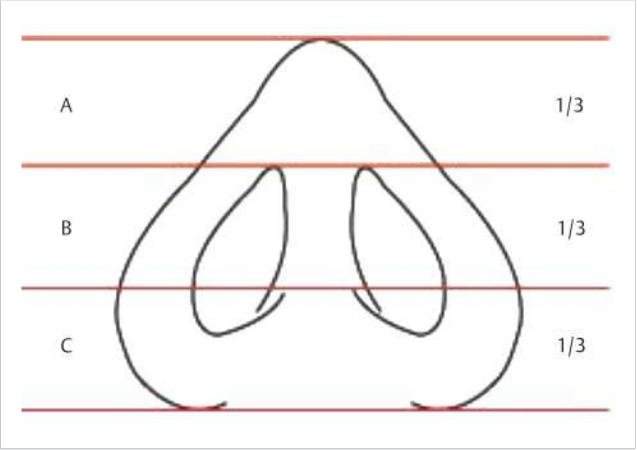


Fig. 6.20 Basal view of the nose divided into equal thirds.

formed by the intersection of a line tangent to the glabella through the nasion and a line tangent to the nasal dorsum with a range of 115 to 135 degrees (▶ Fig. 6.16). The nasolabial angle is the angulation of the columella and upper lip (▶ Fig. 6.21). This angle is formed by the intersection of a line tangent to the labrale superius and subnasale and a line tangent to the subnasale and the most anterior portion of the columella. The ideal measurement is 95 to 110 degrees in females and 90 to 105 degrees in males. The nasofacial angle is the relationship of the nasal dorsum to the facial plane and is the angle formed by the intersection of a vertical line tangent from the glabella through the pogonion and a line from the nasion through the nasal tip with an ideal angle of 36 degrees with a range 30 to 40 (▶ Fig. 6.22). The nasomental angle is the angle formed by the intersection of a line tangent from the nasion to the nasal tip and a line from the tip to the pogonion (▶ Fig. 6.23). The ideal range of this angle is 120 to 132 degrees.

6.14.5 Aging

The nose suffers significant changes with aging, notwithstanding that it has a cartilaginous and skeletal framework. The overlying skin thins and stretches, revealing a more prominent dorsal hump and nasal tip and columella. The upper and lower lateral nasal cartilages weaken and descend, giving the appearance of a ptotic nasal tip. Remodeling of the alar base and maxillary resorption around the pyriform aperture also results in accentuated nasal droop. Recent reports indicate that the nasal tip itself may not drop; rather, the structures around it including the muscles and ligaments may retract giving the appearance of nasal tip ptosis (verbal communication with Val Lambros, MD).

6.14.6 Management

Cosmetic rhinoplasty for the aging nose takes into consideration the resorptive and atrophic changes that occur in the hard and soft tissues. These also have an effect on breathing that involves the septum and turbinates as well. Septoplasty and turbinate reduction procedures have been well described. Through open and closed techniques, several modifications can be made which will affect the shape and profile of the nose. The nasal dorsum can be reduced or narrowed and the nasion burred or grafted. This can also give the appearance of a smaller or larger nose. The nasal tip can be elevated or rotated to give a sharper profile. A wide or bulbous tip can be narrowed and thinned or even grafted to provide more ideal tip defining points. A hanging or retracted columella or ala can be adjusted with suture or grafting. The width of the caudal nose can be adjusted with alar excisions (Weir) or a cinching stitch that can be later modified or removed. Various grafting and suture techniques have been described to try to arrive at a more ideal and harmonious result. Methods to increase nasal projection and rotation include columellar strut graft, tip graft, lateral crural batten grafts, tongue-in-groove techniques, and others.

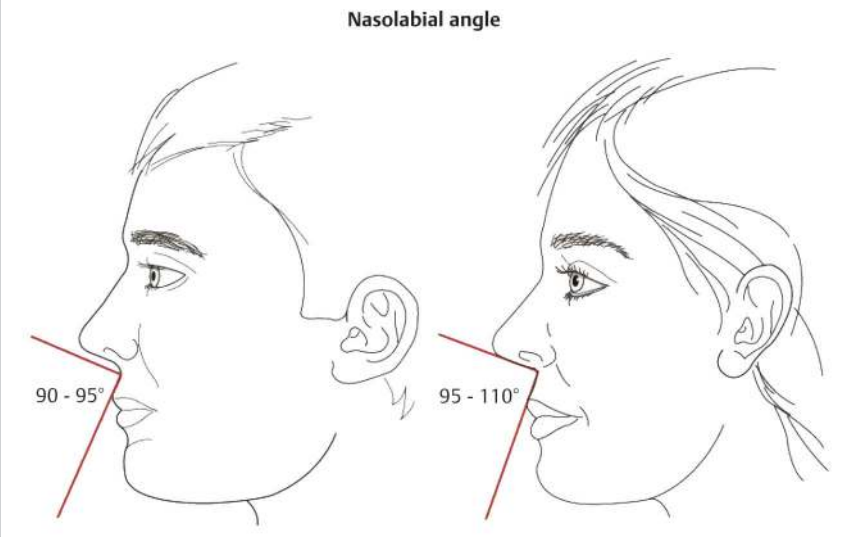


Fig. 6.21 Nasolabial angle.

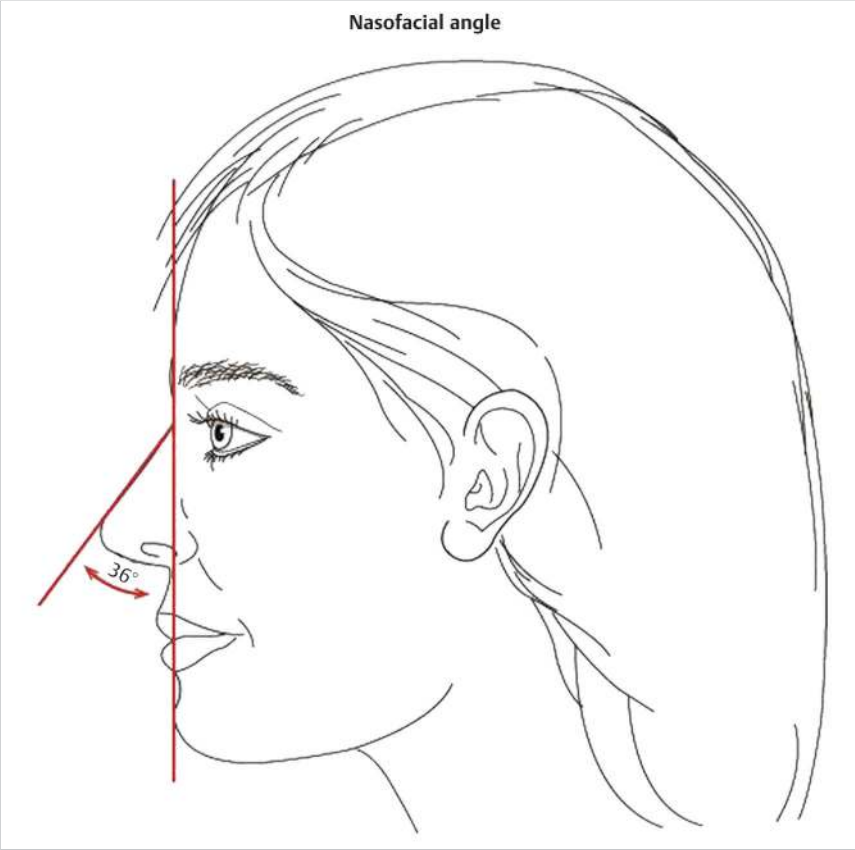


Fig. 6.22 Nasofacial angle.

6.14.7 Ears

Principles

Landmarks of the ear include the concha, helix, antihelix, tragus, anti-tragus, lobule, and scapha (▶ Fig. 6.24). These structures should be examined for deformity. The thickness and flexibility of the cartilage should also be determined. The ear can be divided vertically into three approximately equal parts with the tragus at the midpoint. Ear height is about equal to the midfacial height. The width of the ear is about 60% of the height with the range of 50 to 65%. The long axis of the ear is inclined posteriorly about 15 to 20% from the true vertical plane. The top of the ear is usually at the level of the lateral eyebrow and the bottom at the level of the base of the nose. Several craniofacial syndromes are associated with low-set ears.

6.14.8 Aging

The aging ear will tend to descend and stretch, especially the earlobe. The rest of the ear that contains cartilage better maintains its structure although they may appear larger and more prominent with time.

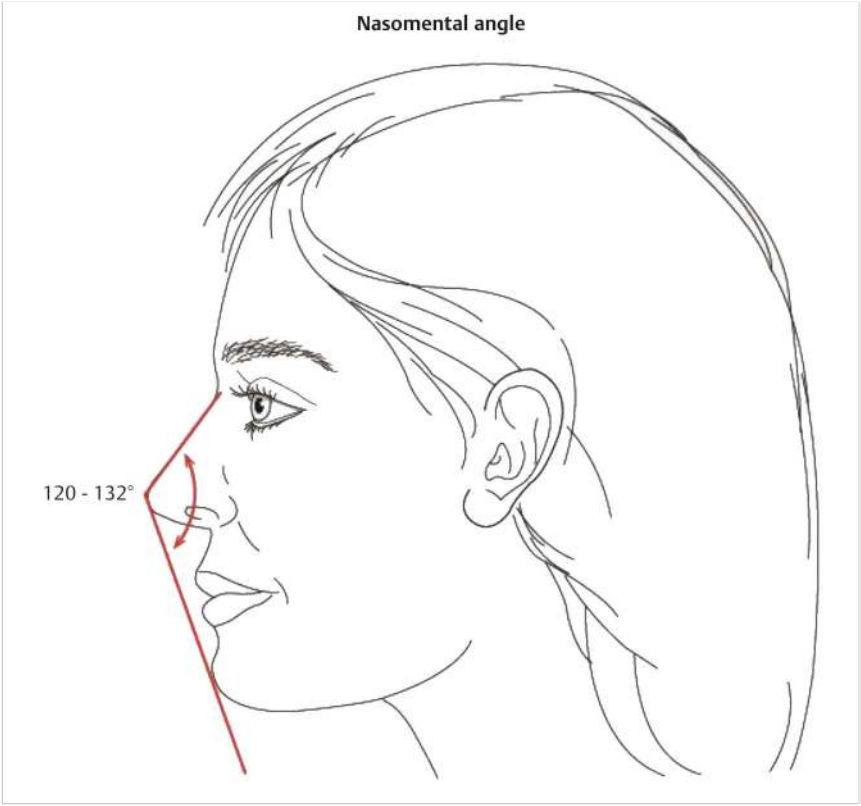


Fig. 6.23 Nasomental angle.

Fig. 6.24 Image of the right ear. (1) Concha. (2) Helix. (3) Anti-helix. (4) Tragus. (5) Anti-tragus. (6) Lobule. (7) Fossa triangularis. (8) Scapha.

6.14.9 Management

The lobe can be corrected usually at the time of a facelift by removing a wedge and making it smaller and more rounded. Various types of otoplasty can be performed to correct prominent ears and the upper ear can be lifted slightly.

6.15 Lower Third

6.15.1 Perioral

Principles

The perioral region and mouth express our emotional state, conveying happiness or sadness and anger or fear. The lip shape should be evaluated according to the shape in profile, the lip length in relation to the frontal incisors, and the degree of vermillion inversion. The lip surface should be evaluated according to the presence and degree of radial wrinkles and the visibility of the structural elements of Cupid's bow, philtrum, and white roll (▶ Fig. 6.25).

Aging

Perioral aging is characterized by fine lines, marionette lines, flattening of the Cupid's bow, long and ill-defined philtrum, and a narrow vertical and wider transverse smile.[37] These changes are the result of genetics, photoaging, smoking, gravity, repetitive pursing of the orbicularis oris, changes of dentition, and maxillomandibular resorption. Resorption of the maxilla can also contribute to loss of upper lip support, resulting in perioral wrinkling.

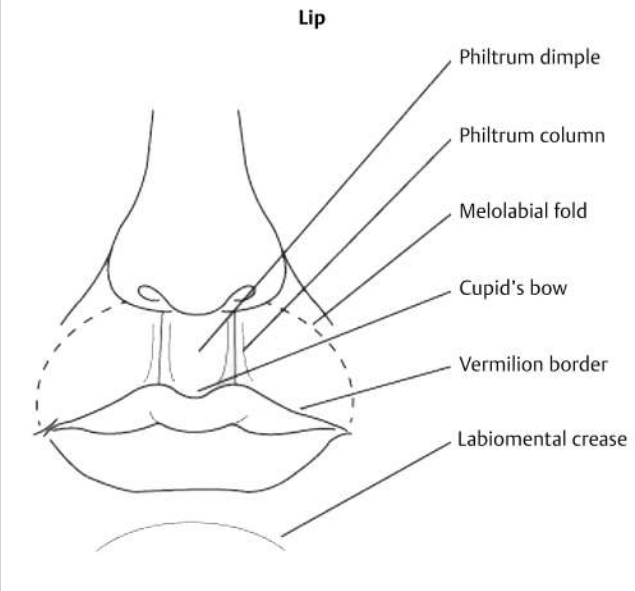


Fig. 6.25 Aesthetic subunits of the lip.

Management

Treatment of fine and coarse rhytids can be addressed with various chemical peels, laser resurfacing, or the injection of filler materials. Injection of botulinum toxin A to relax the perioral musculature has also been described. Care should be exercised with botulinum toxin A to relax the periorbicularis oris contributing to perioral rhytids so as not to produce loss of adequate dynamic function.

6.16 Lips

6.16.1 Principles

The upper lip is measured from the subnasale to the stomion superius and the lower lip is measured from the stomion inferius to the labial-mental crease. The aesthetic units of the lip are illustrated in ▶ Fig. 6.25. The oral commissures should be located along vertical lines drawn from the medial limbus of the iris. In youthful Caucasian lips, the ideal vertical height ratio of the upper lip to the lower lip is 1:1.6; however, there is wide patient variation in ideals of lip size and contour. The typical goal of lip augmentation in Caucasian women is to augment the lip size beyond the natural size. In aged populations, the goal is to restore the size of the lip to the original shape and volume. The anterior-posterior position of the lips is evaluated with the Holdaway Harmony line, which is a line 10 degrees anterior to a line from the pogonion and a line from the pogonion to the glabella.

The horizontal lip position can be determined with the nasomental angle, where the lips are approximately 4 mm behind the line for the upper lip and 2 mm behind for the lower lip. The horizontal lip position can also be determined by a vertical line from the subnasale through the labrale inferius to the pogonion. The upper lip and lower lip should lie approximately 3.5 mm and 2.2 mm anterior to the line drawn perpendicular to the most anterior portion of each lip, respectively (▶ Fig. 6.26). The

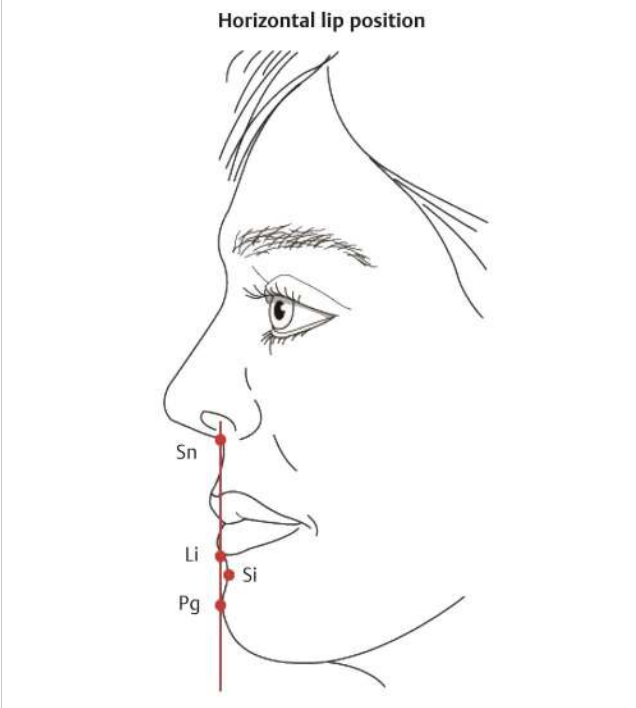


Fig. 6.26 Horizontal lip position. Sn is subnasale. Li is labrale inferius. Si is menotlabial sulcus. Pg is pogonion.

interlabial gap and degree of incisor show with smiling is also important. When smiling, no more than two-thirds of the maxillary incisors should be exposed and there should be no gingival show.

6.16.2 Aging

Well-defined and full lips are a sign of youth. With aging, the proportion of the lips change with depression of the corners of the mouth, lengthening of the cutaneous portion of the upper lip and loss of volume of the upper lip vermillion, decreased elasticity of the subcutaneous soft tissue, retraction of the lip, and loss of teeth contribute to perioral aging. Flattening of the philtral columns occurs with age, and re-creation of the philtral columns can restore a youthful appearance.

6.16.3 Management

Hyaluronic acid products are the most commonly used fillers for the lips and demonstrate excellent safety. Restylane is the only FDA-approved hyaluronic acid filler specifically for lip augmentation, but several hyaluronic acid products are used off-label for lip augmentation (such as Juvéderm and Perlane). Fat grafting has become popular for lip augmentation, but entails a more invasive surgical procedure with subsequent bruising, swelling, and a longer recovery period when compared with fillers. A long vertical lip is common in the aged female; a very sure and dramatic method of treatment is a gull-wing excision at the junction of the upper lip and the alar bases. The scar in the aged patient heals very well and can give satisfactory cosmetic results. Alternatively, an extended cupid's bow excision of the lip skin at the vermillion junction reduces the length of the

lip and provides greater mucosal show. The permanent scar is usually acceptable, but often will require camouflage lip liner makeup.

6.17 Dentition

Dentition should be evaluated and considered in the context of facial aesthetics as it influences facial soft tissues and overall cosmesis. Occlusal planes, alignment, incisor flare and length, and many other factors influence facial analysis. In normal occlusion, or Type I occlusion, the mesiobuccal cusp of the maxillary first molar occludes the buccal groove of the mandibular first molar; in facial profile, this is mesognathic. Type II occlusion occurs when the buccal groove of the maxillary first molar is distal to the mesiobuccal cusp of the maxillary first molar; in facial profile, this is termed retrognathic. Type III occlusion occurs when the buccal groove of the mandibular first molar is mesial to the mesiobuccal cusp of the maxillary first molar; in facial profile, this is prognathic. Severe dentoalveolar relationships can be addressed with orthognatic surgery.

6.18 Chin and Neck

6.18.1 Principles

The protruding chin is unique to humans. A larger cranium, taller forehead, reduced prominence of the brow ridge, reduced bimaxillary protrusion, and a pronounced bony chin distinguish *Homo sapiens* from *Homo erectus*. It confers strength to the face and forms the lower portion of the face. On profile, it is key to establish the character of the lower face and to speak of a strong or weak chin may imply personality. Gonzalez-Ulloa described the ideal chin position to be on a line perpendicular to the Frankfort horizontal passing through the nasion and pogonion (±2 mm).

The neck is a cylindrical structure that connects the lower face with the upper chest. It contains a plethora of very important structures that are invested by muscles, fascia, subcutaneous tissue, and skin. The neck is usually divided into anterior and posterior triangles that are bordered by the sternocleidomastoid and trapezius muscles laterally, the sternum and clavicles inferiorly, and the chin and lower border of the mandible superiorly. The hyoid bone lies in the central region of the neck between the chin and thyroid cartilage and serves as a place of attachment of several muscles of the neck, but does not have any direct bony attachments or articulations. It also serves to stabilize the larynx, epiglottis, and pharynx. It provides the junction of the cervicomental angle which is distinctive to a beautiful neck profile.

The cervicomental angle is an angle formed by a line drawn from the glabella to the pogonion and from the menton to the cervical point; this should be 105 to 120 degrees and is more acute in males compared to females (▶ Fig. 6.27). The mentolabial sulcus (Si) separates the cutaneous lower lip from the chin (▶ Fig. 6.26). The deepest point of the Si should lie -4 mm behind the line drawn between the vermillion border of the lower lip (labrale inferius, LI) and the pogonion (Pg). Gonzalez-Ulloa described the ideal chin position as a tangential line through the N to the Pg (▶ Fig. 6.13). The nasomental angle is a line drawn from the nasal tip to the pogonion.

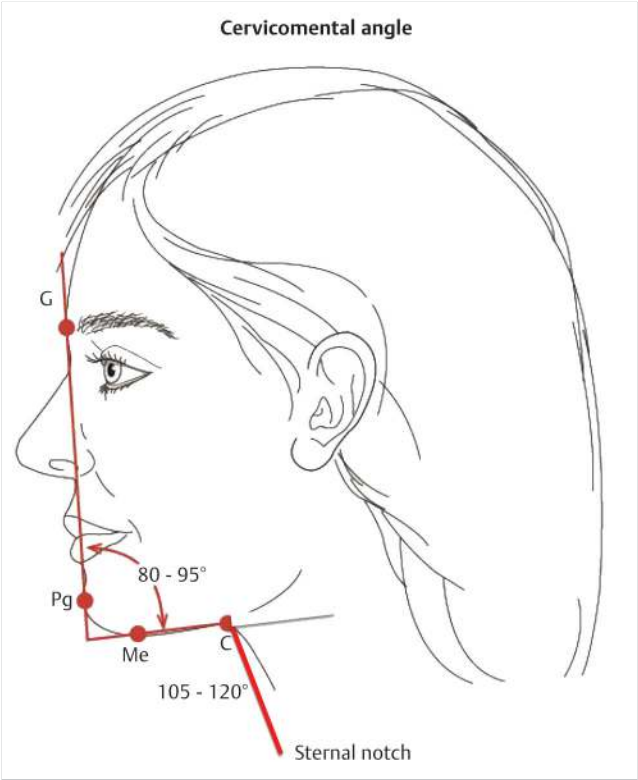


Fig. 6.27 Cervicomental angle.

6.18.2 Aging

The cervicomental angle is typically blurred or lost with aging of the neck and is a common source of concern and consultation in those seeking rejuvenation of the face and neck. The skin, subcutaneous tissues, superficial fascia, and platysma are mostly involved in the aging features of the neck and are the structures most commonly addressed in rejuvenation. The anterior digastric muscles, submandibular glands, and superficial and deep fat deposits are also important in treating the aging neck.

Aging of the neck essentially involves volumetric and directional or vector-based changes. Visibly, it starts with sagging and stretching of the skin and soft tissues; they can become thin and wrinkled or dense and full and everything in between. Fat deposits in the superficial and deep areas may accumulate or droop, adding to an aged appearance. The platysma muscle will become lax and lose its investing properties, contributing to sagging. The muscle also commonly forms bands at sites of continual contraction. The submandibular glands will experience descent as their surrounding fascia relaxes and may become quite visible.

6.18.3 Management

Genioplasty can be achieved through osteoplastic or alloplastic means. It can address projection and width, but soft tissue considerations include jowl sulci, marionette lines, and contouring of the mental crease with injectables.

Treatment of the aging neck will depend on other areas on the extent of deformity from the ideal and the concern of the

patient. The ideal may be related to the principles of aesthetics discussed or what the patient looked like or remembers she looked like at a younger age. Classifications of the aging neck can help to guide the surgeon in addressing certain deformities. The Dedo classification has been proposed, but has not received wide use although it mentions many of the different deformities and structures that can be corrected. A series of minimally invasive and less invasive techniques have been described including neuromodulators, fillers, and energy-based technologies. Liposuction can be effective in cases of mild to moderate fat accumulation. The more aggressive open techniques are without a doubt much more effective for the moderate to severe aged neck.

6.19 Conclusion

Facial aesthetics is a subject of interest not only to plastic surgeons, but also to observers of beauty throughout history and in many areas of nature and art. We have described some of the principles of facial aesthetics, especially as it relates to the evaluation and management of patients desiring aesthetic improvement and rejuvenation. The multifaceted aspects of aging are becoming better understood and therefore we are in a better position to correct them appropriately. A good and qualified plastic surgeon has an excellent sense of facial aesthetic based on his or her trained eye and experience.

6.20 Review Questions

6.20.1 Choose the Best Answer

1. The Fibonacci ratio is often quoted when discussing aesthetic facial proportions. Which one of the following numbers represents this “golden ratio”?
   a) 1:0.33
   b) 1:0.67
   c) 1:1.33
   d) 1:1.62
   e) 1:2
2. Which of the following statements is incorrect when considering the ideal facial proportions on frontal view?
   a) The facial frontal view is divided into vertical fifths.
   b) With a receding hairline, the most superior border of the upper third can be determined by identifying the most superior movement of the frontalis muscle.
   c) The width of one eye opening should equal one fifth of the facial width and this in turn is the width of the intercanthal distance.
   d) The upper lip vermilion marks the vertical midpoint of the lower facial third.
   e) The female eyebrow is generally located 1 cm above the superior orbital rim and has a more prominent arch compared to males.
3. Which of the following is increased in the aging skin?
   a) Epidermal thickness.
   b) Melanocyte number.
   c) Dermal matrix.
   d) Number of fibroblasts.
   e) Ratio of type III to I collagen.
4. With regard to the lower face, which of the following statements is incorrect?
   a) The cervicomental angle is generally more acute in females compared to males.
   b) When smiling, no more than two-thirds of the maxillary incisors should be exposed.
   c) In type I occlusion, the mesiobuccal cusp of the maxillary first molar occludes the buccal groove of the mandibular first molar.
   d) The ideal chin position has been described as a line perpendicular to the Frankfort horizontal passing through the nasion and pogonion.
   e) Perioral aging is characterized by flattening of the Cupid’s bow.

6.20.2 Answers

1. d. 1:1.62. The Fibonacci sequence is a set of numbers starting with 1 or 0 and proceeds as 1, 2, 3, 5, 8… where two consecutive numbers add up to form the next number. The “golden ratio” or Phi is a ratio established by the Greeks and used by the Renaissance artists that equals 1:1.62. This ratio is characteristic of forms of aesthetic proportions in nature and in facial proportions.
2. d. The upper lip vermilion marks the vertical midpoint of the lower facial third.

Facial proportions and contours can vary with sex, age, and race, but some aesthetic ideals persist. The facial frontal view is divided into vertical fifths and horizontal thirds. The boundaries are from the trichion to the glabella (upper face), from the glabella to the subnasale (middle face), and from the subnasale to the menton (lower face). With a receding hairline, the most superior border of the upper third can be determined by identifying the most superior movement of the frontalis muscle. The lateral and medial canthi can be used to divide the face into vertical fifths. The lower lip vermillion, and not the upper lip vermillion, marks the vertical midpoint of the lower third of the face. The female eyebrow is generally located 1 cm above the superior orbital rim and has a more prominent arch compared to males.

3. e. Ratio of type III to I collagen. The ratio of type III to I collagen increases with age. In the epidermis, decreased cellular turnover, reduced keratinocytes and epidermal lipids, and decreased moisture result in dry skin. Altered pigmentation results from fewer melanocytes and melanin production. In the dermis, reduced repair and turnover and reduced function of the sebaceous and sweat glands cause dry skin. Histologically, there are fewer fibroblasts and collagen production, reduced dermal matrix, and thicker elastin fibers with less elasticity.
4. a. The cervicomental angle is generally more acute in females compared to males. When smiling, no more than two-thirds of the maxillary incisors should be exposed and there should be no gingival show. Type I occlusion refers to when the mesiobuccal cusp of the maxillary first molar occludes the buccal groove of the mandibular first molar.

One description of the ideal chin position is a line perpendicular to the Frankfort horizontal passing through the nasion and pogonion. The cervicomental angle is generally more acute in males compared to females. Perioral aging is characterized by fine lines, marionette lines, flattening of the Cupid's bow, long and ill-defined philtrum, and a narrow vertical and wider transverse smile.

References

[1] Kurzban R, Weeden J. HurryDate: Mate preferences in action. Evol Hum Behav. 26:227–244

[2] Buss DM, Barnes M. Preferences in human mate selection. J Pers Soc Psychol. 50:559–570

[3] Cash TF, Kilcullen RN. The eye of the beholder: susceptibility to sexism and beautyism in the evaluation of managerial applicants. J Appl Soc Psychol. 15: 591–605

[4] Riggio RE, Woll SB. The role of nonverbal cues and physical attractiveness in the selection of dating partners. J Soc Pers Relat. 1:347–357

[5] Griffin AM, Langlois JH. Stereotype directionality and attractiveness stereotyping: is beauty good or is ugly bad? Soc Cogn. 24(2):187–206

[6] Pivonkova V, Rubesova A, Lindova J, Havlicek J. Sexual dimorphism and personality attributions of male faces. Arch Sex Behav. 40(6):1137–1143

[7] Thakerar JN, Iwawaki S. Cross-cultural comparisons in interpersonal attraction of females toward males. J Soc Psychol. 108(1):121–122

[8] Langlois JH, Kalakanis L, Rubenstein AJ, Larson A, Hallam M, Smoot M. Maxims or myths of beauty? A meta-analytic and theoretical review. Psychol Bull. 126(3):390–423

[9] Cunningham MR, Roberts AR, Barbee AP, et al. "Their ideas of beauty are, on the whole, the same as ours": Consistency and variability in the cross-cultural perception of female physical attractiveness. J Pers Soc Psychol. 68:261–279

[10] Martin JG. Racial ethnocentrism and judgment of beauty. J Soc Psychol. 63: 59–63

[11] Langlois JH, Roggman LA, Rieser-Danner LA. Infants' differential social responses to attractive and unattractive faces. Dev Psychol. 26:153–159

[12] Langlois JH, Roggman LA, Casey RJ, et al. Infant preferences for attractive faces: Rudiments of a stereotype? Dev Psychol. 23:363–369

[13] Samuels CA, Butterworth G, Roberts T, Graupner L, Hole G. Facial aesthetics: babies prefer attractiveness to symmetry. Perception. 42(11):1244–1252

[14] Cellerino A. Psychobiology of facial attractiveness. J Endocrinol Invest. 26(3) Suppl:45–48

[15] Baudouin JY, Tiberghien G. Symmetry, averageness, and feature size in the facial attractiveness of women. Acta Psychol (Amst). 117(3):313–332

[16] Langlois JH, Roggman LA. Attractive faces are only average. Psychol Sci. 1: 115–121

[17] Galton JC. Recent observations upon the placentation of the sloths. Nature. 18:686–687

[18] Rhodes G, Sumich A, Byatt G. Are average facial configurations attractive only because of their symmetry? Psychol Sci. 10:52–58

[19] Grammer K, Thornhill R. Human (Homo sapiens) facial attractiveness and sexual selection: the role of symmetry and averageness. J Comp Psychol. 108 (3):233–242

[20] Keating CF. Gender and the physiognomy of dominance and attractiveness. Soc Psychol Q. 48:61

[21] Cunningham MR. Measuring the physical in physical attractiveness: Quasi-experiments on the sociobiology of female facial beauty. J Pers Soc Psychol. 50:925–935

[22] Prokopakis EP, Vlastos IM, Picavet VA, et al. The golden ratio in facial symmetry. Rhinology. 51(1):18–21

[23] Marquardt SR. Dr. Stephen R. Marquardt on the Golden Decagon and human facial beauty. Interview by Dr. Gottlieb. J Clin Orthod. 36(6):339–347

[24] Kim YH. Easy facial analysis using the facial golden mask. J Craniofac Surg. 18 (3):643–649

[25] Holland E. Marquardt's Phi mask: pitfalls of relying on fashion models and the golden ratio to describe a beautiful face. Aesthetic Plast Surg. 32(2):200–208

[26] Pallett PM, Link S, Lee K. New "golden" ratios for facial beauty. Vision Res. 50 (2):149–154

[27] Rossetti A, De Menezes M, Rosati R, Ferrario VF, Sforza C. The role of the golden proportion in the evaluation of facial esthetics. Angle Orthod. 83(5): 801–808

[28] Milutinovic J, Zelic K, Nedeljkovic N. Evaluation of facial beauty using anthropometric proportions. Sci World J. 2014:428250

[29] Mommaerts MY, Moerenhout BA. Ideal proportions in full face front view, contemporary versus antique. J Craniomaxillofac Surg. 39(2):107–110

[30] Powell N, Humphries B. Proportions of the Aesthetic Face. New York, NY: Thieme-Stratton; 1984

[31] Coleman SR, Grover R. The anatomy of the aging face: volume loss and changes in 3-dimensional topography. Aesthet Surg J. 26 1S:S4–S9

[32] Gosain AK, Klein MH, Sudhakar PV, Prost RW. A volumetric analysis of soft-tissue changes in the aging midface using high-resolution MRI: implications for facial rejuvenation. Plast Reconstr Surg. 115(4):1143–1152, discussion 1153–1155

[33] Wan D, Amirlak B, Rohrich R, Davis K. The clinical importance of the fat compartments in midfacial aging. Plast Reconstr Surg Glob Open. 1(9):e92

[34] Bartlett SP, Grossman R, Whitaker LA. Age-related changes of the craniofacial skeleton: an anthropometric and histologic analysis. Plast Reconstr Surg. 90 (4):592–600

[35] Choi JY, Lee SH, Baek SH. Effects of facial hard tissue surgery on facial aesthetics: changes in facial content and frames. J Craniofac Surg. 23(6):1683–1686

[36] Hom DB, Marentette LJ. A practical methodology to analyze facial deformities. Head Neck Surg. 109:826–838

[37] Iblher N, Stark GB, Penna V. The aging perioral region – Do we really know what is happening? J Nutr Health Aging. 16(6):581–585

7 Lasers and Radiant Energy in Plastic Surgery

Richard Gregory

Abstract

This chapter is an introduction to laser use in plastic surgery. It is meant as a stimulus to the plastic surgeon who wants to use lasers in plastic surgery or the skin medicine field. The laser is a useful tool in the armamentarium of a cosmetic and reconstructive plastic surgeon. Knowledge of its many uses will complement the skills of the surgeon and facilitate the treatment of the aging process, as well as treating photodamaged skin, birthmarks, and a variety of dermatological disorders such as unwanted hair. Avoiding complications and building a practice are just two of the many ancillary benefits of laser education. There are many good resources to expand on the information presented herein, and a few are listed in the selected references at the end of the chapter. Those wanting to use lasers in medicine would be well advised to join the American Society of Laser Medicine and Surgery (ASLMS). Further information can be obtained online at www.aslms.org.

Keywords: laser, Nd:YAG, KTP, carbon dioxide, Alexandrite, photothermolysis, pulsed dye

7.1 Objectives

- Introduce the plastic surgeon to the theory and practice of laser use.
- Understand the basics of physics and physiology of lasers and radiant energy.
- Gain insight into the various laser technologies available and the pathology that can be treated.
- Provide the foundation for further study, ultimately incorporating lasers into the plastic surgery practice.

7.2 Introduction

This chapter on "Lasers and Radiant Energy in Plastic Surgery" is meant solely as an introduction to laser use in plastic surgery. It is not meant to be comprehensive, but as a stimulus to the person who wants to use lasers in plastic surgery or skin medicine field. The laser is a useful tool in the armamentarium of a cosmetic and reconstructive plastic surgeon. Knowledge of its many uses will complement the skills of the surgeon and facilitate the treatment of the aging process as well as photodamaged skin, birthmarks, and a variety of dermatological disorders such as unwanted hair. Avoiding complications and building a practice are just two of the many ancillary benefits of laser education.

There are many good resources to expand on the information herein, a few listed in the selected references at the end of the chapter. Those wanting to use lasers in medicine would be well advised to join the American Society of Laser Medicine and Surgery (ASLMS). Further information can be obtained online at *www.aslms.org*.

7.3 Laser Physics and Physiology

LASER is an acronym for **L**ight **A**mplification by **S**timulated **E**mission of **R**adiation. It was first invented in 1960 and has evolved to be a useful tool in medicine as well as in many industrial applications. Conceptually, laser can be defined as a heat generator, as nearly all medical applications of the laser involve the conversion of laser energy into heat. An exception to this generalization would be photobiomodulation (PBM), which will be discussed toward the end of the chapter. The heat generated by the laser energy impacting tissue becomes a stimulus for tissue change. The change due to heating of the tissue may range from simple swelling to charring, and eventually evaporation as the tissue disappears into a plume. Healing of the stimulated tissues then occurs as the body reacts to the stimulus affecting the results which hopefully was the objective of the laser treatment. This is where the knowledge and skill of a laser therapist are essential.[1,2]

Lasers come in many forms and have many purposes. Not every laser is suitable for every purpose. Only a few of the lasers are commonly used in plastic surgery, which will be reviewed here. Lasers are usually defined by the wave-length of the emitted energy. The emitted energy may be visible light or invisible to the human eye (▶ Fig. 7.1). The wavelength, intensity, and duration of the laser beam will determine the tissue response. Tissue interaction with the energy is also determined by a variety a tissue factors, including the absorption and spread of the energy. Tissue absorbers, sometimes referred to as chromophores, include water, hemoglobin, melanin, collagen, and other tissue components. Each will react differently depending on the factors described above. These tissue absorbers then become the target, intended, or otherwise. Understanding the characteristics of the target tissue as well as the laser wavelength and parameters is key to achieving a satisfactory result.

7.3.1 Selective Photothermolysis (SPTL)

The concept of selective photothermolysis (SPTL) was introduced by Anderson and Parrish in 1983 to explain the laser tissue interaction.[3] Briefly, the concept states that the combination of laser wavelength, pulse duration, laser energy, and tissue absorption will largely determine the tissue response. A brief explanation of these various factors will help the reader understand many of the laser technologies and techniques. There are many tissue absorbers (chromophores) in the skin, which are competing for absorption of the laser energy. Specific absorption curves can be plotted showing the percent absorption versus the wavelength (▶ Fig. 7.2, ▶ Fig. 7.3). By overlaying these various curves for the major tissue absorbers, one can select the best available laser wavelength and laser, thus optimizing the absorption in the target tissue and minimizing the absorption in the competing chromophores. A tissue target consisting mostly of melanin would require a laser wavelength

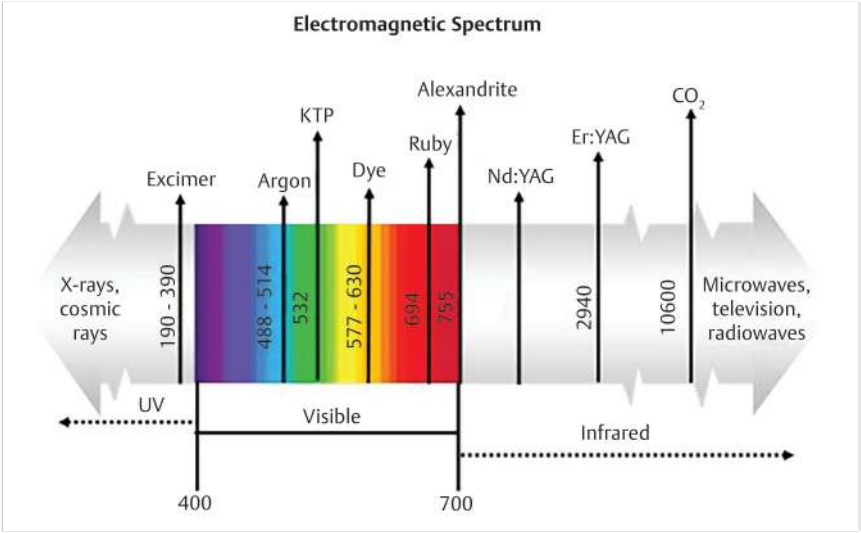


Fig. 7.1 The electromagnetic spectrum.

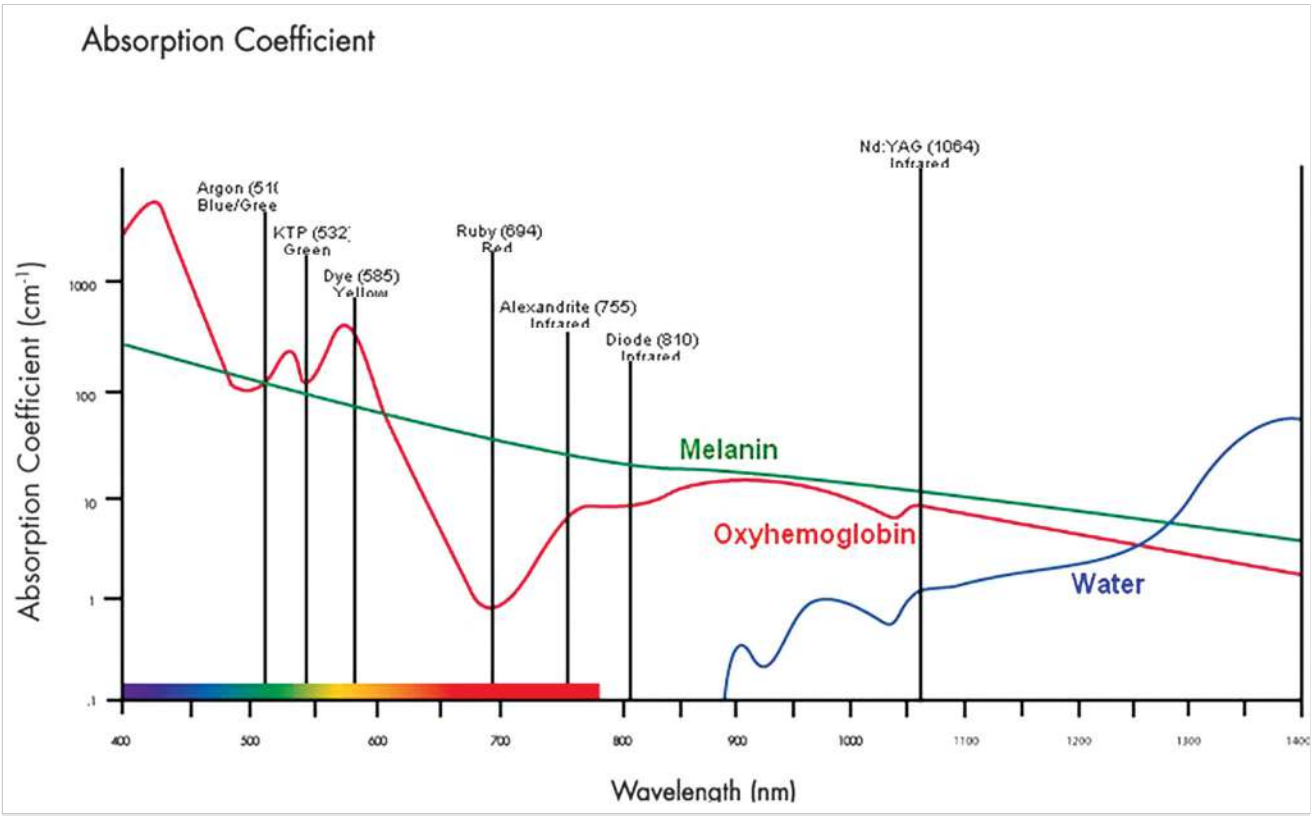


Fig. 7.2 Varying absorption of different chromophores (melanin, oxyhemoglobin, and water) in relation to the wavelength of the emitted light.

near the ultraviolet end of the spectrum while avoiding to a large extent water absorption, which is heavy in the infrared portion of the spectrum. The ruby laser (594 nm) would be a good choice for melanin targets, whereas the carbon dioxide (CO_2) laser (10,600 nm) in the far infrared portion of the spectrum in general works well on cells containing much water.

A second consideration of SPTL is the pulse duration or length of time the laser energy is impacting the target. Ideally, this time period, known as the pulse duration or pulse length, would be long enough to effectively heat the target and short enough to avoid the spread of heat to the surrounding nontarget tissues. Thus, the thermal relaxation time (TRT) of the specific target should determine the pulse duration of the laser beam. The TRT is proportional to the size of the target. Melanophores, the extremely small, several microns wide packets of melanin, would require a nanosecond pulse, whereas the much larger hair follicle bulbs would require a pulse duration of perhaps hundreds of milliseconds.

The goal of almost every laser treatment is to selectively heat the target to a level sufficient to achieve the given purpose without heating the surrounding nontarget tissue that causes complications. This is where the experience and knowledge of the laser therapist are critical to a good outcome. Other factors can be equally important, such as the patient tissue factors including pigment and patient cooperation. Keeping in mind that every patient is different and conditions vary widely within a given lesion (vessel size, etc.), it is a wonder that a favorable outcome can be achieved.

Several different lasers can be used for a given purpose and different pathologies may respond to different lasers. It is our intent now to examine several of the major laser systems, including their characteristics and uses. This will then be followed by a discussion on the various pathologic problems that can be treated, including which lasers may have acceptable outcome.

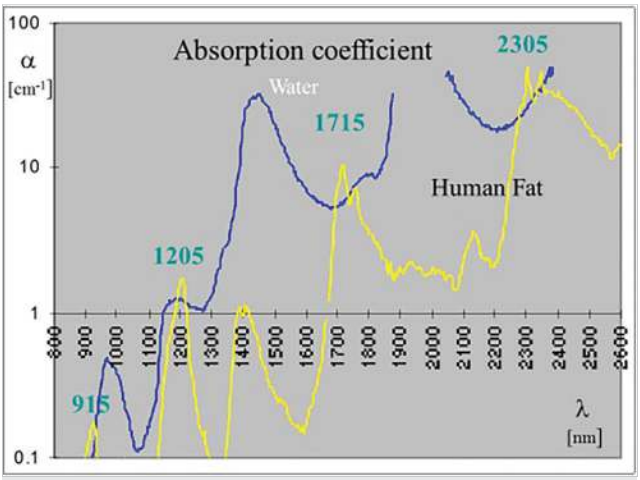


Fig. 7.3 Differing absorption of light energy by water and fat in relation to the wavelength of the emitted light.

7.3.2 Carbon Dioxide (CO_2) Laser

Although the first laser was the ruby laser (1960), the carbon dioxide laser was developed early in laser history (1964) and has found extensive use in plastic surgery. In fact, in many practices, the carbon dioxide laser is the main wavelength used as it is excellent for rejuvenating tissue as well as ablating lesions and even cutting tissue with the modest hemostatic quality. At 10,600 nm, this laser falls in the far infrared portion of the electromagnetic (EM) spectrum. It is heavily absorbed by water and minimally absorbed by melanin. Nevertheless, even pigmented lesions respond as the laser evaporates the water-laden cells containing the pigment.[4]

Probably, the first cosmetic laser was the Coherent UltraPulse® carbon dioxide laser. This carbon dioxide laser had a short-enough pulse to effectively ablate tissue without leaving major heat damage. Popularity of laser resurfacing grew rapidly in spite of the long recovery following treatment and modestly high incidence of hypopigmentation. Heating caused shortening of the collagen bundles and thus tissue shrinkage was achieved. In addition, heat damage to the tissue cells released cytokines and other tissue constituents which ultimately recruited fibroblasts and collagen production ensued. The reduction of tissue weathering, pigmentation issues, and improvement of fine lines and scars with the carbon dioxide laser caused a rapid advancement of laser use in cosmetic surgery, largely replacing many of the chemical peels and dermabrasion procedures previously done. Many competitors to the Coherent laser appeared and technology rapidly improved with the advent of computerized scanning hand pieces and other developments which speeded the resurfacing, making it more uniform and predictable.

Laser resurfacing of the skin is the most common cosmetic use of the laser in my practice. While frequently done under topical anesthesia as an outpatient procedure, it also can be combined with surgical procedures such as facelifts to complement the restoration of the youthful appearance of the skin. Although the procedure was introduced with a single spot hand piece, a great advance was seen with the introduction of the computerized pattern generator (CPG) which speeded the procedure and made it more uniform and precise.

Laser resurfacing consists of ablation of the cornified layer of the skin and heating of the superficial dermis. As explained above, heating tightens the collagen bundles and initiates production of new collagen in the superficial dermis. In addition to alleviating the weathered appearance of the skin and superficial disorders such as pigmentation issues, smoothing and thickening of the skin are produced. While immediate improvement is seen, final results may not be appreciated for six months or more due to the time required for collagen production. Patients undergoing laser resurfacing may not fully appreciate the improvement, and although healing of the skin limits the depth and intensity of a single treatment, multiple treatments can result in increased improvement.

7.3.3 Fractional Laser Resurfacing

The most recent technological advance for laser rejuvenation skin was introduced in 2004 as fractional photothermolysis.[5] Because of the disadvantages of laser resurfacing such as prolonged healing, pain, need for greater anesthesia, and occasional pigmentation issues, fractional technology has been a significant development in laser resurfacing. Although the results are not as dramatic, reduction in these disadvantages is considerable. Thus, many patients would prefer multiple treatments with fractional laser as contrasted to a single treatment with the conventional laser resurfacing.[6,7]

Fractional laser resurfacing consists of creating numerous microscopic laser beam penetrations of the skin known as microthermal zones (MTZ). Although the number and density of these MTZ can be adjusted, the concept involves spacing them since there is a microscopic space between each thermal reaction. This is what accounts for faster healing and other advantages. Generally speaking, each MTZ can be deeper than the heating of conventional laser resurfacing. Most laser resurfacing of the skin is currently done with the fractional technique.[8] Although other wavelengths have been used with the fractional technology, carbon dioxide remains the most common and effective in most instances. The concept has been applied to other energy sources other than laser. As in many advances in technology, the concept sometimes becomes bastardized and thus claims of fractional technology may involve something completely different.

7.3.4 Other Applications of the Carbon Dioxide Laser

The carbon dioxide laser is extremely versatile and can be used for a variety of cosmetic applications. Other than resurfacing and rejuvenation of the skin surface, a focused handpiece can be used for ablating lesions, such as seborrheic keratoses, syringomas and rhinophyma. Because of the wide range of energies available with this laser and the modest ability of achieving hemostasis by defocusing the beam, the carbon dioxide laser is extremely useful in plastic surgery. A cutting mode with a focused beam can be used for surgical applications such as blepharoplasty where precision and hemostasis are critical.

7.3.5 Nd:YAG (Neodymium:Yttrium-Aluminum Garnet) and KTP Lasers

Another useful laser in the plastic surgery practice is the Neodymium:YAG (Nd:YAG) and (Potassium-Titanyl Phosphate) KTP laser combination. Although somewhat different in application, these two are together because they originate from the same laser source. Nd:YAG lasers, like the ruby and alexandrite lasers, are crystal lasers. While ruby and alexandrite lasers produce a light in the visible portion of the spectrum, the Nd:YAG is in the near infrared 1,064 nm and thus is invisible to the human eye. This wavelength allows very deep penetration because of the poor absorption by the chromophores in the superficial skin. Thus, targets such as deep, large vessels may be best treated by the Nd:YAG laser. This laser technology is also very stable and reliable. Its versatility has increased by placing a KTP crystal in the beam path of the laser which halves the original 1064 nm wavelength to 532 nm, thus placing it in the green portion of the visible light spectrum. This significantly alters the absorption characteristics of the laser energy. The laser is useful for pigmented and vascular disorders as well as hair removal (▶ Fig. 7.3).

7.3.6 Ruby Laser (594 nm) and Alexandrite Laser (755 nm)

Both of these crystal-based lasers serve common purposes such as pigmented lesions and hair removal and are thus lumped together for brief discussion. The ruby laser light is heavily absorbed in pigmented lesions, and thus, this laser can be used for most superficial pigmented pathologies. Both of these lasers in the Q-switched version can be used for removing tattoos and other embedded foreign bodies in the superficial dermis.

A number of lasers can be Q-switched, thus changing the effect on tissue. The Q-switched lasers have electronic shutters that allow extremely short (nanosecond) bursts of energy which generally are very high peak power. As stated above, according to the theory of SPTL, the heat is largely confined to the target, thus preventing damage to the surrounding tissue. The high peak power causes rapid buildup of heat within the target, thus creating "microexplosions" which disrupt the tissue target such as tattoo pigment and melanin. It may also require a different delivery system such as a mirrored waveguide rather than a fiber-optic delivery system as with the longer pulsed laser energies.

7.3.7 Picosecond (One Trillionth Second or 10–12 Seconds) Lasers

Technically not a specific laser, but in fact a technology, one of the latest innovations in laser medicine is the picosecond laser. It has long been realized that the extremely short pulse and very high energy of the picosecond laser is useful in tissue disruption. As discussed above, this extremely short pulse creates very limited microexplosions in the tissue, thus confining the heat to an extremely small area while creating maximal disruption within a few cell diameters distance. Like the ultraviolet excimer laser, this has been used for ophthalmology applications for this reason. In plastic surgery, the picosecond laser utilizing a variety of wavelengths (1064 nm, 758 nm, etc.) has been used in tattoo removal as well as tissue rejuvenation. The clinical usefulness of these lasers however is limited by their extreme cost at present. As an aside, ophthalmological and laboratory work is being conducted with the femtosecond (one quadrillionth of a second, 10–15 seconds) laser.

7.3.8 Pulsed Dye Laser (PDL)

This is really a class of lasers, some of which have found extensive use within the plastic surgery genre, particularly for pediatric vascular lesions. As with every laser, there is an energy source, which in this case may be another laser, a laser medium which is most commonly a stream of dye circulated across the output of the energy source by a pump, as well as the usual mirrors and control mechanisms. These are sometimes referred to as pumped dye lasers or tunable dye lasers as the wavelength of the output energy can be changed by changing the dye source. The seminal work with the pulsed dye laser originated due to the fact that the argon laser was much less than ideal for treating pediatric port wine stains. The early argon laser frequently left scars and pigmentation disorders although shrinking the vessels of the lesion. SPTL, as aforementioned, has its roots in the development of the pulsed dye laser, which largely was motivated by the need for improved treatment of these pediatric port wine stains. The PDL was originally designed at 577 nm, 450 µs pulse duration. Although it was perhaps ideal for a superficial population of microvessels, it was largely insufficient for the larger deeper vessels commonly associated with the more mature port wine stains. Thus, the laser has been configured to deliver energy at 595 nm with the pulse duration in the 1 to 10 ms range. This allowed the PDL to adequately treat a much larger population of vessels.

7.3.9 Diode Lasers

In an effort to miniaturize lasers and make them more reliable, a variety of semiconductor lasers have been developed. We are familiar with the typical green laser pointer. A variety of wavelengths have been developed. Medical and dental uses of diode lasers have utilized 800–980 nm wavelengths because of the high hemoglobin absorption. These have also found use in the hair reduction lasers (808 nm).

7.3.10 Other Lasers

There are many other lasers that have found occasional use in medicine, and specifically in plastic surgery. These would include the argon laser, the copper vapor laser, the gold vapor laser, and a variety of other lasers, including a variety of the dye lasers. It is not feasible to cover all of these lasers in this chapter. Other radiant energy sources used in plastic surgery will be reviewed below.

7.4 Non-Laser Radiant Energy Sources

7.4.1 Photobiomodulation (PBM)

Also known as low-level laser therapy (LLLT), soft laser, or biostimulation, this interesting field of research is based on the knowledge that cellular metabolism can be altered by radiant energy. The most common example of this is the common suntan caused by the induction of melanin production by sunlight. This has been used to treat sports injuries for pain and swelling, healing of chronic wounds, and many other uses, including more recently hair growth. Although there is substantial evidence that PBM can be used to manipulate cell repair and metabolism, the science has been corrupted by commercial interests as well as less than scientific methodology in the research. It is likely that we are on the threshold of this future quantum leap in laser medicine.

7.4.2 Photodynamic Therapy (PDT)

This combination of light energy and drug therapy induces cellular necrosis, specifically in malignant tissue. Even after having been researched for many years, the present uses are confined to skin and a few internal cancers, including esophageal and certain lung cancers. FDA approved topical therapy of photo sensitizer can be combined with a variety of light sources including lasers to treat superficial skin cancers and premalignant disease.

7.4.3 Intense Pulsed Light (IPL)

Although technically not a laser, this broadband light source is similar in many respects to a laser and finds a prominent place in the cosmetic laser practice. First introduced as a treatment for telangiectasias in 1995, the IPL was preceded by a variety of light sources such as the infrared coagulator.[8] This xenon bulb light source produces light ranging in wavelength from 500 nm to 1200 nm. By introducing filters in the beam path, portions of the broadband can be removed, creating more selectivity in the tissue interaction, thereby increasing the usefulness of this technology. In addition to treating telangiectasias, this technology has found use in hair removal, treatment of pigmentation disorders, and other areas of skin pathology.[9,10] Limitations include the relative non-selectivity of the broadband light, the modest energy available in each portion of the light spectrum, and lack of precision.

7.4.4 Related Technologies and Radiant Energy

Many years ago, it became evident to the leaders of the American Society for Laser Medicine and Surgery (ASLMS) that there were other energy sources in addition to lasers which play a significant role in medicine. Because of the relationship of these energy sources to lasers, the mission of the society was expanded to include "related technologies" and radiant energy sources.

Practically, everything done in surgery as well as other fields of medicine is to stimulate a given response in the human body. Lasers, ultrasound, radiofrequency, etc. are all energy sources which stimulate the body in a variety of ways. These not only depend on the technology as well as the technical expertise of the therapist, but also on the patient response which is multifactorial in nature. Some of these factors can be predicted and thus manipulated. Others are beyond the control of the therapist and sometimes even beyond the control of the patient.

Almost all laser and energy treatments involve tissue interaction, which results in heat generation. Lasers that have the precision may not have the physical ability to reach and thus stimulate a response in the target tissue. For this reason, other energy sources have become increasingly important in plastic surgery. Notably, among these are ultrasound, microwave, and radiofrequency. These are all extensions of the electromagnetic (EM) energy spectrum.

Continuing the laser theme, a promising area of laser science is photobiomodulation (PBM). This for the most part is subcellular, nonlethal (nonthermal) molecular stimulation which then alters the metabolism of the cell. Research with this technology includes investigations for use in pain control, wound healing, scar therapy, and even hair growth stimulation. Combination of laser and drug therapies such as PDT for the treatment of cancer and other entities have proven to work, but have found limited application to date.

7.5 Common Disorders Treated by the Laser

7.5.1 Aging Skin

In the cosmetic practice, the most common use for lasers is in the treatment of disorders of aging. The laser is an ideal instrument for treating not only the lesions associated with aging, such as lentigines, seborrheic keratoses, and telangiectasias, but also other problems resulting from years of sun exposure as well as general deterioration. The laser really comes into its own in treating the morphologic changes of the aging process, including thickening of the cornified layer, deterioration of the collagen-elastin substrate, and general pigmentation disorders. The fine lines and wrinkles resulting from this process give away a person's age in spite of the best efforts to maintain physical fitness and general physique. Dermabrasion and chemical peels have been the preferred treatments in the past, and there is still a place for these treatments in the armamentarium. However, lasers have largely replaced these treatments in many practices because of the precision and general predictability. Arguments against the laser discuss the cost as well as the technical challenges associated with the technology (▶ Table 7.1; ▶ Table 7.2).

Table 7.1 Therapeutic applicability of commonly used lasers

Lasers/Applications			
Device	Wave length	Tissue	Pathology
CO_2 Superpulse	10,600	Cells, water	Aging, lesions, tumors
CO_2 Fractional	10,600	Cells, water	Aging, lesions, tumors
Erbium	2,940	Water	Pigment, aging
1550 Fractional	1,550	Cells, water	Scars, aging
KTP 532	532	Melanin, hemoglobin	Pigment, vascular
PDL 585/595	585/595	Hemoglobin	PWS, vascular
Ruby/Alexandrite	694/755	Melanin	Dyschromia, birthmarks
IPL Green	500–670, 870–1,200	Melanin, hemoglobin	Pigment, vascular,
IPL Yellow	525–1,200	Melanin, hemoglobin	Pigment, vascular,
IPL Red	650–1,200	Melanin	Hair

Table 7.2 Lasers used to treat skin-based conditions

Lesions/Laser		
Lesion type	Pathology	Laser
Vascular		
	Telangiectasias	Small, shallow-IPL, large, deep-KTP
	Capillary, PWS	Pediatric-PDL, mature-KTP/Nd:YAG
	Pyogenic granuloma	KTP/ CO_2
	Hemangioma	KTP/Nd:YAG
	AVM	KTP/Nd:YAG
	Leg veins	Injection/KTP/ND:YAG
	Rosacea	IPL/KTP
	Poikiloderma	IPL/KTP
	Angioma fibroma	KTP/ CO_2
Pigmented		
	Dyschromia/PIH	IPL/Ruby/KTP(Q-Sw)
	Melasma	IPL/Ruby/KTP(Q-Sw)/Fractional !550/Erb
	Lentigo	Fractional CO_2
	C.A.L.M.	Q Sw Ruby/KTP/IPL
	Seb keratosis	Fractional CO_2
	Congen nevi	IPL/Ruby/KTP
	Tattoos	Picosecond otherwise Q-Sw Ruby/Alex/KTP
Cysts, Tumors		
	Warts	Pulsed CO_2
	Syringoma/Seb hyperplasia	Pulsed CO_2
	Xanthelasma	Pulsed CO_2
	Seb keratosis	Fractional or pulsed CO_2
	Rhinophyma	Pulsed CO_2

The fractional CO_2 laser is the preferred laser for reducing pigmented and other benign lesions, correcting pigmentation disorders, dyschromia, etc. while simultaneously tightening skin and inducing the production of collagen in the dermis. A thorough laser treatment of the entire face can be accomplished in 30 to 45 minutes under topical anesthesia with resolution of the flakiness phase in 5 to 7 days. Most patients are functional during this period of time although restricting the public appearance is an option. Sun exposure precautions are advisable. Improved results can be obtained by combining the laser with skincare treatments as most cosmeceuticals are better absorbed following laser treatment due to the reduction in the cornified epidermal layer. In addition, a maintenance skincare program should be instituted in order to prolong the benefits of the laser treatment. More thorough results can be obtained by using more aggressive techniques, using the older style carbon dioxide laser resurfacing as with the Coherent UltraPulse® laser, or repeating the treatment within a few weeks.

Evaporation of specific lesions can be accomplished with a superpulsed carbon dioxide laser and a focused handpiece. It should be noted, however, that there is no histologic confirmation of the diagnosis or adequacy of treatment. Xanthelasma and syringomas as well as other benign lesions of the skin are ideally treated by the carbon dioxide laser.

While the laser has been used for facelift dissection, there are very few advantages and some potentially serious risks to this. However, it is a well-accepted procedure to use the laser for blepharoplasties. The benefit of precise dissection and hemostasis potentially speeds up the procedure and may reduce the postoperative swelling and bruising.

7.5.2 Vascular Lesions

The earliest use of lasers in plastic surgery was to treat vascular lesions, particularly of childhood, including hemangiomas and capillary malformations (e.g., port wine stains). A variety of vascular lesions of adults can also be treated with laser. Starting with the youngest, the best treatment for neonatal capillary malformations is the pulsed dye laser. Although the argon laser was used in the past, it is not advisable. Aggressive and threatening hemangiomas in infants can be treated with the KTP laser, or the Nd:YAG laser can be used with significant advantage for deeper penetration. Adult vascular lesions can be treated with the KTP or Nd:YAG laser and other lasers have been used. For generalized erythema such as rosacea, the IPL may be used although it fails to treat deeper and larger vessels very well.

7.5.3 Tumors and Hyperplastic Skin Lesions

Debulking of large tumors, such angiofibroma and even neurofibroma, as well as hyperplastic conditions, such as rhinophyma, can be accomplished using the carbon dioxide laser. Improved hemostasis in large vascular tumors can sometimes be achieved using the Nd:YAG laser with contact fiber, but the surgeon should be prepared to control bleeding of large, high-pressure vessels with conventional techniques. Rhinophyma can be effectively treated by debulking with the carbon dioxide laser which usually can be accomplished under local anesthesia. Healing by epithelialization may require three or four weeks, depending on the extent of the lesion.

7.5.4 Pigmentation Disorders

A variety of lasers can be used for many of the more common pigmentation disorders, dyschromia, lentigo, and even melasma. For the more superficial, thin lesions, the IPL may be the ideal treatment. Thicker lesions, however, require a more aggressive approach with the KTP laser or other pigmentation lasers such as ruby or alexandrite. Raised and thickened benign lesions will probably require ablation, and the fractional or superpulsed carbon dioxide laser would be an excellent choice for most of these. It should be noted that as with all conditions, they may require more than one treatment and certainly can return. Melasma, although responsive to the laser, has an extremely high recurrence rate. A full skincare medical program should be outlined for the patient before undertaking a laser treatment. This may require restriction of hormones as well as sun exposure and other precipitating influences.

7.5.5 Tattoos and Other Foreign Body Granulomas

With the recent increase in popularity of tattoos, and the potential dissatisfaction by those with tattoos, a boom in the laser removal would be expected. The history of tattoo removal has been poor because of the slow response, considerable cost, and generally poor result with incomplete removal, pigmentation changes, and scars. Few people having a tattoo or wanting one removed consider these factors. Although many attempts have been made to overcome these obstacles, progress has been slow. The recent added technology of picosecond lasers may change this scenario. Time will tell whether this technology will survive and thrive. The results look promising at present, but the cost of these lasers is extreme. In addition to the body art, embedded foreign bodies such as road dirt can be removed by the laser. Many lasers may be useful in this regard, but in most instances, one would start with the Q-switched laser such as a KTP, ruby, or alexandrite. Deeper and larger particles can be evaporated with the carbon dioxide laser handpiece.

7.6 Related Technologies

Other energy sources can heat tissue and thus affect many of the results achieved by stimulating the tissue with the laser. While having been present for a long time, many of these are still yet to be fully accepted in the medical community. Ultrasound and radiofrequency are among the energy sources developed or being developed for treating not only the aging processes within the body but also localized lipodystrophy. In most instances, these energy sources must pass through the skin, the organ most affected by the laser, into the deeper tissue to achieve the goals of treatment. Deeper, direct application of energy, as with radiofrequency (RF), through a probe inserted through the skin will have greater effect. There is also the added possibility of combining laser to treat the skin and other energy sources to treat the deeper tissue. The development of these energy sources is still in its early stages, and even though many results achieved to date may have minimal benefit, progress is being made and thus the laser therapist should be alert and yet conservative in adopting them.

7.7 Safety

The new laser therapist should be fully risk averse. In addition to the usual risks of surgery including infections, scars, etc., the laser introduces new and potentially dangerous risks. All laser wavelengths can be harmful to the eyes and require eye protection for both the patient as well as therapist and most people in the room. In addition, the risk of fire due to the laser igniting flammable objects within the operative field is greatly increased in the presence of oxygen, whether flowing through a tube or trapped under drapes. Moistened or wet towels surrounding the treated site will guard against fire ignitions. Similarly, alcohol-based preps should be avoided. When using a laser around the mouth, the teeth should be protected from potential deleterious effect on the enamel. Moist gauze occlusion works well in this regard. Furthermore, if the treatment room has a mirror, this should be covered while the laser is in use, thereby protecting from inadvertent reflection of the laser beam. Lastly, appropriate signage is required to alert those entering the treatment room that a laser is in use.

7.8 Conclusion

Lasers are powerful tools applicable to many clinical conditions encountered by plastic surgeons. Significant improvement in such conditions can be provided with the potential for minimal morbidity and short recovery times. Creating a laser practice is a substantial commitment, largely related to the expense of these devices, but also due to the need for maintaining a current knowledge base of new technology and applications. Nevertheless, with proper planning and execution, the use of lasers and related technology can be an extremely rewarding part of one's practice.

7.9 Review Questions

7.9.1 Choose the Best Answer

1. All the following are true about lasers EXCEPT the following:
   a) LASER is an acronym for Light Amplification by Stimulated Emission of Radiation.
   b) Lasers create their clinical effect dependent on the light spectrum (wavelength) emitted.
   c) Lasers do not create heat or thermal tissue injury.
   d) Lasers are defined by the wavelength of light emitted.
2. The tissue response from laser treatment is dependent on
   a) Laser wavelength.
   b) Pulse duration.
   c) Laser energy.
   d) Tissue absorption.
   e) All of the above.
3. Laser energy is dependent on energy absorption into
   a) Water.
   b) Hemoglobin.
   c) Melanin.
   d) Collagen.
   e) All of the above.
4. The carbon dioxide laser is
   a) Heavily absorbed by melanin.
   b) Primarily absorbed by water and causes cellular vaporization.
   c) Ineffective at skin resurfacing.
   d) Best used for vascular lesions.
5. The Neodynium:YAG laser
   a) Provides only superficial penetration of skin.
   b) Cannot be used for hair removal.
   c) Is useful for pigmented and vascular lesions.
   d) Can be used for skin ablation and tightening.

7.9.2 Answers

1. Lasers create their clinical effect dependent on the light spectrum (wavelength) emitted (b).
2. All of the above (e).
3. All of the above (e).
4. Primarily absorbed by water and causes cellular vaporization (b).
5. Can be used for skin ablation and tightening (d).

References

[1] Achauer BM, Vander Kam VM, Berns MW, eds. Lasers in Plastic Surgery and Dermatology. New York, NY: Thieme; 1992
[2] Biesman BS, ed. Lasers in Facial Aesthetic and Reconstructive Surgery. Baltimore, MD: Williams and Wilkins; 1999
[3] Anderson RR, Parrish JA. Selective photothermolysis: precise microsurgery by selective absorption of pulsed radiation. Science. 220(4596):524–527
[4] Laubach HJ, Tannous Z, Anderson RR, Manstein D. Skin responses to fractional photothermolysis. Lasers Surg Med. 38(2):142–149
[5] Cassuto DA, Scrimali L, Sirago P. Energy, pulse width and density: the role of each parameter in microfractional CO2 laser resurfacing. Lasers Surg Med. Supp S20:30
[6] Abasssi N, LaTowsky B, Arndt K, et al. Comparison of four ablative fractional devices in the treatment of Photoaging. Lasers Surg Med. Supp 22:25
[7] Ortiz AE, Tremaine AM, Zachary CB. Long-term efficacy of a fractional resurfacing device. Lasers Surg Med. 42(2):168–170
[8] Raulin C, Hellwig S, Schönermark MP. Treatment of a nonresponding port-wine stain with a new pulsed light source (PhotoDerm VL). Lasers Surg Med. 21(2):203–208
[9] Babilas P, Schreml S, Szeimies R-M, Landthaler M. Intense pulsed light (IPL): a review. Lasers Surg Med. 42(2):93–104
[10] Goldberg DJ. Current trends in intense pulsed light. J Clin Aesthet Dermatol. 5 (6):45–53

Part II

Skin and Soft Tissue

II

8 Burn and Thermal Injury

Steven J. Hermiz, Paul Diegidio, and C. Scott Hultman

Abstract

This chapter addresses the critical issues resulting from injuries due to fire in its various manifestations. Included are discussions of burn management, airway management, resuscitation, wound care, and surgery. The pathophysiology of burns is detailed, along with procedures for accurately assessing the extent and damage due to burns. The multidisciplinary approaches to burn treatment are also reviewed and include topical antimicrobial therapy, nutrition and local injury guidance, anesthesia, infection, airway treatment, excision and grafting, and reconstruction. The chapter includes two clinical cases that apply the treatment procedures covered.

Keywords: Parkland formula, enteral feeding, antimicrobial therapy, thermal injury, chemical burns, frostbite, electrical burns, radiation burns, crystalloid, cytokine

8.1 Goals and Objectives

- Familiarize with the first steps of burn management, inclusive of airway management, resuscitative support, wound care, and surgery.
- Understand effective resuscitation starting with accurate assessment of the extent and depth of the burn injury, followed by the application of the Parkland formula for fluid replacement, to ensure adequate end-organ perfusion, including skin.
- Review the need for early enteral feeding, within the first 24 hours, in burn patients as it decreases the catabolic response, improves nitrogen balance, maintains gut mucosal integrity, and decreases hospital stay.
- Learn the basics for successful surgical management of the burn patient: burn wound excision, topical antimicrobial therapies, and resurfacing with skin grafts are key to successful management.
- Become cognizant of the multiple treatment modalities that can be offered to a patient with hypertrophic burn scars, including nonsurgical care (silicone sheets, pressure garments, and splinting) as well as several different types of laser therapies. Contractures and unstable wounds can be reconstructed with tissue rearrangements, flaps, or skin substitutes.
- Familiarize with the different types of burns, including thermal injury, chemical burns, frostbite, electrical burns, and radiation burns.

8.2 Patient Presentation

8.2.1 History and Epidemiology

Since the discovery of fire, burn injuries have posed a threat to human wellbeing due to direct contact and scald.[1] The foundations of burn treatment can be traced back several thousands of years in time. However, the confluence of medical, surgical, and technological advancements since the mid-1900s has revolutionized burn care and have drastically improved patient outcomes. The major advances documented throughout history are listed in ▶ Table 8.1.[1,2,3,4,5,6,7,8,9,10,11,12,13,14,15,16,17,18,19,20]

Burns are the fourth most common type of trauma injury following traffic accidents, falls, and interpersonal violence.[21] In 2004, 11 million people globally experienced burns severe enough to require medical attention.[21] Each year, burn injuries and fires claim over 300,000 lives worldwide, despite the vast majority of burn injuries being nonfatal.[22,23] In low and middle-income countries, where over 90% of all burn deaths occur, infrastructure to prevent and treat acute burns is lacking, and the healthcare burden is particularly high.[22]

However, even in the United States, burns are a sizable source of morbidity and mortality. According to the American Burn Association, approximately 450,000 patients receive treatment for burns in a hospital or emergency room setting each year in the United States. Of those acute burn injuries, roughly 3,400 result in mortality each year. According to the Centers for Disease Control and Prevention (CDC), males account for 64% of the $7.5 billion total medical cost and productivity loss burden from burn-related injuries per year in the United States.[24] Additional factors and information related to burn epidemiology are described in ▶ Table 8.2.[21,22,23,24,25,26,27]

Burn injuries are a leading cause of death in children living in developing countries.[28] Burns are physically, emotionally, and psychologically devastating to the patient, family members, and the provider. They are a common household injury as children explore new surroundings and objects.[29,30,31,32,33,34] Delgado et al determined that 77.5% of burn cases occurred in a patient's home (67.8% in the kitchen) and 74% were due to scalding. In order to prevent this debilitating injury, the clinician must be cognizant of the risk factors and protective factors aimed at burn prevention (▶ Table 8.3).[27,28,29,30,31,32,33,34,35,36]

8.3 Pathophysiologic Basis of Clinical Presentation

Burn injury results in coagulation necrosis of the skin and possibly the underlying subcutaneous tissue. The tissue surrounding this central zone of coagulation necrosis sustains a moderate degree of vascular injury, which decreases tissue perfusion and is known as the zone of stasis. Local mediators produced from the burn wound, such as arachidonic acid, are able to propel this zone into a partial thickness or full-thickness injury. They also cause arterial/venous dilation and platelet aggregation, thereby decreasing flow and perpetuating stasis. Thromboxane A2 is found in high concentrations in the burn wound and increases neutrophil migration in addition to platelet aggregation.[37]

There are multiple cytokines involved in burn injury (▶ Table 8.4) and their actions are responsible for both the local and systemic effects seen in burn patients.[34] Burn wound colonization and bacterial translocation (▶ Table 8.5) are the nidus for endotoxin production and its effects.[37]

Table 8.1 Major advances in burn care

Author/Event	Year	Contribution
Ebers Papyrus	1500 BC	Earliest description: burns treated with oils, plant extracts, honey and animal tissues
Hippocrates	500 BC	Pig fat and resin to heal burns
Celsius	1st Century AD	Lotion of wine and myrtle to treat burns
Galen	2nd Century AD	Greek surgeon; vinegar and open-air exposure for wounds
Amboise Pare	16th Century AD	Onions to treat burns; early burn wound excision
Guihelmus Fabricius Hildanus	1607	1st systematic classification of burns/treatment of contractures (*De Combustionibus*)
Edward Kentish	1790	Pressure dressings to alleviate pain and blisters from burns
Marjolin	1790s	Squamous cell carcinoma in nonhealing burn wounds
Guillaume Dupuytren	Early 1800s	Occlusive dressing treatment, allowed development of burn depth classification
Topical antimicrobials	18th century	Sodium hypochlorite for infection control
Edinburgh Hospital	Mid-19th century	1st hospital to treat large burns surgically
Burn Education	Late 19th century	Textbook and reference books detailing medical and surgical management of burns
Nutrition	Early 20th century	High-calorie intake recommended
Wars/Industrial accidents	20th century	Disaster burn management development
World War I	1914	Surgical skin transplantation, scar reduction, and pain management were most effective burn management
William Monafo	1941	0.5% silver nitrate—Agent of choice
Boston's Cocoanut Grove nightclub fire	1942	1st comprehensive description of inhalational injury Improvements in topical antimicrobials Shock resuscitation Antibiotic use Understanding the metabolic response after burn injury Beginning of burn care facilities, public safety legislation
Truman G. Blocker Jr.	1947	Deadly ammonium nitrate fertilizer explosion; demonstrated value of multidisciplinary team approach for burn treatment
O. Cope, F.D. Moore	1940s	Quantified fluid amount needed for resuscitation
I.E. Evans	1952	Fluid requirements for resuscitation based on surface area burned and body mass (Parkland formula)
Douglas Jackson	1960	Excision and grafting up to 30% body surface area on day of injury
J.C. Tanner Jr.	1964	Meshed skin graft
Z. Janzekovic	1970	Tangential excision and grafting (large burns)
Engrav, Herndon	1980s	Early excision/tangential excision—decrease mortality, reduce hospitalization, reduce hypertrophic scarring
J. Wesley Alexander	1981	"Sandwich technique"—covering expanded autograft with cadaver skin
John Burke, Ioannis Yannas	1981	Artificial skin (Integra)
J.F. Hansbrough, S.T. Boyce	1989	Composite skin graft
D.N. Herndon	1989	Continuous enteral feeding recommended in burn care Metabolic support, including anabolic steroids (oxandrolone)

The extent of damage done by a burn is not limited to the body surface area involved.[38] The insult of a burn disrupts hemodynamic, respiratory, and metabolic systems.[37] Burn injury releases inflammatory mediators that are responsible for the downstream cascade of events that ultimately results in burn shock, specifically hypovolemic and distributive shock (▶ Fig. 8.1).[38,39,40] Distributive shock is the result of total body fluid expansion resulting from third spacing to include the intravascular, intracellular, and interstitial spaces. Hypovolemic shock ensues from massive interstitial fluid sequestration, and fluid is lost from the wound, resulting in decreased circulating plasma volume, and consequently, preload and cardiac output are decreased.[38,39,40] Demling et al concluded that edema continues and reaches a maximum level of 24 hours after injury and begins to resolve 1 to 2 days after the injury.[38,39]

The degree of metabolic derangement is related to the extent of the burn injury. The first phase is referred to as the "Ebb phase," which is characterized by a decrease in cardiac output and metabolic rate. After adequate fluid resuscitation, the cardiac output increases and there is an increase in resting energy expenditure. Cytokines activate other inflammatory mediators, which cause alterations in the hypothalamic control of temperature and metabolism resulting in fever and hypermetabolism.[41] The thermoregulatory set point increases 5 to 15 days postburn and remains elevated for up to 2 months.[41] The metabolic rate in burn patients is estimated to be twice normal.[41]

Table 8.2 Burn epidemiology

Global health issue	11 Million people world-wide and 450,000 in the United States
Large healthcare burden	Low- and middle-income countries with highest mortality Mortality rate 3,400/y $7.5 billion total medical cost and productivity loss burden/y
Age discrepancy	Pediatric population more at risk, especially for scald burns (non-Caucasian children)
Risk factors and higher morbidity and mortality rates	Male sex Impoverished socioeconomic status Children Elderly Disabled Military personnel
Incidence	50% reduction in fire and burn related deaths and hospitalizations for acute burn injury
Types of Burns	Scald (most common) Fire and flame (most common to require hospitalization)
Geographic discrepancy	Increased incidence in Africa

Table 8.3 Pediatric burns: risk factors and protective factors aimed at burn prevention

Risk factors	Protective factors
Age < 6 y old	Mother literacy
Girls	Education on risks for burns
Impoverished	Living rooms separate from kitchen
Disabled	Smoke detectors
Small kerosene stoves	Emergency response systems
Candles	Good quality healthcare
Volatile substances	Intervention programs (developing countries)
Low rate of literacy	Residential sprinklers
Overcrowded living areas	Child resistant/fire-safe lighters
No supervision	Laws regulating temperature of hot-water taps
History of burns (siblings)	
No regulations on smoke detectors/building codes	
No access to water	

Table 8.4 Cytokines involved in burn injury and their actions

Cytokines	Action
TNF-Alpha	Neutrophil sequestration (microvascular injury/burn size extension into zone of stasis)
IL-1, IL-2, IL-4, IL-6, IL-8, IL-12, INF-gamma	Active other classes of inflammatory mediators (potentiating their actions) Alterations in hypothalamic control of temperature and metabolism Potentiate organ failure progression

Abbreviations: TNF, tumor necrosis factor; IL, interleukin; INF, interferon.

Table 8.5 Burn wound inflammatory response

Bacterial colonization and dermal necrosis	Associated actions
Endotoxins (translocation)	Activates neutrophils and macrophages: inflammation/ tissue damage
Inflammatory response	Activates Proapoptotic signaling pathway
Proinflammatory mediators and neutrophils	SIRS response

Abbreviation: SIRS: systemic inflammatory response syndrome.

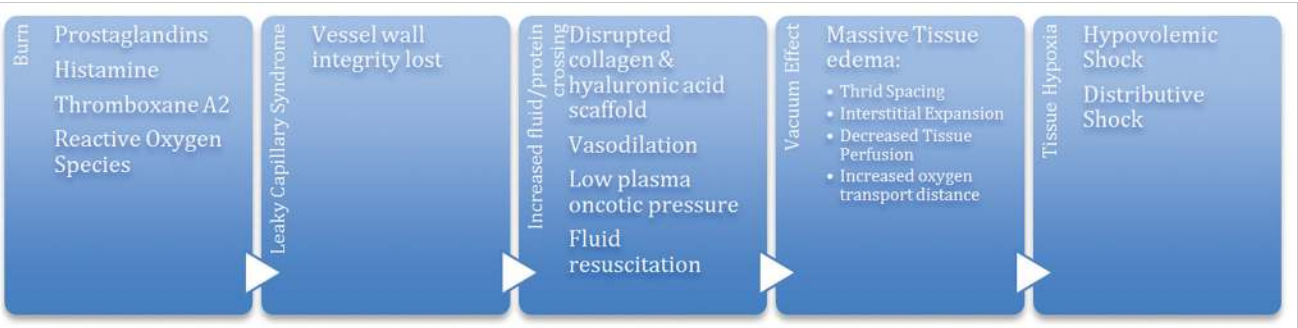


Fig. 8.1 Burn pathophysiology flow chart.

Cortisol, glucagon, and catecholamines are elevated in the burn patient. Cortisol is responsible for a catabolic state and creates a negative nitrogen and calcium balance. It stimulates gluconeogenesis and proteolysis. Catecholamines stimulate glycogenolysis, hepatic gluconeogenesis, and lipolysis and also create peripheral insulin resistance. Therefore, as a result of elevated glucagon levels, both glucose and insulin levels are elevated. Muscle protein catabolism leads to high concentrations of amino acids and decreased protein anabolism as a result of decreased levels of growth hormone and insulin-like growth factor.[41]

In the postburn patient, increased levels of catecholamines and catabolic hormones lead to a global hypermetabolism syndrome. This is evident by tachycardia, fever, hepatic protein synthesis derangement, and muscle protein catabolism. A heightened response leads to immunodeficiency, impaired

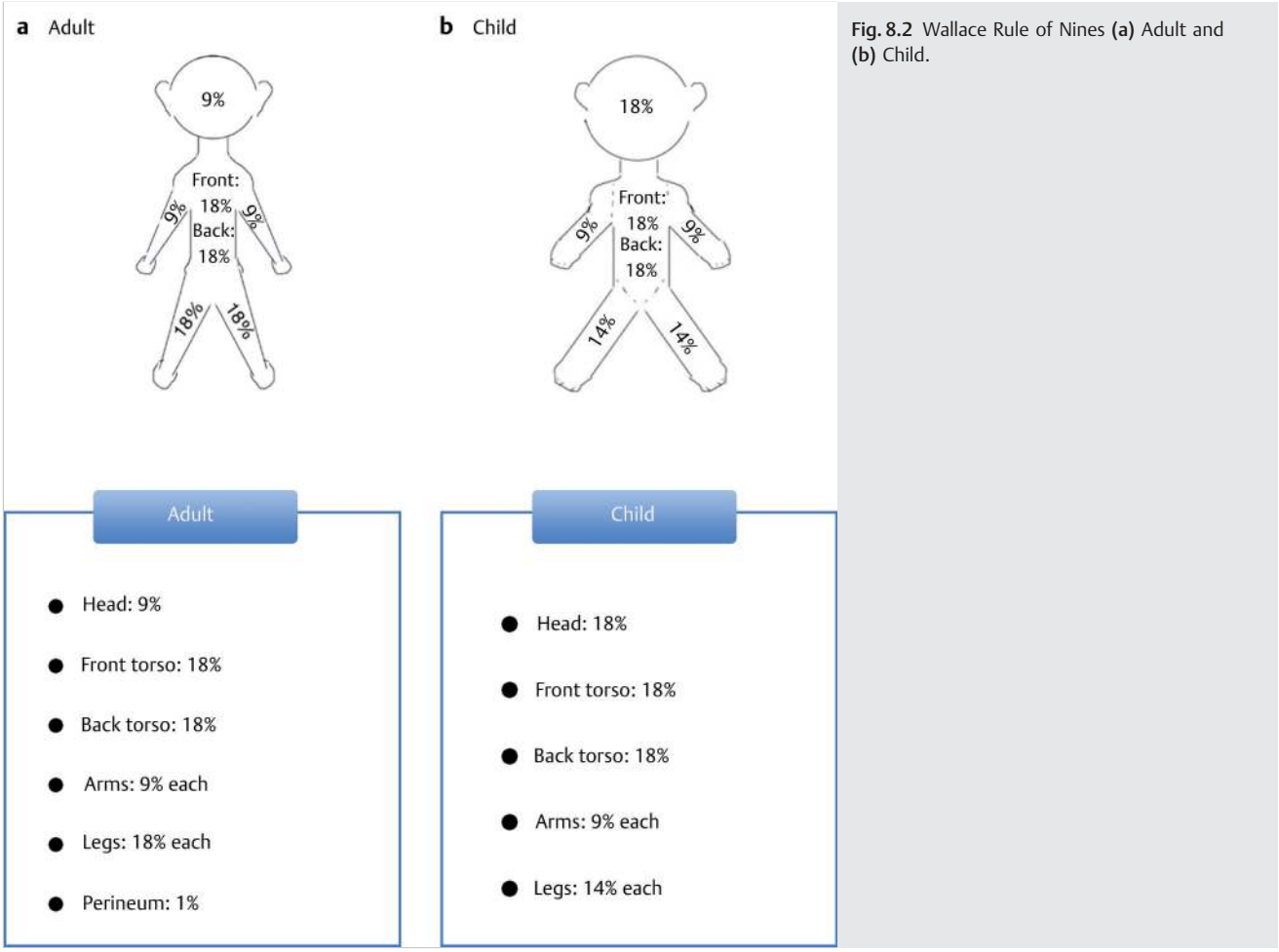


Fig. 8.2 Wallace Rule of Nines **(a)** Adult and **(b)** Child.

wound healing, loss of lean body mass, cardiac ischemia, and sepsis.[37]

Complications from this disruption may lead to acute lung injury, systemic inflammatory response syndrome (SIRS)/sepsis, immunosuppression, acute respiratory distress syndrome (ARDS), and multisystem organ failure (MSOF), potentially culminating in death.[37]

8.4 Preparation for Surgery

8.4.1 Initial Evaluation and Treatment

The first step in effective resuscitation is accurate assessment of the extent and depth of the burn injury. The second step is to determine if smoke inhalation injury is present. Smoke inhalation is suspected with facial burns, carbonaceous sputum, or a history of being in a closed space.[41] The goal of fluid resuscitation is to support the patient through the initial 24 to 48 hours postburn, which results in hypovolemia from third spacing and fluid shifts. This led Baxter and Shires to develop and implement the Parkland formula, which estimates the amount of replacement fluids in the first 24 hours in a burn patient. Total replacements needs are calculated by the formula: total fluids = 4 mL × weight (kilograms) × % total body surface area (TBSA) of second and third-degree burns. The first half is given within 8 hours from the injury, and the second half is given over the remaining 16 hours. The body surface area is estimated using the Wallace rule of nines (▶ Fig. 8.2).[37,38,39,40,41]

The patient's response to resuscitation is dependent upon age, depth of burn, pre-existing comorbidities, associated injuries, and concomitant inhalation injury.[38] Smoke inhalation injuries require up to one third more fluid during acute resuscitation compared to burn patients without inhalational injury.[41] The current first-line crystalloid solution is lactated ringers.[37,38] During the inflammatory state, 25% of the infused volume remains intravascular; therefore, larger amounts of crystalloid are needed, contributing to the edema seen in burn patients.[38,40] The Parkland formula grossly underestimates requirements with inhalational injury, alcohol intoxication, electrical injury, and postescharotomy. Hypertonic saline has been used in early resuscitation with benefits seen in decreased tissue edema and abdominal compartment pressures, but is currently not routinely used.[37,38,40]

Colloid fluid administration (albumin and fresh frozen plasma) can be given 12 to 72 hours postinjury after the capillary leak phase has ceased in patients with low urine output and hypotension, despite crystalloid administration. The standard belief that colloid use increases mortality in burn injury patients and may be due to the notion that colloids leak into lung parenchyma. Blood transfusions increase mortality in burn

patients and a restrictive strategy with hemoglobin goals of 7 to 9 g/dL is recommended.[37]

Intravenous fluid administration is critical to reverse the pathophysiology of burn shock by restoring plasma fluid loss, increasing body fluid reservoir, and restoring preload.[38,40] Care should be taken not to over-resuscitate the patient, which can lead to abdominal compartment syndrome, renal failure, pulmonary edema, extremity compartment syndrome, and orbital compartment syndrome.[38,40] The adequacy of resuscitation is monitored constantly and urine output of 0.5 to 1.0 mL/kg/h is an adequate indicator of vital organ perfusion.[41] Some studies suggest that the use of IV ascorbic acid decreases edema, intravascular fluid requirement, and respiratory dysfunction severity.[37,41]

Upon initial evaluation, the clinician should be aware of both the admission and transfer criteria for burn-injured patients. Current recommendations for admission and transfer are listed in Box 8.1.[42]

Box 8.1 Admission/Transfer criteria after burn injury (American Burn Association Guidelines)

- Second-/third-degree burns: > 10% TBSA (< 10 years old/ > 50 years old).
- Second-degree burns: > 20% TBSA.
- Hands/Face/Feet/Genitalia/Perineum burns.
- Third-degree burns: > 5%.
- Electrical/Chemical burns.
- Inhalational burn.
- Polytrauma.
- Significant comorbidities.
- Child abuse/neglect.
- Social/Emotional/Long-term rehabilitation.

8.4.2 Topical Antimicrobial Therapy

Topical agents are used on burn wound to hinder bacterial proliferation and fungal colonization. The three most commonly used topical antimicrobials are silver sulfadiazine (Silvadene), mafenide acetate (Sulfamylon), and silver nitrate, all of which have varying coverage of bacterial pathogens. Silver sulfadiazine is mainly used for prevention of burn wound bacterial infection, rather than treatment, because of poor eschar penetration. Also, it should not be used on the face. Mafenide acetate is used for both treatment and prevention of bacterial infection of burn wounds because of excellent eschar penetration. However, mafenide's disadvantages include painful application in partial thickness burns and inhibition of carbonic anhydrase, leading to metabolic acidosis. Silver nitrate is another topical agent with broad-spectrum antibacterial activity; however, it has poor eschar penetration and is associated with electrolyte abnormalities. Mupirocin and bacitracin are commonly used for superficial facial burns and care should be taken to only apply to small areas given their nephrotoxic quality (▶ Table 8.6).[41]

8.4.3 Nutrition and Local Injury

Severely burned patients (> 40% TBSA) have a metabolic rate that approaches 200% of the basal rate, resulting in greater energy and protein requirements.[41] Providing nutrition is crucial for wound healing, cellular function, and resistance to infection.[41] Early enteral feeding, within the first 24 hours, in burn patients decreases the catabolic response, improves nitrogen balance, maintains gut mucosal integrity, and decreases length of hospital stay. The recommended formula consists of 20% of calories from protein, 30% as fat, and 50% as carbohydrates. The general formula is 25 kcal/kg + 40 kcal/5% burn.[41] High-carbohydrate diet improves the net balance of skeletal muscle protein, but aggressive monitoring and treatment of hyperglycemia is

Table 8.6 Topical antimicrobial agents

Drug	Mechanism of action	Side effects
Silver sulfadiazine (Silvadene)	Bactericidal (cell membrane/wall) Broad spectrum anti-microbial coverage Exact MOA unknown No eschar penetration Use: superficial and intermediate thickness burns	• Sulfa allergy • Neutropenia • Thrombocytopenia • Not for facial burns or ear burns • Contraindicated on areas of new skin grafting (destructive to grafts)
Mafenide Acetate (Sulfamylon)	Bacteriostatic Inhibits folic acid synthesis Pseudomonal coverage Good eschar penetration Good for cartilage (ears) Prevention and Treatment Use: Intermediate thickness and deep burns (full thickness)	• Painful • Metabolic Acidosis (difficult ventilator management) • Sulfa allergy • Skin hypersensitivity reactions
Silver Nitrate	Antiseptic Denatures proteins (cell wall/membrane) No eschar penetration Advantage: doesn't need to be removed	• Hyponatremia • Hypochloremia • Hypocalcemia • Hypokalemia • Methemoglobinemia • Black tissue staining
Neomycin	Use: superficial partial thickness burns on face	• Nephrotoxic
Polymyxin B	Use: superficial facial burns	• Nephrotoxic
Bacitracin	Use: superficial burns, raw wounds Helps keep wounds moist	• Anaphylaxis
Mupirocin	Use: superficial MRSA infection	• Nephrotoxic

recommended. Uncontrolled glucose levels are associated with increased bacteremia, reduced skin graft take, and increased mortality.[37] The protein requirement in severely burned patients is 1.5 to 2.0 g/kg per day, which attenuates the increased oxidation rate of amino acids.[37]

8.4.4 Anesthesia

Pain management after burn injury is difficult and requires a methodical, rational approach.[40] Burn injury is one of the most painful types of trauma due to injury of both sensory organelle receptors in the dermis and afferent nerve fibers leading to the skin.[42] Debridement, daily wound care, excision and grafting, and physical therapy, all further affect sensory feedback loops. The guidelines for providing adequate pain relief for burn patients begin with differentiating background pain from procedural and breakthrough pain, while treating from anxiety, depression, and possible substance abuse. The goal is for the patient to be comfortably awake and alert.[43]

Classic recommendations of avoiding succinylcholine after 72 hours are still valid.[40] Some studies suggest the use of intravenous opioids from the time resuscitation begins and addressing breakthrough pain with short-acting opioids and nonsteroidal anti-inflammatory medications. During procedures or dressing changes, ketamine, inhaled nitrous oxide, and benzodiazepines have been used with success. Propofol or Precedex has been used for sedation while the patient is mechanically ventilated; however, it is important to note that these infusions do not provide analgesia. Opioids are the mainstay of treatment; however, anxiolytics, anticonvulsants, and nonsteroidal anti-inflammatory drugs (NSAIDs) can be used as adjuncts to control the patient's relentless pain.[43] The prevention of chronic neuropathic pain is critical and therefore other adjuncts are utilized, such as laser therapy, acupuncture, fat grafting, and nerve decompression.

8.4.5 Infection/SIRS/MSOF

Burn wound infections are common after burn injury and the practitioner must be vigilant during clinical assessment (▶ Table 8.7). The larger the size of the burn injury, the greater the risk of infection, which is due to decreased cell mediated immunity, as thermal injury results in less phagocytic activity and lymphokine production by macrophages.[44] Specifically, the large surface burns have a systemic immunomodulatory effect by skewing the system toward an interleukin-mediated response.[45] Multiple studies have looked at the role of prophylactic antibiotics and current recommendations do not support their use.[46]

Hospital-associated infections (HAIs) in severely burned patients remain a major cause of morbidity and mortality. Weber et al used HAI surveillance data at a single institution over 5 years and found the most common sites to be the respiratory tract (ventilator-associated pneumonia [VAP] and tracheobronchitis), the urinary tract, burn surgical site infection, burn wound cellulitis, superficial thrombophlebitis, peritonitis, *Clostridium difficile* colitis, and device-related bacteremias (central-line associated bloodstream infections).[44]

Pneumonia, specifically VAP, is one of the most common infections and most common cause of death in severely burned patients (greater than 30% TBSA). Burn patients become susceptible secondary to immunosuppression, impaired secretory clearance of pulmonary secretions, inflammatory cascade activation, and leakage of plasma into the lung parenchyma. Diagnosis remains difficult in the burned patient, but the cornerstone of accurate diagnosis involves the use of bronchoalveolar lavage with quantitative cultures.[37]

Central line-associated blood stream infections (CLABSIs) are common among patients with burn injuries due to the immunosuppressed state and potential burn wound bacterial colonization. Van Duin et al implemented interventions to decrease CLABSIs in a burn intensive care unit. These interventions included enhanced education of medical staff, mandatory nursing training on IV line care and maintenance, central line changes over a guide-wire every three days with the use of a new site every six days, introduction of antibiotic-impregnated central venous catheters, universal glove and gown use, and use of chlorhexidine patch at insertion site. The interventions decreased the incidence of CLABSIs and the number of CLABSIs caused by *Staphylococcus aureus*.[45]

The most common pathogens among burn intensive care units vary among institutions. Throughout the literature, *Pseudomonas aeurginosa* is recognized as the most common pathogen isolated, followed by *Staphylococcus aureus.* At UNC, the most common pathogens isolated among the burn ICU patients are *Pseudomonas* and *Acinetobacter* spp (▶ Table 8.7).[44]

8.5 Treatment

8.5.1 Airway

Treating the burn patient is complex and requires a multidisciplinary approach in order to limit morbidity and mortality. Initial management should start with airway, breathing, and circulation under Advanced Trauma Life Support guidelines. In the severely burned patient, early intubation and ventilator support is appropriate for airway management. The burned patient has reduced pulmonary compliance and increased chest

Table 8.7 Burn wound infections

Signs	Common bacteria	Other infectious agents	Diagnosis
Rapid eschar separation Edema Partial-thickness conversion to full thickness Hemorrhage within the wound Green fat discoloration Necrotic skin around wound bed Erythema Gangrenosum Malodor from wound bed	*P. aeruginosa* (most common) *S. aureus* (2nd most common) *E. coli* Acinetobacter Enterobacter Enterococcus *C. difficile* Stenotrophomonas	Herpes simplex Virus (most common virus) *C. Albicans* (most common fungus) Mucormycosis Aspergillus	Biopsy (gold standard): > 10^5 organisms

wall rigidity, leading to high airway pressures and exacerbation of the lung injury. Therefore, using of low tidal volumes with permissive hypercapnia is recommended. Another ventilator strategy is using high-frequency oscillator or percussive ventilation.[37] As a last resort, extracorporeal membrane oxygenation can be considered. The benefits have been studied in the pediatric population and adult patients with inhalation injury. Supplemental therapies with inhaled nitric oxide, aerosolized heparin, and N-acetylcysteine have some benefit in selected patients. Early tracheostomy provides shorter time to extubation and increases patient comfort, but offers no advantage in ventilatory support, length of stay, or survival.[37] Burn-induced hypermetabolism is an important sequela that leads to complications and death; therefore, the management recommendations are outlined in ▶ Table 8.8.[37,47,48]

8.5.2 Excision and Grafting

Burn wound excision, moisture dressings, and resurfacing with skin grafts are key to successful management. Early excision of burned tissue, within 24 to 48 hours, halts the sequela of the postburn inflammatory response and improves survival. Bedside evaluation remains the most common method to measure burn depth; however, other modalities such as biopsy and histology, thermography, and indocyanine green video angiography are other modalities that are beneficial. First-degree burns, referred to as superficial burns, are characterized as painful and blanching and heal without scar formation. Second-degree burns are further categorized as superficial partial and deep-partial thickness burns. Superficial partial thickness burns are painful, blanch, blister, and have intact epithelial appendages. Deep-partial thickness burns have loss of hair follicles and are white or mottled in color. Third-degree burns are full-thickness burns, have a leathery appearance, and extend to the subcutaneous fat. Fourth-degree burns are characterized by their involvement of adipose tissue, muscle, or bone. The burn depth, characteristics, and associated treatments are further listed in ▶ Table 8.9. There are many options for wound coverage, which include autografts, allografts, xenografts, or synthetics (▶ Table 8.10).[41,49,50,51,52,53]

Autografts revascularize through imbibition, inosculation, and neovascularization and are more successful in split-thickness skin grafts. Once burn wounds are closed, emphasis shifts to rehabilitation. Burn scars are kept hydrated with emollients. Pressure garments decrease lymphedema, which helps reorient collagen and makes scars flatter and smoother. Early splinting and pressure garments reduce the formation of joint contractures.[49,50,51,52,53]

8.5.3 Reconstruction

Burn injury has downstream consequences that affect a patient's confidence and self-esteem. The burn itself is a constant reminder of the traumatic event. While some burn wounds heal uneventfully, others can exhibit degrees of normal and abnormal scarring, which is one of the major sequelae that affect the patients' psyche. Hypertrophic burn scars are the result of disorganized wound healing and can cause disabling contractures, dysesthesias, paresthesias, pain, stiffness, surface irregularities, chronic folliculitis, hyperemia, and persistent pruritus.[54] They are characterized by excessive inflammation, prolonged re-epithelialization, excess collagen production, abnormal extracellular matrix remodeling, and fibroblast proliferation. The scar also replaces the skin's epithelial appendages that are critical for thermoregulation, UV protection, and mechanical trauma protection.[55,56]

Table 8.8 Burn induced hyper-metabolism management

Recommendation	Effect
Early excision of full-thickness burns	Halts inflammatory mediators
Strict glucose control: Insulin	Target: 80–110 mg/dL Decreases infectious complications and mortality rates
Non-selective beta-blocker: propranolol	Blunt cardiac response Reverses muscle protein catabolism Decreases wound infection rates Decreases wound healing time Decreases mortality
Testosterone analog: Oxandrolone	May improve catabolic response, skeletal muscle growth, wound healing

Table 8.9 Burn classification

Burn depth	Characteristics	Treatment
1st degree/Superficial	Painful, blanches, dermal vasculature dilation, epidermis (sunburn), heals w/o scar, no debridement needed, Intact epithelial appendages	Local wound care: • clean: warm soap, antiseptics • Excise nonviable tissue/blisters • topical antimicrobials • occlusive dressing
2nd degree Superficial dermis (papillary)—superficial partial thickness Deep dermis (reticular)—Deep partial thickness	Superficial dermis (papillary): Painful, blanch, blister. Intact epithelial appendages Deep dermis (reticular): loss of hair follicles, blisters, pink, white mottled	Superficial dermis: Local wound care, no excision needed Deep dermis: skin grafts
3rd degree Full-thickness burns	Leathery, sub-dermal (down to subcutaneous fat)	Skin grafts +/− escharotomy and fasciotomy Early excision and wound closure (> 30% TBSA)
4th degree	Down to adipose tissue, muscle, or bone (periosteum)	Skin grafts +/− escharotomy and fasciotomy Early excision and wound closure (> 30% TBSA)

Table 8.10 Wound coverage options

	Product	Use	Pros	Cons
Temporary	Integra	Allows early wound coverage Minimizes granulation tissue Limited donor sites Unstable patients Burns > 50% TBSA (combo w/ autograft) Hand/Face burns Burns with exposed tendons/bone	Decreases need for painful dressing changes	Expensive Can become infected under bolster/ vacuum dressing
	Allografts: Cadaver	Early wound coverage Wound bed infection Unstable Scarce donor sites	Best option for bridging and later autografting	Limited 2–4 wk; undergoes thrombosis and rejection requiring removal
	Xenografts: porcine	Early wound coverage	Good bridging and later autografting Decreases need for painful dressing changes	Limited 1–2 weeks; undergoes thrombosis and rejection requiring removal (liquefaction)
Permanent	Split thickness skin graft (STSG): 0.013–0.016 cm thick	Non-cosmetic areas Small burns	More likely to survive (thinner) Donor sites can be used again (lateral thighs, back, buttocks) Local wound care for donor site dependent on institution (Silvadene, bacitracin and Xeroform, Tegaderm, etc)	Contract (Meshing increases surface area) Seroma/Hematoma formation (Meshing prevents this)
	Full-thickness skin graft (FTSG):	Face, genital, hand and palm burns (2nd week)	Better skin color match Do not contract Added thickness useful	Limited donor sites (behind ear, above clavicle, groin) Less likely to survive than STSG Requires donor site primary closure

There are many subjective scar assessments, such as the Vancouver burn scar assessment score, the clinical scar assessment scale, and the patient and observer scar assessment scale.[57] The timing for scar revision is between 12 and 18 months because of the continuous collagen remodeling that takes place.

The treatment of hypertrophic burn scars is a challenge for both the burn survivors and burn care providers. Many solutions have been proposed to address hypertrophic burn scars, but it is difficult to predict whether a given therapy will work for each patient. There are multiple treatment modalities that can be offered to a patient. Nonsurgical management includes compression dressings, silicone gel sheeting, scar massage, and topical moisturizing agents.[55,56,57] Other adjunct therapies such as imiquimod, tacrolimus, and corticosteroids have been used with some success.[58]

Silicone gel sheeting was first reported in the 1980s and is proposed to improve skin hydration from occlusion of the silicone, increase oxygen tension, and electrical charge changes due to the silicone molecule.[54] The recommended use is to wear the garment for 12 hours a day for several months. However, they do require frequent cleaning, are costly, and may cause skin maceration if not removed to allow drying.[54] Although the use of silicone gel sheeting is promising, a recent Cochrane review concluded that "trials evaluating silicone gel sheeting as a treatment for hypertrophic and keloid scarring showed improvements in scar thickness and scar color, but are poor quality and highly susceptible to bias."[54]

The other major nonsurgical scar therapy is the use of compression dressings. In 1968, at the Shriners Hospital for Children in Galveston, the first major work on studying and using compression dressings (garments) was done. Their recommendations were to wear the garments continuously for at least 6 months or until scars started to fade away. Multiple studies exist on compression dressing; however, the studies have been underpowered and are difficult to draw conclusions to state whether compression dressing is beneficial.[54]

More invasive therapies for hypertrophic burn scars include intralesional therapy and multiple surgical techniques including laser therapies. Surgical options include excision, contracture release, tissue rearrangements, wound closure with autografts, tissue expanders, and pedicled- free-flap interposition.[54,55,56] One of the most commonly used techniques is the Z-plasty. The Z-plasty is used for scar contracture, scar camouflage, and to increase the scar length. Rohrich et al described the 60° Z-plasty that allows a 25% increase in scar length.[59] A half Z-plasty is indicated when one side of the scar is elastic and the other side is not. This technique is useful to release scar contractures at the skin interface between normal tissue and burned tissue.[57] Another technique is the W-plasty that involves a series of consecutive triangles made on one side of the scar with its mirror image on the other side of the scar.[57] Indications for W-plasty are linear scars greater than 2 cm with an angle greater than 35 degrees; short scars on the forehead or cheek; closure in pretrichial areas; and closure over curved surfaces. This technique is not suitable for longer scars.[60]

The most recent development in hypertrophic burn scar treatment has been laser therapy. Laser therapy has several advantages over surgical and nonsurgical treatments. Laser treatments are relatively short procedures with low risk, short patient recovery, and have the potential to dramatically improve the final appearance and texture of hypertrophic burn scars.[54] Laser therapies include the vascular-specific pulsed dye laser (PDL), ablative/non-ablative fractional laser, and the intense pulsed light. The PDL causes selective photothermolysis and leads to capillary coagulation necrosis. This laser has been used to reduce hyperemia and hypertrophic scar formation and improves burn scar texture, erythema, pruritus, pain, and scar volume.[55,56] Ablative methods consist of fractionated (not whole field) CO_2 and Erbium-YAG laser treatments. The ablative fractional CO_2 laser is used to improve burn scar abnormal texture, thickness, and stiffness. The ablative laser targets intracellular water that leads to vaporization of tissue and denaturation of surrounding extracellular proteins. Therefore, microscopic columns of abnormal dermis are vaporized and as a result stimulate collagen production and remodeling. Nonablative lasers induce coagulation only. Intense pulsed light devices are used for burn scar dyschromia and chronic folliculitis, as well as refractory hyperemia. This platform works by delivering focused, noncoherent, controlled light energy through a coupling gel. It is able to coagulate vascular lesions, remove unwanted pigment, and destroy hair follicles.[56]

8.6 Clinical Case Examples

8.6.1 Case 1

This 26-year-old patient sustained mostly full-thickness burns to the head, neck, and chest, after he came into contact with a > 10,000 volt electrical source. Following resuscitation, oral intubation, and conversion to tracheostomy, he underwent debridement of these wounds, including a partially necrotic sternocleidomastoid muscle, leaving the internal and external jugular veins exposed (▶ Fig. 8.3; ▶ Fig. 8.4). Carotid arteriogram had demonstrated no intimal injury or aneurysmal changes. He was reconstructed with a pedicled pectoralis major myocutaneous flap and split-thickness skin grafting. Ten months after injury, he has closure of his trachea-cutaneous fistula, stable coverage of zone 2 of the neck, and minimal tethering on the left (▶ Fig. 8.5). He is currently undergoing laser resurfacing and modulation of his hypertrophic scars to improve his neck range of motion.

8.6.2 Case 2

This 55-year-old man sustained a partial and full-thickness thermal blast injury from a flare gun shot at close range (▶ Fig. 8.6). He sustained no facial fractures, but did have some weakness of the buccal branch of the facial nerve as well as a salivary leak from the underlying parotid gland. Following excision of the full-thickness burns, removal of a foreign body (the casing from the flare pellet), and debridement of the superficial lobe of the parotid gland (with confirmation of an intact Stenson's duct), we performed cheek reconstruction with a pedicled platysmal myocutaneous flap, based on the branches of the facial artery and vein (▶ Fig. 8.7). The patient returned to clinic only once postoperatively for drain and suture removal (▶ Fig. 8.8). He had no evidence of parotid leak or facial nerve dysfunction.

8.7 Postoperative Care and Outcomes

8.7.1 Rehabilitation

Rehabilitation is critical in the postburn-injured patient to regain maximal functional capacity. Physical and occupational therapists provide exercises for early mobilization, range of motion, strengthening, and conditioning. These exercises with splinting have promoted healing in the postburn patients. Splinting and various adaptive devices are utilized to prevent and treat contractures, while soft tissues are remodeling. Burn-injured patients are in a great deal of pain, and the most comfortable position is the position of deformity. Therefore, recommendations include placing the neck in full extension,

Fig. 8.3 Full-thickness electrical burn to neck and chest

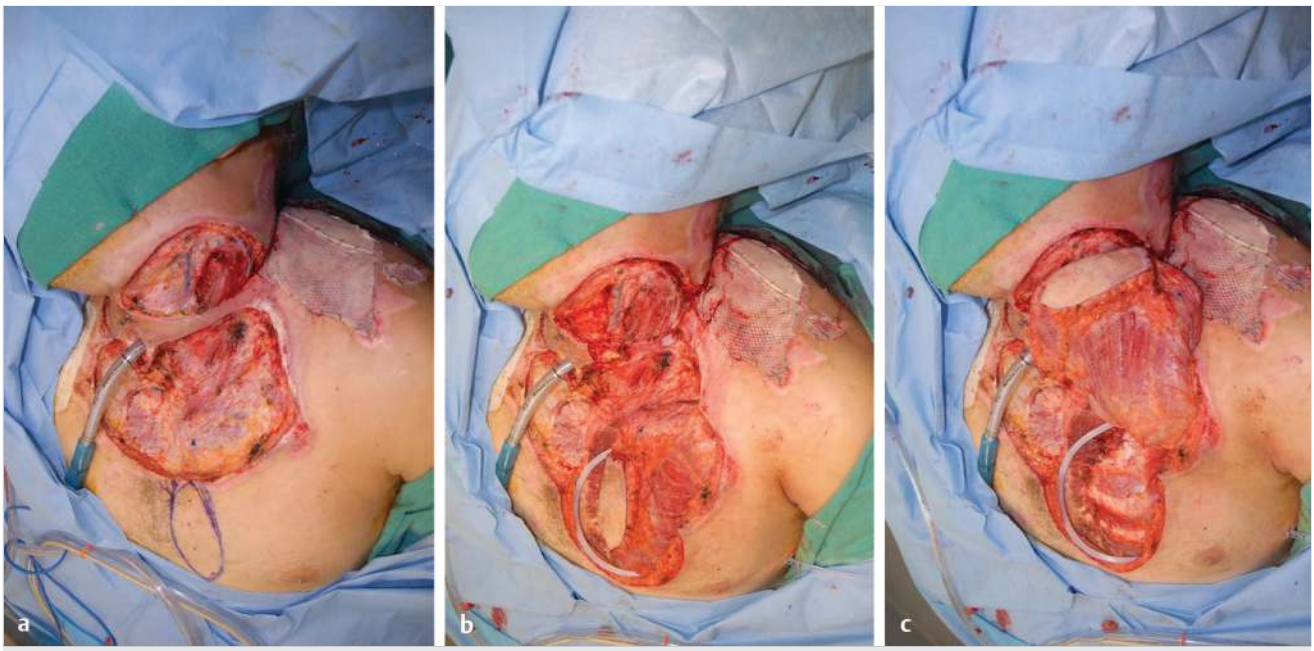


Fig. 8.4 (a-c) Debridement of neck and chest burn woulds, with coverage of using pectoralis myocutaneous flap and split skin graft.

Fig. 8.5 (a,b) S/P high voltage electrical burn to neck and chest, with postoperative results at 10 months.

Fig. 8.6 Partial and full-thickness burns to the face from a flare gun, shot at close range, with involvement of superficial lobe of the parotid gland.

arms to the sides, and legs straight with 10-degree flexion of the knees. Repositioning the patient frequently is important to prevent pressure ulcer formation at the sacrum, hips, and heels. The use of air-fluidized bed has been used with success.[61]

Burn injury causes physical and psychological impairment. Burn patients have been noted to have distress, low self-esteem, anxiety, depression, and posttraumatic stress disorder.[62] Recovery in burn patients often depends upon emotional, psychosocial, and spiritual healing. Burn providers should consider and address the spiritual needs of burns patients as a component of recovery. Hultman et al found that utilization of pastoral care appeared to be linked to the size of burn, financial charges, and length of stay, with religious affiliation serving as a marker for improved survival.[63] Once a patient is discharged from the hospital, the importance of implementing self-care and lifestyle changes are crucial for successful recovery. Therefore, patient education and access to both medical and psychological care is critical for restoring quality of life.[62]

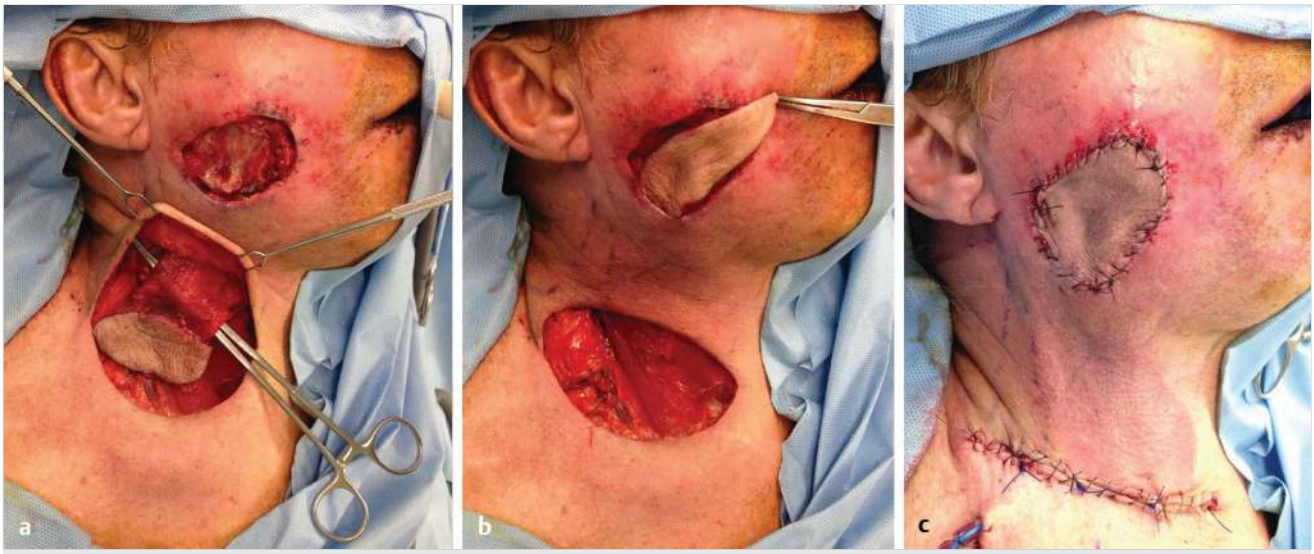


Fig. 8.7 **(a-c)** Debridement of burn wounds, including parotid gland, with harvest and insetting of platysmal myocutaneous flap.

Fig. 8.8 **(a,b)** S/P cheek reconstruction with platysmal myocutaneous flap, intact facial nerve function, no parotid fistula or gustatory sweating.

8.8 Other Thermal-Type Injuries

8.8.1 Inhalation Injury

Presence of inhalation injury is known to increase mortality of burn injury by as much as 50%, but advances in diagnostic methods have improved outcomes, allowing for earlier identification and more efficient treatment.[61]

The pathophysiology involves activation of pulmonary macrophages and neutrophil migration to the lung. These inflammatory cells produce proteases and oxygen-free radicals, which increase the microvascular permeability and the result is pulmonary edema within 48 hours of injury.[41,43] Respiratory failure in severely burned patients can be either direct or indirect. Direct causes are secondary to inhalation of heat or caustic materials in the air. Indirect causes are due to activation of the SIRS response, sepsis, pneumonia, and ventilator-associated lung injury.[37,43]

Inhalational injury is due to carbonaceous material and smoke, not usually from direct thermal injury. Risk factors for inhalational injury include patient and environmental factors (▶ Table 8.11).[41,43] The diagnostic test of choice is flexible bronchoscopy, where soot can be visualized. Soot is the best predictor for development of adult respiratory distress syndrome and death. The most common complication from inhalation injury is upper airway obstruction from edema, which is exacerbated by fluid resuscitation. Another often-concomitant complication is carbon monoxide poisoning. Clinicians should have a high index of suspicion and remember to check the CO levels on arterial blood gases. Treatment of carbon monoxide is 100% oxygen administration to accelerate the dissolution of carbon monoxide from hemoglobin.[41,43]

8.8.2 Frostbite

Frostbite is a special type of burn injury that results from exposure to low temperatures. The pathophysiology involved is local tissue freezing that results in ice crystal formation in the extracellular matrix and cellular dehydration.[59] Mechanical tissue injury ensues and results in protein denaturation and inhibition of DNA synthesis. The global effect on the cellular level leads to endothelial damage, blood flow slowing, thrombosis, and tissue ischemia.[64,65]

The first step in evaluation is a full history and physical examination with attention to a thorough examination of the involved limb and determination of depth of injury.[64,65] Frostbite is categorized as first through fourth degrees (▶ Table 8.12).[65]

Table 8.11 Inhalational injury

Risk factors	Soft signs	Hard signs	Reasons for elective intubation
Alcohol intoxication	stridor	Carbonaceous sputum	$PaO_2/FiO_2 < 250$
Confinement to closed space during burn	Facial burns	Soot (bronchoscopy)	$PaO_2 < 60$
Rapid combustion burns	wheezing		Pa $CO_2 > 50$ or RR > 30
Extremes of age			Severe upper airway edema
			Altered mental status

Table 8.12 Frostbite classification

Degree	Characteristics	Physical exam
1st degree	Superficial Central white area surrounded by erythema	
2nd degree	Blisters (clear fluid)	
3rd degree	Severe Deep tissue involvement Hemorrhagic blisters	+/− No pulses +/− No capillary refill Purple discoloration Hemorrhagic blisters +findings→ Angiography
4th degree	Severe Deep tissue involvement Full thickness tissue necrosis	+/− No pulses +/− No capillary refill Purple discoloration +findings→ Angiography

Table 8.13 Chemical burns

Chemical	Pathophysiology	Clinical	Treatment
Hydrofluoric Acid: Industrial workers Household cleaning solutions Germicides	Liquefaction necrosis	Pain White blisters with surrounding redness Ventricular fibrillation (low Calcium)	Copious irrigation Topical calcium gluconate/chloride gel Cardiac monitor Refractory→ IV calcium gluconate Refractory still → excise
Acid: Hydrochloric acid Sulfur	Coagulation necrosis	Pain	Copious irrigation
Alkali: Bleach	Liquefaction necrosis More tissue damage than acid burn	Pain	Copious irrigation
Tar/Phenol: Disinfectants solvents	Skin necrosis	Ventricular arrhythmias Coma Seizures	Copious irrigation Topical Polyethylene glycol

Current management involves supportive therapy, transfer to a high-level of care, and rapid rewarming in a circulating 40 C° warm water for approximately thirty minutes. Debridement of all clear blisters, to prevent or diminish thromboxane A2 release, is recommended, leaving hemorrhagic blisters intact.[64,65] Initial wound treatment involves limb elevation, avoidance of pressure dressings, adequate pain control, Silvadene for blistered areas, and antibiotics for any signs of infection. For severe upper extremity frostbites, treatment with systemic or directed arterial thrombolytics has been suggested with salvage rates up to 85 to 90%.[65] Amputation should be delayed for approximately three months until final demarcation of viable and necrotic tissue is completed.[64,65]

8.8.3 Chemical Burns

Chemical burn injuries are commonly seen in the emergency department and practitioners should be cognizant of early and definitive management (▶ Table 8.13).[66,67] Obtaining an accurate history is critical as one must consider the strength of the agent and duration of contact. Initial management includes removal of all clothing and copious water irrigation with special attention to the patient's eyes and respiratory status. Evaluation under advanced trauma life support (ATLS) guidelines is useful for the assessment of these patients in order for concomitant injuries not to be missed.[66,67]

8.8.4 Electrical Injury

Electricity is the flow of electrons through a conductor. According to Ohm's law, current is proportional to the voltage divided by the resistance. Bone, fat, and tendon have the highest resistance, which create the most heat. Nerves, blood, and mucous membranes have a lesser resistance. Furthermore, the cross-sectional area through which the current travels affects the resistance; the wrist, for example, generates more heat due to its smaller cross-sectional area than the forearm and arm. The mechanisms of electrical injury include Joule heating and dielectric heating. Joule heating increases the permeability of cell membranes and induces protein denaturation, which leads to tissue damage.[68] Electrical injuries account for 3% of burn injuries that occur yearly in the United States.[69] Electrical injury accounts for 5 to 7% of hospital burn admissions yearly.[70]

Table 8.14 Electrical burns

Degree	Characteristics	Treatment
1st degree	Superficial Erythema	Local wound care
2nd degree	Deeper Edema between superficial injury and deeper viable tissue	Debride intact blisters Topical antimicrobials
3rd degree	White, black, dry, leather	> 2 cm → debridement + skin graft
4th degree	High voltage Massive soft-tissue destruction (subcutaneous fat, muscle, bone)	As above

Electrical burns are either high voltage (> 1,000 volts) or low voltage (< 1,000 volts). High-voltage injuries are seen in industrial accidents, whereas low-voltage injuries are seen in household accidents.[69] Patients with high-voltage injuries have larger total body surface area burns, longer ICU stays, longer hospitalizations, and higher rates of fasciotomy, amputation, nerve decompression, and outpatient reconstruction. Patients with high- and low-voltage injuries appear to have similar rates of neuropsychiatric sequelae (memory loss, depression, insomnia, and posttraumatic stress disorder) and delays in return to work.[70] Electrical injuries are devastating injuries that are predicted to increase in incidence. Prevention is the preferred strategy for treatment with patient education on the frontline.[69]

The first step in evaluation is determining the extent of the injury. The injuries are categorized from first degree to fourth degree (▶ Table 8.14).[69] Electrical injury affects other organ systems beyond the wound. Neurologic complications include paralysis, Guillain-Barre syndrome, amyotrophic lateral sclerosis, and transverse myelitis.[69] The most common neurological finding is peripheral weakness.[69] Electrical injuries affect the myocardium with ventricular fibrillation as the most common cause of death at the scene of an electrical injury.[69] Electrical injuries require increased amounts of intravenous fluids because of occult tissue damage and associated hemoglobin and myoglobin precipitation in the renal tubules as a result of large volume muscle loss.[38] Aggressive volume loading to increase urine output to 1 to 2 cc/kg/hr is recommended, and mannitol can be utilized to maintain renal perfusion. Furthermore, early fasciotomies must be considered as the underlying tissue damage may exceed that of the skin, resulting in compartment syndrome and ongoing rhabdomyolysis.[38,69]

8.8.5 Radiation Injury

Electromagnetic radiation emits ionizing radiation that damages biological tissue. The extent and depth of injury depend on the dosage of radiation. Local effects of radiation injury range from mild erythema to desquamation. Obliterating endarteritis is a common feature seen in radiation injury, which eventually leads to necrosis. Systemically, radiation injury causes acute radiation syndrome, which is characterized by prodromal symptoms followed by refractory hypovolemic shock and death.[71]

The initial management is similar to other types of burns, which includes removal of contaminated clothing and copious water irrigation. A thorough history and physical examination should be ensued, followed by appropriate laboratory studies, including the lymphocyte count. The management of these burns is similar to other burns described in the previous sections. One caveat includes addition of hyperbaric oxygen therapy that may enhance wound healing in the irradiated tissue.[71]

8.9 Review Questions

1. What is a hard sign for smoke inhalation injury?
   a) Soot.
   b) Stridor.
   c) Wheezing.
   d) Facial burn.
2. What is the most common side effect of Mafenide?
   a) Hyponatremia.
   b) Hypocalcemia.
   c) Thrombocytopenia.
   d) Metabolic acidosis.
3. What is the most common bacterium involved in burn wound infections?
   a) *S. epidermidis*.
   b) *P. aeruginosa*.
   c) Herpes simplex virus.
   d) *C. albicans*.
4. What is the crystalloid of choice for fluid resuscitation in burn patients?
   a) Hypertonic saline (3%).
   b) Normal saline.
   c) Lactated ringers.
   d) Dextrose 5%.
5. Which cytokine increases the hypothalamic thermoregulatory set point?
   a) INF-gamma.
   b) IL-8.
   c) IL-4.
   d) IL-1.

8.9.1 Answers

1. a. Soot.
2. d. Metabolic acidosis.
3. b. *P. aeruginosa*.
4. c. Lactated ringers.
5. d. IL-1.

References

[1] Pećanac M, Janjić Z, Komarcević A, Pajić M, Dobanovacki D, Misković SS. Burns treatment in ancient times. Med Pregl. 66(5–6):263–267
[2] Majno G. The Healing Hand - Man and Wound in the Ancient World. Cambridge: Harvard University Press; 1975
[3] Dupuytren G, Brierre de Boismont AJ, ALM P. Oral lessons of clinical surgery, faites à l'Hôtel-Dieu de Paris: Baillière Paris; 1839

[4] Weeks BS. "Brief introduction to the history of burns medical science." Karger 2004: 1–3

[5] Jackson D, Topley E, Cason JS, Lowbury EJ. Primary excision and grafting of large burns. Ann Surg. 152:167–189

[6] Engrav LH, Heimbach DM, Reus JL, Harnar TJ, Marvin JA. Early excision and grafting vs. nonoperative treatment of burns of indeterminant depth: a randomized prospective study. J Trauma. 23(11):1001–1004

[7] Herndon DN, Barrow RE, Rutan RL, Rutan TC, Desai MH, Abston S. A comparison of conservative versus early excision. Therapies in severely burned patients. Ann Surg. 209(5):547–552, discussion 552–553

[8] Tanner JC , Jr, Vandeput J, Olley JF. The mesh skin graft. Plast Reconstr Surg. 34:287–292

[9] Alexander JW, MacMillan BG, Law E, Kittur DS. Treatment of severe burns with widely meshed skin autograft and meshed skin allograft overlay. J Trauma. 21(6):433–438

[10] Heimbach D, Luterman A, Burke J, et al. Artificial dermis for major burns. A multi-center randomized clinical trial. Ann Surg. 208(3):313–320

[11] Hansbrough JF, Boyce ST, Cooper ML, Foreman TJ. Burn wound closure with cultured autologous keratinocytes and fibroblasts attached to a collagen-glycosaminoglycan substrate. JAMA. 262(15):2125–2130

[12] Moyer CA, Brentano L, Gravens DL, Margraf HW, Monafo WW , Jr. Treatment of large human burns with 0.5 per cent silver nitrate solution. Arch Surg. 90: 812–867

[13] Shaffer PA, Coleman W. Protein metabolism in typhoid fever. Arch Intern Med (Chic). IV(6):538–600

[14] Cope O, Moore FD. The redistribution of body water and the fluid therapy of the burned patient. Ann Surg. 126(6):1010–1045

[15] Moore FD. The body-weight burn budget. Basic fluid therapy for the early burn. Surg Clin North Am. 50(6):1249–1265

[16] Evans EI, Purnell OJ, Robinett PW, Batchelor A, Martin M. Fluid and electrolyte requirements in severe burns. Ann Surg. 135(6):804–817

[17] Baxter CR, Shires T. Physiological response to crystalloid resuscitation of severe burns. Ann N Y Acad Sci. 150(3):874–894

[18] Shirani KZ, Pruitt BA , Jr, Mason AD , Jr. The influence of inhalation injury and pneumonia on burn mortality. Ann Surg. 205(1):82–87

[19] Zwischenberger JB, Cardenas VJ , Jr, Tao W, Niranjan SC, Clark JW, Bidani A. Intravascular membrane oxygenation and carbon dioxide removal with IVOX: can improved design and permissive hypercapnia achieve adequate respiratory support during severe respiratory failure? Artif Organs. 18(11):833–839

[20] Hultman, CS. 50 Studies Every Plastic Surgeon Should Know. Taylor & Francis Group, LLC; 2015

[21] World Health Organization. The Global Burden of Disease: 2004 Update. World Health Organization, Geneva 2008. Available at: www.who.int/health-info/global_burden_disease/ GBD_report_2004update_full.pdf

[22] Peck MD. Epidemiology of burns throughout the world. Part I: Distribution and risk factors. Burns. 37(7):1087–1100

[23] Facts about injuries: Burns World Health Organization. Available at: https://www.who.int/violence_injury_prevention/publiations/other_injury/on/burns_fact/sheet.pdf

[24] "Fire Deaths and Injuries: Fact Sheet." Centers for Disease Control and Prevention. U.S. Department of Health and Human Services, October 11, 2011. Web. December 20, 2014. Available at: http://www.cdc.gov/homeandrecreationalsafety/fire-prevention/fires-factsheet.html

[25] Finkelstein EA. Corso Ps, Miller TR, et al. The Incidence and Economic Burden of Injuries in the United States. New York: Oxford University Press; 2006:9–169

[26] Bell NJ, Schuurman N, Morad Hameed S. A small-area population analysis of socioeconomic status and incidence of severe burn/fire-related injury in British Columbia, Canada. Burns. 35(8):1133–1141

[27] Poulos R, Hayen A, Finch C, Zwi A. Area socioeconomic status and childhood injury morbidity in New South Wales, Australia. Inj Prev. 13(5): 322–327

[28] Delgado J, Ramírez-Cardich ME, Gilman RH, et al. Risk factors for burns in children: crowding, poverty, and poor maternal education. Inj Prev. 8(1):38–41

[29] Forjuoh SN, Guyer B, Strobino DM, Keyl PM, Diener-West M, Smith GS. Risk factors for childhood burns: a case-control study of Ghanaian children. J Epidemiol Community Health. 49(2):189–193

[30] Adesunkanmi AR, Oginni LM, Oyelami AO, Badru OS. Epidemiology of childhood injury. J Trauma. 44(3):506–512

[31] Rivara FP, Aitken M. Prevention of injuries to children and adolescents. Adv Pediatr. 45:37–72

[32] Laffoy M. Childhood accidents at home. Ir Med J. 90(1):26–27

[33] De-Souza DA, Marchesan WG, Greene LJ. Epidemiological data and mortality rate of patients hospitalized with burns in Brazil. Burns. 24(5): 433–438

[34] Morrow SE, Smith DL, Cairns BA, et al. Aetiologies and outcomes of burns in infants under one year old. Ann Burns Fire Disasters. 12:81–83

[35] Koupil J, Brychta P, Říhová H, Kincová S. Special features of burn injuries in elderly patients. Acta Chir Plast. 43(2):57–60

[36] Peden M, et al. World Report on Child Injury Prevention. World Health Organization, 2008

[37] Ipaktchi K, Arbabi S. Advances in burn critical care. Crit Care Med. 34(9) Suppl:S239–S244

[38] Ahrns KS. Trends in burn resuscitation: shifting the focus from fluids to adequate endpoint monitoring, edema control, and adjuvant therapies. Crit Care Nurs Clin North Am. 16(1):75–98

[39] Demling RH. The burn edema process: current concepts. J Burn Care Rehabil. 26(3):207–227

[40] Berger MM, Bernath M-A, Chioléro RL. Resuscitation, anaesthesia and analgesia of the burned patient. Curr Opin Anaesthesiol. 14(4):431–435

[41] Rose JK, Herndon DN. Advances in the treatment of burn patients. Burns. 23 Suppl 1:S19–S26

[42] American Burn Association. Available at: http://www.ameriburn.org

[43] Faucher L, Furukawa K. Practice guidelines for the management of pain. J Burn Care Res. 27(5):659–668

[44] Weber DJ, van Duin D, DiBiase LM, et al. Healthcare-associated infections among patients in a large burn intensive care unit: incidence and pathogens, 2008–2012. Infect Control Hosp Epidemiol. 35(10):1304–1306

[45] van Duin D, Jones SW, Dibiase L, et al. Reduction in central line-associated bloodstream infections in patients with burns. Infect Control Hosp Epidemiol. 35(8):1066–1068

[46] Pruitt BA , Jr, McManus AT, Kim SH, Goodwin CW. Burn wound infections: current status. World J Surg. 22(2):135–145

[47] Hemmila MR, Taddonio MA, Arbabi S, Maggio PM, Wahl WL. Intensive insulin therapy is associated with reduced infectious complications in burn patients. Surgery. 144(4):629–635, discussion 635–637

[48] Pidcoke HF, Wolf SE, Loo F, et al. Decreased mortality in burns with improved glucose control. Burns. 33(1) Suppl:S33

[49] Asuku ME, Milner SM. Burn surgery. In: Granick MS, Teot L, eds. Surgical wound healing and management. 2nd ed. New York, NY: Informa Healthcare; 2012:49–57

[50] Janzekovic Z. A new concept in the early excision and immediate grafting of burns. J Trauma. 10(12):1103–1108

[51] Klein MB, Heimbach D, Gibran N. Management of the Burn Wound. New York, NY: Wiley; 2004

[52] Mosier MJ, Gibran NS. Surgical excision of the burn wound. Clin Plast Surg. 36 (4):617–625

[53] Saffle JR. Closure of the excised burn wound: temporary skin substitutes. Clin Plast Surg. 36(4):627–641

[54] Friedstat JS, Hultman CS. Hypertrophic burn scar management: what does the evidence show? A systematic review of randomized controlled trials. Ann Plast Surg. 72(6):S198–S201

[55] Clayton JL, Edkins R, Cairns BA, Hultman CS. Incidence and management of adverse events after the use of laser therapies for the treatment of hypertrophic burn scars. Ann Plast Surg. 70(5):500–505

[56] Hultman CS, Edkins RE, Lee CN, Calvert CT, Cairns BA. Shine on: review of laser-and light-based therapies for the treatment of burn scars. Dermatol Res Pract. 2012:243651

[57] Garg S, Dahiya N, Gupta S. Surgical scar revision: an overview. J Cutan Aesthet Surg. 7(1):3–13

[58] Berman B, Viera MH, Amini S, Huo R, Jones IS. Prevention and management of hypertrophic scars and keloids after burns in children. J Craniofac Surg. 19 (4):989–1006

[59] Rohrich RJ, Zbar RI. A simplified algorithm for the use of Z-plasty. Plast Reconstr Surg. 103(5):1513–1517, quiz 1518

[60] Rodgers BJ, Williams EF, Hove CR. W-plasty and geometric broken line closure. Facial Plast Surg. 17(4):239–244

[61] Warden GD. Fluid resuscitation and early management. In: Herndon DN, ed. Total Burn Care. 3rd ed. Philadelphia, PA. Elsevier Saunders; 2007;107–118

[62] Liang CY, Wang HJ, Yao KP, Pan HH, Wang KY. Predictors of health-care needs in discharged burn patients. Burns. 38(2):172–179
[63] Hultman CS, Saou MA, Roach ST, et al. To heal and restore broken bodies: a retrospective, descriptive study of the role and impact of pastoral care in the treatment of patients with burn injury. Ann Plast Surg. 72(3):289–294
[64] Robson MC, Heggers JP. Evaluation of hand frostbite blister fluid as a clue to pathogenesis. J Hand Surg Am. 6(1):43–47
[65] Bruen KJ, Gowski WF. Treatment of digital frostbite: current concepts. J Hand Surg Am. 34(3):553–554
[66] Dibbell DG, Iverson RE, Jones W, Laub DR, Madison MS. Hydrofluoric acid burns of the hand. J Bone Joint Surg Am. 52(5):931–936
[67] Andrews K, Mowlavi A, Milner SM. The treatment of alkaline burns of the skin by neutralization. Plast Reconstr Surg. 111(6):1918–1921
[68] Kidd M, Hultman CS, Van Aalst J, Calvert C, Peck MD, Cairns BA. The contemporary management of electrical injuries: resuscitation, reconstruction, rehabilitation. Ann Plast Surg. 58(3):273–278
[69] Sanford A, Gamelli RL. Lightning and thermal injuries. Handb Clin Neurol. 120:981–986
[70] Chudasama S, Goverman J, Donaldson JH, van Aalst J, Cairns BA, Hultman CS. Does voltage predict return to work and neuropsychiatric sequelae following electrical burn injury? Ann Plast Surg. 64(5):522–525
[71] Milnder SM. Radiation injury, vesicular burns, and mass casualties. Total Burn Care. 2nd ed. Philadelphia, PA: WB Saunders; 2001:481–492

9 Skin Cancer

Lauren C. Nigro, Shuhao Zhang, Maryann E. Martinovic, and Andrea L. Pozez

Abstract

This chapter reviews the most common form of cancer worldwide. Over 3.5 million new cases of skin cancer are diagnosed each year in the United States alone, consisting primarily of basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and malignant melanoma (MM). Despite increased awareness and knowledge of modifiable risk factors, the incidence of cutaneous malignancy of all forms continues to rise. Melanoma, which is much less common than BCC or SCC, is responsible for a disproportionate number of skin cancer deaths, with projections of nearly 10,000 anticipated deaths yearly. Plastic surgeons are often called upon to assess and treat suspicious cutaneous lesions or to participate in the reconstruction after their excision. To provide a firm understanding of this significant health concern, topics discussed include surgical preparation, lymph node evaluation, risk stratification and staging, anticoagulation, and postoperative care.

Keywords: basal cell carcinoma (BCC), squamous cell carcinoma (SCC), malignant melanoma (MM)

9.1 Goals and Objectives

- Understand the prevalence and predisposing factors for basal cell carcinoma, squamous cell carcinoma, and malignant melanoma.
- Identify and diagnose high-risk lesions.
- Familiarize with the subtypes of basal cell carcinoma, squamous cell carcinoma, and malignant melanoma.
- Familiarize with the preoperative workup, including staging, of cutaneous malignancies.
- Know treatment options, including the appropriate surgical and nonsurgical treatment modalities.

9.2 Introduction

Skin cancer is the most common form of cancer worldwide. Over 3.5 million new cases of skin cancer are diagnosed each year in the United States alone, consisting primarily of basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and malignant melanoma (MM). Despite increased awareness and knowledge of modifiable risk factors, the incidence of cutaneous malignancy of all forms continues to rise. Melanoma in particular, which is much less common than BCC or SCC, is responsible for a disproportionate number of skin cancer deaths, with an anticipated 9,320 deaths in 2018.[1,2] Plastic surgeons are often called upon to assess and treat suspicious cutaneous lesions or participate in the reconstruction after their excision. Therefore, a firm understanding of this significant healthcare burden is necessary.

9.3 Patient Presentation

9.3.1 Basal Cell Carcinoma

As the most common skin malignancy, BCC is recognized in approximately two million people annually with the most at risk populations being Caucasians, males, and individuals over age 65.[3,4] Incidence in males is 1.7 times that in females.[3] Risk factors for development include chronic sun exposure, particularly intermittent, intense ultraviolet (UV) light exposure, and sunburns at any age.[1] Other predisposing factors include exposure to chemical carcinogens, ionizing radiation, solid organ transplantation, predisposing skin lesions (nevus sebaceous), and history of genetic syndromes (Bayez, Gorlin, Xeroderma Pigmentosum).[1] Clinically, patients present with BCC most commonly in sun-exposed areas with 86% occurring on the head and neck.[5]

Both clinical and histologic features contribute to the subtyping of BCC. While over 26 types have been described, we will focus on the most distinctive. These can be classified into two general categories: well-circumscribed (nodular, basosquamous) and diffuse (superficial, morpheaform, infiltrative, micronodular).[6] Nodular BCC is the most common, often recognized as a pearly, flesh-colored papule with a raised border and central dell. There are often arborizing telangiectasias, which can best be appreciated on skin surface microscopy or epiluminescence microscopy (dermoscopy) and ulceration (▶ Fig. 9.1a). Basosquamous types have a squamous component and carry a higher risk of metastasis (unlike BCC, with a metastatic rate of < 0.1%).[5] Diffuse BCC generally exhibits a more plaque-like appearance with ill-defined margins. Superficial spreading is the second most common type of BCC; however, the most common in younger patients.[1] It often has a scale or crust with an erythematous border that slowly expands, making it easily mistaken for a localized fungal infection or inflammatory reaction, such as eczema (▶ Fig. 9.1b).[6] Micronodular BCC is defined by finely palpable nodules with nests of malignant cells that invade the dermis, making recurrence high. Morpheaform or sclerosing type is a firm lesion that can resemble a scar. It can be challenging to treat as it often extends several millimeters radially within the dermis beyond what is visually appreciated and thus positive margins are often noted on final pathology.[5] Lastly, infiltrative BCC presents with an irregular configuration and has a very aggressive course. It should be noted that while a majority of BCC are flesh-colored, some may exhibit pigmentation.

9.3.2 Squamous Cell Carcinoma

SCC is the second most common skin malignancy in the United States. Like BCC, it is most commonly found in sun-exposed areas such as the face, hands, and forearms and is strongly correlated with a history of sunburns. SCC shares many of the same

Fig. 9.1 Basal cell carcinoma: (**a**) nodular type and (**b**) superficial spreading type.

Fig. 9.2 Squamous cell carcinoma. Photo courtesy of Alex Ortega, MD, Department of Dermatology, Virginia Commonwealth University.

genetic and environmental risk factors as BCC. Other additional risk factors include the human papilloma virus (HPV), chronic inflammation, and chronic scar (i.e., Marjolin's ulcer).

Cutaneous SCC appears as a raised, pink papule or plaque, often scaly and at times ulcerated (▶ Fig. 9.2). Approximately 80% of SCC arise from actinic keratosis (AK), premalignant lesions marked by erythematous, and rough scaly plaques (▶ Fig. 9.3).[7] Approximately 20% of all AK transform into SCC, though less than 1% undergo malignant transformation into SCC annually.[8,9] Invasive SCC can also develop de novo, most commonly in those who are immunocompromised, transplanted organ recipients, or in areas of chronic inflammation. Although it is less common, de novo SCC is much more aggressive, with a higher likelihood of metastasis and worse prognosis. High-risk pathologic subtypes include acantholytic (adenoid), adenosquamous, desmoplastic, and metaplastic or carcinosarcomatosis subtypes.

SCC in situ, also known as Bowen's disease, presents as an erythematous, scaly patch. It can be found anywhere on the body, including the mucosal surfaces. When it is found on the mucosa of the glans penis or labia majora, it is known as erythroplasia of Queyrat. Ten percent of Bowen's disease and a significantly higher percentage of erythroplasia of Queyrat transform into invasive SCC.

Keratoacanthoma is a rapidly growing, ulcerated nodule with a central keratin plug that often regresses spontaneously (▶ Fig. 9.4). It is clinically and histologically difficult to distinguish from SCC, though is considered a low-grade variant of SCC.

9.3.3 Malignant Melanoma

Melanoma arises from melanocytes, the dendritic cells in the basal layer of the skin. It is most commonly cutaneous in origin, though it can be present on the mucosal surfaces, uveal tract of the eye, leptomeninges, and in lymph node capsules.[1] This chapter will focus on the cutaneous form. Melanomas typically proliferate in a radial fashion in the epidermis (melanoma in situ, M*is*) before shifting to vertical growth to the dermis and underlying tissue layers (▶ Fig. 9.5). Downward growth suggests invasion and carries a worse prognosis; however, depending on the subset of melanoma, this can be a slow progression. There are four commonly recognized clinical and histologic growth patterns.

Superficial spreading melanoma (SSM) accounts for the majority of melanomas (60–70%) and most often affects those with fair skin and increased sun exposure. It presents as a brown-to-black macule with variegate pigmentation and irregular borders, most frequently on the trunk and legs of middle-aged men and women, respectively (▶ Fig. 9.6a). [1] Approximately half of these lesions arise from a pre-existing nevus.[10] SSM extends horizontally in the superficial epidermal or papillary dermal layers and proceeds slowly before a more rapid, vertical growth phase. In many of these lesions, partial regression may occur as a result of attack by the host's immune system, evident as areas of hypo- or depigmentation.

Nodular melanoma (NM) is the second most common subtype, representing approximately 15 to 30% of melanomas.[1] Unlike SSM, this lesion can develop rapidly (over months) as it is thought to lack a horizontal growth phase.[1] To the examiner, it can appear as a blue-to-brown/black, or sometimes pink-to-red, smooth nodule (▶ Fig. 9.6b). Alternatively, it may be ulcerated or bleeding (▶ Fig. 9.6c). NM is most commonly diagnosed in fair-skinned men in their sixth decade of life.[1] It confers a worse prognosis than SSM.

Lentigo maligna (LM), also known as a Hutchinson melanotic freckle, is a less common, slow-growing M*is* that develops in

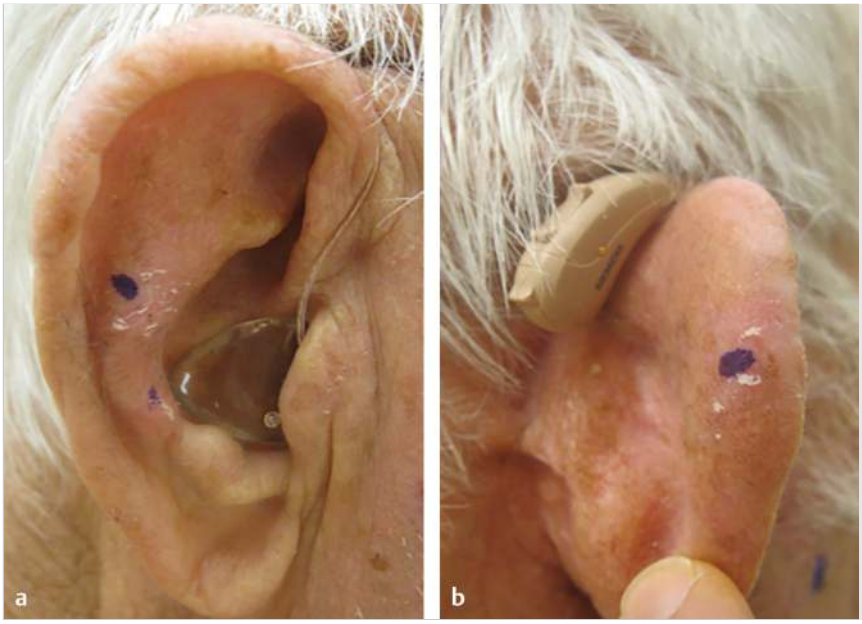


Fig. 9.3 Actinic keratosis of the (**a**) antihelix and (**b**) posterior pinna. Photo courtesy of Alex Ortega, MD, Department of Dermatology, Virginia Commonwealth University.

Fig. 9.4 Keratoacanthoma of the ear. Note the large, ulcerated nodule with a central keratin plug.

Fig. 9.5 Melanoma in situ of the back. Photo courtesy of Alex Ortega, MD, Department of Dermatology, Virginia Commonwealth University.

fair-skinned, older individuals in the seventh decade of life. It presents as a brown-black macule, although may also be hypopigmented, with asymmetric borders in areas of chronic, solar-damage (e.g., face, arms). Approximately 5% of LM progress to invasive growth as LM melanoma (▶ Fig. 9.6d).[11]

Acral lentiginous melanoma (ALM) is a genetically distinct subtype of melanoma responsible for a small fraction of melanoma in Caucasians (5%), but a disproportionate amount in African Americans (up to 70%) and Asians (45%). It arises on the palmar and plantar aspects of the hands and feet or as a subungual line of pigment (melanonychia, Hutchinson sign). Given the likelihood of misdiagnosis or delay in biopsy, these lesions are typically identified at an advanced stage.[12]

A variety of risk factors for developing melanoma, both host and environment-specific, have been identified, many of which have roles in the development of nonmelanoma skin cancer (NMSC), as described above (▶ Table 9.1). Host factors are nonmodifiable genetic or phenotypic characteristics, such as having *CDKN2A*, a high-penetrance susceptibility gene locus for familial melanoma.[13] Environmental factors, on the other hand, are

Fig. 9.6 Cutaneous melanoma. (**a**) Superficial spreading melanoma demonstrating a brown-to-black macule with variegate pigmentation and irregular borders. (**b**) Nodular melanoma demonstrating a blue-to-brown/black, smooth nodule. (**c**) Nodular melanoma demonstrating ulceration and bleeding. (**d**) Lentigo maligna melanoma demonstrating a brown-black macule with asymmetric borders. Photos courtesy of Alex Ortega, MD, Department of Dermatology, Virginia Commonwealth University; Naomi Lawrence, MD, Department of Dermatology, Cooper University Hospital.

Table 9.1 Risk factors for developing melanoma

Nonmodifiable host factors	Modifiable environmental factors
Fair features (Fitzpatrick type I–II skin, light eyes, light hair, ephilides)	Intermittent, intense UVA or UVB exposure (including PUVA)[a]
Numerous congenital, giant, or atypical nevi; solar lentigines	One or more blistering sunburns early in life or > 5 at any age
Family or personal history of melanoma or other skin cancers	Tanning beds, sun lamps
Weakened immune system	Iatrogenic immunosuppression
Advanced age	Chronic sun exposure
Male gender (after age 45)	
DNA repair defects (e.g., Xeroderma pigmentosum)	

[a]PUVA: psoralen and ultraviolet A therapy.

modifiable and are therefore areas of focus for prevention. Sunscreen use, for example, has been demonstrated to decrease the risk of cutaneous melanoma and the use of hats, non-transparent clothing, and shade coverings to block UV radiation are highly encouraged.[14] Ultimately, a patient's overall risk is dictated by the interaction between the host and environmental factors.

9.4 Preparation for Surgery

Evaluating a patient with a concerning cutaneous lesion should start with a thorough history and physical examination. History gathering should include assessment for risk factors/exposures, systemic symptoms such as weight loss and fatigue, and details of the lesion(s) (e.g., duration the lesion has been present, rate of growth, and prior treatment modalities). Particular vigilance should be used in evaluating solid-organ transplant recipients as these patients require more timely treatment.[15] Paresthesias and/or local weakness should also be assessed to rule out perineural invasion. Physical examination should include a thorough skin assessment, addressing the lesion's size, defining characteristics, depth (superficial versus deep), and evidence of prior treatment (i.e., scarring or hypopigmentation from prior cryotherapy). Examination should also assess the local nerve function, lymph node basins, and mucosal surfaces. For melanoma detection in particular, both patients and providers are instructed to use the simple, yet effective, ABCDE tool: asymmetry, border irregularity, color variegation, diameter greater than 6 mm, and evolution of the lesion with time.[16] Other strategies include looking for the "ugly duckling" or single striking lesion unlike surrounding benign lesions and the "Little Red Riding Hood" sign (erythema/inflammation around the lesion). Dermoscopy supplements the skin examination by detecting subtle lesions and improving diagnostic accuracy.[17] Baseline photographs are also recommended to detect new or changing lesions.

Concerning lesions should be biopsied as early detection is key and histologic subtype and features will impact risk stratification and ultimately treatment options. For NMSC, a shave biopsy is most commonly performed and yields pathologic diagnosis with minimal morbidity or disruption of existing architecture. If there is concern for a deep lesion, a punch biopsy should be performed with inclusion of the deep reticular dermis.[18] One should be mindful that complete excision of a lesion via biopsy, if not intended as definitive treatment, may obscure margins of the native lesion with scar tissue. This may ultimately lead to the unnecessary excision of additional tissue, which can be problematic in cosmetically sensitive areas. Imaging studies, such as MRI and CT, are warranted in patients with extensive disease (e.g. bone involvement, perineural invasion, deep soft tissue involvement), with the former better defining perineural disease (55).

An *excisional* biopsy should be completed for lesions concerning for melanoma using 1 to 3 mm margins.[1,19] Shave biopsies in this form of malignancy should be avoided. In cosmetically sensitive or technically challenging areas such as the face, incisional or punch biopsies may be considered. All specimens should be sent for permanent section evaluation as frozen sectioning can obscure the diagnosis or depth of the lesion.[20] From the histologic examination of melanoma, it is essential to determine a lesion's depth (in millimeters) from the most superficial cell layer at the epidermis or ulcer to the deepest point of tumor penetration. This concept, introduced in 1970 by Alexander Breslow, replaced the less-accurate Clark level of invasion and is still in use today as the most important local prognostic factor.[21,22] It is also important to determine the presence of ulceration and mitotic rate (mitoses/mm^2) as these also contribute to prognostic evaluation.[23] Immunohistochemical and molecular profiling studies can help in diagnosis, prognosis, and distinguishing benign from malignant melanoma.[1,23,24]

Preoperative assessment of patients with melanoma often includes imaging (radiographs, CT, PET/CT, MRI, and bone scan) to evaluate specific signs or symptoms in those with stage I or II disease, or to establish a baseline in those with stage III/IV disease.[19] A complete blood count, liver function tests, serum lactate dehydrogenase, alkaline phosphatase, and creatinine should be obtained for more advanced stages of disease.[25] Regional lymph node metastases are best demonstrated by ultrasound, while distant metastases are best demonstrated by PET-CT.[26,27]

9.4.1 Lymph Node Evaluation

As SCC metastasizes preferentially to lymph nodes, it is important to perform a biopsy of clinically significant lymphadenopathy. Palpable regional nodes or abnormal nodes noted on imaging should undergo FNA or core biopsy. Biopsy-proven positive lymph nodes require appropriate complete lymphadenectomy or radiation therapy. There has been interest in performing sentinel lymph node biopsy (SLNB) to detect subclinical nodal metastasis and assist in staging for high-risk lesions; however, no large randomized control studies have been carried out to demonstrate clinical benefit; therefore, routine SLNB is not currently recommended.[28,29]

In patients with cutaneous melanoma, nodal status is a strong prognostic factor for recurrence and survival.[25,30] Although elective lymph node dissection was once recommended for all patients with intermediate to high-risk tumors, the current standard of care for evaluation of a lymph node basin to detect subclinical nodal metastases is an SLNB by lymphoscintigraphy followed by intraoperative injection of a blue dye. When used together, lymphoscintigraphy and the blue dye method can detect up to 95% of positive nodes.[31,32] Given the likelihood of nodal involvement despite clinically negative basins or negative radiographs, SLNB should be considered for those with Breslow depth < 0.8 mm with ulceration or depth 0.8-1.0 mm without ulceration. It is also is recommended for those with tumors greater than 1 mm thick, lymphovascular invasion, younger than 40 years, significant vertical growth phase, increased mitotic rate, or larger than 4.00 mm tumors.[19,23,33,34] Wide local excision plus SLNB has improved disease-free survival and decreased regional recurrence in particular patient populations.[35,36] The role of SLNB in head and neck melanoma is more controversial as this region has a complex drainage pattern and therefore may be more prone to complication. Numerous studies demonstrate improved disease-free and distant metastases survival, reduced lymph node metastases, and better overall survival in head and neck melanoma.[37,38,39,40] Ultimately, given an overall lack of consensus in recommendations, the decision for SLNB should be up to the individual patient and clinician.

For patients with melanoma, a positive SLNB, in which micrometastases are identified, currently warrants a complete regional lymph node dissection (CLND).[19] CLND is also recommended in patients with stage III disease, detectable lymph nodes by clinical examination or imaging, or for those with positive fine needle aspiration results.[19,25,36] A critical appraisal of the multicenter selective lymphadenectomy trial-I by Sladden et al and several other studies question the all-together utility of SLNB and/or CLND, particularly in thin melanomas, given the possible morbidities.[19,36,41,42] Lymphedema can result in 3 to 7% of those status post SLNB and 30 to 60% of those status post CLND.[1] The concern that SLNB can increase the risk of in-transit metastasis has been refuted.[34,36]

9.4.2 Risk Stratification and Staging

The American Joint Committee on Cancer (AJCC) established guidelines categorizing BCC into low and high risk based on clinical and pathologic findings and their subsequent propensity for recurrence (▶ Table 9.2).[43]Low-risk lesions are often well-defined, nodular, or superficial BCC with no perineural involvement and measure less than 2 cm on the trunk/extremities, 6 mm on the

Table 9.2 BCC and SCC risk stratification and risk of recurrence

Lesion characteristics	Low risk	High risk
Location[a]		
• "Mask" areas of face, chin, mandible, temple, ear, pre/postauricular, hands, feet, genitalia		All lesions in this area are considered high risk, however one must account for significant anatomic/functional considerations. Consider Mohs for lesions > 6 mm.
• Cheeks, forehead, scalp, neck, pretibia	< 10 mm	≥ 10 mm
• Trunk, extremities (except hands, feet, and pretibia)	< 20 mm	≥ 20 mm
Borders	Well-defined	Poorly defined
Primary vs. recurrent	Primary	Recurrent
Immunosuppression	No	Yes
Site of prior radiation therapy/chronic inflammation	No	Yes
Histopathology subtype (BCC only)	Nodular, superficial	Aggressive growth pattern[b]
Perineural involvement or neurologic symptoms	No	Yes

[a]Lesions may be high risk independent of size, based on location alone.
[b]Morpheaform, sclerosing, mixed infiltrative, or micronodular features.
Adapted from Edge, Stephen B, Byrd DR, Compton C, et al. AJCC cancer staging manual. 7th ed. New York: Springer; 2010. (55)

"mask" areas of the face, and 1 cm on the remainder of the head and neck. In contrast, high-risk lesions are often morpheaform or infiltrative with poorly defined margins and perineural involvement. These lesions are often much larger in size.

In addition to the factors listed in Table 9.2, SCC lesions that are well or moderately differentiated, less than or equal to 6 mm in depth, and lack lymphatic or vascular involvement, are considered "low risk" for local recurrence of metastasis. High risk lesions are poorly differentiated, greater than 6 mm in depth, may involve lymphatics or vascular structures, and are over 2 mm thick. The AJCC introduced a TNM staging system for SCC to provide better prognostic outcomes (▶ Table 9.3).[43,44]

Table 9.3 Staging of cutaneous squamous cell carcinoma

T Classification	**Tumor size**		
Tis	Carcinoma in situ		
T1	Tumors < 2 cm in greatest dimension		
T2	Tumors ≥ 2 cm < 4 cm in greatest dimension		
T3	Tumor with minor bone erosion, perineural invasion, or deep invasion (> 6 mm or beyond subcutaneous fat)		
T4	Tumor with invasion of a) gross cortical bone or marrow; b) skull base		
N classification*	**Nodal metastatic burden**		
N0	No regional lymph node metastasis		
N1	Metastasis in single ipsilateral lymph node, ≤ 3 cm in greatest dimension, no extranodal extension (ENE)		
N2a	Metastasis in single ipsilateral lymph node, > 3 cm but ≤ 6 cm in greatest dimension, no ENE		
N2b	Metastasis in multiple ipsilateral lymph nodes, ≤ 6 cm in greatest dimension, no ENE		
N2c	Metastasis in bilateral or contralateral lymph nodes, ≤ 6 cm in greatest dimension, no ENE		
N3	Metastasis in lymph node, > 6 cm in greatest dimension, a) no ENE; b) + ENE		
M classification	**Site**		
M0	No distant metastasis		
M1	Distant metastasis present		
*Clinical staging, not pathological. Extranodal extension (ENE) status is the primary difference.			
Anatomic stage/Prognostic groups			
Stage 0	Tis	N0	M0
Stage I	T1	N0	N0
Stage II	T2	N0	M0
Stage III	T3	N0	M0
	T1	N1	M0
	T2	N1	M0
	T3	N1	M0
Stage IV	T1	N2	M0
	T2	N2	M0
	T3	N2	M0
	T Any	N3	M0
	T4	N Any	M0
	T Any	N Any	M1

Adapted from Edge, Stephen B, Byrd DR, Compton C et al. AJCC cancer staging manual. 7th ed. New York: Springer; 2010. Updated based on NCCN Guidelines and AJCC 2017 8th edition updates.

The 7th edition was revised to focus on head and neck SCC in the 8th edition. These high-risk features not only influence staging, but also surgical treatment.

In 2001, the AJCC released a TNM-based melanoma staging system that was subsequently revised in 2009 to include ulceration, mitotic rate (mitoses per mm^2), and updated tumor thickness strata, with stages 1 and 2 representing localized disease, stage 3 representing regional disease, and stage 4 representing distant metastatic disease (▶ Table 9.4).[23,43,45] Staging systems will continue to evolve with the production of more evidence-based medicine in this field.

Predictions regarding prognosis for patients with melanoma are made using Breslow thickness, ulceration, mitotic rate, and nodal status.[46] A predictive, electronic tool was recently created that also includes other prognostic factors such as age, gender, and location of the primary lesion: http://www.melanomaprognosis.org.[47] Future prognosis will perhaps be predicted from molecular profiling and genetic information.[24]

9.4.3 Anticoagulation

In our experience, many patients with cutaneous malignancies are also actively taking anticoagulation medication, whether low-dose daily aspirin as cardiac prophylaxis, long-term anti-platelet therapy after vascular stent placement, or lifelong anticoagulation for arrhythmias or other chronic vascular conditions. Studies have shown that the risk of a severe bleeding complication (as defined by hematoma, flap/graft failure, bleeding requiring a procedure or wound necrosis/dehiscence) following cutaneous surgery is generally less than 1% when performed without anticoagulation.[48,49] This risk increases to 6% in patients taking warfarin within the therapeutic range. Although there is a trend toward increased bleeding risk in patients taking aspirin or nonsteroidal anti-inflammatory drugs (NSAIDs), this is not statistically significant.[50] It is generally considered safe to proceed with low-risk surgery in patients taking only aspirin or NSAIDs, though many practicing surgeons will temporarily hold those medications if it is medically safe. Ultimately, it is important to consult with the prescribing physician regarding the timing and plausibility of cessation of anticoagulants to minimize unwanted bleeding complications while preventing thrombotic events.

9.5 Treatment

With *all* forms of cutaneous malignancy, the goal of therapy should be to eliminate malignancy while preserving anatomic form and function. There are numerous modalities that may be employed to treat NMSC. Selection of the optimal treatment depends on the risk for recurrence, patient age, number of lesions, anatomic location of lesion, and prior therapies utilized.[5]

Superficial BCC may be responsive to topical agents such as 5-fluorouracil (5-FU), Imiquimod, or photodynamic therapy, and cryotherapy. Such superifical therapies are also often "reserved" for those who cannot undergo surgery or radiation. 5-FU (commercially available as Efudex, Fluoroplex, and Carac) is an antineoplastic pyrimidine analog.[51] It is typically applied once or twice daily for 4 to 6 weeks depending on the formulation. For superficial BCC, studies have shown clearance rates greater than 90%, however this method remains lower than

Table 9.4 Staging of malignant melanoma

T Classification	Tumor thickness (mm)	Ulceration, mitoses
T1	≤1.0	a) < 0.8 mm w/o ulceration
		b) < 0.8 mm w/ ulceration or 0.8-1 mm with or without ulceration
T2	1.01–2.0	a) w/o ulceration
		b) w/ ulceration
T3	2.01–4.0	a) w/o ulceration
		b) w/ ulceration
T4	>4.0	a) w/o ulceration
		b) w/ ulceration
N classification	**Number of metastatic nodes**	
Nx	Cannot be assessed	
N0	No regional metastases detected	
N1	1 node	a) micrometastasis
		b) macrometastasis
N2	2–3 nodes	a) micrometastasis
		b) macrometastasis
N3	≥4 nodes (including intralymphatic metastases)[a]	a) micrometastasis
		b) macrometastasis
M classification	**Site**	
M0	No detectable distant metastasis	
M1a	Metastases to skin, subcutaneous, or distant nodes M1a (0) LDH not elevated; M1a (1) LDH elevated	
M1b	Metastases to lung M1b (0) LDH not elevated; M1b (1) LDH elevated	
M1c & M1d	Metastases to other visceral sites, any distant site, elevated serum LDH	

[a]Intralymphatic metastases includes in transit or satellite metastases.

Anatomic stage/Prognostic groups							
Clinical staging[a]							
Stage 0	Tis	N0	M0	Stage IIB	T3b	N0	M0
Stage IA	T1a	N0	M0		T4a	N0	M0
Stage IB	T1b	N0	M0	Stage IIC	T4b	N0	M0
	T2a	N0	M0				
Stage IIA	T2b	N0	M0	Stage III	Any T	≥N1	M0
	T3a	N0	M0	Stage IV	Any T	Any N	M1

[a]Clinical staging is to be used after complete excision of the primary lesion with clinical assessment for regional and distant metastases. Pathologic staging expands on nodal status, including microstaging and pathologic information about the regional lymph nodes after partial or complete lymphadenectomy. Adapted from Edge, Stephen B, Byrd DR, Compton C et al. AJCC cancer staging manual. 7th ed. New York: Springer; 2010. Updated with NCCN Guidelines, Cutaneous Melanoma, 2018.

surgery.[51] It causes an inflammatory response, including as erythema, erosion, and crusting, which may limit patient compliance. Imiquimod (commercially available as Aldara) is a toll-like receptor agonist that up-regulates a cytokine response.[1] Specific dosing for Imiquimod cream is not unanimous with studies reporting once to twice daily to thrice weekly dosing for 6 to 12 weeks. Dosage should be titrated until the skin becomes irritated and demonstrates mild dermatitis.[51] Imiquimod is postulated to have immunologic memory and thus may continue to be effective even after cessation of use. Both of these agents may also be helpful as a prequel to definitive therapy in helping to elicit margins of poorly defined BCC prior to excision.

Topical therapies have limited indication in SCC. Imiquimod and 5-FU are FDA-approved for use for AK and have shown efficacy against Bowen's disease. A few case reports document effectiveness of Imiquimod against low-risk invasive SCC; however, it should not be considered as a first-line option as its efficacy is not well established.[52]

Liquid nitrogen freeze-thaw cycles may be considered for small, superficial BCC. Its greatest benefit may be in the patient with multiple lesions who does not desire a more invasive procedure. Cryotherapy often results in hypopigmentation and postinflammatory scarring that can make detecting recurrence challenging.[1]

Electrodessication and curettage (ED&C) is another destructive option frequently performed by dermatologists. This modality involves curetting down to normal, firm dermis in three directions followed by electrodesiccation to denature the wound bed. The process is repeated for up to two to three cycles. It is only indicated for superficial NMSC lesions that do not have deep, dermal penetration. The curetted tissue should be sent for permanent pathology to confirm no concerning features warranting further excision however this method will not provide margin assessment. Additionally, if curetting leads to exposure of subcutis, traditional excision should be performed. While 97 to 98% cure rates have been reported, this technique creates a hypopigmented or hypertrophic scar and potentially unsatisfactory

cosmetic result, particularly in hair-bearing areas.[1] ED&C should not be used to treat SCC in areas of terminal hair growth (scalp, groin, axilla, beard) given that SCC can extend deep down the hair follicles to a deeper depth.

Radiation therapy is indicated for NMSC when a patient's medical comorbidities or size/location of the lesion preclude surgical excision. Radiation therapy can also serve as adjuvant therapy for incompletely excised large tumors or tumors at high risk for metastasis. The risk of recurrence is greater than that of the other therapies mentioned.

Mohs micrographic surgery (MMS) has become an increasingly popular option for management of NMSC. MMS allows tissue conservation while ensuring histopathologic clearance. Indications for MMS are tumors greater than 2 cm, recurrent lesions, tumors with poorly defined or indistinct margins, and those that may be in cosmetically sensitive or high-risk areas.[6] Five-year recurrence rates for MMS removal of BCC have been reported to be as low as 1%.[1] MMS is contraindicated when the tumor pathology is difficult to assess under frozen section, such as in multifocal tumors and recurrent tumors arising from a scar.

Surgical excision with appropriate margins remains the most effective therapy for NMSC.[53] For BCC, the NCCN consortium recommends a 4-mm margin for any lesion < 2 cm diameter.[1,18] Intraoperative frozen section assessment should be performed. However for high risk lesions, Mohs is preferred. High-risk BCC lesions require a wider margin of excision often from 6 mm in a cosmetically sensitive area (i.e., eyelid) to 1 cm in more well-tolerated regions (trunk and extremities).[1,54] This achieves a 98% disease-free state over five years (55). Similarly, for low-risk SCC, < 2 cm in diameter, 4 mm margins are recommended while for lesions > 2 cm in diameter, 6 mm margins are recommended. For high risk SCC, lesions < 1 cm diameter, 1-1.9 cm diameter, or > or = 2 cm in diameter require 4 mm, 6 mm, and 9 mm margins, respectively. If closure of the excised defect requires local tissue rearrangement, skin grafting, or free tissue transfer, then definitive clear margins should be obtained prior to reconstructive repair as these modalities may distort the location of the residual malignancy. Routine frozen sections are not needed in well-circumscribed, small (< 1 cm), low-risk lesions or lesions located in noncritical areas where a wide margin can be taken and primary closure is possible. For high-risk lesions, surgical excision with intraoperative frozen section or MMS is recommended.[55] Positive margins from a standard excision should be addressed with re-excision, MMS, or radiation therapy.

A vast majority (90%) of melanomas are diagnosed as primary tumors without metastasis.[56] Full-thickness surgical excision in a fusiform or elliptical pattern, extending through subcutaneous tissue to underlying fascia, is the method of choice for definitive diagnosis and management of these lesions.[20,57] Given that melanoma cells can extend beyond the visible tumor origin, surgical margins, measured from the edge of the lesion or previous biopsy scar, are recommended. Previously, 5-cm margins were advocated regardless of tumor depth or location; however, this practice has been abandoned.[30,58,59] The optimal margin(s) remain unclear; however, numerous studies and expert guidelines support the following[60,61,62,63,64,65,66]:

- M*is:* 0.5-1.0 cm margin.
- Lesions ≤ 1 mm: 1.0 cm margins.
- Lesions 1.01–2 mm: 1.0–2.0 cm margins.
- Lesions ≥ 2 mm: 2.0 cm margins.

For tumors > 4 mm, removal of fascia may be necessary.[20] In cases of advanced disease burden (stage IV) and no or limited metastasis, surgical intervention may provide survival benefit.[67]

In special circumstances, one may need to deviate from the recommended surgical margins. For example, 3 cm margins should be considered for desmoplastic melanoma given its high propensity for recurrence.[57] M*is*, including LM, may extend beyond visible margins and may therefore require staged excision or topical agents.[61] For difficult or aesthetically sensitive anatomic sites, such as at the face or distal extremities, an individualized surgical approach with narrower margins may be considered, including the use of MMS, to minimize tissue loss.[30,65,68] On the face, MMS surgery has demonstrated a similar local recurrence, distant recurrence, and overall survival to that of wide local excision.[69,70] Additionally, it can provide an immediate examination of the surgical margin, which allows for immediate reconstruction.[71] However, MMS is not fully advocated as immediate frozen sections may distort identification of malignant cells.[57] Full-thickness wedge excision is suggested for the ear. For subungual melanoma of the fingers and toes, amputation at the midphalanx is often required. Palmar or plantar lesions require excision down to fascia as with other areas of the body, while dorsal surfaces of the hand and foot require resection down to tendon or bone. Metastatic lesions may be excised for palliative purposes, but have also been demonstrated to improve long-term survival.[72]

Postexcision reconstruction is dependent on the anatomical region and individual surgeon comfort. Primary closure should be considered, if possible, and can often be achieved with undermining. Skin grafts, local flaps, and complex, regional soft tissue rearrangement may be warranted if tension-free primary closure is not feasible, particularly in the head, neck, and extremities, however margin assessment is necessary before closure (▶ Fig. 9.7).[20]

Nonsurgical treatment options, some of which take advantage of melanoma's immunogenic nature, are considered in patients who are not amenable to surgery, lesions in cosmetically sensitive areas, or who have unresectable disease (e.g., satellite or in-transit metastases, distant metastases). Options include cytotoxic regimens and biochemotherapy (interleukin-2, IFN-alfa), immunotherapy (monoclonal antibodies), targeted therapy (e.g., BRAF inhibitors), isolated limb perfusion or infusion, cryotherapy, laser therapy, intralesional injections, topical agents (Imiquimod), electrochemotherapy, and radiotherapy. While some of these modalities have activity against melanoma, most unfortunately have not shown a significant improvement in survival.[19,56,73,74,75,76] In the case of radiation therapy, it may be most useful as an adjuvant treatment for palliative purposes or in those with brain, soft tissue, or bony metastases. These nonsurgical options are an area of ongoing innovation and active research.

9.6 Postoperative Care

One should ensure that final pathology demonstrates complete clearance of the lesion. Postoperative care is unique to the extent of the excision and the degree of reconstruction required. Patients may be discharged the day of surgery (primary closure, skin grafts, local tissue rearrangement) or may require hospital admission for closer monitoring (larger/regional flaps, free flaps). Typically, dressings are removed postoperative day 2, and the

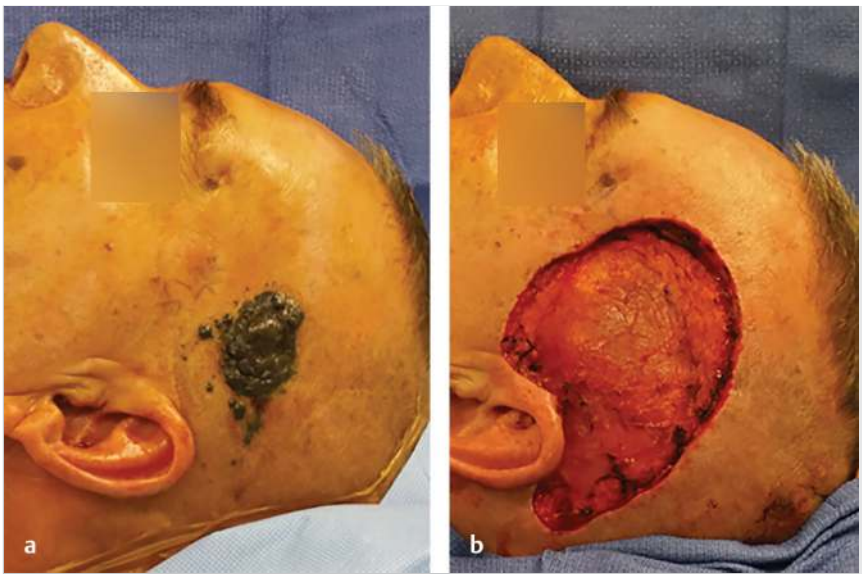


Fig. 9.7 T4 superficial spreading melanoma pre (**a**) and post (**b**) surgical excision with 2 cm margins along the temporal aspect. Reconstruction was completed with Integra and split-thickness skin grafting.

wound is inspected for signs of infection, hematoma, or seroma. Postoperative antibiotics are not routinely prescribed. Sutures are removed in 3 to 5 days for head and neck wounds and 7 to 10 days for wounds of the trunk or extremities.

After discharge, postoperative monitoring is dictated by the type and stage/severity of the diagnosed malignancy. For NMSC, patients should be monitored with full skin assessment every 3 to 6 months for the first year following diagnosis and then every 6 to 12 months thereafter based on the risk to ensure no recurrence or new lesions concerning for malignancy. In addition to plastic surgeons, dermatologists, surgical oncologists, and medical oncologists are often involved in the management and follow-up of cutaneous malignancies. Patients with more advanced disease are often followed in a multidisciplinary fashion.

Unfortunately, there are no collectively accepted guidelines for melanoma follow-up.[19,30] In general, patients with stage I and IIa lesions should be followed up every 3 months for 1 year, every 6 months for the next 5 years, and annually thereafter. Stage IIb and greater may also need imaging (US, CT, PET/CT) every 6 to 12 months and annual brain MRI for 5 years.[27] Alternatively, some experts recommend dermatologic examinations one to four times per year for 2 years, followed by visits every 6 to 12 months for life.[1]

9.7 Outcomes

Surgical excision of NMSC can achieve cure rates above 90%. Patients with stage IA melanoma have 5- and 10-year survival rates of 97 and 95%, respectively. However, these rates are inversely proportional to stage, with stage IV patients having only 15 and 10% 5- and 10-year survival rates.[43] Unfortunately, survival rates for melanoma have not increased at the rate anticipated given the many new treatment options and increased vigilance in diagnosis.

Patients with prior NMSC are at high-risk for developing further cutaneous malignancies. The 5-year risk of developing another NMSC is approximately 30 to 50%.[77] Additionally, patients with prior NMSC are also at increased risk for developing a melanoma.[78] Melanoma has been demonstrated to recur even over a decade after treatment; however, the vast majority recur within 5 years of diagnosis, with an overall 4% risk of recurrence.[1,57,62] Not surprisingly, recurrence confers a worse prognosis.[62]

For NMSC, local recurrence can be treated in the same fashion as the primary lesion. Regional recurrence should be treated by surgical excision or radiation, whereas distant metastases may require evaluation in a multidisciplinary tumor board or clinical trials.[4,55] For melanoma, local scar recurrence is confirmed by biopsy. Subsequent re-excision proceeds as would that of a primary tumor. Local, satellite, or in-transit recurrence is confirmed by FNA or biopsy. Baseline imaging is also often needed. Surgical excision with negative margins is recommended when possible, with or without SLNB. For unresectable disease, hypothermic limb perfusion/infusion, intralesional injections, topical Imiquimod, laser ablation, radiation, or clinical trials may be considered. CLND is recommended for those with regional nodal recurrence. Adjuvant therapy includes high-dose pegylated IFN-alfa and radiation. Distal recurrence is treated in the same fashion as primary stage IV disease.[19]

Despite important advances and increased knowledge of cutaneous malignancies, these devastating diseases remain a significant health problem and warrant advocacy for prevention and diligent management.

9.8 Review Questions

9.8.1 True or False

1. Particular vigilance in diagnosis is warranted in patients with immunocompromise or a solid-organ transplant.
2. Sentinel lymph node biopsy is recommended for all stages of melanoma.

9.8.2 Fill in the Correct Answer

3. What are the modifiable risk factors for the development of cutaneous malignancies?
4. What is the most common type of basal cell carcinoma? Of malignant melanoma?
5. What are the recommended surgical margins for low-risk NMSC?

9.8.3 Answers

1. True. Patients who have undergone solid-organ transplant or who are immunocompromised are at higher risk for developing a cutaneous malignancy than the general population.
2. False. Sentinel lymph node biopsy is recommended for those with tumors > 1 mm thick, lymphovascular invasion, younger than 40 years of age, significant vertical growth phase, high mitotic rate, or larger than 4 mm. SLNB should be discussed with those with tumors < 0.8 mm Breslow depth with ulceration or 0.8-1 mm depth with or without ulceration.
3. Basal cell carcinoma, squamous cell carcinoma, and malignant melanoma share similar modifiable risk factors, including chronic sun exposure, intense, intermittent UV light exposure, one or more blistering sunburns early in life or more than five at any age, chemical carcinogens, use of tanning beds and sun lamps, ionizing radiation, and iatrogenic immunosuppression.
4. Nodular is the most common type of BCC. It is well circumscribed and often recognized as a pearly, flesh-colored papule with a raised border, central dell, ulceration, and arborizing telangiectasias, visible by dermoscopy. Superficial spreading is the most common type of melanoma, accounting for 60 to 70% of all melanomas. It commonly affects those with fair skin and increased sun exposure, appearing as a brown-to-black macule with variegate pigmentation and irregular borders.
5. Surgical excision with appropriate margins remains the most effective therapy for NMSC. For low-risk BCC and SCC, 4 mm and 4 to 6 mm margins are recommended, respectively.

References

[1] Bolognia JL, Jorizzo JL, Schaffer JV, eds. Dermatology. 3rd ed. Philadelphia: Elsevier Ltd; 2012
[2] American Cancer Society. Cancer Facts & Figures. 2018
[3] Asgari MM, Moffet HH, Ray GT, Quesenberry CP. Trends in basal cell carcinoma incidence and identification of high-risk subgroups, 1998–2012. JAMA Dermatol. 151(9):976–981
[4] NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines). Basal Cell Skin Cancer. National Comprehensive Cancer Network; 2018
[5] Netscher DT, Spira M. Basal cell carcinoma: an overview of tumor biology and treatment. Plast Reconstr Surg. 113(5):74e–94e
[6] Netscher DT, Leong M, Orengo I, Yang D, Berg C, Krishnan B. Cutaneous malignancies: melanoma and nonmelanoma types. Plast Reconstr Surg. 127(3): 37e–56e
[7] Mittelbronn MA, Mullins DL, Ramos-Caro FA, Flowers FP. Frequency of preexisting actinic keratosis in cutaneous squamous cell carcinoma. Int J Dermatol. 37(9):677–681
[8] Criscione VD, Weinstock MA, Naylor MF, Luque C, Eide MJ, Bingham SF, Department of Veteran Affairs Topical Tretinoin Chemoprevention Trial Group. Actinic keratoses: natural history and risk of malignant transformation in the Veterans Affairs Topical Tretinoin Chemoprevention Trial. Cancer. 115(11):2523–2530
[9] Glogau RG. The risk of progression to invasive disease. J Am Acad Dermatol. 42(1, Pt 2):23–24
[10] Skender-Kalnenas TM, English DR, Heenan PJ. Benign melanocytic lesions: risk markers or precursors of cutaneous melanoma? J Am Acad Dermatol. 33 (6):1000–1007
[11] Weinstock MA, Sober AJ. The risk of progression of lentigo maligna to lentigo maligna melanoma. Br J Dermatol. 116(3):303–310
[12] Metzger S, Ellwanger U, Stroebel W, Schiebel U, Rassner G, Fierlbeck G. Extent and consequences of physician delay in the diagnosis of acral melanoma. Melanoma Res. 8(2):181–186
[13] Goldstein AM, Chan M, Harland M, et al. Lund Melanoma Study Group, Melanoma Genetics Consortium (GenoMEL). Features associated with germline CDKN2A mutations: a GenoMEL study of melanoma-prone families from three continents. J Med Genet. 44(2):99–106
[14] Green AC, Williams GM, Logan V, Strutton GM. Reduced melanoma after regular sunscreen use: randomized trial follow-up. J Clin Oncol. 29(3):257–263
[15] Euvrard S, Kanitakis J, Claudy A. Skin cancers after organ transplantation. N Engl J Med. 348(17):1681–1691
[16] Abbasi NR, Shaw HM, Rigel DS, et al. Early diagnosis of cutaneous melanoma: revisiting the ABCD criteria. JAMA. 292(22):2771–2776
[17] Kittler H, Pehamberger H, Wolff K, Binder M. Diagnostic accuracy of dermoscopy. Lancet Oncol. 3(3):159–165
[18] National Comprehensive Cancer Network (NCCN) Guidelines, 2015
[19] NCCN Clinical Practice Guidelines in Oncology. Melanoma. National Comprehensive Cancer Network; 2015:1–75
[20] Kimbrough CW, McMasters KM, Davis EG. Principles of surgical treatment of malignant melanoma. Surg Clin North Am. 94(5):973–988, vii
[21] Breslow A. Thickness, cross-sectional areas and depth of invasion in the prognosis of cutaneous melanoma. Ann Surg. 172(5):902–908
[22] Balch CM, Buzaid AC, Soong SJ, et al. Final version of the American Joint Committee on Cancer staging system for cutaneous melanoma. J Clin Oncol. 19 (16):3635–3648
[23] Compton CEA. Cancer Staging Atlas: A Companion to the Seventh Editions of the AJCC Cancer Staging Manual and Handbook; 2012:23–32
[24] Dickson PV, Gershenwald JE. Staging and prognosis of cutaneous melanoma. Surg Oncol Clin N Am. 20(1):1–17
[25] Diller ML, Martin BM, Delman KA. Lymph node dissection for stage III melanoma. Surg Oncol Clin N Am. 24(2):261–277
[26] Ceradini DJ, Blechman KM. Dermatology for Plastic Surgeons II—Cutaneous Malignancies. In: Thorne C, Chung K, Gosain A, Gurtner G, Mehrara B, Rubin J, et al., editors. Grabb and Smith's Plastic Surgery. 7th ed. Lippincott Williams & Wilkins; 2014
[27] Xing Y, Bronstein Y, Ross MI, et al. Contemporary diagnostic imaging modalities for the staging and surveillance of melanoma patients: a meta-analysis. J Natl Cancer Inst. 103(2):129–142
[28] Allen JE, Stolle LB. Utility of sentinel node biopsy in patients with high-risk cutaneous squamous cell carcinoma. Eur J Surg Oncol. 41(2):197–200
[29] Ahmed MM, Moore BA, Schmalbach CE. Utility of head and neck cutaneous squamous cell carcinoma sentinel node biopsy: a systematic review. Otolaryngol Head Neck Surg.
[30] Leiter U, Eigentler TK, Forschner A, et al. Excision guidelines and follow-up strategies in cutaneous melanoma: facts and controversies. Clin Dermatol. 28 (3):311–315
[31] Thompson JF, Shaw HM. Sentinel node mapping for melanoma: results of trials and current applications. Surg Oncol Clin N Am. 16(1):35–54
[32] Landi G, Polverelli M, Moscatelli G, et al. Sentinel lymph node biopsy in patients with primary cutaneous melanoma: study of 455 cases. J Eur Acad Dermatol Venereol. 14(1):35–45
[33] Kirkland EB, Zitelli JA. Mitotic rate for thin melanomas. Dermatol Surg. 40(9): 937–945
[34] Ross MI. Sentinel node biopsy for melanoma: an update after two decades of experience. Semin Cutan Med Surg. 29(4):238–248
[35] van der Ploeg APT, Haydu LE, Spillane AJ, et al. Outcome following sentinel node biopsy plus wide local excision versus wide local excision only for primary cutaneous melanoma: analysis of 5840 patients treated at a single institution. Ann Surg. 260(1):149
[36] Morton DL, Thompson JF, Cochran AJ, et al. MSLT Group. Final trial report of sentinel-node biopsy versus nodal observation in melanoma. N Engl J Med. 370(7):599–609
[37] Leiter U, Eigentler TK, Häfner H-M, et al. Sentinel lymph node dissection in head and neck melanoma has prognostic impact on disease-free and overall survival. ann surg oncol.
[38] Patel SG, Coit DG, Shaha AR, et al. Sentinel lymph node biopsy for cutaneous head and neck melanomas. Arch Otolaryngol Head Neck Surg. 128(3):285–291
[39] Schmalbach CE, Bradford CR. Is sentinel lymph node biopsy the standard of care for cutaneous head and neck melanoma? Laryngoscope. 125(1): 153–160
[40] Dwojak S, Emerick KS. Sentinel lymph node biopsy for cutaneous head and neck malignancies. Expert Rev Anticancer Ther. 15(3):305–315
[41] Nagaraja V, Eslick GD. Is complete lymph node dissection after a positive sentinel lymph node biopsy for cutaneous melanoma always necessary? A meta-analysis. Eur J Surg Oncol. 39(7):669–680

[42] Sladden M, Zagarella S, Popescu C, Bigby M. No survival benefit for patients with melanoma undergoing sentinel lymph node biopsy: critical appraisal of the Multicenter Selective Lymphadenectomy Trial-I final report. Br J Dermatol. 172(3):566–571
[43] Edge S, Byrd D, Compton C, Eds. AJCC Cancer Staging Manual. 8th ed. New York, NY: Springer; 2017
[44] Farasat S, Yu SS, Neel VA, et al. A new American Joint Committee on Cancer staging system for cutaneous squamous cell carcinoma: creation and rationale for inclusion of tumor (T) characteristics. J Am Acad Dermatol. 64(6): 1051–1059
[45] Balch CM, Gershenwald JE, Soong SJ, et al. Final version of 2009 AJCC melanoma staging and classification. J Clin Oncol. 27(36):6199–6206
[46] Mandalà M, Massi D. Tissue prognostic biomarkers in primary cutaneous melanoma. Virchows Arch. 464(3):265–281
[47] Soong SJ, Ding S, Coit D, et al. AJCC Melanoma Task Force. Predicting survival outcome of localized melanoma: an electronic prediction tool based on the AJCC Melanoma Database. Ann Surg Oncol. 17(8):2006–2014
[48] Otley CC, Fewkes JL, Frank W, Olbricht SM. Complications of cutaneous surgery in patients who are taking warfarin, aspirin, or nonsteroidal anti-inflammatory drugs. Arch Dermatol. 132(2):161–166
[49] Dixon AJ, Dixon MP, Dixon JB. Bleeding complications in skin cancer surgery are associated with warfarin but not aspirin therapy. Br J Surg. 94(11):1356–1360
[50] Lewis KG, Dufresne RG , Jr. A meta-analysis of complications attributed to anticoagulation among patients following cutaneous surgery. Dermatol Surg. 34(2):160–164, discussion 164–165
[51] McGillis ST, Fein H. Topical treatment strategies for non-melanoma skin cancer and precursor lesions. Semin Cutan Med Surg. 23(3):174–183
[52] Martín-García RF. Imiquimod: an effective alternative for the treatment of invasive cutaneous squamous cell carcinoma. Dermatol Surg. 31(3):371–374
[53] Bath-Hextall F, Bong J, Perkins W, Williams H. Interventions for basal cell carcinoma of the skin: systematic review. BMJ. 329(7468):705
[54] Rogers-Vizena CR, Lalonde DH, Menick FJ, Bentz ML. Surgical treatment and reconstruction of nonmelanoma facial skin cancers. Plast Reconstr Surg. 135 (5):895e–908e
[55] National Comprehensive Cancer Network (NCCN) Guidelines, 2018
[56] Garbe C, Peris K, Hauschild A, et al. European Dermatology Forum, European Association of Dermato-Oncology, European Organization of Research and Treatment of Cancer. Diagnosis and treatment of melanoma. European consensus-based interdisciplinary guideline–Update 2012. Eur J Cancer. 48(15): 2375–2390
[57] Dzwierzynski WW. Managing malignant melanoma. Plast Reconstr Surg. 132 (3):446e–460e
[58] Cohn-Cedermark G, Rutqvist LE, Andersson R, et al. Long term results of a randomized study by the Swedish Melanoma Study Group on 2-cm versus 5-cm resection margins for patients with cutaneous melanoma with a tumor thickness of 0.8–2.0 mm. Cancer. 89(7):1495–1501
[59] Khayat D, Rixe O, Martin G, et al. French Group of Research on Malignant Melanoma. Surgical margins in cutaneous melanoma (2 cm versus 5 cm for lesions measuring less than 2.1-mm thick). Cancer. 97(8):1941–1946
[60] Sladden MJ, Balch C, Barzilai DA, et al. Surgical excision margins for primary cutaneous melanoma. Cochrane 2010:2009–2011
[61] Tzellos T, Kyrgidis A, Mocellin S, et al. Interventions for melanoma in situ, including lentigo maligna (Review). Cochrane Collab 2014
[62] Balch CM, Soong SJ, Smith T, et al. Investigators from the Intergroup Melanoma Surgical Trial. Long-term results of a prospective surgical trial comparing 2 cm vs. 4 cm excision margins for 740 patients with 1–4 mm melanomas. Ann Surg Oncol. 8(2):101–108
[63] Gillgren P, Drzewiecki KT, Niin M, et al. 2-cm versus 4-cm surgical excision margins for primary cutaneous melanoma thicker than 2 mm: a randomised, multicentre trial. Lancet. 378(9803):1635–1642
[64] Lamboo LG, Haydu LE, Scolyer RA, et al. The optimum excision margin and regional node management for primary cutaneous T3 melanomas (2–4 mm in Thickness): a retrospective study of 1587 patients treated at a single center. Ann Surg. 260(6):1095–1102
[65] Thomas JM, Newton-Bishop J, A'Hern R, et al. United Kingdom Melanoma Study Group, British Association of Plastic Surgeons, Scottish Cancer Therapy Network. Excision margins in high-risk malignant melanoma. N Engl J Med. 350(8):757–766
[66] Haigh PI, DiFronzo LA, McCready DR. Optimal excision margins for primary cutaneous melanoma: a systematic review and meta-analysis. Can J Surg. 46 (6):419–426
[67] Hussussian CJ. Surgical treatment of advanced melanoma. Clin Plast Surg. 37 (1):161–168
[68] Hou JL, Reed KB, Knudson RM, et al. Five-year outcomes of wide excision and Mohs micrographic surgery for Primary Lentigo Maligna in an Academic Practice Cohort. Dermatol Surg. 41(2):211–268
[69] Chin-Lenn L, Murynka T, McKinnon JG, Arlette JP. Comparison of outcomes for malignant melanoma of the face treated using Mohs micrographic surgery and wide local excision. Dermatol Surg. 39(11):1637–1645
[70] Then SY, Malhotra R, Barlow R, et al. Early cure rates with narrow-margin slow-Mohs surgery for periocular malignant melanoma. Dermatol Surg. 35 (1):17–23
[71] Etzkorn JR, Sobanko JF, Elenitsas R, et al. Low recurrence rates for in situ and invasive melanomas using Mohs micrographic surgery with melanoma antigen recognized by T cells 1 (MART-1) immunostaining: tissue processing methodology to optimize pathologic staging and margin assessment. J Am Acad Dermatol. 72(5):840–850
[72] Young SE, Martinez SR, Essner R. The role of surgery in treatment of stage IV melanoma. J Surg Oncol. 94(4):344–351
[73] Eggermont AM, Spatz A, Robert C. Cutaneous melanoma. Lancet. 383(9919): 816–827
[74] Verma S, Quirt I, McCready D, Bak K, Charette M, Iscoe N. Systematic review of systemic adjuvant therapy for patients at high risk for recurrent melanoma. Cancer. 106(7):1431–1442
[75] Ji Z, Flaherty KT, Tsao H. Molecular therapeutic approaches to melanoma. Mol Aspects Med. 31(2):194–204
[76] Schild SE. Role of radiation therapy in the treatment of melanoma. Expert Rev Anticancer Ther. 9(5):583–586
[77] Marcil I, Stern RS. Risk of developing a subsequent nonmelanoma skin cancer in patients with a history of nonmelanoma skin cancer: a critical review of the literature and meta-analysis. Arch Dermatol. 136(12):1524–1530
[78] Chen J, Ruczinski I, Jorgensen TJ, et al. Nonmelanoma skin cancer and risk for subsequent malignancy. J Natl Cancer Inst. 100(17):1215–1222

10 Hidradenitis Suppurativa, Necrotizing Fasciitis, and Soft Tissue Infections

J. Blair Summitt, Yee Cheng Low, and Nishant Ganesh Kumar

Abstract

This chapter examines issues germane to the disease of hidradenitis. The nature and presentation of the disease is delineated, and treatments for its various stages are outlined. The life-threatening potential of necrotizing fasciitis and soft tissue infections is also examined, with treatment options listed. In each case, relevant classifications systems are cited in the text and in table format, to facilitate rapid diagnosis, which can lead to a rapid treatment response.

Keywords: hidradenitis suppurative, necrotizing fasciitis, Hurley classification, Sartorius' classification, soft tissue infection

10.1 Hidradenitis Suppurativa

10.1.1 Goals and Objectives

- Understand the proper evaluation of hidradenitis suppurativa.
- Define the disease process and different treatment modalities.
- Understand the available reconstructive options.
- Appreciate perioperative care options to improve patient satisfaction and quality of life.
- Understand the complications of hidradenitis suppurativa and chronic nature of the disease.

10.2 Patient Presentation

Hidradenitis was first identified and described by Velpeau in 1839. Since that time the disease has undergone many changes in its name. Although its diagnosis has been well established for 176 years, its pathophysiology remains elusive. In 1922, Schieferdecker proposed a possible link between "acne inversa" with obstruction of the apocrine sweat glands. In 1956, Pillsbury published the main characteristics of hidradenitis in a dermatological journal and dubbed it the "acne triad," consisting of hidradenitis suppurativa, perifolliculitis capitis abscendens et suffodiens, and acne conglobata. In 1975, Plewig and Kligman modified the "acne triad" by adding another disease into the spectrum, namely, pilonidal sinus, making it the "acne tetrad."

Hidradenitis is often a disease of the young, with onset generally starting in late teens to early 20 s. The disease is chronic in nature and oftentimes characterized by a cluster of abscesses that affect the apocrine gland bearing areas in the axilla, inner thighs, groin, and perineum.[1] The disease will usually "burn out" with time, but most will remain active for years. Although there is no concrete evidence, there are indications that there could be a hereditary and/or autoimmune component to its pathophysiology. It is more common in women, African Americans, and those with a history of acne. Hidradenitis flares are commonly associated with emotional stress, hormonal changes, heat, and humidity.[2,3]

The typical patient is a 20-year old obese/overweight female who presents with recurrent painful abscesses or drainage in the axilla. The physical assessment should focus on the location, area of involvement, and if there is an active disease. If there is an active infection or fluctuance with expression of purulent material, incision and drainage of the abscess may provide symptomatic relief. The lesions usually start as inflammatory papules that develop into pustules that drain and further progress to odiferous draining sinus tracts with eventual scarring, fibrosis, and deformity of the area involved. When patients present, they typically have suffered with the disease for years and often have avoided diagnosis and treatment due to fear of social embarrassment. At this point, complete healing is often not possible without surgical intervention. Some patients have remission of symptoms for months to years at a time, while others may worsen and require multiple operations.[4]

Hidradenitis affects patients with a wide spectrum of clinical severity, so reliable classification systems are in use for direct management of the disease. The two main classification systems are the Hurley classification system and Sartorius system.[5,6] Hurley's staging system is historically the first classification system in place and is still in use today. Patients are divided into three stages, which rely upon the subjective extent of disease present (▶ Table 10.1).

The second system was proposed by Sartorius, who did not think Hurley's system was sufficient to capture the wide clinical spectrum of hidradenitis. This classification system is more complex, but allows better dynamic monitoring of the disease severity. The Sartorius system uses a point system and disease severity is based on four elements (▶ Table 10.2).

Table 10.1 Hurley's classification of hidradenitis suppurativa

Stage	Clinical features
I	Single or multiple abscesses, without sinus tracts or cicatrization
II	Single or multiple recurrent abscesses, with tract formation and cicatrization, widely separated lesions
III	Multiple interconnected abscesses and tracts across entire area, with diffuse or near-diffuse involvement

Table 10.2 Sartorius' classification of hidradenitis suppurativa

Points	Clinical Element
3 points per region involved	Anatomic regions involved
2 points for abscess, 4 points for fistulae, 1 point for scarring, and 1 point for others	Number and type of lesions involved
2 points if < 5 cm, 4 points if > 5 cm and < 10 cm, and 8 points if > 10 cm	Distance between lesions, especially the length between two relevant lesions
0 points if yes and 6 points if no	The presence of normal skin between lesions

In general, Hurley stage I can be treated nonoperatively, whereas stage II is treated both medically and surgically, and stage III is most often surgical. One should be aware that in the management of hidradenitis, surgery and medical management are not mutually exclusive and oftentimes patients will receive a combination. The Sartorius system, however, is more dynamic and useful in the follow up of patients with hidradenitis after receiving different modalities of treatment. There are other proposed classification systems that incorporate the frequency of flare ups, quality of life, and pain scale. They are not addressed in this chapter.

10.3 Preparation for Surgery

Diagnostic data in the preparation for surgery are the same as that of any elective procedure and are dependent upon comorbid conditions, age, medications, and the standard requirements from the surgical center where the procedure is to be performed. In general, patients with hidradenitis are young adults with little or no significant comorbidities, and preoperative workup is usually not extensive other than a type and screen along with a baseline hemoglobin and hematocrit. The most important component for the preparation of surgery is the counseling of the patient with regards to wound healing issues postoperatively and to match the patient's expectations. It is in the author's experience that patients with hidradenitis often have wound healing complications, and if the incision breaks down, wound care with dressing changes is the management option of choice. Furthermore, patients should be made aware that a single procedure is unlikely to succeed in eradicating the disease, and they will frequently require serial excisions and reconstructive procedures. Imaging studies are rarely necessary prior to surgery.

10.4 Treatment

Medical management alone is indicated for Hurley stage I or early stage II, and anything more severe is treated through a multimodal approach. Initial treatment can be started by using sitz baths and topical cleansing agents. Antibiotic use has little proven effectiveness given that most cultures seen in hidradenitis are sterile, but it continues to be in use. The most effective regimen is a combination of rifampin and clindamycin, as described by Gener et al, in a case series of 116 patients. Antibiotics can be helpful when the culture results return with bacterial superinfection.[7]

Other options are hormonal therapy, which includes oral contraceptive pills that contain a high estrogen to progesterone ratio, or antiandrogens, such as cyproterone acetate, which has shown benefit in some studies and in use in Europe.[8] Oral retinoic acids have also been used, especially to reduce inflammation prior to surgery. However, there is no strict consensus or guidelines with regard to the duration and dosage of retinoic acid, and the side effects from this treatment are not without consequences.[9] Finasteride has been used in some cases and results have ranged from complete healing and remissions lasting 8–18 months. Female patients may note breast enlargement.[10]

Additional treatments have attempted to address the etiology of hidradenitis as an autoimmune entity, so steroids have been used in its treatment. Intralesional steroid injections have some efficacy in small localized areas of disease.[11] Tumor necrosis factor-alpha (TNF-alpha) inhibitors like Humira, Enbrel, and Remicade have also been shown to be effective in some studies, but are not currently approved by the Food and Drug Administration (FDA) for its use in the treatment of hidradenitis.[12,13,14] Cyclosporine has also been used for the treatment of severe disease, but its use is limited by its toxicity. Surgical management is indicated for late Hurley stage II or stage III disease (▶ Fig. 10.1). For small, limited areas of fluctuance, incision and drainage are usually sufficient to provide short-term relief, but it has no influence on the progression of the disease. When there are well-formed sinus tracts that are superficial, some surgeons prefer to unroof or marsupialize the tracts.[15] They are usually epithelized, so this approach promotes rapid healing with minimal scarring. However, recurrence rates are high.

Another approach for patients with advanced disease is radical resection of all apocrine gland-bearing areas. This approach creates a large defect and will require either flap reconstruction or split thickness skin grafting. Skin and subcutaneous tissue are excised down to deep fascia with a 1–2 cm margin. Depending on the area excised, recurrence rates are low. Patients

Fig. 10.1 (a, b) Preoperative and postoperative views of patient with axillary hidradenitis treated with excision and primary closure (Hurley type II).

should be counseled about the fact that radical resection only treats the area resected and new disease may develop if any apocrine-bearing tissue remains. Some surgeons advocate using serial excisions to reduce the need for skin grafts.[16]

In general, wound healing of the axilla, breast, and inguinal areas is better than perineal and gluteal regions. Kagan et al, developed an algorithm for surgical resection of hidradenitis by area of involvement.[17] In this study, patients with limited disease of the axilla, breast, and inguinal region were given excision and primary closure, and those with extensive disease were treated with excision and skin grafting or excision alone with closure by secondary intention. Patients with limited disease in the perianal region and buttocks, however, were treated by excision only with healing by secondary intention. Patients with extensive perianal and gluteal involvement were treated with excision and staged grafting.

Other treatment options include radiation therapy for recalcitrant disease, and this has been shown to be effective in Europe. It is less popular with radiation oncologists in the United States because of the concern of malignancy in the radiated areas. CO_2 lasers and photodynamic therapy have also been used with some studies showing success.[18]

10.5 Postoperative Care

Postoperative care is dependent on the type of reconstructive method selected. For simple incision and drainage, the wound is left open and allowed to heal by secondary intention. If there is a large cavity, the cavity is packed and allowed to drain, granulate, and contract. Dressing changes are performed daily.

For larger resections, if a skin graft is used to reconstruct the defect, a bolster dressing or a negative pressure vacuum-assisted wound dressing (wound VAC) is typically placed for compression to avoid a shear injury and allow the graft to contour to an often irregular surface area. The bolster or wound VAC is left in place for 5 to 7 days. However, it should be taken into consideration that at times an adequate seal with a negative pressure dressing is not achievable in certain anatomical areas, for example, the perianal area. Fecal diversion is rarely undertaken for perianal and perineal hidradenitis.

If a local tissue arrangement or local flaps are used to reconstruct the defect, care should be taken to obtain as tension-free of a closure as possible. Patients should be counseled regarding the increased incidence of wound breakdown. The wounds are then allowed to contract, granulate, and heal secondarily versus further attempts at soft tissue reconstruction.

There are also occasions when the resultant defect is too large for primary closure. A staged approach must be considered (▶ Fig. 10.2). The most common location for such a resection with soft tissue reconstruction is in the perineal and perianal regions. Wound healing complications are increased in this area due to its close proximity to the anus, increased moisture, and motion. This author's preference is to perform daily wound care until the defect either closes primarily or is acceptable for skin grafting or local flap reconstruction (▶ Fig. 10.3).

10.6 Outcomes

Medical management of hidradenitis suppurativa alone is often unsatisfactory except for the mildest form of the disease. Secondary to lack of understanding of the disease process, many treatments have been tried, but there are few randomized controlled trials and results are unpredictable. Antibiotics have been traditionally used in the treatment of the disease, and the largest trial to date by Gener et al, as mentioned previously, showed in his series of 116 patients treated with rifampin and clindamycin to have improvement in their Sartorius score and quality of life indices.[7]

Isoretinoids have been used in the treatment of hidradenitis, and results from studies show limited success with the largest trial by Soria et al, indicating improvement in only 16% and 7% actually worsened with treatment.[19] A review of hormonal therapy by Kraft et al, showed response to antihormonal therapy to be superior to antibiotics alone in a retrospective review of 64 patients.[8] Finasteride has also been used, but there is limited data on its effectiveness. Joseph et al, showed in his series of seven patients treated with finasteride that six had improvement and three of the seven had complete healing of the lesions.[10]

Fig. 10.2 **(a)** Preoperative Hurley type III perianal and gluteal hidradenitis; **(b)** initial resection.

Fig. 10.3 (a, b) At second stage, initial area of resection contracted, well granulated and ready for grafting. Additional area of hidradenitis excised.

There has been a lot of recent interest in the use of TNF-alpha inhibitors such as infliximab in the treatment of hidradenitis. This has been spurred by Hanauer et al's article reporting dramatic improvement in a patient's hidradenitis after receiving infliximab for Crohn's disease.[20] Van Rappard performed a systematic review of the response of hidradenitis to TNF-alpha inhibitors and showed that a good response was seen in 82% of patients treated with infliximab, 76% of patients treated with adalimumab, and 68% of patients treated with etanercept.[21]

Surgery remains the mainstay therapy for recurrent and severe hidradenitis, and possibly the only option of obtaining a cure from this debilitating disease. However, surgery is only beneficial in removing the disease in the area of resection and has little or no effect on preventing future disease in the immediate area around the resection site. Recurrence rates also vary by region after resection. Harrison et al, showed in his case series of 82 patients that after resection, recurrence was 0% in the perianal region, 3% in the axilla, and 37% in the inguinoperineal region.[22] It is felt that a combined approach using surgery to debulk the disease and medical management to prevent future recurrence will offer optimal therapy for patients afflicted with the disease.

10.7 Necrotizing Fasciitis and Soft Tissue Infections

10.7.1 Goals and Objectives

- Define the etiology, frequent causative organisms, and diagnosis of the disease.
- Appreciate the rapid progression of the disease and need for early and aggressive intervention.
- Understand the need for combined medical and surgical management.
- Understand the reconstructive options.

10.8 Patient Presentation

Necrotizing fasciitis (NF) is a soft tissue infection that is rapidly progressing and can be life-threating if not promptly identified and treated. Soft tissue infections include infections of the dermis, subcutaneous tissue, superficial or deep fascia, and muscle.[23] Necrotizing soft tissue infections can be classified depending on anatomic location, depth of infection, and microbial cause.[24] Anatomically, for instance, NF of the perianal, genitourinary, and perineal regions is referred to as Fournier's gangrene.[25] When considering the depth of infection, soft tissue infections can be characterized as adipositis, fasciitis, or myositis. Perhaps the most common classification is the microbial nature of the infection (▶ Table 10.3). Type I is the most common and involves mixed infections with aerobic and anaerobic bacteria, which are typically more indolent and associated with better prognosis. Type II is often monomicrobial and involves group A streptococcus and *S. aureus* and tend to be more aggressive than Type I. Type III is found more commonly in Asia and involves gram-negative and marine-related organisms and is usually associated with seafood or contaminated water. It includes *Clostridium* and *Vibrio* species. Finally, Type IV is of fungal origin, usually seen in immunocompromised or trauma patients and includes *Candida* spp. Like Type II, this type is aggressive in nature, often due to the underlying comorbidities of the patient.[26,27,28]

Table 10.3 Types of soft-tissue infections and causative organisms

Soft-tissue infection type	Causative organisms
Type I	Polymicrobial (aerobic and anaerobic)
Type II	Monomicrobial (Group A strep., *S. aureus*)
Type III	Gram-negative, marine related (*Clostridium, Vibrio*)
Type IV	Fungal (*Candida* spp.)

10.8.1 Epidemiology

Up to 70 to 80% of cases of NF are Type I followed by 20–30% of cases that are Type II.[24,27] The incidence of necrotizing soft tissue infections in the United States is approximately 1,000 cases per year, but some studies estimate the range from 500 to 1500.[23,26,27] In the United Kingdom, the overall incidence is

Fig. 10.4 Skin discoloration associated with necrotizing fasciitis from an infected Portacath. In difficult-to-diagnose cases, imaging can be a useful asset in confirmation. CT and MRI provide more sensitivity and specificity than plain radiography and show gas formation, extent of tissue infection, and fascial swelling. CT is useful in assessing tissue or osseous involvement, while MRI is better for evaluation of soft tissue infection. However, MRI is not often used due to delays in intervention. As pointed out by Anaya et al, imaging studies compare the involved site with the contralateral uninvolved site and do not necessarily help in differentiating between necrotizing and non-necrotizing soft tissue infection, so this must be used with caution when interpreting the findings.

estimated at 0.24 to 0.4 per 100,000 adults. Many risk factors have been attributed toward NF, including age older than 50 to 60 years, diabetes mellitus, IV drug use, peripheral vascular disease, obesity, chronic renal failure, HIV, penetrating trauma, and immunosuppression.[24,26,27,29] Chen et al, reported that NF has been seen in cases of Systemic Lupus Erythematosus (SLE), and other rheumatic diseases such as polymyositis, dermatomyositis, rheumatoid arthritis, and ankylosing spondylitis.[30] More recently, Dinc et al, reported NF (specifically Fournier's gangrene) as a postoperative complication of inguinal hernia repair.[25]

10.8.2 Pathophysiology and Clinical Presentation

The pathophysiology involves a disease that spreads at rates of up to 2 to 3 cm/hour from superficial to deep fascial layers and causes necrosis secondary to microvascular occlusion.[26,31] Depending on the causative microbe, toxins released can increase the aggressiveness of the infection through the influx of various pro-inflammatory and cytokine-mediated injuries. For instance, toxins from *Staph aureus* and Streptococci can, via TNF-alpha, IL-1, and IL-6, trigger a Systemic Inflammatory Response Syndrome (SIRS) response and eventually septic shock that can lead to patient death. There is also coagulation cascade activation that results in tissue ischemia. Understanding the pathophysiology is paramount to guiding management as in the face of significant ischemia, antibiotics may not be adequately delivered, requiring surgical intervention and debridement in most cases.[24] Recently, Kim et al, characterized three stages in the clinical features of progressing NF. Stage 1 begins early and involves tenderness and warmth to palpation, erythema, and swelling.[32] Clinically, this picture can resemble a cellulitis delaying diagnosis. Stage 2 involves formation of blisters or bulla and skin induration. Finally, in Stage 3, hemorrhagic bullae, skin anesthesia secondary to dying nerves from the spreading infection, and discoloration characteristic of gangrene can be seen. Hasham et al, classify the clinical findings in NF into "early findings" that include pain, cellulitis, pyrexia, tachycardia, swelling, induration, and skin anesthesia and "late findings" that include severe pain, skin discoloration, blistering, hemorrhagic bullae, crepitus, discharge of "dishwater fluid," severe sepsis or SIRS, and multi-organ failure (▶ Fig. 10.4).[29]

10.8.3 Diagnosis

Early identification of NF and prompt management will facilitate lower mortality rates. To this effect, the first part of diagnosis is taking a thorough history from the patient, including associated traumas, medical conditions, immunological status, recent travel, and recent infections as these can point toward certain causative organisms. As reported by Sarani et al, some of the signs and symptoms associated with necrotizing soft tissue infections include erythema, pain, swelling, induration, bullae, and fever.[24] Crepitus is not always found in NF and depends on the organism; hence, clinical judgment is important in timely diagnosis.

To aid in diagnosis using lab values, Wong et al, developed the laboratory risk indicator for necrotizing fasciitis score (LRINEC) to help differentiate between necrotizing and non-necrotizing soft tissue infections (▶ Table 10.4).[33] Using six variables (CRP, WBC, hemoglobin, sodium, creatinine, and glucose), each assigned with different points depending on set values, a total score between 0 and 13 is documented. This demonstrated positive and negative predictive values greater than 90% for scores greater than six, which helps in confirming the diagnosis of a necrotizing soft tissue infection.

Table 10.4 LRINEC scoring system for soft tissue infections

Variable (units)	Value	LRINEC Score
WBC count, cells/mm^3	< 15 15–25 > 25	0 1 2
CRP, mg/L	< 150 ≥ 150	0 4
Hemoglobin, g/dL	> 13.5 11–13.5 < 11	0 1 2
Sodium, mmol/L	≥ 135 < 135	0 2
Creatinine, mg/dL	≤ 1.6 > 1.6	0 2
Glucose, mg/dL	≤ 180 > 180	0 1

10.9 Treatment

10.9.1 Medical Management

Source control is of essence in necrotizing soft tissue infections. As discussed earlier, given significant ischemia to the infectious site, medical management with antibiotics needs to be carried out in combination with surgical intervention to debride the area and remove the necrotizing source tissue. Combinations of antibiotics can be tailored to organism based on culture results; however, early intervention includes ampicillin for gram-positive and gram-negative organisms, clindamycin for anaerobic coverage, and piperacillin-tazobactam for broad gram-negative coverage.[26] In order to cover gram-positive organisms, vancomycin, linezolid, and daptomycin are recommended due to concern for Methicillin Resistant Staph Aureus (MRSA).[24,26] Type 3 infections can be managed with clindamycin and penicillin in the case of *Clostridium* species and tetracyclines in the case of *Vibrio* infections, while for Type 4 infections, amphotericin B can be used.[26,27]

Other interventions needed to stabilize the patient include standard care with supportive fluids and continuous monitoring. Studies have also looked into interventions including IV immunoglobulin and hyperbaric oxygen; however, the efficacy of these strategies is unclear given limited number of studies and patient trials.[23,24,27,34]

10.9.2 Surgical Management

Surgical debridement is the mainstay treatment for NF (▶ Fig. 10.5). Intervention is time sensitive as delays beyond 12 hours in fulminant forms can be fatal, and thus early surgical consultation is necessary in the management of these patients.[26,34] In fact, Wong et al, found the relative risk of death to be more than 9 times greater when primary surgery was delayed by more than 24 hours.[35] Similarly, Mok et al, found the mortality relative risk after poor primary debridement to be greater than 7 times.[36] Procedures involving debridement need to be balanced with maintaining patient stability, and after the first debridement, may need to be repeated in the next 24 and 48 hours depending on the course of the infection and can sometimes involve 2 to 4 debridements.[24,27,28] Sarani et al, recommend that boundaries of excision be as wide as the rim of cellulitis and be composed of healthy, bleeding tissue. They also note that the boundaries of excision can expand intraoperatively due to poor boundaries appreciated on external physical exam.[24] Importance must be given to using strategic skin incisions while performing debridement so as to create flaps during the procedure that can facilitate aesthetic and functional closure and maintain coverage of critical structures.

Wound management after debridement involves leaving the wound open, treating with wet to dry dressings as well as vacuum-assisted closure dressing.[24,27,29] This author prefers dressings soaked with a 2.5 to 5% Sulfamylon solution. Use negative pressure vacuum-assisted dressing only after multiple debridements and establishment of a clean wound. As aforementioned, it is important to continue appropriate medical management not only before and during, but also after surgical intervention in order to address hemodynamic, nutritional, and immunological needs of the patient. Amputation may be a necessary intervention in some cases depending on the course of the infection and the amount of tissue that can be salvaged.[27,29]

On completion of the debridement process and once the patient has been stabilized, soft tissue reconstruction can be

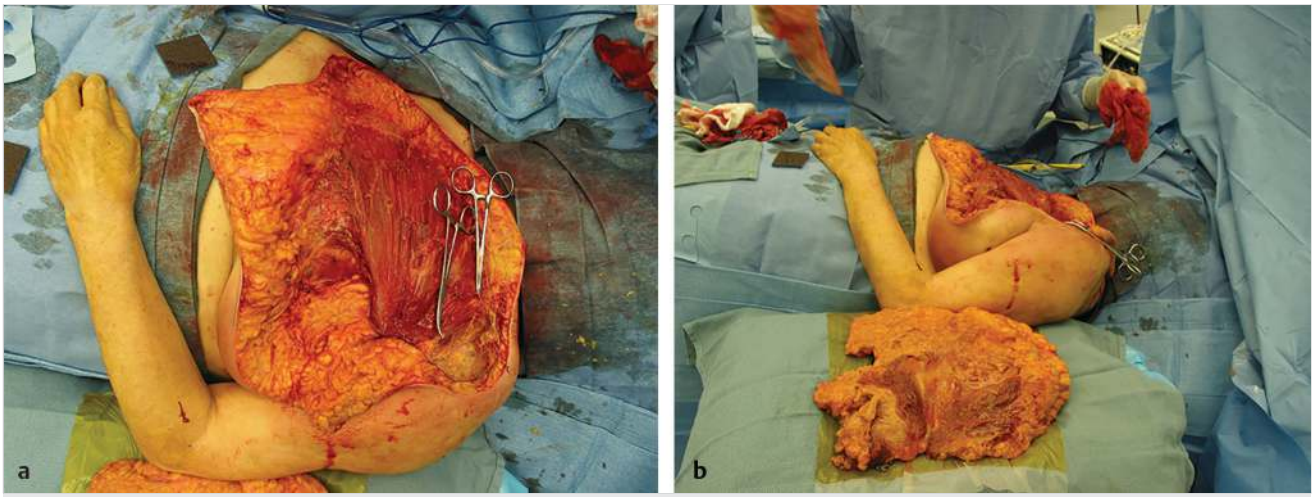


Fig. 10.5 (a, b) Initial resection of necrotizing fasciitis of the chest wall, breast, and upper arm.

Fig. 10.6 (a, b) Skin grafting on the left chest wall and upper arm (immediate and late result).

undertaken. It is important to adhere to the reconstructive ladder in these cases. Edlich et al, state that skin grafts (▶ Fig. 10.6) and flaps can provide sufficient coverage for body surface areas that are less than 25% of the total, and that when there is limited donor-site availability, dermal substitutes need to be considered.[34] Similarly, Sarani et al, state that full-thickness, rotational, or free flaps can help with adequate coverage after debridement.[24]

10.10 Outcomes

Nectrotizing fasciitis is a severe condition with significant morbidity and potentially high mortality. Overall healing and hospital course are dependent on the extent and region of the disease as well as the overall condition of the patients. Typically, these patients are hospitalized for weeks rather than days and required multiple operations and appropriate wound care and rehabilitation. It is essential to keep in mind the risk factors and patient comorbidities in early diagnosis and identification of NF as delayed diagnosis leads to greater risk of mortality. The rate of mortality directly from NF is difficult to assess as most patients have other medical comorbidities; however, rates range from 14% to 56% to as high as 70% to 80% despite treatment, and reach close to 100% without treatment.[23,26,27,31,37,38]

10.11 Review Questions

10.11.1 True or False

1. Early and aggressive management of necrotizing fasciitis is imperative to reduce mortality.
2. There is no role for unroofing or marsupialization of the sinus tracts in hidradenitis.
3. The pathophysiology of hidradenitis is well established and recurrence is rare.

10.11.2 Choose the Best Answer

4. The most common type of necrotizing fasciitis involves
   a) Fungal infections.
   b) Mixed aerobic and anaerobic organisms.
   c) Monomicrobial.
   d) Typically gram-negative and marine-related organisms.
5. Which of the following have been used in the treatment of hidradenitis?
   a) CO_2 lasers and photodynamic therapy.
   b) Isoretinoids.
   c) Finasteride.
   d) TNF-alpha inhibitors.
   e) All of the above.

10.11.3 Answers

1. True.
2. False.
3. False.
4. b. Mixed aerobic and anaerobic organisms.
5. e. All of the above.

References

[1] Alikhan A, Lynch PJ, Eisen DB. Hidradenitis suppurativa: a comprehensive review. J Am Acad Dermatol. 60(4):539–561, quiz 562–563
[2] Vazquez BG, Alikhan A, Weaver AL, Wetter DA, Davis MD. Incidence of hidradenitis suppurativa and associated factors: a population-based study of Olmsted County, Minnesota. J Invest Dermatol. 133(1):97–103
[3] Revuz JE, Canoui-Poitrine F, Wolkenstein P, et al. Prevalence and factors associated with hidradenitis suppurativa: results from two case-control studies. J Am Acad Dermatol. 59(4):596–601
[4] Hurley H. Dermatologic surgery, principles and practice. New York: Marcel Dekker, 1989; 729–739
[5] Sartorius K, Lapins J, Emtestam L, Jemec GB. Suggestions for uniform outcome variables when reporting treatment effects in hidradenitis suppurativa. Br J Dermatol. 149(1):211–213

[6] Rambhatla PV, Lim HW, Hamzavi I. A systematic review of treatments for hidradenitis suppurativa. Arch Dermatol. 148(4):439–446
[7] Gener G, Canoui-Poitrine F, Revuz JE, et al. Combination therapy with clindamycin and rifampicin for hidradenitis suppurativa: a series of 116 consecutive patients. Dermatology. 219(2):148–154
[8] Kraft JN, Searles GE. Hidradenitis suppurativa in 64 female patients: retrospective study comparing oral antibiotics and antiandrogen therapy. J Cutan Med Surg. 11(4):125–131
[9] Boer J. Oral retinoids for hidradenitis suppurativa. In: JemecG, RevuzJ, LeydenJ, eds, 'Hidradenitis suppurativa ' Vol. 1, Springer, 150–160
[10] Joseph MA, Jayaseelan E, Ganapathi B, Stephen J. Hidradenitis suppurativa treated with finasteride. J Dermatolog Treat. 16(2):75–78
[11] Slade DE, Powell BW, Mortimer PS. Hidradenitis suppurativa: pathogenesis and management. Br J Plast Surg. 56(5):451–461
[12] Sullivan TP, Welsh E, Kerdel FA, Burdick AE, Kirsner RS. Infliximab for hidradenitis suppurativa. Br J Dermatol. 149(5):1046–1049
[13] Cusack C, Buckley C. Etanercept: effective in the management of hidradenitis suppurativa. Br J Dermatol. 154(4):726–729
[14] Giamarellos-Bourboulis EJ, Pelekanou E, Antonopoulou A, et al. An open-label phase II study of the safety and efficacy of etanercept for the therapy of hidradenitis suppurativa. Br J Dermatol. 158(3):567–572
[15] Barron J. The surgical treatment of perianal hidradenitis suppurativa. Dis Colon Rectum. 13(6):441–443
[16] Alharbi Z, Kauczok J, Pallua N. A review of wide surgical excision of hidradenitis suppurativa. BMC Dermatol. 12:9
[17] Kagan RJ, Yakuboff KP, Warner P, Warden GD. Surgical treatment of hidradenitis suppurativa: a 10-year experience. Surgery. 138(4):734–740, discussion 740–741
[18] Lapins J, Marcusson JA, Emtestam L. Surgical treatment of chronic hidradenitis suppurativa: CO2 laser stripping-secondary intention technique. Br J Dermatol. 131(4):551–556
[19] Soria A, Canoui-Poitrine F, Wolkenstein P, et al. Absence of efficacy of oral isotretinoin in hidradenitis suppurativa: a retrospective study based on patients' outcome assessment. Dermatology. 218(2):134–135
[20] Hanauer SB. Immunogenicity of infliximab in Crohn's disease. N Engl J Med. 348(21):2155–2156, author reply 2155–2156
[21] van Rappard DC, Leenarts MF, Meijerink-van 't Oost L, Mekkes JR. Comparing treatment outcome of infliximab and adalimumab in patients with severe hidradenitis suppurativa. J Dermatolog Treat. 23(4):284–289
[22] Harrison BJ, Mudge M, Hughes LE. Recurrence after surgical treatment of hidradenitis suppurativa. Br Med J (Clin Res Ed). 294(6570):487–489
[23] Anaya DA, Dellinger EP. Necrotizing soft-tissue infection: diagnosis and management. Clin Infect Dis. 44(5):705–710
[24] Sarani B, Strong M, Pascual J, Schwab CW. Necrotizing fasciitis: current concepts and review of the literature. J Am Coll Surg. 208(2):279–288
[25] Dinc T, Kayilioglu SI, Sozen I, Yildiz BD, Coskun F. Fournier's Gangrene as a Postoperative Complication of Inguinal Hernia Repair. Case Rep Surg. 2014: 408217
[26] Misiakos EP, Bagias G, Patapis P, Sotiropoulos D, Kanavidis P, Machairas A. Current concepts in the management of necrotizing fasciitis. Front Surg. 1:36
[27] Morgan MS. Diagnosis and management of necrotizing fasciitis: a multiparametric approach. J Hosp Infect. 75(4):249–257
[28] Roje Z, Roje Z, Matić D, Librenjak D, Dokuzović S, Varvodić J. Necrotizing fasciitis: literature review of contemporary strategies for diagnosing and management with three case reports: torso, abdominal wall, upper and lower limbs. World J Emerg Surg. 6(1):46
[29] Hasham S, Matteucci P, Stanley PR, Hart NB. Necrotizing fasciitis. BMJ. 330 (7495):830–833
[30] Tung Chen Y, Isenberg D. Necrotizing fasciitis in systemic lupus erythematosus: a case report and literature review. Lupus Sci Med. 1(1):e000008
[31] Chaudhry AA, Baker KS, Gould ES, Gupta R. Necrotizing fasciitis and its mimics: what radiologists need to know. AJR Am J Roentgenol. 204(1):128–139
[32] Kim KT, Kim YJ, Won Lee J, et al. Can necrotizing infectious fasciitis be differentiated from nonnecrotizing infectious fasciitis with MR imaging? Radiology. 259(3):816–824
[33] Wong CH, Khin LW, Heng KS, Tan KC, Low CO. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med. 32 (7):1535–1541
[34] Edlich RF, Cross CL, Dahlstrom JJ, Long WB , III. Modern concepts of the diagnosis and treatment of necrotizing fasciitis. J Emerg Med. 39(2):261–265
[35] Wong CH, Chang HC, Pasupathy S, Khin LW, Tan JL, Low CO. Necrotizing fasciitis: clinical presentation, microbiology, and determinants of mortality. J Bone Joint Surg Am. 85-A(8):1454–1460
[36] Mok MY, Wong SY, Chan TM, Tang WM, Wong WS, Lau CS. Necrotizing fasciitis in rheumatic diseases. Lupus. 15(6):380–383
[37] Chansoria S, Chansoria H, Wanjari PV, Jain S, Gupta P. Prompt diagnosis and management of cervical necrotizing fasciitis. J Clin Diagn Res. 9(3):ZJ04–ZJ05
[38] Lee LH, Chalmers R, Muir T. Diagnosis, management and outcomes of necrotizing soft tissue infection within a Plastic and Reconstructive Surgery unit. Scott Med J. 59(1):56–61

11 Pressure Ulcers

Noah H. Prince, Rachel Cohen-Shohet, and Bruce A. Mast

Abstract

Pressure ulcers are a major concern in patient care. Left untreated, pressure ulcers can turn into pressure wounds that not only prolong healing, but additionally threaten the health and well being of the patient. Because of this, it is imperative that ulcers be promptly and properly treated. The authors take the reader through presurgical preparation and the operative interventions of debridement and reconstruction (often, flap reconstruction), to the postoperative care treatments that promote a good outcome.

Keywords: Norton scale, Braden scale, National Pressure Ulcer Advisory Panel Staging System, debridement, reconstruction, vacuum-assisted closure (VAC)

11.1 Goals and Objectives

- Understand the risk factors for pressure ulcers and be able to describe how they contribute to the formation of pressure wounds.
- Appreciate the multimodality approach for treatment and prevention of pressure ulcers.
- Be able to assess patients and optimize ancillary treatments prior to surgical intervention.
- Know surgical treatment options in the setting of sacral, ischial, and trochanteric pressure ulcers.
- Be able to counsel patients on postoperative treatment goals and outcomes of intervention.

11.2 Patient Presentation

A growing number of patients are presenting with pressure ulcers. Studies from Europe calculate the incidence in hospitalized patients to be 3% to more than 30%.[1] The cost of pressure ulcer treatment is estimated to be 11 billion dollars per year in the United States and utilizes 4% of the National Health Service's budget in the United Kingdom annually (1.4–2.1 billion U.S. dollars).[2]

Pressure ulcers occur in patients with impaired mobility in association with multiple risk factors. A lack of pressure relief techniques and devices places areas over bony prominences at risk (▶ Fig. 11.1, ▶ Fig. 11.2, ▶ Fig. 11.3, ▶ Fig. 11.4)[2]: A lack of pressure relief techniques and devices place the heels at risk in bedridden and supine patients (▶ Fig. 11.3; ▶ Fig. 11.4).

More worrisome is the resulting cone-shaped distribution of injury, with deeper tissues closest to the bone suffering the greatest damage. In these wounds, small-appearing superficial ulcers can have more severe injuries deep to the surface with an area of soft-tissue necrosis much greater than skin necrosis.[3,4,5]

Although excess or localized pressure remains the center of this problem, other contributing factors should not be ignored. These include shearing forces, which may worsen superficial damage, moisture (usually from bladder or bowel incontinence), which may hasten skin breakdown, and friction (usually from patient transfers), which may tear skin. Other factors that contribute to poor wound healing similarly affect pressure ulcers. Considerations such as poor nutrition, underlying infection, chronic edema, and peripheral vasculature disease can increase the time or resources necessary for wound healing.[6,7]

Fig. 11.1 Sacral, ischial, and trochanteric pressure wounds are frequently seen over bony prominences in bedridden patients.

Fig. 11.2 A lack of pressure relief techniques and devices place the heels at risk in bedridden and supine patients.

Fig. 11.3 Heel ulceration can result, as seen in this patient with calcaneal erosion.

Several recent studies have outlined risk factors such as elderly age, male gender, long-term admission, history of ulcers, diabetes, and falls. Bedbound status, poor nutrition, and a prior stroke also contribute in addition to lymphopenia, dry sacral skin, low weight, high illness severity scores, and residents of nursing homes. In these patients, it is crucial to balance prevention efforts with the level of risk.[8,9,10,11,12,13]

Both the Norton scale and Braden scale can be used with 70 to 90% sensitivity to identify those patients at risk for pressure ulcer formation[14,15] The Norton scale uses mental and physical condition, activity level, mobility, and incontinence factors in its formula, while the Braden scale focuses on sensory perception, moisture level, activity, mobility, nutritional status, friction, and shear force.[15,16] However, evidence supports that good clinical judgment can be equally as effective as a risk predictor.[17,18]

The diagnosis of pressure ulcers is largely clinical, with early recognition of pressure ulcers allowing for early treatment. Grading of pressure ulcers follows the National Pressure Ulcer Advisory Panel Staging System. The original staging system developed by Shea in 1975 was revised in 2007 by the National Pressure Ulcer Advisory Panel. The panel stages pressure ulcers by six categories: stages I, II, III, IV, unstageable, and suspected deep tissue injury as described in ▶ Table 11.1.[19]

Lastly, many patients will present with acute or chronic soft-tissue infection, colonization, or underlying osteomyelitis. In these patients, infection control is paramount to reconstructive interventions.

11.3 Preparation for Surgery

Prior to surgical intervention, it is essential to have an in-depth discussion with the patient regarding postoperative expectations and outcomes. Traditionally, flaps for pressure wounds necessitate prolonged bed-bound status, maintenance of nutritional intake, and other aspects of wound care which may require significant patient cooperation. Given the limited number of wound coverage options, it is imperative to optimize chances for flap success. This frequently requires a fully informed and well-prepared patient prior to surgical intervention.

Before reconstruction can take place, the site must be free of infectious nidus. This typically involves soft-tissue debridement with potential extension to bone. Flap viability is severely

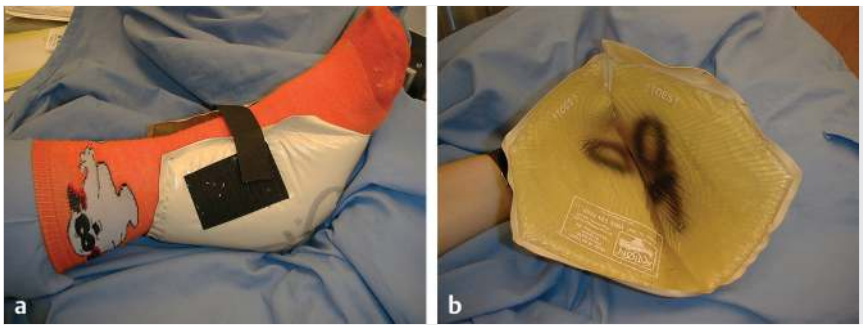


Fig. 11.4 **(a)** Isolated pressure relief of the heel. **(b)** Heel protection provided by gel pad.

Table 11.1 Staging of pressure wounds by National Pressure Ulcer Advisory Panel guidelines.

Stage I	Nonblanchable erythema. The skin over bony prominences may appear different from surrounding skin: painful, warm, cool, soft, or firm
Stage II	Partial-thickness loss of dermis, presenting as an ulcer with a shallow wound bed. Likewise it may appear as a blister, either opened or closed
Stage III	Full-thickness skin loss. The depth of the wound may vary with anatomic location, but there is full loss of dermis. In some cases, subcutaneous fat may be visible. Bone, tendon, or muscle are not be visible
Stage IV	Full-thickness loss with exposed tendon, muscle, and/or bone. Due to exposed structures, the risk for osteomyelitis is higher in stage IV ulcers
Unstageable/unclassified	Full-thickness skin or tissue loss in which the actual or true depth of the ulcer is unknown due to slough or eschar. In order to stage the wound properly, slough or eschar must be removed
Suspected deep tissue injury	Discolored area—usually purple or maroon, which is defined by either discolored intact skin or blood-filled blister from pressure or shearing forces. These wounds may evolve into a true pressure ulcer

Adapted from Black J, Baharestani MM, Cuddigan J, et al; National Pressure Ulcer Advisory Panel. National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system. Adv Skin Wound Care 2007;20(5):269–274

compromised in the setting of high bacterial growth.[20] In that respect, deep wound cultures should be taken to tailor antibiotic therapy. In the setting of suspected osteomyelitis, magnetic resonance imaging (MRI) and bone biopsy may be used for diagnosis and further tailoring of a prolonged antibiotic course. If underlying osteomyelitis is not treated, flap failure may result.

In addition to control of local infection, optimization of nutrition is crucial to promote normal wound healing. Adequate sources of protein (1.5–3 g/kg/day) and nonprotein calories (25–35 cal/kg/day), as well as vitamins and minerals, should be obtained.[21] Nutritional markers such as albumin and prealbumin may be warranted to trend progress, which should be balanced with timing of optimal wound reconstruction.

Diabetes plays an important role in wound healing; hemoglobin A1c greater than 6 has been associated with wound dehiscence and recurrence.[22] Additional factors such as smoking cessation and cardiac risk mitigation should also be discussed with the patient.

Preoperative planning for moisture control should also be considered. Fecal incontinence introduces high moisture, bacterial load, and shearing forces during routine cleaning. While this can sometimes be controlled with local hygiene, fecal diversion via colostomy may be necessary. If urinary incontinence is an issue, then bladder catheterization may be indicated as well.

Generally, it is believed that enzymatic debridement with the use of products such as collagenase, papain, or urea can be used for devitalized tissue in stage I and II pressure ulcers. Many chemical solutions have been used including Dakin's solution, hydrogen peroxide, and iodine to assist with mechanical debridement of devitalized tissue, but should otherwise be used with caution.[20] These agents are effective in controlling bacterial load, but also may be detrimental to human cells needed for healing. Mechanical and surgical debridement is generally recommended for decubitus ulcers stage III or higher.

Despite all operative and ancillary care, surgical reconstruction is of little benefit without plans for future prevention, most importantly pressure relief. In the immediate postoperative period, flap healing is compromised in the setting of ischemic tissue promoted by high pressures; so, plans for support services, mattresses, and cushions must be arranged preoperatively (▶ Fig. 11.5; ▶ Fig. 11.6; ▶ Fig. 11.7).

Furthermore, without sufficient health literacy and social support, many patients are unable to continue to provide themselves with these environments, resulting in development of new pressure wounds. In that sense, coordinated efforts should be made to provide continued assessment, education, and care for these patients.

11.4 Treatment

11.4.1 Indications for Surgical Intervention

The decision to proceed with surgical intervention for pressure ulcers varies with the specific circumstances of the patient and the surgical team involved. It is important that the patient be optimized from a medical standpoint, as discussed previously. Control of infection with appropriate antibiotics and tissue debridement and optimization of nutrition are necessary.[23,24] Generally, a serum prealbumin of greater than 15 mg/dL is considered within the normal range and would represent suitability for surgical intervention. Likewise, factors that created the pressure ulcer should be resolved: ischial pressure ulcers are often formed in the seated position, being caused commonly in the wheelchair bound patient. Patients can often identify a change in brand of cushion, wheelchair, or device that caused the formation of the ulcer. It is important to correct these

Fig. 11.5 (a, b) Pressure-relieving chair cushions are an important part of prevention and treatment of pressure wounds.

Fig. 11.6 Cushioned mattresses may be helpful to minimize pressure over bony prominences.

Fig. 11.7 Bedridden patients may benefit from air-fluidized or low air loss beds which more evenly distribute weight and pressure.

factors as to not cause breakdown postoperatively. Lastly, social hurdles that may prevent the patient from succeeding in flap surgery must be identified. Lack of resources for special equipment including beds and wheelchairs, as well as lack of social support for long-term care, can hinder the patient's recovery and overall success postoperatively.[20] Due to the magnitude of the surgery, the resources required, and the high risk of recurrence, a patient should have been medically optimized and have failed all conservative nonsurgical treatments prior to proceeding into surgical reconstruction. In addition, they should understand the long-term recovery associated with flap reconstruction, need for life-long compliance, and have resources in place prior to proceeding with reconstructive or flap surgery.

11.4.2 Operative Interventions

Surgical intervention can be largely divided into debridement and reconstruction. In most cases, both are required for a successful outcome.

11.5 Debridement

Operative debridement of pressure sores has several key goals. First and foremost begins with removal of necrotic, dead areas as well as chronic scar. This will enable better wound care, including use of enzymatic debridement application, as well as placement of vacuum-assisted closure (VAC) therapy. Second, debridement should remove or control soft tissue and bony infection. Removal of all infected tissues improves chances of success by minimizing bacterial load and biofilms within the wound bed, as these will both impede wound healing and potentiate failure of flap reconstruction. It is recommended that tissue be sent for pathology and culture as to help narrow antibiotic choice postoperatively. Removal of necrotic bone reduces bacterial burden, while also eliminating the bony prominence that was the causative pressure point. Debridement should be performed until healthy bleeding tissue is present in the wound bed. Likewise means of controlling the significant bleeding which is present in chronic wounds, such as sutures and electrocautery, should be readily available. Debridement of small pressure sores is possible at bedside in insensate tissue. However, debridement should ideally be performed in the operating room for large wounds with extensive tissue necrosis, tunneling, and tracking.

11.6 Reconstruction

Although for early stage I and II pressure wounds, debridement may be all that is required for wound healing, deeper stage III and IV wounds will often require surgical closure. Flaps, both myocutaneous and fasciocutaneous, offer volume and blood supply to cover large ulcers. General consensus is that there is no difference in outcomes between closure with fasciocutaneous or myocutaneous flaps; therefore, flap choice can largely be based on the location and size of the wound.[25] Classic pressure wounds include sacral, ischial, and trochanteric, and there are a wide variety of flap options for each. For all, gluteal-based flaps may be used, as they can be advanced superiorly or inferiorly. For trochanteric flaps, the tensor fascia lata flap, which derives its blood supply from the lateral femoral circumflex artery, is usually the first choice. For ischial pressure ulcers, several different posterior thigh flaps may be utilized, which may include the posterior hamstring muscles.

The V-Y advancement flap is a valuable technique often utilized in closure. ▶ Fig. 11.8 and ▶ Fig. 11.9 show an example of an ischial pressure wound and a segmental perforator flap for coverage (▶ Table 11.2). This is performed by making a V incision, followed by advancement of the tissue between the V into the wound site. The resultant donor-site defect from the apex of the original V is then closed linearly such that all scars cumulatively form a Y. This may be used for many pressure sores with closure using the fasciocutaneous components, and not only limited to the pelvic region, but across the body.

No matter what the choice in flap, there are several key principles to be identified. First, suture lines should be avoided over pressure points, to prevent breakdown of closure. Second, one should give thought to the ambulatory status of patient in choice of flap. For instance, a gluteal muscle flap may not be

Fig. 11.8 Left sided ischial pressure ulcer, markings for laterally based perforator flap.

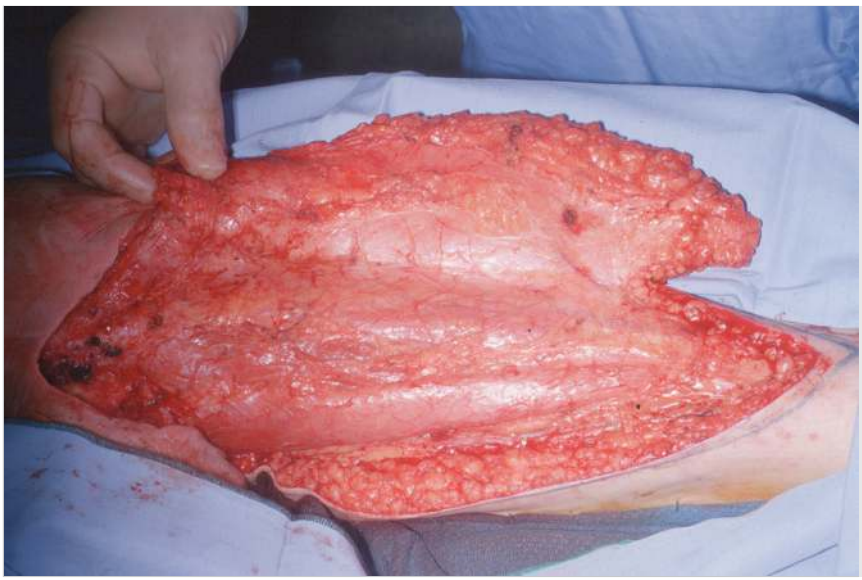


Fig. 11.9 Elevation of flap for coverage of left ischial pressure ulcer.

Table 11.2 Common flap choices for pressure ulcers based on location.

Wound location	Flap choice
Sacral	Gluteal flap (fasciocutaneous vs. myocutaneous)
Ischial	Posterior hamstring myocutaneous flap Posterior thigh flap (fasciocutaneous) Gracilis flap
Trochanteric	TFL flap

Abbreviation: TFL, tensor fascia lata.
Adapted from Power KL, Phillips LG. Pressure sores. In: Thorne CH, Chung KC, Gosain AK, et al. Grabb and Smith's Plastic Surgery. 7th ed. Philadelphia, PA: Wolters Kluwer Health; 2013:989–997.

desirable in a patient who walks. Third, all dead space should be obliterated with vascularized tissue. Drains are necessary within the obliterated dead space as well. Finally, flap reconstruction of pressure sores should include careful planning as to preserve other reconstruction options in the likelihood of future pressure sore formation.

11.7 Postoperative Care

Postoperative care for a patient who has undergone either a debridement of a pressure wound and/or a reconstruction begins with avoidance of pressure on all operative sites. This is first achieved with good nursing care, including turning to offload pressure every 2 hours. Pressure is avoided on operative sites for 4 to 6 weeks.[27] This is often accomplished with the use of specialty air flow beds that provide particularly low pressure to the contact points, as described previously.

After postoperative pressure relief is no longer necessary, a gradual weight/pressure bearing regimen is begun. For instance, progressive sitting is allowed for a patient who has been treated for an ischial ulcer. Pressure on the ischium is introduced gradually to assure stability of healing and avoid tissue damage, as well as providing progressive tissue tolerance to the pressure. The authors generally begin by allowing patients to sit for 30 minutes twice daily for 3 days. If the surgical/healed site remains intact without any ulcerations or evidence of shear or pressure injury, this is increased to 30 minutes three times daily for 3 days. This sort of sitting program has been utilized previously and has been shown effective in postoperative management.[27] Progression is thereby continued until full ad lib sitting with proper interval off-loading and pressure relief.

Venous thromboembolism prophylaxis should be considered in all postoperative patients in the immediate postoperative period including mechanical (such as sequential compression devices) and chemoprophylaxis. Long-term anticoagulation should be assessed individually for each patient.

Long-term antibiotics are usually not required for reconstructed stage II and III ulcers, involving soft tissue only. In stage IV ulcers, bone involvement is frequent. If osteomyelitis is documented by bone culture, long-term antibiotics will be necessary in combination with excision and flap reconstruction. Antibiotic therapy is best directed by an infectious disease specialist and narrowed to reflect intraoperative cultures.

Long-term care should not be taken lightly in these patients. When patients are ready to be discharged, they will likely need continued rehabilitation in a long-term care facility so that they may continue to receive antibiotics, physical therapy, and close nursing care for pressure off-loading. Items that may have caused pressure ulcers in the preoperative period should be removed. Oftentimes, a "home visit" has been used to evaluate pressure-causing items in the home prior to discharge. All long-term care should be focused on avoiding the problems that caused the pressure ulcers: devices, wheelchairs, chronic illnesses causing immobilization. Continued optimization of nutrition, control of diseases such as diabetes, should be continued in the postoperative period and monitored with appropriate labs and follow-up.

11.8 Outcomes

The outcomes for flap reconstruction are well known. Classic literature reports high recurrence rates and high morbidity

after surgical intervention. The dismal reported recurrence is largely based on the severity of the problem. Because the formation of pressure wounds is based on chronic conditions, which are often found in a debilitated population, it is hard to correct such issues in a short time frame, or even at all. The same social issues and requirement for meticulous nursing care, such as keeping surgical areas free of stool and urine as well as turning patients every 2 hours, is not present once patients leave the hospital. Many patients lack the support or finances to purchase the special equipment needed for proper care in the long-term setting and thus recurrence ensues. In severe cases of frequent recurrence, patients may have little surgical options left for closure, and are therefore left to manage with their chronic wounds.

Despite the many challenges, when successful, flap reconstruction of pressure wounds can offer freedom from the pain, infections, expense, and often humiliation that is associated with pressure sores. In addition, flap reconstruction can also prevent progression of infection, which would otherwise necessitate a larger, more morbid surgery. Current data suggest overall improvement in recurrence rates with recent values estimated at 19 to 33%, a large improvement from previous literature.[28,29] Literature also supports the fact that recurrence and complication is multivariable. Higher rates of recurrence were associated with osteomyelitis in the preoperative period.[30] In addition to recurrence of pressure sores in the long term, the surgeon should be prepared to deal with common surgical problems such as hematoma, seroma, or flap necrosis. One study found wound dehiscence rates up to 49%, and another study found an overall surgical complication rate of 40%.[22,31] The authors' experience is that minor dehisce, or distal flap necrosis, can be managed by local wound care. Oftentimes, postoperative complications will require a return to the operating room. This should not be discouraging, as local debridement and advancement of flap can often yield good outcomes.

11.9 Review Questions

11.9.1 True or False

1. Pressure-related tissue ischemia has been shown to occur in 2 hours of high tissue pressure.
2. Operative intervention should be employed as a first-line intervention in the treatment of pressure ulcers.

11.9.2 Choose the Best Answer

3. A pressure wound with full-thickness skin loss, visible subcutaneous tissue but no muscle, tendon, or bone, is classified as
   a) Stage I.
   b) Stage II.
   c) Stage III.
   d) Stage VI.
   e) Deep tissue injury.
   f) Unstageable.
4. Initial treatment of stage I and II ulcers involves
   a) Bone biopsy.
   b) Enzymatic debridement.
   c) Operative debridement.
5. Flaps that are available for ischial pressure ulcers include
   a) Gluteal flaps.
   b) Tensor fascia lata flap.
   c) Medial-based thigh flap.
   d) All of the above.
   e) None of the above.

11.9.3 Answers

1. True.
2. False.
3. c. Stage III.
4. b. Enzymatic debridement.
5. d. All of the above.

References

[1] Moore ZE, Cowman S. Wound cleansing for pressure ulcers. Cochrane Database Syst Rev. 3(3):CD004983

[2] Gillespie BM, Chaboyer WP, McInnes E, Kent B, Whitty JA, Thalib L. Repositioning for pressure ulcer prevention in adults. Cochrane Database Syst Rev. 4 (4):CD009958

[3] Krogh A, Landis EM, Turner AH. The movement of fluid through the human capillary wall in relation to venous pressure and to the colloid osmotic pressure of the blood. J Clin Invest. 11(1):63–95

[4] Landis EM, Jonas L, Angevine M, Erb W. The passage of fluid and protein through the human capillary wall during venous congestion. J Clin Invest. 11 (4):717–734

[5] Landis EM. Factors controlling the movement of fluid through the human capillary wall. Yale J Biol Med. 5(3):201–225

[6] Lindan O, Greenway RM, Piazza JM. Pressure distribution on the surface of the human body. I. Evaluation in lying and sitting positions using a "bed of springs and nails". Arch Phys Med Rehabil. 46:378–385

[7] Daniel RK, Wheatley D, Priest D. Pressure sores and paraplegia: an experimental model. Ann Plast Surg. 15(1):41–49

[8] Berlowitz DR, Wilking SV. Risk factors for pressure sores. A comparison of cross-sectional and cohort-derived data. J Am Geriatr Soc. 37(11): 1043–1050

[9] Allman RM, Goode PS, Patrick MM, Burst N, Bartolucci AA. Pressure ulcer risk factors among hospitalized patients with activity limitation. JAMA. 273(11): 865–870

[10] Nixon J, Cranny G, Iglesias C, et al. Randomised, controlled trial of alternating pressure mattresses compared with alternating pressure overlays for the prevention of pressure ulcers: PRESSURE (pressure relieving support surfaces) trial. BMJ. 332(7555):1413

[11] Horn SD, Bender SA, Bergstrom N, et al. Description of the National Pressure Ulcer Long-Term Care Study. J Am Geriatr Soc. 50(11):1816–1825

[12] Takahashi PY, Chandra A, Cha SS. Risk factors for pressure ulceration in an older community-dwelling population. Adv Skin Wound Care. 24(2):72–77

[13] Baumgarten M, Margolis DJ, Localio AR, et al. Pressure ulcers among elderly patients early in the hospital stay. J Gerontol A Biol Sci Med Sci. 61(7):749–754

[14] Smith DM, Winsemius DK, Besdine RW. Pressure sores in the elderly: can this outcome be improved? J Gen Intern Med. 6(1):81–93

[15] Braden BJ, Bergstrom N. Predictive validity of the Braden Scale for pressure sore risk in a nursing home population. Res Nurs Health. 17(6):459–470

[16] Bergstrom N, Braden BJ, Laguzza A, Holman V. The Braden Scale for predicting pressure sore risk. Nurs Res. 36(4):205–210

[17] Chou R, Dana T, Bougatsos C, et al. Pressure ulcer risk assessment and prevention: a systematic comparative effectiveness review. Ann Intern Med. 159(1): 28–38

[18] Webster J, Coleman K, Mudge A, et al. Pressure ulcers: effectiveness of risk-assessment tools. A randomised controlled trial (the ULCER trial). BMJ Qual Saf. 20(4):297–306

[19] Black J, Baharestani MM, Cuddigan J, et al. National Pressure Ulcer Advisory Panel. National Pressure Ulcer Advisory Panel's updated pressure ulcer staging system. Adv Skin Wound Care. 20(5):269–274

[20] Bauer J, Phillips LG. MOC-PSSM CME article: pressure sores. Plast Reconstr Surg. 121(1) Suppl:1–10

[21] Stratton RJ, Ek AC, Engfer M, et al. Enteral nutritional support in prevention and treatment of pressure ulcers: a systematic review and meta-analysis. Ageing Res Rev. 4(3):422–450
[22] Keys KA, Daniali LN, Warner KJ, Mathes DW. Multivariate predictors of failure after flap coverage of pressure ulcers. Plast Reconstr Surg. 125(6):1725–1734
[23] Cushing CA, Phillips LG. Evidence-based medicine: pressure sores. Plast Reconstr Surg. 132(6):1720–1732
[24] Choo TS, Hayter M, Watson R. The effectiveness of nutritional intervention(s) and the treatment of pressure ulcers–a systematic literature review. Int J Nurs Pract. 19(1) Suppl 1:19–27
[25] Yamamoto Y, Tsutsumida A, Murazumi M, Sugihara T. Long-term outcome of pressure sores treated with flap coverage. Plast Reconstr Surg. 100(5):1212–1217
[26] Power KL, Phillips LG. Pressure sores. In: Thorne CH, Chung KC, Gosain AK, et al. Grabb and Smith's Plastic Surgery. 7th ed. Philadelphia, PA: Wolters Kluwer Health; 2013:989–997
[27] Tchanque-Fossuo CN, Kuzon WM , Jr. An evidence-based approach to pressure sores. Plast Reconstr Surg. 127(2):932–939
[28] Kierney PC, Engrav LH, Isik FF, Esselman PC, Cardenas DD, Rand RP. Results of 268 pressure sores in 158 patients managed jointly by plastic surgery and rehabilitation medicine. Plast Reconstr Surg. 102(3): 765–772
[29] Schryvers OI, Stranc MF, Nance PW. Surgical treatment of pressure ulcers: 20-year experience. Arch Phys Med Rehabil. 81(12):1556–1562
[30] Marriott R, Rubayi S. Successful truncated osteomyelitis treatment for chronic osteomyelitis secondary to pressure ulcers in spinal cord injury patients. Ann Plast Surg. 61(4):425–429
[31] Goodman CM, Cohen V, Armenta A, Thornby J, Netscher DT. Evaluation of results and treatment variables for pressure ulcers in 48 veteran spinal cord-injured patients. Ann Plast Surg. 42(6):665–672

Part III

Pediatric Plastic Surgery

III

12 Cleft Lip Deformity

Ashley K. Lentz

Abstract
Cleft lip deformity not only involves physical distortion, but even more, it commonly carries with it emotional, psychological, and social scarring. For these reasons, surgical intervention is critical, and the author covers every consideration involved in the proper treatment of cleft lip deformity. In preparing for surgery, any comorbities, such as cardiac and pulmonary anomalies, should be identified and addressed, and a presurgical molding technique adopted. Various operative techniques are discussed, and postoperative complications are identified.

Keywords: taping, nasoalveolar molding, Latham appliance, straight line repair, triangular flap, rotation advancement flap, bilateral cleft lip repair

12.1 Goals and Objectives

- Learn the nomenclature and challenges for patients with cleft lip deformity.
- Understand the types of presurgical molding and their benefit.
- Recognize the different types of cleft repair.
- Know the principles for cleft lip repair as well as the indications and timing for additional surgery.

12.2 Patient Presentation

Patients born with cleft lip have a facial deformity that can lead to emotional and physical distortion if unrepaired. These children can suffer from societal exclusion, profound psychological effects, as well as functional compromise.[1] Cleft patients need surgical intervention to help avoid these poor outcomes. In addition, their parents require substantial time investment for education, encouragement, and emotional support. The care of the cleft patient requires a multidisciplinary approach in an effort to provide the necessary resources to optimize the long-term emotional and physical outcome.

Many patients with cleft lip are born into varying levels of socioeconomic class and educational achievement.[2] Therefore, it is important to establish the financial ability and the resources that each family has in order to care for their child. Those with fewer resources and lesser education are more likely to struggle to "make ends meet" while trying to attend the multiple appointments that are needed for their child. A social worker plays a major role in targeting the families that will require more assistance. A unified craniofacial team can lessen the stress on these families and provide an opportunity to see multiple practitioners in one single visit to the center.[3] Additionally, this single visit provides the practitioners an opportunity to meet together and establish a unified and comprehensive plan for each patient.

In addition to a cleft surgeon, a comprehensive craniofacial team offers an evaluation from otolaryngology, oral surgery, psychology, social work, audiology, speech therapy, orthodontics, pediatric dentistry, pediatrics, and genetics. Each specialist plays a significant role in the care of these patients throughout the life of a cleft patient. It is ideal to meet patients within the first couple of weeks of birth. Not only does early involvement help the parents to better cope with the emotional strain, but it also helps parents to better care for their infant. Nutritional support and education for methods of feeding can help ensure that the baby will consume enough nutrition in order to grow and thrive. Finally, this early visit establishes an opportunity for the cleft surgeon to evaluate the severity of the cleft and determine the best approach in preparation for surgical intervention.

12.3 Preparation for Surgery

Patients born with cleft lip may have additional comorbidities that should be addressed prior to surgery. They may have cardiac and pulmonary anomalies that require repair or close monitoring prior to cleft repair. It is also important to assess their growth and weight gain on the pediatric growth chart in order to limit complications with general anesthesia and surgery. Age old recommendations have stated that babies should weigh a minimum of 10 pounds prior to surgery.

There are differing levels of cleft severity, including incomplete clefts all the way to very wide complete clefts. They may be unilateral or bilateral. Larger width clefts will have a greater impact on the nasal and alveolar deformity. ▶ Fig. 12.1 is an example of an incomplete unilateral cleft lip, while ▶ Fig. 12.2 shows a unilateral complete cleft lip. ▶ Fig. 12.3 and ▶ Fig. 12.4 are examples of an incomplete bilateral and complete bilateral cleft lip, respectively. Assessment of the cleft width is necessary in order to determine the need and type of presurgical molding. It is imperative to determine the ability and means for each parent to follow through on presurgical treatment. Time and financial constraints will prevent good outcomes due to patient inability to attend clinic to monitor the success of the treatment.

There are three types of presurgical techniques, which include taping, nasoalveolar molding, and Latham appliance.[4,5] The simplest technique is the act of taping. The infant care provider gently pinches the cleft lip together and applies tape over the gap. This must be performed several times per day, as the tape is likely to fall off when it gets wet during feedings. Taping is very inexpensive and it is very simple to train care providers on the technique. Unfortunately, taping does not address the nasal deformity and it can irritate infant's skin. Nasoalveolar molding requires an intraoral impression to design an acrylic orthopaedic appliance that is placed into the infant's mouth. This device enables the cleft gap to narrow while expanding the tissue to help improve the shape of the nose while preventing palatal collapse. It requires multiple and consistent trips to the pediatric dentist for monitoring of the appliance fit and progression of the molding. This can be difficult for some families. Some infants do not tolerate the appliance and constantly push it out, thereby limiting the potential for benefit.

The Latham appliance is surgically inserted into the infant's mouth early in infancy. The parents are trained to turn the

Fig. 12.1 Patient with a unilateral incomplete cleft lip.

Fig. 12.2 Patient with a unilateral complete cleft lip.

Fig. 12.3 Patient with a bilateral incomplete cleft lip.

Fig. 12.4 Patient with a bilateral complete cleft lip.

Table 12.1 Types of presurgical treatment with pros and cons of each

Type of presurgical treatment	Definition	Pros	Cons
Taping	Tape is applied to the lip to span the gap and an effort is made to approximate the lip segments closer together	• Inexpensive • Simple technique/easy to learn • Nonsurgical	• Requires multiple applications per day • Tape can irritate the skin • Does not address the nasal deformity
Nasoalveolar molding	Impressions of the palate and alveolar gap are made and frequently changed in order to narrow the gap of the cleft	• Provides palatal support to help prevent arch collapse • Addresses the nasal deformity to improve postoperative outcomes • Nonsurgical	• Expensive • Requires multiple visits for proper fitting • Patients may push the appliance out with their tongue
Latham appliance	This device is surgically inserted with pins and screws are turned daily to bring the cleft together	• Does not require removal • Limited number of visits	• Additional anesthesia event • Parents must turn the screws • Infants have an adjustment period while getting used to the device

screws daily in order to bring the cleft together.[6,7] There is an adjustment period for the infant, as the permanent appliance can affect feeding in the early stages. This also requires an additional exposure to anesthesia during the placement of the device. However, this technique limits the number of clinic visits as compared to nasoalveolar molding (▶ Table 12.1).

12.4 Treatment

The face is one of the single most important tools we use to communicate with other individuals in society. We smile, frown, and use other facial expressions to convey our thoughts and feelings. When the face is scarred in some way or not perceived as acceptable by society, individuals suffer emotional consequences and social ostracizing. This also has a long-term negative impact on the patient with a cleft lip and their family.[8] Surgical repair of a cleft lip allows patients to remain an acceptable member of society. In addition, surgery allows the patient to have better oral hygiene while consuming food and beverage.

The overall goal of lip repair is to create a result that addresses many anatomic features. The surgeon must create muscle continuity. The lip height on the cleft side should be equal to the noncleft side. The cupid's bow should be in continuity with the peaks at the same horizontal level. The

Table 12.2 Important anatomy to address during cleft lip repair

Anatomic point	Requirements
Orbicularis oris muscle	Complete muscle continuity is imperative. The surgeon needs to take down the aberrant attachments and place them in the proper anatomic location across the cleft deformity
Lip height	The height of the cleft side needs to match the height of the noncleft side
Cupid's bow	The cupid's bow should be symmetric with the height of the peaks placed at the same horizontal plane
Vermillion cutaneous junction	Step-off deformity between the upper lip skin and the red lip are very noticeable. Therefore, this junction should be in continuity
Red lip	Fullness of the red lip on the cleft side needs to match that of the noncleft side. The junction of the wet and dry lip can be offset preoperatively, and special attention is required to repair this discrepancy
Nose	The flattened ala of the cleft side should be dissected free from the attachments. The ala requires rounding to inset it at the proper anatomic location and to create alar, nare, and nasal base symmetry

vermillion cutaneous junction should be contiguous without step-off deformities. The red lip should be full and symmetric between the cleft and noncleft side, while the wet lip and dry lip junction needs to match at the point of intersection. Finally, the surgeon must spend adequate time addressing the nasal deformity. The flattened ala needs to be contoured at the time of lip repair. The nasal bases should sit at the same horizontal plane and the tip of the nose should be addressed to create better nasal tip projection. Finally, the nares should be equal in diameter and the surgeon needs to try and avoid creating a web in the nasal vestibule of the cleft side (▶ Table 12.2).

The standard time for lip repair is approximately at 3 months. Most surgeons want the infants to be healthy and thriving with a weight equal to or greater than 10 pounds. If an infant undergoes presurgical techniques, they may not undergo surgery until the desired presurgical outcomes are achieved. Additionally, if the infant has any other underlying medical comorbidities, these too should be addressed prior to surgery. Cleft lip repair is the first surgery in a series of surgeries that the patient will have throughout his or her life. Additional surgeries will be needed if the patient has a cleft palate and alveolar cleft (▶ Table 12.3).

Table 12.3 Timeline for cleft lip treatment

Age	Treatment
3 mo	Cleft lip repair
9 mo	Cleft palate repair
7–9 y	Alveolar bone grafting
16–17 y	Orthognathic surgery
16–18 y	Rhinoplasty and lip revision

Fig. 12.5 Example of triangular flap.

12.5 Operative Technique

There have been many techniques described to repair the unilateral cleft lip. In 1844, Mirault published his technique for a straight line repair of the unilateral cleft lip, although there was much controversy regarding his technique.[9] The many successors added modifications in the immediate years to follow. In 1952, Tennison introduced the triangular flap for which he received good results and praise from other surgeons.[10] He used a wire stencil to measure the noncleft side from the peak of cupid's bow to the base of the columella on the cleft side. This wire was then bent into the shape of a Z and imprinted on the cleft side. The final result reveals a zigzag appearance of the scar that extends across the red lip (▶ Fig. 12.5). Millard creatively devised the rotation advancement flap and published his first article on this topic in 1958.[11] This was a clever approach where the noncleft side of the lip was back cut in order to allow for rotation of this lip segment into the vertical position. The cleft side was then advanced to meet the other side. This technique helped to prevent future superior contracture of the lip scar (▶ Fig. 12.6). He again published several articles in the years to follow on modifications of his technique, given the fact that this technique can often be inadequate to create symmetric lip height. Many modifications were made based on Millard's concept of rotation advancement by other surgeons. In 2005, Fisher introduced the anatomical subunit approximation technique. He recognized the limitations of the rotation advancement without modifications.[12] His goal was to prevent the loss of horizontal distance between the peak of cupid's bow on the cleft side to the oral commissure. Finally, this technique places the final scar along the seams of the anatomical subunits of the lip and nose (▶ Fig. 12.7).

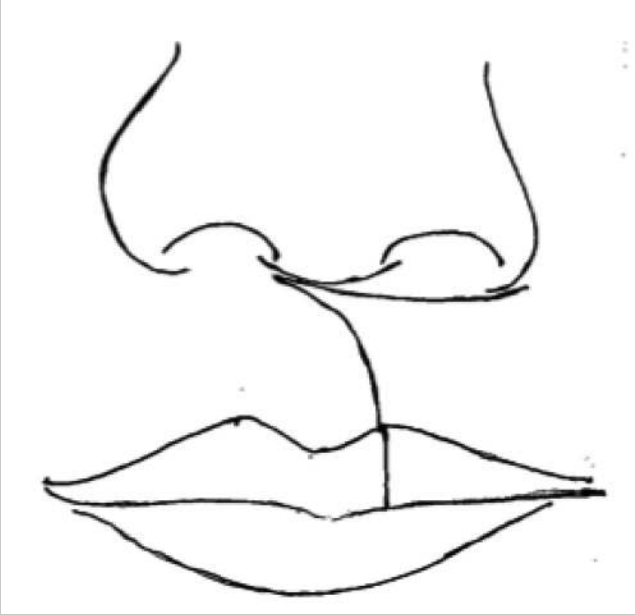


Fig. 12.6 Example of the Millard rotation advancement flap.

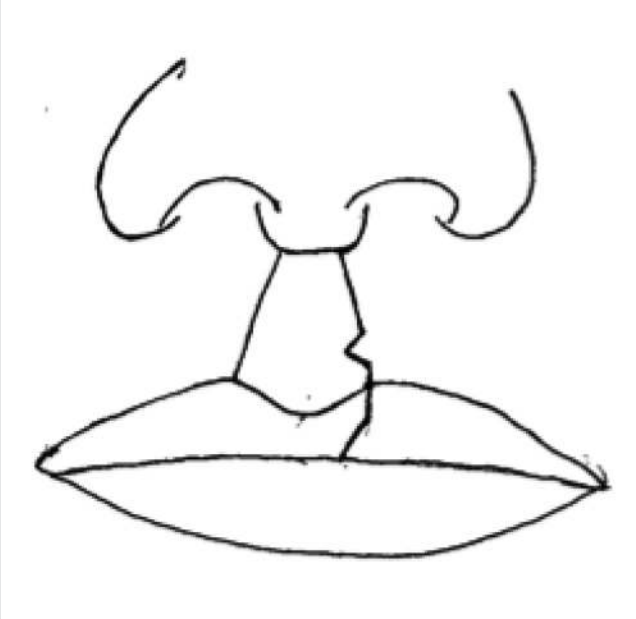


Fig. 12.7 Example of the Fisher flap.

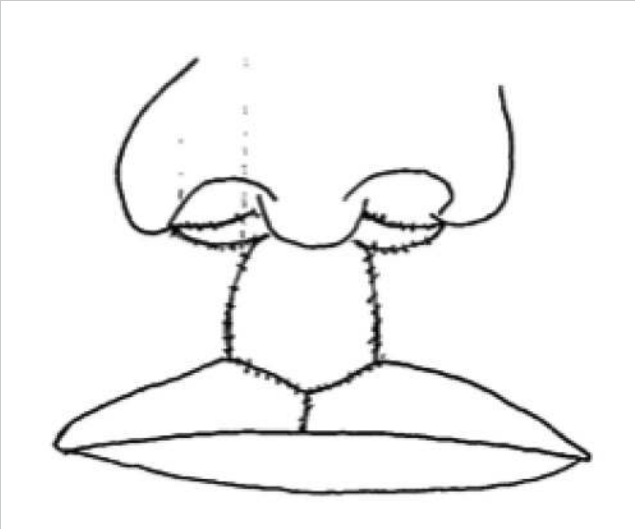


Fig. 12.8 Example of the Millard bilateral cleft lip repair with fork flaps.

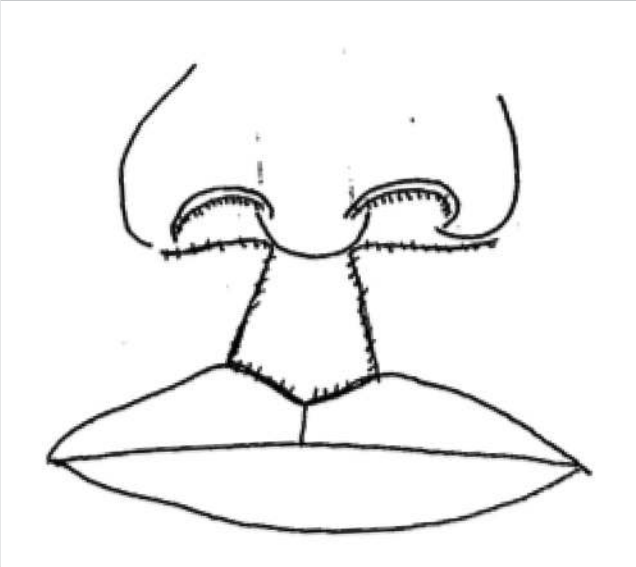


Fig. 12.9 Example of the Mulliken bilateral cleft lip repair.

The bilateral cleft lip repair has evolved similarly. Early on, a straight line repair was used by Veau.[13] Given the shortened prolabium height, additional strategies were used to gain length. These included the use of Z-plasties along the lip.[14,15] Millard recognized the challenge of the shortened prolabium and performed bilateral repair in two stages.[16] He also described forked flaps that were later used to lengthen the columella (▶ Fig. 12.8).[17] As the child with a bilateral cleft lip grows, the prolabium widens and elongates. Recognition of this fact has led to the discontinued use of the forked flap.[18] Mulliken et al used a single-stage closure after patients have undergone presurgical molding. They emphasized the need for primary correction of the nasal deformity. They now use bilateral rim incisions to assist with the exposure to address the nasal cartilage (▶ Fig. 12.9).[19]

12.6 Outcomes

As with any significant surgical procedure, complications or adverse outcomes are possible. These include infection,

bleeding, and dehiscence in the early stages after surgery. Some of the long-term sequelae may be lip height discrepancy, nasal alar collapse, step-off deformity of the vermillion cutaneous junction, malalignment of the wet and dry red lip junction, and loss of red lip fullness on the cleft side leading to a whistle deformity (▶ Fig. 12.8).

Cleft surgery is challenging and the best outcomes will arise when the surgeon pays close attention to all the details as mentioned earlier. It takes years of practice to try and perfect these anatomic points. No two cases are exactly alike, and this fact presents the cleft surgeon with a challenge during each case. A good surgeon constantly analyzes his or her outcomes to provide better care with each successive patient.

12.7 Conclusion

Patients born with cleft lip have varying social circumstances that may have profound impacts on the final outcome of their care. A comprehensive cleft team is imperative to optimize care for these patients. Depending on the defect, an infant may benefit from presurgical techniques. Cleft lip surgery is typically performed at 3 months of age. Subsequent surgeries are performed throughout the first two decades of life. The information above is simply a guide for most standard treatment plans by cleft surgeons. There are many different operative techniques and strategies, and each surgeon develops his or her own method. Constant regard for the outcomes will enable the surgeon to constantly improve the care given. Care of children with cleft lip is personally very challenging and ultimately rewarding, as there is exceptional continuity of care with these patients.

12.8 Review Questions

12.8.1 True or False

1. A comprehensive cleft and craniofacial team provides better outcomes for patients with cleft lip deformity.
2. Presurgical techniques must be used on every cleft patient in order to achieve acceptable surgical outcomes.
3. Cleft lip and cleft palate are often repaired at the same time.

12.8.2 Choose the Best Answer

4. Millard published a new method for cleft lip repair in 1958. This technique is best described as a
   a) Straight line repair.
   b) Rotation advancement flap.
   c) Triangular flap.
   d) Anatomical subunit approximation.
5. Complications following cleft lip repair include all of the following except
   a) Upper lip scar.
   b) Nasal alar collapse.
   c) Increase in the distance from the peak of cupid's bow on the cleft side to the oral commissure.
   d) Step-off deformity of the vermillion cutaneous junction.

12.8.3 Answers

1. True.
2. False.
3. False.
4. b. Rotation advancement flap.
5. c. Increase in the distance from the peak of cupid's bow on the cleft side to the oral commissure.

References

[1] Turner SR, Thomas PWN, Dowell T, Rumsey N, Sandy JR. Psychological outcomes amongst cleft patients and their families. Br J Plast Surg. 50(1):1–9

[2] Acuña-González G, Medina-Solís CE, Maupomé G, et al. Family history and socioeconomic risk factors for non-syndromic cleft lip and palate: a matched case-control study in a less developed country. Biomedica. 31 (3):381–391

[3] Austin AA, Druschel CM, Tyler MC, et al. Interdisciplinary craniofacial teams compared with individual providers: is orofacial cleft care more comprehensive and do parents perceive better outcomes? Cleft Palate Craniofac J. 47(1): 1–8

[4] Pool R, Farnworth TK. Preoperative lip taping in the cleft lip. Ann Plast Surg. 32(3):243–249

[5] Grayson BH, Santiago PE, Brecht LE, Cutting CB. Presurgical nasoalveolar molding in infants with cleft lip and palate. Cleft Palate Craniofac J. 36(6): 486–498

[6] Georgiade NG, Latham RA. Maxillary arch alignment in the bilateral cleft lip and palate infant, using pinned coaxial screw appliance. Plast Reconstr Surg. 56(1):52–60

[7] Latham RA, Kusy RP, Georgiade NG. An extraorally activated expansion appliance for cleft palate infants. Cleft Palate J. 13:253–261

[8] Nelson P, Glenny AM, Kirk S, Caress AL. Parents' experiences of caring for a child with a cleft lip and/or palate: a review of the literature. Child Care Health Dev. 38(1):6–20

[9] Mirault G. Deux lettres sur l'operation du bec-delievre. J Chir (Paris). 2:257

[10] Tennison CW. The repair of the unilateral cleft lip by the stencil method. Plast Reconstr Surg (1946). 9(2):115–120

[11] Millard DR , Jr. A radical rotation in single harelip. Am J Surg. 95(2):318–322

[12] Fisher DM. Unilateral cleft lip repair: an anatomical subunit approximation technique. Plast Reconstr Surg. 116(1):61–71

[13] Veau V. Bec-de-Lievre. Paris: Masson; 1938

[14] Berkeley WT. The concepts of unilateral repair applied to bilateral clefts of the lip and nose. Plast Reconstr Surg. 27:505

[15] Skoog T. The management of the bilateral cleft of the primary palate (lip and alveolus). I. General considerations and soft tissue repair. Plast Reconstr Surg. 35:34–44

[16] Millard DR , Jr. Closure of bilateral cleft lip and elongation of columella by two operations in infancy. Plast Reconstr Surg. 47(4):324–331

[17] Millard DR , Jr. Bilateral cleft lip and a primary forked flap: a preliminary report. Plast Reconstr Surg. 39(1):59–65

[18] Millard DR Jr. Cleft Craft. Vol. 2. Little Brown and Company; 1976

[19] Mulliken JB, Wu JK, Padwa BL. Repair of bilateral cleft lip: review, revisions, and reflections. J Craniofac Surg. 14(5):609–620

13 Cleft Palate Repair by Double Opposing Z-Plasty

Leonard T. Furlow Jr.

Abstract
This chapter is a careful discussion of the function of the palate in normal speech and of the problems caused by cleft palate. After surveying the history of cleft palate repair, the author delves into the steps involved in the double opposing z-plasty repair. Hard palate repairs are also discussed and illustrated. Postoperative care of the airway and bleeding is covered, and the steps in long-term follow-up are outlined.

Keywords: cleft palate, double opposing z-plasty (DOZ-pl), velopharyngeal function

13.1 Goals and Objectives

- Function of palate in speech, both velar and hard palate portions.
- Brief history of cleft palate repair and its importance in the development of palate repair methods.
- The purposes and design of the double opposing Z-plasty palate repair.
- Why velopharyngeal function is critical to normal speech.
- How to determine velopharyngeal function related to cleft palate.

13.2 Patient Presentation

Cleft palate repair carries a large responsibility, because repair does not guarantee normal speech, and because abnormal speech is a serious social disadvantage.

In the United States, the standard of care for those with cleft lip and palate is by a team, which usually includes one or more of the following: plastic surgeons, pediatricians who are familiar with the syndromes associated with patients with cleft palate only **(CPO)** and cleft lip and palate **(CLP)**, otolaryngologists, orthodontists, oral surgeons, speech–language pathologists **(SLP)**, and a supporting team including a nurse and an administrator. Plastic surgeons not associated with a team should consider referring patients to a team.

It is important to consider the concerns and anxieties of the parents and to have the appropriate team members address them at every visit, especially the first. On initial examination, the plastic surgeon should note the child's age, weight, hemoglobin and hematocrit, and a family history of cleft and any congenital abnormality that might be cleft-related; record the characteristics of the cleft; unilateral CLP (UCLP), bilateral CLP (BCLP), CPO; complete/incomplete, width of the cleft, and the slope of the hard palate shelves; evidence of previous surgery (review the chart if the lip has been repaired); and evidence of a syndrome (particularly such as Pierre Robin, which might impact the surgical plans by delaying repair and avoiding a pharyngeal flap, or velocardiofacial syndrome, which is a difficult surgical problem carrying a lower expectation of success). The parents should be asked to try to have the child feeding from a cup before palatal surgery. In older patients, speech characteristics should be noted and velopharyngeal (VP) function tested.

13.3 Preparation for Surgery

After examination of the child and discussion of the patient by the team, the plastic surgeon should explain why the function of the palate is important in speech, what palate repair must accomplish, and that the goals of repair are not always reached at the first operation. The planned operation should be described for the parents with a discussion of postoperative complications; let them know what to expect. The sequence and timing of procedures and operations should be discussed, with the reasons that subsequent operations might be necessary, and an estimate of how frequently they are usually needed. The possibility of operations for velopharyngeal insufficiency (VPI), fistula repair, alveolar bone grafting, or correction of maxillary recession should be emphasized in the discussion. These points should be reiterated at the preoperative visit.

13.3.1 Palatal Function

Proper treatment of the patient with cleft palate requires an understanding of palatal function.

During speech, for various consonants, *the lips and tongue* permit ("really"), restrict ("fish"), or block ("Dad") airflow through the oral cavity. The normal hard palate separates the oral and nasal cavities. It is important for the growth of the midface. The normal soft palate (velum) is a muscular valve that opens and closes to permit, restrict, or prevent airflow and sound into the nasal cavity. This function is required for normal speech.

For most of our English words, the velopharyngeal valve is closed, to direct air and sound through the mouth only ("brother, papa, supercalifragilisticexpialidocious"). The velum provides quick, precisely timed velopharyngeal valving to permit airflow and sound through the nose when the lips, tongue, and teeth block oral airflow ("noon, rain"), and coordinates with the lips and tongue to control airflow and sound through nose and mouth for many words ("mop, king, ban"), or with a mid-word switch ("sentinel, bumper, single, maintain"). Listen to how the oral and nasal vowel sounds relate to the associated consonants.[1,2,3,4]

The palatal cleft mars speech by failing to block airflow into the nasal cavity on consonants, and sound on vowel production. *The prime purpose of cleft palate repair is to make normal speech possible by converting the cleft hard and soft palate into a competent velopharyngeal valve mechanism.*

A successful cleft palate repair (1) produces a velum that permits precise, instantaneous, essentially complete velar closure, and velar opening sufficient for nasal breathing; (2) constructs an intact hard palate that partitions the nasal cavity from the oral cavity and permits adequate midface growth; (3) protects middle ear function; (4) achieves all, preferably by the age of 1 year, before the child learns speech.

Palate repair is by no means always successful. If velopharyngeal closure is inadequate or a hard palate fistula connects the oral and nasal cavities, compensatory articulations, substitute sounds learned when the attempt to make a desired sound by closing the velum does not work, may mar speech after a second operation has provided VP competence.[5,6,7]

13.4 Treatment

13.4.1 History

The history of cleft palate repair is interesting, particularly because improvements have come so slowly and because over its first century and a half, the history has defined the requirements for the best assurance of a successful surgical outcome.

The first successful surgical repair was in 1819 in Paris by Dr. Roux on the Canadian John Stephenson, a medical student in Edinburgh. A thesis was required for graduation, and Stephenson's thesis is a very interesting, revealing personal account: by a person with a cleft palate, and as the patient receiving this seminal surgery.[8] Stephenson returned to Montreal to practice medicine, where he was the key person in the founding of McGill Medical School as the first College of McGill University.

For many years, cleft palate repair was clearly difficult for both patient and surgeon. It was not until 13 years after Stephenson's repair, in 1832 in Jefferson, Georgia, that Dr. Crawford W. Long introduced ether anesthesia, subsequently popularized in Boston. However, general anesthesia was not used for palate repair until about 1867, also the year that Joseph Lister, in Scotland, introduced the concept of asepsis in surgery.[9,10] Surgery was usually performed using outside sunlight until the 1880s, when Tesla's alternating current illuminated the 1883 Chicago World's Fair with Edison's light bulbs. In 1920, Magill introduced the endotracheal tube. Other aids such as epinephrine, intravenous fluids, lidocaine, suction, and the Dingman mouth gag did not become available until sometime in the 20th century.

In 1861, von Langenbeck described tension-relieving mucoperiosteal flap elevation through lateral relaxing incisions, which made hard palate closure easier, and must have reduced fistula rates.[11] It is an operation frequently used today, the repair this author learned and used before the double-opposing Z-plasty (DOZ-pl).[12] Variations include Bardach's two-flap repair, which detached von Langenbeck's flaps anteriorly for easier flap elevation and closure.[13]

In the 1920s, Veau, Dorrance, Wardill, and Kilner's pushback procedures lengthened the velum with hard palate mucoperiosteum. [14,15,16,17] However, without velar muscle lift, the added velar length did not significantly improve speech results, but palatal fistulas and midface retrusion increased.[18,19] The procedure is widely used today. The palatal island flap provided more hard palate mucoperiosteum for velar length,[20] resulting in greater retrusion.[21]

In the 1940s, an increase in midface retrusion, likely from the pushback's increased hard palate scarring, led to Schweckendiek's two-stage repair.[22,23] Its intent was to engage the repaired velum in speech while leaving the unrepaired hard palate mucoperiosteum unscarred for growth.

Maxillary growth was much improved, but the intentional hard palate fistula led to speech seriously marred by compensatory articulations.[24,25] Variations on the two-stage operation have generally moved the hard palate closure into early childhood or later in infancy.

In the 1960s, attention was finally turned to the velar muscles by Ruding, Braithwaite, and Kriens.[26,27,28] By most reports, the end-to-end, anterior intravelar veloplasty (IVV) led to some improvement in speech outcomes, but much room for improvement in speech outcomes remained. Today, IVV is usually a part of palate repair, regardless of design. Sommerlad's recent results with his careful IVV using the operating microscope has produced very good outcomes, even though velar lengthening is not part of the operative plan.[29]

From these history lessons of palate repair and an awareness of the aberrant anatomy, we may conclude that the best characteristics of a repair should

1. Put the levator muscles at functional tension by retroposing and transversely overlapping them.
2. Permanently lengthen the velum.
3. Provide complete oral and nasal mucosal coverage.
4. Leave the hard palate mucoperiosteum where it belongs.
5. Scar the hard palate as little as possible.
6. Complete the cleft repair by or close to age 1 year, in one operation if possible.

13.4.2 Double Opposing Z-Plasty Repair

The design of the double opposing Z-plasty repair achieves the six attributes noted above; patients' VP competency rate of the authors' went from 48% using a von Langenbeck repair either without or with IVV to 91% with double opposing P-plasty (▶ Table 13.1).[12,30,31]

The experience of others has confirmed its effectiveness.[32,33,34,35,36,37,38,39,40]

The operation has been much modified by others, usually by reducing the size of the Z-plasties and by freely using lateral relaxing incisions in the hard and soft palate, more recently by treating the muscles and their Z-plasty flaps as separate units.[40,41]

What follows briefly describes the Furlow version for the hard palate and the velum as the author finally came to perform it after his experience performing some 350 cases: 37 in private practice, and the rest later on volunteer surgery trips.

The DOZ-pl repair was developed in the author's private practice in which a consecutive series of 33 infants and 4 older patients (11 with various syndromes) were treated. Some 300 cases done on volunteer surgery trips added greatly to his operative experience. Much of what is described below was learned on these trips: from other surgeons, from the increasing experience, and the opportunity to do one case after another, remembering problems and solutions from case to case. Unfortunately, the lack of follow-up was very disappointing.

The soft palate cleft is closed with two large mirror-image Z-plasties; the cleft is the central limb of each Z-plasty. The palatal muscles are elevated as part of each posteriorly based flap; the anteriorly based flaps are mucosa only. As the lateral limb incision of the oral posteriorly-based flap is deepened to nasal mucosa, the palatal aponeurosis is *automatically* divided,

Table 13.1 33 infants at operation

	VP competence	Fistulas	Le Fort
UCLP	11/12	0	0
BCLP	9/10	0	2
CP	10/11	2	0
Total	30/33 (91%)	2 (6%)	2 (6%)

Abbreviations: BCLP, bilateral cleft lip and palate; CP, cleft palate; UCLP, unilateral cleft lip and palate; VP, velopharyngeal.

making velar relaxing incisions unnecessary (this author has never used them). The incision is carried to, *but not into* the superior constrictor laterally (▶ Fig. 13.1).[12,42]

The flap with its palatal muscle is *carefully* elevated from the nasal mucosa. A Freer elevator is used to push the levator posteriorly along the medial surface of the superior constrictor, and the flap with its levator is rotated transversely.[12,42]

Transposition of each z-plasty carries the levator in its posterior flap, overlapping the muscles. The anterior flaps close the oral and nasal mucosa. The large z-plasty design gives wide access for precise dissection and positioning of the flaps and their muscles.

In short, the velar Z-plasties repair the cleft, create a retroposed levator sling by rotating the levators transversely and overlapping them, lengthen the velum, leave the hard palate mucoperiosteum where it belongs, and close the oral and nasal mucosa anterior to the muscle sling, *all at the same time*.

Several technical details:

- The end of the lateral limb incisions of the anteriorly-based flaps position the tip of the myomucosal flaps, which sets the A-P direction, dorsal position, and overlap of the velar muscles. As the nasal lateral limb incision is carried laterally its end also curves posteriorly toward the posterior pharyngeal wall, so that a wide z-plasty constructs the levator sling nearer the posterior pharyngeal wall than a small one, under more functional tension, making VP closure easier for the patient.
- The left nasal mucosal Z-plasty flap ends precisely under the levator muscle base to position the right levator tip *directly* under the left levator base, creating a transverse levator sling, its levators under functional tension.[30]
- The oral myomucosal flap tip with its levator muscle is inset directly over the opposite levator base, *not* farther anteriorly over the hamulus ▶ Fig. 13.2.[30]
- An intraoperative view of widely overlapped exactly aligned levators is what one should see before the final oral mucosal z-plasty flap is placed.[31]
- The anteriorly-based flaps provide complete mucosal closure anterior to the velar muscles, so the levator sling is not pulled forward over time by scar contracture, shortening the velum ▶ Fig. 13.3.[12]
- Guneren and Uysal found a mean increased velar length of 12.45 mm (55%) 4.5 years after DOZ-pl.[44]
- Other helpful variations are described in Furlow.[30]

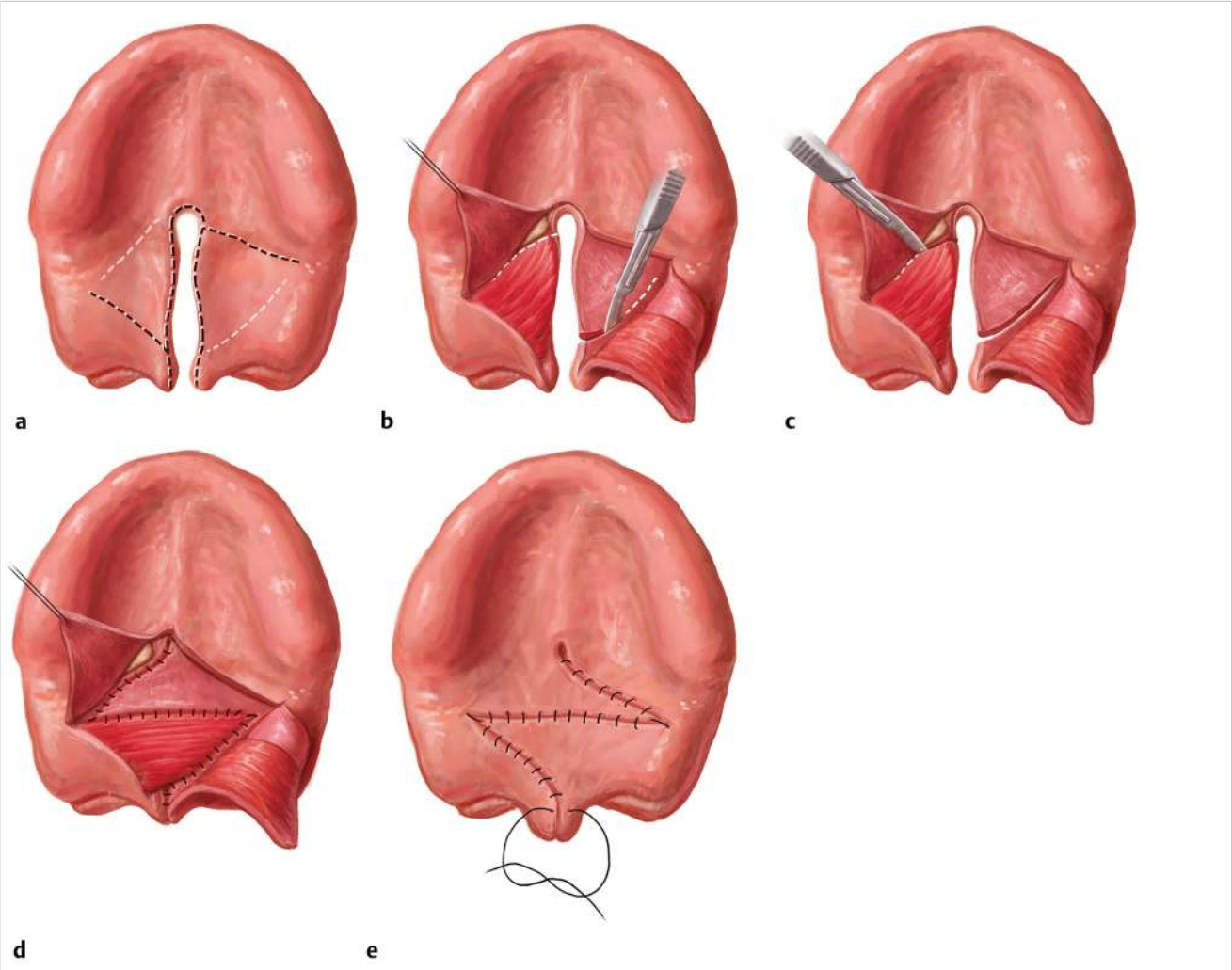


Fig. 13.1 Double opposing Z-plasty: soft palate operative design. (**a**) Oral-side Z-plasties, (**b**) Oral-side flaps reflected, (**c**) Nasal flaps dissected, (**d**) and transposed. (**e**) Levator sling constructed. Anterior flaps close mucosa. Losee J, Kirschner R, ed. Comprehensive Cleft Care. 2nd Edition. New York: Thieme; 2015.

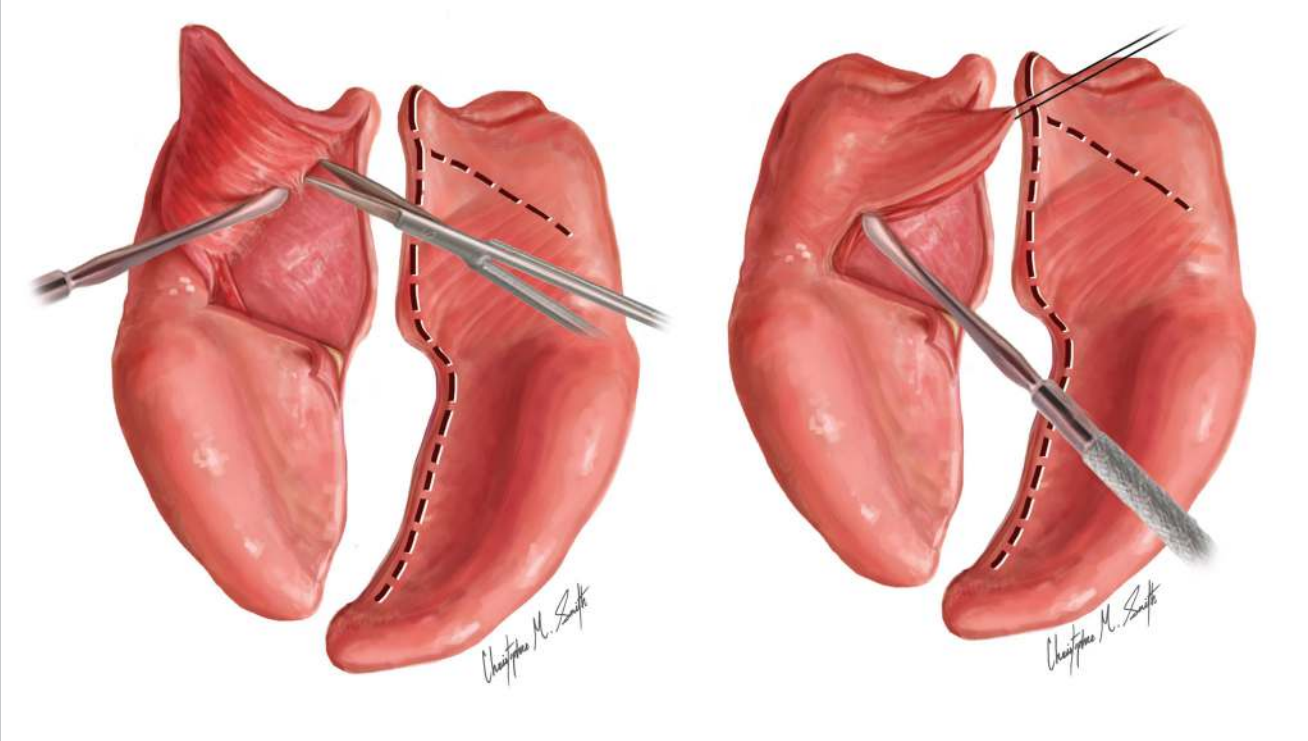


Fig. 13.2 Dissection of the posteriorly-based flap. Losee J, Kirschner R, ed. Comprehensive Cleft Care. 2nd Edition. New York: Thieme; 2015.

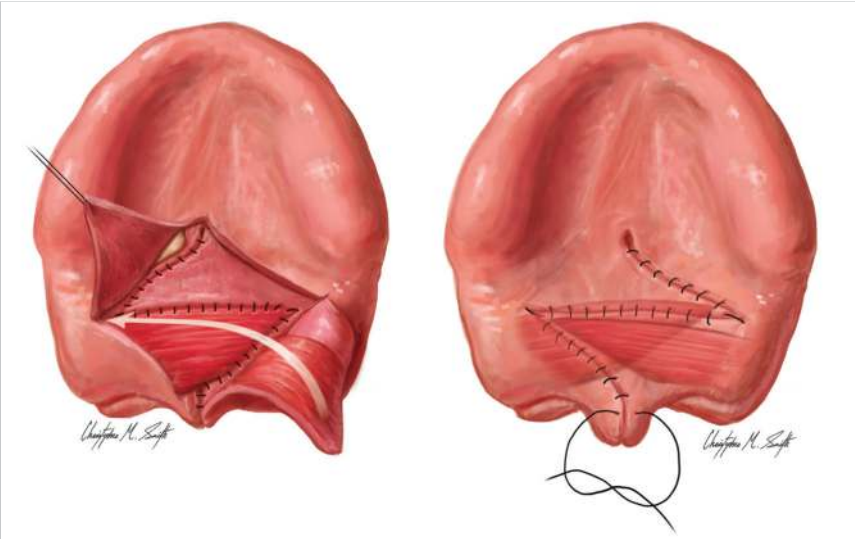


Fig. 13.3 Inset of the double opposing Z-plasty flaps. The anteriorly-based flaps provide complete mucosal closure anterior to the velar muscles, so the levator sling is not pulled forward over time by scar contracture, shortening the velum. Upon inset, the levators widely overlap. Losee J, Kirschner R, ed. Comprehensive Cleft Care. 2nd Edition. New York:Thieme; 2015.

13.5 Hard Palate

Lateral relaxing incisions and pushback procedures are to be avoided because they add hard palate mucosal scars and bare areas. The greater the hard palate mucoperiosteal deficit the greater the midface growth deficit.[18,21]

When the mucoperiosteal flaps are brought from the vault to horizontal, lateral relaxing incisions are infrequently needed.[42]

The relationship of palatal vault to the distance from alveolus to alveolus, *not cleft width*, is critical for closure without lateral relaxing incisions. *To avoid lateral relaxing incisions the mucoperiosteal flaps must reach from the base of each alveolus to each other at the cleft*.

When the cleft is wide, if the vault of the hard palate is high, mucoperiosteal flaps may reach each other when they are brought out of the vault to horizontal, whereas if the cleft is narrower but the vault is flatter, mucoperiosteal flaps may not reach.[31]

To determine if the flaps will reach, plan ahead. *Before beginning surgery*, at the hard-soft palate junction measure the

distance on each side from the cleft margin to the base of the alveolus, which is the breadth of each mucoperiosteal flap that can be brought out of the vault to horizontal to close the cleft. If the two measurements will bridge the gap from alveolus to alveolus, lateral relaxing incisions will not be necessary. If they will not reach, an alternate plan can be made before beginning the operation.

Two q-tips can be used to measure the available mucoperiosteal flap width by placing the end of each q-tip stick (the handle) on the cleft margin of each side at the hard-soft palate junction and marking the base of that side's alveolus with a pen or methylene blue, then adding one handle marking to the other q-tip handle to mark the sum of the mucoperiosteal flap breadths. Bending the q-tip at the mark permits the segment to be put across the gap from alveolus to alveolus. If it reaches, proceed without lateral relaxing incisions.[31]

If it won't reach, after doing velar DOZ-pl, do lateral relaxing incisions, 2-flap elevation and closure, or another plan for hard palate closure.[45,46]

When closing the hard palate, to avoid fistulas, *always use mattress sutures*. The raw surfaces of each mucoperiosteal flap must be in apposition; a raw edge is unlikely to heal to a mucosal surface.

13.6 Postoperative Care

The main immediate concerns are with airway and bleeding; a pulse-oximeter is indicated. Traction on a tongue stitch placed at the end of the palatoplasty can be very helpful in preventing airway obstruction, and sedation and elevation of the head of the infant's bed will stop most postoperative oozing. As soon as the infant is awake, the moistening from drinking from a cup or syringe may be calming and safe, and may reduce the need for pain medicine. If analgesics are needed, acetaminophen or ibuprofen is nearly always effective. Elbow restraints are probably not necessary.

When the infant is drinking clear liquids, the intravenous fluids can be discontinued and the infant sent home on a liquid diet if the parents are reliable and comfortable with providing care.

Initial follow-up should be in about 2 weeks after hospital discharge, and thereafter in craniofacial clinic by its schedule.

13.7 Outcomes

13.7.1 Long-Term Follow-up

Adequate follow-up for speech, hearing, and midface growth should be continued at least until the late teens. Hearing should be evaluated by the team's otolaryngologist or audiologist, and maxillary growth status evaluated by the dental colleagues. Follow-up evaluations should be shared, discussed, gathered, and saved in a way that preserves evidence of outcomes and results for documentation of effectiveness of treatment.

As an example, a 34 year-old patient with a Veau II cleft had never been told it could be repaired (▶ Fig. 13.4). Velum elevates well at two weeks and at two years. She developed velopharyngeal competence, her increased velar length persists, and her "dimple of elevation" remains well posterior. Her articulation was good before her repair and her speech became normal.

13.7.2 Determining Velopharyngeal Function

The prime purpose of cleft palate repair is to effect VP competence to make normal speech. Therefore, our first most important question after surgery should be "is the velopharyngeal valve competent?" If it is not, normal speech is not likely to occur.

After palatoplasty, the SLP's charge is to evaluate speech, and if it is not normal, to determine the cause and to plan treatment. If the VP mechanism cannot close adequately (VPI), surgical or prosthetic treatment is required. If the problem is in the brain, in the form of compensatory articulations mislearned because the patient's VP mechanism could not close when he/she learned to speak, speech therapy by the SLP is needed, not surgery.[5,6,7] If both VPI and compensatory articulations are present, it is likely that speech therapy and surgery will both be needed. The treatment path depends on knowing if the VP mechanism can close, and may involve tests such as videofluoroscopy and videonasendoscopy.[4] Of course, if speech is normal under all circumstances, only follow-up is indicated.

The two direct consequences of VPI are nasal escape of air on consonants and sibilants, and hypernasality on normally nonnasal vowel sounds. To determine their presence or absence, SLPs listen to their patients speak. Accurate detection and grading of nasal escape and hypernasality based on detection in speech has been difficult. For example, the audible "nasal rustle" is heard with smaller but not larger VP gaps.[47,48] Conversely, some people without VPI sound hypernasal; many country music singers are an example. This and failure to discriminate between VPI amd compensatory articulations due to mislearning have made standardizing evaluation of speech outcomes between teams and institutions a vexatious, unsolved problem which has foiled treatment comparisons and identification of "best treatments".[49] Most rating scales include minimal or intermittent VPI in the "success" category, even though when surveyed, SLP's and surgeons say success should be limited to "no VPI" or "normal speech".[49,50,51] Some institutions have used "need for surgery for VPI" as the outcome measure, but there is no standardization among teams and institutions as to the degree of VPI that is minor enough to accept without treatment, which may include nearly half the patients with some degree of residual VPI.[52]

Half a century ago, Warren's pressure-flow studiesgave us "yes/no" physical criteria for VP competence.[1,53] He showed that a residual velopharyngeal opening (or a palatal fistula) smaller than 2.5 mm in diameter (5 mm^2 area) resulted in normal speech, and a mm larger in diameter (3.6 mm, 10 mm^2 area) resulted in speech marred by VPI (a pencil eraser's diameter is 7.1 mm, area 40 mm^2). He also documented that nasal airflow less than 50 cc/second on consonants resulted in normal speech. Unfortunately, his pressure-flow studies are too complex to use at routine clinic visits.

Fortunately for surgeons, "listening tube" turbulence for nasal escape on consonants and sibilants, and cul-de-sac resonance sound change for hypernasality on vowels do not depend on expert evaluation of speech. Rather, test sounds elicited by single syllables that normally cause velopharyngeal closure determine whether the velopharyngeal mechanism is closing. The two tests are quick, free chairside clinic tests that a surgeon can

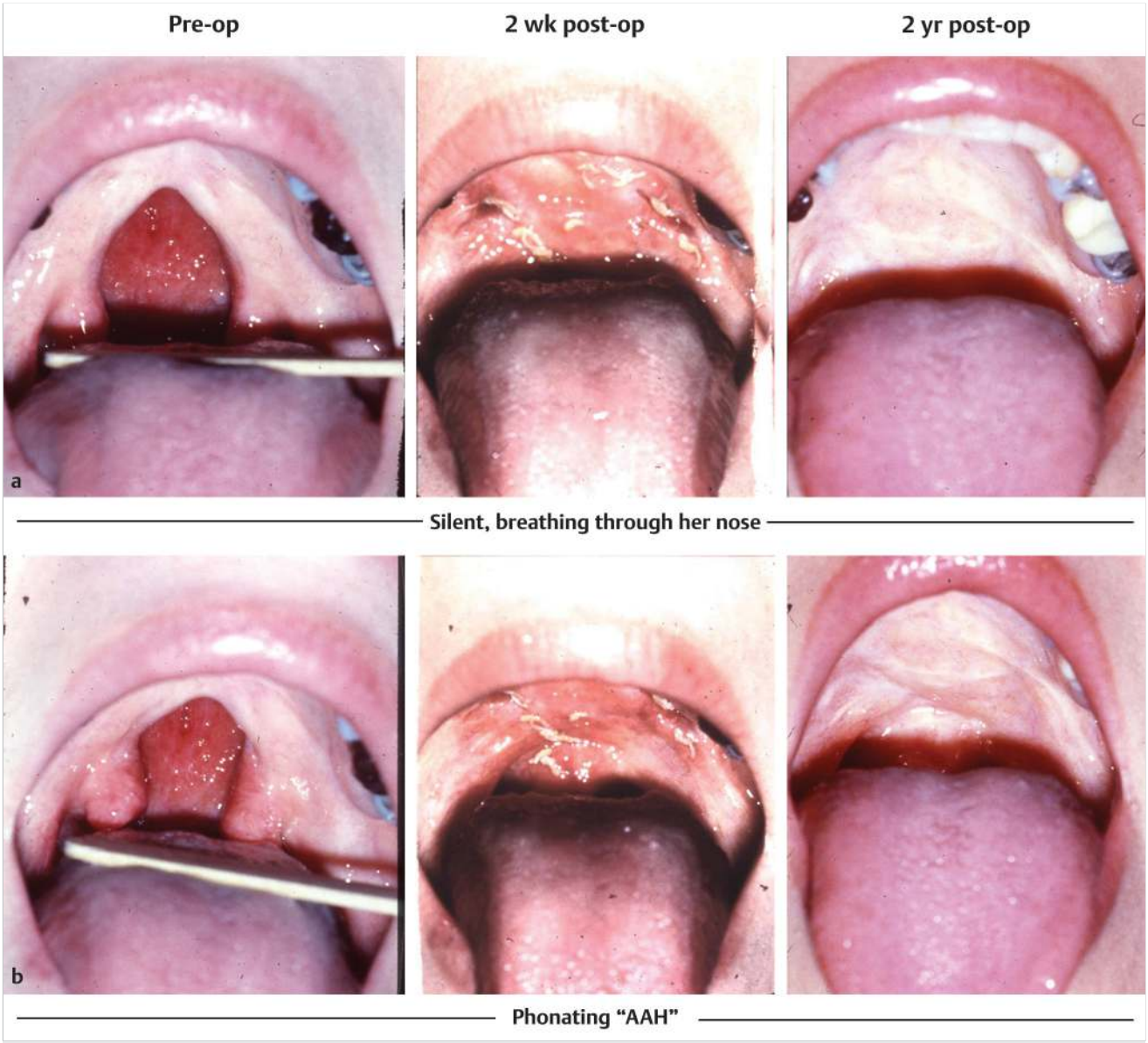


Fig. 13.4 (**a**) The top row of photos shows the palate in the silent, breathing state, while (**b**) the bottom row demonstrates the palate during phonation. The left column is prior to double opposing Z-plasty palatoplasty. The middle is 2 weeks after surgery, and right column is 2 years after surgery. Note the elevation of the velum with phonation, and the dimpling indicative of muscle activity.

perform and evaluate, don't depend on evaluation of speech, do give a "yes / no" answer rather than degrees of VPI, and should therefore be comparable between teams and institutions.

The "Listening tube" test identifies nasal escape by removing the head of a stethoscope, holding the tube end at each of the patient's nostrils as the patient says "beet... bit... bait... bet... bat... bot... boat... boot... but... bert," syllables that should cause the velopharyngeal mechanism to close. Nasal airflow (escape) will create very audible turbulence in the end of the tube. *The sound comes from the turbulence in the stethoscope end, not from the patient*, whose nasal airflow causing the turbulence may be silent, which is more likely the larger the residual velopharyngeal gap.[47]

The cul-de-sac test detects hypernasality by having the patient say each syllable twice: "beet / beet… bit / bit… bait / bait…" etc., pinching the nose for each *second* syllable. If the VP valve is *closed*, the sound will not change, because the nasal cavity is *not* part of the resonating chamber. If the VP valve is *not closed*, the sound will be different with the nose open and with the nostrils pinched because when the nostrils are pinched, the nasal cavity is changed from an open-ended to a closed-ended resonating chamber.[52]

If these two tests were the standard, it might well solve the problem of comparison of results between teams and institutions, and would tell us whether to plan for surgery or speech therapy, or both.

13.8 Review Questions

13.8.1 Choose the Best Answer

1. The soft palate
   a) Is an insignificant structure.
   b) Functions to support the uvula.
   c) Acts as a muscular valve controlling airflow into the nasal cavity during speech.
   d) Is inconsequential for speech development.
2. The timing of palate repair is
   a) Solely related to the size of the cleft.
   b) Dictated by degree of facial growth in order to allow surgery.
   c) Should be done prior to permanent abnormal compensatory speech patterns develop.
   d) Dependent of the patient obtaining a minimum weight of 10 kg.
3. The double opposing Z-plasty
   a) Creates a functional muscular velum via levator muscle retropositioning and overlap.
   b) Lengthens the velum.
   c) Provides oral and nasal mucosal coverage.
   d) Leaves the hard palate mucoperiosteum where it belongs.
   e) All of the above.
4. Hard palate repair done in conjunction with double opposing Z plasty for the soft palate
   a) Will never work.
   b) Can frequently be done without relaxing incisions.
   c) Is done with mucosal flap leaving periosteum in place.
   d) Should always be done with relaxing incision.
   e) None of the above.
5. Outcomes for cleft palate repair
   a) Are best judged by speech and the absence of VPI.
   b) Rarely require further surgery.
   c) Never have effects on facial growth.
   d) Are independent of the function of the velum.

13.8.2 Answers

1. Acts as a muscular valve controlling airflow into the nasal cavity during speech. (c)
2. Should be done prior to permanent abnormal compensatory speech patterns develop. (c)
3. All of the above. (e)
4. Can frequently be done without relaxing incisions. (b)
5. Are best judged by speech and the absence of VPI. (a)

References

[1] Warren DW. Velopharyngeal orifice size and upper pharyngeal pressure-flow patterns in normal speech. Plast Reconstr Surg. 33:148–162
[2] Warren DW. Aerodynamic Assessments and Procedures to Determine Extent of Velopharyngeal Inadequacy. In: Bzoch, ed. Communicative Disorders Related to Cleft Lip and Palate. 5th ed. Austin Texas: Pro-Ed; 2004: Chap. 17
[3] Thompson AE, Hixon TJ. Nasal air flow during normal speech production. Cleft Palate J. 16(4):412–420
[4] Bzoch KR. A Battery of Clinical Perceptual Tests, Techniques, and Observations for the Reliable Clinical Assessment. In: Bzoch, ed. Communicative Disorders Related to Cleft Lip and Palate. 5th ed. Austin Texas: Pro-Ed; 2004: Chap. 12
[5] Isberg A, Henningsson G. Influence of palatal fistulas on velopharyngeal movements: a cineradiographic study. Plast Reconstr Surg. 79(4):525–530
[6] Henningsson GE, Isberg AM. Velopharyngeal movement patterns in patients alternating between oral and glottal articulation: a clinical and cineradiographical study. Cleft Palate J. 23(1):1–9
[7] Henningsson G, Isberg A. A cineradiographic study of velopharyngeal movements for deviant versus nondeviant articulation. Cleft Palate Craniofac J. 28 (1):115–117, discussion 117–118
[8] Furlow LT , Jr. John Stephenson's cleft palate. Cleft Palate Craniofac J. 47(6): 561–565
[9] Long CW. An account of the first use of sulphuric ether by inhalation as an anesthetic in surgical operations. South Med Surg J. 5(12):705–713
[10] Young JH. Crawford W. Long, M.D., a Georgia innovator. Bull N Y Acad Med. 50(3):421–437
[11] Lindsay WK, Witzel MA. Cleft Palate Repair: Von Langenbeck Technique. In: Bardach J, Morris HL, eds. Multidisciplinary Management of Cleft Lip and Palate, Philadelphia: WB Saunders Co.; 1990: Chapter 36
[12] Furlow LT , Jr. Cleft palate repair by double opposing Z-plasty. Plast Reconstr Surg. 78(6):724–738
[13] Bardach J. Cleft palate repair: Two-flap palatoplasty, research, philosophy, technique and results. In: Bardach J, Morris HL, eds. Multidisciplinary Management of Cleft Lip and Palate, Philadelphia: WB Saunders Co.; 1990:303–308. Chapter 46
[14] Wallace AF. A history of the repair of cleft lip and palate in Britain before World War II. Ann Plast Surg. 19(3):266–275
[15] Dorrance GM, Bransfield JW. The push-back operation for repair of cleft palate. Plast Reconstr Surg (1946). 1:145–169
[16] Calnan JS. V-Y Pushback Palatoplasty. In: Bardach J, Morris HL, eds. Multidisciplinary Management of Cleft Lip and Palate, Philadelphia: WB Saunders Co.; 1990:422–431. Chapter 29
[17] Cronin TD. Pushback Palatorrhaphy with Nasal Mucosal Flaps. In: Bardach J, Morris HL, eds. Multidisciplinary Management of Cleft Lip and Palate, Philadelphia: WB Saunders Co.; 1990:432–440. Chapter 30
[18] Pickrell K, Clifford E, Quinn G, Massengill R. Study of 100 cleft lip-palate patients operated upon 22 to 27 years ago by one surgeon. Plast Reconstr Surg. 49(2):149–155
[19] Blocksma R, Leuz CA, Mellerstig KE. A conservative program for managing cleft palates without the use of mucoperiosteal flaps. Plast Reconstr Surg. 55 (2):160–169
[20] Batstone JHF, Millard DR. Pushback Palatorrhaphy with Island Flap to the Nasal Surface. Chapter 31, pp 441–447. In: Grabb WC, Rosenstein SW, Bzoch KR, eds. Cleft Lip and Palate, Boston: Little Brown and Company; 1971
[21] Seckel NG. The Palatal Island Flap on Retrospection. Discussion: Furlow LT, Jr. Plast Reconstr Surg. 96:1269
[22] Schweckendiek W, Kruse E. Two-Stage Palatoplasty: Schweckendiek Technique. In: Bardach J, Morris HL, eds. Multidisciplinary Management of Cleft Lip and Palate, Philadelphia: WB Saunders Co.; 1990:315–320. Chapter 39
[23] Schweckendiek W, Doz P. Primary veloplasty: long-term results without maxillary deformity. a twenty-five year report. Cleft Palate J. 15(3):268–274
[24] Bardach J, Morris HL, Olin WH. Late results of primary veloplasty: the Marburg Project. Plast Reconstr Surg. 73(2):207–218
[25] Cosman B, Falk AS. Delayed hard palate repair and speech deficiencies: a cautionary report. Cleft Palate J. 17(1):27–33
[26] Ruding R. Cleft palate: anatomic and surgical considerations. Plast Reconstr Surg. 33:132–147
[27] Braithwaite F, Maurice DG. The importance of the levator palati muscle in cleft palate closure. Br J Plast Surg. 21(1):60–62
[28] Kriens OB. An anatomical approach to veloplasty. Plast Reconstr Surg. 43(1): 29–41
[29] Sommerlad BC. A technique for cleft palate repair. Plast Reconstr Surg. 112 (6):1542–1548
[30] Furlow LT , Jr. Cleft palate repair by double opposing z-plasty. Operative Techniques in Plast Reconstr Surg. 2(4):223–228
[31] Furlow LT Jr. Double Opposing Z-Plasty Palate Repair. In: Losee JE, Kirschner RE, eds. Comprehensive Cleft Care. New York: McGraw Hill; 2009. Chapter 23, pp. 371–389.
[32] Pet MA, Marty-Grames L, Blount-Stahl M, et al. The Furlow palatoplasty for velopharyngeal dysfunction: velopharyngeal changes, speech improvements, and where they intersect. Cleft Palate Craniofac J. 52(1):12–22
[33] Lin KY, Goldberg D, Williams C, et al. Long-term outcome analysis of two treatment methods for cleft palate: Combined levator retropositioning and pharyngeal flap versus double opposing z-plasty. Cleft Palate Craniofac J. 63: 462

[34] Timbang MR, Gharb BB, Rampazzo A, et al. A systematic review comparing Furlow double-opposing Z-plasty and straight-line intravelar veloplasty methods of cleft palate repair. Plast Reconstr Surg. 134(5):1014–1022
[35] Spauwen PH, Goorhuis-Brouwer SM, Schutte HK. Cleft palate repair: Furlow versus von Langenbeck. J Craniomaxillofac Surg. 20(1):18–20
[36] Grobbelaar AO, Hudson DA, Fernandes DB, et al. Speech results after repair of the cleft soft palate. Plast Reconstr Surg. 95(7):1150–1154
[37] McWilliams BJ, Randall P, LaRossa D, et al. Speech characteristics associated with the Furlow palatoplasty as compared with other surgical techniques. Plast Reconstr Surg. 98(4):610–619, discussion 620–621
[38] Gunther E, Wisser JR, Cohen MA, et al. Palatoplasty: Furlow's double reversing Z-plasty versus intravelar veloplasty. Cleft Palate Craniofac J. 35(6):546–549
[39] Kirschner RE, Wang P, Jawad AF, et al. Cleft-palate repair by modified Furlow double-opposing Z-plasty: the Children's Hospital of Philadelphia experience. Plast Reconstr Surg. 104(7):1998–2010, discussion 2011–2014.–Furlow LT
[40] LaRossa D, Kirschner RE, Low D. The Children's Hospital Modification of the Furlow Double Opposing Z-palatoplasty. In: Losee JE, Kirschner RE, eds. Comprehensive Cleft Care. New York: McGraw Hill; 2009:391–397. Chapter 24
[41] Furlow LT, Randall P, Mathes SJ. Moderator. Double Opposing Z-Plasty in Cleft Palate Repair: Technique, Results and Analysis. Perspect Plast Surg. 7:55
[42] Furlow LT , Jr. Flaps for cleft lip and palate surgery. Clin Plast Surg. 17(4):633–644
[43] Procedures in Plastic and Reconstructive Surgery. How They Do It. In: Vistnes LM, ed. Boston: Little Brown; 1993. Ch 19. Furlow L. Cleft Palate, Fig. 19.5, Pg. 354
[44] Guneren E, Uysal OA. The quantitative evaluation of palatal elongation after Furlow palatoplasty. J Oral Maxillofac Surg. 62(4):446–450
[45] Bozola AR, Gasques JA, Carriquiry CE, et al. The buccinator musculomucosal flap: anatomic study and clinical application. Plast Reconstr Surg. 84(2):250–257
[46] Mann RJ, Fisher DM. Bilateral buccal flaps with double opposing Z-plasty for wider palatal clefts. Plast Reconstr Surg. 100(5):1139–1143, discussion 1144–1145
[47] Kummer AW, Briggs M, Lee L. The relationship between the characteristics of speech and velopharyngeal gap size. Cleft Palate Craniofac J. 40(6):590–596
[48] Kummer AW. Disorders of resonance and airflow secondary to cleft palate and/or velopharyngeal dysfunction. Semin Speech Lang. 32(2):141–149
[49] Chapman KL, Baylis A, Trost-Cardamone J, et al. The Americleft Speech Project: A Training and Reliability Study. Cleft Palate Craniofac J. 53(1):93–108
[50] Kummer AW, Clark SL, Redle EE, Thomsen LL, Billmire DA. Current practice in assessing and reporting speech outcomes of cleft palate and velopharyngeal surgery: a survey of cleft palate/craniofacial professionals. Cleft Palate Craniofac J. 49(2):146–152
[51] Witt PD, O'Daniel TG, Marsh JL, Grames LM, Muntz HR, Pilgram TK. Surgical management of velopharyngeal dysfunction: outcome analysis of autogenous posterior pharyngeal wall augmentation. Plast Reconstr Surg. 99(5):1287–1296, discussion 1297–1300.
[52] Furlow LT. Secondary Cleft Palate Surgery. In: Reoperative Aesthetic and Reconstructive Surgery. 2nd ed. In: Grotting JC, ed. St. Louis, Missouri: Quality Medical Publishing, 2007. Ch. 30, Pp. 1053- 1112. Pp. 1073, 1075. Figs. pg 1058, 1059
[53] Warren DW. Aerodynamic Assessments and Procedures to Determine Extent of Velopharyngeal Inadequacy. In: Bzoch, ed. Communicative disorders Related to Cleft Lip and Palate. 5th ed. Austin Texas: Pro-Ed; 2004:596–605. Chap. 17

14 Craniosynostosis

Jessica A. Ching and John H. Phillips

Abstract
Craniosynostosis refers to the premature fusion of one or more cranial sutures. This often results in characteristic cranial distortions in infancy that prompt investigation with imaging. Once craniosynostosis is confirmed, surgical intervention may include cranioectomy, frontoorbital advancement, posterior cranial vault remodeling, total cranial vault remodeling, or posterior vault distraction. The intervention is chosen based on the sutures involved, severity of symptoms, and if an underlying syndrome is present. A discussion of postoperative care guidelines and outcomes conclude the chapter.

Keywords: syndromic craniosynostosis, posterior plagiocephaly, sagittal synostosis, metopic synostosis, cranioectomy, Virchow's law

14.1 Goals and Objectives

- Recognize the key historical and physical findings needed for diagnosing and treating craniosynostosis.
- Clearly define the objectives of surgical treatment of craniosynostosis.
- Appreciate the complex surgical procedures utilized in treating patients with craniosynostosis, including the appropriate indications and timing of these procedures.
- Understand the evidence-based preoperative and postoperative concerns related to craniosynostosis.

14.2 Patient Presentation

During the first year of life, infants are frequently referred to a plastic surgeon or neurosurgeon for an evaluation of their head shape. The primary concern of the referring provider is usually underlying craniosynostosis, or the premature fusion of one or more cranial sutures. Craniosynostosis may occur as the result of an isolated, sporadic genetic mutation or as part of a complex syndromic diagnosis. Overall, craniosynostosis occurs in 1:2,000, and syndromic craniosynostosis is even more rare, affecting 1:25,000 to 1:100,000 infants.[1] The most common type of craniosynostosis is sagittal synostosis, followed by metopic synostosis, then unicoronal synostosis.[1] Since the "Back to Sleep" campaign began by the American Academy of Pediatrics in 1992, deformational plagiocephaly is much more common than craniosynostosis, nearly as frequent as 1:12.[1,2,3] Positional plagiocephaly is managed nonsurgically with observation or helmeting, while craniosynostosis is often treated surgically. This makes correctly diagnosing craniosynostosis of critical importance.

History and physical examination will assist in determining if further diagnostic workup is necessary. Neonatal and gestational history including pregnancy complications and exposures should be obtained. A description of the child's head shape at birth and subsequent shape changes leading to the present visit is important in assessing if the deformity is improving, worsening, or stable. Craniosynostosis generally worsens as the skull growth attempts to compensate for the fused suture. Family histories of similar cranial findings or genetic disorders is pertinent. Developmental milestone achievement should be delineated, noting any signs of delay or regression that may raise concern of increased intracranial pressure secondary to craniosynostosis or other pathology. Other symptoms of increased intracranial pressure can include inconsolability, head holding, drowsiness, or emesis.[1,4]

Clinical examination first focuses on general head shape and identifying asymmetries. According to Virchow's law, early closure of a cranial suture restricts perpendicular growth but causes a compensatory increase of parallel skull growth (▶ Fig. 14.1).[5] Therefore, characteristic head shapes are often linked with specific craniosynostosis patterns (▶ Fig. 14.2). However, these associations are not always present, and in some cases, the head shape may appear normal even with multiple fused sutures.[4,6] Conversely, the head shape may appear grossly abnormal without any underlying craniosynostosis, such as with positional plagiocephaly. During examination, it is also necessary to palpate sutures and fontanelles. A ridge of bone at a suture line or fontanelle closure may indicate premature suture fusion. An open fontanelle should be soft when an infant is calm and upright. If it is full or tense, this merits consideration of increased intracranial pressure. Additional examination of facial features and extremities should be performed as these may indicate an underlying syndromic diagnosis (▶ Table 14.1). The patient's neck should be examined for range of motion to evaluate for contributing torticollis. If exorbitism, eye irritation, or exposure keratitis is noted, prompt referral to an ophthalmologist is needed.

Some patients present with radiographic imaging. Plain radiographs of the skull are of low diagnostic value in this setting, and their results should not overshadow historical and clinical findings. In cases where preoperative imaging is desired, plain radiographs are likely inadequate as well.

14.3 Preparation for Surgery

If the initial consultation yields suspicion of craniosynostosis, referral to a craniofacial surgeon at a tertiary center is necessary, and further consultations and imaging should be considered. Standard consultations at most specialized tertiary centers include neurosurgery and ophthalmology. Neurosurgery will evaluate for cranial pathology and potential surgical assistance, and ophthalmology will evaluate for papilledema or optic atrophy indicative of increased intracranial pressure. Additional consultations may include genetics to assess for a syndromic diagnosis, associated laboratory evaluations, and counseling. If a syndromic diagnosis is suspected or the patient has complex medical issues, a formal craniofacial team evaluation and follow-up is warranted. At tertiary centers, the craniofacial surgeon is often one member of the craniofacial team that will care for complex craniofacial patients. Patients with syndromic associations and complex medical issues are appropriately referred by

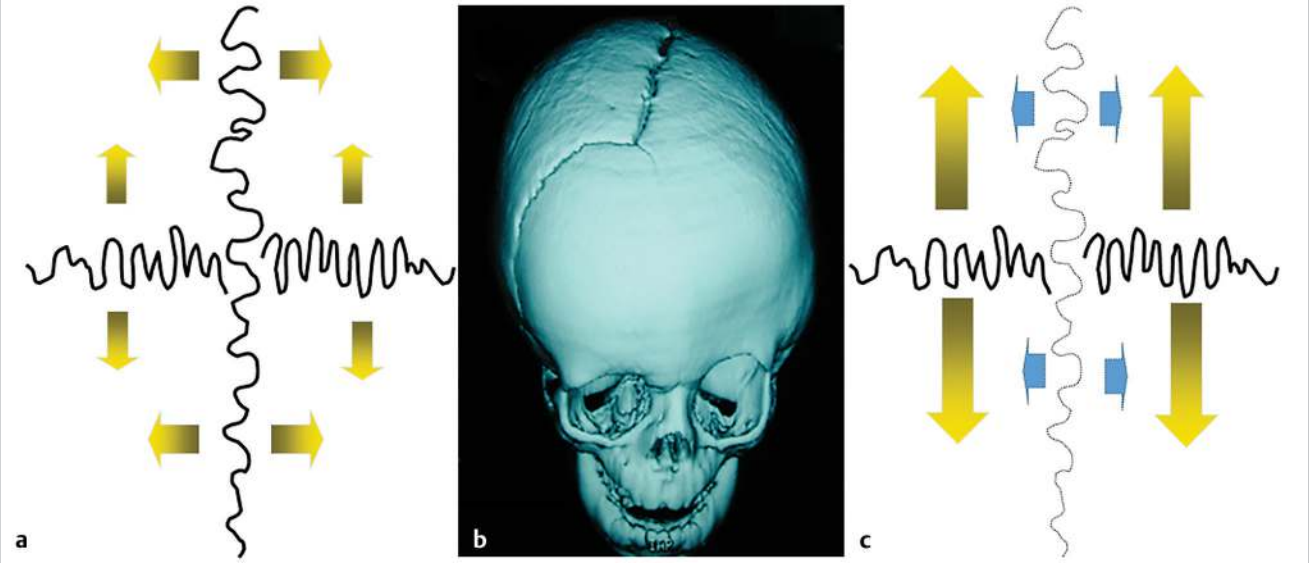


Fig. 14.1 **(a)** Virchow's law is represented with a diagram of an open, unfused suture displaying both perpendicular and parallel growth to the suture. **(b)** This is contrasted to the patient with left unicoronal synostosis and **(c)** diagram of Virchow's law at a prematurely fused suture, exhibiting restricted perpendicular growth and increased parallel growth along the suture.

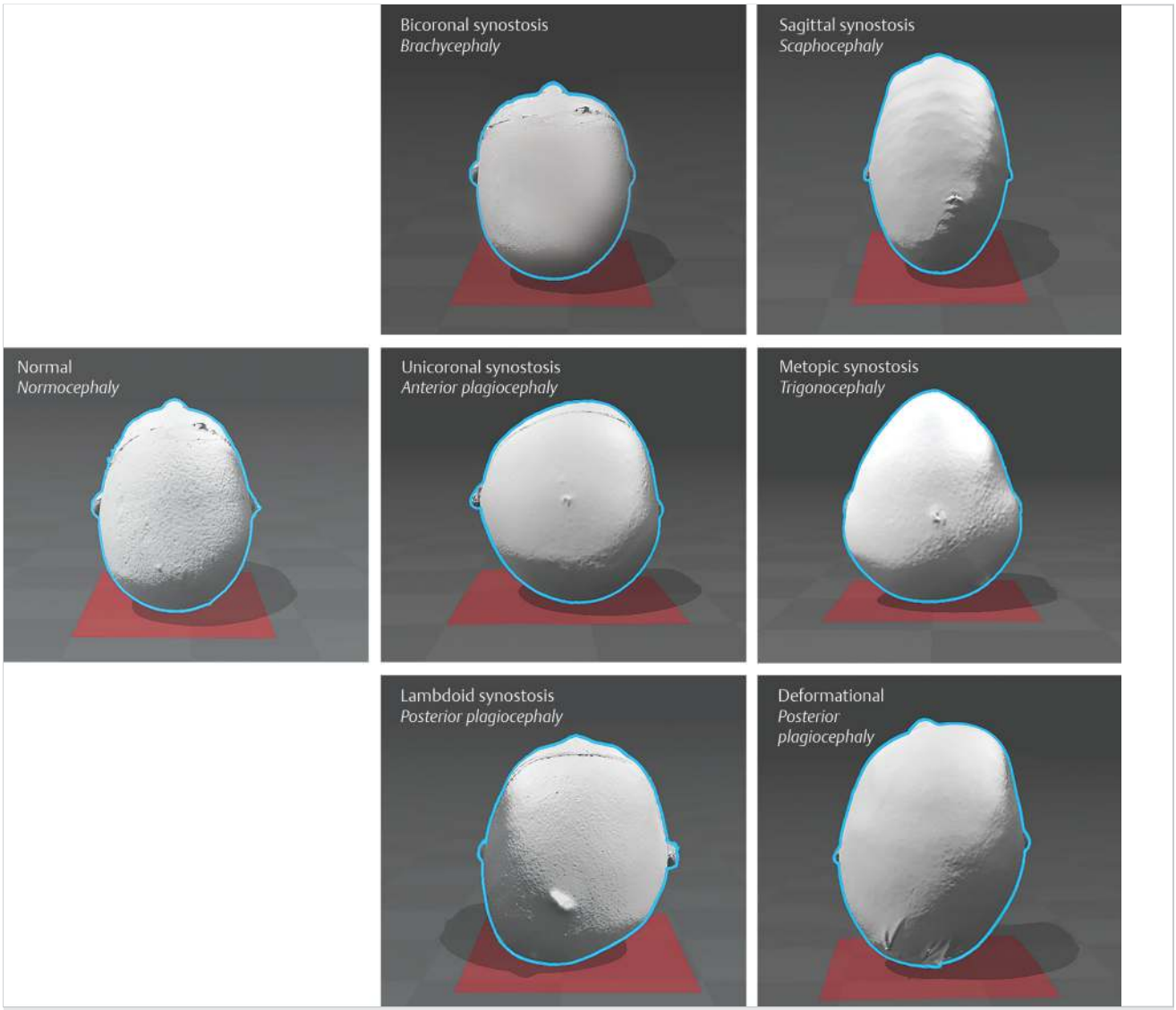


Fig. 14.2 A schematic of typical head shapes associated with specific patterns of sutural fusion.

Table 14.1 Additional exam findings associated with syndromic craniosynostosis

Syndrome	Suture fusion pattern	Facial appearance	Upper extremity features	Lower extremity features
Crouzon	Bicoronal	Maxillary hypoplasia with relative mandibular prognathism, exorbitism, hypertelorism	Normal	Normal
Apert	Bicoronal	Midface retrusion, exorbitism, hypertelorism	Complex syndactyly, radiohumeral fusion	Complex syndactyly
Pfeiffer	Multisuture, variable	Midface retrusion, exorbitism, hypertelorism	Broad thumb with medial deviation, possible brachydactyly	Broad great toes with medial deviation, possible brachydactyly
Saethre-Chotzen	Unilateral or bilateral coronal	Bilateral upper lid ptosis, low hairline, prominent superior transverse crus of the helix, rare midface retrusion	Simple partial syndactyly of the second and third web spaces	Simple partial syndactyly of the second and third web spaces
Muenke	Unilateral or bilateral coronal	No midface retrusion	Brachydactyly, possible carpal fusion	Broad great toes, possible tarsal fusion

the team to additional specialists for evaluation and treatment as needed. Preoperative evaluation by an anesthesiologist familiar with pediatric craniofacial patients is performed to ensure perioperative risk is avoided or mitigated as much as possible.

In most cases, preoperative imaging is desired to confirm the diagnosis, identify other abnormal anatomy, such as emissary veins, and assist with overall surgical planning. A computed tomography (CT) scan without contrast is commonly performed, and three-dimensional reconstruction images are obtained. The CT is performed with contrast if vascular drainage is a concern, as in syndromic craniosynostosis.[7] If warranted, magnetic resonance imaging (MRI) may provide additional information about intracranial pathology or posterior fossa malformations, such as tonsillar herniation.

Timing of the operation depends on the severity of the patient's symptoms. If there are signs of increased intracranial pressure or developmental concerns, surgery proceeds as soon as is feasible. In the absence of increased intracranial pressure, surgery is generally performed within the first year of life, while the exact timing depends on the underlying diagnosis and surgical technique.

Once the surgical strategy and date are determined, patients can be placed on iron supplementation or given recombinant erythropoietin several weeks prior to surgery to optimize hemoglobin levels.[8] Preoperative labs generally include a complete blood count and basic metabolic panel within 30 days prior to surgery.

The most common complications are asymmetry, contour deformities, superficial wound infection, scarring, and wound healing issues. Other risks of any operation for craniosynostosis include bleeding, pain, deep wound infection, blood transfusion, dural tears, cerebrospinal fluid leaks, meningitis, encephalitis, neurologic damage and impairment, and death. These risks should be discussed with caregivers well in advance of surgery.

14.4 Treatment

14.4.1 Indications

Elevated intracranial pressure is an urgent indication for surgical intervention, as it means the brain is externally compressed by the calvarium. In the case of craniosynostosis, the calvarium is unable to accommodate brain growth. The most rapid growth occurs in the first year of life, and the brain nearly triples in size by 2 years of age.[4] Thus, if craniosynostosis is present, as the brain grows the elevated intracranial pressure will likely worsen. Generally, the risk of elevated intracranial pressure increases with each additional suture fusion, and those with syndromic diagnoses have the highest rate of elevated intracranial pressure.[4,9,10] A craniofacial dysostoses syndrome may be associated with ventriculomegaly, venous outflow obstruction, or obstructive sleep apnea, all of which contribute to elevated intracranial pressure. It is associated with multisuture craniosynostosis over 40% of the time and associated with single suture nonsyndromic craniosynostosis over 15% of the time.[1,11] If guardians elect not to proceed with surgery, the patient should be followed up at regular intervals until several years of age to avoid an undiagnosed late presentation of increased intracranial pressure.[6] Untreated increased intracranial pressure can result in neurodevelopmental delay and permanent impairment.[12]

Psychosocial development is another indication for surgery. The cranial deformity may result in social dysfunction in both the short and long term. This directly relates to self-image, confidence, and ultimately, quality of life.[13] Therefore, although psychosocial impact may not be an urgent surgical indication, it is a substantial indication for surgery. For these cases, surgery is still generally recommended before 1 year of age to optimize postsurgical ossification.

14.5 Operative Techniques

Surgical interventions for craniosynostosis aim at expanding cranial volume to accommodate the growing brain as well as normalizing overall head shape. The intervention is selected based on patient age, suture synostosis pattern, and associated syndromic deformities. All procedures are performed under general anesthesia with an anesthesiologist familiar with pediatric craniofacial surgery. Blood transfusion reduction strategies, such as tranexamic acid, should be employed where possible.[14] Even with attempts to minimize transfusion rates, surgery should not begin until blood products are available in the operating room.

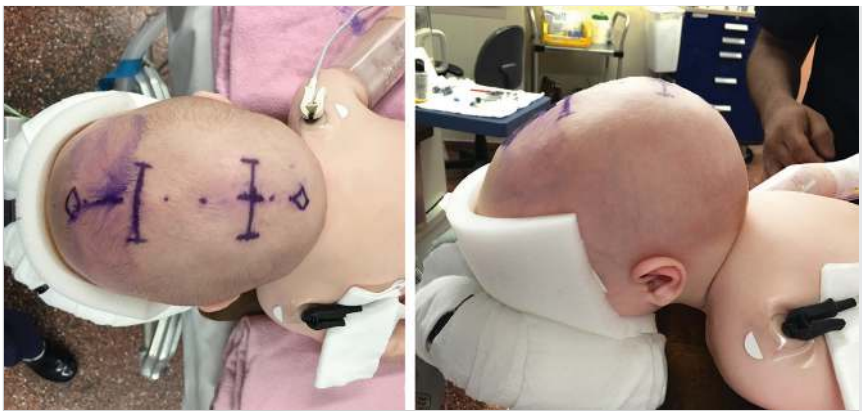


Fig. 14.3 A preoperative view of a patient with isolated sagittal synostosis. Endoscopic access incisions are marked in preparation for the endoscopic-assisted strip craniectomy. Note the patient's scaphocephalic head shape.

14.5.1 Strip Craniectomy

A strip craniectomy may be performed through a full coronal approach, partial coronal incision, or multiple endoscopic access incisions (▶ Fig. 14.3). This technique is most commonly utilized for single suture, nonsyndromic craniosynostosis involving the sagittal suture. For sagittal synostosis, strip craniectomy is most effective in a patient younger than 4 to 6 months and in conjunction with postoperative helmet shaping therapy for 4 to 6 months. Strip craniectomy relies on the malleability of the cranial vault to respond to postoperative helmet therapy. The older the child, the thicker and less malleable the cranial bone, making the child not a candidate for the procedure after 4 to 6 months of age. This makes prompt referral to a craniofacial surgeon imperative if this is to be considered as a treatment option. A craniofacial surgeon and neurosurgeon typically work together to complete the operation, although in some centers the neurosurgeon may perform it alone.

The patient is placed in the supine position on a neurosurgical head rest. Foley catheter, arterial line, and intravenous access are placed. Local anesthetic containing epinephrine is injected along the incision, and then skin is incised. The skin and subcutaneous tissue are undermined as one flap from the periosteum. The area undermined extends a few centimeters beyond the synostotic suture to be removed. The craniectomy is marked. For the sagittal suture, this is approximately 2 to 4 cm in width extending to the anterior and posterior fontanelles. The periosteum is cautiously incised along these markings and undermined from the calvarium. Craniotomy is performed with a burr at the four corners of the desired craniectomy. A curved elevator is gently used to dissect the dura from the inner surface of the calvarium along the craniectomy markings. Extreme caution must be taken near midline as the sagittal sinus may be tethered or partially encased in bone. Rupture of the sagittal sinus can result in significant rapid blood loss that can be fatal. If the sagittal sinus is entered, digital pressure is applied, anesthesia is notified to mobilize blood products, and the cranial vault exposure is widened to access the area of bleeding.

After craniotomy is complete, the craniectomy is performed along the previous markings using a combination of craniotome, Mayo scissors, and endoscope. The piece of bone is slowly removed exposing the sagittal sinus. Barrel stave osteotomies are performed at the left and right sides of the anterior and posterior margins, abutting the fontanelles. These osteotomies are triangular with the apex oriented laterally. With postoperative helmet therapy, these triangular excisions will assist in shortening the anteroposterior length of the cranium and rounding the head shape. The triangles are marked, dissected, and removed in similar fashion to the midline craniectomy. Sealants and coagulative foam are placed along bony margins and the exposed dural surface to assist in hemostasis. Incisions are closed with absorbable suture in a layered fashion. There is no need for a drain, as the dissection is limited.

14.5.2 Frontoorbital Advancement and Anterior Cranial Vault Remodeling

Frontoorbital advancement and anterior cranial vault remodeling are effective for addressing anterior cranial deformities and asymmetry, such as often seen with metopic and unicoronal synostosis. Frontoorbital advancement indicates removal of the bandeau with a bifrontal craniotomy. Anterior cranial vault remodeling indicates removal of the bandeau and bifrontal craniotomy, as well as craniotomy posterior to the coronal suture for partial removal of the bilateral parietal bones. Access and technique are very similar, and thus, they are grouped together in this section.

The patient is placed in the supine position on a neurosurgical head rest. Foley catheter, arterial line, and intravenous access are placed. A bicoronal access incision is marked and injected with local anesthetic containing epinephrine. After vasoconstrictive effect is obtained, the coronal incision is made. The anterior and posterior cranial flaps are dissected either in the supraperiosteal or subperiosteal plane. If supraperiosteal dissection is performed, subperiosteal dissection is done next, and the pericranial flaps and temporalis muscles should be resuspended with absorbable suture during closure.

Flaps are dissected subperiosteally to expose the orbital roof and lateral orbital wall anteriorly, laterally to the level of the squamosal suture or beneath a temporal bulge deformity, and posteriorly as far as necessary to address the cranial deformity. The bandeau is marked, keeping a minimum of 12 to 15 mm of width at all points to prevent fracture with handling and removal (▶ Fig. 14.4). The lateral tenon of the bandeau extends from the superolateral orbits posteriorly several centimeters to provide room for anchoring the advancement later in the procedure. Bifrontal craniotomy is marked with or without

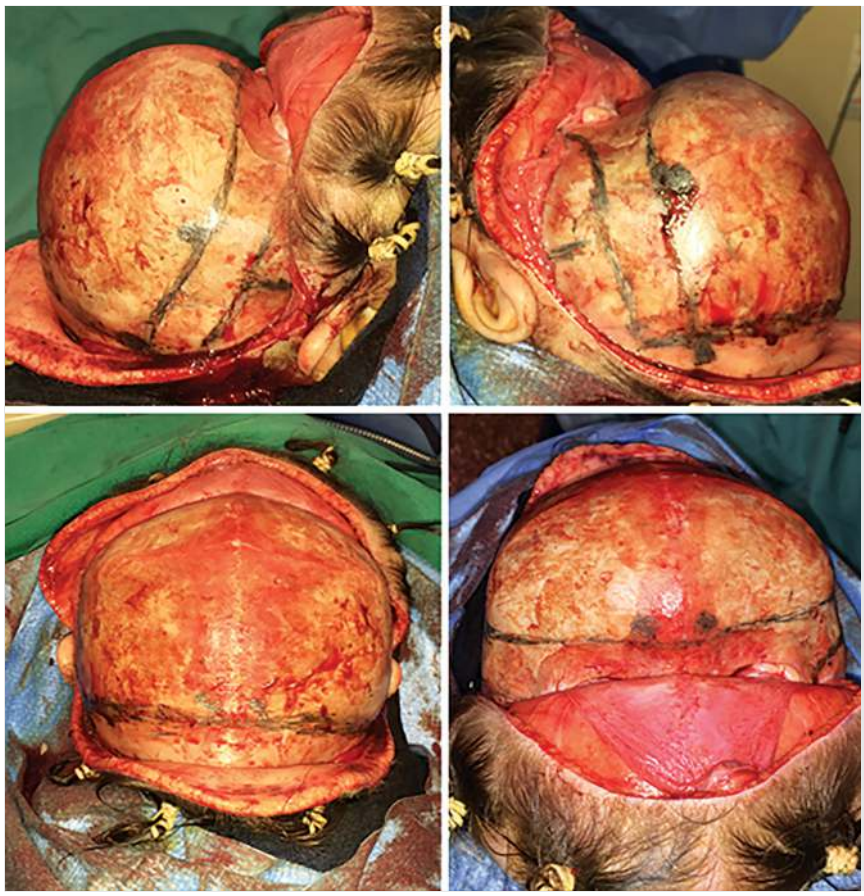


Fig. 14.4 An intraoperative view of frontal orbital advancement in a patient with bicoronal synostosis. The bandeau and bifrontal craniotomy are marked.

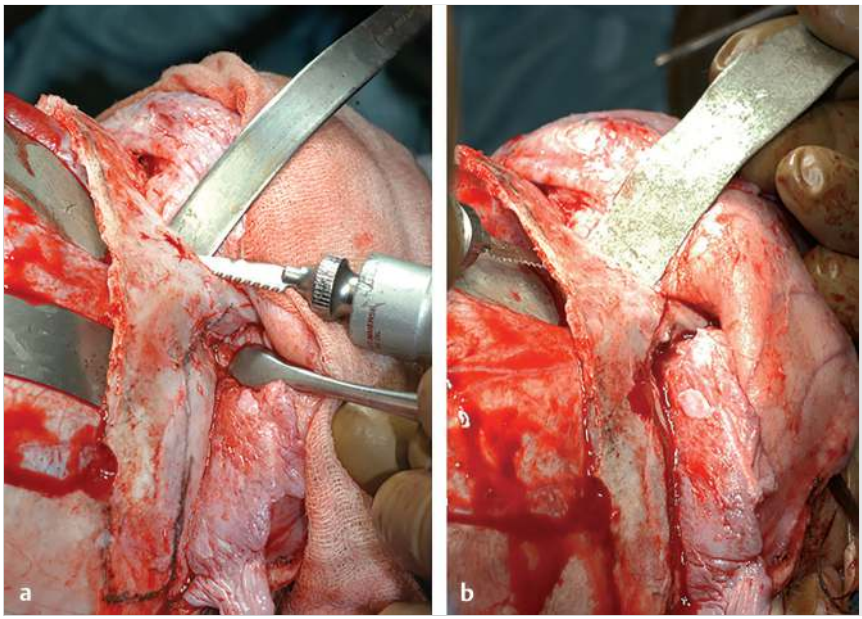


Fig. 14.5 The bandeau is being removed with careful intracranial and extracranial retraction, lateral view. The bifrontal craniotomy has already been performed. **(a)** The zygomaticofrontal osteotomy is performed, **(b)** followed by an osteotomy of the mid orbit extending laterally.

inclusion of the parietal bones. Neurosurgery will perform the craniotomy using a burr and craniotome, first for the bifrontal area, then for the bandeau. The frontal bones are saved in a moist sponge or towel on the back table for replacement later. With the frontal bones removed, the bandeau is dissected free along the anterior and lateral intracranial surface, to allow safe, gentle brain retraction during osteotomies. A reciprocating saw is used to cut extracranially from the zygomaticofrontal suture to the temporal tenon area. A reciprocating saw or side-cutting burr is next used to cut intracranially from mid orbital roof laterally in order to connect with the first osteotomy (▶ Fig. 14.5). Then an intracranial cut is made from the mid orbital roof medially to reach midline. All osteotomies are performed under direct visualization and with cautious retraction of both

Fig. 14.6 **(a)** An asymmetric bandeau of a patient with unicoronal synostosis is reshaped in preparation for **(b)** inset. Note the resorbable plates securing the newly molded contour of the bandeau.

intracranial and extracranial structures. At midline, the cut is positioned anterior to the cribriform plate. The same osteotomies are performed on the contralateral side. The last cut is made externally at midline above the nasofrontal suture and is partial thickness to avoid the cribriform plate; this connects the osteotomies of both sides. The bandeau is then digitally outfractured to complete the midline osteotomy and free the bandeau as one piece.

On the back table, the bandeau is shaped into a symmetric forehead contour, and the orbital apertures are rongeured for symmetric size and shape (▶ Fig. 14.6). Its contour is secured with resorbable plates and screws. If a temporal bulge is present, this may be addressed by removing the convex bone segment with a saw, turning over the bone segment, and replacing it with the convex surface inward so that the external surface is now concave. The bandeau is inset with resorbable sutures through drill holes at the nasofrontal osteotomy and bilateral zygomaticofrontal sutures to stabilize the desired contour. Resorbable plates are used at regular intervals to finalize its position. The anterior vault is inset, cutting as needed, to use the optimal curvature for forehead reconstruction. The remaining pieces are fit accordingly and are secured with resorbable plates and screws. As the cranial volume is increased, gaps between bone segments are anticipated. The gaps are minimized and distributed as much as possible, while avoiding gaps in non–hair-bearing areas (e.g., the forehead). The vast majority of gaps ossify in this age group before 5 years of age, and those that do not, may be addressed by cranioplasty.

Pericranial flaps and temporalis muscles are resuspended with resorbable suture. A drain is typically placed. The galea is closed followed by skin, using resorbable suture.

14.5.3 Posterior Cranial Vault Remodeling

Remodeling of the posterior cranial vault is performed to address asymmetry or deformity of the posterior cranium, such as that seen with lambdoid synostosis. It may also be performed to increase calvarial volume, although this may be more effectively accomplished by posterior vault distraction or total cranial vault remodeling, to be discussed in subsequent sections.

For posterior cranial vault remodeling, Foley catheter, arterial line, and intravenous access are placed with the patient in the supine position. The patient is then transferred to the prone position on a neurosurgical head rest. Incision and exposure are performed similarly to an anterior cranial vault remodeling using a bicoronal approach. The dissection focuses over the area of deformity and planned craniotomy, which usually extends anteriorly to the temporal high point in front of the coronal sutures and posteriorly to the superior nuchal line. If pericranial flaps are separately dissected, an "H" incision may be used to preserve large anteriorly and posteriorly based flaps.

Once the craniotomy is marked by the craniofacial surgeon, it is carried out by the neurosurgeon. The occipital bone is positioned to expand any flattened areas and improve skull height. The occipital bone is cut and reshaped as needed to optimize contour. The bone may be split into inner and outer table segments to increase available bone coverage. All bone segments are secured with resorbable hardware. The pericranial flaps and temporalis muscles are resuspended with absorbable suture. A drain is placed. The incision is closed in layers, galeal then skin, with resorbable suture.

14.5.4 Total Cranial Vault Remodeling

Remodeling of the total cranial vault combines the anterior cranial vault remodeling and posterior vault remodeling. It is utilized in patients with deformities of the entire cranium or if significant volume increase is desired, as in cases with elevated intracranial pressure. In order to fully address the anterior and posterior cranium, a position change is normally required between prone and supine positions, unless adequate exposure can be obtained through the sniffing position. The authors' preference is to start prone and then change to supine to complete the operation. Some practitioners prefer to perform anterior cranial vault and posterior cranial vault remodeling separately

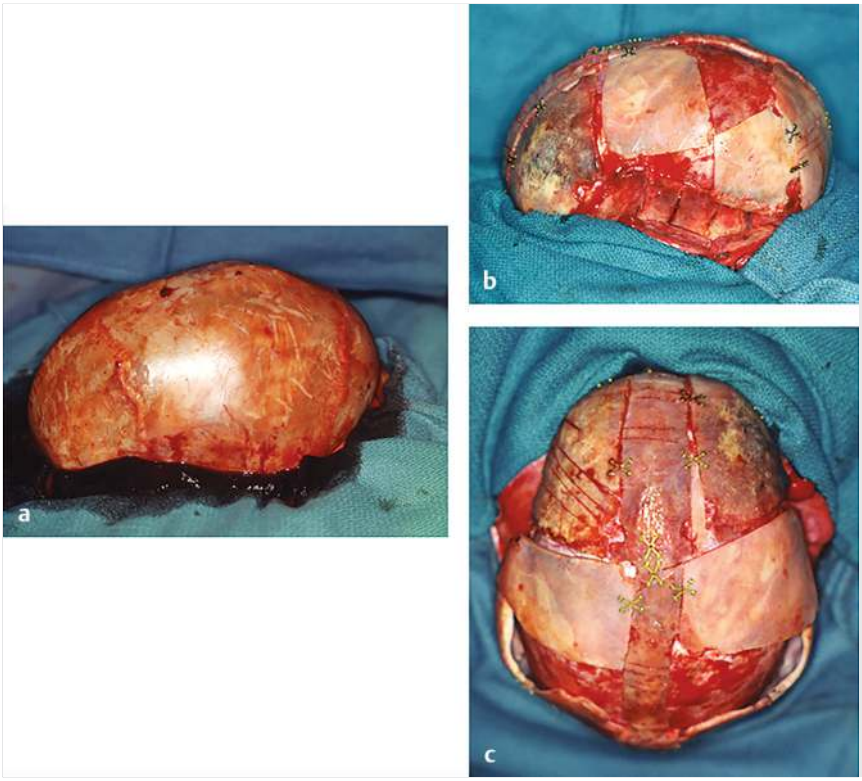


Fig. 14.7 Total cranial vault reconstruction is performed on a patient with sagittal synostosis. **(a)** Intraoperative exposure of cranium is followed by **(b,c)** removal of the cranial bones and replacement in an optimized arrangement that increased intracranial volume and improves aesthetic appearance.

as staged procedures, rather than a total cranial vault remodeling in one operation.

Foley catheter, arterial line, and intravenous access are placed with the patient in the supine position. The patient is then transferred to the prone position on a neurosurgical head rest. Incision and exposure are performed similarly to previously described techniques using a bicoronal approach. Focus is first on the posterior vault, with dissection to the superior nuchal line and anteriorly to the coronal sutures. Craniotomy is marked, taking into account the position of the sagittal sinus. The neurosurgeon performs the craniotomy. The posterior vault bone segments are cut and contoured to optimize the position, shape, and intracranial volume (▶ Fig. 14.7). The segments are secured with resorbable fixation. The pericranial flaps are resuspended, and the incision is temporarily closed with running nylon.

The patient is turned to the supine position, and they are again prepped and draped. The nylon suture is removed. Dissection proceeds anteriorly as with a frontoorbital advancement. The bifrontal craniotomy and bandeau are marked similar to a frontoorbital advancement. The neurosurgeon performs the bifrontal craniotomy, and the craniofacial surgeon removes the bandeau. The bandeau and orbital apertures are contoured. The bandeau is then replaced with sutures and resorbable hardware as in a frontoorbital advancement. The pericranial flaps and temporalis muscles are resuspended. A drain is placed. The galea and skin are closed as separate layers with absorbable suture.

14.5.5 Posterior Vault Distraction

In patients with brachycephaly, turricephaly, or elevated intracranial pressure, posterior vault distraction may be employed to lengthen the calvarium relative to skull height and increase intracranial volume. Underlying diagnoses typically include bicoronal synostosis or multisuture synostosis, both syndromic and nonsyndromic. Often in syndromic patients, posterior vault distraction is done first with plans to perform a frontal advancement procedure later.

Foley catheter, arterial line, and intravenous access are placed with the patient in the supine position. The patient is then transferred to the prone position on a neurosurgical head rest. Incision and exposure are performed similarly to previously described techniques using a bicoronal approach. Dissection proceeds to expose the intended osteotomies, no further posteriorly than the superior nuchal line. The osteotomies generally are posterior to the coronal suture and then connect with a transverse osteotomy just above the superior nuchal line. Two internal distractors are placed parallel to each other at the lateral aspect of the parietal bones, and they are aimed to distract the occipital segment posteriorly and slightly inferiorly. The distractors are secured according to the manufacturer's directions. A separate stab incision is made for the distractor arm to exit the skin anteriorly to the coronal incision. The incision is then approximated with absorbable suture, galea then skin. Activating arms are attached to the internal distractors. At our institutions, activation begins the morning after placement, turning 0.5 mm twice daily or 0.3 mm three times daily until desired distraction is reached. The patient is monitored with lateral skull radiographs as needed (▶ Fig. 14.8). The activation arms are removed once distraction is complete, and consolidation lasts 6 to 8 weeks. After consolidation, the internal distractors are removed in the operating room through a limited opening and dissection of the original approach.

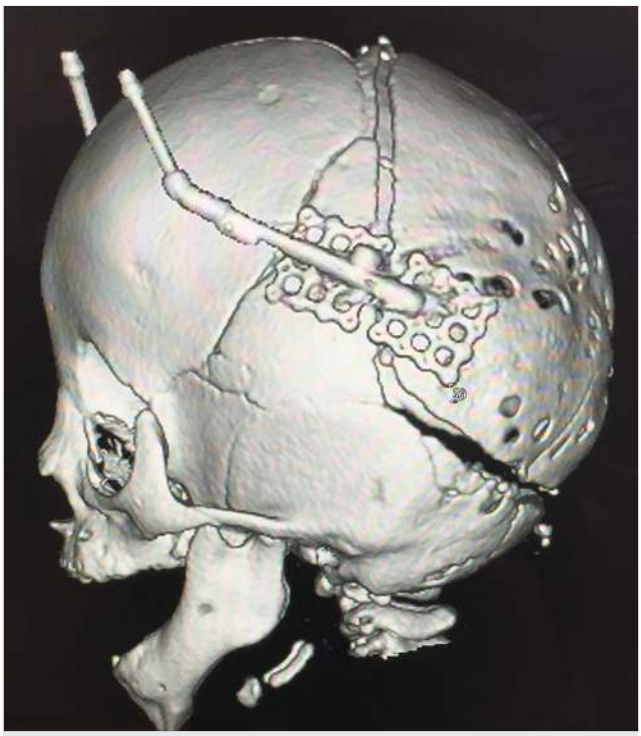


Fig. 14.8 A lateral skull radiograph demonstrating the completed osteotomies and placement of the bilateral internal distractors.

14.6 Postoperative Care

With the procedure complete, the hair is washed with shampoo and conditioner. Antibiotic ointment is placed along the incision. A head wrap or dressing is not necessary. If a dressing is desired, it should be well-padded, loosely placed, and removed the day after surgery to minimize the risk of skin compromise.

Patients are monitored at least one night in the intensive care unit. Most meet criteria for transfer to the floor on postoperative day 1 in our experience. Diet is advanced from clears to regular as tolerated. The activity of the patient is not restricted, and caregivers can hold their child immediately after surgery. Drains are typically removed on postoperative day 1 or 2, unless persistently draining a significant amount. Fluid should be monitored for appearance to ensure cerebrospinal fluid is not being suctioned by the drain, especially if a dural tear was encountered intraoperatively. Perioperative antibiotics are administered for 24 hours after surgery.

Wound care consists of cleaning with cotton swabs and normal saline to remove dried blood and exudate twice daily, followed by application of antibiotic ointment. This should be continued by caregivers at home after discharge. Once discharged, patients may be bathed including the surgical site, as long as the surgical site is not submerged underwater.

Caregivers should be counseled to expect significant periorbital and facial edema postoperatively. Often the eyes are unable to be seen on postoperative day 2 or 3 due to the edema. After this begins to recede and patients can see, oral intake generally improves, and the patient is ready for discharge. Oral intake must be at baseline or nearly at baseline prior to discharge.

Typically patients return to their baseline energy level 2 to 4 weeks after surgery, although their nighttime sleep schedule may be disrupted for several months. The palpable soft spots indicative of gaps between bone segments slowly heal over subsequent months to years. Cranioplasty to repair the gaps is considered if they are not healed by 5 years of age. If gaps are substantially large, a protective helmet may be considered as the child becomes more mobile, until cranioplasty is performed.

14.7 Outcomes

Blood loss and subsequent coagulopathies drive immediate postoperative concerns. Strategies to reduce blood loss should be emphasized, such as meticulous hemostasis and tranexamic acid administration.[14] Transfusion rates vary widely depending on institution protocols, with open procedures generally reporting a higher transfusion rate than endoscopic procedures. Infection rates reported by a recent large database assessment were 0.2 to 0.8%, whereas smaller studies in the literature report infection rates as high as 7.5%.[15,16]

The neurodevelopment of children with craniosynostosis has been of concern for some time. While intervention aims to improve long-term cognition and psychosocial well-being, data are inconclusive as to what optimizes these outcomes. Conversely, data are clear that children with syndromic craniosynostosis have poorer outcomes than those with nonsyndromic craniosynostosis, and multisuture craniosynostosis portends a worse outcome than single suture craniosynostosis.[17,18,19,20]

The rate of revision surgery is influenced by the age of cranial vault remodeling in syndromic and nonsyndromic craniosynostosis. There is an increased risk of major revision (e.g., repeat of the original operation) if the patient is younger than 6 months, with either syndromic or nonsyndromic craniosynostosis.[21,22] While there is an increased risk of minor revision in syndromic patients older than 9 months and nonsyndromic patients older than 6 months.[21,22]

14.8 Review Questions

14.8.1 True or False

1. Posterior plagiocephaly is more common than craniosynostosis.
2. Virchow's law dictates that when premature suture fusion occurs, growth of the skull is typically restricted parallel to the fused suture and enhanced in a plane perpendicular to it.

14.8.2 Choose the Best Answer

3. Strip craniectomy for sagittal synostosis is best performed at
   a) Greater than 6–9 months of age.
   b) Generally less than 6 months of age.
   c) Age is not an important factor in planning strip craniectomy for sagittal synostosis.
4. The most common complications in craniosynostosis corrective surgery include
   a) Neurological damage, coagulopathy, subdural hematomas.
   b) Hydrocephalus, dural tears, blood transfusion reactions.
   c) Asymmetry, contour deformities, superficial wound infections.

d) All of the above complications are equally common.

5. Elevated intracranial pressure is associated with
   a) Developmental delay.
   b) Headaches.
   c) Vomiting.
   d) Increased fussiness.
   e) All of the above.

14.8.3 Answers

1. True.
2. False. Virchow's law dictates that when premature suture fusion occurs, growth of the skull is typically restricted *perpendicular* to the fused suture and enhanced in a plane *parallel* to it.
3. b. Generally less than 6 months of age.
4. c. Asymmetry, contour deformities, superficial wound infections.
5. e. All of the above.

References

[1] Forrest CR, Hopper RA. Craniofacial syndromes and surgery. Plast Reconstr Surg. 131(1):86e–109e

[2] Boere-Boonekamp MM, van der Linden-Kuiper LT. Positional preference: prevalence in infants and follow-up after two years. Pediatrics. 107(2):339–343

[3] Kalra R, Walker ML. Posterior plagiocephaly. Childs Nerv Syst. 28(9):1389–1393

[4] Persing JA. MOC-PS(SM) CME article: management considerations in the treatment of craniosynostosis. Plast Reconstr Surg. 121(4) Suppl:1–11

[5] Virchow R. Uber den Cretinismus, namentlich in Franken, und uber pathologische Schadelformen. Verh Phys Med Gesell Wurzburg.. 2:230–271

[6] Rogers GF, Greene AK, Proctor MR, Mulliken JB, Goobie SM, Stoler JM. Progressive postnatal pansynostosis. Cleft Palate Craniofac J. 52(6):751–757

[7] Jeevan DS, Anlsow P, Jayamohan J. Abnormal venous drainage in syndromic craniosynostosis and the role of CT venography. Childs Nerv Syst. 24(12): 1413–1420

[8] Fearon JA, Weinthal J. The use of recombinant erythropoietin in the reduction of blood transfusion rates in craniosynostosis repair in infants and children. Plast Reconstr Surg. 109(7):2190–2196

[9] Renier D. Intracranial pressure in craniosynostosis: Pre and postoperative recordings–Correlation with functional results. In: Persing JA, Edgerton J, Jane JA, eds. Scientific Foundations and Surgical Treatment of Craniosynostosis. Baltimore, MD: Williams & Wilkins; 1989:263–269

[10] Mathijssen IM. Guideline for care of patients with the diagnoses of craniosynostosis: working group on craniosynostosis. J Craniofac Surg. 26(6):1735–1807

[11] Tamburrini G, Caldarelli M, Massimi L, Santini P, Di Rocco C. Intracranial pressure monitoring in children with single suture and complex craniosynostosis: a review. Childs Nerv Syst. 21(10):913–921

[12] Renier D, Sainte-Rose C, Marchac D, Hirsch JF. Intracranial pressure in craniostenosis. J Neurosurg. 57(3):370–377

[13] Langlois JH, Kalakanis L, Rubenstein AJ, Larson A, Hallam M, Smoot M. Maxims or myths of beauty? A meta-analytic and theoretical review. Psychol Bull. 126(3):390–423

[14] Martin DT, Gries H, Esmonde N, et al. Implementation of a tranexamic acid protocol to reduce blood loss during cranial vault remodeling for craniosynostosis. J Craniofac Surg. 27(6):1527–1531

[15] Lin Y, Pan IW, Mayer RR, Lam S. Complications after craniosynostosis surgery: comparison of the 2012 Kids' Inpatient Database and Pediatric NSQIP Database. Neurosurg Focus. 39(6):E11

[16] Esparza J, Hinojosa J, García-Recuero I, Romance A, Pascual B, Martínez de Aragón A. Surgical treatment of isolated and syndromic craniosynostosis. Results and complications in 283 consecutive cases. Neurocirugia (Astur). 19(6):509–529

[17] Kapp-Simon KA, Wallace E, Collett BR, Cradock MM, Crerand CE, Speltz ML. Language, learning, and memory in children with and without single-suture craniosynostosis. J Neurosurg Pediatr. 17(5):578–588

[18] Speltz ML, Collett BR, Wallace ER, et al. Intellectual and academic functioning of school-age children with single-suture craniosynostosis. Pediatrics. 135 (3):e615–e623

[19] Shim KW, Park EK, Kim JS, Kim YO, Kim DS. Neurodevelopmental problems in non-syndromic craniosynostosis. J Korean Neurosurg Soc. 59(3):242–246

[20] de Jong T, Maliepaard M, Bannink N, Raat H, Mathijssen IM. Health-related problems and quality of life in patients with syndromic and complex craniosynostosis. Childs Nerv Syst. 28(6):879–882

[21] Utria AF, Lopez J, Cho RS, et al. Timing of cranial vault remodeling in nonsyndromic craniosynostosis: a single-institution 30-year experience. J Neurosurg Pediatr. 18(5):629–634

[22] Utria AF, Mundinger GS, Bellamy JL, et al. The importance of timing in optimizing cranial vault remodeling in syndromic craniosynostosis. Plast Reconstr Surg. 135(4):1077–1084

15 Ear Reconstruction and Otoplasty

Stephanie Suprenant, Ruston Sanchez, and Timothy W. King

Abstract

This chapter examines the anatomy of the ear and suggests treatment options when there are significant variations or deformities in development, size, position, and shape. Each of the three major design features—axis/position, proportions, contour—are discussed. Preoperative surgical preparations are detailed, and the pioneering techniques of Radford Chapple Tanzer, Burt Brent, Satoru Nagata, and Françoise Firmin are discussed. The alternative ear reconstruction approach known as alloplastic reconstruction is covered, and paragraphs are dedicated to the ear variations/deformities known as constricted ears, prominent ears, cryptotia, Stahl's ear, and question mark ear. Guidelines for postoperative care and outcomes concludes the chapter.

Keywords: microtia, macrotia, constricted ears, prominent ears, cryptotia, Stahl's ear, shell ear, question mark ear

15.1 Goals and Objectives

- Review the anatomy of the ear in regard to normal development, size, position, and shape.
- Appreciate the rationale for surgical timing and when to operate to maximize outcomes.
- Review the various surgical techniques available for ear reconstruction for both microtia as well as ear deformities.

15.2 Patient Presentation

The patient with a malformed, misformed, deformed, or absent ear presents early in life. As a generality, the greater the deformity, the earlier the presentation due to parent concern. Most abnormalities of the external ear occur as a lone abnormality, but others are syndromic, such as that which occurs with Treacher Collins syndrome. Overall presentation and decisions regarding treatment will depend on multiple factors. Ear reconstruction surgery is predicated on a sound understanding of normal ear development and anatomy.

15.3 Ear Embryology and Development

Malformations of the auricle occur in approximately 1 out of 12,500 births, and these deformities can occur in isolation or in association with genetically determined syndromes.[1] The embryologic origin of the middle and external ear differs from that of the inner ear, leading to conductive but not sensorineural hearing loss in association with auricular deformities. The auricle develops from the first and second branchial arches, which form six auricular hillocks that later fuse to form the external ear. Dysmorphogenesis occurs between the fourth and twelfth weeks of embryonic life. By week 20, development is complete and there is no further development after birth.[2]

Growth of the ear occurs rapidly in the early years, reaching 85% of final adult size by 3 years of age. The distance from the ear to the scalp and the ear width vary little after 10 years. The remaining 15% of vertical height will be complete by age 20 years. Further small increases with advancing age are due to elongation of the lobule.[3,4,5]

15.4 Ear Anatomy

At its most basic, the ear is a multicontoured cartilage flap covered by closely adherent thin skin which protrudes from the head and is positioned within narrow limits relative to the adjacent structures. Refer to ▶ Fig. 15.1 for a depiction of the normal ear, its parts, and its proportions as described by Tolleth.[6,7] When considering the basic anatomical elements of the ear, it becomes evident that there are three main design features to consider when performing an otoplasty—axis/position, proportions, and contour.

15.4.1 Axis and Position

The axis of the ear is defined as the line of balance through the long dimension of the ear. An inclination of 15 to 30 degrees from the vertical is acceptable with 20 degrees being optimal. The most important aspect of this measurement is that the ear tends to incline posteriorly on visual inspection. With regard to position, the distance of the ear from the orbit should be roughly one ear length posterior from the lateral orbital rim or approximately 6.5 to 7.5 cm. The level of the superior aspect of the helix correlates with the brow, and the inferior aspect of the lobule correlates with the base of the columella. These positions are depicted in ▶ Fig. 15.2.

15.4.2 Proportions and Protrusion

The ear varies in height from 5.5 to 6.5 cm. Width varies from 3.0 to 4.5 cm or 30 to 60% of the height. Protrusion from the scalp to the superior helix is approximately 1.5 to 2.0 cm.

15.4.3 Contour

The most noticeable portion of the external ear is the helix, which frames the pinna. The root of the helix begins in the concha, and it then travels in an arc to end at the lobule. At its origin, the helix is wide and gradually narrows as it approaches the lobule. Additionally, the helix transitions from having an overhanging curled portion to become more tubular at the midpoint of the ear. The antihelix begins as a narrow and tightly constricted structure inferiorly and then splits into a smaller, sharp-edged, inferior (anterior) crus and a broader, rounded, superior (posterior) crus. The area between the inferior and superior crus is the fossa triangularis. The scapha is directly posterior to the superior crus (Fig. 15.1).

The concha is triangular in shape and roughly measures 1.5 × 2.5 cm, with a width two-thirds its height. The tragus is a

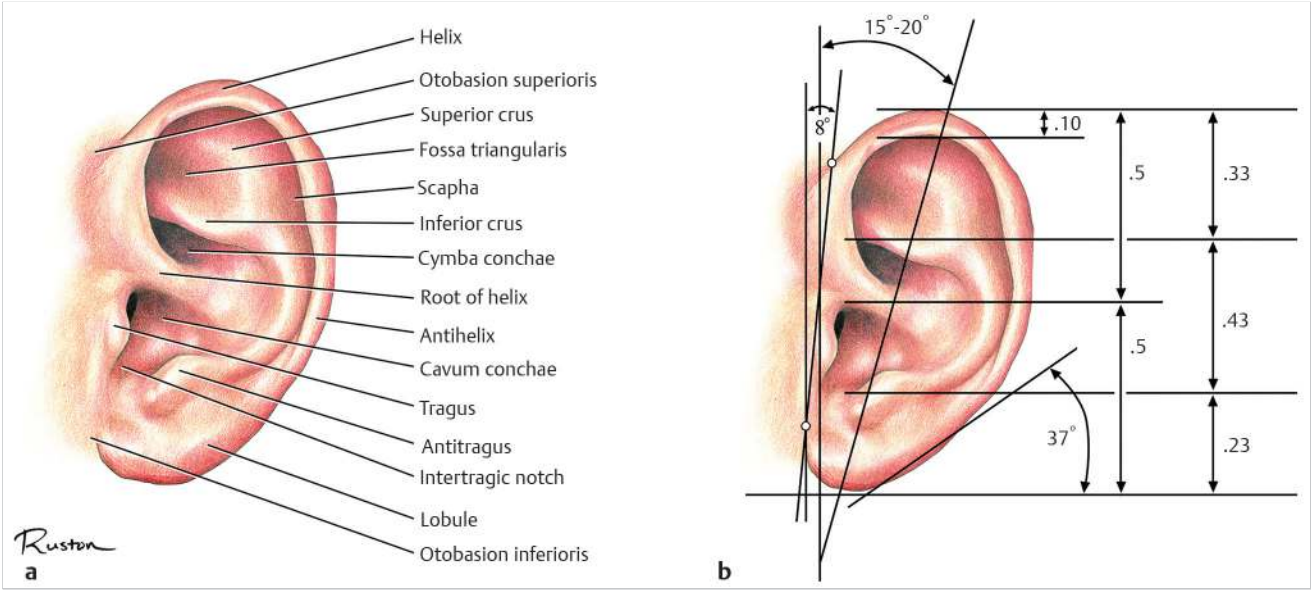


Fig. 15.1 (**a**) The normal ear and its parts. (**b**) The normal ear and its proportions.

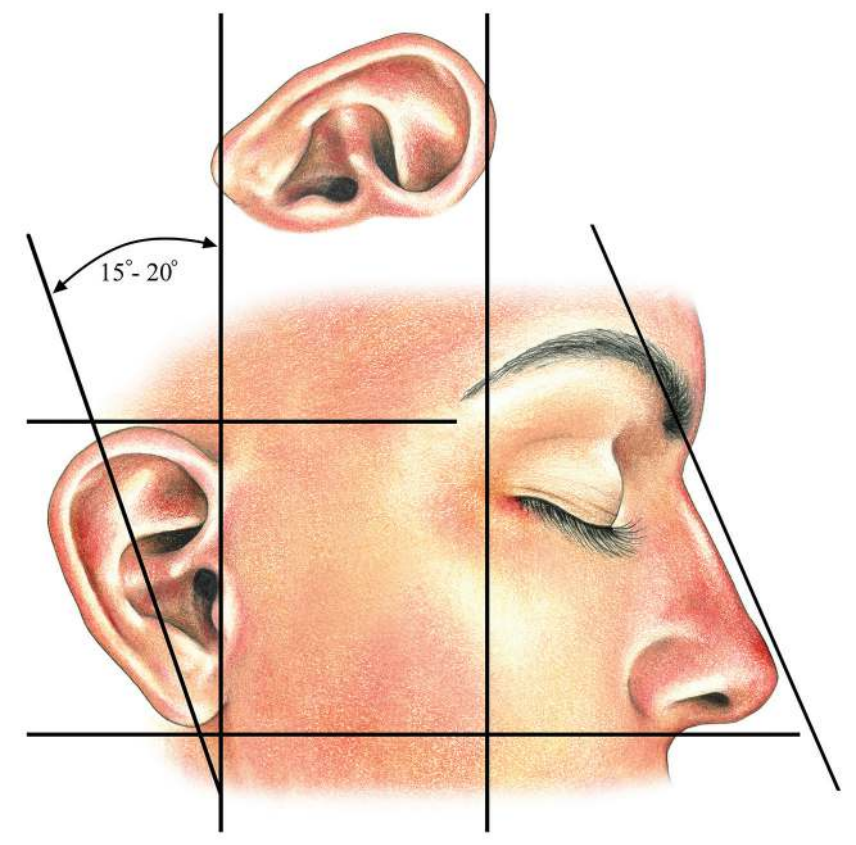


Fig. 15.2 The normal ear size and position.

firm nodule anterior to the ear canal that is a congruent piece of cartilage which becomes the intertragic notch and antitragus, and then blends into the antihelix as it completes its inferior arc. The lobule is a flap of soft fibroadipose tissue, which has three common shapes: round, flat, and triangular. There are varying degrees of attachment of the lobule, ranging from completely free to totally adherent.

15.5 Preparation for Surgery

15.5.1 Ethics, Motivation, and Timing of Surgery

An ethical dilemma in ear reconstruction and otoplasty concerns the timing of surgery. Families typically request early surgical correction to avoid psychological and social trauma, but the child is often not disturbed by the deformity during the early childhood years.[8] Furthermore, the ear is not at its full adult size until later in childhood, making surgical correction at a later age more optimal.[9] Considerations on severity of the malformation, ear growth, patient participation, and psychological impact of the deformity all need to be discussed as a part of the decision to proceed with surgery.[10]

The American Association of Plastic Surgeons were surveyed and 57% responded that they delay otoplasty until age 5 years or older due to the following reasons: near adult size of ear, greater patient cooperation, increased peer ridicule, decreased need for general anesthesia, greater patient self-image, and traditional teachings.[11] A similar survey was sent to members of the British Association of Plastic Surgeons and 79% believed that children should be older than 6 years at the time of otoplasty.[8]

Gosain et al reviewed their patients undergoing otoplasty before the age of 4 years and found that there was no growth restriction when comparing the native normal ear and the reconstructed ear.[11] All of the families in this study stated that they would choose to pursue surgery before the age of 4 years again. Tanzer and Brent found that their reconstructed ears grew with their patients regardless of the age at which surgery was performed. On the other end of the spectrum, Bauer transitioned from reconstructing ears at age 5 to waiting until age 10 due to having sufficient donor cartilage and a lack of psychological impact with greater patient participation.[12]

The only prospective trial to research the impact of timing on ear reconstruction was performed by Bradbury et al in 1992.[13] They found that there was a wide disparity of the age of ear self-consciousness from 4 to 13 years. Patient motivation for surgery was primarily due to psychological and social distress followed by aesthetic concerns and anticipated problems by parents. Levels of teasing were higher in otoplasty patients than control children until age 13 when the levels equalized, although all reported teasing levels were high. There were no correlations between patients, parents, or an independent panel on the degree of observed ear prominence. There was no correlation with severity of deformity and degree of satisfaction. Postoperative interviews revealed that 63% of patients were happier, but only 13% reported improved social circumstances, which led to a significant difference between social and emotional outcomes.

15.5.2 Antimicrobial Considerations and Intraoperative Preparation

While practice patterns during the perioperative period vary greatly, it is generally accepted that at a minimum, a prophylactic dose of intraoperative antibiotics should be given prior to otoplasty or ear reconstruction.[12] When considering ear reconstruction, there is a significant association with patent external auditory canals (EAC) and risk of infection.[14] For this reason, strict operative cleansing of the EAC and treatment based on swab cultures are recommended. This intervention seeks to prevent complications of infection, cartilage exposure, surgical debridement, and loss of graft. The operative field is prepared with Betadine and the head is draped in a fashion to allow visualization of both ears. This is beneficial when making corrections in position or symmetry to the contralateral ear.

15.6 Treatment

When discussing a topic as broad as ear reconstruction and otoplasty, there are many different approaches that one can take. Often, the most memorable approach is to tell the stories of those who most greatly contributed to the topic. There are four key characters in the story of otoplasty, and they all serve unique roles to fully explain the evolution of this technique. The first major character is Radford Chapple Tanzer, MD, the professor; the second is Burt Brent, MD, the artist; the third is Satoru Nagata, MD, PhD, the engineer; and the fourth is Françoise Firmin, MD, the surgeon.[15] All of these individuals have added their own contribution to the study of otoplasty and ear reconstruction, but they have also demonstrated that the fundamentals of ear embryology, anatomy, timing of surgery, and perioperative considerations must be established prior to undertaking this procedure. In this chapter, we will use the story of these four individuals to describe the basics of ear reconstruction for microtia, and will detail the use of otoplasty for the most common auricular deformities.

15.7 Microtia

15.7.1 Radford Chapple Tanzer, MD —"The Professor"

R.C. Tanzer saw his first microtia patient in 1951 and he reports, "I spent one year just thinking about the problem with paper and pencil. I finally used a 6-stage procedure on the boy, borrowing a flap of skin and bringing it up, with its own blood supply, to make a total construction of an external ear."[16] He clearly outlined the difficulties in producing a surgical substitute for a missing ear: furnishing adequate skin of matching quality, producing a framework that will remain permanently inert, creating delicacy along with contour, and positioning the restored ear properly to furnish a symmetric counterpart.[17,18]

His keys to success were simple. He advocated using an autologous cartilage framework, and recommended that soft tissue used to supplement integument should be a free graft. Lastly, he recommended that no visible scars should be created other than those that are completely necessary. He followed four core concepts. The first was that the ear is a structure composed of four separate planes at right angles to each other: conchal floor, conchal wall, antihelix scapha complex, and the helix. The second concept was to use a central incision in the conchal region as opposed to a peripheral helical rim incision. The third was to understand the limited supply of available cartilage in a pediatric patient in which to form a framework. The fourth concept was to form a tragus from rolling tissue from the conchal floor onto itself.[17]

His initial six-stage procedure evolved into the following four-stage procedure[19,20,21,22,23,24,25]:

1. A contour pattern of the normal ear is made from X-ray film and reversed. The lobule is put into a transversely oriented pattern based on an inferior pedicle.
2. Two months later, rib cartilage is harvested from the sixth and seventh ribs en bloc and the eighth rib separately. The contour pattern is applied to the sixth and seventh ribs and a base block is carved. The helical rim is carved out of the eighth rib and secured to the block with steel wire sutures. The framework is then placed into an undermined pocket with an incision at the former vertical lobular incision. Mattress sutures are placed to enforce the entire frameworks along with the helical sulcus and conchal wall. A full-thickness skin graft may be needed for the conchal floor.
3. After 3 months, the posterior aspect of the framework is elevated and a thick-split thickness skin graft is placed from the thigh. A packing dressing in applied to reinforce the sulcus.
4. One month later, conchal and tragal reconstruction is performed. A **U**-shaped flap from the conchal floor is rolled onto itself to make the tragus and a full-thickness skin graft from the opposite inferior auriculocephalic sulcus is secured into the conchal floor defect.

15.7.2 Burt Brent, MD—"The Artist"

Burt Brent furthered the field of ear reconstruction by first classifying microtia into classic and atypical deformities while providing an artist's insight into reconstruction.[26,27] In the classic deformity, the vestige is a sausage-shaped appendage with a relatively normal but displaced earlobe.[28] Atypical microtia deformities were diverse, and were typically addressed on an individual basis for surgical planning.[29] However, for the classic microtia deformity, he used the following stages of reconstruction[28]:

1. *Creation and implantation of the rib cartilage graft*. An X-ray pattern is made of the normal ear, reversed, and made several millimeters smaller to accommodate for thickness of the skin. The position is marked from comparing the height of the contralateral ear and in a parallel line to the nasal bridge. Cartilage is harvested as Tanzer described from the contralateral sixth to eighth ribs. An incision is made anterior to the vestige, all vestigial cartilage is removed, and a pocket is undermined just deep to the subdermal plexus. A suction drain is placed under the framework and replaces mattress sutures.
2. *Transposition of the lobule*. This can be performed on an outpatient basis as early as 6 weeks after the first stage of reconstruction.
3. *Tragus reconstruction, conchal excavation, and simultaneous contralateral otoplasty*. Taking a cartilage-containing composite graft from the contralateral auricle creates a more delicate tragus reconstruction and can often aid in reduction of the contralateral ear.

Further surgery is sought only based on patient's desire. If auricular projection has not been achieved by exaggerating the framework's height, the framework can be elevated and skin grafted to create an auriculocephalic sulcus. Helical definition is accomplished by excising a deep wedge of scar tissue and cartilage from an incision in the helical sulcus. The closed incision is compressed with mattress sutures and bolsters behind the ear.

Skin Coverage

Although there has been an emphasis on formation of a meticulous auricular framework, the quality and quantity of available skin is equally important to a successful ear reconstruction. This problem most commonly arises in cases where prior surgery has been performed and inelastic scars have been created, making helical projection impossible. One technique to correct this problem is to remove the former framework and place an expander. After inflation, the expander can be removed and the new framework placed. This will result in a thick capsule, which requires another procedure for sulcus deepening.[29] A second technique is the use of a temporoparietal fascial flap that can be placed over the cartilage framework and skin grafted.[30,31,32]

Low hairlines are associated with previously reconstructed ears, acquired traumatic deformities, and almost always in the case of anotia. Brent dealt with this problem by excising the unwanted hair-bearing skin, immediately placing a framework, covering with a temporoparietal flap, and covering with a skin graft. Anotia, or the lack of any residual vestige, presents the challenges of a low hairline, shortage of skin, and absence of an earlobe. Anotia is treated as previously described with a temporoparietal flap and the unique addition of a lobule component to the cartilage framework. The earlobe portion is then elevated as a secondary procedure.[29]

Furrowed, Pitted, or Grooved Microtic Vestige

It is tempting to incorporate a furrowed, pitted, or grooved vestige into the reconstruction. However, this produces an unnatural result with an obvious line of demarcation. These vestiges should either be discarded or modified into a classic microtia vestige prior to initial framework insertion. The remainder of reconstruction can proceed as previously described in classic cases.[29]

Microtia with Conchal Remnant

A conchal remnant can sometimes be used in the reconstruction and saves a step of conchal formation. However, this remnant often causes constriction and prevents the use of the standard preauricular incision for framework insertion. This reduces the amount of skin to cover the cartilage framework. Since there is such a variety of presentation, each case must be assessed individually and parts discarded or incorporated depending on their value.[29]

15.7.3 Satoru Nagata, MD, PhD—"The Engineer"

Nagata defined five types of microtia based on the surgical technique for each deformity.[33,34] In the anotia type, the external ear is completely absent. The lobule type presents with a variable cartilage remnant, vertically oriented lobule, no acoustic meatus, no concha, and no tragus. A third type, large conchal, is attributed with the presence of a lobule, tragus, intertragal notch, concha (with or without acoustic meatus),

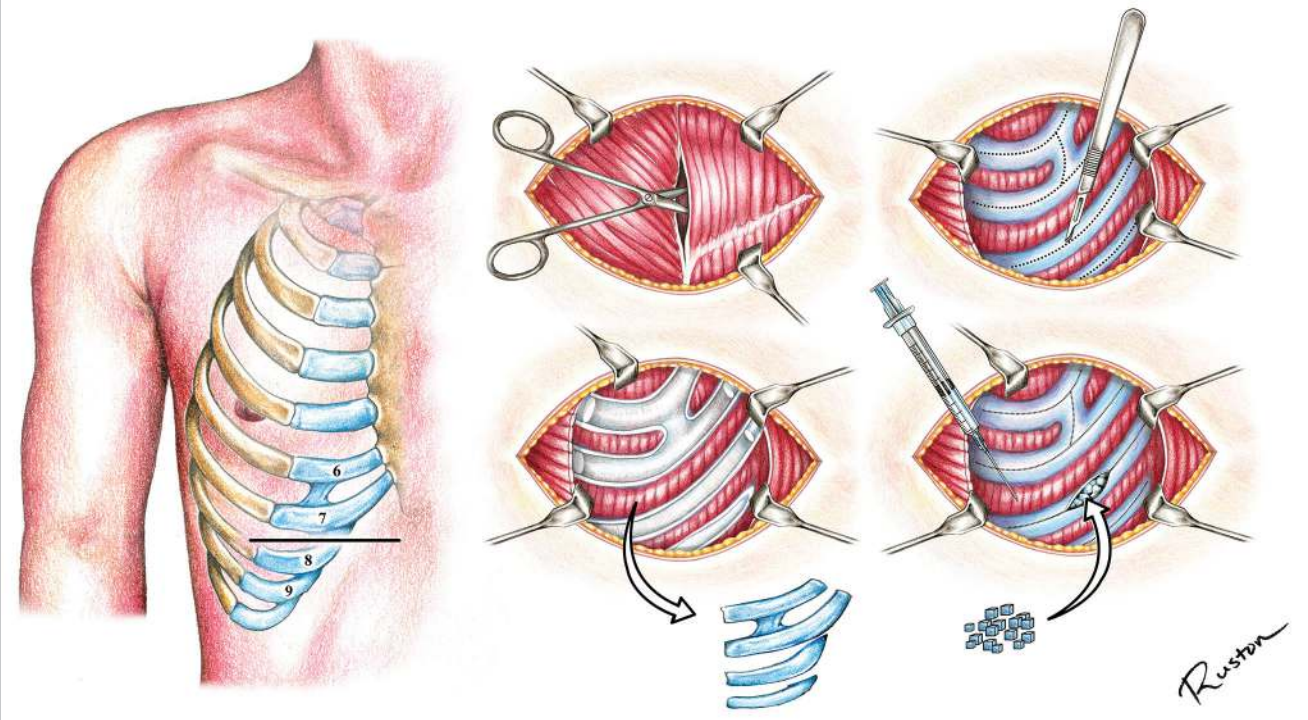


Fig. 15.3 Nagata's technique for cartilage harvest.

and with varying deformities of the upper pole of the auricle. The small conchal type is the lobule type but with a small indentation in the conchal bowl. The last classification is the atypical type, which includes all other deformities which do not fit into other categories.

Nagata's technique for ear reconstruction involves a two-stage procedure with the first being fabrication and grafting of a three-dimensional costal cartilage framework and the second being ear elevation.

Harvest of Costal Cartilage for Auricular Reconstruction

In an attempt to correct chest wall deformities, Nagata changed his practice to minimize the amount of cartilage harvested and improve the technique used in harvesting, leading to fewer deformities and complications.[33,35,36] He described an ipsilateral 9-cm transverse incision over the seventh rib. He then dissected the external oblique and rectus abdominis fascia from each other in a longitudinal fashion to expose the perichondrium of the sixth through ninth costal cartilages. A central incision is made with a scalpel over each costal cartilage to elevate the perichondrium while not incising the costal cartilage. A perichondrial elevator is then used to incise the cartilage, with a Doyen rib elevator used concurrently as an intrathoracic protector. An incision is made medial to the costochondral junction. Costal cartilages 6 and 7 are harvested en bloc with 8 and 9 harvested separately. A leak test is performed to ensure there is no pneumothorax and a layered closure is performed with 4–0 and 5–0 nylon suture. Unused cartilage is cut into 2- to 3-mm blocks and placed back into the cartilage harvest site with securing perichondral sutures. A Penrose drain is left deep to the muscle closure and intercostal nerve blocks provide anesthesia. In their review of patients, it was noted that there were no chest wall deformities and that cartilage regenerated. The steps for cartilage harvest are demonstrated in ▶ Fig. 15.3 and the modifications of ear reconstruction based on each unique microtia type are demonstrated in ▶ Fig. 15.4.

Modification of Framework for Lobule-Type Microtia

Nagata transitioned from using a **V**-shaped incision described by Tanzer to a large **W**-shaped incision over the posterior aspect of the lobule. Tanzer's anterior lobule incision of a straight oblique line was modified to a curvilinear vertical incision with a circular component at the base. The one piece three-dimensional framework was made in two layers with a base frame and a second layer forming the crus helicis, helix, superior crus, inferior crus, antihelix, antitragus, incisura intertragica, and tragus secured on top with nonexposed wire suture. These modifications eliminated the need for skin grafting at the first stage, created better contour definition, and built in tragal reconstruction into the first stage.[37]

Modification of Framework for Concha-Type Microtia

While concha-type microtia seems to be a more simple deformity to correct, it presents unique challenges by incorporating the remnant cartilage into the three-dimensional framework in a seamless fashion. A large **W**-shaped incision is made over the

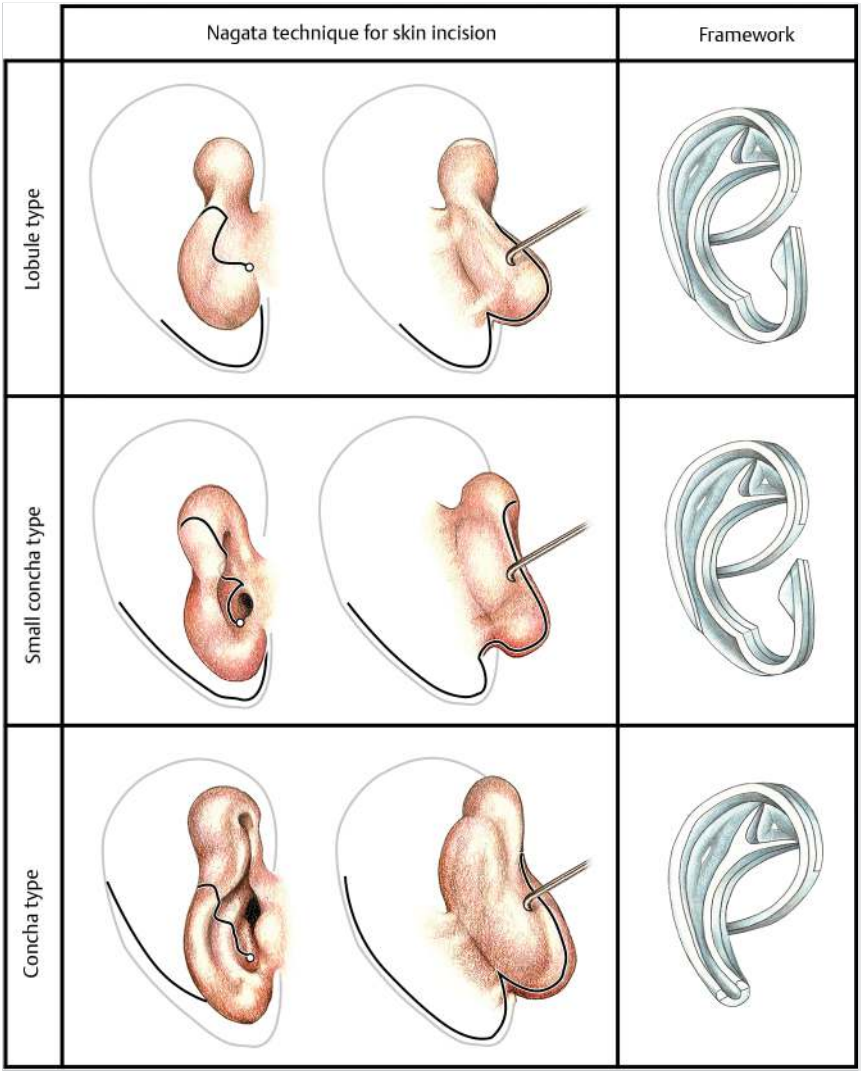


Fig. 15.4 Nagata's technique for skin incision and framework creation based on each microtia classification.

posterior surface along with curvilinear and circular incision anteriorly much like that made in lobule-type microtia. The costal cartilage framework is a two-layer frame in which a base frame is made and the antihelix, superior crus, inferior crus, and helix are then attached. The crus helicis is extended to reach the posterior aspect of the antihelix. Nagata became more aggressive with excision of the remnant ear and conchal cartilages. At his final modification, he removed portions of the posterior conchal cartilage, antihelix, anterior antitragus, and all of the remnant ear cartilage.[38]

Modification of Framework for Small Concha-Type Microtia

There is only a small difference between lobule type and small type microtia, but there are major differences in their reconstructions. Incisions are made identical to that described for the previous two types of microtia. The three-dimensional cartilage framework has two layers. The helix, antihelix, superior crus, inferior crus, incisura intertragica, and tragus were all placed on top of the base layer. The crus helicis was extended to attach on the posterior surface of the antihelix. The remnant ear cartilage was removed in entirety.[39]

Second Stage: Ear Elevation, Fascial Flap, and Skin Grafting

The posterior ear incision is made 5 mm from the edge of the helix. A costal cartilage graft is harvested and made into a concave block with a height of 12 mm. This block is grafted to the posterior aspect of the three-dimensional framework with 4–0 nylon sutures. A temporoparietal fascia flap is then harvested and used to cover the costal cartilage block. Three Penrose drains are placed. A full-thickness skin graft from the groin had been used to cover the fascial flap, but this was later replaced with a split-thickness skin graft. This split-thickness skin graft was elevated with a 15-blade scalpel from the posterior hair-bearing scalp up to the level of Nagata's original release incision (at 5 mm from the helical rim), where it is transitioned to a full-thickness skin graft maintaining congruity.[40,41,42]

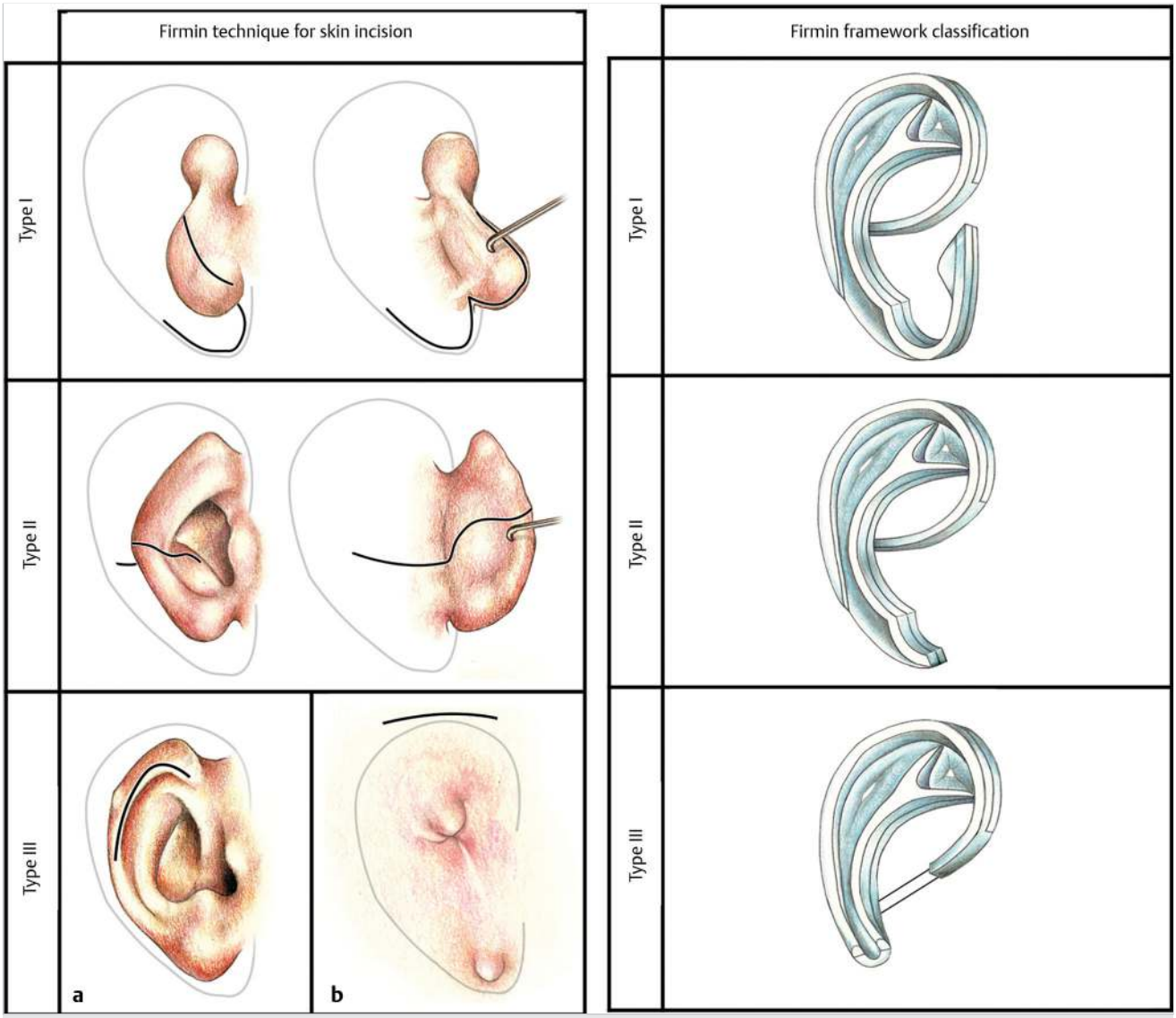


Fig. 15.5 Firmin's technique for skin incision and framework creation based on each microtia classification.

15.7.4 Françoise Firmin, MD—"The Surgeon"

Firmin, an advocate for surgeon experience, studied with both Brent and Nagata. She performed reconstructions with both techniques and eventually blended these into her own approach. She adopted a two-stage reconstruction. The first stage is cartilage harvest, framework sculpture, and placement. Firmin harvests ipsilateral sixth to ninth rib costal cartilages from a 5-cm oblique incision. The anterior perichondrium is harvested, but the posterior perichondrium is left intact with no chest wall deformities. Intercostal blocks are performed and no drains are left. Cartilage is banked for further stage of framework elevation.[43,44,45]

Next, a three-dimensional framework is carved with a base and pieces (antihelix, helix, and tragus–antitragus) attached with stainless steel threads. A modification was the addition of an extra piece of cartilage, behind the helix root and the tragus to enhance anterior projection. Three classifications were made based on the type of framework needed to correct the unique microtia defect: type I (complete framework), type II (no tragus), and type III (no tragus or antitragus). In addition, three skin incisions were described based on the microtic remnants and this is demonstrated in ▶ Fig. 15.5. Type I is a Z-plasty, which allows for transposition of the lobule. Type II is a transfixion incision with a backcut that re-creates the lobule. Type III is a skin incision and only used in rare circumstances. Suction drains are placed.[43,44,45]

The second stage of reconstruction involves creating a retroauricular sulcus 6 months after the initial reconstruction. A posterior incision is made along the surface of the framework and the entire posterior surface of the framework is exposed. The banked cartilage is then harvested and formed in a shape that resembles the antihelix. This is then transfixed with wire sutures in three planes. A temporoparietal fascia flap covers the cartilage and a split-thickness skin graft from the scalp is then placed.[43,44,45]

15.8 Alloplastic Ear Reconstruction

Reinisch and Lewin proposed and perfected an alternative approach to ear reconstruction using a polyethylene or MEDPOR framework covered by a large temporoparietal fascia flap and subgaleal fascia.[46,47] They sought to eliminate the donor-site morbidity from ear cartilage harvest, perform reconstruction at an earlier age, complete reconstruction in 1 stage, and solve soft-tissue restrictions in autologous reconstruction. The original polyethylene framework was welded in four main areas, but based on fracture patterns, the two areas that were the most common for fracture occurred between the two welded areas. This was corrected by soldering additional polyethylene into the areas at risk for fracture. This results in a one-piece implant.

A temporoparietal flap is designed to include both the anterior and posterior branches of the superficial temporal artery. The flap is at least 11 cm wide by 12 cm in vertical length. Many different approaches were initially used for harvest with Reinisch now using a mastoid incision along with a lighted retractor and long, angled electrocautery.[46] The subgaleal fascia is then elevated to provide a two-layered flap. A drain is placed behind the framework and a full-thickness skin graft is placed on the subgaleal aspect of the covered framework. Complications were dramatically improved with modification of the implant design, transposition of a larger temporoparietal fascial flap to cover the entire implant, and elevation of the subgaleal fascia with the TPF flap.[47]

15.9 Macrotia

Macrotia is defined as an ear that has dimensions that exceed the upper ranges of normal width or height.[48,49] Excess tissue is most commonly found in the upper third of the auricle. The ear can also appear abnormally large if it encompasses more than the middle third of the ear head. Rarely, the entire ear can be large and in that case earlobe reduction can decrease size by 1.2 cm. Most commonly, patients present with a component of macrotia and prominent ears and are candidates for reduction otoplasty.[48]

Incisions are made in the retroauricular sulcus and inside the helical rim on the lateral surface of the ear. The retroauricular incision allows access to the concha and this technique will be described in more detail later in the "Prominent Ears" section. The lateral incision, inside the helical rim, extends through the cartilage. A crescent-shaped segment of cartilage is removed from the scaphal area. Mustardé mattress sutures can then be placed between the concha, scapha, and triangular fossa areas to define the upper auricle. Lastly, a wedge-shaped piece of helical rim is removed to match the reduced scapha. Scapha reduction alone can reduce the ear by 7 mm.[48]

15.10 Constricted (Cup and Lop) Ears

Tanzer was the first to classify the constricted ear, and he created three groups and two subgroups.[50] In group I, patients have involvement of only the helix, which is folded on itself along the superior rim causing a diminution in height. There are a variety of treatment options depending on the severity of the deformity. At the most simple, an incision is made 1 cm posterior to the helical rim. The cartilage is completely exposed while maintaining a medial segment attached. The deformed cartilage is then relocated into an upright position as a banner flap. Scoring can be done to add further curvature. The skin is re-draped, closed, and mattress sutures are applied to reinforce the helical sulcus. For more severe constrictions, a bipedicled D flap can be used in an Antia-Buch advancement fashion to restore contour and height.[50]

Group II anomalies involve the helix and scapha with further division into two subgroups. Group IIA patients have a failure of folding of the superior antihelical crus, which causes a tightened purse string effect on the helical rim with the helix and scapha rolling over like a hood. The soft tissue envelope is still adequate to expand the helical margin. Treatment for group IIA deformities are similar to that of group I anomalies. Three principles guide correction—adjustment of the anterior helix, filleting the deformed helix and scapha, and the floating cartilage principle. The anterior helix can be mobilized and advanced superiorly either with a Z-flap at the tip or with a V-Y advancement. Tanzer modified the Ragnell technique to filet the deformed helix and scapha. He created anterior and posterior cartilage leaves which were rotated 180 degrees, like that of the banner flap, and secured to each other to form the desired increase in height. A strip of hooded cartilage can be mobilized as a free segment and combined with scoring to increase auricular height in the floating cartilage principle.[50]

Group IIB anomalies are a more severe cup deformity and require the addition of supplemental skin to expand the helical margin. There is a loss of folding involving the inferior crus, superior crus, and body of the antihelix with pronounced hooding and reduction in height. New skin can be introduced by rotating a skin flap from the medial ear, rotating an inferiorly based auriculocephalic skin flap, or introducing a wedge-shaped composite graft from the opposite ear.[50]

Group III represents the most severe constriction in which the auricle is in a tubular form. The anterior helix is close to the lobule and the entire ear is low set. Stenosis of the external auditory canal and deafness are associated with this deformity. Treatment is focused on first placing the ear in the correct position by elevating the ear using a V-Y advancement and tilting back the ear to be parallel with the nasal bridge. The ear is then completely opened at the tubular structure and set back to the mastoid. The defect is usually the entire superior third of the ear, which can be built up with conchal cartilage and covered with an anteriorly based retroauricular skin flap.[50]

15.11 Prominent Ears

Prominent ears are one of the most common congenital deformities of the head and neck, affecting 5% of the general population. Prominent ears are those that deviate from the normal values described earlier. The three most common causes of prominent ears are (1) underdeveloped antihelical fold, (2) prominent concha, and (3) protruding earlobe. Thorne's technique of otoplasty for prominent ears will be described as well as his methods for evaluation.[49,51]

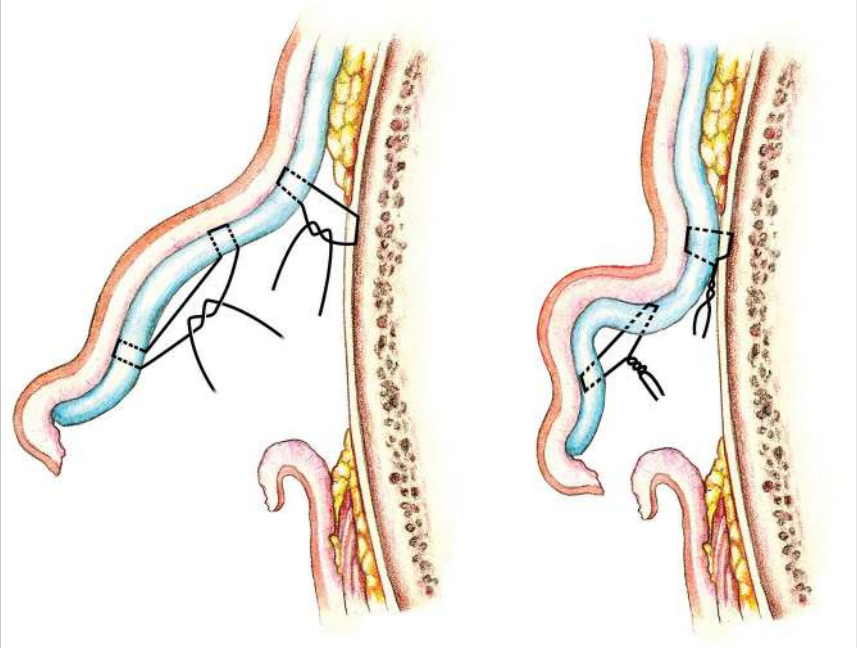


Fig. 15.6 Mustarde (scapha to concha) and Furnas (concha to mastoid) sutures for correction of prominent ears.

An incision is made in the retroauricular sulcus and extends to the medial surface of the earlobe. A small triangular piece of lobular skin and tissue is removed to correct earlobe prominence. At the end of the case, this area is closed with a bite of a small piece of concha to pull the lobule toward the head.

To address the underdeveloped antihelical fold, Mustardé sutures are placed like spokes on a wheel between the scapha to the concha or triangular fossa.[52,53] Needles can be used to mark where mattress sutures should be placed and permanent suture, such as 4–0 clear Nylon, should be used. Scoring has been described in which the cartilage will bend away from the scoring, but this is not necessary to be used.

Prominent concha can be addressed with two methods most commonly used in combination. One is to resect concha at the junction between the posterior wall and floor. This is a minimal resection usually limited to 2 mm. The other method places a 3–0 Nylon Furnas mattress suture between the concha and mastoid to set back the ear.[54] Refer to ▶ Fig. 15.6 for a demonstration of suture placement. The skin incision is then closed with a 5–0 gut suture.

15.12 Cryptotia

Cryptotia is a congenital abnormality common in Asian populations in which the upper part of the retroauricular sulcus is absent and the skin merges directly with the temporal region. The buried part of the helix can be revealed with traction. If there is a mild deformity, it can be treated conservatively before the age of 2 years. For the more severe deformities or those present past childhood, Nagata has described a combined Z-plasty and advancement flap for his surgical technique.[55]

With the ear held in an extended position, the Z-flap is designed. The anterior limb is 5 to 10 mm anterior to the ascending margin of the helix. Each limb is 1.5 to 2.0 cm long, with wider flaps producing better results. Another incision is used from the end of the posterior Z-limb onto the retroauricular skin to fully expose cartilage. Scissors are used to completely separate the soft tissue from the buried cartilage. Depending on the severity of cartilage deformity, parallel scores can be made over the posterior aspect of the superior and inferior crus and mattress sutures can be applied. The temporal and mastoid skin is then advanced anteriorly and the Z-flap is rotated to cover the posterior ear. The Z-flap donor site is closed in a straight line.[55]

15.13 Stahl's Ear

The Stahl ear deformity has a third crus or an abnormal bar of cartilage extending from the antihelix to the helix at the junction between the upper and middle thirds of the ear. The third crus is removed and this cartilage is used to augment the superior crus of the antihelix.[49]

15.14 Inadequate Helical Rim (Shell Ear)

Shell ear is characterized by absence of a helical overhang. An incision is made just inside the helical rim through the skin and cartilage is usually enough to create helical definition. However, if this does not adequately address the deformity, a wedge of helical rim skin and cartilage can be removed to curl the helical rim.[49]

15.15 Question Mark Ear

The question mark ear deformity is rare and can occur spontaneously or in an autosomal dominant fashion. It is characterized by a cleft between the lower part of the helix and the earlobe that is due to an arrest of fusion between the fifth and

sixth auricular hillocks. Ear reconstruction should be delayed until after mandibular deficiency has been addressed. Surgery reduces the size and prominence of the upper pole and corrects the cleft. Cartilage deficiency is addressed by rib or contralateral conchal cartilage grafts. Skin deficiency is corrected by a V-Y flap from the posterior ear. A Z-plasty can be performed to improve the notch.[56]

15.16 Postoperative Care

Some surgeons prescribe broad-spectrum oral antibiotics for the first few postoperative days to decrease the risk of chondritis, although no high-level evidence documents efficacy. Postoperatively, the ear is packed with Vaseline gauze or Xeroform and a bulky dressing is applied. Care is taken to not compress the ear with a bandage since suction drains are already functioning. These drains are usually removed on postoperative day 5. Sutures are removed at 1 week and bandages can be discontinued after 2 weeks. Children return to school after 2 weeks and can return to sports after approximately 5 weeks.[12]

15.17 Outcomes

The more complex the surgical procedure, the more likelihood for complications. Immediate complications related to Tanzer's microtia reconstruction included exposure of cartilage, small losses in skin grafts, ischemic changes from compression sutures, extrusion of metal sutures, contour irregularities, pleural tears during cartilage harvest, and seromas of the abdominal wall.[20] Tanzer reviewed his work after 44 cases and found that late complications were blurring of contours, metal suture extrusion, and chest depression or hypertrophic scarring from cartilage harvest.[24] There was no extrusion of framework. In addition, Tanzer found that the reconstructed framework grew with the child and affirmed the established knowledge that ear growth tapers after 10 years of age.[20]

Brent published a series of follow-up articles based on his total 600, 1,000, and 1,200 cases to provide recommendations based on outcomes.[57,58,59] To prevent chest wall deformities, he would leave a superior rim of sixth rib cartilage. In fabricating the framework, he only used chisels and scalpels, while preserving perichondrium when able. He secured the helical cartilage graft to the framework with a series of 4–0 and 5–0 clear nylon sutures to avoid problems with wire suture extrusion. He incorporated a tragal cartilage strut into the initial framework to avoid another procedure. In older patients with rib cartilage that is fused, the framework was carved from a single block of cartilage and, only if needed, was the helix portion detached and advanced for projection. For issues with a low hairline, Brent evolved to pretreat the ideal hairline with laser hair removal prior to the first stage of reconstruction. To create a posterior auriculocephalic sulcus, the framework was elevated and a wedge of rib graft was placed behind the ear and covered with a turnover of occipitalis fascia. This cartilage is harvested at the first stage and banked posteriorly to the framework insertion site.

Outcomes for correction of existent, but abnormal, ears largely depend on the severity of malformation. Complications include overcorrection, sharp edges, unnatural contours, telephone deformity, infection, and suture complications. Contours and appearance are maximized by proper evaluation. Thorne simplifies the evaluation of the ear deformity into thirds and stresses that in order to achieve a natural look the ear must be observed from all angles.[51]

Ear reconstruction and otoplasty both have a rich history, with several prominent individuals contributing new ideas aimed at eliminating complications and improving outcomes for patients. It is essential to know the normal anatomy in order to correct various abnormalities, whether for microtia or acquired ear deformities. While many options exist for patients, the goal, at its most basic, is to make an ear that looks right for the right patient.[15] With proper attention to detail inclusive of preoperative evaluation, meticulous surgical technique, proper timing, and technical selection, excellent results can be achieved, providing a lifetime improvement for these patients.

15.18 Review Questions

15.18.1 Choose the Best Answer

1. Patients with congenital ear deformities
   a) Usually have multiple facial deformities along with the ear abnormality.
   b) Most often have middle and inner ear problems with hearing loss.
   c) Present with a lone deformity, usually confined to the pinna.
   d) Usually have an autosomal dominant transmission.
2. Otoplasty surgery is usually done at age 5 years or later due to
   a) Ear is at near adult size by this age.
   b) Patient can more readily cooperate with postoperative care.
   c) There is limited peer ridicule until school age, around 5 years.
   d) Patients have greater awareness of appearance and self image.
   e) All of the above.
3. Total ear reconstruction for Microtia is
   a) Performed prior to age 5 so as to avoid social ridicule.
   b) Must be done when children are near teenage years since the ear constructs do not grow if done at much younger ages.
   c) Most often a single stage procedure.
   d) Done in older children to assure enough cartilage availability for the carves framework.
4. Total ear reconstruction for Microtia is
   a) Performed prior to age 5 so as to avoid social ridicule.
   b) Must be done when children are near teenage years since the ear constructs do not grow if done at much younger ages.
   c) Most often a single stage procedure.
   d) Done in older children to assure enough cartilage availability for the carves framework.
5. Prominent ears are
   a) Usually due to under developed antihelical fold, a prominent conchal bowl or protruding earlobe.
   b) No matter the defect, prominent ears are surgically corrected via conchal bowl resection.

c) Antihelical fold creation is most often done by cartilage resection and suture reshaping.
d) Scoring or rasping cartilage has no effect on cartilage shape or bend.

15.18.2 Answers

1. Present with a lone deformity, usually confined to the pinna (c).
2. All of the above (e).
3. Done in older children to assure enough cartilage availability for the carves framework (d).
4. Performed prior to age 5 so as to avoid social ridicule (a).
5. Usually due to under developed antihelical fold, a prominent conchal bowl or protruding earlobe (a).

References

[1] Karmody CS, Annino DJ , Jr. Embryology and anomalies of the external ear. Facial Plast Surg. 11(4):251–256
[2] Wright CG. Development of the human external ear. J Am Acad Audiol. 8(6): 379–382
[3] Adamson JE, Horton CE, Crawford HH. The growth pattern of the external ear. Plast Reconstr Surg. 36(4):466–470
[4] Farkas LG. Anthropometry of normal and anomalous ears. Clin Plast Surg. 5 (3):401–412
[5] Farkas LG. Anthropometry of the normal and defective ear. Clin Plast Surg. 17 (2):213–221
[6] Tolleth H. Artistic anatomy, dimensions, and proportions of the external ear. Clin Plast Surg. 5(3):337–345
[7] Tolleth H. A hierarchy of values in the design and construction of the ear. Clin Plast Surg. 17(2):193–207
[8] Spielmann PM, Harpur RH, Stewart KJ. Timing of otoplasty in children: what age? Eur Arch Otorhinolaryngol. 266(6):941–942
[9] Spira M. Otoplasty: what I do now–a 30-year perspective. Plast Reconstr Surg. 104(3):834–840, discussion 841
[10] Wolfe SA. Timing of otoplasty for prominent ears. Plast Reconstr Surg. 117(2): 680–681, author reply 680–681
[11] Gosain AK, Kumar A, Huang G. Prominent ears in children younger than 4 years of age: what is the appropriate timing for otoplasty? Plast Reconstr Surg. 114(5):1042–1054
[12] Bauer B. Congenital and acquired deformities of the ear. In: Bentz ML, Bauer BS, Zuker RM, eds. Principles and Practice of Pediatric Plastic Surgery. St. Louis, MO: Quality Medical Publishing; 2008: 801–850
[13] Bradbury ET, Hewison J, Timmons MJ. Psychological and social outcome of prominent ear correction in children. Br J Plast Surg. 45(2):97–100
[14] Vardanian A, Tong A, Lee JC, Federico C, Bradley JP. Abstract 49: Increased infection in total ear reconstruction patients with patent external auditory canal warrants a perioperative treatment protocol. Plast Reconstr Surg. 131 5S:45
[15] Thorne CH. Ear Reconstruction for Microtia, Otoplasty, and Ear Reduction. Visiting Professor Lecture. University of Wisconsin Plastic and Reconstructive Surgery Division. Madison, Wisconsin; 2015
[16] Tanzer RC. Dartmouth College Office of Public Affairs. Press Release. June 16, 2003. Accessed on January, 15, 2016. Available at: http://www.dartmouth.edu/~news/releases/2003/june/061603a.html. Accessed November 28, 2017
[17] Tanzer RC. Total reconstruction of the external ear. Plast Reconstr Surg Transplant Bull. 23(1):1–15
[18] Tanzer RC. An analysis of ear reconstruction. Plast Reconstr Surg. 31:16–30
[19] Tanzer RC. The reconstruction of acquired defects of the ear. Plast Reconstr Surg. 35:355–365
[20] Tanzer RC. Total reconstruction of the auricle: a 10-year report. Plast Reconstr Surg. 40(6):547–550
[21] Tanzer RC. Secondary reconstruction of microtia. Plast Reconstr Surg. 43(4): 345–350
[22] Tanzer RC. Total reconstruction of the auricle. The evolution of a plan of treatment. Plast Reconstr Surg. 47(6):523–533
[23] Tanzer RC. The aesthetics of ear placement. Plast Reconstr Surg. 73(4):702–703
[24] Tanzer RC. Microtia–a long-term follow-up of 44 reconstructed auricles. Plast Reconstr Surg. 61(2):161–166
[25] Tanzer RC. An interview with Radford C, Tanzer, M.D. conducted by William D. Morain, M.D. Ann Plast Surg. 10(1):72–85
[26] Brent B, Tanzer RC. The correction of microtia with autogenous cartilage grafts. A discussion by Radford Tanzer. Plast Reconstr Surg. 66(1):22
[27] Brent B. The reconstruction of venus: following our legacy. Plast Reconstr Surg. 121(6):2170–2171
[28] Brent B. The correction of mi-rotia with autogenous cartilage grafts: I. The classic deformity.? Plast Reconstr Surg. 66(1):1–12
[29] Brent B. The correction of microtia with autogenous cartilage grafts: II. Atypical and complex deformities. Plast Reconstr Surg. 66(1):13–21
[30] Brent B, Byrd HS. Secondary ear reconstruction with cartilage grafts covered by axial, random, and free flaps of temporoparietal fascia. Plast Reconstr Surg. 72(2):141–152
[31] Yanai A, Fukuda O, Yamada A. Problems encountered in contouring a reconstructed ear of autogenous cartilage. Plast Reconstr Surg. 75(2): 185–191
[32] Brent B, Upton J, Acland RD, et al. Experience with the temporoparietal fascial free flap. Plast Reconstr Surg. 76(2):177–188
[33] Nagata S. A new method of total reconstruction of the auricle for microtia. Plast Reconstr Surg. 92(2):187–201
[34] Benmeir P, Weinberg A, Neuman A, Wexler MR. Reconstruction of the auricle. Plast Reconstr Surg. 96(1):232–233
[35] Kawanabe Y, Nagata S. A new method of costal cartilage harvest for total auricular reconstruction: part I. Avoidance and prevention of intraoperative and postoperative complications and problems. Plast Reconstr Surg. 117(6): 2011–2018
[36] Kawanabe Y, Nagata S. A new method of costal cartilage harvest for total auricular reconstruction: part II. Evaluation and analysis of the regenerated costal cartilage. Plast Reconstr Surg. 119(1):308–315
[37] Nagata S. Modification of the stages in total reconstruction of the auricle: Part I. Grafting the three-dimensional costal cartilage framework for lobule-type microtia. Plast Reconstr Surg. 93(2):221–230, discussion 267–268
[38] Nagata S. Modification of the stages in total reconstruction of the auricle: Part II. Grafting the three-dimensional costal cartilage framework for concha-type microtia. Plast Reconstr Surg. 93(2):231–242, discussion 267–268
[39] Nagata S. Modification of the stages in total reconstruction of the auricle: Part III. Grafting the three-dimensional costal cartilage framework for small concha-type microtia. Plast Reconstr Surg. 93(2):243–253, discussion 267–268
[40] Nagata S. Modification of the stages in total reconstruction of the auricle: Part IV. Ear elevation for the constructed auricle. Plast Reconstr Surg. 93(2):254–266, discussion 267–268
[41] Chen ZC, Goh RC, Chen PK, Lo LJ, Wang SY, Nagata S. A new method for the second-stage auricular projection of the Nagata method: ultra-delicate split-thickness skin graft in continuity with full-thickness skin. Plast Reconstr Surg. 124(5):1477–1485
[42] Baluch N, Nagata S, Park C, et al. Auricular reconstruction for microtia: a review of available methods. Plast Surg (Oakv). 22(1):39–43
[43] Firmin F. Ear reconstruction in cases of typical microtia. Personal experience based on 352 microtic ear corrections. Scand J Plast Reconstr Surg Hand Surg. 32(1):35–47
[44] Firmin F. La reconstruction auriculaire en cas de microtie. Principes, méthodes et classification. Ann Chir Plast Esthet. 46(5):447–466
[45] Firmin F, Marchac A. A novel algorithm for autologous ear reconstruction. Semin Plast Surg. 25(4):257–264
[46] Reinisch JF, Lewin S. Ear reconstruction using a porous polyethylene framework and temporoparietal fascia flap. Facial Plast Surg. 25(3):181–189
[47] Lewin S. Complications after total porous implant ear reconstruction and their management. Facial Plast Surg. 31(6):617–625
[48] Sinno S, Chang JB, Thorne CH. Precision in otoplasty: combining reduction otoplasty with traditional otoplasty. Plast Reconstr Surg. 135(5):1342–1348
[49] Thorne CH, Wilkes G. Ear deformities, otoplasty, and ear reconstruction. Plast Reconstr Surg. 129(4):701e–716e
[50] Tanzer RC. The constricted (cup and lop) ear. Plast Reconstr Surg. 55(4):406–415
[51] Thorne CH. Otoplasty. Plast Reconstr Surg. 122(1):291–292
[52] Mustardé JC. The treatment of prominent ears by buried mattress sutures: a ten-year survey. Plast Reconstr Surg. 39(4):382–386

[53] Mustardé JC. The correction of different types of prominent ears. Aesthetic Plast Surg. 7(3):163–168
[54] Furnas DW. Correction of prominent ears by concha mastoid sutures. Plast Reconstr Surg. 42(3):189–193
[55] Yanai A, Tange I, Bandoh Y, Tsuzuki K, Sugino H, Nagata S. Our method of correcting cryptotia. Plast Reconstr Surg. 82(6):965–972
[56] Greig AV, Podda S, Thorne CH, McCarthy JG. The question mark ear in patients with mandibular hypoplasia. Plast Reconstr Surg. 129(2):368e–369e
[57] Brent B. Auricular repair with autogenous rib cartilage grafts: two decades of experience with 600 cases. Plast Reconstr Surg. 90(3):355–374, discussion 375–376
[58] Brent B. Microtia repair with rib cartilage grafts: a review of personal experience with 1000 cases. Clin Plast Surg. 29(2):257–271, vii
[59] Brent B. Technical advances in ear reconstruction with autogenous rib cartilage grafts: personal experience with 1200 cases. Plast Reconstr Surg. 104(2): 319–334, discussion 335–338

16 Nevi and Vascular Malformations

Christopher J.M. Brooks

Abstract
This chapter discusses the salient considerations involved in determining the status and treatment of nevi (moles) and vascular malformations. The ABCDE criteria for screening nevi and determining possible malignancies are detailed. Guidelines for excision and biopsy are reviewed, and special consideration is given to giant congenital melanocytic nevi. Vascular malformations—including hemangioma, capillary, venous, lymphatic, and arteriovenous malformations—are also addressed. Postoperative care and outcome guidelines, including cosmetic considerations, are reviewed.

Keywords: nevi, dysplastic nevus, atypical nevus, ABCDE, giant congenital melanocytic nevi, vascular malformation, hemangioma

16.1 Goals and Objectives

- Determine which nevi are suspicious and require excision or biopsy.
- Understand the risks and treatment options for giant congenital pigmented nevi.
- Appreciate the difference in presentation and typical course of the subtypes of vascular malformation.
- Understand the treatment options available for each subtype.

16.2 Patient Presentation

Patients with nevi frequently present either on their own or referred by another physician (primary care or dermatologist). Frequently referred to colloquially in the United States as "moles," the term "nevus" encompasses a broad range of different dermatologic findings. Typically, a nevus is a pigmented lesion on the skin which may or may not be raised and may or may not be hair bearing. Most are benign, but some need to be followed closely, biopsied or excised.

The standard benign pigmented nevus is called a melanocytic nevus. These are typically small, brown, or black lesions with smooth borders and even coloring. These lesions should be followed over time and any change in the characteristics of that lesion noted. There are risk factors which are determined based on the appearance of the lesion which can determine its likelihood to contain malignancy or progress to malignancy (melanoma). The acronym ABCDE has become a widely publicized tool for screening nevi for the possibility of malignancy.

A: Asymmetry—one half is not like the other half.

B: Border—an irregular, scalloped, or poorly defined border.

C: Color—is varied from one area to another; has shades of tan, brown or black; or is sometimes white, red, or blue.

D: Diameter—melanomas are usually greater than 6 mm (which is around the size of a pencil eraser) when diagnosed, but they can be smaller.

E: Evolving—a skin lesion that looks different from the rest or is changing in size, shape, or color.[1]

Those lesions that meet one or more of the ABCDE criteria need further screening as that lesion may represent a dysplastic nevus or melanoma. A dysplastic nevus, or atypical nevus, is one that meets the clinical criteria above and may also have cellular atypia on histologic examination. These dysplastic nevi are usually benign and stable, but they are considered a risk for transformation to melanoma.

Dysplastic nevi may occur singularly, or as part of a larger disorder known as dysplastic nevus syndrome or familial atypical multiple mole melanoma syndrome (FAMMM). Patients with this condition have had multiple dysplastic nevi and at least one close relative who has had melanoma. These patients are at increased risk of melanoma and need to be followed every 6 months to yearly by a dermatologist for complete examination, total body photography, and dermoscopy.[2]

Aside from the ABCDE lesions, there are other types of nevi which warrant consideration. A pigmented nevus which has a surrounding ring of hypopigmentation is called a halo nevus (▶ Fig. 16.1). The origins of these lesions is not understood, but it is surmised that an immune response was responsible for the involution of the pigmented area.[3] The appearance of a halo nevus should prompt an investigation looking for a primary melanoma of the skin or the eye as these have been associated with occult melanoma. Excision of these lesions is prompted by the clinical history of melanoma, atypical appearance of the lesion (ABCDE), or presence of multiple other atypical nevi.[4]

A salmon-colored nevus usually located on the head and neck area is called a nevus sebaceous (of Jadhasson; ▶ Fig. 16.2). These lesions are present at birth and are flat. They typically become raised and develop a rough surface around puberty. Due to the risk for development of basal cell carcinoma, these lesions have typically been excised before puberty. However, studies have shown the incidence is less than 1%, so many are now excised only for cosmetic reasons.[5]

Another type of nevus is a dome-shaped reddish or dark brown papule typically found on the head and neck and sometimes the thigh. Termed a Spitz nevus, it is common in children and rare in adults. Also known as "benign juvenile melanoma," these nevi were once thought to be a form of childhood melanoma, but are now known to be benign nevi. These lesions usually require close observation or excision due to observational concern or confusion with melanoma. While there is no consensus, simple excision is usually adequate with 5-mm margins reserved for reexcision of pathologically atypical lesions.[6]

When children are born with very large pigmented nevi (> 20 cm in adults or predicted to reach 20 cm in diameter by adulthood. Or, 9 cm on an infant's head or 6 cm on an infant's body), they are termed "giant congenital melanocytic nevi."[7] These lesions are often disfiguring and can be complicated by malignant melanoma and neurocutaneous melanosis. While these lesions are always present at birth, the extent of the lesion may not be known at birth due to variable degrees of pigmentation at birth which may increase over time.[4]

Giant congenital melanocytic nevi most commonly occur on the trunk (sometimes called bathing trunk nevi) (▶ Fig. 16.3) followed by the extremities and then the head and neck.[8]

Fig. 16.1 Example of halo nevus. (From Rocken M, Schaller M, Sattler E, Burgdorf W. Color Atlas of Dermatology. New York, NY: Thieme; 2012:255.)

Fig. 16.2 Nevus sebaceus in the postauricular hairline. (From Rocken M, Schaller M, Sattler E, Burgdorf W. Color Atlas of Dermatology. New York, NY: Thieme; 2012:209.)

Although at birth the lesions may be smooth and flat, over time the appearance and texture may change. They often develop prolific hair growth (1–2 years), a verrucous texture and variegation in color (around 10 years).[9] Most often there are also associated satellite lesions which can be located distant to the site of the main nevus. Children with leptomeningeal involvement sometimes present with seizures.[10]

Typically, these giant lesions will expand in proportion to the body's growth. The changes to the characteristics of these lesions need to be followed closely due to the risk of malignant degeneration to cutaneous melanoma or even noncutaneous melanoma (e.g., mucosa of gastrointestinal tract and retroperitoneum). The overall reported rate of malignant melanoma in these patients has ranged from 1.8 to 45%.[11] More recent studies have reported 2.8 to 8.5% with a lifetime risk of 6.3% and a relative risk of 17. Fifty percent of the malignancies arise during the first 3 years of life and 70% by puberty.[12]

16.2.1 Preparation for Surgery

Most often the patients who will require surgery for nevi will be healthy. Age-related risk factors should be addressed as usual. In general, aside from any prior biopsy results, no further examination is warranted. The exception to this is in the children who present with a giant congenital melanocytic nevus and risk factors for neurocutaneous melanosis. These children have greater than 20 satellite nevi and congenital melanocytic nevi in a midline location over the trunk and calvaria.[8, 13] This subgroup should be screened with MRI imaging of the brain and spinal cord between 4 and 6 months of age. Those with positive MRI imaging should be referred to neurosurgery, but the treatment of the cutaneous nevi need not be delayed.[12]

16.3 Treatment

Pigmented lesions which meet the criteria for suspicion require biopsy or excision. Anesthesia and location for the surgical excision is dependent on the patient's age and comorbidities, as well as the size and location of the lesion. Most young children will require a general anesthesia in an appropriate operating room setting with a pediatric anesthesia provider. Older children and adults with small to medium size nevi can be excised in an office or operating room with local anesthesia alone, local anesthesia with sedation, or general anesthesia. Very large lesions will probably necessitate general anesthesia. The author typically also utilizes 0.25% bupivacaine with epinephrine injected 7 or more minutes prior to incision to improve

Fig. 16.3 Giant congenital melanocytic nevus of the posterior trunk and upper buttocks. (From Rocken M, Schaller M, Sattler E, Burgdorf W. Color Atlas of Dermatology. New York, NY: Thieme; 2012:255.)

Fig. 16.4 A 5-year-old boy with giant congenital hairy nevus undergoing tissue expansion.

intraoperative hemostasis and to assist with postoperative pain control. Patients undergoing excision using local anesthesia alone or in conjunction with sedation will receive a 50:50 mixture of 0.25% bupivacaine with epinephrine and 1% lidocaine with epinephrine at least 7 minutes prior to incision.

Excision of these lesions ***needs*** to be full thickness. This is most commonly done as a full-thickness excision with 0 to 2 mm margins. A punch biopsy may be used if the punch incorporates the entire lesion. Very large lesions may be partially excised with a multiple full-thickness punch biopsies or incisional biopsies, but this introduces a significant amount of sampling error and should therefore be avoided. Shave biopsies of pigmented lesions are prohibited as this will destroy the crucial information of invasive depth of the lesion should the nevus turn out to be malignant. All excised tissue is sent for histologic examination. The results of this examination will dictate the need for any further excision.

For multiple reasons, giant congenital melanocytic nevi are treated in early childhood (as early as age 6 months).[14] The rational for surgical excision is to decrease the risk of malignant degeneration, aesthetic and psychosocial improvement, and to improve the ability to screen the lesions. While some have advocated the use of minimally invasive techniques to treat these lesions (curettage, dermabrasion, chemical peeling, lasers), none of these methods eliminates the risk of malignant degeneration.[12]

Full-thickness excision of a giant congenital melanocytic nevus is most often recommended in the plastic surgery literature. Given the sheer size of these lesions, excision and reconstruction is often a challenge. Techniques include serial excision, tissue expansion, skin grafting, local and regional flaps, and free tissue transfer (▶ Fig. 16.4). A thorough discussion with the patient and/or family is clearly required detailing the risks and benefits for each of the procedures. Most often, if an excision can be accomplished in three or fewer stages, then serial excision with a minimum of 6-month intervals is recommended.[8] If four or more stages would be required, then tissue expansion becomes the preferred choice. Tissue expander techniques can be utilized to expand tissue for use as local, regional, or even free flaps. With the success of tissue expansion, the use of skin grafts, which often have suboptimal aesthetic results, has become much less common.[12]

16.3.1 Postoperative Care

Postoperative care following excisional biopsy of atypical or other nevi is minimal. The wounds generally are small and heal quickly. The author most commonly utilizes dissolving suture

and a skin adhesive as a dressing. This minimizes any necessary wound care and allows the patient a rapid return to normal bathing and other activities including swimming.

Following excision, patients need to be followed closely by a dermatologist. The interval at which they need to be seen will be determined by the diagnosis. Typically patients initially should be followed at 6-month intervals unless the nevus shows no atypia. Those with mild atypia are typically followed clinically. Those with moderate or severe atypia are often reexcised with 2 mm margins.

Postoperative care of the larger reconstructive endeavors for the giant congenital melanocytic nevi will depend on the procedure employed. These patients are frequently admitted after surgery for pain control and flap monitoring if necessary. Cases requiring placement of tissue expanders can be sent home with the majority of the expansion taking place as an outpatient—sometimes even at home by a responsible family member.

16.3.2 Outcomes

In general, complete excision of dysplastic nevi and other benign nevi will remove their malignant potential. Cosmetic outcomes will vary by the location and quality of the scar.

Oncological outcomes of treatment of giant congenital melanocytic nevi are related to the completeness of the excision and the degree of extracutaneous manifestation. Cosmetic outcomes are related to the size and location of the nevi, the techniques employed in reconstruction, and the quality of the scar.

16.4 Vascular Malformations

16.4.1 Patient Presentation

The term "vascular malformation" is steeped in controversy. It is really just a broad term which encompasses several very different lesions with very different presentations, different risks, and different treatment protocol.

Fig. 16.5 A 4-month-old girl with scalp hemangioma.

Since there is no classic presentation for a "vascular malformation," this term will be broken down into its individual components. A vascular malformation can be a hemangioma, a venous malformation, a lymphatic malformation, a capillary malformation, or an arteriovenous malformation. Several classification systems have been proposed. The most commonly used in the plastic surgery literature is that of Mulliken and Glowacki.[15]

A hemangioma has proliferative endothelium, whereas the other types of vascular malformation have a stable endothelium. That means that a hemangioma is technically a type of tumor, while a vascular malformation is typically a benign lesion. The vascular malformations can be further broken down into fast flow (arteriovenous malformations) and slow flow (capillary malformations, venous malformations, lymphatic malformations).[16,17] There are also mixed lesions (lymphaticovenous malformations) which further complicate the discussion. This section will concentrate on the most common.

16.4.2 Hemangioma

Hemangioma typically presents at birth as a very small red dot which often goes unnoticed. Over the ensuing weeks and months, this red lesion will grow very rapidly (out of proportion to the child) and can quickly become very disfiguring. Larger lesions may grow so rapidly that they outpace the development of the blood supply and they can become ulcerated, typically in the center of the lesion. The clinical picture of an inauspicious beginning and incredibly rapid growth of a vascular tumor is typically enough to make the diagnosis of a hemangioma. MRI imaging can confirm this diagnosis, but is usually not necessary (▶ Fig. 16.5).

The life cycle of the hemangioma is quite standard. Most often there is the explosive growth phase which typically lasts from 6 to 12 months. This phase is followed by a quiescent phase which can last from months to years. As the tumor enters this phase, it is common that the bright red skin will become gray and dusky in the center as the rapid growth comes to an end. After an unpredictable period of time in the quiescent phase, an involutional phase begins. During this time, the vascular portion of the tumor begins to shrink and, depending on the size of the original lesion, the entire area may revert to normal without any intervention. It is common for larger lesions to leave behind abnormal skin which is atrophic and inelastic. We have come to expect that 50% of these hemangiomas will resolve within 5 years, 90% within 9 years.

16.4.3 Treatment

Since many of these lesions will resolve spontaneously, watchful waiting can be a valid treatment plan. The exceptions to this are when the tumor is causing an airway obstruction or obstructing the visual axis during development. The latter scenario will lead to amblyopia which is a permanent visual field loss secondary to this obstruction. Obviously, these situations require a more aggressive form of intervention.

Injection of steroids has been shown to slow the growth of these tumors and can even lead to the shrinkage of the lesion. This is a treatment which often needs to be repeated more than one time in order to see a significant effect. The use of a tunable dye laser is practiced by some physicians in an effort to address the discoloration of the skin. While there are a few scattered reports of success with this modality, given the extremely limited depth of penetration of the laser, this treatment is unlikely to be effective in the definitive management of hemangiomas. The laser can, however, be used secondarily to treat any residual superficial skin discoloration or hypervascularity once involution has occurred. Surgical excision is always an option for these tumors. One must balance the morbidity and potential complications and sequelae for surgery against the risks and stigmata of watchful waiting. Clearly there is a continued role for surgical excision—especially in the emergent treatment of lesions obstructing the airway or visual axis. Excision of these lesions can be (not surprisingly) quite bloody and can lead to risks to surrounding vital structures (e.g., nerves) due to the small size of the child and the poor visualization. A fairly new and increasingly popular treatment for hemangioma is the use of orally administered propanolol, a beta-blocker which has been shown to quickly and significantly shrink these tumors. The mode of action is not fully understood. Risks of this medication include changes in sleep, acrocyanosis, hypotension, and hypoglycemia.[18] The safety and efficacy of treatment with propranolol often makes this the first-line treatment even in nonemergent but urgent treatment of airway and visual axis obstruction. Another time to consider this treatment is for rapidly growing and/or large lesions in vital or cosmetically sensitive areas such as the face, or lesions that are beginning to ulcerate and bleed.

16.4.4 Capillary Malformations

Capillary malformations are historically referred to as port wine stains. (This terminology is archaic and should be avoided.) They typically appear as a pink or red skin discoloration at birth. As the child ages, however, these lesions may become increasingly thick, dark, and nodular resulting in severe disfigurement. They can also sometimes result in changes to the underlying bone. They can occur anywhere in the body. Certain locations can be associated with syndromes (Proteus, Klippel–Trenaunay, Sturge–Weber) indicating other anomalies or pathology in other organ systems.[19] Diagnosis is most often based on the clinical appearance of the skin lesion but can be further confirmed with MRI. Suspected syndromes should be worked up to exclude associated pathology. Treatment of the capillary malformation is most often with multiple sessions with a 585-nm pulsed dye laser. Treatment results are variable, but often can lead to the near-complete resolution of the discoloration and a good cosmetic outcome.

16.4.5 Venous Malformations

Venous malformations were once called “cavernous malformations” and are present at birth, though not always visible. They are the most common of the vascular malformations and are most frequently found in the head and neck regions, although may occur throughout the body (▶ Fig. 16.6).[20] They are usually noted as a bluish discoloration which may involve the skin or the subcutaneous tissue, or even the underlying muscle, bone, or viscera. They typically grow as the child grows, but may exhibit growth spurts. Parents may report that the lesions enlarge or become darker when the child is upset and crying. These lesions may be painful or tender from thrombosis within the malformation leading to phleboliths. They can be quite disfiguring and can cause severe asymmetry and even limb hypertrophy. Airway obstruction is possible, though is not as acute as seen in the rapid-growing hemangiomas. Diagnosis is typically clinical, though the extent of the lesion is usually determined by MRI examination. Treatment of a venous malformation depends on its size and location. Where applicable (e.g., the limb), compression garments can be effective in reducing the diameter of the affected area and sometimes also the pain. Anticoagulants such as aspirin can be given to reduce the frequency of painful thrombosis and phlebolith formation. Most commonly used are sclerosants which are injected directly into the malformation to cause thrombosis secondary to endothelial damage. Multiple agents have been used including hypertonic saline, absolute ethanol, and sodium tetradecyl sulfate. Multiple treatments may be required and complications such as ulceration, skin necrosis, nerve injury are not uncommon. More seri-

Fig. 16.6 Venous malformation affected the right buttock and upper lateral thigh. (From Rocken M, Schaller M, Sattler E, Burgdorf W. Color Atlas of Dermatology. New York, NY: Thieme; 2012:23.)

Fig. 16.7 (**a**) A 22-year-old man with upper lip arteriovenous malformation—pre-op and (**b**) 6 months after combined treatment with embolization and immediate surgical resection.

ous complications such as compartment syndrome and systemic toxicity have been reported.[20]

Surgical treatment of venous malformations is sometime warranted and can be done in conjunction with sclerotherapy. Due to the amorphous nature of these lesions, complete excision is often difficult. Indications for surgery include severe functional or appearance-related problems such as bleeding, pain, or immobility.

16.4.6 Lymphatic Malformations

Lymphatic malformations are historically known as cystic hygromas or lymphangiomas. Most are present at birth but some are discovered up to age 2 or even older. The cervicofacial region is most commonly involved, but lymphatic malformations can occur anywhere. These lesions are progressive and can cause soft tissue and bony hypertrophy ultimately leading to soft tissue and skeletal overgrowth such as limb hypertrophy. Steady growth of the lesions can be expected. Complications such as infection or bleeding are not uncommon. Infection can lead to the rapid swelling of the lesion and associated pain and erythema. Antibiotic therapy will usually resolve the infection, but rapid swelling of a cervicofacial lymphatic malformation has been known to cause airway obstruction and a surgical emergency.[21] The lesions are often bluish and can cause skin surface irregularities. MRI is often used to delineate the extent of larger and symptomatic lesions. This modality can also be used to determine whether the malformation is macrocystic, microcystic, or mixed. Macrocystic malformations sometimes spontaneously involute.[22] Treatment of these lesions is limited to compression, sclerotherapy, or surgical excision. Possible sclerosing agents include hypertonic saline, absolute ethanol, sodium tetradecyl sulfate, bleomycin, and OK-432. Surgical excision is reserved for those lesions which are functionally or aesthetically unacceptable.[19] Recurrence is common when the lesions are incompletely excised. Staged excisions and excision in conjunction with sclerotherapy is common. Local wound complications are frequent due to infection and serum formation. Prophylactic drain use and antibiotics are highly recommended.

16.4.7 Arteriovenous Malformations

Arteriovenous malformations are distinct from the aforementioned malformations secondary to their high-flow state. As the name would indicate, these malformations result from the direct connection of an artery and a vein. Due to the pressure differential, the flow through these lesions is significant. Adjacent vessels are then recruited leading to the growth of the malformation. Growth can be steady but punctuated by rapid enlargement secondary to trauma or hormonal influence. These typically progress from discoloration to palpable lesions with pulses and thrills. They then enlarge and may develop ulceration, bleeding, and pain before ultimately leading to a very high flow state and congestive heart failure. The palpable pulsation and thrills in the lesion are usually enough to determine the diagnosis. However, the extent of the lesion is often determined by MRA (magnetic resonance angiography) which can also help determine the appropriate treatment. Small lesions can be directly excised. Larger lesions are most often treated with multidisciplinary approach with an interventional radiologist providing embolization followed by surgical resection within 24 to 48 hours. Surgical excision of large lesions without the assistance of preoperative embolization can lead to significant and dangerous blood loss. Embolization without surgical excision or even with excision delayed beyond the 48-hour window leads to recollateralization and likely recurrence (▶ Fig. 16.7).

Preparation for surgery and treatment depend most on the clinical and sometimes radiographic diagnosis. Other factors are related to the patient's age and comorbidities. The postoperative course and management depend entirely on the type of lesion and the treatment. Larger and more aggressive lesions are likely to require secondary resection or embolization. Completely resected lesions rarely recur, but may lead to aesthetic deformities which require secondary reconstruction or revision.

16.5 Review Questions

16.5.1 True or False

1. Biopsies of pigmented lesions can be performed by a shave, or partial-thickness, technique.
2. A pigmented nevus which has been excised with positive margins and a pathologic diagnosis suggesting moderate atypia should be reexcised.
3. Children who present with a giant congenital melanocytic nevus in the midline or one with greater than 20 satellite lesions should undergo MRI examination.
4. Treatment of a hemangioma with a beta-blocker is an appropriate management strategy.

5. A large mass with a palpable pulse and a thrill may benefit from multidisciplinary approach involving embolization and surgical resection.

16.5.2 Answers

1. False. The important prognostic and treatment information which is provided by the depth of a malignant pigmented lesion (e.g., melanoma) is often destroyed by a partial-thickness or shave technique.
2. True. Lesions with moderate or severe atypia and positive margins are usually reexcised.
3. True. These children are at risk of having neurocutaneous melanosis which is diagnosed with MRI.
4. True. Propanolol has been shown to cause the involution of hemangiomas.
5. True. Lesions with a palpable pulse and thrill are most often arteriovenous malformations. A multidisciplinary approach involving preoperative embolization followed by surgical resection within 24 to 48 hours will reduce blood loss and thereby allow for better intraoperative visualization and reduced risk to adjacent vital structures.

References

[1] American Academy of Dermatology. Available at: AAD.org
[2] de Snoo FA, Kroon MW, Bergman W, et al. From sporadic atypical nevi to familial melanoma: risk analysis for melanoma in sporadic atypical nevus patients. J Am Acad Dermatol. 56(5):748–752
[3] Mollet I, Ongenae K, Naeyaert JM. Origin, clinical presentation, and diagnosis of hypomelanotic skin disorders. Dermatol Clin. 25(3):363–371, ix
[4] Lee EH, Nehal KS, Disa JJ. Benign and premalignant skin lesions. Plast Reconstr Surg. 125(5):188e–198e
[5] Cribier B, Scrivener Y, Grosshans E. Tumors arising in nevus sebaceus: a study of 596 cases. J Am Acad Dermatol. 42(2, Pt 1):263–268
[6] Murphy ME, Boyer JD, Stashower ME, Zitelli JA. The surgical management of Spitz nevi. Dermatol Surg. 28(11):1065–1069, discussion 1069
[7] Bittencourt FV, Marghoob AA, Kopf AW, Koenig KL, Bart RS. Large congenital melanocytic nevi and the risk for development of malignant melanoma and neurocutaneous melanocytosis. Pediatrics. 106(4):736–741
[8] Arneja JS, Gosain AK. Giant congenital melanocytic nevi of the trunk and an algorithm for treatment. J Craniofac Surg. 16(5):886–893
[9] Gonzalez J, Palangio M, Fialkoff CN, Schwartz J, Bisaccia E. Giant congenital melanocytic nevus with a large ulceration at birth: a 5-year follow-up. J Am Acad Dermatol. 49(4):752–754
[10] Lawrence CM. Treatment options for giant congenital naevi. Clin Exp Dermatol. 25(1):7–11
[11] Kaplan EN. The risk of malignancy in large congenital nevi. Plast Reconstr Surg. 53(4):421–428
[12] Arneja JS, Gosain AK. Giant congenital melanocytic nevi. Plast Reconstr Surg. 120(2):26e–40e
[13] Agero AL, Benvenuto-Andrade C, Dusza SW, Halpern AC, Marghoob AA. Asymptomatic neurocutaneous melanocytosis in patients with large congenital melanocytic nevi: a study of cases from an Internet-based registry. J Am Acad Dermatol. 53(6):959–965
[14] Pearson GD, Goodman M, Sadove AM. Congenital nevus: the Indiana University's approach to treatment. J Craniofac Surg. 16(5):915–920
[15] Mulliken JB, Glowacki J. Hemangiomas and vascular malformations in infants and children: a classification based on endothelial characteristics. Plast Reconstr Surg. 69(3):412–422
[16] Jackson IT, Carreño R, Potparic Z, Hussain K. Hemangiomas, vascular malformations, and lymphovenous malformations: classification and methods of treatment. Plast Reconstr Surg. 91(7):1216–1230
[17] Fishman SJ, Mulliken JB. Vascular anomalies. A primer for pediatricians. Pediatr Clin North Am. 45(6):1455–1477
[18] Kum JJ, Khan ZA. Mechanisms of propranolol action in infantile hemangioma. Dermatoendocrinol. 6(1):e979699
[19] Marler JJ, Mulliken JB. Current management of hemangiomas and vascular malformations. Clin Plast Surg. 32(1):99–116, ix
[20] Berenguer B, Burrows PE, Zurakowski D, Mulliken JB. Sclerotherapy of craniofacial venous malformations: complications and results. Plast Reconstr Surg. 104(1):1–11, discussion 12–15
[21] Mulliken JB, Fishman SJ, Burrows PE. Vascular anomalies. Curr Probl Surg. 37 (8):517–584
[22] Arneja JS, Gosain AK. Vascular malformations. Plast Reconstr Surg. 121(4): 195e–206e

Part IV

Craniofacial

IV

17 Reconstruction of the Scalp and Forehead

Edward A. Luce

Abstract
This chapter thoroughly discusses the anatomy and various defects of the scalp and forehead that require reconstruction and introduces an algorithmic approach to the matter. Emphasis is given the etiology, diagnosis, and treatment of the three usual sources of scalp defects: congenital, posttraumatic, and oncologic. Substantial differences between scalp and forehead defects are identified, and treatment proper to the forehead is examined. Numerous photographs illustrate the irregularities and treatments discussed.

Keywords: here-there operative planning, subgaleal plane, asplasia cutis, galeal scoring

17.1 Goals and Objectives

- Understand the relevant anatomy of the scalp and forehead in regard to reconstruction.
- Review the various etiologies of scalp and forehead defects requiring reconstruction.
- Gain an appreciation and algorithmic approach to reconstruction of defects of the scalp and forehead.

17.2 Scalp

17.2.1 Patient Presentation

Anatomy

The pertinent anatomy of the scalp that has clinical relevance can be divided into the specific regions as well as the cross-sectional components. The cross-section anatomy is summed in the acronym SCALP (Skin, Connective Tissue, Aponeurosis, Loose Areolar, Periosteum)—the skin containing hair follicles, sebaceous and sweat glands, a thin layer of subcutaneous fat, the galeal aponeurosis, a zone of subgaleal loose areolar tissue, and lastly, periosteum or pericranium. Skin, in contrast to the subgaleal space, is tightly adherent to the underlying galea. The galea has an interesting teleological history since the aponeurosis may represent a vestige of the panniculus carnosus, a layer of immediate subdermal striated muscle seen in nonprimate mammals that enables twitching of isolated areas of skin. In that vein, the galea is a fusion sheet between the frontalis of the forehead and the occipitalis posteriorly, two other structures that represent vestigial remnants of the panniculus carnosus. Laterally, the galea is contiguous with the temporoparietal fascia.

The tightly adhered skin to the underlying galea and the lack of laxity of the skin envelope to the supporting skeleton, the skull, implies that primary closure of soft-tissue defects can be challenging. The looseness of the subgaleal areolar layer has clinical implications: undermining is most easily accomplished in this plane for the creation of scalp flaps or to facilitate closure, and avulsions occur within this plane. Also, any hematoma or abscess in this plane is easily extended to more remote areas of the scalp (▶ Fig. 17.1).

Regionally, the scalp can be divided into the same areas of the skull: temporal, parietal, and occipital. The modified anatomy of the temporal scalp region is of interest because of the surgical corollaries. The additional presence of the temporalis muscle and the investing fascia, the deep and superficial layers adds a level of complexity. Approaches to the zygomatic arch from the bicoronal route dictates a knowledge of this anatomy to avoid injury to the temporal branch of the frontal nerve. Division of the superficial layer of the deep temporal fascia at a point approximately 2 cm superior to the arch and a traverse through the superficial temporal fat pad will provide access to the arch, deep to the plane of the temporal branch that innervates the frontalis muscle.

The scalp is richly vascularized, setting the table for design of soft-tissue flaps of generous length and width proportions if oriented properly. The principal arterial supply is the supraorbital arteries anteriorly, the superficial temporal arteries laterally, the occipital, and to a lesser degree, the posterior auricular arteries posterior. The paired supraorbital nerves of the ophthalmic division of cranial nerve V provides sensation to the forehead and anterior one-half of the scalp. The greater occipital nerves, branches of C_2, are the sensory innervation of the posterior scalp. The temporal region is innervated by the auriculotemporal nerve, a branch of the mandibular division of V.

17.2.2 Etiology of Scalp Defects

The causation of scalp defects falls into one of three of the usual broad group: congenital, posttraumatic, and oncologic.

17.2.3 Congenital Defects

The principal presentation of scalp defects of congenital etiology is aplasia cutis congenital.[1,2] Other causes such as giant nevus and sebaceous nevus are discussed under the oncologic section. Aplasia cutis congenita, a rare condition (1 in 10,000 births), is absence of skin, subcutaneous tissue, and in approximately 25% of the cases, skull and dura as well. An autosomal dominant with variable penetrance inheritance pattern has been described, although many, if not most, cases appear sporadic in nature. Treatment and the urgency of same hinges on size and severity (see "Treatment" section).[3]

Another set of congenital soft-tissue entities is melanocytic and sebaceous nevi. Both are of a treatment concern because of the potential of malignant conversion. Estimations vary but melanoma in giant congenital melanocytic nevi, GCMN, and basal cell carcinoma within sebaceous nevi has been described (▶ Fig. 17.2). The nature of the pathology will dictate the reconstructive approach to oncologic defects. Certainly, in instances in GCMN, the parents will have both aesthetic and cancer conversion concerns.

17.2.4 Traumatic

Although the possible causes of posttraumatic scalp defects are protean, the vast majority of cases are secondary to avulsive loss, direct contusion and loss, or burn injury, of which a

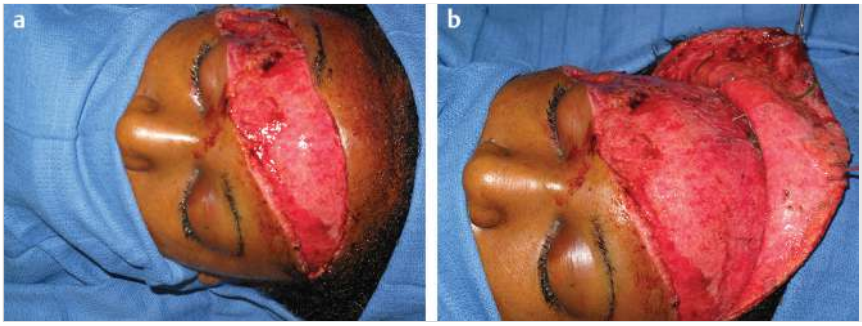


Fig. 17.1 (a,b) Avulsive wound of the forehead and scalp at the subgaleal level in the loose areolar plane.

Fig. 17.2 (a,b) Basal cell within a sebaceous nevus of the scalp in an adult.

significant portion are from electrical burns. Avulsive scalp injuries, particularly when total or subtotal, occur either at the level of pericranium for the reasons cited in the "Anatomy" subsection, but frequently and unfortunately result in loss of pericranium or periosteum as well with significant treatment implications.

Similarly, although flame burns may be full thickness and yet spare subcutaneous tissue, electrical burns or injury, particularly high voltage, may destroy not only all layers of the scalp but perhaps also may involve cranial bone, again with a different set of treatment implications (▶ Fig. 17.3).

17.2.5 Oncologic

The most frequent scalp reconstructive setting for most surgeons will be oncologic. Mentioned earlier were the benign conditions of congenital melanocytic nevus and sebaceous nevus. Any skin tumor, benign or malignant, drawn from the lengthy list of benign and malignant skin and soft-tissue neoplasms, can occur on the scalp. In addition, the treatment of intracranial neoplasms may have scalp reconstructive connotations. The occurrence of postcraniotomy scalp necrosis and/or bone flap infection may require intervention by the reconstructive surgeon. In any

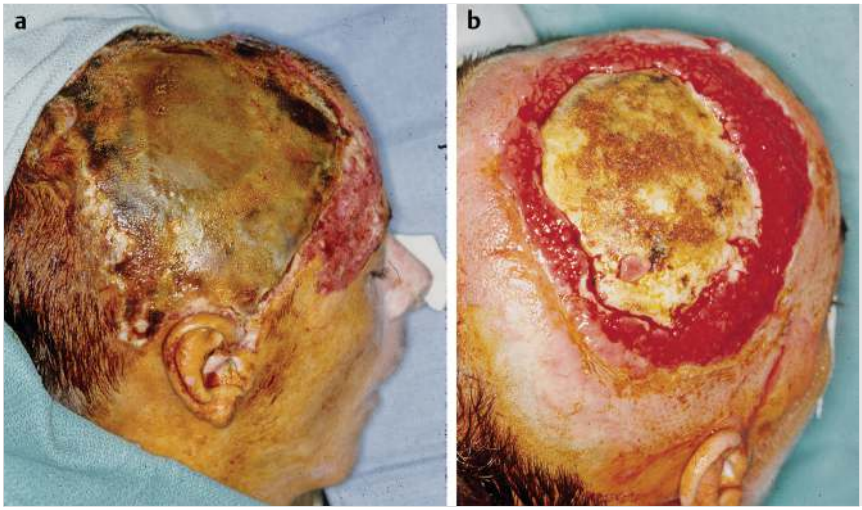


Fig. 17.3 (a, b) Full-thickness electrical burn of scalp and skull.

Fig. 17.4 Extensive congenital aplasia cutis of scalp.

oncologic setting, reconstructive needs may be rendered more complex by the presence of radiation damage from prior radiotherapy, prior incisions and scars, and, of course, the extent of the tumor.

17.3 Treatment

The assessment in preparation for reconstruction of any defect, present or planned, requires adherence to a logic framework to ensure as much as feasible a successful result. One such framework is the Here–There concept[4] that elicits analysis of the defect, anatomically and functionally ("Here"), the forces responsible and the desired objective ("There"), and finally the treatment plan to arrive at the objective. Implicit in each of the treatment scenarios described below is the use of this framework.

17.3.1 Here–There Operative Planning

1. Where is Here (What are the components, functional and structural, of the defect?)
2. How did we get There? (What were the forces, traumatic, oncologic, congenital responsible for the defect?)
3. Where is There? (What are the reconstructive needs?)
4. So do we get There? (Treatment or operative plan including steps and sequences.)
5. How do we want to get There? (A mutual decision-making process of patient and surgeon.)

17.3.2 Congenital

As outlined earlier, management of aplasia cutis congenita pivots on the magnitude and severity of the defect from simple to complex. The simplest of presentations, a defect of 2 to 4 cm^2 skin can only be managed nonoperatively with dressings. Larger defects (▶ Fig. 17.4) require a more aggressive approach, particularly in the absence of bone and the presence of exposed dura since the described complications of hemorrhage from exposed intradural sinuses as the sagittal or large dural veins can result in a fatal outcome. In the absence of coverage, the thin membrane becomes desiccated and an eschar forms, the debridement of which can result in massive bleeding. Contamination of the cerebrospinal fluid can result in meningitis and intracerebral infection. Coverage with split-thickness autografts, with or without a preliminary allograft, does protect the underlying dura and brain. The neonate has the osteogenic capability to generate new bone to heal the osseous defect. The negative aspects of the use of skin grafts include the difficulty and hazards of dissection and excision in preparation for eventual cranioplasty if needed. In addition, some concerns exist that the contractile nature of the grafts does not permit normal dural and brain growth.[3]

Although described earlier, the use of local flaps would seem fraught with hazard. In addition to the concerns about inevitable blood loss and operating time on a neonate without good temperature regulation and physiologic homeostasis of an older infant, an abnormal vascularity of the intact scalp may exist, the result of an in utero epithelialization of perhaps an even larger defect.

An intriguing solution to this treatment dilemma is the use of a dermal substitute. The placement of Integra once incorporated in 2 to 3 weeks, followed by removal of the silicone layer

and placement of a thin split-thickness skin graft has been described in a case report and has the potential to obviate the shortcomings of skin graft alone.[5] Perhaps, a full-thickness autograft could accomplish the same objective with a single stage but with a considerable size donor site.

17.3.3 Traumatic

Again, the analysis begins with the nature of the defect. Intact periosteum permits simple wound closure with split-thickness skin graft (STSG) to set the stage perhaps for definitive reconstruction with tissue expansion. The absence of periosteum implies the usual necessity of provision of full-thickness coverage with vascularized tissue, either pedicled or by microvascular free flaps. An exception is the creation of a vascularized bed by decortication of the skull and exposure of the cancellous layer with delayed skin grafting after stimulation of a granulating surface, hastened by the use of negative wound pressure therapy (NWPT) (▶ Fig. 17.5).[6]

The long-term durability of split-thickness skin grafts placed on decorticated bone is of concern, since late ulceration and wound breakdown is a frequent sequela, perhaps because of the thin layer of dermis.

The employment of a dermal substitute in the reconstructive plan may, as in the management of the aplasia cutis congenita defect, provide a course between the two horns of the dilemma.[7] Both Integra, a dermal substitute composed of bovine collagen and ground substance derived from shark, and Alloderm, an acellular human dermal matrix, are the two most widely used dermal substitutes (▶ Fig. 17.6).

The incorporation of both requires vascularization and the use of NWPT appears to be of benefit in this process. The final wound closure still requires a split-thickness skin graft, albeit considerably thinner than without the use of a dermal substitute. In addition, infection beneath the dermal template can occur, an enhanced probability in the presence of contamination and certainly with potential dire consequences.

Fig. 17.5 (a–c) Full-thickness burn of scalp and skull managed with decortication, negative wound pressure therapy, and split-thickness skin graft.

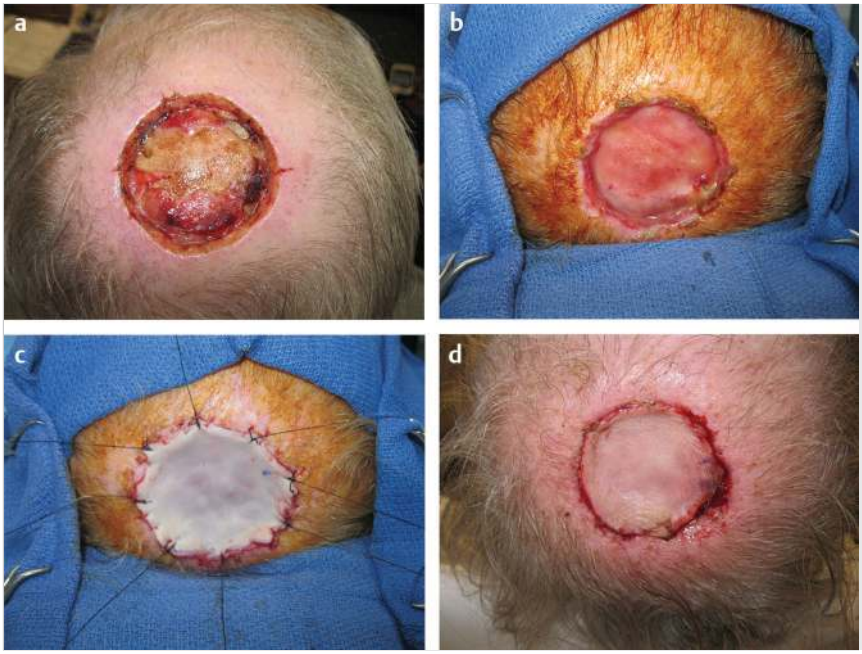


Fig. 17.6 (a-d) Full-thickness Moh's defect including periosteum managed with decortication, immediate application of Integra, and delayed split-thickness skin graft.

Fig. 17.7 (a,b) Full-thickness injury of scalp and skull. Decortication will not be possible because of lack of a viable diploe. Rotational scalp flap will provide wound closure and reossification of the devitalized skull.

The one set of circumstances that prescribes a discreetly different plan is an electrical injury of scalp and skull. In this instance, injury of scalp and skull, none of the above options of acellular dermis plus skin graft are available because of the full-thickness burn of the bony skull. In these circumstances, vascularized coverage, pedicled or free flaps, will be necessary. If performed in the early postinjury period before colonization of the bone, the evidence suggests that the devitalized bone can be regarded as an in situ graft with a perfect fit that will be reconstituted by the process of bone graft take or "creeping substitution" (▶ Fig. 17.7).[8]

The next rung on the reconstructive ladder is the use of local and regional flaps, discussed in the following section. The principles would still be applied in posttraumatic defects, if so indicated.

17.3.4 Oncologic

Extensive excisions of lesions as congenital melanocytic nevi are not amenable to direct primary closure and may be managed by serial tissue expansion (▶ Fig. 17.8). Patients most often present in early childhood, attended by parents with concerns about malignant transformation as well as the inherent aesthetic problem. The elective setting allows a cyclic approach of expansion, excision, and repeat expansion and excision.

As mentioned earlier, virtually any skin-soft tissue tumor that occurs elsewhere can present on the scalp. An additional dimension is the reconstructive deficit that involves the treatment of intracranial neoplasms, complicated by skin flap necrosis and/or infected bone flap. Both scenarios, resective defects created by excision of skin-soft tissue malignancies and the complications of treatment of intracerebral tumors, may be rendered more complex by the presence of previously irradiated tissue.

Defects of the scalp larger than 2.5 to 3.0 cm may be difficult to close without extensive undermining and the excessive tension can produce a widened, alopecic scar.[9] Local flaps such as a yin and yang design still permit primary closure of the donor site but larger, rotation-style flaps do not and a commitment to a skin-grafted donor site is unavoidable. Advancement, transposition, or rhomboid flaps perform poorly on the scalp because of the lack of skin laxity. An option for vertex defects, even of a considerable size, is the Orticochea flap tissue rearrangement approach, mobilization of the entire scalp by division into three flaps, similar to peeling a banana (▶ Fig. 17.9).[10]

Two paired flaps are each based on their respective superficial temporal artery, the posterior flap on the occipital arteries. Descriptions of the Orticochea flaps universally include scoring of the galea. Galeal scoring can be problematic from two aspects: one, the additional length obtained is modest and because the vasculature lies in the immediate superficial plane to the galeal aponeurosis, the blood supply can be damaged, creating a tradeoff of length versus ischemia.[9] If performed, the design is one of cross hatching to create the maximal relaxation (▶ Fig. 17.10).

In the author's experience, galeal scoring is bloody and of little advantage. If a scalp rotation flap is selected, a compulsive and carefully considered plan is mandatory. The elements include incorporation of a flap shrinkage factor (25%) into the defect dimensions, determination of the length and pivot point by simulation with gauze or suture, and orientation of the flap base to include a named blood supply (▶ Fig. 17.11).

The scalp, because of the convexity of the skull and absence of laxity, dictates the design of larger flaps than what the novice may anticipate. A large, unsightly dog ear will occur at the site of the "standing cone" that begs immediate release/excision. To do so will compromise the vascular supply and with time the dog ear will shrink remarkably.

The above scenarios assume the absence of prior irradiation. Prior irradiation to skin-soft tissue, particularly with larger radiotherapy portals, will usually imply regional pedicled or free flap coverage if the dimensions of the reconstructive defect, present or proposed, is of substantial size.

The presentation of a previously irradiated brain tumor patient to the reconstructive surgeon is most commonly as wound dehiscence/infection with or without infection of the underlying bone flap. Some clinical judgment is necessary in the estimation of the degree of radiation injury to the scalp. Review of the radiotherapy plan can be helpful, but the essence of the judgment is the vascularity or lack of same or proposed wound closure. The tautness of the scalp skin, the magnitude of radiation-induced alopecia, and the size and location of the defect all play a role. Wound dehiscence only without evidence of infection can be managed with wide undermining and advancement to close relatively small defects of 2 to 3 cm. More extensive defects that include debridement of necrotic tissue will require a rotational flap plus STSG. Management of the underlying cranial bone is a separate topic, but the treatment will be determined by the extent of infection. Localized osteomyelitis secondary to hardware alone can be managed with removal of

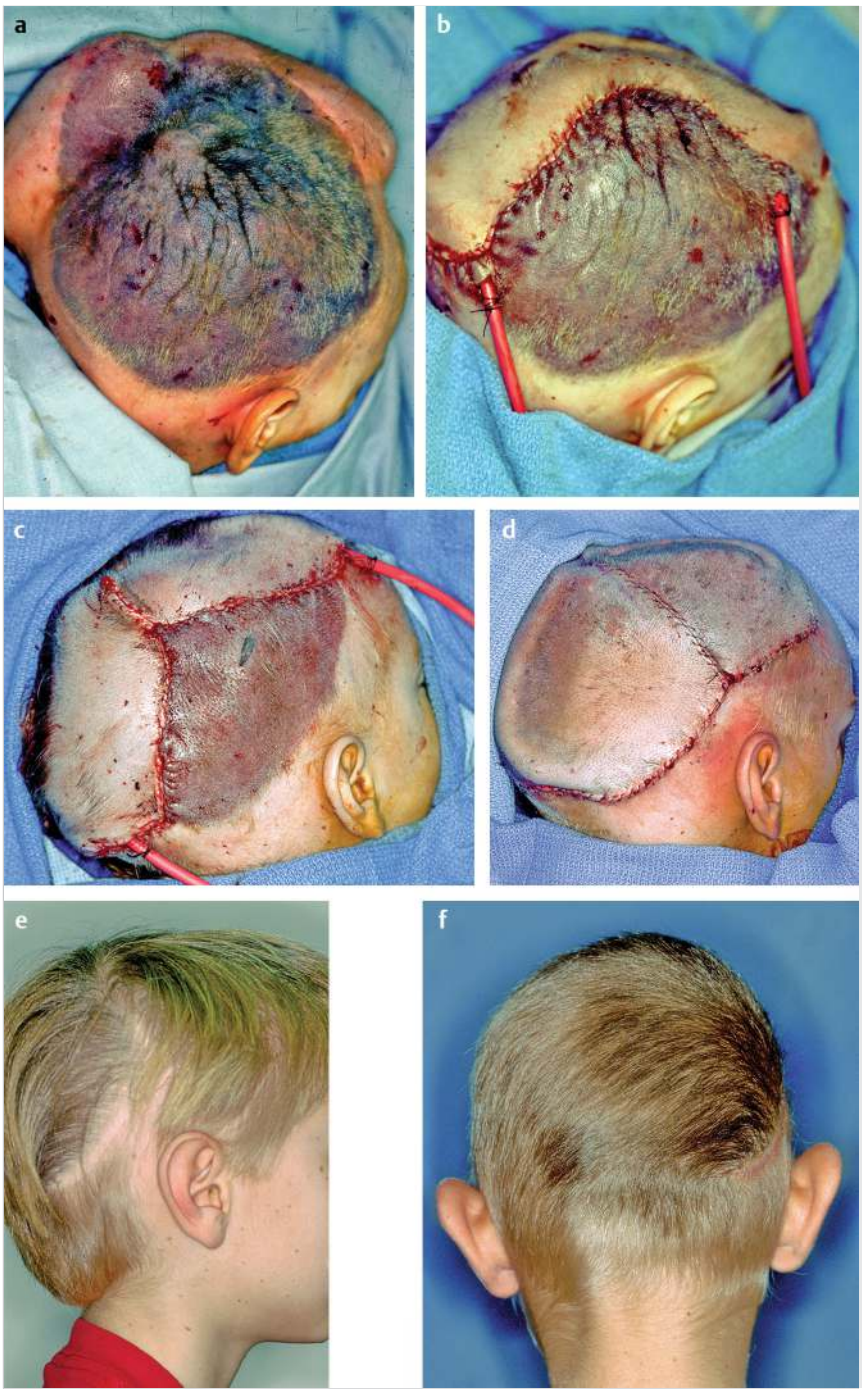


Fig. 17.8 (a–f) Large congenital nevus of the scalp managed with serial tissue expansion and repeated excisions.

the plates and screws and limited debridement, while more extensive infection requires removal of the bone flap and late secondary reconstruction.

Larger postoncologic defects, greater than 50% of the scalp, often cannot be satisfactorily addressed with the above methods (▶ Fig. 17.12).

If the scalp has been previously irradiated for skin-soft tissue malignancy, even considerably smaller defects will compel the use of regional or distant flap coverage, although the use of Integra and delayed skin graft has been described for smaller irradiated scalp defects.[11] The use of Integra has been described for the coverage of a total scalp loss in the pediatric patient.[12] The only scalp location for which the choice of a pedicled regional flap can be entertained is the temporal region, for example, after a temporal bone resection, reconstructed with an extended pectoralis major flap (▶ Fig. 17.13).

The inferior occipital scalp is amenable to a posterior trapezius perforator flap, perfused by the dorsal scapular artery. More often, the challenge is one of previously treated, often inadequately, because of positive margins and/or bone involvement with the employment of postoperative radiation in an adjunct setting.[13] The hostile environment created by

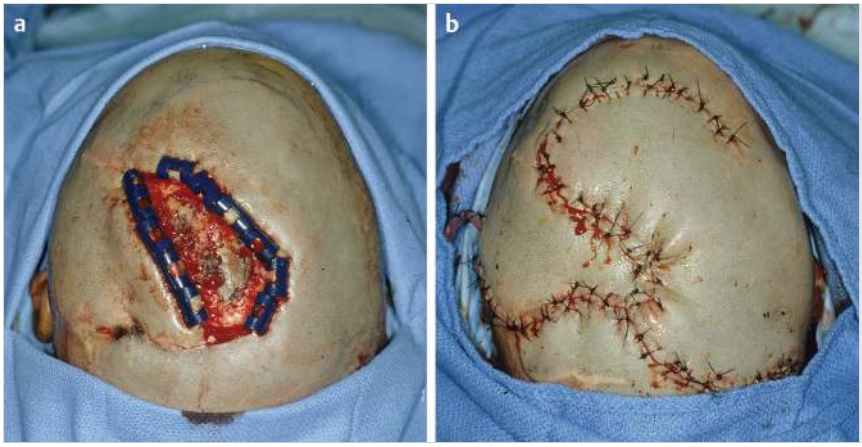


Fig. 17.9 (a,b) Exposed dura secondary to soft tissue and skull necrosis, managed with Orticochea flaps.

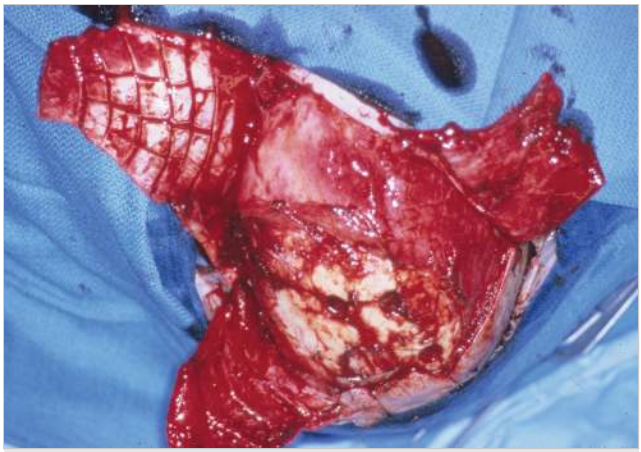


Fig. 17.10 Cross-hatching of galea in a "tic-tac-toe" design.

irradiation, recurrence-persistence of the neoplasm, and possibly contamination, if not of limited size and within the inferior or occipital region, all point to the indication for microvascular flap coverage.

Although an array of free flaps are available, essentially three (and possibly a fourth) have been most frequently selected and described. Large defects, for example, a subtotal scalp excision, covered with a latissimus muscle and skin graft, may be ideal from several perspectives: size, ability to tailor the muscle, and the value of well-vascularized muscle in a contaminated field. A skin island may be included if the size of the defect permits primary closure of the back and patient is not obese. The radial forearm as a fasciocutaneous flap provides, as does the latissimus, a pedicle of adequate length and is suitable for smaller, medium-size defects. The anterolateral thigh flap does provide a skin island, has a long and sizable pedicle, and offers, as does the radial forearm, the opportunity for a two-team approach, a harvest simultaneously with independent preparation of the recipient site. The fourth flap possibility is the scapular or parascapular, both fasciocutaneous flaps in nature. Similar to the latissimus, a patient position change may be necessary.

The most readily accessible recipient vessels are the superficial temporal artery and vein. At times, the vessels, particularly the vein, may be friable and difficult to use. The next echelon is within the neck, access to the superior thyroid or facial vessels. More extreme situations may require the use of vein grafts to access undamaged, nonradiated recipient vessels (▶ Fig. 17.14).

17.4 Forehead

17.4.1 Patient Presentation

Anatomy

The cross-sectional anatomy of the forehead is similar to the scalp but the topography differs substantially from the featureless expanse of the latter. Distinct boundaries define the forehead as an aesthetic unit. Displacement of these boundaries create visual distortion of the aesthetics of the forehead. Inferiorly, the paired eyebrows determine the inferior limit and dictate symmetry, since even minor alterations in brow position are readily perceptible to the casual observer (▶ Fig. 17.15).

The frontal and temporal hairlines determine the superior and lateral limits of the forehead unit and both, dependent on hair pattern, are relatively distinct. The forehead can be further divided into central and lateral units, the central defined laterally by the superior temporal line. The convexity of the central forehead is a reflection of the frontal bony skull, terminating laterally at the aforementioned superior temporal ridge, the bony landmark at the point of fusion of the soft tissue layers of the forehead to the periosteum. The supraorbital ridge is the inferior bony unit. Superiorly, no bony landmarks exist to differentiate forehead from scalp. In that differentiation is the frontal–temporal hairline. In the absence of the hairline, the junction of frontalis of the forehead to the galea of the scalp is a superior boundary, albeit arbitrary.

The cross-sectional anatomy does mirror the scalp consisting of skin, a thin layer of fat, the frontalis muscles, a loose areolar layer, and periosteum. Subspecialization of the layer occurs at the glabella and medial supraorbital ridge in the form of the depressor supercilii, corrugator, and the supraorbital orbicularis muscles—all of which have implications in aesthetic surgery of this region. The most accurate description of the relationship of the galea would include the division of the galea at the superior aspect of the frontalis into a deep and superficial split that in turn envelops the muscle. About 2 cm superior to the superior orbital rim, the deep portion, until this point only loosely

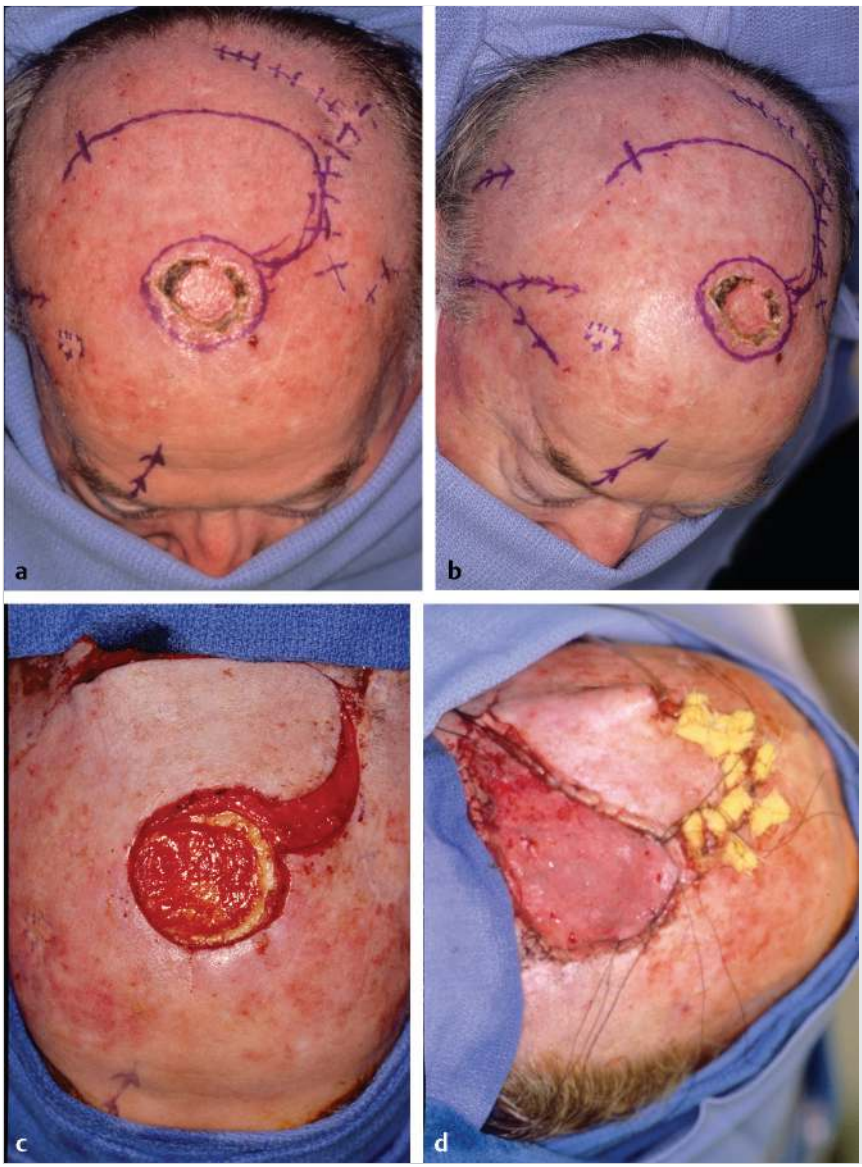


Fig. 17.11 (a–d) Patient with a central anterior scalp defect, prior history of multiple excisions and scalp flaps as well as irradiation, managed with reexcision of soft tissue, outer cortex, and rotational scalp flap.

attached to the underlying periosteum becomes fused to that structure, also a key aspect when elevating a bicoronal flap.

The sensory innervation of the forehead arises principally from the supraorbital and supratrochlear nerves emanating from the supraorbital and the lateral orbital roof, respectably. Both are branches of the ophthalmic division of cranial nerve V, the trigeminal. Sensation of the most lateral portion of the forehead is supplied by the zygomatico-temporal nerve through the small foramen in the most anterior temporal bone and is a branch of the maxillary division. The supraorbital nerve also supplies sensation to the frontoparietal scalp in addition to the forehead by division into a superficial and deep branch.[14] The superficial division courses over the surface of the frontalis, the deep branch passes beneath frontalis to provide sensation to the scalp. Motor innervation of the frontalis is via the temporal, frontal, branch of cranial nerve VII, the facial. The nerve enters the deep aspect of the muscle laterally until that point. After exit from the superior aspect of the parotid, the nerve courses over the zygomatic arch as branch or branches contained within the superficial portion of the deep temporal fascia, a key anatomical fact when elevating bicoronal flap. In addition to the depth dimension of the location of the nerve, the course to the frontalis can also be defined topographically.

Vascular supply to the forehead parallels that of the sensory innervation, namely, the supraorbital and supratrochlear arteries to their respective foramina centrally or medially in a transverse anterior branch of the superficial temporal artery laterally. The venous drainage parallels the arterial inflow.

Etiology

Similar to the scalp, conditions that will require reconstruction are congenital, traumatic, and oncologic in causative nature. The two congenital conditions or etiology that may require reconstruction are congenital melanocytic nevus and hemangioma. Congenital melanocytic nevus, particularly the giant variety, similar to those of the scalp, may mandate surgical management both from oncologic and esthetic aspect

Fig. 17.12 (a–d) Multiple satellite lesions from melanoma, mandating a subtotal excision of the scalp.

(▶ Fig. 17.16). Hemangiomas of the noninvoluting category often, perhaps in a majority of the instances, involve adjacent aesthetic units, the orbit, in addition to forehead (▶ Fig. 17.17).

Traumatic deformities occur via similar mechanisms as described for the scalp. Lacerations and avulsions are most frequent and are often related to blunt-force injury. Diagnosis of underlying fracture and possible intracranial as well as cervical injuries is necessary. Involvement with surrounding structures that can affect treatment and reconstruction must be fully assessed. Benign tumors of the subcutaneous tissue can be approached through a bicoronal approach to avoid direct scars on the forehead (▶ Fig. 17.18).

Any skin-soft tissue tumor that occurs elsewhere can present in the soft tissues of the forehead. Margins are dictated to some degree by particular facial aesthetic unit—margins are inferentially narrow in the midface: periorbita, nose, as well as the ears and less so in the cheek and forehead. The same caveats with respect to prior radiation as discussed in the scalp should be incorporated into the reconstructive plan and apply also to the forehead. Challenges posed by reconstruction of excisional defects are similar to those of the scalp with an important difference, the necessity to respect the topographical landmarks that define the forehead and include brow position and hairline.

In the absence of prior irradiated tissue, the principles of reconstruction of oncologic and congenital defects are similar and will be discussed conjointly.

17.5 Treatment

Reconstruction of forehead defects is almost entirely accomplished with local flaps and/or skin grafts both full and split thickness. As discussed in section “Anatomy,” although the forehead is considered a single aesthetic unit, the central portion from superior bony ridge to superior bony ridge is more demanding than the temporal forehead, at least in the transverse or horizontal vector. Closure of central vertical defects such as the donor site of the median forehead flap are more readily accomplished. Horizontal defects can be closed primarily if particular attention is paid to the maintenance of brow position. Excisional defects as large as 3 cm, even in the suprabrow region, can be closed by wide undermining superiorly. The advanced superior flap as well as the nonundermined inferior brow-bearing flap must be anchored to the underlying

Fig. 17.13 (a–e) Advanced cancer of the ear treated with neck dissection and lateral temporal bone resection. Closure obtained by a pedicled pectoralis major flap.

periosteum with a row of permanent sutures (▶ Fig. 17.19). Smaller defects can be closed with a transverse advancement flaps (▶ Fig. 17.20). Small rotational flaps are possible if designed to place the skin-graft in a location that can be concealed with hair (▶ Fig. 17.21).

Large defects, 4 cm or greater, may be closed with full-thickness skin grafts harvested from the postauricular area or supraclavicular fossa of the lower cervical region. If the defect consists of forehead skin and frontalis, a contour and defect may be noticeable even with full-thickness skin grafts harvested from the head and neck "blush" area. Skin grafts obtained from below the head and neck will be more yellow-brown in hue and match poorly with forehead skin (▶ Fig. 17.22). One modification to address potential contour defects is the use of a galeofrontalis flap and full-thickness skin graft (▶ Fig. 17.23).

In the absence of periosteum, larger defects can be addressed with a two-step approach of placement of Integra, followed 3 weeks later by removal of the silicone layer and application of a split-thickness or full-thickness skin graft harvested from the blush area. The combination of artificial and autogenous dermis can also address contour concerns (▶ Fig. 17.24).

Defects of the lateral forehead can be reconstructed by a combination of advancement of a cheek flap inferiorly and judicious used of advancement of the temporal frontal hairline. Large defects can be closed with full-thickness skin grafts since 7 to 8 cm can be harvested from bilateral lower cervical donor sites and contour defects are not as obvious in the lateral compared to the central forehead (▶ Fig. 17.25). Large defects associated with cranial base or frontal bone resections for advanced cancers most commonly require free tissue transfer (▶ Fig. 17.26).

17.6 Scalp and Forehead

17.6.1 Postoperative Care

The care of these patients is largely dependent on the age, comorbidities, and the type of condition reconstructed, as well as the type of reconstruction done. The inpatient or ambulatory setting is predicated on the size of the defect and the magnitude of reconstruction. Those for whom skin grafting is done will generally require a bolster dressing over the graft for about 5 days and then dressings thereafter to prevent desiccation, such as antibiotic ointment or petrolatum impregnated dressings. Antibiotic therapy is needed for reconstruction involving osteomyelitis. Duration and type of treatment is directed by culture and input from infectious disease colleagues.

Fig. 17.14 (a–h) Recurrent fibrosarcoma of the scalp resected including full-thickness skull. A mirror image bone template used for a split calvarial graft for closure of the bony defect and serratus anterior muscle flap and skin graft. The recipient vessels were accessed in the neck via vein grafts.

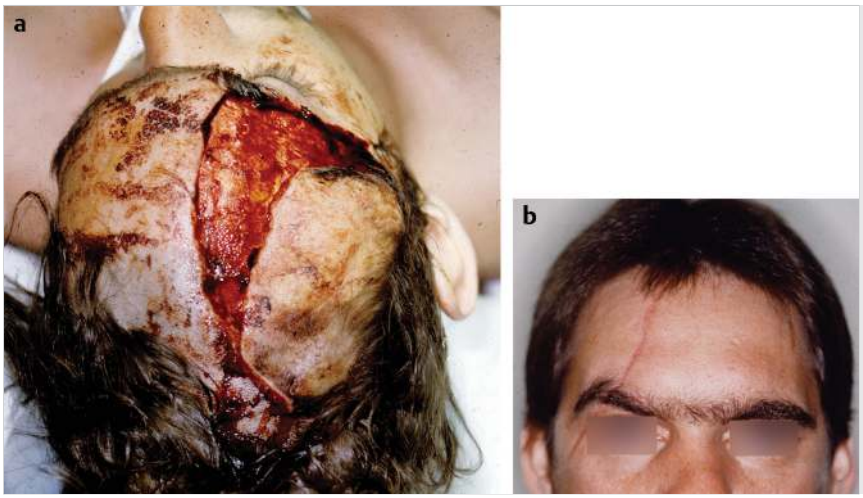


Fig. 17.15 (a,b) Malalignment of the brow.

Fig. 17.16 (a–c) Nevus of Ota of forehead, brow, and medial canthal region, excised and closed with full-thickness skin grafts, harvested from upper chest.

Fig. 17.17 (a,b) Involuting hemangioma of the forehead.

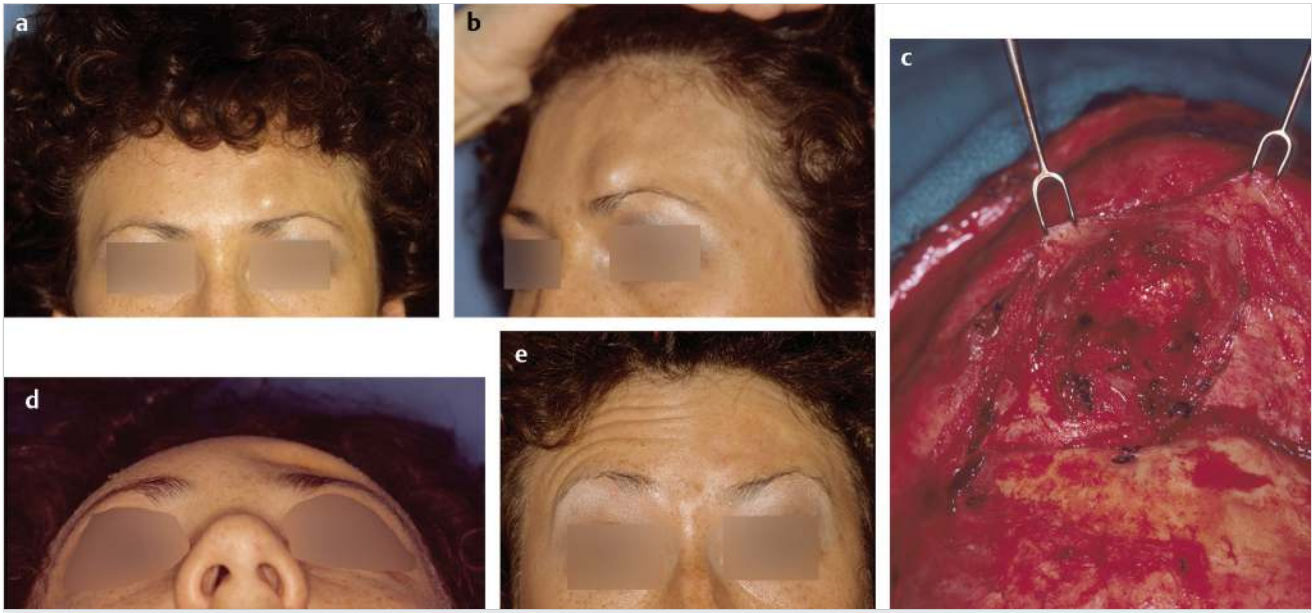


Fig. 17.18 (a–e) Recurrent A-V malformation of the supraorbital foramen, resected via a bicoronal approach.

Fig. 17.19 Malignant melanoma of intermediate thickness of the left brow region. Closure by undermining is possible but mandates that the inferior brow-bearing flap be tacked to the underlying bone and periosteum.

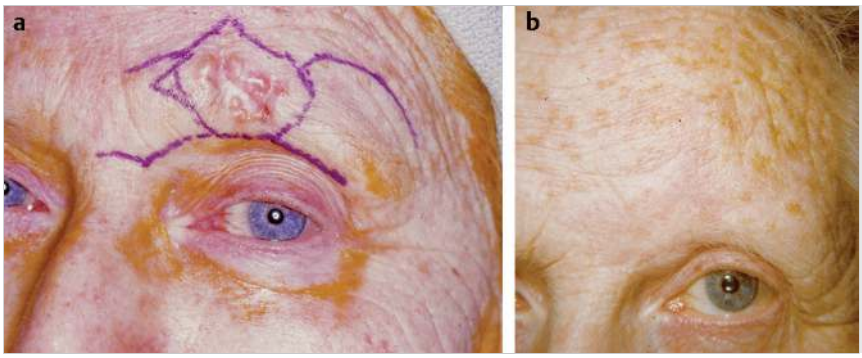


Fig. 17.20 (a,b) Closure of basal cell excision by transverse advancement flaps.

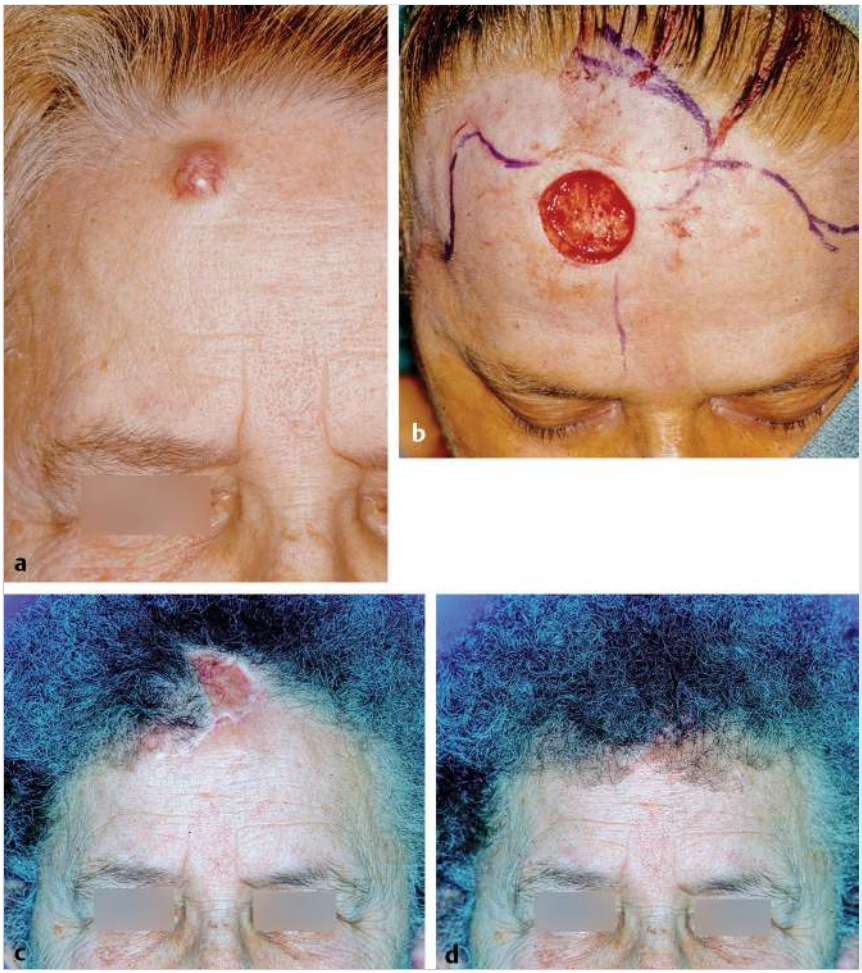


Fig. 17.21 (a–d) Excision of basal cell at the frontal hairline, closed with a small rotational flap with placement of the skin-graft donor site in a location that can be concealed with scalp hair.

Fig. 17.22 Donor site of forehead flap closed with a thigh graft.

Fig. 17.23 (a–f) An irradiated skin-soft tissue defect of the temporal forehead closed with rotation galeofrontalis flap and skin graft.

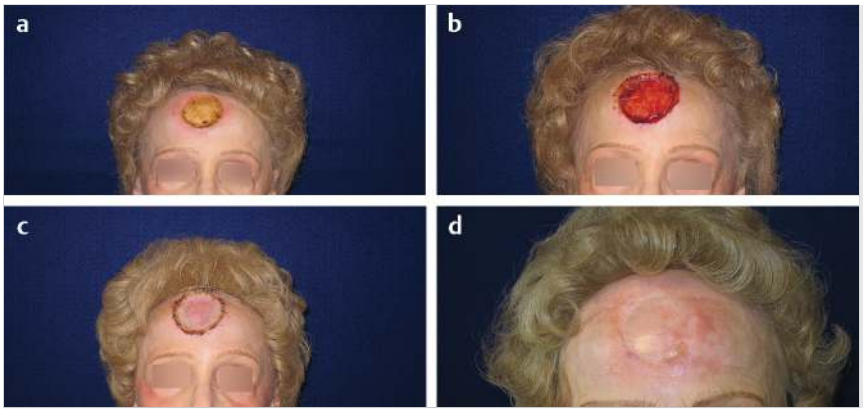


Fig. 17.24 (a–d) Moh's defect of the central forehead; placement of Integra on bleeding decorticated skull followed by delayed split-thickness skin graft.

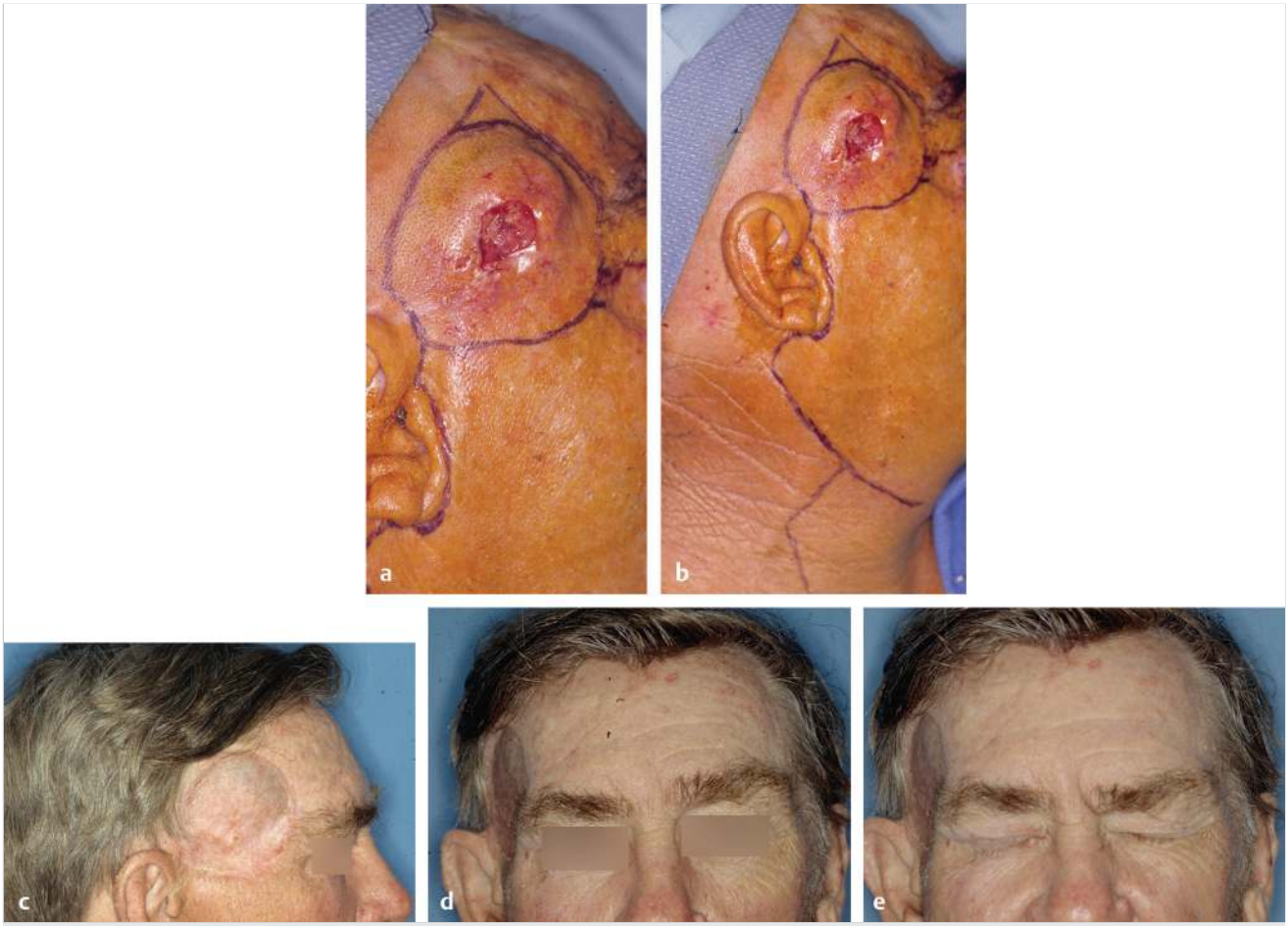


Fig. 17.25 (a–e) Large squamous cell carcinoma of the temporal area. Resection in conjunction with a superficial parotidectomy and neck dissection. Closure by rotation of temporalis muscle and skin graft obtained from the blush area. Normal facial function.

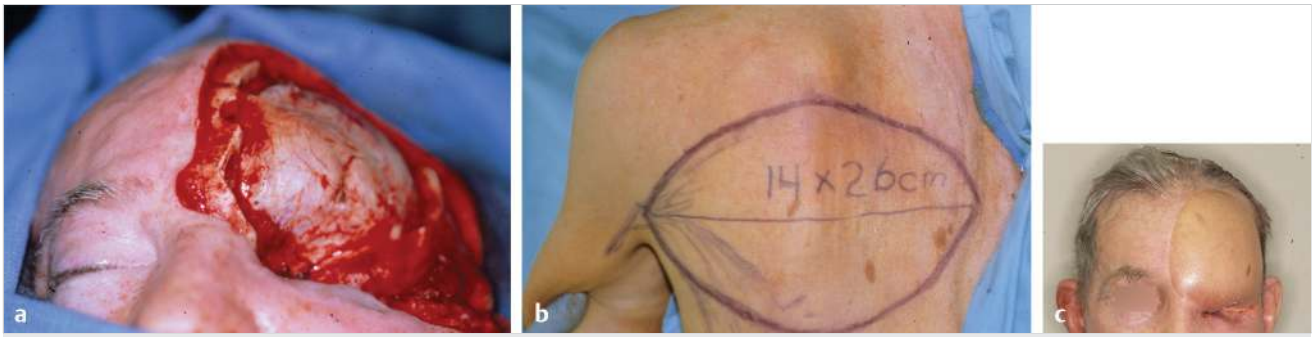


Fig. 17.26 (a–c) Cranial base resection and scapular flap.

17.6.2 Outcomes

Evidence-based data regarding this heterogeneous group of patients is cumbersome to decipher. Complications occur similar to any skin graft, local flap, regional flap, or free flap. These include partial or complete flap necrosis, as well partial or complete loss of skin graft. In properly selected and prepared patients, such complications should occur in a very small minority. Scalp defects will often be associated with alopecia as a result of scarring or skin grafting. As such, all attempts should be made to design flaps such that scars and skin grafts are hidden as much as possible in and around the scalp. Similarly, the aesthetic units and boundaries of the forehead should be respected. Secondary procedures such as serial excision and tissue expansion may be necessary for correction of unsightly alopecia or scarring. Oncologic patients need to be observed carefully for recurrence, either locally, regionally, or distally, if malignant disease is being treated. Overall, when planned carefully, complications are low and patient satisfaction is high.

17.7 Review Questions

17.7.1 True or False

1. The subgaleal plane in the scalp is heavily vascularized and should be avoided.

17.7.2 Choose the Best Answer

2. Aplasia cutis
   a) Is a very common condition of the scalp in newborns.
   b) Is a congenital condition characterized by the absence of skin and subcutaneous tissue, but can also include skull and dura.
   c) Develops within the first 3 months of life and gradually enlarges.
   d) Is another name for congenital nevus of the scalp.
3. Galeal scoring of scalp flaps
   a) Can be done with impunity.
   b) Has no bearing on the vascularity of the flap.
   c) Can be helpful by lengthening the flap.
   d) Is only needed if dura is exposed.
4. Which is true regarding forehead anatomy?
   a) Only two nerves provide innervation of the forehead.
   b) The supratrochlear nerves are variable in their location.
   c) Motor innervation of the frontalis is from the supraorbital nerve.
   d) Vascular supply to the forehead parallels the sensory innervation.
5. While planning closure of forehead defects,
   a) Vertically oriented primary closure should be avoided.
   b) Horizontally oriented primary closure should be done with consideration of alteration of brow position.
   c) Skin grafts are rarely indicated.
   d) Local flaps are limited to meager forehead blood supply.

17.7.3 Answers

1. False.
2. b. Is a congenital condition characterized by the absence of skin and subcutaneous tissue, but can also include skull and dura.
3. c. Can be helpful by lengthening the flap.
4. d. Vascular supply to the forehead parallels the sensory innervation.
5. b. Horizontally oriented primary closure should be done with consideration of alteration of brow position.

References

[1] Frieden IJ. Aplasia cutis congenita: a clinical review and proposal for classification. J Am Acad Dermatol. 14(4):646–660

[2] Silberstein E, Pagkalos VA, Landau D, et al. Aplasia cutis congenita: clinical management and a new classification system. Plast Reconstr Surg. 134(5): 766e–774e

[3] Bharti G, Groves L, David LR, Sanger C, Argenta LC. Aplasia cutis congenita: clinical management of a rare congenital anomaly. J Craniofac Surg. 22(1):159–165

[4] Luce EA. Analysis of complex deformity: the Here-There model. Clin Plast Surg. 22(1):1–8

[5] Smartt JM , Jr, Kim EM, Tobias AM, Yan AC, Kirschner RE. Aplasia cutis congenita with calvarial defects: a simplified management strategy using acellular dermal matrix. Plast Reconstr Surg. 121(4):1224–1229

[6] Marathe US, Sniezek JC. Use of the vacuum-assisted closure device in enhancing closure of a massive skull defect. Laryngoscope. 114(6):961–964

[7] Khan MA, Ali SN, Farid M, Pancholi M, Rayatt S, Yap LH. Use of dermal regeneration template (Integra) for reconstruction of full-thickness complex oncologic scalp defects. J Craniofac Surg. 21(3):905–909

[8] Luce EA, Hoopes JE. Electrical burn of the scalp and skull. Case report. Plast Reconstr Surg. 54(3):359–363

[9] Raposio E, Nordström RE, Santi PL. Undermining of the scalp: quantitative effects. Plast Reconstr Surg. 101(5):1218–1222

[10] Orticochea M. New three-flap reconstruction technique. Br J Plast Surg. 24 (2):184–188

[11] Gonyon DL , Jr, Zenn MR. Simple approach to the radiated scalp wound using INTEGRA skin substitute. Ann Plast Surg. 50(3):315–320

[12] Konofaos P, Kashyap A, Wallace RD. Total scalp reconstruction following a dog bite in a pediatric patient. J Craniofac Surg. 25(4):1362–1364

[13] Lutz BS, Wei FC, Chen HC, Lin CH, Wei CY. Reconstruction of scalp defects with free flaps in 30 cases. Br J Plast Surg. 51(3):186–190

[14] Knize DM. The Forehead and Temporal Fossa. Philadelphia, PA: Lippincott Williams & Wilkins; 2001:28

18 Nasal Reconstruction

Patrick J. Buchanan, William J. Campbell, and Bruce A. Mast

Abstract
This chapter assists in understanding the anatomy of normal noses and defects requiring reconstruction. A critical issue in nose reconstruction involves aesthetics, and the authors fully address the sensitive considerations of which surgeons should be aware. Treatment options covered include replacement of the bone/cartilage framework, reconstructing the nasal lining, and soft-tissue reconstruction (skin grafts, composite grafts, local flaps, forehead flap). Guidelines involving postoperative care and outcomes conclude the discussion.

Keywords: external skin, supporting bone and cartilage, mucoperichondrium lining, rhinoplasty, septoplasty

18.1 Goals and Objectives

- Understand the typical patient presentation for nasal reconstruction.
- Understand the subunit approach to nasal reconstruction.
- Clearly define the indications for the various reconstructive options of the nose.
- Know the evidence-based perioperative care to maximize patient safety and quality outcomes.

18.2 Patient Presentation

Anatomically, the nose consists of three central elements: external skin, a supporting middle layer of bone and cartilage, and a mucoperichondrium lining. Maintaining the central position of the face, the nose is often considered the most difficult facial feature to reconstruct well. Nasal defects are most commonly encountered after cancer resection or trauma, and reconstruction can vary from simple to complex. Depending on the patient's age, comorbidities, and aesthetic desire, a less complicated, quicker repair with minimal surgery or stages may suffice. These less complicated reconstructions include healing by secondary intention or suturing the nasal lining and skin together. Both of these techniques will inevitably lead to a permanent nasal deformity or distortion.[1,2]

The majority of patients want their nasal defect healed without distortion of their appearance. In these cases, the surgeon must be acutely aware of the previous surgical treatments for skin cancer, radiation, trauma, or other scars. These previous interventions may interfere with blood supply, impair healing, or preclude specific flap options.[3] Likewise, operative time, anesthetic requirements, hospitalization, and number of stages must be taken into account prior to choosing a reconstruction option.

18.2.1 Defining the Defect

The defect's size, depth, site, and surrounding skin condition greatly influence the reconstructive decision. A small, superficial defect is characterized as less than 1.5 cm in size with an uninjured cartilaginous framework. A large, deep defect is defined as greater than 1.5 cm in size and often requires reconstruction of the underlying cartilaginous framework and/or nasal lining.[1,2,4] However, in all cases, the missing tissue and framework must be replaced in the exact amount. If too little is replaced then this will distort adjacent landmarks and collapse underlying cartilage grafts. Excess replacement tissue will lead to airway obstruction.[1,2,4] The lateral ala and alar base consist of thick fibrous tissue providing support to alar shape. Full-thickness defects in this area will often need cartilage grafts to prevent tissue contraction, although no cartilage is part of the nascent anatomy.

Topographically, the nose is defined by its nasal subunits, including lateral nasal sidewall (2), alar lobule (2), soft triangle (2), the dorsum, tip, and the columella.[5] These subunits also need to be appreciated in the context of the subunits of the entire face (▶ Fig. 18.1). Scars that lie within the boundaries of subunits tend to heal in the least conspicuous manner. A superficial defect with a base of healthy, well-vascularized subcutaneous tissue is easily reconstructed with a skin graft. Skin graft reconstruction is best used when the defect is within the nasal dorsum, sidewall, alar rims, or columella, as these areas naturally have thin, adherent skin.[2] Local flaps are a better option when the defect is within the thicker skin of the nasal tip and ala.[6,7,8,9,10]

Overall, the process of defining the nasal defect and choosing a reconstructive approach should be thoughtful and comprehensive. The disease process causing the defect, past treatments, patient health, and a review of old photographs should be thoroughly evaluated. The aesthetic subunits need to be appreciated, both for reconstruction of the defect itself and potential use of flaps from the nose or face, in order to provide the best aesthetic outcomes (▶ Fig. 18.2).

18.3 Preparation for Surgery

For nasal reconstruction, the principles of regional subunit repair are applied.[5] If the defect encompasses greater than 50% of a subunit, then it is recommended that the entire subunit be excised and reconstructed (▶ Fig. 18.3). It is always the assumption that patients wish to restore their normal facial appearance. Therefore, it is necessary to use the contralateral, normal side of the nose as a guide. In cases of a bilateral nasal defect, use an old photograph. Exact templates are helpful to define replacement tissue dimensions and for marking landmark positions.[2]

In instances of severe or complex defects, a preliminary operation is beneficial. The preliminary procedure can help ensure that the margins of the defect are clear of cancer or necrotic debris. It also allows for an intraoperative evaluation under anesthesia and to reopen the airway, if needed.[2,4]

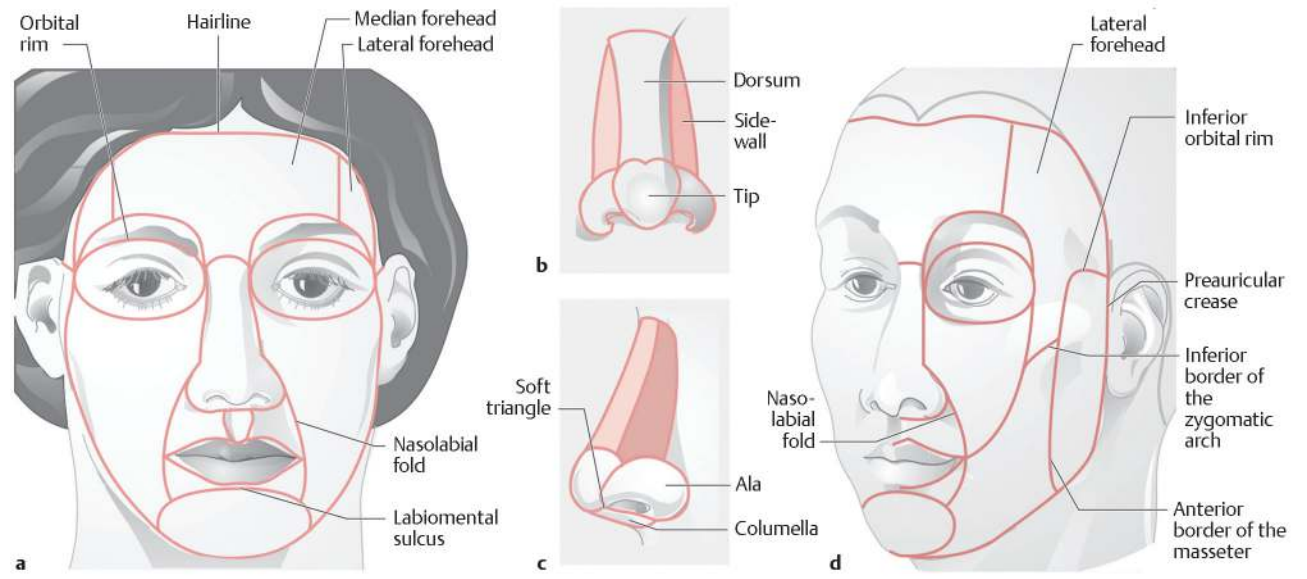


Fig. 18.1 (**a-d**) Aesthetic subunits of the nose and face. (From Weerda H. Reconstructive Facial Plastic Surgery 2e. New York, NY: Thieme; 2014:33.)

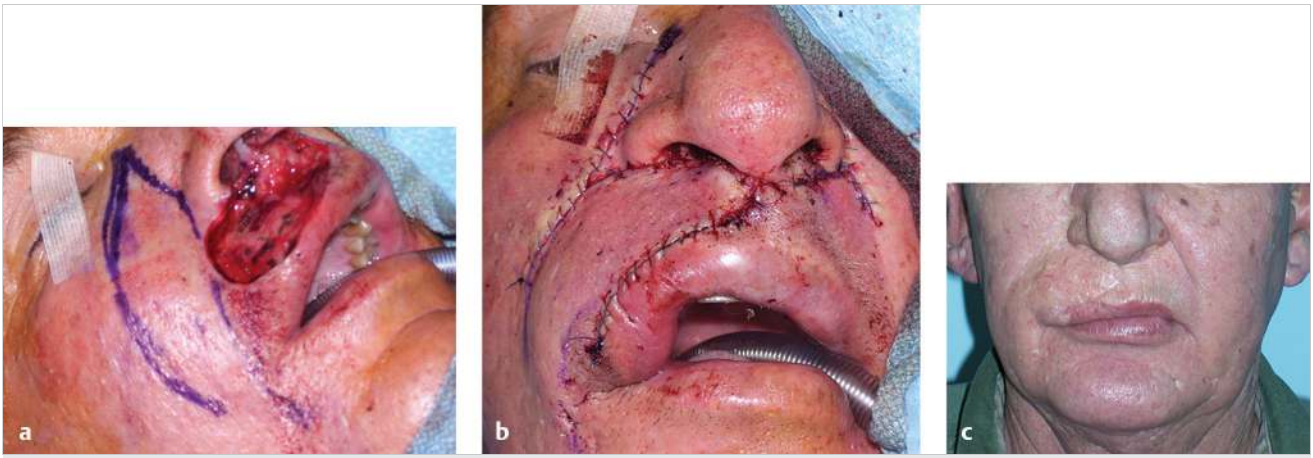


Fig. 18.2 Patient with a large oncologically induced defect of the upper lip in conjunction with columella base and nostril sill. **(a)** Reconstruction is planned with an inferiorly based nasolabial flap. **(b)** Flap harvest and inset was done following the principles of facial aesthetic subunits. **(c)** Inconspicuous scarring as a result of using the subunit principle.

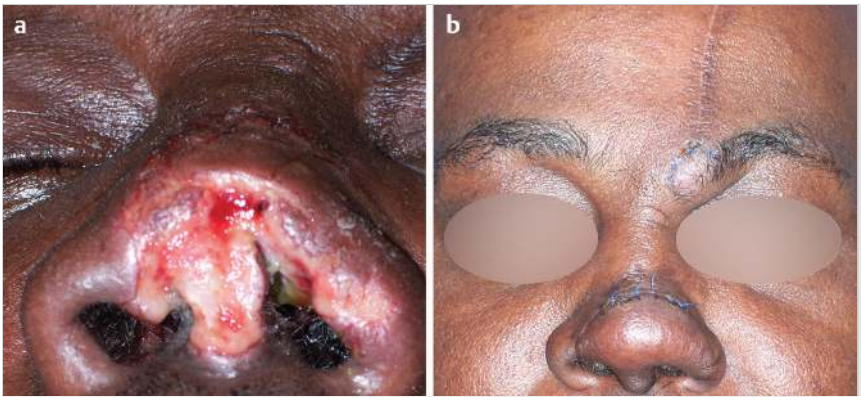


Fig. 18.3 Example of enlarging the nasal defect to encompass the entire subunit. **(a)** In this traumatic defect, the columella and soft triangle subunits were entirely missing, the tip subunit was partially missing. **(b)** The defect was enlarged using the subunit principle, as demonstrated by the inset of the forehead flap, as seen in this early postoperative result after division and inset of the forehead flap.

18.4 Treatment

18.4.1 Bone/Cartilage Foundation

Multiple options exist for the replacement of the bony and cartilaginous nasal framework. The cartilaginous options include septal cartilage, conchal cartilage, and costal cartilage. Bony options include cranial bone and other autogenous bone grafts.[11,12,13]

Septal cartilage provides a strong and straight construct but can be limited in quantity. Conchal cartilage is often useful for alar batten grafts and tip shaping, including replacement of lower lateral cartilage (▶ Fig. 18.4). It is weaker than septal cartilage and has an intrinsic curve. Costal cartilage is taken from the sixth through ninth ribs and provides a strong construct. However, this cartilaginous option does produce donor-site morbidity and has a tendency toward warping over time.

For nasal reconstruction requiring bone grafts, cranial bone can be harvested from the parietal area. These bone grafts are useful in reconstructing the bony nasal pyramid, but are prone to resorption and distortion. Other areas for bone graft harvest are the ribs and iliac crest. Likewise, these are subject to resorption and donor-site morbidity.

18.4.2 Nasal Lining

When reconstructing the nasal lining, there are several considerations. The first is to use a well-vascularized flap to provide internal support for needed cartilage or bone grafts. The second is to select a material thin enough to prevent airway obstruction. The flap must also not distort the structural framework of the nose by tension or contracture. Several options are available.[11,12,13]

Fig. 18.4 Same patient as in ▶ Fig. 18.2. Use of conchal cartilage graft in the nostril sill to prevent soft tissue contraction and medial retraction of the ala.

The nasal turnover flap has limited vascularity and is thick. The nasolabial flap, although with good vascularity, is bulky and may require multiple stages. The forehead turn-in flap has good vascularity but is also bulky and may require multiple stages. A septal mucoperichondrial flap is well vascularized and useful in reconstructing the nasal midvault and alar domes. This flap is a large, caudally based rectangle of mucosa or mucosa and perichondrium. A mucosal advancement flap is a bipedicled flap with limited availability but highly vascularized. This flap option is capable of supporting primary cartilage grafts. A septal door flap involves folding down the ipsilateral nasal mucosa and then "swinging" a septal door of contralateral mucosa with septal cartilage. Care must be taken to leave a sufficient amount of septum to prevent instability. The facial artery musculomucosal (FAMM) flap is superiorly based on the facial artery axial blood supply. To reconstruct nasal lining by free tissue transfer, the radial forearm is the most commonly used donor site. This method of nasal lining reconstruction is performed in multiple stages and requires aggressive thinning. To reduce the number of stages required, prelaminated flaps can be created. For prelaminated flaps, skin grafts or mucosal grafts can be placed on the undersurface of free flaps or forehead flaps to create the nasal lining prior to transfer. Cartilage grafts can be placed between the healed, vascularized, laminated layers of the transferred flap.

18.4.3 Soft-Tissue Reconstruction

Skin Grafting

Skin grafting to reconstruct a nasal defect is advantageous in that no new scars are added to the nasal surface. The locally available excess tissue does not limit this technique. Historically, skin grafts for nasal reconstruction are harvested from preauricular, postauricular, supraclavicular, or forehead donor sites.[1,2] The quality of the donor skin graft is compromised by the transient ischemia incurred prior to imbibition and inosculation. This transient ischemia leads to a pale, smooth, and atrophic appearance of healed skin grafts. The preauricular skin provides a better skin color match when healed, especially when correcting a defect within the nasal sidewall or columella. Postauricular skin grafts remain red in appearance. Supraclavicular skin grafts heal with a brown and shiny appearance. The forehead skin and underlying fibrofatty subcutaneous layer make this donor site thicker, stiffer, and more ideal for resurfacing nasal defects of the tip and ala.

First, a pattern of the nasal defect must be created prior to debridement to ensure a clean and vascular wound bed. If a pre- or postauricular skin graft is chosen, elevate the graft within the subcutaneous layer. A forehead skin graft should be elevated superficial to the frontalis muscle. With curved iris scissors, the skin graft is thinned to the thickness of the nasal defect. Next, the skin graft is transferred onto the prepared recipient site and inset using a single layer of sutures placed around the periphery. A bolster is then affixed on top of the

skin graft with tie-over sutures to prohibit lateral shearing or fluid accumulation underneath the graft. The bolster is best left in place for 7 days. Initially, the skin graft will appear white in color, and then gradually change from blue to pink.

Composite Grafts

A composite graft consists of more than one tissue type (▶ Fig. 18.5). Full-thickness defects of the tip, hemi-tip, alar rim, or alar base can be reconstructed by a composite skin and cartilage graft harvested from the ear, usually at the helical root. Such grafts, similar to simple skin grafts, survive by vascularization from the wound bed. However, since these defects are full thickness, the wound bed is only the periphery of the wound. Therefore, defects must be less than 2 cm in diameter otherwise vascularization from the periphery is unreliable and the graft has a high risk of failure. The portion of the ear that is harvested should yield a graft that has the shape of the missing tissue of the nose. For alar rim defects, this is usually the helical root with donor-site closure done in an inconspicuous manner.

Local Flaps

In contrast to skin grafts, the color and quality of local flaps are very predictable. Skin flaps are thicker than skin grafts and provide more bulk to replace missing tissue. A local flap is a viable reconstructive option if the nasal defect is small, superficial, greater than 5 to 10 mm from the nostril margin, and is located above the nasal tip defining point. If the nasal defect is larger than 1.5 cm, then the excess skin of the dorsum and nasal sidewall is insufficient to use as a local flap.

A single-lobe transposition flap, designed as a Banner or Romberg flap, is used for small nasal defects. Excess skin from one axis is pivoted 90 degrees to fill a deficiency in another axis. The superior aspect of the nose and sidewalls are composed of an excess of mobile skin allowing for closure of small defects without distorting the nasal tip or alar margins. These single-lobe flaps should not be used within the thick, rigid skin of the nasal tip or ala.[6,8]

The dorsal nasal flap is an option for defects within the nasal dorsum and superior tip subunit. Excess skin, subcutaneous tissue, and muscle are elevated from the glabella region and transferred toward the nasal tip. Advancing skin from the cheek upward on to the nasal sidewall closes the defect. The vascular supply is from the facial and angular vessels along the nasal sidewall and medial canthus. The drawback to the dorsal nasal flap is that the thicker glabella skin is transferred downward onto the nasal sidewall, near the medial canthus. This creates a skin thickness mismatch and can create an iatrogenic epicanthal fold.[6,9]

For defects measuring 0.5 to 1.5 cm, located within the thick skin of the nasal tip and alar subunits, the bilobed flap is the preferred reconstructive local flap technique (▶ Fig. 18.6). The first lobe is designed adjacent to the nasal tip or alar defect.

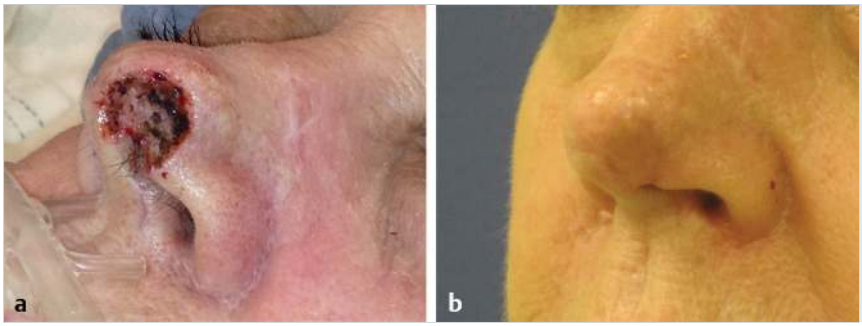


Fig. 18.5 **(a)** Full-thickness nasal left hemi-tip defect involving the alar rim. **(b)** Reconstruction with composite graft harvested from the helical root of the left ear.

Fig. 18.6 **(a)** Nasal sidewall defect extending onto the ala with marked bilobed flap for reconstruction. **(b)** Healed site after reconstruction.

Then, a second lobe is created within the excess tissue of the nasal dorsum or upper sidewall. These two lobes are created in continuity. The first lobe shifts to reconstruct the defect, while the secondary lobe resurfaces the defect caused by the first lobe. The defect caused by the second lobe is closed primarily. The base of this rotation-advancement flap must be positioned away from the margin of the nares to avoid distortion. The flap's overall rotation should be limited to 90 to 100 degrees to avoid the creation of excessive standing cutaneous deformities.[9,10,14]

In designing a bilobed flap, the pivot point is placed at a distance from the nasal defect equal to the radius of the defect. For tip defects, position the flap laterally. If the defect is within the alar subunit, position the flap medially. Once the pivot point is positioned, draw two concentric circles, a smaller inner circle and a larger outer circle. The smaller inner circle is drawn equal to the size of the diameter of the nasal defect. The larger outer circle is drawn at a distance three times the radius of the defect. Next, a template of the nasal defect is positioned adjacent to the defect, along the outer concentric circle. Ensure the first lobe is the exact size of the defect to avoid alar or tip distortion. The second lobe is positioned within the mobile nasal skin and is drawn slightly smaller than the secondary defect. These lobes are elevated superficial to the level of the periosteum and includes skin, subcutaneous adipose tissue, and the nasalis muscle. A lateral incision is created lateral to the outer circle to excise the standing cutaneous deformity caused by the transposition of the second lobe. This excision is then closed primarily.

The nasolabial flap is preferred for defects of 2 cm located on the nasal sidewall or ala, or hemi-tip. This technique utilizes the nearby medial cheek tissue to reconstruct the nasal defect.[7] To start, the nasal sidewall and alar subunits are marked. Depending on the size and location of the defect, it may be desirable to excise the skin remaining between the defect and the inferior nostril rim or alar base to help blend the flap's skin onto the nostril border. Next, the nasolabial crease is marked with the ink pen and a pattern of the nasal defect is outlined adjacent to this crease. This positioning allows the future cheek scar to lie within the nasolabial crease. The width of the flap should be equal to the width of the defect. The flap is then elevated. Undermine approximately 3 to 5 cm laterally. The cheek is then advanced. The raw undersurface of the cheek flap should be anchored to the deep tissues along the nasal facial groove. Excise any excess subcutaneous adipose tissue so that the thickness of the flap matches that of the defect.

Forehead Flap

A forehead flap is the workhorse flap in nasal reconstruction. It helps reconstruct defects larger than 1.5 cm located within the tip or columella subunits. This flap is universally acknowledged as an ideal reconstructive technique because of the forehead skin quality, size of the flap, and robust vascularity. Unlike local flaps, the forehead flap recruits skin to reduce undue tension placed on the other nasal subunits.[15,16,17]

To date, the vertical paramedian forehead flap has emerged as the standard, most universally used, forehead flap for nasal reconstruction (▶ Fig. 18.2, ▶ Fig. 18.7). This flap has a low pivot point providing easy reachability to the nasal defect extending to the columella. Its donor site is closed primarily. If the most superior aspect cannot be approximated, secondary healing usually occurs satisfactorily without the need for a skin graft. The vascular pedicle to this flap is based on the supratrochlear vessels, which exit the orbit superficial to the periosteum and penetrate through corrugator muscles. Two centimeters above the supraorbital rim, the supratrochlear vessels enter the frontalis musculature and travel within the subcutaneous adipose tissue toward the hairline.[17]

Traditionally, the forehead flap is transferred in two stages: a flap transfer stage and then the pedicle division and inset stage. The first stage allows for thinning distally and neovascularization at the point of inset. Two to 3 weeks later, the second stage is performed with division of the pedicle and debulking of the proximal aspect.

During the flap transfer stage, the remaining nasal subunits are marked and the residual skin of the tip or ala is excised. A

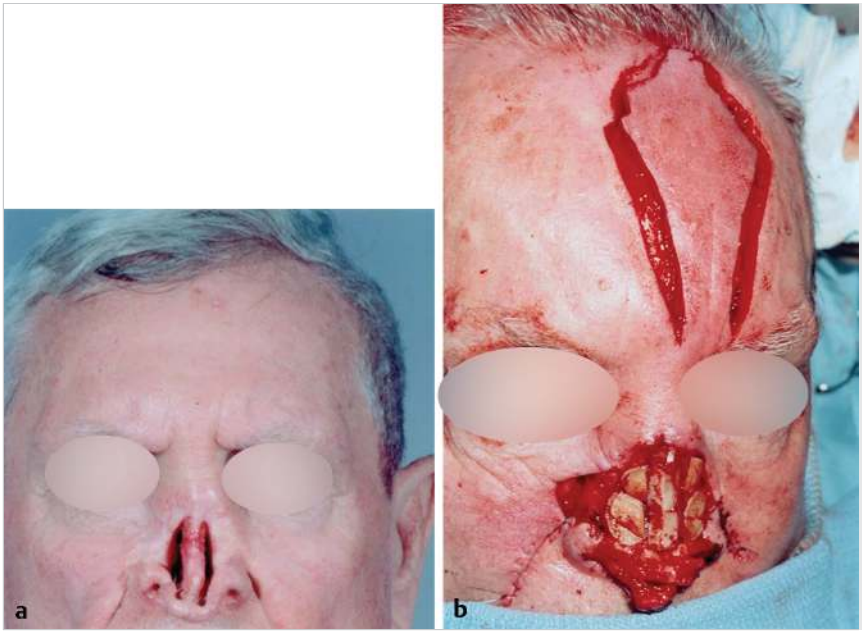


Fig. 18.7 Patient with a subtotal rhinectomy defect after basal cell cancer resection. **(a)** Defect violates most subunits and has loss of lining, skeletal support, and skin. **(b)** Reconstruction was accomplished using a septal pivot flap for the dorsum, conchal, and rib cartilage for tip and sidewall skeletal support; turnover cheek flaps for lining; and paramedian forehead flap for skin coverage.

nasal pattern using the unaffected contralateral side, or ideal, is traced overlying the supratrochlear vasculature, directly underneath the hairline. The supratrochlear artery pedicle is approximately 1.2 to 1.5 cm in width at the brow.

The flap harvest is started at the distal 1.5 to 2 cm in the upper forehead and includes the frontalis muscle and the subcutaneous adipose tissue, while leaving the frontal bone periosteum intact. This produces a skin flap thickness of approximately 2 to 3 mm that will reconstruct the most inferior aspect of the nasal defect. The dissection is continued deep to the frontalis muscle and superficial to the periosteum so that the pivot point is within the medial brow. Alternatively, in thick skinned individuals, the dissection can begin in the upper forehead superficial to the frontalis muscle. This dissection plane is carried inferiorly toward the brow to the level at which inset into the defect is not needed. The dissection is then taken deep to the frontalis so as to preserve the supratrochlear blood supply. The tensionless flap is then sutured into position over the nasal defect with a single layer of sutures. The forehead donor site is widely undermined and closed primarily, in layers.

The second stage, pedicle division, is performed 3 to 4 weeks after the first stage. The proximal portion of the flap is re-elevated, including the 2 to 3 mm of adipose tissue, frontalis muscle, and scar, and divided from its main blood supply. The flap remains perfused through its distal inset. The superior aspect of the nasal defect is contoured. The proximal pedicle is closed in an inverted "V" within the medial brow.

Other forehead flap variants include the median forehead flap, the horizontal forehead flap, and the sickle forehead flap. The median forehead flap pedicle is based on the supratrochlear vasculature and has a wide base above the brows.[16] The horizontal forehead flap, or the Gillies up-and-down flap, is used to increase flap length. However, the additional donor-site scaring eliminates the ability to perform a second flap harvest from the contralateral forehead.[2] The sickle forehead flap is based on the ipsilateral superficial temporal vasculature; however, the morbidity to the donor site requires a permanent skin graft.[2]

18.5 Postoperative Care

The postoperative care for nasal reconstruction is similar to that of other facial reconstructive procedures. There is some variation among surgeons, but in general the patient should keep their head elevated, avoid trauma, avoid exposure to sunlight, and avoid placing pressure on the repair by other mechanisms (glasses, etc.). The remainder of the postoperative care is typical of that for other facial surgeries.

18.6 Outcomes

The vascularity of the face is robust and all but mitigates the risk of ischemia or infection. Therefore, overall outcomes are usually favorable. If a complication does occur, the secondary repair is usually always salvageable; however, this usually comes at the expense of an increased number of operative procedures and stages. Secondary procedures are classified into three categories: minor revision, major revision, and a complete "redo."

In the case of a minor revision, the overall dimension and volume of the nasal reconstruction are correct; however, fine definition of the subunits is needed. These revisions are typically performed using direct incisions that are hidden within the subunits themselves. Most often minor revisions are completed in one stage.

A major revision is needed when the nasal reconstruction results in a shapeless and/or bulky nasal appearance. Using peripheral incisions, wide exposure is possible for gross debulking, as needed. Major revisions may require more than one procedure.

A complete "redo" procedure is necessary when a gross tissue deficit remains. The defect is re-created and the deficiencies are identified and corrected. These types of secondary procedures typically require multiple procedures.

Nasal reconstruction outcomes are functional and aesthetically acceptable. Using the nasal subunit based reconstructive technique, scars are easily concealed within the anatomic break lines and natural creases. Multiple stages may be required; however, with a proper reconstructive plan, tissue selection, and nasal support, an optimal aesthetic outcome is attainable.

18.7 Review Questions

18.7.1 True or False

1. The maximum rotation of a bilobed flap is 100 degrees.
2. The nose consists of three central elements: external skin, supporting bone and cartilage, and a mucoperichondrium lining.
3. The subunit principle of nasal reconstruction states that if 30% or more of a subunit is missing, then the remaining portion of the subunit should be excised completely to achieve an optimal aesthetic outcome.

18.7.2 Choose the Best Answer

4. A 65-year-old woman presents with a 1.7-cm defect within the nasal tip from a Mohs' operation with clear margins. She had no previous facial operations or rhinoplasties. What is the best reconstructive procedure for this patient?
   a) Skin graft.
   b) Nasolabial flap.
   c) Forehead flap.
   d) Single-lobe transposition flap.
5. A 46-year-old man presents with a 0.8-cm nasal defect within the left ala. The patient had no previous rhinoplasty or septoplasty. The defect is from a Mohs' excision of a nonmalignant lesion. What is the best reconstructive option for this patient?
   a) Skin graft.
   b) Dorsal nasal flap.
   c) Bilobed flap.
   d) FAMM flap.

18.7.3 Answers

1. True.
2. True.
3. False.
4. c. Forehead flap.
5. c. Bilobed flap.

References

[1] Antia NH, Daver BM. Reconstructive surgery for nasal defects. Clin Plast Surg. 8(3):535–563

[2] Menick FJ. Nasal reconstruction. Plast Reconstr Surg. 125(4):138e–150e

[3] Buchanan PJ, Kung TA, Cederna PS. Evidence-based medicine: wound closure. Plast Reconstr Surg. 134(6):1391–1404

[4] Millard DR , Jr. Aesthetic reconstructive rhinoplasty. Clin Plast Surg. 8(2): 169–175

[5] Burget GC, Menick FJ. The subunit principle in nasal reconstruction. Plast Reconstr Surg. 76(2):239–247

[6] Elliott RA , Jr. Rotation flaps of the nose. Plast Reconstr Surg. 44(2):147–149

[7] Herbert DC, Harrison RG. Nasolabial subcutaneous pedicle flaps. Br J Plast Surg. 28(2):85–89

[8] Masson JK, Mendelson BC. The banner flap. Am J Surg. 134(3):419–423

[9] Rieger RA. A local flap for repair of the nasal tip. Plast Reconstr Surg. 40(2): 147–149

[10] Zitelli JA. The bilobed flap for nasal reconstruction. Arch Dermatol. 125(7): 957–959

[11] Bloom JD, Antunes MB, Becker DG. Anatomy, physiology, and general concepts in nasal reconstruction. Facial Plast Surg Clin North Am. 19(1):1–11

[12] Menick FJ. Nasal reconstruction. In: Thorne CH, ed. Grabb and Smith's Plastic Surgery. Philadelphia, PA: Lippincott Williams & Wilkins; 2007

[13] Parrett BM, Pribaz JJ. An algorithm for treatment of nasal defects. Clin Plast Surg. 36(3):407–420

[14] Zimany A. The bi-lobed flap. Plast Reconstr Surg (1946). 11(6):424–434

[15] Mazzola RF, Marcus S. History of total nasal reconstruction with particular emphasis on the folded forehead flap technique. Plast Reconstr Surg. 72(3): 408–414

[16] McCarthy JG, Lorenc ZP, Cutting C, Rachesky M. The median forehead flap revisited: the blood supply. Plast Reconstr Surg. 76(6):866–869

[17] Reece EM, Schaverien M, Rohrich RJ. The paramedian forehead flap: a dynamic anatomical vascular study verifying safety and clinical implications. Plast Reconstr Surg. 121(6):1956–1963

19 Facial Fractures

Russell Walther, Luke Pearson, Sushmita Mittal, and Dhruv Singhal

Abstract
This chapter offers a concise discussion of fracture types, surgical management, and areas of concern for each type of fracture involving the head, neck, and scalp. Areas of concern during the physical examination are listed so that no structure potentially affected by a fracture is overlooked. Diagnostic procedures, involving tools such as computed tomography imaging, are reviewed, and then each major type of facial fracture—frontal bone, nasoorbitoethmoid, nasal bone, zygomaticomaxillary complex, orbital, LeFort/maxillary, mandible, panfacial—is thoroughly detailed. Postoperative care guidelines and long-term outcomes conclude the chapter.

Keywords: frontal bone, nasoorbitoethmoid, nasal bone, zygomaticomaxillary complex, orbital, LeFort/maxillary

19.1 Goals and Objectives

- Understand the physical exam findings and diagnostic maneuvers associated with each fracture type.
- Define the surgical indications for each fracture type.
- Understand the general surgical management and specific areas of concern for each fracture type.
- Discuss routine postoperative care of the facial trauma patient.

19.2 Patient Presentation

Patients with facial fractures have typically been involved in assaults, motor vehicle accidents, falls, sporting accidents, or work related injuries. With exception, most facial fractures are non-emergent, and other injuries should be thoroughly evaluated and potentially treated prior to management of facial fractures. Evaluation and management of the airway, hemorrhage, and neurologic injury are priority during initial assessment.

A thorough physical examination of the hard and soft tissues of the head and neck is paramount for the diagnosis of maxillofacial fractures. Frequently missed soft-tissue injuries include the posterior neck and scalp due to cervical collar placement, external auditory canal, nasal mucosa, and intraoral soft-tissue structures. Palpation of the bony skeleton for irregularities, crepitus, and mobility, should be performed over the zygomatic arches, orbital rims, zygomas, frontal bone, nose, and mandible.

Otoscopic evaluation should be performed with assessment of the tympanic membrane and soft tissues of the external auditory canal. Hemotympanum or mastoid ecchymosis may indicate a skull base fracture. An anterior canal laceration can be seen with condylar fractures or anterior auditory canal fractures.

The ophthalmic examination should include evaluation of the cornea, conjunctiva, anterior chamber, lacrimal apparatus, intercanthal distance, lateral and medial canthus symmetry in the frontal plane, and fundus examination. Any patient with a known orbital wall fracture with forehead paresthesia should be closely scrutinized for superior orbital fissure syndrome or orbital apex syndrome. Hyphema, findings supporting globe rupture, intraocular foreign bodies, change in visual acuity, or a lid margin laceration warrant ophthalmologic consultation.

Subconjunctival hemorrhage, an inferiorly displaced lateral canthus, and zygomaticofrontal suture step-offs can be seen with zygomaticomaxillary complex (ZMC) fractures.

Increased intercanthal distance (normal female: 32 mm, normal male: 34 mm), lacrimal injury with epiphora, decreased nasal air flow, and rounding of the medial canthus are seen with nasoorbitoethmoid (NOE) fractures. A Jones I and II test can confirm the presence of lacrimal duct injury. The bowstring test may demonstrate an undiagnosed medial canthus avulsion. ZMC, NOE, and orbital wall fractures may present with diplopia and restricted movement of the eye. Fractures involving the orbital floor or LeFort 2 fractures will often have concurrent infraorbital nerve injury, with paresthesia of the cheek, lateral ala, lower lid, maxillary teeth, and buccal mucosa. Fracture of the frontal sinus may demonstrate clear rhinorrhea, anosmia, or epistaxis. A beta 2 transferrin test of clear nasal discharge is used to diagnose a cerebrospinal fluid (CSF) leak.

An anterior open bite is the most common physical exam finding in patients with maxillary fractures. Midface mobility may be present. Lack of midface mobility does not preclude a fracture, as an impacted maxilla may not be grossly mobile. Epistaxis is common with maxillary, nasal, and NOE fractures.

A step in occlusion with associated open bite is common in mandible fractures. Floor of mouth ecchymosis is pathognomonic of symphysis or body fractures. Vertical collapse of the posterior ramus/condyle height causes a contralateral dental open bite. Bilateral condyle fractures result in an anterior open bite, which may be confused with a midface fracture.

19.3 Preparation for Surgery

Laboratory data in preparation for facial fracture repair is dependent on age and other medical comorbidities. In general, extensive laboratory workup specific for facial fracture repair is limited to only those tests needed to ensure that the patient can tolerate a general anesthetic.

A baseline ophthalmologic examination is cursory, and expert consultation should be obtained should there be any question as to ocular injury. While not warranted in every instance, complex fractures involving the orbit, concern for ocular injury, or extraocular muscle impairment require expert evaluation. In our practice, any patient requiring orbital fracture repair is evaluated by ophthalmology preoperatively to document a baseline examination. Neurosurgery evaluation should be obtained in every patient with concern for a CSF leak post trauma. Patients with hemotympanum, tympanic membrane perforation, external auditory canal wall laceration or fracture, temporal bone fracture, or significant change in auditory acuity warrant evaluation with an otolaryngologist.

Computed tomography (CT) imaging is the gold standard for diagnosis of maxillofacial trauma. In our practice, any patient with significant facial fractures undergo CT imaging with 1 mm

cuts reformatted in coronal and sagittal planes. Panorex imaging has some utility in the diagnosis and management of injuries involving any tooth-bearing segment of the jaws. Fractures isolated to the mandible only, with no other distracting injury, may be diagnosed by Panorex in conjunction with other plain film modalities like the submentovertex or reverse Towne's view plain films. Reliance on a single plain film view of the mandible can result in undiagnosed fractures. No matter the modality, a systematic approach to reviewing facial radiographs is critical given the complexity of the anatomy.

Most facial trauma involves the oral cavity or nose, creating specific concerns for the provider managing the airway. Airway emergencies are minimized with discussion of airway management with the anesthesia provider. Any dentate patient with a fracture involving the mandible or maxilla should undergo nasal intubation to allow maxillomandibular fixation. A sphenoid sinus fracture, or fracture involving the skull base, should alert the surgeon to possible inadvertent entry of a nasal endotracheal tube or any tube into the middle cranial fossa or adjacent spaces.[1] In pan facial fractures involving the mandible and maxilla, surgical airway management should be performed. In patients not requiring long term ventilator support, submental intubation provides the ability to perform maxillomandibular fixation (MMF) without the morbidity of tracheostomy.[2] In patients requiring long term ventilator support, tracheostomy should be performed.

19.4 Treatment

19.4.1 Frontal Bone Fracture

Frontal sinus fractures are generally caused by anterior blunt force trauma. The leading mechanism is motor vehicle accidents. With the increasing use of seat belts and airbags, there has been on overall decrease in incidence of frontal sinus fractures.[3] Other mechanisms of fracture include sports injuries, falls, falling objects, and interpersonal violence.

While many classifications of frontal sinus management exist, consideration for repair hinges on displacement of the anterior or posterior table and the patency of the nasofrontal duct. Patients with non-displaced frontal sinus fractures that involve the anterior or posterior tables, with an intact nasofrontal duct (NFD), and without the need for neurosurgical access should be managed non-operatively. Nasal precautions with avoidance of Valsalva maneuvers, closed mouth sneezing, or heavy lifting should be instated.

Any patient with a comminuted/significantly displaced (greater than the width of the outer table) posterior table fracture or with significant brain injury or dural tear should undergo cranialization of the frontal sinus and repair of the anterior table. In patients with a displaced anterior table fracture and intact NFD, anterior table repair only, without obliteration, should be performed. In patients with a displaced anterior table fracture with NFD involvement, obliteration of the frontal sinus should be performed with complete removal of all sinus lining. In patients with non-displaced anterior or posterior table fractures with NFD injury, reduction of the NOE component should be performed with evaluation of the NFD. If the NFD is not patent, then obliteration with removal of all sinus lining should be performed.[4]

The frontal sinus is approached through existing lacerations, or via a bicoronal incision. Anterior table reduction techniques vary from simple elevation of bone fragments without fixation to removal of bone fragments and orientation extracorporeal with fixation and reimplantation. It is important to maintain orientation of the fragments. Traumatic distortion of the segments may occur. It may be necessary to trim the margins of the fragments to assist in the reduction of comminuted segments.

Exploration, sinus mucosa removal, closure of the NFD, and obliteration of the frontal sinus presents specific challenges. When sinus mucosa removal is indicated, removal of the entirety of the anterior table is indicated. A forceps can be placed into the sinus to define the boundaries of the sinus which are subsequently marked. The anterior table is indexed with fixation, the plates are removed, and the bone is subsequently osteotomized. The anterior table is then elevated and removed. All sinus mucosa and septae should be removed with diamond burs. Placement of methylene blue in the sinus prior to mucosa removal may help in preventing missed areas of sinus mucosa (▶ Fig. 19.1).

NFD obstruction is accomplished with elevation of the sinus mucosa in the frontal recess, which is then inverted and compressed inferiorly to obstruct the ducts. Bone, fat, fascia, or muscle may be placed into the recess. Obliteration of the sinus is accomplished with bone, bone substitutes, fat, pericranium, and muscle. Fat remains the most commonly used and studied material.[5] Synthetic resins should be avoided as they create an avascular medium with high potential for infection.[6] Posterior table fractures, dural repair, and cranialization are generally managed in conjunction with neurosurgery and are beyond the scope of this text.

Paramount to any frontal sinus repair is evaluation for a CSF leak. Clear rhinorrhea may be evaluated intraoperatively with a halo test, though this has low specificity and sensitivity. Fluid testing positive for beta-2-transferrin is diagnostic of a CSF leak. Most CSF leaks resolve spontaneously; however, persistent leaks may require cranialization and dural repair. Lumbar diversion may play a role in management of some leaks.

Complications of frontal sinus injury can be subdivided into early (< 6 months) and late (> 6 months). Early complications include sinusitis, CSF leak, and meningitis. Late complications include mucoceles, mucopyoceles, and brain abscess.[7] Late complications may present years later, therefore long-term surveillance with imaging is indicated.

Advancements in endoscopic sinus surgery have generated interest in treatment of anterior table fractures with NFD injury. Anterior table repair without obliteration with antibiotic administration and serial computed tomography is advocated by some authors.[8] NFD patency may spontaneously occur after repair of the anterior table in a majority of patients, with select patients requiring secondary endoscopic sinus surgery for NFD outflow obstruction. Expert consultation with an otolaryngologist may be warranted in select cases.

19.4.2 Nasoorbitoethmoid Fractures

The nasoorbitoethmoid (NOE) complex is the confluence of the nose, orbits, ethmoids, inferior frontal sinus, and floor of the anterior cranial base. Like frontal sinus fractures, NOE fractures

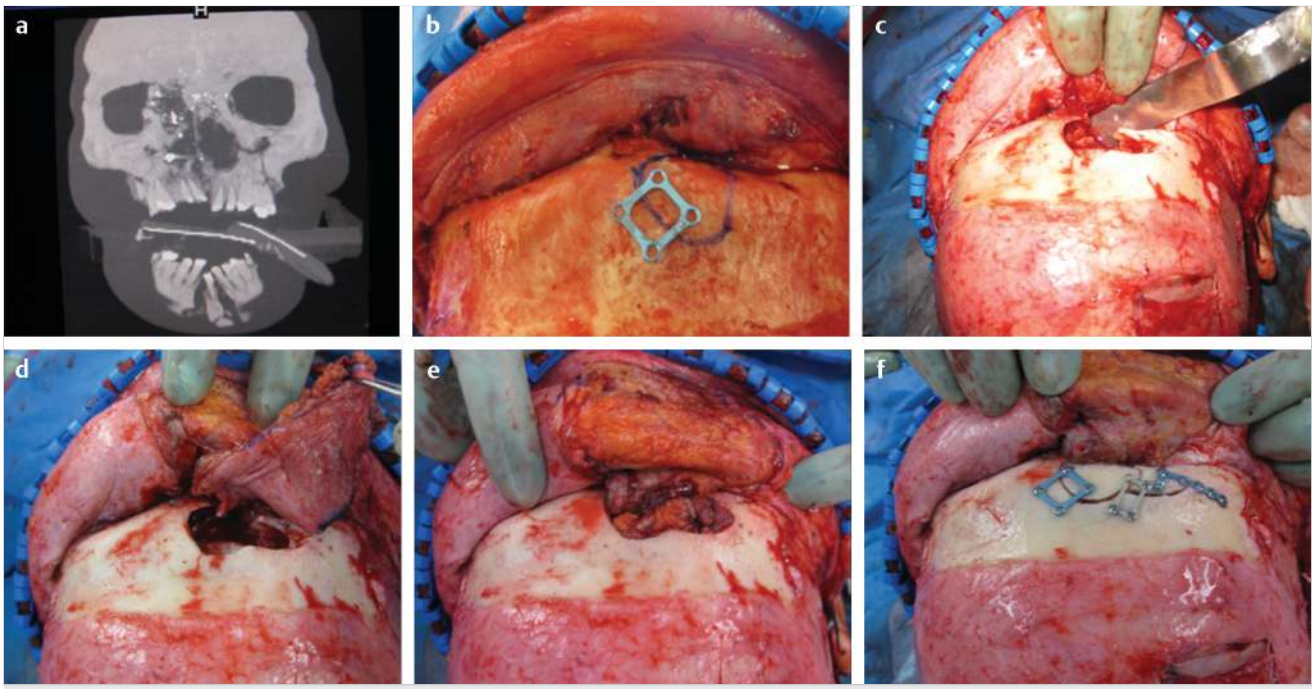


Fig. 19.1 **(a)** A 32-year-old male s/p inflicted GSW to face with compromise of right frontal sinus floor and nasofrontal duct (NFD). **(b)** A coronal incision was performed and the anterior table is indexed and removed with a 701 Burr **(c)** Removal of the anterior table reveals **(d)** a defect of the frontal sinus floor. **(e)** Following burring of the sinus, obliteration of the NFD with bone fragments and a fronto-galeal-pericranial flap is raised. **(f)** The flap obliterated the sinus. The anterior table was replaced with 0.4 and 0.6 mm previously indexed plates with attention paid to burr down the inferior aspect of the anterior table to allow passage of the flap. A split calvarial graft, donor site visible in the lower right, was utilized for nasal reconstruction and cantilever bone grafting.

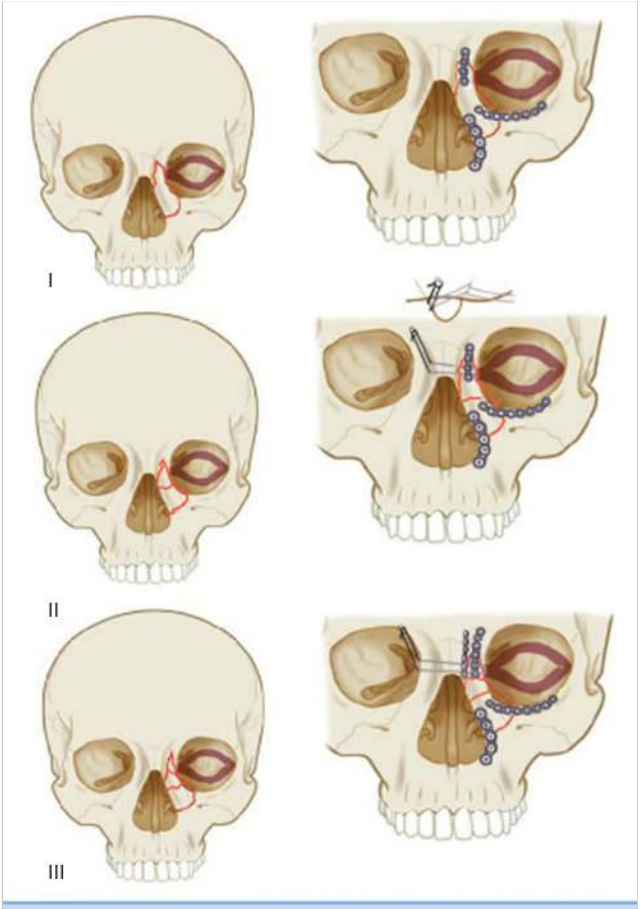
are generally the result of blunt-force trauma to the midface. CSF leak, orbit, globe, nasolacrimal duct, nasal, and intracranial injuries are frequently seen in association with NOE fractures. The classic fracture pattern involves lateral nasal bones, inferior orbital rim, medial orbit/ethmoid wall, nasal maxillary buttress at the piriform aperture, and the junction of the frontal process of the maxilla with the frontal bone. The NOE region involves the major central vertical (nasomaxillary) and horizontal (frontal and inferior rim) buttresses of the face. Re-establishment of an ideal nasofrontal angle (115–130 degrees), nasal projection, and intercanthal distance are critical in appropriate repair.

The medial canthal tendon (MCT) divides into anterior and posterior limbs which attach anteriorly along the anterior lacrimal crest and posteriorly along the posterior lacrimal crest. The MCT envelopes the lacrimal sac. The classification developed by Markowitz et al evaluated the MCT attachment and degree of comminution of the NOE fragment.[9] In type 1 fractures, the MCT remains attached to a large bony segment (central fragment) without comminution. In type 2 fractures, the NOE segment is comminuted with the MCT remaining attached to the central fragment. In type 3 fractures, there is comminution of the NOE segment with detachment of the MCT. A type 1 fracture is typically discernable from a type 2 or 3 fracture radiographically. MCT avulsion is generally determined intraoperatively making preoperative diagnosis of a type 2 versus type 3 fracture difficult (▶ Fig. 19.2).

Open reduction internal fixation for NOE fractures is indicated in all type 2 and 3 fractures, and in type 1 fractures with clinically significant displacement resulting in telecanthus or malposition of the MCT. In type 1 fractures with no clinically significant displacement and no mobility of the MCT, close observation of the fracture may be employed.

The NOE complex requires superior and inferior component access. As with all fractures, existing lacerations may be used. The superior NOE complex (nasofrontal region) is approached through a bicoronal incision with degloving of the superior and medial orbits. Scoring of the periosteal envelope over the radix reduces tension when retracting inferiorly. The inferior NOE complex is approached through a transconjunctival or external lower lid incision. Medial orbital/NOE exploration is facilitated with a transcaruncular incision if needed. The gullwing, extended glabellar incision, and Lynch incision are generally not recommended due to poor cosmesis. The piriform rim may be plated via an intraoral maxillary vestibule incision.

The sequencing of NOE complex repair after surgical exposure is as follows: the MCT is identified, the medial orbital rims followed by medial orbital walls are reconstructed, transnasal canthopexy is performed, the nasolacrimal apparatus is repaired, the nasal dorsum is reconstructed, the soft tissue is closed and the medial canthal soft tissue is bolstered.[10] If the MCT is not readily apparent, identifying the nasofrontal suture and dissecting inferiorly and posteriorly leads to the apex of the lacrimal fossa. The medial orbital rims are reduced and fixated with rigid fixation. Transnasal wiring posterior to the lacrimal fossa may assist in reduction of the medial orbital walls. Roughly half of all patients with NOE fractures required bone grafting or alloplastic reconstruction of the medial orbital wall. Inadequate medial reduction and anterior projection leads to



I	Detachment of isolated, large fragment with attached canthus. Fixation of the fragment is sufficient.
II	Several fragments. The so-called "central fragment," to which the medial canthus is attached by its tendon, is still sufficiently large for osteosynthetic fixation, though it must be adequately stabilized against rotary forces to prevent redisplacement of fragments.
III	Avulsion of the medial canthus from the bony fragments as isolated or additional injury. Additional fixation of the canthus necessary using a hypomochlion with a wired suture.

Fig. 19.2 Schematic illustration of fractures based on Markowitz management. (From Ernst A, Herzop M, Seidl RO. Head and Neck Trauma: An Interdisciplinary Approach. Stuttgart, Germany: Thieme; 2006:78)

cosmetic deformity with telecanthus that is exceedingly difficult to repair secondarily. Over-reduction is effectively impossible and leads to less postoperative complaints than under-reduction (telecanthus).

The transnasal canthopexy is the most difficult aspect of NOE repair. Fortunately, only 3% of patients require canthopexy. The MCT may be posteriorly approached through the coronal approach or anteriorly with a small incision through dermis 3 mm to the medial canthus. There are multiple techniques to anchor the MCT to a point of attachment (transnasal wire, plate, screw). Regardless of the technique, the point of attachment should be located superior and posterior to the posterior lacrimal crest. Primary repair of the nasolacrimal apparatus is controversial and beyond the scope of this text (▶ Fig. 19.3).

Dorsal nasal support should be evaluated and repaired primarily. Dorsal nasal grafting is indicated if the cartilaginous or bony septum is significantly comminuted with anticipated loss of vertical support of the nasal bones and upper lateral cartilages.[11] A calvarial or costochondral graft may be utilized. The distal margin of the graft should be tucked under the lower lateral cartilages to prevent palpability.

The soft tissue of the medial canthal region is degloved during NOE repair. Tight adaptation of this region may be accomplished with medial canthal bolstering. A large nonresorbable suture may be passed from the medial canthal region, transnasal, to the contralateral medial canthal region. The suture may be passed back, and any type of bolster material (Xeroform) can be secured. The bolster is removed at 7 to 10 days.[12]

Two main complications arise after NOE repair. Persistent telecanthus results from inadequate medial reduction and soft-tissue management postoperatively. Secondary repair is exceedingly difficult and often is unsuccessful. Like primary repair, secondary repair should focus on addressing both the hard and soft tissue. Epiphora occurs in approximately half of all NOE fractures immediately postoperatively.[13] Most resolve after soft-tissue swelling decreases with only 5–10% of patients having persistent nasolacrimal duct obstruction.[9] Persistent epiphora should be treated no earlier than 6 months after NOE complex repair.

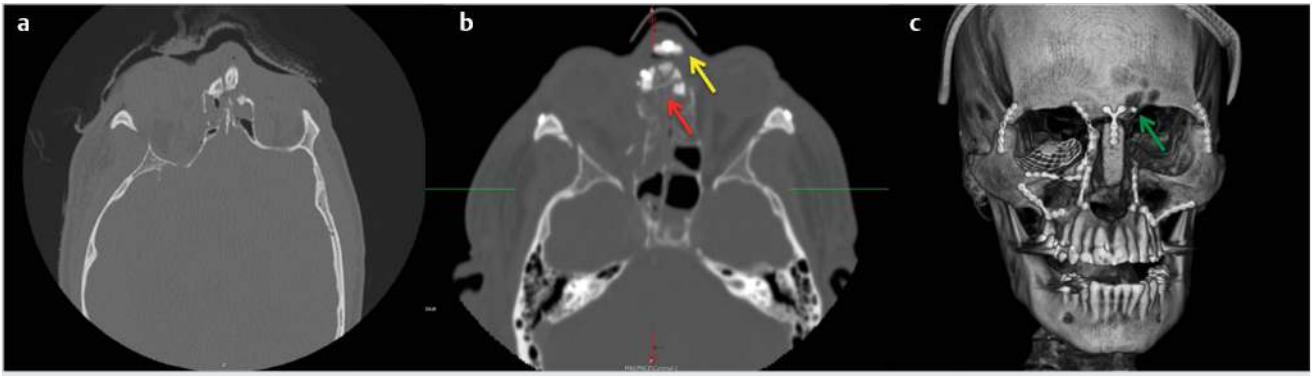


Fig. 19.3 **(a)** A 53-year-old male s/p a crush injury after placement of face into an industrial hydraulic compressor. Injuries included a right Markowitz Type 2 NOE fracture, right orbital floor blowout fracture, and LeFort I, II, and III fractures. **(b,c)** Axial and 3D imaging of NOE fracture highlighting transnasal wiring utilizing a Synthes 28-gauge canthal wire with barbed needle (red arrow) anchored to the left superomedial orbit (green arrow), fixation of the medial orbital rims, and a cantilever bone graft (yellow arrow).

19.4.3 Nasal Bone Fractures

The nasal bones are the most commonly fractured facial bone.[14] While primary repair of nasal bone fractures is generally "simple," unfavorable results occur frequently. Open repair and posttraumatic repair are beyond the scope of this text. History should include mechanism of trauma, direction of impact, history of prior nasal trauma, discussion of prior nasal asymmetry and photographic evaluation. All patients should be evaluated for the presence of a nasal septal hematoma. If present, incision and drainage of the hematoma should be performed.

Indications for repair are esthetic and functional when nasal obstruction or airway compromise has occurred. Contraindications to closed reduction include severe comminution of the nasal bones or septum, associated displaced orbital wall fracture, caudal septum fracture dislocation, open septal fracture, and fractures greater than 3 weeks old. In the case of displaced orbital wall fractures, the orbital wall should be repaired prior to closed reduction of the nasal bones.

Fracture repair may be performed under local anesthesia or general anesthesia. In our practice, a general anesthetic is preferred as epistaxis is common following closed reduction. Local anesthesia administration should focus on regional blocks to the infraorbital, infratrochlear, supratrochlear, external nasal, and nasociliary nerves. The administration of an appropriate amount of local anesthesia with epinephrine to regional blocks prevents distortion of the nasal dorsum and ala. Judicious use of oxymetazoline spray, local anesthesia with epinephrine, and phenylephrine soaked cottonoids reduce the volume and incidence of epistaxis.

After reduction of the nasal bones, splinting should focus on three anatomic regions of importance. The external nose should be protected with an external splint, and the nasal septum should be maintained at midline with internal splints (Doyle, Merocel, etc.). The internal nasal valve is often overlooked and inadequately supported. Placement of bacitracin coated 1/4" strip gauze into the internal nasal valve helps prevent internal valve collapse. All patients with nasal packing should be placed on prophylactic antibiotics to prevent or limit the development of sinusitis and Staphylococcus aureus infection that could lead to toxic shock syndrome.

Pediatric nasal trauma is uncommon due to lack of projection and elasticity of the underdeveloped nasal bones and associated cartilage. There is significant nasomaxillary growth that occurs from the cranial base centered at the nasofrontal suture and nasal septum.[15] Most nasal growth is complete by age 5; however, significant vertical growth limited to cartilage and soft tissue occurs after this age. Because of this, pediatric patients should be counseled that secondary rhinoplasty is highly likely. Addressing nasal airflow and traumatic nasal deformities should be delayed as long as reasonably possible until growth of the nasomaxillary complex is complete.

19.4.4 Zygomaticomaxillary Complex Fractures

Zygomaticomaxillary Complex (ZMC) fractures are the most common midface fracture. The ZMC is a tetrapod structure with articulations classically described in four areas: (1) the zygomaticomaxillary buttress (ZMB), (2) the infraorbital rim (IOR), (3) the frontozygomatic suture (FZ), and (4) the zygomatic arch (ZA). The fifth articulation, and most reliable in determination of adequate reduction, is the zygomaticosphenoid suture (ZSS). The ZMC fracture propagates through the floor and lateral walls of the orbit (▶ Fig. 19.4).

Separate classifications by Manson et al and Zingg et al evaluated the degree of displacement and comminution of the ZMC.[16,17] The ZMC can be fractured at one or more of the previously noted articulations in the Manson low energy fracture, or Zingg type A fracture. Fracture of all articulations of the ZMC without comminution occurs in Manson medium energy fractures, or Zingg type B fractures. Manson high energy fractures, or Zingg type C fractures, result in comminution and displacement of the ZMC.

Indications for surgery are both aesthetic and functional. Significant aesthetic compromise can occur with bizygomatic widening, decreased malar projection, and displacement of the lateral canthus. Functional indications for repair are enophthalmos, diplopia, or restriction of mandibular movement due to compression of the muscles of mastication below the zygomatic arch. Contraindications to surgical repair include no functional impairment, a high-risk surgical candidate, minimally or nondisplaced fractures, and severe contralateral globe injury with vision loss or impairment.

Given the variation in displacement and location of fracture of the ZMC, difficulty surrounds three questions: (1) When should the orbit be explored? (2) How much exposure is required to verify adequate reduction? (3) Should fixation be applied, and where?

With the advent of CT scans, orbital exploration when treating ZMC fractures decreased from 90 to 30% in the late 1980s.[18] Surgical exploration and repair of orbital wall fractures center around function (diplopia) which cannot be fully determined at the time of repair. Evaluation of entrapment with a forced duction test is mandatory after reduction of the ZMC. The presence of entrapment or significant enophthalmos are indications for orbital exploration. Preoperative CT evaluation with greater that 50% of the floor fractured with soft-tissue herniation has been advocated as a guideline for repair of the floor.[19] In general, low-energy fractures involving isolated articulations do not require orbital exploration. High-energy ZMC fractures often require orbital exploration and repair (▶ Fig. 19.5).

Rotation of the ZMC, in addition to displacement, creates challenges to ensure appropriate reduction. Axial rotation of the complex around any articulation may demonstrate adequate reduction at an isolated articulation, while globally the ZMC is still significantly displaced. The zygomaticosphenoid suture (ZSS) is the longest line of fracture and provides the most reliable indication of adequate reduction. The frontozygomatic suture does not reliably demonstrate adequate reduction of the ZMC due to ease of rotation of the complex around one point. Open evaluation of at least 3 points of articulation is typically recommended in significantly displaced medium and all high-energy fractures. Utilization of a Carroll-Girard screw or a Rowe elevator can be useful in mobilization of the ZMC.

While discussion of fixation is important, it should be stressed that reduction is the key to operative success. Ellis demonstrated that there was no perceptible change in ZMC position post-operatively when comparing fractures with and without fixation.[20] He suggested that post-operative asymmetry was likely

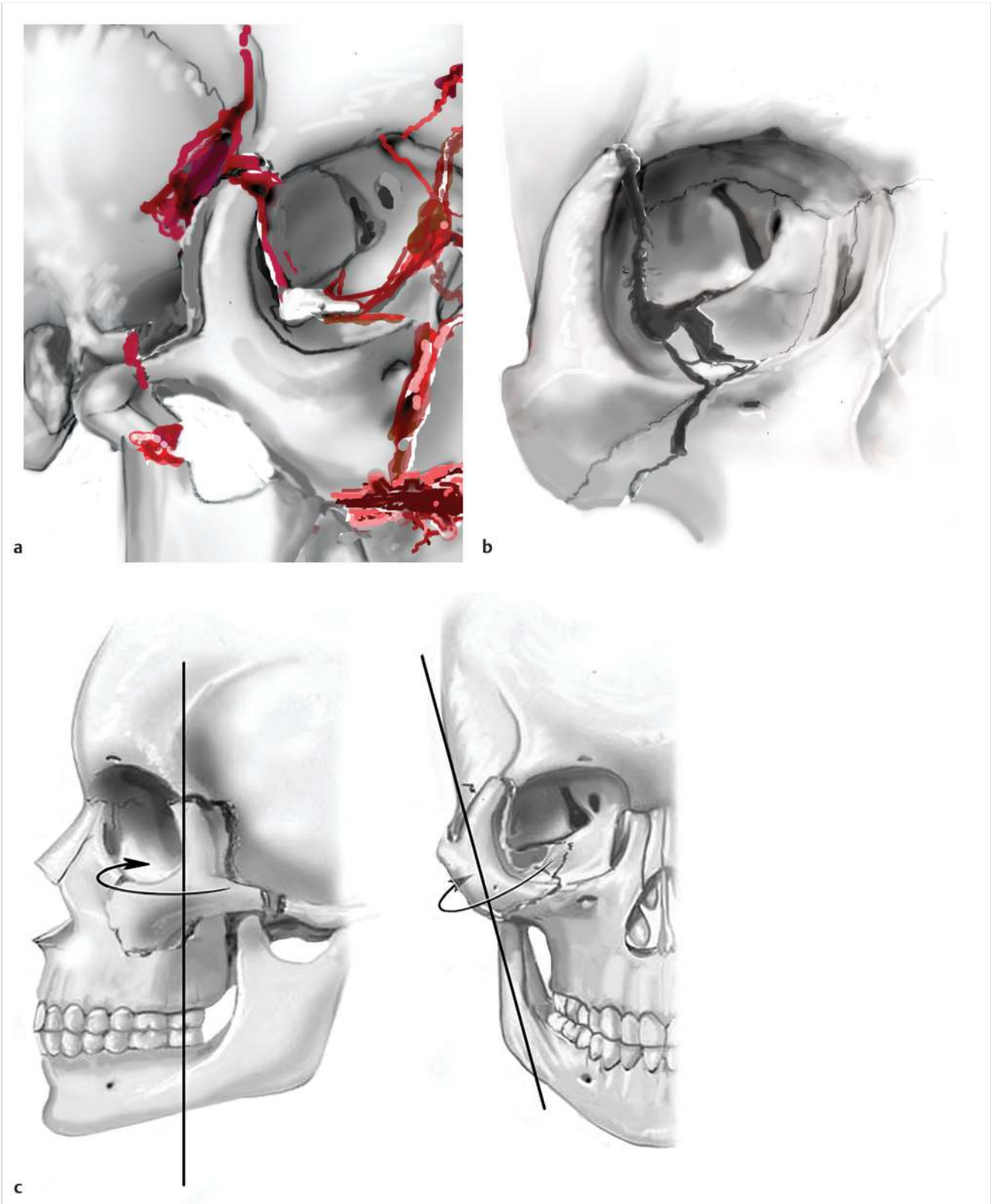


Fig. 19.4 The articulations of the zygoma with the craniofacial skeleton are shown in (**a**) and (**b**). The best areas to visualize fracture reduction are the internal surface of the orbit (1) and the zygomaticomaxillary buttress (2) infraorbital rim (3) and the zygomatic arch (4). The multiple articulations of the zygoma and its relationship to the orbit, temporal bone, and maxilla. Engaging a bone hook within the temporal aspect of the zygoma facilitates rotation and reduction. Pollock, R. Craniomaxillofacial Buttresses: Anatomy and Operative Repair. New York; Thieme: 2012.

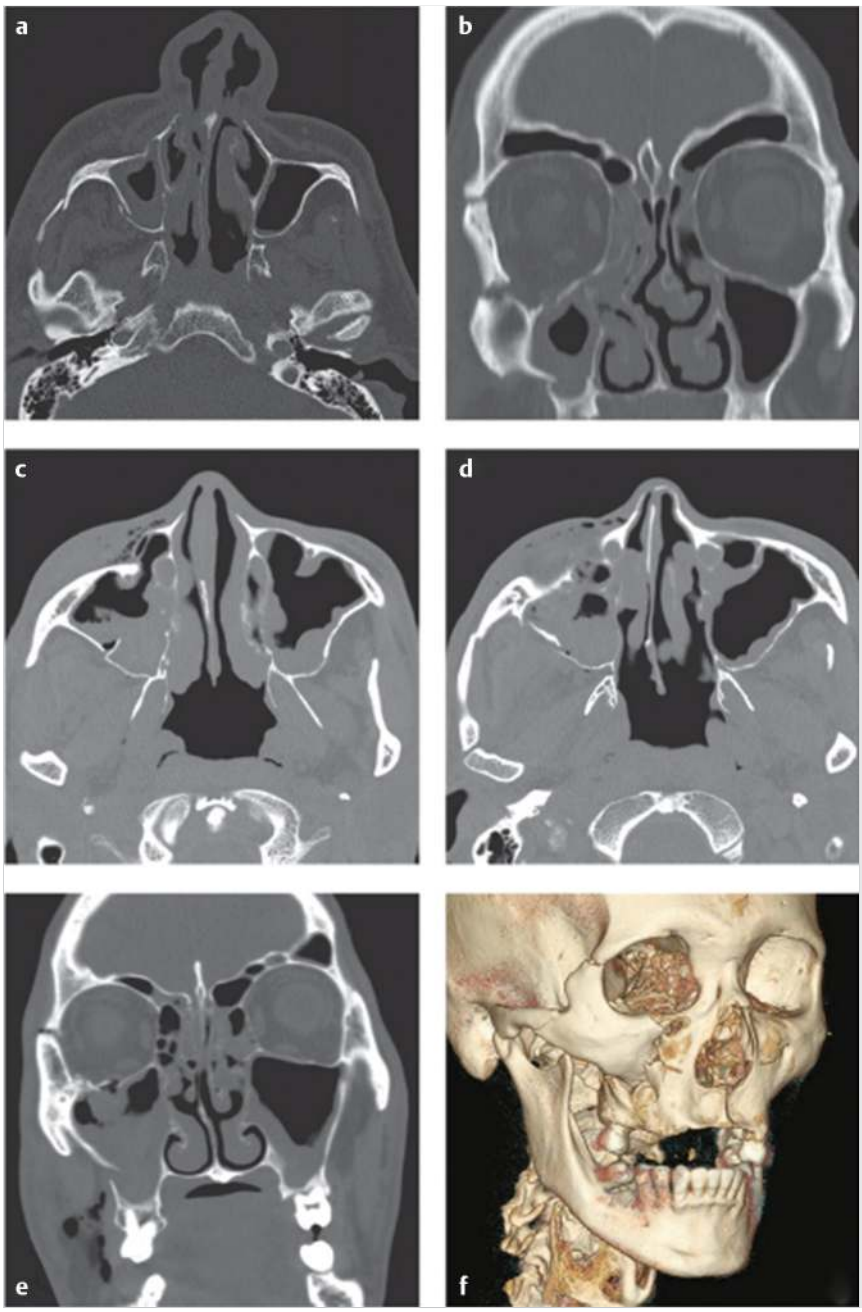


Fig. 19.5 (a,b) Zygomaticomaxillary complex fracture. Minimally displaced right zygomatic arch, anterior and posterolateral maxillary sinus wall, orbital floor fractures. Right zygomaticofrontal suture diastasis. **(c-f)** Zygomaticomaxillary complex fracture. displacement of the malar fragment with soft-tissue emphysema. Right zygomatic arch, anterior and posterolateral maxillary sinus wall, orbital rim, and orbital floor fractures. Right zygomaticofrontal suture diastasis. 3D reconstruction shows the malar fragment. (From Baxter AB. Emergency Imaging: A Practical Guide. New York, NY: Thieme; 2016)

secondary to unsatisfactory primary reduction. Fixation of the ZMC is primarily at the FZ and ZMB with the IOR as a secondary location for fixation. Fixation of the zygomatic arch is reserved for severely comminuted fractures or when coronal access is utilized for fixation of other fractures. The ZSS is rarely fixated. One point fixation is typically at the ZMB and two-point fixation is at the ZMB and FZ. Three or four point fixation including the IOR and ZA is reserved for significantly displaced or comminuted fractures. Should three or four point fixation be employed, orbital exploration should be considered given the high likelihood of significant floor displacement. With concomitant NOE and ZMC fractures, one must carefully consider fixation sequencing. When employing an "outside to inside" approach during treatment of multiple midface fractures, ORIF of the IOR to a laterally displaced NOE fracture can appear as an adequately reduced ZMC fracture. Significant bizygomatic widening can occur. It is the preference of the author to approach concomitant NOE and ZMC fractures medial to lateral in all but the most comminuted NOE fractures.

While discussion of all surgical approaches is beyond the scope of this chapter, careful consideration of the surgical approach is important. If evaluation of the ZF is indicated, the ZF and ZSS should be accessed simultaneously. An upper blepharoplasty incision is preferred approach for ZF/ZSS evaluation. The lateral brow incision should be discouraged due to poor cosmesis. If the IOR and floor are to be explored in addition to the ZF/ZSS, a transconjunctival incision with lateral canthotomy or subtarsal incision provides access to all of the

aforementioned articulations. The ZMB should be accessed intraorally through a maxillary vestibular incision. Orbital floor exploration typically requires an orbital incision; however, endoscopic evaluation and management can be employed via the maxillary vestibular incision. The inferior orbital rim incision (directly over the infraorbital rim) is contraindicated due to morbidity and poor cosmesis.

The isolated zygomatic arch fracture has specific management concerns. Given the morbidity of a direct open approach, two alternative surgical approaches exist. The indirect open "Gillies" approach via a temporal incision allows insertion of an elevator deep to the temporalis fascia and superficial to the temporalis muscle to reduce the zygomatic arch fracture. Careful dissection prevents hemorrhage from the temporalis muscle. The "Keen" approach utilizes a small 2–3 cm maxillary vestibular incision along the posterior maxillary buttress allowing indirect open access to the arch. It should be noted that both techniques are supraperiosteal, and rough manipulation of the tissues may result in neurovascular damage. Reduction of the arch confirmed by manual palpation is often adequate. Transcutaneous circumzygomatic arch wiring has been described with external splinting. Open fixation of the arch is via coronal access.

There are multiple complications that may occur related to ZMC fractures. In general, inappropriate reduction perioperatively is the cause of a preventable bony cosmetic deficit, and surgical access leads to the majority of soft-tissue complaints.

19.4.5 Orbital Fractures

The orbit is approximately 30 mL in volume. It is composed of seven bones with six fissures or canals transmitting various structures. Knowledge of the relationship of various foramina and nerve canals or fissures to known anatomic structures is essential.[21] While detailed anatomic review is beyond the scope of this text, several important surgical landmarks warrant further discussion. The nasolacrimal sac is located 4 mm posterior to the inferomedial orbital rim and is enveloped anteriorly and posteriorly by the medial canthal tendon. Whitnall's tubercle lies 1 cm below the frontozygomatic suture and 3–5 mm posterior to the lateral orbital rim. It serves as the attachment point for the lateral horn of the levator aponeurosis, the lateral canthal ligament, and Lockwood's ligament (inferior suspensory ligament).

Orbital fractures may be divided into pure fractures involving only the internal bony orbit, and impure fractures associated with other mid-face fractures (ZMC, LeFort maxillary fractures, etc).[22] The majority of this section will focus on pure orbit fractures. Traumatic force is applied to the periorbital region resulting in fracture of the orbital walls. Multiple theories explain the mechanism of fracture as direct bony contact from the compressing object does not occur in most fractures. Given the compressive mechanism of energy transfer, the incidence of ocular injury is high, generally reported to be in the 20–30% range. Ophthalmology consultation is recommended in all pure orbital fractures (▶ Fig. 19.6).

Determination of which internal orbital fractures require treatment is difficult. Absolute indications for orbit repair include acute (immediately evident on initial examination) enophthalmos, hypoglobus, or mechanical restriction of ocular mobility. Contraindications for orbit repair include hyphema, retinal tears, lens displacement, or the fracture has occurred in the only seeing eye. Two main reasons drive repair when enophthalmos, hypoglobus, or entrapment are not grossly evident; they are globe malposition and nonresolving diplopia. Patients readily identify 2 to 4 mm of globe malposition.[23] The volume changes of 1cc equate to approximately 1 mm of globe malposition.[24] Unfortunately, volumetric analysis of orbital volume is difficult to perform. The use of an exophthalmometer assists in quantifying globe malposition. Fractures with greater than half of the width of the orbital floor fractures generally need surgical repair.[25] Most patients have binocular diplopia immediately after injury secondary to orbital edema. Binocular diplopia immediately evident on initial examination is not an indication for repair. Persistent diplopia may result from tissue entrapment, globe displacement, or displacement of the origins of extraocular muscles.

Immediate orbital repair should occur in cases of entrapped tissue within the fracture that cause severe functional limitations or when the patient has a non-resolving oculocardiac reflex. "Trapdoor" fractures are more common in the pediatric population. Periorbital contents, including the inferior rectus, may be displaced into the sinus with an orbital floor that appears grossly intact ("trapdoor fracture"). A non-resolving oculocardiac reflex (bradycardia, heart block, nausea, vomiting) may occur with orbital fractures and should be treated with anticholinergics and emergent surgical exploration and repair.

Most orbital fractures should be repaired within 2 weeks. Fractures with no restriction of ocular motility or a negative forced duction test, but with diplopia and globe malposition may be repaired non-urgently. Delaying repair for seven days permits resolution of periorbital edema, making examination and surgical repair easier. Orbital fractures with no diplopia or none in primary or down gaze, good ocular motility, no globe malposition, and imaging demonstrating a small defect do not need surgical repair. Patients should be followed up and treated in a delayed fashion if signs or symptoms arise.

Prior to repair, patients may be given a short course of dexamethasone (8 mg three times daily for 1 day), instructed to sleep in an inclined position, and instructed to apply cold compresses to the periorbital region to minimize edema. All patients should be placed on sinus precautions (no nose blowing, sneeze with mouth open, avoid Valsalva) with decongestants and expectorants. If intraocular pressures are elevated prior to repair, ophthalmology may medically reduce the intraocular pressure. If the pressure is excessive (> 40, or with acuity changes), then surgical decompression of the orbit should be performed with a lateral canthotomy and inferior cantholysis.

Intraoperatively, bag mask ventilation should be minimized to prevent periorbital emphysema. The globe should be protected with a corneal shield. Multiple orbital approaches exist with multiple advantages and disadvantages of each. The selection of approach is beyond this text. All orbital dissection is limited by the inferior orbital fissure due to invagination of the periosteum into the fissure. Bipolar cauterization of the contents of the inferior orbital fissure may be utilized. This facilitates surgical exposure of the medial and lateral orbital walls. All margins of the fracture should be directly visualized, with the most important being the posterior margin or ledge. Care should be taken to avoid posterior dissection into the superior orbital fissure, optic foramen, or through the anterior or posterior ethmoidal arteries without bipolar cauterization.

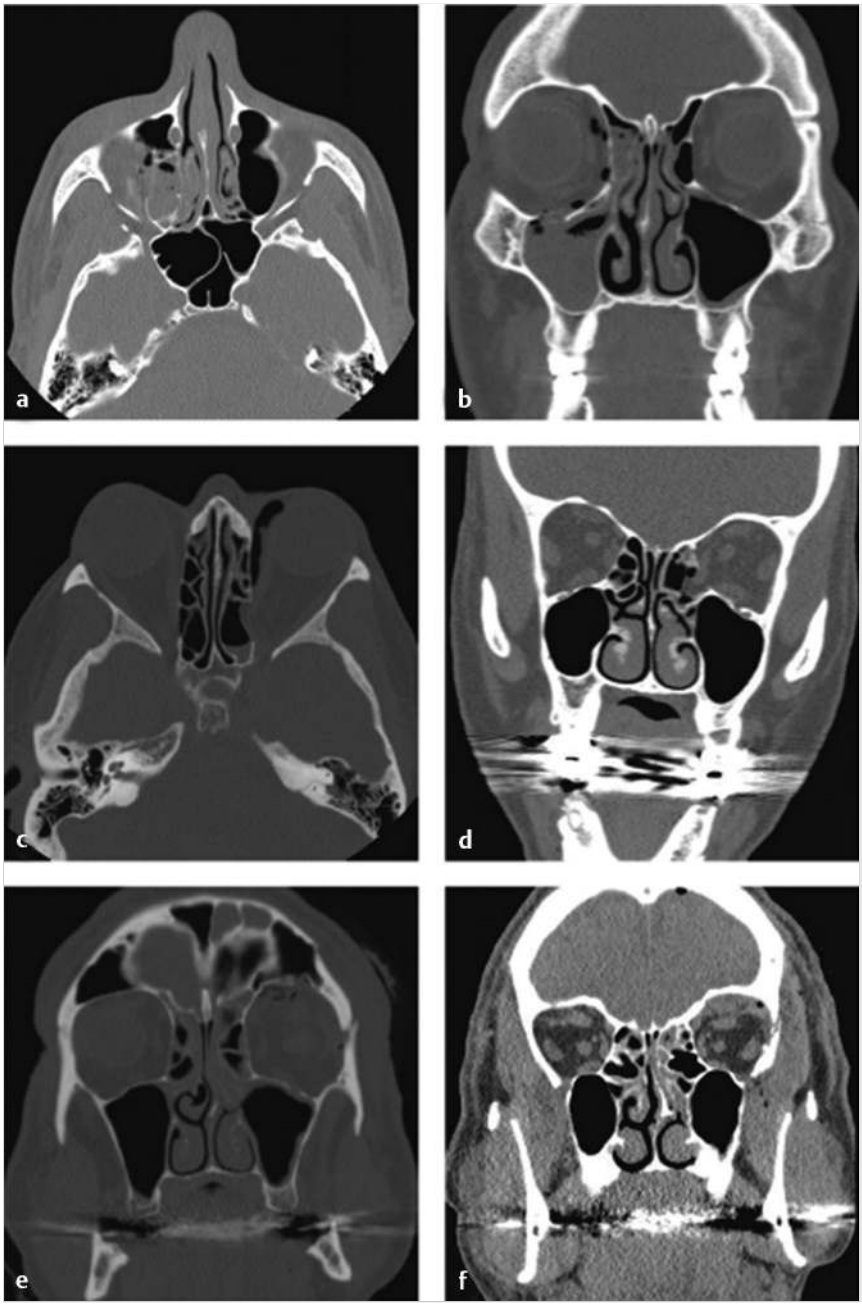


Fig. 19.6 **(a,b)** Orbital floor fracture, Right orbital floor fracture with depression of the lateral orbital floor. The infraorbital canal (V2 branch) is intact infraorbital air and intrusions hemorrhage. (**c,d)** Medial orbital wall fracture. Left posterior medial orbital wall fracture with intraorbital emphysema and fat and mesial rectus herniation into the posterior ethmoid air cells. The mesial rectus is tethered at the anterior margin of the fracture. **(e,f**) Orbital roof fracture. Left orbital roof fracture with extension into aerated frontal sinus. Superior orbital extraconal hematoma and orbital emphysema. (From Baxter AB. Emergency Imaging: A Practical Guide. New York, NY: Thieme; 2016)

Reconstruction of the orbital walls should be performed with a thin, rigid material that provides appropriate resistance against sagging or displacement. Bioresorbable materials are not recommended.[25] The material should be trimmed to cover all margins of the fracture without impingement on vital structures. In cases of multiple orbital wall reconstruction, a preformed metallic implant is of great utility in appropriate contouring of the missing walls. Tension-free placement should be ensured with utilization of a forced duction test. This test should be performed prior to incision, after dissection, after implant placement and stabilization, and after closure. Comparison to the contralateral eye may be of utility if any question of entrapment arises. All orbital reconstruction materials should be stabilized with screws. Intraoperative or postoperative imaging should be obtained to ensure adequate repair if any question of position of the implant arises. The most common mistake is implant placement inferior to the posterior ledge thereby not appropriately reducing orbital contents.

Postoperatively the patient should be instructed to elevate the head, be placed on sinus precautions, and given a short course of steroids and nasal decongestants. A retrobulbar hematoma and orbital compartment syndrome is possible post repair. Any patient with worsening ocular pain, orbital apex syndrome, or superior orbital fissure syndrome should be carefully evaluated for postoperative bleeding. The use of fenestrated mesh reconstruction plates may assist in mitigating the ocular complications of postoperative orbital bleeding. Use of antibiotics is controversial with no convincing evidence that

they prevent postoperative infection. Patients should be instructed to avoid non-pressurized aircraft use and scuba diving for 8 weeks. Commercial aircraft travel may commence after 4 weeks due to pressurization of the cabin. It should be avoided unless absolutely necessary. The patient should be warned that orbital pain due to intraorbital air expansion may occur and that Valsalva maneuvers to equalize middle ear pressure may cause periorbital emphysema.

19.4.6 LeFort/Maxillary fractures

Forces directed through the midfacial skeleton are absorbed and transmitted through vertical and horizontal buttresses, which are composed of areas of denser, thicker bone. The midface is more resistant to vertical forces than horizontal and lateral forces. The vertical buttresses include the nasomaxillary, zygomaticomaxillary, pterygomaxillary, and ethmoid-vomerian (septal). The horizontal buttresses include the superior orbital bar (rims), the inferior orbital rims/zygomatic arches, and the maxillary alveolus.

Rene LeFort described the three "great lines of weakness," now referred to as LeFort 1, 2, and 3 fractures.[26] The LeFort 1 fracture is a transverse fracture extending across the three walls of the maxillary sinus, piriform rims, and the pterygoid plates, often associated with a nasal septum fracture. Pyramidal in shape, the LeFort 2 fracture extends from the maxillary tuberosity superomedially through the inferior orbital rim, lacrimal bones, and transversely across the nasofrontal suture. The LeFort 3 fracture traverses through both zygomatic arches and transversely across the medial and lateral orbits, also including the nasofrontal sutures. The LeFort 3 fracture is a craniofacial disjunction, separating the midfacial skeleton from the cranial base. While useful for classifying fracture patterns, patients rarely have a pure LeFort 1, 2, or 3 fracture, but more often have some combination or component of the LeFort classification (▶ Fig. 19.7).

The goals of operative treatment for fractures of the maxilla are to restore dental function to preinjury occlusion and reconstruct and stabilize the horizontal and vertical buttresses of the face. Cases with minimally displaced fractures without change in occlusion may be treated closed with a soft diet alone. Closed treatment with maxillomandibular fixation alone of displaced fractures is indicated only when there are significant medical comorbidities precluding open repair internal fixation (ORIF). Closed reduction with MMF alone may allow significant midface vertical shortening, as it is difficult to control the vertical position of the maxilla. ORIF is ideally performed within two weeks post fracture; delaying surgery at least seven to ten days is typically advantageous as edema has significantly improved at this point.

Airway management in midfacial fractures requires special consideration. The occlusion provides the most stable guide for operative repair. The final occlusion must be established without interference from the endotracheal tube. In cases of LeFort 1 level fractures, a nasoendotracheal tube may be employed. In cases of LeFort 2 and 3 level fractures, a nasoendotracheal tube may interfere with surgical management of the nasofrontal region. Basilar skull fractures and sphenoid sinus fractures preclude the use of a nasoendotracheal tube due to risk of intracranial intubation. Submental intubation, placement of the tube through interdental spaces in areas of missing teeth, routing the tube behind the dentition, or tracheostomy are all airway management techniques to allow MMF.

Arch bars, or other MMF devices, are placed prior to incision. Surgical access is accomplished through a multitude of surgical approaches. The minimal number of approaches to ensure appropriate reduction depends on the degree of displacement. After surgical exposure, the fractures are mobilized. Rowe disimpaction forceps may assist in mobilizing the fracture. Following fracture mobilization, the patient is placed into MMF. At least three, and most often four, points of fixation are generally recommended for each fracture. The zygomaticomaxillary buttresses and piriform rims are plated in LeFort 1 fractures. The zygomaticomaxillary buttresses, nasofrontal region, and infraorbital rims may be plated in LeFort 2 fractures. The zygomaticofrontal and nasofrontal sutures require fixation in LeFort 3 fractures. After fixation, the patient is released from MMF (▶ Fig. 19.8).

The most common postfixation malocclusion is an anterior open bite. This is the result of inadequate fracture mobilization and reduction. The condyles of the mandible are distracted from the fossa when the patient is placed into MMF if the mobilization is inadequate. When released from MMF, the condyles reseat, reducing posterior mandible vertical dimension, subsequently presenting with an anterior open bite. The surgeon may either remove the fixation, mobilize the fracture, place the patient in MMF and refixate, or perform a LeFort 1 osteotomy. The LeFort 1 osteotomy is reserved for cases in which unusual difficulty was encountered during mobilization and fixation.[27]

Palatal fractures occur in conjunction with approximately 8% of LeFort fractures. Multiple classifications of palatal fractures

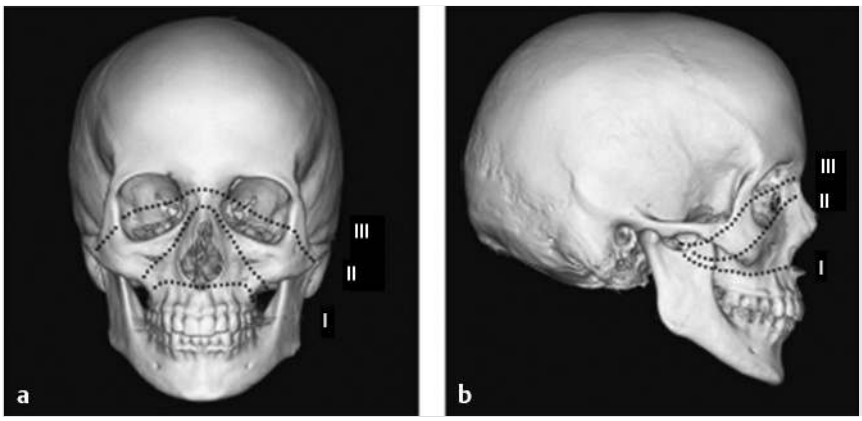


Fig. 19.7 (a,b) LeFort I, II, and III patterns. (From Baxter AB. Emergency Imaging: A Practical Guide. New York, NY: Thieme; 2016)

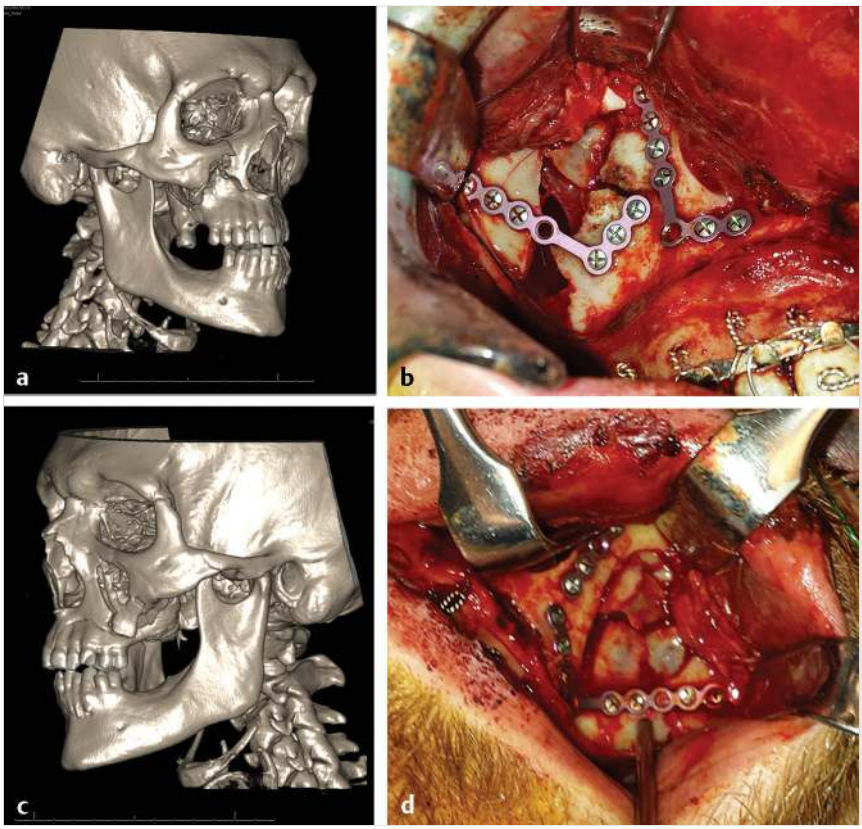


Fig. 19.8 (a–d) A 44-year-old man who sustained a Le Fort 1 level fracture. The patient presented with bilateral V2 paresthesia and an anterior open bite. He underwent open reduction internal fixation with 0.7-mm mini-plates along his bilateral piriform rims and zygomaticomaxillary buttresses.

exist.[28] The discussion of each classification is beyond the scope of this text. Evaluation of the occlusion may be performed preoperatively by obtaining dental impressions and fabricating dental casts. Segmenting the casts along lines of fracture and placing the patient into ideal occlusion with fabrication of a maxillary acrylic splint assists in obtaining proper dental occlusion and restoring the transverse maxillary dimension. The treatment sequencing is similar to the repair of LeFort fractures, with several exceptions regarding MMF and treatment sequencing. Arch bars should be utilized and placed loosely adjacent to fracture lines in the alveolus. The patient is transversely reduced manually or with the assistance of a maxillary splint. The patient is placed into MMF. After placement into MMF, alveolar fractures undergo ORIF, followed by palatal fractures if indicated. The arch bar may be definitively tightened adjacent to the fractures before, during, or after ORIF. The patient should be temporarily released from MMF to ensure adequate stability and reproducibility of the occlusion and dentoalveolar segments.

The majority of patient complaints after ORIF of maxillary fractures relate to inadequate reduction of upper midface fracture lines with subsequent cosmetic deformity, and malocclusion. Careful attention to detail during initial ORIF prevents subsequent need for secondary repair of malocclusion.

19.4.7 Mandible Fractures

The incidence and etiology of mandible fractures vary depending on social, cultural, and environmental factors. Fractures of the mandible are commonly classified by anatomic region (condyle, angle, body, symphysis, coronoid) (▶ Fig. 19.9). Understanding the biomechanics of each region mandible fractures assists in determining the appropriate course of treatment. Common to angle, body, and symphysis fractures are the dentition. Teeth should be extracted from the line of fracture when the tooth prevents reduction of the fracture, has fractured roots, has compromised periodontium (bone loss around tooth), or has an active periapical lesion.[29] The tooth may aid in reduction and stabilization and can be extracted after ORIF. Two general principles guide sequencing of mandible fracture repair: (1) the least displaced fracture should be fixated first and (2) the reduction of tooth-bearing segments should be performed before that of a tooth-free segment.

The pull of the lateral pterygoid muscle usually displaces the condyle anteromedially. Rarely, the condyle may be displaced laterally, or into the middle cranial fossa. Condylar fractures are the most controversial mandible fractures in determining treatment. Four factors should be evaluated when considering opening a condyle fracture: (1) whether there are unilateral or bilateral condylar fractures, (2) whether the patient is edentulous, (3) whether there is adequate bony contact of the fragments, and (4) whether there are other fractures of the mandible. Closed treatment is indicated in unilateral and bilateral cases if there is adequate bony contact between segments and the occlusion is stable without "drop-back," or if the fractures are intracapsular.[30] Open treatment is indicated when there is inadequate bony contact between fracture segments, when re-establishing posterior vertical dimension in panfacial fractures, if the condyle prevents obtaining adequate occlusion, if the condyle is laterally displaced or displaced into the middle cranial fossa, and if there is a medical contraindication to MMF.[31] Treating condyle fractures closed because of lack of

Fig. 19.9 Mandible anatomy and fracture orientation: **(a)** (1) condyle. (2) coronoid process (3) subcondyle or ramus (4) angle (5) body (6) symphysis/parasymphysis (7) alveolus. **(b)** Favorable orientation. **(c)** Unfavorable orientation. (From Baxter AB. Emergency Imaging: A Practical Guide. New York, NY: Thieme; 2016: 580.)

Fig. 19.10 **(a)** A 20-year-old male s/p assault with complex condylar process and coronoid fracture and malocclusion. **(b)** Panorex following endoscopically assisted intra-oral ORIF of condylar process fracture with a strut plate.

open operative experience of the mandibular condyle is inappropriate, and referral or consultation to a provider experienced in open management of condyle fractures is indicated. For closed treatment the patient should generally be placed into three to four weeks of MMF followed by intensive physical therapy to prevent ankylosis or limited maximum incisal opening (▶ Fig. 19.10).

Fractures of the mandibular angle are the most common mandible fracture. The pull of the muscles of mastication creates a line of tension along the superior portion of the fracture line and a line of compression along the inferior portion of the fracture line. Open versus closed treatment is dependent on whether the fracture is favorable (not displaced by the muscles of mastication) and if the angle fracture is isolated. Displaced fractures with inadequate reduction should be fixated. Minimally displaced fractures of the angle should also be considered for fixation given the highly mobile nature of the mandible. Fractures of the angle that are unfavorable should be treated open. Favorable fractures may be treated closed with a 6-week course of MMF. Strong consideration of ORIF of all angle fractures should be considered as there is minimal surgical morbidity associated with open treatment, and it obviates the need for a 6-week course of MMF. An isolated superior border plate utilizing Champy principles is adequate for isolated angle fractures.[32] If there are multiple mandible fractures, then rigid fixation should be utilized with a load-bearing plate, a strut plate with monocortical screws, or a two-plate fixation scheme.[33]

Fractures of the symphysis and body region of the mandible comprise about half of all fractures of the mandible. In the body of the mandible, masticatory forces create tension along the superior border and compression along the inferior border. Moving anteriorly, this pattern transitions into torsion, with no organized zone of compression or tension requiring additional stabilization. Champy describes a neutral zone, below the apices of the teeth and above the mandibular canal, where a single miniplate may be placed for rigid internal fixation.[32] In the symphysis region, two miniplates, one along the inferior border of the mandible and one at least five mm superior to this, are required for fixation. Lag screws may also be utilized in the

symphysis region. Alternatively, a load-bearing reconstruction style plate may be utilized along the inferior border of the mandible in both body and symphysis fractures. Closed reduction may be utilized when the fracture is favorable, there is a stable occlusion, and patient compliance is expected. In cases of severely comminuted body or symphysis fractures, closed treatment may be recommended over open treatment to prevent devitalizing small fragments of bone.

Coronoid process fractures are rare. Management is focused on preventing limitation in mouth opening. Coronoid process fractures do not generally require ORIF. Usually, conservative approaches are utilized with passive range of motion exercises and a soft diet initially, followed by active exercises.[34]

Two populations deserve special discussion: the atrophic edentulous patient and the pediatric patient. Atrophic mandible fractures traditionally were treated closed with circummandibular wiring or Gunning splint placement prior to the advent of rigid internal fixation. Patients should be treated closed if medical comorbidities preclude open treatment. Open reduction internal fixation should be approached via an extraoral approach due to decreased risk of infection and nonunion compared to transoral approaches.[35] Load-bearing reconstruction plates with bicortical screws should be utilized. Miniplate placement may assist in temporary reduction of the fracture prior to placement of the reconstruction plate. In the severely atrophic mandible, primary bone grafting may be indicated. Post-operative paresthesia should be discussed prior to surgery. The morbidity and risk of a non-union outweighs the morbidity of paresthesia for most surgeons.

Treatment of the pediatric patient in the primary (age 6 or less) or mixed dentition (age 6–12) varies considerably compared to the adult patient. Although most adult patients undergo open treatment, most children should undergo closed treatment. The goals of pediatric mandible fracture treatment are to obtain a bony union, normalize the occlusion, and avoid growth disturbances.[36] Growth disturbance avoidance is paramount in evaluation of the pediatric patient. Minor malocclusions will usually self-correct if in the primary or mixed dentition. Avoidance of injury to the developing tooth buds generally prevents the use of rigid internal fixation and IMF screws in the body and symphysis regions. MMF with Risdon cables, Ivy loops, or standard Erich arch bars should be utilized. Significant difficulty is encountered with placement of these tooth-borne MMF devices. If unable to obtain satisfactory MMF device placement, a lingual splint may be fabricated after obtaining dental impressions. Two weeks of MMF immobilization is sufficient in the very young, and three to four weeks will suffice for most fractures treated closed in the pediatric patient. Condyle fractures should be treated for significantly less time than the adult patient, generally 2 weeks or less.[37] The risk of bony ankylosis of the TMJ is highest in the pediatric population.

19.4.8 Panfacial Fractures

Panfacial fractures are defined as fractures involving the lower, middle, and upper face. These fractures result from high-energy mechanisms resulting in multi-vector injury. Reconstruction of panfacial fractures requires re-establishment of the horizontal and vertical bony buttresses in all three planes of space.

The most challenging aspect of panfacial trauma repair involves treatment sequencing.[38] Re-establishment of an appropriately dimensioned and oriented occlusal unit provides the framework for panfacial reconstruction.[39] Re-establishment of the occlusion with appropriate width is essential. In cases of maxilla or mandibular widening (mid-palatal fracture, symphysis fracture), consultation with an oral and maxillofacial surgeon for fabrication of occlusal splints may be of great utility in establishment of the buttresses of the face (▶ Fig. 19.11).

Once the occlusal unit has been established, the patient may be placed in MMF. Two general approaches are utilized: (1) the bottom up approach addressing the mandible first or (2) the top-down approach addressing the frontal sinus, central and lateral midface first. Both approaches have advantages. Common to both are the necessity to fixate from "known to unknown." In both approaches, insufficient fracture reduction and stabilization will lead to error in establishment of the buttresses of the face. This error is magnified as subsequent fixation is completed.

With the bottom up approach, the vertical height of the mandibular ramus should be established. In cases of bilateral condylar fractures, at least one condyle should be reduced and

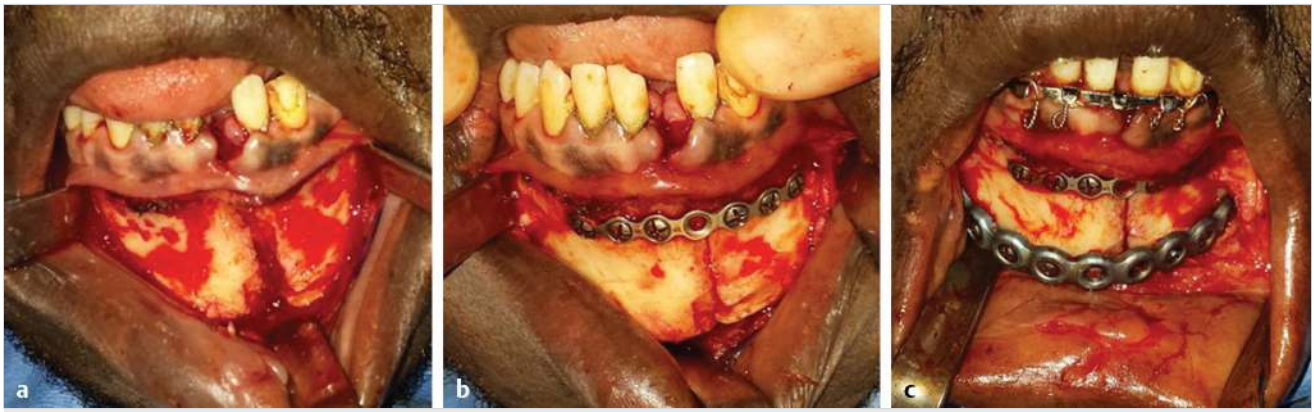


Fig. 19.11 (a-c) A 20-year-old male ejected during MVC sustaining panface fracture with bilateral condyle and left parasymphysis fracture. Given his loss of vertical and horizontal facial buttress, he was treated with a bottom up approach. Care was taken during ORIF of his parasymphysis fracture to narrow the mandible at the angles to prevent facial widening. Tooth no. 23 was avulsed during the injury which was clearly evident during ORIF. A superior tension band plate was placed first, followed by a load bearing reconstruction plate.

fixated.[38,40] Any body or ramus fractures should be reduced and fixated, followed by re-establishment of the vertical ramus height with ORIF of the condyle fracture. Mandible fractures should be addressed with careful attention to avoid mandibular widening. After mandible fixation, attention is then turned to the midface.

With the top-down approach, the patient is placed into MMF. Evaluation of comminution and displacement of each fracture subunit should be performed preoperatively. Reduction starts at a point of minimal displacement and comminution, either laterally at the zygomas or centrally in the NOE region. When able, establishing a pillar of vertical height (i.e., frontal bone → NOE → piriform aperture → maxillary dentition) that is solid allows the surgeon to establish a reference from which to frame the remainder of the midface. This may also be accomplished laterally (FZ → ZMB → dentition). In a similar fashion, the horizontal framework of the face is established from the frontal bar or zygomas. As previously discussed, to prevent significant facial widening care should be taken to appropriately reduce NOE fractures, and evaluate lateral displacement of the medial rim when plating the infraorbital rim. After ORIF of the midface, the mandible is then reduced and fixated.

Panfacial trauma results rely heavily on preoperative planning and sequencing. Secondary revision and reconstruction is difficult.[41] In cases of severely comminuted or displaced fractures, staged repair may be beneficial. On several occasions, the author has completed re-establishing the occlusal unit and ORIF of either the mid and upper face or mandible utilizing a second surgery to complete the panfacial repair.

19.5 Postoperative Care

Postoperative care of the maxillofacial trauma patient depends on the site of injury. Wound care, nutrition, and other supportive care are necessary to minimize postoperative scarring and ensure wound healing. Lack of adherence to sinus precautions and inadequate mandibular range of motion exercises are two common areas of non-compliance that should be evaluated on each postoperative visit. Normal maximum incisal opening (measure from maxillary central incisal edge to mandibular incisal edge) should be greater than 45 mm in adults.

19.6 Outcomes

Facial fractures occur due to significant blunt force trauma. As such, notable soft-tissue contusion or lacerations occur concomitant to the fractures. Overall healing is dependent on both the soft- and hard-tissue components. In general, several weeks are usually necessary for soft-tissue edema and induration to fully recede. Bone healing with rigid fixation occurs in the standard manner such that full healing is present within a few months as well. Persistent pain at the fracture sites is normal, but should diminish with time. However, pain and discomfort may persist for a prolonged period. Increasing pain or visible irregularity raise concern for nonunion or malunion. Specifically, for mandible fractures, increasing pain, trismus and malocclusion should be considered failure to heal until proven otherwise. Nevertheless, for the vast majority of patients, healing occurs with favorable results and infrequent need for hardware removal.

19.7 Review Questions

19.7.1 Fill in the Correct Answer

1. In treating a symphysis and unilateral condyle fracture, which fracture is reduced and fixated first?
2. What is the distance from the anterior lacrimal crest to the anterior ethmoidal artery and optic canal?
3. When evaluating the zygomaticomaxillary complex fracture, which area of fracture allows best determination of reduction?
4. When should the nasofrontal duct be surgically obstructed?
5. What is the most common cause of an anterior open bite after MMF release after ORIF of a LeFort level fracture?

19.7.2 Answers

1. Tooth-bearing fracture followed by non–tooth-bearing fracture.
2. 24 mm to the anterior ethmoidal artery and 42 mm to the optic canal.
3. Zygomaticosphenoid suture.
4. When the patient is cranialized or undergoes frontal sinus obliteration.
5. Inadequate midface mobilization with inferior distraction of the condyles when the patient was placed into MMF.

References

[1] Green JG, Wood JM, Davis LF. Asystole after inadvertent intubation of the orbit. J Oral Maxillofac Surg. 55(8):856–859

[2] Jundt JS, Cattano D, Hagberg CA, Wilson JW. Submental intubation: a literature review. Int J Oral Maxillofac Surg. 41(1):46–54

[3] Metzinger SE, Guerra AB, Garcia RE. Frontal sinus fractures: management guidelines. Facial Plast Surg. 21(3):199–206

[4] Bell RB, Dierks EJ, Brar P, Potter JK, Potter BE. A protocol for the management of frontal sinus fractures emphasizing sinus preservation. J Oral Maxillofac Surg. 65(5):825–839

[5] Hardy JM, Montgomery WW. Osteoplastic frontal sinusotomy: an analysis of 250 operations. Ann Otol Rhinol Laryngol. 85(4, Pt 1):523–532

[6] Tiwari P, Higuera S, Thornton J, Hollier LH. The management of frontal sinus fractures. J Oral Maxillofac Surg. 63(9):1354–1360

[7] Metzinger SE, Metzinger RC. Complications of frontal sinus fractures. Craniomaxillofac Trauma Reconstr. 2(1):27–34

[8] Smith TL, Han JK, Loehrl TA, Rhee JS. Endoscopic management of the frontal recess in frontal sinus fractures: a shift in the paradigm? Laryngoscope. 112 (5):784–790

[9] Markowitz BL, Manson PN, Sargent L, et al. Management of the medial canthal tendon in nasoethmoid orbital fractures: the importance of the central fragment in classification and treatment. Plast Reconstr Surg. 87(5):843–853

[10] Ellis E , III. Sequencing treatment for naso-orbito-ethmoid fractures. J Oral Maxillofac Surg. 51(5):543–558

[11] Vora NM, Fedok FG. Management of the central nasal support complex in naso-orbital ethmoid fractures. Facial Plast Surg. 16(2):181–191

[12] Nguyen M, Koshy JC, Hollier LH , Jr. Pearls of nasoorbitoethmoid trauma management. Semin Plast Surg. 24(4):383–388

[13] Becelli R, Renzi G, Mannino G, Cerulli G, Iannetti G. Posttraumatic obstruction of lacrimal pathways: a retrospective analysis of 58 consecutive naso-orbitoethmoid fractures. J Craniofac Surg. 15(1):29–33

[14] Atighechi S, Karimi G. Serial nasal bone reduction: a new approach to the management of nasal bone fracture. J Craniofac Surg. 20(1):49–52

[15] Desrosiers AE , III, Thaller SR. Pediatric nasal fractures: evaluation and management. J Craniofac Surg. 22(4):1327–1329
[16] Manson PN, Markowitz B, Mirvis S, Dunham M, Yaremchuk M. Toward CT-based facial fracture treatment. Plast Reconstr Surg. 85(2):202–212, discussion 213–214
[17] Zingg M, Laedrach K, Chen J, et al. Classification and treatment of zygomatic fractures: a review of 1,025 cases. J Oral Maxillofac Surg. 50(8):778–790
[18] Covington DS, Wainwright DJ, Teichgraeber JF, Parks DH. Changing patterns in the epidemiology and treatment of zygoma fractures: 10-year review. J Trauma. 37(2):243–248
[19] Shumrick KA, Kersten RC, Kulwin DR, Smith CP. Criteria for selective management of the orbital rim and floor in zygomatic complex and midface fractures. Arch Otolaryngol Head Neck Surg. 123(4):378–384
[20] Ellis E , III, Kittidumkerng W. Analysis of treatment for isolated zygomaticomaxillary complex fractures. J Oral Maxillofac Surg. 54(4):386–400, discussion 400–401
[21] Milora M. Peterson's Principles of Oral and Maxillofacial Surgery. 2nd ed. Hamilton, Ontario: BC Decker, Inc; 2004:465 (Table 24–2)
[22] Ellis E , III. Orbital trauma. Oral Maxillofac Surg Clin North Am. 24(4):629–648
[23] Koo L, Hatton MP, Rubin PA. When is enophthalmos "significant"? Ophthal Plast Reconstr Surg. 22(4):274–277
[24] Raskin EM, Millman AL, Lubkin V, della Rocca RC, Lisman RD, Maher EA. Prediction of late enophthalmos by volumetric analysis of orbital fractures. Ophthal Plast Reconstr Surg. 14(1):19–26
[25] Baumann A, Burggasser G, Gauss N, Ewers R. Orbital floor reconstruction with an alloplastic resorbable polydioxanone sheet. Int J Oral Maxillofac Surg. 31 (4):367–373
[26] LeFort R. Experimental study of fractures of the upper jaw parts I and II. Rev Chir Paris. 23:208
[27] Scolozzi P, Imholz B. Completion of nonreducible Le Fort fractures by Le Fort I osteotomy: sometimes an inevitable choice to avoid postoperative malocclusion. J Craniofac Surg. 26(1):e59–e61
[28] Hendrickson M, Clark N, Manson PN, et al. Palatal fractures: classification, patterns, and treatment with rigid internal fixation. Plast Reconstr Surg. 101 (2):319–332
[29] Freitag V, Landau H. Healing of dentate or edentulous mandibular fractures treated with rigid or semirigid plate fixation–an experimental study in dogs. J Craniomaxillofac Surg. 24(2):83–87
[30] Ellis E , III. Method to determine when open treatment of condylar process fractures is not necessary. J Oral Maxillofac Surg. 67(8):1685–1690
[31] Al-Moraissi EA, Ellis E , III. Surgical treatment of adult mandibular condylar fractures provides better outcomes than closed treatment: a systematic review and meta-analysis. J Oral Maxillofac Surg. 73(3):482–493
[32] Champy M, Loddé JP, Schmitt R, Jaeger JH, Muster D. Mandibular osteosynthesis by miniature screwed plates via a buccal approach. J Maxillofac Surg. 6(1):14–21
[33] Al-Moraissi EA, Ellis E , III. What method for management of unilateral mandibular angle fractures has the lowest rate of postoperative complications? A systematic review and meta-analysis. J Oral Maxillofac Surg. 72(11):2197–2211
[34] Shen L, Li J, Li P, Long J, Tian W, Tang W. Mandibular coronoid fractures: treatment options. Int J Oral Maxillofac Surg. 42(6):721–726
[35] Toma VS, Mathog RH, Toma RS, Meleca RJ. Transoral versus extraoral reduction of mandible fractures: a comparison of complication rates and other factors. Otolaryngol Head Neck Surg. 128(2):215–219
[36] Myall RW. Management of mandibular fractures in children. Oral Maxillofac Surg Clin North Am. 21(2):197–201, vi
[37] Chrcanovic BR. Open versus closed reduction: mandibular condylar fractures in children. Oral Maxillofac Surg. 16(3):245–255
[38] Fritz M, Koltai P. Sequencing and organization of the repair of panfacial fractures. Oper Tech Otolaryngol–Head Neck Surg. 13:261–264
[39] Curtis W, Horswell BB. Panfacial fractures: an approach to management. Oral Maxillofac Surg Clin North Am. 25(4):649–660
[40] Tullio A, Sesenna E. Role of surgical reduction of condylar fractures in the management of panfacial fractures. Br J Oral Maxillofac Surg. 38(5):472–476
[41] Khader R, Wallender A, Van Sickels JE, Cunningham LL. Secondary reconstruction of panfacial fractures. Oral Maxillofac Surg. 18(1):99–109

20 Rhinoplasty

Geo N. Tabbal and Alan Matarasso

Abstract

"Rhinoplasty" covers the spectrum of issues related to the aesthetic and functional components of nose plastic surgery. Factors that influence overall outcome—including patient goals, proper patient selection, psychological fit, etc.—are discussed, as well as up-to-date approaches and techniques used (e.g., software imaging systems). Salient considerations during the presurgical physical examination are listed (e.g., physical deformities and/or abnormalities). Preoperative preparation is stressed, and preoperative considerations are reviewed. A systematic method of facial analysis—involving the frontal, lateral, and basal views—is presented in table format and in the text. Surgical techniques, including the critical area of incisions, are identified, discussed, and compared. Guidelines for postoperative care, complications, airway obstructions, deformities, and outcomes/patient satisfaction conclude the discussion.

Keywords: closed rhinoplasty, headlight, speculum, vasoconstriction, seasonal obstruction, rhinitis medicamentosus, hump reduction, osteotomy, septal perforation

20.1 Goals and Objectives

After reading this article, the participant should be able to

- Discuss essential aesthetic, functional, and other components of the preoperative assessment of patients seeking rhinoplasty.
- Identify factors that can influence overall outcome so that successful patient selection is performed in those seeking nasal surgery.
- Understand the common approaches and techniques used in rhinoplasty as well as their advantages and disadvantages.
- Understand common rhinoplasty complications and be able to identify the principles and methods used in the management and corrective treatment of these sequelae.

20.2 Patient Presentation

Patients seeking rhinoplasty may present in a variety of ways. Regardless of the variability in which patients seek consultation, the initial preoperative assessment is similar. The following methodological approach allows the surgeon to critically understand why the patients are dissatisfied and what their levels of expectation are for surgery. This allows the surgeon to evaluate whether expectations can be met while also allowing the physician to accurately assess and comprehend the patient's psychological fit for the intended procedure. The goals for surgery should be clearly stated by the patient and easily understood by the surgeon. The single most important determinant of procedural success in rhinoplasty is proper patient selection. It ensures that consistent, reproducible outcomes are achieved while maximizing patient satisfaction.[1]

Standardized two-dimensional photographs, including the frontal, oblique, lateral, and basal views, must be obtained in every consultation as they are an important component of the medical record, are instrumental in preoperative planning, and aid in the critical evaluation of the postoperative results.[2,3,4] Additionally, with the evolution and implementation of software imaging systems commonly employed among practicing rhinoplasty surgeons, this can be helpful for some patients to improve communication between surgeons and their patients.[2,5] Most recently, advancements in computer imaging, specifically 3D modeling, can assist in unifying the intended postoperative goals of the surgeon with the intended vision of the patient.[6,7]

Careful assessment of the psychiatric state of each patient should be performed, as it is instrumental in proper patient selection. This includes evaluating the emotional stability and motivating factors in patients seeking surgery. In a report by Picavet et al, moderate to severe body dysmorphic syndromes were reported in 33% of the overall rhinoplasty population, while this number increased to 43% in patients seeking aesthetic rhinoplasty (level of evidence: risk, III).[8] A significant correlation was found between the prevalence of body dysmorphic disorder in patients who sought aesthetic consultation, in patients who had undergone previous rhinoplasty procedures, or in patients who had an existing psychiatric history.

Obtaining a thorough nasal history is an important component of the patient evaluation. In patients who present with aberrant nasal physiology, identifying the laterality, duration, and time of onset of such symptoms should be noted. Careful inquiry into any mitigating factors that contribute to worsening or alleviation of these symptoms, the presence of headaches, visual disturbances, other otologic symptoms, and/or seasonal allergies should be recorded. Other aspects of the nasal history, including any history of trauma, previous surgery, medication use (with particular attention to vasoactive nasal sprays), and/or use of tobacco, alcohol, or drugs should be documented.

Physical examination of the patient plays a critical role during the consultation, with particular attention to the nasal anatomy (▶ Fig. 20.1). Careful identification is made of any physical deformities and/or abnormalities of the external and internal nasal valves, the nasal septum, and inferior turbinates. Completion of the physical assessment can be performed with the aid of a headlight, speculum, and vasoconstriction, if needed. The presence of uncharacteristic masses found on intranasal exam or those with obstruction of unidentifiable source dictates further workup by rhinomanometry or sinus computed tomography.[9,10,11] External inspection should note the presence of inspiratory collapse of the external nasal valve(s) or subjective improvement in inspiratory breathing upon application of the Cottle maneuver (where the examiner places gentle lateral traction on the cheek to simulate improved suspected airway obstruction), either of which should raise the suspicion for nasal airway obstruction.[12]

Successful treatment of rhinoplasty patients demands a keen understanding of nasal physiology. Approximately 50% of overall airway resistance stems from the nasal airway which builds up negative pressure during inspiration, causing the nostrils to enlarge, the upper lateral cartilages (ULCs) to move medially toward the septum, the internal nasal valve to narrow, and the tip to plunge. A variety of factors, however, can influence this process and result in various degrees of obstruction with two

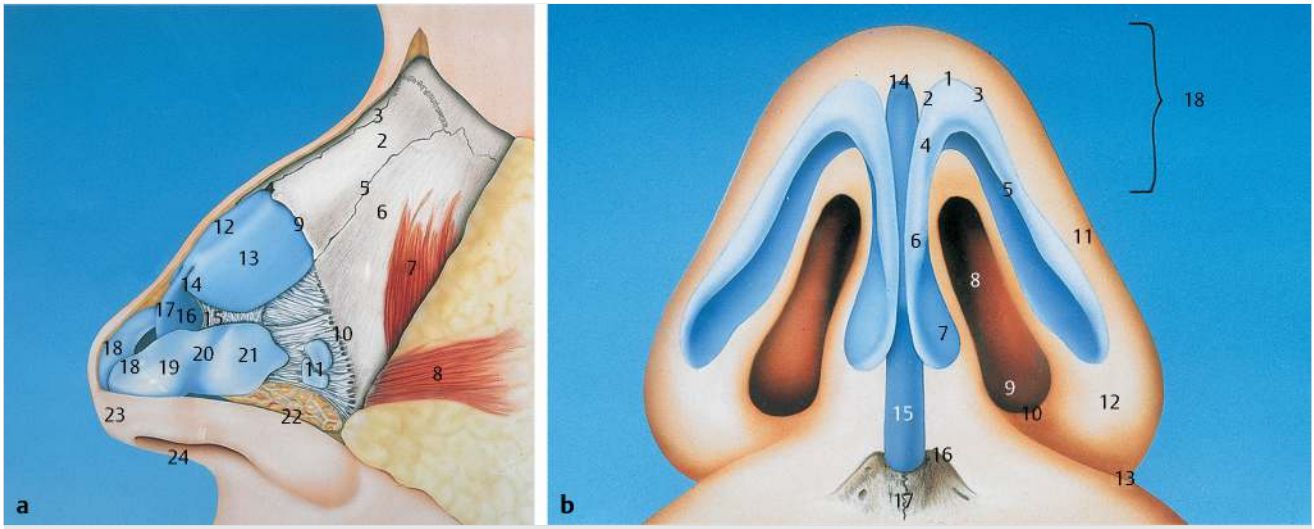


Fig. 20.1 Anatomical landmarks and standard terminology. **(a)** 1. Nasofrontal suture line, 2. Nasal bone, 3. Internasal suture line, 4. Osseocartilaginous junction (rhinion), 5. Nasomaxillary suture line, 6. Ascending process of maxilla, 7. Levator labii superioris muscle, 8. Transverse nasalis muscle, 9. Cephalic portion of upper lateral cartilage (articulates with undersurface of nasal bone), 10. Pyriform margin, 11. Sesamoid cartilages, 12. Cartilaginous dorsum, 13. Upper lateral cartilage, 14. Caudal free margin of upper lateral cartilage, 15. Intercartilaginous tissue condensation, 16. Quadrangular cartilage, 17. Anterior septal angle, 18. Tip-defining point alar cartilage, 19. Lateral crus of alar cartilage, 20. Concavity (hinge) of lateral crus, 21. Lateral aspect of lateral crus, 22. Alar lobule, 23. Infratip lobule, 24. Columella. **(b)** 1. Apex of alar cartilage, 2. Medial angle of dome, 3. Lateral angle of dome, 4. Alar cartilage transitional segment (intermediate crus), 5. Lateral crus alar cartilage, 6. Medial crus alar cartilage, 7. Medial crural footplate, 8. Nostril aperture, 9. Nostril floor, 10. Nostril sill, 11. Lateral alar sidewall, 12. Alar lobule, 13. Alarfacial junction, 14. Anterior septal angle, 15. Caudal septum, 16. Maxillary crest 17. Nasal spine, 18. Infratip lobule. (Used with permission from Behrbohm H, Tardy E, Essentials of Septorhinoplasty. New York, NY: Thieme; 2004)

broad categories including those secondary to an anatomic etiology or those that result from a physiologic response. Patients who complain of constant obstructions are more likely to suffer from a fixed anatomical etiology and are often unilateral while those who have bilateral obstruction, which varies in severity, often suffer from mucosal disease.

Seasonal obstruction relates to obstruction from pollen, dust, mold, or other allergens and should raise suspicion for allergic rhinitis in patients with such history. Physical examination may be notable for "allergic shiners" which appear as dark circles under the eye. Often, these patients have a history of past or ongoing medication use, which must be documented prior to surgical correction. In patients who do not present with clear findings, trial medication management for nasal airway obstruction can help clarify the clinical presentation.

Rhinitis medicamentosus refers to the rebound engorgement and vasodilation of nasal mucosa secondary to the excessive use of topical nasal decongestants including Afrin (Bayer, Pittsburg, PA) or Neo-Synephrine (Hospira Inc, Lake Forest, IL). When diagnosed, treatment consists of patient education, stopping the offending agent, use of combination of oral antihistamines and decongestants, topical nasal steroid sprays, and potentially, a steroid taper. Asthmatics often have a crease in the skin just cephalic to the dome area secondary to long-standing forces that elevate the tip upward.

20.3 Preparation for Surgery

Nasal surgery is considered a clean contaminated field due to intranasal colonization by commonly identified organisms including *Staphylococcus epidermidis*, *Staphylococcus aureus*, and *Staphylococcus viridans*. Commonly, first- or second-generation cephalosporins are used for perioperative antibiotic prophylaxis which should be given 30 minutes prior to surgical incision.[13,14,15] However, the available literature includes several studies that have demonstrated no difference in infection rates with the use of postoperative prophylactic antibiotics.

Currently, there is no high-level, evidence-based research which finds benefit in preoperative screening and treatment of those colonized with methicillin-sensitive *S. aureus*. In one review by Nicholas et al, only a history of methicillin-resistant *S. aureus* (MRSA) was found to significantly correlate with colonization at the time they presented for outpatient rhinoplasty procedures.[16] Other presumed risk factors including occupational health workers, recent hospitalization, and recent antibiotic use were not found to be lone significant risk factors. Difficulty treating MRSA infections following septorhinoplasty does seem to warrant careful identification of patients at risk for MRSA colonization with swab and culture, while identification of at-risk patients who present with suspected surgical site infection is prudent so that empiric treatment may be initiated.

Paramount to successful rhinoplasty is a thorough preoperative preparation, which helps to provide consistent, reproducible results that minimize undue sequelae. Comprehensive analysis includes review of nasal history, anatomic examination of both internal and external nasal structures, identification of the patient's deformities and their expectations for the surgical outcome are critical factors. Most importantly, the treating surgeon must perform a thorough nasal analysis to establish a prudent surgical plan of correction. Skin type, thickness, and quality are noted, as thicker, more sebaceous skin tends to camouflage changes to the osseocartilaginous framework

necessitating greater and more aggressive modification of the osseocartilaginous framework. Thin skin, on the other hand, may allow for identification of even slight change and reveal the presence of minor irregularities that would have otherwise been obscured. The nose is examined within the context of the entire face, including the chin and neck, helping to ensure that facial harmony and balance are achieved while preexisting asymmetries are identified and communicated to the patient.

A systematic method of facial analysis must then be performed. A variety of orderly approaches exist which allow for through analysis which include using the frontal, lateral, and basal views to determine key findings (Box 20.1).

Box 20.1 Systematic Nasal Analysis

- Frontal View
  - Facial proportions
  - Skin type/quality—Fitzpatrick type, thin or thick, sebaceous
  - Symmetry and nasal deviation—midline, C-, reverse-C-, S- or S-shaped deviation
  - Bony vault—narrow or wide, asymmetrical, short or long nasal bones
  - Midvault—narrow or wide, collapse, inverted-V deformity
  - Dorsal aesthetic lines—straight, symmetrical or asymmetrical, well or ill defined, narrow or wide
  - Nasal tip—ideal/bulbous/boxy/pinched, supratip, tip-defining points, infratip lobule
  - Alar rims—gull shaped, facets, notching, retraction
  - Alar base—width
  - Upper lip—long or short, dynamic depressor septi nasi muscles, upper lip crease
- Lateral View
  - Nasofrontal angle—acute or obtuse, high or low radix
  - Nasal length—long or short
  - Dorsum—smooth, hump, scooped out
  - Supratip—break, fullness, pollybeak
  - Tip projection—over- or underprojected
  - Tip rotation—over- or underrotated
  - Alar-columellar relationship—hanging or retracted alae, hanging or retracted columella
  - Periapical hypoplasia—maxillary or soft tissue deficiency
  - Lip-chin relationship—normal, deficient
- Basal View
  - Nasal projection—over- or underprojected, columellar-lobular ratio
  - Caudal septal deviation
  - Nostril—symmetrical or asymmetrical, long or short
  - Columella—septal tilt, flaring of medial crura
  - Alar base—width
  - Alar flaring

From Rohrich R, Adams W, Ahmad J et al., ed. Dallas Rhinoplasty. Nasal Surgery by the Masters.. 3rd Edition. Thieme; 2014.

20.3.1 Frontal View

Facial proportions—three horizontal lines, tangent to the level of the hairline, brow, nasal base and chin (menton)—are used to divide the face into thirds. The lower third is further subdivided into thirds by placing a horizontal line at the level of the oral commissures (stomion). Concern for a craniofacial anomaly, such as vertical maxillary excess or maxillary hypoplasia, should be raised when deviation is identified.

Symmetry and nasal deviation are examined by using an imaginary vertical line from the midglabellar to the menton to determine if the nose is straight or crooked. Deviation from the midline is suggestive of either bone and/or cartilaginous origin. Cartilaginous deviation is indicative of underlying deviation of the septum that can be classified as midline, C-, reverse C-, S or S-shaped.

Analysis of the dorsal aesthetic lines should be performed. These curvilinear lines should appear as two slightly curved lines which originate at the supraorbital ridges, converge at the level of the medial canthus, and then diverge slightly as they extend to the tip defining points. They should be characterized based on their symmetry, width, and degree of definition.

The cartilaginous and bony vault evaluation should include the length of the nasal bones, their shape, and width. Ideally, the bone base should be approximately 75 to 80% of the alar base width and, when excessive, may warrant performing osteotomies, although over-narrowing should be avoided, especially in males as this may have a feminizing effect. The mid-vault should be assessed for the presence of an inverted-V deformity (where an inverted V-shape depression is palpated between the ends of the nasal bones and the start of the upper lateral cartilage), contour irregularities, and possible nasal wall collapse.

The width of the alar base is observed and should be equivalent to the intercanthal distance. When in excess, the degree of nasal flaring should then be ascertained such that greater than 1 to 2 mL difference between the maximum alar width and the alar base is maintained, suggesting the etiology is alar flaring, often corrected by alar base resection. If less than 1 to 2 mm difference is found, the underlying cause is intrinsic to a primarily excessively wide alar base. The alar rims are examined for the presence of retraction, notching, symmetry, and overall shape. The outline of the rims and columella should maintain a slight inferolateral flare resembling a gull in flight. Angles more steep in quality suggest increased infratip lobular height.

The nasal tip is analyzed by drawing two opposing triangles that connect the two tip-defining points, the supratip point and the columellar-lobular angle point. By connecting these four points with lines drawn within this diamond, the two triangles that are formed should appear equilateral. Further analysis of the tip should characterize the tip as bulbous or boxy and identify the possibility of alar malposition.

20.3.2 Lateral View

The lip–chin relationship is analyzed by drawing a one-half the ideal nasal length tangent to the vermillion of the upper lip. In the resting position, the lower lip should lie approximately 2 mm posterior to the upper lip. The ideal chin position in men should have the chin at the level of the lower lip, while in females, the chin should be slightly posterior. Deviation from this should raise the suspicion that orthodontics, a chin implant, or orthognathic surgery might be necessary (▶ Fig. 20.2).

On profile view, analysis of the nose should include the dorsum and tip. The radix should be ideally placed at a level between the upper lash line and the supratarsal fold. The slope

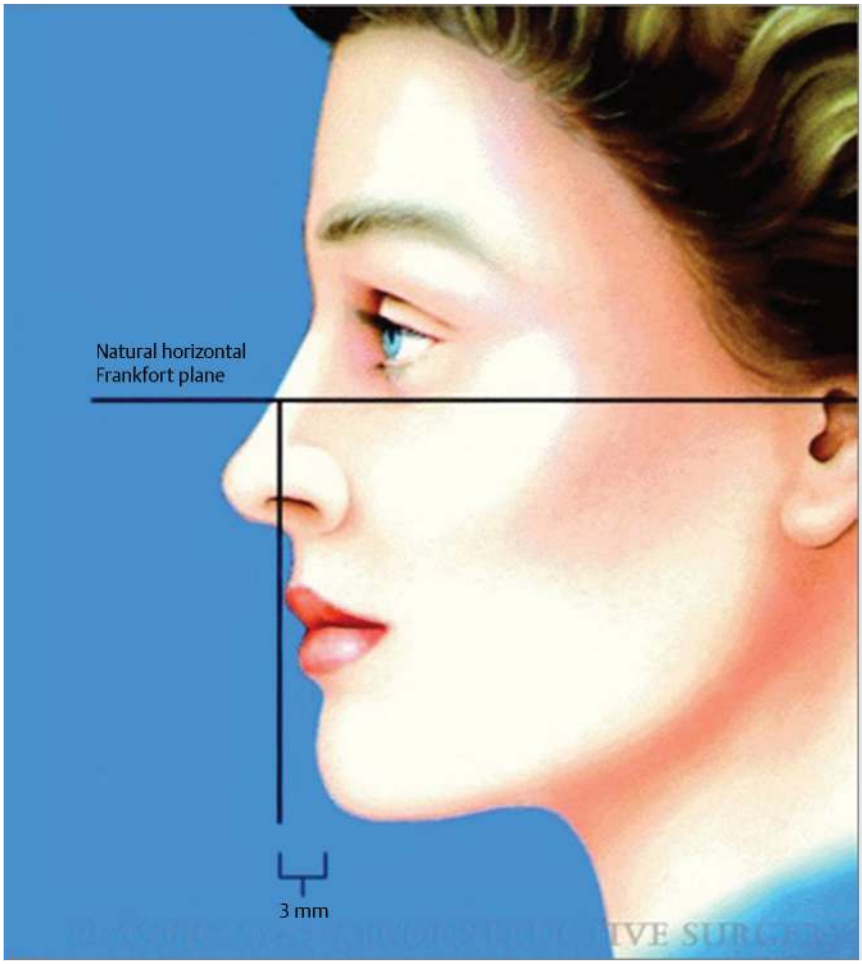


Fig. 20.2 Evaluation of nose-lip-chin balance relative to the natural horizontal Frankfort plane. From Rohrich R, Adams W, Ahmad J et al., ed. Dallas Rhinoplasty. Nasal Surgery by the Masters. 3rd Edition. Thieme; 2014.

of the dorsum, identified by drawing a line from the radix to the tip should be straight in males while a slight concavity approximately 2 mm inferior to this line with a supratip break is considered ideal. Excessive quadrangle cartilage with inferior descent of the nasal tip cartilages is defined as a tension nose.

Tip projection is defined as the distance from the alar cheek junction to the nasal tip and should equal width of the alar base as well as two-thirds of the distance from radix to the tip. An additional method of analysis involves drawing a vertical line tangent through the most projecting portion of the upper lip vermillion. Fifty to 60% of the tip should lie anterior to this line (▶ Fig. 20.3).

The degree of tip rotation is assessed by calculating the nasolabial angle. This is the angle formed between a line connecting the anterior and posterior portions of the nostril and plumb line dropped perpendicular to the natural horizontal facial plane. Men should have angles between 90 and 95 degrees, and women between 95 and 100 degrees (▶ Fig. 20.4). Commonly confused with tip rotation is the columellar–labial angle which is the angle formed by the junction of the columella with the upper lip. Ideally 30 to 45 degrees, fullness in this region usually relates to excessive caudal septum.

20.3.3 Basal View

Examination of the nose from the basal view should demonstrate two equilateral triangles each formed by the alar rims and nasal tip with a 1:2 relationship of the nasal tip to the columella. The orientation of the nostril should have a slight medial direction of the long axis and an overall teardrop geometry (▶ Fig. 20.5).

20.4 Treatment

Surgery may be performed under either local anesthesia with IV sedation or general anesthesia with the use of either laryngeal mask or endotracheal intubation. Upon induction, the patient is prepped for surgery by first trimming the nasal hairs and swabbing the nostrils with Betadine. For an open rhinoplasty, the planned columellar incision is then marked with an indelible marker if using the open approach. Next, the soft-tissue envelope of the nose, the septum, and nasal mucosa are infiltrated with 1% lidocaine with 1:100,000 epinephrine. Additional infiltrate is injected into the inferior turbinates if their manipulation is anticipated. Cotton pledgets soaked in either 4% cocaine or oxymetazoline are then inserted into each nare to help shrink the nasal mucosa, facilitate exposure, and minimize blood loss.

The debate of whether an open versus closed (endonasal) approach to rhinoplasty has been long-standing with firm supporters of each method arguing the merits of why one is better than the other. Regardless of the generic advantages and disadvantages of each method, rhinoplasty should ideally be

Fig. 20.3 Proper tip projection on profile view should be measured as 0.67 times the ideal nasal length. From Rohrich R, Adams W, Ahmad J et al., ed. Dallas Rhinoplasty. Nasal Surgery by the Masters.. 3rd Edition. Thieme; 2014.

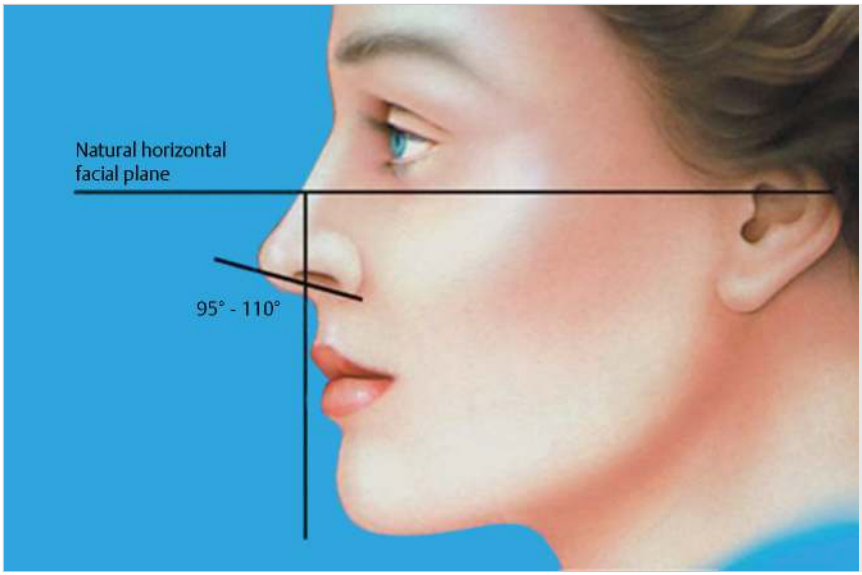


Fig. 20.4 Tip rotation is assessed by calculating the nasolabial angle. From Rohrich R, Adams W, Ahmad J et al., ed. Dallas Rhinoplasty. Nasal Surgery by the Masters.. 3rd Edition. Thieme; 2014.

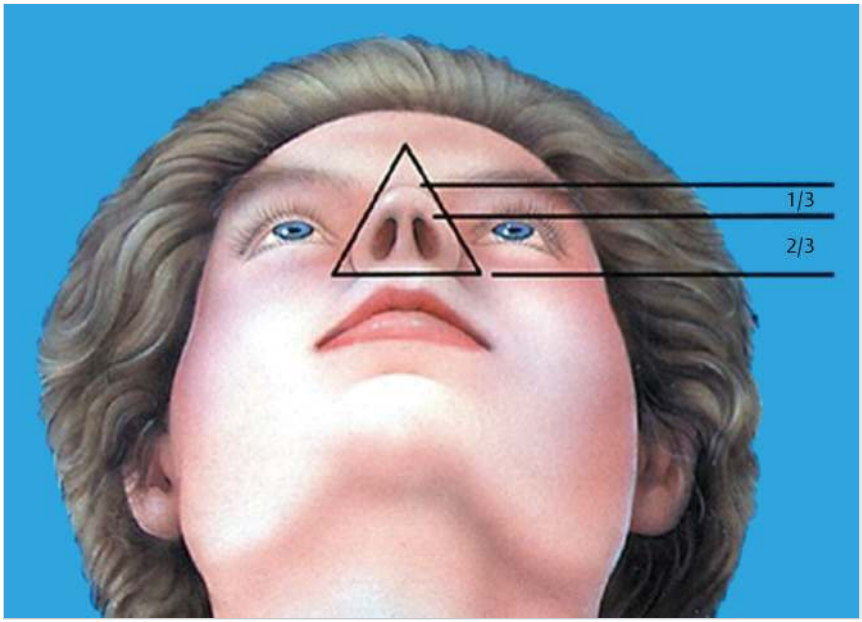


Fig. 20.5 Nostril-tip proportion should be evaluated on the basal view. The ideal nostril-tip relationship should be approximately 2:1. From Rohrich R, Adams W, Ahmad J et al., ed. Dallas Rhinoplasty. Nasal Surgery by the Masters. 3rd Edition. Thieme; 2014.

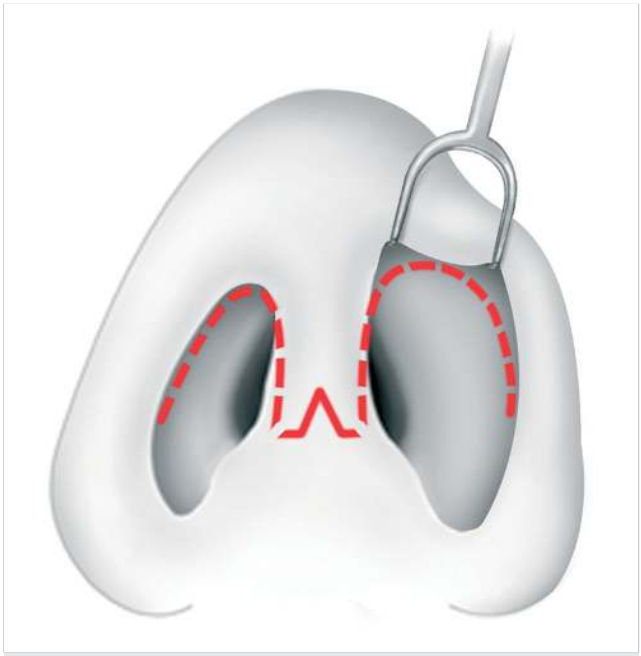


Fig. 20.6 Open approach to nasal tip cartilages. (Used with permission from Behrbohm H, Tardy E. Essentials of Septorhinoplasty. New York, NY: Thieme; 2004:50)

performed by a surgeon who tailors the operation to the specific anatomic deformities of each patient; thus, one should be familiar with both approaches.

Many surgeons feel that the exposure provided by the open approach allows for more precise identification of the diagnosis or etiology for the nasal obstruction or aesthetic deformity. Moreover, manipulation of these structures and the consequence produced by their dynamic interplay is best appreciated. Although the open approach can be used for all rhinoplasties, three particular circumstances may lend themselves to the open approach, including revisional surgery, posttraumatic deformities in which complete release of the intrinsic and extrinsic deforming forces is necessary, when complex tip modification is necessary.[1,2,3]

Several types of transcolumellar incisions exist and include the stair-step and inverted-V (▶ Fig. 20.6). The ideal incision should minimize scar contracture, provide for optimal scar camouflage, and ease closure by the provision of anatomic landmarks. Infracartilaginous extensions of the incision are then made bilaterally and connected to the columellar incisions with careful and meticulous identification of the caudal borders of the lower lateral cartilages (LLCs) so that iatrogenic injury to these structures and particularly the domes is prevented.

The closed approach lends itself to patients with isolated deformities of the nasal dorsum and/or tip which require minimal tip modification. A combination of incisions is often used to access the regions of concern. They include both alar and septal incisions.

20.5 Alar Incisions Used During Closed Rhinoplasty

1. Intercartilaginous: incisional placement between the upper and lower lateral cartilages.
2. Transcartilaginous: incision placed at the level of the lower lateral cartilages with the LLCs delivered via a cartilage-splitting approach.
3. Marginal: incision made at the alar rim and in conjunction with an intercartilaginous incision that allows for delivery of the LLC.

20.6 Surgical Incisions for Both Open and Closed Rhinoplasty

1. Complete (transfixion) incision: the membranous and caudal cartilaginous junction is incised in its entirety, exposing the nasal spine and the depressor septi muscle.
2. Limited partial transfixion: allows for preservation of the attachments of the caudal septum to the medial crural footplates but decreases access to the nasal tip.
3. Partial transfixion: starts caudal to the anterior septal angle and ends just short of the medial crural attachments to the septum.
4. Hemitransfixion: placed unilaterally at the junction of the caudal septum and columella.
5. High septal transfixion: preserves the junction of the caudal septum to the medial crura and membranous septum.

Regardless of the method of approach, the skin envelope is then sharply skeletonized immediately superficial to the perichondrial surfaces of the cartilages in a cephalad direction until the bony pyramid is reached. At this point, a subperiosteal dissection is carried out in limited fashion over the portion of the bony dorsum that needs modification while minimizing disruption to the periosteal attachments which maintain nasal bone position and attachment to the ULCs. The periosteum of the nasal bones is elevated with a Joseph periosteal elevator, and with the same instrument the depressor septi nasi are swept off the maxilla in order to reduce nasal tip plunging.

20.6.1 Nasal Dorsum

Hump reduction can be performed in either a composite or component fashion. Several techniques have been described with osteotome and rasp being the most common method of reducing the dorsal hump (▶ Fig. 20.7). In the composite method where the upper lateral cartilages and nasal bones are reduced together, the risk of separating the nasal bones from the ULCs, resulting in an inverted-V deformity, is possible and necessitates restoration of the ULC position to maintain the internal nasal valve.

In the component method of reduction, the ULCs are separated from the septum by creating bilateral superior sub-muco-perichondrial tunnels which allows for sharp division of these transverse processes while not violating the nasal mucosa. Alternatively, the upper lateral cartilages can be separated from the septum with a no. 11 blade. Upon separation into three distinct components (the septum and the ULCs on each side), the cartilaginous dorsum can then be sharply lowered under direct visualization. The ULCs are reduced if indicated. This type of separation of the ULCs from the septum is necessary when spreader cartilage grafts are placed for treatment of airway obstruction (▶ Fig. 20.8).

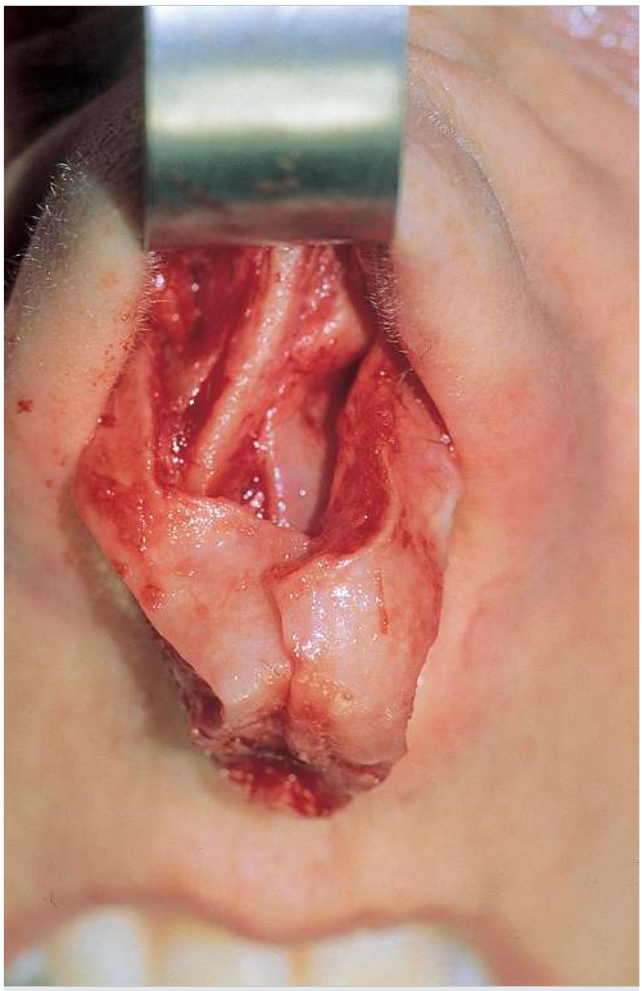


Fig. 20.8 Open approach with inferior retraction of the lower lateral cartilages, and separation of the upper lateral cartilages from the septum. If spreader grafts are used, they are positioned in this space.

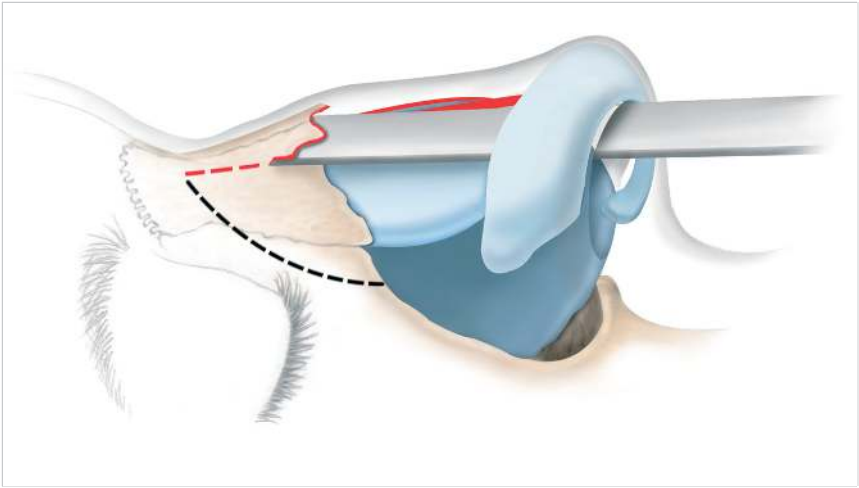


Fig. 20.7 Guarded Rubin osteotome completing removal of the bony dorsal hump. (Used with permission from Behrbohm H, Tardy E. Essentials of Septorhinoplasty. New York, NY: Thieme; 2004:60)

Incremental dorsal bony reduction is then performed with either a power burr or a guarded 8-mm osteotome when large (> 5 mm) or with a sharp, down-biting diamond rasp if smaller. Verification using three-point palpation and a saline moistened dominant index fingertip is performed repeatedly throughout this process to assess for dorsal irregularities or contour depressions of both the left and right dorsal aesthetic lines and the septum centrally.

Dorsal augmentation can be carried out using a variety of techniques with the use of autologous cartilage being most common. Dorsal onlay grafts can be fashioned by performing partial-thickness cuts into various shaped grafts. Care must be taken to minimize graft visibility as skin thickness and graft placement increase this potential risk. More commonly used in Asia, synthetic implants include silicone (expanded polytetrafluoroethylene Gore-Tex) and high-density polyethylene (Medpor; Porex Surgical, Newman, GA).

20.6.2 Septal Reconstruction/Cartilage Graft Harvest

Cartilage grafts have been used in a wide variety of unique applications to help augment areas where contour deformities show deficiency as well as provide structural support (Box 20.2). Septal cartilage obviates the need for an additional donor site but may not be present in revisionary cases. In the closed approach, a Killian or hemitransfixion incision is often employed. If the open approach is used, the middle crura are separated from the anterior septal angle by incising the interdomal suspensory ligament. The septal mucoperichondrium is then incised with a scalpel and a Cottle elevator used to dissect the sub-mucoperichondrial plane posteriorly to the perpendicular plate of the ethmoid bone and down to the nasal floor. A similar dissection is then performed on the contralateral side. Care to maintain structural integrity of the cartilaginous framework necessitates the preservation of an L strut with at least 10 mm of caudal and dorsal septum and at least one side of intact septal mucosa.

Auricular cartilage lacks in volume and rigidity when compared to the other sources. Costal cartilage offers ample volume and support but has the propensity to warp which may be minimized by balanced, cross-sectional carving. Irradiated costal cartilage avoids the morbidity of an additional donor site, but warping does exist despite irradiation.[18] Diced or morselized cartilage offers the advantage of minimizing the forces of cartilage warping and includes several technical variations including the Turkish delight as described by Erol, when diced cartilage wrapped in Surgicel (Ethicon, Somerville, NJ), or wrapped in fascia as described as Daniel.[19,20]

20.6.3 Osteotomies

A variety of techniques have been described, including medial, lateral, and transverse, as well as both internal and external methods of approaches. Three main goals, however, are shared by such variations, including narrowing the lateral walls of the nose, closing an open roof deformity after dorsal hump reduction, and to create symmetry by straightening the nasal bony framework.[1,2,3] Regardless of the specific technique employed, care must be taken to ensure that Webster's triangle, the bony triangular area of the caudal aspect of the maxillary bony process, is not violated so as to prevent functional nasal airway obstruction. Additionally, a step-off deformity at the junction of the osteotomy should be avoided by maintaining a smooth fracture line low along the bony vault with the cephalic border of the osteotomy no higher than the medial canthal ligament. The thick nasal bones above this area increase technical difficulty and allow for potential iatrogenic injury to the lacrimal system.

Lateral osteotomies may be performed in one of three main varieties, with the classification nomenclature referencing "height" relative to the face of the maxilla.

Low-to-high: Begins "low" (lateral) at the pyriform aperture and ends "high" medially along the dorsum. Used to close a small open-roof deformity or mobilize a wide nasal base (▶ Fig. 20.9).

Low-to-low: Begins "low" along the pyriform aperture and continues "low" along the base of the nasal bony vault to end laterally along the dorsum near the intercanthal line. Considered a more powerful technique that is useful to mobilize the

Box 20.2 Overview of Rhinoplasty Grafts by Region

- Dorsum
  - Autospreader flap
  - Dorsal onlay graft
  - Dorsal sidewall onlay graft (lateral nasal wall graft)
  - Radix graft
  - Spreader grafts
  - Septal extension graft
- Tip
  - Anchor graft
  - Cap graft
  - Columellar strut graft (fixed)
  - Columellar strut graft (floating/fixed floating)
  - Extended columellar strut–tip graft (extended shield graft)
  - Onlay tip graft
  - Shield graft (Sheen or infralobular graft)
  - Subdomal graft
  - Umbrella graft
- Alar region
  - Alar batten graft
  - Alar contour graft (alar rim graft)
  - Alar spreader graft (lateral crural spanning graft)
  - Composite alar rim graft
  - Lateral crural onlay graft
  - Lateral crural strut graft
  - Lateral crural turnover flap
- Base
  - Alar base graft
  - Columellar plumping grafts
  - Premaxillary graft

From Rohrich R, Adams W, Ahmad J et al., ed. Dallas Rhinoplasty. Nasal Surgery by the Masters, 3rd Edition. Thieme; 2014.

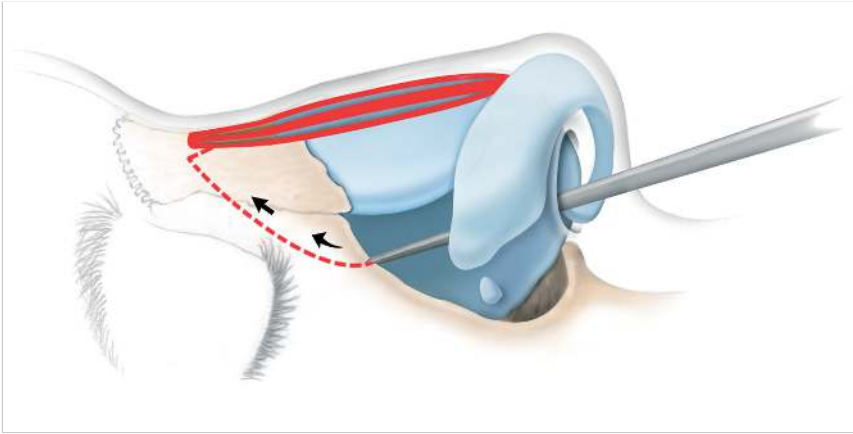


Fig. 20.9 Curved low lateral osteotomy, beginning higher on the ascending process at the level of the inferior concha, coursing lower onto the ascending and finally curving upward toward the medial- oblique osteotomy site. (Used with permission from Behrbohm H, Tardy W. Essentials of Septorhinoplasty. New York, NY: Thieme; 2004:62.)

bony roof, correct a large open roof deformity, or mobilize an excessively wide nasal base.

Double-level: In cases with severe lateral wall asymmetry or with excessive lateral wall convexity that is too great to be corrected with a single lateral osteotomy, two lateral osteotomies may be performed. The first one performed should be the medial of the two, and is carried out along the nasomaxillary suture. The second, more lateral osteotomy, is then performed in a standard low-to-low fashion.

Medial osteotomies can be performed in isolated fashion or in conjunction with aforementioned lateral osteotomies to facilitate medial repositioning of the nasal bones. In the latter case, the medial osteotomy could be performed first as this aids in technical execution of the lateral osteotomy. The cant of the medial ostomy can vary in orientation from medial, oblique, to paramedian or transverse direction. However, the cephalic extent of the osteotomy should not cross the intercanthal line and care taken to not place the osteotome too far medially as this may result in a "rocker deformity" where the bony dorsum is widened from the fractured nasal bone kicking out. Percutaneous osteotomies with a 2-mm osteotome can also be used and do not require closure of the skin incisions.

20.7 Postoperative Care

Patients are typically discharged home the day of surgery with an Aquaplast nasal splint, rarely nasal packing, and a mustache gauze dressing is used for the first 24 to 48 hours. A narcotic prescription (typically hydrocodone/acetaminophen 5/500 mg) is provided to aid in postoperative pain control and used as necessary. Strict postoperative care instructions include avoidance of nose blowing and sneezing thru the mouth when unavoidable. Other movements which may manipulate the nose, including rubbing, blotting are to be avoided for the first 3 weeks.

The nasal splint and any external sutures are removed at the first postoperative visit between days 5 and 7 after surgery. Normal sensation will expect to return over the course of 3 to 6 months. The patient's activities should be restricted for 3 weeks postoperatively while contact sports or activities that may subject the nose to deforming forces withheld for 4 to 6 weeks.

Edema will resolve considerably over the first several weeks and months following surgery, although prolonged edema may take up to and beyond a year to resolve, particularly in the nasal tip. Generally, the patient should be counseled that "identifiable" swelling by those other than the patient should last less than 4 to 6 weeks. During the first 48 to 72 hours following surgery, the patient is instructed to keep the head of the bed elevated and to apply ice packs or cool compresses, such as chilled gel eye masks (Swiss Eye Therapy), to minimize swelling. These two interventions were found to be most commonly employed by a recent survey of facial plastic surgeons (93 and 75%, respectively) while only 21% of high-volume rhinoplasty surgeons routinely prescribe corticosteroids at any time within the perioperative period.[21]

Of note, within this same limited group of high-volume surgeons, 61% prescribed *Arnica montana*, an herbal agent thought to have anti-inflammatory properties, but which have not been unequivocally demonstrated to produce any benefit (level of evidence: therapeutic, II).[22] Further review of the role of corticosteroids in rhinoplasty demonstrates considerable variance in the dose strength, timing, route, and duration of treatment, resulting in confusion regarding their clinical efficacy. Hatef et al published a systematic review to evaluate the efficacy of perioperative steroid dosing in helping reduce edema and ecchymosis following rhinoplasty (level of evidence: therapeutic, II).[23] They found that perioperative administration before surgical induction helped decrease edema and ecchymosis with continued oral administration up to 3 days postoperatively helping to significantly decrease the severity of both even more effectively.

20.7.1 Complication

The incidence of reported complications following rhinoplasty varies widely from 1.7 to 18% with the most common events includes bleeding, infection, nasal airway obstruction, and aesthetic deformities.[24,25,26,27,28]

Bleeding is more prone to occur in patients with a history of hypertension, family or personal history of diathesis (factor XI deficiency), recent use of aspirin, and other nonsteroidal anti-inflammatory agents or nutritional supplements. Pre- and perioperative diligence can minimize these potential risk factors by maintaining close communication with the anesthesia

provider regarding intraoperative blood pressure control. If needed for perioperative treatment of hypertension, the patient can be given a 0.2-mg clonidine transdermal patch (Catapress-TTS-2; Boehringer Ingelheim Pharmaceuticals Inc., Ridgefield, CT).[2,28] Patients should be advised to avoid aspirin and other nonsteroidal anti-inflammatory drugs for at least 7 days before surgery and 2 weeks thereafter.

Two patterns of excessive intraoperative bleeding have been described by Dr. Mark Constantian. Type I presents as a constant, slow, multifocal bleeding starting with the first injection and is indicative of factor VIIII deficiency which is treated with DDAVP (see below). Pattern II, however, typically presents with normal hemostasis for 2 to 3 hours followed by multifocal areas of bleeding from previously treated areas and indicates fibrinolysis. This process is treated by the administration of a 4 to 6 g loading dose of intravenous Amicar (aminocaproic acid), a fibrinolytic inhibitor that inhibits plasminogen activators. This is followed by subsequent administration of Amicar 1 g intravenously every hour until discharge at which point the patient is instructed to take a 1-g parenteral dose daily until the risk of hemorrhage has passed. This pattern has been found to be linked to the use of selective serotonin reuptake inhibitors which are ideally stopped 2 weeks preoperatively. Because this is often not practical, a urinalysis should be obtained preoperatively to rule out microscopic hematuria because of the risk for glomerular capillary thrombosis with Amicar should its administration be necessary.[29,30]

Epistaxis is mild in nature and commonly occurs from either the incisions or traumatized mucosa. Head elevation, oxymetazoline nasal spray (such as Afrin; Bayer, Pittsburg, PA), and 15 minutes of gentle pressure typically resolve minor cases. If bleeding persists, the patient should be seen and anterior nasal packing with Surgicel (Ethicon, Inc., West Somerville, NJ), lubricated with antistaphylococcal antibiotic ointment placed into the affected nare.[17]

Cases of continuous bleeding may benefit from the administration of desmopressin, a synthetic analogue of vasopressin, which increases coagulation activity by rising plasma concentrations of factor VIII. In a study by Faber et al (level of evidence: therapeutic, IV), 9 of 268 consecutive rhinoplasty patients who presented to the emergency department with refractory bleeding were administered 0.2 µg/kg of intravenous desmopressin over 30 minutes.[31] This effectively controlled the bleeding in all cases. If all measures fail to control the bleeding, consultation for angiographic embolization is necessary. Should bleeding persist despite the aforementioned interventions, operative exploration is mandated with exploration and cauterization employed.

Hematomas necessitate drainage and packing, as they will invariably lead to fibrosis, scarring, and contour deformities. Septal hematomas can lead to perforations, necrosis of septal cartilage and ultimately, saddle nose deformity when severe.

Cases of soft tissue infection are best avoided by judicious use of perioperative antibiotics as discussed earlier. However, when mild cases of cellulitis do present, the patient should be started on cephalosporin antibiotic for 24 to 48 hours.[28] Symptoms which fail to resolve could be secondary to MRSA and empiric antibiotic therapy initiated based on local antibiotic resistance patters.[32,33]

20.7.2 Nasal Airway Obstruction

Most patients can expect some degree of transient, operative induced nasal airway obstruction for 2 to 3 weeks following rhinoplasty as edema slowly resolves. If persistent, internal examination of the nasal airway with the aid of a vasoconstrictor should be carried out to elicit the inciting cause. Confirmation that persistent edema is the underlying etiology may be managed with nasal decongestants although topical vasoconstrictors should be used for less than 7 days as rebound congestion will occur. If, on the other hand, anatomic causes of obstruction are found, surgical intervention will be necessary but should be delayed for at least 1 year to allow for the resolution of edema and scar maturation.

20.7.3 Deformities

Mild deformities discovered during the postoperative period should be conservatively managed with observation, and surgical intervention performed when persistent after one year. Significant deformities, on the other hand, should be corrected when identified to minimize patient dissatisfaction. Contour irregularities can be masked temporarily with filler agents such as Restylane (Galderma Laboratories, L.P., Fort Worth, TX) which is placed in a specific, judicious manner. However, permanent correction can be achieved by injection of permanent silicone based agents, Scar proliferation and other deformities resulting from the healing process may sometimes occur. In general, they are encountered in the supratip area. These can be treated with the injection of 3 to 5 mg of triamcinolone acetate (10 mg/mL) mixed in a one-to-one fashion with 2% lidocaine using a 27-gauge needle with care taken to avoid superficial injections that can lead to dermal atrophy or intravascular injection.[17,28]

20.8 Outcomes

Although several authors have attempted to describe methods of assessing patient satisfaction with others attempting to quantify both functional and aesthetic results (often in relation to the impact of various technical maneuvers performed at the time of surgery), a lack of standardized methods in which to assess both functional and aesthetic outcomes in rhinoplasty persists.[34,35,36,37,38] In a 2009 survey of board certified plastic surgeons and otolaryngologists, most reported revisionary rhinoplasty to be required in less than 10% of patients.[39] Numerous factors play a role in the underlying etiology that necessitates reoperation, but can be classified into one or a combination of the following: displaced anatomic structures including grafts, under correction during the time of primary rhinoplasty from an overly conservative surgeon, or, conversely, over-resection/overcorrection from an overzealous surgeon.

The increased complexity of revisionary rhinoplasty is attributable to altered anatomy, scar formation, previous graft placement with potential warping, and potential cartilage deficiency.[40] These factors tend to influence most surgeons to adopt an open approach in secondary cases where superior operative exposure of the nasal framework allows for accurate diagnosis and proper correction of the deformity.

20.9 Review Questions

20.9.1 Choose the Best Answer

1. Closed rhinoplasty is best suited for
   a) Revisional surgery.
   b) Complex tip modifications.
   c) Minimal tip modification.
   d) Posttraumatic deformities.
2. All are key factors that can increase the chances for excessive perioperative bleeding, *except*
   a) Hypertension.
   b) Aspirin.
   c) Nonsteroidal anti-inflammatory drugs.
   d) Personal or family history of bleeding diathesis.
   e) History of nasal trauma.
3. The following is a true statement about cartilage grafting for rhinoplasty:
   a) Septal cartilage should not be used.
   b) Costal cartilage is too soft.
   c) Auricular cartilage is as rigid as septal cartilage.
   d) Costal cartilage tends to warp.
   e) Diced cartilage is ineffective.
4. The preoperative assessment during the preparation for surgery should include
   a) Assessment of patient's goals and objectives for the surgery.
   b) Examination in multiple views.
   c) Intranasal examination.
   d) Detailed history of breathing issues and/or allergic rhinitis.
   e) All of the above.
5. The most common postoperative complications encountered in rhinoplasty include the following, *except*
   a) Epistaxis.
   b) Nasal airway obstruction.
   c) Septal perforation.
   d) Aesthetic deformity.
   e) Infection.

20.9.2 Answers

1. Minimal tip modification (c).
2. History of nasal trauma (e).
3. Costal cartilage tends to warp (d).
4. All of the above (e).
5. Septal perforation (c).

References

[1] Beck DO, Kenkel JM. Evidence-based medicine: rhinoplasty. Plast Reconstr Surg. 134(6):1356–1371
[2] Rohrich RJ. Open Technique Rhinoplasty, 3rd ed. New York: Saunders Elsevier; 2012
[3] Swamy RS, Sykes JM, Most SP. Principles of photography in rhinoplasty for the digital photographer. Clin Plast Surg. 37(2):213–221
[4] Punthakee X, Rival R, Solomon P. Digital imaging in rhinoplasty. Aesthetic Plast Surg. 33(4):635–638
[5] Janis JE, Ahmad J, and Rohrich RJ: Primary Rhinoplasty. In: Nahai F. (ed.), The Art of Aesthetic Surgery: Principles and Techniques, 2nd ed. New York: Thieme Medical Publishers; 2011, pp 1895-1974
[6] Toriumi DM, Dixon TK. Assessment of rhinoplasty techniques by overlay of before-and-after 3D images. Facial Plast Surg Clin North Am. 19(4):711–723, ix
[7] Mehta U, Mazhar K, Frankel AS. Accuracy of preoperative computer imaging in rhinoplasty. Arch Facial Plast Surg. 12(6):394–398
[8] Picavet VA, Prokopakis EP, Gabriëls L, Jorissen M, Hellings PW. High prevalence of body dysmorphic disorder symptoms in patients seeking rhinoplasty. Plast Reconstr Surg. 128(2):509–517
[9] Becker DG, Bloom JD, Gudis D. A patient seeking aesthetic revision rhinoplasty and correction of nasal obstruction. Otolaryngol Clin North Am. 42(3): 557–565
[10] Becker DG, Ransom E, Guy C, Bloom J. Surgical treatment of nasal obstruction in rhinoplasty. Aesthet Surg J. 30(3):347–378, quiz 379–380
[11] Chandra RK, Patadia MO, Raviv J. Diagnosis of nasal airway obstruction. Otolaryngol Clin North Am. 42(2):207–225, vii
[12] Constantian MB, Clardy RB. The relative importance of septal and nasal valvular surgery in correcting airway obstruction in primary and secondary rhinoplasty. Plast Reconstr Surg. 98(1):38–54, discussion 55–58
[13] Rajan GP, Fergie N, Fischer U, Romer M, Radivojevic V, Hee GK. Antibiotic prophylaxis in septorhinoplasty? A prospective, randomized study. Plast Reconstr Surg. 116(7):1995–1998
[14] Lyle WG, Outlaw K, Krizek TJ, Koss N, Payne WG, Robson MC. Prophylactic antibiotics in plastic surgery: trends of use over 25 years of an evolving specialty. Aesthet Surg J. 23(3):177–183
[15] Georgiou I, Farber N, Mendes D, Winkler E. The role of antibiotics in rhinoplasty and septoplasty: a literature review. Rhinology. 46(4):267–270
[16] Nicholas BD, et al. Preoperative prevalence of methicillin-resistant Staphylococcus aureus (MRSA) colonization in patients undergoing intranasal surgery. Med Sci Monit. 2010; 16(8):365-368
[17] Rohrich RJ, Ahmad J. Rhinoplasty. Plast Reconstr Surg. 128(2):49e–73e
[18] Adams WP , Jr, Rohrich RJ, Gunter JP, Clark CP, Robinson JB , Jr. The rate of warping in irradiated and nonirradiated homograft rib cartilage: a controlled comparison and clinical implications. Plast Reconstr Surg. 103(1):265–270
[19] Velidedeoğlu H, Demir Z, Sahin U, Kurtay A, Erol OO. Block and Surgicel-wrapped diced solvent-preserved costal cartilage homograft application for nasal augmentation. Plast Reconstr Surg. 115(7):2081–2093, discussion 2094–2097
[20] Daniel RK, Calvert JW. Diced cartilage grafts in rhinoplasty surgery. Plast Reconstr Surg. 113(7):2156–2171
[21] Shadfar S, Deal AM, Jarchow AM, Yang H, Shockley WW. Practice patterns in the perioperative treatment of patients undergoing septorhinoplasty: a survey of facial plastic surgeons. JAMA Facial Plast Surg. 16(2):113–119
[22] Totonchi A, Guyuron B. A randomized, controlled comparison between arnica and steroids in the management of postrhinoplasty ecchymosis and edema. Plast Reconstr Surg. 120(1):271–274
[23] Hatef DA, Ellsworth WA, Allen JN, Bullocks JM, Hollier LH , Jr, Stal S. Perioperative steroids for minimizing edema and ecchymosis after rhinoplasty: a meta-analysis. Aesthet Surg J. 31(6):648–657
[24] McKinney P, Cook JQ. A critical evaluation of 200 rhinoplasties. Ann Plast Surg. 7(5):357–361
[25] Holt GR, Garner ET, McLarey D. Postoperative sequelae and complications of rhinoplasty. Otolaryngol Clin North Am. 20(4):853–876
[26] Dziewulski P, Dujon D, Spyriounis P, Griffiths RW, Shaw JD. A retrospective analysis of the results of 218 consecutive rhinoplasties. Br J Plast Surg. 48(7): 451–454
[27] Cochran CS, Landecker A. Prevention and management of rhinoplasty complications. Plast Reconstr Surg. 122(2):60e–67e
[28] Rohrich RJ. P. J., Landecker A. Postoperative management of the rhinoplasty patient, 2nd Ed. St. Louis: Quality Medical; 2007
[29] Andrade C, Sandarsh S, Chethan KB, Nagesh KS. Serotonin reuptake inhibitor antidepressants and abnormal bleeding: a review for clinicians and a reconsideration of mechanisms. J Clin Psychiatry. 71(12):1565–1575
[30] Constantian, MC. (2015, June). Email with Alan Matarasso, MD
[31] Faber C, Larson K, Amirlak B, Guyuron B. Use of desmopressin for unremitting epistaxis following septorhinoplasty and turbinectomy. Plast Reconstr Surg. 128(6):728e–732e
[32] Elward AM, McAndrews JM, Young VL. Methicillin-sensitive and methicillin-resistant Staphylococcus aureus: preventing surgical site infections following plastic surgery. Aesthet Surg J. 29(3):232–244
[33] Angelos PC, Wang TD. Methicillin-resistant Staphylococcus aureus infection in septorhinoplasty. Laryngoscope. 120(7):1309–1311
[34] McKiernan DC, Banfield G, Kumar R, Hinton AE. Patient benefit from functional and cosmetic rhinoplasty. Clin Otolaryngol Allied Sci. 26(1):50–52

[35] Kosowski TR, McCarthy C, Reavey PL, et al. A systematic review of patient-reported outcome measures after facial cosmetic surgery and/or nonsurgical facial rejuvenation. Plast Reconstr Surg. 123(6):1819–1827
[36] Meningaud JP, Lantieri L, Bertrand JC. Rhinoplasty: an outcome research. Plast Reconstr Surg. 121(1):251–257
[37] Courtiss EH, Goldwyn RM. The effects of nasal surgery on airflow. Plast Reconstr Surg. 72(1):9–21
[38] Tollefson TT, Sykes JM. Computer imaging software for profile photograph analysis. Arch Facial Plast Surg. 9(2):113–119
[39] Warner J, Gutowski K, Shama L, Marcus B. National interdisciplinary rhinoplasty. Aesthet Surg J. 29(4):295–301
[40] Rohrich RJ, Lee MR. External approach for secondary rhinoplasty: advances over the past 25 years. Plast Reconstr Surg. 131(2):404–416

21 Browlift and Blepharoplasty

Jorge de la Torre and Cristiano Boneti

Abstract
This chapter surveys the salient considerations for periorbital rejuvenation procedures. Foremost in the discussion are patient concerns involving function and aesthetics. Areas of emphasis during the presurgical physical examination are listed, and tests for assessing remedial action are thoroughly covered. Common surgical techniques—resection of excess skin, repositioning of anatomical structures, excision of herniated fat pads—are discussed. Measures involved in both brow lifting and upper and lower blepharoplasty are detailed and illustrated, and the chapter concludes with the authors advising on postoperative care, expected outcomes, and complications that can potentially (though rarely) ensue.

Keywords: browlift, blepharoplasty, senile ptosis, transverse forehead rhytids, snap test, Schirmer test, endobrow technique

21.1 Goals and Objectives

- Understand the proper evaluation of prospective periorbital rejuvenation patients.
- Clearly define the benefits for the various periorbital procedures.
- Appreciate the technical aspects of addressing each anatomic component of age-related changes and/or neuromuscular abnormalities.
- Know the evidence-based perioperative care to maximize patient safety and quality outcomes.

21.2 Patient Presentation

Patients seeking for blepharoplasty or forehead lift present with concerns regarding function, aesthetics, or a combination. Functional impairment may present as a decrease in visual field as a result of senile ptosis, with associated “tired” appearance, or the result of trauma with or without facial nerve paralysis. Other less common neuromuscular disorders such as myasthenia gravis, tumors, or Horner’s syndrome represent a minority of the cases. On the other hand, patients seeking periorbital improvement of age-related changes will be peculiar about what aspect of their appearance bothers them the most. Since the aging face does so at different rate between patients and between anatomic regions in the same patient, a thorough understanding of the patient’s concerns is essential to achieve a satisfactory result. Therefore, the history should include subjective assessment of vision, use of corrective lenses/contacts, symptoms of dry eyes, facial nerve disorders, hypertension, bleeding disorders, endocrine disorders, cataracts, glaucoma, diabetes, corneal or previous lid surgery, psychiatric disorders, trauma, and medications.

The physical examination should document not only any existing skin lesions and dermatologic conditions but also determine the quality of the skin and if chemical or laser peeling would improve the final result.[1] Integrity of the extraocular muscles should also be documented.

A documentation of the orbital fissure will also guide the treatment plan. Guidelines for the aesthetic goals have been suggested by Farkas and Kolar, Flowers, and Wolford et al as follows[2,3,4]:

- The forehead—distance from the anterior hairline to brow—measures 5 to 6 cm.
- The distance from brow to orbital rim should be 1 cm; brow to supratarsal crease, 1.6; and brow to midpupil, 2.5 cm.[5]
- Canthal tilt should be 3 to 4 degrees higher on the lateral canthus.[6] Lid margin to lid fold ranging from 8 to 10 mm.[7]
- The upper lid may drape over 2 to 3 mm of upper limbus, while the lower lid should just touch the lower limbus.[8]
- The medial canthus should line up on a vertical plane with the medial brow and lateral edge of the nasal ala. The intercanthal distance should be one-fifth of the facial width.[9,10]

The upper eyelid exam should evaluate for asymmetries, ptosis, levator function, skin and fat excesses, and lid retraction. The examination must be performed in the resting position and in an elevated brow position if a brow lift is being considered. Further evaluation of the lower eyelid includes assessment of scleral show, lid position, lid tone and support, entropion/ectropion, malar bags, nasojugal folds, and skin/muscle/fat excess.[11]

The age-related changes in the orbicularis oculi result in the crow's feet and contribute to lid ptosis. Such changes are the result of muscle relaxation as well as increasing laxity and attenuation of the orbicularis ligamentous attachments.[12] Observation of the skeletal anatomy is also very important, as a negative vector between the globe, the lower eyelid margin, and the malar eminence also known as “polar bear syndrome” portends an inferior result after lower lid blepharoplasty unless appropriate modifications of lower lid surgery are made.[1,8] Finally, documentation of Bell's phenomenon is important as it is a protective reflex.

21.2.1 Preparation for Surgery

Patient evaluation should begin with a visual field test to assess and validate the need for ptosis correction. Once that is established, bilateral ptosis correction is invariably necessary even if unilateral ptosis is observed. As the pathologic side is corrected, decrease lid elevation reflex will unmask a compensated ptosis on the contralateral side.

Lower eyelid laxity must be assessed with a snap test, and a Schirmer test may be helpful to diagnose dry eyes. Noteworthy is the fact that Rees and LaTrenta found that 65% of patients with postoperative dry eye syndrome had a normal Schirmer test.[13]

Next, it is important to determine if brow elevation is indicated. If blepharoplasty is to be performed in combination with brow lift, the eyelids are addressed first to avoid the distortion resulting from edema after the brow lift. It is important to allow for the brow elevation during marking of the upper lids to avoid overcorrection.

Currently, the preferred method of brow lift is the endobrow technique because of comparable brow elevation, long-lasting

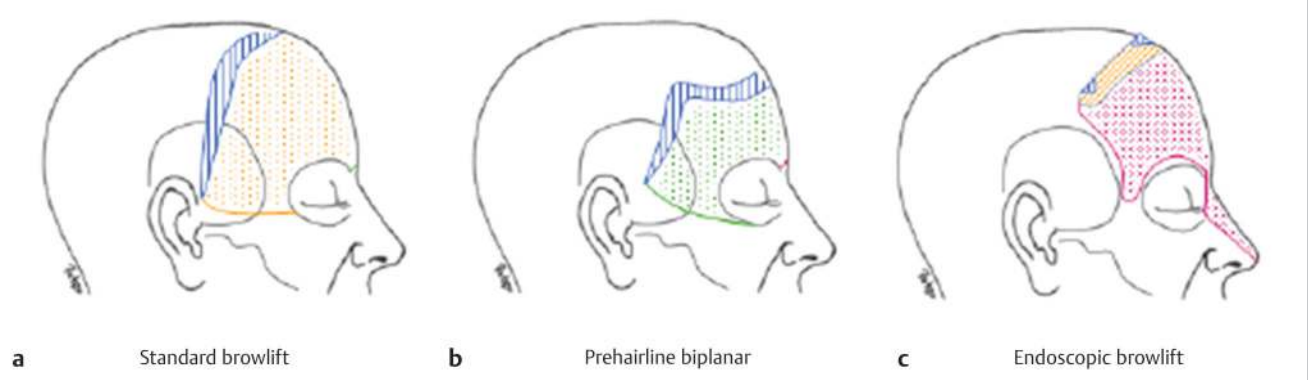


Fig. 21.1 Standard browlift approach— (**a**) skin excision (*blue*) with subperiosteal undermining (*yellow*); (**b**) pre-hairline biplanar approach—skin excision (*blue*) with subcutaneous undermining (*green*); (**c**) endoscopic browlift approach—–endoscopic access incisions with subperiosteal undermining (*red*).

results, and improved cosmesis. However, if an elevation or advancement of the hairline is intended, then an open technique must be used.

In a healthy patient and with no history of bleeding disorder, no further workup is necessary. Comorbidities are evaluated accordingly.

21.3 Treatment

The goals of periorbital surgery are to restore a youthful appearance. The steps used to accomplish it consist of resection of excess skin, repositioning of the anatomical structures, and excision of herniated fat pads.

The essential elements of a successful operation are judicious resection of redundant skin, fat, and muscle while preserving or restoring symmetry and function. Goals for the upper lid include the restoration of sharp, crisp tarsal folds and a pretarsal show with deepening of the orbitopalpebral sulcus. The lower lids must appear smooth and soft. Distortion of lid shape and position must be avoided. The brow should be elevated to the level of the superior orbital rim in males and 1 cm above in females. It is critical to evaluate both the eyelids and the brow with regard to ptosis.

The procedure may be performed under general anesthesia or local anesthesia plus anxiolytics. If local anesthetic route is chosen, 1 mL of 1% lidocaine with epinephrine is injected on each eyelid prior to the procedure. A diluted lidocaine 0.25% with epinephrine is infiltrated in the forehead and a supratrochlear, supraorbital, and infraorbital nerve block is obtained. With general anesthesia, lidocaine is not necessary and diluted epinephrine 1:1,000,000 is sufficient.

The patient is placed supine with a headband exposing the entire face and ears. Considering the good vascular perfusion and extremely low risk of infection, skin cleansing with peroxide is enough. We believe the peroxide alone is adequate preparation of the skin as long as there is no bone exposure or work performed.

21.4 Brow Lifting

Rejuvenation of the brow is designed to address three main areas: (1) position of the eyebrow, (2) transverse wrinkles, and (3) forehead height (distance from the eyebrow to the hairline). The anatomy of the forehead muscles includes the frontalis which elevates the brow and results in the transverse rhytids. The depressor muscle includes the procerus and corrugators which cause vertical glabellar rhytids and the orbicularis oculi muscle. Various approaches to perform browlift procedures include the standard approach, the pre-hairline incision, endoscopy, direct incision, and upper blepharoplasty approach (▶ Fig. 21.1).

21.4.1 Open Forehead Lift

Patients with significant to severe forehead ptosis and significant asymmetry of the brows or deep forehead wrinkles may require an open or combined approach. The open approach can be very successful in re-creating lateral brow elevation. The preferred incision varies according to the sex, hair pattern, and desired elevation of the forehead. In females, an incision within the hair-bearing scalp is well tolerated and will hide the scars. It avoids shortening of the forehead, but can result in posterior displacement of the hairline. Therefore, it is best suited for patients with short forehead distances (< 6 cm), and should be carefully considered in patients with high hairlines. According to Connell and Marten, every 1 mm of eyebrow elevation produces 1.5 mm of elevation of the hairline.[14] It should also be carefully considered in men at risk for male pattern baldness, as the scar will become apparent with hair loss. In bald patients, superciliary or midforehead incisions can avoid the visibility of a scalp incision and be disguised within the normal transverse forehead rhytids.

Once the incision is made, a subperiosteal dissection will provide the most reliable and lasting elevation. Gentle blunt dissection can be performed with a periosteal elevator. This allows maximum mobilization of the skin and soft tissues. Lateral dissection to release the orbital retaining ligament is helpful in obtaining lateral elevation. Several approaches have been described with particular advantages and disadvantages.

A hairline incision is preferable in patients with long foreheads (> 10 cm), as it avoids lengthening the forehead. Incisions are placed perpendicular to the skin and parallel to the hair follicles, as it allows the growth of hair through the scar making it very acceptable to patients.[15] A subcutaneous approach will

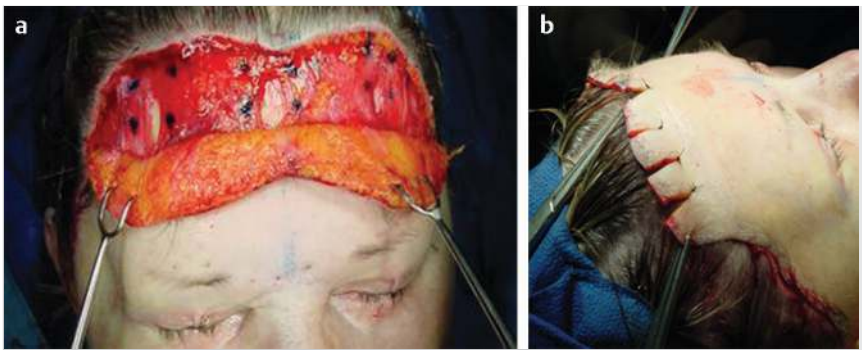


Fig. 21.2 **(a)** Open pre-hairline approach with subcutaneous elevation and endoscopic approach through the frontalis muscle to release the supraorbital periosteum. **(b)** Tension-free redraping of excess skin for excision after plication of the frontalis muscle.

Fig. 21.3 Various blunt dissectors, note hook scalpel on far right.

Fig. 21.4 Lighted endoscope with protective sheath.

directly remove the skin attachments to the muscle, effectively addressing the transverse wrinkles (▶ Fig. 21.2).

21.5 Endoscopic Forehead Lift

Patients with mild to moderate ptosis will benefit the most from this approach. It is also preferable that forehead length is with the normal range of 6 to 10 cm. The principle behind endoscopic forehead lift is that with ablation of the eyebrow depressors, forehead elevation will be unopposed by the frontalis muscle function. Division of the periosteum allows free reposition of the eyebrows and once it adheres to the underlying bone, a natural, dynamic, long-term result can be expected.[16,17] Although the use of firm fixation with screws or sutures has been described, physiologic correction alone provides excellent outcomes and helps avoid the undesirable excessively elevated appearance that can occur with rigid suspension of the brow.

Access for the endoscopic cavity is obtained through three triangular incisions marked anterior to the frontal hairline. As indicated, the incisions are beveled to match the direction of the follicles. This helps preserve the hair follicles and avoids alopecia at the scars. The incisions need to be just sufficient for introduction of the endoscopic equipment.

The triangular shape allows for additional access to permit passage of the endoscope through the incision without extending its length. In addition, the horizontal closure of the triangular incision provides a slight amount of extra brow elevation. The three minimal access incisions are placed at the frontal pre-hairline level, one at the midline and the two at lateral incisions in line with the lateral limbus. The size and location can be adjusted to account for asymmetries with regard to forehead ptosis.

The marked skin and subcutaneous tissue are excised above the musculo-aponeurotic tissue. The galea and the periosteum are bluntly separated with a closed hemostat dissecting along the muscle fibers to avoid damage of the local sensory nerves. Using a curved periosteal elevator, the subperiosteal dissection extends to the supraorbital rims and to the frontotemporal line (▶ Fig. 21.3). Dissection is extended to the nasal tip to release the procerus. Direct visualization is obtained using a lighted rigid endoscope (▶ Fig. 21.4) and endoscopic video monitor (▶ Fig. 21.5). An optical cavity is created using a sheath around the scope to elevate the soft tissue off the lens. In addition, three nylon suspension sutures are placed externally just above the brow line (▶ Fig. 21.6). Divergent traction is placed on the sutures. The traction on the sutures helps create an optical cavity.

The supraorbital periosteum is divided completely to the level of the lateral orbital rims with an endoscopic carpal tunnel hook knife scalpel. Resection of the corrugators, procerus, and other depressors is achieved bluntly with a grasping forceps. The supratrochlear and supraorbital nerves are identified and preserved. The glabellar region is inspected after resection to ensure that the contour is smooth and not depressed or irregular. If there is any depression, a small amount of fat or SMAS can be used to fill the area (▶ Fig. 21.7, ▶ Fig. 21.8, ▶ Fig. 21.9).

Fig. 21.5 Note operative surgeon is at the head of the patient, the monitor is at the patient's feet, and the assistant is to the side.

Fig. 21.6 Triangular incisions and three nylon sutures to create optical cavity. The sutures a and c are pulled "divergently."

At the end of the procedure, the incisions are closed transversely. After closure of the access incisions, temporary suspension is obtained with cable sutures. A single staple is placed across the closure of each incision and approximately 4 cm posterior within the hair-bearing scalp. A 3–0 Nylon suture is passed through both staples and tightened to suitably suspend the brow during the immediate postoperative period. This direct, external suspension allows for adjustment to avoid postoperative asymmetries. The staples and nylon sutures are left in place 3 to 5 days. This time period allows for adhesion of the periosteum and long-term effect. The resection of the depressor muscles obviates the need for other methods of suspension. Postoperative suspension has been described with internal sutures securing through tunnels drilled in the external table of the frontal skull or using bone screws. Absorbable tacks are also available to stabilize the soft tissue. A simple method is the use of external nylon sutures thread through staples across the three endoscopic incisions and several centimeters posterior in the hair-bearing scalp (▶ Fig. 21.10).

21.6 Upper Blepharoplasty

The preferred lower skin incision is placed at the supratarsal crease incision, 9 to 12 mm above the eyelid margin. The upper skin incision is determined based on the amount of excess skin. The amount of skin to be resected is determined before surgery, marking the patient in an upright position and by pinching the lid skin using a fine forceps. Conservative skin resection is recommended, especially if the forehead lift is to be performed after the blepharoplasty. If blepharoplasty is performed in combination with forehead lift, estimation of the skin resection should be done with manual elevation of the brow simulating the expected forehead lift effect. Medial and lateral aspects of the incision for dealing with skin redundancy at either end have been addressed in various ways, removing the skin with a resection design that will result in a scar with an acute angle superior and lateral results in improved scar appearance. The resection can be performed separately where first one remove the skin alone dissected off the muscle followed by muscle resection separately or an en bloc of skin with orbicularis muscle. Skin resection alone is safer and allows for judicious resection of orbicularis muscle under direct visualization. It is critical to avoid injury to the levator muscle when resecting orbicularis muscle (▶ Fig. 21.11a).

Excess or herniated orbital fat is removed through small openings in the orbital septum. Gentle separation through the septum with a mosquito hemostat provides access to the fat compartments. The medial compartment is often more medial than anticipated and is usually pale in color compared to the other compartments. Careful hemostasis is essential to avoid complications such as hematomas or even blindness. Finally,

Fig. 21.7 Resection of depressor muscles under direct visualization; note suspension sutures to create optical cavity. (Note the surgeon is using bimanual palpation and the assistant is controlling the endoscope.)

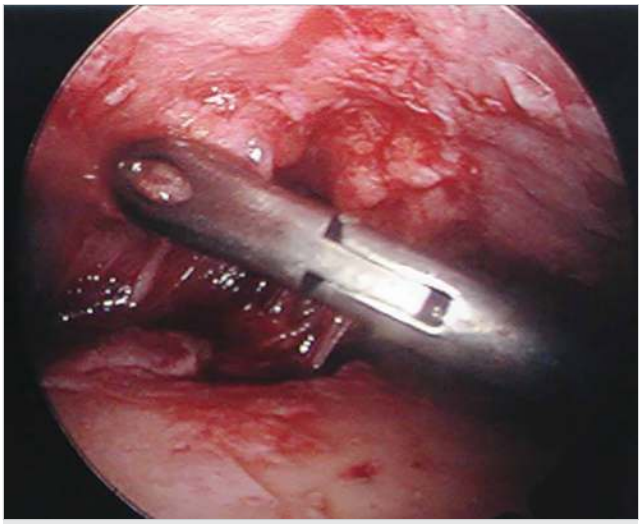


Fig. 21.8 Endoscopic view of grasper resecting depressor muscles.

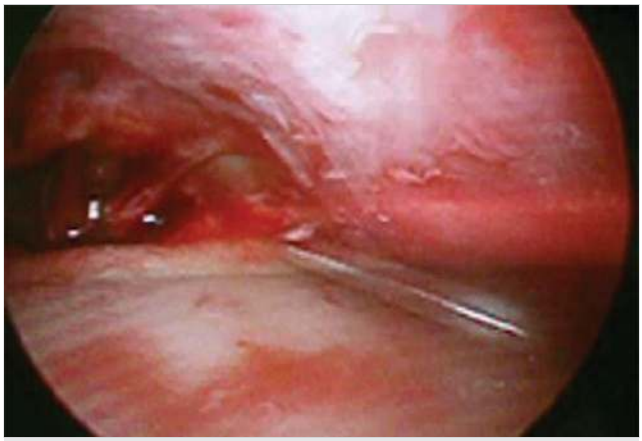


Fig. 21.9 Release of periosteum with hook scalpel.

plication of the levator muscle to tarsus is performed as needed for ptosis (▶ Fig. 21.11b, ▶ Fig. 21.12).

21.7 Lower Blepharoplasty

The approach to the lower lid includes the use of transconjunctival incision or an external skin incision. Eye protectors are highly recommended, particularly with the transconjunctival approach, to avoid corneal abrasion or injury. A temporary frost suture to close the lids is an option to protect the cornea when an external skin incision is used. External incisions allow resection of skin; however, it should be noted that skin excisions should be very conservative to avoid postoperative ectropion. Although lower lid incisions can be placed in a skin crease within the lower lid, typically a subciliary incision is preferred. Pinching the lower lid skin to determine the amount of excess skin to be resected is a reliable technique. This method preserves the blood supply to the lower lid, avoiding complications associated with skin-only techniques. Reported complications include ectropion and denervation of the orbicularis muscle (▶ Fig. 21.13a).

A subciliary skin–muscle flap technique exposes the orbital septum down to the level of the orbital rim.[18] It is important also to avoid injury to the lower lid orbicularis muscle and to preserve innervation to the muscle in order to avoid ectropion.

Gentle pressure on the globe will demonstrate the herniated fat compartments. The periorbital fat is removed through

perforating incisions in the orbital septum. Conservative fat resection can improve the lid–cheek junction and the appearance of the transition from the lower lid to the malar fat pad by removing the fat from the bulging lower lid compartments. Over-resection of fat results in a hollowed, aged appearance.

One option to avoid this is the release of the arcus marginalis as described by Hamra and others.[12] After release of the arcus marginalis, the lower lid fat is dissected out from the compartment, but left attached to preserve the vascular supply. It is draped into the tear trough beneath the lower lid skin flap. This helps ameliorate the transition from the lower lid to the cheek.

Alternatively, the malar fat pad can be elevated superiorly through the lower lid incision (▶ Fig. 21.11) to address the tear trough area. In addition to improving the transition from the lower lid to cheek, this adjunct has greatly reduced the risk of lower lid ectropion in patients undergoing lower lid blepharoplasty (▶ Fig. 21.13b).

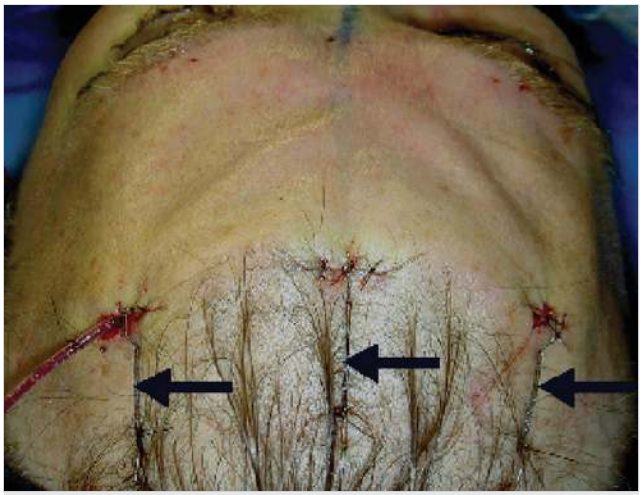


Fig. 21.10 Closure of access incisions and placement of nylon suspension sutures to provide temporary suspension.

Senile horizontal laxity of the lower lid may be addressed by wedge excision. As suggested by Codner et al, pulling the incised lower lid laterally will confirm intraoperatively the degree of laxity.[19] When there is less than 3 mm of laxity overlapping the lateral orbital rim, it can be managed with a canthopexy alone. More severe redundancy usually requires a formal canthoplasty.

21.8 Postoperative Care

In the immediate postoperative period, cool packs are recommended as is elevation of the head (▶ Fig. 21.14). Direct contact with ice pack should be avoided as they can result in thermal injury.

Patients are instructed to wash their face and hair with soap and water as usual. However, they are cautioned to be especially cautious about hot water as the protective sensation may be impaired for a few weeks. We ask patients to avoid hair driers and makeup until the scabs fall off. Once that happens, we recommend avoiding sun exposure and to apply sunscreen.

Fig. 21.12 Upper blepharoplasty incisions 1 week postoperatively.

Fig. 21.11 **(a)** Surgical landmarks. Upper lid margin covers 1 to 2 mm of the upper limbus (*red arrow*); Lower lid is at the lower limbus (*green arrow*); the distance from the upper lid margin to the lid crease is approximately 8 mm (*blue arrow*). **(b)** Immediate postoperative appearance following biplanar open browlift, upper and lower blepharoplasty. Note there is some anticipated lagophthalmos in the immediate postoperative period, which will resolve.

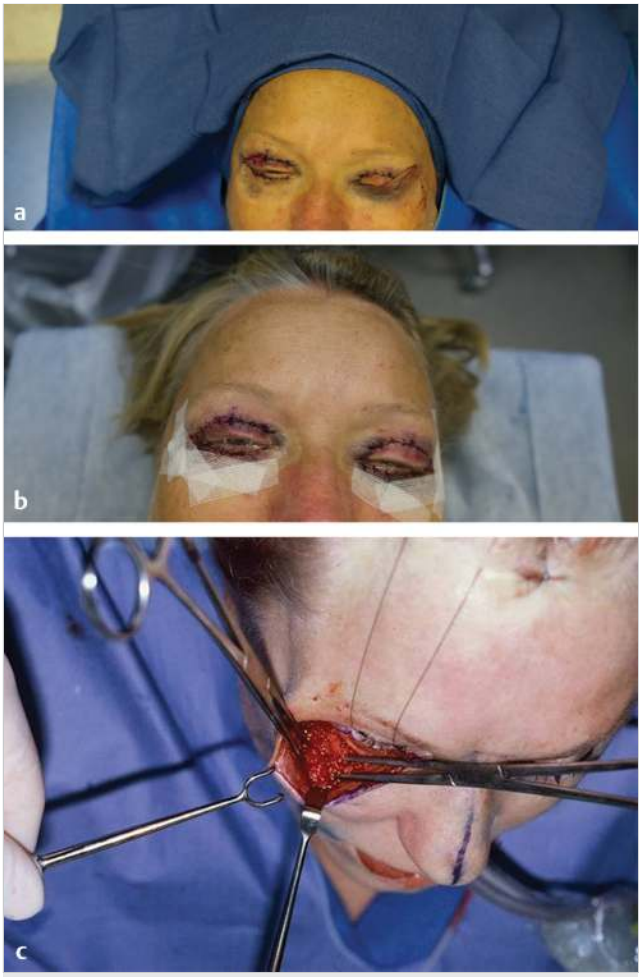


Fig. 21.13 (**a**) Intraoperative view demonstrates excess skin, draped tension-free, (**b**) immediately postoperatively with suspension tape in place. (**c**) Use of subciliary incision to suspend malar fat pad.

Retention sutures from endoscopic forehead lift are removed in 3 to 5 days.

21.9 Outcomes

Most patients heal uneventfully with very favorable improvement in their appearance. Nevertheless, various complications can occur, with serious complications occurring infrequently or rarely (▶ Fig. 21.15, ▶ Fig. 21.16).

21.10 Complications

21.10.1 Visual Loss

Blindness after blepharoplasty is thought to be a result of increased intraorbital pressure secondary to hemorrhage. The estimate incidence is 0.04%.[20] Several cases of visual loss after blepharoplasty have been reported.[21,22] Treatment of retrobulbar hemorrhage requires immediate decompression of the orbit by opening the skin incisions and releasing the septum and the lateral canthus; reduction of intraocular pressure with an intravenous osmotic agent (20% mannitol, 1–2 mg/kg body weight, 12.5 g administered over 3 minutes and the remainder over 30 minutes, and 500 mg acetazolamide [Diamox] IV bolus followed by 250 mg by mouth every 6 hours); and administration of 95% oxygen/5% CO_2 mixture to dilate intraocular and intracerebral vessels.[5,23]

Fig. 21.14 Placement of cool packs rather than ice.

21.10.2 Corneal Injury

Prevention with corneal shields and gentle handling of tissues is the main strategy; however, they must be used carefully as their placement can lead to corneal injuries as well. Once a corneal injury is suspected, fluorescein and Wood's lamp can confirm the diagnosis. Where superficial injuries can be managed with topical antibiotics and patching of the eye, large or deep corneal abrasions should be referred to an ophthalmologist. Upper blepharoplasty that sufficiently corrects skin excess and ptosis may result in temporary lagophthalmos. This will resolve within 2 weeks or less. In the interim, patients must be educated on using artificial tears and, if needed, ophthalmic ointment at night. In some cases, taping or an eyepatch while sleeping may be required to protect the cornea.

21.10.3 Bleeding

Meticulous hemostasis, good technique, and good intraoperative and perioperative blood pressure control are the best strategy to prevent this complication. Many authors have suggested that traction on the posterior vessels during aggressive fat resection, poorly controlled fat pad vessels that retract into the orbit, and bleeding deep into the orbit from the cut edges of the orbicularis are the likely sources of bleeding.[24,25]

21.10.4 Diplopia

Where temporary diplopia is likely the result of healing inflammatory reaction, edema, or small hematomas, permanent diplopia or strabismus is thought to occur from damage to the extraocular muscles or nerves.[26] The most commonly injured muscle is the inferior oblique muscle, followed closely by the superior oblique muscle.[22]

Fig. 21.15 **(a)** Pre-op and **(b)** post-op views after upper and lower blepharoplasty and endoscopic forehead lift.

Fig. 21.16 **(a)** Patient with dermatochalasis, asymmetric brow ptosis, and vertical glabella rhytids. (**b**) Postoperative appearance after upper and lower blepharoplasty and endoscopic forehead lift.

21.10.5 Ptosis

Postoperative ptosis is usually the result of failure to diagnose preoperatively, or unilateral correction without addressing the contralateral ptosis. It may also result from injury to the levator muscle.[27]

21.10.6 Lagophthalmos

Some degree of transient lagophthalmos is normal for a few days after blepharoplasty. It usually resolves spontaneously or with conservative treatment such as lubrication, massage, lid taping, and patching. Permanent lagophthalmos is the result of excessive skin resection or incorporation of the orbital septum in the incisional scar. In this case, correction requires a full-thickness skin graft.[22,28]

21.10.7 Ectropion

The eversion of the lid margin may result from excessive skin, fat or muscle removal; scar contracture, adhesion of the orbital septum; failure to address lid laxity; and proptosis.

21.10.8 Inadequate Fat Resection

Excessive intraorbital fat resection can result in a hollowed out socket.[29] Insufficient fat resection is most common in the upper and lower medial and lower lateral compartments.

21.10.9 Chemosis

Chemosis occurs in up to 11% of blepharoplasty patients. It is characterized as conjunctival edema and hyperemia with tearing and can progress to periorbital edema and ectropion. This is most often due to mild lid malposition and incomplete lid opposition in the early postoperative period. Initial treatment is ocular lubrication and corticosteroid ophthalmic drops. If symptoms persist, oral corticosteroids can be added, with minor procedures such as an incision in the conjunctiva for drainage and tarsorrhaphy added as needed.[30]

21.11 Review Questions

21.11.1 Choose the Best Answer

1. Transverse forehead rhytids are caused by
   a) Descent of the eyebrows.
   b) Hyperactivity of the frontalis muscle.
   c) Inactivity of the corrugator muscle.
   d) Hypoactivity of the forehead elevators.
2. The endoscopic forehead procedure is well suited for which of the following situations:
   a) A patient who has a very long forehead.
   b) A patient with a very short forehead.
   c) A patient with moderate brow ptosis.
   d) Significant asymmetry of the eyebrows.
3. The upper eyelid incision should be marked
   a) In the supratarsal crease.

b) 9 to 12 mm above the lid margin.
c) With the patient in the upright position.
d) All of the above.

4. Which of the following is correct regarding lower lid blepharoplasty:
   a) Aggressive skin resection helps improve the tear trough deformity.
   b) Corneal eye protectors must always be used.
   c) Senile laxity is best addressed with vertical skin excision.
   d) Injury to the orbicularis muscle can cause ectropion.
5. The first step in the management of postoperative blindness should be managed by
   a) Intravenous mannitol.
   b) Releasing the septum and the lateral canthus.
   c) 500 mg acetazolamide (Diamox) IV bolus.
   d) Ophthalmology consult.

21.11.2 Answers

1. b. Hyperactivity of the frontalis muscle.
2. c. A patient with moderate brow ptosis.
3. d. All of the above.
4. d. Injury to the orbicularis muscle can cause ectropion.
5. b. Releasing the septum and the lateral canthus.

References

[1] Jelks GW, Jelks EB. Preoperative evaluation of the blepharoplasty patient. Bypassing the pitfalls. Clin Plast Surg. 20(2):213–223, discussion 224
[2] Farkas LG, Kolar JC. Anthropometrics and art in the aesthetics of women's faces. Clin Plast Surg. 14(4):599–616
[3] Flowers RS. The art of eyelid and orbital aesthetics: multiracial surgical considerations. Clin Plast Surg. 14(4):703–721
[4] Wolfort FG, Baker T, Kanter WR: Aesthetic goals in blepharoplasty. In: Wolfort FG, Kanter WR, eds. Aesthetic Blepharoplasty. Boston, MA: Little Brown; 1995
[5] Matarasso A, Terino EO. Forehead-brow rhytidoplasty: reassessing the goals. Plast Reconstr Surg. 93(7):1378–1389, discussion 1390–1391
[6] Bartlett SP, Wornom I , III, Whitaker LA. Evaluation of facial skeletal aesthetics and surgical planning. Clin Plast Surg. 18(1):1–9
[7] Baker TJ, Gordon HL, Mosienko P. Upper lid blepharoplasty. Plast Reconstr Surg. 60(5):692–698
[8] Jelks GW, Jelks EB. The influence of orbital and eyelid anatomy on the palpebral aperture. Clin Plast Surg. 18(1):183–195
[9] Ellenbogen R. Transcoronal eyebrow lift with concomitant upper blepharoplasty. Plast Reconstr Surg. 71(4):490–499
[10] Tolleth H. Concepts for the plastic surgeon from art and sculpture. Clin Plast Surg. 14(4):585–598
[11] Carraway JH, Mellow CG. The prevention and treatment of lower lid ectropion following blepharoplasty. Plast Reconstr Surg. 85(6):971–981
[12] Hamra ST. The zygorbicular dissection in composite rhytidectomy: an ideal midface plane. Plast Reconstr Surg. 102(5):1646–1657
[13] Rees TD, LaTrenta GS. The role of the Schirmer's test and orbital morphology in predicting dry-eye syndrome after blepharoplasty. Plast Reconstr Surg. 82 (4):619–625
[14] Connell BF, Marten TJ. The male forehead plasty. Recognizing and treating aging in the upper face. Clin Plast Surg. 18(4):653–687
[15] Camirand A, Doucet J. A comparison between parallel hairline incisions and perpendicular incisions when performing a face lift. Plast Reconstr Surg. 99 (1):10–15
[16] Vasconez LO, Core GB, Oslin B. Endoscopy in plastic surgery. An overview. Clin Plast Surg. 22(4):585–589
[17] Ramirez OM. Why I prefer the endoscopic forehead lift. Plast Reconstr Surg. 100(4):1033–1039, discussion 1043–1046
[18] Beare R. Surgical treatment of senile changes in the eyelids: the McIndoe-Beare technique. In: Smith B, Converse JM, eds. Proceedings of the Second International Symposium on Plastic and Reconstructive Surgery of the Eye and Adnexa. St Louis, MO: Mosby; 1967
[19] Codner MA, McCord CD, Hester TR , Jr. The lateral canthoplasty. Oper Tech Plast Reconstr Surg. 5(2):90
[20] DeMere M, Wood T, Austin W. Eye complications with blepharoplasty or other eyelid surgery. A national survey. Plast Reconstr Surg. 53(6):634–637
[21] Hepler RS, Sugimura GI, Straatsma BR. On the occurrence of blindness in association with blepharoplasty. Plast Reconstr Surg. 57(2):233–235
[22] Lowry JC, Bartley GB. Complications of blepharoplasty. Surv Ophthalmol. 38 (4):327–350
[23] Hueston JT, Heinze JB. Successful early relief of blindness occurring after blepharoplasty. Case report. Plast Reconstr Surg. 53(5):588–592
[24] Callahan MA. Prevention of blindness after blepharoplasty. Ophthalmology. 90(9):1047–1051
[25] Lisman RD, Hyde K, Smith B. Complications of blepharoplasty. Clin Plast Surg. 15(2):309–335
[26] Smith B, Lisman RD, Simonton J, Della Rocca R. Volkmann's contracture of the extraocular muscles following blowout fracture. Plast Reconstr Surg. 74(2): 200–216
[27] Patipa M, Wilkins RB. Acquired ptosis in patients undergoing upper eyelid blepharoplasty. Ann Ophthalmol. 16(3):266–, 268–270
[28] Baylis HI, Cies WA, Kamin DF. Correction of upper eyelid retraction. Am J Ophthalmol. 82(5):790–794
[29] Tenzel RR. Complications of blepharoplasty. Orbital hematoma, ectropion, and scleral show. Clin Plast Surg. 8(4):797–802
[30] Weinfeld AB, Burke R, Codner MA. The comprehensive management of chemosis following cosmetic lower blepharoplasty. Plast Reconstr Surg. 122(2): 579–586

22 Face and Neck Rejuvenation

Ahmed M. Hashem, Rafael A. Couto, Cagri Cakmakoglu, Eliana F. R. Duraes, and James E. Zins

Abstract

This chapter explores in detail the range of considerations, historical perspectives, assessments, and surgical and nonsurgical options involved in offsetting facial and neck aging. Changes that occur due to facial aging, skin laxity, sun damage, lipoatrophy, soft-tissue descent, and potential skeletal changes are covered, along with effective remedies for restoring balance and harmony to the facial parts. Treatment options for every region of the face and neck are reviewed, including skin resurfacing, soft-tissue augmentation, chemodenervation, and surgery. Operative techniques—incisions, facelifts, skin management, etc.—are listed, discussed, and illustrated. Particular emphasis is paid to the risks involving the facial nerve branches. Seasoned advice on postoperative care and complications conclude the chapter.

Keywords: minimal access cranial suspension (MACS), lateral SMASectomy, extended superficial musculoaponeurotic system (SMAS), composite rhytidectomy, lipo-filling, botulinum toxin, fillers, peeling, laser, facelift

22.1 Goals and Objectives

- Recite the landmark contributions which define the historic evolution of face and neck rejuvenation surgery.
- Describe the three-dimensional surgical anatomy of the SMAS, retaining ligaments of the face, facial nerve branches, safe dissection planes, and danger zones.
- Outline the basics of clinical evaluation of face and neck rejuvenation patients.
- Differentiate short-scar-limited SMAS interventions from traditional surgical techniques.
- Explain the principles and steps of the MACS lift, the lateral SMASectomy, the extended SMAS, and composite rhytidectomy.
- Summarize the potential complications and their management.

22.2 Patient Presentation

22.2.1 Clinical Evaluation

Given the plethora of surgical and nonsurgical options, a comprehensive clinical assessment is crucial to guide treatment strategies. Particular attention is given to the various aspects of facial aging including skin laxity, sun damage, lipoatrophy, soft-tissue descent, and potential skeletal changes.

22.2.2 Aesthetic Facial Evaluation with a Cephalometric Perspective

This assessment should analyze age-related deformities in the context of soft tissue and skeletal cephalometrics. The goal is to determine balance and proportions (harmony) of different facial parts, establish limitations of soft-tissue procedures, and identify potential adjunctive skeletal interventions (e.g., genioplasty).[1,2] The patient should be assessed with the head in "natural" position (straight forward gaze; the Frankfort horizontal is generally parallel to the ground in that posture) and with the eyebrows and lips relaxed.[1] Any deviation results in soft-tissue distortion that can lead to inaccurate assessment.[1] Although assessment should be three dimensional, frontal aesthetics hold prime significance since this is how patients picture themselves (▶ Fig. 22.1, ▶ Fig. 22.2).[1]

Upper Third

Aging is associated with (1) hairline recession, (2) transverse forehead rhytids, (3) vertical glabellar lines, and (4) eyebrow ptosis.

Middle Third

Aging is associated with (1) blepharochalasis, (2) ptosis, (3) lower lid laxity ± ectropion, (4) lower lid fat bulge, (5) Crow's feet, (6) tear trough deformity, (7) palpebromalar groove, and (8) malar mound.

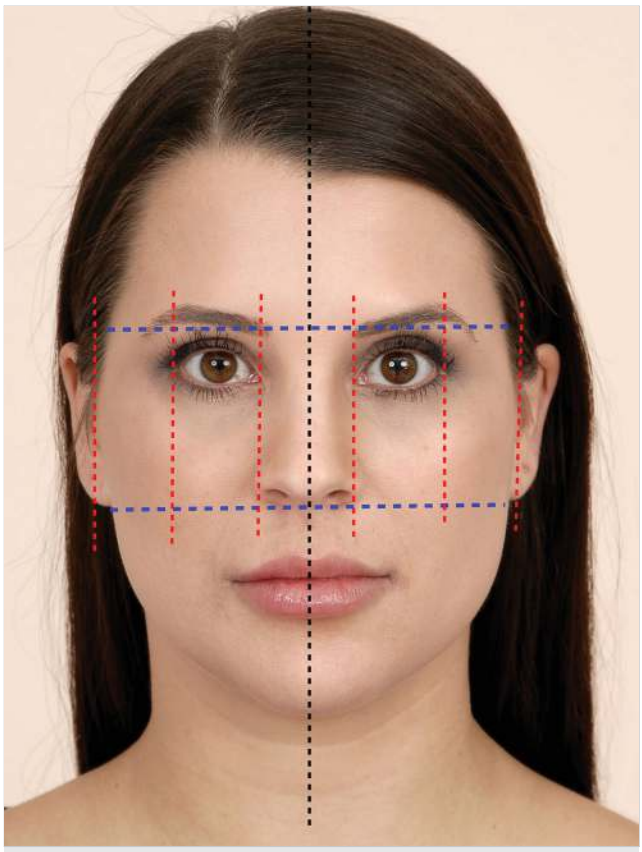


Fig. 22.1 The face can be divided into three almost equal thirds along the glabella and subnasale. In transverse dimension, it can be divided into five almost equal thirds as depicted. The midline helps identify areas of asymmetry.

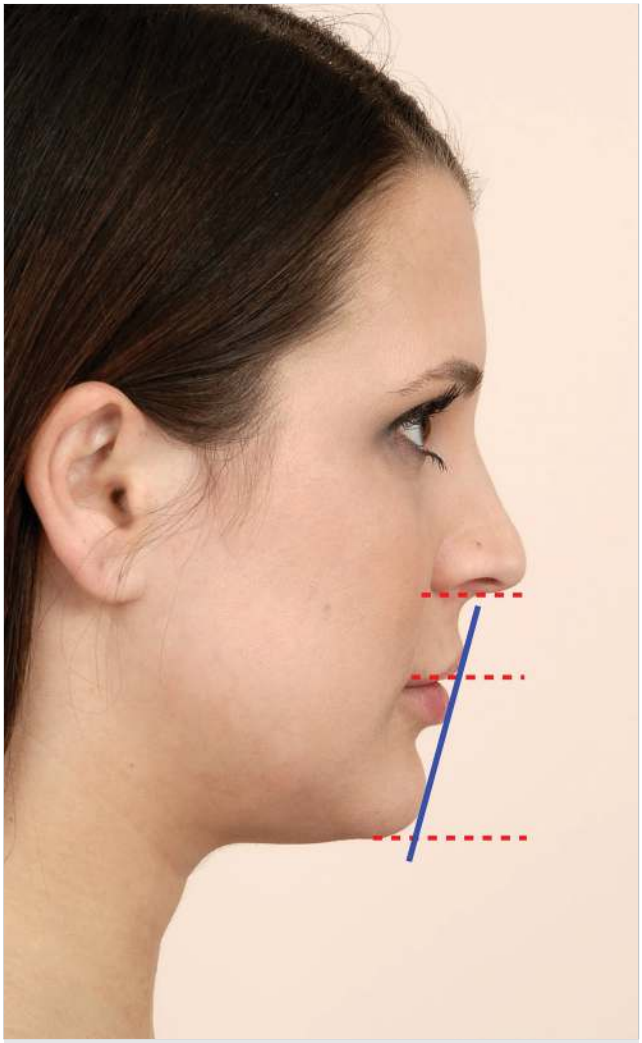


Fig. 22.2 The relationships of the lower third of the face. Red dotted lines show the ratio of upper to lower lip and chin of 1:2. Blue line shows the anteroposterior relation of upper and lower jaw.

The Lower Third and Neck

Aging is associated with (1) nasolabial fold bulge, (2) nasolabial crease deepening, (3) marionette lines, (4) exaggeration of the labiomental fold, (5) perioral wrinkles, (6) loss of the cervicomental angle, and (7) platysmal bands.

22.3 Preparation for Surgery

Like many other aesthetic procedures, rigorous patient selection is crucial. Medically unfit patients may benefit from alternatives such as botulinum toxin and fillers. A thorough screening for hemorrhagic diathesis is essential to avoid bleeding complications.[3] Antiplatelets and vitamin K suppressors should be stopped 1 to 2 weeks prior to surgery.[3] A medical consult is occasionally necessary to document fitness for surgery, the feasibility of drug cessation, and to suggest alternatives.[3] Smoking should be stopped for at least 4 weeks in advance.[4,5] Finally, identifying patients with unrealistic expectations, psychiatric disease, psychosocial impairment, and body dysmorphic disorder is also critical.[6] Chronological age does not seem to be an independent risk factor for higher complications in rhytidectomy surgery. In fact, a study demonstrated that elderly patients (older than 65 years) were not at a higher risk for complications, compared with a younger control group (younger than 65 years), provided proper preoperative screening is undertaken.[3]

22.4 Treatment

Face and neck rejuvenation involve multifaceted clinical treatment of cervicofacial aging using skin resurfacing, soft-tissue augmentation, chemodenervation, and surgery. The objective is to positively modify skin, cervicofacial contour, and restore volume. Safe and effective treatment requires precise knowledge of anatomy and technique. Understanding anatomical planes, the SMAS's (Superficial Muscular and Aponeurotic System) relations to the facial nerves, and retaining ligaments is critical to safe sub-SMAS surgery. No versatile surgical technique can fit all needs. Rather surgeons should modify surgery to meet the specific requirements of their patients. While the *MACS (minimal access cranial suspension) lift* and the *lateral SMASectomy* are examples of widely used *short-scar-limited SMAS interventions*, the *extended SMAS* and the *composite rhytidectomy* represent prototypes of *conventional sub-SMAS procedures* that suit patients with significant laxity and advanced signs of aging. *Lipo-filling* is assuming growing significance as a regenerative and volume restoring adjunct. Nonsurgical interventions, especially *botulinum toxin* and soft-tissue *fillers*, are rising as standalone and ancillary interventions. Finally, skin-resurfacing techniques, like *peeling* and *LASER*, further optimize the surgical outcome. This chapter focuses on surgical treatment.

22.5 Historical Perspectives

Prior to Skoog's contributions,[7] facial rejuvenation was addressed with conservative skin resection, and limited subcutaneous undermining. Skoog reported the elevation of a cervicofacial flap deep to the superficial fascia, lifting the platysma in the neck, and the "superficial fascia" in the face, opening a new era for facelift surgery.[7] This paved the way for defining the applied anatomy of the SMAS by Mitz and Peyronie in 1976.[8] Furnas then outlined the retaining ligaments of the cheek in 1989.[9] Stuzin et al further highlighted the importance of the masseteric ligaments, and the relationship between superficial and deep fascia, defining safe planes and danger zones of sub-SMAS dissections.[10] These anatomical studies stimulated different "SMAS-platysma modifications" and deeper tissue repositioning techniques in an attempt to identify procedures that are more durable.

Owsley described the SMAS-platysma facelift, which formed the foundation of SMAS flap techniques.[11,12,13] The subperiosteal approach was presented by Psillakis et al and Ramirez et al who explored deep tissue repositioning in this anatomical plane.[11,12,13,14,15] Hamra took Skoog's operation a step further, describing first the deep plane facelift,[16] followed by the composite facelift,[17,18,19] as well as different modifications of the lower eyelid myofascial complex.[20,21,22,23,24,25] Barton reported his modification of Skoog's procedure which became known as

the "high SMAS technique."[26,27,28,29]While the refinements by Hamra and Barton adopted a composite dissection, where skin and SMAS are elevated as one unit, other SMAS-based techniques employed a bilamellar dissection, where the SMAS is undermined and redraped independently in a vertical direction, while the skin vector is more horizontally positioned.[18,28] These latter procedures described variable SMAS flap designs, for example, the extended SMAS.[12,18,28,30,31,32,33,34,35,36,37]

In the neck, Connell combined comprehensive lipectomy with full platysma transection and mobilization.[38] Various other platysma-modifying techniques were also proposed. The most well-known of these being the "corset platysmaplasty" described by Feldman in 1990.[39] Knize, Zins, and Feldman further highlighted the utility of the isolated anterior approach to improve the neck in selected patients.[40,41,42,43] A trend toward less invasive techniques in facial rejuvenation was initiated by the endoscopic brow lift, intended to address drawbacks of conventional open brow lift surgery.[44] "Short-scar-limited SMAS" procedures, including the lateral SMASectomy and the MACS lift, then followed.[45,46,47,48,49,50,51,52,53,54,55] These "less invasive" procedures were designed to serve patients with minimal laxity seeking shorter recovery and less surgical risk.[48,49,50,53,55] Recently, more emphasis has been placed on ancillary facelift techniques including volume restoration with lipofilling based on Coleman's pioneering work.[56,57] Finally, the evolution of botulinum toxin further allowed myogenic manipulation of wrinkles, and brow position, and soft-tissue fillers enabled wrinkle and volume adjustment in less surgery-committed patients.[58,59]

22.6 Anatomical Considerations

"No amount of improvement in facial contouring is worth an injury to the facial nerve."[35]

22.6.1 The SMAS-platysma-superficial Temporal Fascia plane

The SMAS is a subcutaneous fibromuscular structure continuous with the platysma in the neck and the superficial temporal fascia in the temporal region.[8] Further cephalad it lays in the same plane as the frontalis and galea aponeurotica.[8] More anteriorly it invests the muscles of facial expression.[10,27] Posteriorly it is fixed to the thick parotid fascia in addition to the thick sternocleidomastoid fascia.[8,10] The facial nerve branches are invariably located deep to this layer (▶ Fig. 22.3).[8,10] The SMAS is muscular caudally, where it merges with platysma, but becomes progressively aponeurotic (fibrous) more cephalad (▶ Fig. 22.4).[8] Posteriorly, it blends with the parotid fascia, forming a thick layer.[8] More anteriorly, it thins out and becomes more tenuous.[8,10]

22.6.2 The Support System of the Face (Retaining Ligaments of the Face)

A series of retaining ligaments or more recently termed "SMAS fusion zones" supports the facial soft tissues against gravitational forces.[60,61,62] Their constant location and predictive nature with relation to facial nerve branches renders them surgically significant (▶ Fig. 22.3).[40,41,42,63,64,65,66] The classic plastic surgery literature has described this support system as osteocutaneous or "true" ligaments such as the zygomatic and mandibular ligaments, or false ligaments.[9,10,63,67] True ligaments extend from bone to dermis passing through the SMAS and arborizing to attach to dermis, similar to the trunk of a tree passing from bone and arborizing after passing through the SMAS to insert into dermis.[33] Arborization allows an enhanced effect on skin superficially.[33] Myofascial or false ligaments, such as the masseteric and parotid cutaneous ligaments, are fibrous structures fixing deeper fascial layers to overlying superficial soft tissue.[10]

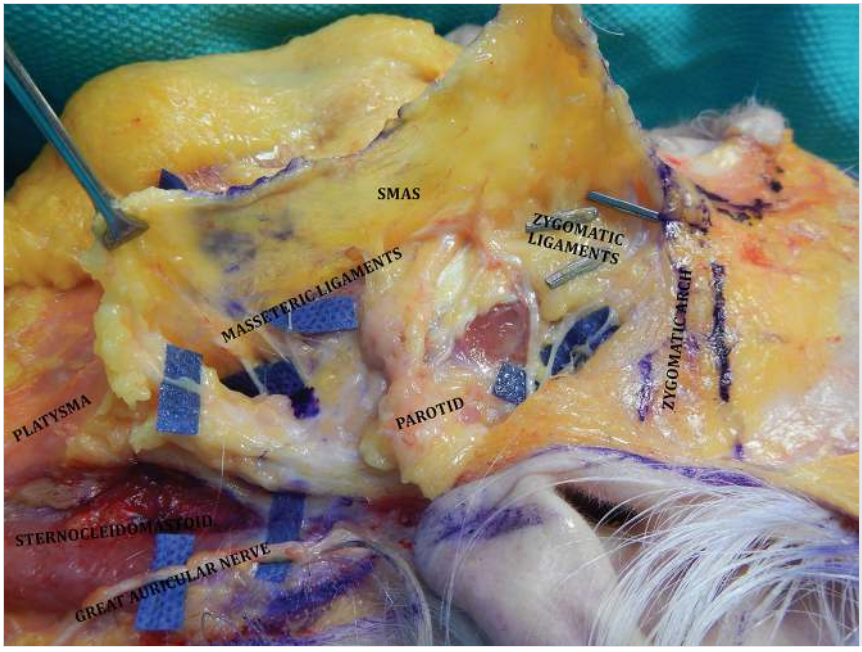


Fig. 22.3 Cadaver dissection of the left side of the face. The SMAS layer has been elevated and held with Allis clamps up. Underneath, the parotid gland and the facial nerve branches can be seen. The zygomatic ligaments were tagged with clips before their release. The relation of the zygomatic ligaments to the zygomatic rami of the facial nerve can be appreciated. Lower down in the sub-SMAS area, fibrous strands extending from the deeper tissues to the SMAS constitute the masseteric ligaments.

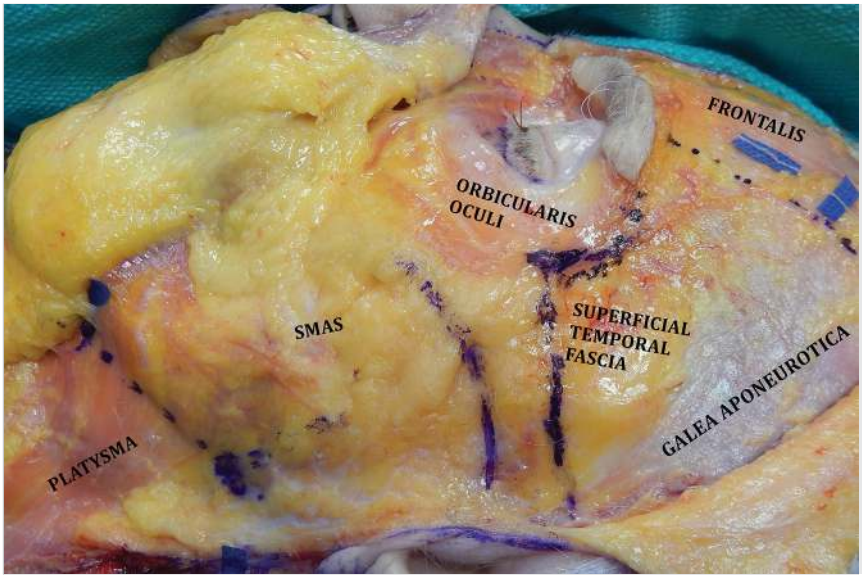


Fig. 22.4 Cadaver dissection of the left side of the face. The skin flap is reflected at the medial part of the face. The outline of the mandibular border and the zygomatic arch are emphasized with blue marking. Note the SMAS is continuous with the platysma in the neck and the superficial temporal fascia in the temple. This layer blends with galea aponeurotica and frontalis more cephalad and the orbicularis oculi around the eye. The SMAS is muscular in the lower face and aponeurotic further cranially.

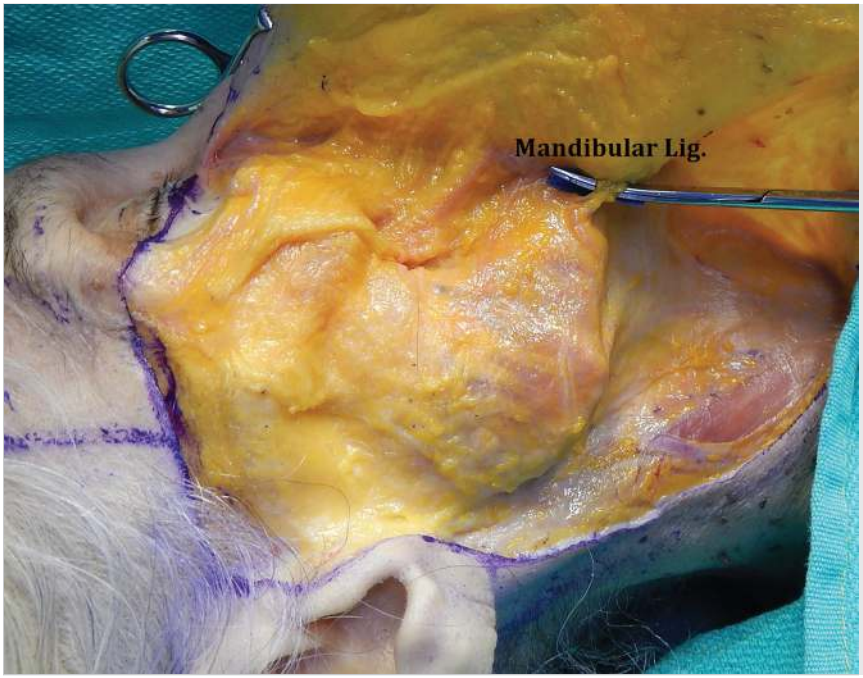


Fig. 22.5 Cadaver dissection of the right side of the face showing the mandibular ligament in the lower face extending from the anterior mandible to the skin. An instrument is placed behind the ligament.

Moss et al, for greater precision, advocated a distinction between (1) True ligaments which are cylindrical fibrous structures holding the superficial layer (SMAS and extensions) to deeper periosteum or deep fascia, and are mostly located in the mid-, and lower face.[66] The zygomatic, masseteric, and mandibular ligaments (▶ Fig. 22.3, ▶ Fig. 22.5) constitute the best-known examples of these.[66] (2) Septa, on the contrary, are fibrous walls extending from deep fascia or periosteum to the superficial layer and are basically found in the temporal and periorbital region. The superior and inferior temporal septa exemplify the prototypes of these (▶ Fig. 22.6).[66] (3) Finally, adhesions represent areas of low-density fibrous or fibrofatty adhesions between the superficial layer and deeper tissues. The temporal and supraorbital ligamentous adhesions are models of this latter configuration (▶ Fig. 22.6).[66] The mobility of the superficial layer is generally dictated by the type of underlying support, with true ligaments providing greatest mobility, septa allowing only motion perpendicular to their line of attachment, and adhesions restricting movement in all directions.[66] Furthermore, septa and adhesions represent areas of extended bonding between superficial and deep fascia generally requiring wider dissection to obtain satisfactory repositioning.[66]

Recently, Pessa described this anatomy as SMAS fusion zones rather than ligaments.[60] In his analysis, these structures exist as bilaminar membranes extending between superficial and deep fascia, and constitute the boundaries of the fat compartments and the anatomical spaces of the face (e.g., the buccal space).[57,60] Blood vessels, nerves, and lymphatics are stabilized by traveling

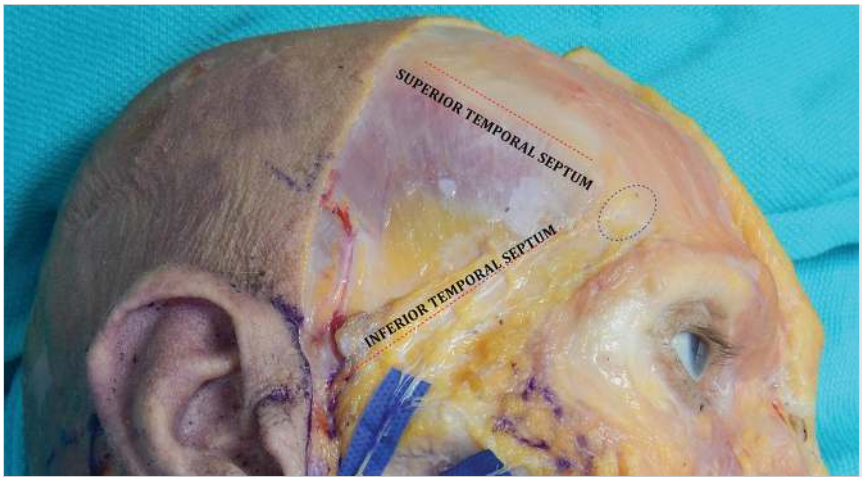


Fig. 22.6 Cadaver dissection of the right upper face showing the superior temporal septum (red dots), the inferior temporal septum (red dots), and the temporal ligamentous adhesion (blue ellipse). Note the relation of the frontal rami of the facial nerve with blue background underneath the inferior temporal septum.

within these fusion zones.[60] Between superficial fascia (SMAS) and skin, these membranes further continue as unilaminar structures limiting the superficial fat compartments.[60]

The disparity in nomenclature and variation in reporting have added significant confusion to the understanding of these anatomical structures.[60,61,62]

The Temporal Region

The Superior Temporal Septum

This coincides with the superior temporal fusion line where the galea and periosteum adhere to bone just superior and medial to the deep temporal fascia (▶ Fig. 22.6).[66] It starts superior to the temporalis muscle and arches downward toward the outer corner of the superior orbital rim, defining the transition of the superficial layer from temporoparietal fascia to galea and frontalis.[66] Its lower end is expanded and forms the temporal ligamentous adhesion.[66] The septum forms the upper border of the superior temporal compartment, whose inferior boundary is formed by the inferior temporal septum. Dissection can proceed rapidly in this anatomical space since no vital structure is encountered.[66]

The Temporal Ligamentous Adhesion

This is a triangular fibrous condensation 1 cm above the craniolateral corner of the orbital rim at the juncture of frontal, temporal, and periorbital areas (▶ Fig. 22.6).[66] Three retaining ligaments radiate from it: the supraorbital ligamentous adhesion medially, the superior temporal septum superolaterally, and the inferior temporal septum inferolaterally.[66]

The Inferior Temporal Septum (Orbicularis-Temporal Ligament)

This is an area of fusion between the superficial temporal fascia (temporoparietal fascia) and the superficial layer of deep temporal fascia extending from the craniolateral corner of the orbicularis oculi to the external acoustic meatus which constitutes this structure (▶ Fig. 22.6).[66] It divides the temporal compartment into superior (see earlier) and inferior temporal compartments; the latter is further bordered caudally by the zygomatic ligaments.[66] This space is surgically significant for housing the frontal branches of the facial nerve, the zygomaticotemporal nerve, and the sentinel veins (▶ Fig. 22.7).[66] The frontal branches of the facial nerve travel medial and parallel to the inferior temporal septum. The septum thus acts as a warning sign during craniocaudal dissection in the temporal region, alerting surgeons to stay strictly on top of the superficial layer of the deep temporal fascia (or deep to it) to avoid nerve injury (▶ Fig. 22.6).[63,66]

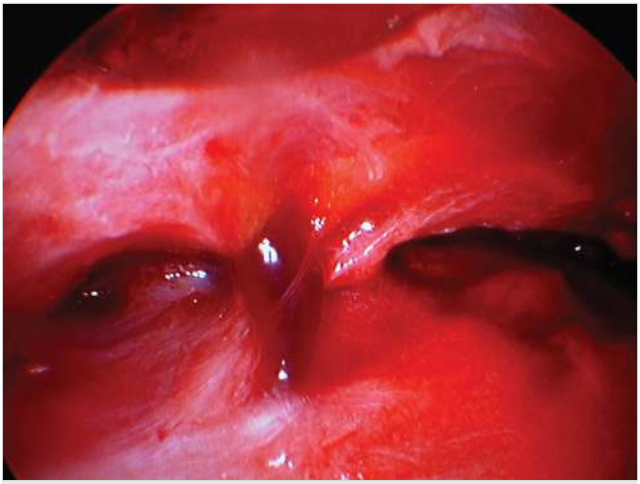


Fig. 22.7 Intraoperative endoscopic view of the sentinel vein. The frontal rami of the facial nerve usually lie within 10mm cranial to the vein.

The Periorbital Area

The Orbicularis-Retaining Ligament (Orbitomalar Ligament)

The anatomical delineation of this structure stems from the clinical observation of constant strong attachments between the orbicularis oculi and the inferolateral orbital rim that often need to be released for adequate periorbital mobilization.[66,67,68,69]

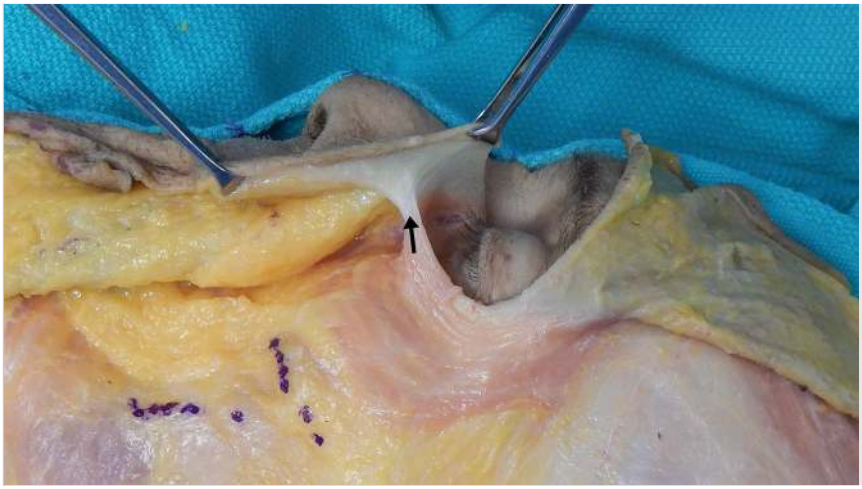


Fig. 22.8 Cadaver dissection of the left lateral orbital region and left side of face, demonstrating the orbitomalar ligament (*blue arrow*).

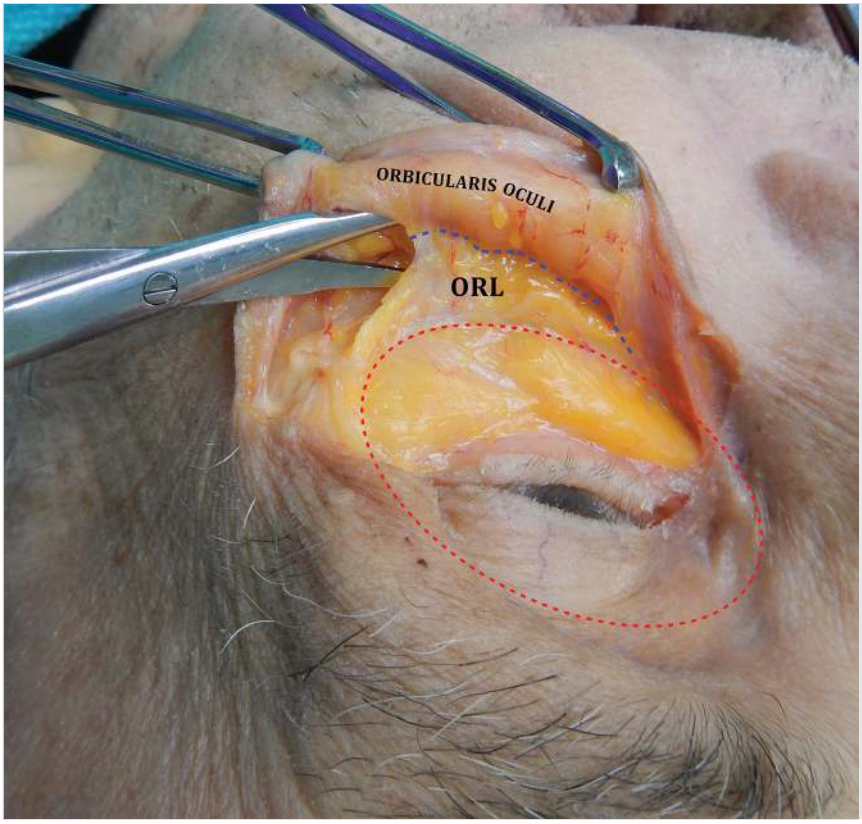


Fig. 22.9 Cadaver dissection of the left orbit view from above. The red dotted line marks the bony orbital margin. The blue dotted line marks the junction of the ORL and the orbicularis oculi muscle. The ORL is in between.

The *orbitomalar ligament* (ORL) is an osteocutaneous ligament arising from the periosteum of the orbital rim and traversing the orbicularis oculi to insert into the dermis of the lid–cheek junction (▶ Fig. 22.8).[68,70] It is not well defined medially where the orbicularis oculi is tightly adherent to the inferior orbital rim (▶ Fig. 22.9).[68] The ligament extends circumferentially around the orbital rim, and thus forms the roof of the preseptal space in the upper eyelid, the floor of the preseptal space in the lower eyelid, and the roof of the prezygomatic space in the midface, which is bordered caudally by the medial extent of the zygomatic ligaments.[67,68,69] Morphologically, the ligament is a bilaminar structure where the cranial leaf is a reflection of the orbital septum, and the caudal leaf a continuation of the fascia covering the preperiosteal fat of the prezygomatic space.[68,69] The zygomaticofacial nerve and foramen always lie peripheral to the ORL.[68,69] The ORL can be thought of as the leash of a dog running from bone to dermis. The leash is tight medially, loose centrally, and again tight laterally; where it fuses with the lateral orbital thickening or superficial canthal tendon, the laxity of the ligament thus creates a V-shaped structure (▶ Fig. 22.9).[68] Lysis of the

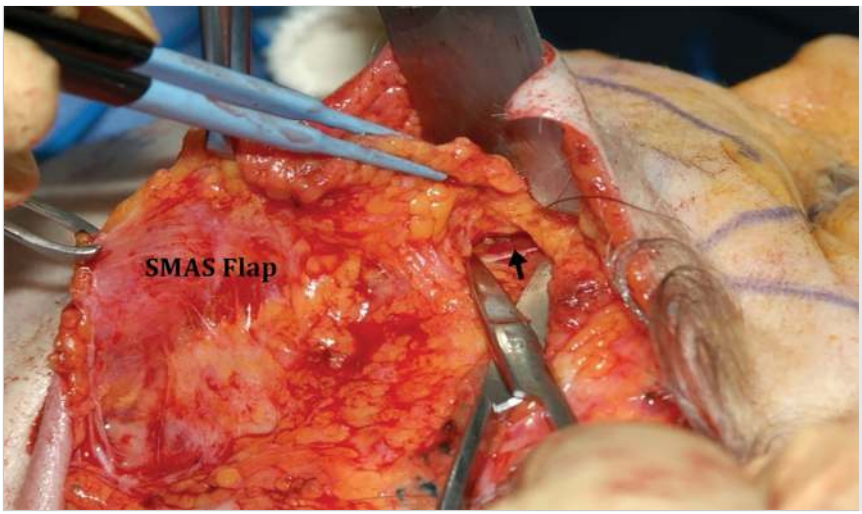


Fig. 22.10 Intraoperative view of sub-SMAS dissection. After release of the major zygomatic ligament, the zygomaticus major (ZM) muscle (black arrow) comes into view and this constitutes an important surgical landmark in all sub-SMAS techniques. The masseteric ligaments inferior to the ZM still need to be released. Note that sub-SMAS fat is kept down protecting the facial nerve branches.

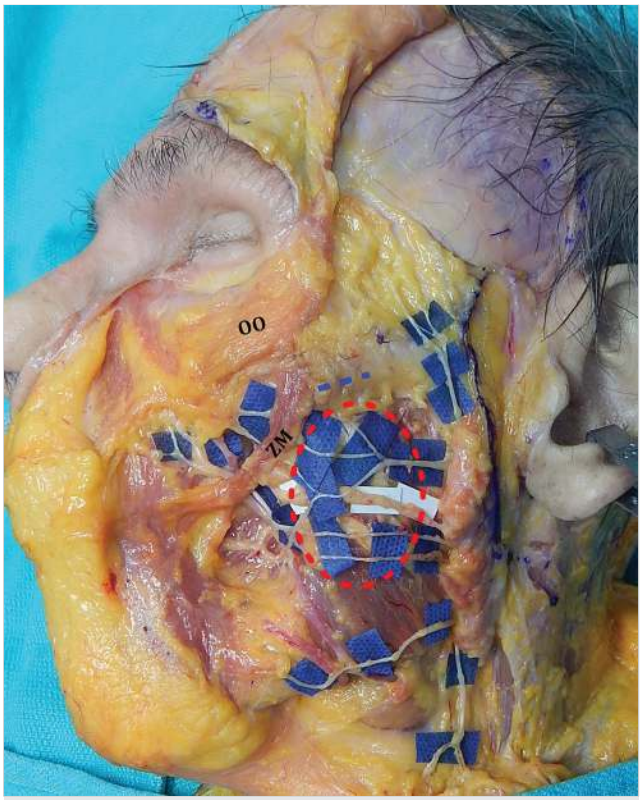


Fig. 22.11 Cadaver dissection of the left face. Note the zygomaticus major (ZM) and orbicularis oculi (OO) muscles. The position of the zygomatic ligaments to the arch and body of the zygoma are highlighted with a blue dotted line. The danger zone is located caudal to the zygomaticus major muscle and is marked with a red dotted circle. The zygomatic and buccal facial nerve branches are located in this area (blue background). Note the parotid duct (white background).

medial orbicularis oculi and varying portions of the ORL have become an essential part of a variety of procedures used to treat the tear trough, palpebromalar groove, and malar mound.[24,69,71,72,73,74,75,76]

The Cheek Area

The zygomatic ligaments arise from the lower border of the zygomatic arch and from the body of the zygoma.[9,10,64] They attach directly to the dermis.[9,10,64] The medial extent of these ligaments forms the floor of the prezygomatic space, while the lateral extent constitutes the lower border of the inferior temporal compartment.[66,67,69] Furthermore, these ligaments act as the roof of the lateral cheek compartment.[63] The major zygomatic cutaneous ligament lies at the junction of the arch and body of the zygoma.[63,64] This ligament is surgically important because when released, the zygomaticus major (ZM) muscle comes into view.[9,10,35] This marks the end of the sub-SMAS dissection. All sub-SMAS procedures invariably pass superficial or subcutaneous at this point, in order to avoid injury to facial nerve branches (▶ Fig. 22.10). [9,17,35,37]

The masseteric ligaments have their origin from the fascia overlying the masseter muscle. Together with the zygomatic ligaments they form a T-shaped configuration (the confluence of the T is at the origin of the ZM muscle).[10,33,63,64] One centimeter inferior to the ZM in the vicinity of the stout upper masseteric ligaments is the sub-SMAS "danger zone," where care needs to be taken when lysing these ligaments to avoid injury to zygomatic branches of the facial nerve (▶ Fig. 22.11).[9,10,63,64] The lower masseteric ligaments or zones of fusion separate the premasseteric space from the masticatory space that houses the buccal fat pad.[10,60,63]

The platysma auricular ligament/fascia is the first ligament severed during a facelift. It is formed of fibrous bands connecting the platysma to the parotid fascia, securing it to the preauricular skin.[9,10,63]

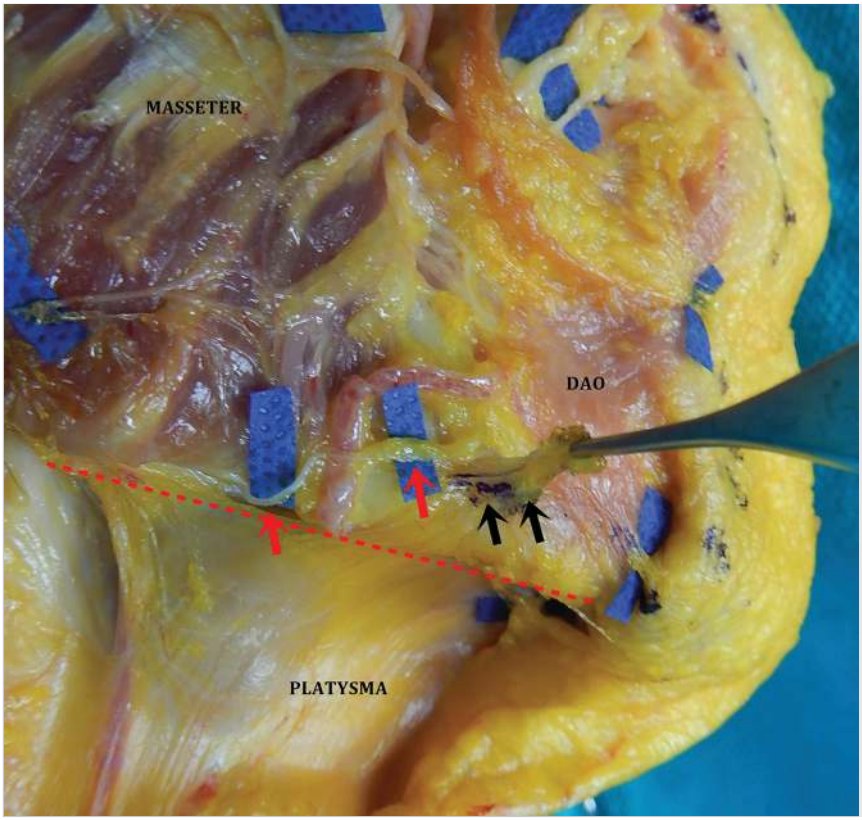


Fig. 22.12 adaver dissection of the right lower face. The red dotted line marks the lower mandibular border. The mandibular ligament is held with forceps (black arrows). The marginal mandibular nerve can be seen crossing superficial to the facial vessels and then travels immediately cephalic to the mandibular ligament (red arrows).

The Mandibular Area

The mandibular ligament is an osteocutaneous ligament arising from the anterior third of the mandible, 1 cm above the inferior border.[9] Its presence marks the anterior boundary of the jowl (▶ Fig. 22.5).[9,10] The marginal mandibular branches of the facial nerve are located posterior and approximately 1 cm cranial to it (▶ Fig. 22.12).[65]

22.7 The Facial Nerve

22.7.1 Beyond the Parotid Gland, Five Groups of Branches Can Be Identified

Frontal (Temporal) Branches

The limited number of communications with other facial nerve branches is thought to account for the higher risk of permanent injury.[77] The classic surface anatomy is the Pitanguy line which extends from 0.5 cm below the tragus to 1.5 cm above the lateral eyebrow.[78] The nerve exists in multiple (2–5) rami that intercommunicate, which may explain the return of function after injury. The branches were found to reside 2 cm posterior to the lateral orbital rim at the level of the lateral canthus.[79,80] At the level of the zygomatic arch, the branches run over the middle third approximately 1.8 cm anterior to the helical crus, and 2 cm posterior to the lateral orbital rim (▶ Fig. 22.13).[79,81] In terms of depth, these branches are closely applied to the periosteum of the zygomatic arch, deep to both the SMAS and the parotid masseteric fascia (parotid temporal fascia).[82] Superior to the arch, they travel on top of the superficial layer of the deep temporal fascia.[82,83,84] Trussler et al and Agarwal et al in separate studies showed the presence of two superficial fascial layers covering the zygomatic arch and the deep temporal fascia.[82,84] The first layer is the superficial temporal fascia, which is in continuity with the SMAS.[82,84] Deeper to it they showed the existence of another distinct layer (the parotid temporal fascia per Trussler et al, and innominate fascia per Agarwal et al).[82,84] The frontal branches travel either deep or within this deeper layer.[82,84] This relationship continues for at least 1.5 to 3 cm above the arch from where the nerves start to transition through the superficial temporal fascia to merge with the anterior branch of the superficial temporal artery as they approach orbicularis oculi and frontalis.[82,84] Appreciation of this anatomy is important during craniocaudal dissections in the temporal region, and in elevating a high SMAS flap.[82] Surgeons should stay strictly on top of the superficial layer of the deep temporal fascia (or deep to it) elevating the frontal branches with the flap. Two centimeters cephalad to the zygomatic arch, dissecting deeper to the superficial layer of the deep temporal fascia has been advocated to avoid nerve injury.[77,82,84] In endoscopic brow lift, the sentinel vein (located approximately 0.5 cm lateral to the frontozygomatic suture) defines a danger zone about 10 mm cephalad, where the frontal branch is liable to injury (▶ Fig. 22.7).[81] A second vein 1.5 cm lateral to the first is occasionally identified.[81]

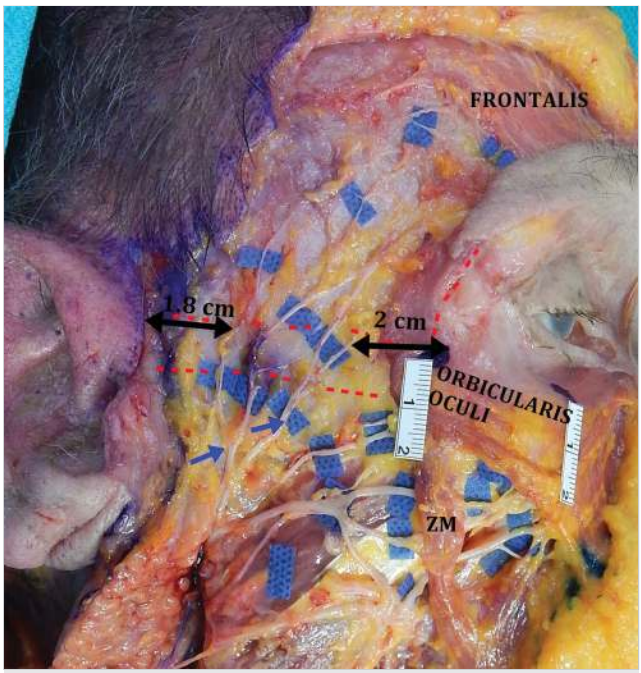


Fig. 22.13 Cadaver dissection of the right side of the face. The frontal rami of the facial nerve have been dissected (blue arrows). The relationship as they cross the zygomatic arch is shown. The arch is outlined with red dotted lines.

Zygomatic and Buccal Branches

These branches display higher number of rami and interconnections which may account for spontaneous recovery following injury (▶ Fig. 22.3 and ▶ Fig. 22.11).[77] The buccal ramus is thought to be the most frequently injured facial nerve branch in rhytidectomy.[36,77] Due to extensive arborization, clinical effect is usually short-lived and rarely discernible.[36,77] Injury to the branches that supply the elevators of the upper lip (e.g., ZM), however, can potentially affect smile. The area inferior to the zygomatic ligaments and anterior to the upper masseteric ligaments is a danger zone[9,10,63,77] (▶ Fig. 22.9). Thus, sub-SMAS dissection in this region should proceed with extreme caution, always hugging the undersurface of the SMAS and keeping all fat down.

Marginal Mandibular Branch

The limited number of rami and communications with other facial nerve branches is thought to account for higher risk of permanent injury (▶ Fig. 22.11 and ▶ Fig. 22.12).[77] Anatomical studies have shown the nerve to be composed of at least two rami. Posterior to the fascial vessels they travel within 1 cm (rarely 2 cm or more) of the mandibular border (more frequently cephalad than caudad).[65,77,83,85] In contrast, anterior to the facial vessels the marginal mandibular is always cranial to the mandibular border traveling toward the depressor anguli oris.[77,83,85] Here, it lies posterior and approximately 1 cm cranial to the mandibular ligament[65] (▶ Fig. 22.10). In terms of depth, the nerve emerges from the lower end of the parotid gland and travels deep to the parotid-masseteric fascia, and under cover of platysma.[65,77,83,85] Inferior to the mandible it courses on the surface of the posterior belly of digastric and submandibular gland remaining deep to platysma.[77,83,85] At the level of the facial vessels, the nerve crosses the mandibular border superficial to these vessels. It is at this point that the nerve is believed to be at higher risk of injury as it becomes more superficial.[77] The nerve remains deep to the platysma throughout its course, and thus keeping dissections above the platysma is a safe dissection plane. In sub-SMAS dissections, the lower masseteric ligaments are a sign of caution to the proximity of the marginal mandibular branches.

Cervical Branch

Injury to this branch is believed to result in marginal mandibular pseudo-paralysis secondary to dysfunction of the platysma which is likewise a depressor of the lower lip (▶ Fig. 22.11).[86,87] Clinical differentiation is possible by demonstrating retained ability to pucker the lower lip, which signifies residual orbicularis oris and mentalis function.[86,87] Return of function is almost always complete.[86,87] This nerve (composed of one to three rami with occasional communication to the marginal mandibular) travels close to the gonial angle and branches approximately 1.74 mm inferior to the mandibular border.[88,89] Platysmal incisions should be designed at least 15 mm posterior to the gonial angle, and 45 mm caudal to the mandibular border to avoid cervical nerve injury.[88,89] It emerges from the lower border of the parotid gland and remains deep but close to the undersurface of the platysma.[77,88,89] Thus, subplatysmal dissection in proximity to the gonial angle should proceed with caution staying superficial to the underlying fat. Blunt dissection has been advocated in this plane inferior to the tail of the parotid to minimize injuries of the marginal mandibular and cervical branches.[77,89]

Sensory Nerves

The great auricular nerve (C2 and C3) is thought to be the most commonly injured nerve in rhytidectomy.[90] Arising from the cervical plexus, it winds around the posterior border of the sternocleidomastoid, and passes vertically upward toward the middle of the lobule of the ear.[91] The classic surface landmark is McKinney's point which lies 6.5 cm caudal to the external acoustic meatus approximately 0.5 to 1 cm posterior to the external jugular vein.[90] Ozturk et al have identified a triangle, where the nerve can be found 100% of the time.[92] The triangle is defined by a line perpendicular to the Frankfort horizontal bisecting the lobule of the ear and another diverging 30 degrees posterior to it (▶ Fig. 22.14).[92] Maintaining the dissection superficial to the sternocleidomastoid fascia in this region prevents nerve injury and consequent neuroma formation.[92]

22.8 Operative Techniques

22.8.1 Incisions

The Preauricular Part

A preauricular retrotragal incision results in less discernible postoperative scarring.[32] If properly executed, the aesthetic outcome is excellent. The potential for tragal deformity is readily

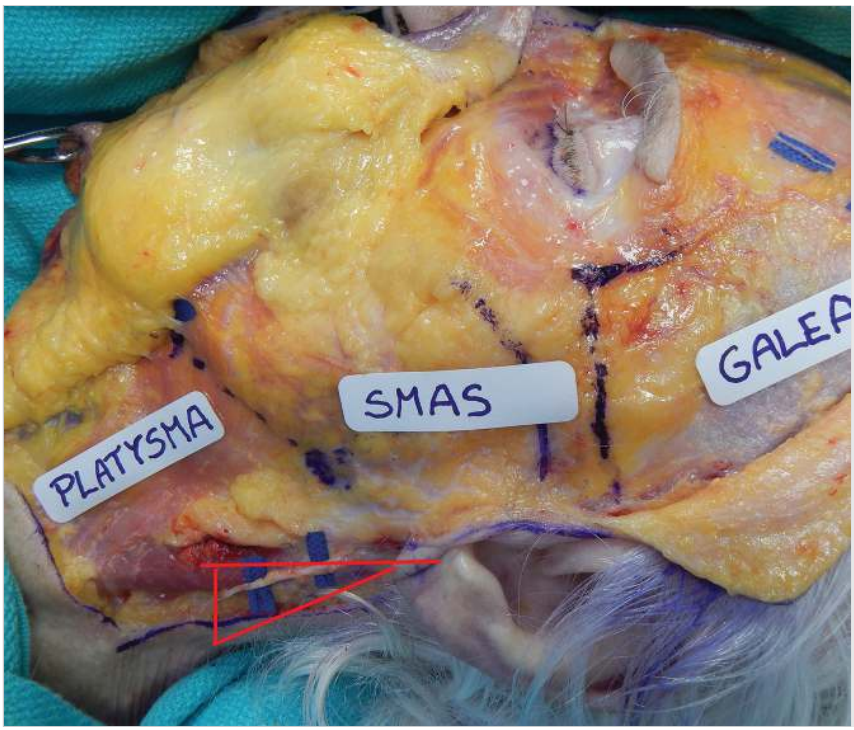


Fig. 22.14 Cadaver dissection of the left face. A dissection of the great auricular nerve is shown. Ozturk's triangle is defined by a perpendicular to the Frankfort horizontal bisecting the lobule of the ear and another line diverging from it at 30 degrees starting at the tip of the lobule.

preventable if care is taken to avoid cartilage injury and ensure tensionless closure (▶ Fig. 22.15).[32]

The Temporal Extension

An outer canthus to temple distance less than 5 cm permits a temporal scalp extension. A distance greater than 5 cm mandates a temporal hairline incision to avoid aggravation of temporal recession (▶ Fig. 22.15).[32,47,48,93]

The Postauricular Extension

The incision follows the postauricular sulcus and crosses the postauricular skin where the helix and postauricular hairline touches (Feldman's "touch point").[94] It either continues horizontally into the occipital scalp or follows within the occipital hairline.[32,55,94] The latter prevents posterior hairline recession especially if neck skin laxity is profound (▶ Fig. 22.16).[32,55,94]

Submental Incision

A 3.5-cm horizontal incision is centered over the midline approximately 5 mm caudal to the submental crease if anterior cervicoplasty/platysmaplasty is contemplated.[41,42]

22.8.2 The Extended SMAS Facelift (Senior Author's [JZ] Preferred Technique)

Principle

The extended SMAS technique delaminates the face by raising separate skin and SMAS flaps.[35] The independent mobility of SMAS and skin permits greater versatility in vector selection and direction of tissue repositioning.[34,36,37] Precise control of dissection plane and skin flap thickness is critical, however, to avoid tearing of the SMAS flap.[34,37] The SMAS thins considerably as dissection extends medially and SMAS tearing can complicate flap fixation.[8,34,37,46]

Dissection

The Temporal Region

It is prudent to stay on top of the superficial temporal fascia. This is the layer immediately deep to the hair follicles. Proceeding anteriorly, the orbicularis oculi muscle is identified, and more caudal dissection should continue superficial to the plane of this muscle (▶ Fig. 22.17).[32]

The Preauricular and Cheek Part

Immediately anterior to the helical crus, dissection should be very superficial (subdermal), to avoid injury to the superficial temporal vessels; this part of the skin is usually discarded anyway except in secondary cases. At the tragus, caution is necessary to prevent cartilage injury. In the cheek area (from the zygomatic arch to the mandible), dissection should be more superficial to preserve fat on top of the SMAS, thus providing a thicker SMAS flap.[32,35] Moving across the mandible to the neck, the plane transitions smoothly to a slightly deeper level. Dissection anterior to the sternocleidomastoid muscle should be hugging the superficial surface of the platysma (▶ Fig. 22.18).

The Postauricular Part

Skin should be elevated immediately superficial to the sternocleidomastoid fascia. The great auricular nerve is protected

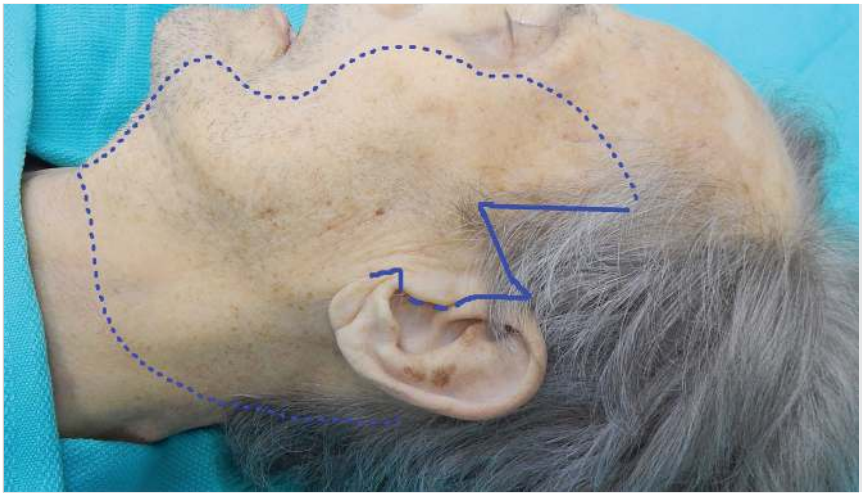


Fig. 22.15 The design of the preferred preauricular retrotragal incision by the senior author is depicted in *blue*. The *dotted line* along the central face and neck represents the usual extent of skin undermining as performed by the senior author.

Fig. 22.16 The design of the preferred postauricular incision by the senior author is marked in *blue*. The horizontal *blue* mark in the neck marks the extent of undermining in the neck.

by superficial dissection over Ozturk's triangle (see above; ▶ Fig. 22.14).[92]

The Neck

The neck dissection continues immediately on top of the platysma and is taken across the midline in the majority of cases.[39,94]

The SMAS Flap and Sub-SMAS Dissection

Outline of the SMAS Flap

The Upper Border

Different variations in designing the upper border have been described. While Stuzin et al place it 1 cm caudal to the zygomatic arch, others incise at or above the level of the arch.[32,35,36,37] It is our preference to draw this line flush with the lower border of the zygomatic arch in order to capture most of the malar fat pad in the SMAS flap. The proximity of the frontal branches, their surface anatomy, and their depth (see section 22.6 Anatomical Considerations) should be appreciated.

The Lateral Border

This line is designed approximately 1 to 2 cm in front of the ear and continues downward for 4 to 5 cm into the neck (at least 1.5 cm lateral to the gonial angle to avoid the marginal mandibular nerve). This line generally coincides with the posterior border of the platysma.[32,35,36,37]

SMAS Flap Elevation

Starting sub-SMAS dissection over the parotid gland mitigates potential nerve injury, since parotid tissue serves as an extra layer protecting the nerves. The SMAS is also thicker, muscular, and more distinct in this region (▶ Fig. 22.10). Anterior to the parotid, it is critical to hug the deep surface of the SMAS flap, maintaining ***all*** fat over the masseteric fascia protecting the facial nerve branches lying deep (▶ Fig. 22.10). Once the proper plane is identified, more cephalad dissection can be undertaken. Malar SMAS is mobilized severing zygomatic and upper masseteric ligaments with extreme care (see section 22.6 Anatomical Considerations).[32,35,36,37] Amidst this dissection, ZM muscle is identified (▶ Fig. 22.10).[32,35] Dissecting medial to ZM disconnects any remaining zygomatic ligaments, while caudal to it, the masseteric ligaments are split.[35] Maintaining the dissection superficial to the plane of this muscle protects the nerves. Using blunt spreading motion, dissection continues to the nasolabial crease.[35] This maneuver ameliorates the depth of the crease and simultaneously lifts the malar fat pad.[35] Further, malar fat mobility can be achieved using finger dissection in the same plane: "FAME" (finger-assisted malar elevation).[95] An effective SMAS flap should improve the jowl and the neck and reposition the malar fat pad.[32,35] The SMAS elevation has a decidedly positive and powerful effect on platysma tightening and gonial angle improvement because the platysma is continuous with the SMAS and has no bony attachments.

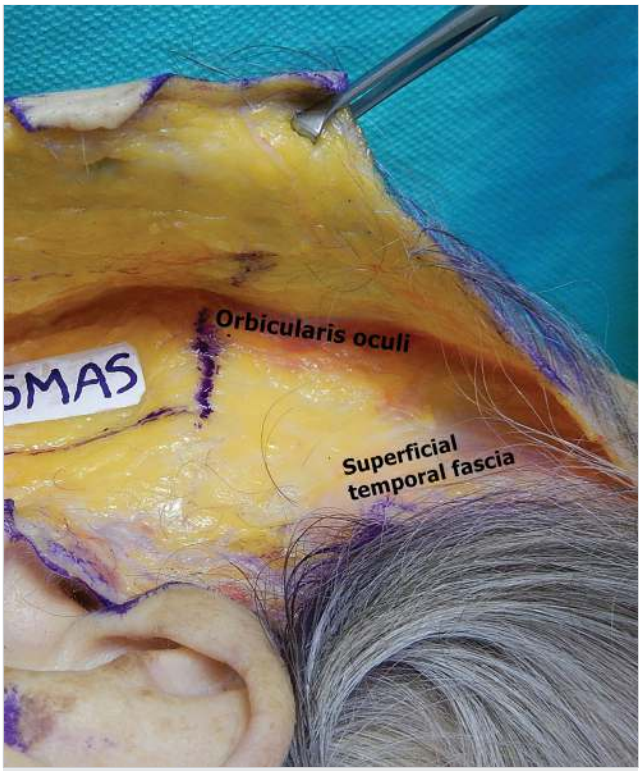


Fig. 22.17 Cadaver dissection of the left face. The undermining in the temporal area is superficial to the superficial temporal fascia until the orbicularis oculi is identified. More caudal dissection should maintain the same plan.

SMAS Flap Fixation

Temporal Fixation

Before fixation, it is useful to draw the trajectory of the frontal branches to avoid inadvertent injury. The vector of SMAS-flap transposition is vertical (▶ Fig. 22.19). The exact anchor point is determined by pulling on the flap and observing the effect on the face and neck.[32] Once determined, blunt scissor spreading exposes the deep temporal fascia for safe and robust fixation.

Cervical Fixation

The lateral (posterior) edge of the SMAS flap is incised from tragal level to the level of the gonial angle creating a tongue of inferiorly based SMAS flap (▶ Fig. 22.20a). This is rotated posteriorly and fixed to the mastoid fascia and periosteum pulling

Fig. 22.18 Cadaver dissection of the left face showing the plane of undermining in the cheek area and the neck. Note the plane in the neck (left side of the figure) is immediately supraplatysmal. In the cheek, the plane is more superficial leaving fat on top of the SMAS. A transition from the more superficial to the deeper plane occurs across the mandibular border.

Fig. 22.19 The SMAS flap is elevated vertically (*blue dotted line*). In this case, a 2.5 cm of vertical SMAS-flap advancement above the zygomatic arch was documented.

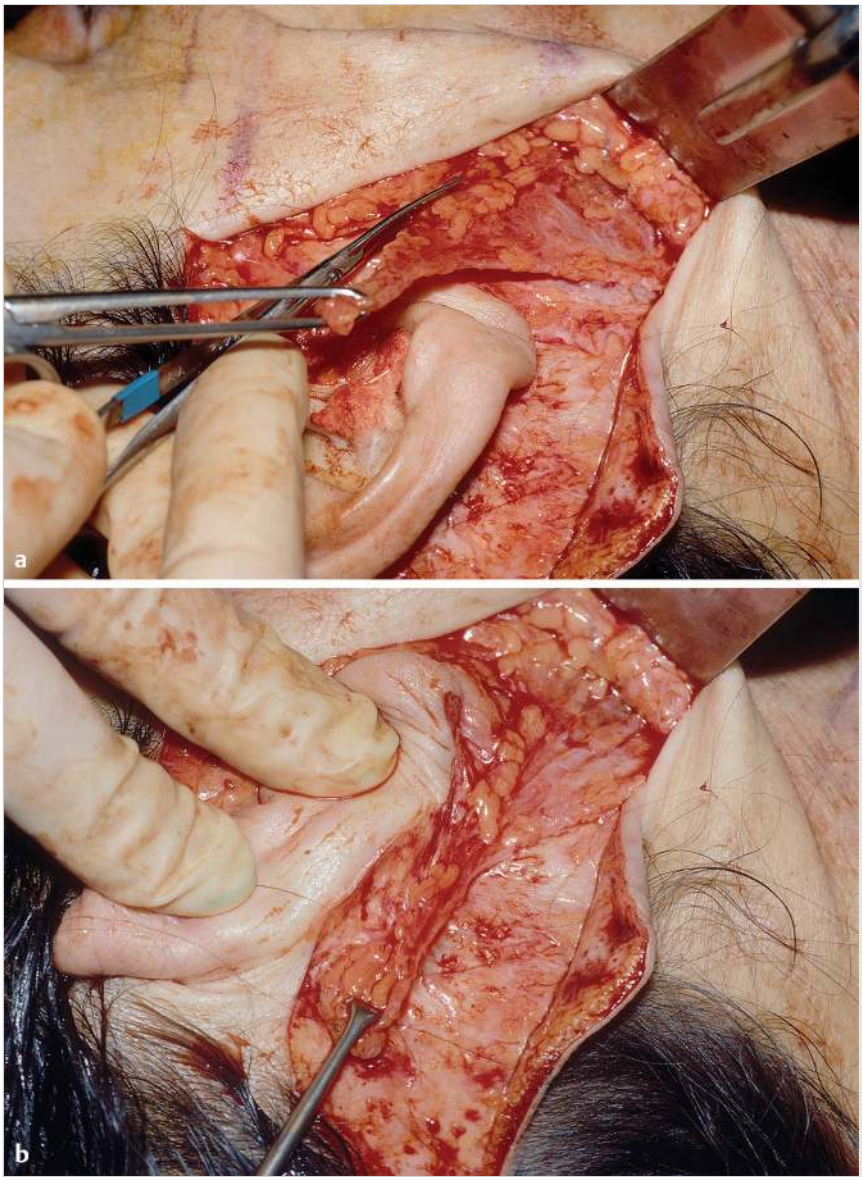


Fig. 22.20 **(a)** Intraoperative view showing the incision of an inferiorly based tongue from the parent SMAS flap. **(b)** This is rotated and fixed behind the ear to mastoid periosteum.

Fig. 22.21 **(a,b)** Preoperative front and profile photograph of a 59-year-old woman. **(c,d)** Postoperative front and profile view 2 years after extended SMAS facelift combined with anterior neck lipectomy and corset platysmaplasty. Note improvements in the neck.

the platysma horizontally (the vector of neck tightening is horizontal; ▶ Fig. 22.20b).[35]

22.8.3 Management of the Skin

The undermined skin/subcutaneous flap is gently pulled perpendicular to the nasolabial crease, redraping skin without tension. The ear lobule is inserted first, by incising the flap at this level parallel to the mandibular border. A permanent suture at the helical crus and another at the cephalic end of the postauricular incision sets the skin tension. Skin resection and flap inset then continues as feasible.[32] Extensive skin undermining in the neck in the superficial plane combined with anterior lipectomy, platysmaplasty, and extended SMAS dissection and fixation in a vertical direction addresses the malar, cheek, and neck areas and results in long-lasting results (▶ Fig. 22.21).

22.8.4 The MACS Lift

Principle

The **MACS** ("**m**inimal **a**ccess **c**ranial **s**uspension") technique consists of a limited incision with restricted subcutaneous undermining (The "**MA**" part).[51,53] Purse string sutures in loop configuration elevate and tighten the SMAS and platysma by imbrication and suspension to the deep temporal fascia (the "**CS**" part).[51,52,53] The aim is to address laxity and contour without the risks of deep dissections.[51,52,53] Submental/submandibular liposculpture and facial microfat grafting are important adjuncts.[55] The procedure is performed using a short scar technique. Conceptually, the procedure is based on posterior vertical vectors of correction and the absence of "delamination" of planes of dissection.

Incision

The MACS incision differs by lack of a postauricular component.[53] The temporal incision is designed in a zigzag manner to accommodate skin excess.[51,52,53] For the ***standard*** MACS lift, the temporal incision ends opposite the external canthus, while for the ***extended*** MACS lift it terminates opposite the tail of the brow.[51,52,53,54,55]

Dissection

Subcutaneous skin undermining is limited to 5 cm anterior to the ear and up to the mandibular border.[51,52,53,54,55] It is critical to extend the neck dissection until the craniolateral part of the platysma is visualized.

The Anchor Point

This is placed 1 cm above to the zygomatic arch and 1 cm anterior to the helical rim. This point is anterior to the superficial temporal vessels and posterior to the course of the frontal nerve. Blunt scissor spreading allows identification of the deep temporal fascia as a glistening white shiny layer.[51,52,53,54,55]

The Neck Loop

This is a vertical narrow (1 cm wide) **U**-shaped purse string loop. It starts at the anchor point, and proceeds inferiorly along the pretragal sulcus down to craniolateral part of the platysma.[51,52,53,54,55] From there it turns upward again toward the anchor point. Stitches are placed in an imbricating fashion and tissues should bunch-up upon tightening correcting cervico mental angle and upper neck. Grasping several bites of the craniolateral platysma in the neck is critical (▶ Fig. 22.22).[51,52,53,54,55]

The Cheek Loop

A wider "**O**"-shaped loop is designed at 30 degrees to the neck loop. It extends downward in the direction of the jowls. Again bites are placed grasping the SMAS in an imbricating fashion leading to bunching of tissue and improving jowls, marionette lines, and corner of the mouth (▶ Fig. 22.22).[51,52,53,54,55]

The Malar Loop

The malar suture is optional and constitutes the ***"extended"*** MACS. The anchor point lies 1.5 cm lateral to the external canthus. It can be either deep temporal fascia or periosteum of the frontal process of the zygoma (medial to the frontal rami).[51,52,53,54,55] Subcutaneous dissection is performed above the temporoparietal (superficial temporal) fascia until the orbicularis oculi vertical fibers are identified. A window is created through these fibers by spreading scissors. Next, a small and narrow **U**-shaped loop grasps the edge of the malar fat pad, which is usually 2 cm below the lateral canthus. The pull should be as vertical as possible to achieve malar fat pad–midface lift.[51,52,53,54,55] Secondary skin bunching of the lower lid mandates a pinch blepharoplasty.[54] More recently, Tonnard has discontinued the extended MACS in favor of fat transfers to the malar area (▶ Fig. 22.23).[96]

Fig. 22.22 Cadaver simulation of the MACS lift. Note the direction and design of the **U**-shaped neck loop and the **O**-shaped cheek loop. Also, note the anchor point position (*black arrow*).

Flap Advancement and Skin Resection

Skin redraping is purely vertical with all skin excised in the temporal region under no tension. No skin should need to be resected in the preauricular area. If significant skin is excised preauricularly, the vector of pull is incorrect. The zigzag design facilitates accurate and tensionless closure.[51,52,53,54,55]

22.8.5 Lateral SMASectomy

Principle

The technique consists of SMAS excision and closure at the anterior border of the parotid gland via a short scar incision.[45,46,47,48,49] Following resection, loose cheek SMAS is sutured to more fixed SMAS overlying the parotid.[45,46,47,48,49] Patients with thin faces benefit from SMAS plication rather than resection. Based on skin/neck laxity, patients with minimal laxity are excellent candidates and the procedure is preceded by closed liposuction of the neck and jowls.[48,49] Patients with significant

Fig. 22.23 Marking of the three MACS loops on the skin of a cadaver. Note the position and direction of the malar loop comprising the extended MACS lift.

Fig. 22.24 A cadaver model showing the extent of undermining for a lateral SMASectomy procedure.

laxity are better served with a standard facelift in addition to a submental incision and platysmaplasty.[48,49] Baker graded facial aging by grades I to IV and alters his approach accordingly.[48,49]

Dissection

Skin dissection is taken to the lateral canthus separating orbicularis oculi fibers from the skin flap. The cheek flap is dissected anteriorly releasing zygomatic and masseteric ligaments as well as the mandibular ligament if necessary.[48,49] Dissection stops well short of the nasolabial fold. Fat is left on the skin flap. Dissection is taken 5 to 6 cm into the neck over the platysma and sternocleidomastoid muscle.[48,49] If a submental incision is made, dissection is extended across the midline and the platysmaplasty is performed in grade IV (severe facial aging patients; ▶ Fig. 22.24).[48,49]

The SMASectomy

The lateral SMASectomy is performed from lateral canthus to gonial angle and into the neck. Depending on the degree of laxity, a strip (2–4 cm) of superficial fascia is resected starting inferiorly.[46,47,48,49] The direction of SMASectomy is parallel to the nasolabial fold (▶ Fig. 22.25). Care is taken to resect superficial fascia only since the deep fascia protects the facial nerve (▶ Fig. 22.25).[46,47,48,49]

SMAS Closure

Interrupted sutures approximate distal loose SMAS to proximal fixed SMAS in a posterior and cranial vector (▶ Fig. 22.26). The first suture approximates the platysma at the gonial angle and the final suture lifts the malar fat pad.[46,47,48,49] The orientation of the SMASectomy can be varied to provide more vertical lift superiorly when indicated.

Skin Management

Skin closure is accomplished by advancing skin in a more horizontal direction. Skin resection is performed under minimal tension. Skin edges should kiss at completion of procedure.[46,47,48,49] Clearly, ligaments are not released deep to the SMAS. Baker feels this is unnecessary in the lower face where there is enough "play" in the ligaments.

22.8.6 The Composite Rhytidectomy

Principle

A composite flap containing SMAS, malar fat pad, and orbicularis oculi is elevated.[17] The lateral vector of the flap is counterbalanced by superomedial malar fat repositioning via a subciliary incision using the orbicularis oculi as a vehicle.[25,97,98,99] The aim is to overcome the "lateral sweep" and "hollow eye" phenomena of the traditional facelift.[25] Significant redundancy secondary to malar lift mandates obligatory forehead and brow lift, which are combined with an upper lid blepharoplasty.[99,100] The obligatory forehead lift is accomplished via either an open coronal or a subgaleal hairline approach (according to forehead length); endoscopic techniques are not utilized.[99,100] Arcus marginalis release and septal reset are now integral components of the procedure.[99,100,101] A submental incision and platysmaplasty are always used to address the neck.[99,100,101]

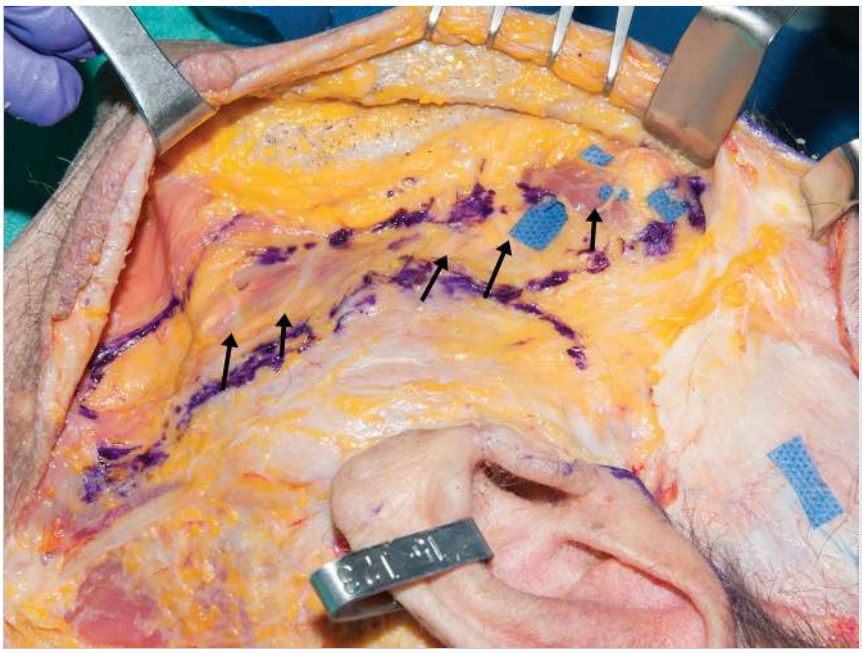


Fig. 22.25 Same cadaver model as in ▶ Fig. 22.24. After the SMASectomy is performed, note that the excision is anterior to the parotid gland and parallel to the nasolabial crease. Also, note the facial nerve branches and the zygomaticus major muscle (*black arrows*).

Fig. 22.26 Same cadaver model as in ▶ Fig. 22.24 and ▶ Fig. 22.25. Showing the SMAS tightening after the SMASectomy closure approximating loose SMAS anterior to parotid to fixed SMAS overlying the parotid.

Marking

The markings include the jawline, the gonial angle, and the course of the zygomaticus muscles (major and minor) from the malar eminence to the corner of the mouth and alar base (▶ Fig. 22.27).[100]

Incisions

The following incisions are necessary: (1) hairline or coronal browlift incisions, (2) upper and lower blepharoplasty incisions, (3) temporal scalp (temporal hairline incision is never utilized), (4) periauricular facelift incisions, and (5) submental incision.[100]

Dissection

Composite rhytidectomy includes dissections in the following planes: (1) subgaleal forehead/temporal dissection, (2) zygo-orbicular dissection through a subciliary/lower eyelid incision, (3) deep/sub-SMAS dissection in the face, and (4) subcutaneous/preplatysmal neck dissection.[20,21,23,24,99,100,101]

An upper blepharoplasty is completed first leaving the lateral incision open for later lower orbicularis suspension to the periosteum of the lateral orbital rim.[100] Through a lower blepharoplasty, strict suborbicularis oculi dissection is continued deep to ZM ("zygo-orbicular dissection") in a pre-periosteal plane using scissor spreading or occasionally a "Kitner" to avoid potential nerve injury (this dissection should not detach the origin of the ZM from the zygoma).[23,100,101] This is followed by arcus marginalis release and septal reset.[21,24,101] A laterally based orbicularis oculi muscle flap is developed for later fixation to the lateral orbital rim (to achieve the malar lift).[100] A lateral canthopexy or canthoplasty is mandatory.[100] A mesentery is preserved between the zygo-orbicular dissection and the face-lift dissection. This mesentery contains the branches of the nerves and orbicularis oculi muscle and is called the "meso-orbicularis."[23,100] The neck dissection is performed in a preplatysmal plane from the jawline

Fig. 22.27 Cadaver model showing the marking for the composite facelift on the left hemiface.

Fig. 22.28 Same cadaver as ▶ Fig. 22.27. Initial dissection shows the mesotemporalis (*red arrow*). The line of incision to enter the sub-SMAS plane is marked *blue*. Once dissection in the sub-SMAS plane is accomplished, the neck will be separated from the face by the mesomandibularis (*black arrow*).

to the inferior cervical crease.[100] A mesentery of tissue "meso-mandibularis" is maintained undissected over the gonial angle and along the mandibular border, separating neck and face dissections.[17,19,99,100] The preauricular subcutaneous dissection transitions to a deep sub-SMAS plane from a line joining the malar eminence to the jawline (a point 2 cm anterior to the ear lobule; ▶ Fig. 22.28).[17] The dissection in the lower face proceeds past the facial vessels.[17] In the upper face, the identification of ZM is critical (▶ Fig. 22.29).[17] As with all sub-SMAS techniques, dissection continues superficial to the ZM using scissor spreading motion to and past the nasolabial fold.[17,18,101,102]

A mesentery "meso-temporalis" separates the face and forehead dissections and contains the frontal branches and the anterior branch of the superficial temporal vessels.[99,100]

Closure

The cheek lift (the laterally based orbicularis oculi muscle flap) takes precedence.[100] The forehead and preauricular closures are simulated with temporary towel clamps, to judge the tension on the cheek lift precisely.[100] The laterally based orbicularis oculi muscle flap is passed under the lateral raphe and sutured to the lateral orbital rim through the ***upper*** blepharoplasty incision.[100] This is followed by closure of postauricular, preauricular, and submental incisions after judicial skin trimming.[100] Forehead closure proceeds next while the lower eyelid skin excision and suturing is performed last.[100] As emphasized by Hamra, "extraordinary tension is exerted for all closures particularly the orbicularis to periosteum approximation."[17,23]

22.8.7 The Neck (Senior Author's [JZ] Preferred Technique)

Direct neck procedures include the following: (1) defatting superficial to the platysma, (2) subplatysmal and interplatysmal defatting, (3) medial platysmaplasty/platysma tightening, (4) platysma plication over the submandibular gland, (5) submandibular gland resection, and (6) alloplastic anatomical chin implant placement when indicated.[2,41,42]

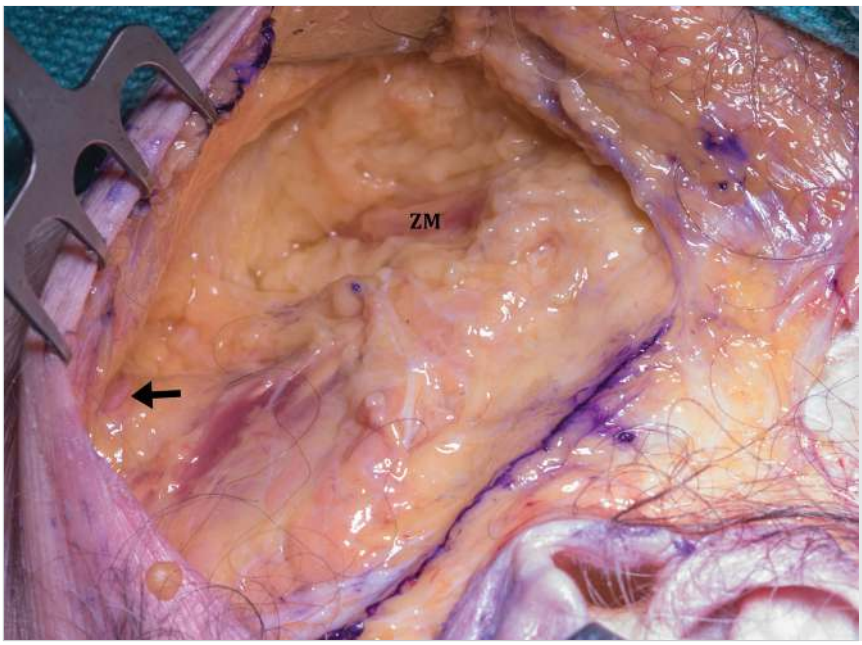


Fig. 22.29 Same cadaver as ▶ Fig. 22.27 and ▶ Fig. 22.28. Note the sub-SMAS dissection is limited by the zygomaticus major (ZM) muscle in the upper face and the facial vessels in the lower face (*black arrow*).

Operative Technique

Surgical manipulation of the aging neck starts from the lateral facelift approach. We prefer extensive lateral strict preplatysmal dissection through the periauricular facelift incision, which we almost always take across the midline.[2] This greatly facilitates subsequent submental dissection. During subcutaneous undermining of the cervicofacial flap, we try to achieve a smooth transition from the cheek where we keep extra fat on top of the SMAS to the neck where we dissect strictly preplatysmal. This transition occurs across the mandibular border and invariably results in some preplatysmal fat remaining immediately below the mandibular border.[2] Some of this fat is transferred to the face during cranial SMAS flap mobilization. Thorough removal of all remaining fat below the mandibular border and gonial angle enhances jawline definition.[2,41,42] A 3.5-cm-long submental incision is centered over the midline, approximately 5 mm caudal to the submental crease.[42] Dissection is taken through the subcutaneous tissue identifying the medial borders of the platysma muscle. It is possible to inadvertently get too deep and transect the platysma especially in revision cases. Extensive preplatysmal dissection from the lateral approach across the midline facilitates later correct plane identification through the submental incision.[2] The undersurface of the platysma is dissected from underlying fat by dissecting the medial edges of the platysma laterally until the anterior bellies of the digastric muscles are identified. Interplatysmal/subplatysmal fat is removed flush with the digastric muscles (▶ Fig. 22.30).[2,41,42] Fat removal extends inferiorly down to the thyroid cartilage. Fat distribution is in the shape of an inverted "T" with fat extending laterally as dissection continues inferiorly. If the anterior bellies of the digastrics are full, they can be shaved flush with subplatysmal fat.[2,41,42] The medial edges of the platysma are sutured together either using interrupted or continuous 3–0 PDS suture. Our preference is a running layer of platysma plication down to thyroid cartilage and up again to the chin outside the first layer using the same suture progressively tightening the "corset" (corset platysmaplasty of Feldman).[39,94] The open approach to the submental area is controversial. Many excellent facelift surgeons frequently avoid the anterior approach to the platysma, as it does increase complications.[103] But in our opinion, it is critical to consistently excellent results in most patients.[39,41,42,94] Subplatysmal fat is to be excised flush with anterior digastrics so as not to create a cavity between the muscles. A midline ridge is avoided by careful approximation of the platysma, taking small bites when suturing the platysma closed. When the platysmaplasty is well done, the patients will complain of tightness in the neck postoperatively. This invariably resolves in a manner of several weeks. If the platysma plication stops short of the thyroid cartilage bands will recur below the plication. The corset platysmaplasty does not include horizontal platysma transection. Transection is contrary to the corset concept as described by Feldman.[39,41,42,94]

22.9 Postoperative Care

At the completion of the procedure, a gauze padded, mildly compressive, cheek and head wrap inclusive of the neck is placed. The head is kept elevated the first 24 hours to minimize facial edema. Elevation is continued as much as possible for the first 1 to 2 weeks after the procedure. The compressive wrap and dressing is removed the day following the operation, and examination is focused on hematoma, facial nerve function, and flap viability. Most surgeons continue use of elastic neck and cheek support for the first week after surgery to minimize edema in the neck and lower face. Physical restrictions are maintained for the postoperative period to minimize facial edema and reduce the risk of incisional dehiscence. Gradual resumption to normal activities is permitted after about 3 weeks. Strict sun avoidance is necessary for 1 month, after which sun protection is maintained.

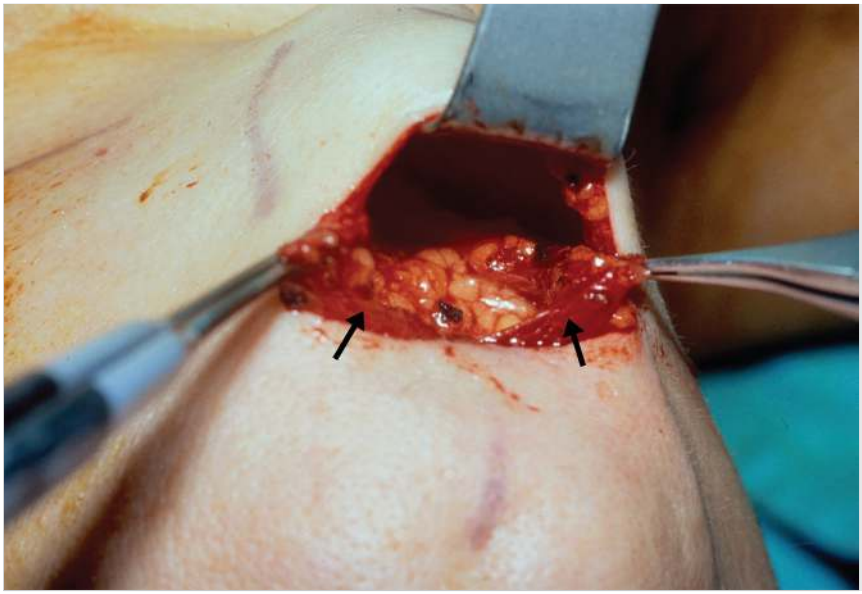


Fig. 22.30 Intraoperative picture. Bird's eye view of the submental dissection. Note the medial platysmal edge held in forceps (*black arrows*) and the subplatysmal fat in between. Fat is excised flush with the digastric muscles.

22.10 Outcomes

22.10.1 Early Complications

Hematoma

The incidence of hematoma varies greatly, from as low as 1% to higher numbers.[3,48,94,103,104,105,106,107,108,109,110] Preoperative screening for bleeding diathesis and relevant medications in addition to meticulous surgery can reduce the risk.[3,103,104,106] Maintaining blood pressure at or above baseline before closure helps recognize potential postoperative bleeding as rebound from epinephrine effect wanes.[94] Avoidance of postoperative hypertension, nausea, and vomiting is also critical.[103,106,111] Platelet-rich plasma and fibrin glue have been utilized to minimize bleeding complications, but evidence of effectiveness is still debatable.[103,106] An expanding hematoma requires immediate evacuation due to the risk of airway compromise (if subplatysmal in the neck) and flap ischemia. Small hematomas can be missed because of facial edema and lead to postoperative contour irregularities. Large hematomas should be evacuated in the operating room, while small blood collections can be cleared through minimal interventions (e.g., milked out through drains or aspirated).

Seroma

Seromas occur most frequently in the neck region and if unrecognized they can lead to contour irregularities that are difficult to correct. Although controversial, many believe suction drains obliterate the dead space and minimize fluid collection. If seroma forms after drain removal, repeated aspirations are mandatory until the seromas subside.

Sialoma

Sialoma is a rare complication that may occur after a sub-SMAS operation or submandibular gland resection. Clear fluid drainage should raise suspicion and a parotid fistula is diagnosed if a high amylase level is found. Obliterating the dead space with suction drains rather than repeated aspirations is the most effective management strategy. In the absence of distal obstruction, the leak will invariably stop.

Facial Nerve Injury

Permanent facial nerve paralysis is rare. The incidence is 0.4 to 2.6%.[83] The most common branches are the marginal mandibular and frontal branches.[77,83] Since injury is usually distal and spontaneous resolution is common, surgical exploration is not indicated.[77,83] Botulinum toxin to the contralateral muscles is a reasonable temporizing measure to achieve symmetry until recovery.

Flap Ischemia

This usually occurs in the postauricular area and frequently follows a hematoma. It is less common with deep plane (composite) rhytidectomy. When suspected, limited suture removal to relieve tension can result in improvement. Once established, expectant management and reassurance is sufficient. Scar revision may be necessary later.

22.10.2 Late Complications

These are generally aesthetic and include (1) contour irregularities that can result from asymmetric fat excision and inadequate skin undermining; (2) unmasking of ptotic submandibular glands; (3) pixie ears (avoid any tension on the lobule during closure); (4) unsatisfactory scars (tensionless closure is mandatory); (5) loss of tragal definition; (6) temporal or occipital hairline recession (see before); (7) alopecia; and (8) recurrent neck banding caused by aggressive defatting, unrecognized seromas, or irregular platysma plication. Conservative resection of fat is the best prevention.

The results of surgical rejuvenation are not permanent. Ageing and its effects on facial anatomy continue beyond the surgical procedure. Eventually, further surgery can address the progressive aging process. Several studies have looked at the longevity of results, with the endpoint of repeat face lift. The consensus is that 8 to 10 years is the timeframe for which a "redo" surgical procedure can be done to correct the continued aging of the face. Individual results and longevity often depend on individual patient characteristics such as age, changes in weight, level of activity, sun exposure, and general health. Nevertheless, the "clock is reset" and patients will appear younger after the surgical rejuvenation and restart the aging process from the new starting point. In that regard, the changes have some permanency.

22.11 Review Questions

22.11.1 Choose the Best Answer

1. A 60-year-old female is to undergo an extended SMAS procedure. The details of the surgical anatomy of the SMAS were described by
   a) Tord Skoog.
   b) David Furnas.
   c) Vladimir Mitz and Martine Peyronie.
   d) James Stuzin et al.
2. A 62-year-old woman is undergoing a facelift. To avoid injury to the frontal rami of the facial nerve, skin undermining in the temporal region should be superficial to
   a) The deep temporal fascia.
   b) The superficial temporal fascia.
   c) The innominate fascia/parotid temporal fascia.
   d) The periosteum.
3. A 47-year-old woman is in your office for consultation regarding face and neck rejuvenation surgery. She has read some information on the internet and is interested in a short-scar-limited SMAS intervention. The following technique best describes her wishes:
   a) Composite rhytidectomy.
   b) Lateral SMASectomy.
   c) High SMAS technique.
   d) SMAS-platysma facelift.
4. A 49-year-old woman is undergoing an endoscopic brow lift. In your craniocaudal dissection in the temporal region, the following structure when encountered indicates that the frontal rami of the facial nerve are in close caudal proximity:
   a) Sentinel vein.
   b) Lateral orbital thickening.
   c) Supraorbital ligamentous adhesion.
   d) Inferior temporal septum.
5. A 50-year-old woman is undergoing face and neck lift surgery. Her clinical evaluation discloses significant cervicofacial laxity, pronounced vertical neck bands, an obtuse cervicomental angle, and prominent jowling. The surgical technique to achieve long-term improvement in the neck bands most consistently would involve
   a) Aggressive neck liposculpture.
   b) Vertical SMAS flap transposition and fixation.
   c) Corset platysmaplasty.
   d) Lateral SMASectomy.

22.11.2 Answers

1. c. Vladimir Mitz and Martine Peyronie.
2. b. The superficial temporal fascia.
3. b. Lateral SMASectomy.
4. d. Inferior temporal septum.
5. c. Corset platysmaplasty.

References

[1] Reyneke JP. Systematic patient evaluation. In: Essentials of Orthognathic Surgery, 1st ed. Chicago, IL. Quintessence Publishing Co, Inc; 2003:13-68
[2] Fardo D, Zins JE, Nahai F. Facelift (lower face); current technique. In: Mathes, S ed. Plastic Surgery, 2nd ed. Philadelphia,PA. Elservier;2006:275-296
[3] Martén E, Langevin C-J, Kaswan S, Zins JE. The safety of rhytidectomy in the elderly. Plast Reconstr Surg. 127(6):2455–2463
[4] Rees TD, Liverett DM, Guy CL. The effect of cigarette smoking on skin-flap survival in the face lift patient. Plast Reconstr Surg. 73(6):911–915
[5] Kuri M, Nakagawa M, Tanaka H, Hasuo S, Kishi Y. Determination of the duration of preoperative smoking cessation to improve wound healing after head and neck surgery. Anesthesiology. 102(5):892–896
[6] de Brito MJA, Nahas FX, Cordás TA, Tavares H, Ferreira LM. Body dysmorphic disorder in patients seeking abdominoplasty, rhinoplasty, and rhytidectomy. Plast Reconstr Surg. 137(2):462–471
[7] Skoog T. Plastic Surgery – New Methods and Refinements. Philadelphia, PA: WB Saunders; 1974
[8] Mitz V, Peyronie M. The superficial musculo-aponeurotic system (SMAS) in the parotid and cheek area. Plast Reconstr Surg. 58(1):80–88
[9] Furnas DW. The retaining ligaments of the cheek. Plast Reconstr Surg. 83(1): 11–16
[10] Stuzin JM, Baker TJ, Gordon HL. The relationship of the superficial and deep facial fascias: relevance to rhytidectomy and aging. Plast Reconstr Surg. 89 (3):441–449, discussion 450–451
[11] Owsley JQ , Jr. Platysma-fascial rhytidectomy: a preliminary report. Plast Reconstr Surg. 60(6):843–850
[12] Owsley JQ , Jr. SMAS-platysma face lift. Plast Reconstr Surg. 71(4):573–576
[13] Owsley JQ , Jr. SMAS-platysma facelift. A bidirectional cervicofacial rhytidectomy. Clin Plast Surg. 10(3):429–440
[14] Psillakis JM, Rumley TO, Camargos A. Subperiosteal approach as an improved concept for correction of the aging face. Plast Reconstr Surg. 82 (3):383–394
[15] Ramirez OM, Maillard GF, Musolas A. The extended subperiosteal face lift: a definitive soft-tissue remodeling for facial rejuvenation. Plast Reconstr Surg. 88(2):227–236, discussion 237–238
[16] Hamra ST. The deep-plane rhytidectomy. Plast Reconstr Surg. 86(1):53–61, discussion 62–63
[17] Hamra ST. Composite rhytidectomy. Plast Reconstr Surg. 90(1):1–13
[18] Hamra ST. Composite rhytidectomy and the nasolabial fold. Clin Plast Surg. 22(2):313–324
[19] Hamra ST. Composite rhytidectomy. Finesse and refinements in technique. Clin Plast Surg. 24(2):337–346
[20] Hamra ST. Repositioning the orbicularis oculi muscle in the composite rhytidectomy. Plast Reconstr Surg. 90(1):14–22
[21] Hamra ST. Arcus marginalis release and orbital fat preservation in midface rejuvenation. Plast Reconstr Surg. 96(2):354–362
[22] Hamra ST. The role of orbital fat preservation in facial aesthetic surgery. A new concept. Clin Plast Surg. 23(1):17–28
[23] Hamra ST. The zygorbicular dissection in composite rhytidectomy: an ideal midface plane. Plast Reconstr Surg. 102(5):1646–1657
[24] Hamra ST. The role of the septal reset in creating a youthful eyelid-cheek complex in facial rejuvenation. Plast Reconstr Surg. 113(7):2124–2141, discussion 2142–2144
[25] Hamra ST. Frequent face lift sequelae: hollow eyes and the lateral sweep: cause and repair. Plast Reconstr Surg. 102(5):1658–1666
[26] Barton FE , Jr. Rhytidectomy and the nasolabial fold. Plast Reconstr Surg. 90 (4):601–607
[27] Barton FE , Jr. The SMAS and the nasolabial fold. Plast Reconstr Surg. 89(6): 1054–1057, discussion 1058–1059
[28] Barton FE , Jr. The "high SMAS" face lift technique. Aesthet Surg J. 22(5):481–486

[29] Barton FE , Jr. Aesthetic surgery of the face and neck. Aesthet Surg J. 29(6): 449–463, quiz 464–466
[30] Aston SJ. Platysma-SMAS cervicofacial rhytidoplasty. Clin Plast Surg. 10(3): 507–520
[31] Connell BF, Marten TJ. The trifurcated SMAS flap: three-part segmentation of the conventional flap for improved results in the midface, cheek, and neck. Aesthetic Plast Surg. 19(5):415–420
[32] Marten TJ. High SMAS facelift: combined single flap lifting of the jawline, cheek, and midface. Clin Plast Surg. 35(4):569–603, vi–vii
[33] Mendelson BC. Extended sub-SMAS dissection and cheek elevation. Clin Plast Surg. 22(2):325–339
[34] Stuzin JM, Baker TJ, Baker TM. Refinements in face lifting: enhanced facial contour using Vicryl mesh incorporated into SMAS fixation. Plast Reconstr Surg. 105(1):290–301
[35] Stuzin JM, Baker TJ, Gordon HL, Baker TM. Extended SMAS dissection as an approach to midface rejuvenation. Clin Plast Surg. 22(2):295–311
[36] Stuzin JM. MOC-PSSM CME article: face lifting. Plast Reconstr Surg. 121(1) Suppl:1–19
[37] Stuzin JM. Restoring facial shape in face lifting: the role of skeletal support in facial analysis and midface soft-tissue repositioning. Plast Reconstr Surg. 119(1):362–376, discussion 377–378
[38] Connell BF. Contouring the neck in rhytidectomy by lipectomy and a muscle sling. Plast Reconstr Surg. 61(3):376–383
[39] Feldman JJ. Corset platysmaplasty. Plast Reconstr Surg. 85(3):333–343
[40] Knize DM. Limited incision submental lipectomy and platysmaplasty. Plast Reconstr Surg. 101(2):473–481
[41] Zins JE, Fardo D. The “anterior-only” approach to neck rejuvenation: an alternative to face lift surgery. Plast Reconstr Surg. 115(6):1761–1768
[42] Zins JE, Menon N. Anterior approach to neck rejuvenation. Aesthet Surg J. 30 (3):477–484
[43] Feldman JJ. The isolated necklift. In: Necklift, 1st ed. Stuttgart, Germany. Thieme;2006:106-113
[44] Vasconez LO, Core GB, Gamboa-Bobadilla M, Guzman G, Askren C, Yamamoto Y. Endoscopic techniques in coronal brow lifting. Plast Reconstr Surg. 94(6):788–793
[45] Baker DC. Lateral SMASectomy. Plast Reconstr Surg. 100(2):509–513
[46] Baker D. Rhytidectomy with lateral SMASectomy. Facial Plast Surg. 16(3): 209–213
[47] Baker DC. Minimal incision rhytidectomy (short scar face lift) with lateral SMASectomy. Aesthet Surg J. 21(1):68–79
[48] Baker DC. Minimal incision rhytidectomy (short scar face lift) with lateral SMASectomy: evolution and application. Aesthet Surg J. 21(1):14–26
[49] Baker DC. Lateral SMASectomy, plication and short scar facelifts: indications and techniques. Clin Plast Surg. 35(4):533–550, vi
[50] Baker DC, Nahai F, Massiha H, Tonnard P. Short scar face lift. Aesthet Surg J. 25(6):607–617
[51] Tonnard P, Verpaele A, Monstrey S, et al. Minimal access cranial suspension lift: a modified S-lift. Plast Reconstr Surg. 109(6):2074–2086
[52] Tonnard PL, Verpaele A, Gaia S. Optimising results from minimal access cranial suspension lifting (MACS-lift). Aesthetic Plast Surg. 29(4):213–220, discussion 221
[53] Tonnard P, Verpaele A. The MACS-lift short scar rhytidectomy. Aesthet Surg J. 27(2):188–198
[54] Verpaele A, Tonnard P, Gaia S, Guerao FP, Pirayesh A. The third suture in MACS-lifting: making midface-lifting simple and safe. J Plast Reconstr Aesthet Surg. 60(12):1287–1295
[55] Verpaele A, Tonnard P. Lower third of the face: indications and limitations of the minimal access cranial suspension lift. Clin Plast Surg. 35(4):645–659, vii
[56] Coleman SR. Long-term survival of fat transplants: controlled demonstrations. Aesthetic Plast Surg. 19(5):421–425
[57] Rohrich RJ, Pessa JE. The fat compartments of the face: anatomy and clinical implications for cosmetic surgery. Plast Reconstr Surg. 119(7):2219–2227, discussion 2228–2231
[58] Carruthers JD, Carruthers JA. Treatment of glabellar frown lines with C. botulinum-A exotoxin. J Dermatol Surg Oncol. 18(1):17–21
[59] Duranti F, Salti G, Bovani B, Calandra M, Rosati ML. Injectable hyaluronic acid gel for soft tissue augmentation. A clinical and histological study. Dermatol Surg. 24(12):1317–1325
[60] Pessa JE. SMAS fusion zones determine the subfascial and subcutaneous anatomy of the human face: fascial spaces, fat compartments, and models of facial aging. Aesthet Surg J. 36(5):515–526
[61] Mendelson B, Wong C-H. Commentary on: SMAS fusion zones determine the subfacial and subcutaneous anatomy of the human face: fascial spaces, fat compartments, and models of facial aging. Aesthet Surg J. 36 (5):529–532
[62] Zins JE, Hashem AM. Commentary on: SMAS fusion zones determine the subfascial and subcutaneous anatomy of the human face: fascial spaces, fat compartments, and models of facial aging. Aesthet Surg J. 36(5):527–528
[63] Alghoul M, Codner MA. Retaining ligaments of the face: review of anatomy and clinical applications. Aesthet Surg J. 33(6):769–782
[64] Alghoul M, Bitik O, McBride J, Zins JE. Relationship of the zygomatic facial nerve to the retaining ligaments of the face: the Sub-SMAS danger zone. Plast Reconstr Surg. 131(2):245e–252e
[65] Huettner F, Rueda S, Ozturk CN, et al. The relationship of the marginal mandibular nerve to the mandibular osseocutaneous ligament and lesser ligaments of the lower face. Aesthet Surg J. 35(2):111–120
[66] Moss CJ, Mendelson BC, Taylor GI. Surgical anatomy of the ligamentous attachments in the temple and periorbital regions. Plast Reconstr Surg. 105 (4):1475–1490, discussion 1491–1498
[67] Mendelson BC, Muzaffar AR, Adams WP , Jr. Surgical anatomy of the midcheek and malar mounds. Plast Reconstr Surg. 110(3):885–896, discussion 897–911
[68] Muzaffar AR, Mendelson BC, Adams WP , Jr. Surgical anatomy of the ligamentous attachments of the lower lid and lateral canthus. Plast Reconstr Surg. 110(3):873–884, discussion 897–911
[69] Wong C-H, Mendelson B. Midcheek lift using facial soft-tissue spaces of the midcheek. Plast Reconstr Surg. 136(6):1155–1165
[70] Kikkawa DO, Lemke BN, Dortzbach RK. Relations of the superficial musculoaponeurotic system to the orbit and characterization of the orbitomalar ligament. Ophthal Plast Reconstr Surg. 12(2):77–88
[71] Rohrich RJ, Ghavami A, Mojallal A. The five-step lower blepharoplasty: blending the eyelid-cheek junction. Plast Reconstr Surg. 128(3):775–783
[72] Kawamoto HK, Bradley JP. The tear “TROUF” procedure: transconjunctival repositioning of orbital unipedicled fat. Plast Reconstr Surg. 112(7):1903–1907, discussion 1908–1909
[73] Stutman RL, Codner MA. Tear trough deformity: review of anatomy and treatment options. Aesthet Surg J. 32(4):426–440
[74] Ghavami A, Pessa JE, Janis J, Khosla R, Reece EM, Rohrich RJ. The orbicularis retaining ligament of the medial orbit: closing the circle. Plast Reconstr Surg. 121(3):994–1001
[75] Schiller JD. Lysis of the orbicularis retaining ligament and orbicularis oculi insertion: a powerful modality for lower eyelid and cheek rejuvenation. Plast Reconstr Surg. 129(4):692e–700e
[76] Hidalgo DA. An integrated approach to lower blepharoplasty. Plast Reconstr Surg. 127(1):386–395
[77] Roostaeian J, Rohrich RJ, Stuzin JM. Anatomical considerations to prevent facial nerve injury. Plast Reconstr Surg. 135(5):1318–1327
[78] Pitanguy I, Ramos AS. The frontal branch of the facial nerve: the importance of its variations in face lifting. Plast Reconstr Surg. 38(4):352–356
[79] Bernstein L, Nelson RH. Surgical anatomy of the extraparotid distribution of the facial nerve. Arch Otolaryngol. 110(3):177–183
[80] de Bonnecaze G, Chaput B, Filleron T, Al Hawat A, Vergez S, Chaynes P. The frontal branch of the facial nerve: can we define a safety zone? Surg Radiol Anat. 37(5):499–506
[81] Trinei FA, Januszkiewicz J, Nahai F. The sentinel vein: an important reference point for surgery in the temporal region. Plast Reconstr Surg. 101(1):27–32
[82] Trussler AP, Stephan P, Hatef D, Schaverien M, Meade R, Barton FE. The frontal branch of the facial nerve across the zygomatic arch: anatomical relevance of the high-SMAS technique. Plast Reconstr Surg. 125(4):1221–1229
[83] Baker DC, Conley J. Avoiding facial nerve injuries in rhytidectomy. Anatomical variations and pitfalls. Plast Reconstr Surg. 64(6):781–795
[84] Agarwal CA, Mendenhall SD , III, Foreman KB, Owsley JQ. The course of the frontal branch of the facial nerve in relation to fascial planes: an anatomic study. Plast Reconstr Surg. 125(2):532–537
[85] Dingman RO, Grabb WC. Surgical anatomy of the mandibular ramus of the facial nerve based on the dissection of 100 facial halves. Plast Reconstr Surg Transplant Bull. 29:266–272
[86] Ellenbogen R. Pseudo-paralysis of the mandibular branch of the facial nerve after platysmal face-lift operation. Plast Reconstr Surg. 63(3):364–368
[87] Daane SP, Owsley JQ. Incidence of cervical branch injury with “marginal mandibular nerve pseudo-paralysis” in patients undergoing face lift. Plast Reconstr Surg. 111(7):2414–2418
[88] Chowdhry S, Yoder EM, Cooperman RD, Yoder VR, Wilhelmi BJ. Locating the cervical motor branch of the facial nerve: anatomy and clinical application. Plast Reconstr Surg. 126(3):875–879

[89] Salinas NL, Jackson O, Dunham B, Bartlett SP. Anatomical dissection and modified Sihler stain of the lower branches of the facial nerve. Plast Reconstr Surg. 124(6):1905–1915
[90] McKinney P, Katrana DJ. Prevention of injury to the great auricular nerve during rhytidectomy. Plast Reconstr Surg. 66(5):675–679
[91] Last RJ. The head and neck. Anatomy Regional and Applied. 7th ed. New York: Churchill Livingstone; 1984:381-388
[92] Ozturk CN, Ozturk C, Huettner F, Drake RL, Zins JE. A failsafe method to avoid injury to the great auricular nerve. Aesthet Surg J. 34(1):16–21
[93] Stuzin JM, Baker TJ, Gordon HL. Treatment of photoaging. Facial chemical peeling (phenol and trichloroacetic acid) and dermabrasion. Clin Plast Surg. 20(1):9–25
[94] Feldman JJ. Neck lift my way: an update. Plast Reconstr Surg. 134(6):1173–1183
[95] Ferreira LM, Horibe EK. Understanding the finger-assisted malar elevation technique in face lift. Plast Reconstr Surg. 118(3):731–740
[96] Tonnard P, Little J, Stuzin J, Zins JE, Hamra ST. Facial Rejuvenation Part 2:My Recent Refinements. In: Zins JE, ed. ASPS-The Meeting. Boston; 2015
[97] Hamra ST. Correcting the unfavorable outcomes following facelift surgery. Clin Plast Surg. 28(4):621–638
[98] Hamra ST. Prevention and correction of the “face-lifted” appearance. Facial Plast Surg. 16(3):215–229
[99] Choucair RJ, Hamra ST. Extended superficial musculaponeurotic system dissection and composite rhytidectomy. Clin Plast Surg. 35(4):607–622, vii
[100] Choucair RJ, Hamra ST. Nuances of the composite face-lift technique. Semin Plast Surg. 23(4):247–256
[101] Hamra ST. Building the composite face lift: a personal odyssey. Plast Reconstr Surg. 138(1):85–96
[102] Hamra ST. A study of the long-term effect of malar fat repositioning in face lift surgery: short-term success but long-term failure. Plast Reconstr Surg. 110(3):940–951, discussion 952–959
[103] Grover R, Jones BM, Waterhouse N. The prevention of haematoma following rhytidectomy: a review of 1078 consecutive facelifts. Br J Plast Surg. 54(6):481–486
[104] Baker DC, Aston SJ, Guy CL, Rees TD. The male rhytidectomy. Plast Reconstr Surg. 60(4):514–522
[105] Gupta V, Winocour J, Shi H, Shack RB, Grotting JC, Higdon KK. Preoperative risk factors and complication rates in facelift: analysis of 11,300 patients. Aesthet Surg J. 36(1):1–13
[106] Jones BM, Grover R. Avoiding hematoma in cervicofacial rhytidectomy: a personal 8-year quest. Reviewing 910 patients. Plast Reconstr Surg. 113(1):381–387, discussion 388–390
[107] Jones BM, Grover R. Reducing complications in cervicofacial rhytidectomy by tumescent infiltration: a comparative trial evaluating 678 consecutive face lifts. Plast Reconstr Surg. 113(1):398–403
[108] Man D. Premedication with oral clonidine for facial rhytidectomy. Plast Reconstr Surg. 94(1):214–215
[109] Rohrich RJ, Ghavami A, Constantine FC, Unger J, Mojallal A. Lift-and-fill face lift: integrating the fat compartments. Plast Reconstr Surg. 133(6):756e–767e
[110] Rohrich RJ, Ghavami A, Lemmon JA, Brown SA. The individualized component face lift: developing a systematic approach to facial rejuvenation. Plast Reconstr Surg. 123(3):1050–1063
[111] Ramanadham SR, Costa CR, Narasimhan K, Coleman JE, Rohrich RJ. Refining the anesthesia management of the face-lift patient: lessons learned from 1089 consecutive face lifts. Plast Reconstr Surg. 135(3):723–730

23 Facial Rejuvenation with Injectables

Lynn A. Damitz

Abstract
This chapter covers the subject of botulinum toxins and the different classes of fillers—collagen, hyaluronic acid, calcium hydroxylapatite, poly-L-lactic acid, permanent fillers, liquid injectable silicone—available for facial rejuvenation. The authors sequentially describe each step of the presurgical, surgical, and postsurgical phases. Indications that respond best to Botox are cited, and techniques for filler injection are covered. The chapter concludes with guidelines on postsurgical care and, importantly, potential complications and their treatments.

Keywords: Botox, fillers, G prime value, collagen, silicone

23.1 Botox: Goals and Objectives

- Know the indications for use of botulinum toxins including FDA-approved indications and off-label uses.
- Know the different types of botulinum toxins and the advantages and disadvantages of each.
- Know the contraindications and cautions for use of botulinum toxin.
- Know the potential complications and side effects of treatment with Botox and what should be discussed in order to obtain informed consent.
- Know proper reconstitution, storage, preparation, and the techniques for injection of Botox for different areas of the face.

23.2 Fillers: Goals and Objectives

- Know the different classes of fillers and the pros and cons of each and their use in appropriate areas.
- Know what the G prime value is and how it guides the use of the filler.
- Know the complications that can be associated with injection of fillers.
- Know the injection techniques for the upper, mid, and lower face.
- Know aftercare procedures and cautions after injection with fillers.

23.3 Botox

Botulinum neurotoxin (BoNT) is an exotoxin that is secreted by the *Clostridium botulinum* bacteria. There are seven serotypes of botulinum toxin labeled A to G, but only types A and B are produced for clinical use. Although serotypes A and B have the same clinical effect of chemodenervation, they have different mechanisms of action. Type A botulinum toxin (BoNT-A) inhibits SNAP-24 which inhibits vesicular release of acetylcholine at the neuromuscular endplate. Type B botulinum toxin (BoNT-B) binds to and cleaves synaptobrevin which blocks presynaptic vesical fusion and neurotransmitter release. The first reported use of BoNT in humans was in 1980 for the treatment of strabismus by Dr. Alan Scott. Dr. Carruthers pioneered the use of BoNT for cosmetic treatment of glabellar rhytids in 1987.[1]

BoNT is a safe drug when used appropriately. Most cosmetic patients receive 25 to 75 units per treatment. The most commonly used BoNT used for cosmetic applications is Type A. The different formulations of injectable BoNT-A available are OnabotulinumA (Botox Cosmetic, Allergan), AbobotulinumtoxinA (Dysport, Medicis), and IncobotulinumtoxinA (Xeomin, Merz). There are minor differences between the different formulations. Compared to Botox, Dysport is thought to have a faster onset, but its spread is wider which may be a factor when deciding if to use it around the ocular muscles. It requires 2.5 to 4 times the dose of Botox for the same effectiveness. Xeomin has the advantage of not needing refrigeration and its dosage is 1:1 with Botox. It does not have hemagglutinin proteins which give it a lower theoretic hypersensitivity profile than Botox or Dysport. Botox is approved by the Food and Drug Administration (FDA) for the treatment of glabellar rhytids and crow's feet. Dysport and Xeomin are FDA approved for glabellar rhytids. RimabotulinumtoxinB (Myobloc, US WorldMeds LLC) is a BoNT-B, but it is not commonly used for cosmetic applications as it has a lower pH which makes injection more painful and it has a shorter duration of action.[1]

23.4 Fillers

There are a wide variety of fillers that are FDA approved for use in facial rejuvenation. Fillers can be first divided into two main categories: biodegradable fillers which are impermanent and non-biodegradable fillers which are permanent. The biodegradable fillers can be further subdivided into collagen, hyaluronic acid (HA), calcium hydroxylapatite (CaHA), and polylactic acid fillers and within these different categories, the fillers can vary in their concentration of material, size of the particles, the viscosity, and side-effect profile. Common uses for fillers are for correction of moderate-to-severe facial wrinkles, lip augmentation, cheek augmentation to address midface age-related volume deflation, and tear trough deformities.

The fillers have different properties which guide which ones are used for different areas. The size of the particles, the amount of cross-linking, the G-prime, the viscosity, and the concentration of the product determine its properties. G-prime refers to the elastic modulus, or stiffness of the filler. The more cross-linking of the polymer, the higher the viscosity and G-prime of the product and the more volume and lift it will give and the longer it will last.[2]

23.4.1 Collagen

The first facial fillers were made of bovine collagen, but they fell out of favor due to the need for skin testing before injection to detect possible hypersensitivity reactions and a short duration of action. Zyderm I and II and Zyplast (Inamed Aesthetics, Santa Barbara, CA) were derived from bovine collagen and to address

the concerns of hypersensitivity, CosmoDerm I and II and CosmoPlast (Inamed Aesthetics, Santa Barbara, CA) were developed from highly purified human collagen.[3]

23.4.2 Hyaluronic Acid

HA is a naturally occurring glycosaminoglycan in the skin. It is synthesized using non-animal sources and consists of cross-linked polysaccharide chains which combine with the skin's HA and water to increase the volume and lift in the area. It is also thought to contribute to neocollagenesis. HA lasts between 4 and 12 months and is cleared by the body via hepatic metabolism. HA fillers can be used for correction of moderate-to-severe rhytides and folds and is usually injected into the mid-to-deep dermis. The side-effect and complication profile of HA includes mild pain, edema, and erythema which can be related to injection technique. Hyaluronidase can be used to dissolve HA which makes HA the only currently reversible filler available today. Currently available HA fillers include Restylane (Galderma), Juvederm (Allergan), Prevelle (Mentor), and Belotero (Merz).[4]

23.4.3 Calcium Hydroxylapatite

Calcium hydroxylapatite microspheres are made of material that is similar to bone. The microspheres measure 25 to 45 µg and are suspended in an aqueous sodium carboxymethylcellulose carrier gel. The microspheres stimulate the neocollagenesis which lasts for 15 months or longer due to the slower degradation of the particles. CaHA is degraded into calcium and phosphate which is resorbed. It is currently FDA approved for HIV-associated lipoatrophy and moderate-to-severe rhytids. It should not be used for superficial injections or in the lips, tear trough, or glabella due to granuloma formation. There is no reversal agent for CaHA. Radiesse is a CaHA filler currently available in the United States.[4]

23.4.4 Poly-L-Lactic Acid

Poly-L-lactic acid (PLLA) is a synthetic polymer that is broken down in the body via hydroxylation. Sculptra is a PLLA-based filler that is available in the United States. The microspheres usually measure 40 to 63 µg and after injection, it causes a subclinical inflammation which stimulates collagenesis. It is FDA approved for the treatment of HIV-related lipoatrophy and correction of moderate-to-deep rhytides. It has also been used to treat acne scars and for larger volume correction of facial asymmetry. The product requires reconstitution with sterile water hours prior to injection and may take several treatments to achieve the needed volume correction, but the results can last as long as 2 years. Complications associated with PLLA include nodule formation and clumping of the filler in dynamic areas.[4]

23.4.5 Permanent Fillers

Permanent fillers are polymethylmethacrylate (PMMA) microspheres and liquid injectable silicone (LIS). PMMA microspheres measure 30 to 50 µg and are suspended in a gel that contains a small amount of bovine collagen. The first generation of PMMA fillers used smaller and less uniform microspheres which resulted in granuloma formation 6 months to 6 years after implantation. The newest generation of PMMA fillers, such as Bellafill (Suneva Medical, San Diego, CA), utilizes larger, more uniform beads and have a better side-effect profile. PMMA microspheres stimulate neocollagenesis which occurs months to more than a year after initial injection. This filler is good for the treatment for contour deformities, facial wasting, acne scars, and deep rhytids. It is meant for deep plane injection and should not be used for superficial injection and in thin skin areas due to the risk of nodules, beading, and scarring, especially with first generation of PMMA fillers.[4] The permanence of the filler is both an advantage and disadvantage of the filler. If injected correctly, the results are permanent and do not require reinjections, but if injected incorrectly, there is no way to remove the filler except surgically.

23.4.6 Liquid Injectable Silicone

Silikon 1000 (Alcon, Fort Worth, TX) and ADATO SIL-OL 5000 (Bausch & Lomb, San Dimas, CA) are two FDA-approved LIS. The FDA approval is for intraocular use only and LIS is not commonly used for facial rejuvenation due to the risk of granuloma formation and migration of the silicone. The use of a microdroplet injection technique has been shown to decrease incidence of complications.[5]

23.5 Patient Presentation

The chief concern that patients may present with in the office can vary from general requests to look younger or "less tired" to specific concerns about particular areas of the face and a request for "Botox." Patients may complain of looking angry or upset due to rhytids that are present when their face is at rest. Others will complain of feeling like their eyes are tired at the end of the day, especially after being outside for an extended period of time. Others may present with specific concerns such as dark circles under the eyes (tear trough), deep marionette lines, or nasolabial folds (▶ Fig. 23.1).

The initial patient assessment includes obtaining a full history and information about prior cosmetic procedures and injections. When asking if the patient has had prior BoNT injections, the question should be directed as queries about any facial injections, not just Botox injections as some patients are not aware that Dysport and Xeomin and Myobloc are in the same class of medications as Botox.

Past medical history should be screened for neuromuscular diseases such as amyotrophic lateral sclerosis, myasthenia gravis, and Eaton–Lambert's syndrome, which are a relative contraindication to BoNT injection.[6] Review of systems should include inquiries about problems with swallowing, bleeding problems, and eye difficulties such as ptosis and corneal exposure. It is not recommended to perform BoNT injections on patients who have recently had LASIK surgery and blepharoplasty due to a higher risk for dry eyes and corneal exposure.

Medications to watch for are any blood thinning medications including warfarin, clopidogrel, and aspirin and recent use of nonsteroidal anti-inflammatory drugs or any of the many blood thinners that are now available. The use of over-the-counter medicine such as fish oil and vitamin E should also be investigated as these may also increase the risk of bleeding and

Fig. 23.1 (a-c) Prior to injection with Botox and Radiesse Plus. Botox: glabellar = 25 units, right lateral brow = 4 units, left lateral brow = 4 units, crow's feet = 8 units.

bruising. The patient will need to be off any blood-thinning medications for 2 weeks prior to injection. Aminoglycoside medications and other medications that affect neuromuscular transmission are contraindicated as they can potentiate the effect of BoNT.

Other contraindications to treatment with BoNT are allergies to botulinum toxin or human albumin. If the patient is allergic to milk protein, Dysport cannot be used as it contains milk protein.

Although BoNT and fillers have not been found to be a teratogen, it is a category C drug and injections are not recommended for women who are pregnant, nursing, or are attempting to become pregnant.

If the patient is concerned about perioral rhytids and is requesting injection around the mouth, obtaining information about his or her jobs and activities such as playing a musical instrument, any job that requires a lot of talking, and other job/activities that require a lot of movement of the mouth is important, as it may be compromised by BoNT injections around the mouth.[7]

23.6 Evaluation of the Patient

The patient's face is first examined at rest and existing rhytids and resting facial expression are noted. Common patterns of rhytids on the upper face are transverse rhytids across the forehead, vertical rhytids overlying the glabella, transverse rhytids over the nasion, and transverse and radial rhytids lateral to the lateral canthus, also known as "crow's feet." A snap test can be performed on the lower eyelid to test for risk for ectropion. In the lower face, the common patterns of rhytids are along the nasolabial folds, marionette lines, and vertical perioral rhytids. The resting position of the oral commissures may be downturned due to overactive depressor anguli oris (DAO) muscles. There may be a mental crease along the chin and platysmal banding on the neck.

Next, have the patient animate his or her face. The amount of movement and deepening of the rhytids should be noted. Forehead rhytids will deepen with contraction of the frontalis. The position of the brow as the frontalis contracts should be observed. Hyperactivity of the lateral brow in contrast with the medial brow may indicate a lateralized frontalis which could lead to excessive elevation of the lateral brow if not enough Botox is placed laterally. Next, have the patient frown to identify the position of the corrugator muscles. Having the patient sniff will activate the procerus and the transverse nasion rhytids can be examined. When the patient smiles, activation of the zygomaticus major (ZM)/minor will result in deepening of the nasolabial folds and the radially oriented folds at the lateral canthus. Having the patient strain his or her neck will make any platysmal banding more prominent.

BoNT injection for the treatment of a "gummy smile" has been reported. This appearance can be due to hyperactive lip elevator muscles. For patients with this complaint, there should be special consideration on the type of smile the patient has and which muscles are mainly responsible for the smile. Rubin described three basic forms of smiles. The "Mona Lisa" smile is the most common smile and is where the corners of the mouth are pulled up and out due to a dominant ZM muscle and is followed by contraction of the levator labii superioris (LLS). The "canine" smile occurs when the LLS contracts first, exposing the canine teeth followed by contraction of the ZM. The third type of smile is the "full denture" smile where the ZM and LLS contract at the same time showing both the upper and lower teeth.[8]

If the patient has deep forehead rhytids at rest and there is evidence of dermatochalasis, the patient should also be evaluated to see if he or she is using his or her frontalis muscle to keep the eyelids in a position that allows for adequate peripheral vision. This exam can be done by relaxing the forehead and having the patient look down and then straight ahead while keeping a hand on the forehead. The amount of dermatochalasis should be evaluated and the patient's peripheral vision evaluated. The patient should be counseled that by relaxing the brow with BoNT, worsening of peripheral vision may occur.

Existing asymmetries of the face should be discussed with the patient during the preinjection evaluation and there may be differences in the way the muscles respond to the injection. Since there are differences between patients in response to BoNT, an incomplete block may occur with dosages that produce a complete block in another patient.

23.6.1 Managing Expectations

It is a misperception that Botox removes all wrinkles in the area injected. In a young patient with minimal rhytids, paralysis of the muscle can result in a smooth brow due to decreased activation of muscles in the area. In a patient with deep rhytids, they need to understand that the wrinkles will remain even if the muscle is inactivated and further treatments such as skin care, resurfacing treatments, or filler will be needed to lessen the appearance of the rhytids.

The onset of Botox occurs within 3 days and the maximal effect will not be seen until 7 to 14 days after injection. If the patient has any significant events within in a week, as a wedding or public appearance, injection may want to be delayed until after the event to give time for any bruising or swelling to resolve.

The paralysis of the muscle is temporary and on average, the patient can expect it to last for 3 to 4 months with full reversal of the effects taking 3 to 6 months.[9]

After injection with fillers, the patient may have bruising for 7 to 10 days after injection. Nonpermanent filler injections can last 6 to 12 months based on their properties and placement. Fillers placed in dynamic areas will not last long and more frequent injections may be needed.

23.7 Preparation for Procedure

23.7.1 Consent

Prior to injection, the patient needs to be fully informed of the risks, benefits, and alternatives to the treatment.

FDA approval for cosmetic uses currently for Botox, Dysport, Xeomin, and Myobloc is only for treatment of glabellar rhytids. Botox has the additional approval for treatment of crow's feet. The use of BoNT in any other areas of the face is off-label.[10]

The approval for fillers is generally for injection into the mid to deep dermis for treatment of moderate-to-severe facial wrinkles and folds. Restylane silk has approval for treatment of perioral rhytids and lip augmentation. Juvederm Voluma has been approved for supraperiosteal injection for mid-face volume restoration. Sculptra and Radiesse have been approved for HIV-related lipoatrophy.[11]

Since injections with BoNT and fillers are often cosmetic procedures, insurance does not typically cover the treatment and the patient will be financially responsible for the treatment, follow-up visits, and for the costs for treatment of any complications that arise from the injections.

The American Society of Plastic Surgeons (ASPS) has designed a general consent for BoNT and filler injections that can be utilized by ASPS members.

23.8 Treatment

23.8.1 Prep

The skin is cleaned with a Betadine and/or alcohol prep in the area to be injected. Prior to this, the patient can place ice packs over the skin to decrease sensation over the area. EMLA or LET cream can be placed on the skin to induce some anesthesia. Some practitioners will perform regional blocks prior to injections of filler if multiple injections are needed.[12]

23.8.2 Reconstitution

Botox comes lyophilized and needs to be reconstituted with saline for injection. Recommended reconstitution procedures are given by the manufacturer. For Botox, it is recommended that 2.5 mL of preservative-free 0.9% sodium chloride is added to the vial which results in 4 units per 0.1 mL. The liquid is then drawn up into a 1-cc syringe. A short 30- to 33-gauge needle is used for the injection. Large reconstitution volumes afford greater control over the amount dosed, but more concentrated formulas are less painful on injection due to the smaller volume. It is recommended that after reconstitution, it is used within 24 hours and stored in a refrigerator at 2 to 8 °C.[13]

Fillers come ready to use and do not require reconstitution, but some practitioners will dilute some of the fillers to facilitate the injection process and prevent clumping.[14]

23.8.3 Botox Injection Techniques

Botox is the most commonly used BoNT currently; so, all dosages reported below are for Botox. Men typically require higher dosages that are 10 units higher than women.

Glabella and Vertical Forehead Lines

The targeted muscles are the corrugator supercilii, procerus, and orbicularis oculi. Prior to injection and prep, the patient is asked to frown and scrunch the nose to evaluate the pattern of frowning and location of the greatest activation of the muscles. The recommended dosage of Botox in this area is 20 units divided into five injection sites; two 4 units of injection into each corrugator, and one 4 units of injection into the procerus. All injections need to be at least 1 cm over the supraorbital rim to avoid brow or eyelid ptosis.[15,16]

Bunny Lines

Transverse nasal dorsal rhytids can be treated by injecting the nasalis muscle with 3 to 5 units in one injection or divided into two superficially on the dorsum of the nose or on the nasal sidewall.[16]

Horizontal Forehead Lines

The target muscle is the frontalis and a total of 10 to 20 units are used in this area for women. The frontalis is anatomically variable in this area but generally, there are two heads with variable amounts of overlap centrally. When the patient raises his or her eyebrows, look for asymmetry and if a higher dosage is needed on one side. Most forehead elevation originates more caudal on the forehead; so, the injections should be located lower on the forehead. Four to six areas are injected across the forehead taking care to stay 2 cm above the supraorbital rim to avoid brow or eyelid ptosis. If the injection pattern is too centralized, there will be excessive elevation of the lateral brow. Recommended dosage is 6 to 15 units divided into four to eight injection points for women.[15,16]

Crow's Feet and Lower Eyelid

The target muscle is the lateral portion of the orbicularis oculi muscle. Have the patient smile to evaluate the pattern of rhytids from the lateral eye. The most common pattern is radially oriented rhytids from the lateral canthus. Two to four injections are given on each side, one centimeter lateral to the orbit, avoiding any veins. The injection is superficial at a level where the needle should be seen under the skin. The usual dosage is 8 to 16 units per side divided into three to five injection sites. If the patient has lax lower eyelids, medial injections should be avoided to prevent inability to close the eyelid and development of ectropion.[16]

Perioral Lines

The orbicularis oris is the targeted muscle for treatment of perioral lines. Prior to injection, the patient purses his or her lips for evaluation of the strength of the muscle. Two to five units in total are injected across the upper lip within 5 mm of the vermillion, avoiding the philtrum and commissures.[16]

Neck

For patients with platysmal banding, injection of 15 to 30 units of Botox can be injected directly into the muscle in three to five sites in 1-cm intervals from the mandibular border to where the band is visible. Care must be taken to inject only into the platysma and not deeper in order to avoid chemodenervation of the underlying strap muscles.[15,16]

Downturned Commissures

Injection of the DAO can help raise downturned commissures of the mouth and reduce the appearance of marionette lines and an angry or sad appearance. To locate the locations of the DAO, the patient is asked to pull down the corners of the mouth. Injection of 2 to 5 units of BoNT is performed 1 cm lateral to the oral commissure along the mandibular border.[17]

Dimpled Chin

If the patient has dimpling of the chin, this can be improved with injection of 5 to 10 units of Botox into the mentalis muscle in the midline just under the prominence of the chin.[15]

Gummy Smile

The injection of the lip elevators to treat excess gingival show is not often performed but has been described in the literature. Hwang et al described injection of Botox at "Yonsei's point" to target the lip elevators, the LLS, levator labii superioris alaeque nasi, and zygomaticus minor. Yonsei's point is a triangular area of convergence of the three lip elevators. The points of the triangle are the lateral point of the ala, the midpoint of the nasolabial fold between the ala and the commissure, and the maxillary point one-fourth distance between the ala and tragus. Five units of Botox are injected into the central point of this triangle which is usually 1 cm lateral to the ala and 3 cm cephalad to the commissure.[18]

23.9 Filler Injection Techniques

23.9.1 Upper Face

Fillers can be used to treat temporal hollowing related to aging. Fillers that have a high G-prime and viscosity are used in this area. The superficial temporal artery should be palpated and injection kept away from the vessels. The needle is inserted near the temporal crest to the level of the bone which is a relatively avascular area. The needle is maintained against the bone during the injection and the product will spread circumferentially from the injection site. The total amount of filler injected can range from 0.25 to 0.75 mL per side. For more superficial injection, a lower G-prime filler is used. The needle is inserted at a 90-degree angle near the temporal crest and the angle of the needle is changed to 45 degrees to facilitate injection into the subdermal space at the level of the superficial temporal fascia. The injection plane is between the subcutaneous tissue and the superficial temporal fascia and aliquots of 0.01 mL are injected in a retrograde fashion followed by massage to contour the filler.[19,20,21]

23.9.2 Midface

The midface can be divided into three main areas: the lateral zygoma, the midcheek, and the submalar area which are divided by two lines—one extending from the lateral canthus to the oral commissure and the other from the tragus to the base of the ala. To increase the width of the midface, augmentation with filler is performed in the area of the lateral zygoma which is lateral to a line drawn from the lateral canthus to the oral commissure. To increase the anterior projection of the midface, the midcheek, which is medial to the lateral canthus and above a line drawn from the tragus to the base of the ala, is augmented. Augmentation of submalar area (lateral to a line drawn from the lateral canthus to the oral commissure and below the line from the tragus to the base of the ala) addresses facial hollowness.[20]

Fillers with a higher G-prime which offer more lift and volume are usually used in this area. The injection is performed with a 25- to 27-gauge needle with aspiration during injection to prevent intra-arterial injection. The injection plane is supraperiosteal and the facial compartments targeted are the suborbicularis oculi fat and the deep medial cheek fat. The injection pattern used is small depot injections versus fanning until the facial shadows are softened. Pitfalls include overinjection medially or in the tear trough.[12]

23.9.3 Tear Trough

Loss of volume in the midface can accentuate the nasojugal groove, also known as the tear trough, giving the appearance of dark circles or bags under the eyes due to shadowing from the contour. Use of filler in this area can improve the appearance of the tear trough deformity. Fillers used in this area include Belotero (HA), Restylane (HA), and Radiesse (CaHA). After preparation of the area, the needle is inserted over the infraorbital rim in line with the center of the eyelid where the skin is thicker. When Restylane or Radiesse is used, it is injected retrograde supraperiosteally in small aliquots below the area of the hollowing. The filler is then massaged over the infraorbital rim and into the volume-deficient areas. The filler is not directly injected into the hollowed areas because of the thin skin and risk of bluish discoloration to the area due to the Tyndall effect. Belotero is less viscous and can be injected more superficially to address superficial irregularities.[12]

23.9.4 Lips

Treatment of the lips with filler can include treatment of perioral lines, fine vermillion lines, augmentation of the lips. The low viscosity, small particle HA fillers such as Restylane Silk and Belotero can be used for the treatment of superficial rhytids of the vermillion and perioral rhytids. For augmentation of the lips, low viscosity small particle HA is also used. Injections into the orbicularis oris or deep fat pads will use HA filler with

intermediate G-prime and viscosity such as Juvederm Ultra. Technique for injection includes linear threading along, but not above, the vermillion border and 1 to 2 mL of filler is injected retrograde. Philtral columns may splay with aging and restoration can be accomplished with injection medial to each philtral column.[14,22] Radiesse is not recommended for injection into the lips due to the risk of nodule formation.[4]

23.9.5 Melomental Folds

Loss of volume around the prejowl sulcus can accentuate the melomental folds or "marionette lines" and may also be accompanied by downturned commissures. Fillers used to treat this area have a high G-prime and viscosity to provide adequate lift and volume. Filler injection may be combined with Botox injection of the DAO. Treatment of the marionette lines involves the injection of filler in the deep and subdermis in a retrograde depot fashion. Additional injections can be done lateral to the commissure to lift the commissure.[14]

23.10 Postprocedural care

After the injection, ice packs can be placed over the areas to reduce swelling and bruising for 10 minutes on, 10 minutes off two to three times a day as needed for swelling for the first 1 to 2 days.

Postinjection instructions vary among providers but generally, the patient should not massage the face and should avoid compression (including headbands and hats) to the areas injected to reduce the risk of the toxin being distributed into surrounding areas for at least 90 minutes. This is especially important for injections in the glabellar region since downward migration of the toxin can affect the ocular muscles leading to difficulties with periorbital and extraocular muscles. They should not lie down for a few hours after injection and strenuous activities which increase heart rate, blood pressure, and make the face hot or flushed after injection should be avoided to decrease chances of bruising.

Blood-thinning medications can be resumed 2 days after injection.

Follow-up is also dependent on the provider. The patient can follow up in 2 to 4 weeks for reevaluation and any additional injections if needed.

23.11 Management of Complications

Risks of injection include swelling, rash, headache, local numbness, pain at the injection site, bruising, respiratory problems, and allergic reaction. Infection is a rare complication but can cause scarring at the site if it does occur.

Side effects of BoNT injection may include a headache that can last from days to weeks. After injection, there may be bruising in the area and swelling at the injection points. If the patient has a significant event coming up in the next week, it is recommended that the injections be postponed until after the event.

Possible complications include eyelid or brow ptosis. There is no reversal agent for Botox but apraclonidine 0.5% drops (Iopidine, Alcon Labs) can be used to help with eyelid elevation. Apraclonidine is an α2-adrenergic agonist which helps with eyelid elevation via Mueller's muscle. Other medications that can be used for mild cases of ptosis include Naphazoline (Naphcon, Alcon Labs) and Phenylephrine 2.5% (Mydfrin 2.5%, Alcon Labs).[23]

Another complication is corneal exposure. Injection of BoNT affecting the orbicularis oculi may result in decreased blinking and difficulty in completely closing the eyes, including ectropion if the lower orbicularis oculi is affected. Migration of BoNT to the extraocular muscles may induce a strabismus.

If the patient has undergone multiple BoNT injections in the past, the body may develop neutralizing antibodies to the toxin which reduces the effectiveness of the injection. Rarely, patients have a hypersensitivity reaction to BoNT which may be related to the proteins in the formula. Reactions can range from a mild rash to anaphylaxis.[7]

Complications stemming from injection of fillers are rare, but can range from poor aesthetic outcome to skin necrosis and blindness. Skin necrosis and blindness can occur from vascular occlusion due to intravascular injection which can present as pain and blanching. Proper training and knowledge of filler properties and facial anatomy will help prevent these significant complications. Incidence of immunologic reactions to fillers has been reported to be 0.06% and there has been no identification of a definitive risk factor. Overcorrection or vascular occlusion with HA fillers can be treated with hyaluronidase and is recommended as soon as possible or optimally 4 hours of injection. Large volume of hyaluronidase (450-1500 units) should be infiltrated over the entire area including the course of the vessel as hyaluronidase can permeate vessel walls.[24]

Injection of HA filler in areas of thinner skin such as under the eye may result in bluish discoloration due to the Tyndall effect (also known as Rayleigh's scattering) which describes the light reflex off filler that is injected too superficially.[2] Treatment for this is hyaluronidase to dissolve the HA.[25]

The aftercare instructions should be reviewed with the patient and if they are not able to comply with the restrictions and instructions, injection should be deferred.

23.12 Outcomes

Patient satisfaction with injections is high. They are quick procedures with minimal downtime and minimal complications for the experienced and educated injector. Objective analysis of the decrease in rhytids after BoNT injection has been attempted through digital analysis. Using digital analysis, Cavallini showed a decrease in static lines of 12.4% and a 41.2% decrease in dynamic lines after injection of 15 units of Botox into the glabellar region.[26]

It has been suggested that injection treatments are more than an "indulgence therapy" since it can result in a better emotional and functional state after treatment.[27] Standardization of injection sites and dosages is difficult due to variations in patient anatomy and outcomes are dependent on the skill of the injector. There are many med spas and offices that offer Botox and filler injections but not all injectors are the same. Knowledge of facial analysis, anatomy, and mechanisms of action of botulinum toxin will produce improved aesthetic results and avoid a frozen, artificial look and complications from treatment.

23.13 Review Questions

23.13.1 Fill in the Correct Answer

1. A patient experiences ptosis of the upper eyelid after injection of Botox into the corrugator muscles to treat glabellar rhytides. What muscle is affected by the Botox leading to this complication?
2. After injection of the frontalis muscle for the treatment of forehead rhytides, the patient has new excessive elevation of the lateral brow. What caused this and how do you treat it?
3. What are the FDA-approved cosmetic uses of BoNT?
4. A patient has sudden blindness after injection of filler in the glabellar regions. What caused this and how do you treat it?
5. After injection of the tear trough with filler, the patient complains of a bluish discoloration under the eyes. How do you prevent and treat this complication?

23.13.2 Answers

1. When BoNT is injected too close to the levator palpebrae superioris muscle or it migrates downward, paralysis of the levator muscle can occur leading to ptosis of the upper eyelid. This is usually associated with injection of the corrugator muscles and in the glabellar region. This can be prevented by injecting at least 1 cm above the supraorbital rim and instructing the patient to avoid massaging that area after injection. BoNT with more diffuse effects such as Xeomin should also be avoided in the glabellar region. There is no reversal agent for Botox, but apraclonidine 0.5% drops (Iopidine, Alcon Labs) can be used to help with eyelid elevation. Apraclonidine is an α2-adrenergic agonist which helps with eyelid elevation via Mueller's muscle. Other medications that can be used for mild cases of ptosis include Naphazoline (Naphcon, Alcon Labs) and Phenylephrine 2.5% (Mydfrin 2.5%, Alcon Labs).
2. Excessive elevation of the lateral brow after BoNT injection of the frontalis muscle is due to insufficient treatment with BoNT of the lateral frontalis and can be treated with additional injection of BoNT laterally. Hyperactivity of the lateral brow in contrast with the medial brow may indicate a lateralized frontalis.
3. Botox is FDA approved for the treatment of glabellar rhytides and crow's feet. Dysport and Xeomin are FDA approved for glabellar rhytides.
4. Complications stemming from injection of fillers are rare, but can include blindness when injections are performed in the glabellar region due to vascular occlusion from the filler if there is an intraarterial injection. Vascular occlusion with HA fillers can be treated with Hyaluronidase. One mL of hyaluronidase will dilute 1 mL of HA, but the total dosage should be titrated to effect. In the case of blindness, an ophthalmologist should be immediately consulted. It has been recommended that clinicians performing filler injections have a "filler crash kit" consisting of hyaluronidase available in the case of emergencies such as this.
5. The bluish discoloration seen under the eye after injection of the tear trough with HA fillers is due to the Tyndall effect. The Tyndall effect, also known as Rayleigh's scattering, is due to differential scattering of light, so a seemingly clear material may appear blue. For this reason, filler is not directly injected into the tear trough because of the thin skin. A recommended HA filler for injection into the tear trough is Belotero, which is less viscous and has a low G-prime and can spread under the thin superficial tissues without causing the Tyndall effect. Treatment of this is injection with hyaluronidase which will dissolve the HA.

References

[1] Walker TJ, Dayan SH. Comparison and overview of currently available neurotoxins. J Clin Aesthet Dermatol. 7(2):31–39

[2] Sundaram H. Going with the flow: An overview of soft tissue filler rheology and its potential clinical applications (2 of 3). 2011:23–28. Available at: http://practicaldermatology.com/pdfs/PD0611_dpt_Cosmetics.pdf. Accessed April 17, 2016

[3] Eppley BL, Dadvand B. Injectable soft-tissue fillers: clinical overview. Plast Reconstr Surg. 118(4):98e–106e

[4] Carruthers J, Carruthers A, Humphrey S. Introduction to fillers. Plast Reconstr Surg. 136(5) Suppl:120S–131S

[5] Broder KW, Cohen SR. An overview of permanent and semipermanent fillers. Plast Reconstr Surg. 118(3) Suppl:7S–14S

[6] Jabeen A, Kandadai RM, Kannikannan MA, Borgohain R. Guidelines for the use of botulinum toxin in movement disorders and spasticity. Ann Indian Acad Neurol. 14 Suppl 1:S31–S34

[7] Sundaram H, Signorini M, Liew S, et al. Global Aesthetics Consensus Group. Global Aesthetics Consensus: Botulinum Toxin Type A–Evidence-based review, emerging concepts, and consensus recommendations for aesthetic use, including updates on complications. Plast Reconstr Surg. 137(3):518e–529e

[8] Rubin LR. The anatomy of a smile: its importance in the treatment of facial paralysis. Plast Reconstr Surg. 53(4):384–387

[9] Glogau R, Kane M, Beddingfield F, et al. OnabotulinumtoxinA: a meta-analysis of duration of effect in the treatment of glabellar lines. Dermatol Surg. 38 (11):1794–1803

[10] Drug Safety Information for Healthcare Professionals - Information for Healthcare Professionals. OnabotulinumtoxinA (marketed as Botox/Botox Cosmetic), AbobotulinumtoxinA (marketed as Dysport) and RimabotulinumtoxinB (marketed as Myobloc). Available at: http://www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/DrugSafetyInformationforHeathcareProfessionals/ucm174949.htm. Accessed April 17, 2016

[11] Health C for D and R. Soft Tissue Fillers (Dermal Fillers) - Soft Tissue Fillers Approved by the Center for Devices and Radiological Health. Available at: http://www.fda.gov/MedicalDevices/ProductsandMedicalProcedures/CosmeticDevices/WrinkleFillers/ucm227749.htm. Accessed April 17, 2016

[12] Cotofana S, Schenck TL, Trevidic P, et al. Midface: clinical anatomy and regional approaches with injectable fillers. Plast Reconstr Surg. 136(5) Suppl: 219S–234S

[13] Dosage, Dilution, Reconstitution | BOTOX® Cosmetic. Available at: http://hcp.botoxcosmetic.com/support/science_of_botox/dosage_admin/dilution_reconstitution. Accessed April 17, 2016

[14] Braz A, Humphrey S, Weinkle S, et al. Lower face: clinical anatomy and regional approaches with injectable fillers. Plast Reconstr Surg. 136(5) Suppl: 235S–257S

[15] Carruthers JDA, Glogau RG, Blitzer A, Facial Aesthetics Consensus Group Faculty. Advances in facial rejuvenation: botulinum toxin type a, hyaluronic acid dermal fillers, and combination therapies–consensus recommendations. Plast Reconstr Surg. 121(5) Suppl:5S–30S, quiz 31S–36S

[16] Gart MS, Gutowski KA. Aesthetic uses of neuromodulators: current uses and future directions. Plast Reconstr Surg. 136(5) Suppl:62S–71S

[17] Wu DC, Fabi SG, Goldman MP. Neurotoxins: current concepts in cosmetic use on the face and neck–lower face. Plast Reconstr Surg. 136(5) Suppl:76S–79S

[18] Hwang WS, Hur MS, Hu KS, et al. Surface anatomy of the lip elevator muscles for the treatment of gummy smile using botulinum toxin. Angle Orthod. 79 (1):70–77

[19] Sykes JM, Cotofana S, Trevidic P, et al. Upper face: clinical anatomy and regional approaches with injectable fillers. Plast Reconstr Surg. 136(5) Suppl: 204s–218s

[20] Moradi A, Watson J. Current concepts in filler injection. Facial Plast Surg Clin North Am. 23(4):489–494
[21] Eviatar J, Lo C, Kirszrot J. Radiesse: advanced techniques and applications for a unique and versatile implant. Plast Reconstr Surg. 136(5) Suppl:164S–170S
[22] Bertucci V, Lynde CB. Current concepts in the use of small-particle hyaluronic acid. Plast Reconstr Surg. 136(5) Suppl:132S–138S
[23] Scheinfeld N. The use of apraclonidine eyedrops to treat ptosis after the administration of botulinum toxin to the upper face. Dermatol Online J. 11(1):9
[24] Martyn King, MD, Cormac Convery, MD, and Emma Davies, RN, NP. HYPERLINK "https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6011868/" J Clin Aesthet Dermatol. 2018 Jun; 11(6): E61E68. This month s guideline: The Use of Hyaluronidase in Aesthetic Practice (v2.4) Published online 2018 Jun 1
[25] Rzany B, DeLorenzi C. Understanding, avoiding, and managing severe filler complications. Plast Reconstr Surg. 136(5) Suppl:196S–203S
[26] Cavallini M. Preliminary report on an objective, fast, and reproducible method to measure the effectiveness of Botulinum Toxin Type A. Aesthet Surg J. 35(6):715–720
[27] Fried RG, Philip Werschler W, Floirendo T. The botulinum toxin experience: results of a patient self-report questionnaire. J Clin Aesthet Dermatol. 2(11): 37–40

Part V

Hand and Upper Extremity

24 Hand Fractures

Lisa M. Block and A. Neil Salyapongse

Abstract
This chapter discusses the range of injuries and remedies involved in a fracture to the hand. Practical guidelines are offered for imaging modalities to assess injuries, regional anesthetic techniques, and the procedures of reduction, immobilization, and fixation. Each structure of the hand commonly involved in a fracture—distal phalanx, middle and proximal phalanx, metacarpal, thumb, carpus—is individually treated. Discussions of postoperative care, therapy, and outcomes conclude the chapter.

Keywords: digital block, reduction, immobilization, fixation

24.1 Goals and Objectives

- Know the key components of the history and physical exam to perform when evaluating a patient who has sustained a hand injury.
- Understand which imaging modalities best evaluate various hand fractures.
- Be able to accurately diagnose and describe the type and pattern of fracture.
- Appreciate the various regional block techniques that can be utilized when performing the hand exam, fracture reduction and splinting in the emergency room, or operative fixation.
- Understand the options for surgical approaches to the fingers, thumb, and hand and fixation techniques for various fracture types.

24.2 Patient Presentation

Fractures of the long bones of the hand are among the most common fractures sustained by patients of all ages. Patients who have sustained a traumatic injury to their hand most often present to the emergency department or urgent care facility for treatment; however, some may present to the office as their initial contact. As the treating physician, the plastic surgeon should be able to evaluate and treat fractures of the phalanges, metacarpals, thumb, and carpus. The goal of appropriate hand fracture treatment is ideally to achieve clinical union with anatomic alignment and articular congruity with rapid mobilization to minimize postinjury stiffness and associated comorbidities.

24.3 History and Physical Exam

When taking a patient's history who has sustained a hand injury, the physician should always document the patient's age, sex, hand dominance, injury laterality, occupation, and hand-specific hobbies, as well as specific details of the mechanism of injury and the timing of the injury. The initial physical exam should take place before any regional or local anesthetic is applied, in order to accurately determine sensation. Oral or intravenous (IV) analgesia may be given to minimize discomfort during examination. All dressings should be removed, and the wound thoroughly irrigated to clear away blood and debris for a clear view of the injury and structures involved. Physical exam should start with observation of the injured hand, and any associated bony and soft-tissue deformity. The examiner should note how the hand is held at rest, whether there is any significant hematoma or active bleeding, whether there is any bone exposed, whether the fingers are all appropriate length, and whether there is any malrotation of the fingers. Gentle palpation of the hand and fingers may elicit focal areas of tenderness, prompting focal examination of individual digits as the survey progresses. Finger length and rotation should be examined both with the fingers extended and with the fingers flexed into the palm. Any scissoring of the fingertips indicates that there is likely a metacarpal or phalangeal fracture with malrotation (▶ Fig. 24.1). Comparison to the unaffected hand is key in differentiating between normal anatomic variations and new pathology. Exposed structures should be noted as well as any soft-tissue deficits. Perfusion to all fingertips should be assessed by observing fingertip temperature, color, and capillary refill; hand-held Doppler exam of involved digital arteries may be warranted if observation and palpation alone is unable to confidently determine perfusion to the involved structures. Sensation should be first grossly assessed by determining whether or not light touch can be perceived, then evaluated and documented specifically by performing two-point discrimination on the radial and ulnar sides of each fingertip. Finally, first passive then active range of motion of the wrist and all fingers should be assessed and recorded.

24.4 Imaging

In the radiographic evaluation of hand fractures, plain radiographs are the mainstay of diagnosis. All hand injuries should be evaluated with anteroposterior (AP), lateral, and oblique radiographs. These three views are essential for visualizing the small bones in the hand to best characterize, and avoid missing, any fractures. In addition, it is best to order radiographs as specific to the injury as possible for the best view. For example, if the small finger has a deformity and a fracture is suspected, a three-view series of the small finger will yield higher quality images than a three-view series of the hand. This is because the sharpest images are obtained when the X-ray beam is centered over the area of interest; for example, hand series X-rays center the beam over the middle finger metacarpal which contrasts with dedicated finger series which center over the proximal phalanx. As a result, the AP image of a hand will capture the small finger slightly supinated potentially obscuring subtle fractures (▶ Fig. 24.2). In some injury patterns, additional radiograph views are useful, such as hook of the hamate view, navicular view, Robert's view for a true AP view of the thumb, and dynamic studies such as the clenched fist view (▶ Fig. 24.3).[1,2] Computed tomography (CT) imaging is required only occasionally for fracture evaluation. Indications for CT imaging are primarily for operative planning in highly comminuted, intra-articular

Fig. 24.1 Left ring finger malrotation deformity after PIP fracture dislocation.

Fig. 24.2 Hand series X-ray of right small finger middle phalanx.

Fig. 24.3 Scaphoid fracture X-ray **(a)** posteroanterior view compared with **(b)** navicular view, which allows better visualization of scaphoid waist fractures.

fractures. Magnetic resonance imaging is likewise rarely required for hand fractures, indicated mainly for detecting occult scaphoid fractures or early avascular necrosis such as in Kienbock's disease.[2] While ultrasound imaging can be useful for soft-tissue hand injuries, it does not have utility in evaluating fractures.

When reading the radiographs, the treating physician should be able to accurately diagnose and describe the type of fracture sustained. This not only allows for determination of appropriate treatment but also ensures accurate and efficient communication with other physicians. Possible fracture patterns include transverse, oblique, spiral, and comminuted (▶ Fig. 24.4). Fractures are described by identifying the type of fracture, whether it is open or closed, commenting on the degree of comminution if present, the presence and direction of fracture fragment

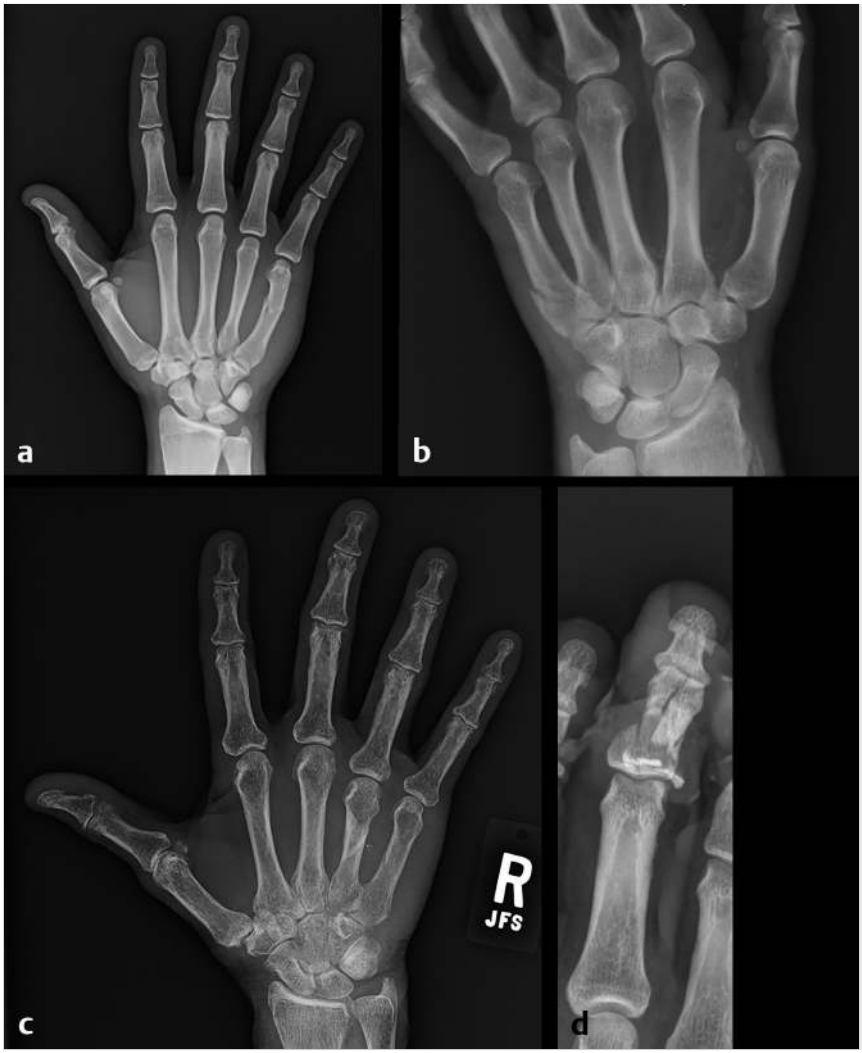


Fig. 24.4 **(a)** Transverse fracture, **(b)** oblique fracture, **(c)** spiral fracture, **(d)** comminuted fracture.

angulation, whether the fracture is apex-volar or apex-dorsal, whether there is any shortening or segmental loss of the overall bone length, measurement of any step-off of the fracture fragments, description of any rotational deformity, quantifying any articular surface involvement, and whether there is any associated joint dislocation (▶ Fig. 24.5).

24.5 Preparation for Surgery

If surgical treatment is indicated for a patient with a hand fracture, additional workup may be required. Whether the patient is currently an operative candidate is the first consideration; if the patient has significant polytrauma involving other major injuries, or if they are in poor health with significant comorbidities, they may require either initial or ultimate nonoperative management. Determination of general versus regional anesthesia with or without sedation should be made together with the patient. Generally, all hand fractures requiring surgical treatment can be done under regional or local anesthesia with sedation; however, some patients strongly prefer general anesthesia. If the patient has an appropriate safety profile for general anesthesia, American Society of Anesthesiologists class I or II, this may be accommodated. However, the risks and complications of general anesthesia should be fully considered. Prior to undergoing anesthesia, a heart and lung exam should be performed, as well as an ECG if indicated, and any laboratory tests as indicated, such as checking coagulation factors in patients who are taking anticoagulation medications.

After the fracture is fully evaluated through history, physical exam, and radiographic imaging, the treating physician must determine appropriate treatment. Most hand fractures can be managed nonoperatively, but there are clear indications for surgical intervention. These include open fracture, displaced intra-articular fractures, fractures with associated soft-tissue injury requiring repair or reconstruction, malrotation, segmental bone loss, multiple contiguous fractures, or an irreducible or highly unstable fracture.[3,4] However, the surgeon must bear in mind that any surgical intervention, even when carried out with delicate and precise tissue handling, creates additional trauma to the surrounding soft tissue and increases the risk of adhesions and stiffness.

Fig. 24.5 Small finger PIP joint fracture dislocation.

24.5.1 Treatment

Unless a patient has a clear indication for surgical intervention, closed reduction and immobilization should be attempted. This can be done in the emergency room, urgent care, or in the office, provided appropriate imaging and splinting materials are available. One of the primary determinants in achieving a good reduction is satisfactory pain control for the patient so appropriate manipulation of the fracture fragments can be performed. Regional anesthetic techniques prove very useful in this setting to avoid dangerously large volumes of local anesthetic, and to achieve good pain control with lower doses of opiate pain medication. Mastering the following regional anesthetic techniques will allow for the surgeon to perform reduction maneuvers on the full range of hand fractures: ring block, digital nerve block, flexor tendon sheath block, wrist block, hematoma block, and Bier block.

24.6 Regional Anesthetic Techniques

When choosing the anesthetic for a regional block, the onset and duration of action should be considered. In general, when performing a regional block for fracture examination and reduction, a rapid onset is preferred to expedite the procedure and 1% lidocaine is the usual choice. When performing a regional block for postoperative pain control, a longer duration is preferred and 0.25% bupivacaine, which lasts twice as long as lidocaine, is the preferred agent.[5] A 1:1 mixture is an additional option when performing regional blocks in which both rapid onset and longer duration are desired. The following principles apply to all regional block techniques in order to avoid complications: never administer a block in an area of soft-tissue infection, always withdraw the syringe prior to injecting to avoid intravascular injection, use a small-gauge needle and slow injection speed to minimize discomfort, use a short needle to control the location of the tip, and calculate the maximum dose of the chosen local anesthetic prior to drawing up the medication to avoid using a toxic dose. The use of epinephrine in concentrations of 1:100,000 to 1:200,000 is a common adjunct to local anesthetics in order to limit systemic absorption and increase the maximum safe dose of administration. Despite historic concern regarding the safety of epinephrine use in the fingers with a theoretic risk of skin or tissue loss, current evidence has clearly shown that the use of epinephrine with local anesthetic injection into the finger or hand is very safe, with no incidence of digital tissue loss recorded in more than 3,000 cases in the literature.[6]

Regional blocks useful for reducing finger fractures include ring block, digital nerve block, and flexor tendon sheath block. The finger is innervated by four digital nerve branches: two dorsal and two volar along the radial and ulnar sides of the finger. The ring block is performed by injecting local anesthetic in a subcutaneous plane around the base of the proximal phalanx circumferentially around the finger. Large volumes of dilute anesthetic may be used; however, the volume should not be so great as to "balloon" the skin and induce pallor at the site of injection as well as the distal digit. The needle is inserted just proximal to the web space on the dorsal surface of the finger and a small subcutaneous wheal created; the needle is then advanced toward the palm and 1 mL anesthetic injected just under the skin on the palmar surface to anesthetize the volar digital nerve. The needle is then withdrawn to the level of the skin but not removed entirely, rather is redirected toward the opposite side of the finger, just proximal to the web space, and a small subcutaneous wheal created. The needle is then withdrawn completely, inserted through the new wheal created, and advanced toward the palmar surface where an additional 1 mL anesthetic is injected as previously.[5,7] Alternatively, a single injection to the volar surface at the level of the metacarpophalangeal (MCP) joint with instillation of approximately 5 mL of anesthetic is another accepted technique with good results and reduction of multiple needle passes (▶ Fig. 24.6).[5] Keep in mind that injuries to the dorsal surface of the proximal phalanx will likely be inadequately anesthetized with this technique, as the terminal innervation from either the radial sensory nerve or dorsal branch of the ulnar nerve will not be blocked.

The common digital nerve is blocked by inserting the needle at the level of the distal palmar crease, just to one side of the metacarpal neck. Two milliliters of local anesthetic is injected, blocking the common digital nerve as it travels toward the finger. The flexor sheath block is performed by inserting the needle at the level of the palmar digital crease, in the center of the long axis of the finger. The needle is inserted until it touches

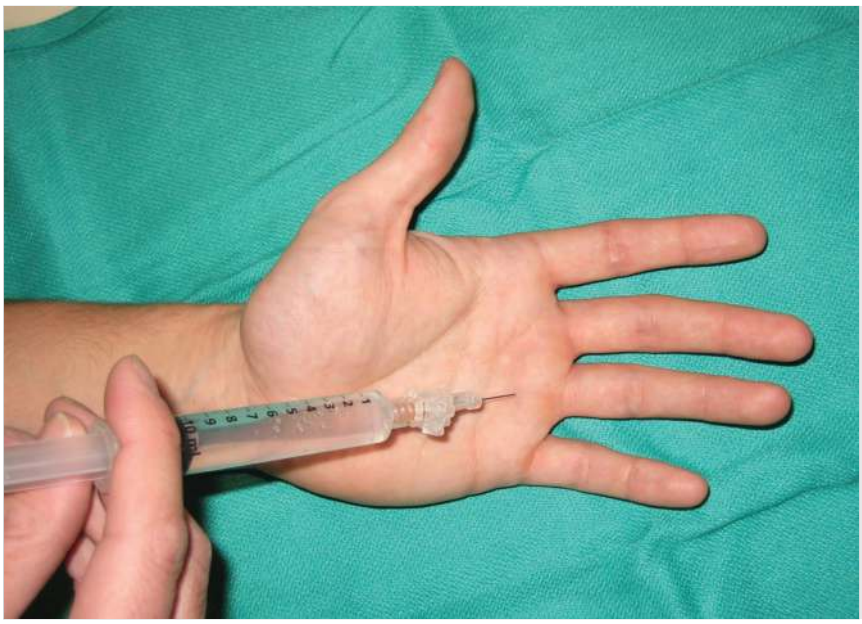


Fig. 24.6 Digital block.

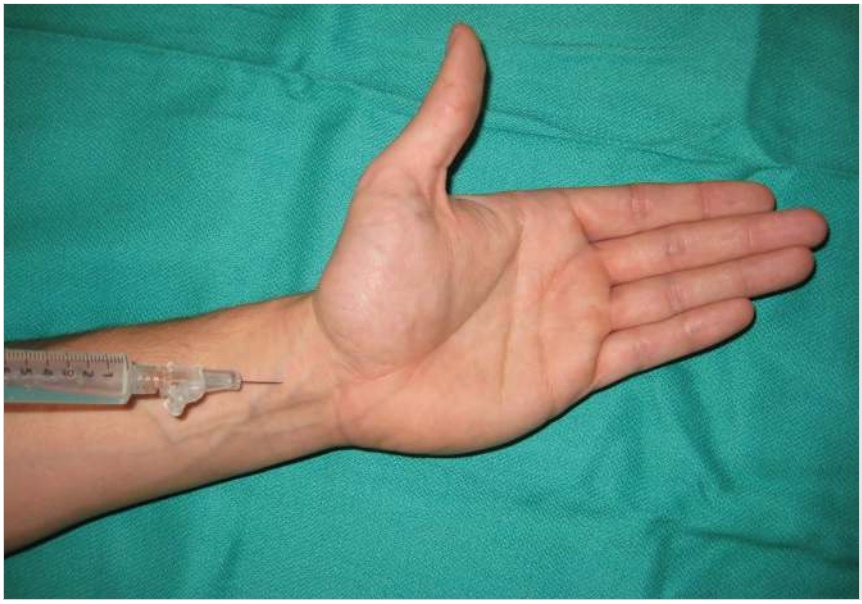


Fig. 24.7 Median nerve block.

the bone, then while slight pressure is put on the syringe plunger, the needle is withdrawn until the anesthetic begins flowing easily into the flexor sheath. Two milliliters is injected. While this technique does provide good pain relief, patients can experience discomfort from the pressure of the volume in the closed space of the flexor sheath. Care should be taken to avoid injecting too much volume into this potential space.[5,7]

Regional blocks useful for reducing hand and wrist fractures include wrist block, hematoma block, and Bier block. The wrist block involves blocking the median, ulnar, and radial nerves at the level of the wrist, rendering the hand insensate and intrinsic muscles paralyzed; it does allow for extrinsic hand muscle movement. The median nerve is blocked by inserting the needle on either the ulnar side of the palmaris longus or, alternately, through the flexor carpi radialis at the level of the proximal wrist crease; it is inserted just until the flexor retinaculum is penetrated, approximately 1 cm deep (▶ Fig. 24.7). After confirming that the patient is not experiencing paresthesias from the needle near the median nerve, 5 mL of local anesthetic is injected, and an additional 1 mL injected superficial to the flexor retinaculum to block the superficial palmar branch. The ulnar nerve is blocked by inserting the needle on the dorsal ulnar side of the flexor carpi ulnaris (FCU) and injecting 5 mL of anesthetic deep to the FCU and an additional 1 mL superficial to block the dorsal cutaneous branch (▶ Fig. 24.8). The radial nerve has many superficial branches; these are blocked by injecting 5 to 10 mL of local anesthetic in a subcutaneous plane starting at the level of the radial styloid and proceeding dorsally. The skin should be pinched and the needle tip inserted and a wheal injected first prior to advancing; this maneuver protects the dorsal radial sensory nerve.[5,7]

A hematoma block is performed by inserting the needle into the area of the fracture, feeling the needle tip along the fracture fragments until it falls easily into the fracture site. The plunger should

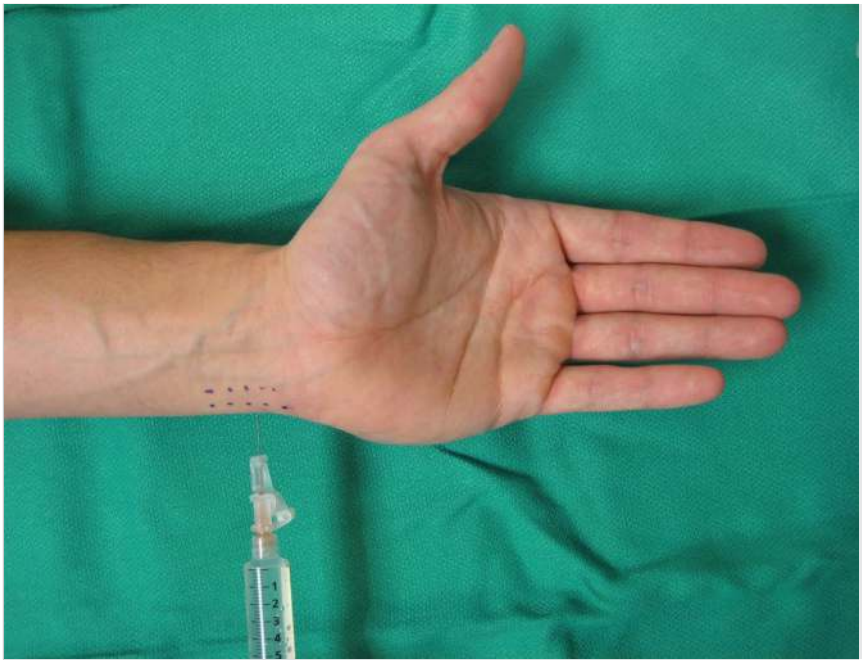


Fig. 24.8 Ulnar nerve block.

be withdrawn and a flash of blood seen in the syringe, confirming the location of the needle tip in the fracture hematoma.[7] Five to 10 mL anesthetic should be infiltrated, proportional to the size and location of the fracture. A Bier block, or IV regional block, involves first placing an IV catheter into the dorsum of the hand, followed by exsanguination of the hand and forearm and inflation of a tourniquet. Next, 0.5% lidocaine at 3 mg/kg or approximately 30 to 50 mL total for an adult is injected via the IV, and the IV subsequently removed.[7] Historically, the full Bier block involved placing the tourniquet on the proximal arm to achieve complete upper extremity anesthesia. However, this involves using a higher dose of local anesthesia, which can increase the risk of systemic toxicity when the tourniquet is released at the end of the procedure. The mini-Bier block technique has thus been developed, which involves placing the tourniquet on the proximal forearm which allows for smaller volumes of anesthesia to be used. Upon releasing the tourniquet at the end of the procedure, the patient should be counseled to notify the surgeon of any metallic taste in their mouth or ringing of their ears, as this may indicate a systemic reaction to the sudden inflow of local anesthetic agent into the circulation. Hemodynamics should be monitored closely throughout this procedure with continuous telemetry and frequent blood pressure checks.

24.6.1 Reduction

After analgesia has been achieved, reduction is performed. Utilizing fluoroscopy, if available, can significantly improve the precision of the reduction. In general, a combination of traction and apex depression is applied to the fracture fragments to achieve reduction. Several specialized maneuvers have been described for specific fracture patterns. For metacarpal neck fractures with apex–dorsal angulation, the Jahss maneuver is the technique of choice for closed reduction. This maneuver involves flexing the MCP and PIP joints to 90 degrees and then applying upward force through the PIP joint while applying downward force on the metacarpal shaft to reduce the metacarpal head into an appropriate alignment with the metacarpal shaft. Rotational deformity can be reduced by using the finger, flexed at the MCP joint, as a crank.[4] Reduction is confirmed with either fluoroscopy or plain radiographs in three views to verify satisfactory reduction in all planes. Soft-tissue integrity and perfusion are ensured by postreduction examination. Reduction is held while either a cast or a splint is applied for immobilization.

24.6.2 Immobilization

Once satisfactory reduction has been achieved, the fracture must be immobilized. Successful bony healing after closed reduction relies on satisfactory immobilization with appropriate splinting or casting. As with all immobilization of the hand, either via splinting or casting or via internal fixation, a balance must be achieved between immobilizing the bone long enough for bony healing to occur but avoiding prolonged immobilization and subsequent stiffness and adhesion formation. Finger splints for phalangeal fractures can be applied by utilizing foam-padded malleable metal splinting material and wrapping the finger with an elastic bandage. The surgeon should avoid applying too much compression during splinting that could cause decreased perfusion to the fingertip. For metacarpal fractures of the ring or little fingers, an ulnar gutter splint is applied, ensuring to include the adjacent stable finger for additional support. For metacarpal fractures of the index or middle fingers, a volar or radial gutter splint is applied, again ensuring to include the adjacent stable finger for additional support. For multiple fractures, a volar slab splint with the hand placed in safety position is applied (wrist extension of 30–40 degrees, MCP flexion 70–90 degrees, IP joints extended). Important points in splint and cast application are to ensure appropriate padding of all bony prominences to avoid pressure points and to always leave all fingertips visible to ensure adequate perfusion. After the splint or cast is applied, final radiographs are taken to ensure reduction stability.

24.6.3 Surgical Approach

If surgical indication is warranted based on either the characteristics of the fracture or failure of closed reduction or instability, reduction and immobilization are performed in the operating room. As discussed earlier, anesthesia can be obtained with either general anesthesia or regional anesthesia with sedation. If general anesthesia is used, local anesthesia should still be administered for improved postoperative pain control. A tourniquet is often used to minimize blood loss and improve visualization of the operative field. The incision chosen will be largely determined by the location of the fracture and the type of fixation planned. For closed reduction and percutaneous pinning, no incision is made. For plating and screw fixation, adequate bony exposure is required for fracture fragment visualization and adequate space to place hardware. For phalangeal fractures, a longitudinal incision either in dorsal or midaxial lateral plane is often utilized for adequate exposure of bone with minimal disruption of soft tissue. For dorsal approach, the extensor mechanism can either be split longitudinally and repaired at the end of the case, or retracted laterally if possible. For the midlateral approach, the neurovascular bundle must be visualized and protected to avoid injury. For metacarpal fractures, the incision is usually placed longitudinally over the dorsum of the involved metacarpal. If two sequential metacarpals are affected, the incision is placed longitudinally between them to avoid multiple incisions. As with all hand surgery, the surgeon should avoid placing the incision on protective surfaces such as the fingertip, the radial side of the index finger, and the ulnar side of the small finger or hand.

Fig. 24.9 Kirschner wires.

24.6.4 Type of Fixation

A wide variety of materials and techniques exist for bony fixation, including Kirschner wires (▶ Fig. 24.9), tension banding (▶ Fig. 24.10), lag screws (▶ Fig. 24.11), plating (▶ Fig. 24.12), and external fixation (▶ Fig. 24.13). The type of fixation chosen may depend on fracture pattern, material availability, and surgeon's preference. Kirschner wires are arguably the most popular choice for internal fixation in hand fractures given their ease of insertion and minimal disruption to the soft-tissue envelope. They are less bulky than plates and screws, and have minimal

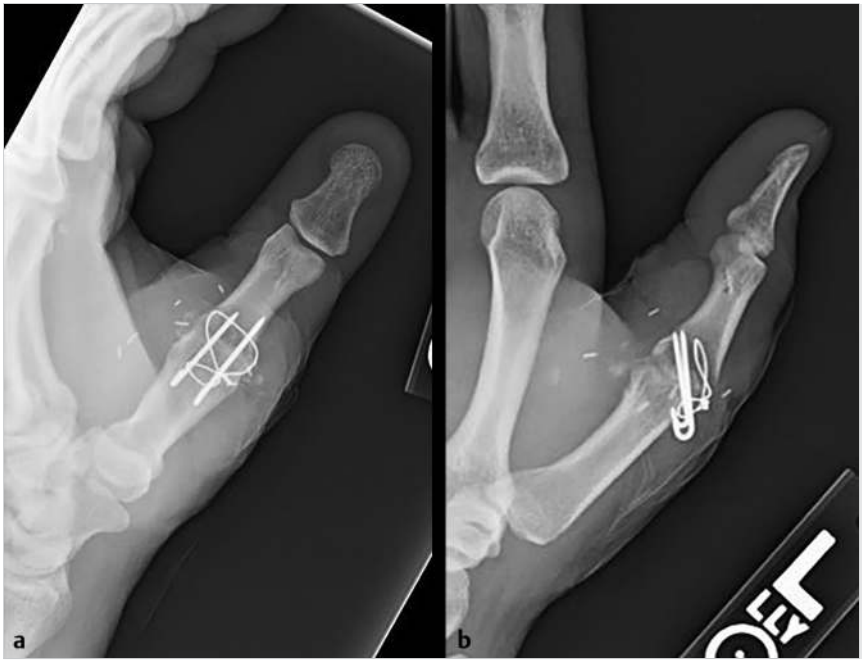


Fig. 24.10 Tension banding **(a)** posteroanterior and **(b)** lateral.

Fig. 24.11 Screws.

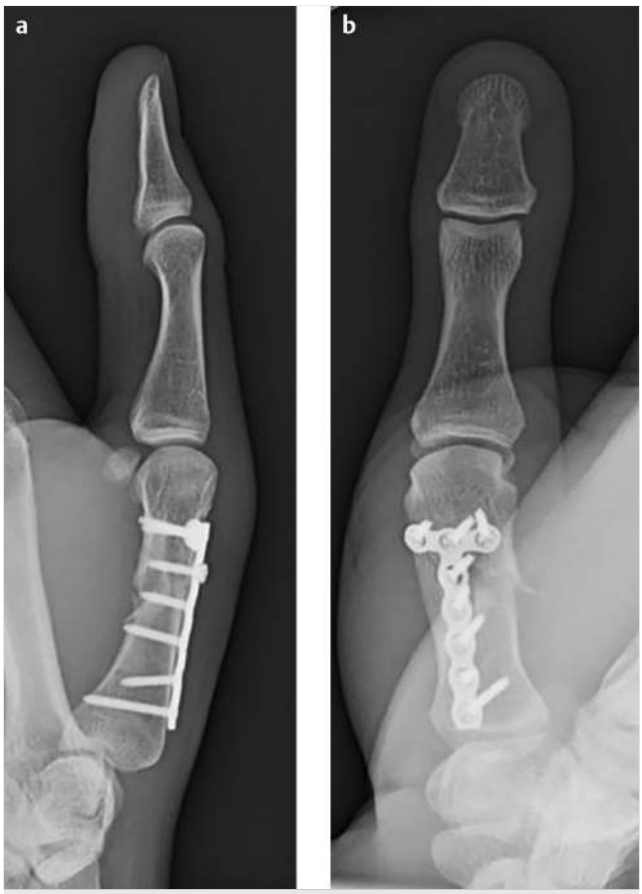


Fig. 24.12 Plating **(a)** posteroanterior and **(b)** lateral.

Fig. 24.13 External fixation.

disruption on bone blood supply. However, smooth Kirschner wires do not provide rigid fixation and do not apply compression across the fracture. Furthermore, a single Kirschner wire does not provide rotational stability, requiring that at least two wires in different places are placed to prevent rotation around the axis. Tension banding is a technique that builds upon Kirschner wire placement for enhanced fracture stability and compression across the fracture line. With this technique, a 26-gauge stainless steel wire is looped under and around crossed Kirschner wires in a figure-of-eight fashion and tightened down to apply compression.[3,4]

If the fracture fragments are able to be reduced but are unable to be maintained in appropriate reduction with cast or splint immobilization, percutaneous Kirschner wire fixation can be utilized. In addition, closed reduction and percutaneous pinning may be used when associated soft-tissue injury requires frequent dressing changes not amenable to cast or splint immobilization. The fracture is reduced and alignment

verified under fluoroscopy. With one hand, the surgeon holds the alignment, and with the other advances the Kirschner wire approximately perpendicularly across fracture line, capturing the fracture fragments. As discussed earlier, at least two Kirschner wires are normally required to prevent rotational deformity of the fracture fragments. Kirschner wires can be placed parallel to each other or crossed. Due to soft-tissue movement relative to the underlying bone, the Kirschner wire should first be advanced through the skin by hand until the sharp point touches the bone. The wire should then be directed in the desired angle, and the wire pushed in to gain some purchase in the bone stock. The drill should then be used for advancing the wire through the bone. This maneuver helps improve accuracy of the placement.

Screw fixation achieves rigid stabilization and can apply compression across the fracture by using the lag technique. Screw fixation can be especially useful in spiral and long oblique fractures. Screws can additionally be counter-sunk to reduce bulk on the bone surface to prevent interference with tendon glide. Plate and screw stabilization, like screw fixation, provides rigid fixation and allows for early motion. Additionally, plates help maintain bone length. However, plate and screw stabilization provides minimal interfragmentary compression. Furthermore, plate placement necessitates wider soft-tissue dissection and periosteal stripping, disrupting the blood supply to the bone fragments, and adds more bulk than other methods of fixation which can contribute to adhesions.

External fixation may be indicated in extensively comminuted fractures, especially those with segmental bone loss, significant shortening, and associated soft-tissue injury. External fixation bridges the fracture site, stabilizing the fracture fragments and maintaining bone length, while the callus forms and the soft tissues heal. A mini-external fixation device may be applied by placing one transverse pin distal to the fracture, and one transverse pin proximal to the fracture, both through mid-axial or dorsolateral incisions. Connecting rods and swivel clamps are then applied, the fracture reduced, and swivel clamps tightened to maintain reduction.[4]

24.7 Surgical Techniques

24.7.1 Distal Phalanx

The distal phalanx is the most commonly fractured bone in the hand.[4] Fractures of the distal phalanx can be divided into three main categories: tuft fractures, shaft fractures, and epiphyseal fractures (▶ Fig. 24.14). Tuft fractures are further characterized into either comminuted or noncomminuted fractures. If open, they are often associated with nail bed or pulp laceration. If closed, they can present with a subungual hematoma which may require drainage. As tuft fractures rarely require internal fixation, stability should be achieved by careful repair of any nail bed and pulp injury, followed by application of simple palmar-based finger splint.[4] Shaft fractures can present as longitudinal or transverse fractures. Nondisplaced fractures do not require internal fixation as the surrounding nail plate and soft tissues provide adequate stability; these should be treated with splinting.[4] Displaced fractures may require internal fixation with Kirschner wire placement either perpendicular to the fracture orientation in oblique and spiral fractures or crossed in the case of transverse fractures.[4] Displaced fractures have a higher likelihood of presenting with a concomitant nail bed injury; if this is the case, the nail matrix should be repaired at the same time. Fractures of the epiphysis, if associated with avulsion fracture of the terminal extensor tendon, are also known as mallet fractures (▶ Fig. 24.15). These often occur from a jamming injury with hyperflexion against a hard surface. If associated with a nail bed injury, the nail matrix must be repaired. If possible, the nail plate should be replaced to aid in soft-tissue stability. The finger should then be splinted with the DIP in 5 to 10 degrees of extension to minimize the risk of extensor lag.[3,4]

Fig. 24.14 Tuft fracture **(a)** posteroanterior and **(b)** lateral.

24.7.2 Middle and Proximal Phalanx

For middle and proximal phalanx fractures, fractures can occur at the shaft, neck, head, base, or in combination. Shaft fractures can be transverse, oblique, spiral, or comminuted. Stable, nondisplaced, or minimally displaced fractures can be treated by reduction and splinting in safety position to prevent collateral ligament contracture. Spiral and oblique fractures have a higher propensity to shorten after reduction and splinting when compared with transverse fractures; they therefore require close follow-up with serial radiographs.[3,4] If loss of length or alignment occurs, internal fixation is indicated.[4] Fractures with transverse, oblique, or spiral orientations that are not comminuted may be treated with Kirschner wire fixation to allow for early mobility with minimal injury to the soft tissues. Condylar fractures are inherently unstable and usually require operative

fixation with either Kirschner wires or lag screws.[4] Fractures of the base of the middle phalanx warrant special consideration due to the tendinous and ligamentous attachments. Fractures of the dorsal base can result in detachment of the central slip insertion of the extensor mechanism, and often require internal fixation to prevent boutonniere deformity. Fractures of the lateral base can result in detachment of the collateral ligaments; if the fracture fragment is significantly displaced, the joint is likely unstable and requires internal fixation.[4] Severely comminuted or "pilon" style fractures can be stabilized and rehabilitated using dynamic traction. A variety of methods have been described to allow dynamic traction; however, one of the simplest involves use of readily available Kirschner wires and rubber bands.[8] Outcomes from even severe intra-articular fractures using this method demonstrate reasonable motion and stability.[9,10]

Fig. 24.15 Mallet fracture.

24.7.3 Metacarpal

Metacarpal fractures, such as phalangeal fractures, can occur at the head, neck, shaft, base, or combination thereof. Metacarpal head fractures that constitute more than 25% of the articular surface or have more than 1 mm articular step-off require open reduction and internal fixation, often with screw fixation for enhanced stability enabling early joint mobilization.[4] Metacarpal neck fractures, particularly of the ring and small finger, are very common. Often referred to as boxer's fractures, these occur when the ulnar surface of the clenched fist strikes a hard object (▶ Fig. 24.16). These fractures usually have an apex dorsal configuration due to the pull of the intrinsic musculature and can exhibit significant angulation.[3] Decision for operative management is based on the degree of angulation and rotational deformity. The carpometacarpal (CMC) joints have an increasing degree of flexion, extension, and rotation the more ulnarly they are located, with up to 20 to 30 degrees of flexion and extension in the small finger CMC joint.[4] The small and ring fingers can therefore compensate for higher degrees of angulation and rotation in metacarpal neck fractures; up to 30 to 40 degrees of angulation in the ring finger and 50 to 60 degrees in the small finger are acceptable.[4] The index and middle fingers, conversely, can tolerate very little angulation or rotational

Fig. 24.16 Boxer's fracture **(a)** posteroanterior, **(b)** oblique, and **(c)** lateral.

deformity and more often require operative treatment; angulation more than 10 to 15 degrees is unacceptable and requires reduction and fixation.[4] Any rotational deformity that results in finger scissoring or crossing when fingers are flexed into the palm requires reduction and immobilization or fixation.

24.7.4 Thumb

For thumb fractures, phalangeal fractures are treated similarly to finger phalangeal fractures. However, a notable difference is the ability of the thumb to tolerate greater rotational and angular deformity than the fingers due to its greater degree of compensatory movement in all planes. Intra-articular fractures, however, must be treated aggressively with precise reduction and internal fixation to prevent loss of motion and posttraumatic arthritis; this is particularly important in the thumb MCP and CMC joints due to their high degree of mobility and importance to overall hand function.[4] Closed reduction of the thumb can usually be accomplished with axial traction, extension, and pronation of the thumb while applying dorsal pressure to the fracture site.[1]

Fracture patterns unique to the thumb include Bennett's fracture and Rolando's fracture. Bennett's fracture occurs when the thumb metacarpal is axially loaded while partially flexed, resulting in an articular fracture at the metacarpal base consisting of a single volar ulnar fracture fragment.[1,4] The metacarpal subluxes radially, proximally, and dorsally while the fracture fragment is held in anatomic position against the trapezium by the anterior oblique ligament. Closed reduction is attempted by applying axial traction, palmar abduction, and pronation to the thumb while applying pressure to the metacarpal base.[1] If the fracture is able to be reduced, and the fracture fragment small, consisting of less than 20% of the articular surface, fixation can be performed with percutaneous pinning and immobilization with a thumb spica cast.[4] If the fracture is irreducible, has a large fracture fragment, or has more than 2 mm of articular step-off despite closed reduction, open reduction and internal fixation with lag screws is the recommended treatment, again with postoperative immobilization in a thumb spica cast.[4]

Rolando's fracture is a comminuted, intra-articular fracture of the thumb metacarpal base. This fracture pattern nearly always requires open reduction and internal fixation to restore articular congruency.[1,4] For minimally comminuted fractures, anatomic reduction may be achieved with multiple Kirschner wires or screws or with plate application. However, for highly comminuted fractures with small fracture fragments, a mini-external fixation device may be required.

24.7.5 Carpus

Carpal fractures account for 18% of all hand fractures, with the scaphoid and triquetrum being the most frequently fractured carpal bones.[2] Scaphoid fractures often are associated with a fall on an outstretched hand and will have tenderness to palpation in the anatomic snuffbox. Approximately 8 to 20% of scaphoid fractures are not seen on initial radiographs; if there is a high clinical suspicion of scaphoid fracture, the patient is placed into a thumb spica cast and repeat radiographs are performed 10 to 14 days later.[2] Most scaphoid fractures can be treated nonoperatively with a thumb spica cast for 8 to 12 weeks, until radiographic union is seen.[2] Scaphoid fractures are immobilized longer than most hand and wrist fractures due to the relatively poor blood supply to the scaphoid and subsequently relatively higher incidence of nonunion or malunion. Indications for surgical fixation of scaphoid fractures include more than 1 mm displacement, comminution, angulation, carpal instability with scapholunate angle more than 60 degrees or radiolunate angle more than 15 degrees, or an open fracture.[2] Open reduction and internal fixation with a headless compression screw oriented along the long angle of the scaphoid is the standard surgical treatment. Fractures of the other carpal bones can generally be treated with cast immobilization for 4 to 6 weeks, unless the fracture fragments are displaced or the fracture pattern is associated with carpal instability.[2,11]

24.8 Postoperative Care

After surgical fixation is complete, hemostasis achieved, and the skin closed, a dressing and splint is applied. Postoperative dressings vary widely due to surgeon's preference, but generally an occlusive layer such as petroleum gauze is applied to the incision site, fluffed gauze is then applied for padding and fluid absorption, and an appropriate splint is applied to maintain immobilization of the repaired bone. For Kirschner pins, petroleum gauze strips are wrapped around the external pin site, fluffed gauze applied on top or around the pins for padding and fluid absorption, and again a splint applied for immobilization. All fingertips should be visible for monitoring perfusion. The patient is instructed to keep the splint clean and dry. Pain control is achieved with infiltration of local anesthetic at the end of the case, and initiation of oral analgesics such as scheduled acetaminophen and as needed narcotic medication after the patient is fully awakened from anesthesia. Patients who have received regional blocks are advised that they may experience significant pain after the block wears off, approximately 8 to 12 hours after surgery depending on the type of local anesthetic used. They are therefore counseled to begin taking their oral analgesics prior to onset of significant pain.

The dressing will be removed at the first postoperative clinic visit, usually 3 to 5 days after surgery. At that time, a removable splint will be applied that should be worn at all times except when bathing or when working on exercises as directed by hand therapy. The patient is instructed to gently wash the incision or pin sites daily with soap and water, and pat dry prior to reapplying the splint. Radiographs may be taken at the first postoperative visit to ensure repair stability. Patients are usually started on hand therapy at the first postoperative visit and will continue to visit hand therapy for the duration of their healing. Postoperative visit schedules will vary based on the fracture and surgeon's preference, but in general, the patient will return to clinic every 2 to 3 weeks until clinical and radiographic union is documented.

Clinical union occurs when the fracture has healed with good stability at rest and with motion, and without pain to palpation or with motion.[3] This usually occurs within 3 to 6 weeks in hand fractures.[3] Radiographic union often lags behind clinical union by 2 to 4 weeks. Early range of motion is important in hand fractures to prevent postinjury stiffness and adhesions; however, motion must not begin too early and jeopardize stable bony healing. Uninvolved fingers are mobilized immediately

after treatment. Mobilization of the injured bone depends on the type of immobilization method chosen. For splint and cast immobilization, mobility is initiated at the time of clinical union. For stable fixation with pins, plates, screws, or external fixators, mobility can begin very early, usually at 3 to 7 days postoperative.[4] Mobility and weight bearing are gradually increased under the direction of a hand therapist. Patients are initially not allowed to bear any weight on the injured hand, but at the time of clinical union are allowed full weight bearing. Patients may return to work once their pain is adequately controlled, but must adhere to their specific mobility and weight-bearing restrictions, in addition to keeping their hand splint clean and dry. If the patient's job cannot accommodate these restrictions, they cannot return to work until they have achieved clinical union.

24.8.1 Hand Therapy

Hand therapy is a key component of successful hand fracture management and rehabilitation. Hand therapists are specialized occupational therapists with additional training and certification in the management of hand injuries. They help guide the patient through protected and controlled mobilization to minimize the risk of joint stiffness and soft-tissue adhesions, apply techniques to minimize soft-tissue edema, and help remodel restrictive scar tissue. The hand therapist and surgeon work closely together to maintain an optimum balance of stability to ensure bony healing and mobility to minimize postinjury stiffness.

24.9 Outcomes

As discussed earlier, the majority of hand fractures can be managed nonoperatively. Surgical treatment is reserved for management of open, irreducible, unstable, or multiple fractures. Although surgery provides benefits of precise reduction and rigid fixation allowing early movement, it also causes additional soft-tissue trauma that increases the risk of postinjury stiffness. Furthermore, appropriate follow-up care including surveillance radiographs to ensure bony stability and healing, dressing management to aid soft-tissue healing, and hand therapy for appropriate hand mobility and rehabilitation are critical to the successful outcome of hand fracture treatment.

24.10 Review Questions

24.10.1 True or False

1. The initial hand exam should be performed prior to administering local anesthetic.
2. Radiographs should not be taken after fracture reduction and splinting.
3. Kirschner wires apply compression across the fracture.

24.10.2 Choose the Best Answer

4. A 23-year-old right-hand–dominant man presents with ulnar-sided right-hand pain after punching a wall. Physical exam is notable for ecchymosis and edema of the hand as well as pain to palpation over the small finger metacarpal head. What image(s) should be ordered to best evaluate this injury?
   a) Ultrasound of the hand.
   b) CT of the hand.
   c) Three-view radiographs of the hand (AP, lateral, and oblique).
   d) Three-view radiographs of the small finger (AP, lateral, and oblique).
5. Incisions should be avoided on which of the following surfaces:
   a) Central fingertip.
   b) Radial side of the index finger.
   c) Ulnar side of the index finger.
   d) Ulnar side of the small finger.
   e) A, B, and D.
   f) A, C, and D.

24.10.3 Answers

1. True.
2. False.
3. False.
4. Three-view radiographs of the hand (AP, lateral, and oblique) (c).
5. A, B, and D (e).

References

[1] Carlsen BT, Moran SL. Thumb trauma: Bennett fractures, Rolando fractures, and ulnar collateral ligament injuries. J Hand Surg Am. 34(5):945–952

[2] Sebastin SJ, Chung KC. Management of wrist fractures. In: Thorne CH, ed. Grabb's and Smith's Plastic Surgery. Philadelphia, PA: Lippincott Williams & Wilkins; 2014:767–783

[3] Choi MS, Chang J. Management of hand fractures. In: Thorne CH, ed. Grabb and Smith's Plastic Surgery. Philadelphia, PA: Lippincott Williams & Wilkins; 2014:758–766

[4] Day CS, Stern PJ. Fractures of the metacarpals and phalanges. In: Wolfe SW, Hotchkiss RN, Pederson WC, Kozin SH, eds. Green's Operative Hand Surgery. Philadelphia, PA: Churchill Livingstone; 2011:239–290

[5] Hammert WC. Anesthesia techniques. In: Thorne CH, ed. Grabb and Smith's Plastic Surgery. Philadelphia, PA: Lippincott Williams & Wilkins; 2014:727–730

[6] Lalonde D, Bell M, Benoit P, Sparkes G, Denkler K, Chang P. A multicenter prospective study of 3,110 consecutive cases of elective epinephrine use in the fingers and hand: the Dalhousie Project clinical phase. J Hand Surg Am. 30 (5):1061–1067

[7] Fisher L, Gordon M. Anesthesia for hand surgery. In: Wolfe SW, Hotchkiss RN, Pederson WC, Kozin SH, eds. Green's Operative Hand Surgery. Philadelphia, PA: Churchill Livingstone; 2011: 25–38

[8] Ruland RT, Hogan CJ, Cannon DL, Slade JF. Use of dynamic distraction external fixation for unstable fracture-dislocations of the proximal interphalangeal joint. J Hand Surg Am. 33(1):19–25

[9] Ellis SJ, Cheng R, Prokopis P, et al. Treatment of proximal interphalangeal dorsal fracture-dislocation injuries with dynamic external fixation: a pins and rubber band system. J Hand Surg Am. 32(8):1242–1250

[10] Haase SC, Chung KC. Current concepts in treatment of fracture-dislocations of the proximal interphalangeal joint. Plast Reconstr Surg. 134(6): 1246–1257

[11] Geissler WB, Slade JF. Fractures of the carpal bones. In: Wolfe SW, Hotchkiss RN, Pederson WC, Kozin SH, eds. Green's Operative Hand Surgery. Philadelphia, PA: Churchill Livingstone; 2011:639–707

25 Soft-Tissue Reconstruction of the Hand and Upper Extremity

Wendy Lynne Czerwinski and Robert A. Weber

Abstract
This chapter covers the various issues involved in soft-tissue reconstruction of the hand due to defects from various etiologies, such as trauma, tumor extirpation, thermal injuries, and infectious complications. The primary concerns of stable coverage, mechanical loading, pliability, sensation, and aesthetics are individually addressed. Diagnostic options for assessing injuries are listed and surgical techniques—skin grafts and local, regional, chest, and free flaps—are reviewed. Three case examples help illustrate the techniques presented, and seasoned advice on postoperative care and outcomes is provided.

Keywords: stable coverage, mechanical loading, pliability, sensation, skin grafts, flaps

25.1 Goals and Objectives

- Help the reader learn the anatomy and functional needs of the hand that must be considered when planning soft-tissue reconstruction.
- Identify the important information the reconstructive surgeon must gather to plan coverage.
- Understand how the nature and site of the defect lends itself to various reconstructive options.
- Comprehend the surgical technique for some of the more commonly used reconstructive modalities.
- Know the appropriate postoperative regimen to improve outcomes for these reconstructive patients.

25.2 Patient Presentation

25.2.1 Hand Soft Tissue and Function

The hand is the most commonly injured part of the body; as such the surgeon who treats the hand is frequently asked to reconstruct soft-tissue injuries. Soft-tissue deficits of the hand arise from a variety of etiologies including trauma, tumor extirpation, thermal injuries, and infectious complications. The resultant defects can range from only localized skin loss to extensive exposure of tendons, joints, and bone with varying degrees of involvement of these structures. The goal of treatment is not only to provide soft-tissue coverage but also to restore optimal function and appearance. A reconstructed hand that is insensate and does not move is of little benefit to a patient. Limiting long-term sequelae of scarring and hypersensitivity is a priority. At the same time, patients now have a heightened awareness of aesthetic outcomes, and this has been shown to be an important factor in satisfaction of reconstruction.[1,2,3]

The hand has three anatomical structures that make reconstruction a unique challenge. First, the glabrous skin on the volar surface is thicker and has a higher density of sensory end organs than does most skin. Second, the vital structures of the hand are padded by a layer of mechanical fat directly beneath the skin. This adipose tissue has smaller globules, more fibrous tissue, and is tethered to its position, thereby absorbing force and resisting migration. Third, the palmar fascia holds the overlying skin and mechanical fat in place with multiple fibrils which allows the hand to grip firmly and resist shear stress.

Ideal reconstruction of these structures when missing provides stable coverage, allows mechanical loading, does not restrict movement, provides protective sensation, and does not draw visual attention to itself, all with minimal donor-site morbidity (▶ Table 25.1). Achieving all these goals can be challenging; however, attention to the primary treatment effort will help avoid the need for later complex procedures to treat unstable wounds, painful function, or scarred coverage. In addition, the choice of treatment must be practical for the patient and this will vary based on culture, age, and patient demographics such as smoking. The same defect presenting in a young female with high aesthetic goals could be managed differently than in an older male manual laborer. Technically demanding retrograde facial flaps in smokers have higher failure rates and poorer outcomes. Thus, the reconstructive choices for soft-tissue defects of the hand and upper extremity must take into account all of these variables.

From the aforementioned information, the wide range of possible defects, multiple goals that must be achieved, and numerous patient considerations combine to make standardized cookbook approaches to hand soft-tissue deficits limited in their applicability. This chapter aims to provide the reader with practical options that offer reliable outcomes. The reconstructions described are a way to address the challenge and demonstrate the applicability of a specific technique. More important is to see how the surgical choice addresses the underlying needs of the hand within the parameters of a particular patient. Each surgeon will develop his or her own algorithm and tools to manage their particular patient population.

25.2.2 Evaluation of the Patient

Clinical Presentation

Patients with hand and upper extremity soft-tissue deficits can present in both an urgent or elective manner based on their etiology. The mechanism of soft-tissue loss should be described as precisely as possible as it may give information as to the extent of the deficit and potential reconstructive needs. The history may include symptoms of pain, loss of extensor or flexor function, joint or bone instability, and/or sensory disturbance. Questions should also be answered regarding any previous trauma or functional deficits, prior radiation, and functional demands the patient places on the hand.

During the initial evaluation, a surgeon has the advantage of temporizing care with dressings until formal evaluation of the defect is made and a reconstructive plan can be agreed upon

Table 25.1 Demands on the soft tissue of the hand and upper extremity

Stable coverage—the hand sustains shear stress and must withstand friction. The glabrous skin of the volar hand is unique in its durability and capacity to adapt	• Split-thickness skin graft—does not withstand shear stress well • Full-thickness skin graft—handles shear stress well once mature, can even form calluses on the volar surface • Local flap—good vs. shear • Regional skin flap—good vs. shear • Free flap—good vs. shear
Mechanical loading—when used in grip, the hand must provide sufficient padding to the underlying structures. The mechanical fat on the volar hand is very difficult to replace	• Split-thickness skin graft—does not provide any cushion • Full-thickness skin graft—does not provide sufficient cushion for the volar surface of the hand, is adequate for the dorsal hand and entire forearm • Local flap—very good when it includes the underlying mechanical fat, limited donor sites on the hand and fingers • Regional skin flap—can be designed to include fat, but may deform too much with shear stress • Free flap—can be designed to include fat, but may deform too much with shear stress
Pliable—tissue on the dorsum of the hand must be able to stretch when the fingers flex; tissue on the volar surface must be supple and thin enough to bend to 90 degrees	• Split-thickness skin graft—moderately extensible, very pliable • Full-thickness skin graft—both extensible and pliable • Local flap—can stretch, pliability determined by thickness of included fat • Local flap—can stretch, pliability determined by thickness of included fat • Free flap—can stretch, pliability determined by thickness of included fat, fascial flap can be combined with a skin graft
Sensate—the hand is often the first body part to encounter the environment, so it must be able to detect sharpness and temperature	• Split-thickness skin graft—does not have sensory end organs • Full-thickness skin graft—sensory potential dependent on donor site and the presence of underlying nerves • Local flap—good sensation if sensory pedicle retained • Regional skin flap—difficult to achieve sensation • Free flap—can be designed to include nerves
Aesthetics—color mismatch and contour deformities are quickly identified	• Split-thickness skin graft—frequently differs in color and leaves a significant contour depression • Full-thickness skin graft—can match well if proper donor site chosen but may have a significant contour depression • Local flap—ideal color and contour match • Regional skin flap—can match color well, may be too thick • Free flap—can match color well, may be too thick

with the patient. The primary means of evaluating the deficit is by a thorough motor and sensory exam including tissues proximal and distal to the zone of injury. The exam should note the surface area of soft-tissue loss as well as the location, paying particular attention to joint creases that are involved. Depth and degree of involvement of the glabrous skin, mechanical fat, and palmar fascia should also be assessed and documented (▶ Table 25.2).

Table 25.2 Components of physical examination of the hand for soft-tissue coverage

Goal	Evaluation
Motor	• Flexor and extensor tendon function including forearm musculature • Thenar and hypothenar musculature • Intrinsic musculature
Sensory	• Median, ulnar, and radial nerve distributions
Range of motion	• Arc of joint rotation: ◦ DIP, PIP, MP ◦ Wrist, elbow ◦ Forearm rotation • Joint stability
Soft-tissue defect	• Dimension • Location • Contamination or presence of foreign body • Tissue involvement: ◦ Skin, subcutaneous, fascia, ligament, tendon, nerve, vessel, nail
Bone injury	• Angulation/rotational deformity • Bone fracture or defect • Supported by radiographs

Abbreviations: DIP, distal interphalangeal; MP, metacarpophalangeal; PIP, proximal interphalangeal.

25.3 Preparation for Surgery

25.3.1 Diagnostic Adjuncts

Standard 3 view radiographs are obtained for all patients with traumatic etiology. These are also used selectively in tumor extirpation, thermal injuries, and infectious complications as there may be bone involvement. Other imaging modalities such as computed tomography, magnetic resonance imaging, and angiography may be of value in select cases where bone reconstruction may accompany soft-tissue coverage or free tissue transfer is being considered. Photographic documentation of the original and subsequent examinations is helpful for providing a more objective basis for comparisons over time and among providers.

In large traumas where significant blood loss was encountered, a hemogram may be valuable if large immediate reconstructive efforts are planned. As the time from creation of the

defect to planned reconstruction increases, a nutrition workup including albumin and prealbumin is indicated to ensure the patient will be able to heal after the surgery.

25.4 Treatment

25.4.1 Anatomic Classification

Understanding the anatomic nature of the defect and its location will provide the surgeon the key elements in choosing an appropriate reconstructive option for soft-tissue coverage of hand and upper extremity defects. ▶ Table 25.3 describes four anatomic sites or zones of injury requiring soft-tissue reconstruction. Alternatives for care for each of these sites are listed in the right column. The actual reconstruction chosen will depend on the volar or dorsal nature of the defect as well as the degree of tissue loss.

Table 25.3 Options for soft-tissue reconstruction in the hand and upper extremity

Zone 1 (DIP joint to fingertip)	• Secondary intention healing
	• Split- or full-thickness skin grafts
	• Revision amputation with local flap cover
	• Volar V-Y Atasoy advancement • Bilateral V-Y Kutler advancement • Thenar flap • Moberg flap
	• Neurovascular island flaps (retro- or antegrade)
	• Random chest flaps[a]
	• Toe pulp transfers
Zone 2 (finger from MP to DIP joint)	• Full-thickness skin grafts • Composite synthetic dermis and autograft skin[a] • Cross finger flaps • First dorsal metacarpal artery flap (Foucher's kite flap) • Adipofascial turnover flap
Zone 3 (hand from distal wrist crease to MP joint crease)	• Full-thickness skin grafts • Composite synthetic dermis and autograft • Pedicled groin and abdominal flaps • Reverse radial forearm flap and adipofascial variants • Retrograde PIN flap • Dorsal ulnar artery perforator flap • Free tissue transfer (radial forearm, lateral arm, TPF, ALT)[a]
Zone 4 (forearm and elbow)	• Skin graft • Flank flap • Free tissue transfer

Abbreviations: ALT, anterolateral thigh; DIP, distal interphalangeal; MP, metacarpophalangeal; PIN, posterior interosseous nerve; TPF, temporoparietal fascia.
[a]Case examples.

Specific Considerations

Secondary Intention

For most skin-only tissue loss, particularly those wounds 1 to 2 cm^2 or less and not involving a volar joint crease, allowing the wound to heal by secondary intention is a good option. The resultant scar is sufficiently durable for long-term function, and the contracture pulls normally sensate skin into the area. This is particularly a useful way to treat volar zone 1 injuries.

The patient is instructed to wash the wound twice a day with plain soap and water (hydrogen peroxide should be specifically prohibited as many people habitually use it on open wounds of the hand). The wound is then dressed with antibiotic ointment and a nonadherent dressing. Most wounds should close within 2 to 3 weeks.

Skin Grafts

If skin is the only tissue missing, then a skin graft is frequently the first reconstructive choice. Split thickness (0.012–0.014 inch) grafts are acceptable for dorsal finger and hand defects that do not cross a joint. An alternative is to harvest these from the hypothenar area. Some surgeons have begun to place acellular dermal matrices beneath the grafts for improved padding and durability, particularly for wounds with subcutaneous fat loss and thin vascularized tissue covering vital structures (▶ Fig. 25.1). For those wounds missing skin over dorsal joints and for all volar defects, full-thickness skin grafts are preferred. The antecubital fossa, medial arm, and groin provide adequate skin to cover most defects.

An important design note for volar finger defects is that full-thickness grafts used to cover a volar finger crease should extend from midlateral line to midlateral line at the crease (▶ Fig. 25.1). This important step helps decrease the risk of a flexion contracture due to a longitudinal scar crossing a flexion crease. Synthetic composite tissues are an alternative.[4] These can be placed to provide coverage and later skin grafted if needed.

Local Flaps

Cross finger flaps, thenar flaps, etc., are indicated when there is volar skin plus fat loss in the finger, zone 2. The first dorsal

Fig. 25.1 **(a)** Grinding abrasion injury of the dorsal right hand resulting in a 9 × 7 cm soft-tissue deficit with underlying extensor hood and disruption of joint capsules. **(b)** Joint stabilization and closures, extensor tendon repairs, and coverage with a synthetic dermal substitute (Integra).

metacarpal artery can be used to cover zone 2 defects of the thumb and select zone 3 injuries. These wounds often have exposed tendon, neurovascular structure, or bone. Local flaps offer durable skin and padding and can be placed over avascular tissue. Their disadvantage lies in the fact that they are insensate and can produce a noticeable donor-site defect. They are also limited to smaller, single defects.[5]

Regional Flaps

For larger soft-tissue defects or multiple separate defects, regional flaps such as a reverse radial forearm flap or posterior interosseous flap may be needed.[6] Like local flaps, these reconstructive options provide durable, padded skin which can be used to cover exposed vital structures. Also, like local flaps, they are insensate and produce donor-site morbidity. Large volar areas in zone 3 that have palmar fascia loss can be reconstructed with these flaps, but the patient will notice the shifting skin in the palm when shearing forces are applied, so it is important to inset the skin under slight tension around the edges.

Chest Flaps

The chest flap is a frequently overlooked but highly useful flap for the hand (▶ Fig. 25.2, ▶ Fig. 25.3, ▶ Fig. 25.4, ▶ Fig. 25.5). The chest flap is an ideal choice for the hand with multiple zone 2 defects with exposed structures and in those patients for whom a regional or free flap is not an option due to anatomic or patient health factors. Raised as a random pattern skin flap that can also carry a layer of adipose tissue, chest flaps can be tailored to fit small and large defects (a single flap can provide circumferential coverage of an entire zone 2 defect), and are often the best choice when having to reconstruct defects of multiple

Fig. 25.2 (a,b) Traumatic avulsion amputation of the right ring finger with volar and dorsal soft-tissue deficit. **(c)** Bony avulsion at the level of the distal interphalangeal joint.

Fig. 25.3 (a–c) Intraoperative images of the contralateral chest flap elevation and attachment.

Fig. 25.4 (a–c) Intraoperative images of the contralateral chest flap division and insetting.

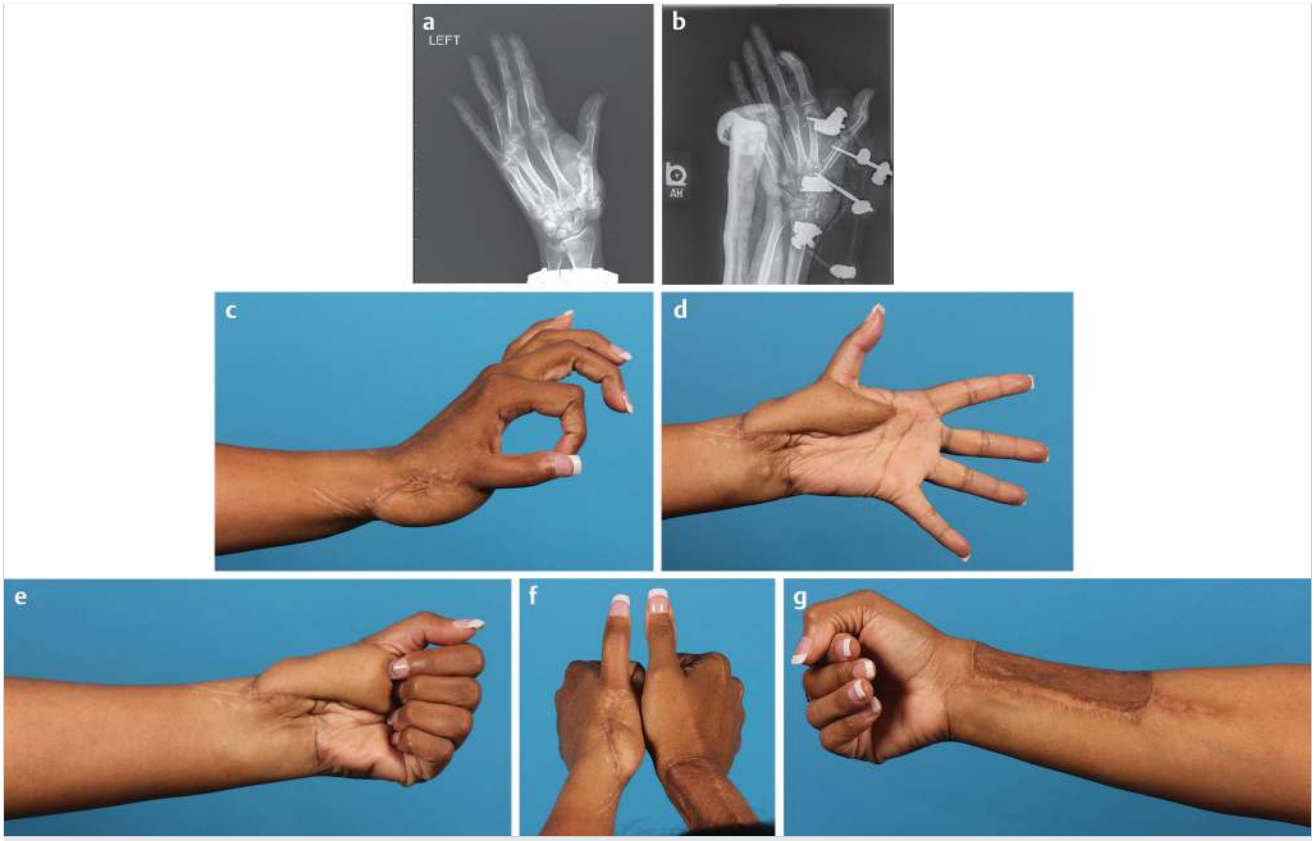


Fig. 25.5 **(a)** Radiograph demonstrating level of traumatic injury to the left hand. **(b)** The patient underwent debridement, placement of an external fixator, iliac crest bone grafting with internal pin stabilization, and free radial forearm soft-tissue coverage. **(c-g)** Postoperative aesthetic and functional outcomes are very good.

digits. The abdomen and groin can also be used, but hand placement is more dependent (resulting in significant edema that must be mobilized for motion) and more awkward.

The choice of donor site is made by placing the hand over the chest and determining if the elbow and shoulder joints are more relaxed with the hand at the ipsilateral or contralateral inframammary crease. The base of the donor site is then placed in the inframammary crease, thereby making the final scar less obvious. The length of the flap must be designed to cover the defect as well as the distance from the defect to the chest wall. Multiple fingers can be covered by adjacent flaps, but the final scars become more obvious on the chest wall.

The flaps are elevated with a thin layer of fat, the donor site partially closed, and the flaps are inset onto the finger or hand. Additional sutures are placed temporarily securing the hand to the chest, so the patient does not inadvertently pull the hand off the chest. A circumferential wrap can be placed around the chest and arm to further secure the flap.

The flaps are usually small enough to be divided at 3 weeks without flap loss, but if there is a concern about flap viability, the pedicle can be partially divided at 1 week. At final division and inset, the flap can be divided during the wound prep so that the hand and chest wall can be closed simultaneously. The donor site can be revised to create a less noticeable scar, and the flap trimmed to produce less functional interference.

Free Flaps

Large soft-tissue defects of the hand and forearm with exposed tendon and/or bone often require free flap coverage (▶ Fig. 25.5). With the development of improved microsurgery skills and knowledge of more fasciocutaneous free tissue donor sites, the original reconstructive option of bulky rectus or latissimus muscle and skin graft is now much lower on the surgical options list. The anterolateral thigh (ALT) flap can provide a large area of fascia with or without skin, and sensory innervation can frequently be a part of free flap reconstruction. Similarly, radial forearm free or pedicled variants can also provide a large area of coverage without bulk. The key to using free flaps for forearm and hand reconstruction is to minimize bulk. Muscle flaps will be thinned over time and can be acceptable in the forearm, but the hand requires more supple restoration.

25.4.2 Case Examples

Case 1—Composite Synthetic Dermis and Autograft Skin

A 76-year-old man presented to the emergency department (ED) after sustaining a grinding abrasion injury of the dorsal right hand resulting in a 9×7 cm soft-tissue deficit. He had underlying extensor hood injuries at and just proximal to the

metacarpophalangeal (MP) joints with unstable joints and disruption of joint capsules. Because of his advanced age and medical comorbidities, a reconstructive plan that minimized operative time and systemic stress was outlined.

He underwent joint stabilization and closures, extensor tendon repairs, and coverage with a synthetic dermal substitute (Integra; ▶ Fig. 25.1). Several weeks later, neovascularization of the dermal graft was achieved; so, we proceeded with a split-thickness skin graft for final coverage. He had a good outcome with stable coverage, acceptable range of motion (ROM), and minimal donor-site deficit.

Case 2—Chest Flap

A 26-year-old man presented to the hand center with an avulsion injury to his right ring finger (▶ Fig. 25.2). There was loss of soft-tissue proximal to the proximal interphalangeal joint and bone loss through the joint. The patient desired to maintain length of the digit. He was taken to the procedure room and local anesthetic was used to infiltrate the left anterior chest and establish a ring finger digital block. Nonviable tissue was debrided from the digit and the cartilage cap removed from the proximal phalanx.

A chest flap was designed along the contralateral inframammary crease and elevated to include skin and subcutaneous tissue (▶ Fig. 25.3). The flap was sutured to the digit and the patient remained in this position with a supportive sling for the next 3 weeks. Under local anesthetic, the flap was then divided and inset and the donor site closed primarily. The patient did well with stable wound coverage, preservation of length, and minimal donor deformity (▶ Fig. 25.4).

Case 3—Free Radial Forearm Flap

A 43-year-old right-handed woman sustained a gunshot wound to the radial border of the left hand. She had skin and subcutaneous tissue loss including the dorsal/radial aspect of the wrist and thumb, the volar radial palm, and the entire thenar musculature. There was complete destruction of the base of thumb and first carpometacarpal (CMC) joint.

She underwent a staged reconstruction. Her initial procedure was a washout, debridement, and thorough assessment of injury. She then underwent multiple vessel and nerve repairs with placement of an external fixator. The next stage involved reconstruction of the base of thumb and first CMC joint with placement of an iliac crest cortical/cancellous block and rolled abductor pollicis longus tendon interposition. Coverage of the soft-tissue defect was achieved with a neurotized contralateral radial forearm free flap incorporating the cephalic vein for outflow (▶ Fig. 25.5). She has a very good functional and aesthetic result with acceptable donor-site morbidity.

25.5 Postoperative Care

Postoperative care for hand and upper extremity patients is critical to the successful outcome of their reconstruction. Intraoperative splinting is done with plaster which is converted to thermoplastic alternatives by our occupational hand therapy (OT) colleagues at first follow-up. The splints maintain safe joint position and protect healing bone, tendon, and ligamentous injuries. Care is taken to avoid any pressure points over flaps or grafts. OT plays a pivotal role in the rehabilitation of these patients. The preference in our practice is to maintain the intraoperative dressing until the patient's first follow-up visit, up to 2 weeks. This avoids patient manipulation of their wounds and splints. This has not led to any increase in wound healing complications.

For grafting procedures, patients are typically evaluated at 1 week postoperative to assess graft take and to start local wound care and begin ROM if not otherwise contraindicated. Local flap reconstruction follow-up is normally at 2 weeks to permit wound evaluation and suture removal simultaneously.

For regional and distant pedicle flaps, patients are evaluated for their wounds at 1 week, but division and flap inset are normally planned at about 3 weeks after the initial operation. Free tissue transfer patients remain in hospital from 3 to 5 days depending on the severity of the injury, patient health, and flap progress. These patients are routinely maintained on an 81 mg aspirin, kept in warm environments, and not permitted to use nicotine products.

Hand therapy will vary widely in this patient population dependent on the nature of the defect.

25.5.1 Outcomes

Due to the diverse etiologies and treatment strategies for soft-tissue reconstruction of the hand, well-conducted comparative outcome studies are virtually nonexistent. There are, however, some case series evaluating and advocating various surgical strategies such as healing by secondary intension, toe pulp transfer for digit coverage, thinned ALT flaps, and synthetic substitutes.[7,8,9,10,11,12,13,14,15] Prospective comparative studies are needed. Due to the wide variety and small numbers treated in single institutions, multicenter cooperation is necessary.

As noted at the beginning, the variety of soft-tissue deficit etiologies, coupled with multiple anatomic and functional needs, makes strict algorithm-based treatment paradigms difficult. The focus needs to remain on stable reconstruction, functional rehabilitation, and sensitivity to aesthetics tailored to each individual's circumstances. The surgeon operating on the hand should be comfortable with a wide range of reconstructive techniques that can be employed, choosing the simplest approach to attain the patient's goals.

25.6 Review Questions

25.6.1 Fill in the Correct Answer

1. A 30-year-old man is seen in the ED with a 1 × 2 cm abrasion injury over the dorsum of the left long finger. There is complete loss of the skin; the extensor tendon is not exposed. What reconstructive option would you offer this patient?
2. A 15-year-old girl has a full-thickness electrical burn to the volar surface of her right index finger. What structures are likely to be exposed after debridement? What would you use to reconstruct the finger if the flexor tendon and radial neurovascular bundle are exposed?
3. A 42-year-old man is transferred to your clinic 2 days after an avulsion injury with near circumferential avulsion injury of the right long, ring, and little finger tips. He has 12 mm of

exposed distal phalanx with no fracture. What would be this patient's best reconstructive option?

4. You are asked to see a 71-year-old woman with multiple medical comorbidities who developed a 3 × 5 cm full-thickness defect over the dorsum of her hand from a hematoma she sustained during a fall. What reconstructive option is safest for her? If she has exposed extensor tendons, would that change your plan?
5. A 53-year-old man presents with a 6 × 4 cm wound of the dorsoradial left hand with exposed thumb CMC joint, extensor tendons, metacarpal, MP joint, and index CMC joint after resection of a benign tumor. The dorsal branch of the radial artery is intact. How would you reconstruct this defect?
6. Assuming you chose a reverse radial forearm flap, as you clamp the proximal radial artery having completely dissected out the flap, you notice the skin paddle turns blue with no bleeding. How would you proceed?

25.6.2 Answers

1. FTSG, secondary intention is likely to result in a scar contracture.
2. Cross finger flap, FTSG will not provide coverage of the tendon and nerve.
3. Chest flaps to the finger tips; this allows for the maintenance of useful length. Three amputations would produce significantly decreased function of the hand.
4. Her safest option is a synthetic dermal substitute and wound vacuum-assisted closure with skin grafting once she has formed sufficient granulation tissue. This is suitable for exposed tendons as most patients are able to regain sufficient tendon gliding to allow adequate hand function.
5. Reverse or free radial forearm flap.
6. Examine the radial artery. If reverse flow can be established, continue with the flap. Otherwise, use the flap as a free flap once suitable recipient vessels can be found.

References

[1] Kovacs L, Grob M, Zimmermann A, et al. Quality of life after severe hand injury. J Plast Reconstr Aesthet Surg. 64(11):1495–1502

[2] Papadopulos NA, Kovacs L, Krammer S, Herschbach P, Henrich G, Biemer E. Quality of life following aesthetic plastic surgery: a prospective study. J Plast Reconstr Aesthet Surg. 60(8):915–921

[3] von Soest T, Kvalem IL, Roald HE, Skolleborg KC. The effects of cosmetic surgery on body image, self-esteem, and psychological problems. J Plast Reconstr Aesthet Surg. 62(10):1238–1244

[4] Friedrich JB, Katolik LI, Vedder NB. Soft tissue reconstruction of the hand. J Hand Surg Am. 34(6):1148–1155

[5] Panattoni JB, De Ona IR, Ahmed MM. Reconstruction of fingertip injuries: surgical tips and avoiding complications. J Hand Surg Am. 40(5):1016–1024

[6] Page R, Chang J. Reconstruction of hand soft-tissue defects: alternatives to the radial forearm fasciocutaneous flap. J Hand Surg Am. 31(5):847–856

[7] Adani R, Rossati L, Tarallo L, Corain M. Use of Integra artificial dermis to reduce donor site morbidity after pedicle flaps in hand surgery. J Hand Surg Am. 39(11):2228–2234

[8] Chen C, Tang P, Zhang L. Reconstruction of a large soft-tissue defect in the single finger using the modified cross-finger flap. J Plast Reconstr Aesthet Surg. 68(7):990–994

[9] Chen QZ, Sun YC, Chen J, Kong J, Gong YP, Mao T. Comparative study of functional and aesthetical outcomes of reverse digital artery and reverse dorsal homodigital island flaps for fingertip repair. J Hand Surg Eur Vol.

[10] Jin YT, Guan WX, Shi TM, Quian YL, Xu LG, Chang TS. Reversed island forearm fascial flap in hand surgery. Ann Plast Surg. 15(4):340–347

[11] Lemmon JA, Janis JE, Rohrich RJ. Soft-tissue injuries of the fingertip: methods of evaluation and treatment. An algorithmic approach. Plast Reconstr Surg. 122(3):105e–117e

[12] Lin CH, Lin YT, Sassu P, Lin CH, Wei FC. Functional assessment of the reconstructed fingertips after free toe pulp transfer. Plast Reconstr Surg. 120(5):1315–1321

[13] Mennen U, Wiese A. Fingertip injuries management with semi-occlusive dressing. J Hand Surg [Br]. 18(4):416–422

[14] Torres-Ortíz Zermeño CA, López Mendoza J. Aesthetic and functional outcomes of the innervated and thinned anterolateral thigh flap in reconstruction of upper limb defects. Plast Surg Int. 2014:489012

[15] Zheng H, Liu J, Dai X, Machens HG, Schilling AF. Free lateral great toe flap for the reconstruction of finger pulp defects. J Reconstr Microsurg. 31(4):277–282

26 Tendon Repair

Brian D. Rinker

Abstract
This chapter covers the range of issues involved in the remediation of tendon injuries. The advances that have been made in tendon repair and rehabilitation are discussed in detail. The five zones of the flexor system are delineated for the purpose of diagnosis and treatment. Surgical techniques for repair of routine tendon lacerations in each zone are reviewed and recommendations made regarding proper anesthesia (general, regional), incisions, and antibiotic regimens. The chapter concludes with information pertinent to postoperative care and possible outcomes.

Keywords: flexor tendon, extensor tendon, bowstringing, Brunner incision, epitendinous suture

26.1 Goals and Objectives

- Understand the proper clinical evaluation of patients with tendon injuries.
- Understand the technical aspects of tendon repair and the physiologic basis for effective tendon repair techniques.
- Appreciate the key role of postoperative rehabilitation in the recovery of function following tendon repair.
- Know the common adverse outcomes following tendon repair and the evidence-based perioperative care to maximize patient safety and quality.

26.2 Patient Presentation

Tendon injuries pose a formidable challenge to the reconstructive surgeon. The natural responses of the body to injury, inflammation, edema, and scar formation are detrimental to tendon excursion and limit the degree of function that can be regained after tendon repair. However, repair techniques have been developed to restore tendon continuity and preserve tendon glide. Likewise, rehabilitation strategies have focused on limiting edema and preventing adhesion formation through early motion. Tendon injuries are still often devastating to patients and challenging to surgeons, but with modern techniques, an acceptable return of function can be anticipated in the majority of cases.

The patient with a tendon injury will present with loss of flexor or extensor function. However, the deficit may be subtle and may escape the notice of the patient, especially in the setting of multiple or complex injuries. A careful assessment of tendon function should be performed in every patient with a laceration through the skin in the forearm, wrist, or hand. Tendon injuries can occur in the absence of a laceration, as well. Patients with traumatic or attritional tendon ruptures may present with pain, ecchymosis, sudden loss of function, and tenderness at the rupture site, usually at the tendon insertion or the musculotendinous junction.

26.2.1 Flexor Tendon Injuries

The flexor system has been divided into five zones for the purpose of diagnosis and treatment (▶ Fig. 26.1).[1,2] Zones 1 and 2 are characterized by the presence of the fibro-osseus digital sheath and a flattened layer of fibroblasts on the surface of the tendons, termed the "epitenon," which must be restored in order for tendon repair to be successful. The pulley system of the flexor tendon sheath consists of thick annular pulleys which provide a gliding surface for the tendon and resist palmar translation, and cruciform pulleys which are collapsible, allowing digital flexion to occur without buckling of the tendon sheath (▶ Fig. 26.2). The A2 and A4 annular pulleys, which arise from the proximal and middle phalanges, respectively, are the key pulleys which must be preserved or restored in tendon repair to prevent tendon "bowstringing."[3]

On inspection, the patient with a flexor tendon injury may have a loss of the normal finger cascade. A digit will display loss of active distal interphalangeal (DIP) and proximal interphalangeal (PIP) joint flexion if the flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP) tendons are both divided, or loss of only DIP flexion if the FDP alone has been divided. As only the FDP tendon traverses the DIP joint, the FDP function can be assessed by asking the patient to actively flex the DIP joint of the affected finger. The FDS function is tested by holding the DIP joints of the adjacent fingers in extension, stabilizing the proximal phalanx of the affected finger to eliminate intrinsic muscle action, and asking the patient to flex the PIP joint of the affected finger. This takes advantage of the fact that the FDP tendons to the long, ring, and small fingers share a common muscle belly and function as a single unit. As the FDP to the index finger is usually an independent musculotendinous unit, this test is unreliable for the index finger. Additionally, the FDS to the small finger may be dependent on the FDS to the ring finger, or it may be absent altogether.[4] Weakness, pain, or triggering of the digit may be indicative of a partial tendon laceration.

Patients with flexor tendon injuries in the digits or palm should be carefully examined for concomitant injuries of the arteries and nerves due to the close anatomic relationship between the digital bundles and flexor tendon sheath.

26.2.2 Extensor Tendon Injuries

Extensor tendon injuries should be suspected with any laceration of the extensor surface. Extensor tendon injuries are categorized into eight zones, with odd zones corresponding to injuries occurring over joints and even zones referring to injuries occurring between joints (▶ Fig. 26.3). Due to the vulnerable superficial location of the extensor tendons, they are injured more commonly than flexor tendons,[5] and functional deficits may be subtle due to the numerous interconnections between the extensor tendons. Injuries in zones 1 and 2 present with extensor lag and inability to extend at the DIP joint. In

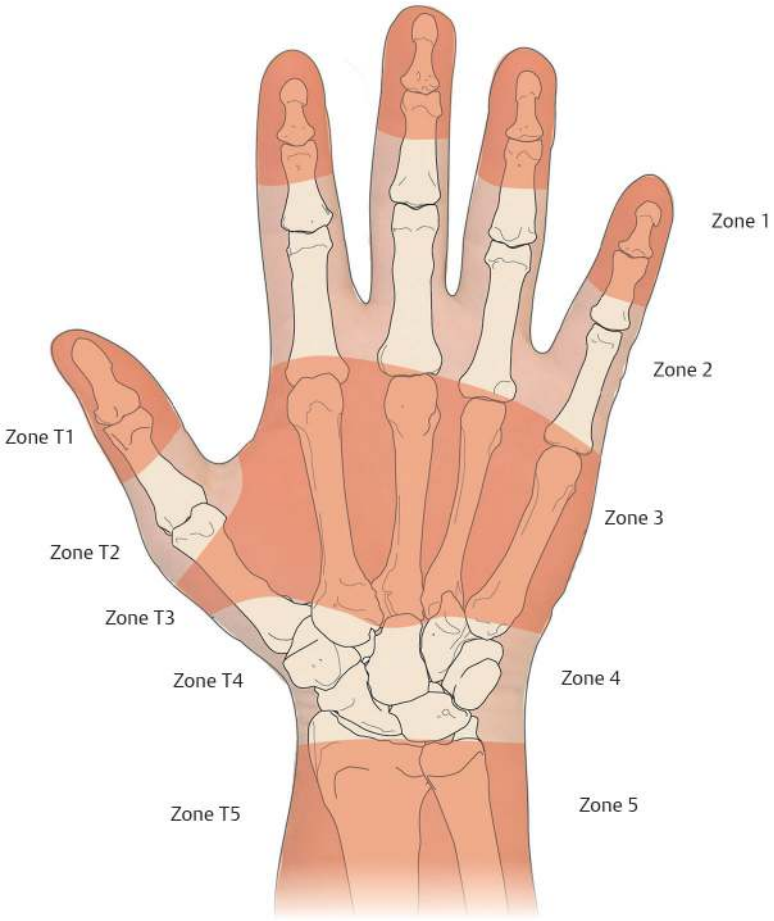


Fig. 26.1 Zones of flexor tendon repair.

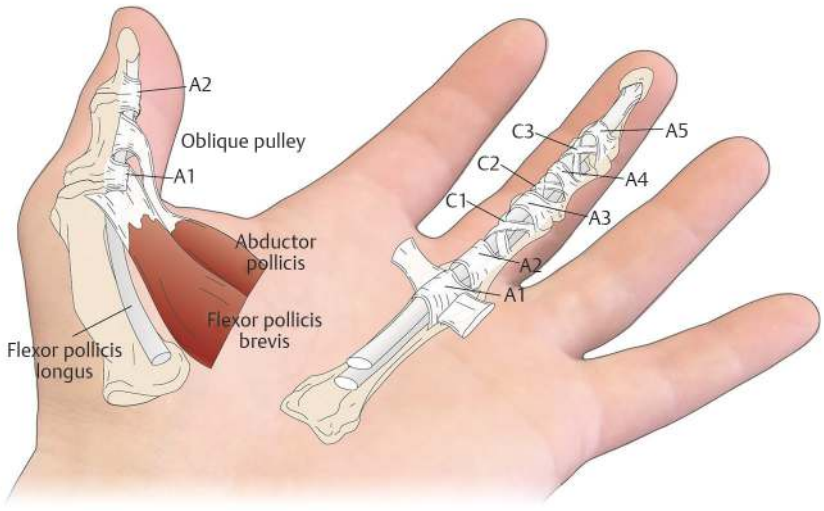


Fig. 26.2 Digital pulley system.

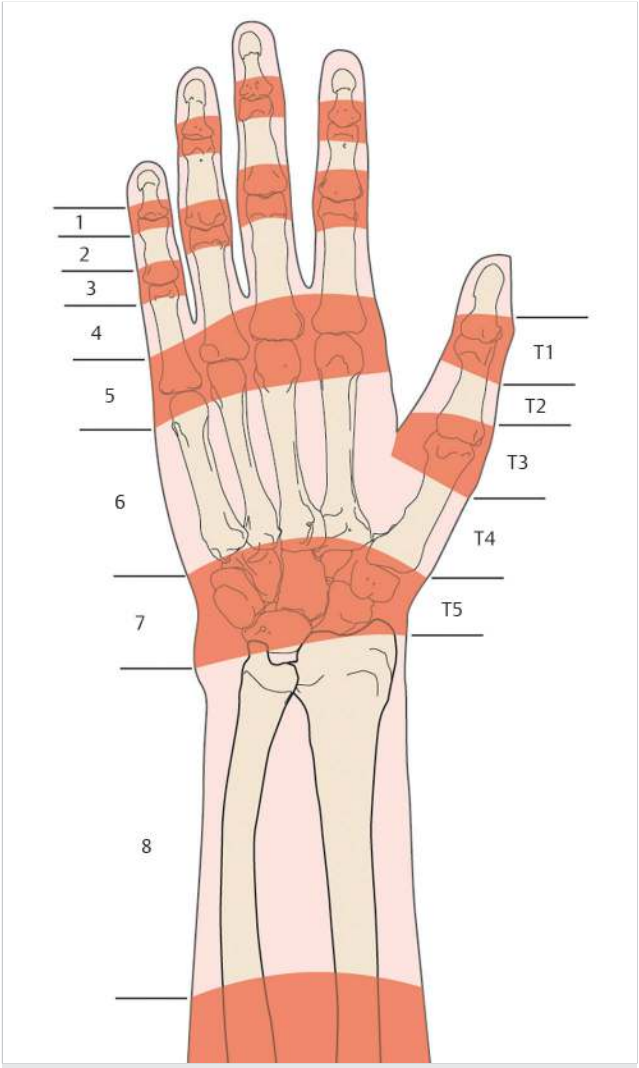


Fig. 26.3 Zones of extensor tendon repair.

zones 3 and 4, the extensor mechanism is broad and flat and covers up to three-fourths of the surface area of the phalanx; therefore, partial injuries are common. Patients may not present with extensor lag at the PIP joint, even with complete rupture or severance of the central slip, due to the action of the lateral bands (▶ Fig. 26.4). A subtle extensor lag may be detected by examining the PIP extension with the wrist and metacarpophalangeal (MP) joints in full flexion. Tendon lacerations in zones 5 and 6 are common, and may result in minimal MP extensor lag due to the presence of the juncturae tendinum. However, its effects can be eliminated by holding all of the MP joints in flexion and having the patient individually extend the proximal phalanx of the affected digit. Lacerations in zones 7 and 8 are often multiple due to the close relationship of the tendons at the extensor retinaculum, and present with inability to extend one or more digits. In small children or patients who are unable to cooperate with the exam, a passive wrist tenodesis test may be helpful.

26.3 Preparation for Surgery

The diagnosis of flexor tendon injuries can usually be made on the basis of physical examination alone. However, radiologic studies can be helpful in many cases. For example, extensor tendon ruptures in the digits are often associated with avulsive fractures which are best visualized on lateral radiographs. Routine radiographs should also be obtained for sharp tendon lacerations in the digits or hand, as there may be concomitant fractures or radiopaque foreign bodies. Although not generally necessary, ultrasonography can be used to diagnose flexor tendon ruptures or lacerations in questionable cases, or to determine the level of proximal tendon stump retraction.[6,7] This is particularly important where there has been a delay between injury and presentation, and the degree of retraction will determine the necessary treatment. Laboratory tests are not necessary on a routine basis, but are dictated by the presence of comorbidities or concomitant injuries.

Several authors have stressed the importance of early primary repair of flexor tendons; however, the repair need not be done immediately after injury unless there is a vascular injury requiring emergency revascularization.[8,9,10,11] Routine tendon lacerations can be treated acutely with wound irrigation and closure in the emergency department, with application of a forearm-based resting splint to prevent motion and tendon retraction. Flexor tendons should be repaired within 5 to 7 days following injury to avoid myostatic retraction of the musculotendinous unit which may preclude a primary repair. Extensor tendons, with the exception of the extensor pollicis longus tendon, do not retract significantly after laceration, and the timing of repair can safely be delayed up to 14 days, if necessary. However, there are little objective data evaluating the effect of timing on outcomes in tendon repair.

26.4 Treatment

Surgical exploration and repair are indicated for all patients with a diagnosis of an acute tendon laceration, with the exception of patients whose medical conditions or life-threatening injuries render the risk of surgery unacceptably high. Tendon repair surgery can be performed under general or regional anesthesia, as dictated by patient and surgeon preference. Recently, there has been an increased interest in wide-awake flexor tendon repair, with local infiltration of lidocaine with epinephrine. This technique allows the patient to follow commands, so that active tendon glide can be assessed on the table immediately following the repair.[12,13] However, the technique may not be appropriate for patients with a nervous temperament or who have difficulty lying still for extended periods.

Prophylactic antibiotics, usually consisting of a first-generation cephalosporin, are administered prior to tourniquet elevation. The upper arm tourniquet is used to provide a bloodless surgical field. The wounds are irrigated and any devitalized tissue is excised. Flexor tendons are exposed through zigzag (Bruner's) incisions on the volar digits, or through extensions of the laceration along the midlateral line (Bunnell's incisions; ▶ Fig. 26.5).[14,15] Flaps should be elevated in as deep a plane as possible to avoid devitalizing the skin, but care must be taken to

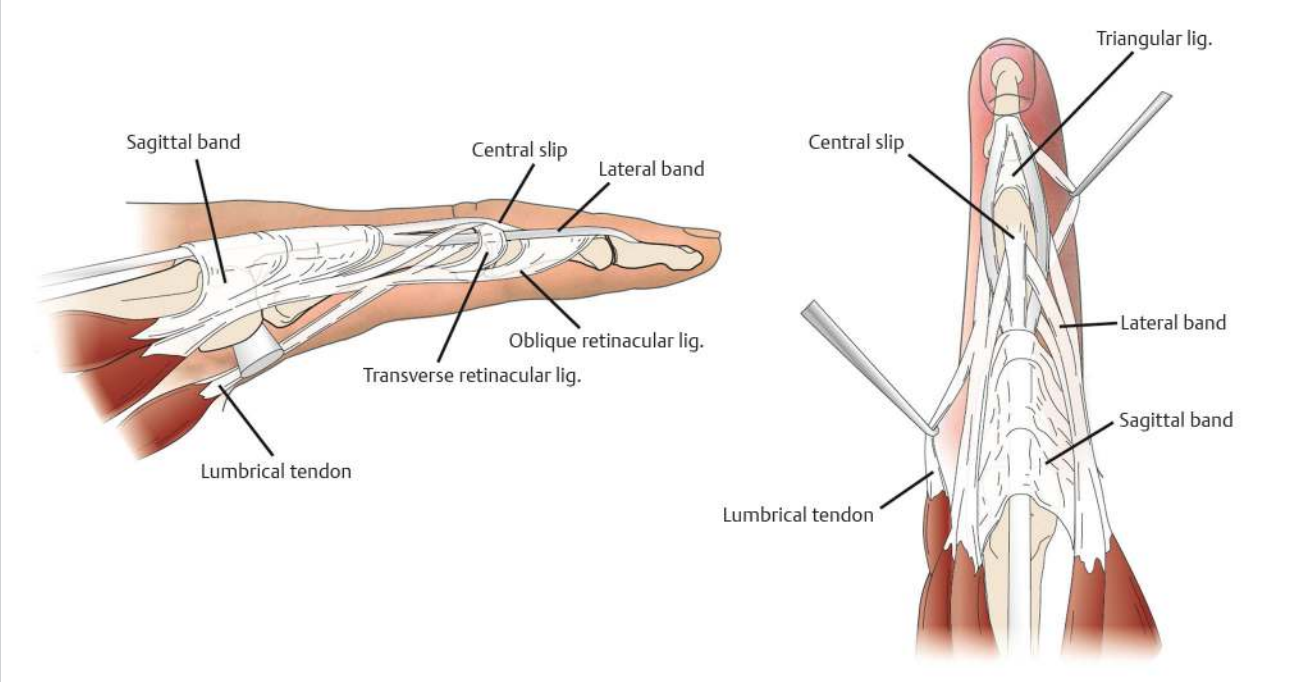


Fig. 26.4 Extensor tendon anatomy.

Fig. 26.5 Common incisions for exposure of flexor tendon injuries.

identify and protect the digital arteries and nerves. Extensor tendons are exposed through curvilinear extensions of the lacerations, avoiding incisions over the apex of joints when possible.

26.4.1 Flexor Tendon Repair—Zone 1

In zone 1, only the FDP tendon is present. The proximal end of the tendon is often held by a vinculum and readily visualized beneath the sheath. If so, it is grasped gently by the epitenon with forceps, retrieved into the wound, and held in place with a hypodermic needle passed through the skin. Occasionally, the tendon retracts into the proximal finger or palm. If so, the Bruner's incisions must be extended proximally until the tendon is visualized. A small opening is made in the tendon sheath to access the proximal cut end of the tendon. It can be sutured to a fine pediatric feeding tube, which is used to draw the tendon distally through the fibro-osseous sheath to the repair site (▶ Fig. 26.6). Blindly instrumenting or otherwise traumatizing the tendons and sheath must be avoided. Any frayed tendon ends are sharply debrided with a scalpel using a tongue depressor as a "cutting board." Excessive trimming must be avoided, as shortening a lacerated FDP tendon by more than 1 cm may result in a "quadrigia" effect on the intact FDP tendons, limiting their excursion.

The distal cut end of the tendon is assessed. If 1 cm or more of tendon remains, repair by primary suture can be performed, similar to treatment in zone 2. If less than 1 cm of distal tendon remains, the proximal cut tendon must be reattached directly to the distal phalanx. The insertion site is prepared by elevating a distally based flap of periosteum and residual tendon and roughening the volar cortex with a rongeur. In the traditional method for reinserting an FDP tendon to the distal phalanx, a core suture is placed in the tendon and used to secure it to the insertion site. The free ends of the suture are passed either around the distal phalanx or through a drill hole in the distal

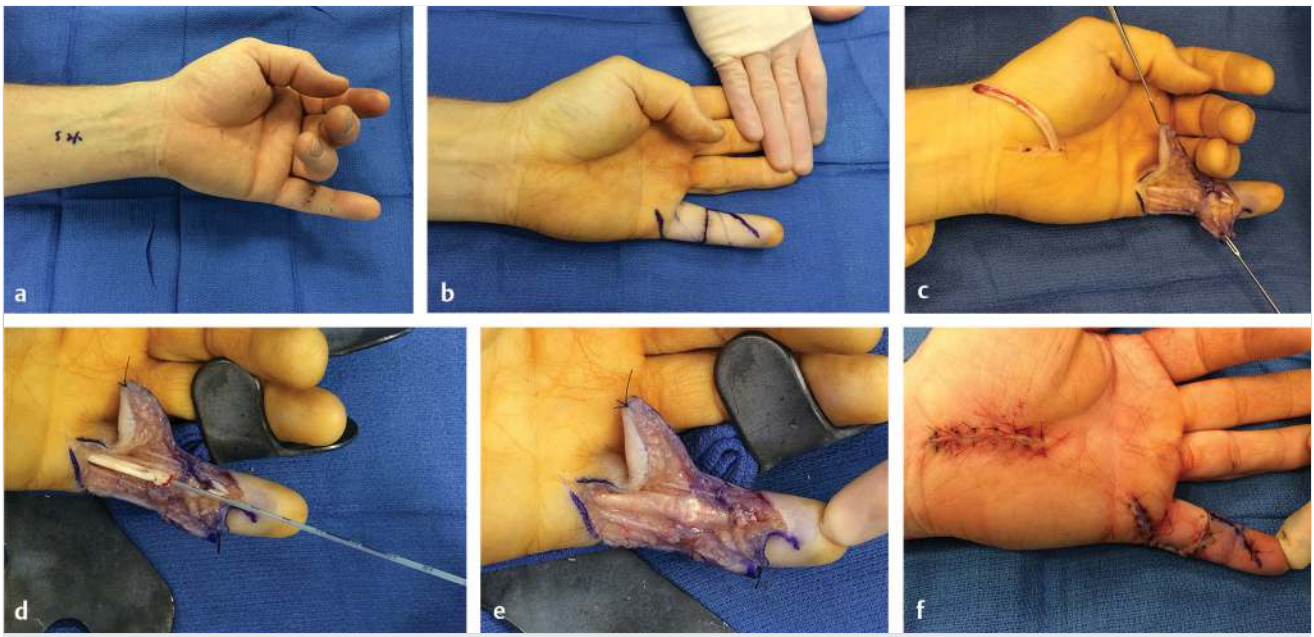


Fig. 26.6 **(a)** A 35-year-old man 2 weeks following knife injury to the left small finger with loss of active DIP flexion. **(b)** Planned incisions for exposure of the tendon sheath. **(c)** Exposure showing an intact FDS tendon and a zone 1 laceration of the FDP tendon. The proximal FDP had retracted and was retrieved through a carpal tunnel incision. **(d)** A pediatric feeding tube was used to pass the tendon down the tendon sheath. **(e)** Following tendon repair distal to the A4 pulley. **(f)** Immediate postoperative appearance.

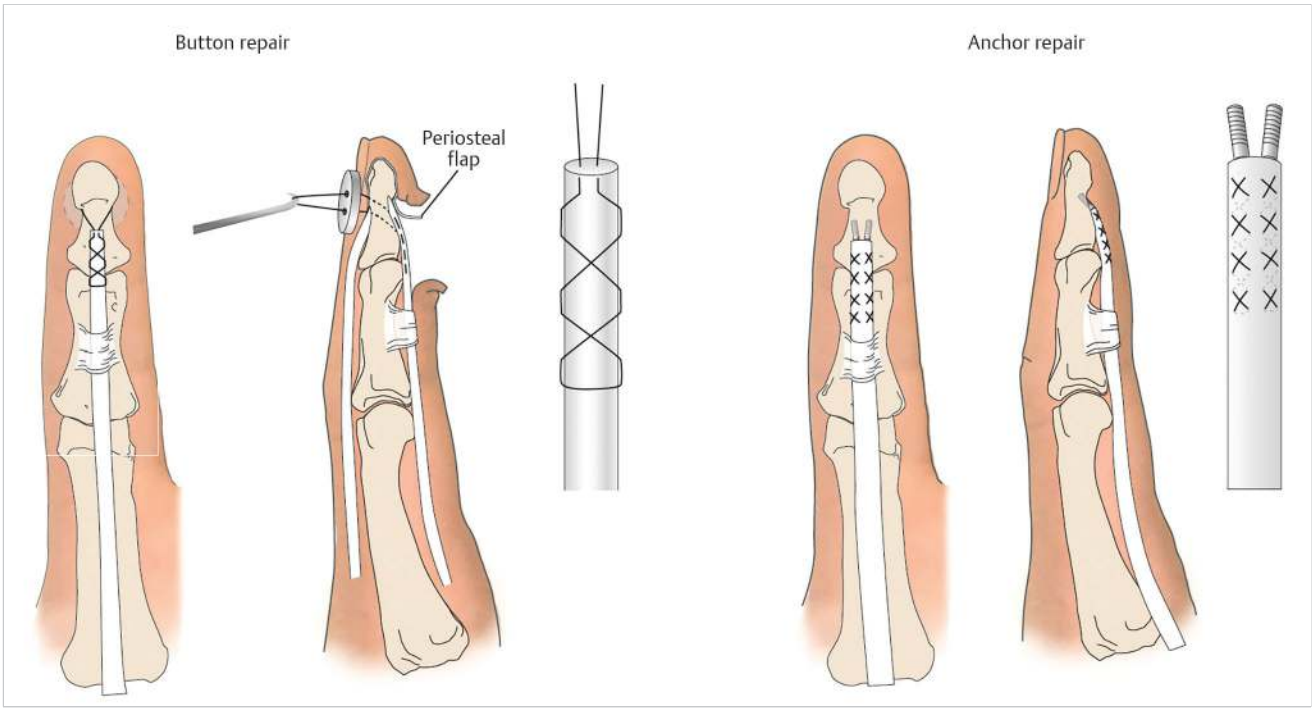


Fig. 26.7 Zone 1 flexor tendon repair techniques.

phalanx and secured over the fingernail with a button. Alternatively, one or two miniature suture anchors can be used to secure the tendon to the phalanx (▶ Fig. 26.7). Both techniques have been shown to provide equivalent repair strength, sufficient to allow early motion.[16]

Distal tendon ruptures are managed in a similar fashion as lacerations. If a small avulsive bone fragment is present, it can be excised to facilitate tendon repair. If the fragment is large, it should be secured to the distal phalanx with a small lag screw or Kirschner wire.

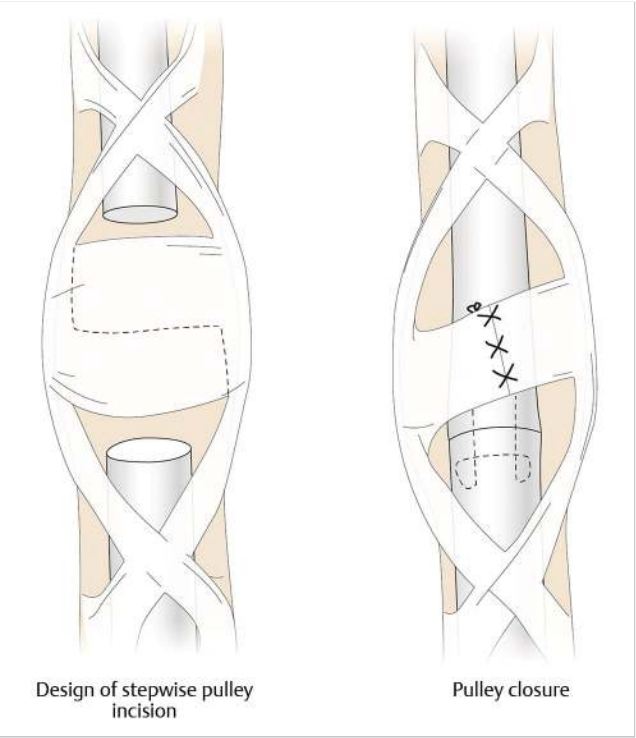


Fig. 26.8 Stepwise division and closure of annular pulley to accommodate repair.

26.4.2 Flexor Tendon Repair—Zone 2

The challenges inherent to zone 2 flexor tendon repair stem from the need to preserve differential gliding of two flexor tendons within a tight fibro-osseous sheath. The wounds are extended proximally and distally as needed to expose the tendon ends and to permit repair (▶ Fig. 26.5). Knowledge of the mechanism of injury can determine whether to extend the laceration proximally or distally. If the finger was lacerated while in a flexed position, as when grasping a knife blade, the distal end of the tendon will likely be found distal to the skin laceration. The distal end of a tendon cut while the digit was extended will be found close to the skin laceration.

If the cut tendon ends are visible, they can be gently grasped with a forceps or hemostat by the epineurium and retrieved. If not, flexing the wrist and MP joints while gently "milking" the tendon sheath from proximal to distal will often deliver the cut proximal ends into the wound. If this maneuver fails, the tendon sheath should not be instrumented blindly, but rather opened proximally to retrieve the tendons. Occasionally, an FDS tendon will retract to the carpal tunnel level, but the FDP tendons are prevented from migrating proximally by the lumbrical muscle. The tendon can then be passed down the sheath with a feeding tube as described earlier. The tendon sheath should be preserved as much as possible, but the pulleys may need to be opened slightly to access the tendon for repair and to permit gliding. An annular pulley can be cut in a stepwise fashion and repaired as a Z-plasty to increase the volume in the sheath to accommodate the repair site (▶ Fig. 26.8).

Repair techniques vary greatly among surgeons but are guided by some basic underlying principles. The tendons are generally repaired with core sutures consisting of 3–0 or 4–0 polypropylene or nylon. A number of techniques have been described for the application of core sutures (▶ Fig. 26.9), but

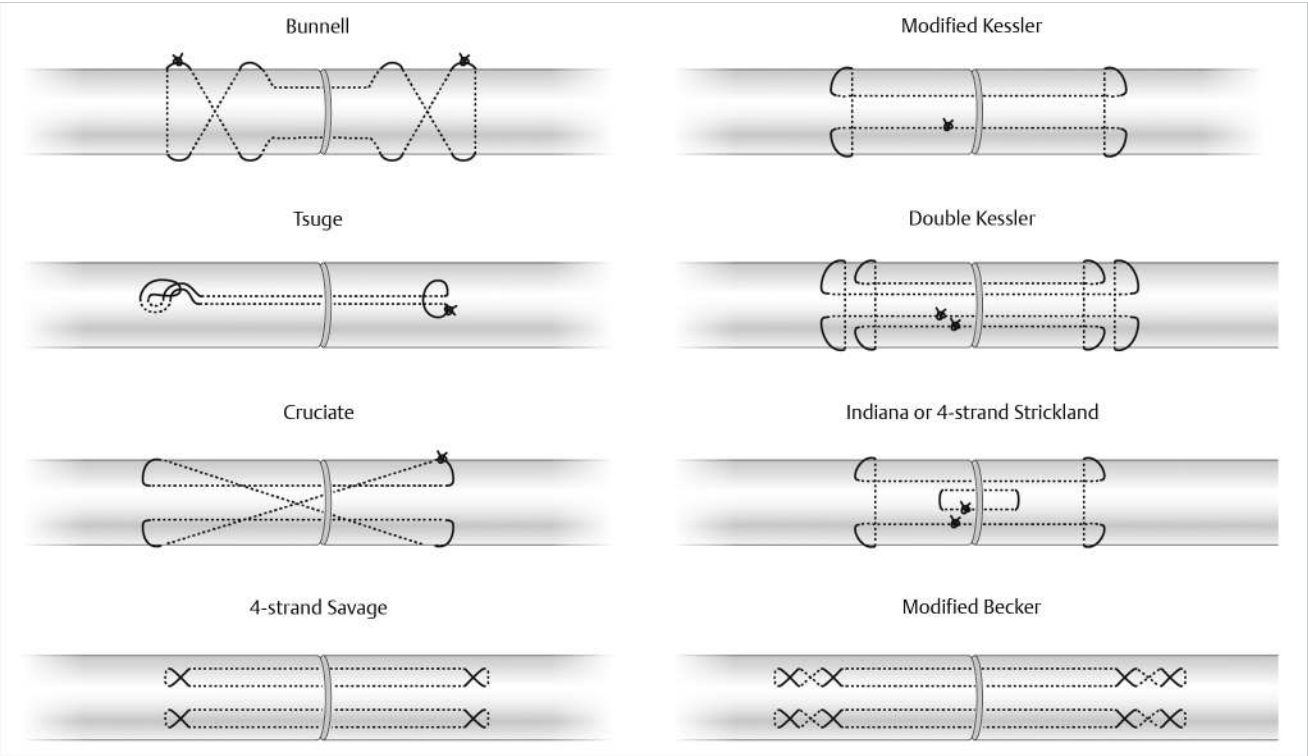


Fig. 26.9 Common flexor tendon repair techniques.

Principles of Successful Zone 2 Flexor Tendon Repair

- Extensile exposure.
- Adequate lighting and magnification.
- Atraumatic handling of tendons and sheath.
- Use at least a four-core suture technique (with 3–0 or 4–0 suture).
- Avoid bulky repair.
- Running circumferential epitendinous suture to resist gapping and add strength.
- Repair the sheath only if it improves tendon glide.

studies have shown that a four-strand repair (such as the Becker, modified Savage, or Strickland's repair) or greater provides enough early repair strength to permit early motion rehabilitation protocols.[17,18] The author's preference is to use the Strickland's four-core repair with 4–0 polypropylene followed by a 6–0 polypropylene running epitendinous suture.

The FDS tendon is repaired first. Near its insertion, where the tendon divides and flattens, it can be approximated with one figure-of-eight or horizontal mattress suture in each tendon slip, followed by a 6–0 running epitendinous suture. FDS tendon lacerations which are proximal to the insertion and FDP tendons are then repaired (▶ Fig. 26.10). Core sutures should be placed 7 to 10 mm from the tendon ends and in the volar two-thirds of the tendon, in order to prevent injury to the longitudinal vascularity of the tendon. The epitendinous suture can be placed in a simple running or locking fashion and has been shown to augment repair strength by resisting gap formation on cyclic loading (▶ Fig. 26.11).[19,20] Closure of the tendon sheath is not necessary and may constrict the repair site. The sheath may be judiciously opened to allow the tendon repair site to glide freely throughout the range of digital motion. If the tendon sheath is too tight to accommodate repair of both tendons, as is commonly seen in the small finger, one slip of FDS may be excised.

26.4.3 Flexor Tendon Repair—Zones 3 to 5

The principles of flexor tendon repair in zones 3 to 5 are the same as for zone 2. In general, lacerations in these zones have a better prognosis than those in zone 2, due to the lack of a fibro-osseous tunnel and more space to accommodate the repair. Tendon lacerations in zone 4 should be exposed by dividing the transverse carpal ligament. The superficial palmar arch and

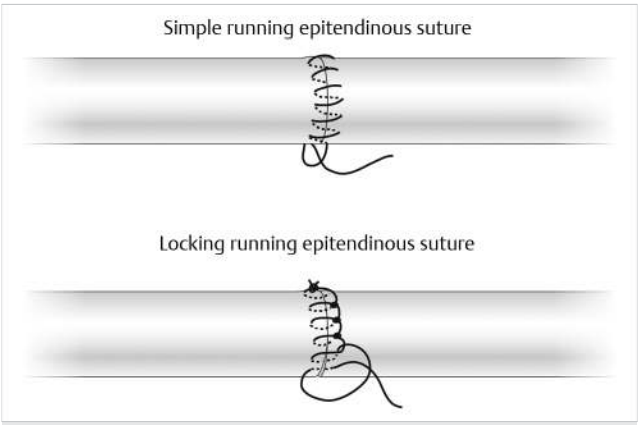


Fig. 26.11 Epitendinous suture techniques.

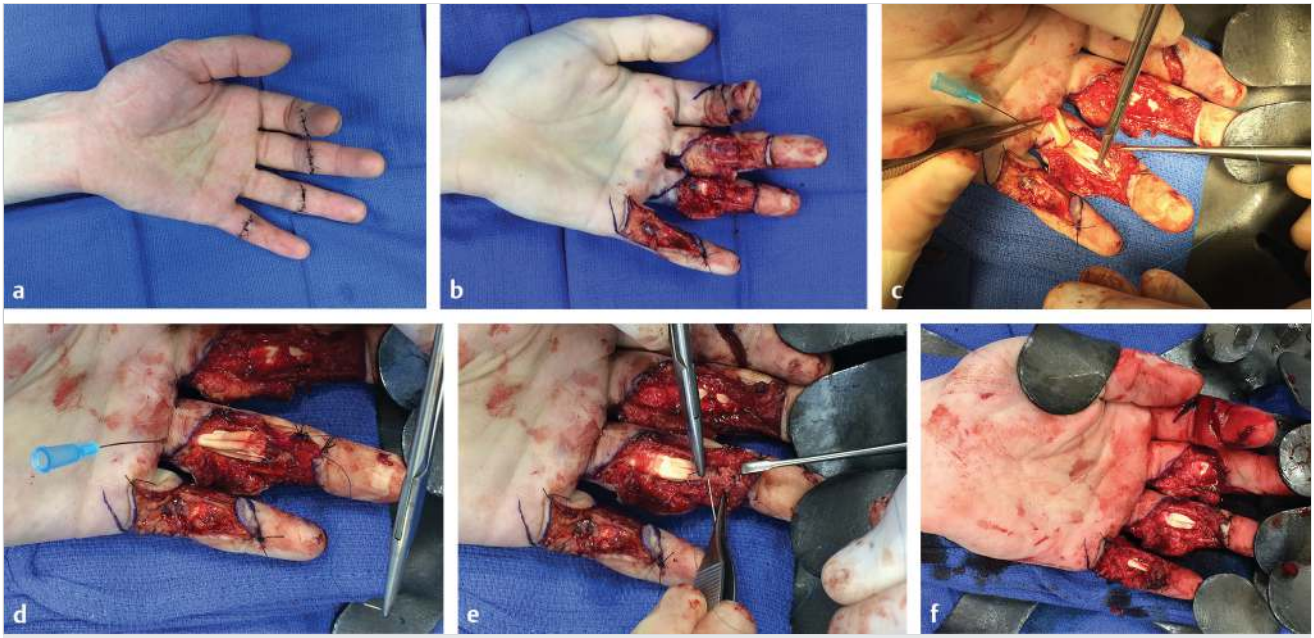


Fig. 26.10 **(a)** An 18-year-old man 5 days after a knife laceration to the left hand. Note the loss of the normal digital cascade. **(b)** Exposure revealing zone 2 lacerations of the FDS and FDP tendons in all three digits. **(c)** Tendons are retrieved and held in place with a 25-gauge hypodermic needle. The FDS tendon is repaired. **(d)** Core sutures are placed in the FDP tendon. **(e)** A running epitendinous suture completes the repair. **(f)** Appearance following completion of all six repairs.

median nerve should be carefully assessed due to a high incidence of concomitant injuries to these structures. Multiple tendon injuries are common in zone 5, due to their close proximity. Matching the proximal and distal cut ends can be challenging and is assisted by clues such as the size, shape, and fiber configuration of the tendon, as well as the geometry of the laceration (transverse, oblique, stepwise, etc.). Lacerations close to the musculotendinous junction result in retraction of the proximal tendon into the muscle belly, but with gentle dissection into the muscle, they can be readily identified and retrieved.

26.4.4 Extensor Tendon Repair—Zones 1 and 2

Sharp lacerations of the terminal extensor tendon can be repaired with direct suture if there is sufficient distal tendon substance. The laceration is extended proximally and distally and the tendon is repaired with one or two 4–0 nylon or polypropylene sutures in a figure-of-eight fashion. Alternatively, a primary tenodermodesis may be performed, where the skin and tendon are repaired with one running layer of a 4–0 monofilament suture. A Kirschner wire can be placed to hold the DIP in extension and protect the repair (▶ Fig. 26.12). Lacerations close to the terminal insertion are best treated by reinserting the terminal tendon to the bone, either via a drill hole or suture anchor. Care must be taken to protect the germinal nail matrix, which lies just distal to the terminal extensor insertion.

The terminal tendon is frequently disrupted in a closed hyperflexion injury (mallet finger), which can involve the tendon alone or may include an avulsion fracture from the distal phalanx. Pure avulsions and avulsions involving a small bone fragment are best treated with closed extension splinting for 6 to 8 weeks, followed by passive range-of-motion and blocking exercises. When nonoperative management fails, a secondary tenodermodesis procedure may be effective.[21] Avulsions with larger bone fragments, involving more than 30% of the articular surface, are often associated with volar subluxation of the DIP joint, and require operative fixation. This can be accomplished by passing a stainless steel wire through the terminal tendon, bone fragment, and distal phalanx to the volar pad of the finger, where it is secured over a button. Alternatively, large avulsion fragments can be fixated with Kirschner wires or interfragmentary lag screws.

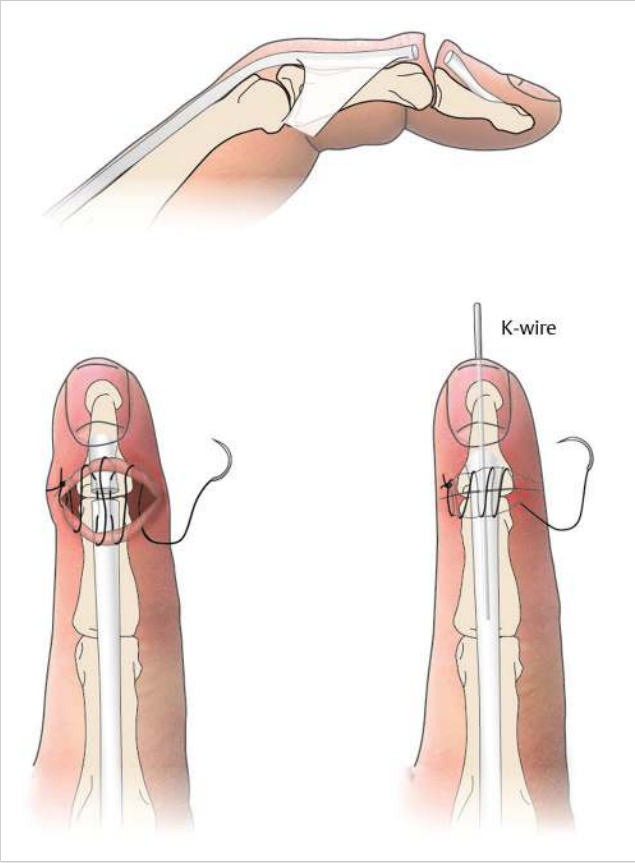


Fig. 26.12 Repair of zone 1 extensor tendon injury by tenodermodesis.

26.4.5 Extensor Tendon Repair—Zones 3 and 4

Open tendon lacerations in zones 3 and 4 can be repaired with 4–0 monofilament sutures placed in a figure-of-eight or modified Kessler fashion, followed by a running superficial "cross-stitch" of 6–0 polypropylene (▶ Fig. 26.13). Care should be taken to bury the knots, as the soft-tissue coverage in this area is limited and sutures may become palpable or extrude. The central slip and lateral bands should be repaired separately. Due to the limited excursion of extensor tendons, the cut ends of the tendon must not be excessively trimmed or overlapped during repair. As little as 2 mm of shortening or overlap could lead to a marked PIP flexion lag. Sharp lacerations of the central slip at the insertion site at the base of the middle phalanx should be managed by securing the tendon to the bone via a suture passed through a transverse osseous tunnel or with a suture anchor. Closed avulsion injuries of the central slip can be managed by splinting the PIP joint in extension for 6 weeks.

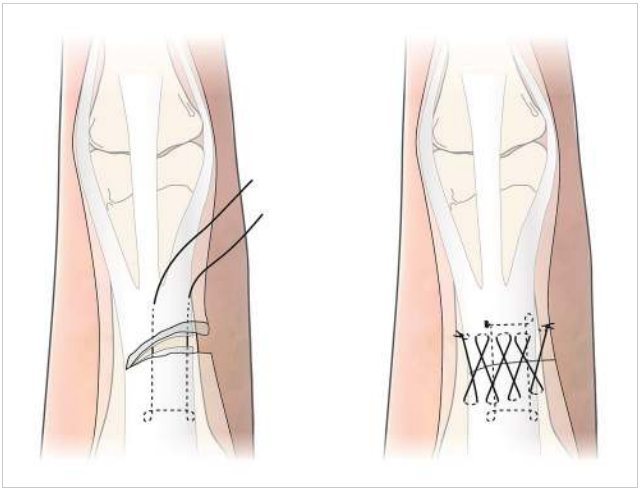


Fig. 26.13 Zone IV partial extensor laceration repair.

26.4.6 Extensor Tendon Repair—Zones 5 to 8

For zones 5 and 6 injuries, skin lacerations should be extended proximally and distally in a curvilinear fashion to allow access to the cut tendon ends. These are gently grasped by the edges with forceps, and the epitenon is trimmed away with fine scissors. Minimal, if any, shortening should be performed. The tendons are repaired with 4–0 monofilament sutures placed in a figure-of-eight or modified Kessler fashion, followed by a running cross-stitch of 6–0 polypropylene for additional strength and to reduce resistance of gliding.

For zone 7 injuries, it may be necessary to elevate the extensor retinaculum to access the tendons for repair. The retinaculum can be incised in a stepwise fashion, incorporating a modified Z-plasty into the retinacular repair. Tendons in zones 7 and 8 are rounder in cross-section than in the digits, and can accommodate core sutures. They should be repaired with 4–0 monofilament sutures placed in a modified Kessler or figure-of-eight fashion. Zone 8 injuries are usually multiple and identification of the tendons can be challenging. To avoid the repetitive identification of tendon ends, a 4–0 monofilament suture can be placed through each cut tendon end as it is identified and held by a labeled hemostat. After all tendons have been labeled, they can be easily matched up and repaired.[22]

26.5 Postoperative Care

The tourniquet is lowered and hemostasis is obtained. The skin is closed with 4–0 nylon sutures, and a nonadherent dressing is applied. A small piece of a saline-moistened and wrung-out gauze sponge can be applied directly over the suture line. It will draw any blood away from the wound and prevent the dressing from sticking painfully to the sutures when removed, without causing the skin maceration associated with petroleum-impregnated dressings.

26.5.1 Flexor Tendon Repair

Postoperative splints are designed with attention to protecting vulnerable structures while allowing uninjured parts to move. Following flexor tendon repair, a well-padded, forearm-based, dorsal-blocking splint is applied with the wrist in 10 to 20 degrees of flexion, the MP joints at 60 to 70 degrees of flexion, and the IP joints in slight flexion. For any flexor tendon repair in the index, long, ring, or small fingers, all four fingers should be included to counteract the effect of the common FDP muscle belly. For repairs of the flexor pollicis longus tendon, a thumb spica splint can be fashioned with the other digits free.

The importance of postoperative rehabilitation in flexor tendon repair cannot be overstated. Immediately following surgery, the repair strength is due to the sutures alone. The strength decreases during the inflammatory phase of healing in the first postoperative week, and then begins to slowly increase. During this period, repairs cannot tolerate unprotected motion. However, early postrepair motion stress has been shown to improve tensile strength, limit adhesion formation, and improve tendon excursion.[23,24] Thus, numerous rehabilitation strategies have been devised to allow motion stress while protecting the repair (▶ Fig. 26.14). Several randomized controlled studies have compared these strategies, without clearly identifying one as superior.[25,26]

The Strickland modification of the passive motion regime of Duran and Houser is a controlled passive finger flexion protocol, which is simpler and less patient dependent than methods involving rubber band traction. On postoperative day 3, the operative splint is removed and an orthoplast dorsal blocking splint is applied. The splint is worn at all times, and the passive motion regime is initiated, which consists of 15 repetitions of passive flexion and extension of the PIP, then the DIP, and then the entire digit. The patient performs these exercises every 2 hours, during the waking hours of the day. At 3 weeks postoperative, the dorsal blocking splint is replaced with a volar wrist cock-up splint, and the wrist is progressively extended to neutral. Place and hold exercises are initiated. At 5 to 6 weeks, active flexion in the splint is allowed. At 8 weeks, active flexion out of the splint is allowed and strengthening is begun. Full activity is resumed at 12 weeks.

During the postoperative period, the patient should be seen frequently by the surgeon and there must be open lines of communication among surgeon, patient, and therapist. The protocol can be adjusted as needed to accommodate differential rates of healing and patient compliance.

26.5.2 Extensor Tendon Repair

For extensor tendon repairs in zones 1 and 2, a DIP extension splint is placed at the time of surgery which is worn

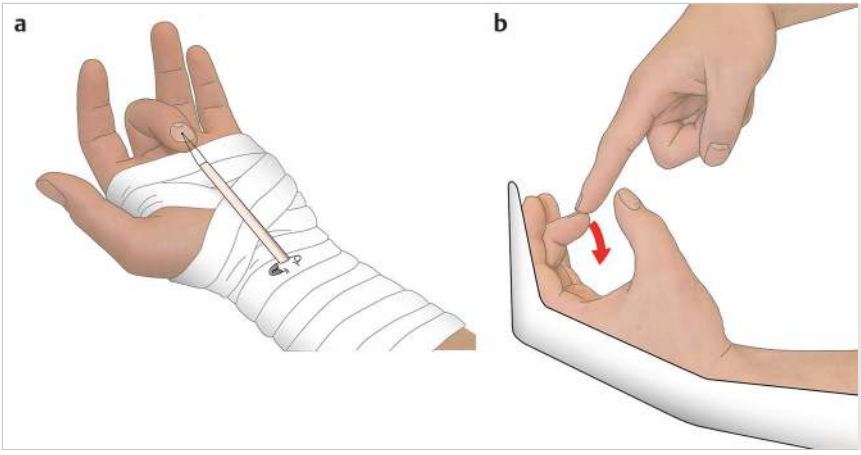


Fig. 26.14 Methods of early mobility following flexor tendon repair. **(a)** Kleinert's method. **(b)** Duran and Houser's passive motion method.

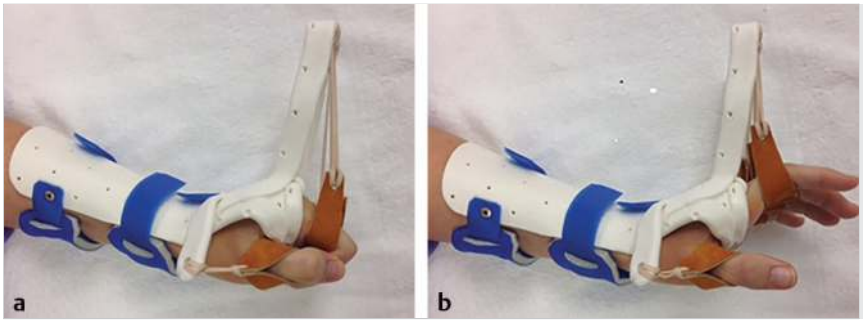


Fig. 26.15 Dynamic splint for rehabilitation after extensor tendon repair. **(a)** Active flexion against resistance. **(b)** Passive extension.

continuously for 6 weeks, following which it is worn at night for an additional 2 to 4 weeks. At 6 weeks postoperative, active range-of-motion exercises are begun, progressing to full use at 12 weeks. For zones 3 and 4 repairs, the PIP is splinted in full extension for 5 to 6 weeks. If the lateral bands were repaired, the DIP should also be splinted; otherwise, it is allowed to move. At 6 weeks, active range-of-motion exercises and night-only splinting are initiated, progressing to resisted exercises at 8 weeks. For zones 5 and 6 repairs, a forearm-based splint is applied at surgery with the wrist in 30 degrees of extension, the MP joints in 20 degrees of flexion, and the IP joints straight. Early active motion is preferable, unless there is a fracture or complex injury which prevents it. Passive range of motion is begun at 1 week, progressing to active motion at 4 weeks, and resisted extension exercises at 8 weeks.

An effective alternative to static splinting and early range of motion involves dynamic splinting (▶ Fig. 26.15). Beginning at days 3 to 5 after surgery, a dorsal outrigger splint is applied for daytime use which maintains the affected digit or digits in extension with rubber band traction. Excursion of the repaired tendon is achieved by active flexion and passive extension, which is performed 15 times per hour. A resting splint is worn at night. The MP flexion is gradually increased over the first 6 weeks, after which the splint is discontinued and resisted extension exercises and grip strengthening is begun. Dynamic extension has been shown to be effective for extensor tendon repairs in zones 3 through 7, but the technique requires a motivated and compliant patient.[27,28,29]

26.6 Outcomes

Common complications following tendon repair include adhesion formation, tendon rupture, and joint stiffness. The incidence of tendon rupture following zone 2 flexor tendon repair ranges from 3 to 9%.[8,30] Rupture occurs most commonly in the first or second weeks following repair, when the repair strength is low and the friction coefficient within the tendon sheath is high due to edema. The rupture commonly occurs during a therapy session and may be accompanied by a "popping" sensation or sound. Prompt recognition and repair result in the best outcomes and avoid the need for delayed tendon reconstruction. Other complications include the "quadrigia effect" from excessive shortening of the FDP tendon, or reduced range of motion from tendon adhesions. When the loss of motion is due to adhesion formation, the measured passive range of motion will exceed the active range of motion. A patient who is at least 3 months after tendon repair and whose progress has stalled despite adequate range-of-motion therapy may be a candidate for operative tenolysis. However, therapy must be undertaken immediately following surgery if the tenolysis is to be effective.[31] Numerous mechanical and chemical methods of reducing adhesions have been devised, but none of them has found clinical utility.[32]

The most common method for assessing postoperative outcomes in flexor tendon repair is the Strickland method which adds the range of motion of the DIP and PIP joints. Motion of more than 85 degrees is considered an "excellent" result.[33] The American Society for Surgery of the Hand endorses the total active motion (TAM) method, by which the extension deficits in the MP, PIP, and DIP joints are added and then subtracted from the total flexion. The outcomes of flexor tendon repair vary greatly and are affected by age, extent of injury, timing of repair, and especially, compliance with the postoperative rehabilitation regime. When early mobilization is performed in a motivated patient, return to 90% of normal grip strength, pinch, and range of motion can be achieved.[34]

Results following extensor tendon repair are largely dependent on associated injuries with worse results expected where there are concomitant fractures. A retrospective study of 62 patients with 101 extensor tendon injuries, treated with standard repair and static splinting, found that patients without associated injuries achieved 64% good or excellent results and a mean TAM of 212 degrees. In this study, loss of flexion was a more common adverse outcome following extensor tendon repair than loss of extension.[35] In general, repairs in distal zones (I–IV) yield poorer results than in proximal zones (V–VIII), largely due to the intimate relationship of the tendons to the phalanges in the distal zones.

Tendon injuries pose a formidable challenge to the reconstructive surgeon, but when principles of repair and rehabilitation are followed which are in harmony with the underlying physiology of tendon healing, good restoration of function can be achieved.

26.7 Review Questions

26.7.1 True or False

1. Tendon injuries are always associated with open lacerations.
2. The FDP tendons to the index and long fingers share a common muscle belly.
3. For extensor tendon injuries, odd-numbered zones correspond to injuries occurring over joints.

26.7.2 Choose the Best Answer

4. A running epitendinous suture reduces the risk of rupture following tendon repair by
   a) Adding to the repair strength.
   b) Resisting gap formation.
   c) Reducing the coefficient of friction within the tendon sheath.
   d) All of the above.
5. Excessive shortening of a flexor tendon can cause a loss of function due to
   a) A lumbrical plus deformity.
   b) The quadrigia effect.
   c) Intrinsic tightness.
   d) Boutonniere deformity.

26.7.3 Answers

1. False.
2. False.
3. True.
4. d. All of the above.
5. b. The quadrigia effect.

References

[1] Kleinert HE, Kutz JE, Atasoy E, Stormo A. Primary repair of flexor tendons. Orthop Clin North Am. 4(4):865–876
[2] Verdan CE. Half a century of flexor-tendon surgery. Current status and changing philosophies. J Bone Joint Surg Am. 54(3):472–491
[3] Lin GT, Amadio PC, An KN, Cooney WP. Functional anatomy of the human digital flexor pulley system. J Hand Surg Am. 14(6):949–956
[4] Gonzalez MH, Whittum J, Kogan M, Weinzweig N. Variations of the flexor digitorum superficialis tendon of the little finger. J Hand Surg [Br]. 22(2):277–280
[5] Tuncali D, Yavuz N, Terzioglu A, Aslan G. The rate of upper-extremity deep-structure injuries through small penetrating lacerations. Ann Plast Surg. 55 (2):146–148
[6] Lee DH, Robbin ML, Galliott R, Graveman VA. Ultrasound evaluation of flexor tendon lacerations. J Hand Surg Am. 25(2):236–241
[7] Wu TS, Roque PJ, Green J, et al. Bedside ultrasound evaluation of tendon injuries. Am J Emerg Med. 30(8):1617–1621
[8] Elliot D, Giesen T. Avoidance of unfavourable results following primary flexor tendon surgery. Indian J Plast Surg. 46(2):312–324
[9] Griffin M, Hindocha S, Jordan D, Saleh M, Khan W. An overview of the management of flexor tendon injuries. Open Orthop J. 6:28–35
[10] Tang JB. Tendon injuries across the world: treatment. Injury. 37(11):1036–1042
[11] Lutsky KF, Giang EL, Matzon JL. Flexor tendon injury, repair and rehabilitation. Orthop Clin North Am. 46(1):67–76
[12] Lalonde DH. Wide-awake flexor tendon repair. Plast Reconstr Surg. 123(2): 623–625
[13] Lalonde DH, Martin AL. Wide-awake flexor tendon repair and early tendon mobilization in zones 1 and 2. Hand Clin. 29(2):207–213
[14] Bruner JM. The zig-zag volar-digital incision for flexor-tendon surgery. Plast Reconstr Surg. 40(6):571–574
[15] Hall RF , Jr, Vliegenthart DH. A modified midlateral incision for volar approach to the digit. J Hand Surg [Br]. 11(2):195–197
[16] Chu JY, Chen T, Awad HA, Elfar J, Hammert WC. Comparison of an all-inside suture technique with traditional pull-out suture and suture anchor repair techniques for flexor digitorum profundus attachment to bone. J Hand Surg Am. 38(6):1084–1090
[17] Boyer MI, Gelberman RH, Burns ME, Dinopoulos H, Hofem R, Silva MJ. Intrasynovial flexor tendon repair. An experimental study comparing low and high levels of in vivo force during rehabilitation in canines. J Bone Joint Surg Am. 83-A(6):891–899
[18] Silva MJ, Hollstien SB, Fayazi AH, Adler P, Gelberman RH, Boyer MI. The effects of multiple-strand suture techniques on the tensile properties of repair of the flexor digitorum profundus tendon to bone. J Bone Joint Surg Am. 80(10): 1507–1514
[19] Wade PJ, Wetherell RG, Amis AA. Flexor tendon repair: significant gain in strength from the Halsted peripheral suture technique. J Hand Surg [Br]. 14 (2):232–235
[20] Kubota H, Aoki M, Pruitt DL, Manske PR. Mechanical properties of various circumferential tendon suture techniques. J Hand Surg [Br]. 21(4):474–480
[21] Sorene ED, Goodwin DR. Tenodermodesis for established mallet finger deformity. Scand J Plast Reconstr Surg Hand Surg. 38(1):43–45
[22] Botte MJ, Cohen MS, von Schroeder HP. Method of tendon labeling in forearm injuries. J Hand Surg Am. 16(4):763–764
[23] Gelberman RH, Botte MJ, Spiegelman JJ, Akeson WH. The excursion and deformation of repaired flexor tendons treated with protected early motion. J Hand Surg Am. 11(1):106–110
[24] Gelberman RH, Nunley JA , II, Osterman AL, Breen TF, Dimick MP, Woo SL. Influences of the protected passive mobilization interval on flexor tendon healing. A prospective randomized clinical study. Clin Orthop Relat Res.(264): 189–196
[25] Adolfsson L, Söderberg G, Larsson M, Karlander LE. The effects of a shortened postoperative mobilization programme after flexor tendon repair in zone 2. J Hand Surg [Br]. 21(1):67–71
[26] Kitis PT, Buker N, Kara IG. Comparison of two methods of controlled mobilisation of repaired flexor tendons in zone 2. Scand J Plast Reconstr Surg Hand Surg. 43(3):160–165
[27] Browne EZ , Jr, Ribik CA. Early dynamic splinting for extensor tendon injuries. J Hand Surg Am. 14(1):72–76
[28] Evans RB. Therapeutic management of extensor tendon injuries. Hand Clin. 2 (1):157–169
[29] Saldana MJ, Choban S, Westerbeck P, Schacherer TG. Results of acute zone III extensor tendon injuries treated with dynamic extension splinting. J Hand Surg Am. 16(6):1145–1150
[30] Elliot D, Moiemen NS, Flemming AF, Harris SB, Foster AJ. The rupture rate of acute flexor tendon repairs mobilized by the controlled active motion regimen. J Hand Surg [Br]. 19(5):607–612
[31] Strickland JW. Results of flexor tendon surgery in zone II. Hand Clin. 1(1): 167–179
[32] Ferguson RE, Rinker B. The use of a hydrogel sealant on flexor tendon repairs to prevent adhesion formation. Ann Plast Surg. 56(1):54–58
[33] Strickland JW, Glogovac SV. Digital function following flexor tendon repair in Zone II: a comparison of immobilization and controlled passive motion techniques. J Hand Surg Am. 5(6):537–543
[34] Yen CH, Chan WL, Wong JW, Mak KH. Clinical results of early active mobilisation after flexor tendon repair. Hand Surg. 13(1):45–50
[35] Newport ML, Blair WF, Steyers CM , Jr. Long-term results of extensor tendon repair. J Hand Surg Am. 15(6):961–966

27 Nerve Compression Syndromes of the Upper Extremity

Loretta Coady-Fariborzian and Winston Richards

Abstract
This chapter addresses the diagnosis and anatomy of the different kinds of nerve compression that cause numbness, tingling, pain, or weakness primarily in the hand. Presurgical considerations are detailed, and surgical options, including open and endoscopic approaches, are discussed for each type of compression (e.g., carpal tunnel, median nerve, cubital tunnel, ulnar tunnel). Postoperative guidelines are provided, and there is an extended discussion of possible outcomes.

Keywords: Tinel's sign, Phalen's test, Durkan's test, anterior interosseous syndrome, carpal tunnel syndrome, radial tunnel syndrome, Froment's sign, Wartenberg's sign

27.1 Goals and Objectives

- Recognize the presentation of patients with upper extremity nerve compression syndromes.
- Understand the anatomy of the different kinds of nerve compression.
- Verify the location of the compression through physical exam and diagnostic tests.
- Know when to offer treatment for a diagnosis of nerve compression.
- Recognize postoperative complications and recurrence of nerve compression.

27.2 Patient Presentation

Patients with nerve compression will usually present to their primary care provider with weeks to months of hand "numbness," "tingling," "pain," or "weakness." Upon further questioning, the symptoms may wake them up at night or be present in the early morning hours upon awakening. The patients may relate the symptoms to certain activities that place tension or compression on the peripheral nerves such as driving, writing, keyboarding, shaving, hairstyling, or holding the phone to his ear. The symptoms may have developed with a change in medical condition such as pregnancy, hypothyroidism, or synovial proliferation, or with an increase or change in work or hobbies. More often than not, however, the condition is idiopathic; there is no clear etiology for the symptoms. The two most common forms of nerve compression are compression of the median nerve at the wrist (carpal tunnel syndrome) and compression of the ulnar nerve at the elbow (cubital tunnel syndrome). The examiner does need to be suspicious of the less common nerve compression syndromes involving the median nerve in the forearm (anterior interosseous and pronator syndrome), ulnar nerve in the wrist (ulnar tunnel), and radial nerve in the forearm (posterior interosseous syndrome, radial tunnel syndrome, and Wartenberg's syndrome).

Physical exam consists of a visual inspection of the hand. Muscle wasting of the interosseous muscles indicates long-standing compression of the ulnar nerve. Wasting of the thenar muscles indicates severe medial nerve compression. Observe how the patient holds his digits; a claw-like (Duchenne's sign) or flattened ("ape hand") appearance indicates severe or prolonged nerve injury. It is more common for patients to present at earlier stages of compression. The examiner will look for signs of nerve "irritability" by tapping on the point of compression and eliciting the "Tinel's" sign. This is found by tapping at the suspected point of compression, which is the distal wrist crease for carpal tunnel or the inside of the elbow for the ulnar nerve. Be aware that there is no standardization and considerable inter-examiner variability exists with this test.[1] The examiner may be able to elicit a positive "Phalen's" test to the carpal tunnel by having the patient flex the wrists and increasing the pressure in the carpal tunnel. Digital pressure over the median nerve at the wrist is known as "Durkan's" test and will reproduce the symptoms of carpal tunnel. The symptoms of cubital tunnel can be reproduced by having the patient fully flex the elbows while the examiner places digital pressure on the nerve.

The patient is asked to abduct and adduct his or her fingers to the examiner's resistance and to cross and uncross fingers. Patients with significant ulnar nerve compression may have difficulty with this exercise. The patient is asked to abduct his or her thumb over and across the palm against resistance. Patients with median nerve compression and thenar muscle involvement may have difficulty with this maneuver.

Anterior interosseous syndrome is a less common but well-described median nerve compression in the forearm. It presents as pain in the forearm and weakness of the innervated muscles (flexor digitorum profundus to the index and middle fingers, pronator quadratus, and flexor pollicis longus) without a sensory deficit. It can be identified on physical exam with a positive Tine's sign over the fibrous bands in the volar forearm. The patient may fail the "pinch test" and not be able to make an "OK" sign with his thumb and index finger due to weakness in the proximally innervated muscles: flexor pollicis longus (index) and flexor digitorum profundus (▶ Fig. 27.1). Anterior interosseous nerve (AIN) syndrome can be confused with pronator syndrome, which presents as forearm pain and sensory disturbance in the median nerve distribution. Provocative tests for pronator syndrome include resisted elbow flexion, resisted middle finger flexion, and resisted forearm pronation.

Ulnar tunnel syndrome is a compression of the ulnar nerve at the wrist. The ulnar tunnel, also known as Guyon's canal, is divided into three zones and contains both the ulnar nerve and the radially located ulnar artery (▶ Fig. 27.2). Zone 1 is the most proximal and the patients will have sensory and motor symptoms. Zone 2 is radial and involves the motor branch of the ulnar nerve. Zone 3 is ulnar and the symptoms will be sensory. Guyon's canal is bordered by the transverse carpal ligament (TCL) below and the volar carpal ligament and pisohamate ligament above. The most common reason for ulnar tunnel syndrome is a ganglion cyst (▶ Fig. 27.3).[2] An ulnar artery aneurysm or hook of the hamate fracture can also cause compression in this tight space. Ulnar tunnel syndrome can be distinguished from cubital tunnel syndrome by retained sensation over the dorsal/ulnar hand due to the takeoff of the sensory branch of the ulnar nerve proximal to the ulnar tunnel.

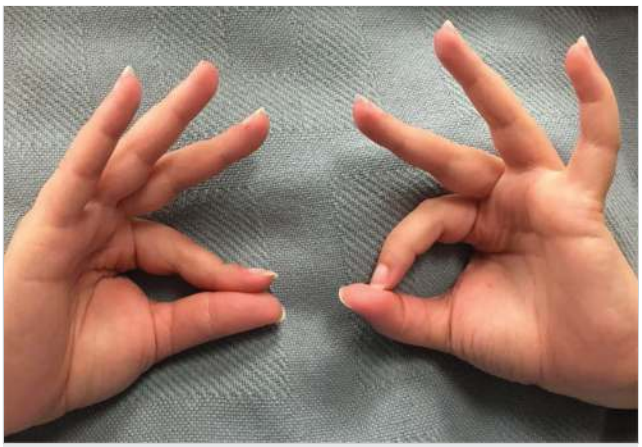


Fig. 27.1 Pinch "OK" test.

Fig. 27.3 Ganglion cyst in Guyon's canal.

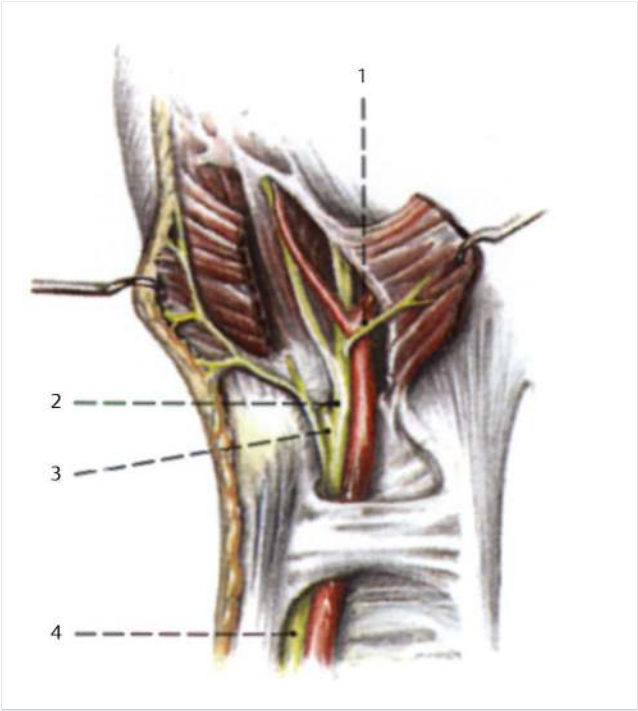


Fig. 27.2 Schematic diagram of Guyon's canal. (1) Muscular branch (palmaris brevis). (2) Superficial branch of the ulnar nerve. (3) Deep branch of the ulnar nerve. (4) Ulnar nerve. (Used with permission from Pechlaner S, Kerschbaumer F. Atlas of Hand Surgery. New York, NY: Thieme Medical Publishers; 2000)

Additional physical exam findings can be noted with ulnar nerve compression. Patients will hold an object, such as a piece of paper, in the first web space by flexing the IP joint using the unaffected flexor pollicis longus instead of using the weakened adductor pollicis. This is known as "Froment's sign" (▶ Fig. 27.4). The patients may not be able to adduct the small finger with the weakened palmar interosseous muscle due to the unopposed extensor digit quinti, innervated by the radial nerve. This is known as the "Wartenberg's sign" (▶ Fig. 27.5).

Radial nerve compression can be found in the distal and proximal forearm. Distally, it is known as "Wartenberg's syndrome." Wartenberg's syndrome is a compression of the superficial sensory radial nerve between the extensor carpi radialis and the brachioradialis. Patients will complain of pain, numbness, and paresthesias over the distribution of the radial sensory nerve. Blunt trauma over the radial/dorsal forearm or tight compression, such as a watch or handcuffs, can provoke this syndrome due to the superficial location of the nerve. Patients will avoid direct contact with clothing sleeves, gloves, or jewelry at the wrist. Symptoms can be provoked with flexion, pronation, and ulnar deviation of the wrist. This can be confused with de Quervain's stenosing tenosynovitis, which can be present simultaneously; however, a distinguishing feature is that Wartenberg's syndrome is present at rest.[3]

Proximal radial nerve compression is known as "posterior interosseous syndrome" or posterior interosseous nerve (PIN). The patients present with complaints of pain in the forearm and weakness of the radial nerve innervated muscles. The patient will have wrist and metacarpophalangeal joint extension weakness. PIN palsy will need to be distinguished from extensor tendon rupture or tendon subluxation by examining for extensor tenodesis and the ability to hold digits already placed in extension. Radial tunnel syndrome is distinguished from PIN with a presentation of pain without motor or sensory disturbances. Symptoms can be provoked with resisted wrist supination or when traction is placed on the nerve by extending the elbow, pronating the arm, and flexing the wrist. Tenderness is found along the mobile wad.

Office-based equipment can help establish a baseline of the patient's weakness and function. The two-point discriminator is a small pocket device used to examine the patient's sensory loss. Normal two-point is 5 to 6 mm.[4] The patient's grip and pinch are measured and compared with the other side using dynamometers. Traditionally, the dominant hand was believed to have a 10% greater grip; however, the difference may be minimal if the patient is left-handed.[5]

Once the history and physical exam is completed, the patient will be asked to continue with diagnostic studies to verify the location of the suspected nerve compression. Nerve conduction

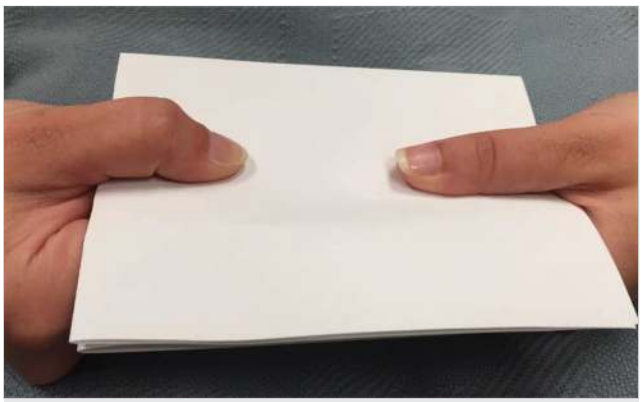


Fig. 27.4 Froment's sign.

Fig. 27.5 Wartenberg's sign.

Fig. 27.6 Depigmentation at wrist after steroid injections.

studies (NCSs) and electromyography (EMG) are performed by a neurologist or physiatrist. NCS allows the examiner to accurately localize a lesion or identify a generalized disease process.

The EMG uses a needle to determine the health and function of the muscles innervated by the nerve in question. Generally, "mild" findings indicate prolonged sensory latencies with normal motor studies; "moderate" findings indicate abnormal sensory latencies and prolongation of median motor distal latencies; and "severe" findings indicate evidence of axonal loss.[6] These studies are helpful by not only verifying the location of the compression but ruling out other more serious, central problems that a plastic surgeon may not recognize such as amyotrophic lateral sclerosis, radiculopathy, or myasthenia gravis.[7] These studies can determine the severity of compression such as progression to muscle involvement. The EMG also serves as a baseline in the event of a patient deciding to pursue nonoperative treatment or presents postoperatively with complaints of worsening or ongoing symptoms. Due to variability in diagnostic equipment and examiners, we recommend having a relationship with a particular physiatrist or group who consistently performs your patients' studies. Postoperative studies are expected to show electrophysiological improvement.[8,9]

X-rays are easy to obtain and are not mandatory; however, the authors prefer to use them to identify or rule out coexisting pathology such as an occult fracture or osteoarthritis that may be contributory to the compression or become symptomatic during the postoperative period. A computed tomography or magnetic resonance imaging (MRI) may be indicated if a mass-occupying lesion is suspected as the etiology for PIN or AIN syndrome. An MRI is useful to differentiate a ganglion cyst from an aneurysm at the wrist.

27.3 Preparation for Surgery

Patients are offered surgical decompression if they are healthy enough to tolerate surgery, are symptomatic, and have evidence of compression on the diagnostic studies. Surgery is most often performed using local plus intravenous sedation for the distal nerve compressions (carpal tunnel and radial sensory) or a regional block for compressions at the elbow or forearm. If the NCS or EMGs are read as "borderline," "mild," or do not indicate compression, conservative treatment is recommended. Conservative treatment for carpal tunnel involves nighttime splinting to prevent prolonged compression on the nerve from wrist flexion while sleeping. A steroid injection at the distal wrist crease adjacent to the median nerve can control or reduce symptoms. Studies have found that patients who respond well to steroid injections also tend to respond well to surgery.[10] We discuss risks such as skin depigmentation in darkly pigmented patients and elevated blood glucose levels in diabetics up to 5 days postinjection (▶ Fig. 27.6).[11,12] Conservative treatment for cubital tunnel syndrome involves elbow padding during the daytime to prevent inadvertent leaning on the nerve and antecubital

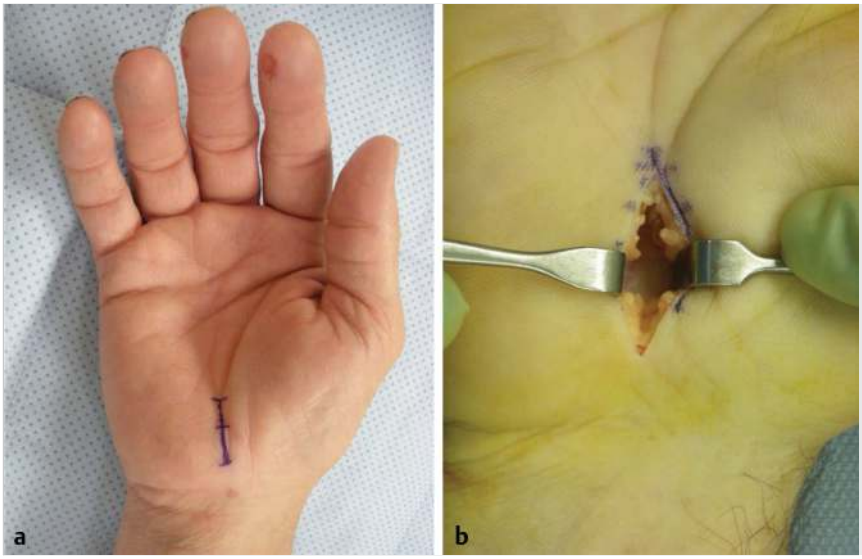


Fig. 27.7 **(a)** Marking for open carpal tunnel release and **(b)** intraoperative view.

padding at nighttime to prevent full elbow flexion that creates prolonged ulnar nerve tension during sleep.

Basic preoperative laboratory tests include a complete blood cell count, coagulations, and a basic metabolic panel. In the event that a patient is on Plavix, Coumadin, or ASA for a cardiac or neurologic reason, the authors will perform indicated hand surgery without stopping anticoagulation. Recent hand literature supports maintaining a patient's anticoagulation regime, if that is in the patient's best interest, with a low risk of complications.[13,14,15] The anesthesiologists often do regional blocks for patients on anticoagulants due to their skill in locating the brachial plexus with ultrasound. We offer in situ release of the cubital tunnel on anticoagulated patients to minimize dissection and bleeding risk. Nerve compression tends to be a chronic, not an acute, problem, and the authors recommend allowing any unresolved medical problems to be addressed first in the best interest of the patient.

Elective hand patients need to have their diabetes and hypertension under control before committing to surgery to avoid postoperative systemic and local complications.[16,17] HbA1c levels can be used to identify patients with poor glycemic control. Values between 6.5 and 8% have been recommended.[18] Antihypertensives are usually continued until the day of surgery. Patients with systolic blood pressure ≥ 180 mm Hg or diastolic blood pressure ≥ 110 should have their pressures lowered gradually over days to weeks as outpatients.[16]

In the event of bilateral compression, one side will be offered, followed by the other side 4 to 6 weeks later. Patients often request bilateral surgery for the sake of time and convenience, but we remind patients that one side has to be free to perform activities of daily living, while the other side can be allowed time to heal while staying clean and dry.

Wartenberg's syndrome, radial tunnel syndrome, and pronator syndrome do not tend to have objective findings on nerve studies. These cases would be best served with conservative treatments of nonsteroidal anti-inflammatory drugs (NSAIDs), activity modification, and splinting for 3 to 6 months. A diagnostic lidocaine injection at the point of compression can help with future surgical planning and expectations.

27.4 Treatment

The surgical treatment of carpal tunnel syndrome is simple decompression of the TCL. This can be done through an open approach (▶ Fig. 27.7) or endoscopic approach (▶ Fig. 27.8). The most popular option is the open release, which requires less specialty training and has a shorter learning curve.[19] The advantage of the endoscopic release is an earlier recovery and return to work. The skin incisions are shallower, allowing for a faster recovery time. Results have been similar over the long term, ranging from 3 months to 1 year.[20,21,22,23]

During open release, synovectomy and manipulation of the nerve are not indicated except for specific cases of synovial hypertrophy that need debulking. A forearm-based tourniquet is well tolerated and is set to 225 mm Hg after manual exsanguination. We recommend a median nerve block using 1% lidocaine and 0.25% Marcaine placed at the distal wrist crease in preoperative holding to allow time for the medication to work and dissipate from the surgical site. The carpal tunnel can be released under direct vision by making a longitudinal incision over and through the TCL from the volar wrist flexion crease, staying in line with the radial border of the fourth ray. Visualization of the palmar fat is the distal end point. A tenotomy scissors can be used to release the distal end of the antebrachial fascia in the forearm, staying ulnar to the palmaris longus tendon. The TCL is about 2 cm in length. The days of large palmar and forearm dissections are long gone and unnecessary, and only contribute to prolonged recovery and scar adhesions.

The endoscopic carpal tunnel release is a minimally invasive alternative to the open release. This is most often performed proximally based, using either a single or double port. An upper arm tourniquet set at 250 mm Hg after manual exsanguination is necessary. A distally based flap of antebrachial fascia is elevated at the distal wrist crease through a transverse incision just above and ulnar to the palmaris longus tendon. A spatula is used to bluntly remove the synovial tissue from the undersurface of the TCL. An endoscopic 4-mm 30-degree camera is placed distally to visualize the TCL. The edge of the ligament is verified with a blunt hook. A sharp hook is used to transect

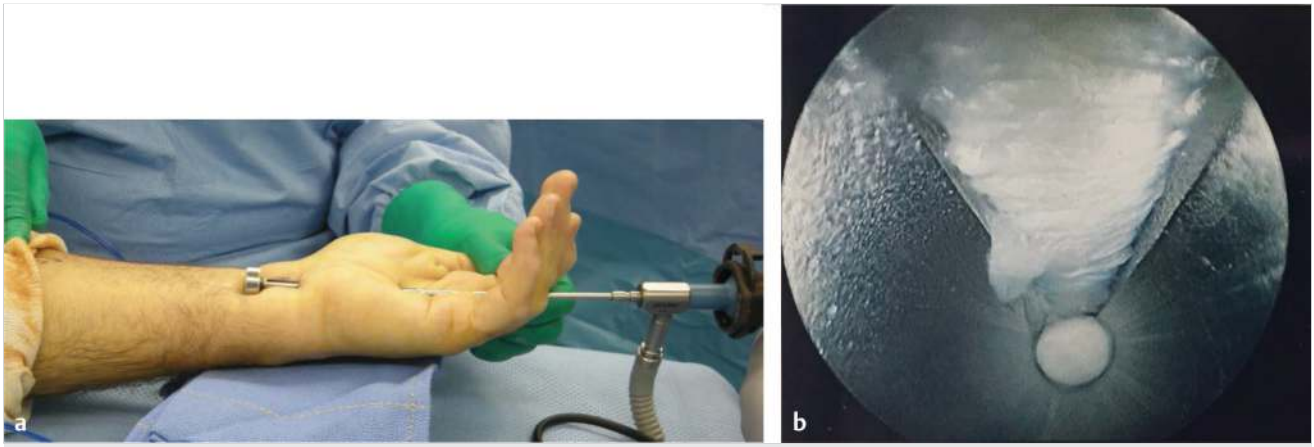


Fig. 27.8 **(a)** Endoscopic carpal tunnel release and **(b)** endoscopic view of transverse carpal ligament.

the ligament under endoscopic vision in a distal to proximal direction.

Postoperatively, the patients are placed in a bulky dressing and encouraged to use their fingers while avoiding lifting or gripping more than 5 lb for 4 to 6 weeks. Open carpal tunnel releases are splinted for a week if they give indication that they are at a higher risk of wound breakdown. Patients are brought back within 10 days for suture removal and one hand therapy visit to assist with mobility and scar management.

Median nerve decompression at the forearm requires an upper extremity tourniquet and regional block. A lazy S incision is made from just proximal to the antebrachial fossa to the midvolar forearm. Points of compression needing to be identified and released or lengthened, from proximal to distal, are ligament of Struthers, bicipital aponeurosis, tendon of the superficial head of the pronator teres, deep head of the pronator teres, and arch from the leading edge of the flexor superficialis (▶ Fig. 27.9). The patient is placed in a bulky dressing restricting full elbow mobility until sutures are removed. Controversy exists over when surgical intervention is appropriate. Most authors recommend surgical decompression after 3 months of conservative treatment or persistent findings on electrodiagnostic studies. There have been reports of spontaneous recovery, but an inflammatory etiology may have been responsible for the AIN palsy.[24]

There are many accepted alternatives for cubital tunnel release. They include open in situ; subcutaneous, intramuscular, transmuscular, and submuscular transposition; medial epicondylectomy; and endoscopic in situ. The points of compression have to be identified and addressed. These include (proximal to distal) arcade of Struthers, intermuscular septum, Osborne's ligament, anconeus muscle (if present), and the flexor pronator fascia (▶ Fig. 27.10). Care needs to be taken to identify and protect large crossing branches of the antebrachial cutaneous nerve, found on average 1.8 cm proximal and 3.1 cm distal to the medial epicondyle as well as the motor branch to the flexor carpi ulnaris (FCU) muscle, located distal to the medial epicondyle.[25] The authors prefer to anteriorly transpose the nerve over the medial epicondyle using the subcutaneous or transmuscular techniques to avoid tension on the nerve during elbow flexion. Dissection of the nerve can temporarily devascularize it, but no correlation has been found between devascularizing the nerve during transposition and postoperative nerve recovery.[26] We offer open in situ release to patients who are less than optimal surgical candidates for the sake of speed and simplicity. Median epicondylectomy is another option that has been found to be successful; however, the authors prefer to avoid the risks of bone tenderness, elbow instability, and heterotopic bone formation.

Postoperatively, we splint patients for 1 week in a padded elbow splint. One to two visits with hand therapy is usually sufficient to recover elbow mobility. Submuscular transposition requires a longer splinting protocol, risking elbow stiffness, which is why it is not our first-line treatment.

Patients with ulnar tunnel syndrome are given a regional block and a forearm tourniquet. The incision is made in a zigzag fashion using the FCU, pisiform, and hook of the hamate as landmarks. The ulnar nerve runs radial to the FCU and between the pisiform and hook of the hamate. The nerve is identified, taking care to identify and protect the ulnar artery. Dissection of the nerve continues until the aponeurotic arch of the hypothenar muscles is released. An anomalous structure, such as a ganglion, fracture fragment, or aneurysm, needs to be identified and removed. The patient is placed in a bulky dressing until return to clinic for suture removal within 10 days.

Spontaneous resolution is typically expected with Wartenberg's syndrome that includes removal of the inciting etiology (compressive force), rest, splinting, and NSAIDs.[3] Surgery is offered after failure of conservative treatment. Decompression of the radial sensory nerve is performed under a proximal forearm tourniquet and can be done under local or regional block with sedation (▶ Fig. 27.11). A longitudinal incision is made over where the radial sensory nerve (RSN) presents between the brachioradialis (BR) and extensor carpi radialis longus tendons, located approximately 9 cm proximal to the radial styloid, staying slightly volar to avoid direct nerve contact. When operating near the RSN, we recommend breaking dermis only with the knife and doing longitudinal blunt dissection. Decompress the fascia between the tendons. Cover with a bulky dressing.

An initial trial of nonsurgical management of PIN and radial tunnel syndrome is recommended. This includes rest, splinting, and anti-inflammatories. An injection of lidocaine and a steroid

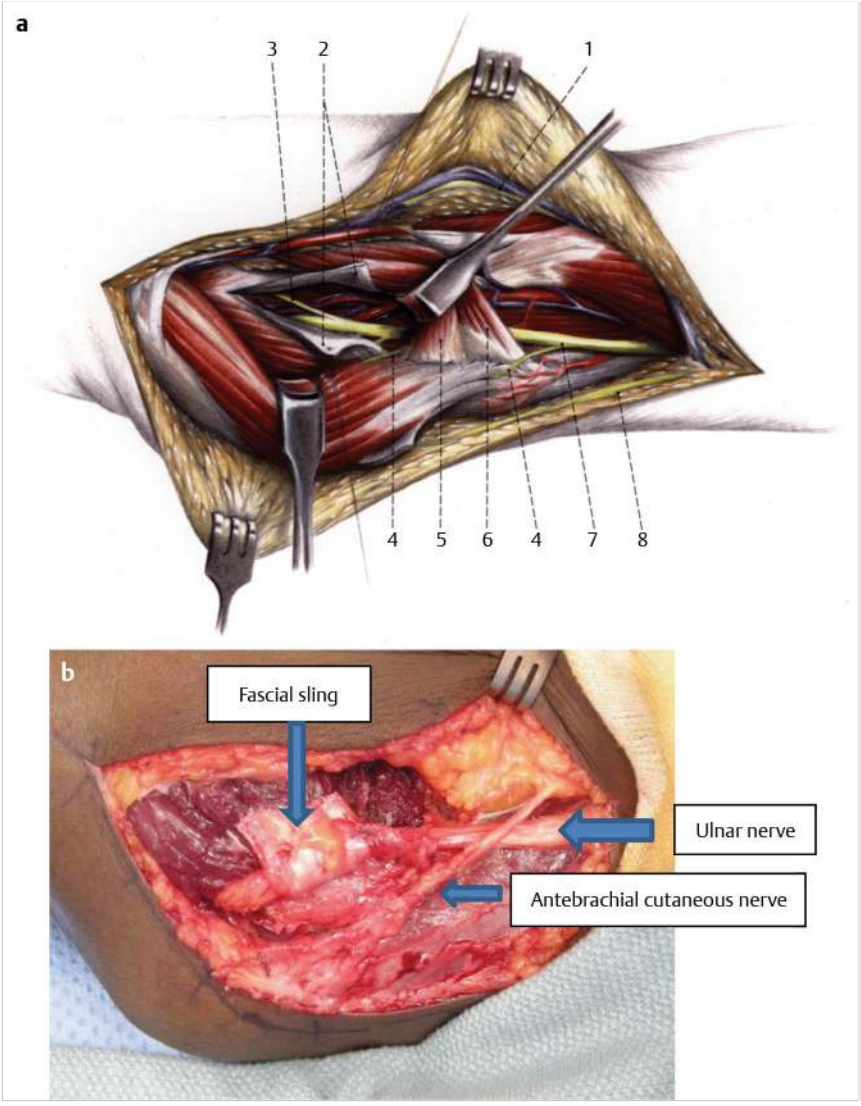


Fig. 27.9 (**a**,**b**) Fascial sling created during cubital tunnel release. ((a) Used with permission from Pechlaner S, Kerschbaumer F. Atlas of Hand Surgery. New York, NY: Thieme Medical Publishers; 2000)

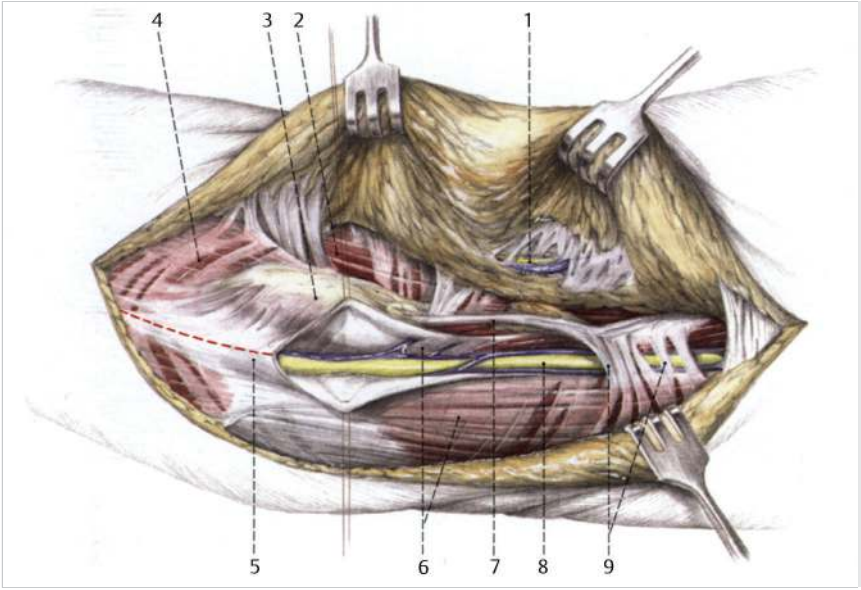


Fig. 27.10 (1) Medial fascicle of the medial antebrachial cutaneous nerve. (2) Brachialis. (3) Medial epicondyle. (4) Common head of the flexors. (5) Canal of the ulnar nerve. (6) Triceps. (7) Medial intermuscular septum. (8) Ulnar nerve. (9) Arcade of Struthers. (Used with permission from Pechlaner S, Kerschbaumer F. Atlas of Hand Surgery. New York, NY: Thieme Medical Publishers; 2000)

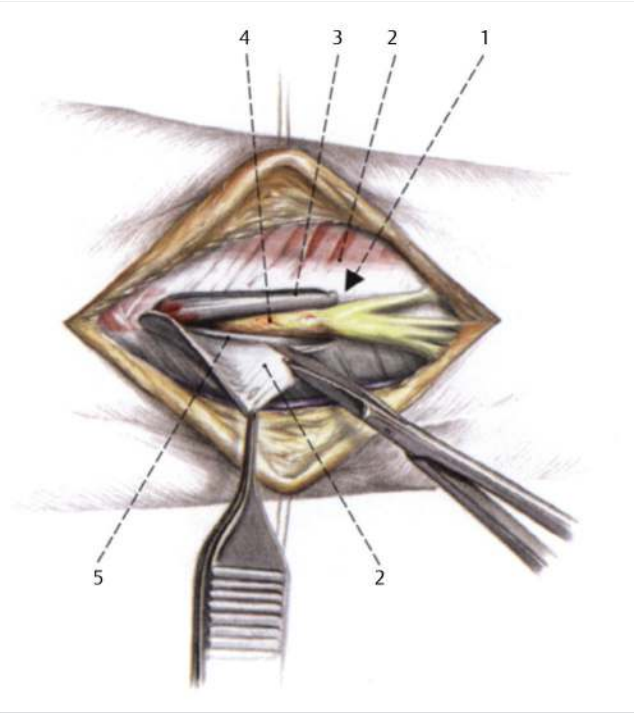


Fig. 27.11 4–44 Decompression of the superficial branch of the radial nerve at its point of entry into the fascia of the forearm between the brachioradialis and extensor carpi radials longus partial resection of the forearm fascia. (1) Point of entry of the superficial branch of the ulnar nerve into the forearm fascia. (2) Forearm fascia (partially resected). (3) Brachioradialis. (4) Superficial branch of the radial nerve. (5) Extensor carpi radials longus. (Used with permission from Pechlaner S, Kerschbaumer F. Atlas of Hand Surgery. New York, NY: Thieme Medical Publishers; 2000)

may be diagnostic and therapeutic for radial tunnel syndrome.[3] If symptoms persist after 3 months, decompression of the PIN is considered. This is performed with an upper extremity tourniquet and under regional block. A longitudinal incision is made over the mobile wad, most easily found when the forearm is pronated on the operating table with the elbow gently flexed. Points of identification and decompression are fibrous tissue anterior to the radiocapitellar joint, "leash of Henry" (recurrent radial vessels), edge of the extensor carpi radialis brevis, arcade of Frohse (proximal edge of the supinator), and distal edge of the supinator (▶ Fig. 27.12). The arm is placed in a bulky dressing. We recommend surgical taping of the dorsal forearm scar for several months in the postoperative period to minimize scar hypertrophy.

All patients with regional blocks are sent home in slings with instructions to remove the sling once sensation and motor function returns. This is to avoid a stiff shoulder.

The use of perioperative antibiotics has not been found to be indicated in elective, clean hand cases.[27] We do give immunocompromised patients, such as diabetics or those on steroids, one perioperative dose of antibiotics before tourniquet inflation. These are clean cases and the antibiotics are not continued in the postoperative period.

27.5 Postoperative Care

In general, we try to minimize immobilization. Patients are treated with a bulky dressing except for the open carpal tunnel patients who are at risk of wound dehiscence from potential falls or pressure onto the wound. Those patients are splinted for a week. Cubital tunnel release patients are placed in an elbow splint, leaving the hand and wrist free, for a week. Patients who are not splinted are advised to remove the dressings on postoperative day 2 and replace with clean, sterile, nonadherent dressing. We allow patients to wash the incision site with mild soap and water at this time. All patients are brought back to

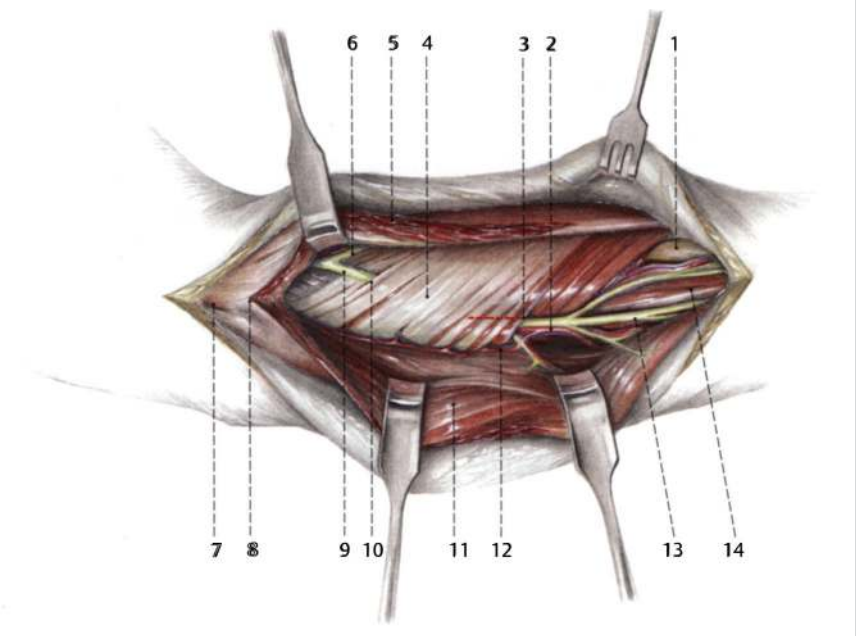


Fig. 27.12 The radial nerve is exposed in the supinator tunnel between the extensor carpi radialis brevis and extensor digitorum. (1) Abductor pollicis longus. (2) Posterior interosseous artery. (3) Point of exit of the deep branch of the radial nerve from the supinator tunnel. (4) Supinator. (5) Extensor carpi radialis brevis. (6) Superficial branch of the radial nerve. (7) Lateral epicondyle. (8) Lateral humeral intermuscular septum. (9) Deep branch of the radial nerve. (10) Arcade of Frohse (point of entry of the radial nerve into the supinator tunnel). (11) Extensor digitorum. (12) Interosseous recurrent artery. (13) Posterior interosseous nerve. (14) Extensor pollicis brevis. (Used with permission from Pechlaner S, Kerschbaumer F. Atlas of Hand Surgery. New York, NY: Thieme Medical Publishers; 2000)

clinic 7 to 10 days postsurgery to have sutures removed and to start hand therapy. Formal hand therapy is usually no more than one to two visits where the patients work on scar mobility, resolution of stiff joints, and gradual strengthening, depending on their work needs. We encourage early mobilization to prevent stiff joints. Patients are allowed to return to light duty within 1 to 2 weeks. We counsel against heavy lifting for the first 6 weeks.

All patients are prescribed anti-inflammatories such as high-dose Motrin or naproxen if there is no contraindication. We try to minimize narcotic prescriptions to prevent inadvertent self-injury due to dizziness and falling and to prompt a quick return to clinic if the patient is having an unusual amount of pain, rather than allowing him to mask the pain with narcotics. We agree with the recommendations of Stanek et al[28] and prefer to minimize the number of unnecessary narcotic pills in circulation that can be misused by better educating the residents and staff. If patients have a history of substance abuse or narcotic dependence, we enlist their primary care physician in the management of their medications prior to surgery.

27.6 Outcomes

Most carpal tunnel release patients are pleased with the surgery, particularly when they are able to sleep through the night without waking up with numb hands. We temper expectations in the elderly, diabetics, and those with long-standing or severe nerve compression; however, positive outcomes have been noted in these patients.[29,30,31] Similar outcomes have been found in both open and endoscopic carpal tunnel release. Advantages of the endoscopic release are a quicker recovery, earlier return to work, and fewer wound complications. The skin incisions are shallower, allowing for a faster recovery time. Results have been similar over the long term, ranging from 3 months to 1 year.[20,21,23,32] There is a learning curve in the endoscopic approach. The most feared complication is nerve injury. A large review of published studies between 1966 and 2001 found that structural injury to nerve, tendons, or arteries, although very uncommon, is more common in the open approach (0.49%) compared with the endoscopic method (0.19%).[33] The open approach is the method that would be offered most often by the surgeon who is not specifically hand trained, which could account for those findings. Neuropraxia is more common in the endoscopic approach, likely due to the blunt dissection of the synovial tissue from the undersurface of the TCL and the placement of the instruments within the closed space of the carpal tunnel.[33] Care should be taken not to push the instruments too far into the palm past the TCL. Open carpal tunnel release has more wound complications such as infection, hypertrophic scar, and scar tenderness.[22] Pillar tenderness is a vague discomfort in the palm over the TCL that can last weeks to months. Theories range from cutaneous nerve injury during the open release, scar tenderness, and alterations to the structure of the carpal arch.[34] Reassurance and hand therapy are the treatment.

The idea of "recurrent" or "persistent" carpal tunnel syndrome is real, but needs to be approached cautiously. A careful history will determine if the symptoms never resolved or if they returned quickly after surgery. Return of preoperative symptoms within 6 months is considered "persistent" carpal tunnel syndrome and is typically due to incomplete release of the TCL. Recurrent carpal tunnel would be from scar compression that recreates the TCL.[35,36] All patients are sent for repeat NCS/EMGs, which are compared to the originals. If findings are unchanged or worse, reoperation is offered under regional block. If scar adhesions are found between the nerve and ligament (or re-created ligament), a hypothenar fat flap transfer or vein wrapping is offered.[37]

All the methods of ulnar nerve decompression at the elbow have their supporters in the literature.[38,39,40,41] In general, open in situ release is recommended for patients with more mild findings on exam and nerve studies or for patients who need a faster surgery with less dissection. Simple decompression has been found to have comparable outcomes with the more complicated procedures, with fewer potential complications. The downside is the potential for ulnar nerve subluxation, which needs to be evaluated intraoperatively by passively mobilizing the elbow.

Anterior transposition is the method preferred by the authors because it relieves the tension on the ulnar nerve during elbow flexion and has the advantage of complete visualization and release. The nerve is held in place with a subcutaneous or fascial sling that is either sutured to the skin (subcutaneous) or to fascia (transmuscular). Good results with improvement in sensation and strength have been noted.

Intramuscular transposition places the nerve within the muscle mass, while submuscular transposition places the nerve under the muscle mass. These techniques allow for more protection of the nerve, but are more technically demanding and require longer immobilization. They also risk recompressing the nerve.[38]

Complications from cubital tunnel release include incomplete relief of symptoms; recovery up to 2 years; injury to the medial antebrachial cutaneous nerve resulting in neuroma, neuropraxia, or loss of sensation to the elbow skin; seroma; infection; hematoma; and, rarely, recurrence that can be due to incomplete release or scar adhesions. In the event of a suspected recurrence, EMGs need to be ordered and original operative notes reviewed. Persistent symptoms are usually a result of incomplete decompression or scaring around the nerve and can be treated with a revision decompression, which has been shown to provide improved, but inferior, results compared with primary surgery.[38,42]

As with all nerve pathology, enthusiasm for treatment needs to be tempered with reality. Patients who are older, have more proximal lesions, show muscle atrophy, or have diabetic neuropathy may never get complete relief. We counsel the patients that the first objective is to prevent persistent long-term and irreparable injury.

27.7 Review Questions

27.7.1 Choose the Best Answer

1. NCSs/EMGs would NOT be helpful for which nerve compression syndrome?
   a) Carpal tunnel syndrome.
   b) Ulnar tunnel syndrome.
   c) PIN syndrome.
   d) Pronator syndrome.
   e) Cubital tunnel syndrome.

2. All are points of ulnar nerve compression except
   a) Arcade of Struthers.
   b) Osborne's ligament.
   c) Arcade of Frohse.
   d) Anconeus muscle.
   e) Flexor pronator fascia.
3. Patients presenting with recurrent or persistent nerve compressions are evaluated by
   a) Physical exam.
   b) Reviewing previous operative notes.
   c) Reviewing preoperative EMGs.
   d) Ordering updated nerve studies.
   e) All the above.

27.7.2 True or False

4. First-line treatment for Wartenberg's syndrome (radial sensory nerve compression) is surgery.
5. Guyon's canal is divided into three sections: section 1 (motor and sensory), section 2 (motor), and section 3 (sensory).

27.7.3 Answers

1. d. Pronator syndrome.
2. c. Arcade of Frohse.
3. e. All the above.
4. False.
5. True.

References

[1] Lifchez SD, Means KR , Jr, Dunn RE, Williams EH, Dellon AL. Intra- and inter-examiner variability in performing Tinel's test. J Hand Surg Am. 35(2):212–216
[2] Chen SH, Tsai TM. Ulnar tunnel syndrome. J Hand Surg Am. 39(3):571–579
[3] Dang AC, Rodner CM. Unusual compression neuropathies of the forearm, part I: radial nerve. J Hand Surg Am. 34(10):1906–1914
[4] Kenney RJ, Hammert WC. Physical examination of the hand. J Hand Surg Am. 39(11):2324–2334, quiz 2334
[5] Petersen P, Petrick M, Connor H, Conklin D. Grip strength and hand dominance: challenging the 10% rule. Am J Occup Ther. 43(7):444–447
[6] Werner RA, Andary M. Electrodiagnostic evaluation of carpal tunnel syndrome. Muscle Nerve. 44(4):597–607
[7] Weiss L, Silver J, Weiss J. Easy EMG. New York, NY: Butterworth-Heinemann; 2004
[8] Shurr DG, Blair WF, Bassett G. Electromyographic changes after carpal tunnel release. J Hand Surg Am. 11(6):876–880
[9] Kanatani T, Nagura I, Kurosaka M, Kokubu T, Sumi M. Electrophysiological assessment of carpal tunnel syndrome in elderly patients: one-year follow-up study. J Hand Surg Am. 39(11):2188–2191
[10] Edgell SE, McCabe SJ, Breidenbach WC, LaJoie AS, Abell TD. Predicting the outcome of carpal tunnel release. J Hand Surg Am. 28(2):255–261
[11] Wang AA, Hutchinson DT. The effect of corticosteroid injection for trigger finger on blood glucose level in diabetic patients. J Hand Surg Am. 31(6):979–981
[12] Stepan JG, London DA, Boyer MI, Calfee RP. Blood glucose levels in diabetic patients following corticosteroid injections into the hand and wrist. J Hand Surg Am. 39(4):706–712
[13] Edmunds I, Avakian Z. Hand surgery on anticoagulated patients: a prospective study of 121 operations. Hand Surg. 15(2):109–113
[14] Naito K, Lequint T, Zemirline A, Gouzou S, Facca S, Liverneaux P. Should we stop oral anticoagulants in the surgical treatment of carpal tunnel syndrome? Hand (NY). 7(3):267–270
[15] Bogunovic L, Gelberman RH, Goldfarb CA, Boyer MI, Calfee RP. The impact of antiplatelet medication on hand and wrist surgery. J Hand Surg Am. 38(6):1063–1070
[16] O'Shaughnessy MA, Adams JE. Perioperative management of hypertension in hand surgery patients. J Hand Surg Am. 40(8):1684–1687
[17] Kang JR, Yao J. Perioperative management of diabetic patients undergoing hand surgery. J Hand Surg Am. 40(5):1028–1031, quiz 1031
[18] Underwood P, Askari R, Hurwitz S, Chamarthi B, Garg R. Preoperative A1C and clinical outcomes in patients with diabetes undergoing major noncardiac surgical procedures. Diabetes Care. 37(3):611–616
[19] Munns J, Awan H. Trends in carpal tunnel surgery: an online survey of members of the ASSH. J Hand Surg Am. 40(4):767–771
[20] Larsen MB, Sørensen AI, Crone KL, Weis T, Boeckstyns ME. Carpal tunnel release: a randomized comparison of three surgical methods. J Hand Surg Eur Vol. 38(6):646–650
[21] Thoma A, Veltri K, Haines T, Duku E. A meta-analysis of randomized controlled trials comparing endoscopic and open carpal tunnel decompression. Plast Reconstr Surg. 114(5):1137–1146
[22] Vasiliadis HS, Xenakis TA, Mitsionis G, Paschos N, Georgoulis A. Endoscopic versus open carpal tunnel release. Arthroscopy. 26(1):26–33
[23] Sayegh ET, Strauch RJ. Open versus endoscopic carpal tunnel release: a meta-analysis of randomized controlled trials. Clin Orthop Relat Res. 473(3):1120–1132
[24] Dang AC, Rodner CM. Unusual compression neuropathies of the forearm, part II: median nerve. J Hand Surg Am. 34(10):1915–1920
[25] Lowe JB , III, Maggi SP, Mackinnon SE. The position of crossing branches of the medial antebrachial cutaneous nerve during cubital tunnel surgery in humans. Plast Reconstr Surg. 114(3):692–696
[26] Nakamura K, Uchiyama S, Ido Y, et al. The effect of vascular pedicle preservation on blood flow and clinical outcome following ulnar nerve transposition. J Hand Surg Am. 39(2):291–302
[27] Tosti R, Fowler J, Dwyer J, Maltenfort M, Thoder JJ, Ilyas AM. Is antibiotic prophylaxis necessary in elective soft tissue hand surgery? Orthopedics. 35(6):e829–e833
[28] Stanek JJ, Renslow MA, Kalliainen LK. The effect of an educational program on opioid prescription patterns in hand surgery: a quality improvement program. J Hand Surg Am. 40(2):341–346
[29] Kronlage SC, Menendez ME. The benefit of carpal tunnel release in patients with electrophysiologically moderate and severe disease. J Hand Surg Am. 40(3):438–44.e1
[30] Hattori Y, Doi K, Koide S, Sakamoto S. Endoscopic release for severe carpal tunnel syndrome in octogenarians. J Hand Surg Am. 39(12):2448–2453
[31] Thomsen NO, Cederlund RI, Andersson GS, Rosén I, Björk J, Dahlin LB. Carpal tunnel release in patients with diabetes: a 5-year follow-up with matched controls. J Hand Surg Am. 39(4):713–720
[32] Vasiliadis HS, Georgoulas P, Shrier I, Salanti G, Scholten RJ. Endoscopic release for carpal tunnel syndrome. Cochrane Database Syst Rev. 1(1):CD008265
[33] Benson LS, Bare AA, Nagle DJ, Harder VS, Williams CS, Visotsky JL. Complications of endoscopic and open carpal tunnel release. Arthroscopy. 22(9):919–924, 924.e1–924.e2
[34] Ludlow KS, Merla JL, Cox JA, Hurst LN. Pillar pain as a postoperative complication of carpal tunnel release: a review of the literature. J Hand Ther. 10(4):277–282
[35] Neuhaus V, Christoforou D, Cheriyan T, Mudgal CS. Evaluation and treatment of failed carpal tunnel release. Orthop Clin North Am. 43(4):439–447
[36] Mosier BA, Hughes TB. Recurrent carpal tunnel syndrome. Hand Clin. 29(3):427–434
[37] Chrysopoulo MT, Greenberg JA, Kleinman WB. The hypothenar fat pad transposition flap: a modified surgical technique. Tech Hand Up Extrem Surg. 10(3):150–156
[38] Elhassan B, Steinmann SP. Entrapment neuropathy of the ulnar nerve. J Am Acad Orthop Surg. 15(11):672–681
[39] Charles YP, Coulet B, Rouzaud JC, Daures JP, Chammas M. Comparative clinical outcomes of submuscular and subcutaneous transposition of the ulnar nerve for cubital tunnel syndrome. J Hand Surg Am. 34(5):866–874
[40] Mitsionis GI, Manoudis GN, Paschos NK, Korompilias AV, Beris AE. Comparative study of surgical treatment of ulnar nerve compression at the elbow. J Shoulder Elbow Surg. 19(4):513–519
[41] Watts AC, Bain GI. Patient-rated outcome of ulnar nerve decompression: a comparison of endoscopic and open in situ decompression. J Hand Surg Am. 34(8):1492–1498
[42] Aleem AW, Krogue JD, Calfee RP. Outcomes of revision surgery for cubital tunnel syndrome. J Hand Surg Am. 39(11):2141–2149

28 Replantation and Amputation of Fingers

Michael Friel and William C. Lineaweaver

Abstract
This chapter deals with a significant trauma that remedially requires the rapid processing of many issues. Surgery is based on the findings of medical, laboratory, and radiologic evaluations; factors that enhance the possibility of success (e.g., wrapping the amputated digit in moist gauze, sealing it in a plastic bag that is placed on ice) are covered, and surgical contraindications are enumerated. The examination of affected bones, tendons, nerves, arteries, and veins is described, and if surgery is deemed possible, the recommended techniques and procedures (including the use of leeches) are sequentially listed. Because postoperative care for both replantation and amputation is critical, the authors conclude their study by going into some detail about practices that increase the odds for a satisfactory outcome.

Keywords: replantation, amputation

28.1 Goals and Objectives

- Review the indications for replantation and amputation of fingers.
- Outline technical aspects of finger replantation and amputation.
- Summarize reported outcomes and evidence-based recommendations for management of fingers evaluated for replantation or amputation.

28.2 Patient Presentation

In the setting of traumatic amputations involving one or more fingers, decisions regarding replantation and amputation have complex implications for patients. It is necessary for patients and surgeons to work through many issues in a relatively short period of time to determine an optimal treatment plan.

Initial evaluation must include an examination of the injured parts (including physical examination and X-ray), review of the mechanism of injury, and thorough assessment of any associated injuries and coexistent medical problems.[1,2,3]

If a patient is first seen in a facility which will transfer the patient elsewhere for definitive management, primary emergency care should include dressing and elevation of the injured hand; wrapping of the amputated parts in moist gauze and subsequent placement into a specimen cup or plastic bag which is then placed on ice; establishment of intravenous access in an uninjured extremity; evaluation of medical problems and associated injuries; performance of indicated laboratory and X-ray studies; and administration of indicated antibiotics, analgesics, and tetanus prophylaxis. If the patient presents to the same hospital where definitive care will be offered, the responsible hand surgeon can directly oversee this primary management.

With accomplishment of initial evaluation, the surgeon can examine the amputated parts. The patient and surgeon then must arrive at a treatment plan.

Replantation can offer possibilities of maximum salvage of function with the drawbacks of prolonged surgery and hospitalization as well as prospects of lengthy postoperative therapy and secondary procedures. Each patient will have individual priorities to bring to the formulation of a surgical plan; each injury will offer the surgeon a range of possibilities and limitations. These elements must be assembled into the best available composite of patient expectation and surgeon performance.

The indications for replantation have been established over decades. Prime opportunities for replantation based on prospects for useful salvage of function include amputation of thumbs, multiple fingers, amputations in children, and finger amputations distal to superficialis tendon insertion. Contraindications can include crushing and multilevel amputations and single-finger injuries proximal to the superficialis insertion. Specific patient issues, including commitments to recovery time, can ultimately determine the surgical plan.[4]

If replantation is not a consideration, or if intraoperative findings or postoperative failure aborts a replantation effort, the surgeon must apply effective amputation strategies.

28.3 Treatment

28.3.1 Preparation for Surgery

Review of the preparations described earlier, including medical, laboratory, and radiologic evaluations, forms the basis for proceeding to surgery. Radiographs of the hand will provide information regarding the level and extent of bone and/or joint trauma. Similarly, radiographs of the amputated part may be desired.

Informed consent should include options for amputation as well as replantation procedures that could include vein, nerve, and skin grafts, as well as anticoagulant use and blood transfusion.

The patient should be further informed of the duration of surgery and hospitalization, risks of replant failure, length of recovery, and possible need for secondary operations.

28.4 Replantation

Initial exploration of the amputated digit includes examination of bone and tendons, as well as identification and tagging of digital nerves, digital arteries, and volar veins. These maneuvers are facilitated by making midlateral incisions in the digit, allowing reflection of dorsal and volar tissue to create a wide field (▶ Fig. 28.1).

The injured hand is explored under tourniquet control. Proximal incisions are made, usually in a Bruner pattern, to identify and tag critical structures to match the components prepared in the amputated part.[5]

Generally, replantation of multiple digits is prioritized from thumb to ulnar digits with the index finger being the most functionally expendable.[6] A cleanly amputated part can be replanted at a more optimally functional site if that site's amputated part is unreplantable. For example, a cleanly amputated

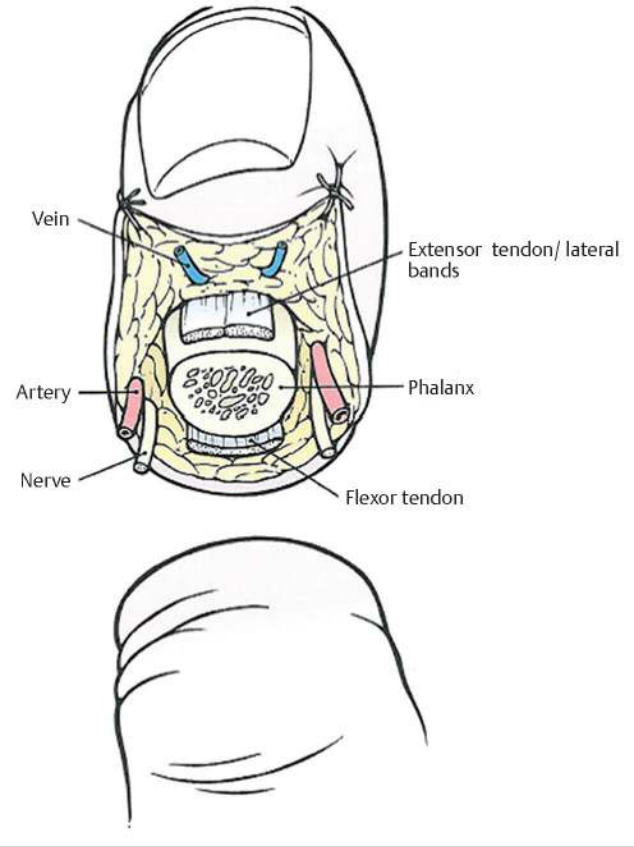


Fig. 28.1 Structures exposed for repair in a middle phalangeal amputation.

index finger can be replanted onto a thumb site when the amputated thumb itself is not replantable.[7]

Replantation follows an organized sequence of bone fixation, volar repairs (flexor tendons, digital arteries, digital nerves, and skin closure), and dorsal repairs (extensor tendon, veins, and skin closure).[4,6]

A number of skeletal fixation techniques have been used in replantation, but crossed Kirschner wires (K-wires) offer speed of application and simplicity of technique with no documented outcome disadvantages relative to more complicated systems. K-wires can be placed through the amputated part at the initial dissection. Replantation can then commence with reduction of the amputated bone into the proximal skeletal stump, usually under direct visualization. The K-wires are then driven into the proximal bone. Wire position and reduction can be confirmed by fluoroscopy.[4,6,8]

Flexor tendon repairs are performed next. At the initial exploration, sutures can be placed in the proximal and distal tendon stumps with each suture representing one half of the selected tendon repair technique. Completion of the repair is accomplished by tying the two segments of sutures together.[4,9]

The tourniquet is released following skeletal and flexor tendon repairs. Under the microscope, the digital arteries and nerves are examined. The proximal and distal margins of these structures are trimmed to grossly uninjured levels. The proximal artery should be treated with mechanical dilation, heparin irrigation, and topical vasodilation (e.g., papaverine) until pulsatile flow is identified. The digital artery segments are then anastomosed, usually with 9–0 or 10–0 nylon sutures. Repairs of both arteries should be considered to guard against failure of a single repaired vessel. Both digital nerves are next repaired using 9–0 or 10–0 nylon sutures. The volar skin is closed as loosely as possible.[4,6,9]

Significant gaps in specific tissues can be addressed with grafts. Vein grafts can be used to bridge arterial defects. These grafts can be harvested from a foot, with veins near a web space being a good match for digital arteries while veins on the distal dorsum of the foot are generally suitable for common digital arteries.[10] Digital nerve gaps rarely have sufficiently secured skin cover for the surgeon to consider autologous nerve grafts, but small vein conduits or cadaveric nerve allografts can be successfully applied.[11,12] Significant gaps in skin cover, including defects exposing underlying neurovascular repairs, can be covered with small split or thinned full-thickness skin grafts.[13]

After completion of volar repairs, the hand is turned for access to the dorsal injury. The extensor tendon is repaired with absorbable sutures. One or more veins are then repaired, and vein grafts can be used here also. Veins can be identified during the initial dissection, but optimal distal vein selection is best accomplished after arterial repairs establish venous outflow from the amputated part.[4] Dorsal skin closure completes this procedure.

For multiple replantations, a functional operative sequence includes preliminary dissection and tagging of all proximal and distal structures; skeletal reduction; flexor tendon repairs; introduction of the microscope and tourniquet release; all arterial repairs; all nerve repairs; volar skin closure; and turning of the hand for all extensor repairs followed by vein repairs and dorsal skin closure (▶ Fig. 28.2). First priority for salvage should be given to the thumb, followed by little, ring, middle, and index fingers.

28.5 Postoperative Care

Preoperative and intraoperative coagulation modifiers are used empirically by many surgeons. These agents can be used in combination and continued postoperatively. Agents include aspirin, low-molecular-weight dextran, and full- or low-dose heparinization. No clear evidence supports any specific recipe for these agents, and prolonged use of combination therapies substantially increases the possible need for blood transfusion.[2,14]

The dressing applied at the end of the surgical procedure should have minimal potential for circumferential pressure, should be easily changed, and should allow inspection of the replanted parts. One technique is a "barrel-stave dressing." Initial layers of nonadherent gauze strips are applied to the dorsal and volar surfaces of the fingers. Dry gauze of 4 × 4 is placed in a sandwich fashion to the dorsal and volar surfaces of the finger and hand, and this layer is secured by a loose wraparound gauze roll. A volar plaster splint is applied to maintain the wrist in 30-degree extension and the fingers in comfortable extension. The splint is held in place by a wraparound bias dressing, and the distal gauze layers are adjusted to expose the finger tips.[6]

Postoperative monitoring can include clinical examinations and techniques such as quantitative fluorimetry, pulse oximetry, temperature monitors, and laser monitors. Clear evidence to support any of these techniques for monitoring replants is

Fig. 28.2 **(a)** Complex injury to the left hand, with pulp laceration of the thumb; amputation of index; near-complete amputation of middle; devascularization of ring; and distal phalangeal fracture-nail bed injury of little. **(b)** Completion of procedure which included replantation of index (K-wire reduction of proximal phalanx, repair of flexor digitorum superficialis [FDS], flexor digitorum profundis [FDP], ulnar digital nerve [UDA], ulnar digital nerve [UDN], extensor tendon, volar vein with graft); middle (fusion; proximal interphalangeal joint [PIPJ]; repair of FDS, FDP, radial digital artery [RDA], UDA, radial digital nerve [RDN], UDN; extensor tendon; identification of patent dorsal vein in skin bridge); and salvage of ring (repair, PIPJ volar plate, FDS, FDP, RDA, RDN, UDN). Procedure time was 7.5 hours. **(c,d)** Healed wounds at 3 months with functional large object grip.

minimal, but the monitoring process should function as a clinical alert for either arterial or venous occlusions.[1,15] With evidence of vascular compromise, the first response should be complete removal of the splint and dressings to relieve any external compression. In cases of arterial occlusion, operative reexploration can be considered. Often, salvage will consist of resection of damaged vessel margins and vein graft reconstruction. Venous occlusion may also be reexplored, but can be treated with leech application for 24 to 72 hours while capillary venous return is established across the replantation site. Leech application can salvage up to 70% of replants with patent arteries and occluded or no veins. Antibiotics effective against leech enteric organisms (especially *Aeromonas* species) should be administered to prevent secondary infections.[16] For any vascular complication, systemic anticoagulation can be considered.

Intensive hand therapy is required for replanted digits to maximize function. Early range of motion is optimal for prevention of tendon adhesions and joint stiffness. Nevertheless, this is limited until fracture healing is stable. Scar and soft-tissue management may also be required.

28.6 Amputation

A great majority of cases considered for replantation include digits too damaged for replantation or, more rarely, patients who are unsuitable for replantation because of medical problems, coincident injuries, or unwillingness to commit to the prolonged surgery, hospitalization, and rehabilitation associated with replantation.

In such cases, the hand surgeon seeks to provide the most functional solutions to the acute injury. Rarely, at the time of initial injury, the surgeon should consider elaborate strategies of ray amputation, digit transfer, or toe transplants. Amputation is the most common acute surgical procedure, and should concentrate on maximal preservation within the limits of available soft-tissue coverage and functional skeletal parts.[17]

Fingertip amputations can often be covered with local volar or midlateral V-Y flaps to preserve the remaining nail bed and distal phalanx. Cross finger flaps can also be utilized for larger defects, achieving coverage of joints and tendons.[18]

More proximal injuries should be treated by establishing the most secure and functional skeletal amputation level and developing volar and dorsal skin flaps from available viable tissue (▶ Fig. 28.3).[18]

Thumb amputations offer additional incentives for stump preservation since the proximal amputation can be the basis for digit pollicization, toe transplantation, or functional lengthening from widening of the first web space. Metacarpophalangeal joint coverage by local or pedicled flaps can be an important element of acute salvage. Excessive debridement of digital nerves or tendons should also be avoided (▶ Fig. 28.4; ▶ Fig. 28.5).[17,19]

28.7 Outcomes

Eighty to 90% of attempted finger replantations are reported as initially successful in large series.[1] The definition of functional success requires a more detailed description including sensation, motion, and grip. To improve functional outcome, 30 to 50% of replanted fingers undergo secondary procedures. Such operations should be sequentially planned to achieve soft-tissue coverage, skeletal stability, sensation, passive joint motion, and active motion as these problems are identified.[20]

Skin coverage options can include application of skin grafts, local flaps, or microsurgical flaps. Skeletal stability requires

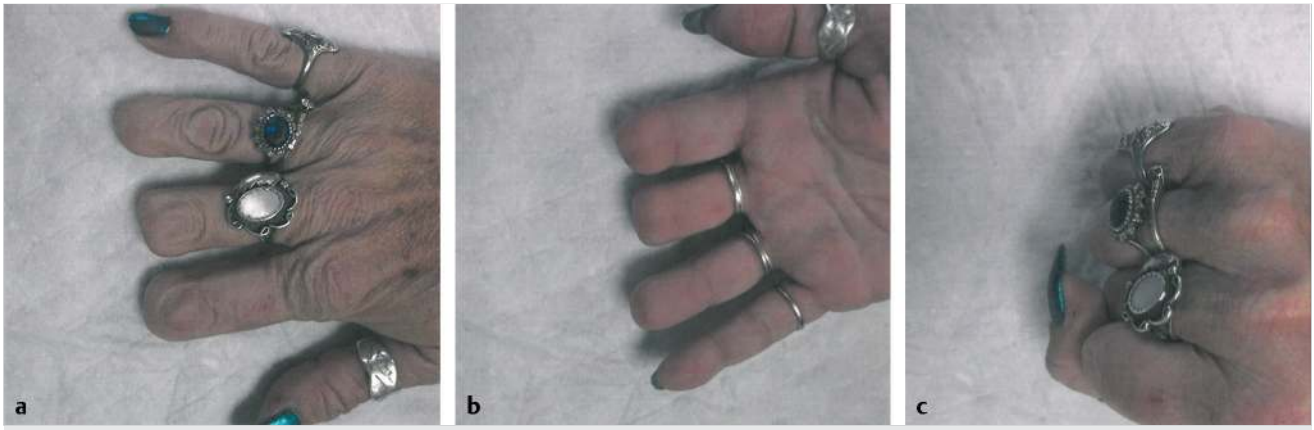


Fig. 28.3 (a–c). Functional late results after primary amputations.

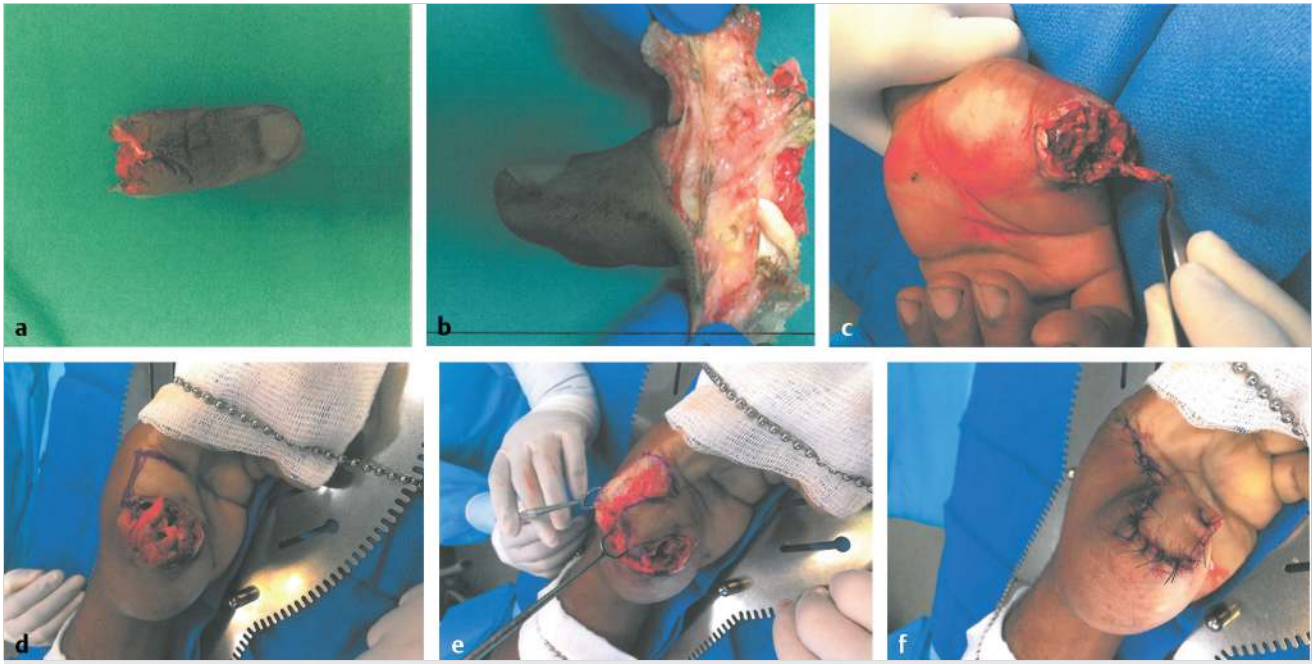


Fig. 28.4 (a) Unreplantable thumb amputation with multiple levels of injury and extensive avulsion. **(b)** Amputation site with avulsed neurovascular bundle. **(c)** Debridement with markings for a transposition flap from the first web space. **(d–f)** Elevation, inset of flap with preservation of the metacarpophalangeal joint.

salvage of nonunions, malunions, or osteomyelitis. Restoration of sensation can require neurolysis, revision of original repairs, or grafting to replace damaged nerve segments. Joint procedures can range from capsulotomies to Silastic replacement prostheses. Tendon function may be restored by tenolysis of original repairs or staged tendon grafting of unsalvageable tendon segments. Optimization of replantation, therefore, requires not only a successful acute procedure but also application of comprehensive hand surgery skills. Reports of secondary procedures following digit replantation document that less than 2% of replants go on to secondary amputation.[20] This low occurrence gives indirect evidence that the great majority of replanted digits are functional.

Acute amputation may also require secondary procedures for problems as diverse as nail remnants, unstable soft-tissue coverage, painful neuromas, chronic infections, or impaired function related to an unfavorable amputation stump. This last circumstance can lead to such procedures as a ray amputation of an index stump to re-create an open first web space, or transfer of a little finger to a ring metacarpal stump to close a gap in grip.[18]

Secondary procedures following amputation should be planned to coincide with maximum return of function in therapy as well as definition of problems. For example, initial pain at the site of the digital nerve transections may resolve in 6 to 12 weeks and not require exploration for neuromas. The stump of an index finger may not cause problems until a patient returns to work where repeated trauma may indicate that a ray amputation can offer more functional first web space.

Fig. 28.5 Digital nerve stumps, prior to neuroma resection and transposition into hypothenar muscles.

28.8 Conclusion

Replantation is feasible only in a small number of patients seen for amputations. A successful replant or an optimal initial amputation can be the basis for a best possible outcome for a patient with a serious hand injury, and hand surgeons should be able to offer this spectrum of reconstructive procedures to best serve their patients.

28.9 Review Questions

28.9.1 Choose the Best Answer

1. Contradictions to replantation include
   a) Crushed amputated parts.
   b) Pediatric patients.
   c) Patients older than 65 years.
   d) Amputations distal to the flexor superficialis insertion.
   e) Multiple digit amputations on the same hand.
2. For transportation to a medical facility, an amputated part should be
   a) Placed in a sterile container filled with saline.
   b) Wrapped in the dressing applied to the injured hand.
   c) Placed directly on ice in a sealed container.
   d) Wrapped in moist gauze, sealed in a plastic bag which is then placed on ice.
   e) Placed in a sterile glove with the cuff tied off and placed on dry ice.
3. In multiple-digit amputations, the highest priority should be given to replantation at the
   a) Thumb site.
   b) Index site.
   c) Middle site.
   d) Ring site.
   e) Little site.
4. The first step in replantation should be
   a) Skeletal fixation.
   b) Flexor tendon repairs.
   c) Exploration and tagging of critical structures.
   d) Arterial repair.
   e) Vein repairs.
5. Leeches can be applied to replanted parts to treat
   a) Arterial occlusion.
   b) Venous occlusion.
   c) Edema.
   d) Pain.
   e) Necrotic skin margins.

28.9.2 Answers

1. a. Crushed amputated parts.
2. d. Wrapped in moist gauze, sealed in a plastic bag which is then placed on ice.
3. a. Thumb site.
4. c. Exploration and tagging of critical structures.
5. b. Venous occlusion.

References

[1] Pederson WC. Replantation. Plast Reconstr Surg. 107(3):823–841
[2] Prucz RB, Friedrich JB. Upper extremity replantation: current concepts. Plast Reconstr Surg. 133(2):333–342
[3] Partington MT, Lineaweaver WC, O'Hara M, et al. Unrecognized injuries in patients referred for emergency microsurgery. J Trauma. 34(2):238–241
[4] Maki Y. Digital replantation. In: Boyd JB, Jones NF, eds. Operative Microsurgery. New York, NY: McGraw-Hill; 2015:685–697
[5] Adamson J, Fleury A. Incisions in the hand and wrist. In: Green DP, ed. Operative Hand Surgery, 2nd ed. New York, NY: Churchill Livingston; 1988:1785–1804
[6] Lineaweaver W, Buncke GM, Alpert BS, Buncke HJ. Pitfalls in replantation surgery. In: Vistnes L, ed. Procedures in Plastic and Reconstructive Surgery. Boston: Little Brown; 1991:699–707
[7] An P, Kuo Y, Lin T, et al. Heterotopic replantation in mutilating hand injury. Ann Plast Surg. 15A:328–334
[8] Whitney TM, Lineaweaver WC, Buncke HJ, Nugent K. Clinical results of bony fixation methods in digital replantation. J Hand Surg Am. 15(2):328–334
[9] Dzwierzynski W. Replantation and revascularization. In: Neligan P, ed. Plastic Surgery, 3rd ed. Vol. 6. London: Elsevier, 2013:228–249
[10] Buncke HJ, Whitney T, Valauri F, Alpert BS. Replantation surgery. In: Buncke HJ, ed. Microsurgery. Philadelphia, PA: Lea & Febiger; 1991:589–591
[11] Chiu D, Straush B. A prospective clinical evaluation of autogenous vein grafts for distal sensory nerve defects of 3 cm or less. Plast Reconstr Surg. 86: 928–934
[12] Brooks DN, Weber RV, Chao JD, et al. Processed nerve allografts for peripheral nerve reconstruction: a multicenter study of utilization and outcomes in sensory, mixed, and motor nerve reconstructions. Microsurgery. 32(1):1–14
[13] Lineaweaver WC. Skin graft coverage of critical marginal wounds in microsurgical cases. Microsurgery. 33(4):315–317
[14] Furnas HJ, Lineaweaver W, Buncke HJ. Blood loss associated with anticoagulation in patients with replanted digits. J Hand Surg Am. 17(2):226–229
[15] Chae MP, Rozen WM, Whitaker IS, et al. Current evidence for postoperative monitoring of microvascular free flaps: a systematic review. Ann Plast Surg. 74(5):621–632
[16] Whitaker IS, Josty IC, Hawkins S, et al. Medicinal leeches and the microsurgeon: a four-year study, clinical series and risk benefit review. Microsurgery. 31(4):281–287
[17] Adamson G, Palmer RE. Amputations. In: Achauer B, Erikson E, Guyuron B, et al, eds. Plastic Surgery. St. Louis, MO: Mosby; 2000:1831–1843
[18] Jebson PJL, Louis DS. Amputations. In: Green DP, Hotckiss RN, Pederson WC, et al, eds. Operative Hand Surgery, 5th ed. Philadelphia, PA: Elsevier; 2005:1939–1983
[19] Lineaweaver W. Great toe to thumb transfer. In: Weinzweig N, Weinzweig J, eds. The Mutilated Hand. Philadelphia, PA: Elsevier; 2005:163–173
[20] Lineaweaver W, Oswald T. Secondary surgery following digit replantation. In: Grotting J, ed. Reoperative Aesthetic and Reconstructive Surgery, 2nd ed. St Louis, MO: QMP; 2007:1939–1951

29 Dupuytren's and Trigger Finger

Chris McCarthy and Sonu A. Jain

Abstract

This chapter examines the anatomy, pathology, and treatment of Dupuytren's disease and the not uncommon malady known as trigger finger. Surgery is often the only remedy for Dupuytren's, and the authors guide the reader through the intervention options. Trigger finger presents as a catching or locking sensation in the digits, and treatment—nonsurgical or surgical—depends on the severity of the problem. For the surgical option, the authors describe each step of the procedure and caution about possible complications. Postoperative dressings are reviewed and expected outcomes cited at the conclusion of the chapter.

Keywords: Dupuytren's disease, trigger finger, fasciectomy, palmar skin management, collagenase botulinum histolyticum

29.1 Dupuytren's Disease (Palmar Fibromatosis)

29.1.1 Goals and Objectives

- To understand the anatomy and pathology of at-risk patient populations for Dupuytren's disease.
- To understand the surgical indications and options for Dupuytren's patients.
- To be able to avoid potential complications of Dupuytren's surgery and educate patients appropriately on expectations of surgery and the risk of recurrence.

29.1.2 Patient Presentation

A typical presentation of a patient with Dupuytren's disease is described: A 60-year-old man of Scandinavian descent presents with progressive flexion contracture of his ring and little fingers over the last 6 months. The deformity is causing inability to perform daily tasks with his right hand which originally began with a painful nodule in his palm. This became nonpainful over time despite developing into a larger cord of tissue. His father had a similar condition in the past. On physical exam, he has a metacarpophalangeal (MCP) joint flexion contracture of 45 degrees and a proximal interphalangeal (PIP) joint contracture of 45 degrees along with palpable cords to both the ring and little fingers. He has normal sensation and vascular flow to the fingers. Radiographs show no arthritis of the hand and no other anomalies.

29.1.3 Preparation for Surgery

Dupuytren's disease is a disease more predominant to males over the age of 40 years and of Northern European descent, traditionally Scandinavian region and the regions in which Viking conquests took place.[1] It is a proliferation of collagen with an increase in ratio of Type III to Type I collagen in the palmar fascia creating pathologic cords from the normal bands. Myofibroblasts and fibroblasts are the implicated cell lines with residual myofibroblasts in adjacent dermal and epidermal tissues being thought to contribute to recurrence.[1,2,3] Typically, Dupuytren's disease is diagnosed by history and physical exam alone with plain radiographs used to evaluate for any concomitant osteoarthritic changes of contracted joints. MRI and CT evaluations are typically unnecessary. Biopsy is also typically unnecessary unless the presentation is atypical and there is a concern for malignant mass; however, this is a rare presentation.

Understanding of the pathoanatomy is critical in Dupuytren's disease (▶ Fig. 29.1). Normally, as the palmar fascia travels distally toward the digits, it becomes pretendinous bands often. The portion that bifurcates to each digit then becomes spiral bands and traverses to the lateral sheets in the ulnar and radial aspect of each digit which also coalesce and connect to the coronally oriented perineurovascular fascial sheets named "Cleland's and Grayson's ligaments" which attach to the flexor apparatus centrally and the dermis peripherally. Grayson's ligament is volar to the neurovascular bundle and can be involved in Dupuytren's disease, while Cleland's ligament is dorsal and not involved in the disease process. In the distal palm and toward the digits, the spiral bands become dorsal and more peripheral than the neurovascular bundle. In the diseased hand, the bands become pathologic cords and thicken to become spiral cords. The neurovascular bundles are then displaced centrally and more superficial. These changes lead to cutaneous pitting, MCP joint flexion contracture, and, sometimes, PIP joint contracture as well (▶ Fig. 29.2; ▶ Fig. 29.3).

29.2 Treatment

Dupuytren's disease does not predictably resolve or improve with nonoperative treatments such as splinting, ointments, creams, or therapy.[4] Patient education is important for self-monitoring for progression of deformity as it can be slowly progressive and painless. The "table top" test whereby a patient attempts placement of his/her palm flush on a table is helpful. Inability to perform this task can be associated with MCP joint contracture of 30 to 40 degrees.[4] Intervention is standardly recommended for MCP joint contracture of 40 degrees or more in one digit, and if treating that digit, then contracture of other digits may be considered even if deformity is only 20 to 30 degrees. PIP joint contracture is more difficult to improve and some authors advocate for intervention in cases of any contracture; however, McFarlane recommends intervention only when it is more than 30 degrees due to potential risk of actually worsening the disease for interventions at lower degrees.

Fasciectomy is the most common surgical intervention in which all the macroscopically diseased tissues are excised (▶ Fig. 29.4).[5,6,7] Recurrence rates range in the literature from approximately 12 to 73% likely due to remaining microscopic diseased tissue.[2,3] It is difficult to interpret recurrence rates in the literature due to lack of standard definitions for recurrence.[2,3,8] Limited open or percutaneous fasciotomy was originally recommended for elderly or debilitated patients or patients who cannot participate in appropriate postprocedure

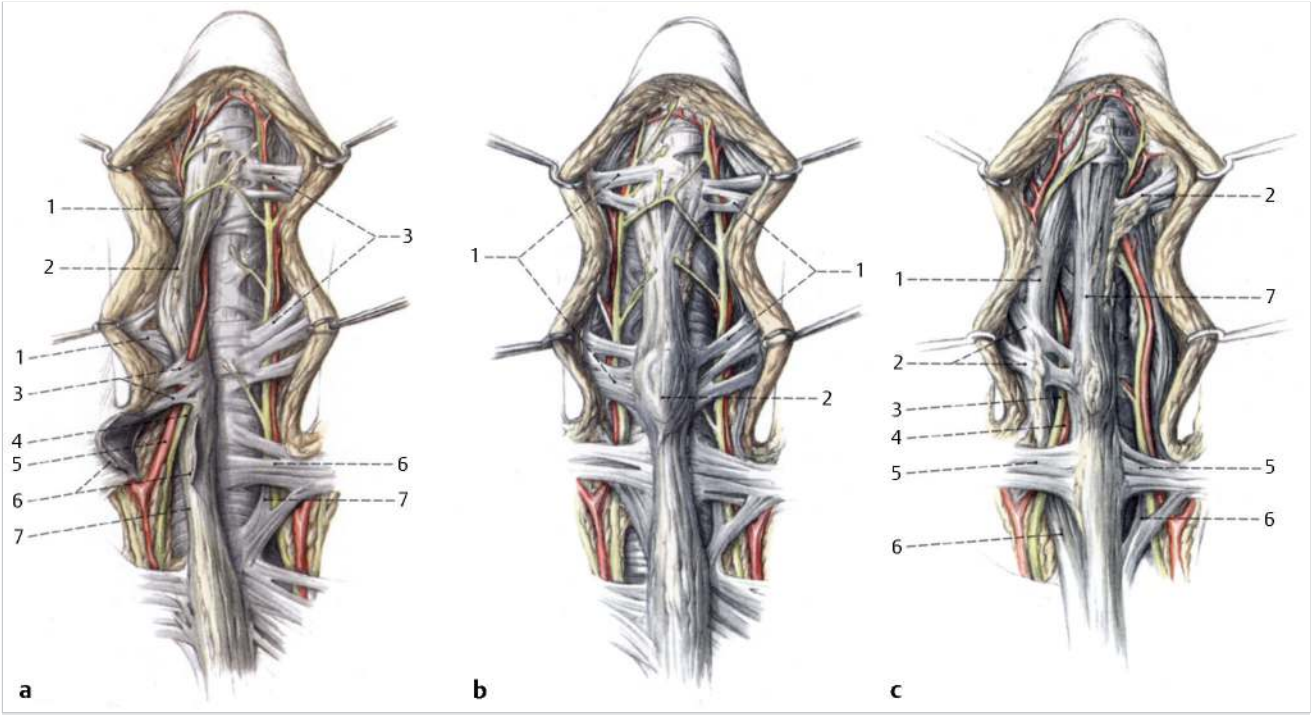


Fig. 29.1 **(a)** Thickening of the longitudinal bands with associated displacement of the neurovascular bundle. (1) Cleland's ligament. (2) Longitudinal bands of the finger. (3) Grayson's ligament. (4) Proper palmar digital nerve. (5) Proper palmar digital artery. (6) Superficial transverse metacarpal ligament (reflected). (7) Mediolateral ligament. **(b)** Formation of fibromatous band over the tendon sheath. (1) Grayson's ligament. (2) Median band. (**c**) Combined disorder with formation of fibromatous median and lateral bands and topographic displacement of the neurovascular bundle. (From Pechlaner S, Hussl H, Kerschbaumer F. Atlas of Hand Surgery. Stuttgart, Germany: Georg Thieme Verlag; 2000)

Fig. 29.2 Cutaneous pitting caused by fibrotic cords in Dupuytren's disease.

Fig. 29.3 Underlying fibrotic process that causes cutaneous pitting and flexion contracture.

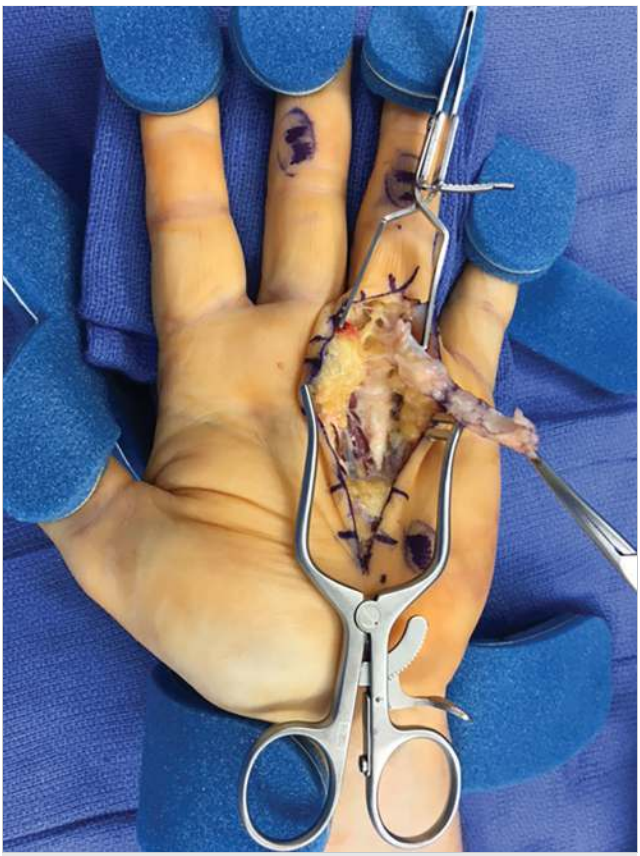


Fig. 29.4 Open excisional fasciectomy with removal of the fibrous cord causing contracture, while preserving the flexor mechanism and neurovascular bundles.

rehabilitation, as they were thought to provide smaller power of correction. However, studies show percutaneous fasciotomy to provide excellent improvement with upward of 72% of MCP joint correction maintained over a 3- to 6-year follow-up period in a series reviewed by Pess et al.[8] PIP joint correction was less well maintained in the long term in their series of more than 1,000 digits. They concluded that percutaneous needle fasciotomy was a safe intervention method but resulted in a larger recurrence rate than open fasciectomy.[9]

Palmar skin management after surgery is treated in three ways: direct closure, full thickness skin grafting, and open technique described by McCash allowing for skin closure by secondary intention through a transverse incision at the mid-palmar crease and then counter incision(s) in the digit(s) being addressed. The biggest complication of primary closure is the risk of postoperative hematoma and subsequent skin edge necrosis. Various incision types can be used including the aforementioned transverse incision as well as a midaxial longitudinal incision which is closed by forming multiple Z-plasties at the flexor creases or Bruner's incisions with excellent exposure (▶ Fig. 29.5). Recurrence necessitating revision surgery is challenging and fasciectomy is recommended. However, the risk of neurovascular injury with devascularization of the digit must be considered. This is at higher risk for recurrent surgery due to the possibility of occult digital artery injury at the primary operation which could have been compensated for by the contralateral vessel. As such a new neurovascular injury could lead to a devascularized digit. Some authors have advocated that percutaneous fasciotomy technique can be utilized in the recurrent patient setting as well. Joint fusion may also be considered in selective cases of the MCP and/or the PIP in settings of painful arthritis, deformity over 90 degrees, dysvascular digit, and patient's preference.[10]

In recent history, the newest and increasingly more popular treatment for Dupuytren's cases is the collagenase injection, *Clostridium histolyticum* (Xiaflex, Auxilium Pharmaceuticals). The collagenase is injected directly into the palpable cord in an office-based procedure and then typically between 24 and 48 hours after the injection, a finger extension manipulation is performed in the office to correct the contracture. The patient is fitted with an extension night time splint to wear for 2-4 months and with daily flexion exercises. The collagenase is approved for both MCP and PIP contractures with an overall recurrence rate (greater than 30 degrees worsening) at 5 year follow up of 26% for MCP joints treated, and 46% of PIP joints treated with an overall recurrence percentage of 47%. Also at 5 years, 16% of treated joints underwent secondary procedures for re-correction of contracture and the most common of which was surgical fasciectomy.[11] Collagenase is a favorable option for many Dupuytren's patients with the proper counseling of recurrence rates and possible complications, the most common of which are swelling and ecchymosis to the hand from the injection followed by skin tearing after the manipulation which is treated with simple dressing changes alone.[12]

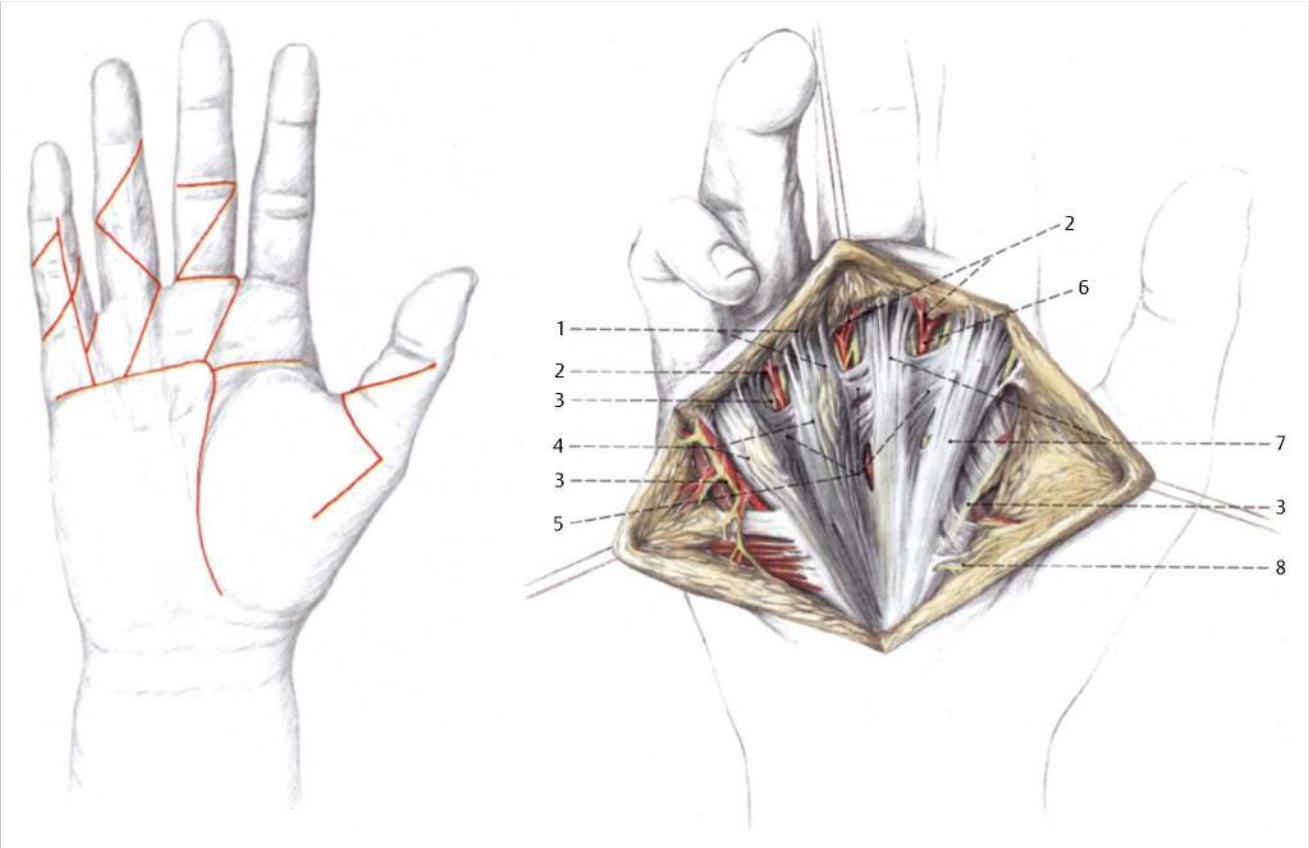


Fig. 29.5 The palmar aponeurosis is dissected to expose the contracted bands of the ring and little fingers. Care is taken to protect and preserve the neurovascular structure supplying the skin. The longitudinal bands of the index and middle fingers are not affected in this illustration. (From Pechlaner S, Hussl H, Kerschbaumer F. Atlas of Hand Surgery. Stuttgart, Germany: Georg Thieme Verlag; 2000)

29.3 Postoperative Care

After surgical excision of the fascial bands, patients are splinted in extension with forearm-based splints, which can be transitioned to removable splints during the first postoperative week. All fingers are splinted in extension to minimize web-space contracture. Occupational therapy is initiated with passive and active range-of-motion exercises for both flexion and extension with passive extension stretching as well. By the second postoperative week, the patients can be weaned to night splinting while encouraging daily home exercises, but splinting usually will last 3 to 6 months. In the case of the McCash technique, patients are covered with a nonadherent dressing in the immediate postoperative period followed by daily dry dressing changes once motion is initiated at 1 week. Strengthening is delayed to approximately 6 weeks in these patients, while it can be initiated by 3 to 4 weeks postoperatively in other techniques.

29.4 Outcomes

Studies show that greater initial correction and PIP joint correction (if applicable) correlate with improved results and hand function at 6 and 12 months of follow-up.[6,13] In a natural history study of patients presenting with nodular disease, Reilly et al followed up 59 patients for 8 years with 30 of 59 patients developing cords; however, only 5 patients meet the criteria for surgical correction and only 3 of those 5 went on to surgery.[10] The primary risk of open fasciectomy is digital artery and/or nerve injury along with hematoma. Risks of percutaneous fasciotomy include skin tears (approximately 3%) and higher recurrence.[7] Collagenase injections have risk of local skin injury such as skin tears and blood blistering which has been reported in up to 46% of patients. Patient education should be held by the surgeon regarding various treatment options related to recurrence rates and risks of intervention to make the surgical decision a very personalized one for patients suffering from Dupuytren's disease.

29.5 Trigger Finger (Stenosing Tenosynovitis)

29.5.1 Goals and Objectives

- To understand the pathoanatomy and types of trigger fingers.
- To understand the treatment algorithm for trigger finger patients.
- To understand the surgical management and risks associated with trigger release.

29.5.2 Patient Presentation

A typical presentation of a patient with trigger finger is described: A 55-year-old woman with diabetes presents with 9 months of ring finger "catching" and "locking" which is increasingly painful and negatively affecting her daily activities as well as hobbies such as knitting.

29.6 Preparation for Surgery

Stenosing tenosynovitis, or trigger finger, comes in two varieties—nodular and diffuse.[14] The condition is more common in women as well as in patients with diabetes and hypothyroidism.[4,15] The most commonly affected digits in order are ring, thumb, index, middle, and little fingers.[4,15] Diagnosis is made by history and physical exam with no need for additional testing. Evaluating the patient for ability to smoothly flex and extend fully the digit in question while palpating over the MCP joint of that digit will yield crepitus with possible tenderness in the area as well. Additionally, the digit may catch or lock causing the need for forceful manipulation toward extension of the digit while suddenly feeling a give way sensation.[4,15] The tenosynovitis occurs at the fulcrum leading edge of the A1 pulley and causes metaplasia both of the pulley and the flexor tendon with histologic changes similar to cartilage metaplasia.[4] Nodular type is a focal inflammation while diffuse is less discrete. The difference can be felt on physical exam with the nodular type having a focally palpable nodule while not present focally in the diffuse type.

29.7 Treatment

Trigger finger management depends on severity, length of symptoms, and age. Several grading classifications have been developed in order to guide treatment. Grade 0 is mild crepitus in a digit without triggering, grade 1 is uneven movement of the digit, grade 2 is a digit which clicks but does not lock, grade 3 is a locking digit, and grade 4 is a locked digit (▶ Table 29.1).[4] Initial nonoperative management includes nonsteroidal anti-inflammatory medicines, ice, massage, avoidance of possible inciting events based on the patient's history, and splinting.[16] Splinting should allow free PIP and DIP joint motion and leave the MCP joint in either 0 degrees or up to 10 to 15 degrees of flexion.[17] Splinting may be necessary for up to 4 months.[4] Grade 4 locked digits do not respond to these nonoperative measures.

Corticosteroid injections may be used and have been used in all grades of trigger finger.[4,17,18] Betamethasone sodium phosphate and acetate suspension are commonly used, although all types of corticosteroids have been reported. Betamethasone has the benefit of being water soluble and has the potential for less risk of fat necrosis.[4] According to the treatment algorithm proposed by Saldana, if the trigger finger has been present for less than 6 months and grade 3 or lower, NSAIDs along with possible splinting, ice, and massage should be attempted.[4,16] If not resolved, then corticosteroid injections should be attempted up to twice at 1-month recheck intervals.[17,18] Effectiveness of corticosteroid injection has been reported to be approximately 70% overall with one study showing a 93% response in nodular type compared to 48% response for diffuse type.[19] If still unresolved, trigger finger release should be offered. In the case of grade 4 digits, surgical release should be offered due to ineffectiveness of nonoperative measures.[4]

Table 29.1 Trigger finger grading

Grade	Features of affected digit
0	Mild crepitus without triggering
1	Uneven movement
2	Clicking without locking
3	Locking
4	Locked

29.8 Surgical Treatment

Open release of the A1 pulley is the standard surgical intervention for trigger finger.[4] This can be performed with a longitudinal or transverse incision at the distal palmar crease and generally under local anesthetic with a tourniquet (▶ Fig. 29.6). The A1 pulley is identified, and radial and ulnar neurovascular bundles protected, followed by longitudinal full release of the A1 pulley taking care not to release the more distal A2 pulley. The patient can then be asked to flex and extend the digit to ensure full release.

Some authors have described a percutaneous release, which can be performed in the office setting. Local anesthetic along with corticosteroid is injected into the A1 pulley region of the digit in question.[17] A 20-gauge needle is inserted at the location of midpoint of the A1 pulley which corresponds to one-third the distance from the distal palmar crease to the digitopalmar crease of the little, ring, and middle fingers.[17] The index finger landmark is one-third the distance from the thenar crease to the index digitopalmar crease and the thumb entry site is located at the midaxial point of the thumb digitopalmar crease.[17] The bevel of the needle is used to cut the pulley proximally and distally until full release is obtained and the patient has relief of the triggering.[17] Complications of A1 pulley release include digital nerve or artery injury, A2 pulley release, stiffness, continued triggering, and painful scar.[19,20] Percutaneous release is thought to have an increased risk of neurovascular injury; however, none reported to date.[17] Without the use of corticosteroid along with local anesthetic, several postprocedure months of painful digit flexion were reported in one series.[17]

29.9 Postoperative Care

Patients can be placed in a soft dressing with immediate motion exercises. In the case of open release, full use of the hand can be recommended once the sutures are removed and the patients can work on scar massage. Patients undergoing percutaneous release may begin full use of the hand 1 day after the procedure without restrictions.

29.9.1 Outcomes

With full release of the A1 pulley, no recurrence should be expected in that digit. Patients may develop symptoms in other digits of the same or contralateral hand. The recurrence rate of

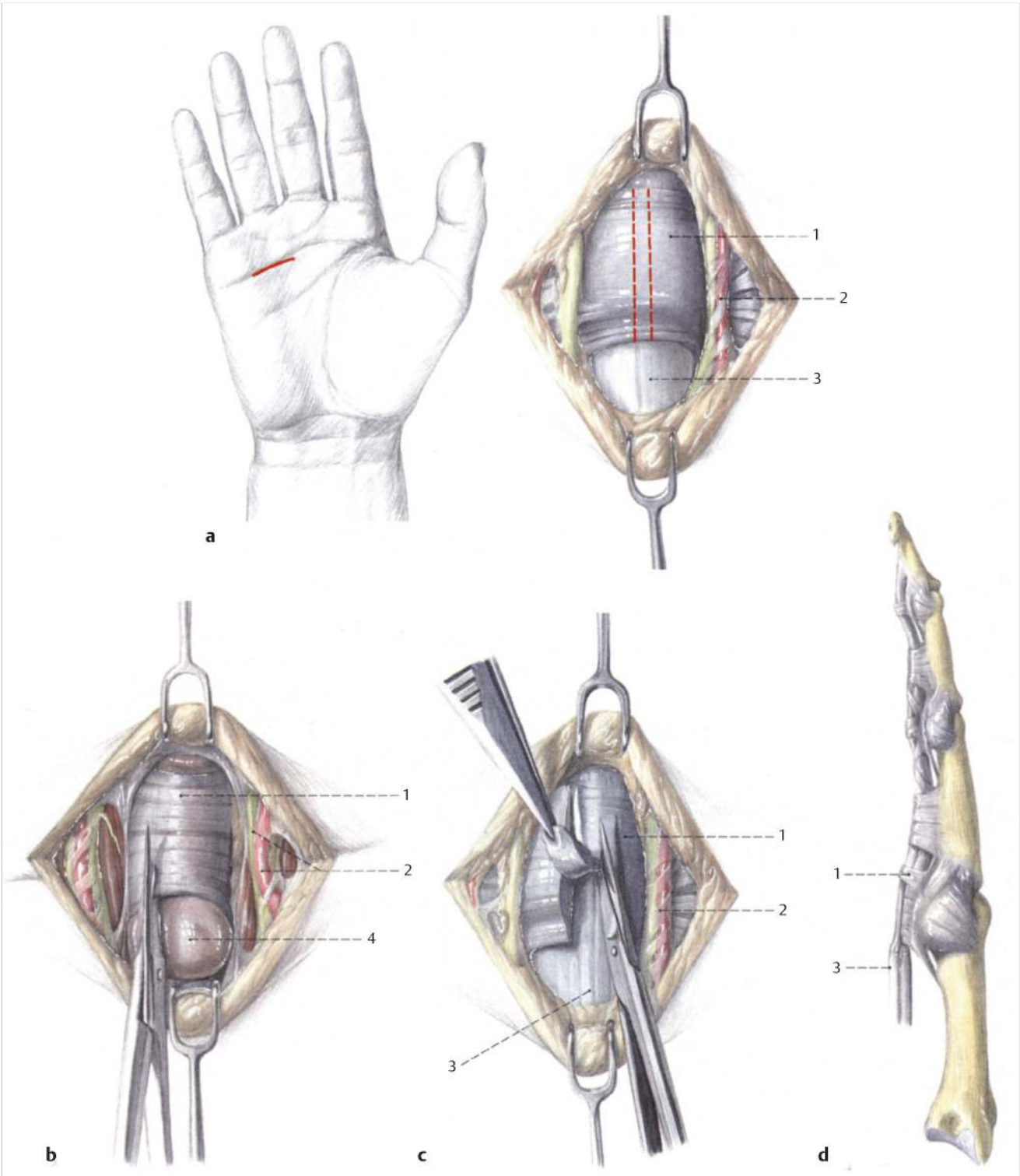


Fig. 29.6 **(a)** Complete decompression is achieved by excising a 2-mm-wide strip from the midline of the A1 pulley (*dashed line*) using microsurgical technique. **(b)** In the presence of tenosynovitis of the flexor tendon, the pulley is incised and the swollen synovial membrane resected. **(c)** Normally, excision of a strip from the midline of the pulley with microscopic scissors will be sufficient, and a synovectomy of the flexor tendon is not preformed. **(d)** If, after decompression, complete passive extension of the finger can be achieved without snapping, then an effort should be made to preserve the distal portion of the A1 pulley. The tendon should be explored with a tendon retractor prior to closure. (From Pechlaner S, Hussl H, Kerschbaumer F. Atlas of Hand Surgery. Stuttgart, Germany: Georg Thieme Verlag; 2000)

trigger finger release was 0% in a prospective study of 78 trigger digits and 0.3% in a large retrospective review of approximately 1,600 digits.[20] The rates of digits having any adverse event, which most commonly was postoperative pain or slow recovery treated with injections and therapy, respectively, have been shown to be between 5 and 28%. Percutaneous release success rate was found to be 94% by Zhao et al in a meta-analysis in 2014.[17]

29.10 Review Questions

29.10.1 Fill in the Correct Answer

1. What patient population is Dupuytren's disease most common in and what is the pathoanatomy of disease?
2. What are the treatment indications for Dupuytren's patients and what are the surgical technique options for fasciectomy?
3. What are the primary complications and recurrence rates for Dupuytren's surgery?
4. What are the recurrence rates with collagenase botulinum histolyticum use in Dupuytren's disease?
5. What patients benefit from open trigger finger release and what is the success rate of percutaneous release?

29.10.2 Answers

1. Dupuytren's disease is most common in males over the age of 40 years and of Scandinavian descent with the pathoanatomy being transition of pathologic cords from the normal bands in the palmar fascia causing contracture of the MCP and sometimes the PIP joints.
2. MCP joint contracture over 30 degrees or PIP joint contracture. Surgical techniques for open fasciectomy include primary closure, full-thickness skin grafting, and the McCash open palm technique.
3. Primary complications include wound healing (20%) and digital artery/nerve injury (3%).
4. Recurrence rates using collagenase botulinum histolyticum for Dupuytren's disease has been reported from 20 to 100% across the literature.
5. Patients with grade 3 triggering or above benefit from open trigger finger release if failing corticosteroid injections (two injection attempts) with success rates of nearly 100%. Percutaneous release results range from 48 to 94% in the literature.[17,21]

References

[1] Degreef I, De Smet L, Sciot R, Cassiman JJ, Tejpar S. Beta-catenin overexpression in Dupuytren's disease is unrelated to disease recurrence. Clin Orthop Relate Res. 467(3):838–845

[2] McCann BG, Logan A, Belcher H, Warn A, Warn RM. The presence of myofibroblasts in the dermis of patients with Dupuytren's contracture. A possible source for recurrence. J Hand Surg [Br]. 18(5):656–661

[3] Wade R, Igali L, Figus A. Skin involvement in Dupuytren's disease. J Hand Surg Eur Vol.

[4] Saldana MJ. Trigger digits: diagnosis and treatment. J Am Acad Orthop Surg. 9 (4):246–252

[5] Draviaraj KP, Chakrabarti I. Functional outcome after surgery for Dupuytren's contracture: a prospective study. J Hand Surg Am. 29(5):804–808

[6] Crean SM, Gerber RA, Le Graverand MP, Boyd DM, Cappelleri JC. The efficacy and safety of fasciectomy and fasciotomy for Dupuytren's contracture in European patients: a structured review of published studies. J Hand Surg Eur Vol. 36(5):396–407

[7] Werker PM, Pess GM, van Rijssen AL, Denkler K. Correction of contracture and recurrence rates of Dupuytren contracture following invasive treatment: the importance of clear definitions. J Hand Surg Am. 37(10):2095–2105.e7

[8] Pess GM, Pess RM, Pess RA. Results of needle aponeurotomy for Dupuytren contracture in over 1,000 fingers. J Hand Surg Am. 37(4):651–656

[9] van Rijssen AL, Werker PM. Percutaneous needle fasciotomy in Dupuytren's disease. J Hand Surg Am. 31A:498–501

[10] Reilly RM, Stern PJ, Goldfarb CA. A retrospective review of the management of Dupuytren's nodules. J Hand Surg Am. 30(5):1014–1018

[11] Peimer C, Blazar P, Coleman S, Kaplan F, Smith T, Lindau T. Dupuytren Contracture Recurrence Following Treatment With Collagenase Clostridium Hisotlyticum (CORDLESS [Collagenase Option for Reduction of Dupuytren Long-Term Evaluation of Safety Study]): 5 Year Data. J Hand Surg Am. 2015; 40(8): 1597-1605

[12] Xiaflex (collagenase clostridium histolyticum). Prescribing information. Chesterbrook, PA: Auxilium Pharmaceuticals, Inc.; October 2014

[13] Everding NG, Bishop GB, Belyea CM, Soong MC. Risk factors for complications of open trigger finger release. Hand (NY). 10(2):297–300

[14] Ryzewicz M, Wolf JM. Trigger digits: principles, management, and complications. J Hand Surg Am. 31(1):135–146

[15] Fleisch SB, Spindler KP, Lee DH. Corticosteroid injections in the treatment of trigger finger: a level I and II systematic review. J Am Acad Orthop Surg. 15 (3):166–171

[16] Freiberg A, Mulholland RS, Levine R. Nonoperative treatment of trigger fingers and thumbs. J Hand Surg Am. 1989 May;14(3):553-8

[17] Zhao JG, Kan SL, Zhao L, et al. Percutaneous first annular pulley release for trigger digits: a systemic review and meta-analysis of current evidence. J Hand Surg Am. 39(11):2192–2202

[18] Turowski GA, Zdankiewicz PD, Thomson JG. The results of surgical treatment of trigger finger. J Hand Surg Am. 22(1):145–149

[19] Will R, Lubahn J. Complications of open trigger finger release. J Hand Surg Am. 35(4):594–596

[20] Bruijnzeel H, Neuhaus V, Fostvedt S, Jupiter JB, Mudgal CS, Ring DC. Adverse events of open A1 pulley release for idiopathic trigger finger. J Hand Surg Am. 37(8):1650–1656

[21] Rodrigo JJ, Niebauer JJ, Brown RL, Doyle JR. Treatment of Dupuytren's contracture. Long-term results after fasciotomy and fascial excision. J Bone Joint Surg Am. 58(3):380–387

Part VI

Breast

VI

30 Breast Reduction

Rukmini S. Rednam and Bruce A. Mast

Abstract
This chapter surveys the anatomy of the breast and covers the matter of breast reduction surgery. The reasons—physical, emotional, reconstructive, aesthetic, etc.—why women of all ages wish to have their breast size reduced vary greatly. Surgical options are varied and the one selected should correspond to the patient's morphology, nipple placement, previous surgeries, and goals, as well as to the surgeon's comfort level, training, and experience. The five key steps common to all breast reduction techniques are listed and recommended surgical options and procedures (e.g., liposuction, Wise pattern reduction, vertical reduction, etc.) are discussed in detail. The chapter concludes with suggestions for postoperative care and an enumeration of possible complications.

Keywords: macromastia, parenchyma, liposuction, Wise pattern reduction, short scar periareolar inferior pedicle reduction, Benelli-type circumareolar reduction, superomedial pedicle with inverted T-scar pattern

30.1 Goals and Objectives

- Understand and describe the anatomy of the breast as it relates to breast reduction surgery.
- Understand the proper evaluation of a patient considering breast reduction surgery.
- Clearly define the indications for breast imaging prior to breast reduction surgery.
- Describe the incidence of invasive breast cancer in surgical specimens.
- Have an understanding of the basic techniques available to treat a patient with macromastia and which patients are best suited for a particular technique.
- Be able to identify and treat the common early and late complications associated with breast reduction surgery.

30.2 Patient Presentation

Women seeking out breast reduction surgery can vary from adolescents to the elderly. Although these patients are from different backgrounds, age groups and body shapes, they consistently present with a similar constellation of symptoms. These symptoms include upper back pain, neck pain, headaches, inframammary intertrigo, difficulty with physical activities, painful bra strap grooving, dissatisfaction with breast appearance, poor sexual well-being, emotional distress, and, though less common, ulnar paresthesias due to pressure on the brachial plexus.

Most patients present for evaluation based on referral from their individual primary care physicians. Though they are physician referred, not all women are candidates for breast reduction surgery due to their current health status, smoking status, or medical history. As such, upon initial presentation, a thorough medical history (including pregnancy, and ability and desire to breastfeed) and examination of pertinent systems is necessary. Specific attention should be given to addressing comorbidities such as diabetes, obesity, and nicotine abuse that can all impair normal wound healing. Any use of antiplatelet and anticoagulant medications should be investigated to determine the exact cause for need of the medication and its implications for the surgery. Personal or family history of breast masses, breast cancer, abnormal mammograms, nipple sensory changes, and current oral contraceptive or hormone replacement therapy should be noted. The patient's current bra size should be recorded as well as her ideal cup size.

Concerning the adolescent population, emotional distress due to peer pressure, or ridicule from others should be delved into as it can be a driving factor for desiring surgery. This can be a significant issue which can lead to stunted social development at an important time in a young woman's life and alone can justify undergoing the procedure.[1]

The physical assessment includes height, weight, and calculation of body mass index (BMI; weight in kilogram/height in meter squared). A full breast exam is performed looking specifically for (1) symmetry of the breast and nipple–areola complex, (2) tissue quality, (3) degree of ptosis, (4) presence of masses in the breasts or axilla, (5) position of inframammary fold and changes to the skin in the fold (intertrigo), and (6) presence of shoulder grooving. The manual exam should be done in supine position with the remainder of the exam in the upright position with arms placed at sides. Standard measurements should also be taken measuring the sternal notch to nipple distance and nipple to inframammary fold distance bilaterally. Many other measurements can be helpful depending on surgeon's preference (▶ Fig. 30.1).

Understanding anatomy of the breast is key for both evaluation and treatment of macromastia. Especially important is a familiarity with the blood and nerve supply to the nipple–areola complex. Sensation to the complex comes from the lateral

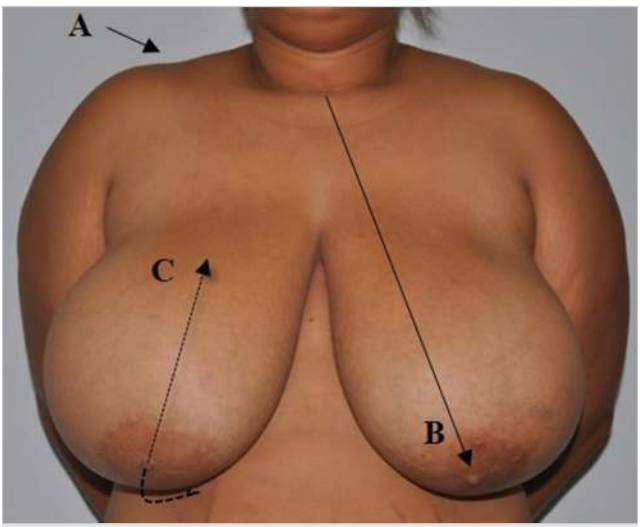


Fig. 30.1 Sample patient evaluation. A: shoulder strap grooving; B: supraclavicular notch to nipple distance; C: inframmary fold to nipple distance. C also indicates new nipple position.

and medial cutaneous branches of the intercostal nerves T3, T4, and T5. The lateral branches exit chest wall at the midaxillary line and travel medially along the pectoralis major fascia terminating in the breast skin and nipple–areola complex. Sensation to the upper breast is provided by the lower cervical plexus.[2] The blood supply is based on musculocutaneous perforators from the internal mammary, anterolateral intercostal and anteromedial intercostal, and fasciocutaneous perforators from the lateral mammary branches of the long thoracic artery (▶ Fig. 30.2). All of these vessels interdigitate and are present in all four quadrants of the nipple–areola complex forming a dense subdermal plexus. This is important as it allows many different pedicles to be created while still allowing the nipple–areola complex to thrive. Factors such as radiation, smoking, and previous surgery can alter the normal blood flow of the breast and result in necrosis if they are not taken into consideration. Coopers suspensory ligaments are the connective tissue support of the breast and course through the parenchyma to attach to the overlying dermis. These attachments can become lax due to pregnancy, significant weight gain, smoking, and aging, which results in increased breast mobility and ptosis.[3]

30.3 Preparation for Surgery

Photographic documentation of the patient's preoperative appearance is important to obtain during the preoperative consultation. The photos serve as a permanent part of the medical record for patient reference when needed, and also are required for any cases submitted through insurance. The photos should frame the patient from the neck down to just below the lowest level of the breast and from just outside each arm. All jewelry should be removed and hair should be swept back or put up. Three views are sufficient including a frontal view and two lateral views. A three-quarter photo, one with hands over the head and one exposing the inframammary fold, can be beneficial as well for documentation purposes (▶ Fig. 30.3).

Diagnostic data in preparation for surgery would be similar for other elective major procedures and would be dependent on age, presence of comorbidities, as well as requirements of the surgical center in which the procedure will be performed. If per history there is reason to suspect preoperative anemia, a hemoglobin and hematocrit are obtained. Cardiac workup is dependent on patient age, history, and the facility in which the procedure is being done. In general, if an electrocardiogram is abnormal, internal medicine or cardiology clearance is mandatory. Breast reduction surgery can safely be done in an outpatient setting; however, patients with multiple medical comorbidities or difficulty with anesthesia may require an inpatient setting.

Deep vein thrombosis screening and appropriate prophylaxis should be undertaken. Using a model such as the Caprini Risk Assessment score was found to be effective for stratifying plastic and reconstructive surgery patients for venous thromboembolism risk.[4] Regardless of risk score, all patients should have pneumatic compression devices placed. While there is no consensus at present for perioperative chemoprophylaxis for many plastic surgery procedures, operative times greater than 2 hours can increase the risk despite the type of surgery.[5] As breast reduction surgery is elective, patients who fall into the highest risk category should only be operated on with extreme caution. Data are still lacking determining the safety profile, specifically incidence of hematomas, for use of chemoprophylaxis at this time. The authors' preference is 40 mg subcutaneous unfractionated heparin in the preoperative holding area for most breast reduction patients with pneumatic compression devices placed prior to and functioning before induction in order for them to be effective.

All patients should be screened for tobacco use, including cigarettes, chew and vapors. For each cigarette, the peripheral blood supply is diminished for up to 90 minutes. The direct link between smoking and delayed wound healing represents an unfavorable shift of the risk-to-benefit ratio for breast reduction such that it should be avoided if possible.[6] Recommendations vary for smoking cessation prior to surgery from 2 to 4 weeks depending on surgeon's preference. If needed patients should be referred to smoking cessation programs to help ensure their success. If there is any question whether the patient has abstained from nicotine, a urine cotinine test may easily be obtained in the preoperative area.

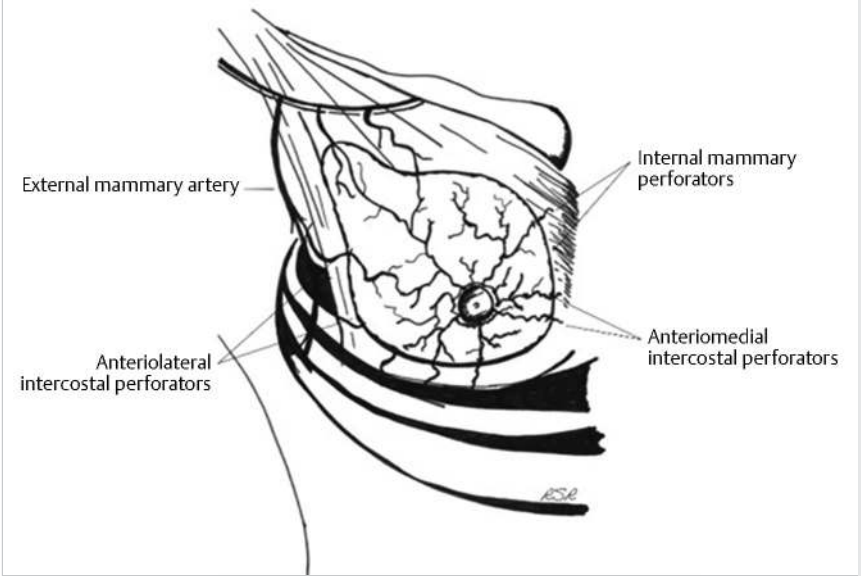


Fig. 30.2 Illustration of the blood supply to the nipple–areola complex.

Fig. 30.3 Standard preoperative views for breast reduction. **(a,b)** Lateral breast views with field including from the neck to below the breast and from just in front of the farthest projection of the breast to behind the back with the arm just slightly posterior. **(c)** Frontal view with field including from the neck to just below the breasts and extending to either side of the arms.

A common question from patients concerns insurance coverage of breast reduction surgery. Most insurance plans require a predetermination. This may simply require the plastic surgeon's office notes documenting a detailed history of the patient's symptoms and assessment of severity from the plastic surgeon, but may also require photographs, and documentation from a primary care physician describing the symptoms the patient has experienced and conservative measures taken and failed. Conservative measures generally include physical therapy, chiropractic treatment, ointments, powders, anti-inflammatory medications, and any other modifications made to treat the symptoms. An estimated weight of resection is made based on experience. Many insurance companies have adopted charts based on body surface area to estimate total resection need for coverage. These should be used as a guide, but the patient's ultimate goals should be taken into consideration if there is any conflict with the amount of tissue needed to be resected for insurance approval. This is best handled talking directly with the patient about the situation in order to move forward. Whether or not insurance is involved, an estimated breast resection volume should always be documented prior to surgery for your reference and for medical record.

It is generally agreed that the same recommendations for screening mammography should be followed for preoperative workup for breast reduction. The American Cancer Society recommends that all women over the age of 40 years obtain yearly mammograms for so long as they are in good health.[7] Among women younger than 40 years, evidence supports use of the screening mammography for only those considered high risk for breast cancer.[8] As a result of breast reduction surgery, the breast parenchyma develops scarring and possible calcifications that may be seen on future mammograms. As such a mammogram prior to surgery in the above appropriate patients is recommended to rule out abnormal findings. Patients should also be informed that a baseline mammogram will be obtained 6 months after surgery to serve as a baseline study for comparison with future mammograms.

30.4 Treatment

30.4.1 Options and Indications

Indications for breast reduction surgery are straightforward for the most part. If a woman presents with a majority of the constellation of symptoms—macromastia with upper back pain, neck pain, headaches, bra strap grooving, and recurrent intertrigo of the inframammary fold—then she should benefit from a breast reduction.

Many procedural options are available for breast reduction and should be based on the patient's morphology, nipple placement, previous surgeries, goals, and very importantly upon surgeon's comfort level, training, and expertise with certain techniques. Many different reduction techniques can yield excellent aesthetic results with symptom relief. All described procedures are based on pedicles designed to support a viable, sensate nipple while allowing resection and reshaping of the breast parenchyma. The only two techniques that are an exception to this are liposuction only and breast reduction techniques involving free nipple grafting.

No matter the technique, five key steps are present in all: (1) Design a pedicle in order to keep the nipple–areola complex viable. (2) Decide how much and from where you will excise breast parenchyma. (3) Plan your skin access incisions. (4) Shape the remaining breast parenchyma. (5) Remove any excess skin. In order to reduce intraoperative blood loss, dilute epinephrine containing solution should be injected into the breast in all areas except the designed pedicle. The safety and efficacy of using epinephrine and tumescent solution in breast reduction has previously been reported.[9]

All patients should be marked in the preoperative holding area with patient sitting completely upright or in the standing position with shoulders even and rolled back and the face looking straightforward. No matter which technique is used, key markings should be placed on the sternal midline, breast meridians, inframammary fold, and desired position for the nipple–areola complex. The nipple–areola complex position is most often marked in the breast meridian at the level of the inframammary fold. This position can be adjusted intraoperatively after completion of breast shaping, if needed. A dire pitfall is marking the nipple–areola complex too high on the breast making it visible in a demi-cup bra or bathing suit. Should this occur, fixing the problem afterward can be almost impossible and should be avoided at all costs. Ideally the nipple–areola complex should sit at the area of most prominent projection of the breast. Keep in mind for vertical procedures that it should be placed somewhat lower to accommodate for the increased projection. If any uncertainty exists for placement of the nipple–areola complex, it is better to err on placing it slightly lower (▶ Fig. 30.4).

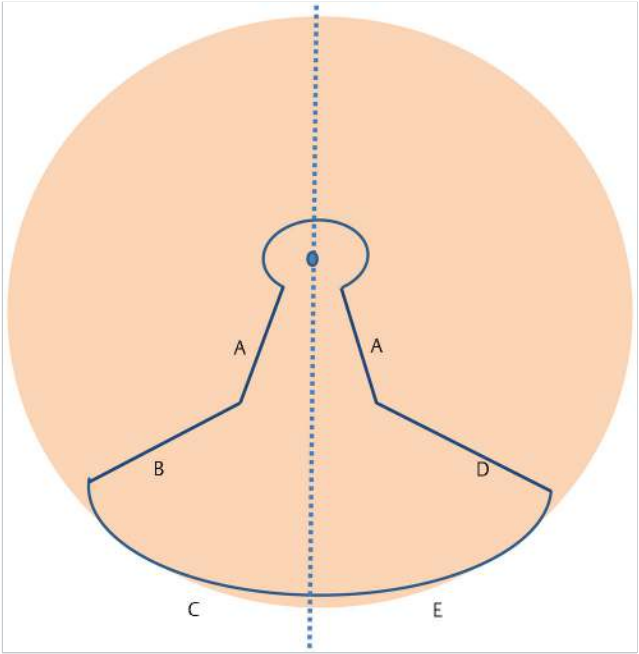


Fig. 30.4 Schematic of the Wise pattern markings, left breast. The areola inset is drawn around the nipple position which is marked at the meridian of the breast (*dashed line*) at the level of the inframammary fold while the patient is upright. The vertical limbs are identical in length typically 5 to 6 cm in length from the base of the areolar inset. The horizontally oriented lines emanating from the vertical lines (B and D) should be about the same distance of the corresponding length of incision line along the inframammary fold (C and E, respectively). This assures appropriate horizontal skin removal and proper alignment of the skin flaps. Lateral lines D and E are often longer than the medial lines due to greater excess of lateral breast tissue. The larger the breast, the greater the divergence of the vertical (A) incision lines.

30.5 Surgery Procedures

Whether done as an outpatient or inpatient, reduction mammoplasty is mainly performed under general anesthesia. Despite the use of local anesthesia, for full patient comfort general anesthesia or deep total intravenous anesthesia is preferred. The patient is placed supine on the operating room table with both arms placed on arm boards and wrapped with padding beneath the elbows. It is very important to make sure the patient is centered and her shoulders are even on the operating table. Overlooking this simple detail can lead to asymmetric results. A single intravenous dose of a first-generation cephalosporin or clindamycin (penicillin-allergic patients) should be administered prior to the incision.[10] Evidence at this time supports the use of a single dose of perioperative intravenous antibiotic. However this is not common practice currently with more than 50% of plastic surgeons in the United States prescribing more than 1 day of antibiotics for breast reduction surgery.[11]

After completion of the nipple–areola marking and delineating the pedicle, a wetting solution of 0.01% lidocaine with 1:1,000,000 epinephrine is then infiltrated into the breast parenchyma along the incision lines avoiding the parenchyma of the pedicle. Tumescent fluid is injected in a subdermal, wheel-like fashion within the pedicle to facilitate de-epithelialization of the skin overlying the pedicle. Any number of epinephrine-containing solutions can be used based on surgeon's preference.

30.5.1 Surgical Options

Multiple methods for breast reduction have been described. Varying skin resection patterns and varying vascular pedicles for the nipple complex exist, each with potential advantages and disadvantages (▶ Table 30.1). The most common methods will be reviewed, with a detailed description of the authors' favored method: extended superomedial pedicle combined with the inverted T scar pattern.

Table 30.1 Guidelines for choosing Breast Reduction Approach

Skin excision pattern and scar appearance				
Breast characteristic	**Liposuction**	**Wise-pattern**	**Vertical**	**Periareolar**
Size				
<500 g	x		x	x
500-100 g	x	x	x	
>1000 g	x	x	x	
Skin elasticity				
Normal	x	x	x	x
Inelastic		x	x	
Skin excess				
Minimal	x	x	x	x
Moderate		x	x	
Massive		x	x	
Parenchyma				
Firm and Fibrous		x	x	x
Soft and fatty	x	x	x	
Skin - Parenchyma relationship				
Firmly adherent		x		
Loosely adherent	x	x	x	x

30.5.2 General Principles

The most common patterns for breast reduction are the vertical and Wise (inverted T) patterns. With either pattern, multiple pedicle options are available. Less common but also an option is the circumareolar pattern which works for a select population but has more limited access to the breast parenchyma with limited pedicle choices. No one approach will fit all patients such that it is up to the surgeon's expertise and experience to decide which will be best suited for a patient. Breast shape is three-dimensional and when planning a breast reduction, this must always be kept in mind. Curvilinear incisions are preferred versus straight incisions in order to maintain a natural breast shape. Medial and upper breast tissue should be preserved as it is desirable in this location. The majority of resected breast tissue will come from the lower and lateral breast parenchyma. Depending on the technique, tissue may be removed from a desirable area; but as long as the breast shaping takes this into consideration and provides volumes to fill these areas the result will still be excellent. Skin plays little role in maintaining the breast shape in the long term and should be kept in mind when shaping the parenchyma.

30.5.3 Liposuction

This technique can be used in a specific population of patients to yield results. It relies on skin elasticity and retraction since no skin is resected and only fatty tissue is removed. It is good for women with mild volume excess with normal skin elasticity and mild ptosis, at most, who have a well-positioned nipple–areola complex at the apex of the breast. Tumescent fluid is injected into the breast and the inferior and lateral border of the breast are liposuctioned to reduce the fatty volume of the breast. The medial and superior poles are avoided to preserve the breast volume in those desirable locations. Liposuction only may also be the best option for a medically compromised patient, as it has a high safety index and a short operating room time compared to open techniques. This procedure can be used in those with greater degrees of ptosis or skin redundancy but will cause the breast to appear deflated with likely increased ptosis after surgery. Liposuction is not appropriate for adolescents and young adults, as their breasts are more fibrous and have little adipose tissue. But, for the rare patient who desires volume reduction for symptoms but is not worried about the skin excess or ptosis, liposuction may be a good option. Also in patients who present for secondary reduction with an unknown pedicle liposuction with skin only retailoring may be a safe and effective option.

30.5.4 The Wise Pattern Reduction

The Wise pattern refers to the inverted T scar pattern. The resultant scars are around the nipple–areola complex, vertically down to the inframammary fold and horizontally within the inframammary fold, oftentimes extending laterally onto the flank. This technique resects skin from the lateral, medial, and inferior aspects of the breast leaving the classic "anchor" scar. It is well established and can produce predictable, reproducible results in women of all breast sizes and types. The Wise pattern, combined with an inferior pedicle, is the most popular breast reduction technique performed in the United States (▶ Fig. 30.4). [12] However, multiple pedicles (medial, lateral, inferior, central, etc.) can be designed using this skin pattern. The *inferior and central pedicle* techniques create large skin flaps which are then wrapped around the designed pedicle. While the technique has the benefit of an easy learning curve and can be used on any volume of breast tissue, shape is largely determined by the skin envelope, therefore a substantial risk of bottoming out and pseudoptosis development over time due to overreliance on skin for shape. This *medial and lateral pedicle* technique creates minimal to no skin undermining and the shape is dependent on the parenchymal pillars. As such, the shape of the breast is maintained over a longer period of time.

The inferior dermoglandular pedicle is most commonly used with the Wise pattern and the nipple–areola complex obtains its blood supply from the perforators within the pedicle. Breast parenchyma is excised from the medial, superior, and lateral portions of the gland. Skin excess is removed horizontally from the lower poles and vertically from the central breast. The classic McKissock method retains a dermoglandular pedicle superior and inferior to the nipple complex. The superior pedicle is imbricated for nipple complex transposition and inset. (▶ Fig. 30.5). A major disadvantage of a true inferior pedicle is its reliance on the skin to completely hold the shape, even if some surgical shaping is done to the pedicle. However, a central/inferior pedicle can be shaped and pexied to the chest wall such that the long-term shape of the breast is less dependent on the overlying skin. This remains a good choice for women with very large breast reductions and women after massive weight loss.

The medial and superomedial pedicle technique resects tissue from the inferior poles of the breast leaving breast parenchyma medially and laterally untouched. A small portion of the lateral upper pole is resected, but the volume is replaced with rotation of the pedicle. This can be done with a vertical or the Wise pattern skin excision. This is a reproducible technique that requires almost no undermining. Unlike the inferior pedicle, medial and lateral pillars are created and the pedicle can be placed in an advantageous position for upper pole volume and shaping. This pedicle can be thinned as needed in order to rotate it into a new position while still preserving its vascularity. The medial and lateral breast pillars are approximated creating breast form and projection without reliance on the overlying skin for shaping (▶ Fig. 30.6; ▶ Fig. 30.7; ▶ Fig. 30.8; ▶ Fig. 30.9).

30.5.5 Vertical Reduction

With a vertical reduction, there is a scar around the nipple–areola complex and a vertical scar and minimal to no inframammary fold incision. The technique was initially described and popularized using a thinned superior pedicle for the nipple–areola complex, while the majority of excess tissue is removed from the inferior pole of the breast, as well as posterior to the nipple complex. However, medial, lateral, and inferior pedicles can still be designed with this technique. A medial pedicle provides for excellent upper pole volume and preserves sensation very well, though it has a higher incidence of nipple necrosis, especially in obese women.[13] Vertical reduction techniques are best suited for smaller reductions with minimal to moderate

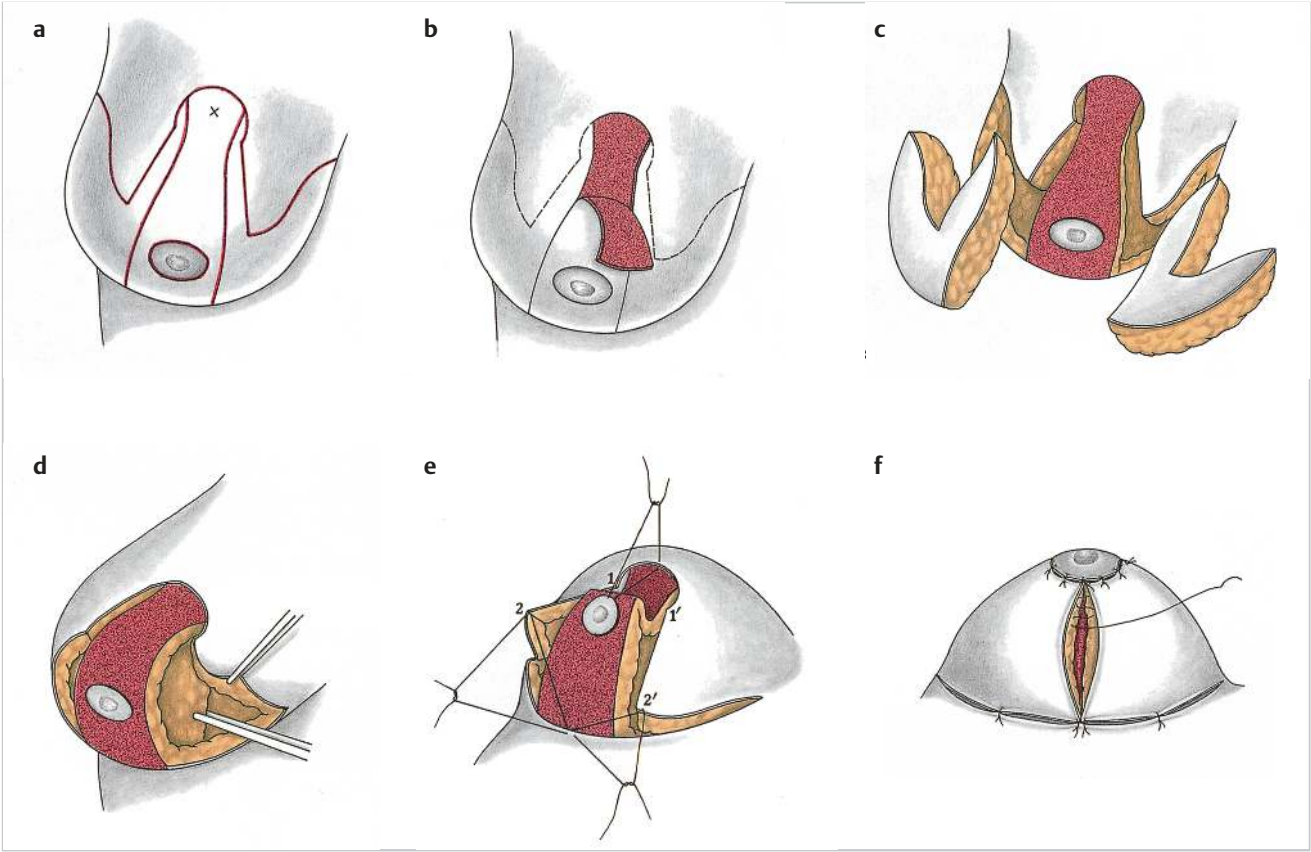


Fig. 30.5 The Mckissock reduction mammoplasty technique. (a) A vertical bipedicled dermal flap is marked. (b) De-epithelialization of the bipedicled flap. (c) After periareolar de-epithelialization of the bipedicled flap, resection of the lateral tissue blocks is performed. (d) Tissue resection behind the bipedicled flap and in the superior portion of the breast. (e,f) Transposition of the nipple on the vertical skin flap to its new site and uniting the lateral and medial portions of the breast over the inferior portion of the nipple flap. (Used with permission from Gabka CJ, Bohmert H. Plastic and Reconstructive Surgery of the Breast, 2nd ed. New York, NY: Thieme; 2009:fig. 4.11, 59.)

Fig. 30.6 Left breast demonstrating the medial aspect of the inverted T or Wise pattern markings with the patient supine and the breast lateralized naturally by its weight. The meridian line of the breast when upright is continued at the inframammary fold to orient the placement of the inverted T. Also the length of the inframammary incision line medial to the meridian is the same as the medial horizontal incision line.

Fig. 30.7 The extended superior medial pedicle is marked, extending inferomedially to preserve medial tissue for retained fullness.

Fig. 30.8 The pedicle is de-epithelialized and the excess tissue is resected, mostly inferomedially, and inferolaterally. No skin undermining is created.

skin excess, with the majority of excess skin removed via the vertical incisions. When marking the vertical limb, the line should stop anywhere from 2 to 6 cm above the inframammary fold. In women with longer nipple to inframammary fold distances (> 15–18 cm), the vertical excision is extended into the inframammary fold as a J, L, or T excision. It is important to note that the curvilinear incision will become longer than marked with vertical closure and that with healing this will cause the inframammary fold to be elevated. Stopping prior to the inframammary fold will help eliminate an unnatural central fold elevation. This technique is excellent for preserving the breast projection and shape and has the added benefit of less scar burden. It is the most commonly used short scar technique in the United States and can make the operative time less. However, the vertical reduction technique does have a steeper learning curve and the initial appearance on the operating room table can seem somewhat distorted and requires a longer time for the breast tissue to settle and reveal the final result. There is a higher revision rate recorded with this technique concerning the puckering at the inferior end of the incision, but these results may be skewed as many surgeons do not revise dog ears after a Wise pattern, though technically it is a similar scar issue.

30.5.6 Short Scar Periareolar Inferior Pedicle Reduction

With a short scar periareolar inferior pedicle reduction (SPAIR) technique, an inferior pedicle is created and excess tissue is removed similar to any other inferior pedicle technique. Excess skin is removed using a circumvertical pattern where the periareolar incision is larger than the areolar incision and the inferior end of the vertical segment courses laterally along the fold as needed. Shaping is limited to suturing the distal part of the pedicle to the upper pole to create fullness. This inferior pedicle

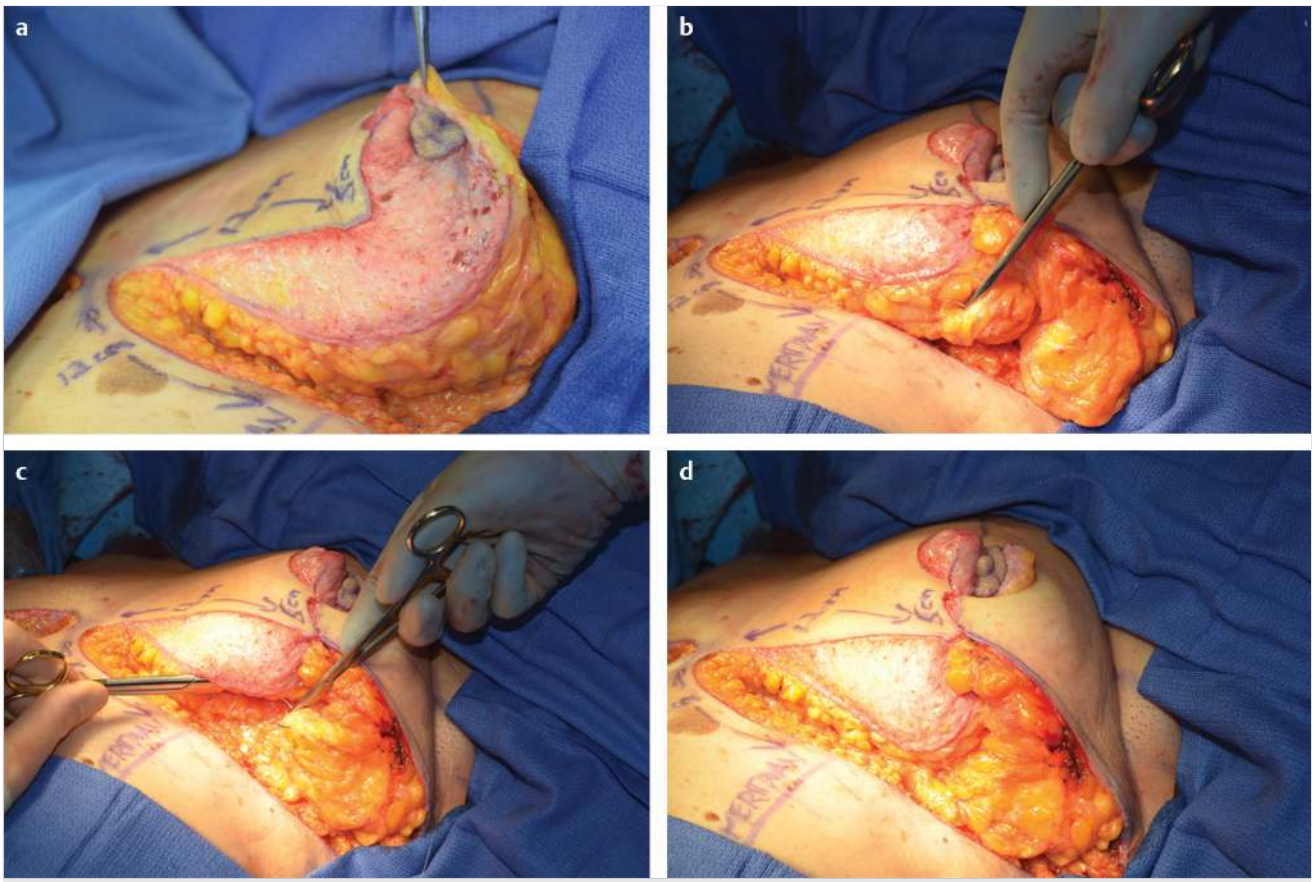


Fig. 30.9 **(a)** The pedicled nipple complex is rotated into position. **(b)** The vertical incision has been approximated and the medial and lateral breast pillars are positioned to maximize breast shape and projection. A monofilament suture is being passed through the parenchyma of the medial pillar. **(c)** The suture is being passed through the lateral pillar. **(d)** The pillars are sutured together providing structural support of the breast mound.

is a design most plastic surgeons are very familiar with and can be used on all breast volumes depending on the skin excess required. This technique does not disrupt the inframammary fold which allows a more rounded natural shape than the Wise pattern inferior pedicle and decreases the potential for bottoming out. It is, however, very technically demanding and requires accurate flap dissection with very little room for error. The skin envelope tailoring has a steep learning curve and a very large reduction with major skin excess may not provide an acceptable appearance to the periareolar scar.

30.5.7 Benelli-Type Circumareolar Reduction

With this technique, a periareolar donut skin excision is performed, a central mound is preserved and the surrounding breast tissue is excised. The final scar is only periareolar and is closed in a purse string fashion to avoid scar widening. This technique is best for mild to moderate reductions (200–300 g) with up to a 3-cm circumareolar reduction. It is not used very often in the United States due the volume limitations. For the appropriate patient, it can shape the breast nicely with minimal scarring, but it can create a flattened-appearing breast and unaesthetic scarring and pleating if used for patients with larger reductions.

30.5.8 Breast Reduction and Free Nipple Grafts

This technique of reduction may be used for very large volume reductions (> 2,000 g) and severe ptosis with long nipple to suprasternal notch distances. With extremely ptotic, large breasts, a reliable pedicle cannot always be preserved and nipple–areola complex viability cannot be assured. In these situations, a central/inferior pedicle with a Wise pattern can be designed and the nipple viability can be assessed intraoperatively. Should there be any concern for nipple ischemia, the nipple–areola complex can be removed, defatted, the dermis thinned, and placed as a free nipple graft. Another more traditional option is amputation of the lower pole of the breast for a horizontal closure and then free nipple grafting. The full-thickness graft is placed on a de-epithelialized portion of the remaining breast flap at the point of maximal projection of the breast. This is a good option for nipple preservation in heavy smokers, but with this technique you are sacrificing some of the aesthetic benefits of other techniques. But, from a safety standpoint and for nipple preservation, it is the best choice for some women. Free nipple grafts are associated with partial pigment loss, nipple projection loss, and sensory loss. The pigment loss can be permanent and will be most obvious and unaesthetic in darkly pigmented patients.

30.5.9 Detailed Description of Superomedial Pedicle with Inverted T Scar Pattern

The senior author's (BAM) favored method for most women is an extended superomedial pedicle with an inverted T scar skin pattern as depicted in ▶ Fig. 30.5, ▶ Fig. 30.6, ▶ Fig. 30.7, ▶ Fig. 30.8, ▶ Fig. 30.9. The procedure begins with skin markings similar to all reductions with the patient in the upright position (▶ Fig. 30.4). The areola inset is drawn around the nipple position which is marked at the meridian of the breast at the level of the inframammary fold while the patient is upright. The vertical limbs are identical in length from the base of the areolar inset, typically 5 to 6 cm. They diverge inferiorly to the extent necessary to remove the excess horizontal skin as judged by retracting the breast medially and laterally. Horizontally oriented incision lines emanating from the bases of vertical lines are brought out medially and laterally to meet the inframammary fold incision lines, respectively. Laterally, the incision line may extend onto the lateral chest wall if needed for contouring. The horizontal incision lines should be about the same distance of the corresponding length of incision line along the inframammary fold. This assures appropriate horizontal skin removal and proper alignment of the skin flaps.

The breast is allowed to lie neutrally on the chest wall and the pedicle is marked beginning at the 12 o'clock position of the areola inset, curving around the areola border and ending at the medial end of the IMF. The medial extension preserves soft tissue for medial fullness. The areola diameter is marked at 38 to 42 mm and infiltration with dilute epinephrine solution is done. The pedicle is de-epithelialized after which the inframammary incision is created and brought down to the muscular fascia. An incision is then made at the borders of the de-epithelialized pedicle and carried down to the chest wall without undercutting the pedicle. The lateral vertical and horizontal incision lines are made and carried straight down to the chest wall. The tissue is then removed en block starting medially and extending laterally. Superolaterally, modest tissue resection is also done to accommodate the pedicle that will be transposed. This leaves a broad superomedial pedicled nipple complex, and no undermined skin flaps.

The areolar complex is then inset superiorly after rotating into position (▶ Fig. 30.9a). The medial and lateral breast parenchymal pillars are approximated with monofilament absorbable suture in a manner that maximizes projection and shape (▶ Fig. 30.9b–d). The skin is then approximated in the standard manner.

30.6 Postoperative Care

Drains: The overall evidence supports not using drains for breast reduction surgery, though almost 50% of plastic surgeons in the United States always use drains with this procedure. Drain placement is not benign and is a cause of significant discomfort for patients. While there may be instances where a drain may be of benefit, level I evidence suggests routine use of drains provides no benefit.[14]

Dressings: This varies among plastic surgeons. While some use paper tape, others use Dermabond and still others place nothing over the incisions. It seems universally accepted that a soft surgical bra should be used for support. In general, no underwire is used for a minimum of 6 weeks after surgery and a front zip sports bra can be used for months until a regular bra can comfortably be worn without causing any pressure or redness on the breast or scars.

Pain control: Most surgeons will prescribe narcotic pain medication for at least the first week after surgery. Intercostal blocks can provide additional pain control in the postoperative period and decrease the need for narcotic medication.

Activity restrictions: Similar to other body and breast contouring procedures, the patient should gradually resume normal activity. Restrictions on heavy physical activity are generally for the first 6 weeks. For women who work in a sedentary office setting returning to work in 1 to 2 weeks is a reasonable goal. For those with more active jobs that require physical work 3 to 4 weeks is more appropriate. Though jogging and heavy exercise is limited until 6 weeks, ambulating is encouraged from day 1, and light exercise is encouraged staring at 2 to 3 weeks.

Follow-up: Patients ideally should be seen within 24 to 48 hours of surgery in order to evaluate for hematoma and any skin flap or nipple compromise. Weekly or every 2-week visits should follow the initial postoperative visit until 6 weeks. At that point, a follow-up should be scheduled for 3, 6, and 12 months in order to see the patient's final results and monitor her progress.

30.7 Outcomes

In the immediate postoperative period, the main complications encountered are hematoma, infection, wound healing issues, and nipple–areola loss. Hematoma occurs within hours after surgery but can present at up to 2 weeks after. Usually the patient complains of unilateral pain, tightness, swelling, discoloration, and excessive bloody output from either a drain or from the incisions. Hematoma may be difficult to detect early in its formation due to the normal postoperative swelling of the breast and newly created dead space combined with breast distensibility. A significant hematoma should be drained in the operating room at the time of diagnosis so that any ongoing bleeding can be controlled and all blood clots can be evacuated. Leaving a large hematoma without associated skin or nipple compromise to resolve on its own is not recommended. Such action (or lack thereof) can lead to spontaneous drainage of liquefied hematoma, large seroma, open wound, abscess, fibrosis, undesirable scarring, and breast shape irregularity.

Deep infections are rare, but cellulitis can occur and present with erythema, fevers, increased pain, and drainage. Small localized infections are not uncommon and most often present at the T point on a Wise pattern reduction. Oral antibiotics with local wound care will resolve the majority of these infections. Rarely an incision and drainage is required.

Wound healing issues and skin flap necrosis can occur for a number of reasons and can be very problematic. Areas of wound healing impairment are often due to excess tension on either the breast parenchyma or skin in the absence of infection. Again this is most common at the inverted T point on a Wise pattern reduction. Skin flap necrosis can be due to over dissection leaving thin flaps, excessive tension due to tight closure, pressure necrosis due to dressings or hematoma, or secondary to nicotine

use. Complication data have revealed that increasing resection weight also correlates with increased risk of complications. Delayed wound healing is directly linked with increased resection weight and inversely with increasing age.[8] For small areas of skin necrosis, local wound care will generally aid in healing but may result in a wider scar due to secondary healing. Often antibiotic ointment with dry dressings is sufficient. Larger areas may require operative debridement and closure.

It has been well demonstrated that there is a direct link between a decrease in safety and increase in BMI. Kerrigan and Slezak analyzed the American Board of Plastic surgery MOC data on breast reductions and found that weight of resection was a significant predictor of adverse events and increased complications were present with increasing weight of resection. They also found that patients with class I, II, and III obesity also had a significant higher occurrence of adverse events.[8] As such a patient with a BMI of 30 or less would have the lowest risk of additional adverse events. However, for some women this is difficult as one of the reasons they are unable to exercise and lose weight is due to their large breast size. Therefore, BMI should be assessed within the context of the individual patient and considered as part of the risk–benefit analysis.

Nipple–areola complex loss is one of the most serious complications that can occur and can be devastating to the patient. It is often accompanied by underlying fat necrosis of the distal end of the pedicle due to insufficient blood supply. This complication should occur rarely if proper patient and procedure selection is done.[15] If after complete closure the nipple–areola complex looks compromised, all surrounding sutures should be released to see if blood flow returns. If however, after the effects of epinephrine have passed and the nipple–areola complex continues to look ischemic, then a conversion to a free nipple graft would be advised to preserve an acceptable result. Total loss of the nipple–areola complex requires reconstruction at least 6 months after surgery and is not ideal.

Fat necrosis usually occurs at distal most edge of the pedicle due to fat without adequate blood supply. It will often resolve with time and massage over a week to months after surgery. Rarely surgical intervention is indicated. It is more likely to occur if too much tension is placed on the parenchymal pillars with closure.

Late complications include untreated seromas, poor scars, shape changes, fat necrosis and asymmetry, poor nipple positioning, and nipple sensory changes.

Small seromas can be managed early on with percutaneous drainage or for many no intervention is needed and the body will resorb the fluid. However, larger seromas can cause wound separation and delayed wound healing. A seroma that continues to reoccur despite multiple aspirations may have to be excised directly in order to remove the cavity and close the space.

Scars can be a difficult issue to treat, as they can be unpredictable and difficult to determine how each person will scar individually. Often poor scarring is due to a combination of undue tension on the skin and genetics. Early hypertrophic or keloid-appearing scars can be treated with intralesional steroid injection. If, however, by 6 months to 1 year if there has been no improvement, surgical scar revision may be undertaken with an improvement in the scar appearance as the breast tissue would have stabilized in its new position by that time. If the puckers seen with vertical reduction techniques do not resolve, then correction may be needed to remove additional subcutaneous tissue in order to flatten the scar.[16] More commonly dog ears on the medial and lateral aspect of a Wise pattern reduction are a cause of irritation for patients and can be removed but will make the incision slightly longer, which the patient should be made aware of before proceeding.

Postoperatively, it is expected that the skin envelope will stretch due to gravity and the weight of the breast. The inframammary fold attachments which are divided with a Wise pattern reduction can allow the breast to migrate lower leading to bottoming out or pseudoptosis. If that occurs, any revisions to the shape must take into account the original pedicle in order to avoid devascularizing the nipple–areola complex and the fold should be re-created in the desired position. The vertical reduction appears to maintain its shape for a longer period of time and may require less revision in some cases concerning breast shape versus the Wise pattern.[16]

Asymmetry can occur with any technique and is much more likely when the patient originally presents with asymmetric breasts. It is important that careful detail goes into preoperative markings to try to avoid this complication. Asymmetry of the nipple is generally less acceptable than minor asymmetry of the breast as a whole. Significant asymmetry is unacceptable and can be managed with further parenchymal resection or liposuction at least 6 months after surgery.

The lifetime risk for a woman to develop breast cancer is one in eight or 12.5%.[7] In some large series, an incidental finding of cancer in a breast reduction specimen was found to be less than 0.5 to 0.8%.[17,18] Though this is a rare finding, prior to surgery the possibility should be discussed with the patient. A positive finding should then be approached as a new cancer finding with a workup, surgical, and medical oncologic consultation and a treatment plan based on the recommendations. Should a suspicious mass be found during reduction surgery, an intraoperative frozen section should be obtained for diagnosis. If the specimen is found to be benign, surgery can proceed as planned. However, if it is malignant or equivocal, the mass should be resected, the biopsy site marked with clips, and the surgical site should be closed when a good stopping point is reached. If a surgical oncologist is available, the resection should be preferably done by them; however, if no one is available in a reasonable amount of time, for safety reasons the resection can be done by the plastic surgeon. As with an incidental finding in a resected specimen, a formal workup and oncologic consultation should then take place.[4]

Patient satisfaction with the appearance and effectiveness of breast reduction surgery is very high. A 30-year review of breast reduction literature found patient satisfaction with end results to be extremely high (> 90%).[19,20,21] In general, patients are happy to have undergone the procedure and have significant improvement and satisfaction with their breast appearance, as well as psychosocial, sexual, and physical well-being based on ratings using the BREAST-Q questionnaire.[22] In a study done by Carty et al, dissatisfaction correlated with patient's age younger than 40 years and postoperative soft-tissue necrosis, with surgeon's experience not found to correlate with satisfaction.[21] Though patients may present due to their symptoms which are a result of their macromastia, after surgery they will rarely concentrate on the improvement of these symptoms but will focus on the aesthetic appearance of their

breasts. Breast reduction surgery, while functional, is also very much an aesthetic procedure and should be approached as such when planning for it.

Breast reduction surgery can be very rewarding for both the patient and the surgeon with extremely high satisfaction rates. Understanding the surgical anatomy and the techniques available allows plastic surgeons to make well informed and educated choices applying their knowledge to achieve the best aesthetic result for their patients. Understanding early and late complications along with key factors for success help provide patients with high level of safety and excellent results.

30.8 Review Questions

30.8.1 True or False

1. Mammograms should be routinely performed prior to breast reduction surgery in all age groups.
2. The skin is the most important structure holding the shape of the breast after reduction.
3. The incidence of invasive breast cancer in reduction pathology specimens is approximately 3%.

30.8.2 Choose the Best Answer

4. Sensation to the nipple–areolar complex is best described as
   a) The medial cutaneous branch of the intercostal nerve to T5.
   b) The medial and lateral cutaneous branches of the intercostal nerves T3, T4, T5.
   c) The medial and lateral cutaneous branches of the intercostal nerve T4.
   d) The lateral cutaneous branch of the intercostal nerve to T3, T4.
5. A 16-year-old girl comes to the office with her mother because she is unhappy about the size of her breasts. She wears a 34 DDD bra and has been this size for the past 2 years. She feels her breasts are too big and would like surgery to make her breasts smaller. She is uncomfortable participating in sports and does not want to socialize with her friends because she is embarrassed by her large breast size. Her exam shows large symmetric fibrous breasts without any palpable masses. Her body mass index is 25. The best treatment option for her:
   a) Physical therapy and exercise/dieting.
   b) Breast reduction surgery.
   c) Breast reduction using liposuction only.
   d) Counseling.

30.8.3 Answers

1. False.
2. False.
3. False.
4. b. The medial and lateral cutaneous branches of the intercostal nerves T3, T4, T5.
5. c. Breast reduction using liposuction only.

References

[1] Hammond D-C, Loffredo M. Breast reduction. Plast Reconstr Surg. 129(5): 829e–839e

[2] Schlenz I, Kuzbari R, Gruber H, Holle J. The sensitivity of the nipple-areola complex: an anatomic study. Plast Reconstr Surg. 105(3):905–909

[3] Rinker B, Veneracion M, Walsh C-P. Breast ptosis: causes and cure. Ann Plast Surg. 64(5):579–584

[4] Pannucci CJ, Bailey SH, Dreszer G, et al. Validation of the Caprini risk assessment model in plastic and reconstructive surgery patients. J Am Coll Surg. 212(1):105–112

[5] Nahai FR, Nahai F. FRMOC-PS(SM) CME article: breast reduction. Plast Reconstr Surg. 121(1, Suppl):1–13

[6] Cunningham B-L, Gear A-J-L, Kerrigan CL, Collins ED. Analysis of breast reduction complications derived from the BRAVO study. Plast Reconstr Surg. 115 (6):1597–1604

[7] American Cancer Society. American Cancer Society guidelines for the early detection of cancer: Breast Cancer. Available at: http://www.cancer.org/healthy/findcancerearly/cancerscreeningguidelines/american-cancer-society-guidelines-for-the-early-detection-of-cancer. Accessed August 22, 2015

[8] Kerrigan C-L, Slezak S-S. Evidence-based medicine: reduction mammaplasty. Plast Reconstr Surg. 132(6):1670–1683

[9] Armour A-D, Rotenberg B-W, Brown M-H. A comparison of two methods of infiltration in breast reduction surgery. Plast Reconstr Surg. 108(2):343–347

[10] American Society of Health-System Pharmacists. Clinical Practice Guidelines for antimicrobial Prophylaxis in Surgery; 2015:582–667. Available at: www.ashp.org/surgical-guidelines. Accessed August 18, 2015

[11] Swanson J-A, Schmitz D, Chung K-C. How to practice evidence-based medicine. Plast Reconstr Surg. 126(1):286–294

[12] Rohrich R-J, Gosman A-A, Brown S-A, Tonadapu P, Foster B. Current preferences for breast reduction techniques: a survey of board-certified plastic surgeons 2002. Plast Reconstr Surg. 114(7):1724–1733, discussion 1734–1736

[13] Hall-Findlay E-J. Breast reduction: the medial pedicle technique. In: Nahai F. ed. The Art of Aesthetic Surgery: Principle and Techniques, 2nd ed. St. Louis: Quality Medical Publishing, Inc.; 2011: 2444–2482

[14] Corion L-U, Smeulders M-J, van Zuijlen P-P, van der Horst CM. Draining after breast reduction: a randomised controlled inter-patient study. J Plast Reconstr Aesthet Surg. 62(7):865–868

[15] Manahan MA, Buretta KJ, Chang D, Mithani SK, Mallalieu J, Shermak MA. An outcomes analysis of 2142 breast reduction procedures. Ann Plast Surg. 74 (3):289–292

[16] Hall-Findlay E-J, Nahai F. Alternative approaches to breast reduction. In: Nahai F, ed. The Art of Aesthetic Surgery: Principles and Techniques, 2nd ed. St. Louis: Quality Medical Publishing, Inc.; 2011:2532–2596

[17] Smith M-L, Evans G-R, Gürlek A, et al. Reduction mammaplasty: its role in breast conservation surgery for early-stage breast cancer. Ann Plast Surg. 41 (3):234–239

[18] Spear S-L, Pelletiere C-V, Wolfe A-J, Tsangaris TN, Pennanen MF. Experience with reduction mammaplasty combined with breast conservation therapy in the treatment of breast cancer. Plast Reconstr Surg. 111(3):1102–1109

[19] Singh K-A, Losken A. Additional benefits of reduction mammoplasty: a systemic review of the literature. Plast Reconstr Surg. 129(3):562–570

[20] Scott GR, Carson CL, Borah GL. Maximizing outcomes in breast reduction surgery: a review of 518 consecutive patients. Plast Reconstr Surg. 116(6):1633–1639, discussion 1640–1641

[21] Carty MJ, Duclos A, Gu X, Elele N, Orgill D. Patient satisfaction and surgeon experience: a follow-up to the reduction mammaplasty learning curve study. Eplasty. 12:e22

[22] Coriddi M, Nadeau M, Taghizadeh M, Taylor A. Analysis of satisfaction and well-being following breast reduction using a validated survey instrument: the BREAST-Q. Plast Reconstr Surg. 132(2):285–290

31 Mastopexy

Francesco M. Egro, Edward H. Davidson, and Kenneth C. Shestak

Abstract

This chapter surveys the types of mastopexy and augmentation-mastopexy procedures. Ptosis refers to the descent of the breast due to various factors (aging, weight loss, pregnancies, etc.) and mastopexy refers to the treatments meant to correct ptosis. Patients are evaluated according to Regnault's classification of ptosis and surgical treatment is determined. The authors provide detailed information in the text and table format regarding the desired outcome for each degree (minor, moderate, major) of ptosis. The advantages and disadvantages of mastopexy techniques (crescent, concentric circumareolar, vertical, etc.) are thoroughly discussed. Augmentation-mastopexy augmentation techniques are also covered. The chapter concludes with guidelines for postoperative care and a listing of anticipated outcomes and possible complications.

Keywords: mastopexy, ptosis, Regnult's classification, augmentation

31.1 Goals and Objectives

- Understand the proper evaluation of patients with breast ptosis.
- Clearly define the indications for the various types of mastopexy and augmentation–mastopexy procedures.
- Appreciate the technical aspects of skin and parenchymal management in mastopexy and augmentation–mastopexy procedures.
- Know the advantages and disadvantages of each technique and their complications.
- Understand the postoperative management of mastopexy and augmentation–mastopexy.

31.2 Patient Presentation

The breast is an important organ to a woman, and it remains the central focus of femininity and the source of attraction. It has been depicted throughout centuries as the source of sensuality and perfection. Nature shapes the breasts in each individual, but the shape often changes due to aging, lifestyle, injury, or congenital deformity. In Greek, *ptosis* means "falling" and describes the descent of breast parenchyma, which is managed by *mastopexy*. In Greek, *mastos* means "breast," *pexy* means "fixation," and it involves a series of principles and techniques to correct breast ptosis. It is essential for the plastic surgeon to know the hallmarks of the youthful breast, and identify those techniques that are most likely to restore the breast to its optimal aesthetic form in a given patient. This chapter presents a comprehensive review of the evolution and principles of the management of the ptotic breast by means of mastopexy or augmentation–mastopexy.

According to the American Society of Plastic Surgeons (ASPS) statistics report, more than 92,000 mastopexies were performed in 2014 (75% increase from 2000), and was ranked the seventh most common aesthetic surgical procedure in the United States.[1] A similar trend is reflected in the American Society for Aesthetic Plastic Surgeons (ASAPS) national statistical data, which indicate that mastopexy was ranked the sixth most common aesthetic surgical procedure. The majority of patients were between the ages of 35 and 50 years, and returned to work within 1 to 2 weeks postoperatively.[2]

Geographical variation of the published mastopexy techniques exists. It appears that North American surgeons use the inverted T incision and inferior pedicle more frequently, use fascial sutures less often, and usually do not incorporate "auto" augmentation in their procedures. Conversely, Latin American and European surgeons prefer the vertical incision and superior pedicles, and use fascial sutures and "auto" augmentation more often.[3]

31.2.1 Etiology

Ptosis results when breast parenchymal volume is relatively deficient within the larger skin envelope. Factors found to have a statistically significant correlation with ptosis include advanced age, history of significant weight loss, high body mass index (BMI), large bra cup size, greater number of pregnancies, and smoking history.[4]

With age, the breast assumes a lower position on the chest wall becoming ptotic and losing the youthful breast contour. Factors that may play a role include glandular hormonal regression, atrophy, gravity, and loss of skin elasticity. This leads to stretching and lengthening of skin and glandular attachments. As glandular tissue settles, the upper pole of the breast loses its fullness and appears deflated.[4] Breast ptosis is a very common in post-bariatric patients who have undergone massive weight loss. Studies have shown that weight loss is an issue if greater than 50 lb is lost.[4] High BMI and large bra cup size (breast weight > 400 g) have been shown to be predisposing factors, most likely because of the increased pull caused by gravity on heavier breasts.[4] Although pregnancy per se was not found to predispose to breast ptosis, an increase in the number of pregnancies does. The reason is uncertain, but weight gain during pregnancy was not found to be associated with ptosis.[4] Smoking has been shown in experimental and clinical studies to impair the production of collagen and increase the production of tropoelastin and matrix metalloproteinases. These cause an abnormal deposition of elastotic material and degradation of collagen, elastic fibers, and proteoglycans, leading to dermal connective tissue degenerative changes, wrinkling, and loss of skin elasticity and tone.[5] Breast-feeding is widely held accountable for breast ptosis and has been shown to be a reason for early termination of breast-feeding.[6] This is a significant misconception, as there is no evidence to support this hypothesis.[4] Another misconception is the role of regular upper body exercise to reduce the risk of ptosis. No evidence exists to support this argument.[4] Other factors mentioned in the literature as predisposing factors for ptosis include congenital deformities such as tubular breasts, thin skin, excessive breast size, and postoperative changes.

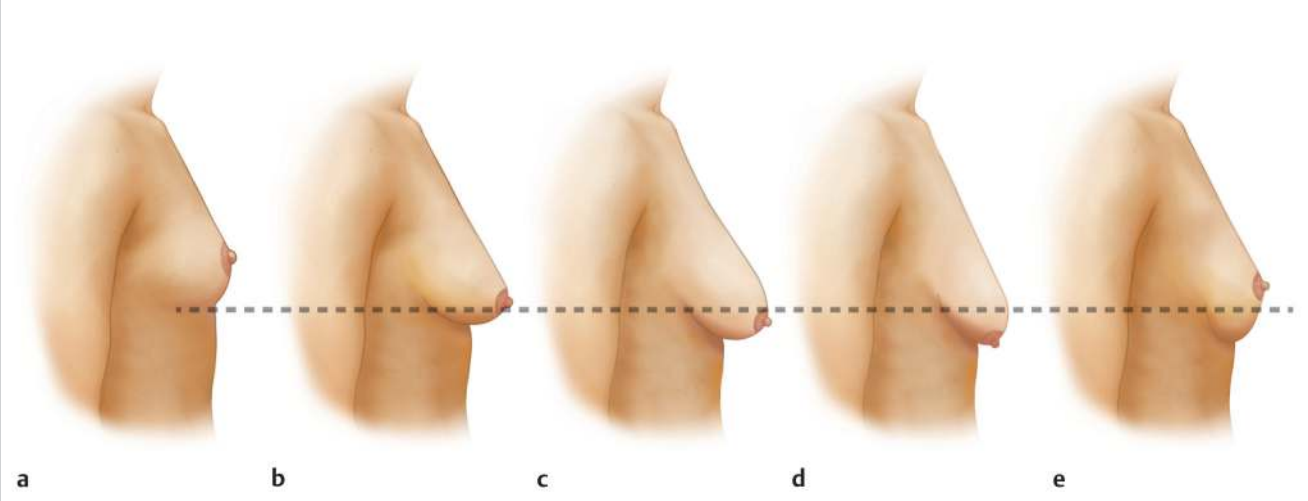


Fig. 31.1 Breast classification. (**a**) Normal. (**b**) Minor or first degree. (**c**) Moderate or second degree. (**d**) Major or third degree. (**e**) Partial or glandular ptosis. (Adapted from Jones GE. Bostwick's Plastic and Reconstructive Breast Surgery, Third Edition. New York: Thieme Medical Publishers; 2009)

31.2.2 Classification of Ptosis

The youthful breast is positioned between the third and sixth intercostal spaces, with the inframammary fold located at the level of the fourth to fifth intercostal space, with a full rounded lower pole contour, a lateral outward convexity that covers the rib cage as it sweeps toward the axilla becoming concave, and a nipple–areola complex (NAC) positioned directly at the point of maximal projection. Several classifications have been introduced to define the degree of ptosis; however, Regnault's classification remains the most widely employed.[7] The degree of ptosis is based on the position of the nipple in relation to the inframammary fold and the gland. Breast ptosis is classified as pseudoptosis, glandular or "partial" ptosis, and three degrees of true ptosis (▶ Fig. 31.1; ▶ Table 31.1).[7]

The terms "pseudoptosis" and "glandular ptosis" to this day are often used incorrectly interchangeably. "Pseudoptosis" describes a breast that is hypoplastic, with a loose skin envelope and the nipple lying above the inframammary fold; this combination of factors results in an appearance similar to the one of minor ptosis, and can be resolved by increasing breast volume (e.g., inserting an implant). "Glandular ptosis" was referred by Regnault as "partial ptosis," and also presents with the nipple above the inframammary fold, but the gland and skin brassiere follow the influence of gravity; this results in the lower portion of the skin brassiere to be elongated, while the upper portion to remain the same length.[7]

Table 31.1 Regnault's classification of ptosis.

Pseudoptosis	The nipple lies above the inframammary fold, but the breast is hypoplastic leading to a loose skin brassiere and this gives the impression of a minor ptosis
Partial ptosis (glandular ptosis)	The gland and skin brassiere follow the influence of gravity, while the nipple remains above the inframammary fold because the upper portion of the skin brassiere has not changed, whereas the lower one has elongated
Minor ptosis (first degree)	The nipple lies at the level of the inframammary fold, and above the lower contour of the breast and skin brassiere
Moderate ptosis (second degree)	The nipple lies below the inframammary fold, but is above the lower contour of the breast and skin brassiere
Major ptosis (third degree)	The nipple lies below the inframammary fold, and at the lower contour of the breast and skin brassiere

Based on Regnault P. Breast ptosis: Definition and treatment. Clin Plast Surg. 1976; 3(2):193–203

31.2.3 Patient Evaluation

A thorough history and physical examination is the basis for a successful surgical strategy. Evaluation of the patient's preoperative medical status must include conditions that can affect the shape and size of the breast (such as breast disease, significant weight gain followed by weight loss, previous breast surgery or biopsies); family history of breast cancer; details of recent mammograms; reproductive history including number of pregnancies, breast-feeding history, and plans for future children.

Examination requires a careful and systematic evaluation of each breast, and includes a detailed visual analysis and thorough palpation. The visual inspection is performed to evaluate the shape, symmetry, contours, skin quality, scars, breast volume, along with the NAC shape, position relative to the inframammary fold, and amount of breast overhanging the inframammary fold. Careful notes are made regarding symmetry of contour, NAC position, presence of striae, and overall skin quality. Additionally, the presence, size, and location of contour deformities are recorded and pointed out to the patient preoperatively. Surface measurements from key breast anatomic points noted above are recorded in the patient's chart. This includes a bio-dimensional analysis of topographic landmarks including supra-sternal notch to nipple distance, breast base width, nipple to inframammary fold distance at rest and on stretch and pinch thickness of the upper and lower poles of the breast.

Fig. 31.2 The maneuver of pinching the lower pole of the breast allows the surgeon to judge the adequacy of breast volume and the ability of this volume to produce upper pole fullness when transposed. If an inadequate shape results, the patient may not be a suitable candidate for mastopexy alone, that is, it may help predict the need for an implant.

Tactile examination includes careful palpation of the skin and breast parenchyma, looking for masses, tenderness, areas of thickening, scar adherence, glandular mobility, along with parenchymal and skin elasticity. The parenchyma is also assessed for volume, position, distribution, and elasticity. Pinching the lower pole or lateral inferior pole of the breast mound can simulate the superior transposition of the parenchyma. The ability to simulate a favorable upper pole shape change this way suggests that the patient is a good candidate for vertical mastopexy (▶ Fig. 31.2). On the other hand, the lack of breast shape change with this maneuver suggests that the patient is more likely to benefit from a mastopexy with a combination of vertical and horizontal skin excision, or a mastopexy, which includes the placement of an implant.

Each patient presents with preconceived ideas of an ideal breast appearance. Sometimes they already have a shape in mind and may attempt to explain or show it by adjusting the contour of their breast with their hands. For this reason, it is vitally important for the plastic surgeon to understand the patient's primary concerns and goals. The patient's expectations must be realistic and she must understand that the surgery will result in permanent scars on the breasts. Once the breasts have been accurately assessed and classified, an honest discussion is undertaken with the patient regarding the various elements of her deformity, the recommended treatment, limitations and complications of operative intervention, and appropriate expectations regarding outcome.

31.3 Preparation for Surgery

Preoperative radiological or laboratory evaluation in preparation for surgery is similar to other major elective procedures and would be dependent on the patient's age and comorbidities. Patients who are of menstruating age must have a urine HCG. Blood work is not routine but directed by significant medical conditions.

Preoperative mammogram is routinely ordered by the senior author (KCS) in patients aged 40 years and over, and in patients aged 30 years with a family history of breast cancer or previous breast tissue diagnosis. The mammogram is used as a baseline study for future mammograms.

Anteroposterior, lateral, and oblique photographs are taken in the upright position in a uniform way, using the same background color, lighting conditions, and patient position. If certain areas of the breasts are of particular interest, other views are taken including photographs from above, below, or in the supine position.

Table 31.2 Guideline to the surgical approach of ptotic breasts

Minor ptosis (first degree)	Augmentation alone. Periareolar mastopexy with or without augmentation depending on whether patient desires larger breasts
Moderate ptosis (second degree)	Vertical mastopexy with or without augmentation depending on whether patient desires larger breasts
Major ptosis (third degree)	Vertical or inverted T mastopexy with or without augmentation depending on whether patient desires larger breasts

31.4 Treatment

Mastopexy entails breast reshaping and varying degrees of nipple elevation in the context of using the most appropriate incisions. Many algorithms have been introduced to guide surgeons on the best approach according to the degree of ptosis.[7,8,9,10] However, no ideal technique has been identified and the shortest scar procedure may not necessarily be the most appropriate in all patients. Each patient requires an individually tailored surgical plan as the surgeon needs to consider the incision pattern, need for tissue rearrangement, parenchymal fixation, or implant placement as dictated by his/her analysis described above. There are three basic incision patterns, with several variations, and the most commonly used techniques are described below.

The degree of ptosis is one of the factors to guide the choice of the surgical approach to be used. Additionally, the patient's preference, aesthetic breast evaluation, careful parenchymal and skin elasticity assessment, and the surgeon's experience and level of comfort all influence the surgical choice. The trend is toward using minimal incision patterns but ultimate selection of incisional technique is best guided by the Regnault classification. ▶ Table 31.2 provides a guideline to the surgical approach of ptotic breasts.

Important technical maneuvers for all mastopexy procedures done without an implant begin with skin marking. This is done with the patient in the standing position. Most importantly these include establishing new nipple position, which will correspond to the newly created maximal projecting portion of the breast as created by movement of breast tissue to the upper pole. As such the new position for the nipple will most often be above the inframammary fold, especially if an implant will be used concomitantly. This may be anywhere from 1 to 4 cm

Fig. 31.3 In mastopexy, volume is always added to the upper pole of the breast. For that reason, presumptive planning always includes planning nipple position between 1 and 3 cm (sometimes as much as 4 cm) above the IMF.

Fig. 31.4 The surgeon can often estimate accurately the shape change and amount of skin to be excised preoperatively with gentle digital manipulation of the breast tissue.

above the inframammary fold in the upright position (▶ Fig. 31.3). In addition, determining the approximate skin resection area (in the periareolar, vertical, and horizontal orientations if necessary) can often be fairly accurately estimated preoperatively by gentle digital manipulation of the tissues (▶ Fig. 31.4).

The plan and operative technique must include

- Flexibility—Preoperative planning is important but it is presumptive and not definitive; the surgeon must develop the ability to intraoperatively alter incision design and nipple position using tailor-tacking.
- Tailor-tacking—skin sutures or skin stapling to simulate changes in breast shape which occur with skin tightening or plication (▶ Fig. 31.5).
- Parenchymal suturing—performed in almost every case. This includes pedicle fixation by "pexy sutures" to pectoralis major muscle fascia, suturing de-epithelialized dermal wings, and lower breast pillar sutures in case of vertical mastopexy (▶ Fig. 31.6).

The insertion of an implant has various effects on the shape of the breast and NAC including increase in upper pole fullness and breast projection, widening of areola, and change in upper pole contour from linear (preoperatively) to slightly outwardly convex (postoperatively).[11] However, it must be kept in mind that the breast mound is only minimally elevated (less than 1 cm), the lower pole expands and may stretch slightly, and although the actual NAC position is not changed, the changes produced by the implant produces an illusion that it is elevated.[11] Therefore, augmentation alone should be reserved for selected patients where the main complaint is a deflated breast with inadequate volume and minimal inferior displacement of the NAC. Spear advocates augmentation alone if less than 2 cm of breast overhangs the fold, the nipple is appropriately positioned on the anterior surface of the breast, nonpigmented skin is visible between the lower areola and the inferior border of the breast, and the nipple to inframammary fold distance is less than 9 cm.[12] In some of these patients, a periareolar excision of skin (periareolar mastopexy) can elevate the NAC 1 to 2 cm and may enhance the result.

A combined augmentation and mastopexy approach to treat deflated and ptotic breasts can either be performed as a one-stage or two-stage procedure. The one-stage procedure was first described in the 1960s by Gonzalez-Ulloa.[13] Since then multiple plastic surgeons have shared their outcomes and opinions on a one-stage procedure, leading to a controversial debate on its reliability and relative safety.[14,15,16,17,18] One-stage procedures are challenging because augmentation and mastopexy work against each other, producing diametrically opposed effects on the breast tissue and skin envelope, at times potentially altering the blood supply to the NAC and skin flaps, and always placing greater tension on the closure. Mastopexy repositions the nipple, reshapes the breast with some element of tension in the line of closure with a reduction of the redundant skin envelope. On the other hand, augmentation expands the breast, increases the size of the skin envelope, and increases the

Fig. 31.5 Skin resection can be most accurately finalized intraoperatively using "tailor-tacking" maneuver done with sutures or skin staples with the patient sitting upright at 90 degrees on the operating table.

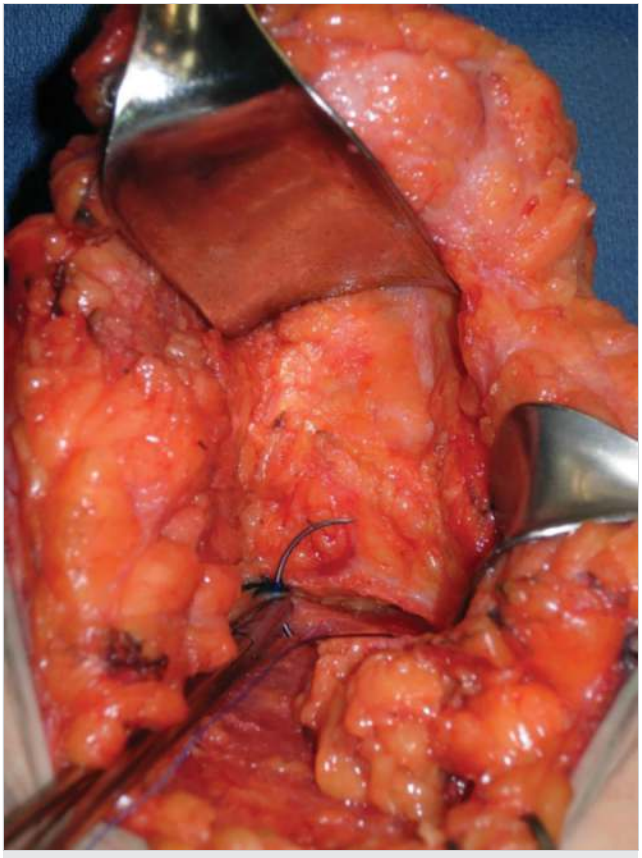


Fig. 31.6 Suture fixation of the breast parenchyma to the underlying pectoralis major muscle (PMM) fascia may be helpful in some mastopexy techniques to stabilize the position of the parenchyma at the time of surgery.

tension of wound closure. Furthermore, augmentation introduces potential implant-related complications such as capsular contracture, potential lower pole stretch, and possible inferior or lateral implant malposition. These possible untoward sequelae must be explained to the patient preoperatively.

Patient selection is key for a successful one-stage augmentation–mastopexy and the decision is based on the patient's preoperative breast evaluation, tissue condition, and suitability to the procedure along with the surgeon's experience and level of comfort with this plan. Favorable characteristics include first and second degree ptosis; using a small implant (less than 300 mL); nipple elevation less than 4 cm; light toned skin with good elasticity; and absence of peri-operative risks (e.g., obesity, smoking history, and massive weight loss). The main indication of a one-stage augmentation–mastopexy includes ptosis combined with significant volume deficiency. Depending on the degree of ptosis, a periareolar, vertical, or inverted T technique can be used. The choice of implant size is an important part of planning regardless of the chosen technique, and depends on the preoperative characteristics of the breast. The larger the implant, the less aggressive the skin excision is. Deflated pendulous breasts require an implant whose diameter matches the base diameter of the breast, and often ptotic breast tissue in the lower pole must be excised in the so called plus/minus mastopexy. Constricted or tuberous breasts almost always require implants with a diameter that exceeds the original breast base width. For this reason, mastopexy must be very conservative in these patients. No evidence exists on the specific association of complication rates and the implant type or pocket used in one-stage augmentation–mastopexy. However, most studies indicate a higher use of silicone implants (up to 75% of cases) placed in a dual submuscular plane (up to 96% of cases).[14,15,16]

The senior author and most experienced surgeons believe that the smallest implant that will provide volume meeting the patient's expectations should be used. It is generally felt by most authors that insertion of the implant is done prior to mastopexy because increased volume within the breast conferred by the implant results in less ptosis and correspondingly less skin that needs to be excised.[19]

However, some patients may present a very high risk for a one-stage augmentation–mastopexy, especially in patients displaying the following unfavorable characteristics: extreme third-degree ptosis; heavy breasts with more than 4 cm of tissue overhanging the inframammary fold; required implant volume greater than 400 mL; nipple elevation greater than 6 cm; poor skin quality or pronounced striae; patients with BMI greater than 35; massive weight loss; extreme asymmetry; medially displaced NACs; and perioperative risks such as smoking history.[20] It is the senior author's (KCS) opinion that patients considered for a for a two-stage augmentation–mastopexy first undergo a mastopexy and subsequently have an implant placed, if still desired, 4 to 6 months later. Other than the safety benefit, a two-stage augmentation–mastopexy allows the patient to evaluate the appearance of her breasts after mastopexy. If satisfied with the result, the patient may opt to forgo breast augmentation.

Important technical pearls in augmentation–mastopexy:

- Using the smallest implant that will satisfy patient expectations.
- Placing the implant first usually through an incision that will be incorporated in the mastopexy plan.
- Planning for conservative skin resection and on-table adjustment with tailor-tacking with the patient sitting at 90 degrees.
- Nipple elevation and final nipple position guided by presumptive preoperative planning and tailor-tacking to be repositioned 2–5 cm (most commonly 3–4 cm) above the inframammary fold, since the implant adds significant fullness to the upper pole of the breast.

31.4.1 Techniques in Mastopexy

A variety of mastopexy techniques have been described. This section provides an overview of the three basic incision patterns (periareolar, vertical, and inverted T) and the techniques used to manage the underlying breast parenchyma.

Periareolar Mastopexy

Crescent Mastopexy

Indications

Rarely indicated and it is mainly used to address upper areola level asymmetry.

Technique

Crescent mastopexy was originally described by Puckett as an eccentric circumareolar skin excision without NAC mobilization nor purse-string suture.[21] A crescent-shaped incision is made around the upper half of the areola, down to the reticular dermis. A wedge of de-epithelialized skin is excised to mobilize the NAC up to 2 cm but most commonly much less. The incision can be as extensive as needed (it can almost resemble a circumareolar shape) depending on the degree of breast ptosis, and the NAC's diameter and circumference. The orientation of the incision can help reshape or reposition the NAC as needed including superior lifting, medial or lateral shifting, inferior repositioning, reduction, or a combination of the above.

Advantages and Disadvantages

The main advantage includes the very short scar that can be camouflaged by the border of the areola. The main disadvantage of this technique is that the force applied to the areola along the vertical axis, can result in an oval-shaped or elongated areola, which frequently results in a suboptimal aesthetic result.

Concentric Circumareolar Mastopexy

Indications

First- and early second-degree ptosis, large areolar diameter, and tuberous breasts.

Technique

Benelli introduced the "round block" technique in 1990.[22] The technique begins by marking an outer ring that defines the outer border of the de-epithelialization area. This ring joins the superior and inferior borders of the new NAC, and varies depending on the amount of excess skin. The new areolar circumference (approximately 4 cm in diameter depending on breast size) is then drawn using an areolar sizer, which defines the inner border of the de-epithelialization area (▶ Fig. 31.7). The redundant skin found between these two markings is excised to reposition the NAC. The skin is then undermined to expose the parenchyma, which is then incised leaving the NAC on a superior pedicle. Depending on the degree of ptosis, the management of the parenchyma differs. For mild degree of ptosis the base of the breast is simply plicated and invaginated. Whereas for more severe ptosis, medial and lateral parenchymal flaps are created and overlapped to provide breast width narrowing, and breast shape lifting and coning. The periareolar incision is then closed in a purse-string fashion with non-absorbable sutures, which allows a reduction in scar tension as described by Benelli.[22] An excellent variation of this technique is described by Goes, which includes the use of a prosthetic mesh layer to support the breast parenchyma.[23] or the use of implants to minimize the skin resection required.[9,17] It is important that the surgeon not excise too much skin as the quality of the aesthetic result depends on breast parenchymal re-shaping with subsequent skin redraping.

Advantages and Disadvantages

The circumareolar mastopexy is more versatile than the crescentic approach and generally provides better aesthetic outcomes. The circumareolar scar can be camouflaged at the border of the areola. The main disadvantages include flattening of the anterior contour of the breast mound and recurrence of ptosis.[8,10,22,24] Benelli notes that this circumareolar technique is not suitable for all breasts and that shape correction must be sacrificed in favor of a reduced scar.[22] It must be emphasized that the learning curve with this procedure is steep.

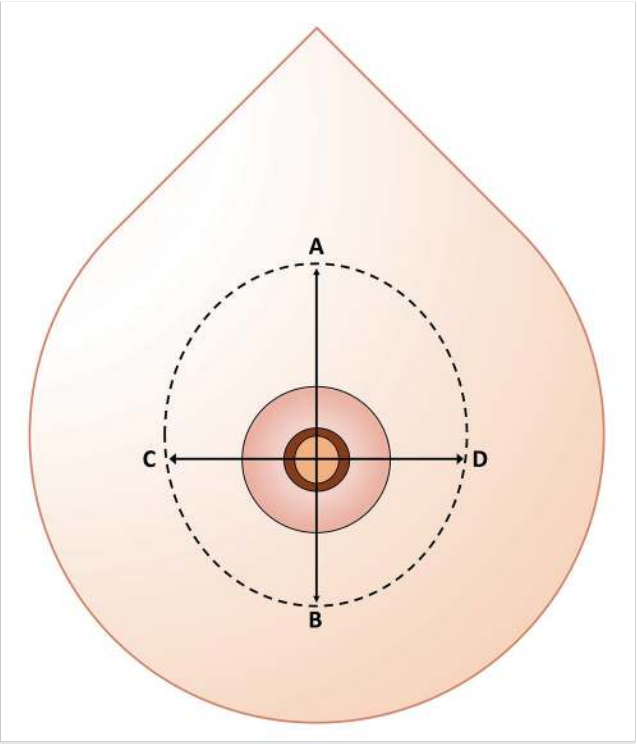


Fig. 31.7 Markings for the Benelli's "round block" technique. An outer ring (dotted line) defines the outer border of the de-epithelialization area, and joins the superior (point A) and inferior borders (point B) of the new NAC. The new areolar circumference (solid line) is drawn using an areolar sizer.

Vertical Mastopexy

Vertical Mastopexy without Undermining (Lassus)

Indications

Moderate (second degree) and early major (third degree) ptosis, but the scar burden may not be justified in mild cases.

Technique

The technique begins by marking the position of the NAC superior border as the intersection point between the vertical axis of the breast and a horizontal line 2 cm below the level of the midpoint between the acromion and the olecranon. The lower end of the vertical incision is marked on the vertical axis of the breast at a variable distance above the inframammary fold depending on the degree of ptosis (▶ Fig. 31.8a). The estimated resection markings are then drawn by gently pushing the breast medially and laterally, and joining the two points described above (▶ Fig. 31.8b). The markings are incised superiorly to create a de-epithelialized superior pedicle, then laterally and inferiorly down to pectoralis fascia to elevate a glandular flap. This may be folded under or more commonly imbricated in the upper pole of the breast by "pexy" sutures anchoring the deep surface of the breast parenchyma to pectoralis fascia, which is helpful for creating increased upper pole fullness at the time of surgery (▶ Fig. 31.6). An inferocentral wedge of skin can be excised from the breast. This tissue may also be folded under the NAC to increase NAC projection in the central breast. The medial and lateral pillars are then drawn together and most often fixed with pillar sutures without further undermining, and reshaped appropriately using tailor-tacking sutures (▶ Fig. 31.5). As the width of the distal superior pedicle increases inferior to the NAC, a relatively greater amount of

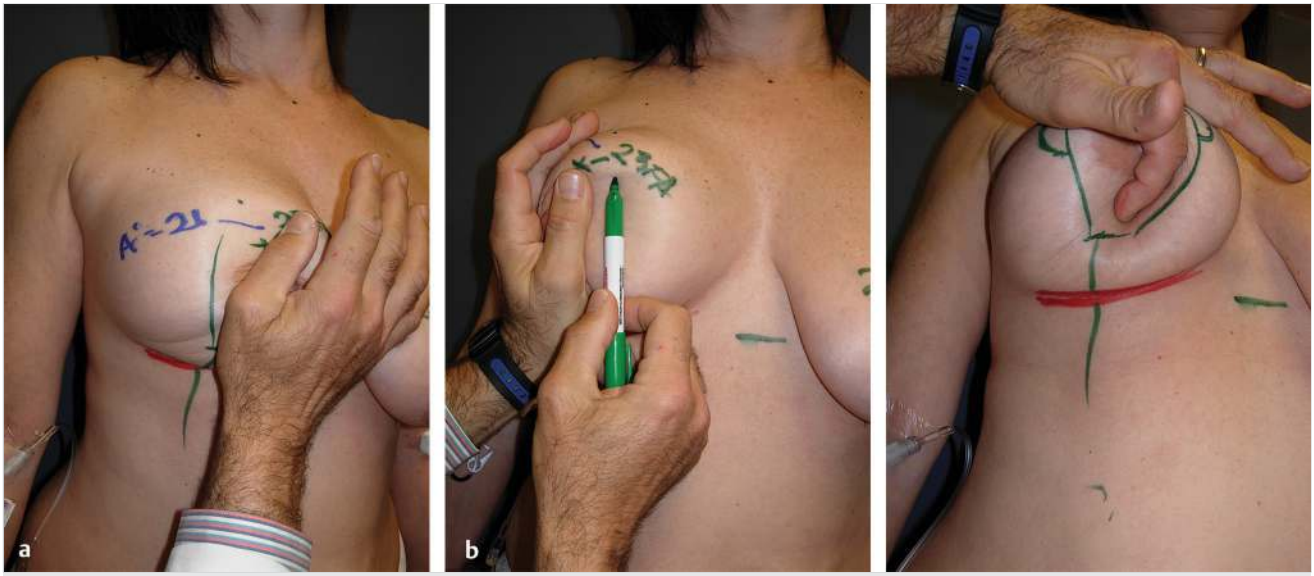


Fig. 31.8 (**a**) Medial breast displacement for marking lateral vertical incision and pillar creation connecting mid-clavicular point with mid breast mark at IM fold. (**b**) Lateral breast displacement for creation of medial vertical incision and medial pillar creation. (**c**) Inferior extent of lateral and medial pillars terminating 4 cm above IMF.

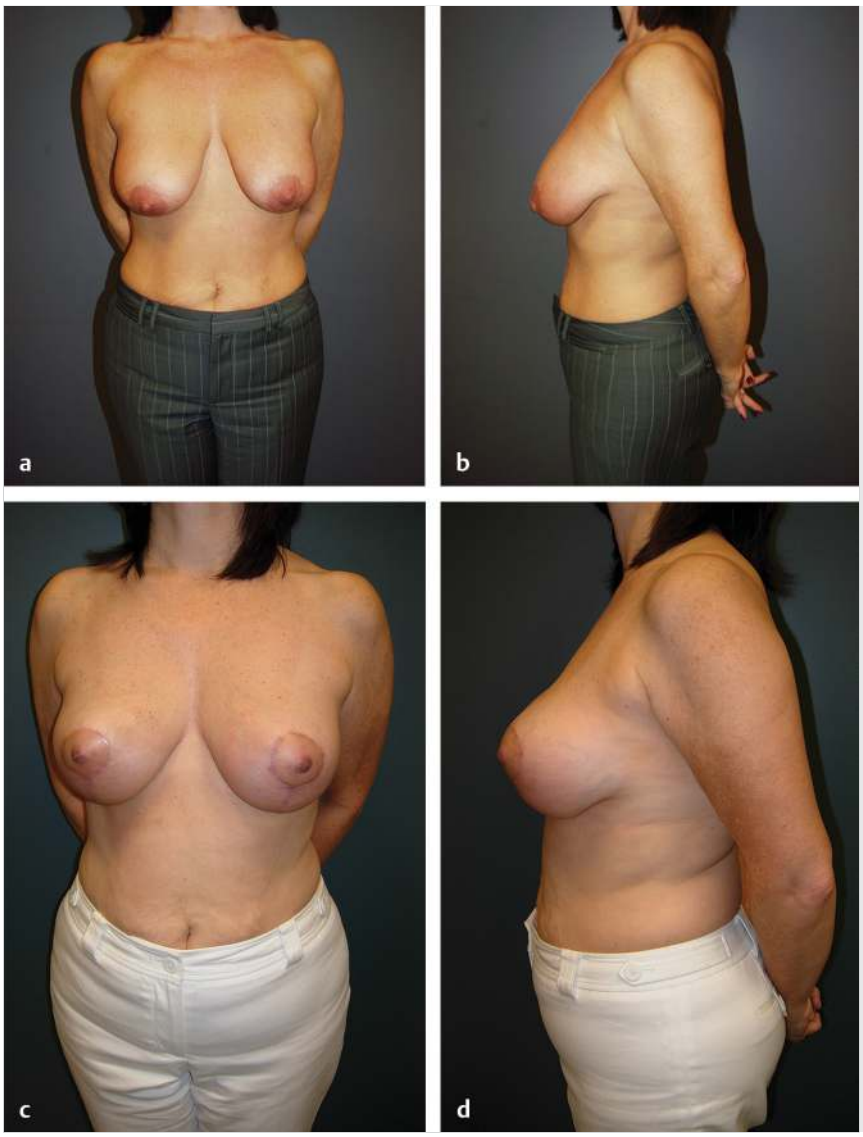


Fig. 31.9 Pre-operative (**a**) AP and (**b**) lateral view of patient desiring a mastopexy. The plan is for a superior-dermo glandular pedicle. Post-operative (**c**) AP and (**d**) lateral views at 6 months post-op.

tissue will be removed from the central lower pole and transferred to the upper pole of the breast. This may result in a somewhat flat shape of the lower breast pole that will regain some convexity as the breast pedicle (which is not fixed to the chest) descends over 2 to 3 months following surgery most often evolving a satisfactory appearance.[25,26] Liposuction is helpful in the lower pole of the breast to adjust the final shape by removal of fat and some breast tissue and in stimulating skin re-draping and mild skin contracture.

Advantages and Disadvantages

The technique is one of breast parenchymal reshaping with solid fixation in the lower pole and provides a reduced scar burden. The disadvantages include a steep learning curve and the need for slight overcorrection on the table. The combination of pronounced immediate postoperative upper pole fullness and "bunched up" lower pole, leads to less aesthetic immediate outcome. Most often this settles over time, but occasionally revision surgery is necessary to optimize scar appearance and contour in the lower pole. In the properly selected patient it often provides a better aesthetic result compared to the inverted T technique in the long term (▶ Fig. 31.9, ▶ Fig. 31.10).[27]

Vertical Mastopexy with Undermining (Lejour)

Indications

All degrees of ptosis, but the scar burden may not be justified in mild cases.

Technique

The technique is similar to the Lassus technique and begins by marking a mosque dome incision pattern and the superior pedicle as already described (▶ Fig. 31.11). Care should be taken not to over resect skin or breast tissue in the lower pole to

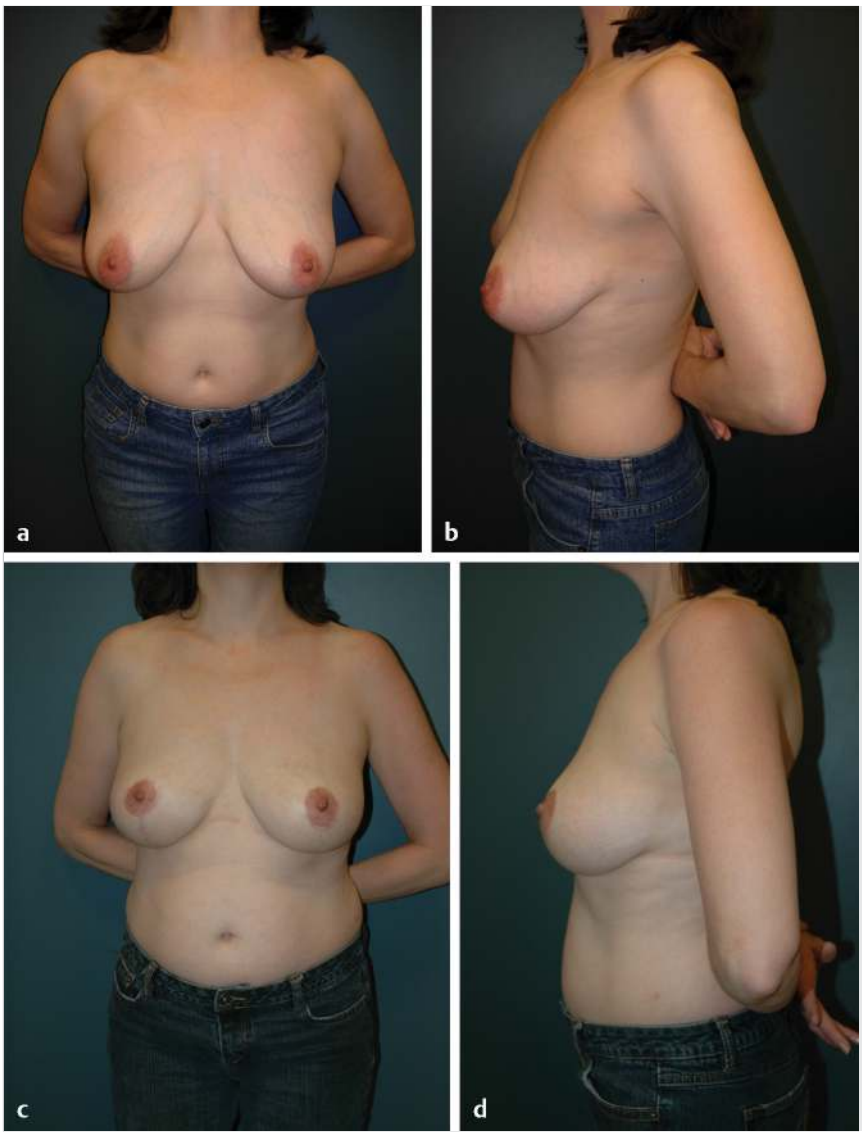


Fig. 31.10 (a,b) Preoperative breast appearance of a patient with second-degree ptosis who underwent Lassus no pillar undermining modification of the superior dermo-glandular vertical mastopexy using "pexy" sutures. **(c,d)** Outcome at 3 years following surgery with good correction of her ptosis.

minimize problems with wound healing and poor scars. The desired areolar circumference is marked around the original nipple, with the superior pedicle (at least 6–8 cm in width) extending from the upper border and continued in a conical shape down around the circumference. The pedicle skin surrounding the areola is de-epithelialized to a point 2 to 3 cm below the areola. Incisions are made along the lateral breast markings, and there is both pillar and skin undermining which is performed medially, laterally, and inferiorly to the level of the inframammary fold. This is one of the technical variations that distinguish the Lejour from the Lassus technique in which there is no undermining of the pillars. The dermoglandular breast pedicle is then moved superiorly as a whole unit and sutured to the superior part of the retro-mammary dissection. This creates medial and lateral pillars of breast tissue that are sutured together. The elevation of the areola may cause tension, which can be improved by cutting the dermis 1 or 2 cm at each site. Lejour also described the use of liposuction and resection of breast parenchyma, but these techniques are mainly utilized in reduction mammaplasty rather than mastopexy.[28,29]

Advantages and Disadvantages

The advantages and disadvantages of the Lejour vertical approach are similar to those outlined for the Lassus technique.

Short Scar Periareolar Inferior Pedicle Reduction Mammaplasty (Hammond)

Indications

All degrees of ptosis, particularly in patients with associated concavity in the upper pole of the breast. The scar burden may not be justified in mild cases.

Technique

The technique begins by marking the top of the periareolar pattern and the new areola position, found on the breast meridian at the level of typically between 3 and 5 cm above the inframammary fold. Then the inferior pedicle is drawn, with an 8 cm wide base, and lines measuring 8 to 10 cm up extending perpendicularly from the inframammary fold. The medial and

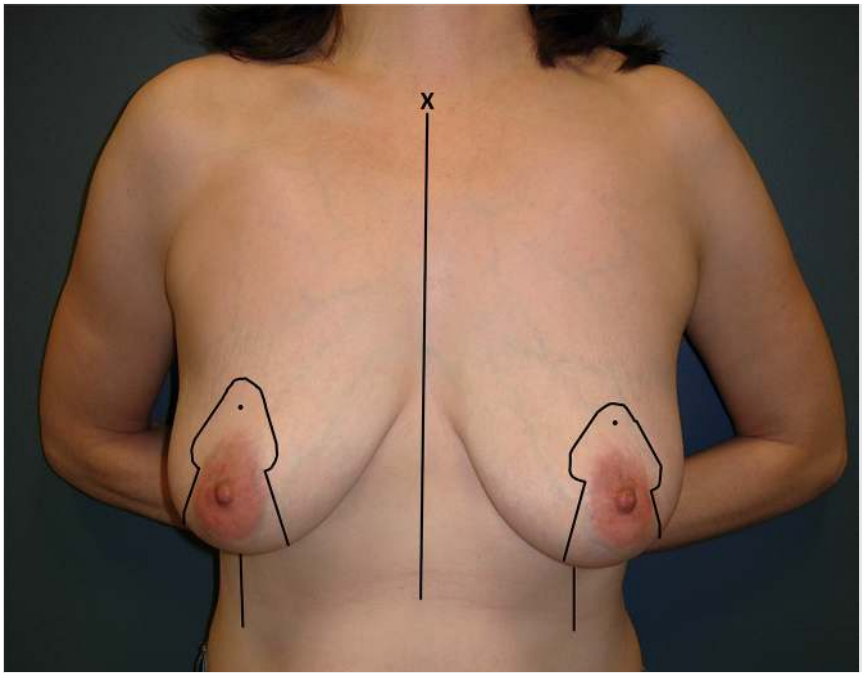


Fig. 31.11 Markings for the Lejour vertical mastopexy with undermining.

lateral borders of the periareolar pattern are then drawn, by displacing the breast medially and laterally in relation to its vertical axis. Lastly the new nipple is drawn measuring 40 mm in diameter. The operation is started by de-epithelializing the inferior pedicle and a 5 mm rim of periareolar skin. Thin medial, superior and lateral skin flaps are created. The former two gradually become thicker (3–6 cm) as the chest wall is approached, and are undermined for 2 to 3 cm along the pectoralis fascia. The deep leading edge of the superior flap is plicated to pectoralis fascia to restore upper pole fullness. The medial flap is plicated onto itself, whereas the inferior pedicle is resuspended to the pectoralis fascia to secure an elevated position. The redundant inferior skin envelope is tailor-tacked to create the desired contour, de-epithelialized, and closed in a vertical pattern using a CV-3 Gore-Tex interlocking periareolar purse-string cinched down to the desired areolar size.[30]

Advantages and Disadvantages

The advantages include a smaller scar compared with the inverted T techniques. Overcorrection of the breast on the table and time for it to settle are no longer needed, as the aesthetic immediate outcome is excellent and the shape of the breast does not appear to significantly change over time. The disadvantages include a very steep learning curve and the highest complication rates of any of the techniques described thus far.[31]

Simplified Vertical Mastopexy (Hall-Findlay)

Indications

All degrees of ptosis, but the scar burden may not be justified in mild cases.

Technique

In 1999, Hall-Findlay introduced modifications to the standard Lejour vertical technique. These include the use of medial (or rarely a lateral) pedicle; minimal or no liposuction; no pectoralis fascia sutures; and no skin undermining. The technique begins with markings that are similar to the inverted T or Wise pattern, except for the medial and lateral limb extensions; instead the limbs are curved downward to meet each other 2 to 4 cm above the inframammary fold (▶ Fig. 31.12). The new nipple is placed on the anterior aspect of the breast at or just above the level of the inframammary fold, with a NAC diameter between 4 and 5 cm. A mosque dome incision pattern similar to that described by Lejour is outlined. Then the medial pedicle is drawn with a base of 6 to 8 cm, and a 1 cm periareolar rim. The pedicle is de-epithelialized and incised down to the chest wall, without undermining. The lateral and superior-lateral skin flaps are beveled, but the inferior skin flap is kept thin. The lower end of the areolar skin is closed and the medial pedicle is rotated up in position. The medially based glandular flap is mobilized, rotated medially, and it provides upper pole fullness. The inferior aspect of the pedicle is sutured to the lateral pillar. Lastly the deep dermis is sutured and the skin is gathered along the vertical closure to help shorten scar length.[32,33]

Advantages and Disadvantages

The advantages include a small vertical scar, the additional support by the inferior parenchymal closure, and faster operative time compared to inverted T technique due to the shorter incision length resulting in quicker time for wound closure. The lack of undermining reduces healing problems. Lastly, the avoidance of pectoralis fascia sutures makes the inset of the pedicle easier. The disadvantages include a steep learning curve, the need for overcorrection on the table, and less aesthetic immediate outcome, which settles over time. The aesthetic concerns are mainly related to the exaggerated upper pole fullness, breast shape, and gathering and bunching of the skin.[32] Fur-

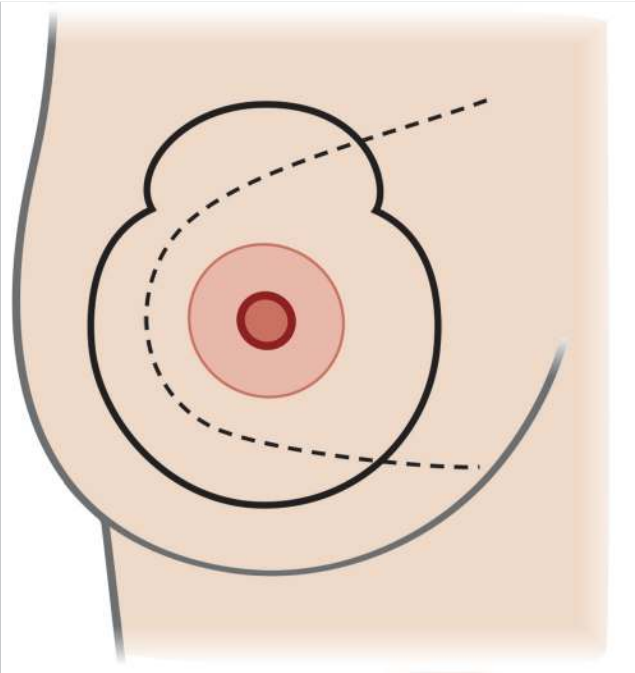


Fig. 31.12 Markings for the Hall-Findlay simplified vertical mastopexy.

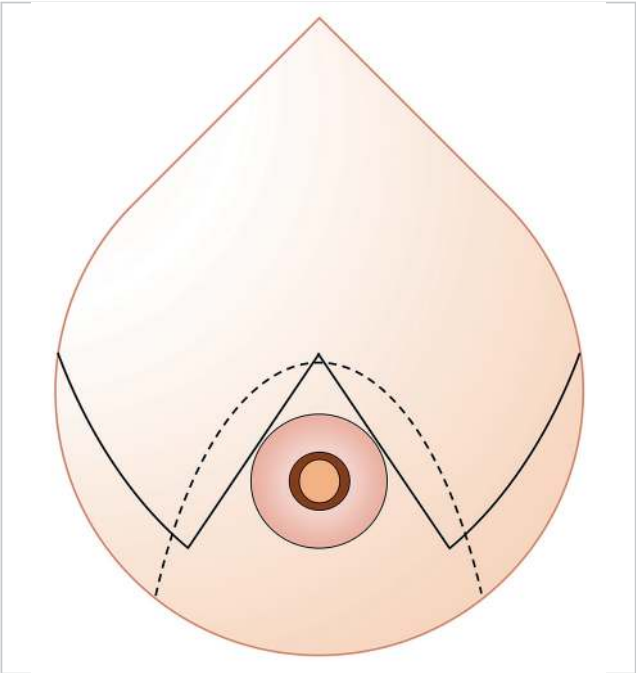


Fig. 31.13 Markings for the inverted T mastopexy.

thermore, a national survey by Rohrich et al has showed that this technique has the second highest complication rate (exceeded only by the SPAIR technique), most commonly including suture spitting, asymmetry, and high revision rate.[31]

Inverted T Mastopexy

Indications

All degrees of ptosis, but the scar burden may be most justified in more severe cases. This includes in patients with severe skin excess (typically seen in massive weight loss patients), poor skin quality, fatty parenchyma, and/or where the NAC requires more than 6 cm of elevation.

Technique

The inverted T or Wise pattern[34] is historically the most commonly used technique in mastopexy, and is the one that all other techniques are compared to in terms of aesthetic outcomes and complications. The technique begins by marking an inverted "V" pattern, in which the limbs of the "V" are located on the anterior aspect of the breast at the level of the inframammary fold. Two limbs measuring 5 to 7 cm are extended inferiorly, skirting the medial and lateral margins of the areola. Medial and lateral extensions are then drawn toward the inframammary fold at a variable angle dependent on the amount of skin needing excision. Lastly, a line running through the inframammary fold joins these two limbs (▶ Fig. 31.13). Once the skin pattern is designed, the surgeon has to elect what to do with the breast parenchyma. The traditional inverted T technique dictates that the breast shape is solely maintained by the skin envelope. This entails de-epithelialization of the marked area, elevation of the skin flaps, excision of the redundant skin, closure of the inframammary and vertical incisions, removal of a circular segment of skin at the apex of the vertical incision and inset of the areola. Modern variations of the traditional inverted T technique rely on parenchymal support (rather than skin alone) to increase the longevity of results plication sutures may be used to shorten and increase the projection of the pedicle. The support can be provided as simple sutures to fixate the parenchyma to the pectoralis muscle or fascia.[35,36] This may be facilitated by de-epithelializing skin that would otherwise be discarded, and using the resulting dermal flaps to aid in pedicle suspension and fixation. The support can also be provided by parenchymal redistribution methods. For example, a superior flap carrying the NAC can be partially raised to create a pocket over the pectoralis muscle, and the inferior parenchyma is then sutured high into this pocket to increase upper pole fullness and decrease lower pole laxity. In patients that have sufficient breast parenchyma, but suffer from severe ptosis (e.g., massive weight loss patients), a dermal suspension "auto" augmentation–mastopexy can be performed. This technique entails elevation of medial and lateral de-epithelialized parenchymal flaps, and their suspension high to rib periosteum to provide support; the breast is then reshaped by plicating the dermis on the surface of the pedicle thus reshaping it.[37]

Advantages and Disadvantages

The main advantage includes the ability to excise large amounts of skin (necessary for severe ptosis with significant skin excess in all dimensions), eliminating any bunching or skin redundancy noted in the periareolar or vertical techniques. For this reason, the inverted T approach is the best option for severe

ptosis, typically seen in massive weight loss patients. This approach is commonly practiced in North America, thus the learning curve is not as steep as other approaches. Also, it is more predictable, and has excellent immediate aesthetic outcomes without the need for intraoperative overcorrection or waiting for the breast to settle. What is seen on the operating table with tailor-tacking maintains itself reasonably well. This technique allows wide exposure of the breast, facilitating NAC elevation and parenchymal fixation, redistribution, and autoaugmentation techniques. Because of all these reasons, the inverted T technique remains the most popular approach for mastopexy in the US and throughout the Americas.[31]

31.5 Other Variations

The number of techniques and variations described in the literature are too many to be listed in this chapter. There have been many attempts to shorten or eliminate the horizontal or the vertical components of the scar. Some of these include the B technique by Regnault, short inframammary scar by Marchac and de Olarte, L short scar technique by Chiari, the J scar technique by Gasperoni et al, the owl technique by Ramirez, and the no vertical scar technique by Lalonde et al.[38,39,40,41,42,43]

31.5.1 Techniques in Augmentation–Mastopexy

The surgical approach to the ptotic breast by means of mastopexy has been thoroughly discussed in this chapter, and for this reason this section will discuss only the variations to the techniques already described.

Periareolar Augmentation–Mastopexy

Indications

Nipple elevation not more than 2 cm. The ptosis presentation should include a NAC at or no more than 1 cm below the inframammary fold and not pointing inferiorly, no more than 3 cm of breast overhanging the fold, and nipple-to-inframammary fold no more than 8 cm.[16]

Technique

Mild ptosis can sometimes be managed by augmentation alone (instead of augmentation–mastopexy) and where uncertainty exists, intraoperative evaluation is needed. Before committing to mastopexy, only the inferior areolar incision is created, to transect the breast inferiorly thorough the zone of de-epithelialization and bevel toward the inframammary fold. This opening allows insertion of the implant by a dual-plane, submuscular, or subglandular technique. After insertion, the incision is stapled closed to assess the breast with the patient sitting upright. If significant ptosis remains, the previously planned periareolar pattern is tailor-tacked, nipple position adjusted, and mastopexy performed. This strategy avoids unnecessary placement of scars and ensures optimal results.[14,16] Recently, there has been increasing enthusiasm for using two incisions for augmentation–mastopexy: an inframammary incision is used for the dual plane placement of the implant to avoid violation of the breast parenchyma; and intra-areolar incisions and breast dissection, which are associated with increased risk of capsular contracture according to several recent studies.[44,45] The periareolar mastopexy is then performed as needed.

Vertical Augmentation–Mastopexy

Indications

Nipple more than 2 cm below the inframammary fold, NAC is not visible on the front of the breast, more than 3 cm of breast overhanging the inframammary fold, and nipple-to-inframammary fold distance more than 9 cm.[16]

Technique

An incision is made in the previously marked vertical resection area extending vertically from the NAC. The implant is placed in the desired pocket by means of a dual-plane, submuscular, or subglandular technique. The incision is then stapled closed and the areas of excess skin are tailor-tacked and marked with the patient sitting upright. The periareolar excess skin is then de-epithelialized and all the remaining skin in the inferior pole within the markings is excised. Breast flaps are undermined as minimally as needed for mobilization to allow closure with minimal tension.[14,16] An important caveat here is that it is important to maintain subcutaneous tissue above the inframammary fold, but below the inferior extent of the vertical incision to help protect the implant from exposure in the event of a wound healing problem.

Inverted T Augmentation–Mastopexy

Indications

Nipple more than 2 cm below the inframammary fold, NAC is not visible on the front of the breast, more than 3 cm of breast overhanging the inframammary fold, nipple-to-inframammary fold distance more than 9 cm, with both horizontal and vertical skin excess.

Technique

The approach to inverted T varies between authors. Some utilize the markings and surgical technique described in the vertical augmentation–mastopexy approach, but add horizontal plications (at the level of the inframammary fold) centered on the vertical segment to take up the redundant skin. Other authors suggest placing the implant through an inframammary fold incision instead, which is then stapled closed and the areas of excess skin are tailor-tacked and marked. The periareolar and inferior pole areas are de-epithelialized, the NAC is elevated, and the incisions are closed appropriately.[14,16]

31.6 Postoperative Care

Postoperative care varies among surgeons; however, common principles can be applied. All patients are asked to carefully wash their chest and abdomen using Hibiclens twice a day for 3 days prior to surgery. Antibiotics are normally used only

perioperatively and not continued postoperatively. Prophylactic intravenous antibiotics are administered less than one hour prior to the incision, and the choice normally includes a first generation cephalosporin such as cefazolin, or an alternative (e.g., vancomycin or clindamycin) in case of allergy. Postoperative analgesia includes a combination of narcotics (e.g., oxycodone) and non-narcotics (e.g., acetaminophen), and is typically needed for the first 2 weeks after surgery. Nausea and vomiting are managed with antiemetics, such as selective 5-HT3 receptor antagonists (e.g., ondansetron) or phenothiazine H-1 receptor antagonists (e.g., promethazine). Drains are rarely used, but if inserted, they are usually removed within the first 1 to 3 days postoperatively. A supportive brassiere is required for at least 6 to 8 weeks postoperatively to ensure full support during the healing process. Strenuous exercise, especially bouncing and jumping is not permitted for at least 4 weeks. At that time, exercise is gradually increased, starting with mild activities such as walking, followed by stationary bicycle or equivalent the week after, and full-on activities during the eighth week. Scar treatment begins during the third week but only after complete wound healing is verified. Scar revision, if needed, is generally postponed 1 year after surgery.

31.7 Outcomes

31.7.1 Outcomes in Mastopexy

Mastopexy produces an improved aesthetic appearance of the breast in most cases and the tradeoff of "scar for shape" is a good one for most patients. However, an undeniable sequela in every case is some degree of recurrent ptosis. This is especially true for augmentation–mastopexy,[14,15,16] and there is a higher re-operation rate following secondary procedures (revision of previously revised mastopexy[46] and augmentation–mastopexy[14,16]). A survey by Rohrich et al showed that most mastopexy techniques had a complication rate less than 5% except for the SPAIR (Hammond) technique, which had complication rates up to 5% (reported by 37.5% of respondents). Complications include suture spitting, excess scarring, breast asymmetry, nipple asymmetry, bottoming out and need for revision. Complications variation by technique is shown in ▶ Table 31.3.

31.7.2 Outcomes in Augmentation–Mastopexy

Good outcomes are possible with all of these approaches. Most incisions heal well if designed appropriately and closed well. The reliability and efficacy of one-stage augmentation–mastopexy has been a controversial topic for many years.[18] A recent systematic review and meta-analysis showed a complication rate of 14.7% and reoperation rate of 12.1%.[47] Complications can be separated into either tissue-related or implant-related categories. Tissue-related complications include recurrent ptosis (4.0%), breast or areola asymmetry (2.7%), poor or hypertrophic scarring (2.5%), and hematoma (0.8%). Implant-related complications include capsular contracture (2.0%); infection (0.7%); and seroma (0.7%).[47] Major skin flap or nipple necrosis, and implant malposition were rarely reported in the literature, thus they were not included in the meta-analysis.[47] However,

Table 31.3 Complications following mastopexy[31]

Mastopexy technique	Complication	%
Overall	Suture spitting	52.6
	Excess scarring	45.2
	Bottoming out	36.1
	Excess scarring in IMF	30.2
	Need for revision	28.1
	Breast asymmetry	19.5
	Nipple asymmetry	6.5
Periareolar	Suture spitting	61.8
	Excess scarring	50.0
	Need for revision	50.0
Vertical	Suture spitting	45.8
	Excess scarring	37.3
	Bottoming out	27.1
SPAIR	Suture spitting	75.0
	General asymmetry	50.0
	Nipple asymmetry	25.0
	Need for revision	25.0
Hall-Findlay	Suture spitting	50.0
	General asymmetry	50.0
	Need for revision	35.7
Inverted T	Suture spitting	53.4
	Excess scarring	46.6
	Bottoming out	41.1

Based on Rohrich RJ, Gosman AA, Brown SA, Reisch J. Mastopexy preferences: a survey of board-certified plastic surgeons. Plast Reconstr Surg. 2006; 118(7):1631–1638

Calobrace et al found partial skin necrosis in 2.6%, and implant-related problems in 1.3% of patients.[14] Tissue-related complications predominated over implant-related complications in most studies, and the possible cause is the weight of the implant contributing to the risk of recurrent ptosis. The combination of tension from an underlying implant, extensive undermining and soft-tissue manipulation around the nipple can lead to nipple and skin flaps necrosis, loss of nipple sensation, and wound dehiscence. The tension also exacerbates areolar widening and hypertrophic scarring. One-stage augmentation–mastopexy has been shown to have comparable complication rates to primary augmentation or mastopexy alone. Risks can be minimized by careful patient selection, thorough preoperative planning, and meticulous execution. In cases where a one-stage augmentation–mastopexy is not safe, a two-stage approach remains a viable and safe alternative.

31.8 Review Questions

31.8.1 True or False

1. According to the American Society of Plastic Surgeons (ASPS) statistics report, mastopexy was the most common aesthetic surgical procedure in the United States.
2. The degree of ptosis is based on the position of the nipple in relation to the inframammary fold and the gland.
3. The plan and operative technique must include intraoperative flexibility to alter incision design and nipple position using tailor-tacking, and in most cases the use of parenchymal suturing.

4. With augmentation–mastopexy, it is ideal to use the smallest implant that will satisfy patient expectations, place the implant first usually through an incision that will be incorporated in the mastopexy plan, and plan for conservative skin resection and on-table adjustment with tailor-tacking.
5. A supportive brassiere is not needed postoperatively and the patient can resume strenuous activities immediately after discharge from hospital.

31.8.2 Answers

1. False.
2. True.
3. True.
4. True.
5. False.

References

[1] American Society of Plastic Surgeons. Plastic Surgery Statistics Report; 2014
[2] American Society of Plastic Surgeons. Cosmetic Surgery National Data Bank Statistics; 2014
[3] Swanson E. A retrospective photometric study of 82 published reports of mastopexy and breast reduction. Plast Reconstr Surg. 128(6):1282–1301
[4] Rinker B, Veneracion M, Walsh CP. Breast ptosis: causes and cure. Ann Plast Surg. 64(5):579–584
[5] Boyd AS, Stasko T, King LE , Jr, Cameron GS, Pearse AD, Gaskell SA. Cigarette smoking-associated elastotic changes in the skin. J Am Acad Dermatol. 41(1):23–26
[6] Pisacane A, Continisio P, Italian Work Group on Breastfeeding. Breastfeeding and perceived changes in the appearance of the breasts: a retrospective study. Acta Paediatr. 93(10):1346–1348
[7] Regnault P. Breast ptosis. Definition and treatment. Clin Plast Surg. 3(2):193–203
[8] Bartels RJ, Strickland DM, Douglas WM. A new mastopexy operation for mild or moderate breast ptosis. Plast Reconstr Surg. 57(6):687–691
[9] Elliott LF. Circumareolar mastopexy with augmentation. Clin Plast Surg. 29 (3):337–347, v
[10] Spear SL, Kassan M, Little JW. Guidelines in concentric mastopexy. Plast Reconstr Surg. 85(6):961–966
[11] Swanson E. Prospective photographic measurement study of 196 cases of breast augmentation, mastopexy, augmentation/mastopexy, and breast reduction. Plast Reconstr Surg. 131(5):802e–819e
[12] Davison SP, Spear SL. Simultaneous breast augmentation with periareolar mastopexy. Semin Plast Surg. 18(3):189–201
[13] Gonzalez-Ulloa M. Correction of hypotrophy of the breast by means of exogenous material. Plast Reconstr Surg Transplant Bull. 25:15–26
[14] Calobrace MB, Herdt DR, Cothron KJ. Simultaneous augmentation/mastopexy: a retrospective 5-year review of 332 consecutive cases. Plast Reconstr Surg. 131(1):145–156
[15] Stevens WG, Macias LH, Spring M, Stoker DA, Chacón CO, Eberlin SA. One-stage augmentation mastopexy: a review of 1192 simultaneous breast augmentation and mastopexy procedures in 615 consecutive patients. Aesthet Surg J. 34(5):723–732
[16] Spear SL, Boehmler JHT , IV, Clemens MW. Augmentation/mastopexy: a 3-year review of a single surgeon's practice. Plast Reconstr Surg. 118(7) Suppl: 136S–147S, discussion 148S–149S, 150S–151S
[17] Regnault P. The hypoplastic and ptotic breast: a combined operation with prosthetic augmentation. Plast Reconstr Surg. 37(1):31–37
[18] Spear S. Augmentation/Mastopexy: “surgeon, beware”. Plast Reconstr Surg. 112(3):905–906
[19] Nahai F, Fisher J, Maxwell PG, Mills DC , II. Augmentation mastopexy: to stage or not. Aesthet Surg J. 27(3):297–305
[20] Khavanin N, Jordan SW, Rambachan A, Kim JY. A systematic review of single-stage augmentation-mastopexy. Plast Reconstr Surg. 134(5):922–931
[21] Puckett CL, Meyer VH, Reinisch JF. Crescent mastopexy and augmentation. Plast Reconstr Surg. 75(4):533–543
[22] Benelli L. A new periareolar mammaplasty: the “round block” technique. Aesthetic Plast Surg. 14(2):93–100
[23] Góes JC. Periareolar mammaplasty: double skin technique with application of polyglactine or mixed mesh. Plast Reconstr Surg. 97(5):959–968
[24] Gruber RP, Jones HW , Jr. The “donut” mastopexy: indications and complications. Plast Reconstr Surg. 65(1):34–38
[25] Lassus C. The Lassus vertical technique. Aesthet Surg J. 31(8):897–913
[26] Lassus C. Breast reduction: evolution of a technique–a single vertical scar. Aesthetic Plast Surg. 11(2):107–112
[27] Cruz-Korchin N, Korchin L. Vertical versus Wise pattern breast reduction: patient satisfaction, revision rates, and complications. Plast Reconstr Surg. 112 (6):1573–1578, discussion 1579–1581
[28] Lejour M. Vertical mammaplasty and liposuction of the breast. Plast Reconstr Surg. 94(1):100–114
[29] Lejour M. Vertical mammaplasty: update and appraisal of late results. Plast Reconstr Surg. 104(3):771–781, discussion 782–784
[30] Hammond DC, Alfonso D, Khuthaila DK. Mastopexy using the short scar periareolar inferior pedicle reduction technique. Plast Reconstr Surg. 121(5): 1533–1539
[31] Rohrich RJ, Gosman AA, Brown SA, Reisch J. Mastopexy preferences: a survey of board-certified plastic surgeons. Plast Reconstr Surg. 118(7): 1631–1638
[32] Hall-Findlay EJ. A simplified vertical reduction mammaplasty: shortening the learning curve. Plast Reconstr Surg. 104(3):748–759, discussion 760–763
[33] Hall-Findlay EJ. Pedicles in vertical breast reduction and mastopexy. Clin Plast Surg. 29(3):379–391
[34] Wise RJ. A preliminary report on a method of planning the mammaplasty. Plast Reconstr Surg (1946). 17(5):367–375
[35] Flowers RS, Smith EM , Jr. “Flip-flap” mastopexy. Aesthetic Plast Surg. 22(6): 425–429
[36] Ritz M, Silfen R, Southwick G. Fascial suspension mastopexy. Plast Reconstr Surg. 117(1):86–94
[37] Rubin JP. Mastopexy after massive weight loss: dermal suspension and total parenchymal reshaping. Aesthet Surg J. 26(2):214–222
[38] Regnault P. Breast reduction: B technique. Plast Reconstr Surg. 65(6):840–845
[39] Marchac D, de Olarte G. Reduction mammaplasty and correction of ptosis with a short inframammary scar. Plast Reconstr Surg. 69(1):45–55
[40] Chiari A , Jr. The L short-scar mammaplasty: 12 years later. Plast Reconstr Surg. 108(2):489–495
[41] Gasperoni C, Salgarello M, Gasperoni P. A personal technique: mammaplasty with J scar. Ann Plast Surg. 48(2):124–130
[42] Ramirez OM. Reduction mammaplasty with the “owl” incision and no undermining. Plast Reconstr Surg. 109(2):512–522, discussion 523–524
[43] Lalonde DH, Lalonde J, French R. The no vertical scar breast reduction: a minor variation that allows to remove vertical scar portion of the inferior pedicle wise pattern T scar. Aesthetic Plast Surg. 27(5):335–344
[44] Wiener TC. Relationship of incision choice to capsular contracture. Aesthetic Plast Surg. 32(2):303–306
[45] Jacobson JM, Gatti ME, Schaffner AD, Hill LM, Spear SL. Effect of incision choice on outcomes in primary breast augmentation. Aesthet Surg J. 32(4): 456–462
[46] Spear SL, Low M, Ducic I. Revision augmentation mastopexy: indications, operations, and outcomes. Ann Plast Surg. 51(6):540–546
[47] Khavanin N, Jordan SW, Kim JY. Reply: A systematic review and meta-analysis of single-stage augmentation mastopexy. Plast Reconstr Surg. 2015; 135(6):1064e–1066e

32 Breast Augmentation

Richard C. Baynosa, Andrew G. Silver, and Rachel I. Mason

Abstract
This chapter deals with the relevant considerations involved in breast enhancement using implants. Specific data to be gathered during the presurgical exam are detailed, and implant shells (smooth, textured), implant fillers (silicone, saline), implant shape, and implant size are discussed. Relevant surgical procedures involving access incision, pocket location, and surgical techniques are thoroughly covered, and the chapter concludes with seasoned advice on postoperative care and potential outcomes and complications.

Keywords: Baker classification and grades of capsular contracture, silicone filled implants, saline filled implants

32.1 Goals and Objectives

- Understand the proper evaluation of prospective breast augmentation patients.
- Clearly define the benefits and drawbacks for the different types of breast implants and various surgical approaches to breast augmentation.
- Appropriately educate patients on the potential complications of breast augmentation.
- Know the evidence-based perioperative care to maximize patient safety and quality outcomes.

32.2 Patient Presentation

Patients seeking breast augmentation can vary in diagnosis from simple hypomastia to breast asymmetry or ptosis, to the more complex tuberous breast deformity.

As with all surgical procedures, a general medical history is obtained in order to identify any contraindications to surgery. Specific focused history for breast augmentation patients should include the following:

1. *Any previous breast pathology or breast cancer*: Our group requires a documented normal mammogram and/or breast examination within 1 year prior to surgery. Any abnormalities encountered on examination or mammography must be thoroughly evaluated by a breast surgeon prior to breast augmentation.
2. *Pregnancy history*: Breasts often undergo substantial changes in both size and shape during pregnancy and breastfeeding. Our group recommends a waiting period of at least 3 months to allow for stabilization of breast size after completion of breastfeeding.
3. *Changes in weight*: Fluctuations in weight can significantly affect breast size and shape as approximately 50% of the breast is composed of fat. It is advisable to delay surgery until the patient's weight has been stable prior to breast augmentation.
4. *Patient's age*: Our group does not perform breast augmentation in patients younger than 18 years. If the patient is younger than 22 years, we do not use silicone implants without a signed waiver acknowledging Food and Drug Administration (FDA) off-label usage.

Physical exam should include a general observation of the patient, including height, weight, body frame, and the presence of any structural asymmetries or deformities. It is imperative to point out any and all asymmetries, as minor differences often go unnoticed by a patient preoperatively, yet can become a significant source of distress after the operation. This is likely due to an increase in the patient's attention to her augmented breasts postoperatively. In the authors' opinion, it is far easier to allay patient concerns about an asymmetry that was discussed and documented preoperatively.

Required measurements include nipple-to-sternal notch distance, nipple-to-inframammary fold (IMF) distance, and the breast base–width (taken just above the nipple). The measurement of breast height and intermammary distance are also recommended. Additional helpful findings on physical exam include a pinch test of the upper pole to assess the amount of soft-tissue coverage and an assessment of skin envelope quality (e.g., stretch marks may indicate an inelastic envelope). These measurements are verbalized as they are being taken to reinforce any asymmetries to the patient.

The lie of the breast on the chest wall (i.e., lateralization of nipple and breast) versus normal positioning should also be documented. It should be discussed with the patient that the postoperative intermammary distance is determined by the preoperative state. Simply stated, with a properly sized implant, the patient with a normal intermammary distance will be normal and patients with a wide intermammary distance will be wide postoperatively. It is imperative that the patient understands that the breast footprint cannot be changed. Essentially, breast augmentation fills out the existing breast behind the existing nipple position.

32.3 Preparation for Surgery

There is no single approach in breast implant surgery that is ideal for every patient. Surgical decision-making should be customized to the needs and expectations of the patient. However, in general, preparation and planning for surgery can be broken down into three major decisions: implant selection, access incision, and pocket location.

32.4 Implant Selection

32.4.1 Implant Shell

Essentially, two types of implant shell are currently available: smooth and textured. While the texturing process varies by manufacturer, these devices were developed to mimic the surface of polyurethane covered implants, which had very low capsular contracture rate, but were removed from the market due to breakdown to a carcinogenic compound. Level I studies have not supported the lower capsular contracture rates utilizing the current textured implants.[1,2,3,4,5,6,7,8,9,10,11,12,13,14] Moreover, there are as many level I studies showing no difference as there are that show a benefit, which likely indicates that surface

texture does not have a major role in capsular contracture, especially in the subpectoral pocket plane. The available literature is less conclusive for subglandular pocket plane, and there seems to be a possible benefit of less capsular contracture with implant texturing in this position.[4,9,10,15] However, recent reports have shown an increased incidence of late seroma development associated with implant texturing.[16]

32.4.2 Implant Filler Material

The type of implant filler is limited to two choices: silicone and saline.

Silicone is beneficial in that it offers the most natural feel. After a period of unavailability due to health concerns, the FDA again approved silicone implants for general aesthetic augmentation in 2006. However, it should be noted that when silicone implants are used for breast augmentation, their use is limited by the FDA to patients older than 22 years.[17] As new generations of implants have been introduced, the silicone gels have become progressively more cohesive through crosslinking of the silicone polymer. Currently, all modern silicone implants contain cohesive gels of varying viscosity. Form stable implants maintain their shape without collapsing under their normal weight or being deformed by the surrounding soft-tissue envelope. These implants are often referred to as highly cohesive gels. There is some evidence that form stability has some long-term advantages in minimizing implant-related complications, such as leak and capsular contracture.[18,19,20] However, for a given volume, form-stable implants require incisions 0.5 to 1.0 cm larger than round non–form-stable implants. For all modern silicone filled implants, the FDA recommends that patients receive MRI screening for silent rupture 3 years after the initial placement and every 2 years thereafter.[21]

Although the feel of saline implants is less natural than that of the silicone gels, leaks are easier to detect and are safely absorbed by the body. Additional disadvantages include an increase in visible implant wrinkling. From a procedural standpoint, these implants require access incisions that are smaller and potentially more easily concealed, since they are inserted in a deflated state. In addition, there is room for intraoperative adjustment of the final implant size based on the manufacturer recommended fill range making these implants particularly advantageous in patients with significant asymmetries.

32.4.3 Implant Shape

Both silicone and saline implants are now available in a large variety of shapes (▶ Fig. 32.1, ▶ Fig. 32.2, ▶ Fig. 32.3, ▶ Fig. 32.4). Most round implants come in different projections for a given

Fig. 32.1 Various smooth round silicone gel implants in multiple styles, base widths, and projections with approximately 300 mL volume (295–325 mL). Front row (L to R): Allergan Natrelle Inspira SRM 310cc, Allergan Natrelle Inspira SRF 295cc. Middle row (L to R): Allergan Natrelle Style 10 300cc, Allergan Natrelle Style 15 304cc, Allegan Natrelle Style 20 325cc. Back row (L to R): Allergan Natrelle Style 40 300cc, Allergan Style 45 320cc.

Fig. 32.2 Side profile of various silicone gel implants. From left to right: Allergan Natrelle Inspira SRM 310cc, Allergan Natrelle Inspira SRF 295cc, Allergan Natrelle Style 10 300cc, Allergan Natrelle Style 15 304cc, and Allegan Natrelle Style 20 325cc.

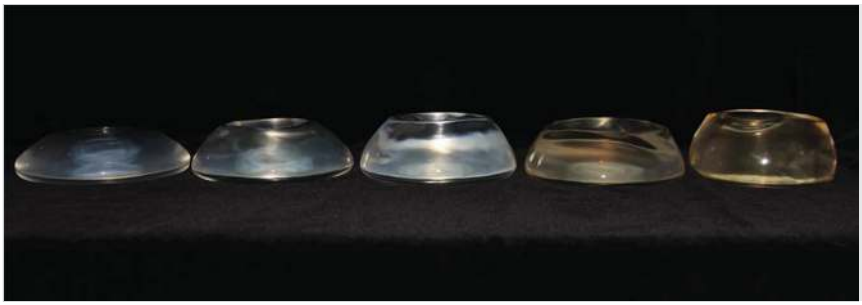


Fig. 32.3 Side profile of various silicone gel implants. From left to right: Allergan Natrelle Style 10 300cc, Allergan Natrelle Style 15 304cc and Allegan Natrelle Style 20 325cc, Allergan Natrelle Style 40 300cc, and Allergan Style 45 320cc.

Fig. 32.4 Side profile of various silicone gel implants. From left to right: Allergan Natrelle Inspira SRM 310cc, Allergan Natrelle Inspira SRF 295cc, Allergan Natrelle Style 40 300cc, and Allergan Style 45 320cc.

volume (low-profile, moderate-profile, moderate-plus-profile, high-profile, and ultra-high-profile). Shaped/anatomic implants come with even more variability because of an asymmetric shape in which width, height, and projection can all be varied. In general, these implants have a tapered upper pole and fuller lower pole, which is intended to give a more natural breast shape. However, utilization of shaped implants requires more precision as orientation must be both appropriate and symmetric. Overdissection of the implant pocket can lead to malposition and rotation, which is highly problematic when utilizing a shaped device. Studies have shown that patient satisfaction is similar in using either a round or shaped device.[19] Further, plastic surgeons saw no difference in regard to overall breast beauty by implant shape and scored round implants higher in regard to naturalness and upper pole appearance (► Fig. 32.8, ► Fig. 32.9).[22]

32.4.4 Implant Size

The primary driving force for many patients regarding size selection remains achieving a desired cup size. However, choosing volume in this manner often leads to reoperation, as the volume selection has not matched the tissue characteristics or the footplate of the breast. Alternatively, and more appropriately, the guiding principles popularized by Tebbetts should be to optimally fill the breast while respecting its natural boundaries.[23,24] Classically, the most important dimension is the width of the breast and therefore the width of the implant itself. The other dimensions and skin envelope may further guide the selection process. Ultimately, we routinely utilize the breast width to select a range of implants available to the patient, and allow the patient to choose the final volume with assistance from the surgeon regarding the type and shape of implant that will best achieve the patient's goals.

32.5 Access Incision

32.5.1 Inframammary

This approach provides the most direct access to all possible pockets and avoids having to enter the breast parenchyma. The scar is generally well concealed if appropriately planned. This is also the most versatile incision for future revisions. There are data to suggest a decreased capsular contracture rate with this incision when compared to the periareolar incision.[25,26]

32.5.2 Periareolar

This approach typically results in a well-concealed scar within the interface of the nipple-areola complex (NAC) and the breast skin. However, a hypertrophic scar in this location is very unsightly and can be difficult to correct. A nipple diameter of at least 3.5 cm is required for adequate access. The contamination theory suggests a higher rate of capsular contracture through this approach due to bacteria present within the breast ductules.[27,28]

32.5.3 Transaxillary

This approach completely avoids the potential for placing a scar on the breast. However, it is the least versatile of the recommended approaches and is furthest from the pocket. In general, only saline implants in the subpectoral pocket are recommended with this approach. Silicone gel implants and the subglandular plane may also be used but this is technically more challenging. With this approach, the utilization of an endoscope results in greatly enhanced visualization.[29]

32.5.4 Transumbilical

This approach is restricted to inflatable saline devices and because of the blind nature of this dissection and imprecise pocket creation, it is not recommended by the authors in any situation.

32.6 Pocket Location

32.6.1 Subglandular

This pocket provides both good projection and shape, and minimizes distortion of the implant in muscular or active patients. This pocket may also provide a small degree of lift in breasts with mild ptosis and volume deflation. Utilization of this pocket requires at least 2 cm of upper pole soft tissue coverage as determined by pinch test. Drawbacks of this pocket include a potentially higher capsular contracture rate, more implant edge palpability, greater visible rippling or implant visibility, and an increased degree of mammography interference.[1,15]

32.6.2 Subpectoral

This pocket provides the thickest soft tissue coverage, lower capsular contracture rates,[1,15] and minimizes implant visibility and palpability. However, there is less upper pole projection when compared to the subglandular plane, and the upper pole projection is more challenging to control. Additional drawbacks of this pocket are related to the overlying pectoralis major, which may distort the implant with activity (animation deformity) and can also cause lateral displacement of the implant over time, resulting in a wider cleavage, or intermammary distance particularly when the patient lies down. A double bubble deformity where the breast tissue migrates

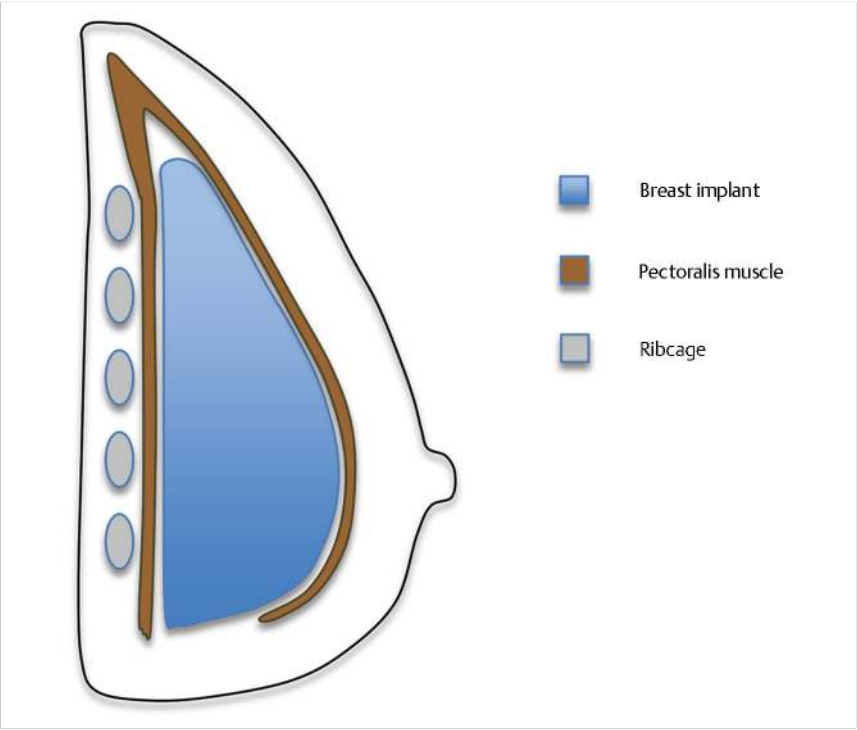


Fig. 32.5 Dual Plane I. Release of pectoralis muscle at the IMF.

inferiorly over the fixed implant is also more common when this pocket is utilized. Finally, muscle spasm should be anticipated and a pretreatment of such spasm should be considered.

32.6.3 Biplanar (Dual Plane)[29]

This approach allows for upper pole coverage by the pectoralis major with varying amounts of lower pole coverage by the breast parenchyma. Division of the origins of the pectoralis at the IMF allows for retraction of a portion of the pectoralis, with a resultant direct exposure of the implant surface to the breast parenchyma along the lower pole of the implant. This allows for the implant to rest at the IMF, thus expanding the lower pole and potentially improving breast ptosis (▶ Fig. 32.5, ▶ Fig. 32.6, ▶ Fig. 32.7).

Dual Plane I

This involves division of the pectoralis major origins along the IMF. No additional separation of the breast parenchyma and underlying pectoralis fascia is performed. The subpectoral space is then dissected for implant placement (▶ Fig. 32.5). This is the approach performed by our practice in most subpectoral breast augmentations (see surgical technique).

Dual Plane II

In addition to the release of the pectoralis and creation of the subpectoral pocket as described in dual plane I, the breast parenchyma is released from the underlying pectoralis fascia to the level of the inferior border of the NAC (▶ Fig. 32.6). This is our group's preferred approach in patients with mild ptosis that do not want a concurrent mastopexy. (▶ Fig. 32.5, ▶ Fig. 32.7)

Dual Plane III

This approach follows that of dual plane II but involves continued release of the breast parenchyma to the level of the superior border of the NAC. This maximizes the amount of parenchymal coverage of the implant while still providing additional soft-tissue muscle coverage of the upper pole (▶ Fig. 32.7).

32.6.4 Preoperative Patient Counseling

In addition to the normal risks of surgery (bleeding, infection, damage to nearby structures), additional considerations that should be routinely discussed include the potential for scarring, loss of nipple sensation, implant rippling, implant rupture, blocking of breast tissue on mammography, animation deformity (with submuscular pocket selection), capsular contracture or pocket malposition requiring further surgery and cost, and development of anaplastic large cell lymphoma (ALCL) at a rate of 1/200,000.[30] The current FDA recommendations for MRI surveillance should also be mentioned in patients undergoing breast augmentation with silicone gel implants and it should be noted that these costs are rarely covered by insurance. The published data regarding rupture and reoperation rates over a 10-year period of approximately 8 and 36%, respectively, is quoted.[31] No guarantees on results or cup size are implied or given.

32.7 Treatment

32.7.1 Surgical Technique: General

In all situations, the patient is initially given prophylactic antibiotics and appropriate deep venous thrombosis prophylaxis

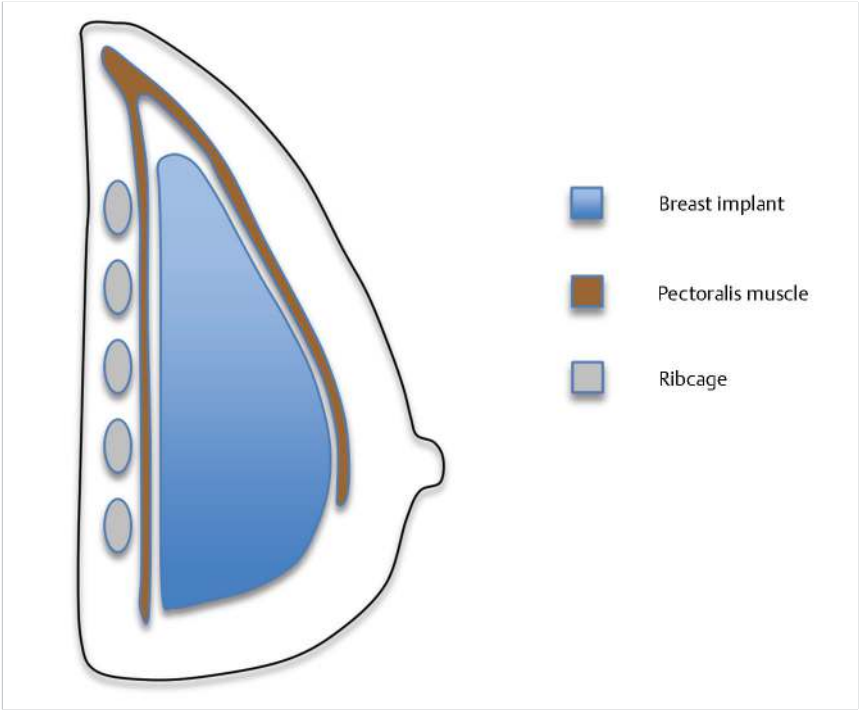


Fig. 32.6 Dual Plane II. Release of pectoralis muscle to inferior border of nipple-areola complex.

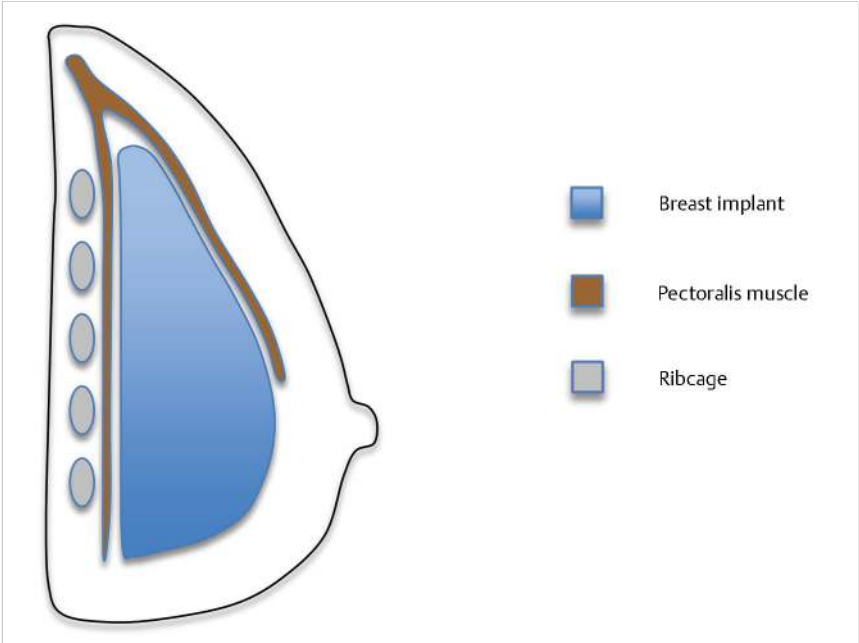


Fig. 32.7 Dual Plane III. Release of pectoralis muscle to superior border of nipple-areola complex.

measures are taken. The patient is then prepped and draped in the usual sterile fashion with the upper extremities secured to arm boards at 90 degrees to the thorax. The planned incision is then marked and carried out in the manner selected as described below (▶ Table 32.1). Dissection of a precise pocket under direct visualization, while maintaining meticulous hemostasis, is aided with the utilization of a lighted retractor and/or endoscope depending on the desired approach. Early in one's career, an exact implant sizer can be utilized to verify the pocket size and positioning. After the implant pocket has been appropriately created, the permanent implant and pocket are then irrigated with an antibiotic solution (50,000 units of bacitracin, 1 g cefazolin, and 80 mg gentamycin in 500 mL of normal saline). The pocket is again visualized to ensure hemostasis. All visible blood is copiously irrigated, after which the implants are inserted utilizing a no-touch technique. Although not utilized in our practice, a funnel device can aid in the placement of silicone implants. The patient is then brought into an

Table 32.1 Surgical approaches for breast augmentation

Access site	Pocket choice	Implant type	Reuse for revisions	Major benefit	Major drawback
Inframammary	Both	All	Best	Ease/Versatility	Possible visible scar on breast
Periareolar	Both	All	Possible	Scar can be well hidden	Possible visible scar on breast
Transaxillary	Both (with endoscope)	All	Not recommended	No scar on breast	Remote from pocket

upright sitting position to verify symmetry and implant position. When the surgeon is satisfied with the result, the patient is returned to the supine position and a meticulous layered closure is performed. We prefer to utilize chromic suture to close both the implant pocket and the deep dermis, in simple interrupted and buried interrupted fashion, respectively. The epidermis is then re-approximated with a subcuticular Monocryl suture. Surgical skin adhesive is applied. A 6-inch ACE wrap is then utilized to provide gentle but firm circumferential support. The details regarding incision and pocket location are detailed below.

32.7.2 Inframammary Approach

The incision is planned in the projected inframammary crease, with the majority of the incision placed lateral to a line projected down from the nipple. The incision length is usually 4.5 to 6 cm in length, depending upon the implant type and size. The incision is made and carried down until the prepectoral fascia is identified. If a subglandular pocket has been chosen, this is the plane of pocket dissection. If a subpectoral pocket has been chosen, dissection is then carried laterally to identify the lateral border of the pectoralis major. The plane beneath the pectoralis is then entered. The subpectoral pocket is then created from the lateral to medial direction. Only the inferior most attachments of the pectoralis to the ribs are divided using electrocautery. The methods of pocket creation, irrigation, implant placement, and closure as detailed above are followed.

32.7.3 Periareolar Approach

The incision is planned from the 3 o'clock to the 9 o'clock position on the inferior half of the areola at the junction of the areola and the breast skin. The dissection proceeds in a vertical direction directly through the breast tissue until the prepectoral fascia is encountered. Alternatively, some surgeons, although not our preference, create an inferiorly based breast skin flap, separating it from the parenchyma, providing access to the pectoral fascia along the inframammary fold. Again, if a subglandular pocket has been chosen, this is the plane of pocket dissection. If a subpectoral pocket has been chosen, the dissection proceeds in a more oblique fashion, and angled inferiorly until the prepectoral fascia is identified. Dissection is then carried laterally to identify the lateral border of the pectoralis major. The subpectoral pocket is then created from the lateral to medial direction. Only the inferior most origins of the pectoralis major to the ribs are divided using electrocautery. The sternal origins of the pectoralis major are not disrupted. The methods of pocket creation, irrigation, implant placement, and closure as detailed above are followed.

32.7.4 Transaxillary Approach

The incision is planned in the lower pole of the hair bearing skin of the axilla within the most anterior skin crease. This incision is then deepened bluntly to identify the lateral border of the pectoralis major. Blunt digital dissection is then utilized to develop a plane between the pectoralis major and pectoralis minor. The methods of pocket creation, irrigation, implant placement, and closure as detailed above are followed. With this approach, utilization of an endoscope can greatly enhance visualization and allow more precise dissection of the subpectoral and even the subglandular pockets.

32.7.5 Alterations in Technique for Tuberous Breast

In order to treat this challenging problem, we prefer to use the subglandular pocket as described above. Utilization of this pocket allows for release of the constricting bands between the pectoralis major and the breast parenchyma. Radial scoring of the breast parenchyma is performed with a resultant release of constricted tissue and a widening of the breast base. A periareolar mastopexy is then performed on both breasts to ensure symmetrical scarring. Finally, a saline filled implant is utilized for greater intraoperative adjustability and correction of asymmetry. (See Chapter 37 for greater details on the tuberous breast condition as well as surgical treatment.)

32.8 Postoperative Care

Dressings involve minimal use of tape to avoid skin irritation. Closed incisions are covered with surgical skin adhesive. A 6-inch ACE bandage is then wrapped circumferentially around the patient's chest to provide some gentle compression in order to help position the implant, control edema, and decrease the risk of hematoma. This is left in place for 24 hours and then changed to a sports bra.

The patient is kept in the post anesthesia care unit until discharge qualifications are met. She is instructed to leave the dressings in place. Most patients are given oral narcotic pain medications and an empiric 7-day course of oral antibiotics to cover skin flora.[32]

The patient follows up in clinic on postoperative day 1. Her dressings are removed and the patient is examined for the development of a hematoma. If her breasts are nontender, she is assisted into a

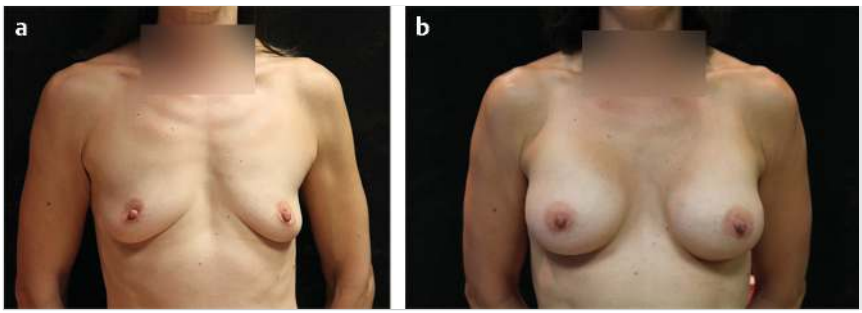


Fig. 32.8 (a,b) Preoperative and postoperative photos, respectively, of a patient with significant breast deflation. Augmentation was performed with shaped anatomical implants in a dual-plane II fashion.

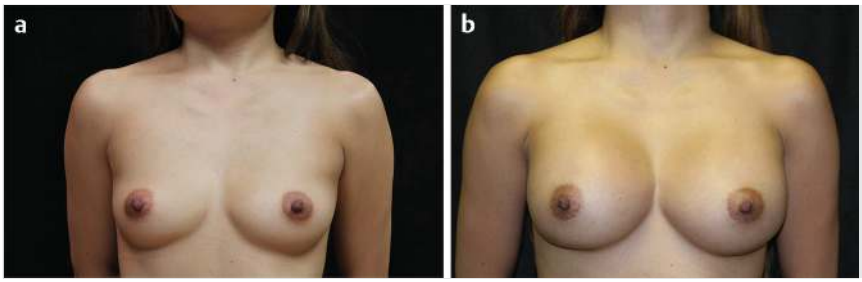


Fig. 32.9 (a,b) Preoperative and postoperative photos, respectively, of a patient desiring larger breasts. A round silicone implant was placed in a dual-plane I fashion.

sports bra. The patient is instructed to wear this bra and avoid breast manipulation for 2 weeks. The patient is told to return to light activity as tolerated, and back to full duty at 2 weeks.

She is seen again in clinic 2 weeks postoperatively. At this point, she is instructed on scar care. She is told to begin scar massage 3 weeks postoperatively with an over-the-counter scar cream, and to wear silicone sheeting as much as possible. Implant massage is not recommended by our group, although others advocate it, particularly in subpectoral or dual-plane implants.[26] Postoperative photographs are taken at 3 months and 1 year. With silicone implants, an MRI may be performed at 1 year to establish a baseline, and the FDA recommends a screening MRI to assess for silent implant rupture at 3 years followed by subsequent imaging studies every 2 years.[21]

32.9 Outcomes and Complications

Patient satisfaction is generally excellent following breast augmentation, even if there is a need for further surgery (▶ Fig. 32.8, ▶ Fig. 32.9). Patients have reported an improved body image, overall sense of well-being, and specifically improved satisfaction with breast appearance as well as psychosocial and sexual well-being.[33,34,35]

Acute complications are often self-limiting. Alterations in nipple sensitivity are common, but resolve over time in most patients.[36,37,38] If a patient presents with a wound infection, an ultrasound may be used to assess for a periprosthetic fluid collection. Simple wound infections with no or minimal periprosthetic fluid will generally respond to oral antibiotics if initiated early. If the infection is severe, does not resolve, or recurs, it should be treated with explantation.[39,40] Hematomas occur in about 1% of patients and must be evacuated if they are large or expanding.[41,42] Mondor's disease is a superficial thrombophlebitis of the breast that may be treated with NSAIDs and warm compresses.[43] Wound breakdown leading to implant exposure must be treated with explantation.[39,40]

Table 32.2 Baker classification of capsular contracture

Grade I	Normal—no palpable capsule
Grade II	Minimal—palpable but not visible
Grade III	Moderate—palpable and visible
Grade IV	Severe—palpable, visible, and painful

Note: Baker grade I and II capsular contractures generally require no intervention, whereas grade III or IV contracture should be treated operatively.

The reoperation rate in our practice is similar to the published rate of 25%.[34] The most common indication for reoperation is capsular contracture, which generally occurs in the first year post operatively.[12,37,44,45,46] As discussed previously, the rate of occurrence is dependent upon implant type and pocket choice.[47] Additionally, bacterial contamination leading to biofilm production around the implant is thought to be a major driving force in the etiology of capsular contracture.[48,49] Factors that have been found to reduce the risk of capsular contracture are the use of an inframammary incision, textured implants, subpectoral placement, and antibiotic irrigation; conversely, hematoma, seroma, infection, smooth implants, and subglandular implant placement increase the risk of capsular contracture.[1,20,26,32,43,47,50]

32.9.1 The Baker Classification and Grades of Capsular Contracture

Baker grade I and II capsular contractures generally require no intervention, whereas grade III or IV contracture should be treated operatively (▶ Table 32.2).[51]

The gold standard for treatment of capsular contracture is total capsulectomy with implant removal. A position or "site"

change is generally recommended, particularly if the original implant was placed subglandular. A subpectoral or dual plane pocket is preferred.[52,53] Due to the technical difficulties in performing a total capsulectomy, the authors have investigated other strategies such as anterior capsulectomies or leaving the capsule intact and creating a new supra- or subcapsular pocket; however, these techniques have not been proven to be as effective as total capsulectomy in preventing recurrence of capsular contracture.[9,54] Acellular dermal matrices are increasingly being used in the treatment of capsular contracture and are showing promise in preventing recurrence, although all studies are compared to historical cohorts.[55,56,57]

Other significant complications are less commonly encountered. Visible implant rippling may be seen, particularly if the implant is too large. Dynamic distortion may occur during pectoralis muscle activation with a submuscular implant. Implant malposition may also occur and can be noted by lateral displacement, symmastia, or an inframammary fold that is too high or too low. If the malposition is minor, the implant may be massaged or compressed in the correct direction. However, these deformities may need to be corrected with pocket change, capsulorrhaphy, or biologic mesh placement.[58,59,60]

Another possible complication is implant rupture. If it is a saline implant, the rupture will be noticeable due to resorption of the saline, and the implant should be explanted and replanted. In silicone implants, if the rupture is symptomatic or extracapsular, the implant should be removed. Significant benefits of removing an asymptomatic, intracapsular leaking silicone implant have not been proven.[61,62] However, if the patient requests removal in this situation, it is our practice to remove the implant.

In addition, there have been rare cases of ALCL reported in association with breast implants, with the vast majority associated with textured implants.[30,63,64] This typically presents as a late periprosthetic fluid accumulation. Any patient with a late appearing seroma should at least have fluid aspirated and sent for cytologic analysis.[65]

Fortunately, complications of breast augmentation are well tolerated and patient satisfaction is high, making breast augmentation a rewarding procedure for both surgeon and patient.

32.10 Review Questions

32.10.1 Choose the Best Answer

1. In breast augmentation patients, the development of anaplastic large cell lymphoma (ALCL) occurs at a rate closest to
   a) 1/20,000.
   b) 1/200,000.
   c) 1/2,000,000.
   d) 1/20,000,000.
2. For all silicone filled implants, the FDA-recommended MRI screening for silent rupture is
   a) Three years after the initial placement and every 2 years thereafter.
   b) Yearly after implantation.
   c) Only if there are symptoms of pain or significant patient concern.
   d) Every 5 years after implantation.
3. According to the authors, the single most important measurement when selecting implant volume is
   a) Nipple to inframammary fold distance.
   b) Sternal notch to nipple distance.
   c) Intermammary distance.
   d) Breast width.
4. The complication more likely to occur with a subpectoral pocket when compared to a submammary pocket is
   a) Implant rippling.
   b) Implant rupture.
   c) Double-bubble deformity.
   d) Capsular contracture.
5. According to the authors, the most versatile access incision for breast augmentation is:
   a) Transumbilical.
   b) Inframammary fold.
   c) Periareolar.
   d) Transaxillary.

32.10.2 Answers

1. b. 1/200,000.
2. a. Three years after the initial placement and every 2 years thereafter.
3. d. Breast width.
4. c. Double-bubble deformity.
5. b. Inframammary fold.

References

[1] Barnsley GP, Sigurdson LJ, Barnsley SE. Textured surface breast implants in the prevention of capsular contracture among breast augmentation patients: a meta-analysis of randomized controlled trials. Plast Reconstr Surg. 117(7):2182–2190

[2] Puckett CL, Croll GH, Reichel CA, Concannon MJ. A critical look at capsule contracture in subglandular versus subpectoral mammary augmentation. Aesthetic Plast Surg. 11(1):23–28

[3] Malata CM, Feldberg L, Coleman DJ, Foo IT, Sharpe DT. Textured or smooth implants for breast augmentation? Three year follow-up of a prospective randomised controlled trial. Br J Plast Surg. 50(2):99–105

[4] Asplund O, Gylbert L, Jurell G, Ward C. Textured or smooth implants for submuscular breast augmentation: a controlled study. Plast Reconstr Surg. 97(6): 1200–1206

[5] Tarpila E, Ghassemifar R, Fagrell D, Berggren A. Capsular contracture with textured versus smooth saline-filled implants for breast augmentation: a prospective clinical study. Plast Reconstr Surg. 99(7):1934–1939

[6] Fagrell D, Berggren A, Tarpila E. Capsular contracture around saline-filled fine textured and smooth mammary implants: a prospective 7.5-year follow-up. Plast Reconstr Surg. 108(7):2108–2112, discussion 2113

[7] Kjøller K, Hölmich LR, Jacobsen PH, et al. Capsular contracture after cosmetic breast implant surgery in Denmark. Ann Plast Surg. 47(4):359–366

[8] Poeppl N, Schreml S, Lichtenegger F, Lenich A, Eisenmann-Klein M, Prantl L. Does the surface structure of implants have an impact on the formation of a capsular contracture? Aesthetic Plast Surg. 31(2):133–139

[9] Collis N, Sharpe DT. Recurrence of subglandular breast implant capsular contracture: anterior versus total capsulectomy. Plast Reconstr Surg. 106(4):792–797

[10] Collis N, Coleman D, Foo IT, Sharpe DT. Ten-year review of a prospective randomized controlled trial of textured versus smooth subglandular silicone gel breast implants. Plast Reconstr Surg. 106(4):786–791

[11] Hakelius L, Ohlsén L. A clinical comparison of the tendency to capsular contracture between smooth and textured gel-filled silicone mammary implants. Plast Reconstr Surg. 90(2):247–254

[12] Hakelius L, Ohlsén L. Tendency to capsular contracture around smooth and textured gel-filled silicone mammary implants: a five-year follow-up. Plast Reconstr Surg. 100(6):1566–1569

[13] Burkhardt BR, Eades E. The effect of Biocell texturing and povidone-iodine irrigation on capsular contracture around saline-inflatable breast implants. Plast Reconstr Surg. 96(6):1317–1325

[14] Burkhardt BR, Demas CP. The effect of Siltex texturing and povidone-iodine irrigation on capsular contracture around saline inflatable breast implants. Plast Reconstr Surg. 93(1):123–128, discussion 129–130

[15] Wong CH, Samuel M, Tan BK, Song C. Capsular contracture in subglandular breast augmentation with textured versus smooth breast implants: a systematic review. Plast Reconstr Surg. 118(5):1224–1236

[16] Spear SL, Rottman SJ, Glicksman C, Brown M, Al-Attar A. Late seromas after breast implants: theory and practice. Plast Reconstr Surg. 130(2):423–435

[17] Adams WP , Jr, Mallucci P. Breast augmentation. Plast Reconstr Surg. 130(4): 597e–611e

[18] Stevens WG, Hirsch EM, Tenenbaum MJ, Acevedo M. A prospective study of 708 form-stable silicone gel breast implants. Aesthet Surg J. 30(5):693–701

[19] Jewell ML, Jewell JL. A comparison of outcomes involving highly cohesive, form-stable breast implants from two manufacturers in patients undergoing primary breast augmentation. Aesthet Surg J. 30(1):51–65

[20] Henriksen TF, Fryzek JP, Hölmich LR, et al. Surgical intervention and capsular contracture after breast augmentation: a prospective study of risk factors. Ann Plast Surg. 54(4):343–351

[21] Center for Devices and Radiological Health, U.S. Food and Drug Administration. FDA update on the safety of silicone gel-filled breast implants, June 2011. Available at: http://www.fda.gov/downloads/MedicalDevices/ProductsandMedicalProcedures/ImplantsandProsthetics/BreastImplants/UCM260090.pdf. Accessed July 31, 2015

[22] Caplin DA. Indications for the use of MemoryShape breast implants in aesthetic and reconstructive breast surgery: long-term clinical outcomes of shaped versus round silicone breast implants. Plast Reconstr Surg. 134(3) Suppl:27S–37S

[23] Friedman T, Davidovitch N, Scheflan M. Comparative double blind clinical study on round versus shaped cohesive gel implants. Aesthet Surg J. 26(5): 530–536

[24] Tebbetts JB. A system for breast implant selection based on patient tissue characteristics and implant-soft tissue dynamics. Plast Reconstr Surg. 109(4): 1396–1409, discussion 1410–1415

[25] Tebbetts JB, Adams WP. Five critical decisions in breast augmentation using five measurements in 5 minutes: the high five decision support process. Plast Reconstr Surg. 116(7):2005–2016

[26] Stevens WG, Nahabedian MY, Calobrace MB, et al. Risk factor analysis for capsular contracture: a 5-year Sientra study analysis using round, smooth, and textured implants for breast augmentation. Plast Reconstr Surg. 132(5):1115–1123

[27] Wiener TC. Relationship of incision choice to capsular contracture. Aesthetic Plast Surg. 32(2):303–306

[28] Bartsich S, Ascherman JA, Whittier S, Yao CA, Rohde C. The breast: a clean-contaminated surgical site. Aesthet Surg J. 31(7):802–806

[29] Tebbetts JB. Dual plane breast augmentation: optimizing implant-soft-tissue relationships in a wide range of breast types. Plast Reconstr Surg. 107(5): 1255–1272

[30] de Jong D, Vasmel WL, de Boer JP, et al. Anaplastic large-cell lymphoma in women with breast implants. JAMA. 300(17):2030–2035

[31] Spear SL, Murphy DK, Allergan Silicone Breast Implant U.S. Core Clinical Study Group. Natrelle round silicone breast implants: core study results at 10 years. Plast Reconstr Surg. 133(6):1354–1361

[32] Huang N, Liu M, Yu P, Wu J. Antibiotic prophylaxis in prosthesis-based mammoplasty: a systematic review. Int J Surg. 15:31–37

[33] Hedén P, Boné B, Murphy DK, Slicton A, Walker PS. Style 410 cohesive silicone breast implants: safety and effectiveness at 5 to 9 years after implantation. Plast Reconstr Surg. 118(6):1281–1287

[34] Murphy DK, Beckstrand M, Sarwer DB. A prospective, multi-center study of psychosocial outcomes after augmentation with Natrelle silicone-filled breast implants. Ann Plast Surg. 62(2):118–121

[35] Spear SL, Murphy DK, Slicton A, Walker PS, Inamed Silicone Breast Implant U. S. Study Group. Inamed silicone breast implant core study results at 6 years. Plast Reconstr Surg. 120(7) Suppl 1:8S–16S, discussion 17S–18S

[36] Stevens WG, Harrington J, Alizadeh K, et al. Five-year follow-up data from the U.S. clinical trial for Sientra's U.S. Food and Drug Administration-approved Silimed® brand round and shaped implants with high-strength silicone gel. Plast Reconstr Surg. 130(5):973–981

[37] Araco A, Araco F, Sorge R, Gravante G. Sensitivity of the nipple-areola complex and areolar pain following aesthetic breast augmentation in a retrospective series of 1200 patients: periareolar versus submammary incision. Plast Reconstr Surg. 128(4):984–989

[38] Okwueze MI, Spear ME, Zwyghuizen AM, et al. Effect of augmentation mammaplasty on breast sensation. Plast Reconstr Surg. 117(1):73–83, discussion 84–85

[39] Adams WP, Bengston BP, Glicksman CA, et al. Decision and management algorithms to address patient and food and drug administration concerns regarding breast augmentation and implants. Plast Reconstr Surg. 114(5):1252–1257

[40] Spear SL, Howard MA, Boehmler JH, Ducic I, Low M, Abbruzzesse MR. The infected or exposed breast implant: management and treatment strategies. Plast Reconstr Surg. 113(6):1634–1644

[41] Alderman AK, Collins ED, Streu R, et al. Benchmarking outcomes in plastic surgery: national complication rates for abdominoplasty and breast augmentation. Plast Reconstr Surg. 124(6):2127–2133

[42] Bengtson BP, Van Natta BW, Murphy DK, Slicton A, Maxwell GP, Style 410 U.S. Core Clinical Study Group. Style 410 highly cohesive silicone breast implant core study results at 3 years. Plast Reconstr Surg. 120(7) Suppl 1:40S–48S

[43] Khan UD. Incidence of Mondor disease in breast augmentation: a retrospective study of 2052 breasts using inframammary incision. Plast Reconstr Surg. 122(2):88e–89e

[44] Handel N, Cordray T, Gutierrez J, Jensen JA. A long-term study of outcomes, complications, and patient satisfaction with breast implants. Plast Reconstr Surg. 117(3):757–767, discussion 768–772

[45] Somogyi RB, Brown MH. Outcomes in primary breast augmentation: a single surgeon's review of 1539 consecutive cases. Plast Reconstr Surg. 135(1):87–97

[46] Cunningham B. The Mentor core study on Silicone MemoryGel breast implants. Plast Reconstr Surg. 120(7) Suppl 1:19S–29S, discussion 30S–32S

[47] Namnoum JD, Largent J, Kaplan HM, Oefelein MG, Brown MH. Primary breast augmentation clinical trial outcomes stratified by surgical incision, anatomical placement and implant device type. J Plast Reconstr Aesthet Surg. 66(9): 1165–1172

[48] Pajkos A, Deva AK, Vickery K, Cope C, Chang L, Cossart YE. Detection of subclinical infection in significant breast implant capsules. Plast Reconstr Surg. 111(5):1605–1611

[49] Rieger UM, Mesina J, Kalbermatten DF, et al. Bacterial biofilms and capsular contracture in patients with breast implants. Br J Surg. 100(6):768–774

[50] Adams WP , Jr, Rios JL, Smith SJ. Enhancing patient outcomes in aesthetic and reconstructive breast surgery using triple antibiotic breast irrigation: six-year prospective clinical study. Plast Reconstr Surg. 117(1):30–36

[51] Baker JL. Classification of Spherical Contractures. Presented at the Aesthetic Breast Symposium at Scottsdale, Arizona; 1975

[52] Adams WP , Jr. Capsular contracture: what is it? What causes it? How can it be prevented and managed? Clin Plast Surg. 36(1):119–126, vii

[53] Spear SL, Carter ME, Ganz JC. The correction of capsular contracture by conversion to "dual-plane" positioning: technique and outcomes. Plast Reconstr Surg. 112(2):456–466

[54] Baran CN, Peker F, Ortak T, Sensoz O, Baran NK. A different strategy in the surgical treatment of capsular contracture: leave capsule intact. Aesthetic Plast Surg. 25(6):427–431

[55] Namnoum JD, Moyer HR. The role of acellular dermal matrix in the treatment of capsular contracture. Clin Plast Surg. 39(2):127–136

[56] Maxwell GP, Gabriel A. Use of the acellular dermal matrix in revisionary aesthetic breast surgery. Aesthet Surg J. 29(6):485–493

[57] Maxwell GP, Gabriel A. Acellular dermal matrix for reoperative breast augmentation. Plast Reconstr Surg. 134(5):932–938

[58] Chasan PE. Breast capsulorrhaphy revisited: a simple technique for complex problems. Plast Reconstr Surg. 115(1):296–301, discussion 302–303

[59] Slavin SA, Lin SJ. The use of acellular dermal matrices in revisional breast reconstruction. Plast Reconstr Surg. 130(5) Suppl 2:70S–85S

[60] Lesavoy MA, Trussler AP, Dickinson BP. Difficulties with subpectoral augmentation mammaplasty and its correction: the role of subglandular site change in revision aesthetic breast surgery. Plast Reconstr Surg. 125(1):363–371

[61] Hölmich LR, Vejborg IM, Conrad C, et al. Untreated silicone breast implant rupture. Plast Reconstr Surg. 114(1):204–214, discussion 215–216

[62] McCarthy CM, Pusic AL, Kerrigan CL. Silicone breast implants and magnetic resonance imaging screening for rupture: do U.S. Food and Drug Administration recommendations reflect an evidence-based practice approach to patient care? Plast Reconstr Surg. 121(4):1127–1134

[63] Jewell M, Spear SL, Largent J, Oefelein MG, Adams WP , Jr. Anaplastic large T-cell lymphoma and breast implants: a review of the literature. Plast Reconstr Surg. 128(3):651–661

[64] Gidengil CA, Predmore Z, Mattke S, van Busum K, Kim B. Breast implant-associated anaplastic large cell lymphoma: a systematic review. Plast Reconstr Surg. 135(3):713–720

[65] Bengtson B, Brody GS, Brown MH, et al. Late Periprosthetic Fluid Collection after Breast Implant Working Group. Managing late periprosthetic fluid collections (seroma) in patients with breast implants: a consensus panel recommendation and review of the literature. Plast Reconstr Surg. 128(1):1–7

33 Breast Reconstruction with Devices

Daniel F. Haynes

Abstract
This chapter covers the sensitive area of breast reconstruction following mastectomy. Every consideration to be addressed during patient presentation is listed, and treatment options are detailed. The focus will be device-based reconstruction, utilizing tissue expanders, implants and dermal matrix. Helpful tables identify the ideal candidates for each treatment option, and actual surgical procedures for each treatment option are thoroughly covered. The chapter concludes with suggestions for postoperative care and some paragraphs on possible complications (skin necrosis, seroma, infection).

Keywords: expander, implant, MRSA screening, chemoprophylaxis, permanent prosthesis, BRCA 1 positive

33.1 Goals and Objectives

- Understand the proper evaluation of prospective breast reconstruction patients.
- Clearly define the indications for the various methods of reconstruction, and which patients are most appropriate for each modality.
- Understand the steps involved in reconstruction, and the anatomic considerations affecting each step.
- Understand appropriate perioperative care to maximize patient safety and quality outcomes.

33.2 Patient Presentation

Patients presenting for breast reconstruction will fall into one of two categories: those desiring immediate reconstruction at the time of mastectomy or those requiring a delayed reconstruction. Some patients will need reconstruction of both breasts, and for others the surgeon must try to match the native breast after a unilateral mastectomy.

With the advent of genetic testing, plastic surgeons are seeing a greater number of women desiring prophylactic mastectomy. In general, these patients are younger, healthier, and will not have the concerns arising from adjuvant chemotherapy or radiation (▶ Fig. 33.1, ▶ Fig. 33.2, ▶ Fig. 33.3).

The initial history should include information about the patient's oncologic status, including planned or on-going chemotherapy, and previous or planned radiation. For patients having immediate reconstruction, the need for radiation therapy may not be well defined at the time of surgery, and will depend on ultimate node status and pathologic examination of the surgical specimen. Additional history should include other surgeries on the breasts, such as previous biopsy or cosmetic procedures, and assessment of co-morbidities, especially cardiac or other major system disease, diabetes, and the use of anticoagulants or anti-platelet agents.

Physical assessment includes evaluation of (1) the diameter and shape of the patient's breast (or previous breast boundaries, if postmastectomy) which will predicate the base diameter of the tissue expander or implant; (2) skin quality, including the presence of stretch marks, amount of subcutaneous fat, and previous surgical scars; (3) skin laxity, including assessment of fibrosis from previous surgery or radiation therapy; (4) thickness and breadth of the underlying pectoralis muscle. The presence of previous scars, either from breast biopsies/lumpectomies, or from breast contouring (i.e., mastopexy), can affect the underlying blood supply, and hence reliability of the skin flaps. Examination should be done in both the supine and sitting positions, to assess the mobility of the skin, areas of redundancy, and thickness of the subcutaneous fat, which in delayed reconstruction, is not always uniform. The hands-on-hips position is beneficial to asses skin redundancy in the lateral chest, variously known as the lateral thoracic fold or lateral chest roll.

Previous radiation therapy does not preclude the use of an expander, but such patients should be approached on a case by case basis, and apprised of the greater risk of complications and implant loss.[1,2] Patients vary in their response to radiation, and the tissue quality should be carefully assessed. If the skin is noticeably tighter and thinner than the un-radiated side, then consider autologous tissue, either as the sole reconstructive technique, or to overlay the expander. If the skin is mobile and appears healthy, and the muscle is pliable and of good caliber, then muscle fibrosis is usually minimal, and subpectoral expander placement may be considered. The patient should be advised that autologous tissue could be needed as a secondary procedure if complications are encountered, or if the tissue will not expand sufficiently.

Fig. 33.1 (a,b) Patient with right breast cancer, well suited to areola-sparing mastectomy and immediate reconstruction.

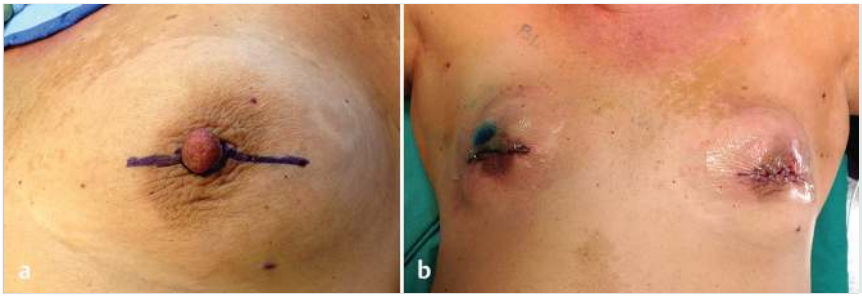


Fig. 33.2 (a,b) Same patient as in ▶ Fig. 33.1, showing preoperative marking, and preservation of the areolae at completion of bilateral mastectomy.

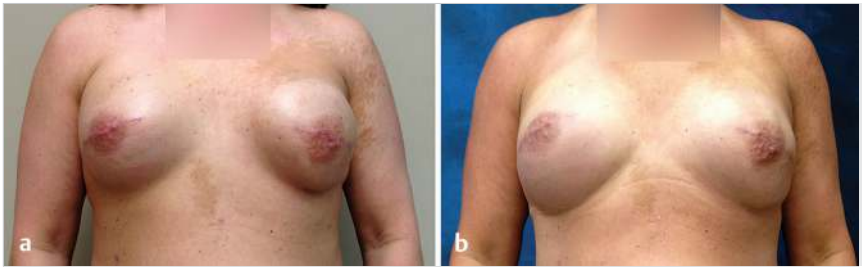


Fig. 33.3 Same patient, showing **(a)** expanders inflated and **(b)** after exchange for cohesive silicone implants.

33.3 Preparation for Surgery

Diagnostic data required in preparation for surgery is similar to other elective major procedures and is dependent on age, the presence of comorbidities, and requirements of the surgical center in which the procedure will be performed. In general, the author requires a baseline hemoglobin and hematocrit, and basic chemistry on most patients. Diabetic patients should have good glucose control, and a hemoglobin A1c within normal range. Patients who have recently undergone chemotherapy should have a total neutrophil count (TNC) > 1,500. Cardiac workup is dependent on patient age, history and the facility in which the procedure is being done. In general, if an electrocardiogram is abnormal, internal medicine or cardiology evaluation should be obtained. Consider nasal MRSA screening, or empiric decolonization with mupirocin.[3] The patient is asked to bathe daily with a chlorhexidine solution, and to use chlorhexidine mouthwash, beginning five days prior to surgery.

33.4 Treatment

Treatment Options and Indications:

The choices for surgical treatment fall into two categories, immediate or delayed reconstruction:

1. *Immediate reconstruction with tissue expander.* If the surgeon performing the mastectomy usually leaves healthy, well-vascularized skin flaps, then immediate placement of a tissue expander is performed (▶ Table 33.1).
2. *Immediate, direct to implant reconstruction.* If the skin flaps appear reliable, and the patient desires the same or smaller breast size, it may be feasible to place the permanent implant at the time of the mastectomy (▶ Table 33.2).
3. *Immediate reconstruction with autologous tissue and underlying expander.* In a patient with less optimal tissue quality, especially if previously irradiated, a latissimus flap will provide additional skin envelope, and a healthy, well vascularized layer over the expander (▶ Table 33.3).

Table 33.1 Ideal candidate for breast reconstruction with tissue expander

Clinical feature	Description
Body type	Fit, but can be mildly overweight; risks increase with increasing BMI. Consider delayed reconstruction or flap, with BMI > 35[4,5]
Chest contour	Normal, without excessive carinatum or excavatum deformity
Skin quality	Adequate laxity must be present to accommodate the underlying expander or implant, without compromising wound closure or tissue perfusion
Psychology	The patient should be psychologically stable. If the patient has recently been diagnosed with cancer, she should be emotionally able to undertake reconstruction, and have adequate social support from family and friends to assist in postoperative care and recuperation It is very helpful if the patient has previous experience with reconstruction in friend or family member
Smoking	While reconstruction can be undertaken in patients who smoke, the risk of complications is increased.[1] The author requires all patients to stop smoking prior to breast reconstruction
General health	The patient should be counseled on the benefits of a balanced diet and regular exercise

Table 33.2 Ideal candidate for direct to implant reconstruction

Clinical feature	Description
Body type	Small to moderate breast size, and wanting to be the same size or smaller
Skin quality	Good, with adequate elasticity
Smoking	Nonsmoker

Table 33.3 Candidates who would benefit from the addition of autologous tissue

Clinical feature	Description
Body type	1. Thin, with minimal subcutaneous fat 2. Small to moderate breast size, and wanting to be significantly larger
Skin quality	Fibrotic or tight, especially if previously radiated
Smoking	Patients who are unable to quit smoking may benefit from the increased vascularity provided by a latissimus myocutaneous flap

Table 33.4 Ideal candidate for areola-sparing mastectomy and reconstruction

Clinical feature	Description
Body type	Small to moderate breast size; desire for larger size after reconstruction can be accommodated Minimal to moderate ptosis Fairly symmetrical, or willing to tolerate postoperative disparity in nipple position
Skin quality	Good, with adequate elasticity
Smoking	The areola is a watershed region in terms of vascularity, and may not survive in patients who smoke

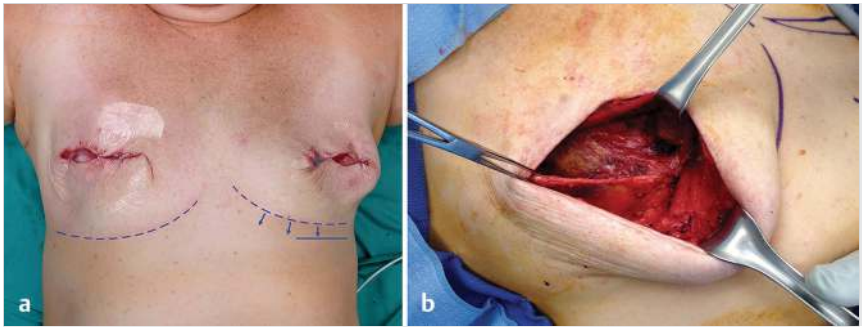


Fig. 33.4 Patient undergoing bilateral submuscular reconstruction with tissue expander. **(a)** Note the higher inframammary fold on the patient's left. **(b)** the pectoralis is retracted superiorly, and the serratus and overlying fascia have been elevated in an inferolateral direction.

4. *Delayed reconstruction*. If the patient's health is not optimal or if the skin flaps after mastectomy are of questionable viability, then it is in the patient's interest to defer the reconstruction to a later date. In the case of a planned immediate reconstruction, but poor quality skin flaps, proceed to verify hemostasis, use appropriate antisepsis, and close the wound gently over a drain. The subsequent loss of a portion of the skin envelope will not jeopardize an underlying device, and chemotherapy will not be delayed for treatment of a device infection. Some surgeons may proceed after objective assessment of the skin flap blood supply via fluorescence or intraoperative angiography, with appropriate resection of compromised skin. The author does not favor this approach, since it does not fully remove the risk of poor healing and subsequent loss of the device and delays in adjuvant therapy (▶ Table 33.4).
5. Immediate or delayed reconstruction with autologous tissue alone. Patients with appropriate body habitus, or a reluctance to use implants, may be reconstructed with either pedicled or free tissue transfer.

33.5 Surgical Preparation

Most patients undergoing mastectomy and immediate reconstruction will stay one night in hospital. The occasional stoic, small breasted, unilateral patient may choose surgery on an outpatient basis. Effective control of postoperative nausea is beneficial, as the straining associated with retching may tear freshly repaired tissue, and increase the risk of postoperative bleeding. Preoperative medications such as ondansetron (Zofran) and aprepitant (Emend) have proven efficacy, as does transdermal scopolamine. Continuous mechanical intraoperative DVT prevention via sequential compression boots is mandated in all breast reconstruction surgeries done under general anesthesia. A DVT risk assessment should be completed preoperatively, and chemoprophylaxis should be used if indicated. The longer operative times associated with more complicated reconstructions will skew the analysis toward adding chemoprophylaxis.

Preoperative marking may be done in either the sitting or the standing position. The author marks the midline and inframammary fold, and also the lateral extent of the breast, provided it is well defined. For immediate reconstruction, the mastectomy surgeon may be willing to place these marks preoperatively. Some surgeons will efface the inframammary fold during the mastectomy, so preoperative marking is beneficial.

For immediate reconstruction, it is the author's practice to have the mastectomy surgeon verify hemostasis, close the wound loosely with staples, and then apply a Tegaderm or OpSite dressing (▶ Fig. 33.4a). The patient is then sterilely reprepped and re-draped, and new instruments are used for the reconstruction. Antibiotics may be redosed, depending on time interval since the initial dose. In a unilateral reconstruction, it is helpful to prep the uninvolved breast into the field, to allow better assessment of symmetry. Covering the exposed nipple with a Tegaderm will isolate bacteria residing in the milk ducts.

33.6 Operative Technique—Expander

33.6.1 Immediate Reconstruction with Tissue Expander

In a bilateral reconstruction, the author prefers to begin on the breast cancer side. If the tissue quality differs appreciably between sides, it will more likely be impaired on the side where

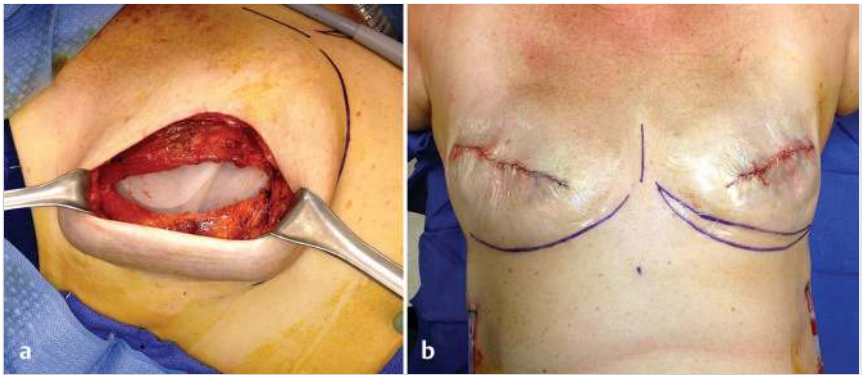


Fig. 33.5 Same patient as in ▶ Fig. 33.4. **(a)** The expander is in place beneath the muscle. **(b)** Immediate postoperative appearance.

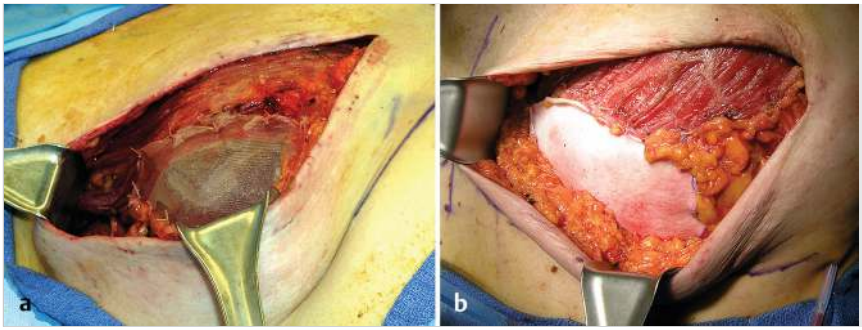


Fig. 33.6 **(a)** use of Vicryl mesh, bridging between the pectoralis and serratus. **(b)** similar use of acellular dermal matrix.

the cancer resection was performed. This allows the surgeon to judge the most appropriate tissue expander or implant, and to determine the need for allograft or autologous tissue. Both sides can be approached simultaneously, alternating from side to side, however this adds operative time, and with experience becomes less useful. Cover the opposite nipple with Tegaderm in unilateral reconstruction.

The mastectomy wound should be irrigated clean, if needed, and reverified for hemostasis. Bleeding points are often present on the deep surface of the skin flaps, and these should be controlled completely but carefully. Infiltration of local anesthetic is then performed to help with postoperative pain relief. The author injects the medial pectoralis, the serratus along the mid-axillary line, and the body of the pectoral muscle, which helps to decrease postoperative muscle spasm (▶ Fig. 33.4, ▶ Fig. 33.5).

Assess the skin flaps for quality and vascularity. If the skin flaps are not adequate in extent or in perfusion, then the reconstructive surgeon may simply close the wound over a drain, and perform the reconstruction in a delayed fashion. Keep in mind that the ultimate objective is a satisfied patient, with a durable, healthy reconstruction. It is worth an extra surgery to obtain this goal, if the skin coverage is not adequate for an immediate procedure.

If the inframammary fold (IMF) has been effaced, it should be reattached for better postoperative definition. Use the minimum number of sutures necessary, to avoid impairing perfusion in the inferior skin flap. At this point, a preliminary decision is made regarding the need for acellular dermal matrix (ADM) or absorbable mesh.[6] If the pectoralis major muscle is of good caliber, extends to the inframammary fold, and there is adequate soft tissue in the serratus and its overlying tissue, then total muscle coverage is performed (▶ Fig. 33.4b). There is published clinical data supporting a slight decrease in infection risk with total sub-muscular placement, as compared to ADM (▶ Fig. 33.6).[3]

The pectoral muscle is elevated, using a lighted retractor for visualization. If the inferior border of the pectoralis is high relative to the IMF, then ADM or absorbable mesh is used to hold the muscle out to length (▶ Fig. 33.6). If the inferior border of the muscle is at or below the IMF, then the muscle is left attached to the overlying skin flap, but is released from its origin on the ribs, to facilitate better expansion of the lower pole. If the serratus is to be elevated, this is done from medial to lateral, dissecting far enough to accommodate the footprint of the desired expander. Perforating vessels from the intercostal arteries can produce troublesome bleeding, and should be carefully controlled. Transection of nerve branches is avoided if possible. The author washes the wound clean at this point, first with chlorhexidine irrigation (Irrisept), and then with triple antibiotic solution.

A variety of expanders are available, with most surgeons choosing a device with an integrated port. Securing the position of smooth surfaced expanders with suture tabs is necessary to prevent rotation of the expander during the period of inflation. The expander is brought onto the field, and checked for leaks. Many surgeons change gloves before handling the expander (or implant), and it is good practice to minimize the number of other scrubbed team members who touch the device. Aspiration of most of the air from the expander will facilitate insertion. The device is checked for correct orientation, and secured with at least one suture tab. Expanders may have a tendency to migrate laterally during inflation, and this can be limited if a medial suture tab is well secured to the underlying tissue.

The device is rinsed with antiseptic or antibiotic after insertion, and if the serratus is utilized, it is sutured to the lateral pectoral edge with horizontal mattress sutures of 2–0 Vicryl. ADM or absorbable mesh may be inset at this point along the inferolateral border of the expander. Port position is verified with the supplied magnet, and an initial saline fill is performed. Consider the quality of the skin flaps when deciding the

amount of initial fill volume, as undue tension will impair circulation in the skin.

Prepectoral placement of the expander is an option for patients with well vascularized skin flap. Acellular dermal matirx is used as a full wrap or anterior coverage of the device. Suturing the dermal matrix, as well as the suture tabs to the pectoralis guards against device rotation. Advantages of prepectoral positioning include less postoperative pain due to reduced surgical dissection and less animation deformity in the long term. However, secondary fat grafting will be necessary in many patients due to the thinner tissue coverage of the final device.

If the lateral thoracic skin has been detached from the chest wall over a wide area, it is beneficial to redrape this against the underlying serratus, and secure with several sutures. At this level, the long thoracic nerve has usually arborized so that it is not at risk. If the sutures are tied loosely there is less likely to be visible skin puckering postoperatively.

In immediate reconstruction, at least one closed suction drain is utilized. In patients undergoing axillary node dissection, a second drain is often placed to drain the axilla. In selected patients having delayed reconstruction, the drain may be omitted if the wound is quite dry, and there is not a large amount of free space that would benefit from suction coaptation. Tunneling the drain through 3 to 4 cm of subcutaneous fat before exiting the skin may decrease the risk of bacterial contamination ascending along the drain tract. The drain is secured to the skin with a double suture of 2–0 silk.

The skin flaps are then redraped, and a final assessment made of skin viability. Any questionable areas should be excised to good dermal bleeding. Significant dog-earing is also contoured at this time. Tissue approximation is done in layers with absorbable tissue. Several tacking sutures to the outer surface of the pectoralis are useful to stabilize the closure, decrease shearing, and lower the risk of seroma. The author uses 2–0 Vicryl in the subcutaneous fat, 3–0 Vicryl in the deep dermis, and subcuticular 4–0 Monocryl in the skin. After skin closure, the incision and drain exit sites are painted with either betadine or chlorhexidine. The author injects 20 mL of 0.25% bupivacaine through the drain tube after skin closure. The suction bulbs are attached, but not placed to suction until the recovery room, allowing time for topical action of the bupivacaine.

Some surgeons prefer a bulky gauze dressing, however it is the author's practice to use Tegaderm or Opsite on the incisions, with an Ace wrap or surgical bra placed directly over the transparent dressing. If an Ace wrap is used, it will need to be re-wrapped at least once daily, and this provides an opportunity for the patient or family member to examine the wound, and report any erythema or other concerns. The Ace wrap should be wrapped comfortably snug, but not tight, sufficient to decrease motion of the expander, and hence postoperative pain, and decrease the risk of venous bleeding. Tight external wrapping could adversely affect skin flap vascularity. The urinary catheter is removed before awakening, to encourage postoperative ambulation.

33.7 Direct to Implant Reconstruction

The surgical procedure is similar to placement of the tissue expander. Precise dissection of the implant pocket is more critical, since there is no planned second step to allow correction of discrepancies. A larger portion of ADM or mesh may be utilized to accommodate the volume of the desired implant. The use of temporary sizers is advisable, both to judge tension on the skin closure before placing the permanent implant, and in bilateral procedures to allow selection of the most appropriate implants for symmetry. Often the cancer side of a bilateral procedure has undergone a more aggressive mastectomy, resulting in less residual tissue remaining. Therefore two different implant sizes may be needed to give the best visual balance.

33.8 Delayed Reconstruction with Tissue Expander

Bilateral: Expander insertion is similar to immediate reconstruction. Preoperative attention should be directed to any asymmetries in the skin envelope, as it is helpful to address these during placement of the expander. Careful preprocedure marking is therefore needed. Significant dog-ears or unilateral skin excess should be tailored at this stage—the subsequent expansion will likely be more uniform, and any touchups that are required to the skin envelope at the second stage will be more apparent.

The tissue expander is usually placed beneath the pectoral muscle, unless the skin flaps are unusually thick and healthy. If the lateral skin is thin, then the expander may be placed beneath the serratus also.

Some patients will have considerable sclerosis on the outer aspect of the pectoralis, and occasionally a mature seroma cavity is found between the muscle and overlying skin. This should be released through cross-hatch scoring, or by removal of the serosal wall, to avoid interference with expansion. The author finds that in most patients a drain is unnecessary, provided the wound is dry. If ADM is used, then a drain is recommended.

Unilateral: It is helpful to perform contralateral symmetry procedures, such as a reduction or mastopexy, at the same time as placing the delayed expander. This allows the contralateral breast to heal well and assume its final form during the process of expansion. The extent of expansion may thus be tailored to the size of the contralateral breast, and the most appropriate implant can be selected for size and shape match.

33.9 Expansion

A period of initial healing as long as several weeks is allowed before starting to stretch the muscle and skin. The expansion process may take several months, depending on tissue coverage, elasticity, and the required volume. Ideally, expansion should be done quickly enough that minimal scar tissue can form around the expander, but slowly enough that the patient is spared undue discomfort. A fill volume of 60 to 100 mL at each expansion works well for most patients.

Expansion can be continued during chemotherapy. It is the author's practice to inflate the expander several days prior to the next dose of chemotherapy, when the white blood count is near its peak. Expansion should not be done during radiotherapy, as shape change in the breast will alter the radiation dose calculations. Avoid overstretching the pocket if planning use of a shaped prosthesis, as a pocket that is too lax will allow the implant to rotate out of correct orientation.

33.10 Replacement with Permanent Prosthesis

One of the benefits of expander based reconstruction is that the surgeon has two opportunities to shape the breast. At the second stage, the expander is removed, and the permanent implant inserted. Take full advantage of this procedure to adjust the implant pocket, the overlying muscle, and the skin envelope to achieve the best symmetry. In a unilateral reconstruction, the patient is often amenable to additional adjustments to the contralateral breast. If a reduction or lift was previously performed, slight adjustments to the nipple position or additional skin tightening along the previous incisions can be quite helpful in better matching the reconstructed breast.

The expander is usually approached through the existing mastectomy scar. Opening the muscle in the direction of its fibers will help preserve vascular supply to the muscle. The tissue expander is deflated and removed, and the pocket inspected with a lighted retractor. Adjustments to the pocket are made as needed, and may include raising or lowering of the IMF, scoring incisions through the capsule, partial removal of the capsule, or capsulorrhaphy to help hold the implant in the desired position. If using a shaped implant, it is useful to have some raw surface within the pocket for the implant to adhere to, to help prevent a rotation deformity. This may be achieved either with partial capsulectomy, multiple scoring incisions, or punctate cautery marking. The surgeon should have a lower threshold for using drains with shaped implants, as fluid accumulation within the pocket can float the implant, and allow malrotation.

The pocket is rinsed with antiseptic and/or antibiotic solution, and verified for hemostasis. Temporary sizers are very useful in selecting the appropriate implant size. As with the expander, minimize the number of personnel who handle the device (ideally only the surgeon), and minimize contact with the skin. Smooth implants may be inserted using the Keller funnel.

Once the implant is inserted, the author performs a temporary closure of the muscle and skin, and the patient is brought to the semi-sitting position to check symmetry. Additional modifications can then be made if needed, and may include fat injection into the subcutaneous tissue to address contour discrepancies. There is some evidence that fat injection may improve tissue quality in radiated breasts.

Once maximal symmetry has been obtained, the pockets are closed in layers, using drains only if necessary. Ten cc of local anesthetic added to the pocket before closure will decrease postoperative pain. If the pockets are quite dry, it is the author's preference to utilize a surgical bra for support. Otherwise, an Ace wrap is applied for additional compression.

33.11 Postoperative Care

The compression wrap is adjusted and reapplied daily. For outpatient procedures, the patient is discharged home after postanesthesia criteria are met. For a bilateral mastectomy, the patient is usually discharged within 24 hours, although occasional patients may need a longer stay for pain management. Surgical drains are removed when output is less than 30 mL/day for 2 consecutive days, with compression wrapping or sports bra continued for several days after drain removal. The patient is instructed to avoid any heavy lifting or repetitive shoulder activity (such as vacuum cleaning) for one month after surgery. Patients undergoing bilateral mastectomy and reconstruction can usually return to office-based work in 2 weeks. Occupations requiring significant upper body motion should wait until one month after surgery.

Postoperative pain relief is provided in multi-modality fashion, using a mixture of narcotic and nonsteroidal anti-inflammatory agents. Narcotic analgesia is provided in the form of hydrocodone or oxycodone, supplemented in hospital with low-dose hydromorphone (2–4 mg) for breakthrough pain. Low-dose diazepam (2 mg) is helpful for muscle spasm in the pectoralis, which can be quite uncomfortable. If the patient can tolerate NSAIDs, such as ibuprofen, it is good practice to have the patient alternate doses between narcotic and nonsteroidal. Postoperative nausea is addressed with promethazine or ondansetron. The scopolamine patch, if used, can be left on for 24 hours postoperatively—if left on longer, the patient will likely experience blurred vision.

DVT chemoprophylaxis should be continued postoperatively if indicated. SCDs are maintained on all patients until discharge, and the nursing staff is asked to ambulate the patient in the hall the evening of surgery. Patients are encouraged to ambulate hourly during the day once at home.

33.12 Complications in the Immediate Postoperative Period

Skin necrosis: Small areas of nonviable skin along the incision margin, also known as edge necrosis, may be managed conservatively if extending 5 mm or less from the incision edge. If kept dry, and painted with betadine or chlorhexidine, the edge necrosis will often form a scab, and eventually separate with healed tissue underneath. If the impaired skin is greater in extent, and still appears viable but in jeopardy, then hyperbaric oxygen may be of benefit. Larger areas of nonviable skin should be debrided expeditiously and reclosed under sterile technique. Removing fluid from the expander may be necessary to aid reclosure.

Seroma: Seroma is best prevented rather than treated—leave the drain if possible until the output is less than 30 mL/day, and maintain counter-pressure for several days after drain removal. Adding fluid to the expander may speed resolution of the seroma, and the seroma fluid may be safely drained as the needle is withdrawn from the integrated port.

Infection: There is no consensus on the duration of postoperative antibiotic therapy, although most surgeons will keep the patient on oral antibiotics until drains are removed. Any new area of erythema should be treated immediately. If the patient has completed the postoperative antibiotic prescription, then antibiotics should be resumed; if still taking the original antibiotic, then either change antibiotics or add a second with broader coverage. Radiated breasts may benefit from a longer duration of antibiotic coverage than is used for non-radiated patients.

33.13 Outcomes

There is considerable variation in the incidence of major complications as reported in national studies; however a large

meta-analysis found total complication incidence for submuscular reconstruction of 14%; with risks of 3.5% for seroma, 4.7% for infection, and 4.9% for flap necrosis, and similar but slightly higher complication rates when using ADM (▶ Fig. 33.7).[7]

Watchful waiting may be acceptable in some aspects of medicine, but is seldom wise in breast reconstruction. If the underlying implant becomes infected, it will require removal—therefore, potential complications should be managed more quickly and aggressively than in patients without an implanted device.

Patients who have undergone radiation therapy, whether pre- or postoperative, experience a higher incidence of both immediate and long-term complications (infection, implant loss, and capsular contracture) and in many cases, an adverse effect on aesthetic outcome (▶ Fig. 33.7).[8,9]

Breast reconstruction provides immediate and sustained psychological benefits to women facing the challenge of mastectomy for breast cancer.[10] Additionally, studies have shown that reconstruction does not affect the risk of breast cancer recurrence, whether local or distant, and does not interfere with the detection of recurrence.[11] Studies have also demonstrated a normalization of body posture when a missing breast is reconstructed.[12]

Restoring the normal anatomy can lessen the burden of breast cancer, and allow a woman to go about day-to-day life without the constant reminder of mastectomy scars, or an ill-fitting external prosthesis (▶ Fig. 33.8). Some of the happiest patients the author has seen are breast cancer patients, post mastectomy and reconstruction, who are able to go to the pool or beach with their children or grandchildren, and are able to enjoy family time while looking and feeling normal.

Fig. 33.7 Sequelae of radiation therapy. Note the thinner appearance of the skin and the multiple telangiectasias on the radiated left chest.

33.14 Review Questions

33.14.1 True or False

1. Unilateral mastectomy and reconstruction may be performed on an outpatient basis.
2. Chemoprophylaxis with heparin is contraindicated in breast reconstruction surgery due to high risk of hematoma.
3. Patients who smoke should be considered either for delayed reconstruction or for the use of autologous tissue.

33.14.2 Choose the Best Answer

4. A 42-year-old woman with a family history of breast cancer, has recently been screened and is BRCA 1 positive. She is interested in bilateral mastectomy and reconstruction. She wears a B cup bra, but would like to be a C cup after surgery. Her body mass index is 26, there is minimal lower abdominal fat, and she does not smoke. Which of the following are viable treatment options?
   a) Nipple or Areola-sparing mastectomy and tissue expander reconstruction.
   b) Bilateral pedicled TRAM flap.
   c) Latissimus flap without underlying implant.
   d) Direct to implant reconstruction.
5. Postoperative pain management
   a) Will benefit from the addition of a benzodiazepine to reduce muscle spasm.
   b) Should avoid non-steroidal agents due to the risk of bleeding.
   c) Should include an anti-emetic to reduce nausea.

33.14.3 Answers

1. True.
2. False.
3. True.
4. a. Nipple or Areola-sparing mastectomy and tissue expander reconstruction.
5. a. Will benefit from the addition of a benzodiazepine to reduce muscle spasm AND c. Should include an anti-emetic to reduce nausea.

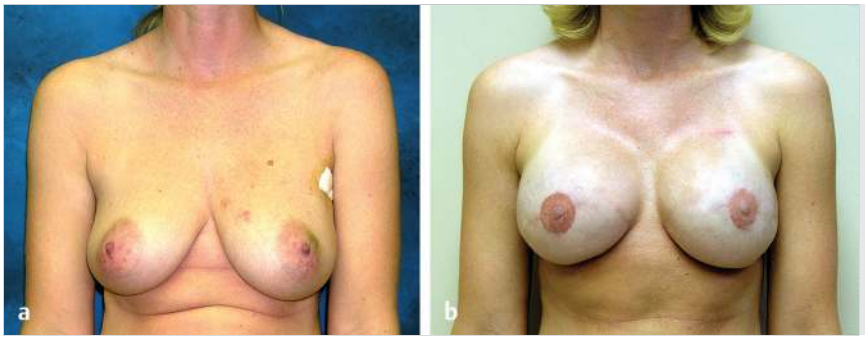


Fig. 33.8 **(a)** preoperative photo, prior to bilateral mastectomy and reconstruction using tissue expanders and Vicryl Mesh. **(b)** postoperative photo, after replacement with round silicone gel prostheses and nipple reconstruction.

References

[1] Sbitany H, Wang F, Peled AW, et al. Immediate implant-based breast reconstruction following total skin-sparing mastectomy: defining the risk of preoperative and postoperative radiation therapy for surgical outcomes. Plast Reconstr Surg. 134(3):396–404

[2] Baschnagel AM, Shah C, Wilkinson JB, Dekhne N, Arthur DW, Vicini FA. Failure rate and cosmesis of immediate tissue expander/implant breast reconstruction after postmastectomy irradiation. Clin Breast Cancer. 12(6):428–432

[3] Craft RO, Damjanovic B, Colwell AS. Evidence-based protocol for infection control in immediate implant-based breast reconstruction. Ann Plast Surg. 69 (4):446–450

[4] McCarthy CM, Mehrara BJ, Riedel E, et al. Predicting complications following expander/implant breast reconstruction: an outcomes analysis based on preoperative clinical risk. Plast Reconstr Surg. 121(6):1886–1892

[5] Hanwright PJ, Davila AA, Mioton LM, Fine NA, Bilimoria KY, Kim JY. A predictive model of risk and outcomes in tissue expander reconstruction: a multivariate analysis of 9786 patients. J Plast Surg Hand Surg. 47(6):513–518

[6] Haynes DF, Kreithen JC. Vicryl mesh in expander/implant breast reconstruction: long-term follow-up in 38 patients. Plast Reconstr Surg. 134(5):892–899

[7] Kim J, Davila AA, Persing S, et al. A meta-analysis of human acellular dermis and submuscular tissue expander breast reconstruction. Plast Reconstr Surg. 129(1):28–41

[8] Sbitany H, Wang F, Peled AW, et al. Immediate implant-based breast reconstruction following total skin-sparing mastectomy: defining the risk of preoperative and postoperative radiation therapy for surgical outcomes. Plast Reconstr Surg. 134(3):396–404

[9] Ho AL, Bovill ES, Macadam SA, Tyldesley S, Giang J, Lennox PA. Postmastectomy radiation therapy after immediate two-stage tissue expander/implant breast reconstruction: a University of British Columbia perspective. Plast Reconstr Surg. 134(1):1e–10e

[10] Atisha D, Alderman AK, Lowery JC, Kuhn LE, Davis J, Wilkins EG. Prospective analysis of long-term psychosocial outcomes in breast reconstruction: two-year postoperative results from the Michigan Breast Reconstruction Outcomes Study. Ann Surg. 247(6):1019–1028

[11] Reddy S, Colakoglu S, Curtis MS, et al. Breast cancer recurrence following postmastectomy reconstruction compared to mastectomy with no reconstruction. Ann Plast Surg. 66(5):466–471

[12] Peres AC, Latorre MD, Maesaka J, Filassi JR, Baracat E, Ferreira E. Body posture after mastectomy: comparison between immediate breast reconstruction versus mastectomy alone. Physiother Res Int.

34 Breast Reconstruction with Free Flaps

Nasim Abedi and Steven Bernard

Abstract
This chapter addresses the subject of breast reconstruction based on free tissue transfer. Preoperatively, the authors stress the importance of the patient factors that will facilitate the selection of the right reconstructive option from the many that are available to the surgeon. The thorough review offered in this chapter discusses the optimum time for reconstruction, diagnostics (CTA, MRA, ultrasound), and the advantages and disadvantages of abdominal-based, free TRAM, muscle-sparing free TRAM, deep inferior epigastric artery perforator, Rubens, gluteal-based, superior and inferior gluteal artery perforator, thigh-based, profunda artery perforator, and transverse fascia lata TFL myocutaenous flaps. Internal mammary and thoracodorsal vessels are also covered. Counsel involving postoperative care and potential outcomes concludes the study.

Keywords: abdominal-based flaps, gluteal artery based reconstruction, free TRAM, Rubens flap

34.1 Goals and Objectives

- Understand the pros and cons of each type of breast reconstruction based on free tissue transfer.
- Understand the advantages, disadvantages, and relative contraindications of each type of flap.
- Understand the preoperative planning for the procedures.
- Know the evidence-based quality outcomes to maximize patient safety and aesthetics.

34.2 Patient Presentation

Restoring the appearance, shape, and texture of breast after mastectomy has shown to improve quality of life and psychological well-being of patients after breast reconstruction. These procedures are broadly divided into two groups: alloplastic reconstruction using tissue expander and/or implants and autologous reconstruction with pedicled or free tissue transfer. Autologous breast reconstruction offers many advantages over implant based reconstruction. It provides superior feel and appearance to the reconstructed breast, helps restore the breast's natural shape and ptosis, has lifetime durability requiring fewer revision surgeries, and ages with the patient. This chapter focuses on the microsurgical options available for breast reconstruction.

With the multitude of options available today for breast reconstruction, patient factors come to play an integral part in individualizing the type of reconstruction. A thorough history and physical exam as well as an understanding of patient's expectations are therefore essential.

Certain patient characteristics affect the decision to proceed with microsurgical breast reconstruction. The factors affecting outcomes can be broadly separated into body habitus, breast shape, comorbidities and the cancer treatment plan. Comorbidities such as coronary artery disease, obesity (body mass index [BMI] > 30), clotting disorders, smoking history, and diabetes, increase the complication rates of free flap surgery. The ideal candidate for microsurgical breast reconstruction is a younger (< 65) patient who is otherwise healthy, does not smoke, has no history of clotting abnormality, has adequate donor tissue, and does not require postoperative radiation therapy.

That being said, patients who do not possess these ideal criteria are the vary patients who may benefit from the advantages of free tissue transfer over implants. Generally, if the patient is less than ideal for autologous methods as listed above, they are an even worse candidate for implants. As implants are limited to approximately 800 mL except in rare cases, breasts larger this of this volume are poor candidates for implant reconstruction. As a result of our experience, we take the patient's comorbidities into account and minimize their effects as best we can and then consider them relative contraindications. Patients who smoke are encouraged to quit prior to semi-elective immediate reconstruction and asked to quit prior to elective or delayed procedures. Patients with large breast size and asymmetry undergo procedures to improve that symmetry such as mastopexy and reduction.

If patient is deemed to be a candidate for microvascular breast reconstruction, the appropriate donor site is selected based on the breast volume and skin requirements, breast size and shape, unilateral versus bilateral reconstruction, patient's size expectations, and availability of donor sites. The abdomen has become the preferred donor site for most patients if sufficient tissue is available to achieve the desired breast volume. When abdominal tissue is not a suitable option either due to paucity of fat or previous abdominal surgeries precluding sufficient tissue transfer, the gluteal or thigh region are considered.

34.2.1 Timing of Reconstruction

Timing of reconstruction in relation to adjuvant radiation therapy remains a topic of controversy. However, the majority of surgeons agree that microsurgical tissue transfer should be performed at 3 to 6 months after adjuvant radiation therapy is completed.[1] This is because radiation damage can cause significant fibrosis and deformation of reconstructed breast. It is generally recommended to consider delayed immediate reconstruction if suspicious of requiring radiation therapy.

Patients with a BMI over 32 are encouraged to lose weight prior to undergoing abdominally based procedures. Where possible nutrition is maximized, diabetes controlled and hypercoagulable states are minimized. Literature suggests that when possible, adjunctive procedures such as reconstruction should be delayed at least 6 weeks postchemotherapy and Tamoxifen have been shown to increase the risk of flap complications.[2]

34.3 Preparation for Surgery

34.3.1 Preoperative Imaging

With development of perforator flaps, the need for assessment of vascular architecture of the flap has become an important part of operative planning. Preoperative imaging has evolved over the past decade to allow for identification and localization of appropriate perforators, especially in abdominally based free flap reconstruction. Modalities used today include Doppler ultrasonography and color duplex sonogram, computed tomographic angiography (CTA), and magnetic resonance angiography (MRA).

34.3.2 Duplex Ultrasound

Doppler ultrasound was one of the first tools used for perforator mapping through the cutaneous territory of the flap. It is also the instrument used by most surgeons intraoperatively and postoperatively to monitor the flap. In case of deep inferior epigastric perforator (DIEP) and transverse rectus abdominis myocutaneous (TRAM) flaps, the majority of dominant perforators have been shown located in the periumbilical area using ultrasound. Color duplex ultrasound can further provide information on the caliber, flow, direction, and velocity of these perforators. In their study Heitland et al showed that TRAM flap has the highest blood flow and velocity followed by DIEP and superior gluteal artery perforator (SGAP) flaps.[3] However, ultrasonography does not provide information on the course and three-dimensional details of the perforator systems. It has been shown to be associated with a high rate of false-positive results and a considerable rate of false-negative results. In their comparative study, Rozen et al demonstrated that CTA was superior to Doppler ultrasound at identifying the course of DIEA and its branching pattern and perforators. Furthermore, it removed the subjective error associated with Doppler ultrasonography.[4,5]

34.3.3 Computed Tomographic Angiography

Although not all high volume practices utilize preoperative imaging, specific anatomic knowledge of the blood vessels can help to plan for variables such as adequate donor size, perforator position and recipient adequacy. CTA has become the gold standard for preoperative imaging of perforator flaps. It has been shown to be accurate in assessing the perforator's caliber and location and their course through the muscle. Most surgeons use CTA for assessment of abdominal flaps and most recently for thigh-based and gluteal flaps. It helps the surgeon make a decision on performing a muscle sparing TRAM versus a DIEP flap, as well as assess the patency of deep and superficial inferior epigastric arteries specially in patients who have had previous abdominal surgeries. Paramedian abdominal scars have been shown to cause the most damage to the DIEA, superficial inferior epigastric artery (SIEA) vessels and the perforators, while laparoscopic incisions cause the least. Casey et al showed that preoperative CTA improved operative efficiency and reduced the incidence of abdominal bulge in DIEP breast reconstruction (▶ Fig. 34.1).[6,7]

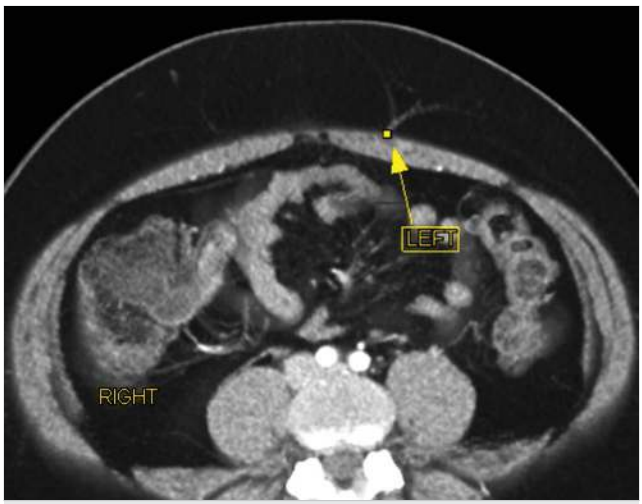


Fig. 34.1 Computed tomography angiography of the abdomen demonstrating a large perforating vessel originating from a medial branch of the deep inferior epigastric artery. The exact position can be determined using this technique.

34.3.4 Magnetic Resonance Angiography

MRA allows reliable visualization of abdominal wall perforators of 1-mm diameter without exposing patients to ionizing radiation or iodinated intravenous contrast. Furthermore, although CTA yields higher spatial resolution, MRA has greater contrast resolution, which allows detection of perforators that may be missed by CTA. Another advantage of MR imaging is that due to absence of ionizing radiation, multiple image acquisitions can be performed after administration of gadolinium-based contrast, resulting in improved ability to obtain images at most optimal times. In this way, patient-related factors can be eliminated.

Gluteal and thigh perforators can also be assessed using MRA imaging. It can help the surgeon identify the location and course of the perforators.[8]

34.3.5 Treatment

Abdominal-Based Flaps

Abdominal-based flaps are the method of choice for autologous breast reconstruction. One of the reasons for this preference is that most women who require breast reconstruction are at an age where they tend to accumulate excess tissue in the lower abdomen. The excess tissue provides an ideal donor site with the added benefit of improved abdominal contour resembling that of an abdominoplasty. Abdominal based flaps include those based on the deep inferior epigastric artery system (the free TRAM flap, muscle sparing free TRAMs, DIEP flap), SIEA flap and the external iliac perforator flap.

Compared to a standard pedicled TRAM, the microvascular flaps have been shown to have lower abdominal morbidity and all offer improved flexibility at the time of inset. Counterintuitively, the flap specific morbidity is lower as well. This may be a result of the typically greater investment in preoperative

Table 34.1 Abdominal based flaps advantages, disadvantages, and relative contraindications

Advantages	Relative contraindications
• Large volume • Improve abdominal contour • No intraoperative position change • Good color and texture match • Acceptable donor scar	• Insufficient abdominal tissue • Previous liposuction • Previous abdominoplasty • Active smoker • Obesity (BMI > 30) • Unfavorable preexisting abdominal scars (midline, paramedian, subcostal, McBurney): C-section scar is generally not a contraindication
Disadvantages • Long recovery time • Donor-site morbidities: ◦ Hernia ◦ Weakness (sitting up) ◦ Umbilical necrosis ◦ Symptomatic bulges	

Abbreviation: BMI, body mass index.

vascularity studies as noted below. Compared to other breast reconstruction free flap option, the abdomen offers the greatest volume of tissue and largest skin paddle. In addition, the abdominally based flaps can be harvested without a position change and can be done concurrently with the mastectomy surgery.

The abdominal free flaps share the disadvantages of a pedicled TRAM versus implant based reconstruction with longer surgical time, longer recovery time and the possibility of abdominal wall weakness, hernia and delayed healing of the donor incision. Relative contraindications are outlined in ▶ Table 34.1.

Although used as first line for breast reconstruction, there are certain contraindications that may preclude use of these flaps. Many surgeons will not offer free flap surgery to active smokers due to significantly higher rate of complications in this group. Furthermore, if the patient has insufficient abdominal tissue to provide the desired breast volume and yet is against prosthetic reconstruction, thigh or gluteal based flaps should be considered. Previous liposuction, obesity, and unfavorable abdominal scars (paramedian, McBurney) are relative contraindications to use of abdominal based flaps, especially perforator flaps. C-section scar is not a contraindication to use of abdominal tissue. However, preoperative imaging is recommended in any patient with abdominal scar.

Knowledge of the perfusion zones of transverse abdominal flap is important when deciding how much of the flap is to be transferred on a single pedicle. Hartrampf et al first described the perfusion zones of TRAM flap. Four zones were identified[9]:

1. Ipsilateral to the pedicle, overlying the rectus abdominis muscle (RAM).
2. Overlying the contralateral RAM.
3. Lateral to the ipsilateral RAM.
4. Lateral to the contralateral RAM.

In 2006, the classic Hartrampf zones II and III were shown by Holm et al to be reversed from the standpoint of blood flow.[10] The ipsilateral half of abdominal flap was shown to have an axial pattern of perfusion, whereas the contralateral half had a random pattern. It is this pattern that should be kept in mind when considering how much tissue can be carried for any individual flap. For clarity in discussion of the tissue to be used for reconstruction, the Hartrampf zones continue to be used.

Perfusion studies since then have shown that there is still further variability in the perfusion patterns depending on whether the medial or lateral row perforators are harvested. Wong et al showed that the medial perforators conform to the Hartrampf zones of perfusion while the lateral perforators follow the Holm theory.[11] In other words, the medial rows tend to perfuse across the midline better than the lateral aspect of the abdomen. These facts should also be taken into consideration when designing the flap. Intra-operative fluorescent angiography and near-infrared spectroscopy can further aid in this assessment.

Free TRAM Flap

First described by Holmstrom in 1979, free TRAM involves removal of the full transverse rectus abdominis muscle and the overlying anterior rectus sheath.[12] The obvious disadvantages is the total loss of muscle strength in the area of the flap as well as loss of abdominal wall strength by removal of the overlying fascia. Its use is indicated in patients who require large volume reconstruction (least fat necrosis) or lack suitable perforators or superficial inferior epigastric vessels based on preoperative imaging. It is ideal for those patients that require maximum blood flow such as active smokers, higher BMI patients and those in need of multiple zones of tissue. All other abdominally-based flaps attempt to maintain a portion of the rectus abdominis muscle and fascia.

The advantages of free TRAM flap include the following:

- Robust blood supply: Free TRAM has the best vascularity of all abdominal based free flaps and thus has the lowest fat necrosis rates (< 10%). Therefore it is the best option in patients with multiple risk factors or those who require large volume reconstruction.
- Compared to pedicle TRAM, free TRAM reconstruction has no pedicle bulge from tunneling.
- It allows versatile shaping into a breast mound to increase projection or match contralateral ptosis.
- Deep inferior epigastric vessel offers a long pedicle with large artery and two venae comitantes.

Muscle Sparing Free TRAM Flap (MS TRAM)

A true free TRAM is rarely performed today for breast reconstruction. Instead, muscle-sparing TRAM is performed preserving a longitudinal portion of muscle as well as the anterior rectus sheath. Depending on the amount of rectus abdominis muscle preserved, a classification system was developed by Nahabedian et al.[13] The rectus muscle is separated into three longitudinal segments: medial, lateral, and central:

- MS-0 → Sacrifice of the full width (partial length).
- MS-1 → Preservation of the lateral segment.
- MS-2 →Preservation of lateral and medial segments (removal of the central one-third makes denervation likely).
- MS-3 →Preservation of entire muscle, sacrificing either no muscle or only a cuff around the perforators (DIEP).

Studies have shown that MS-0 and MS-1 TRAM are associated with greater functional deficit, whereas the difference between MS-2 and DIEP flaps are not as well appreciated (▶ Fig. 34.2).

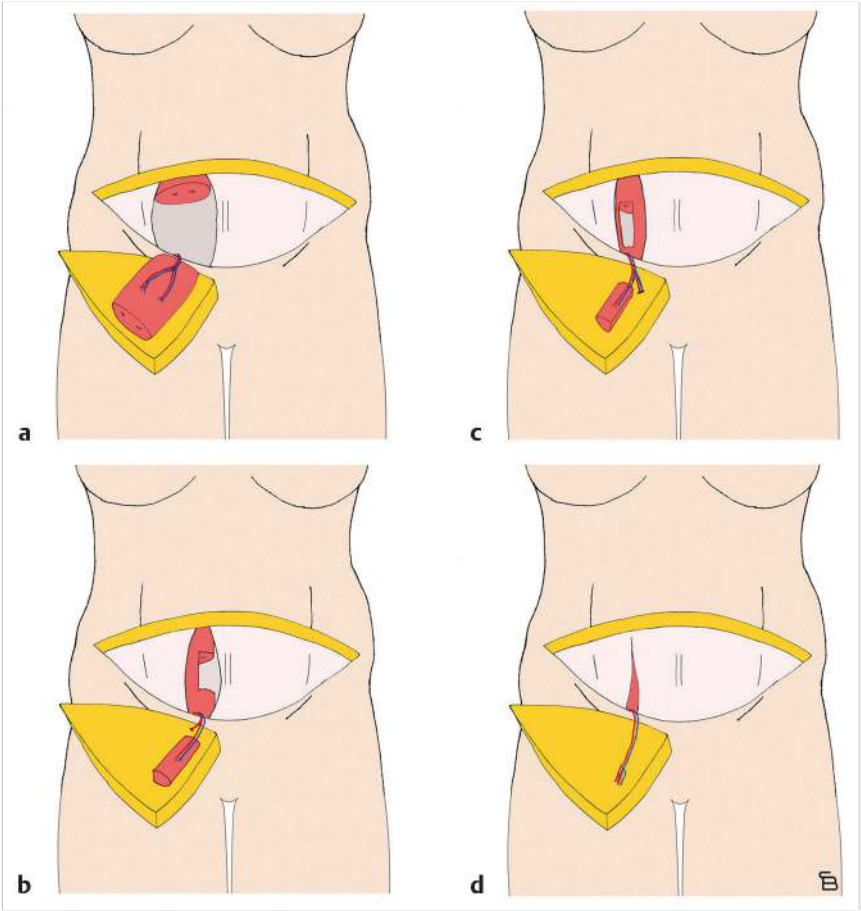


Fig. 34.2 Major types of free flap breast reconstruction. **(a)** MS-0, **(b)** MS-1, **(c)** MS-2, and **(d)** MS-3.

Operative Details

The patient is marked in the standing position. A fusiform ellipse is marked from the suprapubic crease to just above the umbilicus, and laterally to the anterior superior iliac spine (ASIS). Once under general anesthesia in supine position, the markings are confirmed to allow primary closure. If there is any doubt as to how much can be taken, elevate the central upper abdominal flap from above the umbilicus to the xiphoid, break the operating room table into a Fowler Position, and then pull the 〉 flap down to determine the lowest portion of the ellipse. Make the lower abdominal incision. Examine the deep circumflex iliac artery and vein (DCIA/V) laterally and superficial inferior epigastric artery and vein (SIEA/V) medially. If SIEV caliber is more than 1.5 mm, dissect it further until size of the SIEA can be determined. If the size of the artery is adequate, SIEA flap can be considered. In either case, it is safest to dissect both DCIV and SIEV for some length to use them as a lifeboat for venous outflow.

Mark the major perforators, as determined by preoperative imaging and intraoperative Doppler. Elevate the right and left abdominal flaps in the suprafascial plane from lateral to medial direction. Once networks of perforators are identified, incise the anterior rectus sheath surrounding the perforators and extend it caudally. Elevate the fascia off the rectus abdominis muscle and undermine the muscle to visualize the course of deep inferior epigastric vessels. The rectus abdominis muscle is then incised to incorporate all the perforators into the flap and entry point of the pedicle. It is important to preserve the lateral intercostal motor nerves during this dissection to maintain the function of abdominal wall. Once transferred to the breast pocket, the flap is oriented so that the best-perfused portion of the flap (zones I and II) is positioned in the superomedial aspect of the reconstructed breast; this is the least likely site to develop fat necrosis.

Deep Inferior Epigastric Artery Perforator Flap

DIEP flap is an adipocutaneous flap perfused on direct or indirect muscle perforators. Its use is indicated in active patients or women who plan to have a child and therefore require competent abdominal wall. Preoperative CTA or MRA in hand with intraoperative assessment of the perforators allow identification of the eligible abdomen. In general, a single perforating artery or vein of at least 1.5 mm with a palpable pulse is required for a successful perforator flap harvest. Such perforators are typically located in the periumbilical area.

DIEP flap has been shown to offer advantages over the traditional TRAM flap by preserving the rectus muscle. This results in lower donor-site morbidity, specifically risk of abdominal hernia and bulge as well as decreasing postoperative pain and improving recovery time. Perforator flap dissection also

Fig. 34.3 Example of bilateral deep inferior epigastric perforator breast reconstruction done after nipple sparing mastectomies.

increases pedicle length (especially when isolated on a single pedicle), allowing for improved flexibility of flap positioning. On the other hand, due to the need for meticulous dissection, these surgeries are often longer and although the literature varies, older studies demonstrate a higher flap loss rate (5–10% vs. 0.5–1% in free TRAMs). Furthermore, a perforator flap has higher incidence of fat necrosis and venous congestion compared with MS-TRAM as less perforators are included in the flap. More recent studies do not support these findings and show similar complication rates between types of abdominal based free flap breast reconstruction techniques.[14] The improvements may be a result of ever increasing experience among surgeons.

Operative Details

Preoperative markings and initial operative steps are similar to MS-TRAM outlined previously. Once the appropriate perforator (s) are identified, the fascia and muscle are incised as needed to allow perforator dissection; however, no fascia or muscle is harvested. The perforators are dissected by splaying the muscle fibres temporarily and following them down to their origin. The pedicle is identified and elevated off the rectus muscle. In unilateral cases, the contralateral hemi-abdomen is kept intact as a lifeboat until the ipsilateral dissection has been safely completed. The number of perforators necessary to adequately perfuse the flap is largely determined by patient experience. In lieu of this experience, all the perforators can be isolated and individually clamped while assessing the flap for blood flow with either tissue oximetry such as ViOptix or isocyanine green infrared scan. Taking the fewest number of perforators possible will minimize the morbidity of the rectus abdominis muscle. Likewise, taking perforators all from a single row (medial versus lateral) will decrease morbidity as well. Similarly, the SIEV and DCIV are dissected and kept as backup in case they provide the dominant venous outflow to the flap.

In unilateral cases where the entire abdomen needs to be used to achieve the appropriate volume, a stacked, free TRAM, double-pedicled or bipedicled DIEP/MS-TRAM flap can be harvested. In such cases, both hemi-abdomens are dissected as DIEP or MS TRAM. The secondary pedicle is anastomosed either to a side branch of the primary pedicle or to an independence recipient vessel in the chest (retrograde to internal mammaries or to thoracodorsal system). A classification system was described in 2007 and further refined by Murray et al. based on vessels that are used for crossover anastomosis (▶ Fig. 34.3).

Superficial Inferior Epigastric Artery Flap

Of all the abdominal flaps used, the SIEA flap causes the least violation of the abdominal myofascial layer.[15] It is based on superficial inferior epigastric artery and vein, which arises from femoral artery 1 to 3 cm below the inguinal ligament. Although it causes the least trauma to abdominal wall, SIEA flap accounts for well under 30% of all autologous breast reconstructions due to its many limitations:

- A suitable SIEA is present in only 60% of people.
- SIEA caliber is small and adequate (> 1.5 mm) only in 15% of patients.
- Offers a short pedicle (mean of 6 cm).
- Has higher risk of partial or complete flap loss.
- The angiosome territory is restricted to zones Holm's zones 1 and 2 (hemi-abdomen).

Operative Details

The initial operative sequence is as previously described for MS-TRAM. The SIEA/V vessels are identified about one-third the distance from the pubic bone to the ASIS where they cross

the inguinal ligament. At this point, the vessels are dissected down to their origin from femoral vessels and the SIEA feasibility is assessed:

- Vessel caliber greater or equal to 1 to 1.5 mm at the level of the lower abdominal incision with a palpable pulse.
- Pedicle length of at least 6 cm or twice the thickness of the flap for inset purposes.

Special attention should be paid to insetting of the SIEA flap as the pedicle enters the flap at its edge rather than it is under surface as in DIEP or TRAM flaps. Therefore, normal insetting may cause a kink in the pedicle compromising the flow. To prevent this, the inferior 2 to 3 cm of the flap is de-epithelialized and folded, orienting the pedicle in the inferomedial direction and effectively rotating the pedicle superiorly without kinking.

Rubens Flap

Rarely used for breast reconstruction, Rubens flap is based on the deep circumflex iliac artery (DCIA). It allows harvest of the fat pad in the hip and waist region just above the iliac crest. Its use is indicated in the patients who are not candidates for other free flaps and refuse prosthetic reconstruction. It does provide tissue of similar consistency to breast and has an inconspicuous donor site. However, dissection of Rubens flap is quite tedious with a less robust blood supply. Flap perfusion has been shown to improve with harvesting the abdominal wall musculature (external oblique, internal oblique, and transversalis muscle), which on the other hand can cause abdominal wall morbidity such as hernia or bulge. Furthermore, unilateral harvest causes obvious asymmetry that may require contralateral balancing procedures. For all these reasons, Rubens flap has become more of a historic procedure.

Gluteal Based Flaps

Orticochea first performed breast reconstruction using a gluteal flap in 1973.[16] He transferred a musculocutaneous flap in a five-stage procedure. Fujino et al later refined this into a one-stage procedure as a free flap in 1975.[17] The perforator version of these flaps were developed later and first used for breast reconstruction by Allen and Tucker in 1995 and Guerra et al in 2004.[18,19] The perforator flaps include either SGAP flap or the inferior gluteal artery perforator (IGAP).

The gluteal flaps are considered second line choice for autologous breast reconstruction after abdominal based flaps. Their use is indicated in patients with insufficient abdominal tissue (low BMI or previous abdominal surgery such as abdominoplasty) or in those whom abdominal based flaps is contraindicated. Advantages of gluteal-based flaps include

- Concealed scar.
- Adequate volume for medium size breast.
- Concomitant buttock lift.

However, gluteal flaps have a number of shortcomings, rendering their use as first-line reconstructive option:

- Firmer, less pliable, and stiff breast mound.
- Have high donor site morbidity, including contour deformity, buttock asymmetry, sciatic nerve injury, and posterior thigh numbness or paresthesia.
- Have smaller and shorter pedicle compared with DIEP or MS-TRAM.
- Technically among the most difficult flaps to dissect.
- Require intraoperative position changes.
- Have higher rate of flap loss compared with MS-TRAM (5–10%).

Superior Gluteal Artery Perforator

The pedicle for SGAP exits the greater sciatic foramen between the piriformis and gluteus medius muscles, approximately 6 cm below the posterior superior iliac spine (PSIS) and 5 cm lateral to midline. The vessel then perforates the gluteus maximus muscle as it travels to the surface. Pedicle is short with thin walled vein and multiple branches. Pedicle length is approximately 5 to 8 cm, depending on the medial or lateral location of the perforator respectively.

Operative Details

Preoperative markings include PSIS, greater trochanter, and sacrum. The perforators are localized with preoperative imaging and intraoperative handheld Doppler with patient in prone position. Elliptical skin island is then designed with a 30 to 45 degrees slant centered on the suitable perforators. To increase the usable length of the pedicle as well as facilitate the flap inset, peripheral perforators are preferred over central ones. The flap is incised laterally and elevated in the subfascial plane toward midline. The perforator is identified and isolated. It is traced proximally by splitting the gluteus maximus muscle fibres until the main stem is reached as it exists above the piriformis. At this point multiple small venous branches emanating in all directions are encountered and the caliper of the vein can be large (5 mm+). These veins often require tedious dissection and vascular control as significant bleeding can be encountered with loss of proximal control (▶ Fig. 34.4).

Fig. 34.4 Postoperative example of the superior gluteal artery perforator donor on the right.

Inferior Gluteal Artery Perforator

IGAP provides better donor site contour, scar, and pedicle size compared with the SGAP. It is technically easier than the SGAP due to longer pedicle length. The skin paddle can be designed in the gluteal crease as described by Allen et al to camouflage the scar. However, it has higher risk of sciatic nerve irritation as it is often exposed during dissection. Furthermore, due to location of the scar, sitting can be restricted following the surgery. For these reasons, most microsurgeons prefer the SGAP.

34.3.6 Thigh Based Flaps

Transverse Upper Gracilis Myocutaneous Flap

The perforator anatomy of transverse upper gracilis (TUG) flap was first described by Yousif et al in a cadaveric study which demonstrated the transversely running perforators of gracilis muscle.[20] This lead to first use of TUG flaps in breast reconstruction, which is now considered as an alternative to abdominal free flaps in patients with inadequate abdominal tissue. TUG flap offers pliable adipose tissue for moderate size breast reconstruction, mean volume of 330 mL. It also has minimum donor site morbidity, a well-hidden scar, and allows for concomitant inner thigh lift. However, harvesting large skin paddles wider than 10 cm can lead to potential contour deformity, wound dehiscence, and poor scarring. Lower extremity lymphedema, sensory disturbances at the donor site, and loss of flap volume over time with muscle atrophy have also been reported.

Operative Details

The gracilis branch of the medial femoral circumflex artery (MFCA) supplies the TUG flap. It offers 6 to 8 cm pedicle length with 1.5 to 2.5 mm caliber.

The patient is assessed preoperatively in standing position. The medial thigh is pinched to determine the available height of the flap, which usually ranges 8 to 12 cm. Once in supine position and with the thigh abducted, the flap axis is marked from pubic symphysis to the medial femoral condyle, two fingerbreadths below the palpable edge of adductor longus (AL) muscle. The skin paddle is marked transversely on the inner upper thigh just below the inguinal ligament, 1 to 2 cm below the crease, and extending posteriorly into the inferior gluteal crease. This allows maximal use of the angiosome of the dominant gracilis pedicle. The length of the flap in the anterior posterior direction is based on mastectomy defect dimensions.

Starting anteriorly, the flap is elevated in the suprafascial plane. The saphenous vein is identified and preserved if possible. When the septum between AL and gracilis is identified, the fascia is incised; the pedicle to gracilis is identified and traced back toward profunda femoris artery by exposing it in the plane between adductor longus and magnus. The posterior wing of the flap is then elevated toward the posterior gracilis edge and the muscle is divided superiorly and inferiorly.

In order to decrease risk of postoperative limb lymphedema in TUG flaps, several maneuvers are recommended:

- Performing suprafascial dissection anteriorly to preserve deep lymphatic channels.
- Preserving the saphenous vein in the anterior dissection.
- Preserving the subcutaneous fat anterior to gracilis border in the femoral triangle. This can be achieved by designing an S-Shaped skin paddle to avoid the femoral triangle.

TUG flap allows for small to moderate size breast reconstruction (▶ Fig. 34.5). In order to increase the volume of the flap or in cases of unilateral reconstruction, various techniques are available:

- Extended TUG flap includes harvesting fat and subcutaneous tissue beyond the borders of the skin paddle.
- A vertical extension can be added to the skin paddle overlying the gracilis muscle to further increase flap bulk.
- Stacked flaps or double flaps have been described to be used in unilateral cases: Harvest bilateral TUG flaps and perform

Fig. 34.5 Example of the transverse upper gracilis flap in breast reconstruction.

either flap-to-flap anastomoses and then transfer en bloc to the chest to one recipient vessel or double anastomosis to separate recipient vessels in the chest. Flap-to-flap anastomoses can be done when a large branch from the main pedicle going to one of the adductor muscles is present. This branch can be used to anastomose the additional TUG flap too.

Profunda Artery Perforator Flap

The profunda artery perforator flap (PAP) flap is another alternative to abdominal based donor sites for autologous breast reconstruction in patients where the abdominal tissue is insufficient or contraindicated. The PAP flap is a fasciocutaneous flap from the posteromedial thigh, which is based on musculocutaneous perforators from profunda femoris artery. It can be a source of autologous tissue for reconstruction of small to medium-sized breasts. Shortcomings of this flap include its inconsistent perforator location, requiring the need for preoperative imaging studies, and a narrow flap width limiting size of the reconstructed breast.

As per cadaveric studies of Saad et al, the perforator for this flap is found within 8 cm caudal to gluteal crease and on average 6 cm from midline (3–12 cm range).[21] It emerges between the adductor magnus and semitendinosus muscles with a short intramuscular course through the adductor magnus muscle. The flap is designed centered on an identified perforator (by preoperative imaging and intraoperative Doppler assessment) as an ellipse using the gluteal crease to conceal the final scar. Skin paddle can be up to 28 × 8 cm and the pedicle length can be up to 13 cm.

Transverse Fascia Lata TFL Myocutaneous Flap

Based on lateral circumflex femoral artery blood supply to the tensor fascia lata muscle and the overlying skin and fat of the lateral thigh, the TFL myocutaneous flap is an option in those patients where other options have been exhausted.[22] It can be harvested in either a transverse or vertical fashion and may be a consideration for patients with a saddlebag deformity. It leaves a significant scar and causes asymmetry between the upper lateral thighs.

34.3.7 Recipient Vessels

Selection of appropriate recipient vessels is as important as donor flap dissection in microvascular breast reconstruction. Internal mammary artery and vein and thoracodorsal vessels are the most commonly used recipient vessels.

Internal Mammary Recipient

Internal mammary vessels offer a few advantages over the thoracodorsal vessels, which has made them a more popular choice among the breast microsurgeons:

- Easier exposure with no need of axillary dissection. This reduces risk of lymphedema, shoulder stiffness, or brachial plexus injury.
- Easier access for microsurgery, allowing better positioning for the assistant.
- Compatible size matches with excellent flow characteristics. The blood flow rate of the IMA ranges from 15 to 35 mL/min while the blood flow rate of the thoracodorsal artery ranges from 2 to 8 mL/min.
- Allows for more medial placement of the flap with reduced lateral fullness compared with thoracodorsal vessels.

Despite its advantages, there are certain drawbacks associated with use of IMA/IMV as recipient vessels:

- Internal mammary veins tend to be thin walled and fragile especially in postradiated cases, on the left side (where the vena comitans can often be quite small), and below the fourth rib.
- Respiratory movements can make microsurgery difficult.
- Their harvest compromises future use of IMA for coronary artery bypass grafting.
- Has the potential risk of pneumothorax.

Operative Details

The third costal cartilage is identified. Pectoralis major muscle fibres are separated and the cartilage is removed by performing a sub-perichondrial dissection. The deep perichondrium is incised and reflected to expose the internal mammary vessels. Perforating branches of IM are occasionally encountered and if of sufficient size are preserved to be used as recipient vessel.

Thoracodorsal Recipient

Thoracodorsal vessels are occasionally used in cases of modified radical mastectomy where they have been already exposed, or in patients with history of cardiac disease in whom there is a chance that internal mammary vessels will be required in the future for coronary artery bypass graft. The main shortcoming of their use is the need for axillary dissection, the lateral fullness of reconstructed breast, and the potential compromise of future use of pedicled latissimus dorsi musculocutaneous flap for salvage. To avoid the latter point, anastomosis to these vessels should be done proximal to the serratus take off. This preserves the latissimus dorsi flap as a lifeboat via retrograde flow through the serratus branch.

Operative Details

In cases where no axillary dissection has been performed for nodal assessment a separate transverse incision is made between the anterior and posterior axillary folds below the axillary hairline. The lateral border of pectoralis major muscle is identified to enter the axillary fat. The lateral thoracic vein is often first encountered within this fat pad. The vein is traced superiorly to identify the axillary vein. The thoracodorsal vessels are identified posterior to this origin within the fat pad. If an axillary dissection has been done, find the lower portion of the lateral latissimus dorsi muscle and trace it proximally to find the TD vessels.

Other potential recipient vessels for microvascular breast reconstruction include thoracoacromial artery and vein, circumflex scapular artery and vein, lateral thoracic artery and vein, external jugular vein, external carotid artery branch, cephalic vein, and axillary artery and vein. These vessels are only used in cases where internal mammary or thoracodorsal vessels are not available or already used in a failed flap.

The cephalic vein in particular is useful in those cases where the left internal mammary veins are less than 1 mm in size and the thoracodorsal vessels are not available. The use of the

cephalic mitigates the need for vein graft and is an excellent match to the deep inferior epigastric vein at about 3 mm in diameter. If the harvest is taken down to the level of the antecubital vein, its length can reach to the inframammary fold. The vein can be found near the level of the anterior axillary fold below Scarpa's fascia and above the level of the muscular fascia. Skip incisions can be made to minimize the scarring, although the main drawback of this technique is a tendency toward hypertrophic scars on the arm and shoulder.

34.4 Postoperative Care

Postoperative care for the free flap breast reconstruction patient is centered on blood flow in the flap including monitoring and anticoagulation as well as pain control.

For pain control, our current protocol is to perform a transversus abdominis plane (TAP) block. For this, we use a mixture of liposomal bupivacaine (Exparel) and plain bupivacaine injected under ultrasound guidance. At its best, it gives about 3 days of good relief of abdominal pain. A similarly effective relief of pain is obtained with continuous bupivacaine infusion via elastomeric pump (ON-Q).

Flap monitoring is largely based on the preference of the operating surgeon. Techniques vary from exam only, hand-held Doppler, implantable Doppler (Cook or Coupler based), to tissue oximetry. Each method has advantages and disadvantages and vigilance with a hand-held Doppler can be both equally effective and a cost-effective means of monitoring flaps. To be used properly, the hand-held Doppler requires training of the nurses in residents who will be using it.

Typically, free flap patients are in the hospital 3 to 5 days and generally return to work in 2 to 4 weeks. For those with muscle sparing abdominal based flaps, they can expect back to get most of their abdominal strength back by about 3 months.

Follow-up procedures may include flap adjustment via resection, liposuction or lipofilling. Symmetry procedures on the opposite breast are often necessary for maximum aesthetic outcome and those patients without a nipple benefit from either 3D tattoo or nipple reconstruction.

34.5 Outcomes

The versatility and flexibility of free tissue transfer techniques offer improved aesthetics in more difficult situations.[23] Unilateral autologous reconstructions can better match the ptosis of the opposite breast. Autologous reconstruction is possible even where there is inadequate tissue to cover an implant. Implants alone are incapable of making an adequate breast in many obese patients and limited in those patients with very large breasts measured in kilos of removed tissue not grams. The most recent comprehensive data reveal that nearly 64.6% of American women are overweight (BMI > 25) and fully 35.9% are obese (BMI > 30). Alloplastic techniques have the advantage of technical ease and lack of donor morbidity. In those patients who have undergone both procedures, the great majority choose the autologous breast as preferable. The further one asks this question from the original surgery, the greater the gap in satisfaction.[24]

Microsurgery allows for even greater improvements over standard autologous techniques of TRAM flap and latissimus dorsi myocutaneous flap.[19] Free flap inset is less limited by the tether of the pedicle allowing for greater flexibility with inset. It allows for the use of flaps outside of the region of the trunk, offers more options to preserve donor muscle function and offers options to those whose local tissues have been used or compromised. Even with improvements in mastectomy technique, implants manufacturer in and the addition of fat grafting, free flap breast reconstruction remains the standard against which all other techniques are compared.

34.6 Review Questions

34.6.1 True or False

1. Due to its complexity, free flap based breast reconstruction should be limited to only those patients where an implant is not possible or has failed.
2. Pedicled TRAM breast reconstruction has a lower rate of fatty necrosis than free flap based abdominal breast reconstruction.
3. Gluteal artery based breast reconstruction can offer greater recipient breast projection than abdominally based flaps.

34.6.2 Choose the Best Answer

4. A 58-year-old woman with a BMI of 28 is BRCA1 positive with a history of right breast cancer treated by mastectomy and radiation therapy. She has now been found to have a new left-sided breast cancer and her breast surgeons plan to perform a mastectomy for treatment. The patient is interested in reconstruction. Which of the following represent the best treatment plan:
   a) Right breast latissimus dorsi with implant and left breast free TRAM.
   b) Right breast DIEP flap and left breast tissue expander followed by permanent implant.
   c) Bilateral latissimus dorsi flaps with implants.
   d) Bilateral DIEP flaps.
   e) Bilateral TUG.
5. A 48-year-old female with a BMI of 26 is to undergo right-sided mastectomy to treat invasive ductal breast carcinoma. Her history includes a previous abdominoplasty. She is not interested in implant reconstruction and has C-cup breasts. Her best option for treatment is
   a) To help her understand that implant reconstruction remains her best option and other techniques will either result in greater morbidity or poor aesthetics.
   b) Free SGAP flap.
   c) Free TUG flap.
   d) Extended latissimus dorsi myocutaneous flap with no implant.

34.6.3 Answers

1. False.
2. False.
3. True.
4. d. Bilateral DIEP flaps.
5. b. Free SGAP flap.

References

[1] Kronowitz SJ, Robb GL. Radiation therapy and breast reconstruction: a critical review of the literature. Plast Reconstr Surg. 124(2):395–408

[2] Kelley BP, Valero V, Yi M, Kronowitz SJ. Tamoxifen increases the risk of microvascular flap complications in patients undergoing microvascular breast reconstruction. Plast Reconstr Surg. 129(2):305–314

[3] Heitland AS, Markowicz M, Koellensperger E, Schoth F, Feller A, Pallua N. Duplex Ultrasound Imaging in Free Transverse Rectus Abdominis Muscle, Deep Inferior Epigastric Artery Perforator, and Superior Gluteal Artery Perforator Flaps: Early and Long-Term Comparison of Perfusion Changes in Free Flaps Following Breast Reconstruction. Ann Plas Surg. 2005; 55(2):117-121

[4] Aoyagi F, Fujino T, Ohshiro T. Detection of small vessels for microsurgery by a Doppler flowmeter. Plast Reconstr Surg. 55(3):372–373

[5] Blondeel PN, Beyens G, Verhaeghe R, et al. Doppler flowmetry in the planning of perforator flaps. Br J Plast Surg. 51(3):202–209

[6] Casey WJ III, Chew RT, Rebecca AM, Smith AA, Collins JM, Pockaj BA. Advantages of preoperative computed tomography in deep inferior epigastric artery perforator flap breast reconstruction. Plast Reconstr Surg 2009;123:11481155

[7] Masia J, Clavero JA, Larrañaga JR, Alomar X, Pons G, Serret P. Multidetector-row computed tomography in the planning of abdominal perforator flaps. J Plast Reconstr Aesthet Surg. 59(6):594–599

[8] Rozen WM, Stella DL, Bowden J, Taylor GI, Ashton MW. Advances in the preoperative planning of deep inferior epigastric artery perforator flaps: magnetic resonance angiography. Microsurgery. 29(2):119–123

[9] Hartrampf CR, Scheflan M, Black PW. Breast reconstruction with a transverse abdominal island flap. Plast Reconstr Surg. 69(2):216–225

[10] Holm C, Mayr M, Höfter E, Ninkovic M. Perfusion zones of the DIEP flap revisited: a clinical study. Plast Reconstr Surg. 117(1):37–43

[11] Wong C, Saint-Cyr M, Mojallal A, et al. Perforasomes of the DIEP flap: vascular anatomy of the lateral versus medial row perforators and clinical implications. Plast Reconstr Surg. 125(3):772–782

[12] Holmström H. The free abdominoplasty flap and its use in breast reconstruction. An experimental study and clinical case report. Scand J Plast Reconstr Surg. 13(3):423–427

[13] Nahabedian MY, Momen B, Galdino G, Manson PN. Breast reconstruction with the free TRAM or DIEP flap: patient selection, choice of flap, and outcome. Plast Reconstr Surg. 110(2):466–475, discussion 476–477

[14] Macadam SA, Zhong T, Weichman K, et al. Quality of life and patient-reported outcomes in breast cancer survivors: a multicenter comparison of four abdominally based autologous reconstruction methods. Plast Reconstr Surg. 137(3):758–771

[15] Arnez ZM, Khan U, Pogorelec D, Planinsek F. Breast reconstruction using the free superficial inferior epigastric artery (SIEA) flap. Br J Plast Surg. 52(4): 276–279

[16] Orticochea M. Use of the buttock to reconstruct the breast. Br J Plast Surg. 26 (3):304–309

[17] Fujino T, Harasina T, Aoyagi F. Reconstruction for aplasia of the breast and pectoral region by microvascular transfer of a free flap from the buttock. Plast Reconstr Surg. 56(2):178–181

[18] Allen RJ, Tucker C , Jr. Superior gluteal artery perforator free flap for breast reconstruction. Plast Reconstr Surg. 95(7):1207–1212

[19] Guerra AB, Metzinger SE, Bidros RS, Gill PS, Dupin CL, Allen RJ. Breast reconstruction with gluteal artery perforator (GAP) flaps: a critical analysis of 142 cases. Ann Plast Surg. 52(2):118–125

[20] Yousif NJ, Matloub HS, Kolachalam R, Grunert BK, Sanger JR. The transverse gracilis musculocutaneous flap. Ann Plast Surg. 29(6):482–490

[21] Saad A, Sadeghi A, Allen RJ. The Anatomic Basis of the Profunda Femoris Artery Perforator Flap: A New Option for Autologous Breast ReconstructionA Cadaveric and Computer Tomography Angiogram Study. J Recon Microsurg. 2012; 28(6):381-386

[22] Elliott LF, Beegle PH, Hartrampf CR , Jr. The lateral transverse thigh free flap: an alternative for autogenous-tissue breast reconstruction. Plast Reconstr Surg. 85(2):169–178, discussion 179–181

[23] Blondeel PN, Hijjawi J, Depypere H, Roche N, Van Landuyt K. Shaping the breast in aesthetic and reconstructive breast surgery: an easy three-step principle. Plast Reconstr Surg. 123(2):455–462

[24] Hu ES, Pusic AL, Waljee JF, et al. Patient-reported aesthetic satisfaction with breast reconstruction during the long-term survivorship Period. Plast Reconstr Surg. 124(1):1–8

35 Breast Reconstruction Using the Latissimus Dorsi Myocutaneous Flap

Galen Perdikis and Stephanie L. Koonce

Abstract
This chapter discusses the use of the latissimus flap in breast reconstruction. Pre- and postsurgical steps are outlined and operative techniques detailed. Numerous images illustrate the procedures described. Postoperative care guidelines are specified and potential outcomes and complications listed.

Keywords: latissimus, expander, seroma, capsular contracture

35.1 Goals and Objectives

- Understand the assessment of prospective breast reconstruction patients.
- Recognize the indications and contraindications for breast reconstruction with a latissimus dorsi myocutaneous flap.
- Describe the operative technique and postoperative care for a latissimus dorsi myocutaneous flap.
- Review the outcomes and complications following breast reconstruction with a latissimus dorsi myocutaneous flap.

35.2 Patient Presentation

The latissimus dorsi myocutaneous flap (LDMF) is based on the flat triangular latissimus dorsi muscle originating from the iliac crest, the lower third to fourth ribs laterally, the lower six thoracic spines, and the posterior layer of the thoracolumbar fascia with a tendinous insertion upon the intertubercular groove of the humerus. This flap was first described by Tansini in 1897 for coverage of chest wall defects from breast amputation.[1] The dominant pedicle to the latissimus includes the thoracodorsal artery and associated veins and thoracodorsal nerve. The consistency and relatively large diameter of this artery make the latissimus dorsi flap a dependable donor site. In patients with previous axillary surgery in which the thoracodorsal pedicle may have been divided, retrograde flow through the serratus branch of the thoracodorsal artery can maintain adequate vascular supply to the flap.[2] Patients who desire breast reconstruction must be carefully assessed to determine which reconstructive options are best suited to them. A history should be obtained regarding previous surgery to the chest wall, back, axilla, or abdomen, history of or plans for radiation therapy to the breast or chest wall, and also the present condition of the breast. Physical examination should focus on the location and size of the tumor, skin changes, the size of the breast, the presence of ptosis and the amount of skin and subcutaneous tissue available in the dorsal and abdominal regions. Presence of abdominal wall hernias should be noted. A full discussion should be directed toward the patient's willingness to undergo major surgery and the advantages and disadvantages of immediate and delayed reconstruction. Immediate reconstruction avoids the need for a second general anesthesia and may reduce issues related to self-esteem and quality of life. Delayed reconstruction may be indicated in patients who will require adjuvant radiation therapy, those requiring negative margins after a partial mastectomy, or those who desire more time between diagnosis, extirpative surgery, and reconstruction. Delayed reconstruction can be challenging secondary to scar tissue, lack of mobile chest wall skin, or tissue changes from radiation.

35.3 Preparation for Surgery

Preoperatively the patient should undergo an anesthesia clearance evaluation, but otherwise no special testing is required. The diagnostic data desired prior to surgery is dependent on patient's age, comorbidities, and is similar to that required for any other major elective operation. Baseline hemoglobin, hematocrit, and electrolytes are routinely obtained. An assessment of the need for chemoprophylaxis for venous thromboembolism should always be included. We advise all patients to cease the use of tobacco products and maintain a healthy lifestyle.

For those patients who present as a delayed reconstruction, sometimes years following their breast cancer treatment, it is necessary to assure that proper oncologic screening has been maintained. Yearly mammographic screening of the intact breast needs to be confirmed. This similarly applies to those who present with lumpectomy defects, with applicability to both breasts.

35.4 Treatment

35.4.1 Indications, Contraindications, and Alternatives

The LDMF is a good choice for both immediate and delayed breast reconstruction.[3] It is particularly useful in previously irradiated patients as it brings in non-radiated skin and a healthy muscular sling to provide coverage of the final implant. In the majority of patients, the LDMF is used in conjunction with a temporary tissue expander that is subsequently changed to permanent implant; alternatively, it can be used as a one-stage reconstruction by placing an implant at the outset. The two-stage approach allows for flexibility in volume adjustment and has been seen to have low capsular contracture rates compared to one-stage operations.[4,5] More recently, particularly with the use of shaped textured implants, similar capsular contracture rates have been seen in series using a one-stage approach.[6] If an expander is used, the patient should be prepared for multiple office visits for expansion until the desired size is achieved, as well as a second surgery for exchange to permanent implant. Patients may also present with partial mastectomy defects. The latissimus flap alone can provide volume replacement for the absent tissue without a breast device. Furthermore, in selected patients with high body mass index, sufficient tissue from latissimus transfer will be possible such that an implant is not needed.

An absolute contraindication for use of the latissimus dorsi flap is a previous lateral thoracotomy in which the latissimus muscle was divided. Previous surgery or injury to the thoracodorsal nerve resulting in an atrophic muscle can be a relative contraindication if the remaining volume of muscle is inadequate for coverage. In the setting of atrophic muscle after previous dissection, the surgeon must consider the thoracodorsal vascular pedicle may have been damaged which necessitates an intact serratus collateral vasculature for flap survival.

A discussion of all of the reconstructive options is beyond the scope of this chapter but must be included in the discussion with the patient.

35.4.2 Preoperative Evaluation and Markings

With the patient awake and either sitting or standing, the anterior border of the latissimus dorsi muscle is marked. The tip of the scapula is also marked. The skin island may be oriented transversely along the bra line, laterally, or obliquely. It is our preference to orient the skin island transversely in the bra line for improved aesthetics. The pattern is an ellipse that leaves a linear scar at the donor site when closed. The mid sternal line, inframammary folds of both breasts, and, in delayed reconstructions, the mastectomy scars are marked anteriorly. In delayed reconstruction patients with loss of breast landmarks, the opposite breast is used as a template in marking the mastectomy site.

35.4.3 Operative Technique

Patients having immediate breast reconstruction are usually in a supine position during mastectomy allowing confirmation of the integrity of the thoracodorsal vascular pedicle. The patient is then positioned in the lateral decubitus position for a unilateral breast reconstruction. Once the flap is elevated and transferred into the mastectomy defect and the back is closed, the mastectomy defect is temporarily stapled closed, dressed with an occlusive dressing and the patient is placed back into the supine position. The temporary dressing is then removed and the patient re-prepped and draped. On occasion, if the patient is small, the patient can be placed in a “sloppy lateral” position which allows for a surgical approach anteriorly and posteriorly. This avoids re-prepping and draping. Harvesting of the flaps for bilateral reconstruction can be done with the patient in prone positioning as long as direct pressure on the mastectomy flaps is avoided.

Patients undergoing immediate reconstruction have a skin island fashioned to match the skin-sparing mastectomy defect. For all immediate reconstructions, the skin ellipse on the back should be carefully placed along the bra line. In delayed reconstruction, a bigger skin island is needed and the markings can be angled antero-inferiorly (▶ Fig. 35.1). We do not advocate an ellipse that is parallel with the muscle fibers except in chest (not breast) reconstructions that require very larger islands. Although the flap skin inset into the breast is circular, the skin excision on the back is elliptical to allow proper closure (▶ Fig. 35.2). Patients undergoing delayed reconstruction have a larger skin ellipse inset into either the old mastectomy defect or the new inframammary fold to improve the inferior pole contour (▶ Fig. 35.3). This decision depends on the needs of each particular patient as predicated by tissue quality and breast shaping. The skin island is incised beveling the scalpel away from the incision line through the subcutaneous fat. This prevents undermining of the skin island, and allows maximal capturing of perforating vessels into the skin paddle. Dissection is carried out just above the muscular fascia, raising inferiorly and superiorly based skin flaps and exposing the latissimus muscle. Dissection proceeds laterally to identify the edge of the

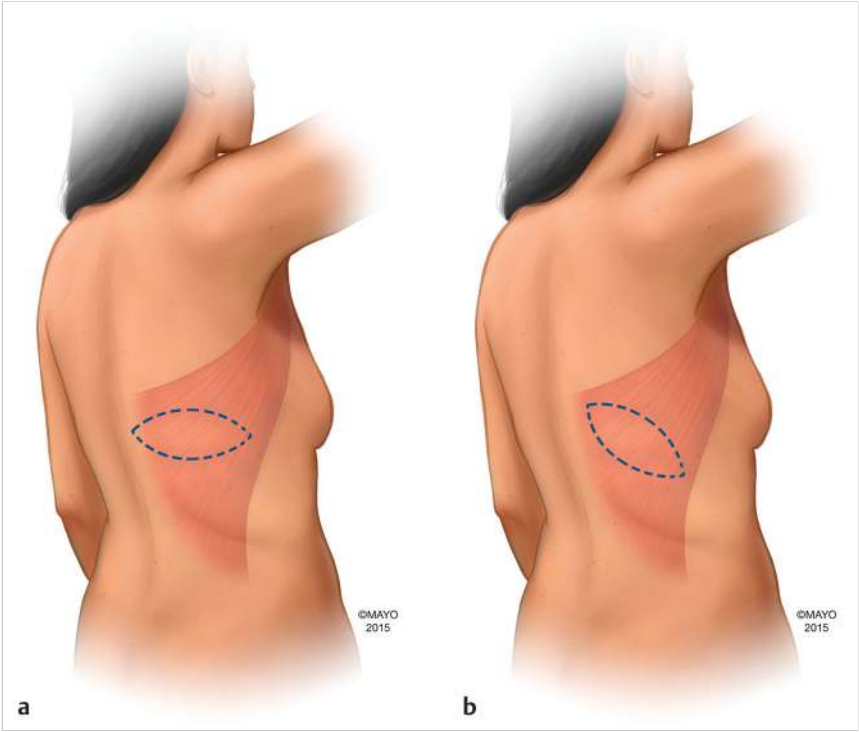


Fig. 35.1 (a,b) Preoperative markings. The back ellipse is placed in the bra line but can be angled anteroinferiorly for a larger skin island if needed in delayed reconstruction. Reprinted with permission of Mayo Foundation for Medical Education and Research. All rights reserved.

latissimus muscle. A thick aponeurotic attachment between the serratus anterior muscle and the latissimus is noted at the level of ribs 10 and 11. This must be divided during latissimus flap elevation to prevent elevation of the serratus anterior muscle along with the latissimus. The flap is then elevated along its lateral edge. The latissimus is then separated from the paraspinous muscle fascia at its origin, as well as inferiorly along its costal attachments. Incision through the paraspinous fascia makes identification of the proper dissection plane challenging and should be avoided. Latissimus fibers originating on the thoracic spines are now divided with meticulous attention paid to hemostasis of the intercostal perforators. The superior border of the latissimus is identified, separated from the overlying trapezius muscle, and dissection proceeds toward the axilla. The thoracodorsal pedicle is identified, and the vessels preserved. Dividing the nerve can help with latissimus animation and spasticity. However, we have not seen this as a problem possibly because the flap if dissected as described, is very loose, and acts as a loose hammock.[7] Furthermore, the intact nerve will minimize long-term latissimus atrophy which can result in deficient soft-tissue coverage, contour irregularities, and implant visibility. Ideally, the serratus branch is identified and preserved when possible, particularly in patients who may have compromised thoracodorsal vessels. Release of the latissimus insertion near its attachment is often done at this point.

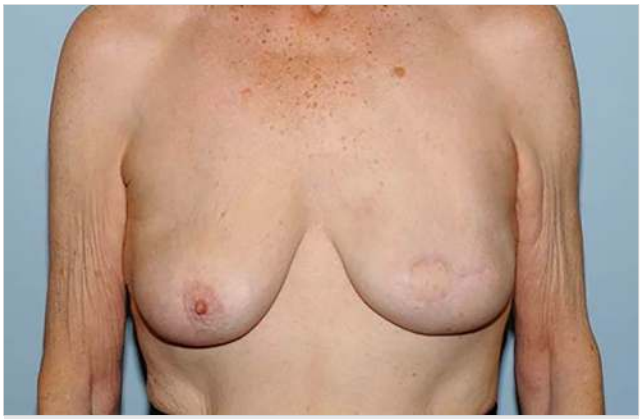


Fig. 35.2 Latissimus dorsi myocutaneous flap (LDMF) skin island used to match the skin-sparing mastectomy defect in a patient who declined any type of mastopexy procedure.

The flap is then transferred to the mastectomy defect via the axilla through a subcutaneous tunnel. Caution is taken to avoid torsion of or tension on the pedicle. The flap is inset. Bolster sutures using dental rolls and nonabsorbable suture are placed through the inframammary fold and inferior margin of the latissimus muscle (▶ Fig. 35.4). These sutures help define the inframammary fold. This can also be achieved with well-placed absorbable sutures inferiorly without the use of bolsters particularly in patients with a well-defined fold. We find the bolsters particularly helpful in delayed reconstruction patients to redefine the fold. The inferomedial attachments of the pectoralis muscle are divided and the muscle is elevated. The tissue expander is placed submuscularly, covered by the latissimus and pectoralis muscle, which are sutured together. The expander is placed meticulously right at the inframammary fold.

In smaller immediate reconstructions placing a shaped silicone implant rather than an expander is a reasonable option. Using an expander though affords the surgeon flexibility. If a shaped implant is chosen, the pocket needs to be sutured in such a way that the implant fits snugly into the pocket so that rotation of the implant does not occur.[6]

Some surgeons do not elevate the pectoralis muscle and use only the latissimus muscle to cover the device. This prevents pectoralis animation deformity and it is less painful. The potential disadvantage is that rippling may develop in the upper pole because of less muscle coverage.[7]

Closure of the back is performed in layers and quilting stitches are often placed to try decrease the risk of seroma. Drainage tubes are placed in the donor site and in the reconstructed breasts. The axillary tunnel is judiciously closed with a few absorbable sutures internally.

Fig. 35.3 Latissimus dorsi myocutaneous flap (LDMF) skin island placed into the inframammary fold. In this case, placing it into the old mastectomy defect would have left the patient with a very high scar.

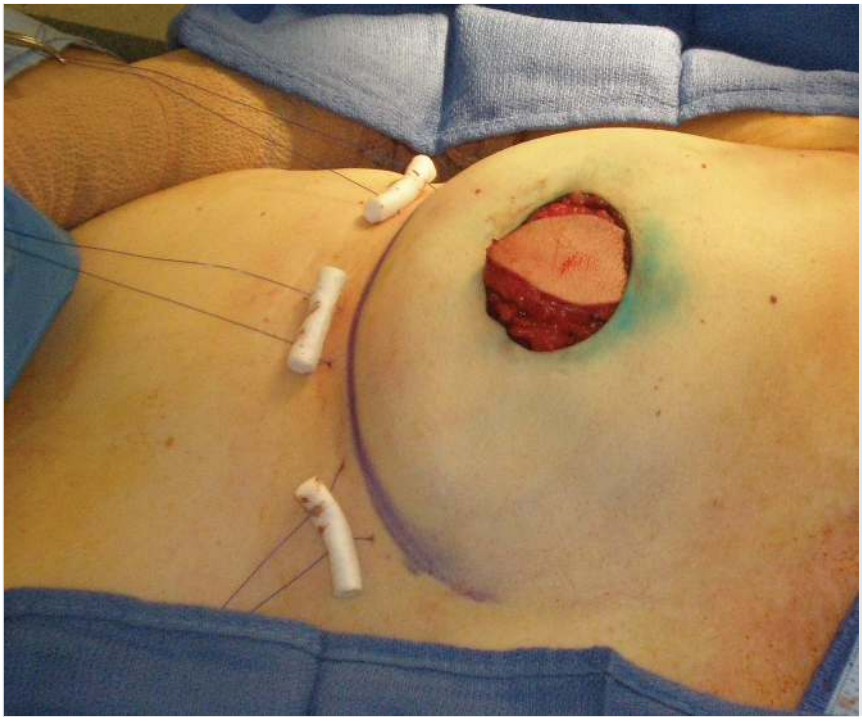


Fig. 35.4 The use of bolsters to inset the latissimus muscle into the inframammary fold is illustrated in this image. The skin will be de-epithelialized to match the skin-sparing mastectomy defect.

35.5 Postoperative Care

Sterile dressings are placed at the conclusion of the operation. A loose circumferential dressing is used, avoiding pressure or direct compression in the axilla to prevent vascular compromise of the pedicled flap. The skin island is dressed in such a manner as to be easily assessed for vascular integrity of the flap. The patient will usually have two to three drains, one or two in the donor site, and one or two in the breast. Pain control is with oral opiates and anti-inflammatory medications. Minimal intravenous narcotics are used in our practice. Urinary catheters are discontinued within 24 hours.

Dressings are removed in 24 to 48 hours. Venous thromboembolism prophylaxis utilizing both chemoprophylaxis and sequential compression devices is continued, and early ambulation is encouraged. Length of hospital stay is on average 1 day with return to work in 3 to 4 weeks. Patients are allowed to use their arm in the immediate postoperative period for very light activity and encouraged to utilize range of motion exercises starting at 2 weeks assuming an uncomplicated postoperative course to prevent shoulder stiffness. The drains are removed once output has decreased to less than 30 mL in 24 hours for 2 consecutive days.

In a two-stage reconstruction, tissue expander expansion begins once the incisions demonstrate adequate healing. This is approximately 2 to 3 weeks postoperatively. Serial expansions are done on average every 1 to 2 weeks in the office until the desired volume is achieved. The volume of expansion per visit is patient dependent but is typically 50 to 100 mL. The tissue expander is exchanged for a permanent prosthetic in the operating room 1 to 3 months after the last expansion. Exchange is done through the previous mastectomy incision. Capsule adjustment and pocket revision may be done during this procedure if indicated.

The thoracodorsal nerve can be proactively divided at the first operation or selectively at the second operation in those patients who complain of post-operative animation. Care must be taken not to injure the vascular pedicle during ligation of the nerve.

35.5.1 Outcomes

Flap necrosis after latissimus dorsi reconstruction is rare owing to the reliable and robust blood supply of the flap.[8] Necrosis of the mastectomy skin flaps is a more common complication, which in most cases can be managed conservatively. Migration of the prosthesis, prosthetic rupture, capsular contracture, or periprosthetic infection are recognized complications requiring operative intervention when a prosthetic device is used.

The most common complication after LDMF is donor-site seroma. The incidence of postoperative seroma is between 12 and 25%.[3,9,10] Most seromas may be managed by serial aspiration. On occasion, the use of a sclerosing agent such as tetracycline can help. Quilting sutures have been shown to help reduce the incidence of this complication.[11] If the seroma is large, painful, or inconvenient to the patient, it may be drained in the office setting. Rarely surgical intervention is required.

Reported patient satisfaction with breast reconstruction using latissimus dorsi flap is high.[3,8,12] Donor-site morbidity may include shoulder weakness, winging of the scapula, and impairment of shoulder mobility. However, most patients note that functionality is minimally impaired with a prospective study demonstrating no limitation of shoulder range of motion at 1 year in LDMF breast reconstruction patients.[9]

Long-term capsular contracture rates were historically as high as 30%; however, we have seen rates as low as 6% with the use of expanders (two-stage) and as low as 8% in irradiated patients.[4,5,6,7,13] More recently, lower rates have also been published using a textured shaped implant.[6] The LDMF is a relatively straightforward reliable technique for breast reconstruction. The use of a latissimus flap with or without tissue expander and permanent implant is an attractive option in patients with or without radiation changes to the breast. The complication rates following its use are low and patient satisfaction is high.

35.6 Review Questions

35.6.1 Fill in the Correct Answer

1. An absolute contraindication to latissimus dorsi myocutaneous flap breast reconstruction is
   a) Ipsilateral axillary irradiation.
   b) Ipsilateral breast irradiation.
   c) Previous rotator cuff surgery.
   d) Previous lateral thoracotomy.
2. What vessels make up the major vascular supply to a latissimus dorsi myocutaneous flap?
   a) Thoracoacromial.
   b) Internal mammary.
   c) Thoracodorsal.
   d) Superficial superior epigastric.
   e) Lateral thoracic.
3. What are the most common causes for latissimus dorsi myocutaneous flap necrosis?
   a) Previous pedicle transection in radiated field.
   b) Absence of secondary blood supply.
   c) Twisted pedicled.
   d) a and c.
   e) b and c.
4. What is the most common complication after latissimus dorsi myocutaneous flap breast reconstruction?
   a) Hematoma.
   b) Seroma.
   c) Partial skin necrosis.
   d) Fat necrosis.

35.6.2 Answers

1. Previous lateral thoracotomy (d).
2. Thoracodorsal (c).
3. a and c (d).
4. Seroma (b).

References

[1] Tansini I. Sopra il mio nuovo processo di amputazione della mammella. Gazz Med Ital. 57:141

[2] Fisher J, Bostwick J , III, Powell RW. Latissimus dorsi blood supply after thoracodorsal vessel division: the serratus collateral. Plast Reconstr Surg. 72(4): 502–511

[3] Lindegren A, Halle M, Docherty Skogh AC, Edsander-Nord A. Postmastectomy breast reconstruction in the irradiated breast: a comparative study of DIEP and latissimus dorsi flap outcome. Plast Reconstr Surg. 130(1):10–18

[4] Freeman ME, Perdikis G, Sternberg EG, TerKonda SP, Waldorf JC. Latissimus dorsi reconstruction: a good option for patients with failed breast conservation therapy. Ann Plast Surg. 57(2):134–137

[5] Sternberg EG, Perdikis G, McLaughlin SA, Terkonda SP, Waldorf JC. Latissimus dorsi flap remains an excellent choice for breast reconstruction. Ann Plast Surg. 56(1):31–35

[6] Hardwicke JT, Prinsloo DJ. An analysis of 277 consecutive latissimus dorsi breast reconstructions: a focus on capsular contracture. Plast Reconstr Surg. 128(1):63–70

[7] Mast BA, Simoneau DK. Latissimus dorsi breast reconstruction utilizing functional muscle transfer and tissue expansion. Ann Plast Surg. 56(1): 36–39

[8] Roy MK, Shrotia S, Holcombe C, Webster DJ, Hughes LE, Mansel RE. Complications of latissimus dorsi myocutaneous flap breast reconstruction. Eur J Surg Oncol. 24(3):162–165

[9] Venus MR, Prinsloo DJ. Immediate breast reconstruction with latissimus dorsi flap and implant: audit of outcomes and patient satisfaction survey. J Plast Reconstr Aesthet Surg. 63(1):101–105

[10] Glassey N, Perks GB, McCulley SJ. A prospective assessment of shoulder morbidity and recovery time scales following latissimus dorsi breast reconstruction. Plast Reconstr Surg. 122(5):1334–1340

[11] Lee KT, Mun GH. Fibrin sealants and quilting suture for prevention of seroma formation following latissimus dorsi muscle harvest: a systematic review and meta-analysis. Aesthetic Plast Surg. 39(3):399–409

[12] Perdikis G, Koonce S, Collis G, Eck D. Latissimus dorsi myocutaneous flap for breast reconstruction: bad rap or good flap? Eplasty. 11:e39

[13] Pinsolle V, Grinfeder C, Mathoulin-Pelissier S, Faucher A. Complications analysis of 266 immediate breast reconstructions. J Plast Reconstr Aesthet Surg. 59(10):1017–1024

36 Oncoplastic Breast Surgery

Albert Losken and Alexandra M. Hart

Abstract
This chapter discusses the elements pertinent to successful oncoplastic surgery. The challenge facing surgeons in breast reconstruction is to avoid common deformities (contour deformities, asymmetry, poor aesthetic outcomes) that stem from breast conservation therapy. The relation of various breast tumors to cosmetic outcome is thoroughly covered, and guidance is offered on oncoplastic resection and subsequent reconstruction employing volume displacement or replacement. Techniques examined include breast flap advancement, mastopexy, and the anterior intercostal artery perforator flap, along with others. Postoperative care guidelines and a review of outcomes and secondary procedures conclude the chapter.

Keywords: oncoplastic resection, oncoplastic reduction, breast conservation therapy (BCT), lumpectomy

36.1 Goals and Objectives

- Recognize the goals, benefits, patient selection process, and indications for performing oncoplastic surgery.
- Understand techniques for breast reshaping, preservation of nipple viability, and filling the tumor resection defect.
- Understand surgical options for treatment of positive margins, postoperative surveillance, and surgical outcomes following oncoplastic surgery.

36.2 Introduction

With the increasing popularity of breast conservation therapy (BCT), we have subsequently become increasingly aware of poor cosmetic results.[1,2,3] The wider the margin of resection, the lower the risk of local recurrence[4,5] and it often becomes a dilemma for the surgeon to meet both these end points. Breast shape becomes compromised and significant contour deformities, breast asymmetry, and poor aesthetic outcomes are not uncommon. Up to 30% of women will have a residual deformity that may require surgical correction,[6] the correction of which is often difficult.[7] This has resulted in attention to the oncoplastic approach whereby partial breast reconstruction is performed along with tumor resection in an attempt to prevent these deformities. The importance of teamwork and communication between the various services is critical for the successful incorporation of the oncoplastic approach. Its popularity will likely continue as long as we continue to demonstrate oncological safety and improved outcomes.[8,9,10,11,12]

36.2.1 Indications

Poor cosmetic results following BCT are not uncommon and usually due to breast shape, tumor size, tumor location, and postoperative radiation.[13] Traditionally, women with large breasts have been deemed poor candidates for breast conservation surgery, because of reduced effectiveness, increased complications, and worse cosmetic outcome. The postradiation sequela in women with *macromastia* is significantly worse. Radiation-induced fibrosis is thought to be greater in women with larger breasts, late radiation fibrosis is higher, and cosmetic results are also reduced.[14,15,16] *Tumor location* also plays a role with central or lower quadrant tumors having a worse cosmetic outcome. Lower quadrant tumors give twice as poor cosmetic results as lumpectomies in other quadrants. Central breast tumors close to the areolar have, in the past, been a contraindication to BCT. The *tumor-to-breast ratio* is one of the most important factors when predicting the potential for a poor outcome. Studies have shown a decline in cosmetic scores for patients with parenchymal resection greater than 70 to 100 cm^3, or when the specimen weight to breast volume ratio exceeds 10:1.[17,18,19] It is important that the reconstructive surgeons have an idea on the extent of the resection, whether lumpectomy or quadrantectomy. In general, when more than 20% of the breast is excised with partial mastectomy, the cosmetic result is likely to be unfavorable.

These are situations where partial breast reconstruction has significantly improved results and broadens the indications for BCT (▶ Table 36.1).

36.2.2 Patient Presentation and Selection

Partial breast reconstruction is indicated whenever the potential for a poor cosmetic result exists, or patients with tumors in whom a standard lumpectomy would lead to breast deformity or gross asymmetry. Factors in addition to cosmetic reasons as an indication for this approach include oncologic issues. Important indications include situations where the surgeon is concerned about the potential for negative margins with standard resection, and based on initial pathology or breast imaging studies, the surgeon needs to perform a *wider excision* in order for the patient to be a candidate for breast conserving surgery. Additional indications include women who desire breast conservation despite potential adverse conditions, as well as older women with large ptotic breasts in whom mastectomy and reconstruction would be difficult.

Table 36.1 Indications and goals of oncoplastic surgery

Cosmetic reasons	Oncological reasons
High tumor to breast ratio (> 20%)	Concern about clear margins
Tumor location—central, inferior, medial	Wide excision required
Macromastia	Poor candidate for mastectomy and reconstruction (i.e., age, breast size)
Large tumor	Patient desires BCT
Patient desires smaller breasts	
Significant ptosis, or breast asymmetry	

Abbreviation: BCT, breast conservation therapy.

36.3 Timing of Partial Breast Reconstruction

In general, partial breast reconstruction, when indicated, is best performed at the time of resection (*immediate reconstruction*). This has the benefits operating on a non-irradiated or surgically scarred defect, resulting in lower complication rates and improved aesthetic results.[7] The main concern with immediate reconstruction is the potential for positive margins. When this concern does exist, the reconstruction can be delayed until final confirmation of negative margins (*delayed-immediate reconstruction*). This then allows the benefits of reconstruction prior to radiation therapy with the luxury of clear margins, although at the expense of a second procedure. Such women at increased risk of positive margins included younger age (< 40 years old), extensive ductal carcinoa insitu (DCIS), high grade cancer, history of neoadjuvant chemotherapy, infiltrating lobular carcinoma, and Her2/neu positivity.[20,21,22] The main disadvantage is the need for a secondary procedure which might be unnecessary in the majority of cases. When a flap reconstruction is required, we prefer to confirm final margin status prior to partial breast reconstruction (▶ Table 36.2).

There are situations where poor results are encountered years following radiation therapy, which then require correction (*delayed reconstruction*). Similar techniques are employed in delayed reconstruction, more often requiring flaps such as the latissimus dorsi myocutaneous flap and associated with higher complication rates (42 vs. 26%) and worse cosmetic outcome.[7]

36.4 Margins

Oncoplastic resections tend to be more generous and have been shown to offer a margin advantage compared to lumpectomy alone.[14] Oncoplastic resections in some series have been over 200 g compared to institutional norms of about 40 to 50 g using lumpectomy alone.[23,24] This does not include the additional glandular excisions necessary to achieve symmetry with the reduced contralateral breast in patients with macromastia. Although there is a lack of randomized control data comparing the two groups, the incidence of positive margins in retrospective comparisons is significantly less in the oncoplastic reduction group compared to BCT alone. Kaur et al performed a prospective trial comparing quadrantectomy alone ($n = 30$) and resection with oncoplastic reconstruction ($n = 30$).[25] They demonstrated larger resection weights (200 vs. 118 g, $p = 0.16$) resulted in fewer close or positive margins (16.7 vs. 43.3%; $p = 0.5$) in the oncoplastic group.[15] Furthermore, DCIS histology was more prevalent in quadrantectomy-alone group and accounted for some of the differences. Giacolone et al performed a similar prospective comparative study comparing quadrantectomy alone ($n = 43$) and resection with oncoplastic reconstruction ($n = 31$).[26] The authors found margins greater than or equal to 5 mm in 67% of oncoplastic group versus 42% in the quadrantectomy-alone group ($p = 0.3$).[4] Losken et al demonstrated a lower positive margin rate (24.1 vs. 41.0%, $p = 0.01$), fewer surgical re-excisions (12.0 vs. 25.9%, $p = 0.01$), and wider margins from the tumor edge when oncoplastic surgery was performed (4.3 vs. 2.8 mm, $p = 0.01$).[14,27] A recent meta-analysis also found a reduction in the positive margin rate for both invasive and in situ disease from 21% with BCT alone to 12% in oncoplastic excisions.[9] The long-term influence of this on cancer recurrence remains to be seen. The use of this approach also allows additional sampling of ipsilateral and contralateral breast tissue with the ability to occasionally diagnose other breast pathology and potentially reduce cancer risk by removing additional breast tissue.[20]

It is important to minimize positive margins as much as possible. Preoperative breast imaging (i.e., MRI, ultrasound, or mammography) is helpful in determining the extent of the disease guiding the necessary resection and should be employed judiciously when indicated. An imaging study showed that tumor size was underestimated 14% by mammography, 18% by ultrasound, whereas MRI showed no difference when compared to the pathological specimen.[26] Wire identification and bracketing wires placed preoperatively will localize the extent of resection.[27] Intraoperative margin assessment requires multidisciplinary coordination between the surgeons, the pathologist, and the radiologist. Multicolored inking kits have proven to be more accurate than traditional stitch markings, especially for the more complex designed oncoplastic specimens.[28] Additional intraoperative confirmatory procedures include gross examination, radiography of the specimen, intraoperative frozen sections for invasive cancer, and touch cytology. Separate cavity margins sent at the time of lumpectomy significantly reduces the need for re-excision. Cao demonstrated that final margin status was negative in 60% of patients with positive margins on initial resection.[29] Rainsbury has established a one-stage approach where bed biopsies are taken from the cavity and subareolar region and sent for frozen section. The entire cavity is then inked and sent as a shave specimen for formal histology.[30] If tumor is still present in the second set of biopsies, then a mastectomy is indicated.

36.4.1 Surgical Planning

Oncoplastic Resection

Although the adverse effects of radiation therapy are often unavoidable, there are principles that can be applied to the resection with or without reconstructive techniques that can be used to minimize the incidence of poor cosmetic results. The

Table 36.2 Terminology of partial breast reconstruction

Partial breast reconstruction			
Timing		**Technique**	
Immediate	At the time of resection	Volume displacement	Volume replacement
Delayed Immediate	1–2 wk following resection (confirmation of margins status)		
Delayed	Following radiation therapy		

oncoplastic approach applies the principles of plastic surgery to the resection as well. A deformity can often be avoided by correctly orienting the breast incisions and parenchymal resection. Neoadjuvant chemotherapy will also downsize the tumor and reduce the required amount of parenchyma resection. Limiting the volume of resection will minimize the incidence of poor cosmetic results. Attention to simple defect closure including breast advancement flaps and full thickness closures are now commonly performed by most breast surgeons and are ways to improve results. The more complex defects with potential for poor cosmesis will often benefit from partial breast reconstruction. Placement of titanium perimeter clips to outline the lumpectomy cavity will guide the radiation therapy with postoperative tumor boost volume during teletherapy (external beam radiation). Communication is necessary between the oncologist and the surgeons especially when glandular remodeling has been performed. These clips will also assist in postoperative surveillance (▶ Fig. 36.1).[25]

36.5 Treatment Algorithm for Partial Reconstruction

Different algorithms have been described in an attempt to simplify the reconstructive process.[31] The decision as to which procedure is more appropriate is multifactorial; however, it is ultimately determined by breast size, tumor size, and tumor location (▶ Table 36.3). Other factors are also important including patient risks and desires, tumor biology, and surgeon's comfort level with the various techniques. Being familiar with the various reconstructive tools will allow reconstruction of almost any partial mastectomy defect. It is important to keep in mind that when the defect is extensive with little remaining breast tissue, then completion mastectomy and immediate reconstruction is often the most appropriate option.

Some simple rules of thumb exist for reconstructing partial mastectomy defects. Large or moderate sized breasts or ptotic breasts with sufficient parenchyma remaining following resection are amenable to *volume displacement or reshaping procedures*. Quadrantectomy-type resections are possible when within the standard Wise pattern markings. In smaller or nonptotic breasts when additional volume is required to match the opposite breast or when skin is required to replace a resection that included parenchyma and skin, *volume replacement procedures* including volume and skin are required. Quadrantectomy-type resections in small breasts, and in the upper or outer quadrant, will invariably require a flap reconstruction to preserve shape.

Table 36.3 Partial mastectomy reconstruction techniques

Volume displacement techniques	Volume replacement techniques
"Parenchymal remodeling, volume shrinkage"	*"Adjacent or distant tissue transfer, volume preserving"*
Primary closure	Implant augmentation—rare
Mirror biopsy/excision Batwing mastopexy	Local flaps: • Fasciocutaneous • Perforator flaps • Latissimus dorsi myocutaneous flap
Breast flap advancement technique	Distant flaps
Nipple areolar centralization	
Reduction mastopexy techniques	

36.5.1 Volume Displacement Techniques

The breast reshaping procedures rely on advancement, rotation, or transposition of a large area of breast to fill a small or moderate sized defect. This absorbs the volume loss over a larger area. In its simplest form, it entails mobilizing the breast plate from the area immediately around the defect in a *breast flap advancement technique*.[32]

Perhaps the most popular and versatile breast reshaping options are the *mastopexy or reduction techniques*. The ideal patient is one where the tumor can be excised within the expected breast reduction specimen, in medium to large or ptotic breasts where sufficient breast parenchyma remains following resection to reshape the mound. Masetti et al described a four-step design for oncoplastic operations: (1) planning skin incisions and parenchymal excisions following reduction/mastopexy templates, (2) parenchymal reshaping following excision, (3) repositioning the nipple, and (4) correction of the contralateral breast for symmetry.[33] Any moderate to large breast can be reconstructed using these techniques unless a skin deformity exists beyond the standard Wise pattern.

Plastic surgeons are all familiar with these techniques, making the incorporation of this approach into their reconstructive practice an easy addition. In women with large or ptotic breasts, the numerous reduction patterns or pedicle designs will invariably allow remodeling of a defect in any location and any size, as long as sufficient breast tissue and skin is available. Creative mammaplasty designs can be made for complete removal of the lesion and reshaping of the mound for both lumpectomy and quadrantectomy-type defects. *Preoperative markings* are important, and a decision is made on pedicle design depending on tumor location. Typically if the pedicle points to or can be rotated into the defect, it can be used. The Wise pattern markings are more versatile allowing tumor resection in any breast quadrant. Once the resection is performed, the cavity is inspected paying attention to the defect location in relation to the nipple, as well as the remaining breast tissue. The reconstructive goals include (1) preservation of nipple viability, (2) reshaping of breast mound, and (3) closure of dead space. The nipple and dermoglandular pedicle are dissected, and remaining tissue is resected if necessary for completion of the reduction. Occasionally, additional dermoglandular or glandular pedicles can be created from tissue that might otherwise have been resected, and rotated to autoaugment the defect. The contralateral procedure is performed using a similar technique. The ipsilateral side is typically kept about 10% larger to allow for radiation fibrosis. Additional tissue sampling from the ipsilateral or contralateral breast is also possible using this technique.[34,35,36]

Lower quadrant tumors in women with larger breasts are ideally suited for the oncoplastic approach.[37] Quadrantectomy-type resections are possible, removing skin and parenchyma from this location, reshaping the breast using a superior or superomedial pedicle. Lower pole tumors in moderate size breasts can be excised along with skin as needed in the usual

Fig. 36.1 **(a,b)** This is a 50-year-old female with a left lower pole breast cancer. **(c,d)** She underwent a generous 120 gram tumor resection including breast tissue and skin. **(e,f)** When the defect is below the Wise pattern markings and a reduction is planned, a generous resection is possible. If additional resection is performed by the reconstructive surgeon during the reduction it is important to mark the specimen appropriately since this would potentially be a new margin if the original margins are positive. A lower pole defect can be reconstructed using any oncoplastic reduction technique except an inferior pedicle. **(g,h)** A superomedial pedicle was chosen because it is a relatively short pedicle and a total of 320 grams was resected on the left including the lumpectomy specimen. The cavity was marked with clips prior to closure. A superomedial reduction was performed on the contralateral side with 360 grams removed. It is common to over resect the contralateral side in anticipation of irradiation fibrosis with time. **(i)** Her result is shown 5 months post operatively and prior to irradiation therapy.

vertical pattern utilizing a superior pedicle followed by plication of the vertical pillars, and vertical reduction on the contralateral side (▶ Fig. 36.1). *Upper quadrant tumors* can be filled as long as the defect is under the skin (lumpectomy type). Autoaugmentation techniques have become popular to fill the dead space and maintain shape. Inferior or medial pedicles allow for safe excisions in the upper half of the breast without impairing nipple viability and parenchyma is often rearranged when insufficient tissue remains in the upper pole to maintain the desired fullness. When skin is resected in the upper half of the breast, such remodeling techniques are not possible. Lateral or upper-outer quadrant defects allow parenchymal remodeling using the superomedial pedicle. These types of reconstructions become difficult when skin is resected with the specimen and are better suited for the lumpectomy-type defects. In women with medium size ptotic breasts, the superomedial pedicle can

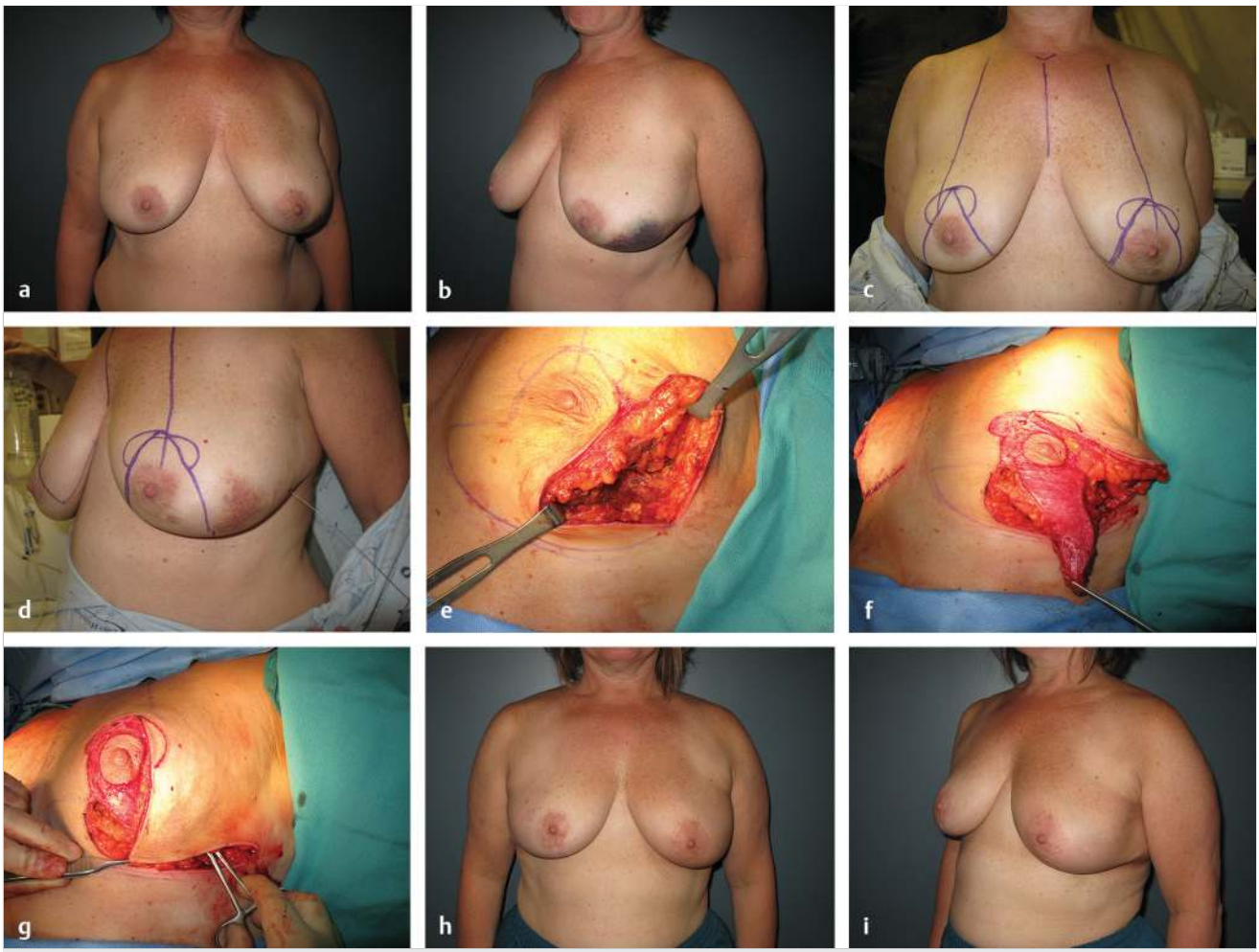


Fig. 36.2 **(a,b)** This is a 51 year old female with a left lower lateral quadrant DCIS with a left lower lateral quadrant DCIS. **(c,d)** She had wire localization and was marked with the Wise pattern. **(e)** After a 60 gram partial mastectomy she was left with a large lateral defect. It was felt that additional tissue needed to be rotated to fill the defect, rather than just using the residual lateral tissue. **(f,g)** This was reconstructed with an extended superomedial pedical and an additional 30 gram resection from the left side. **(h,i)** A contralateral reduction of 120 grams was performed. She is shown 1 year following completion of left sided radiation therapy.

be extended down to the inframammary fold as an autoaugmented pedicle. This can then be rotated to fill a lateral volume void. The vertical pillars are then plicated in the usual fashion to maintain shape. If tissue is removed from above the Wise pattern markings, and flap is often required. In women with macromastia, a reduction can still be performed utilizing the inferior pedicle skin to replace missing breast skin even when above the Wise markings (▶ Fig. 36.2).

Central tumors can be challenging from a cosmetic perspective; however, partial reconstruction often makes these patients a reasonable candidate for BCT.[38] If the NAC is removed with the resection, the mound can be remodeled in the inverted T-closure pattern, and nipple reconstructed at a later stage. Another option if the tumor is located more superiorly or lateral is to perform a central elliptical excision of skin, nipple, and parenchyma, and mirror image contralateral reduction for symmetry. A third option includes creation of a skin island on a dermoglandular pedicle to rotate into the central defect to allow for shape preservation and nipple reconstruction. The breast is marked preoperatively for an inverted T or a vertical approach depending on breast size, and the skin island is brought in from inferior or medial.

Larger quadrantectomy defects, especially above the nipple, can be incorporated into a batwing mastopexy or elliptical incision and provide preservation or improvement of shape and elevation of the ptotic breast along with the tumor resection. A similar mirror image resection is often performed on the opposite side for symmetry. Additional mastopexy options exist for oncoplastic breast conservation.[32] The *donut mastopexy* allows a breast segment to be removed through a periareolar incision, and is useful for segmentally distributed cancers in the upper or lateral portion of the breast. The *batwing mastopexy* involves a full-thickness excision of lesions deep within the breast centrally or adjacent to the nipple-areolar complex. The two similar half-circle incisions with angled wings on either side of the areolar allow advancement of the fibroglandular tissue to close the defect. Since this removes sufficient breast tissue and skin to alter the size of the breast and nipple position, a similar contralateral lift is occasionally required to achieve symmetry. Additionally, if the patient is a candidate for BCT and has

multiple areas that need to be resected, as long as sufficient tissue remains, remodeling techniques can be used in a similar fashion.

36.5.2 Volume Replacement Techniques

Partial mastectomy defects in women with small to medium breasts are often difficult to reconstruct.[39] Women with large tumor-to-breast ratios and women with small to moderate breasts who have insufficient residual breast tissue for rearrangement require partial reconstruction using non-breast local or distant flaps. This is now well accepted in the evolution of breast cancer surgery and provides breast symmetry without remodeling the contralateral breast.

Local flaps are often indicated in small or moderate volume breasts with insufficient tissue remains following resection for volume displacement techniques. The usual techniques include (1) rhomboid flaps, (2) subaxillary flap, (3) superior-based lateral thoracodorsal flap, (4) inferior-based lateral thoracodorsal flap, and (5) the extended lateral thoracodorsal flap. Small lateral defects (< 10% of breast size) can be closed with *local flaps*. Clough et al described using the subaxillary area as a transposition flap, and Munhoz et al more recently demonstrated how the lateral thoracodorsal flap (LTDF) is ideal for lateral defects, especially in obese patients.[40,41] These flaps essentially rotate or transfer skin and subaxillary fat or skin and breast parenchyma into the defect. The same principles can be applied to local flaps taken from outside the breast as described earlier, or even from within the breast (volume displacement techniques). Attention to flap design is important to ensure flap survival, cosmesis, and appropriate conversion to a completion mastectomy if necessary. The latissimus dorsi *musculocutaneous flap* is a common local option for lateral, central, inferior, and even medial defects.[30,42,43] It has excellent blood supply and provides both muscle for filling of glandular defects and skin for cutaneous deficiencies. Avoiding a scar on the back can be achieved by harvesting the LD without skin through the lateral breast incision. The use of an endoscope can assist in raising the muscle.[43] A deinnervated and radiated LD will undergo postoperative atrophy. To compensate for the expected loss in muscle volume, a flap much larger than the defect should be harvested, possibly preserving subscarpal fat on the muscle. A similar skin island to the classical LD musculocutaneous flap can be raised as a *pedicled perforator flap* either from the thoracodorsal or intercostal vessels. Sparing the underlying muscles or using perforator flaps have reduced the donor-site morbidity to the minimum, with no seroma formation at the donor site.[44,45] *The thoracodorsal artery perforator (TDAP) flap* can easily reach defects in the lateral, superolateral, and central regions of the breast. If no suitable perforators are found, the flap is easily converted to a muscle sparing—TDAP or muscle sparing—LD flap. *The lateral intercostal artery perforator (LICAP) flap* is another alternative to the TDAP flap for lateral and inferior breast defects. The lateral intercostal artery perforators are found at 2.7 to 3.5 cm from the anterior border of the LD muscle.[46] *The anterior intercostal artery perforator (AICAP) flap is* similar to the random-designed thoraco-epigastric skin flap, the skin paddle can be harvested as an AICAP flap. The AICAP is based on perforators originating from the intercostal vessels through the rectus abdominis or the external oblique muscles. Since it has a short pedicle, the AICAP flap is suitable to cover close defects that extend over the inferior or medial quadrants of the breast. *The superior epigastric artery perforator (SEAP) flap* is based on perforators arising from the superior epigastric artery or its superficial branch. It has the same indications as the AICAP flap; however, the SEAP flap has longer pedicle and therefore it can cover more remote defect in the breast.

Large medial defects are more difficult for reconstruction. The superficial inferior epigastric artery free flap has been described for this location.[47,48] In situations such as this, or when the partial mastectomy defect is significant with minimal residual breast tissue, a decision needs to be made whether to complete the mastectomy and perform total breast reconstruction for both cosmetic and oncological reasons.

Various other techniques have been described to fill partial mastectomy defects; however, these are currently less common. They include abdominal adipofascial flaps, omental flaps, and autologous fat injections.[49,50,51,52]

Oncologic Safety

- Appropriate patient selection.
- Preoperative planning (imaging, wires) to assist with resection.
- Confirm negative margins in high-risk patients (DCIS, age < 40).
- Consider confirmation of negative margins when performing flap reconstruction.
- Intraoperative margin assessment (multidisciplinary approach).
- Separate cavity sampling.
- Clip cavity for radiation planning and surveillance.
- Appropriate postoperative surveillance protocols.

36.6 Postoperative Care

One main concern with partial breast reconstruction is that it might impair the ability to screen and detect recurrent breast cancer. Some fear that glandular rearrangement, additional scarring, and the disruption of architecture might alter the potential patterns of local recurrence of the ability to screen and detect these lesions. Although this is a valid concern, adherence to appropriate surveillance and cross-specialty communication will reduce this issue. The three main tools when it comes to postoperative surveillance include the physical examination, radiologic imaging, and tissue sampling. It is important that all members of the team are aware of the various surgical components, as differences in presentation might exist depending on the type and technique of reconstruction. We recently demonstrated that mammography following partial breast reconstruction using reduction techniques was just as sensitive as a screening tool when compared to patients with BCT alone.[53] Although the qualitative mammographic findings were similar in the two groups over the average 6-year follow-up, there was a slight trend toward longer times to mammographic stability in the oncoplastic reduction group (25.6 vs. 21.2 months in the BCT-alone group). This means that it might take the oncoplastic reduction patients slightly longer to reach the point where any change in mammographic findings might be suspicious for malignancy. An accurate interpretation requires

familiarity with these temporal changes and mammograms should be compared over time. These data need to be taken into consideration when designing the most appropriate surveillance programs for these patients. Mammographic findings following myocutaneous flap reconstructions typically show areas of radiolucency consistent with a fibrofatty component in most flaps. Microcalcifications and areas of fat necrosis are easily identified, and no interference in postoperative surveillance has been demonstrated. Other imaging techniques such as ultrasound and MRI will likely become more popular as technology improves. Although routine tissue sampling is not recommended for screening, any clinical concern necessitates fine needle aspiration, core needle biopsy, or surgical biopsy to rule out malignancy. Patients who undergo partial breast reconstruction are expected to have an increase in the amount of tissue sampling requirements, as demonstrated in our series (53% in the oncoplastic group compared to 18% in the BCT-alone group over an average of 7 years).[53] Although these are typically benign, additional scarring from the reconstruction might raise clinical suspicion, which is why more biopsies are expected in patients who undergo partial breast reconstruction.

36.7 Outcomes and Secondary Procedures

It is important that complications resulting from oncoplastic techniques do not interfere with the initiation of adjuvant therapy. Careful selection of surgical technique, appropriate patient selection, and meticulous execution will minimize the incidence of postoperative complications. Additional procedures will invariably increase complications; however, most of these are minor. Some larger series with volume displacement techniques report complications such as delayed wound healing (3–15%), fat necrosis (3–10%), and infection (1–5%).[7,20,34] Overall complications following volume replacement techniques are slightly higher (range: 2–77%) and this is likely due to the addition of donor-site complications and potential flap loss issues.[30,42,43] Delayed complications with the oncoplastic approach include breast fibrosis and asymmetry. Although the goal of partial breast reconstruction is to prevent the unfavorable cosmetic result, this approach cannot prevent or reverse the effects of radiation therapy. Since these effects will persist, the assessment of shape and symmetry needs to be made in the long term. However, with partial reconstruction, shape is typically preserved and it is easier to adjust the contralateral side secondarily if necessary than reconstruct a radiated BCT deformity. Asgeirsson et al reviewed numerous series with intermediate follow-up and demonstrated cosmetic failure rates of 0 to 18%.[54] BCT alone has a poor aesthetic result in 20 to 30 patients; however, with the incorporation of oncoplastic breast surgery techniques, this can be dropped to below 7% at 2 years.[55,56] *Local recurrence* is another important outcome that needs to be evaluated in the oncoplastic patient. Most reviews in the literature are of intermediate follow-up (up to 4.5 years), with local recurrence rates varying from 0 to 1.8% per year.[54] Actuarial 5-year local recurrence rates range from 8.5 to 9.4%. Longer term studies are required.

Secondary procedures are not common, and usually needed for size and shape changes in the long term. Although this approach minimizes the potential for poor aesthetic results, the effects of radiation therapy persist and can contribute to changes over time. It is better to wait at least a year following completion of radiation therapy to discuss revisional procedures. Even with resolution of acute radiation changes and tissue edema, it is important to respect tissue planes and blood supply and if possible keep any revisions to a minimum as it is still an irradiated breast and carries the associated risks. Perhaps the largest series of 540 consecutive cases from the Institut Curie demonstrated that in patients with high tumor-to-breast volume ratios, use of the oncoplastic approach provides good outcomes.[53] The 5-year local recurrence (6.8%) and overall survival rates (92.9%) were acceptable with low complication rates (16%) and good aesthetic results at 1 year (97.7%) and at 5 years (90.3%).

When completion mastectomy and reconstruction are required following oncoplastic reduction techniques, the disadvantages of this approach are minimal. All reconstructive options are still available, the contralateral symmetry procedure has already been performed, and it is easier to reconstruct a smaller breast.

36.8 Conclusion

The benefits of using the oncoplastic approach with BCT have been well demonstrated, and will continue to gain popularity and acceptance in the future. The options for women with breast cancer are numerous, and this provides an additional, often favorable one. We need to critically evaluate results measuring functional, oncological, and aesthetic outcomes in an attempt to establish safe and effective practice guidelines to maximize oncological safety.

36.9 Review Questions

1. Which of the following has not been demonstrated in women with breast cancer who undergo reduction at the time of lumpectomy?
   a) Improve the patient's quality of life compared to BCT alone.
   b) Reduce the incidence of positive margins.
   c) Broaden the indications for BCT.
   d) Improve radiation inhomogeneity.
   e) Detection of a synchronous tumor on the opposite breast.
2. Poor cosmetic results following breast conservation therapy is associated with
   a) Women with large breasts.
   b) Large tumor to breast ration (> 20%).
   c) Central tumors.
   d) Medial tumor location.
   e) All of the above.
3. A major concern with doing a flap to reconstruct a partial mastectomy defect at the time of lumpectomy is
   a) Positioning of the patient.
   b) Coordinating care between services.
   c) Concerns about positive margins.
   d) Making radiation therapy more difficult.
   e) Poor surveillance for breast cancer.

4. Regarding postoperative breast surveillance following oncoplastic reduction surgery, which of the following is correct:
   a) There is a longer time to mammographic stabilization compared to BCT alone.
   b) Less need for postoperative tissue sampling.
   c) Additional scarring reduces mammographic sensitivity.
   d) Always do an MRI.
5. An oncoplastic breast reduction at the time of lumpectomy is preferred in
   a) Anyone who undergoes BCT.
   b) Women with smaller breasts and a large tumor.
   c) Women with macromastia who are candidates for BCT.
   d) Any tumor in the lower pole.
   e) When there is a concern about achieving positive margins.

36.9.1 Answers

1. d. Improve radiation inhomogeneity.
2. e. All of the above.
3. c. Concerns about positive margins.
4. a. There is a longer time to mammographic stabilization compared to BCT alone.
5. Women with macromastia who are candidates for BCT.

References

[1] NCI Breast Cancer Database, 2007

[2] Fisher B, Anderson S, Bryant J, et al. Twenty-year follow-up of a randomized trial comparing total mastectomy, lumpectomy, and lumpectomy plus irradiation for the treatment of invasive breast cancer. N Engl J Med. 347(16): 1233–1241

[3] Veronesi U, Cascinelli N, Mariani L, et al. Twenty-year follow-up of a randomized study comparing breast-conserving surgery with radical mastectomy for early breast cancer. N Engl J Med. 347(16):1227–1232

[4] Silverstein MJ, Lagios MD, Groshen S, et al. The influence of margin width on local control of ductal carcinoma in situ of the breast. N Engl J Med. 340(19): 1455–1461

[5] Veronesi U, Volterrani F, Luini A, et al. Quadrantectomy versus lumpectomy for small size breast cancer. Eur J Cancer. 26(6):671–673

[6] Clough KB, Cuminet J, Fitoussi A, Nos C, Mosseri V. Cosmetic sequelae after conservative treatment for breast cancer: classification and results of surgical correction. Ann Plast Surg. 41(5):471–481

[7] Kronowitz SJ, Feledy JA, Hunt KK, et al. Determining the optimal approach to breast reconstruction after partial mastectomy. Plast Reconstr Surg. 117(1): 1–11, discussion 12–14

[8] Losken A, Hamdi M. Partial breast reconstruction: current perspectives. Plast Reconstr Surg. 124(3):722–736

[9] Slavin SA, Love SM, Sadowsky NL. Reconstruction of the radiated partial mastectomy defect with autogenous tissues. Plast Reconstr Surg. 90(5):854–865, discussion 866–869

[10] Audretsch WP. Reconstruction of the partial mastectomy defect: classification and method. In: Spear SL, Willey SC, Robb GL, Hammond DC, Nahabedian MY, eds. Surgery of the Breast: Principles and Art. Philadelphia, PA: Lippincott Williams & Wilkins 2006:179–121

[11] Malycha PL, Gough IR, Margaritoni M, et al. Oncoplastic breast surgery: a global perspective on practice, availability, and training. World J Surg. 32(12): 2570–2577

[12] Munhoz AM, Aldrighi CM, Ferreira MC. Paradigms in oncoplastic breast surgery: a careful assessment of the oncological need and esthetic objective. Breast J. 13(3):326–327

[13] Matory WE , Jr, Wertheimer M, Fitzgerald TJ, Walton RL, Love S, Matory WE. Aesthetic results following partial mastectomy and radiation therapy. Plast Reconstr Surg. 85(5):739–746

[14] Brierley JD, Paterson IC, Lallemand RC, Rostom AY. The influence of breast size on late radiation reaction following excision and radiotherapy for early breast cancer. Clin Oncol (R Coll Radiol). 3(1):6–9

[15] Zierhut D, Flentje M, Frank C, Oetzel D, Wannenmacher M. Conservative treatment of breast cancer: modified irradiation technique for women with large breasts. Radiother Oncol. 31(3):256–261

[16] Clarke DH, Lê MG, Sarrazin D, et al. Analysis of local-regional relapses in patients with early breast cancers treated by excision and radiotherapy: experience of the Institut Gustave-Roussy. Int J Radiat Oncol Biol Phys. 11(1): 137–145

[17] Mills JM, Schultz DJ, Solin LJ. Preservation of cosmesis with low complication risk after conservative surgery and radiotherapy for ductal carcinoma in situ of the breast. Int J Radiat Oncol Biol Phys. 39(3):637–641

[18] Taylor ME, Perez CA, Halverson KJ, et al. Factors influencing cosmetic results after conservation therapy for breast cancer. Int J Radiat Oncol Biol Phys. 31 (4):753–764

[19] Cochrane RA, Valasiadou P, Wilson AR, Al-Ghazal SK, Macmillan RD. Cosmesis and satisfaction after breast-conserving surgery correlates with the percentage of breast volume excised. Br J Surg. 90(12):1505–1509

[20] Losken A, Styblo TM, Carlson GW, Jones GE, Amerson BJ. Management algorithm and outcome evaluation of partial mastectomy defects treated using reduction or mastopexy techniques. Ann Plast Surg. 59(3):235–242

[21] Miller AR, Brandao G, Prihoda TJ, et al. Positive margins following surgical resection of breast carcinoma: analysis of pathologic correlates. Am J Surg. 187(5):647–650

[22] Song HM, Styblo TM, Carlson GW, Losken A. The use of oncoplastic reduction techniques to reconstruct partial mastectomy defects in women with ductal carcinoma in situ. Breast J. 16(2):141–146

[23] Leong C, Boyages J, Jayasinghe UW, et al. Effect of margins on ipsilateral breast tumor recurrence after breast conservation therapy for lymph node-negative breast carcinoma. Cancer. 100(9):1823–1832

[24] Clough KB, Lewis JS, Couturaud B, Fitoussi A, Nos C, Falcou MC. Oncoplastic techniques allow extensive resections for breast-conserving therapy of breast carcinomas. Ann Surg. 237(1):26–34

[25] Kaur N, Petit JY, Rietjens M, et al. Comparative study of surgical margins in oncoplastic surgery and quadrantectomy in breast cancer. Ann Surg Oncol. 12 (7):539–545

[26] Giacolone PL, et al. comparative study of the accuracy of breast resection in oncoplastic surgery and quadrantectomy in breast cancer. Ann Surg Oncol. 2007;14(2):605-614

[27] Losken A, et al. The oncoplastic reduction approach to breast conservation therapy: benefits for margin control. Aesthet Surg J.2014;34 (8):1185-1191

[28] Boetes C, Mus RD, Holland R, et al. Breast tumors: comparative accuracy of MR imaging relative to mammography and US for demonstrating extent. Radiology. 197(3):743–747

[29] Liberman L, Kaplan J, Van Zee KJ, et al. Bracketing wires for preoperative breast needle localization. AJR Am J Roentgenol. 177(3):565–572

[30] Neuschatz AC, DiPetrillo T, Steinhoff M, et al. The value of breast lumpectomy margin assessment as a predictor of residual tumor burden in ductal carcinoma in situ of the breast. Cancer. 94(7):1917–1924

[31] Cao D, Lin C, Woo SH, Vang R, Tsangaris TN, Argani P. Separate cavity margin sampling at the time of initial breast lumpectomy significantly reduces the need for reexcisions. Am J Surg Pathol. 29(12):1625–1632

[32] Rainsbury RM. Breast-sparing reconstruction with latissimus dorsi miniflaps. Eur J Surg Oncol. 28(8):891–895

[33] Kronowitz SJ, Kuerer HM, Buchholz TA, Valero V, Hunt KK. A management algorithm and practical oncoplastic surgical techniques for repairing partial mastectomy defects. Plast Reconstr Surg. 122(6):1631–1647

[34] Anderson BO, Masetti R, Silverstein MJ. Oncoplastic approaches to partial mastectomy: an overview of volume-displacement techniques. Lancet Oncol. 6(3):145–157

[35] Masetti R, Pirulli PG, Magno S, Franceschini G, Chiesa F, Antinori A. Oncoplastic techniques in the conservative surgical treatment of breast cancer. Breast Cancer. 7(4):276–280

[36] Munhoz AM, Montag E, Arruda EG, et al. Critical analysis of reduction mammaplasty techniques in combination with conservative breast surgery for early breast cancer treatment. Plast Reconstr Surg. 117(4):1091–1103, discussion 1104–1107

[37] Goffman TE, Schneider H, Hay K, Elkins DE, Schnarrs RA, Carman C. Cosmesis with bilateral mammoreduction for conservative breast cancer treatment. Breast J. 11(3):195–198

[38] Chang E, Johnson N, Webber B, et al. Bilateral reduction mammoplasty in combination with lumpectomy for treatment of breast cancer in patients with macromastia. Am J Surg. 187(5):647–650, discussion 650–651
[39] Clough KB, Nos C, Salmon RJ, Soussaline M, Durand JC. Conservative treatment of breast cancers by mammaplasty and irradiation: a new approach to lower quadrant tumors. Plast Reconstr Surg. 96(2):363–370
[40] McCulley SJ, Durani P, Macmillan RD. Therapeutic mammaplasty for centrally located breast tumors. Plast Reconstr Surg. 117(2):366–373
[41] Berrino P, Campora E, Santi P. Postquadrantectomy breast deformities: classification and techniques of surgical correction. Plast Reconstr Surg. 79(4):567–572
[42] Clough KB, Kroll SS, Audretsch W. An approach to the repair of partial mastectomy defects. Plast Reconstr Surg. 104(2):409–420
[43] Munhoz AM, Montag E, Arruda EG, et al. The role of the lateral thoracodorsal fasciocutaneous flap in immediate conservative breast surgery reconstruction. Plast Reconstr Surg. 117(6):1699–1710
[44] Munhoz AM, Montag E, Fels KW, et al. Outcome analysis of breast-conservation surgery and immediate latissimus dorsi flap reconstruction in patients with T1 to T2 breast cancer. Plast Reconstr Surg. 116(3):741–752
[45] Losken A, Schaefer TG, Carlson GW, Jones GE, Styblo TM, Bostwick J , III. Immediate endoscopic latissimus dorsi flap: risk or benefit in reconstructing partial mastectomy defects. Ann Plast Surg. 53(1):1–5
[46] Hamdi M, Van Landuyt K, de Frene B, Roche N, Blondeel P, Monstrey S. The versatility of the inter-costal artery perforator (ICAP) flaps. J Plast Reconstr Aesthet Surg. 59(6):644–652
[47] Hamdi M, Van Landuyt K, Monstrey S, Blondeel P. Pedicled perforator flaps in breast reconstruction: a new concept. Br J Plast Surg. 57(6):531–539
[48] Hamdi M, Spano A, Van Landuyt K, et al. The lateral intercostal artery perforators: An anatomical study and functional application in breast surgery. Plast Reconstr Surg. 2007; 121(2):389–396
[49] Spiegel AJ, Khan FN. An intraoperative algorithm for use of the SIEA flap for breast reconstruction. Plast Reconstr Surg. 120(6):1450–1459
[50] Rizzuto RP, Allen RJ. Reconstruction of a partial mastectomy defect with the superficial inferior epigastric artery (SIEA) flap. J Reconstr Microsurg. 20(6): 441–445, discussion 446
[51] Zaha H, Inamine S, Naito T, Nomura H. Laparoscopically harvested omental flap for immediate breast reconstruction. Am J Surg. 192(4): 556–558
[52] Ogawa T, Hanamura N, Yamashita M, Ri Y, Kuriyama N, Isaji S. Usefulness of breast-volume replacement using an inframammary adipofascial flap after breast-conservation therapy. Am J Surg. 193(4):514–518
[53] Kitamura K, et al. Stem cell augmented reconstruction: a new hope for reconstruction after breast conservation therapy. Breast Cancer Res Treat. 106 Supp1:4071
[54] Rageth CJ, Tausch C. Intramammarian flap reconstruction (IFR) technique in breast conserving surgery. Breast. 18(6):387–392
[55] Losken A, Schaefer TG, Newell M, Styblo TM. The impact of partial breast reconstruction using reduction techniques on postoperative cancer surveillance. Plast Reconstr Surg. 124(1):9–17
[56] Asgeirsson KS, Rasheed T, McCulley SJ, Macmillan RD. Oncological and cosmetic outcomes of oncoplastic breast conserving surgery. Eur J Surg Oncol. 31 (8):817–823
[57] Fitoussi AD, Berry MG, Famà F, et al. Oncoplastic breast surgery for cancer: analysis of 540 consecutive cases [outcomes article]. Plast Reconstr Surg. 125 (2):454–462
[58] Munhoz AM, Montag E, Arruda E, et al. Assessment of immediate conservative breast surgery reconstruction: a classification system of defects revisited and an algorithm for selecting the appropriate technique. Plast Reconstr Surg. 121(3):716–727

37 Tuberous Deformity of the Breast (Breast Implantation Base Constriction)

Manuel R. Vegas and José L. M. del Yerro

Abstract

This chapter examines tuberous breast deformities. Beginning with etiopathology at patient presentation, the authors guide the reader through each anatomic component and discuss how to properly contour each anomaly and/or deformity encountered. Discussion of surgical treatment starts with the incision and proceeds to the correction of anomalies/deformities. Remedial techniques proposed include customized dermoglandular flaps, glandular scoring incisions, and gland unfurling, which the authors consider the best option and thus cover in detail. Drawings and photographs greatly assist in illustrating the text. The comprehensiveness of the chapter is seen in the space devoted to other techniques, such as tissue expansion and fat grafting. Paragraphs on postoperative care, outcomes, and complications conclude the study.

Keywords: tuberous breast, breast constriction, gland scoring, gland unfurling

37.1 Goals and Objectives

- Understand the basics of tuberous breast deformity.
- Define the key findings of the varying anomalies and appreciate the benefits of addressing each anatomic component for proper contouring.
- Know the evidence-based perioperative care to maximize patient safety and quality outcomes.

37.2 Patient Presentation

The tuberous breast, which might be better termed "breast implantation breast constriction (BIBC)," is a congenital anomaly that only becomes apparent at the time of breast development. Although rare, it may also occur in males as a form of gynaecomastia.[1] Several other terms to describe the anomaly have been used in the English literature that should likewise be avoided, including tubular breast, constricted breast, doughnut breast, snoopy deformity, nipple breast, breast with narrow base and dome nipple. In severe cases, patients can suffer from social anxiety, depression, peer rejection, psychosexual dysfunction, and low self-esteem.[2] With no exact etiology yet clarified, it is accepted that it has an embryological origin although no family incidence or noxious fetal stimulus has been recognized. Its true incidence is unknown because its clinical definition has not yet been clearly stated.

37.2.1 Etiopathology

In order to understand the tuberous breast and appropriate treatment, an appreciation of breast fascial development and anatomy is mandatory. During gestation, the developing ectodermal breast bud penetrates the underlying mesenchyme and becomes enclosed within the superficial and the deep layers of the fascia superficialis that is continuous with the superficial abdominal fascia of Camper.[3,4] The superficial layer covers the breast parenchyma while the deep layer forms its posterior boundary and is separated from the underlying muscle fascia (pectoralis major and serratus anterior) by the loose areolar space of Chassaignac.[5] Vertical fibrous attachments (suspensory ligaments of Cooper) extend from the deep muscle fascia and the deep fascia of the fascia superficialis through the breast parenchyma and join the superficial fascia and the dermis. The superficial layer of the fascia superficialis, however, is absent in the area underneath the nipple-areola complex (NAC) (▶ Fig. 37.1).[5,6,7] During puberty, ovarian hormonal secretions determine breast development. Breast glandular structures (acini and galactophorous ducts) are under extreme hormonal influences (estradiol and progesterone) and it has been demonstrated that a harmonious growth and final glandular volume and shape are greatly influenced by the delicate balance of these two hormones.

Classically, a congenital fascial anomaly has been considered the underlying cause and two different theories have been proposed. First, the fibrous annular constriction band theory, proposed by Mandrekas et al, suggests that abnormal development of the fascia superficialis leads to restricted growth of the breast in one or more directions at the time of puberty.[6,7] The band is composed of dense fibrous tissue made of large concentrations of collagen and elastic fibers arranged longitudinally which do not allow the developing parenchyma to expand during puberty.[8,9,10,11] The resultant narrow glandular base favors preferential vertical growth toward the areola anteriorly where the fascial layer is naturally attenuated if not almost nonexistent, hence causing it to become oversized and protuberant and creating the tuberous shape and areolar widening.[12,13,14] Existence of this periareolar constriction band has been demonstrated histologically.[6,7] A second theory, less popular and proposed by Pacifico et al and Costagliola et al, suggests that the causative aberrancy is a weakening within the NAC dermis and fascia, and not a constriction band at the breast base, which leads to herniation of tissue through the NAC as the breast develops.[13,15] Despite the recurrent descriptions of a fascial origin, it has been acknowledged that these fascial theories are pure speculations and Klinger et al, in 2011, have demonstrated histological evidence of a disorder in collagen deposition involving all of the stromal components (derma, gland, adipose tissue, and fascia) in patients with tuberous breasts versus normal breasts.[16]

For one reason or another, a glandular growth restriction exists, mainly at the lower quadrants. As a consequence, the inframammary fold (IMF) ascends and the gland herniates through the areola, where the fascia superficialis is naturally nearly absent. Depending on the amount of gland, the growth restriction and the patient's areolar characteristics, the areolar size and protrusion will be more or less evident and a

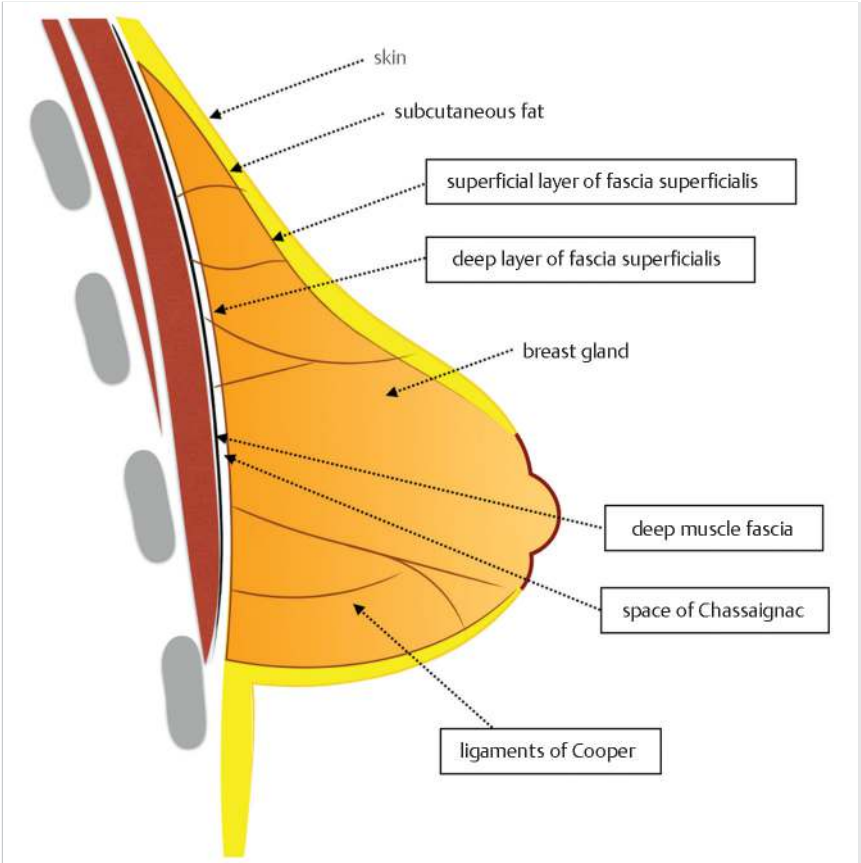


Fig. 37.1 Anatomy of the breast gland and its investing fasciae.

continuous spectrum of anomalies can be found, sharing all the them, in varying degree, the following key features:

1. Breast gland constriction (more severe at the lower quadrants).
2. Ascended inframammary fold (as a consequence of breast growth restriction).
3. Areolar enlargement and protrusion.

37.3 Classification

Rees and Aston found two types of the deformity but it was not until 1996 that Von Heimburg et al proposed the first formal classification, later revised by the first author in 2000.[8,17,18] Grolleau et al in 1999, Meara et al in 2000, Costagliola et al in 2013, and Kolker and Collins, in 2015, further refined Von Heimburg's work.[12,13,19,20] At present, the BIBC (tuberous breast) might be classified in four types, shown in ▶ Fig. 37.2.

- *Type 0*: **Isolated NAC herniation** without further significant anomaly of the breast shape.
- *Type I*: **Deficient lower medial quadrant** characteristically shaped like an italic S. The lateral breast is oversized in comparison. Overall, the breast is frequently ptotic with a normal or enlarged size. The IMF is ascended medially.
- *Type II*: With normal or enlarged upper quadrants, **lower inferior quadrants are deficient** horizontally and vertically. As a result, the IMF is ascended and the areola points downward. Overall, the breast size is usually small and ptotic.
- *Type III*: **All four quadrants are deficient**, resulting in a greatly restricted breast footprint and a resulting small breast. Constriction is both horizontal and vertical, giving the breast the shape of a tubercle.

Patients with severe BIBC or asymmetries have a major aesthetic concern and a related psychological impact that can be variable. On the other side, patients with minor degrees of the deformity however might not even be aware of the anomaly. Patients' expectations are always key in postoperative satisfaction and, consequently, the importance of preoperative counseling cannot be overemphasized. Different classifications have been proposed according to the severity of presentation but clinical practice shows that many patients do not fit a particular type of deformity. Because there is not one single clinical presentation, there is not one single surgical technique. In these patients, standard mammoplasty techniques (reduction, lift, augmentation) have proved to be ineffective. Rather they should be modified according to the patients' particular anatomy and added to the correction of the underlying tuberous findings. More than any other aesthetic procedure of the breast, breast asymmetries and tuberous deformities require a skillful surgeon than can customize the operation to the individual patient.

The usual case is a young woman with a breast asymmetry that shows a more or less tuberous anomaly in one or both breasts. Clinical examination should **not** focus on staging the anomaly because it does not necessarily have a prognostic or

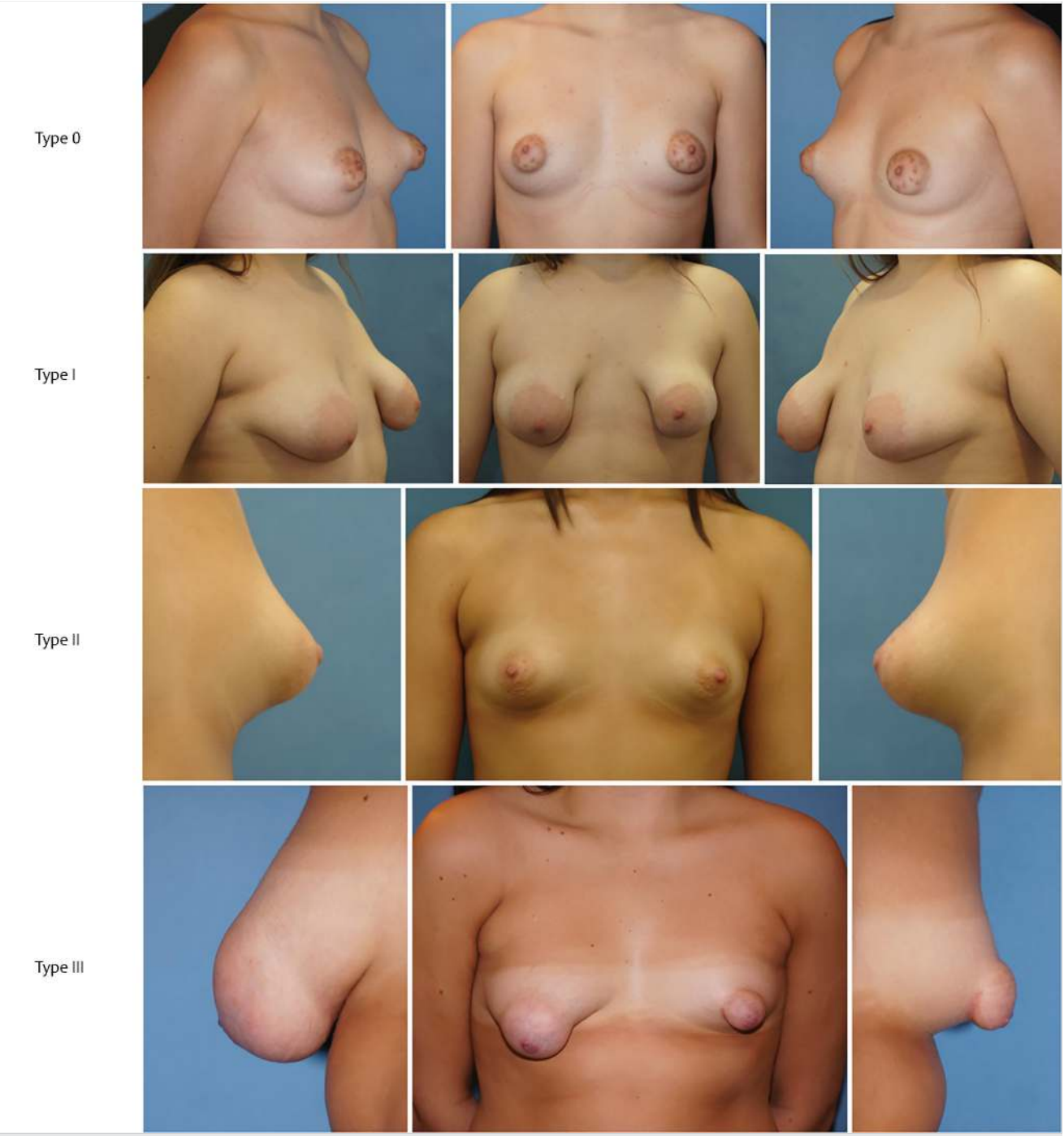


Fig. 37.2 Classification of the breast implantation breast constrictions (tuberous breast).

therapeutic implication. Rather, the following factors should be individually evaluated:

1. Areola:
   - Is the areola enlarged?
   - Is the areola protruding?
   - Should it be elevated?
2. Inframammary fold:
   - Is the inframammary fold elevated?
   - How much should it be lowered?
3. Gland distribution:
   - Which quadrants of the breast are constricted?
   - To what extent?
   - Does it need correction?
4. Breast volume:
   - Should the breast be reduced or lifted?
   - Does the subareolar breast require augmentation?

37.3.1 Preoperative Workup

Preoperative evaluation does not differ from other elective breast procedures. Because this group of patients is generally young and healthy (ASA I), preoperative and preanesthetic workout is quite expeditious. Bleeding diathesis not previously detected should always be ruled out in the medical record. In most patients, only usual preoperative blood tests (including coagulation) are all that is required. Six standard photographs should be taken pre and postoperatively and attached to the medical record (frontal with arms up and down, both oblique and both lateral views). Written informed consent should be taken before operation.

37.4 Treatment

37.4.1 Rationale a Surgical Strategy

Incision

Depending on the specific findings, the incisional approach can be circumareolar, customized circumvertical/inverted T, or inframammary at the new fold. Decision-making as regards to incisions is shown in ▶ Fig. 37.3.

37.4.2 Correction of the Inframammary Fold

Elevation of the inframammary fold (IMF) largely correlates with the underdevelopment of the inferior breast quadrants. Consequently, it will be more ascended in severe type IV cases where the lower breast quadrants are more deficient. How much should the fold be lowered is dependent on two factors, that is, the aesthetic proportions of the breast and the position of the contralateral fold. A balanced decision is thus required.

37.4.3 Correction of the Gland Maldistribution

Gland redistribution is the single most important aspect of a successful treatment in severe cases because it is the improvement of the breast shape, and not size, that really defines a good result. Surgical objectives are three: (1) release of the fascial constriction band, (2) tissue redistribution from excessive to deficient areas, and (3) attain a harmonious, aesthetically pleasant relationship among the four breast quadrants. Different options are available in ▶ Fig. 37.4:

Customized Dermoglandular Flap

Characteristically indicated in type I deformities, where breast tissue is not usually deficient, the customization of the reduction/mastopexy pattern allows the creation of dermoglandular flaps that can move tissue from excessive to deficient areas, usually from inferior-lateral to inferior-medial (▶ Fig. 37.5).

Glandular Scoring Incisions

Single or multiple scoring incisions of the gland, through an anterior or posterior approach, release the fascial thickening,

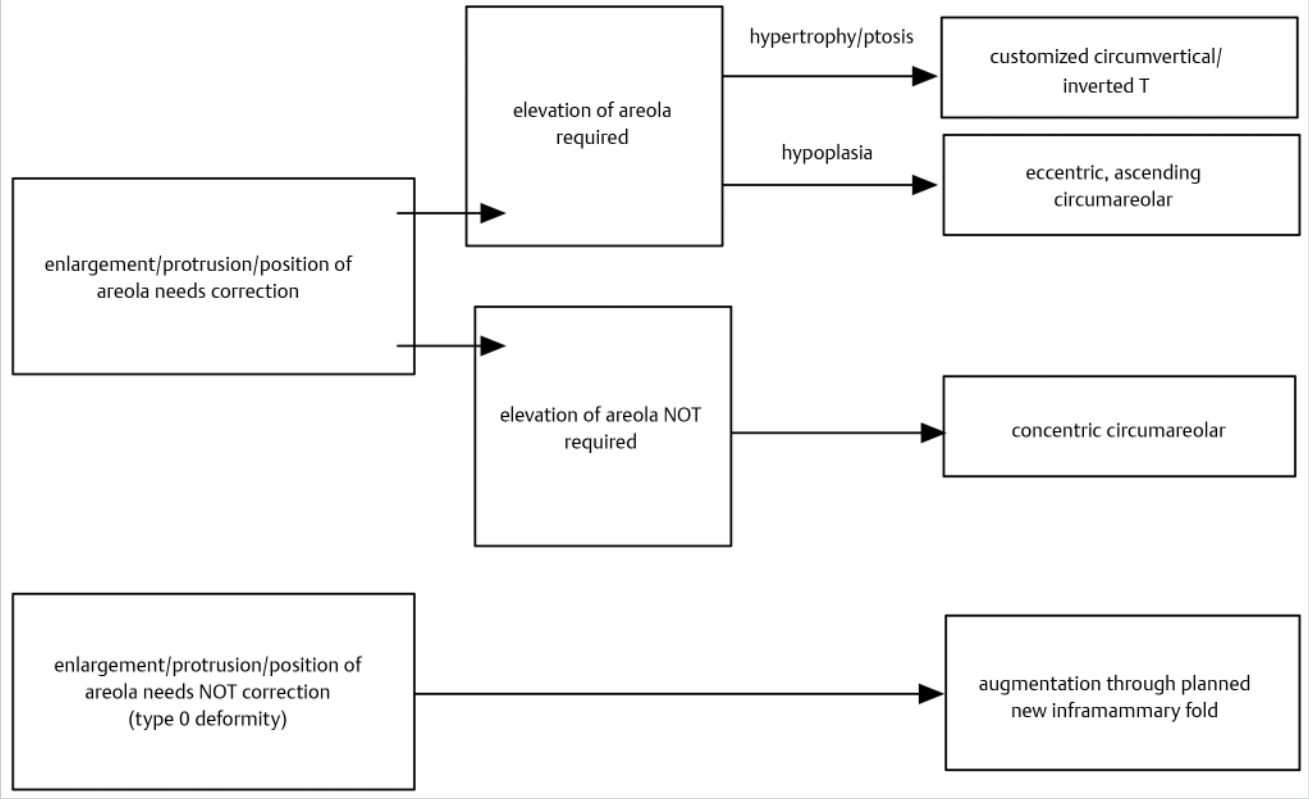


Fig. 37.3 Incisional approach based on the anatomy of the constriction.

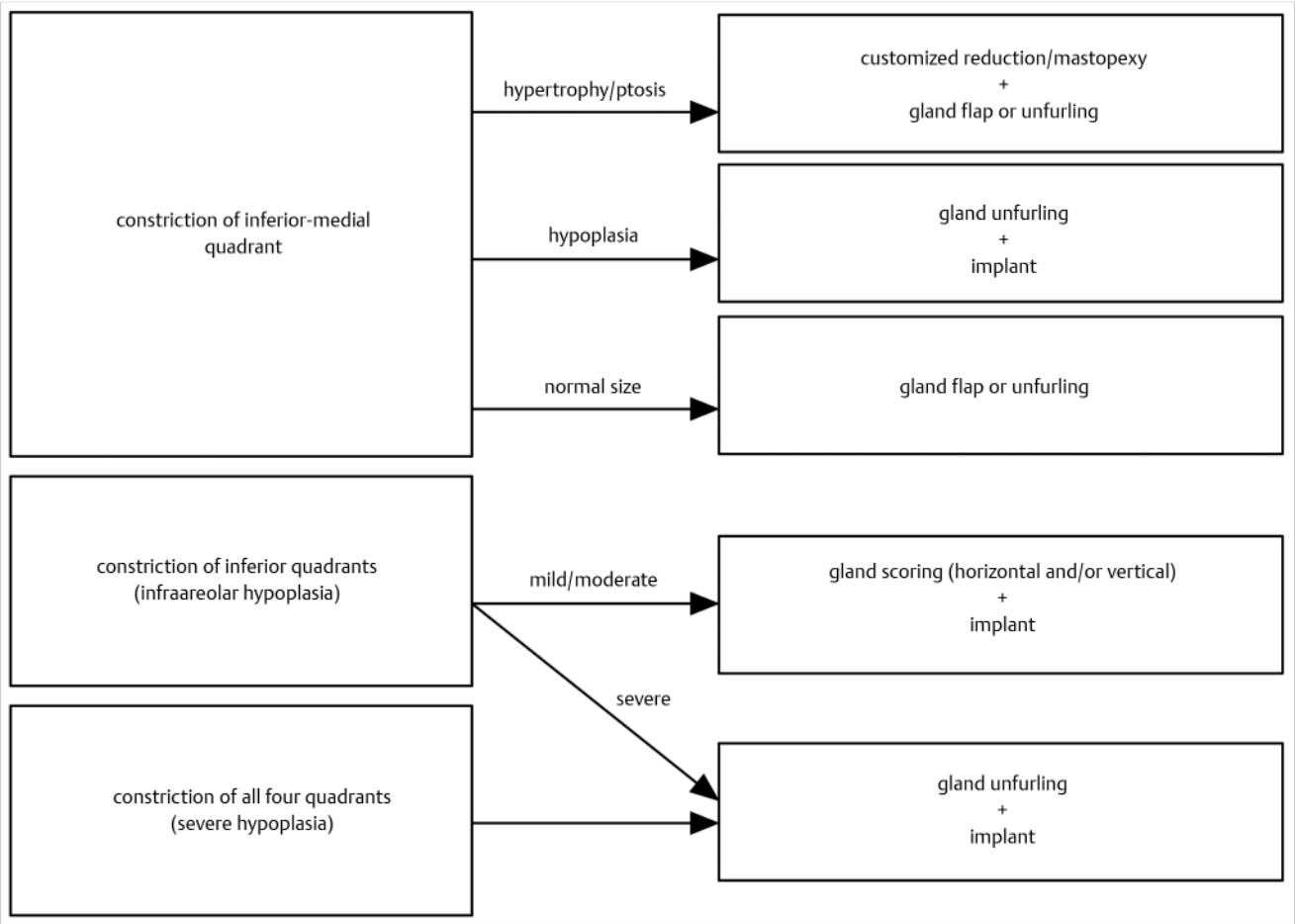


Fig. 37.4 Strategic options in the correction of the constricted tuberous breast.

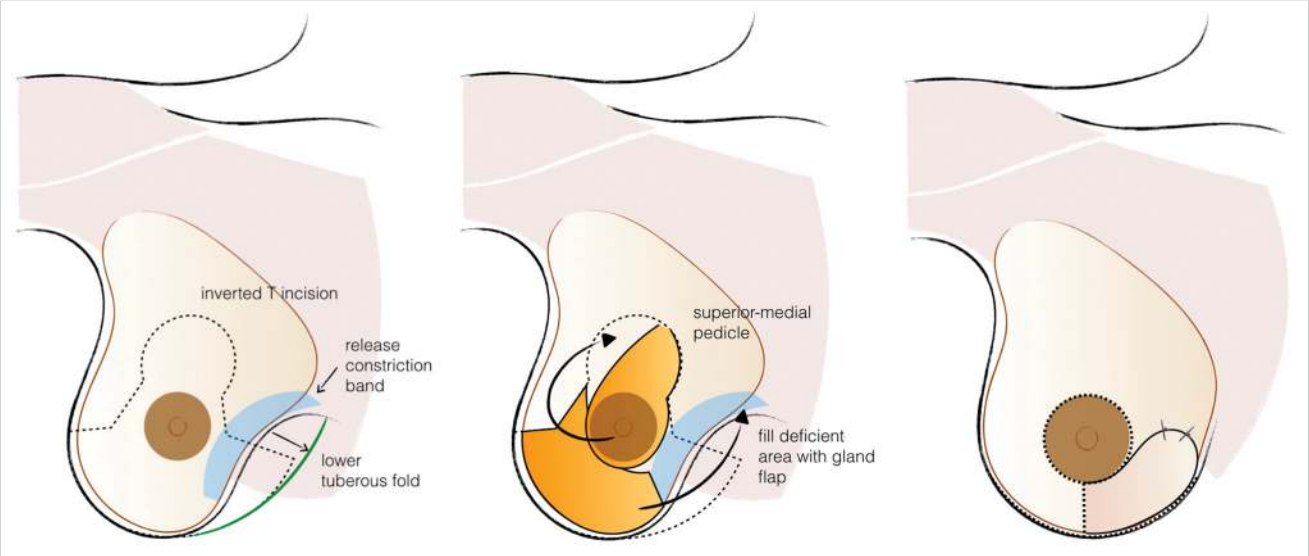


Fig. 37.5 A customized dermoglandular flap can fill the deficient area in the large breast.

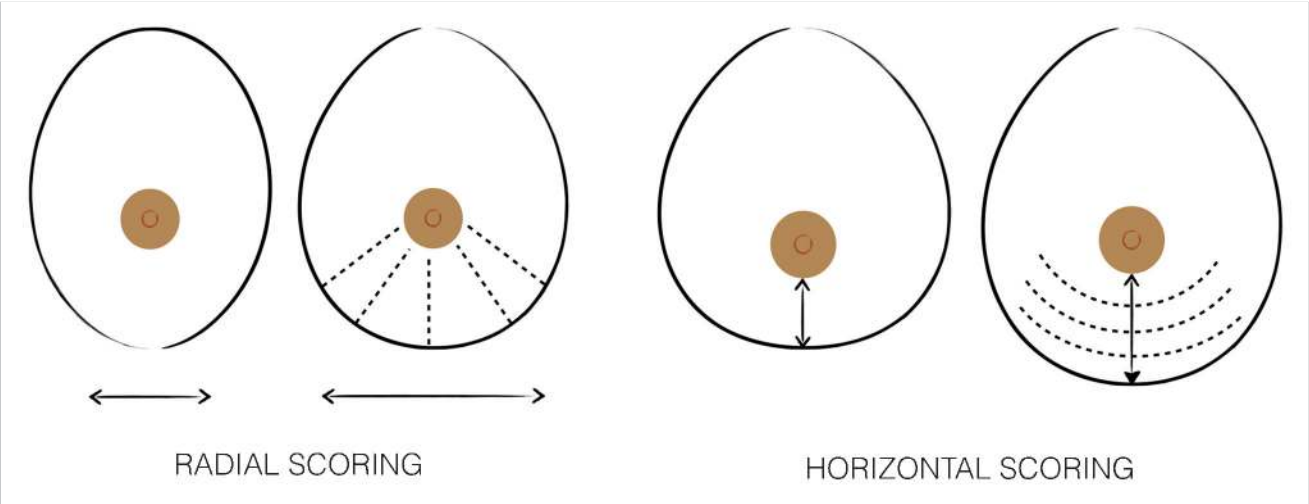


Fig. 37.6 Glandular scoring can expand the breast gland to increase the width and/or height of the infra-areolar breast.

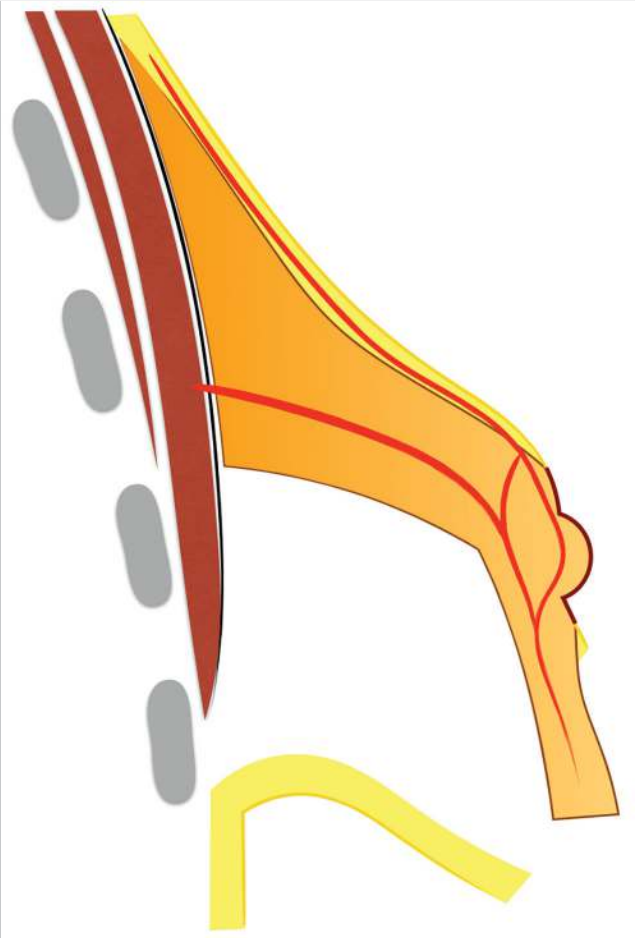


Fig. 37.7 Vascular supply of the unfurling flap.

expand the gland and increase the compliance of the lower pole.[6,10,11,17,19,21] Typically, the technique is indicated in mild to moderate constriction of the inferior quadrants (mild to moderate type III cases) with the addition of implant augmentation. Expansion occurs depending on how the scoring is done (▶ Fig. 37.6):

- *Radial scoring* allows a horizontal expansion of the constricted tissue and, consequently, increases the width and compliance of the inferior breast quadrants.
- *Horizontal scoring* produces a vertical expansion of the infra-areolar breast and, consequently, increases the vertical height and compliance of the lower breast quadrants.

Scoring releases lower pole constriction but has two major limitations in severe cases: (1) it does not significantly improve the areolar protrusion because no retroareolar tissue is (re)moved and (2) it can leave irregularities of the lower pole. Going anterior or posterior will largely depend on the incisional approach (i.e., posterior scoring with the inframammary approach and anterior incisions with the circumareolar approach). Aggressive scoring might endanger vascular supply and end up with irregularities and patchy subcutaneous necrosis that can be palpated under the skin.

Gland Unfurling

First proposed by Puckett and Concannon, gland unfurling is the best technique, if not the only, to correct areolar herniation. It is also very effective in gland redistribution because it moves tissue from excessive to deficient areas.[22,23,24] Consequently, it is the procedure of choice whenever areolar herniation and gland constriction/maldistribution are significant, that is, in moderate to severe hypotrophic tuberous breasts (severe type III and type IV cases). The procedure has distinct benefits: (1) correction of the areolar herniation, (2) minimization of the risk of postoperative double bubble deformity in the usual case of implant augmentation, and (3) enhancement of the soft tissue support of the lower breast.

The vascular supply of the unfurled flap comes from the nipple-areola complex (NAC) vascular network (▶ Fig. 37.7). The NAC has a rich superficial (subcutaneous) and deep vascular supply (via transpectoral perforators) arising from the internal mammary artery, the highest (superior) thoracic artery, the anterior and posterior branches of the intercostal arteries,

the thoracoacromial artery, the superficial thoracic artery, and the lateral thoracic artery.[25,26] Although no studies have been done regarding the vascular supply of the unfurled flap, clinical experience and basic knowledge on microvascular flap hemodynamics indicate that the vascularity of the flap is safe if properly executed: (1) the thickness at the flap base should not be less than 1 cm, (2) the retroglandular dissection should not go beyond the upper areolar margin to preserve any perforating vessels that might reach the NAC through the upper gland, and (3) the width-to-length ratio should be maintained within reasonable limits.

Areolar herniation is a major concern for many patients and relying on the areolar suture technique or tissue removal is not usually stable in the long term.[27,28] The unfurling technique offers long-lasting results because it moves tissue from where it is excessive (retroareolar) to where it is deficient (inferior quadrants).

37.4.4 Gland Unfurling: Surgical Technique

Preoperative Markings

The patient is marked preoperatively in the upright position. A vertical line is first drawn at the body midline followed by a midaxial line on both breasts. The existing fold is marked on both breasts, with a special attention to the usual fold asymmetries (which should be corrected). The planned new fold is then marked as previously published by the authors, albeit more conservatively to minimize the risk of necrosis of the infra-areolar skin.[29,30]A doughnut-shaped circumareolar marking is done for a final 4-cm areolar diameter. Supra-areolar skin can be deepithelialized if an areolar lift is required. Skin de-epithelialization should always be cautious when augmentation is planned (▶ Fig. 37.8).

Technique

Circumareolar de-epithelialization is first performed followed by an inferior hemiareolar incision. The dissection then proceeds inferiorly in the mastectomy plane (just above the superficial layer of fascia superficialis) with sharp dissection or low intensity electrocautery as far as the existing inframammary fold. Keeping the right plane of dissection is essential because, whereas going too superficial endangers the flap viability, going too deep does not adequately release the constriction band. Once the fold is reached, a fascial thickening can usually be found in the shape of a constriction band that needs to be dissected off the skin flap and left behind. Usually this band looks like an apron that hangs caudally from the breast gland. Next, the dissection continues further down, above the thickened superficial fascia, to the level of the planned new inframammary fold (▶ Fig. 37.9). The thickened fascia is then horizontally incised 1–2 cm below the constriction band. Next, the dissection moves upward, behind the constriction band and the breast gland, along the natural plane between the deep layer of the fascia superficialis and the deep prepectoral fascia, as far as the superior areolar margin. Going more cephalad does not

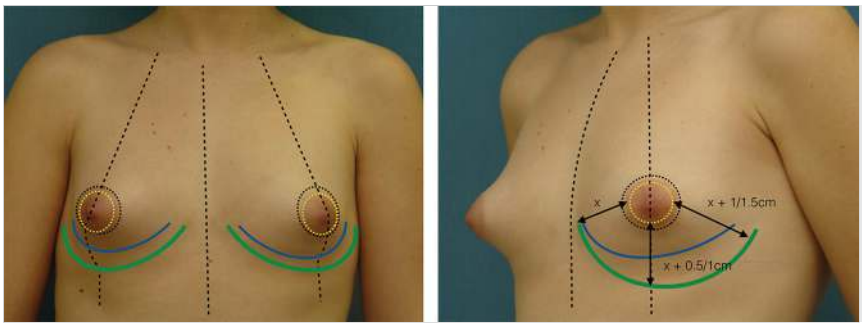


Fig. 37.8 Preoperative marking.

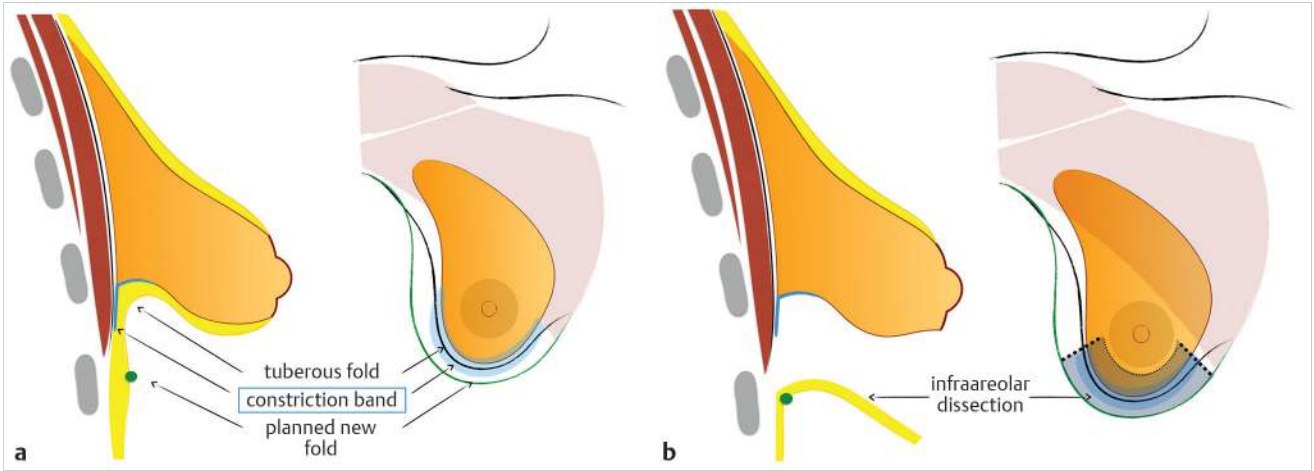


Fig. 37.9 (a,b) Lowering of inframammary fold. Surgical technique.

facilitate unfurling. More on the contrary, it might endanger the vascular supply of the unfurled tissue. This level of dissection is extended laterally and medially for a complete release of the thickened fascia (constriction band). The constriction band can now be fully palpated at the breast base, much like a basket handle, and transected laterally and medially, thus releasing the constriction and leaving a central glandular flap that is exteriorized through the circumareolar opening. The exteriorized glandular flap is cautiously incised in a posterior to anterior direction at the mid-areolar level to allow unfurling. Leaving a tissue thickness of at least 1 cm at the flap base is compulsory to minimize devascularization of the unfurled tissue (▶ Fig. 37.10). Once released, the unfurled flap is redistributed along the inferior breast pole. If the flap does not reach the new fold or shows a tendency toward upward retraction it can be fixed with loose transcutaneous temporary stiches, although this is rarely done in our current practice. If the flap is thick and stiff or too short it should be cautiously beveled to minimize the risk of postoperative double-bubble deformity. Eventually, in thick flaps, a second unfurling incision might be done although caution is advised to minimize the risk of vascular compromise. Again, this is less and less frequent in our current practice. Some glandular reduction of the distal flap tissue might occasionally be advisable in large breasts with large unfurled flaps. Any trimming of the unfurled flap should be done at its posterior aspect, more glandular and prone to leave irregularities postoperatively. As described, tissue unfurls in a cephalad to caudad direction and this is how it should be done when both the medial and the lateral inferior breast quadrants are equally hypoplastic. However, flap design should be modified when the constriction is largely limited to the inferior-medial aspect of the breast. In these cases, the unfurling flap should be designed to move from a superior-lateral to an inferior-medial direction (▶ Fig. 37.11). When an augmentation is planned in moderate-to-severe cases, it should be done retropectoral with high-cohesive anatomical implants. Retropectoral implantation guarantees the preservation of all the perforators coming through the pectoralis major muscle thus diminishing

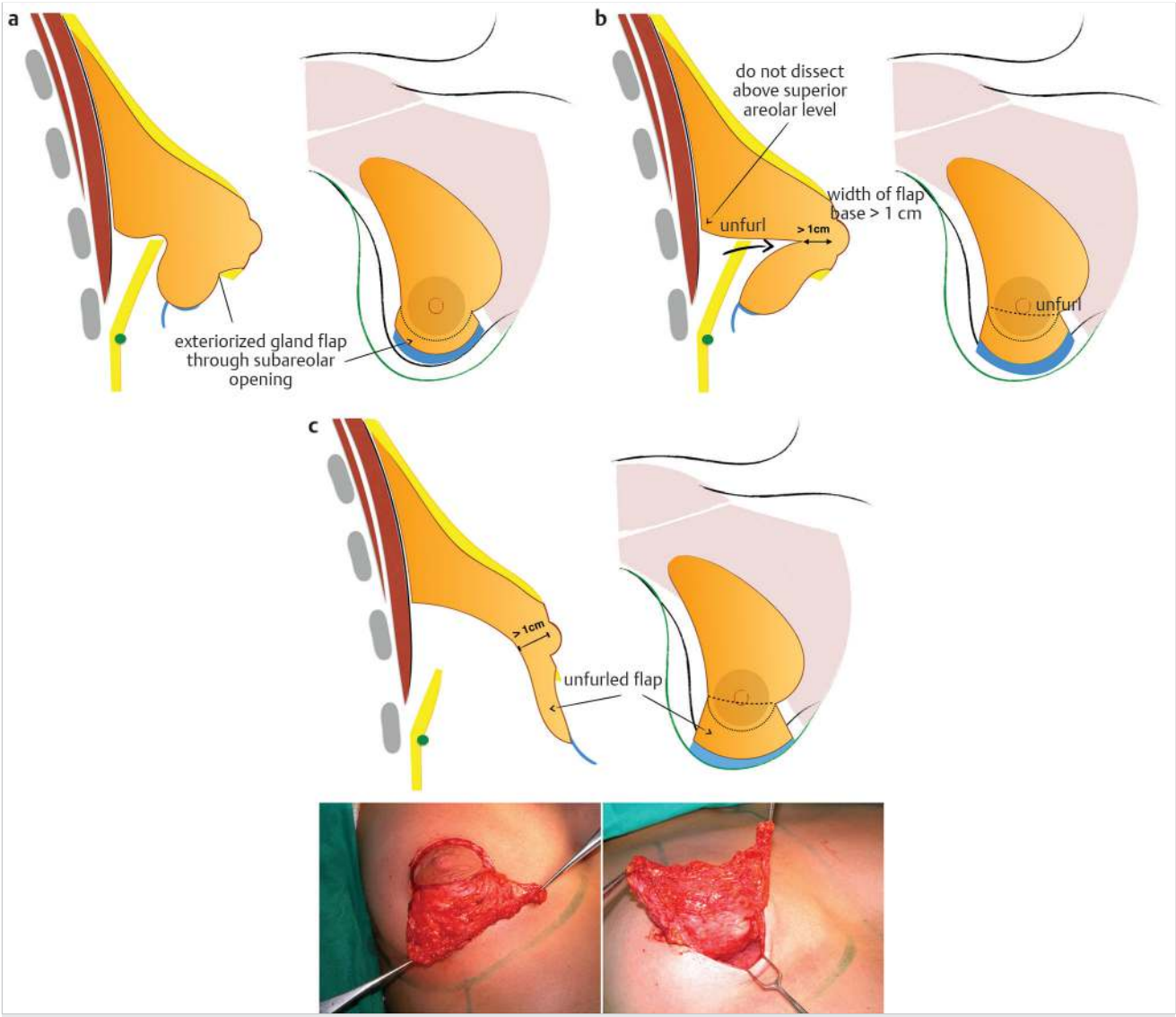


Fig. 37.10 (a–c) Gland unfurling to fill inferior deficit.

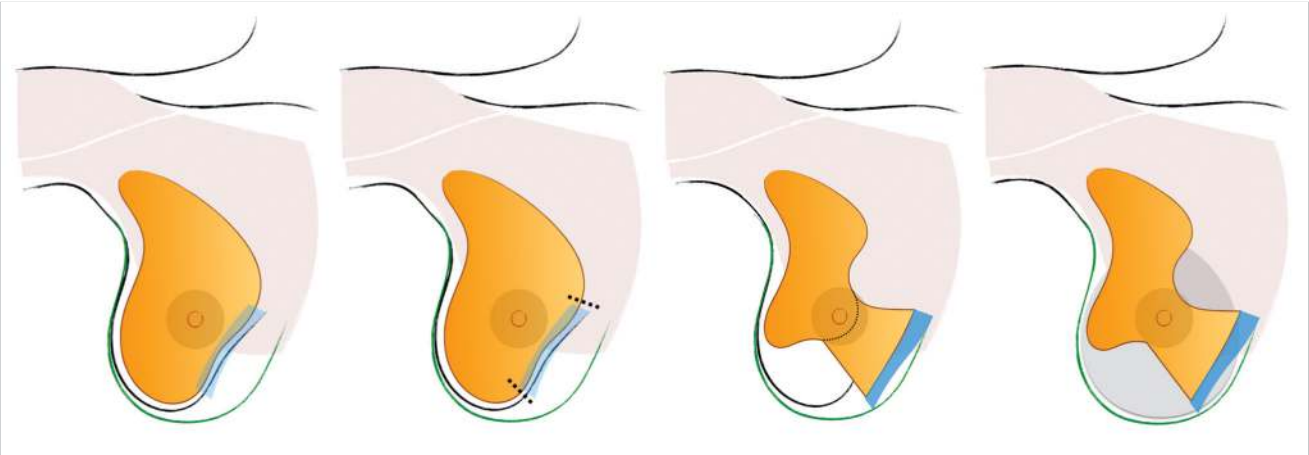


Fig. 37.11 Gland unfurling to fill inferior-medial deficit.

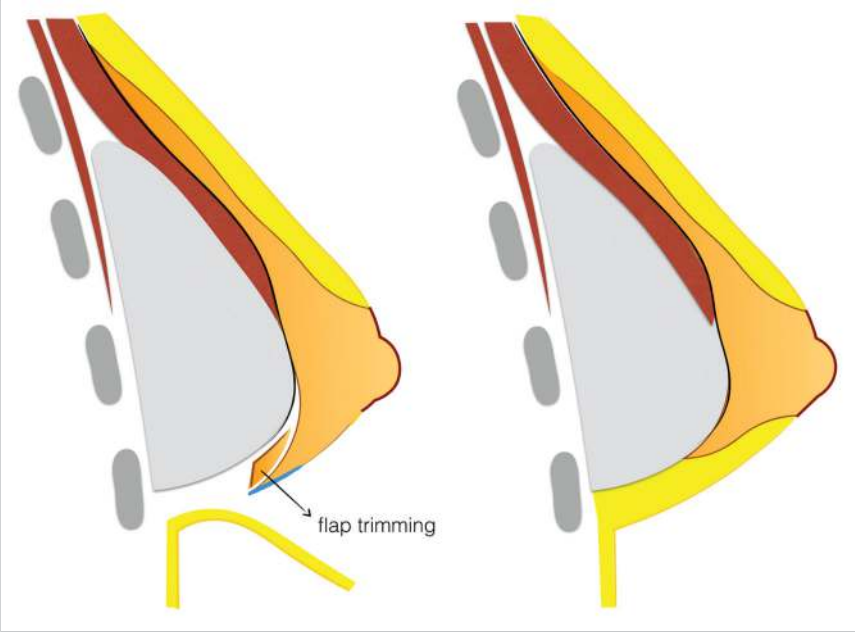


Fig. 37.12 Retropectoral anatomical implants minimize devascularization of the unfurling. flap and maximize projection of the deficient lower pole. Trimming of the flap. minimize in the flap-implant transition.

the risk of devascularization of the unfurled tissue. Anatomical implants are also required to adequately fill the empty lower breast pole, a landmark in severe hypoplastic tuberous breasts (▶ Fig. 37.12).

Implant selection is done according to the authors' previous publications, although with some distinctive features.[29,30] The infra-areolar skin dissection, which must be superficial to the fascia superficialis for an adequate release of the fascial restriction, leaves an infra-areolar random skin flap whose vascularization should be preserved with extreme care. Lowering the inframammary fold increases the flap's length-to-width ratio and jeopardizes viability. Consequently, the descent of the inframammary fold should be conservative and, as compensation, shorter implants than expected in the vertical height should frequently be used. High volume/projecting implants should likewise be discouraged to minimize the pressure over the temporarily devascularized inferior skin flap. Round-block purse-string closure of the areola is finally done over vacuum drains (in our hands, running purse-string closure with non-absorbable TiCron 4–0 suture has given excellent, long-term stable results).

37.4.5 Is There a Place for Tissue Expansion in the Treatment of the Tuberous Breast?

There is some debate in the literature about whether or not there is a skin envelope deficiency at the lower pole in the severe tuberous breasts.[6,31] In the authors' experience, it is the underlying fascial constriction, and not the skin, which is responsible for the tuberous anomaly. We have found that, even in severe cases, the skin of the inferior pole is normal although very tight and poorly compliant, similar to the extreme nulliparous young hypoplastic breast. Consequently, although supported by some publications, we have never needed tissue

expansion in the correction of the tuberous breast.[32,33,34,35] Moreover, breast expansion does not correct areolar herniation (in fact it can be even worsened) or gland maldistribution and can enlarge an already widened areola.

37.4.6 Fat Grafting of the Tuberous Breast

Some recent publications have advocated the use of fat grafting in the corrections of tuberous breasts.[36] We think that presently fat grafting cannot substitute other techniques in the correction of tuberous deformities but, rather, provide the perfect tool to further refine the results, especially in the correction of the double-bubble deformity and surface irregularities of the lower pole.[37] However, more clinical experience is needed to evaluate adequately the role of fat grafting in this group of patients.

37.5 Postoperative Care

As a general rule, patients in our practice are booked for an overnight hospital admission to allow optimized patient comfort and close medical observation. Alternatively, outpatient surgery is often done and safe. Vacuum drains are usually left in place one to several days until fluid collection is less than 25 to 40 mL per 24 hours or largely serous (nonhematic). Contrary to other authors, we think that postoperative suction drains should be used regularly in breast surgery for different reasons: (1) they collapse any dead space and avoid fluid collections, thus minimizing the risk of early postoperative implant rotation and (2) they help in the early detection of postoperative hematomas (quality of drainage). We have not observed an increased incidence of postoperative infection or contracture rate related to the use of drains and favor keeping them as long as reasonably required, especially when implants are used. The use of an appropriate supporting bra is begun the next morning and kept for the next 6 weeks. In those cases when an augmentation is associated with the unfurling technique, the use of bra or compressive bandages is avoided in the first 3 to 4 weeks to minimize the risk of jeopardizing the blood supply of the infra-areolar skin flap. When compliance of the lower pole is low, usually the case in severe tuberous breasts, a compression band of the upper breast might be used two hours per day for 2 to 3 months starting at third to fourth postoperative weeks. The use of prophylactic perioperative antibiotics is recommended until drain removal. Although devoid of scientific evidence, we support the use of oral postoperative antibiotherapy (second-generation cephalosporin) for 1 week when implant augmentation is done, with the rationale of minimizing the risk of capsular contracture. After hospital release, the patient is instructed to gradually reinitiate normal living with caution. Sport activities are restricted until the sixth postoperative week.

37.6 Outcomes

Complications in the correction of the tuberous breast should be rare and include those found in conventional augmentation and mastopexy/reduction procedures. However, the unfurling and scoring techniques have distinct possible complications that merit further explanation. Wound dehiscence and skin necrosis might occur at the lower pole in at-risk patients (heavy smokers, small vessel disease) or when the dissection of the skin flap is inadequate (too superficial). Treatment should generally be conservative until secondary healing, eventually requiring skin grafting. Should it occur, the underlying unfurled flap avoids implant exposure and makes implant extrusion extremely infrequent. Subcutaneous irregularities can be due to aggressive gland scoring or devascularization of the unfurled flap. When clinically relevant, delayed fat grafting might be advisable. Residual or recurrent areolar herniation/widening is rare with the unfurling technique but very frequent when the correction only relies on suture techniques or scoring maneuvers. The chance of a significant residual double bubble deformity (residual tuberous sign) is significantly lowered with the unfurling technique but minor degrees are not rare postoperatively. Again, delayed fat grafting can nicely improve the result (▶ Fig. 37.13).

Long-term stability is a major issue in aesthetic surgery of the breast and different factors will generally affect negatively in the long run: (1) major weight changes, (2) pregnancies and lactation, and (3) hormonal changes. Because breast asymmetries (including tuberous breasts) often require a different technique for each breast, long-term stability of the result might be fragile, especially when implant augmentation is required in one breast and a reduction or lift in the other.

Patient satisfaction outcome is important in the evaluation of any aesthetic procedure. Patients with tuberous breasts (breast implantation base constrictions) are usually satisfied with the results because even less than optimal procedures improve the baseline situation. However, only the selection of the appropriate technique in the individual patient can offer the best possible result and a highly satisfied patient (▶ Fig. 37.14).

37.7 Review Questions

37.7.1 True or False

1. Gland scoring is the best technique in the correction of the areolar herniation.
2. Elevation of the inframammary fold correlates with the underdevelopment of the inferior breast quadrants.
3. The association of the gland unfurling and high projecting implants is recommended in the severe hypoplastic tuberous breast to maximize the aesthetic result.

37.7.2 Choose the Best Answer

4. A 32-year-old woman seeks advice about her breasts (▶ Fig. 37.15). The best treatment option for her would be
   a) Breast augmentation through the periareolar approach.
   b) Circumareolar approach, gland unfurling and implant augmentation.
   c) Circumareolar approach, horizontal gland scoring and augmentation with anatomical implant.
   d) Two-stage expander-implant augmentation.
5. In the tuberous breast anomaly:
   a) It has been demonstrated that the tuberous deformity of the breast is due to an anomalous collagen deposition in the investing fascia.

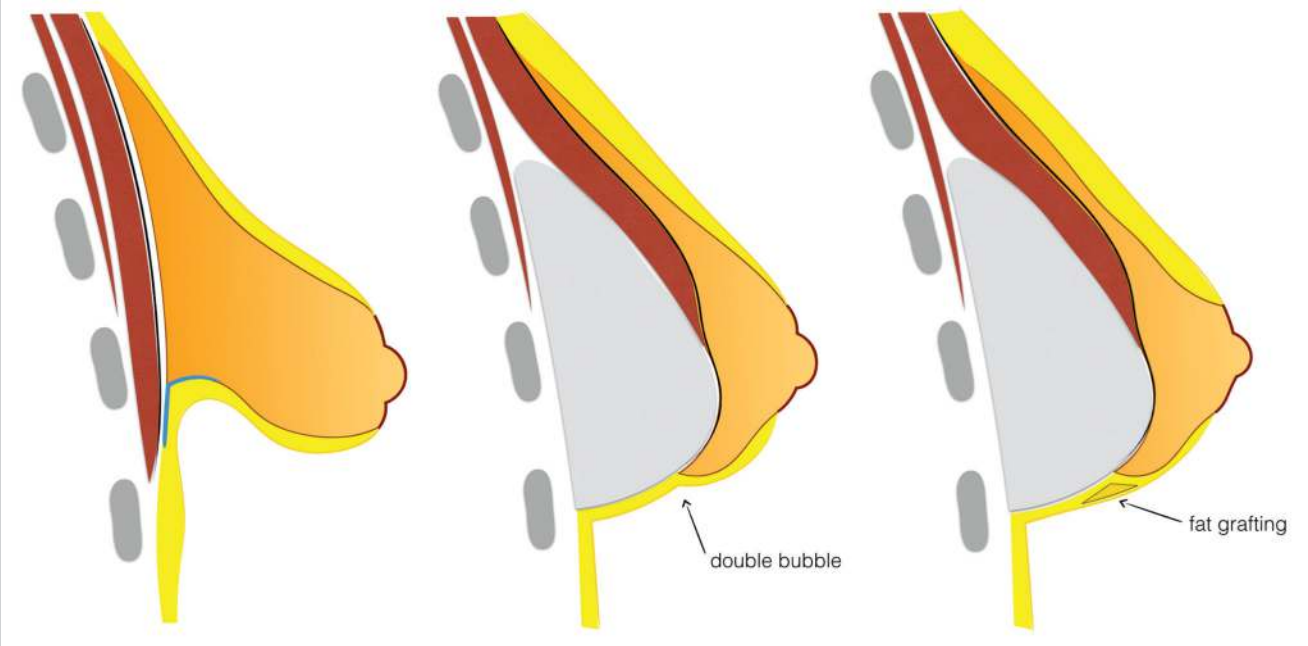


Fig. 37.13 Fat grafting are of great help in the correction of residual deficiencies and irregularities of the lower pole.

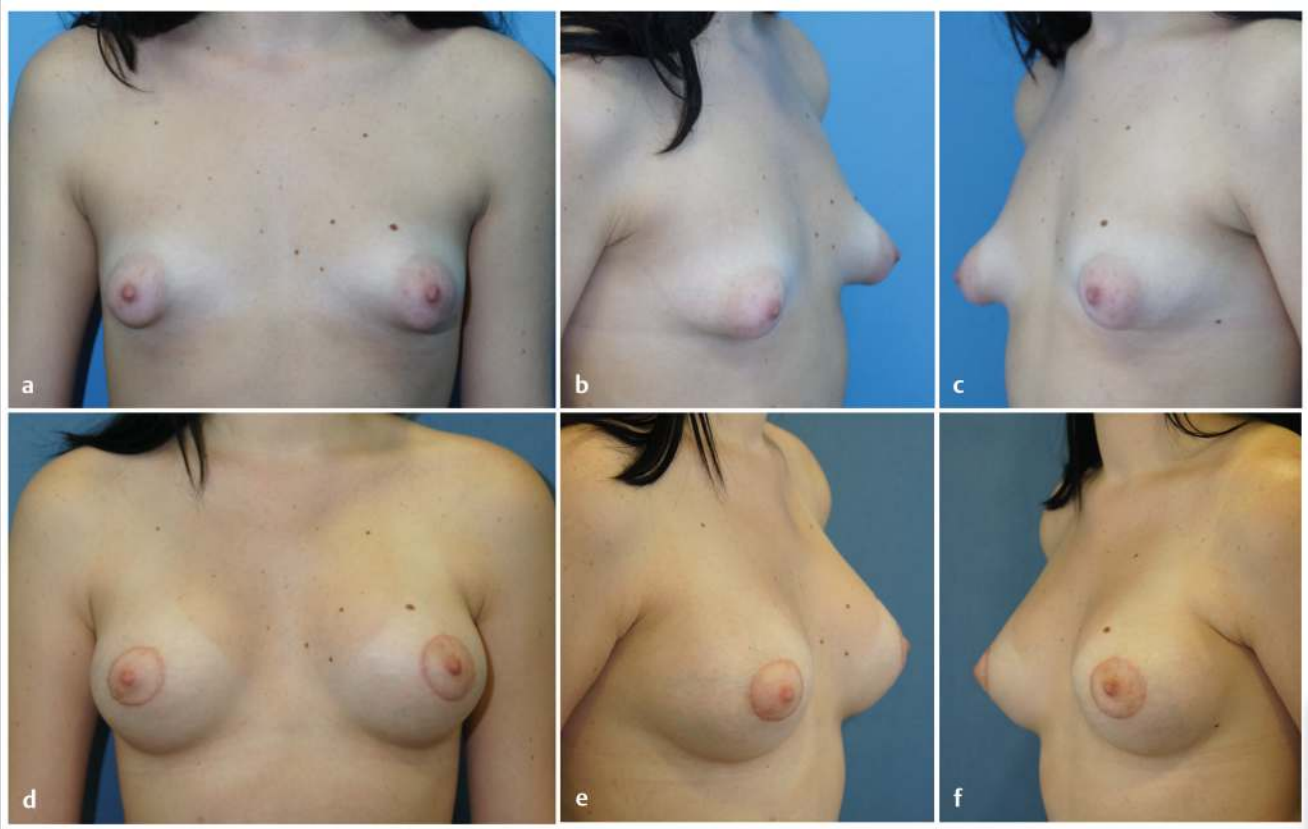


Fig. 37.14 Tuberous breast. (**a-c**) Pre- and (**d-f**) postoperative views.

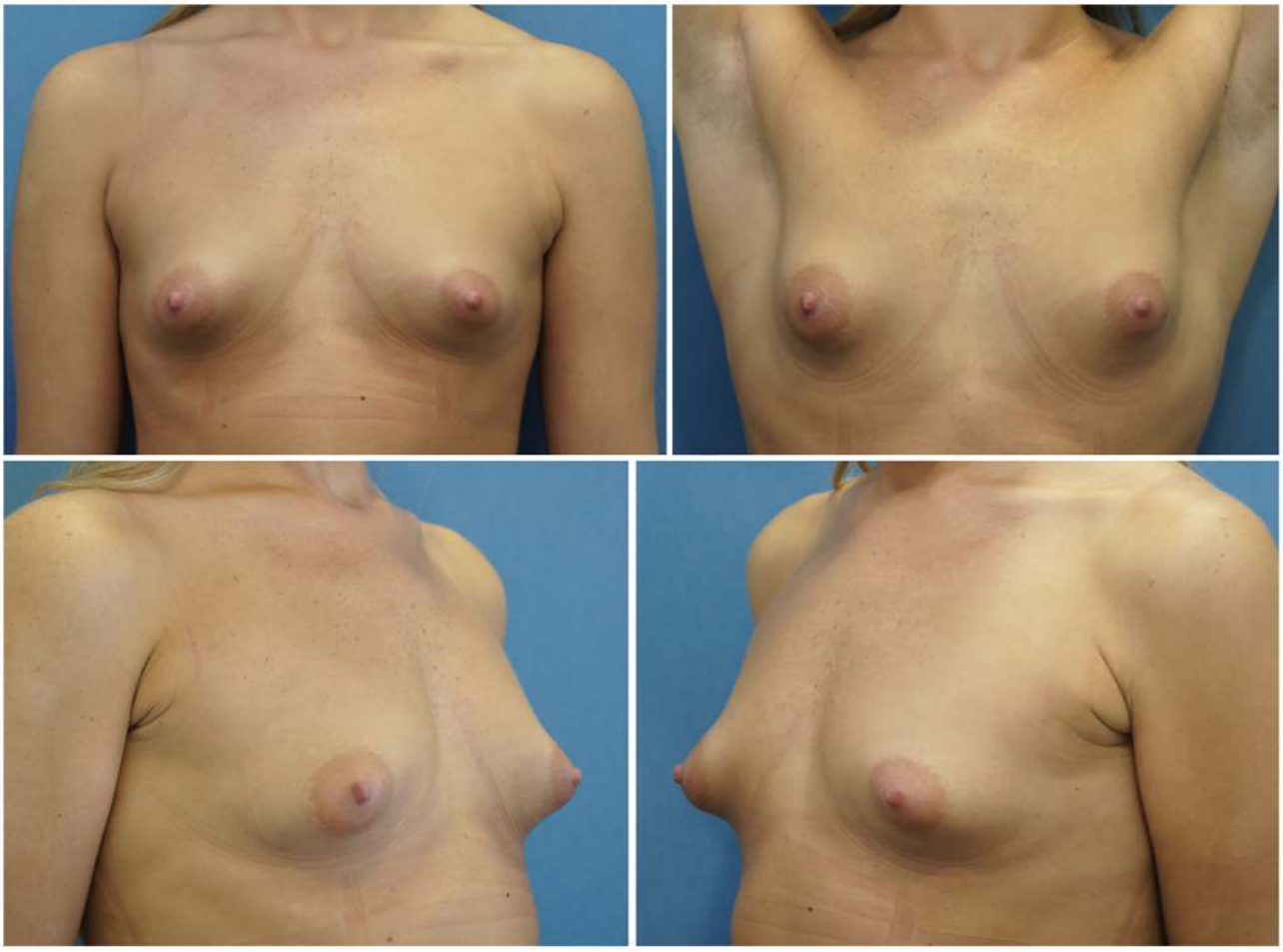


Fig. 37.15 A 32-year-old woman seeks advice about her breasts.

b) Horizontal scoring of the infra-areolar gland increases the compliance and vertical dimension of the lower breast quadrants.
c) Two-stage expansion/implant correction of the lower breast quadrants is the best treatment in the severe hypoplastic tuberous breast.

37.7.3 Answers

1. False.
2. True.
3. False.
4. b. Circumareolar approach, gland unfurling and implant augmentation.
5. b. Horizontal scoring of the infra-areolar gland increases the compliance and vertical dimension of the lower breast quadrants.

References

[1] Hamilton S, Gault D. The tuberous male breast. Br J Plast Surg. 56(3):295–300
[2] Latham K, Fernandez S, Iteld L, Panthaki Z, Armstrong MB, Thaller S. Pediatric breast deformity. J Craniofac Surg. 17(3):454–467
[3] Tanner JM. Growth at Adolescence. Blackwell Scientific Publications; 1969
[4] Osborne MP, Boolbol SK. Breast anatomy and development. In: Harris JR, Lippman ME, Osborne CK, et al., eds. Diseases of the Breast. Philadelphia, PA: Lippincott Williams & Wilkins; 2010
[5] Bostwick J III. Anatomy and physiology. In: Plastic breast surgery. 2nd ed. St. Louis, MO: Quality Medical Publishing; 2000
[6] Mandrekas AD, Zambacos GJ. Aesthetic reconstruction of the tuberous breast deformity. In: Shiffman MA, ed. Breast Augmentation: Principles and Practice. Berlin, Heidelberg: Springer Berlin Heidelberg; 2008
[7] Mandrekas AD, Zambacos GJ, Anastasopoulos A, Hapsas D, Lambrinaki N, Ioannidou-Mouzaka L. Aesthetic reconstruction of the tuberous breast deformity. Plast Reconstr Surg. 112(4):1099–1108, discussion 1109
[8] von Heimburg D, Exner K, Kruft S, Lemperle G. The tuberous breast deformity: classification and treatment. Br J Plast Surg. 49(6):339–345
[9] Zambacos GJ, Mandrekas AD. The incidence of tuberous breast deformity in asymmetric and symmetric mammaplasty patients. Plast Reconstr Surg. 118 (7):1667
[10] Ribeiro L, Canzi W, Buss A , Jr, Accorsi A , Jr. Tuberous breast: a new approach. Plast Reconstr Surg. 101(1):42–50, discussion 51–52
[11] Reynaud JP, Gary-Bobo A, Baron JL. [Tuberous breast: clinical and therapeutic considerations. Report of 20 cases]. Ann Chir Plast Esthet. 35(6):453–458
[12] Grolleau JL, Lanfrey E, Lavigne B, Chavoin JP, Costagliola M. Breast base anomalies: treatment strategy for tuberous breasts, minor deformities, and asymmetry. Plast Reconstr Surg. 104(7):2040–2048
[13] Costagliola M, Atiyeh B, Rampillon F. Tuberous breast: revised classification and a new hypothesis for its development. Aesthetic Plast Surg. 37(5):896–903
[14] Bass CB. Herniated areolar complex. Ann Plast Surg. 1(4):402–406

[15] Pacifico MD, See MS, Cavale N, et al. Preoperative planning for DIEP breast reconstruction: early experience of the use of computerised tomography angiography with VoNavix 3D software for perforator navigation. J Plast Reconstr Aesthet Surg. 62(11):1464–1469
[16] Klinger M, Caviggioli F, Klinger F, Villani F, Arra E, Di Tommaso L. Tuberous breast: Morphological study and overview of a borderline entity. Can J Plast Surg. 19(2):42–44
[17] Rees TD, Aston SJ. The tuberous breast. Clin Plast Surg. 3(2):339–347
[18] von Heimburg D. Refined version of the tuberous breast classification. Plast Reconstr Surg. 105(6):2269–2270
[19] Meara JG, Kolker A, Bartlett G, Theile R, Mutimer K, Holmes AD. Tuberous breast deformity: principles and practice. Ann Plast Surg. 45 (6):607–611
[20] Kolker AR, Collins MS. Tuberous breast deformity: classification and treatment strategy for improving consistency in aesthetic correction. Plast Reconstr Surg. 135(1):73–86
[21] Jones GE. Aesthetic problems and ancillary techniques. In: Bostwick's Plastic and Reconstructive Breast Surgery. St. Louis, MO: Quality Medical Publishing; 2010
[22] Puckett CL, Concannon MJ. Augmenting the narrow-based breast: the unfurling technique to prevent the double-bubble deformity. Aesthetic Plast Surg. 14(1):15–19
[23] Oroz-Torres J, Pelay-Ruata MJ, Escolán-Gonzalvo N, Jordán-Palomar E. Correction of tuberous breasts using the unfolded subareolar gland flap. Aesthetic Plast Surg. 38(4):692–703
[24] Serra-Renom JM, Muñoz-Olmo J, Serra-Mestre JM. Treatment of grade 3 tuberous breasts with Puckett's technique (modified) and fat grafting to correct the constricting ring. Aesthetic Plast Surg. 35(5):773–781
[25] van Deventer PV. The blood supply to the nipple-areola complex of the human mammary gland. Aesthetic Plast Surg. 28(6):393–398
[26] O'Dey DM, Prescher A, Pallua N. Vascular reliability of nipple-areola complex-bearing pedicles: an anatomical microdissection study. Plast Reconstr Surg. 119(4):1167–1177
[27] Atiyeh BS, Hashim HA, El-Douaihy Y, Kayle DI. Perinipple round-block technique for correction of tuberous/tubular breast deformity. Aesthetic Plast Surg. 22(4):284–288
[28] Teimourian B, Adham MN. Surgical correction of the tuberous breast. Ann Plast Surg. 10(3):190–193
[29] del Yerro JL, Vegas MR, Fernandez V, et al. Selecting the implant height in breast augmentation with anatomical prosthesis: the “number Y”. Plast Reconstr Surg. 131(6):1404–1412
[30] Martin del Yerro JL, Vegas MR, Sanz I, et al. Breast augmentation with anatomic implants: a method based on the breast implantation base. Aesthetic Plast Surg. 38(2):329–337
[31] Panettiere P, Del Gaudio GA, Marchetti L, Accorsi D, Del Gaudio A. The tuberous breast syndrome. Aesthetic Plast Surg. 24(6):445–449
[32] Versaci AD, Rozzelle AA. Treatment of tuberous breasts utilizing tissue expansion. Aesthetic Plast Surg. 15(4):307–312
[33] Toranto IR. Two-stage correction of tuberous breasts. Plast Reconstr Surg. 67 (5):642–646
[34] Kneafsey B, Crawford DS, Khoo CT, Saad MN. Correction of developmental breast abnormalities with a permanent expander/implant. Br J Plast Surg. 49 (5):302–306
[35] Foustanos A, Zavrides H. Surgical reconstruction of tuberous breasts. Aesthetic Plast Surg. 30(3):294–300
[36] Derder M, Whitaker IS, Boudana D, et al. The use of lipofilling to treat congenital hypoplastic breast anomalies: preliminary experiences. Ann Plast Surg. 73(4):371–377
[37] Serra-Renom JM, Muñoz-Olmo J, Serra-Mestre JM. Endoscopically assisted aesthetic augmentation of tuberous breasts and fat grafting to correct the double bubble. Aesthetic Plast Surg. 36(5):1114–1119

38 Poland Syndrome

Patrick J. Buchanan and Bruce A. Mast

Abstract
Poland Syndrome is a developmental defect characterized by varying degrees of unilateral pectoralis major aplasia, thoracic skeletal defects, and ipsilateral syndactyly. The etiology of the syndrome is unknown, and most surgical reconstruction is aimed at correcting the aesthetic appearance of the chest. The syndrome presents in two predominant variants: the simple or mild form and the complex or severe form. The surgical treatment for each of these manifestations is discussed by the authors. Treatment options covered include breast implants, latissimus dorsi muscle transfer, tissue expander placement, and endoscopic approaches. The review concludes with seasoned counsel about postoperative care and outcomes and complications.

Keywords: unilateral breast, latissimus dorsi muscle transfer, tissue expander placement

38.1 Goals and Objectives

- Understand the typical patient presentation of Poland syndrome.
- Recognize the different variants of Poland syndrome.
- Understand how to accurately diagnose Poland syndrome.
- Clearly define the indications for the various reconstructive options of the breast/chest wall in Poland syndrome.
- Know the evidence-based perioperative care to maximize patient safety and quality outcomes.

38.2 Patient Presentation

Poland syndrome is a developmental defect characterized by varying degrees of unilateral pectoralis major aplasia, thoracic skeletal defects, and ipsilateral syndactyly.[1,2] It was first written about in 1841, by Alfred Poland, who noted that one of his anatomical cadavers had a deficient sternal- and costal-head of the pectoralis major muscle, but a normal clavicular origin. He also noted, at that time, that there was an ipsilateral hand anomaly. Baudinne later coined this presentation of anatomical abnormalities as "Poland syndrome."[3]

The true incidence and etiology of Poland syndrome still remains unknown; however, it tends to occur more on the right side, 2:1 to 3:1, and is more common in males, 2:1. This syndrome presents with a spectrum of chest wall anomalies ranging from simple to complex. The vast majority of patients with Poland syndrome presenting for treatment do so on a cosmetic basis. The most common presentation is that of a patient with a unilateral absence of the sterno-costal head of the pectoralis major muscle (▶ Fig. 38.1). However, the deformity can be complex and encompass ipsilateral absence of ribs, axillary webbing, and foreshortening of the hemithorax including a derangement of the sternum with absence of the latissimus dorsi, serratus anterior, and/or external oblique muscles.[4]

Conventionally, a congenital unilateral absence of the sternocostal head of the pectoralis major muscle is pathognomonic for Poland syndrome. However, chest wall deformities, including breast maldevelopment, are the leading reasons of why patients seek medical treatment. The maldeveloped breast may be small, or absent, with a small nipple-areola complex that is displaced superiorly and laterally toward the axilla (▶ Fig. 38.1, ▶ Fig. 38.2).

The vast majority of surgical reconstructive cases are aimed at the correction of the aesthetic appearance of the patient's chest. The reconstructive options for patients presenting with chest wall deformities from Poland syndrome depend on several key factors, including anatomical severity, gender, patient's preference, and associated anomalies. Since reconstruction is rarely ever performed to restore a severe functional deficit, providing reassurance to the patient and their family is an important aspect of care that should not be overlooked by the surgeon. Nevertheless, functional reconstruction is possible to regain pectoralis muscle deficiency, a request more like to come from young males.[1]

Reconstructive options include the use of tissue expansion, implants, latissimus dorsi myocutaneous transfers, customized chest wall implants, and rarely, free tissue transfers.[1,5,6,7,8]

38.3 Poland Syndrome Variants

There are two predominant variations of Poland syndrome: (1) simple, or the mild form, and (2) complex, or the severe form. The simple, or mild, form is the most common and is characterized by the absence of the sternocostal head of the pectoralis major muscle. With this absence, the patient will have an effacement of the ipsilateral axillary fold; a cosmetic abnormality that is commonly mentioned by the patient (▶ Fig. 38.1,

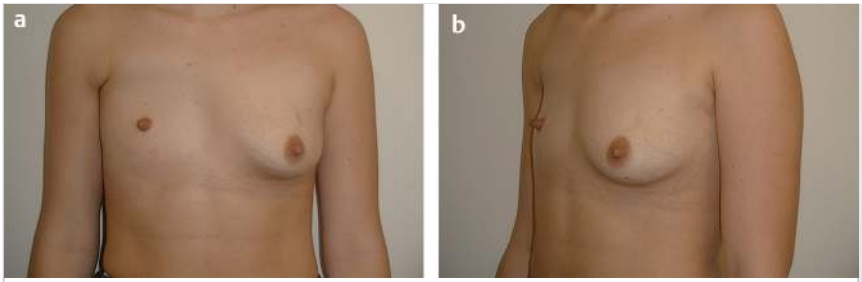


Fig. 38.1 (a,b) Woman with left-side postpartum mammary atrophy and right-side Poland syndrome characterized by absence of the sternocostal head of the pectoralis major and absence of breast development. The clavicular head of the pectoralis major is present as visualized by the infraclavicular soft-tissue fullness on the right. Note the stigmata of lactation and peripartum changes of the left breast whereas no such changes of the right chest skin have occurred.

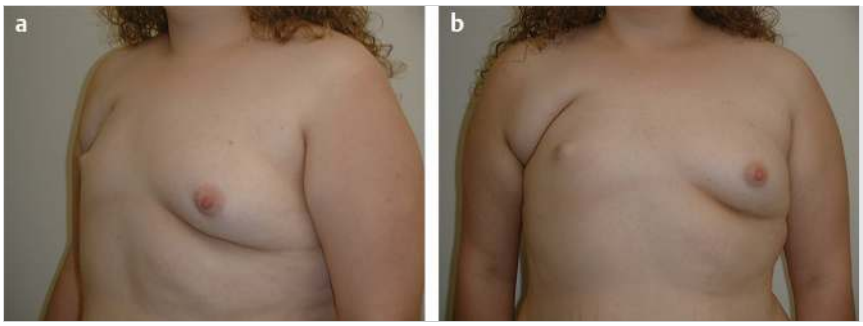


Fig. 38.2 (a,b) Young prepartum woman with right-side Poland syndrome. She has complete absence of the pectoralis major muscle and lack of breast development, as well as hypoplasia of the right nipple complex. Note the much more significant soft-tissue deficit of the chest wall, compared to the patient in ▶ Fig. 38.1.

▶ Fig. 38.2). The clavicular head of the pectoralis major muscle remains as a thin triangular muscle bundle that attaches the humerus to the inferomedial third of the clavicle (▶ Fig. 38.1). The ipsilateral breast is hypoplastic and the smaller nipple-areola complex is displaced laterally toward the axilla (▶ Fig. 38.2). As the pectoralis minor muscle cannot be tested, its presence cannot be confirmed by physical examination alone.

The complex, or severe, form of Poland syndrome is characterized by the absence of the sternocostal head of the pectoralis major muscle, ipsilateral rib and/or sternal hypoplasia or absence, and other ipsilateral muscular abnormalities involving the latissimus dorsi, serratus anterior, and/or external oblique muscles. In addition, the clavicular head of the pectoralis major muscle may be diminutive, the ipsilateral hemithorax may be notably hypoplastic, and axillary webbing may be present. If the serratus anterior muscle is absent, winged scapula is seen on physical examination. The insertion of the ipsilateral rectus abdominis muscle may be displaced cranially. This is caused by a foreshortened and bifid sternum and xiphoid process. Likewise, the ipsilateral scapula is small, rotated, and is cranially displaced when compared to the contralateral side. The anterior second through fifth ribs are thin, short, and missing their superolateral cartilage.

38.4 Diagnosis and Natural History

The surgeon must be acutely aware of the social impact of Poland syndrome on the patient and their parents. Those with the simple form may know of their abnormality earlier than their family, as it can remain unannounced until early adolescence, as muscle and breast development occur. The severe form, on the contrary, is readily known during infancy by the parents, as the chest wall is grossly asymmetrical.

To appropriately evaluate a patient for Poland syndrome, the surgeon must perform a full physical examination of the undressed patient so that their entire torso and upper extremities can be examined. In all patients, there will be a noticeable soft-tissue deficiency of the affected side of the chest due to volume loss from the partial or complete absence of the pectoralis. The full extent of the skeletal and muscular irregularities must be ascertained. Documenting the presence or absence of the serratus anterior and latissimus dorsi muscles is necessary, since this may affect desired reconstruction. The ribs, upper extremity, and torso are palpated and measured. Even in the simple form of Poland syndrome, the affected side will be notably smaller. The sternal notch-to-acromion, acromion-to-olecranon, and olecranon-to-ulnar styloid distances are measured and compared to the contralateral side, in cases of notable skeletal abnormality. In women, the status of the breast and position or presence of the nipple complex is assessed, particularly in regards to the contralateral, normal, side. In the occasional male who seeks muscular strength improvement due to absence of the pectoralis, objective testing of strength of the upper extremity via comparison to the contralateral side is needed.

38.5 Preparation for Surgery

In addition to the surgeon's physical examination, imaging and consultations may be beneficial and are performed selectively based on the physical findings, patient age, and overall condition.

Diagnostic imaging is available but usually not necessary in cases other than the most severe variants. Ultrasonography will show an absence of a portion of the pectoralis major muscle and asymmetry of the rib cage. Mammography is typically not used as an imaging modality for Poland syndrome; however, absence of the pectoralis major muscle on the mediolateral oblique view with a hypoplastic breast can be incidentally found. A CT scan can be used for presurgical planning to confirm the presence of the latissimus dorsi muscle. Likewise, an MRI may be useful to better define the patient's anatomy as it offers a multiplanar view without the ionizing radiation of a computed tomography scan. Both color-coded duplex sonography and contrast-enhanced magnetic resonance angiography allow for the evaluation of selected arteries and vessels.[9,10]

38.6 Treatment

38.6.1 Simple Form

In mild cases of Poland syndrome, the objective is symmetric cosmesis. In women, a breast implant can be used to satisfactorily treat the cosmetic concerns. However, be aware that the subclavicular hollowing can be accentuated with the insertion of an implant due to the tightness of the parasternal tissues causing the implant to be displaced into that hollowing. As such, tissue expansion may be required as a staged procedure and shaped gel implants may be visually superior to round implants, particularly implants with greater height than width. Ultimate results for device-based reconstruction are largely

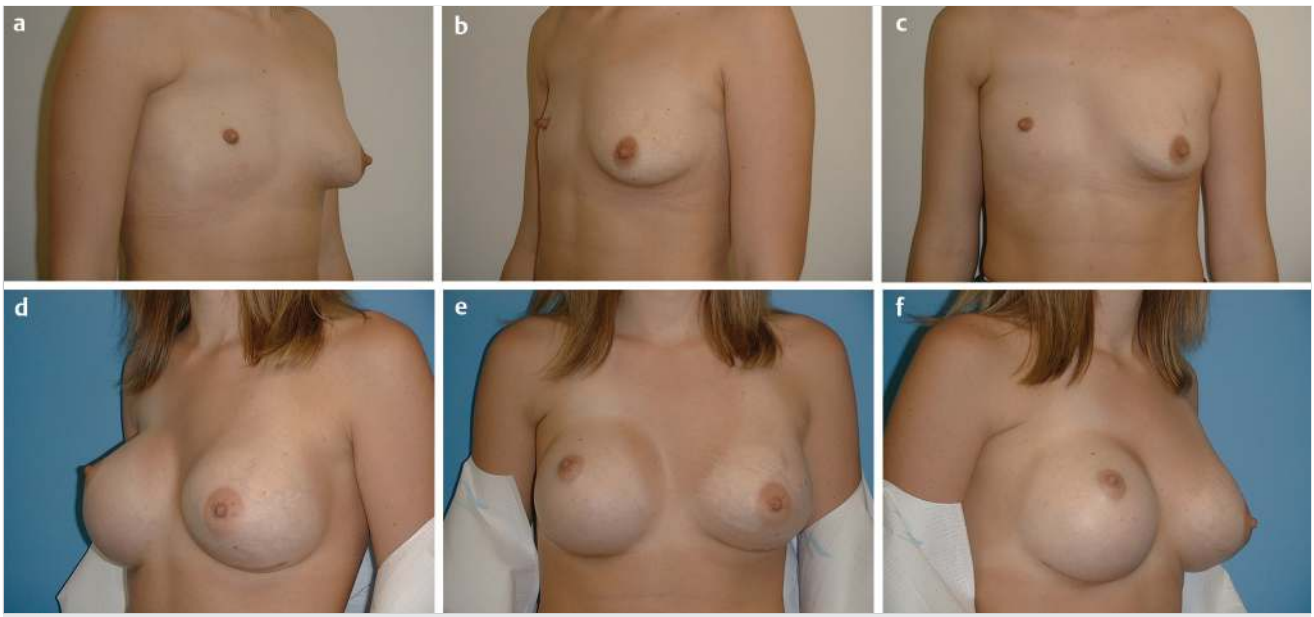


Fig. 38.3 (a-c) Same patient in ▶ Fig. 38.1 with three preoperative views. She has undergone tissue expansion on right followed by round gel implant placement and left augmentation for symmetry. **(d-f)** Although she achieved good shape and symmetry, while clothed, note the persistence of superior and superomedial contour depression due to paucity of soft tissue, as well as continued nipple and inframammary fold asymmetry.

dependent on the degree of breast development, and size of the opposite breast. Symmetry without clothing may not be achievable with device-only reconstruction, even with expansion (▶ Fig. 38.3).

In cases of complete or near complete absence of breast development, or in cases of an ipsilateral large-sized breast, flap reconstruction may be necessary due to soft-tissue demands for symmetry. Latissimus dorsi muscle transfer is the option of choice in prepartum women (▶ Fig. 38.4), while abdominally based, pedicled or free, flaps are generally reserved for women following child bearing. Free tissue transfer using nonabdominal tissue, such as gluteal or transverse thigh flaps, would fully avoid alterations in abdominal anatomy and could be safely used in prepartum women.

Customized chest wall implants may be adjunctively needed in women and are often the treatment of choice in men. The advantages of the customized chest wall implant reside in its ease of fabrication and insertion, and relatively low morbidity compared to flap reconstruction. Fabrication is relatively straightforward and usually done via plaster of Paris molding done in the office. The mold is then sent to the implant manufacturer, from which the customized implant is created. Similar to pectoral implants, the customized implants are created from a soft, solid form of silicone. They provide firm and stable soft-tissue augmentation in the desired shape and form; however, disadvantages include migration that leads to contour irregularities, patient reported discomfort, and potentially palpable or visible edges. The latissimus dorsi muscle transfer, in conjunction with a sub-latissimus breast implant, provides adequate volume to obviate the subclavicular hollowing and has a natural feel and appearance (▶ Fig. 38.4). Likewise, abdominal and other free flaps benefit from their natural appearance and feel, and their ability to obliterate the subclavicular hollowing.[3,6,7,11]

In the male patient, both custom chest wall prostheses and the latissimus dorsi muscle transfer are appropriate reconstruction options. As in the female counterpart, the custom chest wall implant also has the disadvantage of migration and discomfort. The latissimus dorsi muscle transfer allows for the replacement of the missing pectoralis major muscle and has been shown to restore a functional outcome.[1] Minimal re-education consistent of physical therapy is required of the patient to obtain function, as both the latissimus dorsi and pectoralis major muscles insert on the humerus causing adduction and medial rotation.

38.6.2 Latissimus Dorsi Muscle Transfer

Most often, a skin paddle is not required and, as such, a single incision can be used. Therefore, a dorsal incision above the lateral border of the latissimus dorsi muscle, approximately 6 to 10 cm long, should be used to gain access to the muscle. This straight-line incision should be placed within the brassiere line for females. The skin/soft tissue is elevated off the fascia overlying the latissimus muscle for exposure of all borders. The muscle is divided distally and the fascial and muscular attachments to the chest wall are divided, elevating the muscle toward the axilla. In most cases, the tendon to the humerus can remain intact. The thoracodorsal nerve should be transected for breast reconstruction.

If functional transfer is being done in a male, the humeral attachment needs to be relocated. A horizontal incision within the axillary crease is used for access. The latissimus dorsi tendon is visible and transected at its insertion on the humerus.

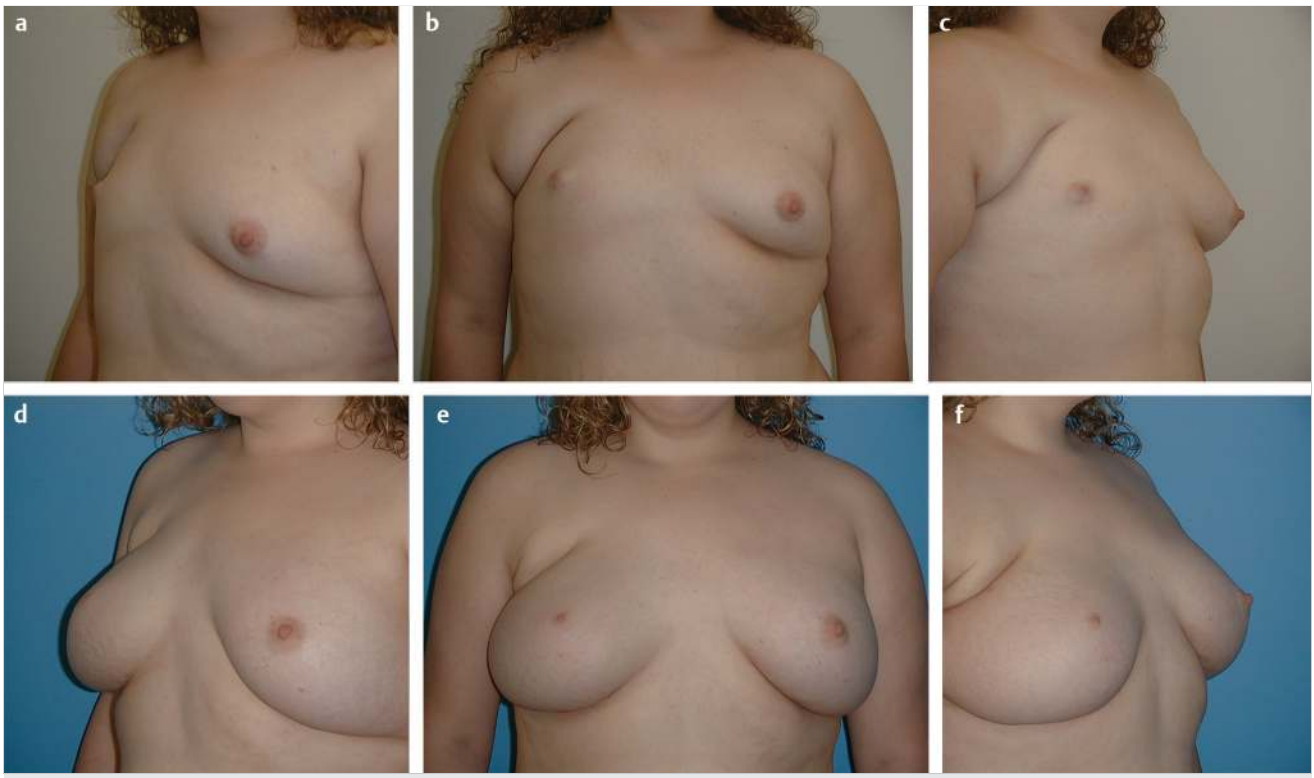


Fig. 38.4 (a-c) Prepartum patient from ▶ Fig. 38.2 with three preoperative views. Reconstruction has been done with latissimus dorsi flap with sublatissimus expansion, followed by bilateral implant placement. **(d-f)** Despite overall good symmetry, the profound preoperative upper lateral soft-tissue depression persists.

The latissimus dorsi tendon is then attached to the anterior bicipital sulcus of the humerus, mimicking the normal position of the pectoralis major muscle. Bone anchoring sutures are most effective (▶ Fig. 38.5, ▶ Fig. 38.6, ▶ Fig. 38.7, ▶ Fig. 38.8, ▶ Fig. 38.9).

In nonfunctional reconstruction, the ipsilateral chest skin is undermined using a periareolar approach. The mammary and previous axillary dissection planes are then joined. The latissimus dorsi muscle should then be transferred anteriorly and sutured to the lateral border of the sternum, the fascia along the inframammary fold, and the lower border of the remaining clavicular pectoralis major muscle.[12,13,14] In a functional transfer, a sternal incision may be desired so that the muscle can be firmly attached to the sternum, again using anchors, and biologic pledgets (▶ Fig. 38.6).

38.6.3 Tissue Expander Placement

A tissue expander based reconstruction is ideal for the female patient with a hypoplastic breast. Most of these patients have fibrotic breast tissue and will require radial scoring of the tissue prior to tissue expander placement to allow for adequate expansion. The scoring should be undertaken on the deep side of the glandular breast tissue perpendicular to the direction of desired expansion. The degree of muscle coverage of the expander is dependent on the extent of pectoralis absence. Partial superior coverage will be achievable in most patients. Subglandular or subcutaneous placement is necessary when the pectoralis major is notably hypoplastic or fully absent. If a latissimus dorsi muscle transfer is being utilized, the expander should be placed beneath the transferred muscle. Expansion is then done as necessary, followed by permanent implant placement, similar to breast reconstruction (see Chapter: 35) Contralateral balancing procedures may be needed: mastopexy, reduction, or augmentation.

A second option is to perform a vascularized flap from the abdomen, either as a free- or pedicled flap. The skin island of the abdominal tissue should be completely deepithelialized and sutured into proper position after removal of the expander. The advantages of this type of reconstruction include a more permanent symmetry and the use of autogenous tissue. Disadvantages include the morbidity incurred to the abdominal wall, including a scar across the abdomen, and a more extensive second surgery.

Another option for reconstruction after tissue expansion is to perform serial fat grafting. The expander can be deflated by one-third of its fully expanded volume and fat grafting can be performed within the subcutaneous plane, superficial to the tissue expander capsule. Fat grafting typically needs to be performed three to five times before the entire volume of the expander is replaced. The advantage of this form of reconstruction is its ability to be performed on an outpatient basis with excellent patient compliance.[12,13,14] Soft-tissue deficiencies can be corrected that occur with device-based reconstruction, but the morbidity of flap reconstruction is avoided.

Fig. 38.5 A 16-year-old man with left-side Poland syndrome complaining of left upper extremity weakness related to sports.

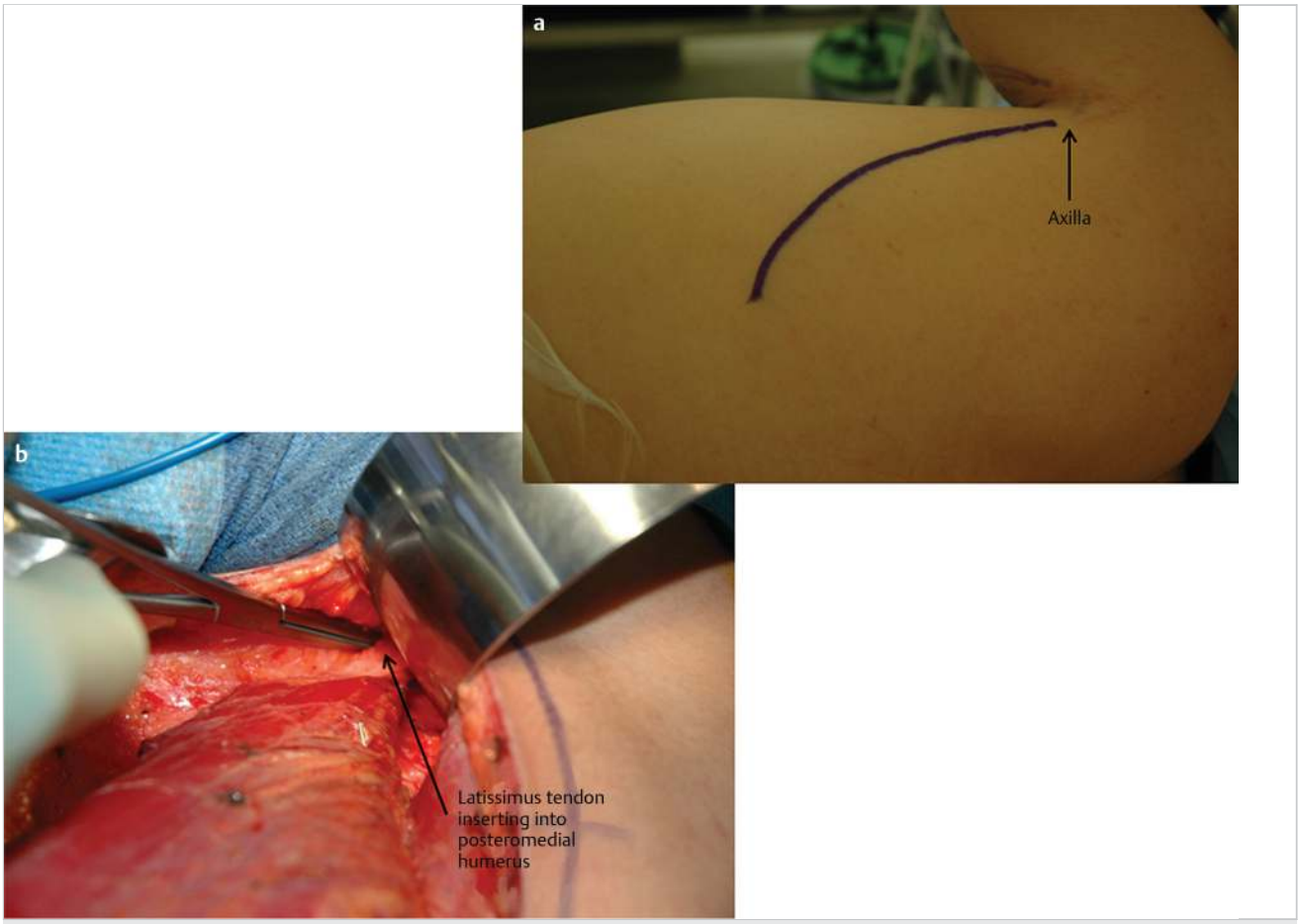


Fig. 38.6 **(a)** Use of oblique flank incision for latissimus harvest and full exposure. **(b)** Identification of the tendinous insertion of the latissimus onto the humerus as indicated by the end of the Kelly clamp.

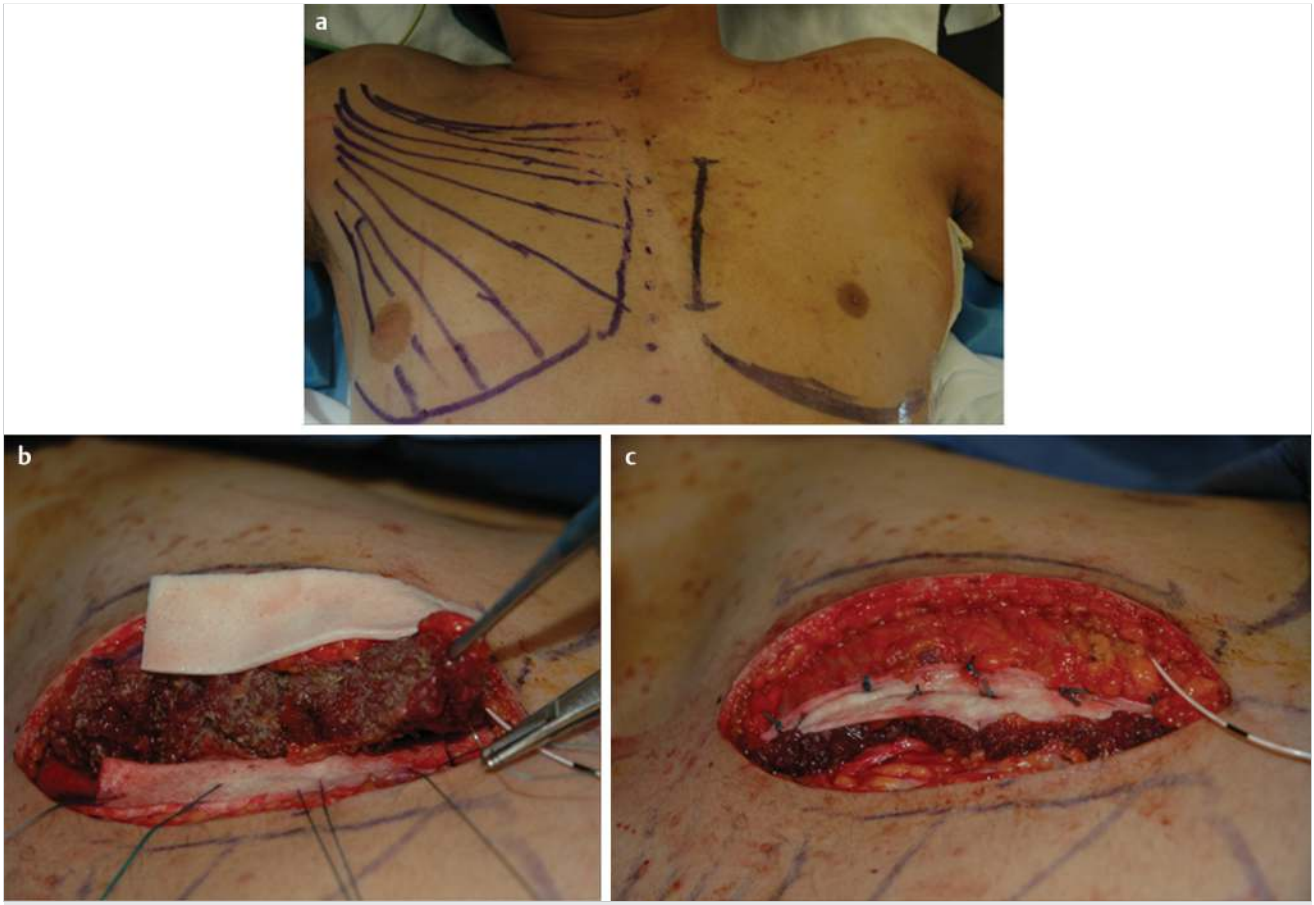


Fig. 38.7 **(a)** Outline of the right pectoralis muscle. **(b)** Through a presternal incision, the leading edge of the muscle is delivered, and sandwiched between acellular dermal matrix for stable inset into the sternum via sutured bone anchors. **(c)** Inset of the muscle onto the sternum.

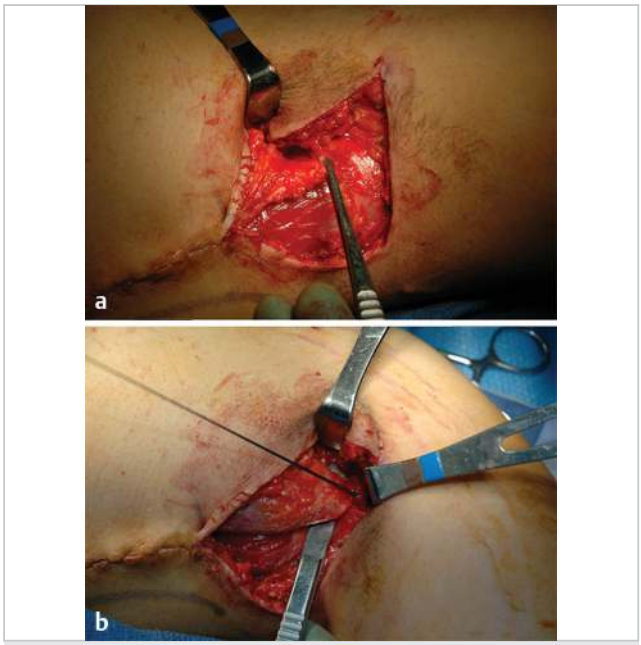


Fig. 38.8 **(a)** Axillary incision used to provide proper inset of the transferred muscle into the humerus. **(b)** Use of sutured bone anchor for creation of the new insertion of the latissimus dorsi tendon into the anteromedial proximal humerus.

38.6.4 Endoscopic Approach

Both tissue expander placement and the latissimus dorsi muscle harvest can be performed using an endoscopic approach. Caution should be taken and only those with previous experience should use this approach. This technique uses a single incision placed within the axilla, thereby negating the scar on the back for the traditional latissimus dorsi muscle harvest. The patient is positioned supine, and a 6-cm incision is placed within the axilla, along the midaxillary plane. The dissection is performed in the subcutaneous plane to expose the lateral border of the clavicular head of the pectoralis major muscle. Then, a 10-mL, 30-degree endoscope is attached to an endoretractor and placed within the dissected pocket to extend it medially to the sternum and inferiorly to the proposed inframammary fold. Once this pocket is developed, a tissue expander is then placed.[8,15]

Once the tissue expander is expanded to its desired volume, the second stage of the operation is performed in the lateral decubitus position. The same 6-cm incision is used for the harvest of the latissimus dorsi muscle. The thoracodorsal neurovascular pedicle can be directly identified and protected. Then, the 10-mL, 30-degree endoscope is inserted to complete the superficial and deep dissection of the latissimus dorsi muscle as previously described. The patient is then repositioned supine and

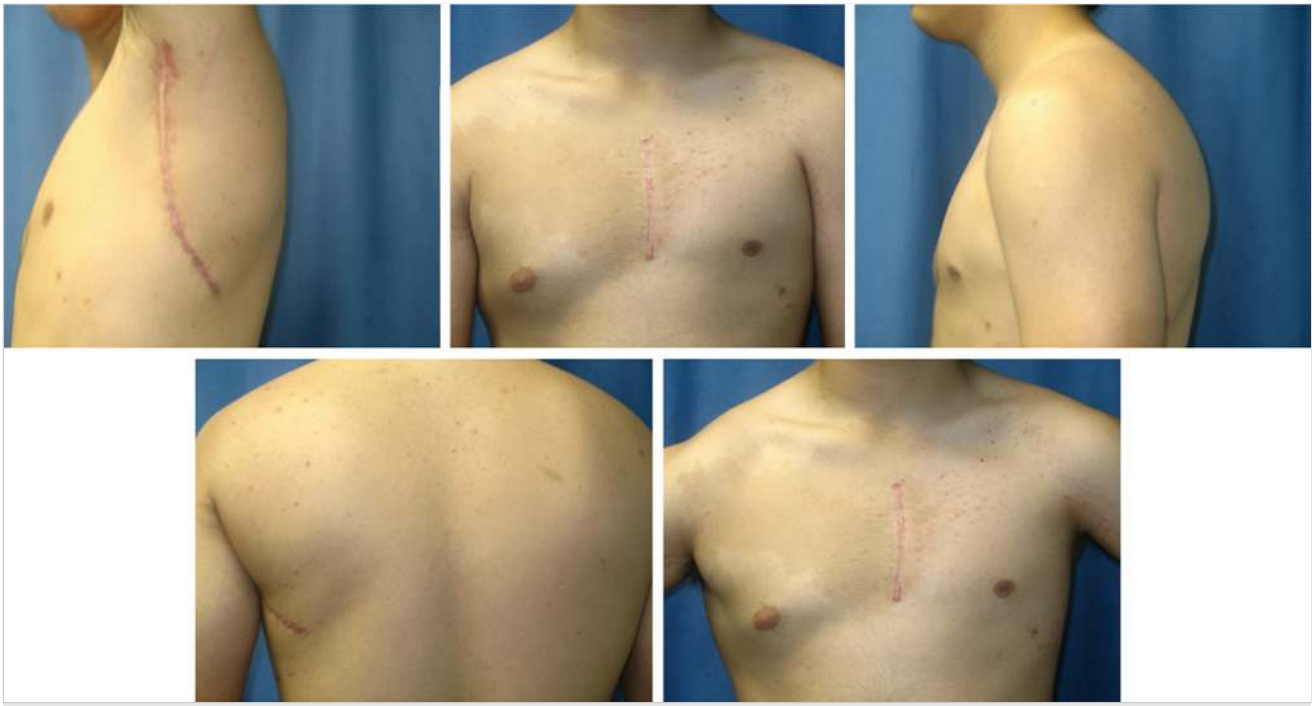


Fig. 38.9 Nine months after functional transfer of the latissimus dorsi. A pectoral shape is visible on the chest wall.

the anterior dissection is performed to regain access to the tissue expander. The tissue expander is removed and the latissimus dorsi muscle is transferred into the anterior pocket and sutured into place. A breast implant can then be positioned deep to the latissimus dorsi muscle.[8]

38.6.5 Complex Form

There are rare cases where proceeding with repair at an early age is necessary, especially in the complex form of Poland syndrome. However, given that the most common indication for surgical intervention is cosmetic in nature, the general preference is to delay reconstruction until late adolescence or adulthood.[11]

Due to the more extensive and visual anatomical abnormality, a latissimus dorsi muscle transfer with concomitant breast implant is the preferred reconstructive method and provides the best aesthetic outcome in the female patient.[3,11,16] At a second procedure, under local or mild sedation, the nipple-areola complex can be repositioned. For the male patient, a latissimus dorsi muscle transfer alone provides an excellent aesthetic result and has the ability to restore a functional outcome as stated previously.[1]

38.7 Postoperative Care

Following Poland syndrome reconstruction, standard incisional postoperative care is required. In male patients with a functional latissimus dorsi muscle transfer, the authors use a graduated load-bearing regimen with the help from physical therapists. Most of these patients are cleared for isotonic exercises 1 month postoperatively and activate the latissimus dorsi muscle with posterior extension of the upper extremity. This activation is slowly progressed over the ensuing 6 weeks and to full weight-training/bearing by 10 weeks postoperatively.[1]

For patients who require an implant-based reconstruction, postoperative protocols apply. At the author's institution, postoperative oral antibiotics are given until the patient's drains are removed. At that time, implant "massaging" commences. This massaging is thought to help reduce the risk of capsular contracture. Patients are also encouraged to walk for at least 10 minutes, every 2 hours, during the day and to do arm/shoulder range of motion exercises four times daily. Patients with an implant-based reconstruction are also encouraged to avoid physical work or lifting anything greater than five pounds for 4 weeks postsurgery. After this, the activity restrictions are liberalized.

Closed-suction drains, when placed, are not removed until they are outputting less than 30 cc of fluid for two consecutive days, individually. Patients are asked to strip and record each drain's volume twice a day. They are cleared to shower 2 days postoperatively; however, a piece of clear, plastic wrapping should be placed over the drain insertion site.

38.8 Outcomes

The general outcome goals for reconstruction of the female patient is to achieve breast symmetry, re-create the anterior axillary fold, and provide adequate infraclavicular fullness. These goals are successfully achieved with any of the aforementioned reconstructive options. However, to optimize breast symmetry, female patients most often request a contralateral mastopexy and/or reduction.

Seyfer et al performed 57 operations on 29 women and incurred no revision operations when they performed the

latissimus dorsi transfer in conjunction with the breast implant.[3] The majority of the complications seen were due to the breast implant, itself. Capsular contractures were revised using open capsulotomies. In four of their female patients, they used the custom-made chest wall prosthesis, alone. All of these subsequently were explanted due to malposition, contour irregularities, and overall patient dissatisfaction.[3]

The general goal for reconstruction of the male patient is to achieve symmetric chest wall contour. As in their female counterpart, the latissimus dorsi transfer, overall, had better outcomes. Most revisions were seen in those patients undergoing reconstruction with the customized chest wall prosthesis. These revisions were for malposition, discomfort, contour irregularities, and overall dissatisfaction.

Like any surgical intervention, Poland syndrome reconstruction can have adverse complications including, but not limited to, seroma, hematoma, infection, scar, delayed wound healing, and pain/paresthesias. Seroma formation was most often seen after the removal of the closed-suction drains in the latissimus dorsi transfer donor site. However, as in the study of Seyfer et al, these seromas are typically small and treated conservatively until they resolve on their own.[3]

The reconstructive results for Poland syndrome tend to be long lasting and with high patient satisfaction. However, as with all cases involving implants, long-term complications can occur and are manifested predominantly by capsular contracture and implant rupture.

38.9 Review Questions

38.9.1 True or False

1. There is a male and right-side predominance in Poland syndrome.
2. There is a normal clavicular head, but a deficient sternocostal head of the pectoralis major muscle in patients with Poland syndrome.
3. Overall, patients are satisfied with their reconstruction using a custom chest wall prosthesis.

38.9.2 Choose the Best Answer

4. A 33-year-old female presents with a unilateral breast and chest wall deformity. During her adolescence, she only experience breast growth on her left side, with contralateral breast, chest, and nipple deformities. Her right pectoralis major muscle is absent. Her BMI is 28. What is the best reconstructive procedure for this patient?
   a) Tissue expansion alone.
   b) Custom chest wall prosthesis.
   c) Latissimus dorsi muscle transfer with breast implant.
   d) Serial fat grafting.
5. A 16-year-old male presents with left-side chest weakness and an asymmetric chest since birth. He was recently diagnosed with Poland syndrome and found to have an absence of the pectoralis major muscle on the left. He states that he would like to play football in high school, but is unable to meet the minimum weightlifting requirements due to the left-side chest weakness. What is the best reconstructive option for this patient?
   a) Custom chest wall prosthesis.
   b) Serial fat grafting.
   c) Latissimus dorsi muscle transfer.
   d) TRAM flap.

38.9.3 Answers

1. True.
2. True.
3. False.
4. c. Latissimus dorsi muscle transfer with breast implant.
5. c. Latissimus dorsi muscle transfer.

References

[1] Buchanan P, Leyngold M, Mast BA. Bipolar latissimus dorsi transfer for restoration of pectoralis major function in Poland syndrome. Ann Plast Surg. 2016; 77(1):85–89

[2] Ram AN, Chung KC. Poland's syndrome: current thoughts in the setting of a controversy. Plast Reconstr Surg. 123(3):949–953, discussion 954–955

[3] Seyfer AE, Fox JP, Hamilton CG. Poland syndrome: evaluation and treatment of the chest wall in 63 patients. Plast Reconstr Surg. 126(3):902–911

[4] Seyfer AE, Icochea R, Graeber GM. Poland's anomaly. Natural history and long-term results of chest wall reconstruction in 33 patients. Ann Surg. 208 (6):776–782

[5] Fokin AA, Robicsek F. Poland's syndrome revisited. Ann Thorac Surg. 74(6): 2218–2225

[6] Haller JA , Jr, Colombani PM, Miller D, Manson P. Early reconstruction of Poland's syndrome using autologous rib grafts combined with a latissimus muscle flap. J Pediatr Surg. 19(4):423–429

[7] Longaker MT, Glat PM, Colen LB, Siebert JW. Reconstruction of breast asymmetry in Poland's chest-wall deformity using microvascular free flaps. Plast Reconstr Surg. 99(2):429–436

[8] Borschel GH, Izenberg PH, Cederna PS. Endoscopically assisted reconstruction of male and female Poland syndrome. Plast Reconstr Surg. 109(5):1536–1543

[9] Hurwitz DJ, Stofman G, Curtin H. Three-dimensional imaging of Poland's syndrome. Plast Reconstr Surg. 94(5):719–723

[10] Wright AR, Milner RH, Bainbridge LC, Wilsdon JB. MR and CT in the assessment of Poland syndrome. J Comput Assist Tomogr. 16(3):442–447

[11] Garcia VF, Seyfer AE, Graeber GM. Reconstruction of congenital chest-wall deformities. Surg Clin North Am. 69(5):1103–1118

[12] Pereira LH, Sabatovich O, Santana KP, Picanço R, Sterodimas A. Surgical correction of Poland's syndrome in males - a purposely designed implant. J Plast Reconstr Aesthet Surg. 61(4):393–399

[13] Santi P, Berrino P, Galli A. Poland's syndrome: correction of thoracic anomaly through minimal incisions. Plast Reconstr Surg. 76(4):639–641

[14] Santi P, Berrino P, Galli A, Quondamcarlo C, Rainero ML. Anterior transposition of the latissimus dorsi muscle through minimal incisions. Scand J Plast Reconstr Surg. 20(1):89–92

[15] Borschel GH, Costantino DA, Cederna PS. Individualized implant-based reconstruction of Poland syndrome breast and soft tissue deformities. Ann Plast Surg. 59(5):507–514

[16] Gatti JE. Poland's deformity reconstructions with a customized, extrasoft silicone prosthesis. Ann Plast Surg. 39(2):122–130

39 Gynecomastia

Brandon K. Richland, Emil Kohan, and Gregory R.D. Evans

Abstract
Gynecomastia is an anomaly involving the enlargement of the male breast due to an increase in glandular, stroma, and/or fat tissue. Physiologic and pathologic etiologies of this condition are reviewed, with special emphasis given to the possibility of breast cancer. Surgical options discussed in detail include traditional suction-assisted lipectomy, ultrasound-assisted liposuction, the pull-through technique, direct periareolar excision, circumareolar resection, vertical resection, inverted T resection, and subcutaneous mastectomy with free nipple graft. The authors conclude their chapter with guidelines on postoperative care and outcomes.

Keywords: true gynecomastia, pseudogynecomastia, liposuction

39.1 Goals and Objectives

- Understand the etiologies for both physiologic and pathologic gynecomastia.
- Appreciate the presentation of gynecomastia and key findings in the history and physical examination that warrant additional testing.
- Understand the various treatments for gynecomastia and when each is indicated.
- Be able to discuss postoperative care, including complications.

39.2 Patient Presentation

39.2.1 Introduction

Gynecomastia remains an important problem encountered by plastic surgeons. It is the most common hyperplastic breast anomaly in children, affecting up to two-thirds of adolescent males during puberty.[1] True gynecomastia is the benign enlargement of the male breast resulting from an increase in glandular (ductal not lobular tissue), stroma, and/or fat. Pseudogynecomastia describes an isolated increase in fat. Patients may present with unilateral or bilateral breast tenderness and pain, a palpable lump, concern for malignancy, and dissatisfaction with their appearance. Gynecomastia is not just a cosmetic problem as patients often experience extreme psychosocial and self-esteem issues.[2] These patients suffer teasing from peers, have difficulty entering into relationships, and may avoid showers, swimming pools, or any sports activity that may result in exposing their chest. In these patients, consideration for psychotherapy as an adjuvant to surgical treatment should be given.[2,3]

39.2.2 Etiology

The most common cause of gynecomastia is idiopathic (25%). Other causes can be broken down into physiologic and pathologic.[4,5] Physiologic gynecomastia has three categories: (1) neonatal, (2) adolescent, and (3) elderly. The cause is an increase in the estrogen to testosterone ratio, or defects in testosterone receptors. Obesity may contribute to the onset of gynecomastia due to increased estrogen secretion from fat cells. At birth, gynecomastia can be seen as higher levels of circulating maternal estrogens are present. This type of gynecomastia is transient and resolves as estrogen levels decline. Adolescence is the most common time period patients experience physiologic gynecomastia and can be seen in up to two-thirds of adolescent males during puberty. While gynecomastia may resolve with time and/or weight loss, surgery is an option when gynecomastia is persistent. Gynecomastia has a third peak in the elderly patients older than 65 years, believed to be from a decline in testosterone.[4,5]

A list of pathologic causes are shown in ▶ Table 39.1 and can be further divided into (1) systemic diseases, such as liver failure, renal failure, and malnutrition; (2) diseases/tumors of endocrine glands such as thyroid, pituitary, adrenal, and testes; and (3) drugs and medications.[4,5]

39.3 Breast Cancer and Gynecomastia

The incidence of breast cancer in patients with gynecomastia is similar to that of the general male population. Males make up approximately 1% of all breast cancer, with an occurance of about 1 in every 1,000 males.[5,6] This means treatment with liposuction, in which no specimens are sent to pathology, can be safely performed without significant concern for occult malignancy.[7] Patients with Klinefelter syndrome, however, have a 20- to 30-fold higher risk of developing breast cancer than the general male population. While this must be considered for preoperative planning and surgical treatment, it is unclear if every male with Klinefelter syndrome should undergo

Table 39.1 Most common etiologies of gynecomastia

Idiopathic		
Obesity		
Physiologic		• Neonatal • Adolescent • Elderly
Pathologic	Systemic	• Liver failure • Renal failure • Malnutrition
	Diseases/tumors of the endocrine glands	• Pituitary—hypopituitarism • Thyroid—hyper or hypo • Adrenal—Cushing's syndrome, congenital adrenal hyperplasia • Testis—hypogonadism, Klinefelter syndrome • Drug induced—estrogens, androgens, spironolactone, cimetidine, ketoconazole, isoniazid, amiodarone, digoxin, alcohol, marijuana

screening mammography prior to breast surgery, as males still have a lower risk of breast cancer than the general female population.[6] In these patients, a discussion about male breast cancer should be initiated, and screening mammography should be performed on a case-by-case basis.

39.3.1 Histology

There are two main histologic types of gynecomastia: florid and fibrous. Florid gynecomastia is seen within the first few months of onset. It is characterized by an increase predominantly in ductal tissue. Fibrous gynecomastia is usually seen greater than 1 year after onset. Histology demonstrates an increase in fibrous stroma with minimal ducts. There is an intermediate subtype which occurs in the transition period from several months to 1 year of onset.[8]

39.3.2 Classification

Gynecomastia may range from minimal enlargement of breast tissue to severe overgrowth with excess skin and/or ptosis (▶ Fig. 39.1). Classification systems take into account volume of breast tissue enlargement and whether or not there is excess skin and/or ptosis. Patients have different severities on presentation, varying from a retroareolar mass with no skin excess (▶ Fig. 39.1a,b), to extensive breast tissue and excess skin with no ptosis (▶ Fig. 39.1c,d), to significant breast tissue with skin excess and ptosis (▶ Fig. 39.1g,h). Although over the last several decades many classification systems have been developed for gynecomastia, there is no universally used system.

39.4 Preparation for Surgery

The history and physical examination is essential in evaluating patients who present with gynecomastia. Questioning should include symptoms, age of presentation, duration, whether the size of the breasts is stable, a careful review of systems and drug/medication use. Patients undergoing surgery must have proper assessment of risk factors that may influence safety and potential complications.

Key physical examination findings include a "pinch test" to help determine consistency of breast tissue (glandular, fibrous, solid, or fatty). The classic presentation for glandular gynecomastia is a mobile well-circumscribed retroareolar mass with a rubbery consistency. Excess skin and/or ptosis will affect treatment decisions. It is important to note that gynecomastia can be unilateral in 14 to 51% of patients, so unilateral breast enlargement alone is not cause for alarm.[9,10,11] Special attention should be given to abnormalities that raise concern for cancer such as spontaneous nipple discharge, irregular skin changes, and nodules with varying consistencies. Physical examination of the thyroid and testes should also be performed to evaluate for pathologic causes.

Laboratory testing and imaging studies are not necessary in every case. Healthy adults and pubertal adolescents having a history consistent with physiologic gynecomastia and a normal physical examination do not require further testing. If abnormalities are found on history or physical examination, or there is concern for pathologic gynecomastia, further testing is indicated. Prepubertal gynecomastia requires additional workup including testicular ultrasound. Patients with feminizing characteristics (small testis, lack of male hair distribution) also require hormone testing. Feminizing characteristics associated with a Marfanoid body habitus are associated with Klinefelter syndrome. Karyotype and hormonal testing are indicated. Testicular or thyroid masses require hormone testing and ultrasound.[4,5] Massive weight loss patients may require additional testing to assess nutrition and blood count.[12]

39.5 Treatment

Surgery remains the gold standard treatment of gynecomastia. Indications include to improve symptoms, cosmesis, and patient well-being. Intervention should be considered no sooner than 12 months after the onset. Any abnormalities, underlying conditions, and causative drugs/medications should be addressed

Fig. 39.1 (a–h) Spectrum of gynecomastia presentation.

and treated prior to surgery. Treatment of the underlying cause and discontinuation of causative medications can cause regression of gynecomastia. Despite different approaches, objectives generally remain the same: to correct the appearance of a feminine chest, eradicate the inframammary fold, remove redundant breast tissue and/or skin, and to achieve symmetry and minimize scaring.

Surgical approaches to gynecomastia include the following techniques: traditional suction-assisted lipectomy (SAL), ultrasound-assisted liposuction (UAL), the pull-through technique, direct periareolar excision, circumareolar resection, vertical resection, inverted T resection, and subcutaneous mastectomy with a free nipple graft. Treatment is based on the degree of excess skin and/or ptosis, and the predominant tissue present in the breast. Mild cases of gynecomastia with no excess skin and/or ptosis can be initially treated by liposuction or excision without skin resection. Fatty tissue can be treated with liposuction, while glandular, fibrous, or solid tissue generally does not respond to liposuction and requires surgical excision. More severe cases of gynecomastia with excess skin and/or ptosis may require a skin resection procedure at the initial surgery.[13,14,15,16,17]

39.6 Operative Technique

39.6.1 Liposuction

Patients with an isolated excess of fatty tissue can usually be treated with liposuction alone. Liposuction is important in achieving a smooth chest wall contour, disrupting the inframammary fold and removing fatty tissue with minimal scarring. The patient is marked in the upright position, highlighting areas of tissue excess and breast landmarks including the inframammary fold. Under general anesthesia, the patient is placed in supine position with arms abducted. Preoperative antibiotics should cover skin flora. Tumescent solution is used with the super wet technique, 1:1 ratio of infiltrate:aspirate. A standard tumescent solution contains 1 L of lactated Ringer solution + 25 to 50 mL of 1% lidocaine + 1 amp of 1:1,000 epinephrine (end concentration of epinephrine is 1:1,000,000) + /– 12.5 mL of 8.4% bicarb. Commonly two incisions are utilized, an incision in the lateral aspect of the inframammary fold and a periareolar incision at the inferior aspect of the areola. Tumescent infiltration is performed in all areas of tissue excess, through both incisions, using a feathering crosshatch pattern. The surgeon's nondominant hand should be used to guide the cannula through the subdermal layer. A prominent inframammary fold in males is abnormal and creates the appearance of feminine breasts. While not every surgeon disrupts this fold, some surgeons feel it allows for a smooth transition between the breast and abdomen and improves postoperative skin retraction.[5] Finally, evacuation is performed with a standard 3-mm cannula for final contouring. The ideal endpoint is a smooth flat chest without palpable or visible excess tissue (▶ Fig. 39.2).

39.6.2 The Pull-Through Technique

After treatment with liposuction, the majority of patients still have glandular, fibrous, or solid tissue remaining. This remaining excess tissue is now stringy or fragmented and can be identified by direct palpation. In this situation, the pull-through technique is useful. The inframammary incision is enlarged to approximately 1.5 cm until the excess tissue can be adequately manipulated. This tissue is grasped, pulled out through the enlarged incision and excised until the desired endpoint is reached. Care should be taken to leave a small amount of tissue deep to the areola, usually 0.5 to 1 cm in thickness. Excising too much retroareolar tissue may cause unwanted nipple and areolar depression resulting in a "saucer" deformity (▶ Fig. 39.2).

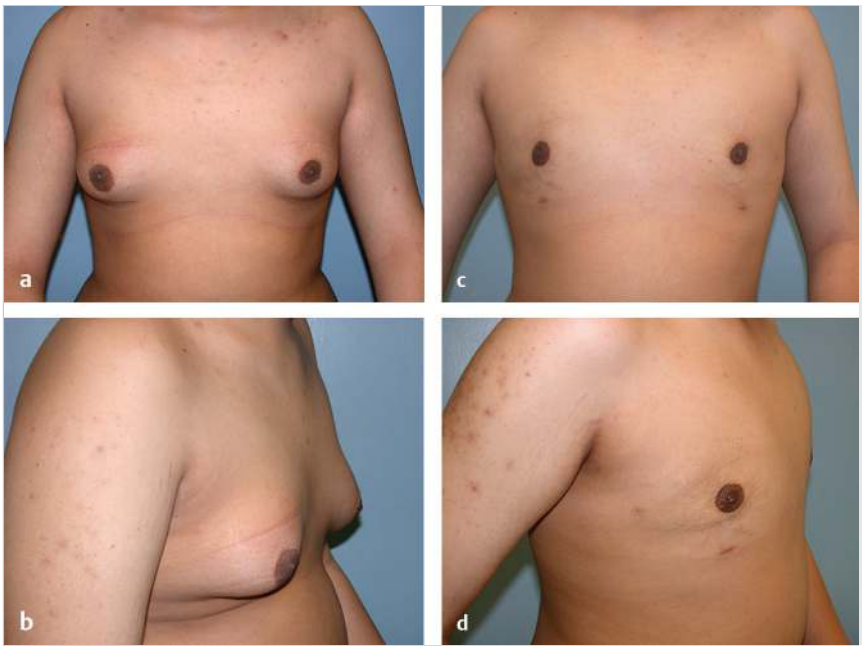


Fig. 39.2 (a,b) 16 year-old with Grade II adolescent gynecomastia. **(c,d)** 1 year following suction lipectomy, and transareolar simple mastectomies.

39.6.3 Direct Periareolar Excision

A common presentation for patients with gynecomastia is an isolated rubbery well-circumscribed mass which resides in the retroareolar position. In these cases, liposuction will be of limited use, and direct periareolar excision can be utilized (▶ Fig. 39.3, ▶ Fig. 39.4). This procedure differs from the pull-through technique in that the initial incision is made around the areola rather than enlarging the liposuction incision. The glandular tissue is well-formed rather than stringy or fragmented because it has not been subjected to liposuction. While these two techniques have some overlap, the liposuction with pull-through is more useful for patients with excess fatty tissue and limited glandular hypertrophy. The direct periareolar excision is more useful for patients with isolated retroareolar glandular hypertrophy.

The direct periareolar excision can be performed under local anesthesia, intravenous sedation, or general anesthesia. A 180-degree semicircular periareolar incision is commonly used along the inferior aspect of the areola. This marking should be exactly at the skin areolar junction and performed before injecting local anesthetic to avoid disruption of landmarks. An incision inside the skin areolar junction may cause a white line while an incision outside the skin areolar junction may cause

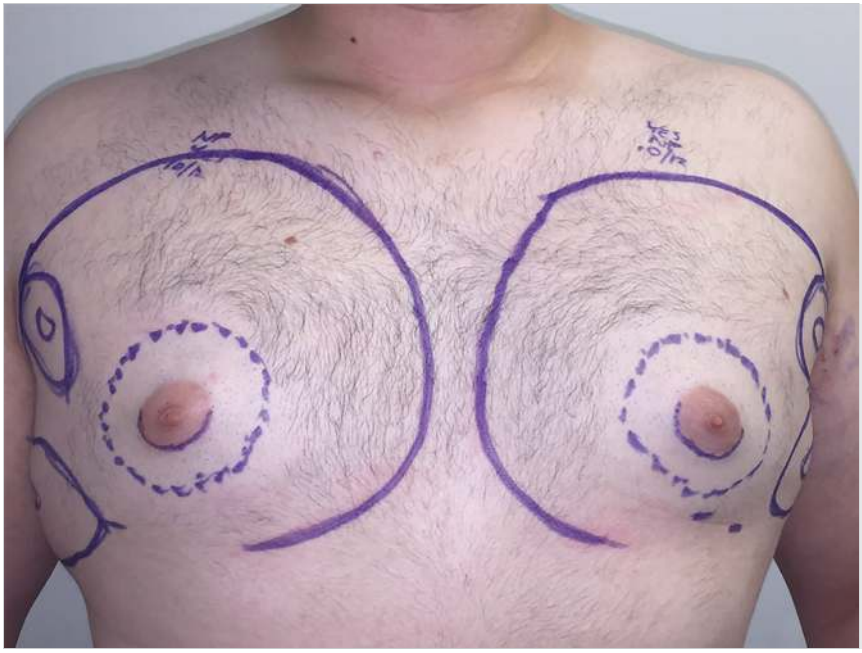


Fig. 39.3 Inner dashed line represents the perimeter of the direct periareolar excision, outer solid line showing areas to be liposuctioned.

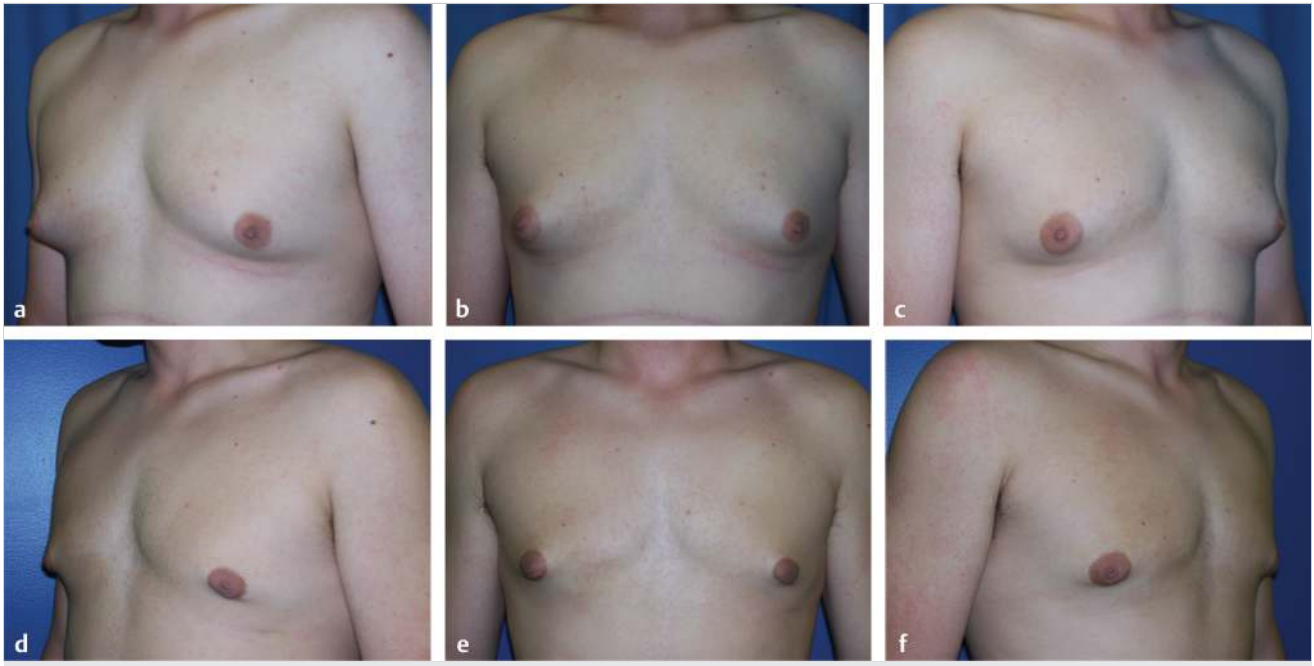


Fig. 39.4 Example of a patient **(a-c)** before and **(d-f)** after direct excision with the use of a compression garment. No skin excision was required.

hypertrophic scaring. During the resection, care should be taken to leave a small amount of tissue deep to the areola, usually 0.5 to 1 cm in thickness. Excising too much retroareolar tissue may cause unwanted nipple areola depression resulting in a "saucer" deformity. If there is also excess tissue in the periphery of the breast, a more extensive resection is warranted. Chest skin flaps are developed (similar to mastectomy flaps) in the plane between the subcutaneous fat and breast tissue. Excess breast tissue is excised and contoured to achieve a smooth chest wall. Alternatively liposuction can be used for contouring as well.

39.6.4 Skin Resection Circumareolar

In patients with severe gynecomastia, the degree of excess skin and/or ptosis will determine whether a skin resection procedure should be performed initially. Younger patients with good quality skin can achieve significant skin envelope contraction once the underlying parenchyma has been removed.[5,9] Therefore, in patients with only mild excess skin/ptosis it is reasonable to either perform a skin resection procedure initially or assess the results of the first gynecomastia procedure prior to skin resection. When reassessment is performed in 6 to 12 months postoperatively, many patients may not require a skin resection procedure at all (Box 39.1).[5,18] Advantages of staging the procedure are ease of contouring the chest at the second operation, and the potential for decreased scarring. Performing skin resection initially may avoid a second operation, which may be of increased importance to cosmetic patients responsible for costs. In either case, it is important to initiate a discussion about the risks and benefits of performing skin resection initially versus a staged procedure.

Box 39.1

In the event that a skin resection procedure is performed, the amount of skin resected is less than what would have been required if the procedure were not staged.

Patients with severe excess skin and/or ptosis will require a skin resection procedure initially. This includes circumareolar resection, formal breast reduction techniques with vertical or inverted T patterns, and free nipple grafts (▶ Fig. 39.6, ▶ Fig. 39.7). An algorithm can be followed that is similar to a female mastopexy. These patients are usually obese, or are massive weight loss patients. Their feminine breast characteristics result from a combination of excess breast tissue, excess skin, breast ptosis, and a prominent inframammary fold. While resection of excess skin may be necessary in these patients, patients are informed that they will be trading improved contour for residual scars.

For a circumareolar resection, the breast landmarks and the inframammary fold are marked with the patient in standing position (▶ Fig. 39.5). The inner circular marking on the areola approximates the new size of the areola. The male areola is approximately 25 to 30 mm (28 mm average) and oval in shape. This is smaller than the female areola. The markings should be made with the skin on moderate radial stretch, as this will approximate the tension after closure. Not placing the skin on stretch may result in an enlarged areola postoperatively. The outer marking is designed to place the resulting nipple–areola complex in the ideal position. This location should be just above the inframammary fold/inferior border of the pectoralis major and slightly lateral to the mid clavicular line. Typical sternal

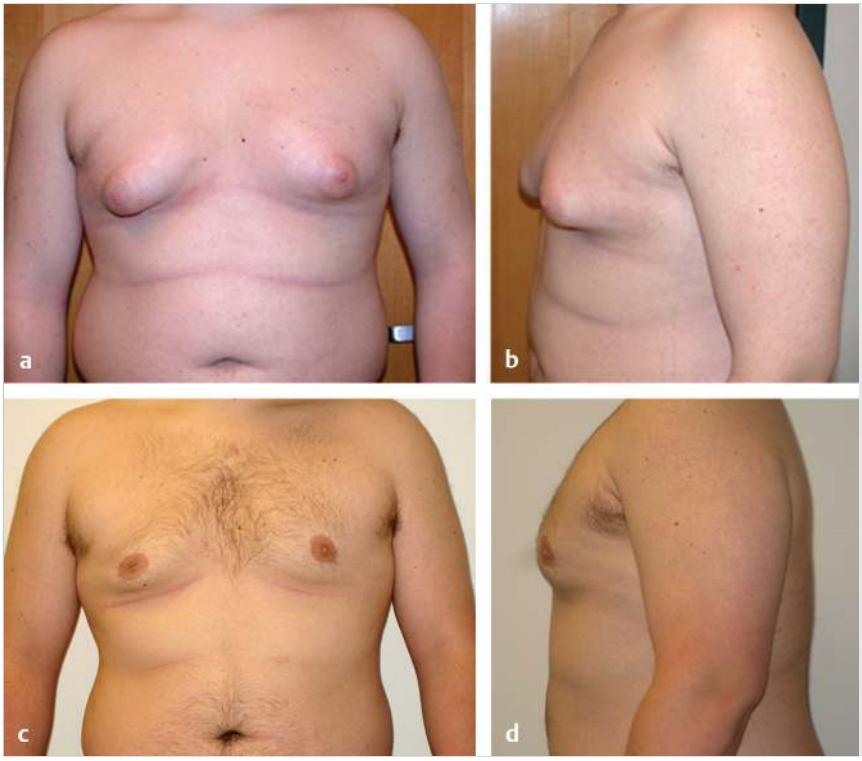


Fig. 39.5 A 42-year-old male presenting with severe bilateral gynecomastia with significant excess in breast tissue and skin, as well as ptosis of the breasts. (**a,b**) Pre-op. (**c,d**) Post-op from liposuction and circumareolar resection.

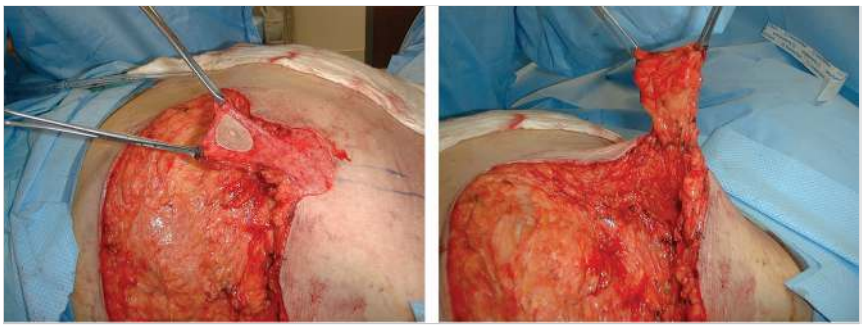


Fig. 39.6 Massive weight loss patient with severe skin excess as well as fibroglandular excess. Treatment was provided using a vertical mastopexy technique. The superiorly pedicled nipple complex and a dermal pedicle is demonstrated with associated skin flap and tissue resection. Courtesy of Bruce A. Mast, MD.

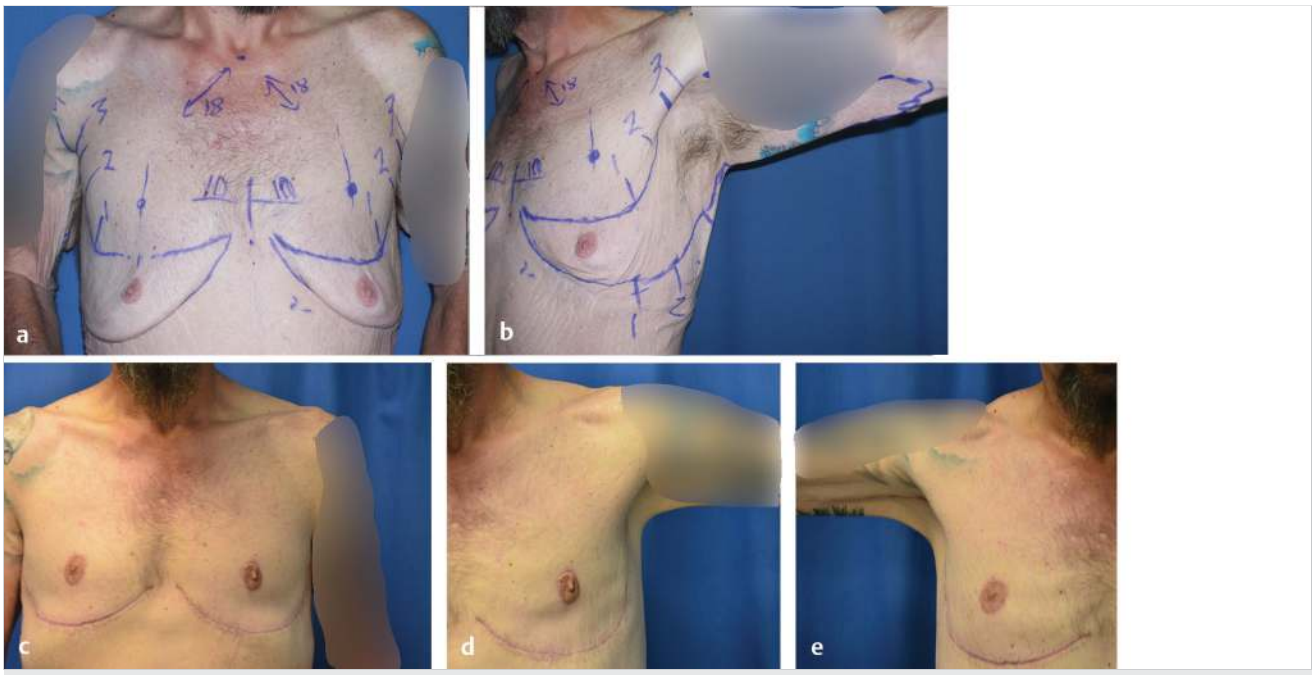


Fig. 39.7 (**a**,**b**) Preoperative and (**c**-**e**) postoperative photo of a massive weight loss patient with severe skin excess and breast ptosis. Preoperative markings for skin resection combined with free nipple grafting, with extension of skin resection into the flanks and continued as brachioplasty. Courtesy of Bruce A. Mast, MD.

notch-to-nipple distance is 20 cm and nipple-to-nipple distance is 21 cm.[19] Incisions are made through the inner and outer markings and the skin between the two incisions is deepithelialized. Liposuction is performed to contour the periphery while any remaining retroareolar glandular, fibrous, or solid tissue can be removed with the pull-through technique or 180-degree inferior periareolar incision through the dermis. If the dermis must be incised circumferentially, a central pedicle can be used to maintain blood supply to the nipple–areolar complex.

39.6.5 Skin Resection, Inframmary Crease, with or without Free Nipple Graft

Massive weight loss patients present an interesting challenge in the surgical approach to gynecomastia. After bariatric surgery most patients lose about 50 to 70% of their excess weight over 1 to 2 years, and stabilize at a body mass index (BMI) of around 30 to 35. No reconstruction should be undertaken until the patient's weight has stabilized for 6 to 12 months. While bariatric surgery can improve hypertension, hyperlipidemia, type 2 diabetes, obstructive sleep apnea, and death from cardiovascular causes, these patients may develop nutritional deficiencies (iron; folate; calcium; albumin; and vitamins B12, A, D, E, and K), protein deficiency, anemia, and increased wound complications.[12,20]

While the rare massive weight loss patient may present with mild gynecomastia that can be treated without skin resection, it is more common to see these patients with severe excess skin and ptosis requiring significant skin resection (▶ Fig. 39.7). They are also more likely to present with varying degrees of pseudogynecomastia, depending on the degree of post bariatric surgery weight loss, rather than hypertrophy of actual glandular tissue. Currently, there are insufficient data to support criteria on when to perform a free nipple graft in massive weight loss patients. However, those patients with severe skin laxity, the nipple–areola complex located well below the inframammary fold, lateral chest rolls, and upper abdominal skin excess, are candidates for this procedure. It is also important to prepare the patient for the possibility of postoperative loss of nipple

sensation, pigmentary changes, a "stuck-on" appearance of the nipple and significant scarring.[9,20]

Breast landmarks and the inframammary fold are marked with the patient in standing position (▶ Fig. 39.7a). Retract downward on breast tissues to mark the superior incision over the desired location of the final horizontal scar. This is usually at the inframammary fold and inferior border of the pectoralis major. The inferior incision is marked using the pinch test to remove all excess tissue. Include lateral chest rolls with an axillary "L" extension and excess upper abdominal skin in the resection if possible. The superior incision is made first and excess breast tissues along with the inferior marking are brought toward the superior incision ensuring enough tissue will be removed without creating excess tension. If the markings are accurate, proceed with the inferior incision and complete the resection. The nipple is either maintained on a pedicle (▶ Fig. 39.6), or placed as a free graft with the appropriate size and location as above (▶ Fig. 39.7a,b).

39.7 Postoperative Care

Gynecomastia surgery is most often done as an outpatient, with the potential exception of the procedure being combined with other body contouring procedures. Standard postoperative office visits are provided with the first visit usually within 48 to 72 hours after surgery. A compression dressing or vest is worn at all times for 2 to 4 weeks postoperatively. Some surgeons continue the compressive dressing for an additional 4 weeks at nighttime only. Restrictions include no strenuous physical activity or exercise for 4 to 6 weeks. Activities of daily living and light exercise or walking are allowed. If closed suction drains are used, they are continued until output is less than 30 mL/day. Overall healing is expected to take several months, with skin retraction and continued contouring occurring over the first year.

39.8 Outcomes

Overall complication rates range from 10 to 30% depending on the study, type of operation, grade of gynecomastia, and patient's BMI.[4,5,20] These include bleeding, seroma, wound healing complications, and need for surgical revision. The most common early complication after gynecomastia surgery is hematoma, reported in 5 to 10% of cases. Evacuation is necessary in all significant hematomas to prevent distortion of the breast as the hematoma is replaced by fibrous tissue.[5,18]

The most common late complication after gynecomastia surgery is under-resection of tissue. This is especially common in patients who undergo liposuction alone. Scarring can also be bothersome to patients, especially when undergoing large skin excisions. The need for revision surgery is common, and seen in up to 10% of patients depending on the study. While most changes in sensation of the nipple–areolar complex improve over time, hypesthesia or hyperalgesia can persist.[5,12,18]

Even with these risks, patients undergoing gynecomastia surgery are extremely satisfied with their experience and report improvement in pain, symptoms, and cosmetic appearance.[5,11] Handschin et al in 2008 determined that while almost half of all gynecomastia patients experienced some degree of mastodynia preoperatively, surgery resolved their pain in all cases.[9] Additionally, the psychosocial improvements experienced by patients undergoing gynecomastia surgery are significant. Patients reported improved interpersonal relationships; an increase in social activities, especially ones that may require removing the shirt; and reduction in peers teasing them about their problem.[2,3,21]

Surgery is an effective treatment for properly selected patients with gynecomastia, providing reduction of symptoms, psychosocial improvements, and enhanced cosmesis.

39.9 Review Questions

39.9.1 Choose the Best Answer

1. Which is most likely to be true gynecomastia?
   a) A 23-year-old male with bilateral isolated retroareolar masses not regressing.
   b) A 23-year-old obese male with bilateral enlarged breasts and ptosis.
   c) A 9-year-old male with bilateral enlarging breasts.
   d) A 33-year-old male 3 years post bariatric surgery with severe skin excess and ptosis.
2. What is the most common etiology of gynecomastia?
   a) Marijuana.
   b) Idiopathic.
   c) Sex tumor induced.
   d) Klinefelter syndrome.
3. A mother brings in her 9-year-old son with bilateral enlarging breasts. She complains that he gets made fun of at school and would like them removed. What is the best next step?
   a) Direct periareolar excision.
   b) Suction-assisted liposuction with pull-through.
   c) Skin resection procedure.
   d) Testicular examination and ultrasound.
   e) No testing or treatment at this time.
4. A 23-year-old male with bilateral, isolated, rubbery, well-circumscribed retroareolar masses presents to your office. What is the next best step after thorough history and physical examination?
   a) Direct periareolar excision.
   b) Suction-assisted liposuction with pull-through.
   c) Skin resection procedure.
   d) Testicular examination and ultrasound.
   e) No testing or treatment at this time.
5. What is the best treatment for a 28-year-old man 2 years post bariatric surgery with severe excess skin and ptosis?
   a) Direct periareolar excision.
   b) Suction-assisted liposuction with pull-through.
   c) Circumareolar skin resection.
   d) Reduction mammaplasty with possible free nipple graft.

39.9.2 Answers

1. a. A 23-year-old male with bilateral isolated retroareolar masses not regressing.
2. b. Idiopathic.
3. d. Testicular examination and ultrasound.
4. a. Direct periareolar excision.
5. d. Reduction mammaplasty with possible free nipple graft.

References

[1] Nydick M, Bustos J, Dale JH , Jr, Rawson RW. Gynecomastia in adolescent boys. JAMA. 178:449–454

[2] Kinsella C , Jr, Landfair A, Rottgers SA, et al. The psychological burden of idiopathic adolescent gynecomastia. Plast Reconstr Surg. 129(1):1–7

[3] Kasielska A, Antoszewski B. Effect of operative treatment on psychosocial problems of men with gynaecomastia. Pol Przegl Chir. 83(11):614–621

[4] Cohen IK, Pozez AL, McKeown JE. Gynecomastia. In: Courtiss EH, ed. Male Aesthetic Surgery. St. Louis, MO: Mosby; 1991

[5] Rohrich RJ, Ha RY, Kenkel JM, Adams WP , Jr. Classification and management of gynecomastia: defining the role of ultrasound-assisted liposuction. Plast Reconstr Surg. 111(2):909–923, discussion 924–925

[6] Brinton LA. Breast cancer risk among patients with Klinefelter syndrome. Acta Paediatr. 100(6):814–818

[7] Koshy JC, Goldberg JS, Wolfswinkel EM, Ge Y, Heller L. Breast cancer incidence in adolescent males undergoing subcutaneous mastectomy for gynecomastia: is pathologic examination justified? A retrospective and literature review. Plast Reconstr Surg. 127(1):1–7

[8] Bannayan GA, Hajdu SI. Gynecomastia: clinicopathologic study of 351 cases. Am J Clin Pathol. 57(4):431–437

[9] Handschin AE, Bietry D, Hüsler R, Banic A, Constantinescu M. Surgical management of gynecomastia–a 10-year analysis. World J Surg. 32(1): 38–44

[10] Colombo-Benkmann M, Buse B, Stern J, Herfarth C. Indications for and results of surgical therapy for male gynecomastia. Am J Surg. 178(1):60–63

[11] Wiesman IM, Lehman JA , Jr, Parker MG, Tantri MD, Wagner DS, Pedersen JC. Gynecomastia: an outcome analysis. Ann Plast Surg. 53(2):97–101

[12] Herman CK, Hoschander AS, Wong A. Post-bariatric body contouring. Aesthet Surg J. 35(6):672–687

[13] Cordova A, Moschella F. Algorithm for clinical evaluation and surgical treatment of gynaecomastia. J Plast Reconstr Aesthet Surg. 61(1):41–49

[14] Petty PM, Solomon M, Buchel EW, Tran NV. Gynecomastia: evolving paradigm of management and comparison of techniques. Plast Reconstr Surg. 125(5): 1301–1308

[15] Esme DL, Beekman WH, Hage JJ, Nipshagen MD. Combined use of ultrasonic-assisted liposuction and semicircular periareolar incision for the treatment of gynecomastia. Ann Plast Surg. 59(6):629–634

[16] Gikas P, Mokbel K. Management of gynaecomastia: an update. Int J Clin Pract. 61(7):1209–1215

[17] Lista F, Ahmad J. Power-assisted liposuction and the pull-through technique for the treatment of gynecomastia. Plast Reconstr Surg. 121(3):740–747

[18] Hammond DC. Surgical correction of gynecomastia. Plast Reconstr Surg. 124 (1) Suppl:61e–68e

[19] Beckenstein MS, Windle BH, Stroup RT , Jr. Anatomical parameters for nipple position and areolar diameter in males. Ann Plast Surg. 36(1):33–36

[20] Gusenoff JA, Coon D, Rubin JP. Pseudogynecomastia after massive weight loss: detectability of technique, patient satisfaction, and classification. Plast Reconstr Surg. 122(5):1301–1311

[21] Crerand CE, Magee L. Cosmetic and reconstructive breast surgery in adolescents: psychological, ethical, and legal considerations. Semin Plast Surg. 27 (1):72–78

Part VII

Chest, Trunk, and Lower Extremity Reconstruction

VII

40 Sternal Reconstruction

R. Brad Nesmith, Zachary T. Young, and Adam J. Katz

Abstract

Sternal reconstruction, as the name implies, is a sternal/chest wall reconstruction made necessary due to infection, oncologic resection, and trauma. A presurgical examination drawing on imaging (CT, nuclear), nutritional markers, and knowledge of any prior reconstruction the patient has had to the chest wall is necessary to determining the proper treatment. The authors guide the reader through each stage of treatment, from debridement of infected and necrotic tissue, to soft tissue coverage options (pectoralis major, rectus abdominus, latissimus dorsi, and omental flaps), to adequate drainage. Pictures from actual cases illustrate the discussion in the text. Guidelines for postoperative precautions are followed by closing paragraphs on outcomes and potential complications.

Keywords: sternal wound reconstruction, sternal wound dehiscence, deep sternal wound infections, ipsilateral rectus muscle

40.1 Goals and Objectives

- Understand the etiology and risk factors for sternal wound breakdown.
- Define the goals of reconstruction and various reconstructive options.
- Have an understanding of the indications for use of sternal plating systems and vacuum-assisted closure devices.

40.2 Patient Presentation

The most common indications for sternal wound reconstruction are infection, oncologic resection (+/– subsequent radiation affects), and trauma.[1] The chest wall, composed of 12 paired ribs and a midline sternum, serves to protect the heart, lungs, and great vessels. Numerous antagonistic muscle groups rely on the rigid thoracic framework to produce functional breathing. When the overall structure of the chest is affected, it can lead to significant physiologic changes and associated morbidity. Thus, it is imperative to address any disruption or wound early to limit the effect on the patient's long-term functional status.

Infection most commonly occurs in the setting of cardiac surgery and can manifest in a number of ways. Patients who undergo sternotomy for coronary artery bypass, in which the internal mammary artery (IMA) is harvested, are at an increased risk for sternal nonunion and subsequent wound infections. Studies have shown sternal blood flow is decreased as much as 90% when the IMA is harvested.[2] Infections that occur between the skin and pectoralis fascia are termed superficial sternal wound infections (SSWI) and carry a mortality/morbidity rate between 0.5 and 9%. However, infections that are deep to the pectoralis musculature, deep sternal wound infections (DSWI) or mediastinitis, are more serious with a mortality rate between 10 and 47%. Risk factors for development of DSWI include diabetes, chronic obstructive pulmonary disease (COPD), obesity, tobacco, osteoporosis, prolonged operative times, and reoperation.[1] In addition, patients who develop sternal wound complications after chest surgery are prone to an average of 20 more days in the hospital and a nearly threefold increase in hospital costs.[1] In 1986, Pairolero and Arnold classified infected sternal wounds based on interval between surgery and presentation of symptoms (▶ Table 40.1).[3]

Most commonly patients present with an open wound or draining sinus tract. Patients may also present with a sternal click on exam or the complaint of "bony pain" with deep inspiration or coughing. Intermittent low-grade fevers and overall malaise can be subtle signs of an indolent chronic infection.

40.3 Preparation for Surgery

Reconstruction can be generalized into soft-tissue coverage and rigid skeletal support. Restoration of skeletal support and reestablishing the thoracic framework prevent paradoxical chest wall motion and facilitate physiologic respiration. Preoperative imaging, including CT and nuclear medicine scans, can assist in planned bony resection and assess for the potential need for rigid fixation. However, the decision to use a sternal plating system must take into account any potential infection remaining in the wound bed. Finally, preoperative antibiotic therapy should be guided by available wound biopsies. A treatment duration of 6 to 8 weeks is usually required after adequate bone and soft-tissue debridement.

Table 40.1 Classification of Infected Sternal Wounds

Type	Timing	Presentation	Treatment
Type I	<7 d	Serosanguineous drainage without signs of osteomyelitis/costochondritis	Intravenous antibiotics with single-stage operative debridement/washout
Type II	1st–4th wk	• Purulent drainage and cellulitis • Costochondritis is rare but osteomyelitis is frequent	• Intravenous antibiotics • Operative wide debridement and removal of sternal wires/plates • Muscle flap coverage • Closure over drains
Type III	Months to years	• Chronic draining sinus tract with localized cellulitis • Costochondritis and osteomyelitis are frequently seen	• Wounds are packed open and treated with frequent dressing changes • When clean these are closed with muscle flap coverage • Closure over drains

Poor nutritional markers (prealbumin/albumin) along with suboptimal glucose control can reduce the likelihood of a successful reconstruction. An effort should be made to maximize nutrition and maintain normal glucose levels perioperatively.

Finally, while there are a number of flaps that can be used to reconstruct thoracic defects, the previous use of the internal mammary vessels in coronary bypass, location of the sternal wound, and prior surgeries may limit the options. If there is any question about the availability or patency of a certain flap, preoperative imaging may help reveal potential flap inadequacies.

40.4 Treatment

Wound Preparation and Rigid Skeletal Reconstruction

The thorough and complete debridement of all infected and necrotic tissue followed by vascularized soft-tissue coverage with adequate drainage is key. Tissue cultures showing > 10^5 organisms/cm^3 are considered positive and should be treated with debridement, antibiotics, and possibly vacuum-assisted wound dressing followed by delayed coverage.[4] The immediate use of rigid plating systems should be avoided when possible in the face of positive wound cultures. Debridement should include all devitalized tissue and removal of previously placed implants including sternal wires, plates, and pacing wires. Bone debridement should be performed until bleeding is seen from edges and the cortex is no longer soft and friable (▶ Fig. 40.1, ▶ Fig. 40.2). Radical sternectomy is not indicated and sternal salvage should be attempted if the bone is viable.[4]

Subatmospheric pressure wound therapy may be utilized to increase peristernal blood flow, even in the face of internal mammary harvest.[5] It has also been shown to decrease bacterial counts and decrease dead space by increasing granulation tissue formation.[6,7] Through these mechanisms, negative pressure wound therapy has been shown to decrease the number of days between wound debridement and definitive closure (8.5–6.2 days) and the number of flaps required per patient (1.5–0.9).[8] Finally, by only having to change the negative pressure dressing three times per week instead of the traditional twice per day gauze changes, there is less exposure of the wound and fewer procedures on potentially debilitated patients. The use of vacuum-assisted wound therapy is now the standard of care in many situations. These include in patients who are unstable, whose chest cannot be closed due to cardiopulmonary compromise, or situations in which pathologic disease-free margins must be confirmed.[9]

Rigid fixation is ideal and paramount for bony union and stabilization of the chest wall. It has been shown to prevent paradoxical chest wall motion, improve patient comfort, accelerate healing, and decrease mediastinal hernias compared to flap coverage alone (▶ Fig. 40.3).[10] Studies have shown plating promotes earlier bony union from accelerated osseous healing.[11] The increased stabilization and re-establishment of normal chest function has been shown to decrease ventilator dependence and reduce overall hospital length of stay.[11] Recurrent plate infections can be minimized with thorough debridement, culture-directed antibiotic therapy, and adequate soft-tissue coverage. Cicilioni et al performed rigid fixation on 50 consecutive cases in which 37 cases were culture positive for osteomyelitis and report only one plate infection.[12]

40.4.1 Soft Tissue Coverage

Since 1980, when Maurice J. Jurkiewicz and his Emory colleagues published their 20-year experience, flap coverage has been the gold standard for sternal wounds.[13] Recruitment of adjacent tissue is necessary, as there is typically a very limited

Fig. 40.1 Open mediastinal wound after heart transplant complicated by fungal mediastinitis.

Fig. 40.2 Sternal wound after complete debridement and removal of sternal wires.

Fig. 40.3 Wound after placement of sternal plating system.

Table 40.2 Summary of Common Flaps Useful for Sternal Reconstruction

Flap	Ideal coverage location	Limitations
Pectoralis major	Anterior upper/middle sternal or sternoclavicular defects	Inferior sternal defects
Rectus abdominus	Anterior chest wall defects, need for skin paddle	Previous abdominal operations that may have disrupted the superior epigastric artery or the associated perforators
Latissimus dorsi	Anterior or anterolateral chest wall defects, posterior midline thoracic defects, need for skin paddle	Previous ipsilateral thoracotomy
Omental	Deep medial chest wounds, large soft-tissue defects	Previous intra-abdominal surgery that may have sacrificed the right or left omental pedicle

amount of soft tissue immediately available over the sternum and even less after wide debridement. The muscle flaps most commonly utilized include the pectoralis major, rectus abdominus, and the latissimus dorsi flaps. The omentum is often used for reconstruction of large, deep wounds and can be used alone or in conjunction with other flaps. It is essential to understand the indications and limitations of each flap, as the resultant wound can become greater than anticipated after debridement and one must be able to improvise (▶ Table 40.2).

Pectoralis Major Flap

The pectoralis major muscle serves to internally rotate and adduct the arm. It originates along the sternum, medial clavicle, and superior six costal cartilages and inserts on the bicipital groove of the humerus. The flap is a type V muscle flap, with the thoracoacromial artery being the major supply and additional segmental flow arising from intercostal perforators derived from the IMA. The thoracoacromial artery is a branch of the second portion of the axillary artery. It emerges from under the clavicle between the middle and lateral third of the clavicle. It passes on the undersurface of the muscle between the pectoralis major and minor. The flap can be raised in a fairly avascular plane as a muscle-only or myocutaneous flap. The dissection for flaps based on the thoracoacromial trunk should start medially at the sternal attachment. The superior attachment to the clavicle should be taken down as well. Lateral dissection should be carried out until adequate medialization into the defect is accomplished. The muscle may also be released from its insertion on the humerus to gain more medial advancement and a greater arc of rotation. This is usually performed through a counter incision near the axillae. Conversely, the flap can be used as a turnover flap based on the internal mammary perforators. This should only be performed once the patency of the ipsilateral internal mammary has been confirmed. This dissection proceeds in a suprafascial dissection above the muscle and is carried out until it can be divided from its humoral insertion. Again, this may require a counter incision to fully visualize the insertion. The thoracoacromial artery is divided as the muscle is flipped into the defect. The majority of sternal wounds can be closed with a single pectoralis flap; however, more extensive defects may require bilateral muscle flaps. Inferior sternal defects are difficult to obtain consistent coverage with pectoralis flaps. In such cases, studies have shown that extending the pectoralis dissection to include anterior rectus fascia or splitting the pectoralis turnover flap can help provide coverage inferiorly.

Rectus Abdominus Flap

Each rectus abdominus muscle serves to flex the trunk. It originates on the pubis and inserts on the costal margin. It is a type III muscle flap with the superior and inferior deep epigastric arteries being the two major pedicles. The deep superior epigastric vessel is the continuation of the internal mammary vessel onto the abdomen. It pierces the posterior rectus sheath superiorly just after the final costal cartilage and travels on the posterior surface of the muscle. It anastomoses with the deep inferior epigastric, an ascending branch of the external iliac, at the level of the umbilicus. The flap can be raised as a muscle-only or myocutaneous flap depending on the reconstruction required. In general, the rectus flap is not a good option if the IMA on the same side has been used for bypass. However, in dire settings, when no other flaps are available, some have reported that the rectus flap can be pedicled superiorly even in

Fig. 40.4 After inset of myocutaneous rectus flap into sternal wound defect.

Fig. 40.5 Two months post-op.

the absence of the internal mammary system, based on accessary blood supply from the eighth subcostal artery.[14,15,16] If a muscle-only flap is to be harvested, a variety of incisions can be used. The most common are the midline and the paramedian incision oriented over the rectus muscle. The dissection is taken down to the anterior rectus fascia. This is divided longitudinally exposing the entire length of the muscle. The anterior sheath is dissected off the muscle without disrupting the inscriptions and violating the integrity of the muscle. Localization and division of the deep inferior epigastric vessels on the lateral side of the muscle near its origin allows for elevation of the flap in an avascular plane from inferior to superior. Care must be taken to avoid injury to the pedicle as it runs along the underside of the muscle. The muscle can be transposed into the defect and covered with local soft tissue and skin, or grafted skin. The divided anterior rectus fascia is approximated and the abdomen is closed. If a myocutaneous flap is desired, a template can be designed from the defect and oriented either vertically, obliquely, or horizontally over the rectus muscle. After division of the skin and soft tissue, the anterior rectus fascia under the skin paddle is excised and is taken with the flap (▶ Fig. 40.4, ▶ Fig. 40.5). The resultant anterior fascial defect may be too large for primary closure, requiring that mesh repair be performed. It is important to explain to the patient preoperatively that this may ultimately result in abdominal wall laxity (i.e., bulging) or hernia. It is critical to assess the patient before surgery for prior abdominal operations that may have compromised cutaneous perforators or violated the intramuscular blood supply.[4]

Latissimus Dorsi Flap

The latissimus dorsi muscle functions to adduct, extend, and internally rotate the arm. Known as the climbing muscle, it originates along the thoracolumbar fascia and iliac crest and inserts into the intertubercular groove on the superior aspect of the humerus. It is a type V muscle flap with the major arterial supply being the thoracodorsal artery in addition to the segmental posterior intercostal perforators. The thoracodorsal artery is the terminal branch of the subscapular circumflex artery which is a branch of the axillary artery. The thoracodorsal artery pedicle runs along the posterior surface and enters the muscle approximately 5 cm from the posterior axillary fold. Similar to the previously described flaps, this can be raised as a muscle-only or a myocutaneous flap. The muscle is very broad with a significant arc of rotation making it ideal for reconstruction/coverage of upper extremity, back, and chest wall defects. For sternal wounds, this flap can be used in patients who do not have usable pectoralis muscles or in combination with other flaps. Preoperative markings should include any desired skin paddle and the location of the scapular tip which marks the superomedial extent of the muscle. The dissection is fairly straightforward and should attempt to harvest the full length

and width of the flap. Exposure should start with defining the borders of the muscle along the lower six thoracic vertebrae, iliac crest, along the scapular tip and its interface with the serratus and oblique musculature. Once the muscle has been fully exposed, it may be raised from its caudal attachments and continued toward the thoracodorsal pedicle. Care must be taken to avoid accidentally elevating the serratus. To avoid complaints of flap animation and postoperative pain, a small portion of the thoracodorsal nerve may be resected. To gain more arc of rotation, the humeral attachment may be divided. Once completely mobilized, the flap may be transposed into the defect. Seromas are very common with this flap donor site; so, closure over drains and possibly the use of quilting sutures can help reduce the incidence.

Fig. 40.6 A 72-year-old male with large basal cell of the anterior chest. Resection resulted in a large full-thickness defect requiring reconstruction.

Omental Flap

The omentum has been used for various reconstructions over the entire body for more than 100 years.[14] The fatty abdominal apron is composed of visceral fat, blood vessels, and an abundance of lymphatics. It originates along the greater curvature of the stomach and is supplied by two dominant pedicles—the right and left gastroepiploic arteries. The two properties that make this flap ideal for reconstruction are the potential flap volume, which can cover and fill large deep wounds (▶ Fig. 40.6, ▶ Fig. 40.7, ▶ Fig. 40.8, ▶ Fig. 40.9), and its pedicle length, which can grow with the division of arterial arcades. For sternal wound reconstruction, the flap is most commonly isolated on the right gastroepiploic artery and can be passed into the defect either through the diaphragm or over the costal margin. It is preferred to pass the omentum through the right hemidiaphragm to avoid the heart and to allow the left lobe of the liver to buttress the defect and prevent hernia.[4] Alternatively, the flap can be brought out through the upper abdomen and tunneled subcutaneously into the sternal defect. Once interpolated, the flap may be covered with local soft tissue or a skin graft. This flap is ideal for an extensive defect, patients with no remaining available muscle flaps secondary to previous surgery or damage due to chest wall radiation. The omentum can also be used in conjunction with other flaps to fill a full-thickness sternal defect prior to coverage with other muscle flaps. Again, a thorough understanding of the patient's prior surgical history, with attention to any previous resection or use of the omentum will help avoid unnecessary laparotomies. The omentum may also be harvested laparoscopically to avoid a laparotomy.

Fig. 40.7 Omentum harvested through small epigastric incision and tunneled into large defect.

Fig. 40.8 Skin grafting of omental flap.

Fig. 40.9 Two months post-op.

In all of these reconstructive options, there is wide and extensive tissue dissection making seromas common. These can be difficult to deal with for the patient and practitioner alike. For this reason, judicious use of closed suction drains is recommended for the donor and the recipient site.

40.4.2 Postoperative Care

All patients undergoing sternal reconstruction are placed on sternal precautions for 6 weeks with strict instructions to avoid direct pressure to the sternum. Postoperative antibiotic therapy is lengthy and should be guided by tissue cultures and possibly the recommendations of the infectious disease team. Drains are continued until outputs drop below 30 mL/day for 2 consecutive days. No routine imaging is required unless concerns arise postoperatively. Patients are routinely seen 1 week post discharge to ensure the wound is progressing well, and then with decreasing frequency after the drains are removed.

40.5 Outcomes

The incidence of DSWI after median sternotomy is between 1 and 5%.[14] The associated mortality ranges in the literature from 10 to 47%.[14,17] These patients should be treated with debridement, antibiotics, and flap coverage. Studies have shown that patients referred to and treated by plastic surgeons have significantly decreased ventilator dependence, tracheostomy, development of stage III/IV pressure sores, major wound dehiscence, hospital length of stay, and mortality compared with patients who are not referred.[18] Brandt and Alvarez compared a group of patients managed with debridement followed by muscle or

omental flap reconstruction to a similar group of patients treated at the same institution with traditional debridement, rewiring, and closed suction drainage. They demonstrated a 22% major complication and 0% mortality rate in their group compared to 92% rate of major complications and a 33% mortality rate in the traditionally treated group.[19]

Complications requiring reoperation following sternal wound reconstruction include recurrent infection and wounds, acute incisional dehiscence, and hematoma. Zahiri and colleagues found that reoperative complications were associated independently with the comorbidities of diabetes, hypertension, coronary artery, congestive heart failure, and renal insufficiency.[20] Furthermore, most surgeons would agree that recurrent infection and their associated wounds are likely due to retained chondritis, since the extent of parasternal costal cartilage debridement at the time of reconstruction is the most difficult to judge clinically.

The disparaging outcomes for patients not treated aggressively are significantly influenced by their overall fragile state. These patients are often ventilator dependent, diabetic, obese, malnourished, and have impaired pulmonary reserve. These patients must be evaluated and treated quickly to help restore normal chest wall function and provide vascularized muscle coverage to help encourage healing and delivery of antibiotics.

40.6 Review Questions

1. What are the risk factors associated with the development of sternal wound dehiscence and subsequent deep sternal wound infections?

Factors that influence wound healing after any surgery-uncontrolled diabetes, smoking, chronic steroid use, and poor nutrition are concerns in sternal wound healing as well. Factors particular to sternal wound healing include the use of the internal mammary artery, previous sternotomies, COPD, obesity/macromastia, osteoporosis, use of intra-aortic balloon pump devices, and prolong operative times.

2. What are the indications for rigid sternal plating?

Rigid fixation of the sternum has been shown to produce earlier boney union due to accelerated osseous healing and increased patient comfort with the elimination of paradoxical sternal movement. Indications for rigid fixation include all patients who require boney debridement due to devitalized tissue and good opposition between the sternal halves is suboptimal. Plating high-risk patients (diabetes, COPD, steroid dependence, obese [BMI > 30], renal failure, or repeat sternotomy) primarily has been shown superior to wire cerclage alone.

3. What are soft-tissue coverage options for sternal wounds?

As outlined above, soft-tissue options should be dictated by the location and extent/size of the sternal wound. Also, making sure to understand any prior surgeries that may eliminate various future reconstructive options.

4. Does harvesting the internal mammary artery for cardiac bypass preclude the use of the ipsilateral rectus muscle for flap coverage?

While the authors do not recommend the use of the ipsilateral rectus muscle in the face of previous IMA harvest, studies have shown that the muscle may be based on an accessory blood supply from the intercostal vessels. This would be used, in our practice, as an option only when no other options exist and not as a first choice.

5. What precautions should these patients be placed on after surgery?

Patients should be instructed to be on strict sternal precautions for 6 weeks postoperatively. This includes the following:

a) No pulling or pushing when getting into/out of a chair or bed.
b) No flexing/extending of arms at the shoulder greater than 90 degrees.
c) Brace yourself with pillow for any coughing/sneezing.
d) Call your physician with any drainage, redness, or clicking.

40.6.1 True or False

1. Use of the internal mammary artery (IMA) in cardiac bypass surgery does not affect blood flow to the sternum because of the compensatory opening of “choke” vessels.
2. Negative pressure wound therapy is an ineffective adjunct in the care of sternal reconstruction patients.
3. More often than not, a rectus abdominus flap harvested from the same side as a previously harvested IMA will be dependably successful if used for reconstruction.

40.6.2 Choose the Best Answer

4. All of the below are associated with deep sternal wound infections:
   a) Mortality rates of nearly 50%.
   b) Increased length of stay.
   c) Increased cost of care.
   d) None of the above.
   e) All of the above.
5. Basic principles of sternal wound reconstruction include:
   a) Thorough debridement of infected, non-vital tissue.
   b) Culture-guided antibiotics.
   c) Removal of all hardware/foreign body.
   d) Soft-tissue coverage with or without bony fixation.
   e) All of the above.

40.6.3 Answers

1. False.
2. False.
3. False.
4. All of the above (e).
5. All of the above (e).

References

[1] Singh K, Anderson E, Harper JG. Overview and management of sternal wound infection. Semin Plast Surg. 25(1):25–33
[2] Peterson MD, Borger MA, Rao V, Peniston CM, Feindel CM. Skeletonization of bilateral internal thoracic artery grafts lowers the risk of sternal infection in patients with diabetes. J Thorac Cardiovasc Surg. 126(5): 1314–1319
[3] Pairolero PC, Arnold PG. Management of infected median sternotomy wounds. Ann Thorac Surg. 42(1):1–2
[4] Song D, Roughton M. Reconstruction of the chest. In: Neligan P, Buck D II, eds. Core Procedures in Plastic Surgery. Elsevier; 2013:239–255

[5] Wackenfors A, Gustafsson R, Sjögren J, Algotsson L, Ingemansson R, Malmsjö M. Blood flow responses in the peristernal thoracic wall during vacuum-assisted closure therapy. Ann Thorac Surg. 79(5):1724–1730, discussion 1730–1731
[6] Argenta LC, Morykwas MJ. Vacuum-assisted closure: a new method for wound control and treatment: clinical experience. Ann Plast Surg. 38(6): 563–576, discussion 577
[7] Morykwas MJ, Argenta LC, Shelton-Brown EI, McGuirt W. Vacuum-assisted closure: a new method for wound control and treatment: animal studies and basic foundation. Ann Plast Surg. 38(6):553–562
[8] Song DH, Wu LC, Lohman RF, Gottlieb LJ, Franczyk M. Vacuum assisted closure for the treatment of sternal wounds: the bridge between débridement and definitive closure. Plast Reconstr Surg. 111(1):92–97
[9] Agarwal JP, Ogilvie M, Wu LC, et al. Vacuum-assisted closure for sternal wounds: a first-line therapeutic management approach. Plast Reconstr Surg. 116(4):1035–1040, discussion 1041–1043
[10] Sargent LA, Seyfer AE, Hollinger J, Hinson RM, Graeber GM. The healing sternum: a comparison of osseous healing with wire versus rigid fixation. Ann Thorac Surg. 52(3):490–494
[11] Voss B, Bauernschmitt R, Will A, et al. Sternal reconstruction with titanium plates in complicated sternal dehiscence. Eur J Cardiothorac Surg. 34(1):139–145
[12] Cicilioni OJ , Jr, Stieg FH , III, Papanicolaou G. Sternal wound reconstruction with transverse plate fixation. Plast Reconstr Surg. 115(5):1297–1303
[13] Jones G, Jurkiewicz MJ, Bostwick J, et al. Management of the infected median sternotomy wound with muscle flaps. The Emory 20-year experience. Ann Surg. 225(6):766–776, discussion 776–778
[14] Gummert JF, Barten MJ, Hans C, et al. Mediastinitis and cardiac surgery–an updated risk factor analysis in 10,373 consecutive adult patients. Thorac Cardiovasc Surg. 50(2):87–91
[15] Fernando B, Muszynski C, Mustoe T. Closure of a sternal defect with the rectus abdominis muscle after sacrifice of both internal mammary arteries. Ann Plast Surg. 21(5):468–471
[16] Jacobs B, Ghersi MM. Intercostal artery-based rectus abdominis transposition flap for sternal wound reconstruction: fifteen-year experience and literature review. Ann Plast Surg. 60(4):410–415
[17] Braxton JH, Marrin CA, McGrath PD, et al. 10-year follow-up of patients with and without mediastinitis. Semin Thorac Cardiovasc Surg. 16(1):70–76
[18] Cabbabe EB, Cabbabe SW. Immediate versus delayed one-stage sternal débridement and pectoralis muscle flap reconstruction of deep sternal wound infections. Plast Reconstr Surg. 123(5):1490–1494
[19] Brandt C, Alvarez JM. First-line treatment of deep sternal infection by a plastic surgical approach: superior results compared with conventional cardiac surgical orthodoxy. Plast Reconstr Surg. 109(7):2231–2237
[20] Zahiri HR, Lumpkins K, Kelishadi SS, et al. Significant predictors of complications after sternal wound reconstruction: a 21-year experience. Ann Plast Surg. 69(4):439–441

41 Abdominal Wall Reconstruction

Gregory A. Dumanian and Bruce A. Mast

Abstract
This chapter addresses the considerations involved in remediating bulges and hernias. The challenge in these procedures is to create a lasting connection between two divided tissues under tension. Bulges result from lessened strength and elasticity of the abdominal wall and generally pose no risk; hernias come with a higher risk that can result in serious and long-term symptoms. Presurgical steps (e.g., advising the patient to lose weight and/or stop smoking) are outlined, and the procedures for hernia repair are detailed. Unsupported repairs rely solely on sutures to hold the abdominal wall; supported repairs add a reinforcing mesh to the repair site; bridged repairs suture spanning material to the abdominal wall. The authors cover the factors, strengths, and contraindications that should be weighed in deciding which repair best suits a patient. Extended discussion is given the critical areas of skin vascularity, force distribution, and components release with perforator preservation and force distribution with mesh. Images from actual cases supplement the discussion in the text. Postoperative care guidelines and information regarding outcomes and complications are also included.

Keywords: bulge, hernia, rectus diastasis, umbilical hernia defect, spanning mesh

41.1 Goals and Objectives

- Understand the anatomy and forces on the abdominal wall.
- Appreciate the blood supply to the skin and the abdominal wall musculature and how to preserve it during abdominal wall closures.
- Establish an algorithm for the treatment of the open abdominal wound.
- Develop a working knowledge of prosthetic and bioprosthetic meshes for elective hernia repair.
- Develop the concept of force distribution and suture pull-through as it relates to abdominal wall closure.

41.2 Patient Presentation

The central issue to abdominal wall reconstruction (AWR) is the optimal means to create a lasting connection between two divided tissues under tension. Understanding the forces and physiology at the suture/tissue interface (STI) is critical to successful closure of the abdominal wall.

Patient presentation is straightforward, and the majority of diagnoses can be established with a history and physical examination that will distinguish between two conditions—bulges and hernias. Bulges occur when the strength and elasticity of the abdominal wall is not enough to contain the viscera uniformly from episodes of increased intra-abdominal pressure. The classic appearances of postpartum women and the epigastrium of heavier older males (the so-called "beer belly") are consistent with rectus diastasis and bulges. In general, there is no risk to abdominal wall bulges, because the intestines face a smooth peritoneal lining without abrupt transitions and will not strangulate. In certain areas of the body such as the groin, umbilicus, and the epigastrium, a failure of the local tissue to contain the viscera creates a true hernia–distinctive from abdominal wall bulges due to a small defined ring that can permit entry of a bowel loop and possibly lead to strangulation. In contradistinction to the smooth peritoneal contour of bulges, these "ventral" hernias (as they are located on the front surface of the abdomen) have rings in the shape of the letter omega with a lip that can catch the preperitoneal fat and bowel. A third category of abdominal wall defects is incisional hernias, notable for the scar demonstrating a prior entry into the abdomen (▶ Fig. 41.1). The scar at the STI has not withstood the abdominal wall tensions and pressures and has failed over time. Mechanistically, the total strength of the physical construct of repair and the biologic healing is less than the loads applied at some point in the postoperative period, leading to deformation of scar and failure. There is some minor overlap between groups; for instance, a bulge from rectus diastasis can be associated with the development of a true umbilical or epigastric hernia. History and physical examination is typically adequate to differentiate these clinical entities, though an abdominal CT scan can add confirmatory information for heavier patients (▶ Fig. 41.2, ▶ Fig. 41.3).

Patients will present with complaints of pain, bowel disorders, and/or change in abdominal wall contour. Large hernias and bulges associated with a loss of intra-abdominal pressure will often be associated with back pain due to a loss of "core strength" due to decreased intraabdominal pressure. Hernia size and location may not be overly correlated with symptoms, as some small hernias will be symptomatic and large hernias can be unnoticed by the patient. The indication for repair of these hernias and bulges is for the decrease of local pain and the avoidance of bowel incarceration at the hernia site. Quality-of-life improvements from repaired contour and core strength are harder to quantitate but are also present.

The majority of patients will present, as above, with a large ventral hernia, often as a recurrent incisional hernia. Less common are patients who have recovered from an abdominal catastrophe such as necrotizing fasciitis, multiple laparotomies with subsequent skin grafting over bowel, stomas, or large wounds with enterocutaneous fistulae. These patients' complaints are similar to those with large hernias, but have the added issues of potentially larger defects of the abdominal wall with possible absence of multiple anatomic components. They often have a level of debilitation due to long illness and convalescence.

41.3 Preparation for Surgery

All patients are encouraged to lose weight prior to surgery, but this is seldom achieved. Actively smoking patients similarly are urged to stop for 1 to 2 months prior to the procedure, though smokers have not had worse outcomes for the procedures described in the following section. Immunosuppressants are

Fig. 41.1 Patient with a 7-cm wide hernia after colostomy takedown.

Fig. 41.2 Cross-sectional abdominal CT scan of patient with large incisional hernia after liver transplantation.

Fig. 41.3 Cross-sectional abdominal CT scan of patient who has had multiple recurrences from previously failed ventral hernia repairs. The abdominal muscles are visualized bilaterally and labeled on the patient's right: R is rectus abdominus; E is external oblique; I is internal oblique.

managed by the transplantation surgery teams, steroids should be at stable (and low) doses, and sirolimus is to be avoided due to its profound wound healing effects. Standard medical evaluation is done if needed, including cardiac clearance by the preoperative clinic or the patient's medical doctor. Specific assessment of nutritional status, including pre-albumin serum levels, may be required dependent on the patients underlying condition. The lead author (G.A.D.) prescribes a gentle bowel preparation the night before surgery, consisting of clear liquids, a half bottle of magnesium citrate, and two bisacodyl tablets to clear the bowel of particulate matter. This is done to decrease intra-abdominal volume and to minimize the early forces at the STI. Many surgeons do not prescribe a bowel prep if the hernia is easily reducible and no loss of domain is present.

Patient who have recovered from a significant intra-abdominal illness with a resultant large hernia should be fully stable and recovered from the initial health crisis that led to the abdominal wall defect. Additionally, patients with enterocutaneous fistula require maximal nutritional support and maturation of their fistula. If a defect is present with skin-grafted small bowel, a degree of scar maturation is needed to be able to remove the skin graft. Often times, gently pinching the skin graft provides an assessment of the degree of adherence to the underlying bowel. These patients with complex defects most often require a combined surgical effort with the primary surgical service. Confirmation of normal lower extremity anatomy and blood supply is required if flaps from the thigh are considered.

41.4 Treatment

41.4.1 Concepts of Pressure, Tension, and Type of Repair

The goal of a hernia repair is to return uniformity to abdominal wall counter pressure against the viscera. “Unsupported” direct repairs rely solely on sutures to hold the abdominal wall. “Supported” direct repairs attempt to distribute the forces on the repair over a larger area by adding a reinforcing mesh to the suture repair site. In bridged repairs, a spanning material is sutured to the abdominal wall to prevent bowel from entering the hernia sac, but the abdominal wall is not approximated. These three methods of abdominal wall repair have *radically* different conceptualizations of the long-term efficacy of approximating the divided abdominal wall.

A suture acts to appose two divided tissues by applying pressure on the inner aspect of a thin flexible loop. A perfectly placed suture would apply just enough force to bring the tissues together to overcome early distraction forces. In an ideal world when forces required to appose tissue are low, the tissue inside the loop of suture remain viable, and inflammation caused by the tissue division and reapproximation incites the normal cascade of wound healing. Unimpeded biologic healing should be expected to regain 70% of the native tissue strength.[1] The long-term strength of the closure in these situations is the physical strength of the suture construct and the strength of the healed biologic tissues. The tensile strength of the physical construct is directly related to the microencapsulation of suture and mesh filaments as a foreign body response. Failure of the physical construct is due to tearing of the filaments away from its encapsulation scar. The ultimate tensile strength of the biologic healing is from scar tissue that joins and unifies the gap between the divided tissues. Therefore, the total strength of any repair is a complex mixture of scar between living tissues and around foreign bodies. High tension closures (such as in abdominal wall surgery and orthopedics) may be different from other situations in surgery, because the forces required for tissue approximation create tissue pressure that in all likelihood can exceed capillary perfusion pressure, causing necrosis and creating a micropressure sore within suture loops. This area of necrosis turns into scar[2] and has the potential to remodel, stretch, and thin over time in response to physical stresses. Visually, this effect is the commonly recognized phenomenon of suture pull-through, as the sutures can be seen to cheese wire through the intact abdominal wall (▶ Fig. 41.4).

The three types of abdominal wall closures (suture unsupported repair, direct supported repair, and spanning mesh repair) have markedly different concepts regarding the healing of high tension approximations. A suture repair of an abdominal wall defect regards the biologic healing and physical strength of sutures to be acceptable to resist the forces at the STI. It is well established that suture repairs trade a higher rate of long-term failure of these high tension closures for decreased local wound issues of mesh implantation.[3] Direct supported repairs attempt to reduce the forces at the STI by increasing the surface area of foreign material and encouraging microencapsulation scar. Proponents of direct supported repairs find the benefits of a stronger physical repair worth the downsides of mesh use and more tension at the closure site due to the reapproximation of divided tissue. Finally, spanning meshes used for abdominal wall defects rely solely on the physical strength of the repair, as there is no attempt to join the abdominal wall at the defect. The abdominal wall muscles are not manipulated and moved, thereby keeping low the tension at the closure (the so-called tension-free repair). The abdominal wall muscles are shortened and not at their optimal lengths, causing them to be at a weaker, less contractile point on their Starling curves. A muscle intentionally left with sarcomere overlap at the microscopic level would not generate as much force against the spanning mesh, with the thought that this lower tension state

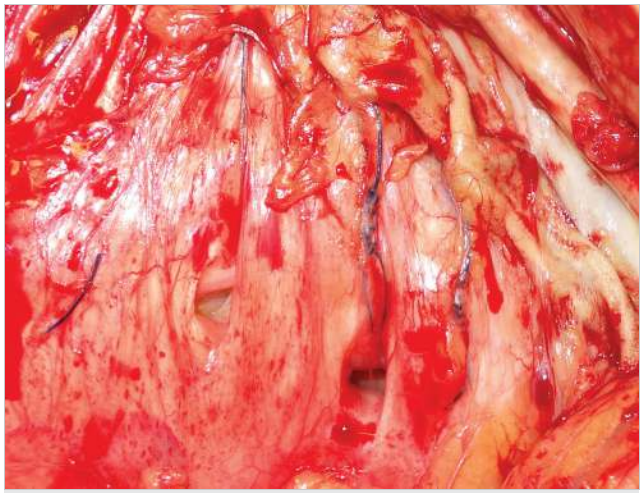


Fig. 41.4 Suture pull-through example.

would lead to a longer-lasting hernia repair. Having presented these three viewpoints, there is general consensus that suture repairs should only be attempted for defects 2 to 3 cm in size. For the two remaining options, it has been shown that direct supported repairs have lower recurrence rates than bridged repairs in open surgery,[4] and therefore will be the focus of this chapter. The efficacy of laparoscopic-bridged repairs is touted by several authors. Such repair is most often done by general surgeons without the input of plastic surgeons. Therefore, this is outside the scope of this review.

41.4.2 Surgical Procedure for Incisional Hernias Based on Skin Vascularity and Force Distribution

Abdominal Skin Vascularity

The dominant blood supply to the abdominal wall comes from perforating blood vessels that emerge through the rectus abdominis muscle and supply the central abdominal skin. In rough terms, below the umbilicus the source of these perforators is the deep inferior epigastric artery and above the umbilicus is the superior epigastric artery. These perforators exist in medial and lateral rows and are located in the periumbilical area. In the inferolateral abdominal skin, the vascular supply is from the superficial inferior epigastric system, and over the external oblique in the upper and mid-abdomen the sources of blood are lateral intercostal perforators. Abdominal skin flap elevation is a commonly performed surgical maneuver, but is not without risk of necrotic tissue loss, as it requires adjacent angiosomes to supply the tissue whose primary perforating vessel was divided.

A working knowledge of abdominal skin blood flow is important in the performance of hernia surgery, as skin flap elevation is a necessary requirement for exposure and manipulation of the abdominal musculature. Old skin incisions interrupt the dermal circulation, and so one should assume that blood *does not* cross a scar. With new skin flap elevation and loss of a primary perforating vessel, the blood flow becomes less pulsatile and more laminar. The choke vessels dampen pulsatility and there is overall lower arteriolar pressure. Tissue perfusion, based on the difference between arterial and venous pressures, is also decreased because outflow is reduced due to choke vessels on the venous side. Decreased total blood flow that lacks pulsatility has been shown experimentally and clinically to have more unreliable healing than when tissue is oxygenated with pulsatile blood flow.[5,6,7] Preservation of perforators will maintain skin vascularity and be a significant aid in healing. Scarred soft-tissue vascular beds only have laminar flow, and therefore should be removed whenever possible.

Force Distribution

If the forces on the closure are greater than the sum of the strength of the physical construct of closure and the strength of biologic healing, then the scar will remodel and widen. The early separation of laparotomy closures as a predictor of eventual incisional hernia formation was demonstrated by Pollock and Evans and later confirmed by Burger et al.[8,9,10] Force distribution is a concept that focuses on lowering the forces experienced at each STI. The lower the STI at each suture and the more evenly distributed the forces for closure, the higher the success of closure. Alternatively, the greater the episodic forces experienced at the STI, the more the sutures will pull through the tissue—the central cause of hernia recurrence.[11,12,13] While the *total* force of closure in *direct supported repairs* will be greater than the forces experienced by bridging meshes, the forces at the STI are lowered in two separate ways. First, the total force of closure is divided by a greater number of sutures, thereby lowering the force at each individual STI. Second, the long-term strength of the repair is achieved both with the physical construct of mesh and the biologic healing of the overlying abdominal wall.

A compliant abdominal wall absorbs energy with coughs, the Valsalva maneuver, and forceful movements, rather than sending energy waves to the STI. Patients with weight loss, treated ascites, and a history of multiple large gestations are all known to have compliant abdominal walls and relative success with closure. Patients with noncompliant abdomens include those patients with prior intra-abdominal sepsis and scarring, a history of radiation therapy, lateral scarring from prior incisions, and patients at their maximal weight. The state of a hernia itself causes an abdominal wall to become less compliant.[14] While many view a components release as a means to bring vascularized tissue to the midline for closure, an alternative conceptualization is to regard the release of the external oblique muscle as a means to improve lateral abdominal wall compliance and thereby protect the midline sutures.

It is often remarked that hernias tend to occur at the end of the repair, and this too can be explained as a function of the STI. The best hernia repairs have smooth transitions between repaired abdominal wall and intact fascia, with a "matching" of compliances. Mismatched compliances will cause high tension at the STI and suture pull-through. Anchoring mesh to noncompliant rib or symphysis pubis in particular leads to areas of imbalance of STI forces and early failure. Another situation where compliance mismatches (and increased chance for failure) exist is when a direct supported repair for a midline incisional hernia ends adjacent to significant rectus diastasis. It is for this reason that recurrences often occur at the superior and inferior borders of mesh. In general, a longer repair with properly balanced forces at each STI (working out the "dog-ears" of the abdominal wall closure) is optimal for long-term success and is preferable to a short repair. *This is opposite to the common belief that one should leave alone stretched but otherwise intact abdominal wall.*

Surgery Technique: Concepts of Components Separation

The primary principle guiding abdominal wall closure is the reduction and minimization of tension. Ramirez et al described a procedure of "components separation" with the goal of spanning large defects in the abdominal wall.[15] The procedure separates the muscular elements of the abdominal wall along natural fascial planes, by incising the external oblique fascia just lateral to the semilunar line which is its fusion with the anterior rectus sheath. The external oblique is dissected away from the internal oblique within the normal avascular plane to the level of the perforating vasculature along the level of the posterior

axillary line, creating a rectus abdominis/internal oblique flap. By doing this bilaterally, gaps of up to 8, 20, and 10 cm can be closed in the epigastrium, mid-abdomen, and hypogastrium, respectively. Additionally, posterior rectus sheath flaps can also be created by incising the sheath laterally and creating a turnover flap toward the midline, if further tissue is required. This last maneuver is not usually done currently by most surgeons due to worries of further weakening the rectus to rectus opposition.

Surgical Technique: Components Release with Perforator Preservation and Force Distribution with Mesh

After a wide skin preparation and under general anesthesia, the previous midline incision is opened and extended both superiorly and inferiorly. The hernia sac is identified and the abdominal cavity entered. Intestinal adhesions to the hernia sac and the posterior aspect of the abdominal wall are widely taken down. When appropriate, no further bowel manipulation or enterolysis is performed in order to lessen postoperative visceral swelling. The anterior rectus fascia is cleared for a distance of 4 cm from its medial border. This requires incision of the hernia sac with cautery, and the division of any medial rectus abdominis perforators. This maneuver will not interrupt any lateral rectus perforators, nor will it reach the semilunar line. At this point, a decision about mesh location is made. Typically, for patients with normal abdominal wall compliance by history and physical examination, a hernia in which the medial aspect of the rectus muscles are separated by less than 6 cm by CT scan can be closed with a direct supported repair using a retro-rectus mesh and without a components release. Between 6 and 14 to 16 cm, a component separation is usually needed to achieve a direct supported repair with a retro-rectus mesh. Over 14 to 16 cm, the author typically uses a components release with an intra-abdominal mesh to achieve fascial closure.

In order to release the external oblique muscle and fascia and to maintain skin vascularity, a transverse incision is made at the inferior costal margin about 6 to 8 cm in length. Dissection to the semilunar lines is performed, and the end of the external oblique muscle is visualized as it turns into the anterior rectus fascia. With blunt dissection using the end of a narrow Deaver retractor both superiorly onto the chest and inferiorly in the direction of the anterior superior iliac spine (ASIS), the semilunar lines are exposed. Using cautery and under direct vision, the external oblique muscle and fascia are divided to create a myofascial rectus abdominis flap. It is important to release high onto the ribs in order to take tension off the superior-most rectus muscle during closure. In the inferior direction, standard instruments and lighting permit the division of the external oblique muscle and fascia to the ASIS. Through the transverse incision, the external oblique is bluntly elevated off of the internal oblique to the level of the mid-axillary line to improve abdominal compliance and to allow for medial movement of the rectus abdominis muscle.

It is important to complete the fascial release all the way to the level of the symphysis pubis so that the rectus muscle is not tethered as it is moved toward the midline. A blunt tunnel is created from the lower aspect of the midline incision in the suprapubic area toward the ASIS and is brought into continuity with the tunnel created from the upper lateral incision. The cut end of the external oblique muscle, in the shape of the letter "V," is captured by a dissecting index finger and pulled into the midline incision where it is divided under direct vision. The tunnels described preserve the soft-tissue attachments between the skin and the rectus muscle, thereby protecting skin vascularity in this perforator-preserving operation. Perforators are not individually skeletonized, thereby simplifying and speeding up the release that takes about 5 minutes on each side to perform.

The perforator-preserving technique described earlier is preferable to the "standard" component separation method in which the skin flaps are elevated off the rectus fascia to the semilunar line. The external oblique fascia is then divided and the rectus flap created. This method divides medial and lateral rectus perforators and leaves only the intercostal perforators and superficial inferior epigastric artery. As described earlier, this creates a high risk for devascularization of the skin flaps with subsequent necrosis and may contribute to seromas and postoperative infections.

When a retro-rectus mesh is placed, the space between the posterior fascia and the rectus muscle is opened for about 4 cm, avoiding the deep inferior epigastric artery, perforators to the posterior fascia, and lateral intercostal segmental nerves. The posterior fascia is closed with a running 2–0 monofilament suture to exclude the bowel (▶ Fig. 41.5). A piece of macroporous monofilament uncoated polypropylene mesh is cut to be 7.5 cm wide and the length of the closure. Bioprosthetic meshes have not been shown to improve outcomes and are not commonly used.[16] 0-Polypropylene sutures are passed through the anterior rectus fascia and rectus muscle, 4 cm lateral to the medial aspect of the rectus muscle and spaced 2 to 3 cm apart. The mesh is inserted flat and tight, with gradual loading of the abdominal wall musculature to achieve closure (▶ Fig. 41.6, ▶ Fig. 41.7, ▶ Fig. 41.8). Using this simple geometric formula, the mesh will be inset under tension and without wrinkles. Mesh wrinkles can create pressure sores into the bowel or the overlying tissues, permit fluid collections, and possibly add to difficulty for later surgical dissections. The medial aspect of the rectus muscles is closed over the mesh to achieve a direct supported repair (▶ Fig. 41.8). For intra-abdominal mesh, an identical technique is performed after mobilization of the falciform ligament and preperitoneal fat. Important to this force distribution technique that routinely requires high tension for closure is the number of sutures. Up to 30 sutures are used for a full abdominal wall closure from xiphoid to symphysis pubis. Therefore, the total force of closure is divided by the number of trans-rectus sutures to decrease the forces experienced at each STI. Done in this manner, while the total tension on the midline may initially seem too tight, clinically the forces at the STI are below the point for suture pull-through.

For truly massive hernias with the rectus muscles separated more than 15 to 20 cm in transverse dimension and clinically with a loss of domain, caution should be used before attempting repair. There are no good methods to accurately predict who will have respiratory difficulties postoperatively and who will require a bridging mesh. At Northwestern, patients with respiratory difficulties on oxygen or with prior tracheostomies for respiratory failure are not offered hernia repair surgery. It is our goal to always achieve a direct supported repair, and so patients

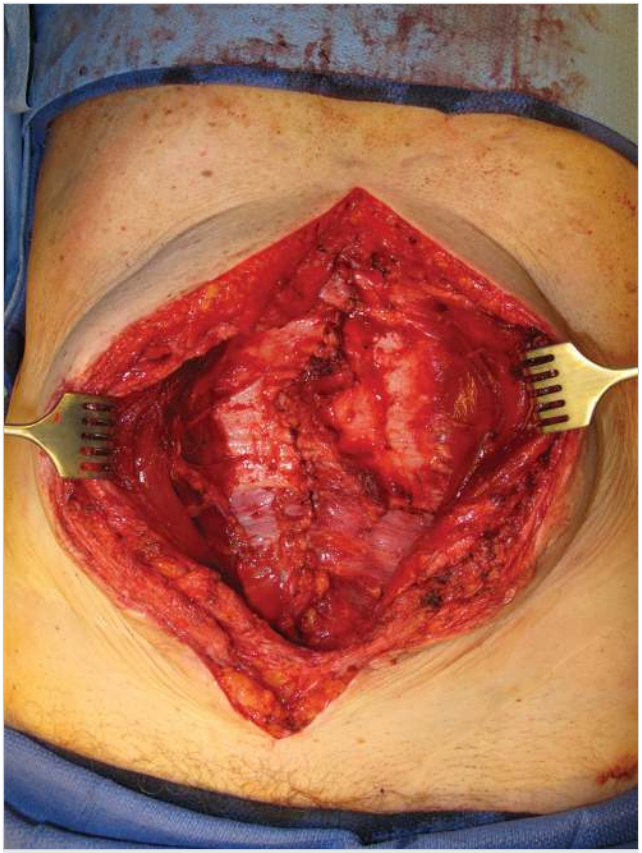


Fig. 41.5 The retro-rectus space has been developed and the posterior sheath approximated to close the defect. Retractors elevate the medial aspect of the rectus muscles.

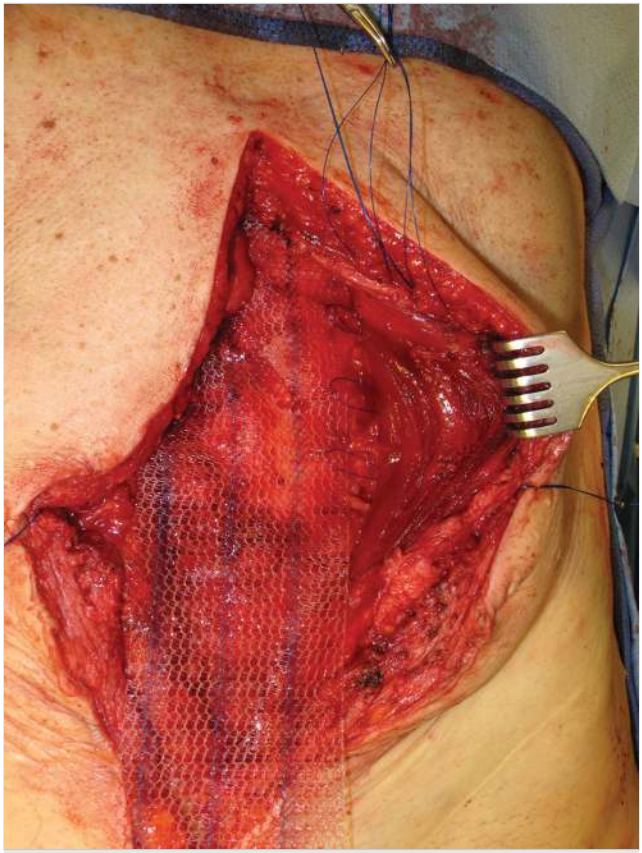


Fig. 41.6 A 7.5-cm mesh is sewn to the underside of the rectus muscles, with sutures placed 4 cm from the free muscle edge.

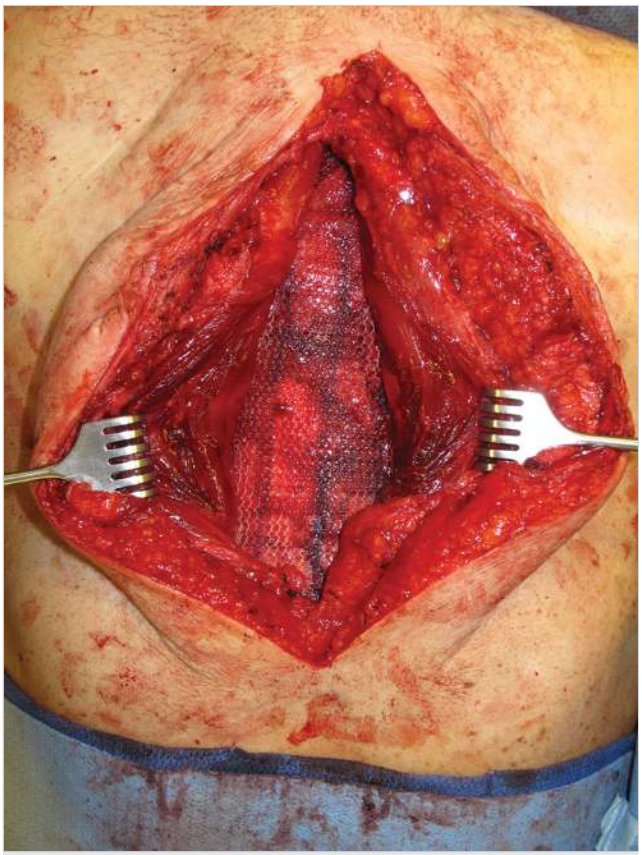


Fig. 41.7 Mesh placed flat and tight in the retro-rectus space.

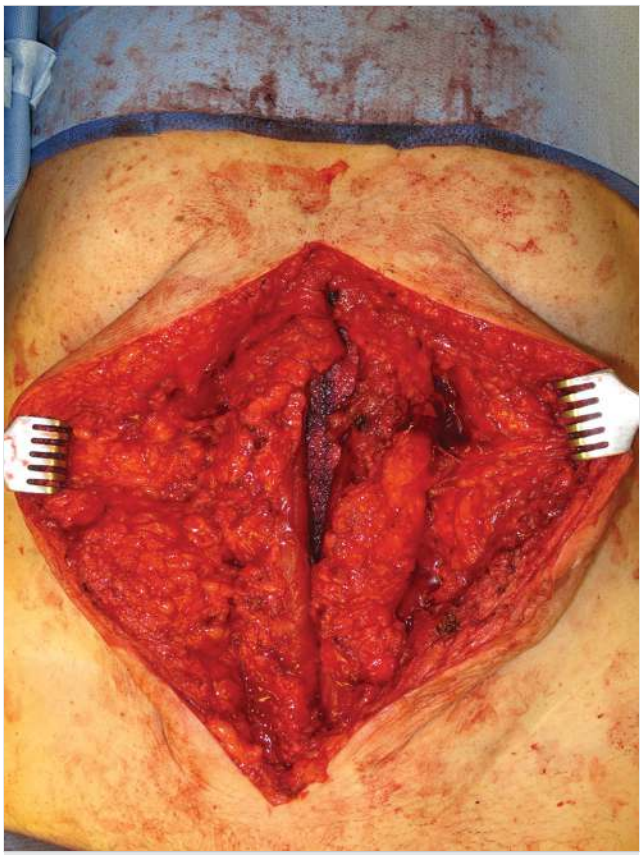


Fig. 41.8 The medial aspect of the rectus muscles is closed over the mesh to achieve a direct supported repair.

Fig. 41.9 Excess skin including old colostomy site excised as vertical panniculectomy.

with large hernias will typically receive an intra-abdominal mesh, as the tissues will accept a higher degree of force without suture tearing. The outcome of these patients using a narrow flat mesh has been favorable with low fistula and hernia recurrence rates.[17,18] More recently, strips of mesh used as sutures are used to close these wider defects without the risk of polypropylene causing adhesions to bowel.[19]

Medialization of the rectus muscles and attached overlying skin by definition cause skin and subcutaneous tissue to become redundant. Routine resection of this tissue as a vertical panniculectomy will serve to cleanse potentially contaminated skin edges, will remove scar and skin without pulsatile blood flow, and decrease potential dead space (▶ Fig. 41.9). Drains or quilting sutures are important to further handle these dead spaces.

Further techniques are required for closure of patients with large complex abdominal wall defects, inclusive of skin/soft tissue loss and/or loss of domain. Tissue expanders may be required for creation of sufficient skin to allow closure and may also facilitate restoration of intestinal domain. Regional/distal flaps from the thigh may also be required, such as the tensor fascia lata flap, rectus femoris flap, anterolateral thigh flap, and vastus lateralis flap. If skin and fascia are required, the most reliable means is via use of the anterolateral thigh flap, sometimes in combination with the tensor fascia lata or vastus lateralis (▶ Fig. 41.10).

41.4.3 Postoperative Care

The patients are given predominantly narcotics for pain control. No nasogastric tubes are used. Clear liquids are started upon the return of bowel function, typically during the third or fourth postoperative day. Binders are used to compress the soft tissues back to the abdominal wall. Antibiotics are given for 24 hours. Unfractionated heparin is given for the time in hospital and occasionally in the early postoperative period at home to limit deep vein thrombosis. Drains are left in place until they are under 30 mL a day. The patients leave 5 to 6 days after surgery only on oral analgesics. The patients are encouraged to walk as tolerated immediately. Nonimpact exercise can commence after 4 to 6 weeks. Isometric core exercise training can begin at 3 months. Light impact exercise is permitted at 6 months, and full exercise is allowed at 12 months.

41.5 Outcomes

There are two critical components to this midline hernia repair. A narrow mesh placed in the retro-rectus position with multiple sutures to distribute forces acts as a pledget to decrease forces at each individual STI thereby limiting suture pull-through. The large number of sutures essentially quilts the mesh to the abdominal wall—following a well-established principle that a well-fixed foreign body does not become infected. Reliable healing is demonstrated in both published and unpublished data from our institution of the reproducibility of this technique. The mesh is kept narrow to resist wrinkling at its edges, to limit the total amount of foreign material, and to avoid unnecessary elevation of wide tissue flaps. As a side benefit, keeping the sutures nearer to the midline also avoids larger segmental intercostal nerves. The Rives-Stoppa repair with large meshes has a 27% chronic pain rate, an issue we believe is avoided with these narrow meshes.[20] Many surgeons who use large meshes avoid suture fixation for reasons of utility, difficulty with suture placement, and avoidance of larger segmental nerves. Downsides to this strategy of using giant meshes include opening much larger tissue planes permitting fluid to collect, the development of mesh wrinkles at the outer edges when trying to fit a flat mesh to a curved surface, and a poorly stabilized mesh that may be slow to incorporate. Finally, large meshes will change overall abdominal wall compliance, leading to lateral stiffness and some measure of discomfort. A narrow mesh designed only to stiffen the midline leaves the lateral wall compliant and protective from the phenomenon of pull-through.

Perforator preservation has been shown to decrease local wound complications in two different reports in the literature in comparison to open component repairs with standard skin undermining. Surgical site infection (SSI) for open procedures has been consistently reported in the 25% range. The report of Dumanian and Saulis in 2002 was the first to directly compare wound complication rates in component procedures with and without perforator preservation.[21] Local wound complications decreased from a baseline of 20% down to 2% with the improved soft tissue vascularity. Butler in 2011 with a similar conceptualization of perforator preservation documented a reduction in local wound complications from 32 to 14%.[22]

Fig. 41.10 **(a)** Patient after resection/takedown of enterocutaneous fistula. Both rectus muscle and overlying skin have been subtotally resected. The large defect was bridged with underlay porcine acellular dermal matrix in preparation for regional flap reconstruction. **(b)** A composite left anterolateral thigh flap and vastus lateralis flap are mobilized on their shared pedicle for reconstruction. **(c)** The flap has been inset providing autologous fascial closure without tension due to the inlay biologic mesh. **(d)** Eight weeks after closure showing abdominal wall integrity with some minor peripheral skin necrosis. **(e)** Healing donor site, which was skin grafted.

Patients with incisional hernias repaired at the Northwestern Feinberg School of Medicine with force distribution techniques that focus on the STI and tissue vascularity have been reviewed. One hundred consecutive retro-rectus repairs, performed between 2010 and 2014 in patients with an average BMI of 29 and with a Ventral Hernia Working Group breakdown of 49:37:13:1 for levels 1 to 4, respectively, were recently reviewed. There were no mesh infections or removals, and no known hernia recurrences to date with a 15-month follow-up. The SSI rate was 2%, the SSO rate was 3%, and the only return to the operating room was for a hematoma. Thirty-three percent of the patients had a prior or simultaneous components release.

41.6 Review Questions

41.6.1 True or False

1. One year after abdominal wall incision and closure, the abdominal wall will regain 90% of its original strength.
2. Rectus diastasis is performed only for cosmetic and not functional issues.

41.6.2 Choose the Best Answer

3. Optimal treatment of a 3-cm umbilical hernia defect would involve
   a) Suture repair.
   b) Mesh repair.
   c) Repair at the time of abdominoplasty and treatment of rectus diastasis.
   d) All of the above.
4. These blood vessels all supply blood to the anterior abdominal wall except
   a) Deep inferior epigastric perforators.
   b) Superficial inferior epigastric perforators.
   c) Superior epigastric perforators.
   d) External oblique perforators.
   e) Thoracodorsal perforators.
5. Optimal closure of abdominal wall defects involve
   a) Direct repair with suture only.
   b) Direct supported repair.
   c) Tension-free (spanning mesh) repair.

41.6.3 Answers

1. False. 70%.
2. False. Rectus diastasis results in decreased core pressure and thereby is associated with the development of back pain.
3. d. All of the above.
4. e. Thoracodorsal perforators.
5. b. Direct supported repair.

References

[1] Hollinsky C, Sandberg S. Measurement of the tensile strength of the ventral abdominal wall in comparison with scar tissue. Clin Biomech (Bristol, Avon). 22(1):88–92

[2] Klink CD, Binnebösel M, Alizai HP, et al. Tension of knotted surgical sutures shows tissue specific rapid loss in a rodent model. BMC Surg. 11:36–45

[3] Nguyen MT, Berger RL, Hicks SC, et al. Comparison of outcomes of synthetic mesh vs suture repair of elective primary ventral herniorrhaphy: a systematic review and meta-analysis. JAMA Surg. 149(5):415–421

[4] Booth JH, Garvey PB, Baumann DP, et al. Primary fascial closure with mesh reinforcement is superior to bridged mesh repair for abdominal wall reconstruction. J Am Coll Surg. 217(6):999–1009

[5] Gibbons GW, Wheelock FC , Jr, Hoar CS , Jr, Rowbotham JL, Siembieda C. Predicting success of forefoot amputations in diabetics by noninvasive testing. Arch Surg. 114(9):1034–1036

[6] Chang N, Mathes SJ. Comparison of the effect of bacterial inoculation in musculocutaneous and random-pattern flaps. Plast Reconstr Surg. 70(1):1–10

[7] Feng LF, Price D, Hohn D, Mathes SJ. Blood flow changes and leukocyte mobilization in infection: a comparison between ischemia and well-perfused skin. Surg Forum. 34:603

[8] Playforth MJ, Sauven PD, Evans M, Pollock AV. The prediction of incisional hernias by radio-opaque markers. Ann R Coll Surg Engl. 68(2):82–84

[9] Pollock AV, Evans M. Early prediction of late incisional hernias. Br J Surg. 76 (9):953–954

[10] Burger JW, Lange JF, Halm JA, Kleinrensink G-J, Jeekel H. Incisional hernia: early complication of abdominal surgery. World J Surg. 29(12):1608–1613

[11] Alexander HC, Prudden JF. The causes of abdominal wound disruption. Surg Gynecol Obstet. 122(6):1223–1229

[12] Rodeheaver GT, Nesbit WS, Edlich RF. Novafil. A dynamic suture for wound closure. Ann Surg. 204(2):193–199

[13] Israelsson LA, Millbourn D. Closing midline abdominal incisions. Langenbecks Arch Surg. 397(8):1201–1207

[14] DuBay DA, Choi W, Urbanchek MG, et al. Incisional herniation induces decreased abdominal wall compliance via oblique muscle atrophy and fibrosis. Ann Surg. 245(1):140–146

[15] Ramirez OM, Ruas E, Dellon AL. Components separation method for closure of abdominal-wall defects: An anatomic and clinical study. Plast Reconstr Surg. 1990;86:519–526

[16] Purnell CA, Souza JM, Park E, Dumanian GA. Repair of recurrent hernia after biologic mesh failure in abdominal wall reconstruction. Am J Surg. 208(5): 788–793

[17] Souza JM, Dumanian GA. An evidence-based approach to abdominal wall reconstruction. Plast Reconstr Surg. 130(1):116–124

[18] Souza JM, Dumanian GA. Routine use of bioprosthetic mesh is not necessary: a retrospective review of 100 consecutive cases of intra-abdominal midweight polypropylene mesh for ventral hernia repair. Surgery. 153(3):393–399

[19] Dumanian GA, Lanier ST, Souza JM, Young MW, Boller AM, Mueller KH, Halverson AL, McGee MF, Stulberg JJ. Mesh sutured repairs of contaminated incisional hernias. Am J Surg. 216: 267-273, 2018. PMID 29108644

[20] Iqbal CW, Pham TH, Joseph A, Mai J, Thompson GB, Sarr MG. Long-term outcome of 254 complex incisional hernia repairs using the modified Rives-Stoppa technique. World J Surg. 31(12):2398–2404

[21] Saulis AS, Dumanian GA. Periumbilical rectus abdominis perforator preservation significantly reduces superficial wound complications in “separation of parts” hernia repairs. Plast Reconstr Surg. 109(7):2275–2280, discussion 2281–2282

[22] Ghali S, Turza KC, Baumann DP, Butler CE. Minimally invasive component separation results in fewer wound-healing complications than open component separation for large ventral hernia repairs. J Am Coll Surg. 214(6):981–989

42 Lower Extremity Reconstruction

Mark M. Leyngold

Abstract

This chapter covers the spectrum of issues and challenges involved in the surgical repair of lower extremity wounds and fractures. The goals of treatment are listed (debride devitalized tissue and obtain healthy wound bed; restore stability, structure, vascularity, and function; etc.), and the sequential procedure for treatment is outlined for both bony and soft-tissue reconstruction. Each area of the lower extremity (thigh, knee, etc.) is covered, and treatment options are enumerated. An important discussion of chronic wounds of the lower extremity is included, pointing out the reasons why a good clinical outcome is difficult to achieve. The author concludes the chapter offering guidance on postoperative care and looking at possible outcomes, good and bad.

Keywords: Gustilo classification, Byrd classification, posterior tibial nerve, split-thickness skin graft (STSG)

42.1 Goals and Objectives

- Understand the proper evaluation, management, and reconstructive options of acute and chronic lower extremity wounds.
- Clearly define the indications for lower extremity reconstruction and be able to choose an appropriate reconstructive option based on the location, size, extent, and etiology of the defect.
- Understand appropriate postoperative care, cost, and outcomes in patients with complex lower extremity wounds.
- Know the evidence-based timing for free flap lower extremity traumatic wound reconstruction to maximize patient safety and quality outcomes.

42.2 Patient Presentation

Patients with lower extremity wounds requiring reconstruction present many challenges to a plastic surgeon, and, thus, have to be addressed methodically. Treatment approaches vary according to the etiology of the wound and patient factors. Lower extremity wounds can be a result of trauma or open fractures, postsurgical dehiscence, compartment syndrome, tumor, radiation damage, vascular insufficiency, and diabetes. Goals of reconstruction should be (1) to debride devitalized tissue and obtain healthy wound bed; (2) restore stability, structure, vascularity, and function; (3) obliterate dead space; (4) provide durable coverage of vital structures; and (5) provide a quality aesthetic result.[1] Evaluation of any trauma patient with a lower extremity wound should always begin with ABCs and evaluation of associated injuries and comorbidities; assessment of wound size, depth, and exposure of vital structures; vascular examination (palpable pulses); neurologic examination; and evaluation of radiographs.

Open tibia fractures are classified based on the severity of bony injury and soft-tissue damage (▶ Fig. 42.1).

The most popular classification of open fractures is by Gustilo and Anderson (▶ Table 42.1).[2]

42.3 Preparation for Surgery and Prognostic Factors

Physicians who treat lower extremity trauma would like to have a reliable way to predict prognosis for each patient. The most important early decision to be made in the event of severe leg trauma is whether to reconstruct or to perform early amputation. Orthopedic and plastic surgeons generally agree that

Fig. 42.1 Patient with chronic Nocardia infection of proximal leg and knee. (Courtesy of Bruce A. Mast, MD.)

Table 42.1 Gustilo classification of open fractures of the tibia

Type	Description
I	Wound < 1 cm with minimal soft-tissue injury
II	Wound is > 1 cm with moderate soft-tissue injury
IIIA	Extensive soft-tissue damage with adequate soft-tissue coverage
IIIB	Extensive soft-tissue loss with periosteal stripping and bony exposure
IIIC	Extensive soft-tissue loss with vascular injury

Source: Adapted from Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. J Bone Joint Surg Am. 1976; 58(4):453–458[2]

Table 42.2 Byrd classification of lower extremity trauma

Type	Criteria
I	Low-energy forces causing a spiral or oblique fracture pattern with skin lacerations < 2 cm and a relatively clean wound
II	Moderate-energy forces causing a comminuted or displaced fracture pattern with skin laceration > 2 cm and a moderate adjacent skin and muscle contusion but *without* devitalized muscle
III	High-energy forces causing a significantly displaced fracture pattern with severe comminution, segmental fracture, or bone defect with extensive associated skin loss and devitalized muscle
IV	Fracture pattern as in type III but with extreme-energy forces as in high-velocity gunshot or shot-gun wounds, a history of crush or degloving, or associated vascular injury requiring repair

Source: Adapted from Byrd HS, Spicer TE, Cierney G, III. Management of open tibial fractures. Plast Reconstr Surg. 1985; 76(5):719–730[5]

many lower extremity injuries are best served by reconstruction; others are candidates for primary amputation. Delayed amputation is considered a treatment failure, as this outcome suggests possible judgment error during the initial management. Delayed amputation has been linked to increased hospital course, increased risk of sepsis, more operations, and increased disability. Multiple scoring systems including Lower Extremity Assessment Project (LEAP) have been designed to compare outcomes of patients with severe lower extremity trauma.[3] Unfortunately, these scoring systems do not reliably predict which injured limbs should undergo primary amputation. Most would agree that absolute indications for lower extremity amputation are complete disruption of posterior tibial nerve in adults and crush injuries with warm ischemia time greater than 6 hours. Amputation should also be considered if there is serious associated polytrauma, severe ipsilateral foot trauma, failed vascular reconstruction with two or more injured tibial vessels, and anticipated protracted course to obtain soft-tissue coverage and tibial reconstitution.[1] Keller reviewed 10,000 tibial shaft fractures and found that the risk of systemic complications is increased in the presence of the following: comminution, displacement, bone loss, distraction, soft-tissue injury, infection, and polytrauma.[4]

42.4 Treatment

Treatment of lower extremity trauma should be done in the following order:

1. Stabilization of fracture (usually external fixator).
2. Restoration of inflow, if required.
3. Four compartment fasciotomies, if required.
4. Debridement and washout of the wound.
   a) If major blood vessel exposure is present then immediate soft-tissue coverage (to avoid blowout).
   b) If no exposed vital structures, then repeat scheduled debridement.

The most important aspect of treatment is thorough debridement. It should be repeated if required to ensure clean and noncontaminated wound.[1]

42.4.1 Timing of Reconstruction

According to Byrd et al, radical bone and soft-tissue debridement with flap coverage should be done in the first 5 to 6 days after injury for type III and IV fractures.[5] A complication rate for Byrd type III wounds averaged 18% during that time frame (▶ Table 42.2). Fractures not treated by early muscle flaps predictably entered a colonized *subacute phase* that extended from 1 to 6 weeks postinjury. Complications after treatment with flaps during this phase averaged 50%. Approximately after 4 to 6 weeks untreated severe injuries enter a *chronic phase* characterized by a granulating wound, adherent soft-tissue, and decreasing areas of infection. After soft-tissue coverage, the complication rate for this chronic group was 40%.[5]

Yaremchuk et al reviewed patients with flap coverage at an average of 17 days after injury with infection rate of only 14%. The key difference was aggressive debridement and complete removal of all bone fragments.[6] According to Godina, free flaps for lower extremity reconstruction performed within the first 75 hours have a failure rate of only 0.75%, postoperative infection rate of 1.5%, and time to union of 6 to 8 months. Consistent with Byrd et al's findings, Godina found that free flaps performed between day 3 and 3 months and flaps performed between 3 months and 12 years had a failure rate of 12 and 9.5%, respectively. Time to union and infection rate were also significantly increased in those groups.[7]

42.4.2 Bony Reconstruction

One of the priorities in patients with open tibial wounds is bony stabilization. Options for stabilizing reduced fractures include plaster immobilization enclosing an open wound; internal fixation with plates, rods, and screws; and external fixation. When a bone gap is present, reconstruction becomes more complicated. Nonvascularized bone grafting can be a good option for smaller bony gaps. Some authors state that cancellous bone grafts can be used for bony defects up to 10 cm. The graft has to be placed under a well-vascularized flap. Intact fibula facilitates bone grafting of longer defects by acting as a strut to keep the extremity at length. When the fibula is not intact, which is quite frequent occurrence in these high-energy fractures, other methods of bony reconstruction should be employed if the defect is greater than 8 cm.

Free osseous or osteocutaneous flap transfer may be required when the bone gap is long. Most commonly free fibula, iliac crest, or scapula is used for bony reconstruction. Weiland et al concluded that vascularized bone grafts are indicated for segmental defects larger than 6 cm.[8] Another useful technique that may be employed when a large bone gap is encountered (generally > 10 cm) is distraction osteogenesis. Fractured ends of the bone are radically debrided and transaction of the cortical bone is made proximally outside of the zone of injury, leaving the medullary bone intact followed by Ilizarov pins near bone ends on either side of the gap and distraction apparatus. After a 7-day waiting period, distraction at 1 mm/day begins until defect is spanned. The frame is usually kept on for about 1 year. Advantage of this method is that the amount of bone generated is anatomically correct for the size of the defect, soft-tissue defects may be closed spontaneously by simultaneous lengthening during the same process and blood transfusions are usually not required. Despite the advantages, this is a very difficult process; thus, patient cooperation and compliance are keys to success. Potential complications may include pin-tract infections, stiffness of adjacent joints, and severe pain.[9]

42.4.3 Soft-Tissue Reconstruction

When it comes to soft-tissue reconstruction, the goals are stable wound coverage, acceptable appearance, and minimal donor-site morbidity. Principles used in many other aspects of plastic surgery also apply in lower extremity reconstruction. They include adequate and possibly serial wound debridements, infection control, with recipient vessels and flaps outside of the zone of injury.

It is useful to think of lower extremity wounds based on the level of injury. For the purposes of reconstruction, defects can be divided into thigh wounds, wounds involving upper third of, middle third, and lower third of the leg respectively. Foot reconstruction can be separated into dorsal and plantar defects as the coverage options may vary between the two (▶ Table 42.3).

Generally, most authors would agree that the majority of thigh wounds can be treated with negative pressure therapy followed by a split-thickness skin graft (STSG) or Integra placement. There are generally large amounts of muscle tissue that can be advanced locally into the wound. Flaps that are available for coverage include tensor fascia lata, gracilis, rectus femoris, vastus lateralis, and biceps muscle flaps.

Medial head of gastrocnemius is a workhorse flap for upper third of the leg defects and the knee. It has a broad muscle belly; single, proximal neurovascular pedicle; and no residual functional deficit. To obtain greater muscular coverage of larger surface area defects, it is useful to score the fascia perpendicular to gastrocnemius muscle fibers (▶ Fig. 42.2, ▶ Fig. 42.3, ▶ Fig. 42.4). Other less common options include lateral head of gastrocnemius (generally smaller), proximally based soleus, or bipedicled tibialis anterior. Tibialis anterior is extremely important for dorsiflexion of the foot and should not be entirely sacrificed. Muscle function is generally preserved when raised as a bipedicled flap. Transfer requires detaching dense anterior tibial connections. One can use muscle-splitting approach ensuring no residual functional deficit.

Middle third of the leg shares similar flap armamentarium compared to proximal third with few differences. Common options in order of preference include proximally based soleus, medial head of gastrocnemius, lateral head of gastrocnemius, flexor digitorum longus, tibialis anterior, extensor digitorum longus, and flexor hallucis longus (FHL). Loss of toe extension and great toe drop results if entire Extensor Digitorum Longus (EDL) and Extensor Digitorum Hallucis (EDH) are used; thus, they should not be the primary flap of choice. Harvest of FHL may result in weakening of the great toe; so, it would not be the best choice in athletes or very active patients. EDH and FHL are mainly useful for lower portions of the middle third and small defects.

Soft-tissue coverage of the lower third of the leg presents many challenges to the reconstructive plastic surgeon. Free flap is generally considered the gold standard for the distal third reconstruction. Local flaps may be an option but generally have limited use. Reverse turndown sural artery flap may be a good option in some patients. It includes the sural nerve and lesser saphenous vein in the flap (▶ Fig. 42.4, ▶ Fig. 42.5).[10] It is important to avoid compression of the pedicle postoperatively, as this flap is notorious for local flap necrosis reported to be up to 21% in some studies. It may also cause a bulge and present less than desired appearance. It may be wise to delay the flap to improve its venous drainage and decrease the rate of flap necrosis. Cross leg flap can be used if free flap is not an option. Cross leg flap is transferred as fasciocutaneous tissue with length/width ratio of 3:1 or 4:1. The blood supply can be axial based on posterior descending subfascial cutaneous branch of popliteal artery. These flaps are notorious for complications including 40% local flap necrosis, infection up to 28%, and potential for significant leg stiffness especially in elderly patients due to prolonged immobilization. As such this flap is rarely used.

Table 42.3 Soft-tissue reconstruction of lower extremity

Level of injury	Reconstructive options
Thigh	**STSG (most commonly),** flap options: TFL, gracilis, rectus femoris, vastus lateralis, ALT, biceps muscle flaps, free flaps
Knee	**Medial head of gastrocnemius (gold standard),** lateral head of gastrocnemius, reverse ALT (delayed), free flap
Upper third of the leg	**Medial head of gastrocnemius (gold standard),** lateral head of gastrocnemius, proximally based soleus, tibialis anterior, free flap, Integra
Middle third of the leg	**Proximally based soleus**, medial or lateral head of gastrocnemius, FDL, EDL, EDH, tibialis anterior, free flap, Integra
Lower third of the leg	**Free flap**, reverse turndown sural artery flap, dorsalis pedis flap, propeller perforator flaps, cross leg flaps, Integra
Foot (dorsal)	Local flaps (i.e., rotation, transposition, etc.), free flaps (i.e., radial forearm, TPF + STSG), STSG
Foot (plantar)	**Free flaps for large wounds,** flexor digiti minimi brevis flap, flexor hallucis longus flap, flexor digitorum brevis, medial plantar flap

Abbreviations: ALT, anterolateral thigh flap; EDH, Extensor Digitorum Hallucis; EDL, extensor digitorum longus; FDL, flexor digitorum longus; STSG, split-thickness skin graft; TFL, tensor fascia lata.
Source: Adapted from Brown D, Borschel G. Lower Extremity Reconstruction. Michigan Manual of Plastic Surgery:361–365.[11]

Fig. 42.2 Patient in ▶ Fig. 42.1 after debridement with violation of joint capsule. (Courtesy of Bruce A. Mast, MD.)

Fig. 42.3 Patient in ▶ Fig. 42.1 and ▶ Fig. 42.2: medial gastrocnemius flap inset and fascial scored/cross hatched to extend surface area for inset. (Courtesy of Bruce A. Mast, MD.)

It is practical to think of the soft-tissue wounds involving the foot as dorsal defects and plantar defects as the reconstructive options are different. For dorsal wounds, thin, pliable tissue is ideal for shoe wear and aesthetics. Skin grafts or local flaps may be used for small defects such as rotation or transposition flaps. For larger defects with exposed vital structures (i.e., bare tendons, nerves, vessels, bone), free flaps are generally required. Some examples of suitable free flaps include radial forearm or temporoparietal fascia with STSG.[11] Latissimus dorsi free flap may be useful in children. For wounds over the plantar aspect of the foot, skin grafts can be used if adequate padding is present and the wound is less than one-third of the weight-bearing surface. In general, however, skin grafts should be avoided for larger and more extensive wounds over the plantar area, as they do not provide durable tissue for weight bearing. Several local flaps are available and are listed in ▶ Table 42.3.

Free flaps are generally required for large wounds with exposed tendons, nerves, vessels, or bone. If osteomyelitis is present, the wound should be treated with aggressive surgical debridement, IV antibiotics, and flap coverage once the wound and patient are optimized.[11]Free flaps should strongly be considered when the defect is large, dead space is present after

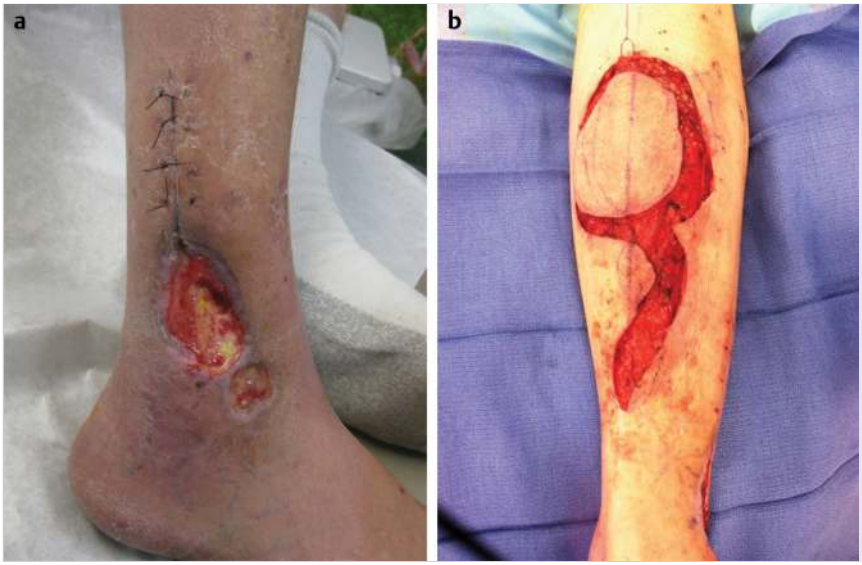


Fig. 42.4 **(a)** Patient with nonhealing lateral leg wound with exposed distal fibula. **(b)** Reversal sural artery flap dissected and ready for transfer. The view is of the posterior leg with the posterior edge of the wound visible at the distal right leg.

Fig. 42.5 **(a)** Flap inset seen distal right. The skin graft in the proximal leg is at the donor site and the graft distally is the portion of the pedicle that could not be closed over with the leg skin. **(b)** Healed wound 3.5 months after surgery.

debridements, local flaps have failed or damaged, the defect is in the distal third with hardware or bone exposure, and sacrifice of local tissues is not desirable. Some of the workhorse flaps include latissimus dorsi, rectus abdominis, gracilis, anterolateral thigh perforator flap (▶ Fig. 42.6, ▶ Fig. 42.7), and radial forearm fasciocutaneous flap. When it comes to lower extremity microsurgery, few points are important to emphasize: (1) perform your anastomosis outside of the zone of injury, (2) consider performing end-to-side arterial anastomosis to preserve flow to the distal extremity especially if there is less than three-vessel runoff present.[12]

42.4.4 Chronic Wounds of the Lower Extremity

Chronic wounds of the lower extremity, a well-known condition with high prevalence, high cost, and poor clinical outcome, are often managed by a nonintegrated health care system. These wounds are often difficult to treat because of poor patient compliance, poor control of underlying disease process, tobacco use, cost of supplies and care, associated medical comorbidities, and patient factors preventing proper wound healing. Etiologic factors for these types of wounds include, but are not limited to, diabetes, vascular insufficiency, venous stasis disease, lymphedema, osteomyelitis, cancer, radiation, and vasculitis. Initial patient evaluation should include history inquiring about onset, duration, location, and pain associated with the wound. Associated comorbidities such as diabetes or vascular disease should be determined. Ambulatory status, shoe wear, history of prior trauma, prior ulcers, and any prior treatment should be assessed. On physical exam, location, size, and depth of the wound should be noted. Pulses in the foot and sensory examination must be performed. The clinician should also look for signs of hemosiderin deposition, edema, skin tempera-

Fig. 42.6 Patient with Gustilo grade IIIB distal third open tibial fracture with exposed hardware.

Fig. 42.7 Patient from ▶ Fig. 42.1 underwent reconstruction with a free anterolateral thigh flap. The photograph demonstrates 3 months postoperative result.

ture, and ankle/brachial index assessment. Next, condition-directed studies should be ordered which may include radiographs, CT angiogram (if concern of poor inflow is present), culture and sensitivities, and possible biopsy. When a chronic ulcerating ulcer is present, suspicion should be high for Marjolin's ulcer and low threshold should exist for tissue biopsy.

Goals of treatment depend on specific etiology of the wound; however, generally the goals should be complete healing of the ulcer, return to ambulatory status, and prevention of recurrence. Diabetic ulcers are usually associated with the presence of peripheral neuropathy which according to studies is present in greater than 40% incidence after 20 years of diabetes. Physical exam generally reveals decreased foot sensation easily assessed with 5.07 Semmes-Weinstein filament. Surgical treatment requires thorough and potentially repeated debridement of all nonviable infected tissue and removing all of the excess callus. Debridement of infected bone is a must if osteomyelitis is present. When a chronic wound has visible or palpable bone, there is 85% chance of osteomyelitis. Unfortunately, skin grafts and flaps have limited success in diabetic wound reconstruction, as there is a very high rate of recurrence if peripheral neuropathy and poor glucose control are present.

Arterial ulcers appear "punched out" and are painful. The patient usually has a history consistent with claudication and/or rest pain. Popliteal and foot pulses are generally either diminished or absent. Extremities can be cool with cyanosis and shiny hairless skin. Smoking history is often present. Transcutaneous O_2, duplex ultrasonography, MRA, or arteriogram will likely confirm the diagnosis in majority of cases. These patients require revascularization by a vascular surgeon prior to any attempts at reconstruction. Moreover, if patient continues to smoke after revascularization and flap coverage, he or she is likely to have recurrent vascular occlusion and flap failure necessitating amputation.

Venous ulcers caused by venous stasis disease may be the most prevalent out of chronic leg wounds. Chronic edema, varicosities, and brownish discoloration of the skin from hemosiderin deposition with "wood-like" feel to the leg typically characterize these patients. The patient may report a history of deep venous thrombosis. The ulcers are generally located in the "gaiter region" between the malleoli and gastrocnemius musculotendinous junction. Medical management with Unna boot, leg elevation, and compression is only moderately effective. It is important to note that unless venous insufficiency is properly addressed and corrected, the ulcer will reoccur despite the best medical management, skin grafting, or flap coverage. Varicose vein stripping and great saphenous vein stripping have been tried with various success. Currently, vascular centers exist with specialized ultrasounds that can determine if insufficiency is mainly present in the deep, superficial, or perforating venous

system, thus allowing the surgeon for a more targeted ablation during operative intervention.

Extremities with lymphedema present with massively swollen leg and decreased skin blood flow. Medical management includes lymph-press pumps and leg wrapping techniques. Excising to fascia and skin grafting has been tried with some success. Lymphovenous bypass and vascularized lymph node transfers are being done in some centers with some promise.

Wounds from osteomyelitis usually present with chronic draining sinus tract that fails to close despite appropriate local wound care. History of prior traumatic event, fracture, or hardware placement is common. MRI, cultures, and aggressive debridement of infected bone with culture-directed intravenous antibiotics are necessary for proper treatment. Once full debridement has been performed, appropriate reconstruction with a vascularized tissue flap is performed.

When patients have history of radiation, there is generally damaged vascularity to the affected area which can be pretreated with hyperbaric oxygen to stimulate angiogenesis at the wound periphery. These wounds usually require aggressive resection of irradiated tissue and free flap reconstruction as local flaps are frequently ineffective because they are within the zone of radiation injury.[13]

42.5 Postoperative Care and Outcomes

Postoperative care depends on the type of wound and reconstruction performed. For example, if an STSG is placed, generally at least 1 week of immobilization with some type of compressive bolster or negative pressure wound therapy is required to improve the chances of graft take. If a local or pedicled flap was performed (i.e., gastrocnemius), a knee brace immobilizer should be worn for a least several weeks postoperatively with extremity elevation.

Postoperatively after lower extremity free flap reconstruction, it is important to keep the leg immobilized and elevated to establish sufficient venous drainage of the flap. Immobilization and dangling protocols vary, with the commonality of gradually dependence and weight bearing as orthopedically appropriate. The author's protocol is that the affected extremity should be immobilized and elevated for at least 10 days postoperatively and dangling should begin with gradual progression starting at about 5 minutes twice a day until at least 30 minutes of dangling 5 times per day are well tolerated. It is also important to look at the flap after each dangling session, and if any signs of venous congestion develop, dangling progression should be postponed until the flap is better acclimated. It is in the author's opinion that tight circumferential Kerlix or ace wrapping should be avoided in first several weeks postoperatively, as this can constrict the flap vascular supply, and potentially result in flap failure if done incorrectly. If the patient is reliable, outpatient dangling can be considered. It may take up to 2 months until full weight bearing status is achieved depending on the presence of fractures, severity of initial injury, and other factors.

The time-consuming and costly nature of the "expensive" and sophisticated reconstructive procedures versus straightforward amputation cannot be ignored. Although the exact cost of leg salvage is difficult to determine, even in high-risk groups such as diabetic patients, salvage may be less expensive than the combined cost of hospitalization, prosthesis fitting, rehabilitation, and disability payments.[14]

Timing of ambulation is controversial; however, surgeons should recognize that the lower extremity lymphatic system is damaged or at least insufficient in the early postoperative period. The reconstructed tissue, particularly with free tissue transfers, has a tendency to become very edematous early. If the edema is not controlled with strict elevation and measured compression, fibrosis can occur and lead to a fixed contour deformity.[12] Soltanian et al recommended that soft-tissue flaps in the lower extremity undergo a 2-month gradual accommodation period of allowing incremental time of the extremity in a dependent position.[12] More extensive reconstructions may require lifelong compression support when the patient has to keep his or her leg in a dependent position for an extended period.

Ideally, the patient suffering from a difficult lower extremity wound will experience stable healing with restoration of adequate function and satisfactory appearance. However, this is not always the case. Complications may intervene, some of which may be insurmountable. Amputation is the last resort, although in the circumstance of intractable pain, even this alternative may be unsatisfactory. Informing the patient of a realistic likely outcome, particularly if that outcome promises poor function, makes the decision for amputation easier.[15]

In conclusion, lower extremity reconstruction presents a challenge to a plastic surgeon that has to be addressed in a practical manner with regard to the mechanism of injury, patient's comorbidities, wound chronicity, and nutrition. Multidisciplinary approach is required for proper management of these complex patients.

42.6 Review Questions

42.6.1 True or False

1. Lower extremity amputation in a trauma adult patient should be considered if a complete disruption of posterior tibial nerve is present with concomitant crush injury.
2. Chronic lower extremity wounds resulting from venous insufficiency reconstructed with a skin graft or a flap generally produce good outcomes even without properly treating the underlying etiology.
3. Lower extremity amputation is always more cost-effective than reconstruction.

42.6.2 Choose the Best Answer

4. A 25-year-old healthy male 3 months after a motor vehicle accident presents to your clinic with a large, 10 cm × 15 cm wound with exposed bone, osteomyelitis, and hardware exposure involving the distal third of the right leg. On physical exam, his foot is sensate and well perfused. Which of the following management strategies would most likely result in stable wound coverage and optimum functional result?
   a) Debridement, pedicled proximally based soleus muscle flap and IV antibiotic therapy.

b) Removal of hardware, debridement, free latissimus dorsi muscle flap with STSG and IV antibiotic therapy.
c) Removal of hardware, debridement, pedicle medial gastrocnemius flap, and IV antibiotic therapy.
d) Amputation.

5. A 50-year-old female presents with a 5 cm × 4 cm wound on the anterior lateral aspect of her thigh just above the knee 1 week after a boating accident. On physical exam, the wound looks clean with healthy granulation bed over the exposed muscle bellies. Which of the following would be an acceptable method of reconstruction?
a) Negative pressure therapy followed by Integra and STSG.
b) Free rectus abdominus myocutaneous flap.
c) Pedicled gracilis muscle flap with STSG.
d) Reverse sural artery flap.

42.6.3 Answers

1. True.
2. False.
3. False.
4. c. Removal of hardware, debridement, pedicle medial gastrocnemius flap and IV antibiotic therapy.
5. a. Negative pressure therapy followed by Integra and STSG.

References

[1] Janis J. Essentials of Plastic Surgery. Second Edition. New York: Thieme Medical Publishers; 2014
[2] Gustilo RB, Anderson JT. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. J Bone Joint Surg Am. 58(4):453–458
[3] Bosse MJ, MacKenzie EJ, Kellam JF, et al. An analysis of outcomes of reconstruction or amputation after leg-threatening injuries. N Engl J Med. 347(24): 1924–1931
[4] Keller CS. The principles of the treatment of tibial shaft fractures. Orthopaedics. 6:993
[5] Byrd HS, Spicer TE, Cierney G , III. Management of open tibial fractures. Plast Reconstr Surg. 76(5):719–730
[6] Yaremchuk MJ, Brumback RJ, Manson PN, Burgess AR, Poka A, Weiland AJ. Acute and definitive management of traumatic osteocutaneous defects of the lower extremity. Plast Reconstr Surg. 80(1):1–14
[7] Godina M. Early microsurgical reconstruction of complex trauma of the extremities. Plast Reconstr Surg. 78(3):285–292
[8] Weiland AJ, Moore JR, Daniel RK. Vascularized bone autografts. Experience with 41 cases. Clin Orthop Relat Res.(174):87–95
[9] Ilizarov GA, Devyatov AA, Kamerin VK. Plastic reconstruction of longitudinal bone defects by means of compression and subsequent distraction. Acta Chir Plast. 22(1):32–41
[10] Ignatiadis IA, Tsiampa VA, Galanakos SP, et al. The reverse sural fasciocutaneous flap for the treatment of traumatic, infectious or diabetic foot and ankle wounds: a retrospective review of 16 patients. Diabet Foot Ankle. 2:2
[11] Brown D, Borschel G, Levi B. Michigan Manual of Plastic Surgery. 2nd Edition. New York: Wolters Kluwers Health; 2014
[12] Soltanian H, Garcia RMD, Hollenbeck ST, Current MD. Current concepts in lower extremity reconstruction. Plast Reconstr Surg. 136(6):815e–829e
[13] Smith APS. Etiology of the problem wound. In: Sheffield PJ, Smith APS, Fife CE, eds. Wound Care Practice. Flagstaff, AZ: Best Publishing Company; 2004:3–49
[14] Lior Heller MD, Scott Levin L. Lower extremity microsurgical reconstruction. Plast Reconstr Surg. 108(4):1029–1041
[15] Reddy V, Stevenson TR. MOC-PS(SM) CME article: lower extremity reconstruction. Plast Reconstr Surg. 121(4) Suppl:1–7

Part VIII

Body Contouring

VIII

43 Liposuction

Julian Winocour, John Layliev, Han Shi, Varun Gupta, and Kent K. Higdon

Abstract

This chapter covers the pertinent considerations involved in this common surgical procedure for the localized or regional (abdomen, flanks, back, buttocks, lower extremity, upper extremity, neck, axilla, breasts) removal of fat. It is a management tool for the purpose of body contouring, not for the management of obesity. Case examples for the most common liposuction sites are included. The ideal candidate is described, as well as contraindications for surgery. The four primary treatment options (suction-assisted, power-assisted, ultrasound-assisted, laser-assisted) are discussed, and each technique employed in the operation is cited to guide the surgeon through the procedure. Seasoned advice on postoperative care and several paragraphs on potential outcomes conclude the study.

Keywords: suction-assisted, power-assisted, ultrasound-assisted, laser-assisted liposuction, tumescent solution, superwet technique

43.1 Goals and Objectives

- Report the current incidence and trends of liposuction in North America and worldwide.
- Effectively evaluate patients preoperatively for liposuction.
- Formulate a surgical treatment plan to safely perform liposuction.
- Understand the differences among the various types of anesthesia and wetting solutions involved in liposuction.
- Institute appropriate perioperative care to maximize patient safety and surgical outcomes.
- Diagnose postoperative complications (both early and late) and formulate appropriate treatment plans.

43.2 Patient Presentation

Suction-assisted lipectomy, or liposuction, involves the surgical aspiration of fat from the subcutaneous space. It involves removal of localized or regional deposits of adipose tissue for the purpose of body contouring, but is not a management of obesity. Liposuction is the most commonly performed aesthetic surgical procedure in the United States according to the American Society of Aesthetic Plastic Surgery (ASAPS) cosmetic national database (342,494 procedures performed in 2014). This represents a 94% increase since the database initiation in 1997.[1] According to the International Society of Aesthetic Plastic Surgery (ISAPS), liposuction ranks second behind eyelid surgeries in worldwide procedures performed in 2014 (1,372,901 procedures performed).[2] Liposuction is being performed increasingly in combination with other surgical procedures to enhance outcomes.

Patients who present for body-contouring procedures desire a reshaping of various regions of their body. Although the trunk has been classically known as the most popular site for liposuction, more and more patients are electing to remove excess adiposity from the upper and lower extremities, breasts/axilla, hips, and neck regions (▶ Fig. 43.1). Equally, patients must understand that liposuction is not a weight loss procedure, but aimed at the reduction of local accumulations of excess fat.[3] An ideal candidate for liposuction is a healthy individual, close to his or her ideal body weight, with realistic body-image expectations, and motivated to making long-term lifestyle changes.[4] Women typically desire to remove excess fat peripherally to obtain a gynecoid body figure, while men aim to reduce excess central fat distributions.[5]

Patient evaluation should begin with a thorough medical and surgical history and a diligent physical exam. The medical history should include specific comorbid conditions, blood clotting disorders, and especially a full medication history (including vitamin supplements, herbal products, and any weight-loss medications). Medications that predispose to bleeding should be avoided. The surgical history should include any surgeries associated with the pertinent body areas in question as well as any major operations and any previous complications. Knowledge of previous weight loss and body-contouring procedures are particularly important. Prevention of venous thromboembolism is a particular concern of all body-contouring procedures, including liposuction. A higher rate of venous thromboembolism has been demonstrated in patients with a body mass index (BMI) greater than 30, patients taking hormone therapy, and those with a higher Caprini risk assessment. For patients receiving a general anesthetic, sequential compression devices (SCD) should be utilized when possible along with proper patient positioning (allowing maximal lower extremity venous drainage) and avoiding external pressure or constriction of the lower extremities. Preoperative chemoprophylaxis may be warranted depending on individual patient risk factors, guided by the venous thromboembolism task force recommendations by the American society of Plastic Surgeons (ASPS).[6,7]

Obtaining a weight history is important in order to determine if a patient has a high risk of gaining weight following liposuction.[4] Candidates within 20% of an ideal body weight ultimately have the best long-term satisfaction. In general, patients with a BMI greater than 30 may benefit from lifestyle modifications before considering most body-contouring procedures.[8]

During the physical examination, the patient should be completely disrobed. The patient's height, weight, BMI, and circumferences of pertinent body regions should be documented.[6] The overall fat distribution should be assessed and gentle pinch testing should be used to assess the thickness of subcutaneous fat. The sites of interest should be examined in multiple positions: standing, sitting, and in the supine position. It is essential to evaluate the location and degree of adiposity as well as the laxity of the targeted areas. A comprehensive skin exam is also crucial to determining a good outcome from surgery. Skin and dermal quality can be assessed by observation and palpation. Thick dermis has more retractile strength, which is favorable for liposuction. Furthermore, elastic skin is favorable compared with flaccid skin for retraction outcome. A snap-test or pinch of skin test can be used to assess skin recoil. Individuals with cellulite, dimples, wrinkles, or very loose skin may have less than ideal results. Individuals with excess skin laxity may require

Fig. 43.1 Demonstrating the problem areas that can be addressed with liposuction. ©American Society of Plastic Surgeons 2008.

skin resection (i.e., abdominoplasty, brachioplasty, thigh plasty, etc.) as liposuction without adequate skin recoil will result in loose and folded skin.

Lipodystrophy can be located in the following areas: abdomen, flanks, back, buttocks, lower extremity (thighs, knees, ankles, and calves), upper extremity, neck, axilla, and breasts. Men who present for abdominal liposuction should be examined diligently, as abdominal bulging can be attributed to intra-abdominal fat, which cannot be addressed with liposuction. The presence of an occult abdominal hernia must be evaluated and abdominal wall scars should be noted; an abdominal hernia can be most easily diagnosed in the supine position. Surface irregularities such as dimpling and cellulite (lipomatous deposits) should be noted and demonstrated to the patient, as these may remain and actually may become more perceptible postoperatively. Patients should understand that while liposuction can be used to contour areas, it does not address cellulite. In contrast, skin dimpling (typically caused by an underlying fascial attachment) may be improved with liposuction.[6] Furthermore, patients should be aware of possible persistent asymmetry, dimpling, and depressions following liposuction. Ideal patient characteristics for liposuction are listed in ▶ Table 43.1 and relative contraindications are listed in ▶ Table 43.2.

Table 43.1 Ideal candidate for liposuction

Clinical feature	Description
Body type	• Stable weight • BMI < 30 (not obese) • Aspirate volume appropriate with patient body surface area
Tissue content	• Less fibrous content (easier penetration by cannula) • Few previous scars, absence of hernias
Excess tissue	• Minimal skin excess and laxity
Symmetry	• Minimal contour irregularities
Other features	• Commitment to making healthy changes in diet, exercise, lifestyle • Specifically, patients who eat a healthy diet and exercise regularly are 1.5–2 times more likely to lose weight, resulting in long-term satisfaction

Source: Data from Iverson and Pao[6]; Rohrich et al[28]; Shiffman M, ed. Cosmetic Surgery Art and Techniques. Berlin: Springer; 2013; Halperin B. Patient safety in plastic surgery. In: Neligan P, ed. Plastic Surgery. 3rd ed. London: Elsevier Saunders; 2013.

43.3 Preparation for Surgery

Healthy patients should have a complete blood count to evaluate preoperative hemoglobin and hematocrit for possible anemia or occult blood loss. Patients who have undergone significant weight loss operations (bariatric surgery) must be carefully assessed for nutritional status (serum albumin or prealbumin), anemia, and vitamin deficiencies contributing to anemia (vitamin B12 and iron).[9] The patient should consult with their primary care physician for discontinuation of herbal supplements (vitamin E, St John's wort, etc.), weight-loss medications, nonsteroidal anti-inflammatory drugs, aspirin, hormone products (hormone replacement therapy [HRT], oral contraceptive pill [OCP], etc.), fish oil, and other supplements for at least 4 to 6 weeks before the procedure date to ensure normal wound healing and blood clotting and to lower the risk of thrombosis. Medications that interact with liver metabolism of lidocaine, one of the main components of tumescent solution, should also be stopped; these include selective serotonin reuptake inhibitors (SSRI), erythromycin, ketoconazole, and other agents.

Table 43.2 Contraindications to liposuction

Clinical feature	Description
Morbid obesity	• Liposuction is not a weight-loss surgery • Patients with BMI > 30 will benefit from counseling, nutrition education, and lifestyle modification prior to body contouring • More likely to suffer from sleep apnea; increased risk for complications
Not surgical candidate	• Medical comorbidities (including severe cardiovascular disease, sleep apnea, etc.) • History of VTE or predisposition (chronic venous insufficiency, family history, severe infection, polycythemia, malignancy, homocystinemia, pelvic or lower extremity radiation, HRT/OCP—not an absolute consideration) • Hypercoagulable conditions (e.g., factor V Leiden mutation) must be controlled with thromboprophylaxis • Pregnancy
Medications	• Specifically those that affect wound healing or blood clotting
Psychiatric	• Mood disorders, thought disorders, severe anxiety, depression, body dysmorphic disorder
Skin conditions and wound healing problems	• Conditions of skin or subcutaneous tissue that make liposuction hazardous • Poor scarring and dehiscence possible
Other features	• Unrealistic expectations

Abbreviations: BMI, body mass index; HRT, hormone replacement therapy; OCP, oral contraceptive pill; VTE, venous thromboembolism.
Source: Data from Data from Iverson and Pao[6]; Halperin B. Patient safety in plastic surgery. In: Neligan P, ed. Plastic Surgery. 3rd ed. London: Elsevier Saunders; 2013; Kenkel J, Stephan P. Liposuction: a comprehensive review of techniques and safety. In: Warren R, Neligan P, eds. Plastic Surgery: Volume 2: Aesthetic Surgery; 2012:507–529

Smoking increases the risk for postoperative complications because of tobacco's primary effects on vasoconstriction and decreased wound healing capacity. The authors agree with the Centers for Disease Control and Prevention (CDC) recommendations of smoking cessation 30 days prior to surgery.

For higher risk individuals and patients in an inpatient setting, assessing coagulation profiles (PT/PTT), a comprehensive metabolic panel (CMP), HIV serology, and a hepatitis viral panel should be considered. An ECG and an echocardiogram should be obtained for individuals with pertinent cardiovascular history. Abnormalities should be cleared by the respective medical specialists prior to consideration of any elective surgery.

43.4 Treatment

Several options exist for liposuction procedures[10]:
1. Suction-assisted liposuction (SAL).
2. Power-assisted liposuction (PAL).
3. Ultrasound-assisted liposuction (UAL).
4. Laser-assisted liposuction (LAL).

Suction-assisted lipoplasty uses an external source of suction to facilitate fatty tissue removal.[6] In *power-assisted lipoplasty*, the system driving the cannula is an electrically or gas-driven power source; a motor moves the cannula tip in a forward and backward motion reducing physician fatigue.[6] This is effective for large-volume fat removal, fibrous areas, and revision procedures. Syringe aspiration of fat is effective for sensitive and smaller volume areas such as the neck. *Ultrasound-assisted liposuction* uses a cannula to deliver ultrasound subcutaneously to liquefy fat.[6] This facilitates removal of fat from fibrous areas such as the upper abdomen and back, especially during secondary or revision procedures. Thermal injury is a risk during this technique but can be avoided by keeping the cannula in constant motion and by using tumescent solution as this cools the heat generated in the process. Additionally, the ultrasound should be used in a time-limited manner, usually at about 1 minute per 100 mL of tumescent solution instilled. *Laser-assisted* liposuction is designed to achieve skin tightening from the thermal effect of laser on the dermis through selective photothermolysis of fat and water chromophores.[11] There are currently no randomized controlled studies reporting laser-assisted liposuction to have significant clinical advantages over "traditional" suction-assisted liposuction.[11]

The indications for liposuction surgical treatment fall broadly under several categories:
1. "Isolated procedure" cosmetic contouring (most common indication, ▶ Fig. 43.2).
2. Adjuncts to other cosmetic surgical procedures (abdominoplasty, body lift, breast reduction, breast augmentation, weight loss skin removal surgeries to debulk adipose tissue content).[12]
   a) Liposuction can be used on abdomen and flanks in combination with full and mini-abdominoplasties after tissue dissection and resection.
3. Treatment for specific medical disorders.
   a) Gynecomastia—often requires additional resection of glandular tissue.
   b) Buffalo hump—reducing uncomfortable fat deposits on back.
   c) Lipomatosis syndromes.
   d) Other—lipodystrophy from HIV, etc.

43.5 Surgical Preparation

Preoperative markings are done in the standing position. Zones of facial adherence are noted and to be avoided. Multiple access incisions are used for cannula placement and may be placed in natural skin folds. This often provides easier access and greater safety, especially for transition areas such as the costal margin where injury to intrathoracic/abdominal structures are at greater risk. A thorough operative record is maintained during the procedure to document the volume of subcutaneous tumescent infiltration and the volume of aspiration at each body area of liposuction.

Assessment and reduction of perioperative morbidity is critical and, thus, it is important to note the American Society of Anesthesiologists (ASA) classification for the given patient. Class I and II ASA patients who are healthy or have mild/stable illness, respectively, can be managed as outpatient procedures if the estimated liposuction aspirations are less than 5,000 mL (▶ Table 43.3). Mortality and morbidity rate increases with more invasive liposuction procedures and an inpatient setting may be considered for ASA class III patients, procedures with an estimated liposuction aspiration more than 5,000 mL, estimated

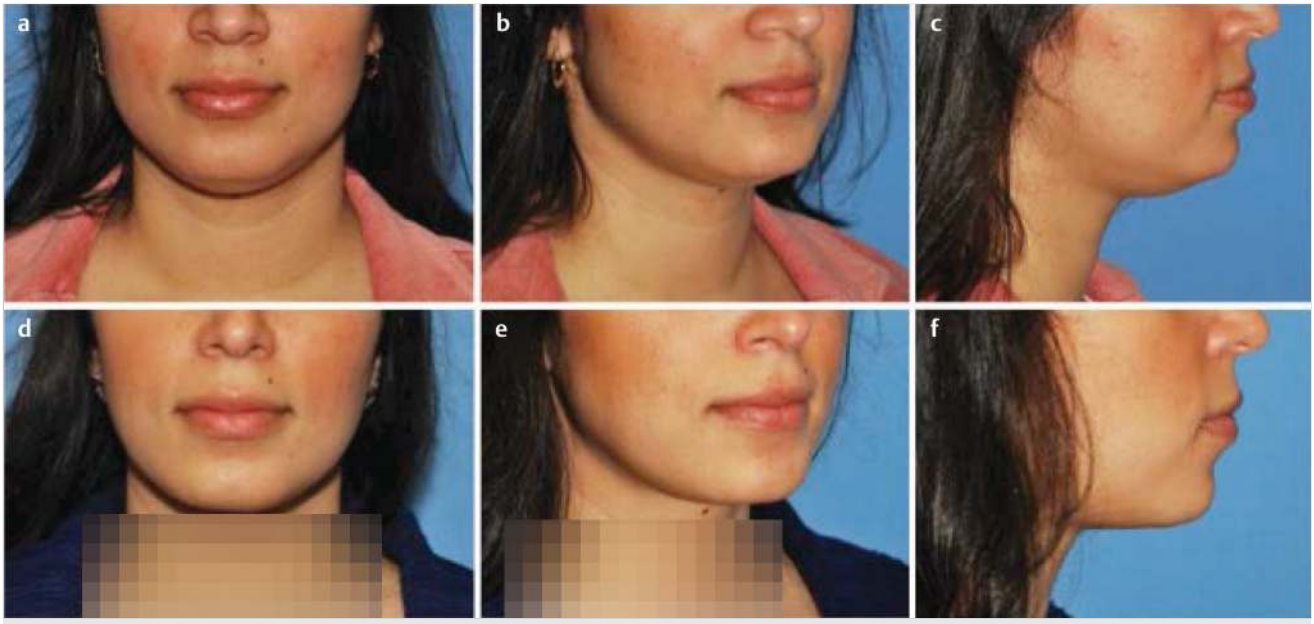


Fig. 43.2 A 28-year-old woman with isolated lipodystrophy to the submental area. Photographs demonstrating **(a-c)** preoperative and **(d-f)** postoperative of isolated liposuction to the submental area.

Table 43.3 American Society of Anesthesiologists Classification

ASA	Description
I	Healthy patient, no medical problems
II	Mild systemic disease
III	Severe systemic disease, but not incapacitating
IV	Severe systemic disease that is a constant threat to life
V	Moribund, not expected to live 24 h, irrespective of operation
VI	Donor patient for organ harvesting

Abbreviation: ASA, American Society of Anesthesiology.

blood loss more than 500 mL, or duration of surgery more than 2 hours.[6,13,14,15,16,17]

Various types of anesthesia are appropriate for liposuction depending on overall patient health and estimated volume of fat to be aspirated. In smaller volume liposuction aspiration cases, anesthetic infiltrate solutions alone may be sufficient to provide adequate pain control during the procedure. This is sometimes combined with a field block performed prior to tumescent infiltration. Several factors allow for increased lidocaine administration over traditional limits. The risk of systemic toxicity from the lidocaine and epinephrine solution is dramatically decreased due to numerous factors including the slow absorption of the infiltrate from fat, the vasoconstriction effect of epinephrine, and because a significant amount of the solution is aspirated. Lidocaine doses up to 35 mg/kg have been generally considered safe to use for solutions containing epinephrine injected into subcutaneous fat; however, doses as high as 55 mg/kg have been described.[6,18,19,20] In patients with low protein states or medical conditions leading to accumulation of lidocaine metabolic byproducts, the maximum lidocaine doses should be reconsidered. It is important to recognize and be cognizant of the signs of lidocaine toxicity, which can vary from lightheadedness, tinnitus and a metallic taste in the mouth to tremors, convulsions, respiratory depression, and even cardiac arrest.[6] Doses of epinephrine should not exceed 0.07 mg/kg, although higher doses have been used safely.[6] Staging the infiltration of multiple anatomical sites can equally reduce the risk of excess epinephrine. Although it is highly unlikely that liposuction would be done in patients who have hyperthyroidism, severe hypertension, cardiac disease, peripheral vascular disease, or a pheochromocytoma, epinephrine should be avoided when such conditions exist.[6] Intravenous sedation can be used in limited smaller volume liposuction cases to maintain patient comfort. General anesthesia is typically required for larger volume liposuction procedures.

Anxiolysis if needed can be maintained with preoperative oral diazepam or lorazepam; less effective are opioids and narcotics. Antihistamines like hydroxyzine can also be used to add sedation and to reduce nausea and pruritus. Finally, as discussed previously, preoperative chemoprophylaxis (either low-molecular-weight heparin or unfractionated heparin) can be used for venous thromboembolism prophylaxis for high-risk patients along with standard lower extremity compression devices.

Multiple techniques of liposuction fluid administration exist and have evolved over time. The dry technique involves liposuction without subcutaneous solution infiltration. This has been abandoned because of substantial swelling, discoloration, and blood loss, with suction aspirates containing 20 to 45% blood.[8] In the wet technique, 200 to 300 mL of infiltrate is injected per body area before insertion of the liposuction cannula; this reduces blood loss up to 4 to 30% of the aspirate.[8] In the superwet technique, 1 mL of solution is injected for each 1 mL total aspirate to be removed; this reduces blood loss to less

Fig. 43.3 Demonstration of infiltration of tumescent fluid with multiple controlled passes in the orthogonal plane.

Fig. 43.4 Demonstration of liposuction of medial thigh with counter pressure from nondominant hand. Fanning markings from entry site drawn out.

Table 43.4 Liposuction fluid resuscitation guidelines[2,2]

Small-volume aspirations (< 5 L)	Large-volume aspirations (> 5 L)
Maintenance fluid[a] Subcutaneous infiltrate[b]	Maintenance fluid[a] Subcutaneous infiltrate[b] 0.25 mL of intravenous crystalloid per mL of aspirate > 5 L

[a]Amount of fluid to be replaced from preoperative, nothing by mouth status.
[b]70% is presumed to become intravascular.

than 1% of the aspirate volume. In the tumescent technique, 3 to 4 mL of infiltrate is infused for each planned 1 mL of aspirate; blood loss in this technique is also estimated to be less than 1% of the aspirate volume.[8] After wetting solution injection in the tumescent technique, the skin should feel firm and indurated indicating complete infusion (▶ Fig. 43.3). Large-volume liposuction, defined as a total aspirate of 5,000 mL or greater, can be safe for selected patients and with appropriate intraoperative fluid balance. Overnight observation is recommended after large-volume liposuction.[21] Liposuction fluid resuscitation guidelines are outlined in ▶ Table 43.4.

43.5.1 Operative Technique

Following infiltration of tumescent solution, 10 to 20 minutes is allotted to allow for the full effect of vasoconstriction prior to liposuction. The same access points used for tumescence are typically used for liposuction. During liposuction, a blunt-ended hollow cannula is used to tunnel under the skin and aspirate fatty tissue. The diameter of the tip typically ranges from 2 to 5 mm and is driven backward and forward manually in SAL or with a motor in PAL. The cannula is first inserted vertically into the incision site, and then readjusted to the orthogonal plane (the coronal plane for abdominal liposuction). The key to safe and adequate aspiration with the cannula is tunneling in an appropriate plane, just deep to the superficial fascia. The operating surgeon should at all moments know the exact location of the cannula to ensure safe technique. This is facilitated by the nondominant hand that can be used to push down and apply gentle pressure to the skin above the deep plane to guide the cannula. Care must be taken not to compress the tissue when lifting skin and subcutaneous tissue with the cannula; skin pinching to assess for asymmetry and lumps is appropriate. Linear and rhythmic strokes should be made in fan-like distribution that circumferentially removes the fat consistently and evenly in the marked out area. Tunneling should be done in the mid-fat and deep-fat planes to ensure even adipose removal (▶ Fig. 43.4). Major lymphatic, nerve, and blood vessels should be avoided; tunneling parallel to the longitudinal axis of the body (e.g., avoiding epifascial lymph vessels in lower extremity) has been shown to reduce the risk of vessel damage.[22,23]

Liposuctioned fat is emulsified and oily, and can be preserved for autologous fat transfer.[24] Incisions can be closed with 5–0 nylon sutures (or fast absorbing suture) or can be left open for drainage if greater than 2,000 mL of lipoaspirate was removed in total.

43.5.2 Postoperative Care

Following closure of incisions, a compression garment or binder should be applied in order to reduce bruising and excess edema. Compression foam can also be used. Compression garments should be worn for 4 to 6 weeks to decrease the postoperative seroma rate. Reduction in edema also maximizes skin recoil and redraping. Outpatient follow-up typically is at 5 days to remove sutures, and 2 weeks, 6 weeks, and at 3 months to assess bruising, edema, and outcome. At the 6-month visit, most, if not all, of the edema should have subsided.

Patients are informed to ambulate on the day of surgery. Physical activity should be limited during the first week but gradually increased with light exercise in the second, third, and fourth weeks. After 1 month, the patient's physical activity can return to baseline. Postoperative pain is typically controlled with acetaminophen; codeine, tramadol, or other oral narcotics

can be added if needed for breakthrough pain. To reduce the risk of infection, patients are asked to not shower or bathe until after 48 hours following surgery. Postoperative antibiotics are typically not used.

43.6 Outcomes

The most common postoperative adverse sequelae of liposuction are contour deformities. These can be considered complications if they persist over 6 months.[6] Because contour deformities may be secondary to postoperative swelling and skin elasticity, they may be treated conservatively for 6 months; treatments generally involve manual lymphatic massage.[6] For areas of excess fat removal or insufficient fat removal, secondary liposuction, fat grafting, or dermolipectomy can be considered. Maintaining a tunneling depth more than 1 cm from the skin is the best way to avoid skin grooving and depressions from excessive fat suctioning.[25] Sites of inadequate skin retraction may require secondary skin resection.

The largest published database (CosmetAssure database) looking at major complications (defined as requiring emergency department visit, admission to hospital, or take back to the operating room) following liposuction reports the most common major complications as infection (0.7%), hematoma (0.6%), and thromboembolism (0.6%).[26] The incidence of significant complications was 0.7% with liposuction alone, 3.8% when combined with abdominoplasty, and 12% when combined with abdominoplasty, breast, and another body-contouring procedure.[2,6] Infections are three times more likely to occur in an inpatient environment and needs to be quickly identified to prevent necrotizing fasciitis and toxic shock syndrome.[25]

Patients with a higher BMI have been found to have a higher risk of developing postoperative seromas.[6] Suggestions for decreasing the incidence of seromas include expressing any remaining fluid before closure, using a single suture to close access incisions, applying a compression garment, and early ambulation.[6] Simple aspiration is the most common treatment for a seroma.

Although quite rare with the proper use of dilute local anesthetic solutions, postoperative lidocaine toxicity must also be recognized. Early postoperative recovery characterized by dizziness, restlessness, tinnitus, mouth numbness, or a metallic taste may be early signs of toxicity (plasma concentration 3–6 µg/mL).[27] Later side effects can include muscle twitching, tremors that can lead to convulsions, CNS depression, and coma.

Most patients are satisfied following liposuction. The most common postoperative complaints are pain, fat return, and weight gain.[22] Eating a healthy diet and exercising regularly were found to be the main factors reducing unwanted postoperative weight gain and ultimately dissatisfaction.[28]

43.7 Case Examples of Common Liposuction Sites

43.7.1 Trunk and Thighs

A 25-year-old woman with lipodystrophy of her abdomen, flanks, anterior/lateral/medial thighs, and medial knees. Standard SAL was performed on flanks/thighs/knees, UAL was performed on abdomen. Superwet infiltration technique was performed with 2,375 mL removed in total (▶ Fig. 43.5; ▶ Fig. 43.6).

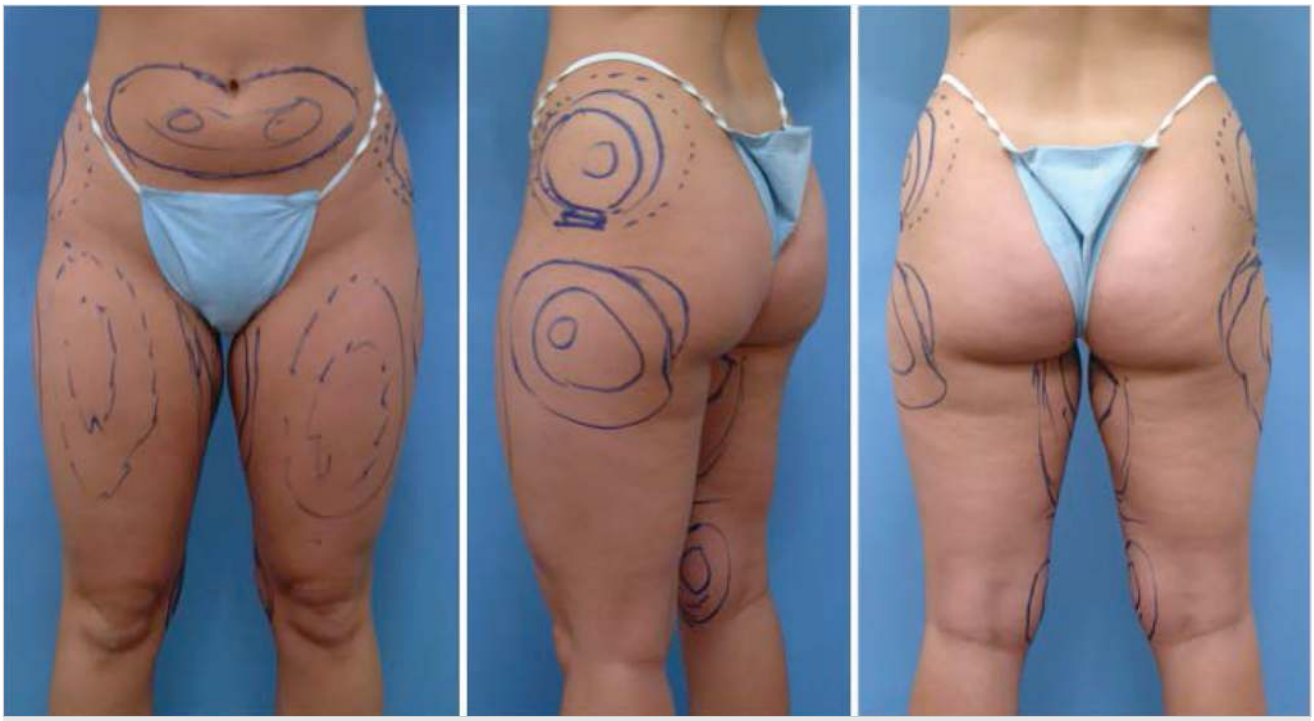


Fig. 43.5 Topographic markings of liposuction of abdomen, flanks, anterior/lateral/medial thighs, and medial knees.

Fig. 43.6 Demonstrating preoperative (upper) and 1 year postoperative (lower) photographs.

43.7.2 Trunk

A 41-year-old woman with localized lipodystrophy of her abdomen/flanks. Standard SAL was performed. Superwet infiltration technique was performed with 2,900 mL removed in total (▶ Fig. 43.7; ▶ Fig. 43.8).

43.7.3 Ankles, Calves Liposuction

Patients undergoing lower leg liposuction require specific consideration and experience. Patients' satisfaction in this area is historically lower due to the difficulty in appreciation of the areas of adiposity. Equally prolonged postoperative pain can be seen (▶ Fig. 43.9).

43.7.4 Gynecomastia

A 38-year-old man with gynecomastia (glandular and fat). UAL was performed. Superwet infiltration technique was performed with a total aspiration volume of 800 mL (▶ Fig. 43.10).

43.7.5 Upper Extremity (Arms)

A 31-year-old woman with lipodystrophy of upper arms. Standard SAL was performed. Superwet infiltration technique was performed with greater than 1,000 mL removed in total (▶ Fig. 43.11).

43.7.6 Submental

A 41-year-old woman with localized lipodystrophy of her neck. Standard SAL was performed. Superwet infiltration technique was performed with 60 mL removed in total (▶ Fig. 43.12).

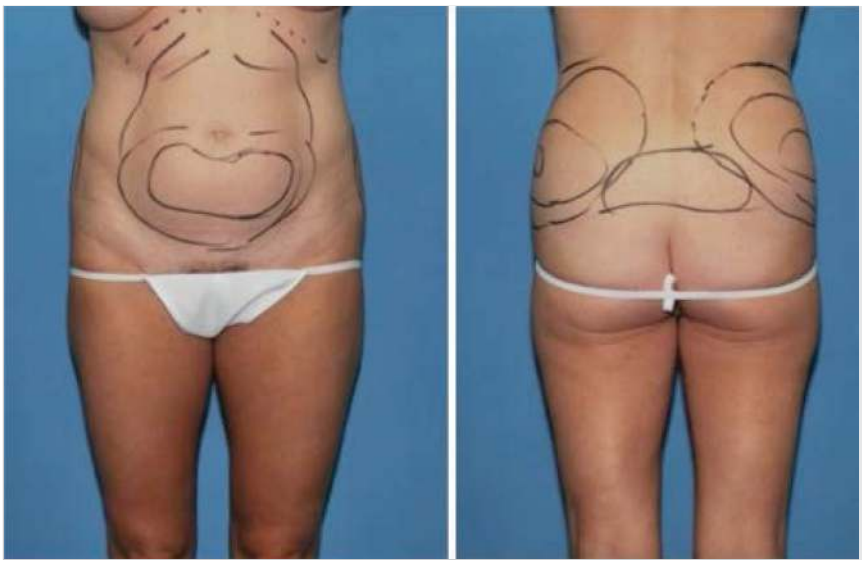


Fig. 43.7 Topographic markings of liposuction of anterior abdomen, flanks, and lower back.

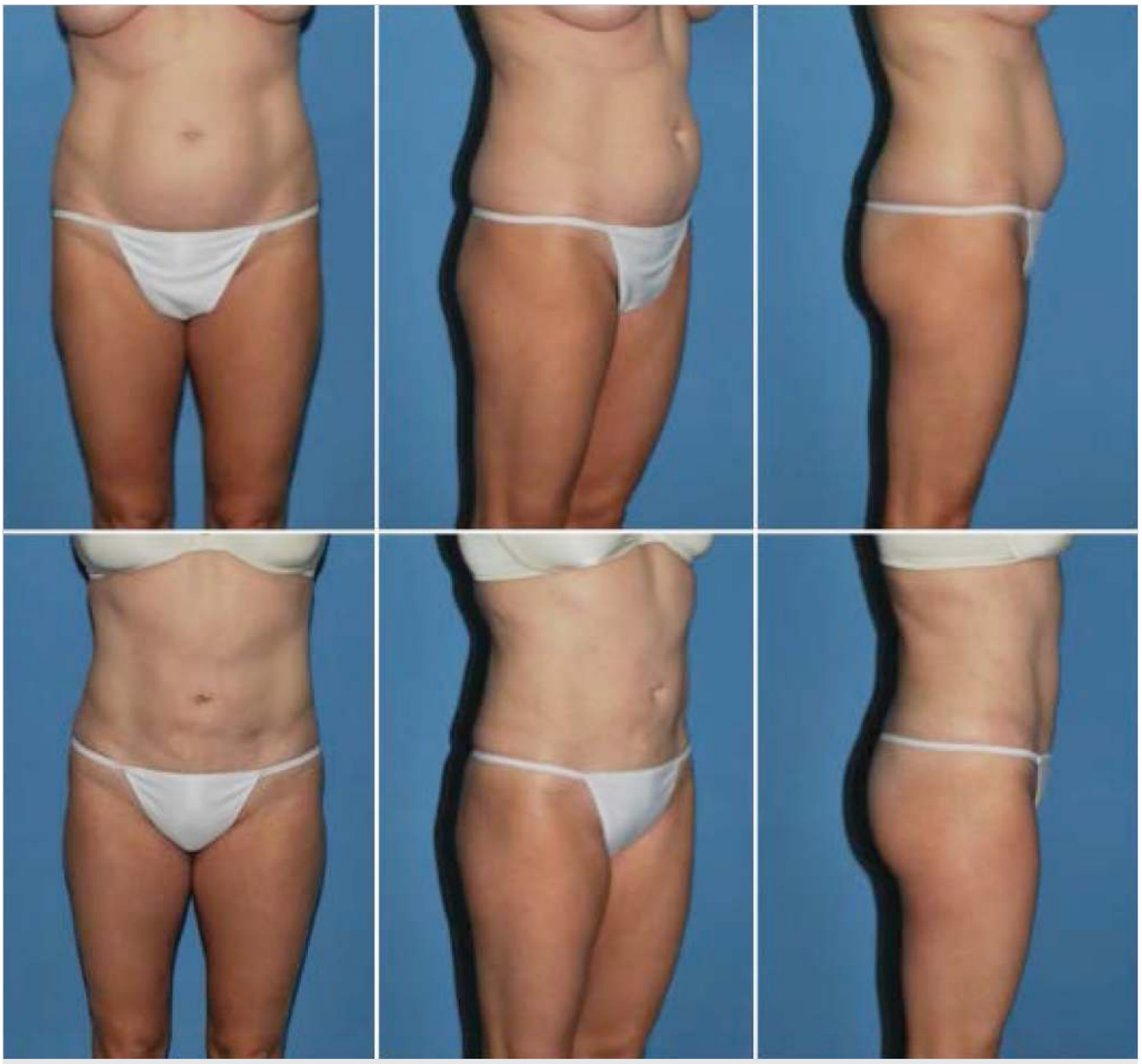


Fig. 43.8 Preoperative (upper) and 6 weeks postoperative (lower) photographs.

Fig. 43.9 Preoperative (upper) and postoperative (lower) results of a 48-year-old woman with isolated adiposity of the lower legs. Isolated ankle/calf liposuction performed.

Fig. 43.10 Preoperative (upper) and 3 months postoperative (lower) photographs of a 38-year-old male patient undergoing liposuction treatment without direct glandular resection for prominent gynecomastia.

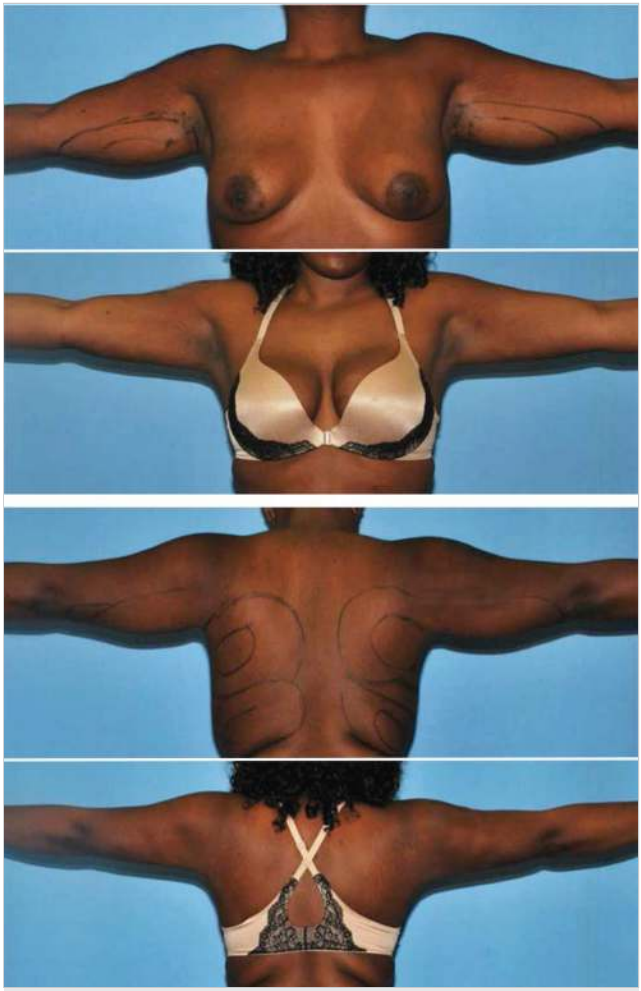


Fig. 43.11 Arm liposuction. Demonstrating anterior (upper photographs) and posterior (lower photographs) preoperative and 6 months postoperative from liposuction to the arms without skin resection.

Fig. 43.12 Preoperative (upper) and postoperative (lower) photographs of isolated liposuction to the submental area.

43.8 Review Questions

1. A healthy 45-year-old woman is scheduled to undergo suction lipectomy of the abdomen and flanks using the superwet technique. When injected into subcutaneous fat with epinephrine, which of the following is the maximum recommended dose of lidocaine?
   a) 7 mg/kg.
   b) 28 mg/kg
   c) 35 mg/kg.
   d) 14 mg/kg.
   e) 21 mg/kg.
2. When performing liposuction using a tumescent technique, the ratio of infiltrate:aspirate is *closest* to which of the following?
   a) 2:1.
   b) 1:2.
   c) 1:3.
   d) 1:1.
   e) 3:1.
3. When performing suction lipectomy using the superwet technique, the amount of blood loss in the suction aspirate is *closest* to which of the following?
   a) 20%.
   b) 40%.
   c) 1%.
   d) 30%.
   e) 10%.
4. A 45-year-old man who has achieved substantial weight loss from massive obesity is scheduled to undergo belt lipectomy for circumferential truncal excess. Which of the following is the most likely postoperative complication?
   a) Deep venous thrombosis.
   b) Contour irregularities.
   c) Skin necrosis.
   d) Dehiscence.
   e) Infection.
5. Compared with traditional suction lipectomy, which of the following is more likely to occur with ultrasonic-assisted suction lipectomy?
   a) Contour irregularity.
   b) Major blood loss.
   c) Nerve injury.
   d) Pulmonary edema.
   e) Thermal burns.

43.8.1 Answers

1. c. 35 mg/kg.
2. e. 3:1.
3. c. 1%.
4. b. Contour irregularities.
5. e. Thermal burns.

References

[1] American Society for Aesthetic Plastic Surgery (ASAPS). "2014 National Totals for Cosmetic Procedures." Available at: www.surgery.org. Accessed September 2015
[2] ISAPS News: Official Newsletter of the International Society of Aesthetic Plastic Surgery (August 2015). ISAPS News. Available at: http://www.isaps.org/Media/Default/newsletters/ISAPS%209–2int-1.pdf. Accessed October 2018
[3] Goldsberry A, Hanke C. Liposuction, Ultrasound-assisted and Powered. In: Orringer J, Dover J, Alam M, eds. Body Shaping: Skin Fat Cellulite. Elsevier Health Sciences; 2014:69–78
[4] Gingrass M. Liposuction. In: Thorne C, ed. Grabb and Smith's Plastic Surgery. Lippincott Williams & Wilkins; 2013:781–789
[5] Kaminer M. Tumescent liposuction. In: Arndt K, Dover J, eds. Atlas of Cosmetic Surgery. Elsevier Health Sciences; 2008:323–365
[6] Iverson RE, Pao VS. MOC-PS(SM) CME article: liposuction. Plast Reconstr Surg. 121(4) Suppl:1–11
[7] Murphy RX , Jr, Alderman A, Gutowski K, et al. Evidence-based practices for thromboembolism prevention: summary of the ASPS Venous Thromboembolism Task Force Report. Plast Reconstr Surg. 130(1):168e–175e
[8] Iverson RE, Lynch DJ, American Society of Plastic Surgeons Committee on Patient Safety. Practice advisory on liposuction. Plast Reconstr Surg. 113(5): 1478–1490, discussion 1491–1495
[9] Kenkel J. Safety considerations and avoiding complications in the massive weight loss patient. Plast Reconstr Surg. 117(1) Suppl:74S–81S, discussion 82S–83S
[10] Ahmad J, Eaves FF , III, Rohrich RJ, Kenkel JM. The American Society for Aesthetic Plastic Surgery (ASAPS) survey: current trends in liposuction. Aesthet Surg J. 31(2):214–224
[11] Fakhouri TM, El Tal AK, Abrou AE, Mehregan DA, Barone F. Laser-assisted lipolysis: a review. Dermatol Surg 2012;38(2):155–169
[12] Rubin J, Gusenoff J. Bodylifts and Post Massive Weight Loss Body Contouring. In: Guyuron B, ed. Plastic Surgery: Indications and Practice. 1st ed. Edinburgh: Saunders Elsevier; 2009:1627–1654
[13] American Academy of Cosmetic Surgery. Guidelines for Liposuction; 1995:2–6
[14] Gold BS, Kitz DS, Lecky JH, Neuhaus JM. Unanticipated admission to the hospital following ambulatory surgery. JAMA. 262(21):3008–3010
[15] Pasternak L. Screening patients: Strategies and studies. In: McGoldrick K, ed. Ambulatory anesthesiology, a problem-oriented approach. Baltimore, MD: Williams & Wilkins; 1995:10
[16] Federated Ambulatory Surgical Association. FASA Special Study I. FASA, Alexandria, VA; 1986
[17] Bennett G. Anesthesia for liposuction and abdominoplasty. In: Shiffman M, Mirrafati S, eds. Aesthetic Surgery of the Abdominal Wall. Berlin: Springer; 2005:29–54
[18] Fodor PB, Watson JP. Wetting solutions in ultrasound-assisted lipoplasty. Clin Plast Surg. 26(2):289–293, ix
[19] Pitman GH, Aker JS, Tripp ZD. Tumescent liposuction. Clin Plast Surg. 26:289
[20] Rohrich RJ, Beran SJ. Is liposuction safe? Plast Reconstr Surg. 104(3):819–822
[21] Beran S, Rohrich R. Body contouring (overview). Selected Readings in Plastic Surgery 1998;8(1)
[22] Broughton G , II, Horton B, Lipschitz A, Kenkel JM, Brown SA, Rohrich RJ. Lifestyle outcomes, satisfaction, and attitudes of patients after liposuction: a Dallas experience. Plast Reconstr Surg. 117(6):1738–1749
[23] Frick A, Hoffmann JN, Baumeister RG, Putz R. Liposuction technique and lymphatic lesions in lower legs: anatomic study to reduce risks. Plast Reconstr Surg. 103(7):1868–1873, discussion 1874–1875
[24] Pereira L, Sterodimas A. Liposuction of the abdomen and trunk. In: Rubin J, Jewell M, Richter D, Uebel C, eds. Body Contouring and Liposuction. Elsevier Health Sciences; 2012:311–320
[25] Shiffman M. Prevention and treatment of liposuction complications. In: Mirrafati S, ed. Aesthetic Surgery of the Abdominal Wall. Springer; 2005:215–220
[26] Ramirez JR, Gupta V, Winocour J, Shack RB, Grotting JC, Higdon K. Liposuction: how safe is it? An analysis of 31,010 cases. Plast Reconstr Surg. 135(4):1180
[27] Klein JA. Tumescent technique for regional anesthesia permits lidocaine doses of 35 mg/kg for liposuction. J Dermatol Surg Oncol. 16(3):248–263
[28] Rohrich RJ, Broughton G , II, Horton B, Lipschitz A, Kenkel JM, Brown SA. The key to long-term success in liposuction: a guide for plastic surgeons and patients. Plast Reconstr Surg. 114(7):1945–1952, discussion 1953

44 Abdominoplasty

Bruce A. Mast

Abstract
This chapter looks at the various ways of correcting contour irregularities of the abdominal wall. Matters to be determined in the presurgical exam (degree/extent of adiposity, redundant skin, laxity of musculofascial units, etc.) are reviewed, and surgical options for every finding are discussed. Treatment options fall under two broad categories: complete/full abdominoplasty and mini-abdominoplasty; liposuction is also an option for contouring the upper abdomen and flanks. A thorough discussion of each major option lays out all techniques, contingencies, and options. Important information on postoperative care is provided, along with advice on potential complications.

Keywords: adiposity, full abdominoplasty, mini-abdominoplasty, lipoabdominoplasty, chemoprophylaxis

44.1 Goals and Objectives

- Understand the proper evaluation of prospective abdominal contouring patients.
- Clearly define the indications for the various type of excision contouring procedures of the abdominal wall.
- Appreciate the technical aspects of addressing each anatomic component of the abdominal wall required for proper contouring.
- Know the evidence-based perioperative care to maximize patient safety and quality outcomes.

44.2 Patient Presentation

Patients with contour irregularities of the abdominal wall present with varied histories and physical findings. Some patients have had a massive weight loss from bariatric surgery or nonsurgical efforts. Others have had more modest weight loss, while many have had changes of weight and skin quality related to aging, pregnancy, and surgery. Accordingly, upon initial presentation, mandatory historical information includes changes in weight, stability of weight loss, desire for future pregnancy, history of abdominal surgery, and overall medical evaluation assessing for comorbidities such as diabetes, coronary disease, and the use of antiplatelet and anticoagulant medications. Each patient needs to be carefully assessed, and based on the characteristics of each individual, the proper type of contouring procedure can be provided.[1]

The physical assessment must evaluate the component irregularities of the abdominal wall: (1) degree and extent of adiposity, (2) quantity and location of loose or redundant skin, and (3) laxity of the musculofascial units.[2] Key in the examination is the determination of abdominal wall hernia or abdominal wall ports such as those used for gastric banding. Full examination of the patient's abdomen is done in several positions: supine, sitting, standing, and the diving position. This provides a full appreciation of the degree of excessive soft tissue and the extent of musculofascial laxity. Careful palpation of the abdomen in the supine position allows assessment of rectus diastasis and the superior extent of the diastasis. It further permits detection of abdominal wall hernias. The quantity of excess tissue is confirmed with the patient supine while downward traction is asserted to the abdominal skin. This will permit fairly accurate assessment of the amount of skin to be removed and whether a full or mini-abdominoplasty would be appropriate. While standing, general visual inspection provides assessment of overall contours and areas of disproportionate fat distribution and skin excess or laxity. The sitting position will help delineate the extent of excessive or loose soft tissue. The diving, or waist-flexed, position allows the loose skin to fall away from the abdominal wall and provide further assessment.

When the affected anatomic components affect the majority of the abdominal wall including the supraumbilical region, a full abdominoplasty with or without liposuction is usually indicated (▶ Table 44.1; ▶ Fig. 44.1). For individuals with more limited or isolated deformities in the lower abdomen, effective treatment can be provided by mini-abdominoplasty, with or without liposuction of the abdominal wall or flank (▶ Table 44.2; ▶ Fig. 44.2; ▶ Fig. 44.3; ▶ Fig. 44.4).[3] Mini-abdominoplasty can provide these patients with excellent correction of their abdominal deformities with significantly less morbidity and faster recovery than that which is associated with a full abdominoplasty.[4,5]

Mini-abdominoplasty can also offer an understandable compromise for the patient who is not an ideal candidate for mini-abdominoplasty, but is unwilling to put forth the physical or financial outlay necessitated by the full abdominoplasty.[6]

Table 44.1 Ideal candidate for full abdominoplasty

Clinical feature	Description
Body type	Fit, but possibly mildly overweight
Abdominal contour	Excessive or disproportionate upper abdominal bulging
Excess tissue	Includes upper abdomen or entire abdomen
Lipodystrophy	Varies, but may involve the entire abdomen and flanks
Musculofascial laxity	Involves supraumbilical abdomen Incisional/ventral hernia
Rectus diastasis	Extends well above umbilicus

Table 44.2 Ideal candidate for mini-abdominoplasty

Clinical feature	Description
Body type	Overall fit and trim; not obese
Abdominal contour	Lower, infraumbilical bulge
Excess tissue	Confined to lower abdomen
Lipodystrophy	Varies, but mild to moderate
Musculofascial laxity	Lower abdomen only
Rectus diastasis	No more than 2 cm above umbilicus

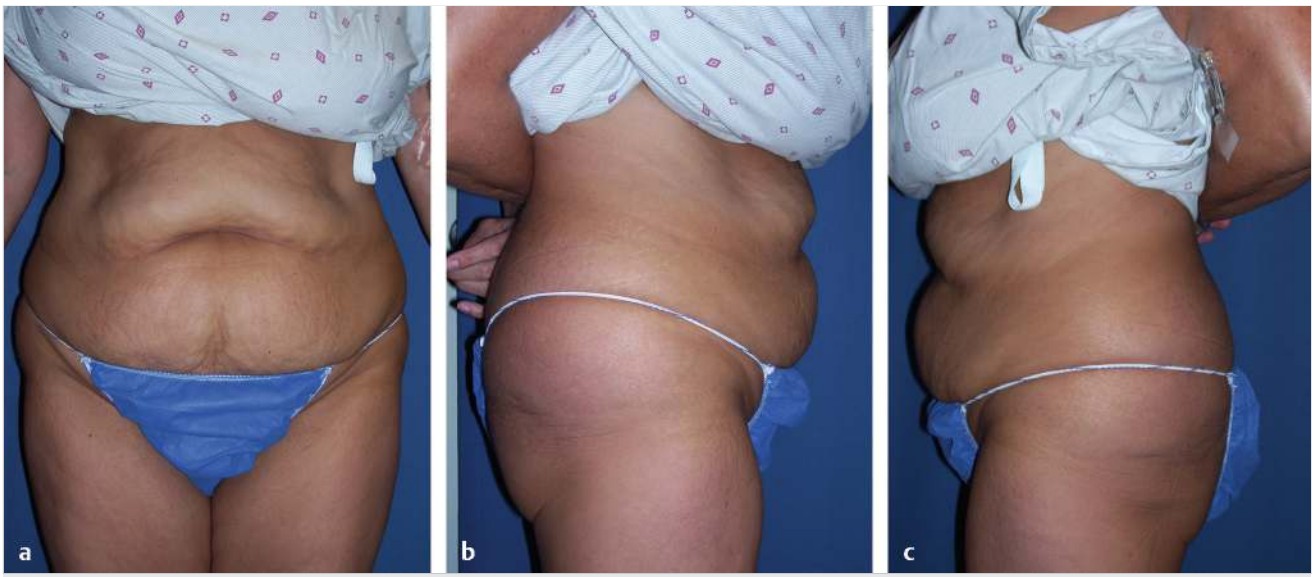


Fig. 44.1 (a–c) Patient with features best treated with lipoabdominoplasty: skin laxity in upper and lower abdomen, combined with lipodystrophy throughout.

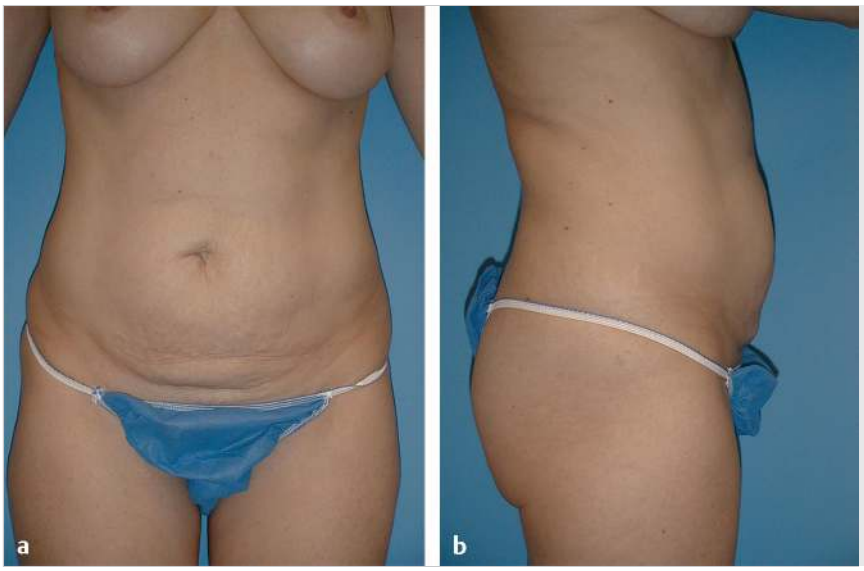


Fig. 44.2 (a,b) Patient with features best treated by mini-abdominoplasty: infraumbilical muscular laxity and mild degree of excess infraumbilical soft tissue and no lipodystrophy.

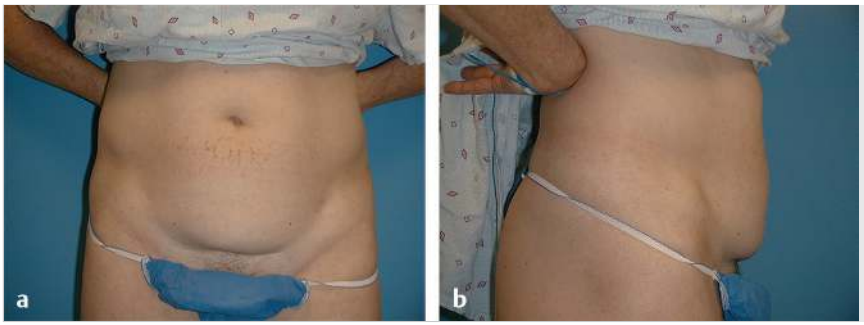


Fig. 44.3 (a,b) Patient with features best treated by mini-abdominoplasty combined with limited liposuction: infraumbilical muscular laxity and mild degree of excess infraumbilical soft tissue and lipodystrophy of the midabdomen and flanks.

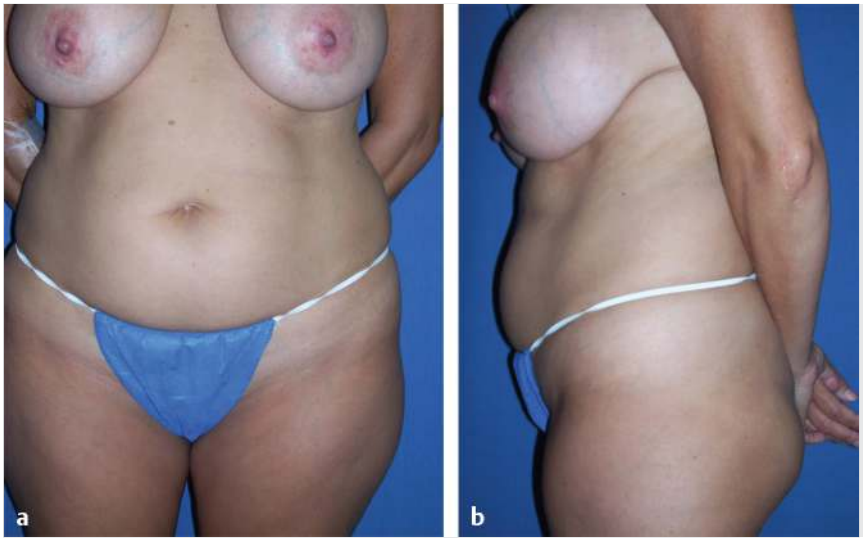


Fig. 44.4 Patient with infraumbilical muscular laxity, mild excess skin but more substantial adiposity throughout the entire abdominal wall and flanks. Best treatment option is aggressive liposuction of the entire abdomen and flanks, combined with mini-abdominoplasty.

Patients with adiposity without skin laxity or significant muscle laxity can be treated with liposuction alone and no skin/soft tissue resection.

44.3 Preparation for Surgery

Diagnostic data in preparation for surgery would be similar for other elective major procedures and would be dependent on age, presence of comorbidities, and requirements of the surgical center in which the procedure will be performed. In general, the author requires a baseline hemoglobin and hematocrit on most patients. Massive weight loss patients also have nutritional parameters assessed via serum albumin level. Iron deficiency anemia is not uncommon in gastric bypass patients and requires correction prior to the surgery. Nongastric bypass patients with anemia require full workup for occult blood loss. Hypoproteinemia should be corrected via high protein diets and subsequent documented correction. Radiographs are not required unless the procedure is being done in combination with large hernia repair. In such a case, computed tomography would be used for assessment of the abdominal wall defect. Cardiac workup is dependent on patient age, history, and the facility in which the procedure is being done. In general, if an electrocardiogram is abnormal, internal medicine or cardiology clearance is mandatory.

44.4 Treatment

44.4.1 Treatment Options and Indications

The choices for surgical treatment fall broadly under two categories:

1. Complete or full abdominoplasty involving infraumbilical soft tissue resection, complete correction of musculofascial laxity, and umbilical translocation and inset. Most abdominoplasty patients fall into this category with laxity and excess of skin that then extends above the umbilicus and muscle laxity that is diffuse, combined with a long-length rectus diastasis. As a general guideline, ptosis of supraumbilical skin over the umbilicus is indicative of excessive laxity of the upper abdominal soft tissue that would not be fully corrected with a standard mini-abdominoplasty (▶ Table 44.1; ▶ Fig. 44.1).
2. Mini-abdominoplasty in which tissue dissection and resection as well as rectus plication are significantly more limited to the inferior abdominal wall and the umbilicus is not translocated. Ideal candidates have loose skin confined to the infraumbilical abdomen. Diastasis of the rectus is limited to the infraumbilical region or only 1 or 2 cm superiorly. Overall muscular tone of the abdomen is good (▶ Fig. 44.2).
3. Liposuction can be added for contouring of the upper abdomen and flanks. When done cautiously, this can be combined with both full and mini-abdominoplasties with safe and reliable results (▶ Table 44.2; ▶ Fig. 44.2; ▶ Fig. 44.3; ▶ Fig. 44.4).[7]

44.4.2 Surgical Preparation

Historically, complete contouring via full abdominoplasties has been an inpatient procedure. However, outpatient surgery in properly selected patients has proven to be safe and effective.[8,9] Outpatient candidates should have limited or no comorbidities and as such are designated American Association of Anesthesiology Class I or II. Patients with systemic diseases, such as insulin-dependent diabetes, may categorize as Class III, and such patients are best treated in the hospital setting. Whether done as an inpatient or outpatient, effective control of postoperative nausea is needed. Preoperative medications such as ondansetron (Zofran) and aprepitant (Emend) have proven efficacy. Continuous mechanical intraoperative prevention via sequential compression boots is mandated in all abdominoplasty operations done under general anesthesia. Additionally, chemoprophylaxis for thromboembolic complications is advised. The American Association of Plastic Surgeons provides practice guidelines that encourage abdominoplasty patients to be treated with postoperative heparin. Preoperative heparin chemoprophylaxis continued postoperatively has also been proven safe and maximizes prophylaxis.

Most abdominoplasty procedures are done under general anesthesia. Choice of anesthesia is dependent on the individual patient, as well as the facility in which the surgery will be done. When combined with liposuction, breast surgery, or another procedure, the total length of the procedure must be considered, affecting the selection of anesthetic, but also mandating consideration of prophylaxis for deep venous thrombosis and the use of body warming devices. Mini-abdominoplasty can be performed under intravenous sedation combined with local anesthetic.[10,11,12,13] In the latter technique, bilateral ilioinguinal nerve blocks are administered combined with direct infiltration of the incision line with local anesthetic. Tumescent liposuction solution consisting of 0.01% lidocaine with 1:000,000 epinephrine is also infiltrated into the lower abdominal wall in the region of flap elevation, in addition to the regions to be treated by liposuction. This augments the anesthetic effect and provides greater patient comfort.[14,15] The use of epidural anesthesia has been sporadically reported, but the potential complications as well as possible prolongation of predischarge recovery must be considered. If the operation to be performed is only a mini-abdominoplasty, then intravenous sedation combined with local anesthetic is easily applicable.

As with all body contouring procedures, the patient is marked preoperatively. Markings are started in the standing position. The proposed placement of the scar is confirmed with the patient such that it will be positioned within the panty line or swimsuit line. The transverse pubic incision is marked in the region of the superior aspect of the pubic hairline. This mark should be no higher than 6 to 8 cm above the superior aspect of the introitus in the midline. If a Pfannenstiel scar is present, the incision line should be inferior to it so that the associated subcutaneous fibrosis that tethers the skin to the fascia can be removed. For full abdominoplasty, the incision extends laterally along the inguinal crease past the anterior superior iliac spine to the position in which skin redundancy tapers away. The mini-abdominoplasty incision line is limited by the extent of tissue excess to be removed. The upper excision line for the full abdominoplasty begins centrally at the supraumbilical border and is carried laterally to meet the inferior line toward the flanks, judging the amount of skin to be removed by downward transection on the infraumbilical skin. For mini-abdominoplasty, the superior incision line is not drawn until the flap is raised intraoperatively. However, for purposes of planning, the skin to be excised usually encompasses about one-third to one-half of the vertical height of the skin between the pubis and the umbilicus. If liposuction is planned for the upper abdomen, flanks, iliac crests, or thighs, these areas are also marked while the patient is standing. At completion, all the markings should be demonstrated and explained to the patient with confirmation of proposed scar placement. However, it should be stressed to the patient that the final position of the scar cannot be guaranteed, rather best efforts are put forth to place the scar favorably.

44.5 Operative Technique

44.5.1 Full Abdominoplasty

The components of abdominoplasty technique address multiple key anatomic features for restoration of abdominal shape and form.[16] The procedure is most often done in combination with upper abdominal and flank/iliac crest liposuction, as well as shaping of the muscular component of the abdominal wall. The procedure is most often commenced with liposuction. After appropriate institution of anesthetic support, cannula insertion incisions are made in the supraumbilical border and the lower abdomen, bilaterally within the tissue to be resected. Infiltration fluid for superwet liposuction need only contain epinephrine (1 mg epinephrine in 10,000 mL of crystalloid solution) if general anesthesia is used. While awaiting the hemostatic effect of the solution, the lower abdominal incision is created and carried through Scarpa's fascia down to the loose areolar plane just superficial to the anterior rectus sheath and external oblique fascial aponeuroses. The skin and subcutaneous tissues are then elevated off the abdominal fascia superiorly and laterally to just below the level of the umbilicus. Liposuction is then done in the deep and superficial planes providing effective contouring. Suction of the upper lateral abdomen can be done aggressively as long as subsequent soft tissue dissection is done properly with perforator preservation (see below). This technique is frequently referred to as a lipoabdominoplasty. If an abdominal wall hernia is present, this must be repaired prior to the liposuction to avoid intestinal injury.

The umbilicus is circumferentially incised when the liposuction is complete. Dissection around the umbilical stalk is carried down to the muscle fascia. The abdominal skin/soft tissue flap is then dissected superior to the umbilicus, leaving the umbilicus attached to the abdominal wall. Dissection in the midline is then carried toward the xiphoid, ceasing when the entire diastasis is visualized or the skin laxity is mobilized. Dissection of the skin/soft tissue lateral to the midline is limited to the extent that permits plication of the rectus diastasis and proper tissue mobilization for contouring. When combined with liposuction, it is essential that the perforators from the rectus muscle are maximally preserved during this dissection so as to avoid inferior skin necrosis.

Dissection and retraction of the abdominal skin flap permits inspection of the musculofascial component of the abdominal wall and the anatomic cause of laxity that contributes to the convex external contour. The laxity is corrected by musculofascial plication, providing reconstruction of the correct anatomic relationships of the abdominal wall musculature. This should result in parallel alignment of the rectus muscles with a straight-line juxtaposition at the linea alba. The plication lines on the anterior rectus sheaths are marked to guide suture placement. The elliptical or crescent marks come together in the midline just above the suprapubic incision line and meet superiorly at the upper aspect of the diastasis (▶ Fig. 44.5; ▶ Fig. 44.6). The author's preferred method of plication is with inverted figure-of-eight braided nylon sutures. These permanent soft sutures require few knots, avoiding postoperative palpability, even in the thinnest patients. Alternatively, a running permanent monofilament suture can be used. Others have described methods of vertical and transverse plication that may be applicable when the extent of laxity is not fully corrected by a standard midline plication.[17] If the external obliques are laterally displaced in patients with exceptional muscular laxity, medial advancement of the oblique is done with running 2–0 polypropylene. This further shapes the abdomen and facilitates creation of a waistline contour (▶ Fig. 44.5; ▶ Fig. 44.6).

Fig. 44.5 Markings for muscle plication. Centrally the midline diastasis recti is marked. Crescent markings bilateral are for external oblique advancement.

Fig. 44.6 Midline plication of the diastasis recti, and medial advancement of bilateral external obliques.

At the completion of midline muscle plication, bilateral transverse abdominis plane (TAP) blocks are administered. This places local anesthetic into the plane deep to the internal oblique and anesthetizes the sensory innervation to the abdominal wall. This can be done under ultrasound guidance, limited open method with an oblique muscle-splitting technique, or a closed means. The author prefers the closed method in which a blunted needle is inserted just lateral to the linea semilunaris marking the junction of the external oblique to the rectus fascia. This is more readily accomplished prior to external oblique advancement, if needed. The needle is inserted with firm, but gentle pressure, and a double pop is appreciated as the needle passes through the fascia of the external and then internal oblique and the TAP is accessed. This is done along the entire length of the abdomen bilaterally with incremental injection of the local anesthetic. Long duration agents are used, such as bupivacaine or ropivacaine in appropriate dosing.[18] This can also be done before or after surgery by the anesthesiologist transcutaneously with ultrasound guidance.

Following plication, the excessive skin and subcutaneous tissue is retracted inferiorly with minimal tension to overlap the lower incision line. The midline point of overlap with the transpubic incision is marked and the flap is incised longitudinally in the midline. This point is secured temporarily to the lower incision line in the midline. This leaves two lateral "triangles" of excess tissue that can be appropriately marked and excised (▶ Fig. 44.7).

Aggressive skin excision that results in a very tight closure should be avoided since excessive tension will contribute to greater discomfort, wound dehiscence, and unfavorable scarring. Also, this will frequently cause gradual superior drift of the scar and subsequent elongation of the pubic hair domain superiorly. To avoid these results, skin excision is judged with the operating table only modestly flexed. It is occasionally

Fig. 44.7 Abdominal skin excision is done via flap advancement over the lower pubic incision with temporary fixation in the midline. The remaining skin bilaterally is then advanced and marked for excision at the line of overlap with the incision line. In this photo, the skin from the left side of the abdominal flap has been excised, and the triangle of skin to be removed on the right is noted and marked.

necessary to leave a small vertical scar in the midline just at the junction of the lower transverse scar, corresponding to the original umbilical site.

Closed suction drains are often placed, but in select patients quilting sutures can be used to eliminate dead space with the added advantage of progressive tension reduction on the skin closure.[19] Prior to closure, the author favors the use of pain pumps that instill 0.25% bupivacaine without epinephrine for 2 to 3 days.

The position of the umbilicus is marked via direct palpation with the flap secured in position but prior to skin closure. As a confirmatory guideline, the proper umbilical position is in the midline at the level of the iliac crests. An inverted triangle of skin and underlying fat is then removed at this site. The author favors this over a circular excision since native umbilici are seldom perfectly circular, and circular insets tend to contract with time. The umbilicus is inset with an inferior midline absorbable monofilament that incorporates the abdominal fascia. This suture pulls the inferior inset closer to the abdominal wall and provides a downward slope of the umbilicus providing an aesthetically pleasing shape. The remaining inset is with absorbable monofilament suture and skin glue.

Tissue approximation is done in layers. Scarpa's fascia, including the subcutaneous aponeurosis, is approximated with interrupted absorbable suture. The central, transpubic sutures also incorporate the abdominal fascia just above the pubis, acting to stabilize the position of the scar and prevent superior migration with time. The skin is closed with subdermal absorbable monofilament and skin adhesive. The soft tissues surrounding the suture line are infiltrated with bupivacaine without epinephrine to provide postoperative analgesia.

44.5.2 Mini-Abdominoplasty

Mini-abdominoplasty is a procedure in which infraumbilical skin is excised and the umbilicus is not translocated. The extent of skin excision varies with individual circumstances and in most cases liposuction is also done. The procedure is commenced in a similar manner as when doing a full abdominoplasty. Liposuction can be done more aggressively to the upper abdomen due to the limited upper abdominal tissue dissection and disruption of perforating blood supply. Additionally, unlike full abdominoplasty, the infra- and periumbilical regions are usually suctioned. As such, suction is usually done prior to elevation of the lower abdominal flap, since this more readily allows the tumescent solution to remain in place without being adjacent to a large open surgical site, through which the fluid can easily egress. The entire upper abdomen extending laterally can be suctioned fairly aggressively since the abdominal flap will not be elevated much above the umbilicus, allowing preservation of perforating vessels. Upon completion of the liposuction, the abdominal incision is created and the infraumbilical skin and subcutaneous tissues are elevated off the abdominal fascia similar to a full abdominoplasty. The superior extent of mobilization is usually the umbilical base, but may extend a few centimeters above the umbilicus. If supraumbilical dissection is necessary, the umbilicus remains attached to the abdominal skin and the muscular wall, and care must be exercised around the umbilical stalk to avoid devascularization. Lateral mobilization of the flap is continued to about the level of the anterior axillary line. If the anesthesia being used is intravenous sedation with local anesthetic, electrocautery should be avoided in ligation and division of larger perforating vessels since these often contain sizable sensory nerves. The electrical current is transmitted into these nerves causing severe pain, despite the rest of the field being completely anesthetic. In such circumstances, suture ligation and sharp division should be employed. These procedures will allow sufficient anterior retraction of the skin unit such that abdominal wall plication can be done under direct visualization.

Rectus diastasis plication is done as in a full abdominoplasty. If diastasis extends well above the umbilicus, a superior periumbilical incision can be made with subsequent dissection of the central supraumbilical skin off the fascia. This will permit direct visualization for suture plication without full tissue dissection in the absence of superior redundant skin. Correction of the inferior diastasis must be done with care so as to avoid bulging of the upper abdomen. This area may now have a relative laxity if the inferior diastasis is corrected aggressively. A

balance between the upper and lower abdominal wall musculofascial unit must be achieved.

Following plication, the excessive infraumbilical skin and subcutaneous tissue is retracted inferiorly, marked and excised in a similar manner as for full abdominoplasty. Aggressive excision that results in a very tight closure should be avoided. This could cause gross distortion of the umbilicus as well as unfavorable scarring due to tension and lead to superior migration of the scar as the looser pubic soft tissue is "pulled" upward. To avoid these results, skin excision is judged with the patient nearly flat with the operating table barely flexed. Closure is then done is a manner similar to the full abdominoplasty. In all cases, the umbilicus will be distorted to some degree, usually with a slight vertically elongated appearance (▶ Fig. 44.4).

An umbilicus to pubis distance of 9 cm is a good parameter to use for a "normal" anatomic relationship that provides a pleasing appearance. If this distance is significantly less than 9 cm, particularly in shorter patients, then translocation to the appropriate position through a vertical, elliptical, midline incision may be necessary. Overall appearance within the parameters of the patient's particular body habitus dictates the necessity of umbilical translocation, and preoperative assessment should allow counseling to alert the patient to the potential need for this maneuver and the resultant scar.

44.6 Postoperative Care

An abdominal binder or appropriate liposuction compression garment is placed after the surgery. For outpatient procedures, the patient is discharged home after postanesthesia criteria are met and instructed to leave the dressings and compression in place until seen for the first postoperative office visit, within 48 hours of the procedure. If surgery is done as an inpatient, discharge from the facility is usually the next morning. Surgical drains are removed when output is less than 30 mL/day for two consecutive days. Pain pump catheters are removed when empty, usually within 72 hours. The patient is instructed to avoid strenuous activities and heavy lifting for at least 1 month after surgery. After 1 month of healing, the patient is permitted to gradually resume a workout regimen and is cleared for all activities as tolerated after 6 weeks of healing. Patients undergoing complete abdominoplasty can usually return to office-based work in 2 weeks, and more vigorous occupations at 3 to 4 weeks. Mini-abdominoplasty patients generally have about 50% less recovery time in this regard.

Postoperative pain relief is provided in multimodality fashion. The author prefers use of bupivacaine pain pumps placed at surgery. Narcotic analgesics are necessary with provision of hydrocodone or oxycodone, supplemented with low-dose hydromorphone (2–4 mg) for breakthrough pain. Additionally, low-dose diazepam (2 mg) is used for muscle spasticity which often occurs after abdominal wall plication. Postoperative nausea is controlled with promethazine or ondansetron. Meclizine is most effective at treating postoperative positional nausea.

Postoperative venous thromboembolism prophylaxis should be continued. If the patient remains within the facility, then sequential compression boots should remain operational continuously until discharged. For patients undergoing full abdominoplasty, chemoprophylaxis should be provided with low-molecular-weight heparin. This can be done as an outpatient with a home prescription, available through most pharmacies. The author continues the chemoprophylaxis for 48 hours postoperatively, during the time of maximum immobilization. Additionally, urinary catheters, if used during the procedure, are removed promptly to encourage mobilization.

Careful preoperative counseling is necessary regarding position and quality of typical scars. Maximum scar quality is achieved by avoidance of excessive tension at closure, complete elimination of ultraviolet radiation (including tanning booths) for at least 6 months following surgery, and the use of various scar lotions/gels or silicone patching for scar management. Occasionally, steroid injections or surgical scar revision is needed (▶ Fig. 44.8; ▶ Fig. 44.9; ▶ Fig. 44.10).

44.7 Outcomes

As with any significant surgical procedure, complications or adverse outcomes are inevitable. Similar complications occur for all abdominal body contouring procedures, but occur with less frequency in mini-abdominoplasties due to the lesser magnitude of the surgery. Large national database evaluations show that major complications following abdominoplasty are infrequent and certainly acceptable for elective procedures: 0.9% for hematoma, 3.5% for infection, and 0.3% for deep venous

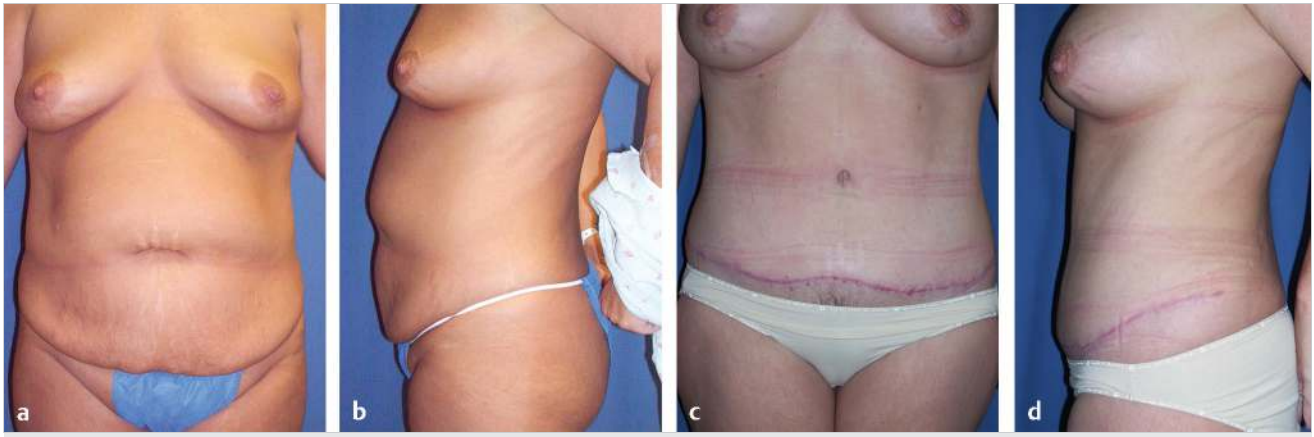


Fig. 44.8 **(a,b)** Prior to full lipoabdominoplasty. **(c,d)** 3.5 months after lipoabdominoplasty.

Fig. 44.9 Same patient in ▶ Fig. 44.3, approximately 4 months after mini-abdominoplasty and limited midabdominal and flank liposuction.

Fig. 44.10 **(a)** Same patient in ▶ Fig. 44.4. **(b)** 4 months after mini-abdominoplasty and abdominal and flank liposuction. Note the elongation of the umbilicus caused by the inferior retraction of the skin.

thrombosis/pulmonary embolism.[20] Seromas occur in approximately 15% of patients, and appear to have increased incidence when liposuction of the abdomen or flanks is part of the procedure.[21] The use of drains or quilting sutures seems to have little effect on the overall seroma rate.[22,23] Most seromas are treated by percutaneous aspiration and occasionally new drain placement is required. Interestingly, overall complications rates do not correlate with years in practice of the operating surgeon.[24]

Abdominoplasty surgery is the aesthetic procedure most often associated with thromboembolic complications. Although the incidence varies in studies, the likely most accurate assessment is an incidence of less than 1%.[25] Despite this very low risk, the complication can be devastating, and as such prophylaxis should be maximized. As stated above, chemoprophylaxis is recommended postoperatively for full abdominoplasty procedures, particularly when done under general anesthetic. Preoperative chemoprophylaxis has also been shown to be safe without increased incidence of hematoma.[26]

Compared to other aesthetic procedures, abdominoplasties are associated with higher complication rates. In the analysis of the CosmetAssure national database, 4.0% of abdominoplasties had a complication, compared to 1.4% of other aesthetic procedures.[27] The study identified risk factors for complications as being male sex, age 55 years or older, body mass index of 30 or greater, surgery done in hospital or ambulatory surgery center rather than an office-based suite, and multiple procedures. An abdominoplasty without any other procedure had a complication rate of 3.1%, with combined procedures ranging up to 10.4%. Nevertheless, in properly selected patients, combining abdominal contouring with other procedures is considered safe.

Patient satisfaction outcomes are important to evaluate, particularly in elective, aesthetic procedures. Indeed, objective assessment has shown that after surgery abdominoplasty patients have enhanced body image and self-awareness as demonstrated by improved satisfaction with their overall appearance, as well as less self-consciousness and avoidance of body exposure during sexual activities.[28] Overall satisfaction is generally very high with the vast majority of patients stating they would do it all over again, and would recommend the procedure to others.[29] As with all body contouring procedures, it is

essential that the patient maintains a healthy lifestyle after surgery to have long-lasting results and satisfaction.[30] Patients need to be reminded of this, since some have reduced dieting and exercising with the notion that it is no longer needed, since their "problem" areas have now been taken away. Such patients are destined for weight gain and disappointment.

Abdominoplasty procedures are highly effective in properly selected patients providing a high level of satisfaction with long-lasting results and minimal risk.

44.8 Review Questions

44.8.1 True or False

1. Complete or full abdominoplasty can safely be done as outpatient surgery.
2. Chemoprophylaxis with heparin is contraindicated in abdominoplasty surgery due to high risk of hematoma.
3. Midline rectus muscle plication is the only means for correcting muscle laxity.

44.8.2 Choose the Best Answer

4. A 36-year-old woman is 2 years s/p delivery of twins and plans no further pregnancies. Her exam shows loose abdominal skin extending to the mid upper abdomen, a notable bulging of periumbilical abdomen, 5-cm diastasis recti for nearly the entire length of the abdomen, and moderate fat accumulation in the flanks and less so in the upper abdomen. Her body mass index is 27. The best treatment option for her is
   a) Mini-abdominoplasty.
   b) Physical therapy and exercise/dieting.
   c) Lipoabdominoplasty.
   d) Liposuction.
5. Postoperative pain management:
   a) Requires multimodality approach to include analgesics and antispasmodics.
   b) Is unaffected by liposomal bupivacaine.
   c) Is only effective with intravenous analgesia in an overnight facility.

44.8.3 Answers

1. True.
2. False.
3. False.
4. c. Lipoabdominoplasty.
5. a. Requires multimodality approach to include analgesics and antispasmodics.

References

[1] Sozer SO, Agullo FJ, Santillan AA, Wolf C. Decision making in abdominoplasty. Aesthetic Plast Surg. 31(2):117–127

[2] Matarasso A. Classification and patient selection in abdominoplasty. Oper Tech Plast Reconstr Surg. 3:7–14

[3] Shestak KC. Marriage abdominoplasty expands the mini-abdominoplasty concept. Plast Reconstr Surg. 103(3):1020–1031, discussion 1032–1035

[4] Greminger RF. The mini-abdominoplasty. Plast Reconstr Surg. 79(3):356–365

[5] Wilkinson TS, Swartz BE. Individual modifications in body contour surgery: the "limited" abdominoplasty. Plast Reconstr Surg. 77(5):779–784

[6] Mast BA, Hurwitz DJ. Mini-abdominoplasty. Oper Tech Plast Reconstr Surg. 3: 38–41

[7] Brauman D, Capocci J. Liposuction abdominoplasty: an advanced body contouring technique. Plast Reconstr Surg. 124(5):1685–1695

[8] Mast BA. Safety and efficacy of outpatient full abdominoplasty. Ann Plast Surg. 54(3):256–259

[9] Buchanan PJ, Nasajpour H, Mast BA. Safety and efficacy of outpatient lower body lifting. Ann Plast Surg. 70(5):493–496

[10] Byun MY, Fine NA, Lee JY, Mustoe TA. The clinical outcome of abdominoplasty performed under conscious sedation: increased use of fentanyl correlated with longer stay in outpatient unit. Plast Reconstr Surg. 103(4):1260–1266

[11] Bitar G, Mullis W, Jacobs W, et al. Safety and efficacy of office-based surgery with monitored anesthesia care/sedation in 4778 consecutive plastic surgery procedures. Plast Reconstr Surg. 111(1):150–156, discussion 157–158

[12] Byrd HS, Barton FE, Orenstein HH, et al. Safety and efficacy in an accredited outpatient plastic surgery facility: a review of 5316 consecutive cases. Plast Reconstr Surg. 112(2):636–641, discussion 642–646

[13] Rosenberg MH, Palaia DA, Bonanno PC. Abdominoplasty with procedural sedation and analgesia. Ann Plast Surg. 46(5):485–487

[14] Abramson DL. Tumescent abdominoplasty: an ambulatory office procedure. Aesthetic Plast Surg. 22(6):404–407

[15] Nguyen TT, Kim KA, Young RB. Tumescent mini abdominoplasty. Ann Plast Surg. 38(3):209–212

[16] Patronella CK. Redefining abdominal anatomy: 10 key elements for restoring form in abdominoplasty. Aesthet Surg J. 35(8):972–986

[17] Cardenas Restrepo JC, Munoz Ahmed JA. New technique of plication for mini-abdominoplasty. Plast Reconstr Surg. 109(3):1170–1177, discussion 1178–1190

[18] Fiala T. Tranversus abdominis plane block during abdominoplasty to improve postoperative patient comfort. Aesthet Surg J. 35(1):72–80

[19] Rodby KA, Stepniak J, Eisenhut N, Lentz CW , III. Abdominoplasty with suction undermining and plication of the superficial fascia without drains: a report of 113 consecutive patients. Plast Reconstr Surg. 128(4):973–981

[20] Alderman AK, Collins ED, Streu R, et al. Benchmarking outcomes in plastic surgery: national complication rates for abdominoplasty and breast augmentation. Plast Reconstr Surg. 124(6):2127–2133

[21] Neaman KC, Armstrong SD, Baca ME, Albert M, Vander Woude DL, Renucci JD. Outcomes of traditional cosmetic abdominoplasty in a community setting: a retrospective analysis of 1008 patients. Plast Reconstr Surg. 131(3):403e–410e

[22] Quaba AA, Conlin S, Quaba O. The no-drain, no-quilt abdominoplasty: a single-surgeon series of 271 patients. Plast Reconstr Surg. 135(3):751–760

[23] Bercial ME, Sabino Neto M, Calil JA, Rossetto LA, Ferreira LM. Suction drains, quilting sutures, and fibrin sealant in the prevention of seroma formation in abdominoplasty: which is the best strategy? Aesthetic Plast Surg. 36(2):370–373

[24] Matarasso A, Swift RW, Rankin M. Abdominoplasty and abdominal contour surgery: a national plastic surgery survey. Plast Reconstr Surg. 117(6):1797–1808

[25] Levesque AY, Daniels MA, Polynice A. Outpatient lipoabdominoplasty: review of the literature and practical considerations for safe practice. Aesthet Surg J. 33(7):1021–1029

[26] Campbell W, Pierson J, Cohen-Shohet R, Mast BA. Maximizing chemoprophylaxis against venous thromboembolism in abdominoplasty patients with the use of preoperative heparin administration. Ann Plast Surg. 72(6):S94–S96

[27] Winocour J, Gupta V, Ramirez JR, Shack RB, Grotting JC, Higdon KK. Abdominoplasty: risk factors, complication rates, and safety of combined procedures. Plast Reconstr Surg. 136(5):597e–606e

[28] Bolton MA, Pruzinsky T, Cash TF, Persing JA. Measuring outcomes in plastic surgery: body image and quality of life in abdominoplasty patients. Plast Reconstr Surg. 112(2):619–625, discussion 626–627

[29] Papadopulos NA, Staffler V, Mirceva V, et al. Does abdominoplasty have a positive influence on quality of life, self-esteem, and emotional stability? Plast Reconstr Surg. 129(6):957e–962e

[30] Rohrich RJ, Broughton G , II, Horton B, Lipschitz A, Kenkel JM, Brown SA. The key to long-term success in liposuction: a guide for plastic surgeons and patients. Plast Reconstr Surg. 114(7):1945–1952, discussion 1953

45 Lower Body Lift

Paul N. Afrooz and Jeffrey A. Gusenoff

Abstract
This chapter describes the procedures, technical aspects, and options for optimal lower body contouring. A lower body lift aims to remove excess skin of the abdomen, thighs, and buttocks (which often results from bariatric surgery) and contour these body parts in a more aesthetically pleasing way. Procedural options fall into two broad categories—belt lipectomy and lower body lift—and each step for each option is carefully described. Numerous images illustrate the techniques described in the text. The authors offer guidance on postoperative care and discuss possible outcomes, including potential complications.

Keywords: lower body lift, belt lipectomy, bariatric surgery, gluteal elongation, autoaugmentation, Lockwood type 1 and 2 lower body lifts

45.1 Goals and Objectives

- Understand the proper evaluation of prospective lower body lift patients.
- Clearly define the indications for the various types of lower body lift procedures.
- Appreciate the technical aspects and options available to achieve optimal contouring.
- Know the evidence-based perioperative care to maximize patient safety and quality outcomes.

45.2 Patient Presentation

As the number of bariatric procedures in the United States increases to temper the growing obesity epidemic, the desire for truncal body contouring procedures has increased. Redundant skin is often not just limited to the abdomen, but extends in a circumferential pattern around the outer thighs and buttocks. Lockwood described two main patterns of lower body lifts. Type 1 lower body lifts addressed the buttocks and merged into the inner thigh lift, while type 2 lower body lifts addressed the buttocks and abdomen in a circumferential fashion.[1,2]

A variety of previously described gluteal deformities occur with aging and massive weight loss (MWL).[3,4,5,6,7,8] In MWL patients, these deformities are typically due to atonic skin quality and a high degree of laxity. Common deformities include an enlarged buttock, a deflated and ptotic buttock with deficient gluteal volume, and gluteal flattening. Patients primarily seek restoration and enhancement of gluteal shape and projection. A variety of procedures are available to address the range of deformities with scar position being the key factor in determining the type of lift, and the obtainable result.

It is important to obtain a thorough weight loss history including maximum weight prior to weight loss, lowest weight, and weight at the time of presentation. Any fluctuations in weight over the past 3 months should be documented, as surgery should only be performed once the patient's weight loss has stabilized. Some degree of weight gain is expected after a nadir is reached following bariatric procedures. The type of weight loss including bariatric procedures versus diet and exercise is reviewed. Any risks of malnutrition are determined including dumping syndrome or poor daily protein intake (< 70–100 g a day for an average 70-kg person). A medical history including pre-weight loss and post-weight loss comorbidities is assessed to determine what issues have resolved or may still require medical attention. Medications, smoking history, and exercise evaluations are performed. A physical exam along with a discussion of the patient's goals and desires is required at the initial consultation. Repeat evaluation prior to surgery is recommended to review the potential risks of surgery and the surgical plan.

The clinical exam of the lower trunk should include the upper and lower abdomen, the umbilical region, the midline region in standing and supine position, the flanks, hips, lateral thighs, inner thighs, and the entire lower back and gluteal region. If an abdominoplasty is to be done concurrently, examination should note the presence or absence of abdominal wall hernia, as well as the location of a fill port in those who had undergone a gastric banding procedure. Careful attention should be directed to the location of back rolls, buttocks volume, and saddlebags. For patients with flank lipodystrophy or low back rolls, a belt lipectomy or circumferential body lift may be more appropriate, thereby placing the scar higher with less control of the lower buttocks and lateral thigh. However, if a patient has more buttocks ptosis and loss of volume, and desires a large skin excision or gluteal autoaugmentation, a more inferiorly based excision is more appropriate, thereby forfeiting control over lower back rolls (▶ Fig. 45.1; ▶ Fig. 45.2). Saddlebag deformities may be challenging, and in some cases may require a lower scar to achieve complete correction and avoid recurrence due to skin relaxation.[9]

The initial and subsequent consultations must involve a thorough and detailed explanation of the entire procedure including possible complications. Complications including wound dehiscence, seroma, delayed wound healing, fat necrosis, visible scars, recurrent skin laxity, blood clots, and lower extremity edema must be discussed preoperatively to temper postoperative disappointment. Optimizing body mass index prior to surgery is a key factor to avoiding postoperative complications.[10] Some authors recommend performing procedures only in those patients with body mass indices less than 35 kg/m^2; however, we believe that each patient has unique anatomic deformities, and should be evaluated on an individual basis to determine their candidacy, rather than preclusion based on body mass index. Gluteal autoaugmentation may lead to an increase in the rate of wound dehiscence or delayed wound healing, but may be worth these minor complications in order to achieve the increased postoperative projection that is otherwise difficult to achieve once the volume has been resected.[11]

Fig. 45.1 **(a)** This patient exhibits significant flank rolls. A more superiorly based belt lipectomy was employed to address the flank rolls and excess lower back tissue. **(b)** Final scar position following belt lipectomy.

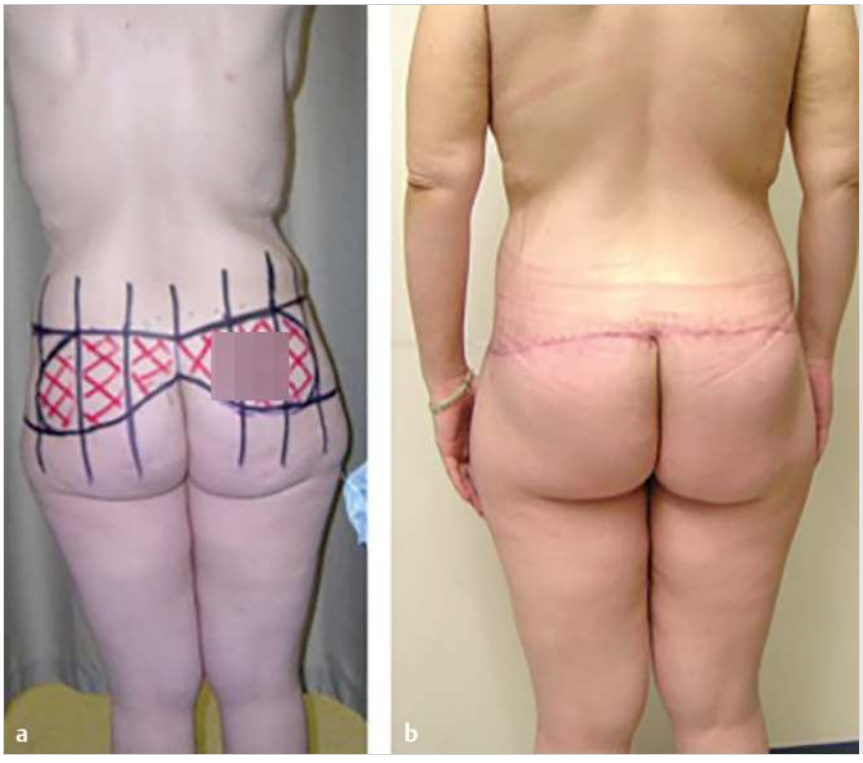


Fig. 45.2 **(a)** This patient exhibits mild buttock ptosis and volume loss. A more inferiorly based lower body lift was employed to address the buttock. **(b)** Final scar position following lower body lift.

45.3 Preparation for Surgery

Most patients should be at least a year out from bariatric procedures and weight stable for at least 3 months.[12,13] Preoperative laboratory data are performed on massive weight loss patients, including a complete metabolic panel, complete blood count, and coagulation parameters. Albumin and prealbumin are obtained if there is concern for malnutrition. Preoperative anemia is suspected in bariatric surgery patients who are not taking iron or vitamin B12 supplementation. If blood work confirms anemia, it must be treated prior to any elective surgery with iron deficiency being the most common type of anemia. Consultation with medical experts in hematology, cardiology, medicine, and psychiatry may be necessary for patients with known preexisting conditions.

Smoking cessation must occur at least 4 weeks prior to surgery, and patients are informed of the increased risks of wound complications secondary to nicotine. Urine cotinine is assessed preoperatively and surgery is canceled in the event of a positive urine cotinine test. Patients should refrain from smoking for 4 weeks after surgery.

Some studies have reported the increased risk of thromboembolism in lower body lift procedures.[14] Chemoprophylaxis should be strongly considered in patients with increased age, residual obesity, varicosities, and long operative procedures, which are common in most massive weight loss patients. Sequential compression stockings are initiated preoperatively and early ambulation is encouraged the evening of surgery.

45.4 Treatment

45.4.1 Treatment Options and Indications

Lower body lift procedures fall under two broad categories (▶ Fig. 45.1; ▶ Fig. 45.2):

1. Belt lipectomy: a superiorly based resection that will directly excise flank rolls and accentuate the waistline.
2. Lower body lift: an inferiorly based resection that will implement a stronger elevation on the lateral thighs and gluteal tissues with the ability to provide additional contour using autologous gluteal flaps.

These procedures are often combined with other areas of skin laxity including the inner thighs (Lockwood type 1 lower body lift) or with the abdomen (Lockwood type 2 lower body lift). In patients who have had prior abdominal or inner thigh contouring, the lower body lift procedure can be performed in isolation and may be an easier recovery than performing multiple procedures at the same time.[15] Staging of lower body procedures in patients with excess of the abdomen, buttocks, and outer and inner thighs can be challenging. It is our preference to combine the lower body lift with an abdominoplasty in the first stage and return for an inner thigh lift in the second stage.[15] This is because as the tissues relax from the lower body lift procedure, the skin laxity will rotate down and inward toward the inner thigh. Excess relaxed skin can then be removed with the full-scar vertical medial thigh plasty procedure. In addition, performing the abdominoplasty first may have a slight lifting effect on the upper part of the thigh, which may limit the length of the medial thigh scarring necessary in the second stage.

45.4.2 Surgical Preparation

An isolated lower body lift that does not combine with an abdominal or inner thigh procedure could be performed in an outpatient setting. Patients combining the procedure with an abdominoplasty or thigh plasty are often encouraged to stay two nights in the hospital. These are often lengthy procedures, and ambulation the evening of surgery as well as pain control may be difficult without appropriate nursing care. However, in selected patients, outpatient surgery has shown to be safe in these combined procedures.[16]

Patient motivation is an important factor in planning the postoperative course. Preoperative chemoprophylaxis and sequential compression stockings are utilized, and chemoprophylaxis continues during the hospital stay until the time of discharge. It is only continued in patients with known coagulopathies in conjunction with recommendations from their hematologist.

Scar placement is critical in preoperative surgical planning. For patients with minimal laxity, precision lower body lifting can be achieved by having the patient wear underwear at the time of marking. The borders of the undergarment can be marked and the incision line can often be concealed within the confines of the garment. This may not be achievable in patients with more superiorly based excisions to address the flanks, or more inferiorly based excisions addressing the saddlebags.

For patients undergoing simultaneous abdominal contouring, the markings begin with the patient in the supine position. With upward stretch on the lower abdominal tissue, a marking is placed 6 cm superior to the anterior vulvar commissure or the base of the penis. This point should rise just above the pubic symphysis when placed on stretch, but may not in some patients with a long torso. In such cases, this marking is moved superiorly by 1 to 2 cm until it is just above the pubic symphysis. The markings continue with the patient standing and facing away from the surgeon.

The first critical decision in posterior design is selecting the superior anchor line. This will be the superior line of incision. This line can be manipulated in the superior and inferior direction in order to achieve the desired effect of either greater waist contouring or greater gluteal and lateral thigh contouring, respectively. The superior anchor line is designed by carrying out the central point to a lateral point on the hip near the posterior superior iliac spine where the body transitions in a sinusoidal fashion. Vertical reference marks are placed at 6-cm intervals to aid in symmetry and final tissue reapproximation. Next, a pinch test is used to elevate the inferior tissues to the superior anchor line to estimate the amount of tissue resection. This involves rolling the inferior tissues under the superior anchor line to most accurately estimate the amount of tissue resection. The lateral tissues are estimated and marked with the legs slightly abducted to facilitate an optimal resection. Each estimated point of resection is marked along the vertical hash marks. These marks are then used to draw the inferior line of resection (▶ Fig. 45.3). If it can be avoided, the inferior line of excision should not violate the native gluteal cleft, as this may cause a direct elevation and undesirable lengthening of the

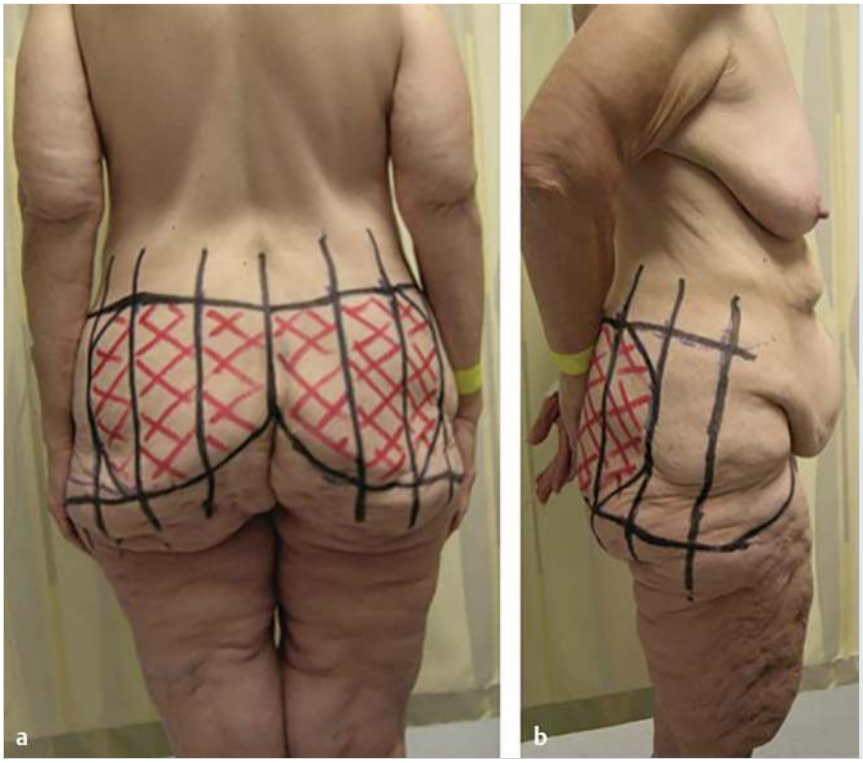


Fig. 45.3 (a,b) Posterior and lateral views of lower body lift markings. The superior line is selected followed by vertical reference marks at 6-cm intervals. Pinch test is used to estimate the amount of tissue resection at each vertical hash. These estimates at each vertical hash are then connected to mark the estimated inferior line of excision. The lateral tissues are marked with the legs in slight abduction to facilitate optimal resection. Notice the red hash marks carried out to the third vertical hash marks representing the area of de-epithelialization for gluteal autoaugmentation.

gluteal cleft postoperatively.[17] The lateral margin of resection is then selected connecting the inferior and superior lines of incision, and is usually at the level of the fourth vertical bar. This point typically coincides with the midaxillary line, and will be the transition zone between the posterior and anterior resections.

The overlying concept in the lower body lift is a repositioning and recontouring of gluteal tissue rather than simply resection of tissue. Therefore, at this point, an estimation is made regarding the amount of adipose tissue that will be preserved to shape the buttock region. Various methods of autoaugmentation have been described including muscle flaps, adipofascial flaps, and dermal fat flaps.[5,18,19,20,21,22] If an autoaugmentation is to be performed, we typically utilize a dermal fat flap 2 to 3 cm lateral from the midline that may range from 5 to 15 cm in width, depending on body type and intended contour. Typically it extends to the third vertical bar (▶ Fig. 45.3). The gluteal autoaugmentation can be performed with either an island of adipose tissue, or a fasciocutaneous flap that is undermined in the lateral region and transposed into the inferior buttock region approximately 45 degrees. The vertical reference lines can serve as reference points for precise measurements of flap design and symmetry. Lower body lifting with autoaugmentation is one of the few body contouring procedures where one must commit to the upper and lower incisions to provide the flap for augmentation. For the novice surgeon, it is advisable to be conservative when committing to the resection between the inferior and superior marking such that there is no excess tension on the closure, thereby increasing the risk for dehiscence.

The surgeon then transitions to additional anterior markings. Upward tension is placed on the patient's hip, and a line is drawn from the inferior margin of resection posteriorly and connected to the point above the mons. This maneuver is repeated on the contralateral side. This line represents the line of incision for the abdominoplasty portion of the operation. The superior extent of the abdominal resection is determined intraoperatively.

For patients undergoing medial thigh plasty, the lower abdominal incision is marked as previously mentioned and the lower body lift is tapered into this lower abdominal line to avoid scar migration inferiorly. A routine vertical medial thigh plasty resection can then be planned while maintaining the groin crease scar close in the mons region to avoid a scar on the front of the thigh.

45.5 Operative Technique

Following orotracheal intubation and bladder catheterization, the patient is placed in the prone position. The table is gently jackknifed to simulate a flexed position as this procedure is often combined with an abdominoplasty requiring subsequent bed flexion. Reverse Trendelenburg positioning prevents excess pressure on the eyes.[23] Warming blankets and appropriate padding are placed for secure, pressure reducing, and symmetrical positioning. Sequential compression devices are placed on each leg prior to the induction of general anesthesia. The electrocautery grounding pads may be placed on the calves beneath the compression boots bilaterally. The legs are prepped in a circumferential manner with Betadine prep. Two well-padded arm boards are placed at the lower end of the table so that the legs can be abducted. The room is kept at 70 degrees Fahrenheit and warmers are used throughout the case to avoid hypothermia.[24] Epinephrine (1:100,000) is injected into the dermis prior to skin incision to control bleeding from dermal edges.

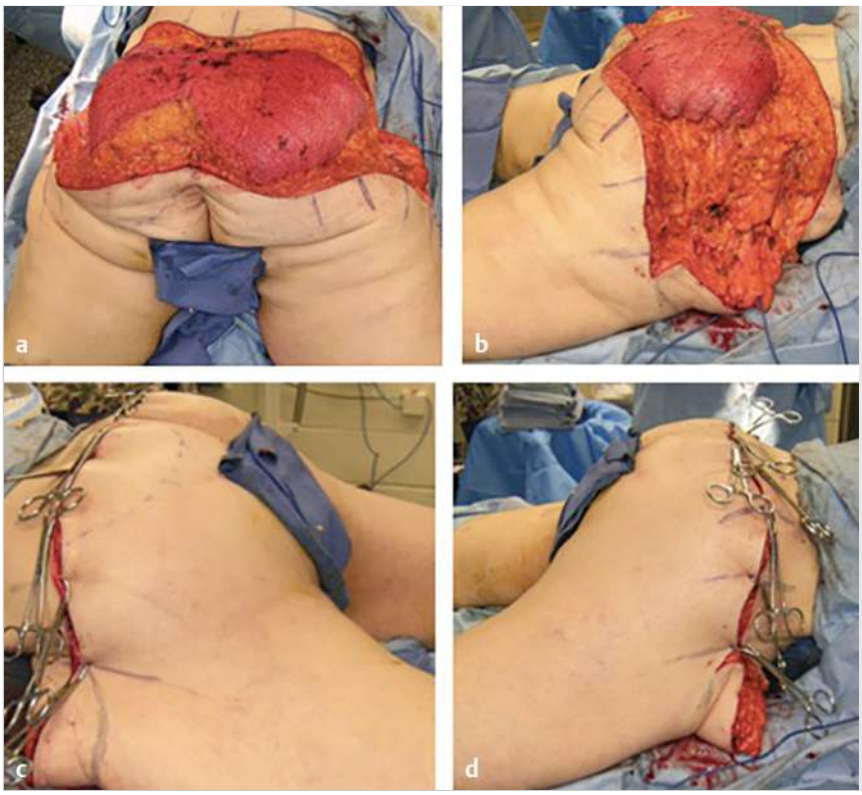


Fig. 45.4 (a,b) Posterior and lateral views of de-epithelialized adipofascial flaps for gluteal autoaugmentation. The flap is undermined in the lateral region at the level of muscle fascia, mobilized and rotated approximately 45 degrees inferiorly, and then inset. **(c,d)** Lateral views of skin edges approximated and secured with towel clips with the legs in slight abduction.

If gluteal autoaugmentation is part of the operative plan, the region of gluteal fat to be preserved is de-epithelialized with a scalpel blade. Following de-epithelialization, the adipose paddle is circumscribed and dissected down to the level of the deep fascia. Excision of the skin and subcutaneous tissue within the borders of the planned resection is carried out down to the level of the deep fascia. The de-epithelialized adipose flap is undermined in the lateral region at the level of the muscle fascia to allow inferior mobilization of the flap. A pocket for the flap is then created by dissection toward the inferior gluteal crease above the muscle fascia. The flap is rotated approximately 45 degrees, inserted into the pocket, and inset with multiple 2–0 braided absorbable sutures placed into the deep muscle fascia to restore the lateral curve of the buttocks (▶ Fig. 45.4a,b). The dermal surface of the tissue flap can be further manipulated and contoured by suture plication to achieve optimal shape and projection. If autoaugmentation is not performed, this tissue is resected just superficial to the level of Scarpa's fascia.

The Lockwood discontinuous undermining device (Byron Medical, USA) is passed in the subcutaneous tissue of the lateral thighs to facilitate mobilization of the thigh tissue during closure. In order to further facilitate a tension-free closure in the lateral aspects of the posterior wound, the legs are abducted onto the extended arm boards placed in this region of the operating table. The skin edges are approximated and secured with towel clips (▶ Fig. 45.4c,d). Saddlebags inferior to the planned resection may be suctioned at this time if desired. The superficial fascial system is then reapproximated with interrupted, braided, 2–0 absorbable suture. The dermis is then closed with deep dermal 2–0 absorbable braided sutures, followed by a running, subcuticular, barbed suture. The large lateral dog ears are stapled shut and addressed with the anterior resection. Closed suction no. 15 Jackson-Pratt drains are placed in the posterior excision site and temporarily buried in the stapled dog ears prior to turning the patient supine. They are then brought out anteriorly for ease of drain care. A cyanoacrylate is applied over the repair. The site is dressed with layered gauze dressings and an occlusive plastic dressing to protect the site during the remainder of the procedure.

The patient is turned to the supine position while care is taken to avoid inadvertent pulling or entanglement of intravenous lines, catheters, and drain tubes. For safety, the patient is rolled onto the stretcher and then transferred back onto the operating room table in the supine position. The patient is prepped and draped once again. The incision line connecting the inferior aspect of the lateral margin of resection with the low transverse abdominoplasty marking is incised. At this point, an abdominoplasty or thigh plasty can be performed as desired (see Chapters 44 and 47, respectively). Closed suction drains are placed in the abdomen or thighs, and closure follows.

45.6 Postoperative Care

An abdominal binder is placed following closure. If a thigh plasty is performed, the thighs are wrapped in ACE wraps from the toes to the groin. Patients are positioned in the beach chair position. Intravenous fluids are administered for the initial 24 hours postoperatively to ensure adequate tissue perfusion. Routine labs are checked in the initial 24 hours postoperatively to monitor for any electrolyte derangements, or postoperative anemia. Chemoprophylaxis is continued, and placement of sequential compression devices on the lower extremities is utilized until the patient is ambulating independently. Patient-controlled analgesia pumps

are routinely used for the initial 24 hours postoperatively. Patients are mobilized on the first postoperative day, and encouraged to perform deep breathing exercises by way of an incentive spirometer. Foley catheters are usually removed by noon on postoperative day 1, and the patient-controlled analgesia is discontinued with a transition to oral pain medication. Drains are removed when drainage is less than 30 mL in 24 hours for 2 consecutive days. Patients are instructed to avoid bending over in any way for at least 3 weeks. Scar massage is initiated after 3 weeks.

45.7 Outcomes

Complications following lower body lift procedures are common and often minor in nature. Due to the significant length of the incisions and pressure on the buttocks during recovery, wound dehiscence, suture extrusion, or delayed wound healing may occur. Srivastava et al found that wound dehiscence occurred in 42.5% of patients who underwent autoaugmentation versus 20% in those who did not.[11] However, local wound breakdown can be treated conservatively with local wound care (▶ Fig. 45.5a,b). Once much of the inflammation has settled, delayed secondary closure can be performed to expedite the healing process. Acute dehiscence can often be managed immediately with local anesthetic, irrigation of the wound with Betadine, and closure with Prolene suture over a Penrose drain (▶ Fig. 45.5c,d). Seromas may contribute to acute dehiscence; therefore, drains remain until drain output is less than 30 mL/day for 2 consecutive days. Recurrent seromas are rare and can be managed with serial aspiration or sclerosis, and rarely require reoperation.[25] Other minor complications include unfavorable scarring, asymmetry of scars, or recurrent skin laxity. These complications can be corrected by secondary revision.

Fig. 45.5 **(a)** Postoperative wound separation. **(b)** Healed wound following **(c,d)** local anesthesia, irrigation, sterile prep and wound closure with Prolene sutures over a Penrose drain.

Fig. 45.6 Undesirable gluteal cleft elongation following lower body lift.

Gluteal deformities among individual patients are variable and, therefore, patterns of excision are subject to variability. Some deformities require lower, aggressive excision patterns in order to achieve optimal gluteal reshaping and resuspension of ptotic tissue. However, undesirable gluteal cleft lengthening can occur due to direct elevation of the gluteal cleft, and the length discrepancy between the superior and inferior incisions. This length discrepancy forces inferior tissue to be worked into the repair, leading to a medial tissue redundancy, which may create the appearance of an elongated gluteal cleft as this redundant tissue redistributes (▶ Fig. 45.6). Therefore, in our experience, lengthening of the gluteal cleft can be mitigated by employing excision pattern designs such that the gluteal cleft is not directly involved in the excision pattern.[17] This can be accomplished by curving the medial extent of the inferior incision superiorly above the gluteal cleft, so as not to incorporate the cleft directly in the excision, and tapering the upper incision downward in a gullwing pattern to avoid major length discrepancies between the upper and lower excision lines. Additionally, the dorsal vector lines of resuspension should be designed judiciously. While a mediocranial direction of resuspension may sculpt the waist and enhance gluteal projection,[25,26,27,28] gluteal cleft elongation may result from the redistribution and gathering of excess tissue medially. Therefore, while it is a delicate balance, we advocate dorsal vector lines that resuspend tissue in a more cranial direction while titrating the medial vector of the resuspension. This will mitigate undesirable gluteal cleft lengthening while maintaining ample waist sculpting and gluteal enhancement. Similar to many other techniques, there are several tradeoffs in lower body lift surgery, and the design of each individual operation must be carefully planned.

Patient satisfaction with body lift procedures is high in the presence or absence of autoaugmentation. For those patients who desire fullness of the gluteal area, autoaugmentation is recommended and has proven to be a safe operation with minor risk of wound healing problems.[11]

45.8 Review Questions

45.8.1 True or False

1. Excision patterns that directly involve the gluteal cleft lower the risk for gluteal elongation.
2. Autoaugmentation of the buttocks can increase the risk of wound healing problems postoperatively.
3. A lower body lift addresses the lower back rolls and flank lipodystrophy.

45.8.2 Choose the Best Answer

4. A 36-year-old woman undergoes 4 years s/p gastric bypass and has lost 100 lb. She has laxity of the abdomen, inner thighs, outer thighs, and buttocks. She wants to limit her recovery times by combining procedures. The best way to stage the procedure is as follows:
   a) Abdominoplasty first, followed by a second-stage lower body lift, and third-stage thigh plasty.
   b) Lockwood type 1 lower body lift, followed by an abdominoplasty as a second stage.
   c) Lockwood type 2 lower body lift, followed by a thigh plasty at a second stage.
   d) Thigh plasty and abdominoplasty first, followed by a lower body lift as a second stage.
5. A 45-year-old woman has lost 120 lb after bariatric Roux-en-Y gastric bypass surgery. She has been weight stable for 3 years. She presents for body contouring surgery and undergoes a lower body lift with autoaugmentation. She returns to the office 3 weeks after surgery with delayed wound healing. The most appropriate next step:
   a) Return to the operating room to close the wounds.
   b) Immediately close the wound in the office over a drain.
   c) Evaluate the patient for nutritional deficiencies.
   d) Instruct the patient to avoid bending over for the next 3 weeks.

45.8.3 Answers

1. False.
2. True.
3. False.
4. c. Lockwood type 2 lower body lift, followed by a thigh plasty at a second stage.
5. c. Lipo-abdominoplasty.

References

[1] Lockwood T. Lower body lift with superficial fascial system suspension. Plast Reconstr Surg. 92(6):1112–1122, discussion 1123–1125
[2] Lockwood TE. Lower-body lift. Aesthet Surg J. 21(4):355–370
[3] Babuccu O, Gözil R, Ozmen S, Bahçelioglu M, Latifoglu O, Celebi MC. Gluteal region morphology: the effect of the weight gain and aging. Aesthetic Plast Surg. 26(2):130–133
[4] Centeno RF. Gluteal aesthetic unit classification: a tool to improve outcomes in body contouring. Aesthet Surg J. 26(2):200–208
[5] Centeno RF. Autologous gluteal augmentation with circumferential body lift in the massive weight loss and aesthetic patient. Clin Plast Surg. 33(3):479–496
[6] Centeno RF, Young VL. Clinical anatomy in aesthetic gluteal body contouring surgery. Clin Plast Surg. 33(3):347–358

[7] Da Rocha RP. Surgical anatomy of the gluteal region's subcutaneous screen and its use in plastic surgery. Aesthetic Plast Surg. 25(2):140–144
[8] Mendieta CG. Classification system for gluteal evaluation. Clin Plast Surg. 33 (3):333–346
[9] Hurwitz DJ, Rubin JP, Risin M, Sajjadian A, Sereika S. Correcting the saddlebag deformity in the massive weight loss patient. Plast Reconstr Surg. 114(5): 1313–1325
[10] Coon D, Gusenoff JA, Kannan N, El Khoudary SR, Naghshineh N, Rubin JP. Body mass and surgical complications in the postbariatric reconstructive patient: analysis of 511 cases. Ann Surg. 249(3):397–401
[11] Srivastava U, Rubin JP, Gusenoff JA. Lower body lift after massive weight loss: autoaugmentation versus no augmentation. Plast Reconstr Surg. 135(3):762–772
[12] Koltz PF, Chen R, Messing S, Gusenoff JA. Prospective assessment of nutrition and exercise parameters before body contouring surgery: optimizing attainability in the massive weight loss population. Plast Reconstr Surg. 125(4):1242–1247
[13] Bossert RP, Rubin JP. Evaluation of the weight loss patient presenting for plastic surgery consultation. Plast Reconstr Surg. 130(6):1361–1369
[14] Aly AS, Cram AE, Chao M, Pang J, McKeon M. Belt lipectomy for circumferential truncal excess: the University of Iowa experience. Plast Reconstr Surg. 111(1):398–413
[15] Coon D, Michaels J , V, Gusenoff JA, Purnell C, Friedman T, Rubin JP. Multiple procedures and staging in the massive weight loss population. Plast Reconstr Surg. 125(2):691–698
[16] Buchanan PJ, Nasajpour H, Mast BA. Safety and efficacy of outpatient lower body lifting. Ann Plast Surg. 70(5):493–496
[17] Afrooz PN, Shakir S, James IB, Rubin JP, Gusenoff JA. Dynamics of gluteal cleft morphology in lower body lift: predictors of unfavorable outcomes. Plast Reconstr Surg. 136(6):1167–1173
[18] Centeno RF, Mendieta CG, Young VL. Gluteal contouring surgery in the massive weight loss patient. Clin Plast Surg. 35(1):73–91, discussion 93
[19] Colwell AS, Borud LJ. Autologous gluteal augmentation after massive weight loss: aesthetic analysis and role of the superior gluteal artery perforator flap. Plast Reconstr Surg. 119(1):345–356
[20] Raposo-Amaral CE, Cetrulo CL , Jr, Guidi MdeC, Ferreira DM, Raposo-Amaral CM. Bilateral lumbar hip dermal fat rotation flaps: a novel technique for autologous augmentation gluteoplasty. Plast Reconstr Surg. 117(6):1781–1788
[21] Rohde C, Gerut ZE. Augmentation buttock-pexy using autologous tissue following massive weight loss. Aesthet Surg J. 25(6):576–581
[22] Sozer SO, Agullo FJ, Wolf C. Autoprosthesis buttock augmentation during lower body lift. Aesthetic Plast Surg. 29(3):133–137, discussion 138–140
[23] Shermak M, Shoo B, Deune EG. Prone positioning precautions in plastic surgery. Plast Reconstr Surg. 117(5):1584–1588, discussion 1589
[24] Coon D, Michaels J , V, Gusenoff JA, Chong T, Purnell C, Rubin JP. Hypothermia and complications in postbariatric body contouring. Plast Reconstr Surg. 130 (2):443–448
[25] Shermak MA, Rotellini-Coltvet LA, Chang D. Seroma development following body contouring surgery for massive weight loss: patient risk factors and treatment strategies. Plast Reconstr Surg. 122(1):280–288
[26] Richter DF, Stoff A. Circumferential body contouring: the lower body lift. Clin Plast Surg. 41(4):775–788
[27] Richter DF, Stoff A, Velasco-Laguardia FJ, Reichenberger MA. Circumferential lower truncal dermatolipectomy. Clin Plast Surg. 35(1):53–71, discussion 93
[28] Rihter DF, Stoff A. Liposuction and Circumferential Lower Truncal Dermatolipectomy. 1st ed. St. Louis, MO: Elsevier; 2012

46 Brachioplasty

J. Peter Rubin

Abstract
Brachioplasty is a surgical procedure that aims to recontour the arm following excessive weight loss. To achieve the desired goal(s), liposuction and excisional surgery are options. The author covers all the vital considerations when preparing the patient for surgery and then looks at the treatment options, explaining which procedure (liposuction or excisional) is best suited to which indications. Each procedure is described in detail, with images illustrating the text. Surgical techniques come in for an extended discussion, giving surgeons the knowledge and insights needed for a successful outcome. Guidelines on postoperative care and potential complications (seroma, hematoma, wound breakdown, prolonged swelling) conclude the chapter.

Keywords: brachioplasty, Pittsburgh rating system, liposuction, excisional surgery, intraoperative flap marking, segmental resection

46.1 Goals and Objectives

- Understand the proper evaluation of patients presenting for brachioplasty.
- Define the indications for brachioplasty, and discuss the risks and benefits with the patient.
- Appreciate the technical aspects of the procedure that will lead to successful outcomes and reduce the risk of complications.
- Be aware of potential complications and their management.

46.2 Patient Presentation

Most patients presenting for brachioplasty will have experienced significant weight loss either through successful diet and exercise or a bariatric procedure. In the setting of a history of massive weight loss (MWL), a thorough evaluation should be done in the context of this weight loss, including an assessment of the patient's nutritional status, maximum body mass index (BMI), current BMI, and overall medical profile. If the patient is still far from his or her goal BMI, nutritional counseling should be offered, as well as having the patient visit with their bariatric surgery team to optimize weight loss.[1,2] A very important historical point relates to previous surgery of the upper extremities as well as the axillae, including lymph node dissection or radiation. Another important historical point relates to any history of chronic swelling of the upper extremities.

The physical examination will help define the deformities and determine the appropriate treatment. The Pittsburgh rating scale provides a grading system for deformities.[3] The physical examination should determine the amount of adipose tissue in the upper arm and the extent of skin laxity. The pattern of skin laxity should be carefully evaluated, including the amount of skin laxity in the axilla, as well as the extent of the deformity onto the forearm distally, and on to the lateral chest proximally. Patients with excess adipose tissue, but good skin tone, may be candidates for liposuction alone, and will not require excisional surgery. Patients with primarily skin laxity can be treated with excisional brachioplasty, and patients with skin laxity and excess adipose tissue can be treated with concurrent liposuction of the posterior arm with excisional brachioplasty. The rate of complications when these procedures are performed together is equivalent to excisional brachioplasty alone.[4] While there are short scar versions of the brachioplasty, most patients with significant skin laxity, especially after MWL, will require a full length longitudinal incision.[5]The incision length can certainly be varied in its distal extent, ending in the midportion of the arm in rare cases. The scar length can also vary in length along the lateral chest wall. The design of the operation that we describe puts the scar in the midaxial position at the level of the elbow, so the elbow can be crossed with a resection extending onto the proximal forearm without impairing range of motion. Some patients, especially those with excessively large amounts of excess skin, may present with the appearance of tight restrictive bands on the arm above the bicep muscle. These bands should be noted (▶ Fig. 46.1) and pointed out to the patient preoperatively. Often times, patient will not notice these constrictive bands until the postoperative stage, and will attribute them to the surgery. However, these findings are evident preoperatively. Unfortunately, a good solution to eliminating these bands has not been developed. Interestingly, a very acceptable contour can be obtained along the posterior surface of the arm despite these bands over the bicep. Additional physical exam finding should be noted such as previous scars on the arms and any evidence of prior trauma. The dorsal surfaces of the hands should be inspected for any signs of acute or chronic edema.

46.3 Preparation for Surgery

Preparation for surgery begins with the vital task of complete preoperative counseling as to the risks and benefits of the procedure. The most important message to convey to the patient is that brachioplasty is the most visible example of making a tradeoff between accepting a scar and improving contour. Patients are counseled that the scar is permanent and visible when the arms are exposed. Placement of the scar in the medial aspect of the upper arm at the bicipital groove allows the patient to hide the scar by placing their arms against the side of their body. Patients are counseled that the scar takes quite a while to mature, and the timeframe that we convey is 1 year for the scar to reach its final appearance. The scar will remodel and change in appearance up until that time, and sometimes beyond 1 year. Patients are also counseled that scars may extend on to the forearm. Of course, the extent of the scar, in each particular case, is reviewed with the patient preoperatively in the office and again during the markings preoperatively. Another common risk that is discussed with the patient is sensory disturbances on the forearm, including paresthesia and dysesthesia. These improve over time and rarely result in

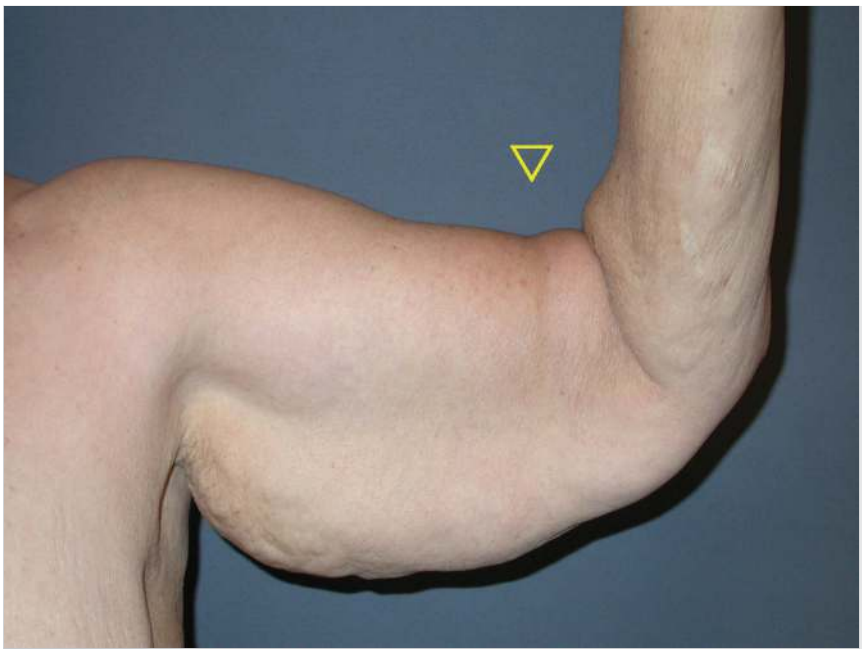


Fig. 46.1 Occasionally, patients will have the appearance of restrictive bands above the biceps muscle. These bands represent a regional soft-tissue deficiency and should be pointed out to the patient. These bands will still be present after brachioplasty, and will often worsen after surgery. Patient and surgeon may speculate that these bands were a complication of the surgery, but a careful review of the preoperative photos usually reveals that they were present before surgery.

long-term discomfort. Seroma can occur, and also wound dehiscence. While prolonged swelling and chronic lymphedema are uncommon after brachioplasty, patients should be counseled that they may have short-term swelling bilaterally.

Laboratory testing, as appropriate for the patient's age and medical profile, should be obtained. Additionally, electrocardiogram and chest X-ray should also be obtained as indicated.

Brachioplasty may be performed as an isolated procedure or, as is commonly seen, may be combined with other body contouring procedures such as abdominoplasty. If the brachioplasty is planned as a single procedure, this can be done as an outpatient procedure. When combining brachioplasty with other procedures, strong consideration should be given to an overnight stay.

46.4 Treatment

46.4.1 Treatment Options and Indications

The choices for surgical treatment of the arms are related to the extent of skin laxity, the extent of adipose tissue, and the relevant skin tone. Patients with excessive adipose tissue and good skin elasticity may be candidates for liposuction as a primary treatment. If this course therapy is selected, patients must be advised that they may have resultant loose skin if the skin does not retract sufficiently after liposuction. Of course, this option should always be considered when there is good skin elasticity present because it will obviate the need for significant scars to achieve results. Most patients with skin laxity will require an excisional brachioplasty. While short-scar versions have been described, patients with significant circumferential excess skin will require a longitudinal incision along the arm in order to achieve adequate results. Liposuction can be planned as an adjunct to brachioplasty in order to decompress adipose deposits in the posterior arm.

46.4.2 Surgical Preparation

Patients are advised to shower with an antibacterial soap the morning of the surgery. Patients are not asked to shave hair-bearing regions within the axilla, and these areas are not shaved by the surgical staff either. Patients who routinely shave the hair-bearing regions in the axilla are advised to not shave within 48 hours of the procedure.

The patient is marked preoperatively in the holding area. Markings are made with the patient holding the arm in the "victory" position in which the upper arm is at 90 degrees to the shoulder (parallel to the floor) and the elbow is bent at 90 degrees. First, the dome of the axilla is marked (▶ Fig. 46.2) and designated point A. Next, an intended line of incision is drawn from point A inferiorly along the lateral chest terminating at a point designated "point B." The length of line A to B is determined by the relative amount of laxity and skin redundancy of the lateral chest wall. Next, a line is drawn along the bicipital groove from the axilla distally. The bicipital grove will be the intended location of the scar. The end of the line extending from the axilla along the bicipital groove to the distal most aspect of the resection is designated as "point C." The length of that line is determined by the relative skin redundancy. The elbow can be crossed in a midaxial position with this line. It is not uncommon to extend this incision past the elbow for several centimeters, although further progression onto the forearm would leave a more visible scar. The next mark is the line of incision above the bicipital groove. The placement of that line is designed in a way that when the line is under tension, it will advance to the bicipital groove. This line is generally 2 cm above the bicipital groove mark, as a general rule, but can be planned more precisely by pulling inferiorly on the bicipital groove marking and noting how far the skin migrates under tension. An important goal is to correct the descent of the axillary fold. A point is picked in the axilla that can be transposed up to point A in the dome of the axilla. We designate this point as A prime. That corner point coming up to the axilla will result in a

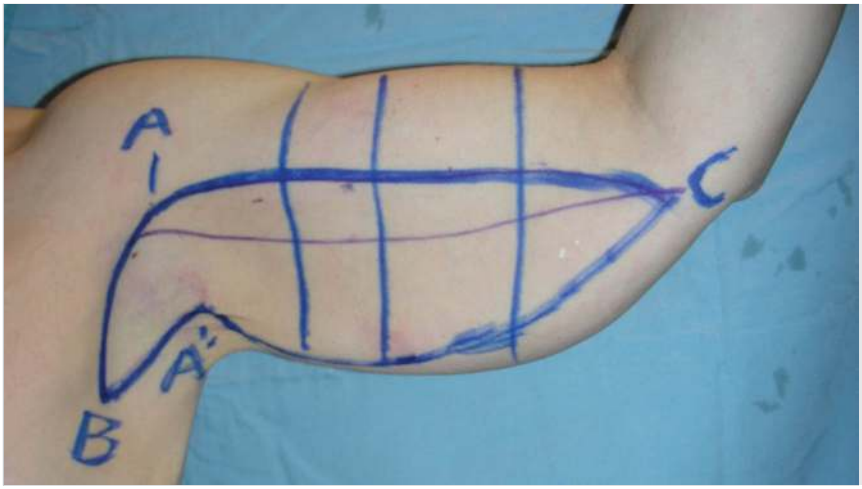


Fig. 46.2 Pattern of markings for brachioplasty. The design is described in detail within the text.

flap covering the axilla, and the offset of scars will occur at the dome of the axilla. This pattern of scars simulates a Brunner incision pattern extending from the extremity on to the trunk. Once this point is selected, a line is drawn connecting point A prime to point B, and point A prime to point C based on a pinch test. It is very important to note that the lines marked by pinch test are only estimates, and will be rechecked and adjusted as needed during the procedure by a segmental marking process. The surgeon will commit *only* to the superior line of incision (drawn above the bicipital groove) and the axillary/lateral chest wall incision (point A to point B). The corner point (A prime) represents the tip of the flap that will be advanced into the axilla and the associated lines connecting. Since the lines marked from A prime to both points B and C are merely estimates, and these will be measured and remarked on the table, this technique minimizes the risk of over-resecting.

Next, other regions of the arm are inspected for excess adipose tissue. If concurrent liposuction is planned, usually on the posterior/lateral aspect of the arm, these regions are marked and estimation of the aspirate volume is determined. Invariably, the anesthesiologists inquire about where they can place an intravenous (IV) catheter. While there is a school of thought that dictates no IV catheters should be placed in the arms during brachioplasty, our experience has shown that it is not a problem to have IVs placed in the arms distal to the elbow. Additionally, a blood pressure cuff can be placed on the forearm opposite the arm with the IV, or on the calf. IV antibiotics are administered prior to beginning the procedure. Placement of a Foley catheter is not necessary if the brachioplasty is performed as an isolated procedure, but should be considered if multiple procedures are being performed.

46.4.3 Operative Technique

After induction of general anesthesia, the patient is prepped in the supine position with each arm extended at slightly less than 90 degrees and resting on a well-padded arm board. The arm is prepped circumferentially including the shoulder and the superior portion of the lateral chest. Draping is very simple with sterile towels used to block off the shoulder and a sterile sheet over the arm board. With an assistant holding the arm up, the arm is prepped with betadine and then laid down over the sterile sheet and the distal part of the arm and hand wrapped in a sterile towel, and the towel secured with staples. Sterile rolled gauze can be used to wrap the IV and preserve sterility. It is important that the arm is prepped circumferentially so that it can be ranged during the case to give exposure to all aspects of the upper extremity and the shoulder. After completing the draping over the remainder of the patients' body, and leaving room for surgeons to stand on either side of both right and left arm boards, the procedure commences. If liposuction is to be performed, an assistant will hold the arm up and a small port site incised with a number 15 blade at the posterior elbow in one of the natural creases. Tumescent solution is infused into the posterior and lateral arm at a ratio of 1 mL of infusion per anticipated 1 mL of aspirated fat volume. A concern is that infusion of fluid into the arm will result in rapid spread of edema through the tissue planes, and difficulty closing the arm and/or adequately estimating the resection. This problem is mitigated in two ways. First, the fluid is infused and no waiting period is allowed between finishing the infusion and starting the liposuction. Liposuction is started with a 3- or 4 mL tri-tip cannula immediately after finishing the infusion. We find that the epinephrine effect is quite adequate in this region without waiting the usual 10 to 15 minutes. Second, no fluid is infused into the contralateral arm until the surgeon is ready to commit to liposuction followed by immediate sharp resection of the tissues. This strategy limits the spread of edema in the tissues. We perform liposuction in approximately one-half of the brachioplasties done at our center. Next, the arm is repositioned and extended on the arm board, and the intended incision lines (the longitudinal line above the bicipital groove and the lateral chest wall mark) are injected with approximately 10 mL of epinephrine 1:100,000. This solution is mixed on the table by adding 1 mg of epinephrine to 100 mL of normal saline. Injecting the solution into the dermis minimizes bleeding from the dermal edges after incision.

The superior longitudinal incision line above the bicipital groove is incised with a scalpel through the dermis, extending from point C to point A. The marked line in the axilla, extending down to the chest wall, is also incised with a scalpel connecting point A with point B. Using electrocautery, the soft tissues are dissected and elevated within the marked pattern, leaving a thin layer of fat over the fascia to protect the sensory nerves. It

is best to start this dissection in the proximal third of the arm, away from the origin of the medial antebrachial cutaneous nerve, which emerges through the deep fascia into the subcutaneous space in the distal third of the forearm. Once the plane of dissection is established, and the proximal third of the arm, safe dissection can proceed both distally and proximally. In a proximal direction, undermining is commenced within the marked area, keeping in mind that the inferior marks representing the other margins of resection are still only estimates at this point. As the axilla is approached, the plane of dissection should be kept at the same level so that the flap thickness is uniform in that region. As the dissection moves more distally, the surgeon should identify the fat pad containing the basilic vein and medial antebrachial cutaneous nerve (MABC). The MABC and the basilic vein run together. In general, a plane of dissection above the main branches of the basilic vein will protect the MABC from injury. Although the MABC is the nerve at highest risk for injury during brachioplasty, it is fortunately a sensory nerve, so injury will not result in any motor impairment. However, there may be changes in sensation on the forearm.

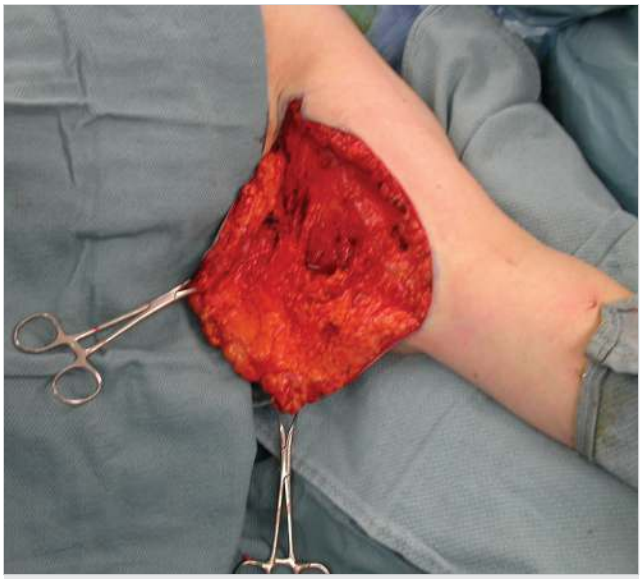


Fig. 46.3 The flap is elevated and dissected to the estimated margins of resection. This is followed by systematic on-table measuring and remarking.

Once the flap is elevated to the estimated margins of resection, on-table marking is performed to refine the margin of resection (▶ Fig. 46.3, ▶ Fig. 46.4). This involves marking and segmental resection at three points along the arm using a flap marking technique: distal third, midpoint, and proximal third. Ensuring that the skin of the posterior arm is not tethered, the first measurement is made at the distal third of the arm. Using two sharp towel clamps to hold up the flap, heavy forceps or a sharp towel clip is secured at the edge of the superior wound edge and advanced under the flap. The tip of the instrument is palpated through the flap after the appropriate amount of tension is applied. This location is marked and then the flap is split and the points secured with a sharp towel clip (▶ Fig. 46.4; ▶ Fig. 46.5; ▶ Fig. 46.6). A key point in marking the resection in the distal third of the arm is not to overtighten the tissues, but to keep a smooth transition in contour between the upper arm and the forearm. In the midportion of the arm, a similar process of flap marking, measurement, and splitting is commenced (▶ Fig. 46.7). As the surgeon moves more proximately along the arm, greater tension can be applied on the flap and the tissues tightened considerably. The third point of measuring, marking, and splitting the flap is done in the proximal third of the arm. Once these points are marked, measured, split, and secured with a sharp towel clip, attention is turned to adjusting the markings of the corner of the axillary flap.

The goal of the axillary flap is to make sure that the tip of the flap comes up high into the dome of axilla so that the longitudinal scar on the upper arm meets the scar from point A to point B along the lateral chest in a manner that no scar crosses the flexion crease. As mentioned earlier, this is similar to a Brunner incision that is made on the hand. Using heavy forceps, the corner point is secured and the flap advanced over this instrument with tension applied and the proper location marked. The tissue surrounding the corner flap is excised (▶ Fig. 46.8). The

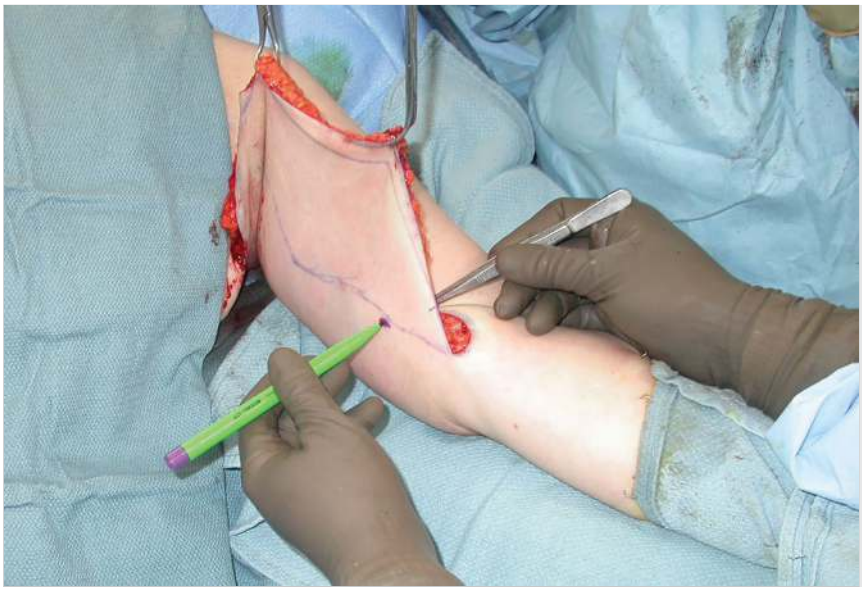


Fig. 46.4 The flap is marked under appropriate tension at three sites along the arm: distal third, midpoint, and proximal third. The distal most site is marked first. The flap is held under less tension while marking the most distal site as to not overtighten and create a more natural transition onto the forearm.

Fig. 46.5 The flap is split with a scalpel to separate the first segment.

Fig. 46.6 A sharp towel clamp is used to approximate the tissues at the base of the incision.

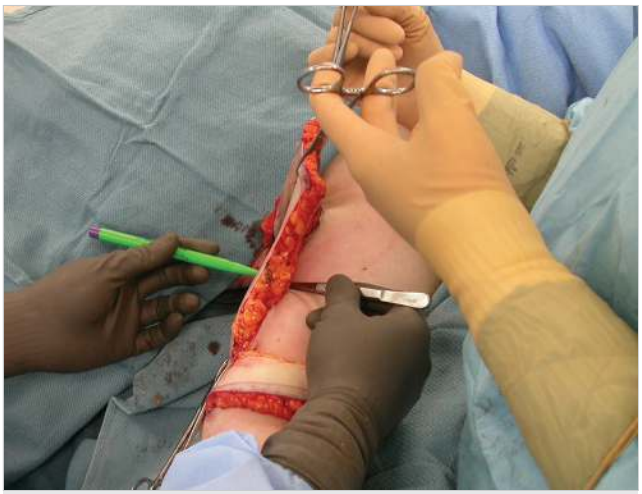


Fig. 46.7 The flap is marked at the midpoint of the arm. Tension on the flap can be increased here to tighten the closure and enhance the contour.

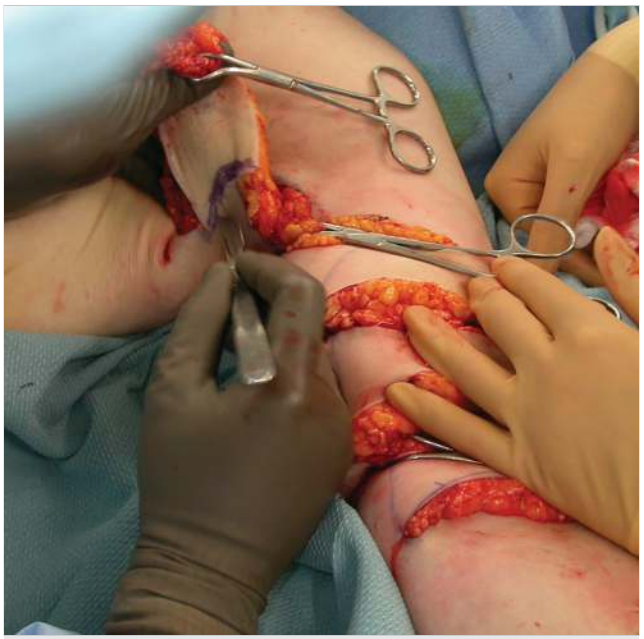


Fig. 46.8 After all three sites have been marked along the arm, the corner flap is also re-marked and excess tissue excised sharply.

corner point of the flap is then secured with one or two deep-braided sutures to the clavipectoral fascia, which is the thick condensation of the subcutaneous tissue above the muscle fascia in the deltoid region (▶ Fig. 46.9). The sutures will hold that corner point in place and prevent it from migrating inferiorly into the axilla. Next, the apices of the flap splitting incisions are connected to mark the resection of the segments (▶ Fig. 46.10). The skin is then marked at the sites of the three towel clamps between the split segments on the arm in order to guide reapproximation, and the towel clips removed. The segments of the flap that had been split are then incised with a scalpel and resection completed with electrocautery.

Fig. 46.9 The corner of the flap (point A prime) is secured to the clavipectoral fascia at the level of the superficial fascial system using a braided 2–0 suture.

Having completed the resection of all of the subcutaneous tissue, the wound is checked for hemostasis and then very quickly the towel clamps are reapplied at the same three points along the arm to approximate the tissues (marking the sites of the towel clamps, as mentioned earlier, will enable accurate reapproximation). A closed suction Jackson-Pratt drain is inserted into the deep tissues and the end of the drain taken out through a separate stab incision near the axilla. The wound is then staple-tacked closed and the sharp towel clips removed. At this point, the contour of the arm is checked. If there is still too much laxity, refinement of the resection can be commenced. Closure of the arm is performed in two layers with 2–0 braided absorbable sutures used in the deep layers and barbed 3–0 myofilament used in the dermal layer. Great care is taken not to sew the drain into the deep tissues when closing those layers. Cyanoacrylate skin adhesive is applied over the wound and this is followed by a rolled gauze sterile dressing. The arm is then wrapped with ACE bandages from the wrist to the shoulder along the entire arm.

46.5 Postoperative Care

ACE wraps are kept in place until the first postoperative visit, along with strict elevation of both arms on pillow stacks until the first postoperative visit, usually between 3 and 5 days following surgery. After the first visit, the ACE wraps are removed and the drains are also removed if output is less than 30 mL per drain in 24 hours. Patients are advised to avoid strenuous exercise and heavy lifting for at least 4 weeks following the procedure. Patients with nonstrenuous jobs can often return to work in 2 weeks while occupations requiring heavy lifting will require 4 weeks of recovery. Patients may use their arms to transfer in and out of bed and chair. Patients are encouraged

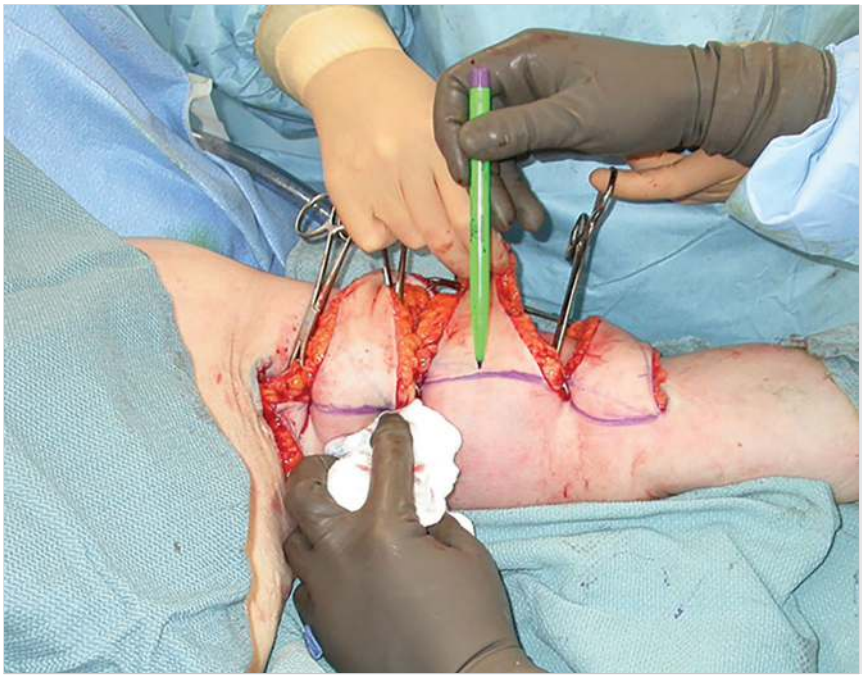


Fig. 46.10 Based on the intraoperative segmental flap marking, the final line of excision is drawn along the arm.

Fig. 46.11 A 57-year-old weight loss patient shown preoperatively in the **(a)** anterior view and **(b)** posterior view. Postoperative anterior views are shown at **(c)** 4 months and **(d)** 2.5 years, demonstrating the scar maturation. Posterior postoperative views are shown at **(e)** 4 months and **(f)** 2.5 years.

not to raise their arms above their heads for 2 weeks. After 2 weeks, gentle full range of motion is recommended.

Scar management is a major concern for these patients. At 1 month following surgery, silicone gel is usually started, applied twice a day, and continued for months. This will help keep the scar flat overtime.

46.6 Outcomes

Complications known to brachioplasty include seroma, hematoma, wound breakdown, and prolonged swelling.[1,4] Of course, a very serious complication is overresection and inability to close the wound. With the technique described earlier, this complication is greatly reduced due to the on-table flap marking prior to definitive resection of both margins. Relying solely on a pinch test, especially in a patient with a large amount of adipose tissue in their arms, is not always 100% reliable.

Hematomas tend to occur within 24 hours of surgery, and may require decompression in the operating room. Seromas tend to occur distally on the arm and be of lower volume, in the range of 5 to 15 mL. Serial aspiration is usually adequate to treat that adverse event. Permanent swelling in the forearms or hands after brachioplasty is uncommon, but patients may experience prolonged swelling following the surgery. This is treated with arm elevation and continued compression. Manual lymphatic drainage can be used in more severe cases. Infection occurs in approximately 5% of cases, and is usually resolved with oral antibiotics.

Clinical case examples are shown in ▶ Fig. 46.11, ▶ Fig. 46.12, and ▶ Fig. 46.13.

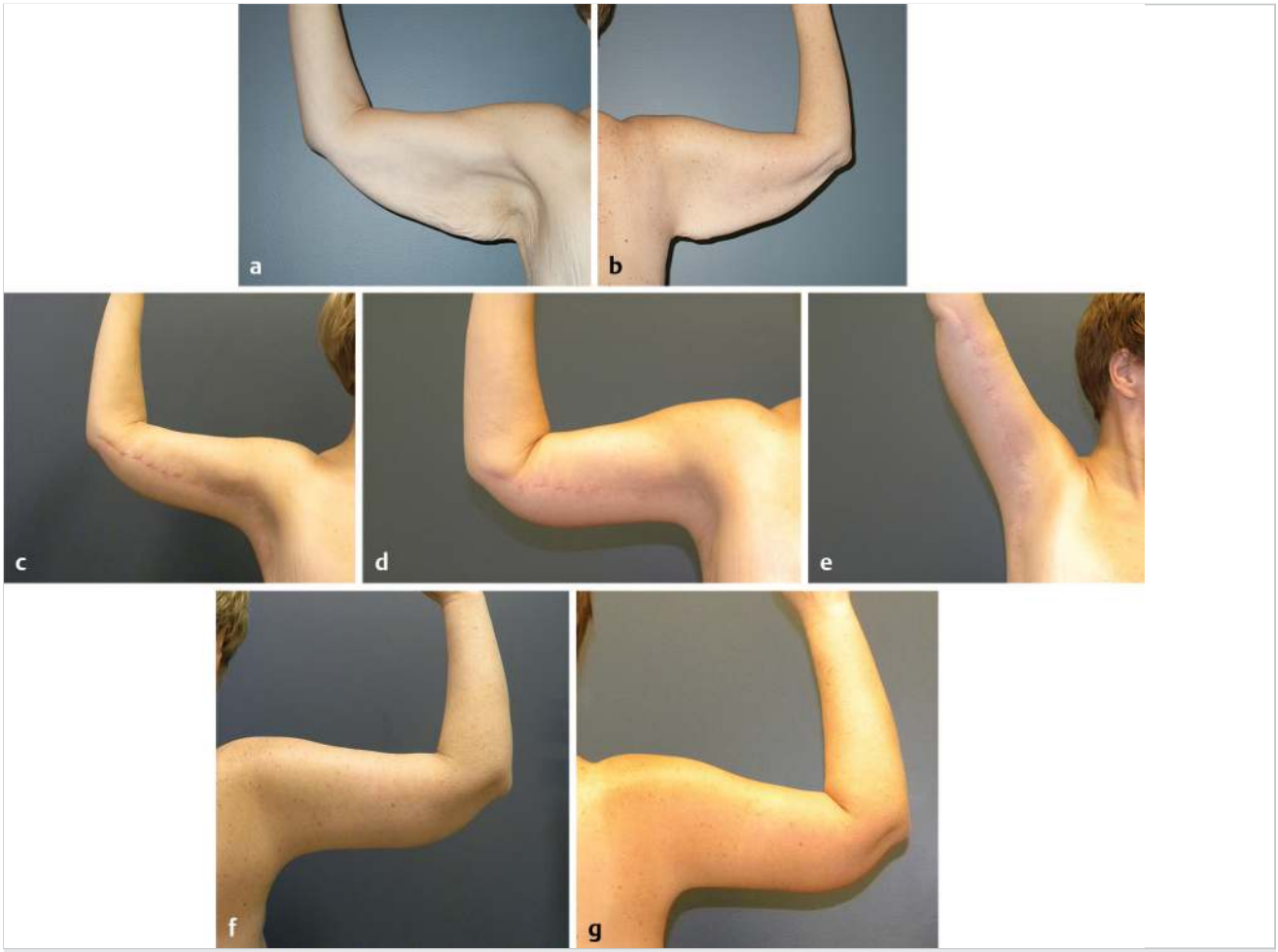


Fig. 46.12 A 46-year-old weight loss patient shown preoperatively in the **(a)** anterior view and **(b)** posterior view. Postoperative anterior views are shown at **(c)** 9 months and **(d)** 19 months, showing that the scar is improving even after 9 months following surgery. The patient is shown comfortably raising her arm over her head 19 months after surgery **(e)** with no scar in the flexion crease and no limitation of movement. Posterior postoperative views are shown at **(f)** 9 months and **(g)** 19 months.

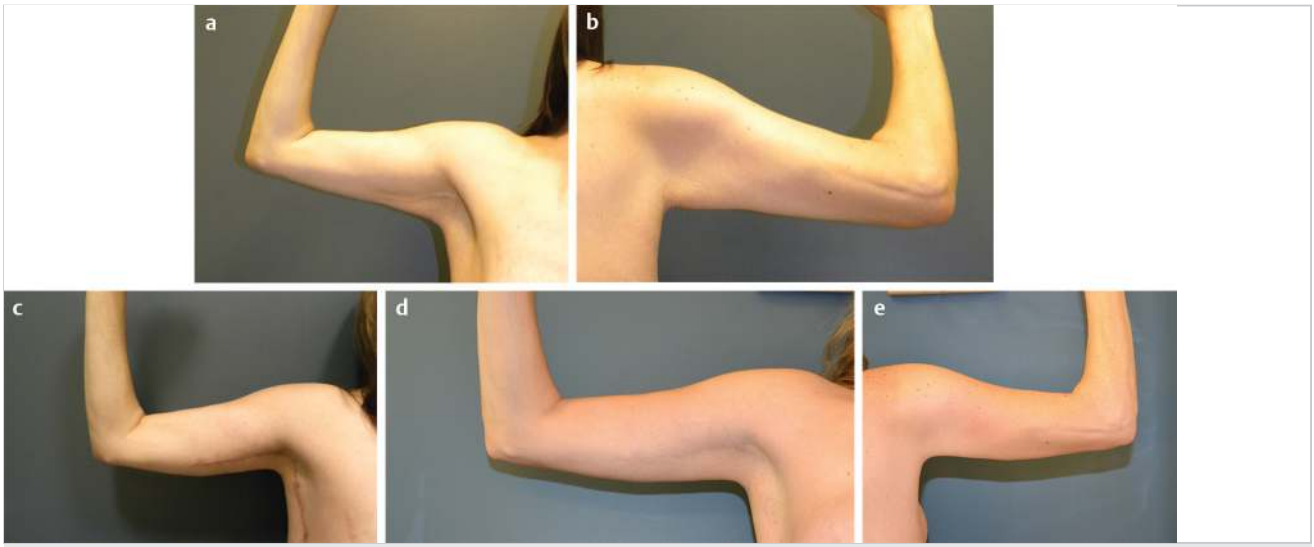


Fig. 46.13 This 35-year-old weight loss patient had more mild deformities of excess skin in the proximal region of the arm, with skin laxity on the lateral chest as well. Preoperative photographs are shown in the **(a)** anterior view and **(b)** posterior view. She was a good candidate for a limited incision along the arm, extending distally to the midhumerus. Postoperative anterior views are shown at **(c)** 3 months and **(d)** 12 months, demonstrating the scar maturation and acceptable contour at the transition between the end of the scar and the distal upper arm. The posterior postoperative view is shown at **(e)** 12 months.

46.7 Review Questions

46.7.1 True or False

1. Liposuction can be performed concurrently with excisional brachioplasty safely.
2. Intraoperative flap marking and segmental resection can help avoid overresection and inability to close.
3. Brachioplasty should only be performed as an isolated procedure because combining this operation with other body contouring procedures would result in worse outcomes.

46.7.2 Choose the Best Answer

4. The nerve most commonly injured during brachioplasty:
   a) Lateral antebrachial cutaneous.
   b) Medial antebrachial cutaneous.
   c) Ulnar.
   d) Musculocutaneous.
5. Scars resulting from brachioplasty
   a) Are rarely a concern for patients.
   b) Are generally acceptable by 3 months postoperative.
   c) Can vary in length to address individual patterns of skin excess.
   d) Can always be limited to the axilla without a longitudinal extension.

46.7.3 Answers

1. True.
2. True.
3. False.
4. b. Medial antebrachial cutaneous.
5. c. Can vary in length to address individual patterns of skin excess.

References

[1] Bossert RP, Rubin JP. Evaluation of the weight loss patient presenting for plastic surgery consultation. Plast Reconstr Surg. 130(6):1361–1369

[2] Rubin JP. Principles of Plastic Surgery after Massive Weight Loss. In: Thorne CH, Chung KC, Gosain AK, eds. Grabb and Smith's Plastic Surgery; 2014:713–719

[3] Song AY, Jean RD, Hurwitz DJ, Fernstrom MH, Scott JA, Rubin JP. A classification of contour deformities after bariatric weight loss: the Pittsburgh Rating Scale. Plast Reconstr Surg. 116(5):1535–1544, discussion 1545–1546

[4] Bossert RP, Dreifuss S, Coon D, et al. Liposuction of the arm concurrent with brachioplasty in the massive weight loss patient: is it safe? Plast Reconstr Surg. 131(2):357–365

[5] Reed RS. Brachioplasty with limited scar. Clin Plast Surg. 41:753–763

47 Thigh Lift and Reduction

Paul A. Ghareeb, Alexandra M. Hart, and Felmont F. Eaves

Abstract
This chapter examines the role of thigh lift and reduction in remediating medial thigh deformities, which are often caused by massive weight loss. The authors lay out the indications for thigh rejuvenation and then provide an in-depth description of how patients should be prepared for surgery, followed by the actual surgical procedure. Postoperative care guidelines are included, and a frank assessment of outcomes and possible complications concludes the chapter.

Keywords: massive weight loss, truncal tissue, greater saphenous vein

47.1 Goals and Objectives

- To educate the reader on the keys to evaluating a patient presenting with medial thigh deformity.
- To describe where thigh lift and reduction fits into the sequence of body contouring.
- To review current concepts in the surgical management of thigh contour deformities, including when to utilize liposuction as well as when to use the vertical, horizontal, or combined approach to excisional lift.
- To outline common techniques for excisional thigh lift, including fascial anchoring and degree of excision.
- To review current evidence on outcomes, complications, and patient satisfaction after thigh-contouring procedures.

47.2 Patient Presentation

The medial thigh is a common area of concern for patients when seeking plastic surgical consultation for body contouring, particularly after massive weight loss (MWL). Historically, the number of patients undergoing thigh lift has been low secondary to the scar burden and risk for complications. However, the technique has gained popularity in patients who have undergone MWL, either secondary to lifestyle modification or bariatric surgery. According to the procedural statistical report of the American Society of Aesthetic Plastic Surgery (ASAPS), over 17,000 thigh lift and reduction procedures were performed in the United States in 2014, marking a 500% increase from 1997.[1] These patients are often willing to trade the resulting scar burden for improved contour in this area.

The vast majority of MWL patients presenting with medial thigh deformity have concurrent excess truncal tissue. This should be addressed first with either an abdominoplasty procedure or lower body lift. Oftentimes, truncal contouring alone can correct mild upper third thigh laxity. However, if the medial thigh deformity persists, many advise waiting at least 3 months after abdominal contouring and it is necessary to address this in a separate procedure.[2]

When evaluating a patient for thigh rejuvenation, it is useful to divide the medial thigh into thirds to accurately determine the location of excess tissue. By doing so, patients can be divided into four distinct groups: (1) patients who have upper third excess, (2) patients who have upper and middle third excess, (3) patients who have excess tissue throughout the entire medial thigh, (4) patients who have excess which extends past the knee (▶ Fig. 47.1). The distinction among these groups defines both the technique for skin excision, horizontal or vertical, and the extent of resection.

It is important to remember that the majority of MWL body-contouring patients may be undergoing multiple body-contouring procedures in one or more stages; so, it is important to consider the timing and sequencing of the medial thigh lift. In general, the abdominoplasty/lower body lift is completed prior to medial thigh lifting, typically separated by a healing period of 3 or more months. Addressing the trunk first includes tightening and anchoring the lateral thigh, making the medial thigh procedure more effective. During the surgical planning, the thigh should also be examined to determine whether there is excess skin and subcutaneous tissue, or simply excess skin involved. In patients in whom excess subcutaneous tissue is present, direct tissue excision can be combined with liposuction at a single stage. More commonly, liposuction is performed at an earlier phase (e.g., combined with the lower body lift) in a pre-resection decompressive strategy.

Fig. 47.1 Preoperative images of a 43-year-old woman presenting for medial thigh lift after massive weight loss. She had previously undergone gastric bypass, which resulted in a 190 lb weight loss (BMI of 32.6 upon presentation). Her weight had been stable for several years, and she had undergone abdominal panniculectomy. These images demonstrate excess skin and tissue which extends distal to the knee.

47.2.1 Preparation for Surgery

A thorough history and physical exam during the initial consultation are key to creating the best treatment plan for the patient. Patients who have undergone MWL should have a stable weight for at least 3 months prior to surgery. The body mass index (BMI) of the patient at the time of surgery is important both to the risk of complications and to the quality of the result which is to be expected. Patients with a BMI above 30 to 32 are usually not offered medial thigh plasty until they are able to reduce their BMI to this more favorable range. Pertinent comorbidities should be documented and optimized prior to surgical intervention, and note should be taken of any history of previous lower extremity surgery. As most patients present for body contouring after MWL, investigation into method of weight reduction should be undertaken, as many nutritional deficiencies that may impair wound healing are often present in this population. Smoking cessation is required for at least 1 month prior to intervention. Appropriate preoperative imaging and labs should be obtained based on patient age and risk factors.

Patient expectations should be clearly defined prior to any intervention. It is particularly important to ensure that patients understand the extent of achievable contouring and the possibilities of residual tissue laxity or excess. Patients should be counseled on the risks of surgical intervention, including poor scarring, wound dehiscence, seroma, hematoma, lower extremity swelling and lymphocele formation, infection, and recurrent deformity.

The first medial thigh lift was described by Lewis in 1957.[3,4] His initial technique involved excising tissue in both a horizontal and vertical vector. Lockwood later described performing a vertical vector excision through a horizontal scar pattern, and also advocated anchoring the medial thigh tissue to Colles' fascia to improve results.[5] Despite the incorporation of these principles, horizontal medial thigh lifting is a procedure that is frequently compromised by scar descent (so that it is visible outside of undergarments or bathing suits) or traction on labial tissues, leading to symptoms of dryness (▶ Fig. 47.2).

For these reasons, patients with laxity limited to the upper third of the thigh may be candidates for horizontal excision. However, the majority of patients presenting for body contouring have laxity that extends beyond the upper third of the thigh. This must be addressed with a vertical excision pattern. The fundamental aspect of this approach to medial thigh lift is to use the vertical tissue resection to reorient the vertical tension vector in a horizontal fashion. This horizontal pull in the anterior and posterior planes allows for successful skin and thigh circumference reduction.

The patient is marked in the preoperative holding area prior to induction of anesthesia. The anterior extent of excision is planned using a variant of the lift and drop technique, with the proximal dog-ear taken out anteriorly along the groin. The final scar is designed to run along the medial aspect of the leg in an area of minimal visibility. The patient is placed in the supine position, antibiotics are administered, and general anesthesia is induced. The legs are abducted and may be supported in this position by stirrups, spreader bars, or bolsters. Pneumatic compression devices are placed prior to induction.

The anterior skin marking site is incised sharply, and dissection is carried out through the superficial fascia. At this plane, the tissue is mobilized from anterior to posterior. It is critical to maintain this level of dissection, which helps preserve the saphenous vein as well as the surrounding deep lymphatics. As the dissection is carried more proximally in the groin region near the femoral triangle, it is important to preserve all fat in this area and merely undermine skin, in an effort to avoid the underlying lymphatics. The posterior saphenous branch is consistently encountered coursing at an oblique angle into the elevated flap, and is ligated.

Fig. 47.2 Postoperative photos of matured scars after horizontal thigh lift and a massive weight loss patient. The scar has descended from the perineal crease.

During the dissection from anterior to posterior, the elevated tissue is periodically advanced to determine the limits of dissection, with the goal being to minimize elevation of nonresected tissue and thus minimize surgical dead space. This measurement ultimately determines the extent of skin resection. In order to help conceal the scar proximally over the groin, we have followed the example of Kenkel and Eaves and added a "bottleneck" proximally, removing slightly more proximal anterior skin to better position the final scar.[6]

Once the final extent of tissue excision is determined, hemostasis is achieved. In most cases, the tissue may be closed without a drain in place. However, drains may be considered for patients with an elevated BMI or those undergoing concomitant liposuction. Our approach to the excision and closure is a "close-as-we-go" philosophy. Closing the tissue in conjunction with the excision allows the closure tension to be continuously assessed for consistency. In the unlikely scenario that resection

in a segment makes closure difficult, a portion of the incised flap can be repositioned back in the wound to assist in closing the wound. The superficial fascia is typically closed in interrupted fashion with a heavy polydioxanone suture (0 or 1). The skin is closed in one or two running layers of 3–0 or 4–0 monofilament absorbable suture. Topical skin adhesives may be utilized to dress the wound. Compression garments may be used immediately postoperatively based on surgeon's and patient's preference.

47.2.2 Postoperative Care

In properly selected patients, medial thigh plasty is safely performed in the ambulatory setting, and may also result in less postoperative complications.[7] Patients are encouraged to ambulate early postoperatively and gradually increase activities as comfort allows. Antibiotics are not routinely prescribed postoperatively. The wounds are examined for any evidence of infection or dehiscence, and minor wound complications are treated with daily wet-to-dry dressings until healing by secondary intention occurs. Compression garments may be utilized on a case-by-case basis depending on the use of concomitant liposuction, the presence and extent of swelling, as well as patient's preference. Postoperative deep vein thrombosis chemoprophylaxis is recommended by some authors, but there is no currently consensus over use, timing, or duration.

47.3 Outcomes and Complications

Medial thigh lift is an effective method of treating thigh excess, especially in patients who have undergone MWL (▶ Fig. 47.3; ▶ Fig. 47.4). In patients seeking body contouring after MWL, quality-of-life (QOL) assessment is an important endpoint to evaluate the effectiveness of therapy. Some small case series have shown that following medial thigh plasty, patients report increased overall satisfaction rates and are willing to trade the scar burden for improved functional status and aesthetic appearance.[8] Overall patients have shown significantly improved mean QOL scores postoperatively when compared to preoperative evaluation.[9,10] Furthermore, these scores did not change over time, revealing that the improvements in quality are sustained. Currently, two new patient-reported outcome instruments are being developed, the Body-QoL and the BODY-Q, which will serve to measure satisfaction after body-contouring procedures specifically.[11,12] Once utilized, these tools will further elucidate the postoperative QOL changes in this patient population.

While the positive impact on patients from medial thigh lifting is significant, reported overall complication rates following medial thigh lift range from 40 to 68%.[9,13,14] In a review by Gusenoff et al, the authors concluded that sex, BMI, smoking, diabetes, hypothyroidism, and concomitant liposuction at the time of thigh plasty were not associated with increased complications postoperatively.[9] The commonly cited risk factors include anemia, older age, elevated BMI prior to weight loss, elevated BMI at the time of surgery, and hypertension.[9,14] Additionally, a larger resection length and additional procedures at the time of thigh plasty have also been associated with increased risk of complications.[9,15] The most common complication is the development of limited open wounds, especially proximally. These can typically be managed conservatively until healing by secondary intention occurs. Other common complications following body-contouring surgery include seroma, hematoma, soft-tissue infection, lower extremity edema, scar widening, pulmonary embolism, and labial distortion. A review by Shermak et al of MWL patients following body-contouring surgery cited a seroma rate of 18% with lower body-contouring procedures. Additionally, the authors concluded that in patients with a BMI greater than 30, the major risk factor for seroma formation was the weight of skin removed.[16]

Development of lower extremity edema can occur after medial thigh plasty. While relatively uncommon, lower extremity edema

Fig. 47.3 (**a,b**) Pre- and (**c,d**) postoperative photos of the patient described in ▶ Fig. 47.1. She ultimately underwent direct medial thigh lift in a two-stage procedure. The initial procedure addressed the medial thigh excess in the upper, middle, and distal thirds of the thigh, while the medial knee and leg excess was excised in a separate second stage. Note the bottleneck extension onto the anterior thigh proximally.

Fig. 47.4 (a) Massive weight loss patient who experienced a 400-pound weight loss by combination of bariatric surgery and lifestyle changes. He underwent three separate procedures: abdominoplasty and lower body lift, full length thigh plasty, and chest contouring/brachioplasty. (b) Photo demonstrates scar burden from full length thigh plasty at approximately 6-month follow-up. Courtesy of Bruce Mast, MD

has been reported to develop in 9 to 29% of cases.[9,13,17,18] Utilizing a superficial dissection superiorly and preserving the saphenous vein are important to reduce this risk.[9,19] Additionally, using a full length vertical incision may be associated with increased risk of lower extremity edema.[9,18] Many of these cases resolve with conservative management after 3 months, and almost all patients have improvement by 1 year.[9] There are various strategies in regard to compression garments postoperatively. Some authors recommend immediate compression, others recommend delayed compression, and some recommend no compression at all. Currently, there is not enough information known to make an evidence-based recommendation on compression strategies to prevent lower extremity edema.

There have been no large prospective studies examining thrombotic complications exclusively in the medial thigh lift population. However, the Caprini risk-assessment score has been validated for use in the plastic surgical population, and can be a useful tool to determine if a patient is at high risk for postoperative thrombosis. The overall rate of deep vein thromboses in body-contouring patients ranges from 0.05 to 3%, and risk factors identified include BMI greater than 35, older age, and inpatient admission.[20,21] However, since a relatively small percentage of these patients had isolated medial thigh plasty, the true incidence of venous thromboembolism or any benefit associated with pharmacologic prophylaxis following thigh lift still remains uncertain.

The medial thigh lift is a safe and effective procedure to address excess thigh tissue. The procedure is most commonly performed in the MWL population, and has been shown to significantly improve QOL postoperatively.

47.4 Review Questions

47.4.1 Fill in the Correct Answer

1. What is the most common population of patients who seek consultation for thigh lift or reduction?
2. What structure is necessary to preserve in order to reduce the risk of postoperative lower extremity edema?
3. What is the most common complication arising from medial thigh lift and how is it treated?
4. In the order of body-contouring surgery, when should the medial thighs be addressed?
5. When would a horizontal excision alone be an acceptable technique for medial thigh lift?

47.4.2 Answers

1. The massive weight loss population.
2. Greater saphenous vein should be preserved with superficial dissection to lower the risk of the development of postoperative lower extremity edema.
3. Minor wound healing complications are the most commonly encountered problems after thigh lift procedures. This is most commonly treated conservatively with local wound care until healing by secondary intention occurs.
4. Patients who undergo medial thigh lift surgery are often massive weight loss patients with concurrent truncal and abdominal obesity and these areas should be addressed prior to medial thigh lift.
5. This is appropriate for patients who present with upper third thigh laxity.

References

[1] American Society of Plastic Surgeons. (2019). 2014 Plastic Surgery Statistics. [online] Available at: https://www.plasticsurgery.org/news/plastic-surgery-statistics?sub=2014+Plastic+Surgery+Statistics [Accessed 24 Mar. 2015]
[2] Herman CK, Hoschander AS, Wong A. Post-bariatric body contouring. Aesthet Surg J. 35(6):672–687
[3] Lewis JR , Jr. The thigh lift. J Int Coll Surg. 27(3):330–334
[4] Lewis JR , Jr. Correction of ptosis of the thighs: the thigh lift. Plast Reconstr Surg. 37(6):494–498
[5] Lockwood TE. Fascial anchoring technique in medial thigh lifts. Plast Reconstr Surg. 82(2):299–304

[6] Kenkel JM, Eaves FF , III. Medial thigh lift. Plast Reconstr Surg. 122(2):621–622
[7] Buchanan PJ, Nasajpour H, Mast BA. Safety and efficacy of outpatient lower body lifting. Ann Plast Surg. 70(5):493–496
[8] Bruschi S, Datta G, Bocchiotti MA, Boriani F, Obbialero FD, Fraccalvieri M. Limb contouring after massive weight loss: functional rather than aesthetic improvement. Obes Surg. 19(4):407–411
[9] Gusenoff JA, Coon D, Nayar H, Kling RE, Rubin JP. Medial thigh lift in the massive weight loss population: outcomes and complications. Plast Reconstr Surg. 135(1):98–106
[10] van der Beek ES, Geenen R, de Heer FA, van der Molen AB, van Ramshorst B. Quality of life long-term after body contouring surgery following bariatric surgery: sustained improvement after 7 years. Plast Reconstr Surg. 130(5): 1133–1139
[11] Danilla S, Dominguez C, Cuevas P, et al. The Body-QoL(®): measuring patient reported outcomes in body contouring surgery patients. Aesthetic Plast Surg. 38(3):575–583
[12] Klassen AF, Cano SJ, Scott A, Tsangaris E, Pusic AL. Assessing outcomes in body contouring. Clin Plast Surg. 41(4):645–654
[13] Bertheuil N, Thienot S, Chaput B, Varin A, Watier E. Quality-of-life assessment after medial thighplasty in patients following massive weight loss. Plast Reconstr Surg. 135(1):67e–73e
[14] Bertheuil N, Thienot S, Huguier V, Ménard C, Watier E. Medial thighplasty after massive weight loss: are there any risk factors for postoperative complications? Aesthetic Plast Surg. 38(1):63–68
[15] Coon D, Michaels J , V, Gusenoff JA, Purnell C, Friedman T, Rubin JP. Multiple procedures and staging in the massive weight loss population. Plast Reconstr Surg. 125(2):691–698
[16] Shermak MA, Rotellini-Coltvet LA, Chang D. Seroma development following body contouring surgery for massive weight loss: patient risk factors and treatment strategies. Plast Reconstr Surg. 122(1):280–288
[17] Capella JF. The vertical medial thigh lift. Clin Plast Surg. 41(4):727–743
[18] Gusenoff JA. Prevention and management of complications in body contouring surgery. Clin Plast Surg. 41(4):805–818
[19] Ellabban MG, Hart NB. Body contouring by combined abdominoplasty and medial vertical thigh reduction: experience of 14 cases. Br J Plast Surg. 57(3): 222–227
[20] Wes AM, Fischer JP, Nelson JA, Serletti JM, Kovach SJ, Wu LC. Venous thromboembolism in body contouring: an analysis of 17,774 patients from the National Surgical Quality Improvement (NSQIP) Databases. Plast Reconstr Surg. 134(4) Suppl 1:121–122
[21] Shermak MA, Chang DC, Heller J. Factors impacting thromboembolism after bariatric body contouring surgery. Plast Reconstr Surg. 119(5):1590–1596, discussion 1597–1598

Index

Note: Page numbers set **bold** or *italic* indicate headings or figures, respectively.

G

H

S

T

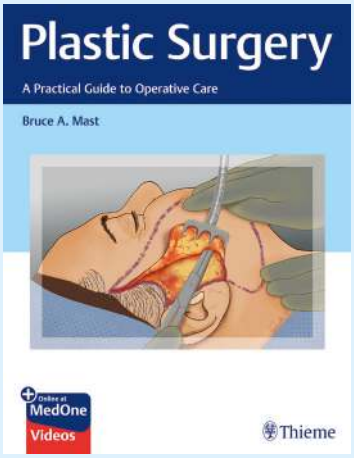
U

V

W

X

Z



Important Notes

- The personal access code is disabled once the e-book is first activated. Use of this product is restricted to the buyer or, for library copies, authorized users.
- Sharing passwords is not permitted. The publisher has the right to take legal steps for violations.
- Access to online material is solely provided to the buyer for private use. Commercial use is not permitted.

MedOne

Medical information how and when you need it.

Access your free e-book now!

With three easy steps, unlock free access to your e-book on MedOne, Thieme's online platform.

1. Note your personal access code below. Once this code is activated, your printed book can no longer be returned.

   EBRqSnthsH8tJ

2. Scan this QR code or enter your access code at **medone.thieme.com/code**.

3. Set up a username on MedOne and sign in to activate your e-book on most phones, tablets, or PCs.

Quick Access

After you successfully register and activate your code, you can find your book and additional online media at **medone.thieme.com/9781626232587** or with this QR code.