Facial Plastic and Reconstructive Surgery

A Comprehensive Study Guide

Brian J.-F. Wong
Michelle G. Arnold
Jacob O. Boeckmann
Editors

Second Edition

Springer

Facial Plastic and Reconstructive Surgery

Brian J.-F. Wong
Michelle G. Arnold
Jacob O. Boeckmann
Editors

Facial Plastic and Reconstructive Surgery

A Comprehensive Study Guide

Second Edition

Springer

Editors
Brian J.-F. Wong
Department of Otolaryngology
Head & Neck Surgery
University of California Irvine
Irvine, CA
USA

Michelle G. Arnold
Department of Otolaryngology
Head & Neck Surgery
Naval Medical Center San Diego
San Diego, CA
USA

Jacob O. Boeckmann
Facial Plastic and Reconstructive
Surgery Private Practice
Pacific Coast Facial Plastic
Surgery Laguna
Niguel, CA
USA

ISBN 978-3-030-45919-2 ISBN 978-3-030-45920-8 (eBook)
https://doi.org/10.1007/978-3-030-45920-8

© The Editor(s) (if applicable) and The Author(s) 2021
This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.
The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.
The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, expressed or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Preface

Introduction to First Edition

This review book was undertaken as a project designed to address a need in the specialty of Facial Plastic and Reconstructive Surgery. There are many wonderful textbooks available in print and hard copy that are exhaustive and detailed on all aspects of this specialty. However, as a resource for review and a guide for study, we found no comprehensive text. In North America and now globally, certifying board examinations in the specialty of Facial Plastic and Reconstructive Surgery are gaining broad acceptance as a metric of certification, professional excellence, and achievement. Hence, an aim of this book is to aid those who wish to pursue these standardized examinations.

Admittedly, part of our motivation was selfish. Each of us will have to take Maintenance of Certification Examinations in the near future, and we recognized a need for a concise study guidebook for Facial Plastic and Reconstructive Surgery. A study guide is softcover and something that lives in your backpack. It is designed to be annotated with notes and scribbled on. It needs to be light and easily carried. And there is something ethereal about paper that remains transcendent at least for the current rising generation of Facial Plastic Surgeons. *Otolaryngology—Head and Neck Surgery* has such guides with *K.J. Lee's Essential Otolaryngology* or Reza Pasha's *Clinical Reference Guide* serving as excellent examples. Hence, we felt there was a need and thus addressed it. Our approach toward developing this review book and study guide is rather novel, and we took our inspiration from "crowdsourcing" and reached out to others for content. Naturally, we focused on those who had a vested interest in producing a practical review book, the actual examinees. For the most part, with this first edition, we identified fellows in training, who would soon take a board certification examination in Facial Plastic and Reconstructive Surgery, and asked them to write the chapters. These are individuals who, at this point in their careers, are focused and most directed at understanding the subtlety as well as esoterica that permeates this field. We feel this multitude of voices and perspectives, though it does lend to some variability in content and organization, provides a richer, more constructive and informed read.

This is the first edition, and with the support of our managing editors at Springer, the first of what we hope to be many yearly revisions. As such, each chapter was written de novo, and each author had a unique view with respect to identifying, structuring, and presenting material pertinent for the advanced reader. This was not designed as a textbook or introductory volume for beginners. Significant base knowledge and understanding is critical and important. This book was conceived as a concise resource that would allow someone to review quickly relevant information in this specialty.

Each year, we will recruit a new set of authors, who will edit and revise each and every chapter. Over time, this iterative approach hopefully will result in an exhaustive and succinct survey of the specialty and evolve into the ideal preparatory text for the various board examinations in North America and abroad. While we feel it will take one or two more iterations before this edition hits its stride, we believe we have a solid foundation. To that end, in this inaugural edition, we are very fortunate to have as contributing authors three AAFPRS Anderson Prize winners, and their chapters are elegantly written, concise, and to the point.

This project has been our labor of love and the product of thousands of emails, innumerable late night phone calls, and brainstorming sessions. We also worked closely with two artists who illustrated this book and generated over 90% of the original artwork contained herein. Brian and Aaron Lemieux are identical twin brothers, and in addition to being first-rate medical illustrators, they also happen to be brilliant medical students. We feel this is yet another reason why our book stands apart.

We believe this review book to be a living body of work, as each year it will be updated and reviewed by a new set of 15–20 surgeons pursuing advanced training in Facial Plastic and Reconstructive Surgery. We are grateful and appreciative of the efforts of Daniel Dominguez and Rebecca Amos at Springer, who have shepherded us through this process and provided guidance along every step of the way.

Lastly, we dedicate this book to our spouses and our mentors.

Irvine, CA, USA Brian J.-F. Wong
San Diego, CA, USA Michelle G. Arnold
San Clemente, CA, USA Jacob O. Boeckmann

Introduction to Second Edition

Our initial goal was to revise this text on an annual basis. However, the economics and realities of contemporary academic publishing have made this a significant challenge. It has been sometime since the first edition was published, and with this second edition we hope to have improved the book by adding additional questions that might guide the reader and further focus their studies. A couple chapters have been condensed, and each and every chapter has been revised under the evaluation of AAFPRS fellows actively preparing for the ABFPRS Board examination. As with the first edition, this text was born out of a labor of love as well as a means of self-preservation in that we editors will at some point take a Maintenance of Certification examination—multiple times. Another trend that we did not consider at the time of the first edition was the growing number of individuals preparing for both the International Board for Certification in Facial Plastic and Reconstructive Surgery and the European Board for Certification in Facial Plastic and Reconstructive Surgery who undergo a similar examination process. Further, many accomplished surgeons have not trained under a structured fellowship program focused on facial plastic surgery. We hope this new edition will provide a meaningful starting point in which to begin studies as well as a concise reference source to rapidly review critical information in the days just before the examination in either Washington, D.C, or London. We would like to thank both the current contributing authors as well as the authors of the first edition for their contributions to this text. We also like to thank both the residents and former fellows in Department of Otolaryngology – Head and Neck at UC Irvine who have provided ad hoc reviews as well as valuable commentary on what information needs to be conveyed, integrated, and presented in a concise manner.

Irvine, CA, USA — Brian J.-F. Wong, MD
San Diego, CA, USA — Michelle G. Arnold, MD
Laguna Niguel, CA, USA — Jacob O. Boeckmann, MD

Contents

Part I Basic Principles, Perioperative Management, and Miscellaneous Topics

1 **Analgesia and Conscious Sedation** 3
Amir Allak and Christian P. Conderman

2 **Basic Techniques, Surgical Anatomy, and Histology** 25
M. A. J. Scott Bevans and Heather E. Lee

3 **Evidence-Based Medicine** 41
Brandyn Dunn, Giriraj K. Sharma, Jacob O. Boeckmann, and Brian J.-F. Wong

4 **General Head and Neck** 47
Taylor Robinson Pollei and Nathan Johnson

5 **Legal, Ethical, and Information Technology Issues in FPRS** . . . 63
Brandyn Dunn and Andrew Kroeker

6 **Psychosocial Aspects of FPRS** 69
Brandyn Dunn and Anita Konka

Part II Reconstruction

7 **Flaps and Grafts** 75
Jennings Boyette, Philip K. Robb Jr., and Irene A. Kim

8 **Facial Paralysis and Facial Reanimation** 107
Eric W. Cerrati and Jon-Paul Pepper

9 **Mohs Surgery** 121
Sim Choroomi and David Sarcu

10 **Neoplasm and Reconstruction** 133
Jeffrey D. Markey and Jon Robitschek

11 **Scar Revision and Dermabrasion** 151
Sagar Patel and James A. Owusu

12 **Trauma** 161
James Teng and John Michael Harbison

Part III Congenital Malformations

13 Cleft Lip and Palate 181
Dana K. Petersen and Christian P. Conderman

14 Craniofacial Anomalies 205
Eugenia Chu and Leslie Irvine

15 Otoplasty 215
Gregory S. Dibelius and Colleen F. Perez

16 Vascular Anomalies 231
Matthew Keller and Tristan B. Klosterman

Part IV Aesthetic Surgery

17 Blepharoplasty 247
Benjamin P. Caughlin

18 Browplasty 263
Boris Paskhover and Virginia P. Teti

19 Cheiloplasty 275
Yuan F. Liu, Jared C. Inman, Farhad Ardeshirpour, and Nikita Gupta

20 Chemical Peels 285
David M. Lieberman and Sudeep Roy

21 Fillers, Botulinum Toxin, Mid-facial Implants, and Tissue Expansion 299
Yuna Larrabee and Nate Jowett

22 Genioplasty 321
Christian Paquet and Matthew D. Johnson

23 Hair Restoration 337
Michelle G. Arnold and Scott R. Owen

24 Lasers in Aesthetic Surgery 351
Jennifer Goodrich

25 Liposuction 361
Andrew D. Beckler and Mehdi Sina

26 Rhinoplasty 371
Gregory Kelts and Jacob O. Boeckmann

27 Rhytidectomy Essentials 395
David Shamouelian and Jordan P. Sand

Index 417

Contributors

Amir Allak, MD, MBA Aspen Facial Plastic Surgery, Salt Lake City, UT, USA

Farhad Ardeshirpour, MD Adresh Facial Plastic Surgery, Los Angeles, CA, USA

Michelle G. Arnold, MD Department of Otolaryngology Head and Neck Surgery, Naval Medical Center San Diego, San Diego, CA, USA

Andrew D. Beckler, MD Department of Head and Neck Surgery, UCLA, Los Angeles, CA, USA

M. A. J. Scott Bevans, MD Otolaryngology, University of Washington, Seattle, WA, USA

Jacob O. Boeckmann, MD Pacific Coast Facial Plastic Surgery, Laguna Niguel, CA, USA

Jennings Boyette, MD Otolaryngology, University of Arkansas for Medical Sciences, Little Rock, AR, USA

Benjamin P. Caughlin, MD, FACS @manyfacesofchicago, Chicago, IL, USA

Eric W. Cerrati, MD Division of Otolaryngology – Head & Neck Surgery, University of Utah, Salt Lake City, UT, USA

Sim Choroomi, BSc (Med), MBBS, FRACS Facial Plastic Surgery, The Lasky Clinic, Beverly Hills, CA, USA

Eugenia Chu, MD Department of Otorhinolaryngology – Head and Neck Surgery, Georgetown University Hospital, Washington, DC, USA

Christian P. Conderman, MD Otolaryngology-Head and Neck Surgery, Division of Facial Plastic and Reconstructive Surgery, Orinda, CA, USA

Gregory S. Dibelius, MD Private Practice, New York, NY, USA

Brandyn Dunn, MD, MPH Department of Otolaryngology Head and Neck Surgery, University of California, Irvine, Orange, CA, USA

Jennifer Goodrich, MD Walter Reed National Military Medical Center, Bethesda, MD, USA

Nikita Gupta, MD Department of Otolaryngology-Head and Neck Surgery, Division of Facial Plastic and Reconstructive Surgery, University of Kentucky Chandler Medical Center, Lexington, KY, USA

John Michael Harbison, MD Otolaryngology-Head and Neck Surgery, University of California-Irvine, Omaha, NE, USA

Jared C. Inman, MD Otolaryngology/Head and Neck Surgery, Loma Linda University, Loma Linda, CA, USA

Leslie Irvine, MD Private Practice, Manhattan Beach, CA, USA

Matthew D. Johnson, MD Department of Surgery/Division of Otolaryngology – Head and Neck, Southern Illinois University, Springfield, IL, USA

Nathan Johnson, MD Ear Nose and Throat Specialty Care, Minneapolis, MN, USA

Nate Jowett, MD Otology and Laryngology, Massachusetts Eye and Ear Hospital/Harvard Medical School, Boston, MA, USA

Matthew Keller, MD Otolaryngology/Head and Neck Surgery, Naval Medical Center San Diego, San Diego, CA, USA

Gregory Kelts, MD Otolaryngology, HealthPartners Specialty Center, Minneapolis, MN, USA

Irene A. Kim, MD Head & Neck Surgery - Facial Plastic & Reconstructive Surgery, UCLA, Santa Monica, CA, USA

Tristan B. Klosterman, MD Otolaryngology, Highland Hospital, Oakland, CA, USA

Anita Konka, MD, MPH Otolaryngology – Head and Neck Surgery, University of South Florida, Tampa, FL, USA

Andrew Kroeker, MD Department of Otolaryngology – Head and Neck Surgery, University of Michigan Health System, Ann Arbor, MI, USA

Yuna Larrabee, MD Otolaryngology, Massachusetts Eye and Ear Infirmary, Quincy, MA, USA

Heather E. Lee, MD Otolaryngology-Head and Neck Surgery,Quatela Center for Plastic Surgery, Lindsay House Surgery Center, Rochester, NY, USA

David M. Lieberman, MD Lieberman & Parikh Facial Plastic Surgery, Palo Alto, CA, USA

Yuan F. Liu, MD Otolaryngology – Head and Neck Surgery, Loma Linda University Medical Center, Loma Linda, CA, USA

Myiam Loyo, MD Division of Facial Plastic and Reconstructive Surgery, Department of Otolaryngology - Head and Neck Surgery, Oregon Health and Science University, Portland, OR, USA

Jeffrey D. Markey, MD Otolaryngology – Head and Neck Surgery, New York University Langone Health, New York, NY, USA

Scott R. Owen, MD Facial Plastic and Reconstructive Surgery, Department of Otolaryngology, University of Iowa Hospitals and Clinics, Iowa City, IA, USA

James A. Owusu, MD Otolaryngology – Head and Neck Surgery, University of Michigan, Ann Arbor, MI, USA

Christian Paquet, BS, MD Biltmore ENT, Facial Plastics & Allergy, Phoenix, AZ, USA

Boris Paskhover, MD Dept. of Otolaryngology, Division of Facial Plastics and Reconstructive Surgery, New York University Langone Medical Center, New York, NY, USA

Sagar Patel, MD Facial Plastic Surgery, Sagar Patel Aesthetics, Beverly Hills, CA, USA

Jon-Paul Pepper, MD Department of Otolaryngology- Head & Neck Surgery, Stanford University, Palo Alto, CA, USA

Colleen F. Perez, MD Otolaryngology-Head and Neck Surgery, Naval Medical Center San Diego, San Diego, CA, USA

Dana K. Petersen, MD, DDS Head and Neck Surgery, Marin General Hospital, Greenbrae, CA, USA

Taylor Robinson Pollei, MD Facial Plastic & Reconstructive Surgery, Williams Plastic Surgery Specialists, Latham, NY, USA

Philip K. Robb Jr, MD Robb Facial Plastic Surgery, Alpharetta, GA, USA

Jon Robitschek, MD Otolaryngology, Brooke Army Medical Center, Fort Sam Houston, TX, USA

Sudeep Roy, MD Refined Facial Plastic Surgery, Los Gatos, CA, USA

Jordan P. Sand, MD Spokane Center for Facial Plastic Surgery, Spokane, WA, USA

David Sarcu, MD Facial Plastic and Reconstructive Surgery, Advocate Medical Group, Aurora, IL, USA

David Shamouelian, MD Otolaryngology – Head and Neck Surgery, University California, Irvine Medical Center, Orange, CA, USA

Giriraj K. Sharma, MD, MS Otolaryngology – Head and Neck Surgery, University of California, Irvine, Orange, CA, USA

Mehdi Sina, MD, MBA Facial Plastic and Reconstructive Surgeon, Pearlman Aesthetic Surgery, New York, NY, USA

James Teng, MD Santa Cruz ENT, Santa Cruz, CA, USA

Virginia P. Teti, MD Department of Otolaryngology, Naval Medical Center Portsmouth, Portsmouth, VA, USA

Brian J.-F. Wong, MD, PhD Department of Otolaryngology Head and Neck Surgery, University of California Irvine, Irvine, CA, USA

Part I

Basic Principles, Perioperative Management, and Miscellaneous Topics

1 Analgesia and Conscious Sedation

Amir Allak and Christian P. Conderman

Sedation and Analgesia

- *Sedation is a continuum* of states from minimal sedation (anxiolysis) to general anesthesia; depth of sedation can be fluid and a patient's clinical status can quickly go from a state of lighter to deeper sedation and vice versa; sedative-analgesics can be given in combination with local and regional anesthesia for greater effect and a reduction in the overall amount of sedative-analgesic medication that may be necessary.
- *Purpose*: (1) allows patients to tolerate unpleasant procedures by relieving anxiety, discomfort, or pain (2) may improve compliance in procedures performed on children and uncooperative adults that are not particularly uncomfortable, but require the patient to not move.
- Sedation can never compensate for an inadequate local anesthetic block; if the regional or local block is deemed inadequate it should be repeated prior to administration of further sedative-analgesic medication.
- Sedation may result in *cardiac or respiratory depression* resulting in hypoxemia and must be appropriately recognized and treated to avoid the risk of hypoxic brain injury, cardiac arrest, and/or death.
- *Primary causes of morbidity* associated with sedation-analgesia are drug-induced respiratory depression and/or airway obstruction.
- Sedatives and analgesics tend to *impair airway reflexes in proportion to level* of sedation-analgesia achieved.
- Practitioners must be able to "*rescue*" patient from a deeper state of sedation than anticipated, i.e., for moderate sedation may include managing a compromised airway or hypoventilation and for deep sedation may include need to manage respiratory or cardiovascular instability with appropriate interventions or medications
- Four variables are used to define the level of sedation: (1) level of responsiveness, (2) airway function, (3) spontaneous ventilation, and (4) cardiovascular function (Table 1.1).

Minimal Sedation drug-induced state facilitating performance of a procedure that maintains normal verbal responsiveness and does not impair airway, ventilation, or cardiovascular function. Cognition and coordination may be impaired. For example, single oral sedative or analgesic or application of <50% nitrous oxide with no other sedative or analgesic.

A. Allak (✉)
Aspen Facial Plastic Surgery, Salt Lake City, UT, USA

C. P. Conderman
Otolaryngology-Head and Neck Surgery, Division of Facial Plastic and Reconstructive Surgery, Orinda, CA, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_1

Table 1.1 Depth of sedation from minimal to a state of general anesthesia is dependent on four variables (figure adapted from ASA practice guidelines for sedation/analgesia by non-anesthesiologists)

Levels of Sedation/Anesthesia				
	Mild	Moderate	Deep	General anesthesia
Neurologic	Normal response	Purposeful response to auditory or physical stimuli	Purposeful response to pain	No response
Airway	No assistance required	No assistance required	Airway may be required	Airway required
Respiratory drive	Spontaneous	Spontaneous	Diminished	Near absent
Cardiovascular	Intact	Intact	Diminished	Diminished

Moderate (Conscious) Sedation depressed state of consciousness with patients remaining purposefully responsive to verbal commands alone or accompanied by light tactile stimulation. *No intervention required to maintain a patent airway*. Ventilation and cardiovascular (CV) function adequate/maintained.

- Example of moderate sedation regimen: initial IV doses of 2 mg versed and 50 mcg fentanyl with titration doses of 0.5–1 mg for versed (up to 1 mg/kg) and incremental doses of 25 mcg of fentanyl until suitable level of sedation is reached.
- Oral sedation cocktails have become popular with in-office procedures and can achieve moderate sedation levels with doses given at specific intervals. These often include a combination of preoperative oral benzodiazepines (valium, versed, etc.), narcotics (hydrocodone, demerol, etc.), and antihistamines/antiemetics with sedative properties (e.g., diphenhydramine, promethazine). Oral sedation does not obviate the need for monitoring and IV access, especially in cases with higher levels of sedation anticipated.

Deep Sedation Depressed state of consciousness during which patients are not easily aroused, even by (repeated) painful stimulation. Purposeful response may be elicited with repeated or painful stimulation, however. *Ability to maintain ventilatory function and a patent airway may be impaired.* CV function usually maintained. It is possible to transition from moderate sedation to deep sedation and the surgeon must be aware of airway and respiratory status to determine when this has occurred. Additional sedation should be delayed and in severe cases, reversal agents should be considered (naloxone, flumazenil). Often these patients require repeated stimulation to increase respiratory drive and/or repositioning/jaw thrusting to assist in airway obstruction.

General Anesthesia state in which patients are not arousable, even by painful stimulation. Ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway, and *positive pressure ventilation may be required* because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. *Cardiovascular (CV) function may be impaired*.

ASA Classification Chart

The ASA (American Society of Anesthesiologists) guidelines are used to measure a patient's overall health and tolerance for procedures (Table 1.2). In general, patients with more severe systemic disease (III and higher) should have a preoperative anesthesia evaluation. Consideration should be given to performing the procedure under general anesthesia as they may be at higher risk of complication(s) during sedation. Certainly if the patient has a difficult airway, general anesthesia is often preferred, as standard noninvasive airway support measures may not be enough if the airway were to be compromised during sedation. Alternatively, the anesthesiologist and surgeon may consider performing the procedure under sedation if the patient's cardio-

Table 1.2 ASA guidelines

ASA I	Healthy patient
ASA II	Mild to moderate systemic disease, medically well controlled
ASA III	Moderate to severe systemic disease limiting activity but not incapacitating
ASA IV	Severe incapacitating systemic disease
ASA V	Profound systemic disease limiting patient life to less than 24 h without surgery
ASA VI	Brain-dead patient, organ donor status

vascular system is deemed too fragile to tolerate the cardiac and vascular effects of general anesthetic agents. The fluctuations these agents can induce in blood pressure and heart rate may induce a cardiac event if preexisting conditions are severe enough.

Summary of Task Force Recommendations for Sedation-Analgesia by Non- anesthesiologists

Patient Evaluation pre-procedure H&P increases likelihood of satisfactory sedation and decreases likelihood of adverse outcomes; components of H&P assessment of major organ system abnormalities, recent illnesses or systemic symptomatology, previous adverse experiences with sedation-analgesia, allergies, current meds and potential drug interactions, time and nature of last oral intake, history of tobacco, alcohol, or substance abuse. Exam should include vital signs (VS), auscultation of heart and lungs, and evaluation of airway, head, and neck (see below).

Assessment of Airway *Anatomic features related to difficult tracheal intubation predisposed to upper airway obstruction*: Hx of previous difficult intubation, stridor, snoring or sleep apnea, history of cervical abnormality or spine surgery, history of head and neck radiation, advanced autoimmune arthritis, chromosomal and other developmental abnormalities; *OSA and obesity increase the risk of airway obstruction during sedation.*

- *STOPBANG* scoring has helped stratify patient likelihood for undiagnosed obstructive sleep apnea. Score >3 significantly increases the risk of OSA
- S-noring, T-iredness, O-bserved apneas, high blood P-ressure, B-MI >35 kg/m^2, A-ge >50, N-eck circumference >43 cm for male/41 cm for female, G-ender = male
- *Exam findings portending difficult airway*: significant obesity, short neck, limited neck extension, decreased hyoid-mental distance (<3 cm in adults), neck masses, cervical spine disease, fibrotic radiation changes, tracheal deviation, and facial dysmorphia
- Oral evaluation: small opening/trismus, edentulous, protruding incisors, loose or capped teeth, dental appliances, high-arched palate, macroglossia, tonsillar hypertrophy, long soft palate with nonvisible uvula (*Mallampati classification*), micrognathia, retrognathia, and malocclusion.
- In general, obstruction of the airway during sedation is the result of lost muscle tone, especially at the level of the tongue base and velopharynx; sedation produces loss of wakefulness and cortical influence on the maintenance of airway tone in addition to a decreased respiratory drive; sedatives can also preferentially depress airway neuromuscular function.

Airway Management Acute airway obstruction or excessive ventilatory depression mandates emergent management—(1) determination that airway obstruction or ventilatory depression exists: visual presence of hypoventilation, paradoxical breathing (abdominal distention and compression of thoracic cavity during inspiratory effort) change in end-tidal CO2 monitor tracing, significant decrease in SpO_2, or lack of patient responsiveness (2) use of verbal and tactile stimuli to prompt patient to breathe and reassess degree of airway obstruction or ventilatory depression (3) if obstruction or ventilatory depression is present, give supplemental O_2, give further verbal and tactile prompting to breathe, jaw-thrust, manual bag mask ventilation, pharmacologic antagonists, calling for help (4) oral airway, mask application and bag ventilation with jaw thrust

Pre-procedure Preparation Electrocardiogram and labwork should be guided by a patient's medical condition and should be considered if results would affect management of sedation-analgesia or there is a pertinent ongoing condition that would need to be assessed (atrial fibrillation, renal failure, etc.); counsel and consent patient before moderate/deep sedation; fasting decreases risks during sedation. In emergency situations (fasting not practical)—consider potential for pulmonary aspiration to determine target level of sedation (less sedated) and consider delaying procedure or protecting trachea with intubation.

Pre-procedure Checklist As is mandated by healthcare governing bodies, a "Time-out" should be performed prior to initiation of any procedure. This is a convenient time to discuss the plan for sedation, patient specific concerns, and contingencies should there be any complications as a result of the sedation. Communication with the procedural staff is critical and may serve as an opportunity to reveal any safety deficiencies (e.g., missing reversal medications, defibrillator not in room) that should be addressed prior to starting the procedure in a proactive fashion.

Monitoring Appropriate and effective monitoring is key to safe sedation and monitoring based on four physiologic variables used to define the level of sedation. Parameters below should be recorded during sedation *at minimum* (1) upon arrival to surgical suite, (2) after first administration of sedative-analgesic/antibiotic, (3) regular intervals during procedure (q 5 min once stable), (4) during initial recovery, and (5) immediately prior to discharge. Importance of respiratory function highlighted by the fact that two of the four characteristics used to define sedation are respiratory in nature and experts agree that *ventilation and oxygenation* are related, albeit different processes, and *should be monitored separately with standard SpO2 and end-tidal CO2 monitoring.*

- *Level of responsiveness*: A patient's response serves as a guide to their level of consciousness and should be routine during all procedures. Many published scales exist, and most rely on two variables—a stimulus (e.g., verbal/tactile stimulation, painful stimulation) and the patient's response to determine the level of sedation. Patients who can communicate verbally likely have an adequate airway and sufficient ventilatory drive. A thumbs-up or similar nonverbal communication can be used in lieu of a verbal response when not feasible (e.g., upper endoscopy) and indicates moderate sedation. Reflex withdrawal to painful stimulation in the absence of the above indicates deep sedation or general anesthesia.
- *Pulmonary ventilation*: Observation of ventilatory function (removal of CO_2) by observation (i.e., watching chest rise), auscultation, or end tidal CO2 monitoring reduces the risk of adverse outcomes and is a means to monitor respiratory rate. This should be continually monitored and end tidal CO_2 monitoring should be considered in all patients undergoing deep sedation and those who cannot be monitored directly during moderate sedation; respiratory disturbances detected by capnography were found to *precede hypoxemia* and serve as an early warning for impending ventilatory compromise.
- *Oxygenation*: Pulse oximetry should be used to monitor *all patients* undergoing sedation-analgesia. Early detection of hypoxemia reduces the risk of adverse events during sedation-analgesia as signs of hypoxemia (e.g., cyanosis, and tachycardia) can be unreliable. SpO_2 in the 80s places a patient's oxyhemoglobin dissociation curve at a tenuous point, and further declines can lead to a rapid decline in saturation levels resulting in low and dangerous oxygen levels. Monitoring oxygenation by pulse oximetry *is not a substitute for monitoring ventilation.*
- *Hemodynamics*: Sedation can produce significant autonomic and hemodynamic reflexes or disturbances including hypo- and hypertension, tachy- and bradycardia, arrhythmias, and myocardial ischemia. BP and HR should be recorded prior and following initial dose of sedation and VS should *be monitored at 5-min intervals* once a stable level of sedation is

established for both moderate and deep levels. Electrocardiographic monitoring can be used to detect more than 80% of ischemia with proper use of modified V5 electrode-chest lead at fifth intercostal space, anterior axillary line. *Continuous EKG* should be provided for *all patients undergoing deep sedation* and those undergoing *moderate sedation with significant comorbidities* (significant CV disease or dysrhythmias) and/or procedures that can evoke autonomic reflexes or hemodynamic disturbances. Also the possibility of hypotension from allergic/anaphylactic responses to perioperative antibiotics should be considered when these are administered and could be accompanied by skin manifestations, airway edema, and other signs.

Availability of personnel A designated individual, i.e., other than the practitioner performing the procedure, should be present to monitor the patient throughout procedures performed with sedation-analgesia. During deep sedation, this individual should have no other responsibilities; however, during moderate sedation, he/she may assist with minor interruptible tasks once the patient's level of sedation-analgesia and VS has stabilized but should always be immediately available for assistance.

Training of personnel The individual responsible for monitoring the patient should be trained in recognition of complications associated with analgesia-sedation. As noted above, since sedation is a continuum, practitioners must be able to rescue a patient from a deeper state of sedation than intended, as outlined above. He/she should understand the pharmacology of the agents used as well as the role and indication for antagonists for opioids and benzodiazepines. At least one individual present besides the surgeon should be capable of establishing a patent airway and positive pressure ventilation when sedation-analgesia is administered. An individual with *advanced life support skills* should be *immediately available (within 5 min) for moderate sedation and in the procedure room for deep sedation.*

Emergency equipment Pharmacologic antagonists and other ACLS/emergency medications (e.g., epinephrine, ephedrine, nitroglycerin, lidocaine), defibrillators, and equipment for establishing an intravenous line and airway (including pediatric endotracheal tubes where appropriate) should be immediately available for all cases of sedation-analgesia.

Supplemental O_2 Supplemental O_2 should be considered for moderate sedation and should be administered *during all cases of deep sedation* to reduce the possibility, frequency, and duration of hypoxemic episodes. Its use has been shown to decrease the magnitude of desaturation and decrease the incidence of ST changes in patient with ischemic heart disease. O_2 at 2–3 L/min or an inspired concentration of approximately 30% O_2 can help maintain normal oxyhemoglobin saturations even in the presence of significantly reduced minute ventilation. Supplemental O_2 can delay recognition of apnea; hypoxemia is not identical to a state of pulmonary hypoventilation.

Use of Sedative-Analgesics

- *Combination*—Sedatives and analgesics used in combination can provide adequate moderate and deep sedation with excellent analgesic effects; however*, combinations may increase likelihood of adverse outcomes,* i.e.*, ventilatory depression, airway obstruction, and hypoxemia*. Each component should be given to achieve the desired effect, e.g., additional analgesic for pain relief and additional sedative for sedation/anxiolysis or reduction in awareness. Respiratory function must be *continually monitored* when given in combination and there should be an appropriate reduction in the dose of each component based on the patient's status.
- *Titration*—Incremental administration improves patient comfort and decreases risks for both moderate and deep sedation, i.e., sedative/analgesic agents should be given in

small, incremental doses that are titrated to the desired end point and not given as one-time bolus. When administered via nonintravenous routes, allowance should be made for the time to bioavailability and potential for unpredictable effect (PO/IM). *Repeated doses of PO meds to supplement sedation are not recommended.*

- *Induction agents for sedation-analgesia*—Agents such as propofol and ketamine can be used to achieve moderate and deep sedation; however, propofol can produce a *rapid, profound decrease in LOC and cardiorespiratory function* culminating in a state of general anesthesia. Ketamine, while associated with less cardiorespiratory depression, can still cause laryngospasm, airway obstruction, and pulmonary aspiration. Moreover, because of its dissociative properties, signs of depth of sedation may be obfuscated. When these meds are used for analgesia-sedation, practitioners should care for the patient *as if it were a case of deep sedation* and should be qualified to rescue the patient from general anesthesia.

Intravenous access When analgesics-sedatives are given intravenously, IV access should be maintained until patient is no longer at risk for CV or respiratory depression (until ready for discharge). In cases where oral sedation is given, the need for access can be determined based on the potential need for additional medication and/or resuscitative drugs. In all cases, an individual with the skills to establish IV access should be immediately available.

Reversal agents Acute reversal for opioids (*Naloxone* 0.4–2 mg initial dose may be repeated every 2–3 min; 0.1 mg/kg in pediatric patients) and benzodiazepines (*flumazenil* 0.2 mg dose, may repeat ×3; 0.01 mg/kg in pediatric patients with max of 3 mg/h) may result in pain, hypertension, tachycardia, and pulmonary edema. The literature supports the use of these agents to reverse opioid-induced sedation and respiratory depression as well as BZD-induced sedation and ventilatory suppression when given alone or in combination with an opioid. Nonetheless, the task force recommended that *respiratory depression be treated initially with encouragement or stimulation to breathe deeply, supplemental O_2 and, if necessary, positive pressure ventilation by mask*. Reversal agents should be immediately available and may be especially helpful where airway control and positive pressure ventilation are difficult. After reversal, patients need to be monitored to ensure that sedation and cardiorespiratory effects do not recur as *flumazenil and naloxone have shorter half-lives than the opioid and BZD they are meant to antagonize*. Routine reversal of sedative or analgesic medication is discouraged; that is, their use was discouraged to routinely awaken patients from a state of sedation at the conclusion of a procedure.

Recovery/discharge Patients may continue to be at significant risk of adverse effects following procedures as stimulation is reduced, delayed drug absorption can occur and slow drug elimination may contribute to residual sedation and cardiorespiratory depression. Patients *should not be discharged until they are near their baseline LOC and are no longer at increased risk for cardiorespiratory depression.* VS *should be stable and the patient should be well hydrated prior to discharge.* Sufficient time should have elapsed after the last administration of reversal agents (if given) to ensure that the patient is not at risk for re-sedation after the reversal has worn off. An outpatient should be discharged in the presence of a responsible adult who will provide transportation and accompany them home. This or another qualified individual should stay with him/her until the pt can function independently. Written instructions should be provided. If a designated adult is unable to assume responsibilities, arrangements should be made to admit the pt to a hospital or aftercare facility for monitoring.

Special situations Patients with severe underlying medical conditions (extremes of age, severe cardiac, renal, pulmonary, hepatic dysfunction, pregnancy, etc.) should be seen by appropriate consultants prior to undergoing moderate or deep sedation. For deep sedation, immediate availability of an individual with postgraduate training in anesthesia will decrease the likelihood of adverse events, and the task force was equivocal in this regard for moderate sedation. An anesthesiologist should be consulted if it is likely that sedation to the point of unresponsiveness is anticipated or in the context of a severely compromised or medically unstable patient (e.g., anticipated difficult airway, severe COPD, CAD, or CHF).

Medications

Local Anesthesia

Acts by preventing depolarization of the ionic electrical gradient across the cell membrane of peripheral nervous system, via *reversible blockade of channels, which prevents rapid influx of sodium ions, which in turn stops propagation of AP* (Fig. 1.1).

- *Amides* (2 "I"s in generic names): amide link more stable, resists changes in pH and temperature, metabolized by hepatic degradation; no plasma metabolism; e.g., lidocaine (xylocaine), bupivicaine (marcaine), prilocaine (citanest), and benzocaine (americaine)
- *Esters*: cocaine, procaine, and tetracaine; ester link is heat labile making esters less stable overall; hydrolyzed by cholinesterases in plasma and liver (cocaine is broken down by plasma pseudocholinesterase)
- *Advantages*: control of airway (when used without sedation), smoother recovery period with better pain control, less stress on CV system, less nausea → early discharge, less cost, less bleeding, awake patient can assist with positioning
- *Disadvantages*: patient apprehension, systemic toxicities of local anesthetics, larger nerves more difficult to block, small risk of permanent nerve damage with intraneural injection, local anesthetics may have decreased potency in acidic (e.g., infected) environment due to configurational changes in anesthetic medication, resuscitation equipment, and personnel should be on hand to manage toxicity

Local anesthetic toxicity and management Toxicity is dose dependent, and risk is reduced by careful calculation and titration of dosage, coadministration of vasoconstrictant, avoidance of intravascular injection (leads to immediate high blood levels that can cause toxicity with small doses), and BZD premedication for its anticonvulsant effect by elevation of seizure threshold. Absorption varies by mucosal surface and application to the tracheobronchial tree can lead to rapid rises to toxic levels if excessive doses are given. Progression from mild toxicity to death is common to all of the aforementioned agents. Supplemental O_2 should be administered at first sign of toxicity and an IV line is mandatory in all but the most minor procedures and especially if the use of powerful sedative-analgesics such as midazolam and fentanyl is anticipated in addition to local.

- *Drug-specific minimum toxic levels*
  - 1% yields 10 mg/mL, 1:1000 yields 1 mg/1 mL
  - *Lidocaine 4.5 mg/kg (7 with epi)*
  - Bupivicaine 3 mg/kg
  - Prilocaine 8 mg/kg
  - Tetracaine 3 mg/kg
  - Procaine 12 mg/kg
- *CNS excitation*—Generally *first* sign of toxicity → agitation, muscle twitching, and hypertension; additional Sx in lower blood levels (1–5 μg/mL of blood lidocaine levels)—lightheadedness, tingling of the lips, tinnitus, and bitter/metallic taste.
- *CNS depression*—Occurs with very high blood levels (20–25 μg/mL), and toxic effects on cardiorespiratory system are seen. Somnolence, coma, and bradycardia accompany shallow respirations and respiratory acidosis ensues. This is followed by apnea, hypotension, and cardiovascular collapse and arrest.
  - Treatment should be directed toward maintenance of respiration and circulation with intubation, positive pressure ventilation, and cardiovascular support with vasopressors and fluids to restore circulation.

	Topical		Infiltrative				
	Concentration	Dose	Concentration	Dose	Onset	Duration	Notes
Epinephrine			1:1000-1:200,000 (1 mg/mL - 1mg/200 mL or 1000 mcg/ml -5 mcg/ml)	10 mcg/kg			
ESTERS							
Cocaine	4-10% (40-100 mg/mL)	3 mg/kg Topical	Not used		Immediate	30-60 mins	
Procaine (Novocaine)			1-2%	14 mcg/kg Adults 5 mg/kg Children	2-5 mins	1 hr	
Tetracaine (Pontocaine)	0.5-2.0% (5-20 mg/mL)	1 mg/kg Topical	0.10 - 0.25 %	1-1.5 mg/kg Infiltrative	20-30 mins	4-5 hrs	
Benzocaine (Americaine)	20%	200 mg max dose	Not used				
AMIDES							
Lidocaine (Xylocaine)	2-4% (20-40 mg/mL)		0.5-2.0 %	4-5 mg/kg(without epi) 7 mg/kg (with epi)	7-15 mins	1.5-2 hrs	
Viepivicaine (Carbocaine	Not Effective		1-2%	7 mg/kg			
Prilocaine (Citanest)	Not Effective		1-2%	7 mg/kg	10-15 mins	1.5-2 hrs	Methemoglobinemia with dose >600 mg
Bupivicaine (Marcaine)	Not Effective		0.25-0.75%	2 mg/kg (without epi) 3 mg/kg (with epi)	30 mins	3 hrs	High degree of cardiotoxicity

Fig. 1.1 Summary of concentration and dosages of common local anesthetic agents. (Table adapted from Essential Otolaryngology and references tables in Eval of Conscious Sedation in FPS)

- *Seizures*: When blood levels reach moderate range (8–12 μg/mL) or higher, a local anesthetic seizure can occur; this can be preceded by a prodrome of slowed speech, jerky tremors, and hallucinations; seizures should be controlled with BZDs (e.g., 0.1 mg/kg of valium or 1–2 mg of versed to raise seizure threshold); once seizure has developed maintenance of oxygenation is of critical importance and supplemental O_2 should be administered immediately with control of airway → patient may require intubation, ventilation, and vasopressors with persistent seizure.
- True *allergies* are rare, although esters more commonly act as allergens and cross-reactivity with amides is not described; that is, amides can be given with a hx of allergy to ester anesthetic; often times allergic reaction is to preservative in solution (e.g., methylparaben, or metabisulfite).
- *Bupivicaine* has a *higher rate of cardiotoxicity* and cardiac depression due to low threshold of myocardium to conduction changes caused by the drug. Intravascular injections can accelerate this process significantly even in lower drug concentrations
- *Prilocaine* has *unique dose-related side effect of causing methemoglobinemia* in doses of 500 mg or more, usually used topically in the oral cavity/oropharynx → Tx: 1–2 mg/kg of IV methylene blue (both application of methylene blue and methemoglobinemia and can cause abnormal Sp02 reading).

Cocaine

- Naturally occurring ester that has both vasoconstrictive and topical anesthetic properties with a narrow therapeutic range.
- Mechanism—blocks norepinephrine reuptake at presynaptic terminal and blocks sodium channels for local anesthetic effect.
- Maximal dose: *3 mg/kg* and the use of more than 200–300 mg should not be necessary (e.g., 200 mg = 5 mL of 4% solution).
- Metabolized by plasma pseudocholinesterase and should not be used in patients with *pseudocholinesterase deficiency* (dx suspected with prolonged apnea after administration of succinylcholine); patients on physostigmine or neostigmine may be prone to the toxic effects of cocaine as these agents inhibit pseudocholinesterase.
- Contraindicated in patients with h/o cardiac arrhythmias or ischemic myocardial disease, patients with h/o epilepsy or seizures, caution is urged in patients with HTN.
- *Toxicity*—primarily CNS in nature—excitability, followed by HA, N/V, tachycardia with rapid progression to delirium, Cheyne-Stokes respiration (cyclical periods of hyperpnea followed by apnea) and convulsions: Thought to be due to overwhelming sympathetic stimulation of CNS and cardiovascular and respiratory systems. If reaction is severe and therapeutic measures are not instituted rapidly, it can progress to respiratory arrest and death.
  - Hypertension and tachycardia can result prior to CNS excitation as well as sensitizing target organs to the effects of SNS stimulation-vasoconstriction, tachycardia, mydriasis, and elevated body temperature.
  - In acute overdose, victim may report confusion, dry throat, and dizziness. Hyperreflexia may be present and tonic–clonic convulsions may occur.
  - Cardiac arrhythmias (especially in patients on MAOis and TCAs)—ventricular ectopy (bigeminy is a cardinal sign of myocardial hypoxia); antiarrhythmics may be necessary, e.g., lidocaine or adrenergic blockade.
- *Tx* - Provide adequate oxygenation, supportive measures, i.e., maintenance of patent airway, ventilation, IV administration of appropriate medications.
- Alpha adrenergic blockade is recommended (*Phentolamine*), as this will prevent *further vasoconstriction and hypertension with selective beta blockade*. The addition of a beta blocker in acute cocaine hypertension is controversial and if utilized, a beta blocker with additional alpha blockade (e.g., labetalol) would be preferred. Sublingual or IV nitro-

glycerin could be of added benefit, especially in the setting of chest pain concerning for cardiac ischemia.
- The latest AHA guidelines state that labetalol is reasonable to treat hypertension >150 mmHg or tachycardia >100 bpm assuming the patient has received a vasodilator (calcium channel blocker or nitroglycerin) within the last hour
- Combination of ketamine and cocaine is best avoided as catecholamine release/effect by cocaine can be enhanced with this combination; halothane also sensitizes myocardium to the effects of catecholamines.

Regional Anesthesia

Knowledge of cervicofacial neural anatomy guides infiltration of anesthetic medications to enhance patient comfort. Blockade of trigeminal and cervical branches achieves desired effect; volume necessary to produce anesthesia is directly proportional to the size of the nerve and larger nerves can take up to 30 min for full anesthetic effect to be seen.

V1— *Supra-orbital* and *supratrochlear* nerves can be blocked by injecting 2 cc at superior orbital rim *using superior orbital notch* as landmark and is done by lateral to medial insertion toward medial eyebrow and ending with ~1 cc over nasal bone (in some cases the lateral/deep branch of the supra-orbital nerve may need to be blocked 1 cm above the superior orbital rim at the ZF suture line in a sub-frontalis plane). *Nasociliary* nerve can be injected with 2 cc injected 2 cm deep to medial brow along medial orbital wall. The *external branch of the anterior ethmoid nerv*e can be blocked at the lower border of the nasal bone *6–10 mm* off the midline (this can reduce painful injections of the nasal tip).

V2— *Maxillary nerve* can be blocked at *foramen rotundum* by pre-auricular approach through sigmoid notch using spinal needle aiming slightly superiorly until the lateral pterygoid plate is reached and redirecting the needle anteriorly and superiorly for ~1.5 cm; 5–10 cc is usually required to block this nerve (paresthesias may be elicited to identify correct location and care must be used to avoid intravascular injection in the infratemporal fossa). *Sphenopalatine ganglion* can be blocked via greater palatine foramen. A 4 cm 27 gauge needle is used, and the *midline and buccal edge of second maxillary molar* serve as landmarks with foramen lying half way between these two points; needle is used to locate foramen and is inserted approximately 1.5 cm and 5 cc injected after withdrawal to avoid intravascular injection. *Infraorbital nerve* can be blocked where it exits infraorbital foramen *5–8 mm below infraorbital rim* (at line dropped from medial limbus on primary gaze); foramen opens downward and medial; 1–1.5 cc is usually sufficient for this block. *Zygomatico-temporal* block can be performed along posterolateral orbital rim with foramen approximately 1 cm below the lateral canthal attachment (needle inserted 10–12 mm posterior to ZF suture line aiming toward posterior concave surface of lateral orbital rim). *Zygomatico-facial* nerve emerges through foramen on anterior surface of zygoma with multiple branches and exits on anterolateral surface of malar eminence—can be inserted by injecting dime-sized area lateral to junction of inferior and lateral orbital rims; lower mid-face can be a difficult area to block (unless anesthetized at foramen rotundum) and often requires field block.

V3— *Mandibular division* can be blocked as it exits *foramen ovale* in a similar fashion as the maxillary division at the foramen rotundum; after needle contacts lateral pterygoid plate, reorient needle more posteriorly to a depth of 6 cm where paresthesias are elicited and 5 cc of local anesthetic is injected. *Inferior alveolar nerve* block can be performed by passing 5 cm needle into retromolar tissue using *lingula* as landmark for injection and injecting 5 cc. *Mental nerve* (foramen usually below *apex of second pre-molar*: can lie 6–10 mm in either anterior or posterior direction) block is done by injecting 2 cc through mandibular gingivo-buccal sulcus between first and second pre-molars (in some cases, e.g., chin augmentation, additional sensation of chin below mentolabial sulcus may need to be anesthetized by injecting additional local to block contribution from nerve to mylohyoid sensory branch or in some cases the lower branch of the mental nerve). Mental nerve is 2.6–2.8 cm from midline and ~1 cm above inferior mandibular border.

Cervical plexus Superficial branches (lesser occipital, great auricular, supraclavicular, and transverse cervical nerves) can be blocked at Erb's point 1/3 of the way between mastoid tip and sternoclavicular joint; ~10 cc injected 3 cm deep to skin at this point will suffice; great auricular nerve (C2/3) can be blocked 6.5 cm down from lower EAC on line drawn delineating mid-SCM.

Sedatives and Analgesics

Basics Opioid analgesics alone produce potent, dose-dependent analgesia, but little sedation; when used alone these do not result in optimal sedation/amnesia for painful procedures and are therefore normally paired with a benzodiazepine. The surgeon should always be aware that a synergistic effect exists when using opioids and sedatives in combination that can result in dose-dependent and potentially profound respiratory depression and apnea. This may require a dose reduction in both BZD and opioid when given in combination as compared with the individual drug doses in isolation. Successful local/regional anesthetic is also key to minimizing the need for sedatives/analgesics and diminishing complications thereof. Moderate-to-severe pain cannot be effectively treated with moderate IV sedation/analgesia and these procedures require general anesthesia if local anesthesia cannot be used. Clinicians who embark upon moderate sedation in the absence of adequate local anesthesia with the notion that it can provide adequate conditions will inevitably end up with a level of sedation deeper than moderate sedation. Naloxone should be given before flumazenil if significant respiratory depression exists as respiratory depression is more likely due to the opioid's effect.

Opioids/narcotics Group of naturally occurring, synthetic, and semisynthetic medications that act on six opiate receptors (Mu, Kappa, and Sigma most important) in CNS, each having a unique activity profile based on affinity for given receptor and its lipid solubility; Narcotics enhance the effect of other sedatives and reduce the amount of local required.

- *Mu* receptor is responsible for analgesia and euphoria, *Kappa* receptor results in sedation and respiratory depression, and *Sigma* receptors cause dysphoria.
- Clinical findings associated with opioid use:
  - Nausea—due to increased tone at GI sphincters and decreased peristalsis
  - Hypotension—due to peripheral histamine release
  - Pinpoint pupil—characteristic of narcosis due to stimulation of Edinger–Westphal nucleus
- Can produce significant respiratory depression in a dose-dependent manner but usually only produce mild hemodynamic depression
- Can lower seizure threshold in patients with seizure disorders
- Should be used carefully in patients taking other CNS-depressing meds, i.e., phenothiazines (thorazine, compazine), antihistamines, sedatives, and in combination with other narcotics
- Full agonists (e.g., morphine, fentanyl, demerol) have no ceiling effect to analgesia and this effect continues in a linear fashion until effect or adverse effect is achieved
- *Fentanyl*—Most commonly administered opioid with nearly ideal characteristics when used for sedation
  - Highly lipid soluble with immediate onset (and peak effect in 5 min) and short duration when given in small analgesic doses
  - Quickly redistributed through fat and skeletal muscle leading to saturation with repeated doses over 4-h period; poor drug for long-term pain control; increased muscular activity can release medication in recovery room; more potent respiratory depression, less N/V and hypotension
- *Demerol (meperidine)*:
  - Greater lipid solubility → hypotension and dysphoria that may be more profound than morphine

  - Respiratory depression equal with equivalent doses
  - Intramuscular doses may be erratically absorbed
  - *Can interact with MAOis and SSRIs → serotonin syndrome: seizures, coma, hypertension, and pyrexia*
  - Tremors, myoclonus, and seizures can result from accumulation of normeperidine (active metabolite with longer half-life than parent compound that may prolong duration of action); normeperidine seizures are *not responsive to naloxone*
- *Morphine*: Prototypical narcotic
  - Long-lasting (half-life 2–3 h), slower onset of action (poorly lipid soluble), and may cause respiratory depression
- *Remifentanil, alfentanil, sufentanil*—opioids related to fentanyl with similar or higher potency and shorter half life/duration
  - Remifentanil is metabolized by a plasma enzyme and accounts for its ultra-short duration of action—usually administered as bolus followed by drip
  - Along with fentanyl, these compounds do not cause histamine release and may be preferred in patients with CV instability.
- *Dilaudid (hydromorphone)*—congener of morphine that is 3–5 times more potent; onset 15–30 min; half-life 2–3 h
- *Naloxone*—Opioid antagonist at all six receptors with *plasma half-life that is less than that of morphine*, i.e., narcotic overdose may recur after having been reversed due to shorter length of action of antagonist. Reversal can lead to catecholamine release that may lead to CV compromise in patients with underlying cardiac disease. Similarly, it can precipitate withdrawal Sx in chronic narcotic users.

Benzodiazepines (BZDS) Class of sedative-hypnotic medications that act on thalamus, hypothalamus, and limbic system via potentiation of GABA neuronal activity. This effect is mediated by BZD receptor (linked to GABA receptor) causing conformational change with a resultant increased affinity for GABA. This causes an increased influx of chloride ions and subsequent hyperpolarization of the neuron that leads to an inhibitory response preventing propagation of further action potentials. BZDs have *amnestic, anxiolytic, sedative-hypnotic, anticonvulsant, and muscle-relaxing effects*. In general BZDs have *little or no analgesic* properties. Three parenteral BZDs are available in the USA: midazolam (versed), diazepam (valium) (both valium and versed are more lipophilic than ativan), and lorazepam (ativan), which has a longer onset and overall effect.

Hepatic degradation is the only route of excretion mandating dose reduction in patients with liver dysfunction for these medications; patients with hepatic encephalopathy are at increased risk of exacerbation of their Sx when BZDs are given. Age and degree of liver dysfunction must be considered prior to administration of BZDs and may require reduction in dosage. All BZDs are highly protein bound in plasma, and patients with hypoalbuminemia may have more active form of medication in circulation.

Hypoventilation that may be seen is likely due to depressant effects of BZD on respiratory center in brain and BZDs usually do not cause CV compromise (except in states of deep sedation where peripheral vasodilation may occur, resulting in decreased cardiac output and peripheral resistance causing systemic hypotension).

- *Versed (midazolam)*—sedative of choice for most clinicians in short, ambulatory procedures
  - Water soluble with pH-dependent structure. Relatively short-acting due to high lipophilicity with rapid onset; context-sensitive half-life predicts that 1–2 h is often required for recovery
  - ~5× more potent than valium
  - Potent respiratory effects—depresses airway function with significant increases in airway resistance
  - Produces anterograde amnesia more commonly than valium
  - Should be given in small incremental doses; large bolus doses should only be

given if intubation and ventilation are anticipated
  - Dose reduction by half should be considered in elderly patients; liver and kidney play roles in excretion with hepatic blood flow being a main determinant of metabolism and excretion
  - Patients receiving concomitant P450 inhibitors (e.g., azoles, phenytoin, diltiazem) and inducers (rifampin) may need dosing modifications
- *Valium (diazepam)*
  - Long half-life (~30 h for parent compound, up to 80 h with metabolites)
  - Undergoes oxidative metabolism in liver via CYP450 and has active metabolites which may prolong medication effect
  - Respiratory depression is a major side effect
  - Effects are prolonged in elderly patients due to increased volume of distribution as compared with young, healthy patients
  - Generally considered safe during gestation, although its use should be tapered or stopped prior to delivery as neonatal toxicity and withdrawal can be seen in infants
  - May cause *phlebitis* at injection site due to preservatives in solution
- *Ativan (lorazepam)*—not routinely used in procedures requiring sedation, as it is less lipophilic than versed and valium. This results in a longer onset of action (30–40 min) and therapeutic concentrations that remain in the CNS for longer periods.
  - Overall duration of action can last for 6–8 h.

Propofol alkylphenol that is used for induction and maintenance of anesthesia and sedation in minor procedures and for induction in general anesthesia

- Possesses sedative, amnestic, and analgesic effects.
- Highly lipophilic with rapid distribution to tissues and CNS and rapid redistribution to blood resulting in a rapid metabolic clearance.
- Exerts its actions through GABA-mediated interactions, specifically at $GABA_A$ receptors (related but not identical to BZD mechanism of action)
- May rapidly produce a state of general anesthesia. Short-acting with more rapid recovery than seen with midazolam and less amnesia at equal sedative doses. Leads to faster recovery times, and less post-op n/v.
- Decreases in cerebral blood flow and cerebral oxygen consumption can also be seen; can cause reduction in BP and decrease in cardiac output without an increase in HR.
  - Concomitant administration of narcotics can increase hemodynamic effects of propofol and the combination of these two medications should be used with caution in patients with CAD.
- Respiratory depression can be significant, and it is accompanied by depression of airway tone and reflexes. Apnea can occur, especially if given as bolus.
- Often can have narrower ideal therapeutic window with elderly patients—high doses can cause airway obstruction and respiratory depression with lighter doses inducing a disinhibitory and confused state
- Patient must be monitored as under deep sedation when using propofol; best titrated to effect by administration of continuous IV infusion based on weight-adjusted dose.
- Disadvantages: Cost, narrow-therapeutic window, cardiopulmonary complications, and requires the presence of specially trained personnel.

Barbiturates classified by duration of action: ultra-short, short, intermediate, and long-acting; primarily provide hypnosis and do not provide analgesia or muscle relaxation. Multiple sites of action in CNS including multiple voltage-regulated ion channels; most prevalent effect on GABA receptors; allosterically enhance binding of BZDs and GABA agonists and inhibit GABA receptor antagonists

- *Thiopental*: ultra-short duration; used in induction of anesthesia
- *Pentobarbital*: short-intermediate; used as sedative-hypnotic and antiepileptic

Ketamine IV anesthetic, structurally related to phencyclidine and cyclohexamine; produces anesthesia with muscular rigidity and open eyes

- Patients may have purposeful movements unrelated to surgical or noxious stimuli.
- Produces prolonged analgesia, blocks pain signal transmission, and produces anesthesia by blocking sodium channels; causes dissociative state upon emergence that causes patients to hallucinate.
- Dose-related increase in HR and BP through direct stimulation of CNS and sympathetic nervous system (SNS).
- Can cause increases in intraocular pressure.
- *Contraindicated* in patients younger than 3 months of age and in those with histories of airway instability, tracheal abnormalities, active pulmonary disease, CV disease, head injury, central nervous system (CNS) masses, hydrocephalus, porphyria, and thyroid disease and in patients with h/o psychosis.
- Can be associated with high *potential for laryngospasm* when used during upper endoscopy.

Dexmedetomidine alpha-2 agonist with similar mechanism to clonidine with much higher selectivity for its receptor.

- Centrally induces sedation, anxiolysis, analgesia, and hypnosis with some anesthetic effects; peripherally, it attenuates the hyperdynamic response and improves hemodynamic stability by attenuating SNS activity, thereby lowering BP and preventing pain-induced hemodynamic fluctuations.
- Spares respiratory drive and decreases need for supplemental O_2.
- Properties lead to less opioid/BZD use intraoperatively and less post-op nausea.
- Can be effective means of sedation-analgesia in facial surgery and may reduce the dosage of opioid and BZD required to achieve sedation and analgesia.
- Stabilizes CV parameters in response to pain; therefore, infiltration of local does not cause wide swings in BP and HR.
- Primary adverse effects are bradycardia and hypotension.

Clonidine alpha-2 agonist, albeit with less selectivity for its receptor compared to dexmedetomidine.

- Provides analgesia and sedation while stabilizing hemodynamic parameters.
- Due to its unfavorable pharmacokinetics, it is not routinely used for sedative purposes:
  - Delayed onset of action (30–60 min) when given IV
  - Long-lasting, making intraoperative titration and fine-tuning of sedation difficult
  - Propensity to cause long-term orthostatic hypotension, making it further unsuitable in this setting
- Primary use is an adjunct in sedation procedures for perioperative blood pressure control.

Total intravenous anesthesia (TIVA) eliminates paralytics and use of volatile gases using IV agents exclusively for patient sedation. The usual regimen includes deep sedation with propofol and varying amounts of ketamine, midazolam, or fentanyl/remifentanyl.

- Should be administered by a provider with postgraduate training in anesthesia due to the use of propofol and requirement of drip management.
- Consider invasive airway management if preferred (laryngeal mask airway, endotracheal tube)

Special Situations

Sedation/Analgesia in Pediatric Patients

- If IV sedation is used in pediatric procedures, the provider(s) must be prepared for poor patient tolerance as children often become restless and combative during standard IV

sedation regimens. Often an initial bolus is used while local anesthesia is administered to help patient tolerance through the procedure.
- Similar to adult procedures, hypoxemia, presumably due to oversedation, was identified as the most common complication. Laryngospasm is more common in the pediatric population.
- Deeper levels of sedation are associated with a child that may be unconscious, is unable to cooperate, and has its protective reflexes blunted; increased risks of sedation and prolonged recovery room stays are significantly correlated with deeper levels of sedation; can be difficult to distinguish between under- and oversedation in an agitated child. Similar to adult ASA criteria, it is generally felt that ASA class 1 and 2 patients may safely undergo moderate sedation, those in class 3 need further evaluation, and those in class 4–5 may be better served by general anesthesia.
- Standard IV sedation with fentanyl and midazolam is safe and efficacious; however, there is reported incidence of 20% of patients with at least one side effect of sedation including bradycardia, agitation, skin rxn, vomiting, and hypoxemia.
- Ketamine alone has been used for shorter procedures and can be given IM or IV. The patient's respiratory status and airway should be closely monitored for laryngospasm.
- Diazepam metabolism is slower in infants compared to school-aged children; fentanyl is variably metabolized in the liver and may not be an ideal sedative for infants as it has been associated with significant apneas in infants <3 months.
- Current standard of care for monitoring patients undergoing pediatric procedures is continuous pulse oximetry and visual assessment of the patient; additionally, ventilatory monitoring should be done continuously by a nurse dedicated to monitoring the child.

Malignant Hyperthermia (MH)

- *Autosomal dominant* transmission most commonly due to abnormality of *ryanodine receptor* (although other mutations exist) resulting in *abnormal calcium metabolism* triggered by a sensitivity to volatile anesthetics (halothane [most potent], enflurane, isoflurane, sevoflurane, desflurane) and depolarizing paralytic agent, succinylcholine. When used in combination, they can result in explosive onset of MH. These agents cause a perturbation of the skeletal muscle membrane resulting in skeletal muscle hypermetabolism, with prolonged contraction and state of O_2 consumption, and release of lactic acid, CO_2, phosphate, and heat. Anaerobic metabolism follows and the plasma membrane begins leaking intracellular ions and myoglobin with a subsequent rise in serum potassium and myoglobinuria.
- Signs of MH include tachycardia and tachypnea, increased $ETCO_2$, hyperkalemia, muscle rigidity and eventual rhabdomyolysis, acidosis, elevated body temperature (*may NOT be immediate*), and masseter rigidity/spasm (may be more pronounced in children) which may serve as an initial warning sign.
- MH-susceptible individuals (those with a first-degree relative with a h/o MH) can be tested via genetic testing or in vitro muscle biopsy (*caffeine halothane contracture test*).
- Local anesthetics, opiates, and nondepolarizing paralytic agents *do not trigger* MH.
- If MH-susceptible individual proceeds with procedure, preoperative precautions must be performed including washing out anesthetic machine (with high-flow oxygen and/or use of charcoal filters) as large amounts of volatile gases can remain in reservoirs of such machines.
- Tx: *Dantrolene should be given immediately* (acts to stabilize ryanodine receptor and reduce efflux of Ca^{2+} from sarcoplasmic reticulum, 2.5 mg/kg as rapid bolus through large-bore IV line repeating at 5-min intervals until signs of acute episode are reversed; once patient has been stabilized [usually following transfer] infusion at 10 mg/kg/day should be given for at least 24 h after initial successful tx). Transfer to an ICU (if procedure is being performed at an outpatient facility) should be instituted immediately. Additional measures

that should be implemented immediately are *cooling of body temperature, correction of electrolyte imbalances, and immediate cessation of all volatile gases*.

Operating Room Fires

- Cutaneous facial surgery is the second most commonly affected site (tonsil #1) for occurrence of OR fires. Additional high-risk procedures include airway procedures and laser procedures of the head and neck.
- Majority of cases occurred while supplemental oxygen was being used, and buildup of oxygen beneath a drape was cited as a reason for the fire in cases of cutaneous surgery.
- Requires vigilance to prevent the fire triad of ignition: *ignition source* (electrocautery, lasers, fiber-optic light cables), *fuel* (ETT, drapes, towels, sponges, pt's hair or skin, alcohol-based solutions), and *oxidizer* (oxygen or nitrous oxide).
- Provision of O_2 via nasopharyngeal tube may reduce the local concentration of O_2 in the environment, thereby reducing the risk of fire; cautery should be performed at least 5 cm away from supplemental oxygen source.
- Selective use of supplemental O_2 can limit the risk, as can the use of the lowest possible inspired O_2, waiting a 60-s period prior to use of ignition source if O_2 is given.
- Presence of fire may be heralded by abnormal sound (cracking) or burning odor, flash, or flame.
- *Management of fire*
  - The presence of a fire should be noted and all team members should be made aware as to the presence of a surgical fire.
  - The flow of all gases should be stopped and the fire should be extinguished as rapidly as possible—initially using water and saline, and smothering the fire.
  - The fuel, such as drapes, tubes, and gauze, should be removed from the patient as rapidly as possible.

    If the fire occurs in the *airway*, the ET tube should be removed as quickly as possible, the gas should be stopped, remove all flammable and burning materials from the airway, and pour saline into patient's airway to cool tissues and extinguish residual embers.

    Additionally, the tube should be evaluated for possibility of any residual pieces/remnants in the patient's airway and rigid bronchoscopy should be performed to assess tissue damage and remove any residual foreign materials.
  - Following control of the fire, airway support should be provided as quickly as possible with bag-mask ventilation without the use of supplemental oxygen and/or nitrous oxide, if possible.

Questions

1. A patient is undergoing a facelift under sedation and lidocaine local anesthesia. She begins to fidget and her limbs seem restless. She denies any pain but seems to be talking excessively and is having some tingling in her hands. The nurse asks if you would like to give more sedation. What is the diagnosis?
2. (Continued from #1) The nurse mistakes the bottles and tells you that you have administered an appropriate amount of 0.5% lidocaine (where you actually injected 2% to toxic levels). The patient requests additional numbing medicine and you inject additional lidocaine. Several minutes later the patient begins convulsing. What is the diagnosis and the mechanism behind it?
3. (Continued from #2) As you look to the monitor you notice that the EKG tracing is abnormal. Your loupes are foggy because you are sweating profusely given the situation. What will this tracing most likely show? What is the mechanism?
4. How does the toxicity of local anesthetics progress with further increases in plasma levels of lidocaine? What is the treatment of local anesthetic toxicity?
5. A 75-year-old female presents for removal of a skin lesion overlying the mid-portion of

her sternocleidomastoid. You draw up 10 cc of 1% lidocaine with 1:100,000 epinephrine and hand it to your resident instructing him to inject the patient with local as you see the next clinic visit. In an effort to be efficient he injects all 10 cc deep to the lesion with one stick. He interrupts your visit to inform you that the patient is unresponsive and convulsing on the procedure table. What has likely occurred?

6. A patient comes to your office for nasal obstruction and discussion of septorhinoplasty. Examination reveals a large polypoid nasal mass. You are out of the topical lidocaine and you find a bottle in the storage room with "Cocaine" your retired partner's handwriting but the concentration is not specified. Assuming this is 4%, you use an appropriate amount to anesthetize the patient. When you return to the room, he is flushed, his pupils are large, and has pressured speech. He says he feels warm and has a headache. What has occurred? What are the other signs/symptoms you might expect?
7. (Continued from #5) You immediately remove the pledgets and go to ask your nurse for help. When you return to the room, the bottle of cocaine is now empty in the patient's hand and he is convulsing on the ground- it is clear that he self-administered the remainder of the drug. What are the signs of advanced cocaine toxicity?
8. What is the mechanism of action of cocaine? How is cocaine toxicity managed?
9. (Continued from #6) You are able to establish an airway and IV access and an ambulance is on its way. His seizures resolve after benzodiazepine administration. The nurse tells you his blood pressure is 245/140. Which antihypertensive will you ask for and why?
10. A patient requests topical anesthetic before injection of his buccal mucosa for a lower eyelid lamellar graft. He is very nervous and requests extra numbing medicine. A generous amount of topical benzocaine spray was administered followed by 5 cc of injectable 0.25% bupivacaine with 1:200,000 epinephrine. He becomes hypoxic and cyanotic several minutes later. What is the diagnosis and how would you treat it?
11. After successful induction, paralytic, and intubation for a rhinoplasty on a 24-year-old female, the anesthesiologist turns the table toward you and comments that she "is still tachy" and her end tidal CO2 is higher than he would expect for someone this healthy. You pack the nasal cavity and inject local anesthetic. As you finish opening the nose he states that his airway pressures have increased and the ventilator is having difficulty providing volumes. You notice the patient is biting the endotracheal tube, and it is very difficult for you to open her mouth as she is contracting her muscles of mastication very firmly. Her skin feels warm to the touch. What could be happening and what is the cause of this?
12. (Continued from #9) You and the anesthesiologist confirm that you are concerned for malignant hyperthermia. What medication should be immediately available if an MH crisis occurs? What other measures should be taken if MH becomes evident?
13. A 63-year-old patient presents for reconstruction of a 4 cm cheek defect and receives a combination of versed and fentanyl in addition to local anesthetic. She is no longer responding verbally and only reflexively withdraws from painful stimuli. What does this indicate with regard to the level of sedation and what should be done next for her?
14. (Continued from #13) What are the four levels of sedation? What variables are used to identify the depth of sedation of a patient?
15. (Continued from #13) The patient is reversed with naloxone and flumazenil. How do the pharmacokinetics of these agents affect the recovery and discharge of such a patient having received antagonist medications?
16. In a preoperative clinic visit in preparation for a facelift to be performed under fentanyl/versed conscious sedation, a 59-year-old female asks "what drugs will you give me? I have some valium at home, can I just take that before the surgery?" How are valium

and versed different? Which is more potent? Which has a more rapid onset of duration?

17. (Continued from 16) Her mother, who is in the clinic visit with her, is 79 and has mild liver dysfunction from steatohepatitis. She is scheduled for a facelift in a month. How would you alter the sedation plan for her surgery?
18. (Continued from 16) During her surgery, after administration of initial doses of fentanyl and versed, the patient is still largely awake. Additional doses of both are given, local anesthetic is injected, and the case is started. Her oxygenation begins to dip into the 80s and she is having audible stertor and obstructive events. What is the primary cause of morbidity in procedures performed under sedation?
19. (Continued from 18) Despite verbal and tactile stimulation, and chin/jaw thrust, she continues to have low saturation and obstruction. The nurse reports her blood pressure to be 65/40. How do you manage a patient who has entered a deeper state of sedation than anticipated, i.e., how do you treat a patient who has lost control of his/her airway or becomes cardiovascularly unstable?
20. (Continued from 19) Reversal is successful and her vitals signs and mental status stabilize. Her mother wants to know if "she will choke when she has her facelift as well?" At the time of the preoperative evaluation, how do you determine if a patient is at risk for airway compromise during a procedure performed under conscious sedation?
21. A 4-year-old boy presents to the emergency room after suffering a forehead laceration while playing with his older brother. The emergency room physician offers to provide conscious sedation to allow you to perform a laceration repair. After administering IV ketamine, you inject 1% lidocaine with epinephrine into the soft tissues surrounding the wound. Several minutes into your procedure the patient develops progressive biphasic stridor. What has occurred? What is the treatment?
22. Why is it necessary to give a combination of opioids and sedatives during a procedure? Are there certain risks associated with the use of a combination of these medications?
23. List the benefits of using dexmedetomidine in combination with other agents to produce sedation during aesthetic facial surgery.
24. A mentoplasty is performed on a patient under sedation. A mental nerve block is performed at the second pre-molar in the gingivolabial sulcus. He continues to be very responsive and reacts to surgical maneuvers and manipulation. What can you do to improve the patient's comfort level? Why is this occurring?
25. An anesthesiologist challenges you to do a full-face CO2 laser resurfacing only using regional nerve blocks. Which area will you have the most trouble anesthetizing in this fashion?
26. A 32-year-old female presents for upper lip filler augmentation. She requests a regional block as the previous one was performed with topical alone and this was uncomfortable for her. What regional block is best for the upper lip? What region of the lip is this most likely to leave sensate?
27. What does the pupil look like when patients have received narcotics? Why does this occur? What does it look like when cocaine is given? Why?
28. During an upper and lower blepharoplasty under sedation, the anesthesiologist accidentally forgets to turn the supplemental oxygen off, which is being delivered via nasal cannula partially under the drapes. Electrocautery is used during the lower blepharoplasty and a flash of flame is seen. What are the steps in management?

Answers

1. This patient is exhibiting common early signs of lidocaine toxicity, largely CNS excitation. It would be prudent to review the volume and concentration of lidocaine given and determine if this could be the cause of her issues.

2. The patient has had a seizure as a result of highly toxic serum levels of lidocaine. As potent Na channel blockers, these create hyperpolarization of neurons in the basal ganglia and brainstem causing aberrant epileptic signals.
3. The cardiac rhythm changes with toxic levels of local anesthetics usually show QRS prolongation that can progress to ventricular tachycardia. The cardiac conduction system is inhibited via dose-dependent blockade of sodium channels and to a lesser extent cardiac contractility is inhibited.
4. The first sign of local anesthetic toxicity is CNS excitation. This can be manifested by psychomotor agitation with muscle twitching that may progress to seizures. Additional early signs/symptoms are lightheadedness, euphoria, tingling of the lips, tinnitus, bitter/metallic taste, and hypertension. As the toxic plasma concentration of local anesthetic rises to higher levels, CNS depression can ensue followed by cardiorespiratory collapse. With the first signs of CNS excitation/toxicity, supplemental oxygen should be given and a benzodiazepine should be administered for its anticonvulsant effects to increase the seizure threshold. Additional measures include supportive care, such as ensuring that an IV is in place, and securing the airway in the case of progression of local anesthetic toxicity.
5. The injection was likely into the external jugular vein. When local anesthetic is injected directly intravascularly, toxicity is much easier to achieve when compared with injection into subcutaneous tissue. It is important to always be aware of local vascular anatomy and distribute the drug more gradually and in a dispersed fashion.
6. The patient is experiencing the early stages of cocaine toxicity (Phase I). The CNS signs are mydriasis, headache, tremor, euphoria, elation, hallucination, nausea/emesis. Other signs are elevated BP/HR, hyperventilation, and elevated body temperature.
7. Phase II and Phase III signs include seizures leading to status epilepticus or coma, incontinence, hypertension/tachycardia leading to ventricular fibrillation, hyperthermia, peripheral cyanosis, gasping breathing, and pulmonary edema.
8. Cocaine is an ester local anesthetic that is broken down by plasma pseudocholinesterase. Vasoconstriction results from blockade of norepinephrine reuptake at the presynaptic membrane. Its local anesthetic effect is produced by blockage of sodium channels similar to other local anesthetics.

   Management of cocaine toxicity is primarily supportive. This includes provision of supplemental oxygen, anticonvulsants, antihypertensives, and support of the airway if progression of toxicity ensues.
9. An alpha adrenergic blocker (such as phentolamine) is the drug of choice in this situation. If a selective beta blocker is used, the alpha receptor stimulation will, in theory, cause worsened vasoconstriction. If a beta blocker must be utilized, a drug with added alpha activity should be used (e.g., labetalol). Nitroglycerin can be of additional therapeutic benefit, especially in cases of chest pain concerning for cardiac ischemia.
10. Benzocaine can cause methemoglobinemia. This can be treated with 1–2 mg/kg of IV methylene blue.
11. Most commonly, malignant hyperthermia is caused by a ryanodine receptor abnormality that is transmitted in an autosomal dominant fashion. Following exposure to a trigger, usually a volatile anesthetic gas, or depolarizing paralytic, such as succinylcholine, membrane instability in skeletal muscle results. This leads to a rapid release of calcium with muscular hyper-contraction with rapid consumption of available oxygen leading to a catabolic state with release of CO_2, and lactic acid. As this cascade progresses, muscle fibers are broken down leading to uncontrolled elevation of central body temperature and significant abnormalities of serum electrolytes, most prominently hyperkalemia. Early signs of MH include tachycardia and tachypnea with an increase of expired end-tidal CO_2. Muscular rigidity

and elevated body temperatures are generally seen later on in the course of disease progression.

12. Dantrolene is the medication of choice if an MH crisis occurs. This medication acts at the ryanodine receptor and acts to stabilize the sarcoplasmic reticulum membrane, thereby limiting further calcium ion egress. Additional measures that should be undertaken if MH becomes evident are institution of cooling measures, correction of electrolyte abnormalities, immediate cessation of volatile gases, or other triggers. Transfer to a facility that has ICU capabilities for continued monitoring of the patient should also be considered.
13. Reflexive withdrawal from noxious stimuli indicates at least a level of deep sedation, if not a state of general anesthesia, and requires that the patient be "rescued" from this level of sedation that may be deeper than anticipated or desired. This includes providing supportive care with administration of supplemental oxygen; chin thrust, bag-masking the patient if necessary, and making preparations to intubate the patient if the airway is deemed at risk. Reversal agents should be attempted prior to invasive airways. Similarly, if the patient is in a state of general anesthesia, he/she may be at risk for cardiovascular deterioration and appropriate management with medications and or IV fluids should be instituted where appropriate.
14. Four levels of sedation: minimal sedation/anxiolysis, moderate/conscious, deep, and a state of general anesthesia. The four variables used to define sedation are (1) a patient's responsiveness, (2) airway function, (3) spontaneous ventilation, and (4) cardiovascular status. See also Fig. 1.1.
15. The patient should be watched for a longer period in recovery, as the effect/half-life of the antagonist medications is shorter than those of the opioids and benzodiazepines they are designed to counteract. As a result, a rebound effect with recurrence of sedation may be seen.
16. Versed is the more potent of the two benzodiazepines and has a more rapid onset of action. Additionally, valium is metabolized and some of its metabolites have a similar mechanistic effect, thereby prolonging the overall effect of valium as compared to versed, which does not have active metabolites. Versed also has an amnestic effect in a much larger portion of patients, which is advantageous for procedural anesthesia.
17. Age and hepatic status must also be considered when these medications are given as reduction in the dosage is usually advised in patients with advanced age and hepatic dysfunction. You may consider having an anesthesiologist consultation and possibly decide to perform her case under general anesthesia.
18. The primary cause of morbidity in procedures under sedation is drug-induced respiratory depression, often with loss of airway support, usually with loss of muscular tone and protective airway reflexes. If this is unrecognized, this can lead to significant complications including anoxic/hypoxic brain injury. Cardiovascular complications can also result from oversedation and can contribute to morbidity, cardiac events, etc.
19. A patient who has lost control of his/her airway should receive immediate interventions that aim to reestablish airway support. This includes prompting the patient to breathe or stimulating the patient to do so, supplemental oxygen, chin thrust, bag-mask ventilation. Reversal agents should be initiated to help support the airway and cardiopulmonary systems and should be attempted prior to invasive airways. Cardiovascular support may be administered via intravenous administration of fluids and or pharmacologic means of blood pressure support, i.e., pressors, if necessary. Consideration of rescue medications in the form of pharmacologic antagonists should also be considered under these circumstances.
20. Exam findings portending airway difficulty include short neck, obesity, neck masses, short hyo-mental distance (<3 cm in adult),

large tongue (preoperative evaluation should include assessment of Mallampati classification), facial dysmorphia, limited neck extension, cervical spine disease, trismus/limited mouth opening, micro- or retrognathia, and dental problems including loose or capped teeth, and/or protrusive incisors.

Historical elements that may indicate a patient with a difficult airway include any history of sleep apnea or prior difficult intubation, previous airway procedures/instrumentation, chromosomal or developmental delays, cervical spine disease, or advanced rheumatoid arthritis. STOPBANG score >3 will increase the risk for OSA and may require further workup if indicated

21. The patient is suffering from ketamine-induced laryngospasm. Initial measures include oxygen supplementation and positive pressure ventilation with bag-masking. Additional measures include propofol administration or neuromuscular paralysis with possible intubation.
22. The two medications serve interrelated but different purposes during a procedure performed under sedation. Opioids primarily serve an analgesic role, and have a stronger inhibitory effect on a patient's respiratory drive. Sedatives induce hypnosis and amnesia without significantly blunting the pain response. As a result, these medications are given in combination and titrated until a suitable level of sedation is reached. When given in combination, however, there is a synergistic effect on the cardiorespiratory system and the risk of complication is increased when given together as compared to when they are given in isolation.
23. Dexmedetomidine is a highly selective alpha-2 agonist. It thereby can induce sedation without reducing a patient's ventilatory drive and without having significant effects on the cardiovascular system. This also reduces the amount of sedative and analgesic that may be required during a case. This further reduces the morbidity of sedation with dexmedetomidine and decreases the likelihood of postoperative nausea/vomiting.
24. This occurs due to inadequate local anesthesia along the inferior border of the mandible. While the mental nerve provides sensation to a large portion of the lower lip and chin, additional sensory branches may stem from sensory branches of the nerve to mylohyoid, nerves that leave the inferior alveolar nerve prior to entering the mandibular foramen, or inferior branches of the mental nerve. These additional sensory branches can be blocked by injecting additional local anesthetic along the inferior border of the mandible via an intraoral or transcervical route.
25. The lower cheek does not have a well-defined nerve or combination of nerves that is able to be covered by a regional block.
26. Bilateral infraorbital nerve blocks will anesthetize the upper lip very well. The area that will most likely be left sensate is in the philtrum as this often has contribution from the nasopalatine nerve.
27. Narcotics—miotic pupil; due to effects at the Edinger-Westphal nucleus

    Cocaine—mydriatic pupil due to sympathomimetic effects on iris sphincter muscles
28. The electrocautery should be stopped (ignition) drapes and nasal cannula should be immediately removed (fuel), the oxygen turned off (oxidizer), and the field doused with saline or water. In the head and neck one of the most common causes of surgical fire is oxygen trapped under surgical drapes.

Additional Resources

1. American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists. Practice guidelines for sedation and analgesia by non-anesthesiologists. Anesthesiology. 2002;96(4):1004–17.
2. Anderson JL, et al. 2012 ACCF/AHA focused update incorporated into the ACCF/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task

Force on Practice Guidelines. J Am Coll Cardiol. 2013;61(23):e179–347.
3. Bailey PL, Pace NL, Ashburn MA, Moll JW, East KA, Stanley TH. Frequent hypoxemia and apnea after sedation with midazolam and fentanyl. Anesthesiology. 1990;73(5):826–30.
4. Blake DR. Office based anesthesia: dispelling common myths. Aesthet Surg J. 2008;28(5):564–70.
5. Butz DR, Gill KK, Randle J, Kampf N, Few JW. Facial aesthetic surgery: the safe use of oral sedation in an office-based facility. Aesthet Surg J. 2016;36(2):127–31.
6. Caplan R, et al. Practice advisory for the prevention and management of operating room fires. Anesthesiology. 2008;108:786–801.
7. Difazio CA. Local anesthetics—action metabolism and toxicity. Otolaryngol Clin N Am. 1981;14:515.
8. Ellis DA, Gage CE. Evaluation of conscious sedation in facial plastic surgery. J Otolaryngol. 1991;20(4):267–73.
9. Failey C, Aburto J, de la Portilla HG, Romero JF, Lapuerta L, Barrera A. Office-based outpatient plastic surgery utilizing total intravenous anesthesia. Aesthet Surg J. 2013;1:33(2):270–4.
10. Gay GR. Clinical management of acute and chronic cocaine poisoning. Ann Emerg Med. 1982;11(10):562–72.
11. Gurunluoglu R, Swanson JA, Haeck PC, ASPS Patient Safety Committee. Evidence-based patient safety advisory: malignant hyperthermia. Plast Reconstr Surg. 2009;124(4 Suppl):68S–81S.
12. Horn E, Nesbit SA. Pharmacology and pharmacokinetics of sedatives and analgesics. Gastrointest Endosc Clin N Am. 2004;14(2):247–68. Review
13. Huang R, Eisen GM. Efficacy, safety, and limitations in current practice of sedation and analgesia. Gastrointest Endosc Clin N Am. 2004;14(2):269–88.
14. Iverson RE. Sedation and analgesia in ambulatory settings. American Society of Plastic and Reconstructive Surgeons. Task Force on Sedation and Analgesia in Ambulatory Settings. Plast Reconstr Surg. 1999;104(5):1559–64.
15. Jackson RS, Archibald DJ, Farrior E, Farrior EH. Ambulatory facial plastic surgery centers and malignant hyperthermia. JAMA Facial Plast Surg. 2014;16(3):167–8.
16. Karan SB, Bailey PL. Update and review of moderate and deep sedation. Gastrointest Endosc Clin N Am. 2004;14(2):269–312. Review
17. Lee KJ. Essential otolaryngology. 10th ed. New York: McGraw Hill Medical; 2012.
18. Lightdale JR. Sedation and analgesia in the pediatric patient. Gastrointest Endosc Clin N Am. 2004;14(2):385–99. Review
19. Marcus JR, Tyrone JW, Few JW, Fine NA. Optimization of conscious sedation in plastic surgery. Plast Reconstr Surg. 1999;104(5):1338–45.
20. Nekhendzy V, Ramaiah VK. Prevention of perioperative and anesthesia-related complications in facial cosmetic surgery. Facial Plast Surg Clin North Am. 2013;21(4):559–77.
21. Reyes RJ, Smith AA, Mascaro JR, Windle BH. Supplemental oxygen: ensuring its safe delivery during facial surgery. Plast Reconstr Surg. 1995;95(5):924–8.
22. Shapiro FE. Anesthesia for outpatient cosmetic surgery. Curr Opin Anaesthesiol. 2008;21(6):704–10.
23. Smith LP, Roy S. Operating room fires in otolaryngology: risk factors and prevention. Am J Otolaryngol. 2011;32(2):109–14.
24. Taghinia AH, Shapiro FE, Slavin SA. Dexmedetomidine in aesthetic facial surgery: improving anesthetic safety and efficacy. Plast Reconstr Surg. 2008;121(1):269–76.
25. Urman RD, Punwani N, Shapiro FE. Patient safety and office-based anesthesia. Curr Opin Anaesthesiol. 2012;25(6):648–53.

2 Basic Techniques, Surgical Anatomy, and Histology

M. A. J. Scott Bevans and Heather E. Lee

Wound Healing

Phases of Wound Healing [1, 2] (See Table 2.1)

1. Inflammatory (up to 7 days after injury)
   (a) Endothelial injury starts clotting cascade
      - Exposes: *Collagen*, laminin, and fibronectin
      - Clot: *Platelets* and *fibrin*
      - Releases: *Histamine*, proteases, prostaglandins, serotonin

   (b) *Vasoconstriction* priority (thromboxane A2—lasts 5–10 min)
   (c) *Vasodilation* from histamine, increased vessel permeability (edema) for 48–72 h
   (d) Key cellular response begins:
      - *Granulocytes* (PMNs) arrive within 6 h, maximum influx by 24 h
         – Neutrophils release proteolytic enzymes, free radial species and antimicrobial peptides to assist in digestion of cellular debris and bacterial elimination
      - *Macrophages* dominate by 3 days, essential for wound healing
         – Release TGF-B, FGF, remodeling factors, nitric oxidie
      - *Fibroblast* appear by 48 hours, maximum influx by 15 days
         – *Predominant producer of collagen*, elastin, and fibronectin
         – Differentiate into *myofibrolast*

   (e) End result: *cells ready for proliferation* in wound bed
   (f) Wound strength: *5–10%* of normal tissue
   (g) To promote optimal healing: use *aseptic technique*, *copiously irrigate* with saline (10–15 psi ideal for cleaning without causing damage/seeding), ±antibiotics (i.e., 50,000 U of bacitracin)
2. Proliferative (1–21 days after injury)
   Mediated by fibroblasts, keratinocytes, and endothelial cells.
   (a) *Re-epithelialization* (1–5 days) mediated by neutrophil and macrophage secretion of IL-8, platelet/macrophage secretion of EGF, and TGF-α
      - Creates protective barrier
      - Begins at *hair follicles/adnexa/sebaceous glands*
      - *Rapid when primarily closed*
      - *Promoted by moist environment*
      - Prolonged (3–5 days) when healing secondarily or over poor vasculature

M. A. J. Scott Bevans
Otolaryngology, University of Washington, Seattle, WA, USA

H. E. Lee (✉)
Otolaryngology-Head and Neck Surgery, Quatela Center for Plastic Surgery, Lindsay House Surgery Center, Rochester, NY, USA
e-mail: hlee@quatela.com

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_2

Table 2.1 Phases and characteristics of wound healing

Wound healing phases	Time after injury	Key cellular components	Strength
Inflammatory	<7 days	Platelets, granulocytes, macrophages	5–10%
Proliferative	1 day–3 weeks	Fibroblasts	40%
Maturation	3 weeks–1 year	Myofibroblasts	80%

(b) *Neovascularization* (3–4 days after injury)
- *Granulation tissue* produced by activity of macrophages
- *VEGF* is secreted by keratinocytes, which promotes endothelial cell proliferation and migration
- *Scaffold* holding vessels, matrix of nutrients, cells, fibronectin/collagen
- Present until epithelialization is complete

(c) Collagen Synthesis
- Mediated by fibroblast—direct collagen, elastin, GAG formation
- Requires Vit C and iron for hydroxylation of proline and lysine chains
- Glycosylation of collagen helix results in *procollagen*, which is secreted by fibroblasts
- Cleavage of procollagen → *tropocollagen* → fibril aggregation → *collagen fibers*
- *Type III collagen* predominates early → *type I collagen* during maturation (see below)
- Maximum deposition at 2–3 weeks

(d) Contracture (1–3 weeks after injury)
- Mediated by *myofibroblasts*
- Maximum at 12–15 days after injury, occurs at 0.7 mm/day
- Worse if left open or inflamed
- Contraction lessened by skin grafts (still contract by 20% depending on type of skin graft)

(e) End result: *disorganized collagen, relatively hypertrophied, erythematous scar*

(f) Wound strength: *increases slowly for 2 weeks, linearly for 4 weeks, 50% of final wound strength (40% of normal skin) at 6 weeks*

(g) To promote optimal healing: *close primarily* if possible/applicable, optimize nutritional status (collagen cross-linking), *keep wound clean and moist*, use *occlusive/semi-occlusive or silicone dressings*. Mederma (onion extract) showed no visible benefit in humans in RCTs.

3. Remodeling/Maturation (3 weeks–12 months after injury)

(a) *Collagen remodeling* (continue transition to type I collagen)—increases strength
- Disarrayed fibers are remodeled, becoming *parallel, organized woven*

(b) Decreased vascularity—resolves erythema

(c) Key cellular components:
- *Myofibroblasts*—mediate contraction, then die (except in keloids)

(d) End result: *fully contracted, avascular scar*

(e) Wound strength: *80% of normal skin*
- 3 weeks: 15% original tensile strength
- 6 weeks: 60% original tensile strength
- 6 months: 70–80% original tensile strength

(f) To promote optimal healing: *pressure dressings, massage, sun avoidance*, reducing inflammation (steroid injections into scar only—not surrounding tissue)

Optimizing Wound Healing

Application of basic wound healing science to clinical practice is necessary for optimal wound healing and patient outcomes. The surgeon should make all attempts to identify and correct factors that could result in suboptimal healing when possible. Compromised wound healing can result from a number of factors including:

- Local: infection, radiation, hematoma, neoplasm, wound desiccation, contamination
- Medical comorbidities: diabetes, malnutrition, hypothyroidism, peripheral vascular disease, smoking, immunodeficiency, keloid history, collagen

- Medications: corticosteroids, immunosuppressants, chemotherapy
- Technical: excessive wound tension, poor hemostasis, traumatic tissue handling

Hyperbaric Oxygen

- Useful in select patients at risk of ischemia or poor wound healing, (osteoradionecrosis, irradiated wounds, smokers, diabetics)
- Increases partial pressure of O2 to create pressure gradient for oxygen uptake
  - 30–50 mm Hg normal subcutaneous O2 tension
  - increasing pressure by 2–2.5 atmospheres increases arterial partial pressure to 1500 mm Hg

Vacuum-Assisted Closure (VAC)

- Useful for difficult chronic wounds not readily amenable to granulation tissue production or massive defects requiring complex reconstruction with compromised patient medical condition (e.g., exposed calvarium following malignancy, necrotizing fasciitis)
- VAC exposes the wound to intermittent subatmospheric pressure of 125 mm Hg for 5 min followed by 2 min without suction
  - 4× increase in blood flow
  - 2× proliferation of granulation tissue
- Advantages: allows option for secondary healing and/or less-complex local flap/skin graft repair of wound; limits infection through increased blood flow, removes excess wound fluid, reduces wound care prior to definitive reconstruction

Growth Factors

Ongoing research is looking into improving faulty wound healing and adjuvant treatment for enhanced wound healing. Exogenous administration of specific growth factors to surgical site.

- Platelet-rich plasma (PRP) supplies high levels of platelet products (PDGF, VEGF, TGF-β) to wound bed for improved production of the ECM, improved wound contracture, and increased vascular ingrowth.
  - Research is ongoing to determine optimal method of extraction, application (injection vs. topical)

Traumatic Wounds (See Chapter 2 for More Detail)

- Evaluation—depth of wound: layers, structures involved determine reconstructive approach
  - *Determine if there is functional or sensory deficit before application of local anesthetic*
  - Facial nerve exploration if the injury is lateral to lateral canthus
  - Parotid duct injury/exploration for lateral cheek
- Tetanus status
  - Booster (*tetanus toxoid*) every 10 years
  - *Contaminated wounds*, deep punctures—tetanus booster within *5 years*
  - Less than two prior doses of toxoid, needs tetanus immunoglobulin
- Post-injury use antibiotics:
  - Immunocompromised
  - Rheumatic heart disease or implants
  - Contaminated wounds (bites)
- Bites
  - Humans—more contaminated, treat with broad-spectrum antibiotics for additional coverage against anaerobes and aerobes. Consider delayed repair. Check offending party for infectious diseases like HIV, Hep
  - Dogs and cats—cover Staphylococci, Streptococci, anaerobes, and Pasteurella multocida species for 5 days (see Table 2.2)

Table 2.2 Antibiotic therapy in animal bites

Dog bites—"*Dog*"-mentin
*C*ats (or PCN allergic)—*C*linda & *C*ipro

    - Augmentin BID or
    - Clindamycin with Cipro or
    - Bactrim
  - Monkeys—treat with antivirals also
  - Rabies (unprovoked attack, test the animal) if high suspicion, start treatment with Rabies Ig and vaccine

Surgical Techniques

- Infection prevention (aseptic technique) and control (wound cleanliness—see Table 2.3)
- WHO guidelines for antibiotic use:
  - Within 60 min but prior to incision for Ancef, Unasyn, clindamycin
  - Within 120 min but prior to incision for vancomycin and fluoroquinolones
  - Re-dose when surgery exceeds half-life of drug or if significant bleeding
  - Place incisions in relaxed skin tension lines (see below)
- Debride necrotic tissue and remove foreign bodies (including unnecessary sutures)
- Atraumatic tissue handling (do not crush)
- Use sharp anatomic dissection of tissue and freshen wound edges
- Obliterate dead space (prevents seroma/hematoma formation)
- Avoid tension on epithelial wound edges
  - *Undermining up to 4 cm* from wound edge can relax tension (more doesn't help)
- Trichophytic incisions
  - Cut between follicles on a bias (so hair grows through the incision)
- Suture choices (see Tables 2.4 and 2.5)
  - Smallest needle and suture caliber that is strong enough to resist deformation
  - Least inflammatory
  - Remove permanent sutures in 5–7 days
- Tissue Glue
  - Three layers recommended by most manufacturers
  - Dermabond (Octylcyanocrylate) is strongest among currently available
  - Equally as effective to sutures in lacerations parallel to RSTLs
  - Superior to sutures in lacerations perpendicular to RSTLs
  - Not good for areas of high motion

Table 2.4 Absorbable suture types

Absorbable suture types and characteristics		
Monofilaments	Absorption	50% strength
Fast gut	10–20 days	5–10 days
Plain gut	30–60 days	15 days
Chromic gut	70–90 days	30 days
Poliglecaprone (Monocryl)	3–4 months	1–2 weeks
Polydioxanone (PDS)	6–8 months	4+ weeks
Braided:		
Polyglactin 910 (Vicryl)	2 months	3 weeks

Table 2.5 Nonabsorbable suture types

Nonabsorbable suture types and uses
Monofilaments:
Nylon—skin, vessels Polypropylene (Prolene)—skin, removable running subcuticular Stainless steel—cartilage or bone
Braided:
Nylon or polyester—high tension for long duration (rhytidectomy, otoplasty) Silk—used for ligating vessels, drains (knots would not slip)

Table 2.3 Surgical wound classification—ACS-NSQIP

Surgical wound classification used by ACS-NSQIP [5]				
Wound class	Class I—clean	Class II—clean contaminated	Class III—contaminated	Class IV—infected/dirty
Example	Uninfected, incision in skin after surgical prep	Incision in the respiratory, alimentary, or urinary systems	Open or fresh wounds, surgical wound with gross spillage	Old wounds, evidence of infection in wound
Infection rate	1–5%	3–11%	8–17%	12–27%

- Secondary healing in *<2 cm concave areas* (nose, eyes, ear, and temple) is acceptable [3, 4]
- *Dermabrasion at 4–8 weeks after injury* improves scar appearance

Histology

- Normal Skin Histology
  - Layers of the skin (see Fig. 2.1)
  - Epidermis- avascular layer made primarily of keratinocytes
  - Stratum corneum: keratin-filled anucleated cells eventually lost through desquamation, helps serve as barrier to trauma and infection
  - Stratum lucidum: thickened in palms and soles of feet
  - Stratum Granulosum: lipids from keratinocytes excreted into extracellular space and desmosomes between cells help prevent fluid loss
  - Stratum spinosum: desmosomes on outer surface give cells a "spiny" appearance, contain Langerhans cells, which are dendritic cells that contain birbeck granules and act as antigen presenting cells

    Stratum basale: contain germinal cells of epidermis, melanocytes, and Merkel cells

    Epidermis is separated from dermis by basement membrane

    28-day cycle from stratum basale to shedding at stratum corneum

    Rete ridges: projections of the epidermis that help anchor into the dermis

    Dermis: vascular layer composed mainly of fibroblasts that secrete collagen, elastin, and ground substance.
  - Papillary dermis: loose connective tissue with vascular network responsible for thermoregulation, surrounds adenexal structures
  - Reticular dermis: thicker, compact collagen, important in giving overall strength and elasticity to skin
  - Collagen—*80–90% type I collagen*
  - Scalp—thicker stratum corneum
- Blood supply
  - *Choke vessels under the dermis–subdermal plexus*
  - Capillary network under epidermis
- Skin cancer histology
  - Sites of origination
  - BCC—basal cell layer
  - SCC—keratinocytes
  - Melanoma—melanocytes
- Keloid vs. hypertrophic scars
  - *Hypertrophic scars—more type III collagen*

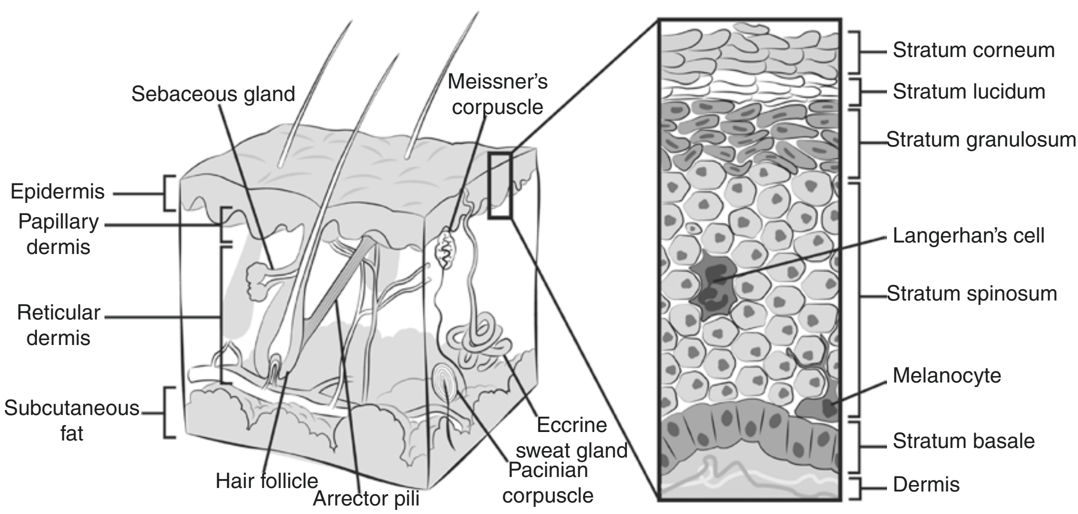


Fig. 2.1 Cross-section of skin (*left*) with cellular component of epidermis (*right*)

  - Keloids—*increased TGF-B expression*, expand *beyond the boundary* of the original injury, failure of myofibroblast apoptosis
- Fitzpatrick skin types (see Table 2.6)
  - Classification of skin response to UV exposure

Surgical Anatomy

- Embryology (see Table 2.7)
- Facial proportions (Figs. 2.2 and 2.3)
  - *Vertically* divided in *1/3 s* by:
    - Trichion
    - Glabella
    - Subnasale
    - Menton
  - *Horizontally* divided in *1/5 s* by:
    - Edge of helix
    - Lateral canthus
    - Medial canthus
  - Divine proportions (Golden Rule)
    - Φ (Greek letter phi) = 1.618…
    - Facial height/facial width
    - Facial height (trichion to midpupillary line to menton)
    - Several others (controversial)
- Superficial landmarks (see Fig. 2.4)
  - *Skin-retaining ligaments of the face*: (Fig. 2.5)
    - Mandibular
    - Masseteric
    - Zygomatic—McGregor's patch
    - Platysmal-auricular
    - Orbicularis
- Facial skeleton
  - Beams (4)

Table 2.6 Fitzpatrick scale

I	Always burns	Pale, fair, freckles
II	Usually burns, sometimes tans	Fair
III	May burn, usually tans	Light brown
IV	Rarely burns, always tans	Olive
V	Moderate constitutional pigmentation	Brown
VI	Marked constitutional pigmentation	Black

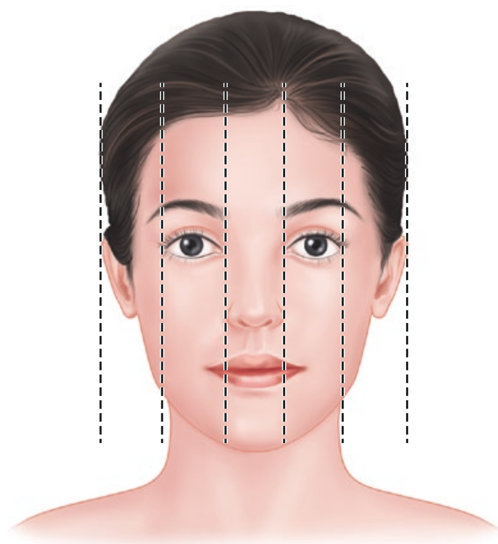


Fig. 2.2 Facial proportions- Vertical

Table 2.7 Embryologic arches and derivatives

Arch	Connective tissue	Artery	Muscles	Nerves
1st	Maxilla, mandible, zygoma, malleus-head and neck, incus-body and short process	Maxillary	Muscles of mastication, mylohyoid, ant, digastric, tensor tympani, tensor veli palatini	V3
2nd	Manubrium of malleus, long and lenticular process of incus, stapes, styloid, hyoid (lesser horn)	Stapedial	Facial expression, post-digastric, stapedius, stylohyoid	VII
3rd	Hyoid (greater horn)	Common carotid	Stylopharyngeus	IX
4th	Thyroid cartilage, cuneiform	Aortic arch, right subclav	Constrictors, cricothyroid, levator veli palatini	X (SLN)
6th	Cricoid cartilage, arytenoid, corniculate	Pulmonary arteries, ductus arteriosus	Intrinsic laryngeal muscles	X (RLN)

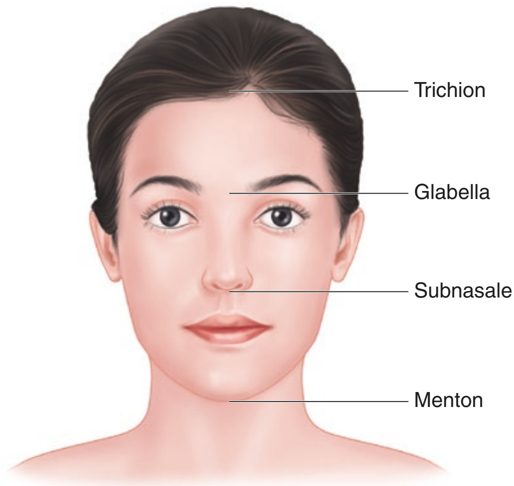


Fig. 2.3 Facial proportions- Horizontal

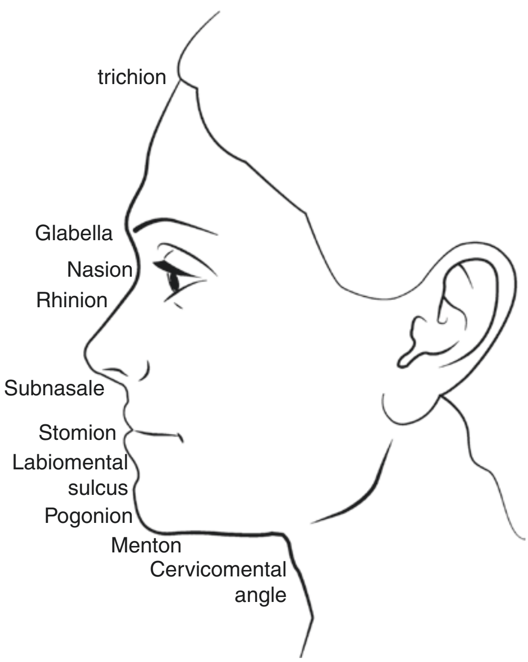


Fig. 2.4 Superficial landmarks (profile view)

Frontal bar
Infraorbital/Zygomatic process
Maxillary alveolus
Mandible

– Buttresses (4)

Nasomaxillary

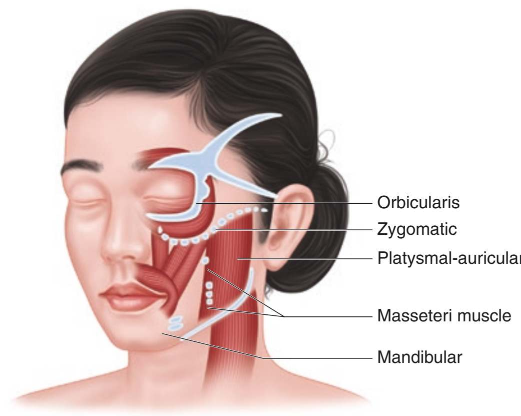


Fig. 2.5 Skin-retaining ligaments of the face

Zygomaticomaxillary—capable of greatest load bearing
Pterygomaxillary
Mandible condyle/ramus

- Cutaneous innervation
  - Anterior ½ from trigeminal nerves
  - Posterior ½ from cervical nerves
- Motor nerves
  - Facial nerve

    Main trunk—five ways to identify it (see Table 2.8)

    Extracranial course—deep to posterior belly of digastric

    Pes anserinus—where temporofacial and cervicofacial branches meet

    - Temporofacial—zygomatic and frontal
    - Cervicofacial—buccal, marginal, cervical

      Temporal: innervates frontalis and upper orbicularis oculi. Runs superficial to the superficial layer of deep temporal facia over ZM arch then divides into 2–4 branches.

      Approximate its course with pitanguy's line—starts 0.5 cm below tragus and extended to brow passing 1.5 cm above lateral brow

      Zygomatic: innervates lateral aspect of orbicularis oculi

Table 2.8 Methods of finding the facial nerve

Identifying the facial nerve:
1. 1 cm deep and 1 cm inferior to tragal pointer
2. Drill vertical portion in the mastoid to the stylomastoid foramen
3. Peripheral branch, retrograde dissection
4. Follow the stylomastoid suture line
5. Deep to posterior belly of digastric muscle attachment to digastric groove

Buccal: innervates procerus, medial lover orbicularis oculi, nasal muscles, upper orbicularis oris

Marginal mandibular: innervates lower orbicularis oris, lip depressors

- Motor nerve to masseter

  Subzygomatic triangle: to identify nerve for facial animation

  - Frontal branch, zygoma, TMJ
  - 3 cm anterior to tragus
  - 1 cm inferior to zygomatic arch
  - 1.5 cm deep to SMAS

- SMAS
  - Continuous with platysmal inferiorly
  - Continuous with temporoparietal fascia, then continuous with galea
  - Invests in nasolabial, peri-ocular, peri-oral musculature
- Scalp
  - Layers—*s*kin, *c*onnective tissue, *a*poneurosis (galeal), *l*oose connective tissue, *p*ericranium
  - Galeal aponeurosis invests the frontalis and occipitalis muscles
  - Temporalis fascia
  - Superficial layer
  - Deep layers—superficial and deep surround temporalis muscle

Frontal branch of VII runs within the temporoparietal fascia (superficial layer of deep temporal fascia

- Muscles of facial expression (see diagram below) (see Fig. 2.8)
  - All *innervated from deep* surface *except* "MLB"

    Mentalis

    Levator anguli oris

    Buccinator

  - Orbicularis oculi parts:

    $\left.\begin{array}{l}\text{Pretarsal}\\ \text{preseptal}\end{array}\right\}$ Palpebar

    Orbital

  - Nasal musculature

    Nasalis dilator—flares lower lateral cartilages

    Nasalis transverse—compresses the nasal wall

    Depressor septi (depresses tip)

    Levator labii superioris alaeque nasi (flares—produces "*gummy smile*"—runs in the alar crease)

    Procerus (elevates tip)

  - Ear wigglers:

    Posterior, superior, and anterior auricular muscles

- Rhytides
  - Glabella

    Vertical rhytides—*c*orrugator

    Horizontal rhytides—*p*rocerus

    Crow's feet—orbicularis oculi

  - Nasolabial and melolabial—investment of SMAS fibers to skin, orbicularis oris
- Orbital anatomy (see Table 2.9, Figs. 2.6, 2.7, 2.9, and 2.10)
  - Medial wall structures (see Table 2.9)
  - Peak of eyebrow should be at the lateral limbus
  - Tarsal show (Caucasians): 7–8 mm
  - Brow to supratarsal crease: 10–11 mm

Table 2.9 Distance to critical structures along the medial orbital wall

Medial orbital wall structures
Anterior lacrimal crest to:
Anterior ethmoid art: 24 mm
Posterior ethmoid art: +12 mm
Optic nerve: +6 mm

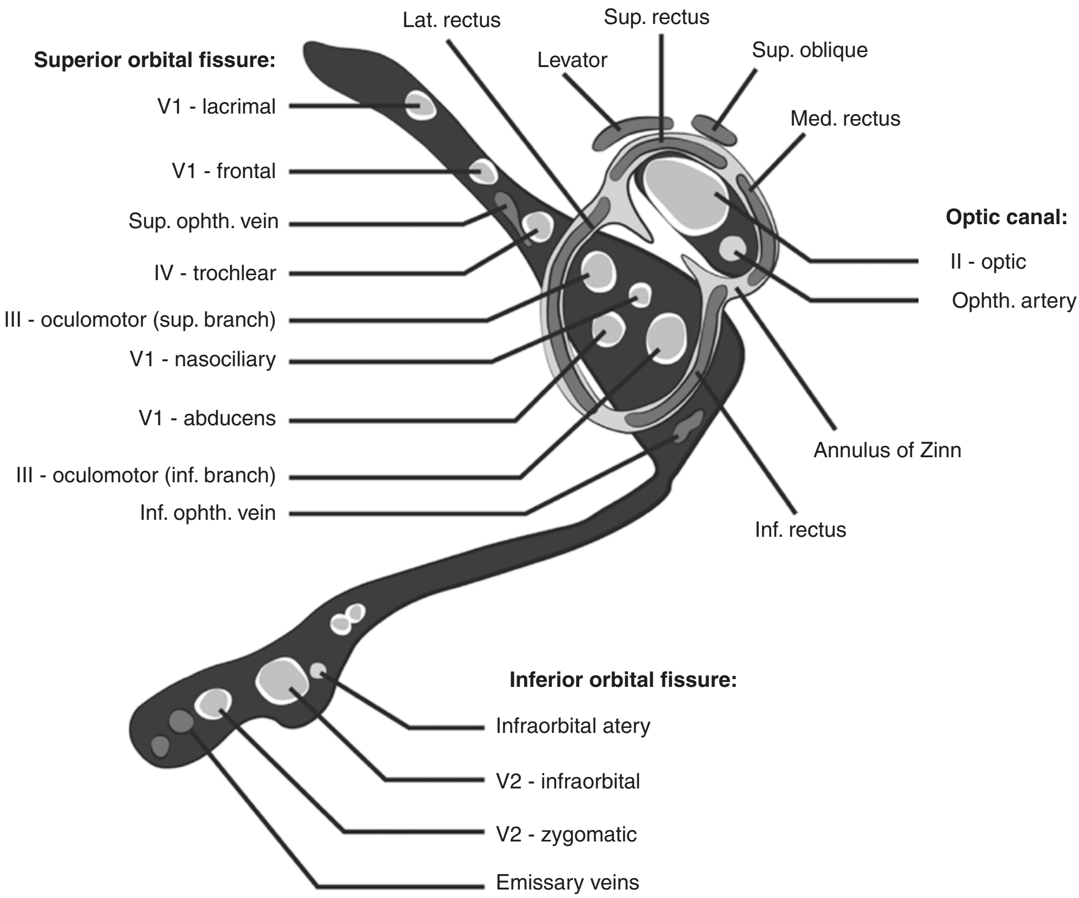


Fig. 2.6 Right orbital fissure contents

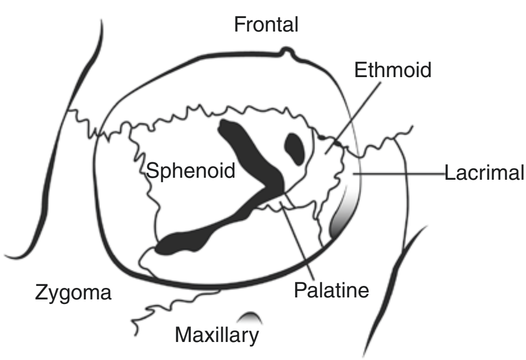


Fig. 2.7 Bone composition of the orbit (right side)

– Superior tarsal plate height: 8–9 mm
– Inferior tarsal plate height: 4–5 mm
– *Asian eyelid*: *lower (or absent) insertion of the levator aponeurosis* to the skin, septum fuses with the levator aponeurosis below superior tarsal border (above in Caucasians) → ptosis of orbital fat down to lid margin gives appearance of fuller eyelid

Anterior lacrimal crest: where medial canthal tendon attaches

Whitnall's tubercle: where Lockwood's ligament and lateral canthal tendon attach; 11 mm from ZF suture and 4 mm posterior to lateral orbital rim

1	Frontalis
2	Procerus
3	Corrugator
4a	Obicularis oculi-orbital
4b	Obicularis oculi-preseptal
4c	Obicularis oculi-pretarsal
5	Zygomaticus Major
6	Zygomaticus Minor
7	Levator labiisuperioris
8	Levator labiisuperioris alawquae nasi
9a	Transverse nasalis
9b	Alar part of nasalis
10	Compressor narium
11	Depressor septi
12	Risorius
13	Obicularis Oris
14	Depressor anguli oris
15	Depressor labii
16	Mentalis
17	Platysma
18	Levator anguli oris
19	Buccinator
20	Parotid duct
21	Masseter

A	Supratrochlear nerve (V1)
B	Supraorbital nerve (V1)
C	Zygomaticotemporal nerve (V2)
D	Zygomaticofacial nerve (V2)
E	Infraorbital nerve (V2)
F	Infratrochlear nerve (V1)
G	External nasal nerve (V1)
H	Mental nerve (V3)

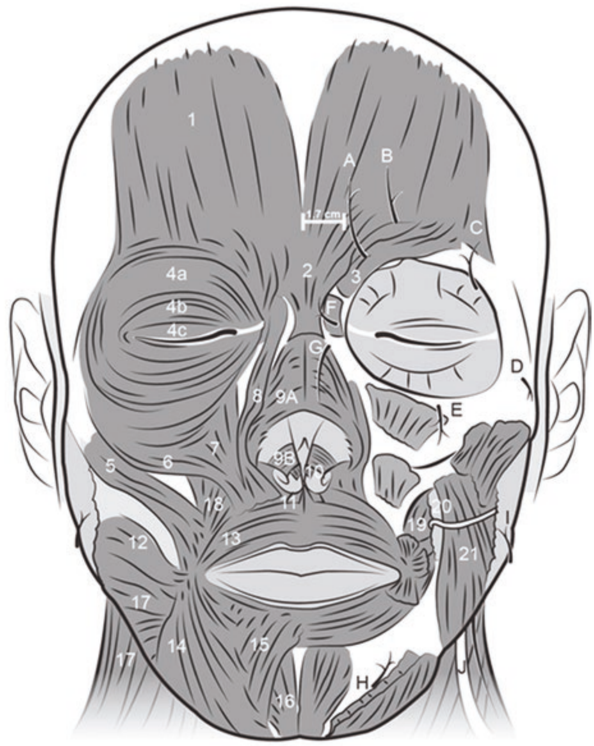


Fig. 2.8 Muscles of facial expression, facial innervation

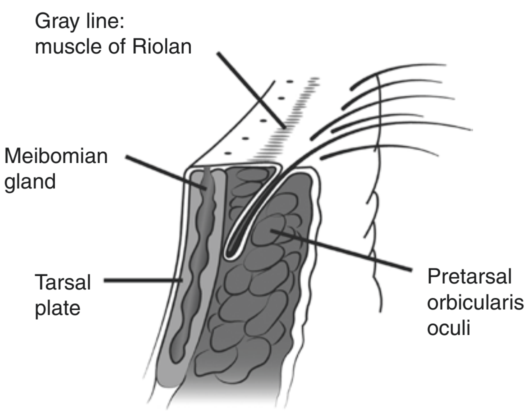


Fig. 2.9 Cross-section of lower eyelid

- Nose
  - *Nasal tip innervated by V1*
  - Vascular supply:
    External carotid
    (a) IMA → greater, lesser palatine
    (b) IMA → sphenopalatine → posterior septal and lateral nasal branches
    (c) Facial artery → superior labial, angular, lateral nasal
    Internal carotid
    (a) Anterior and posterior ethmoid, dorsal nasal
- Mouth
  - External landmarks
    Philtrum
    Modiolus
    White roll
- Wet Line
  Red color due to capillary plexus and non-keratinizing cells
  - Musculature
    Several parts to orbicularis oris
    (a) Originate from buccinators, depressor septi, mentalis
    (b) Philtrum overlies area of decussation
  - *Labial artery runs between orbicularis and mucosa* at or inferior to the level of the vermillion border (see Fig. 2.11)

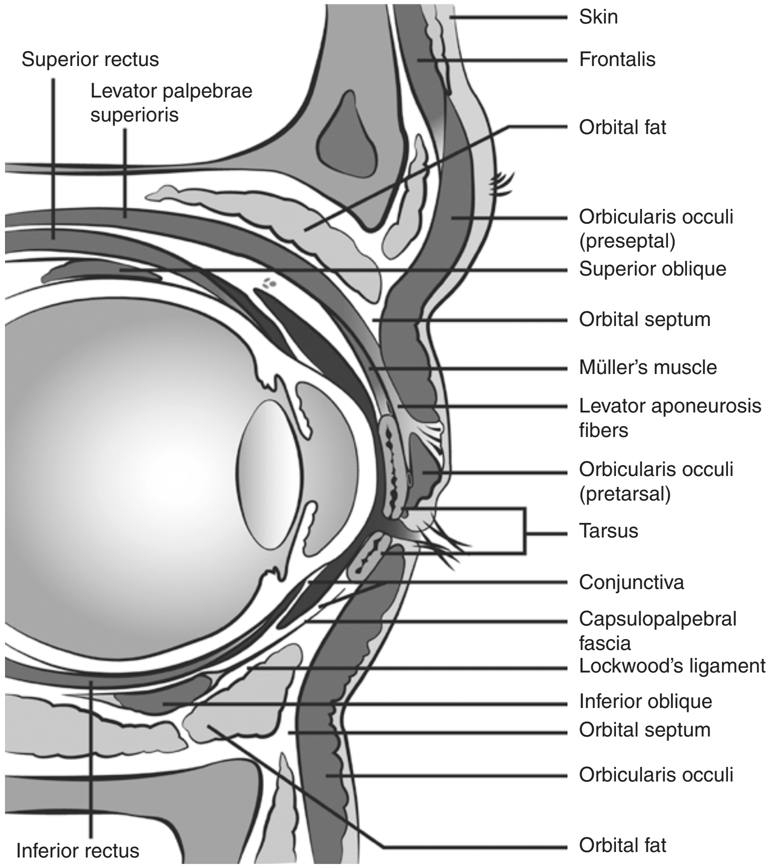


Fig. 2.10 Cross-sectional view of orbital anatomy

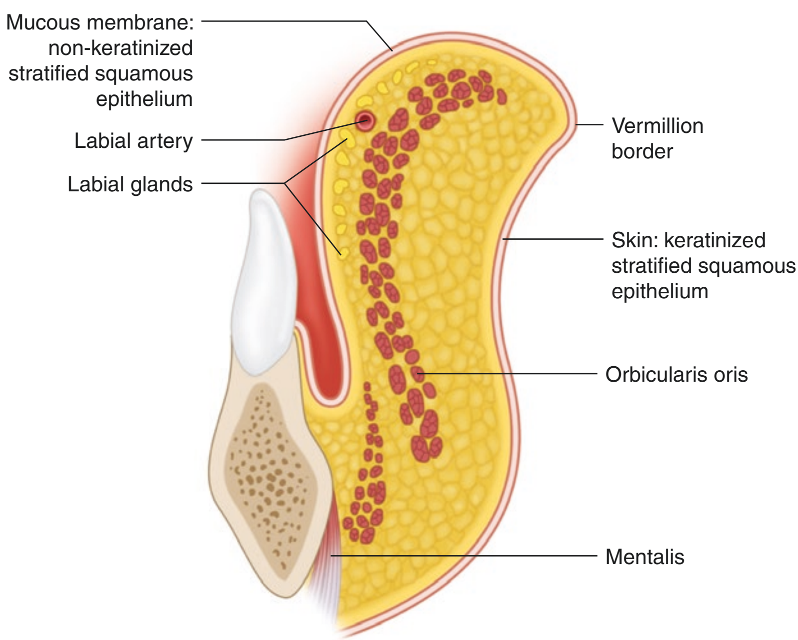


Fig. 2.11 Cross-section of lower lip

- Mandible (See Fig. 2.12)
  - Muscles and motions
    Lateral pterygoid (condyle/coronoid) *opens the jaw*
    Inserts on the lateral aspect of the lateral pterygoid plate while the medial pterygoid inserts on the medial aspect of the lateral pterygoid plate
  - Temporalis (coronoid), Masseter (angle), medial pterygoid (behind the mylohoid groove, medial angle of mandible)—*close the jaw*
- Superficial neck
  - Great auricular nerve
  - External jugular (anterior to great auricular)
- Ear (see Figs. 2.13, 2.14, and 2.15)
  - Vertical height ~6 cm; width: 3.5 cm
  - Orientation: *long axis posteriorly rotated 15°* or roughly "*parallel to nasal dorsum*" (usually less)
  - *Projects 20–30°, 2–3 cm* from mastoid
  - Blood supply from:
  - Posterior auricular
  - Superficial temporal
  - Posterior occipital
  - Deep auricular
  - Cartilage framework
    - *Hillocks of His*
    - *1–3 from first arch*

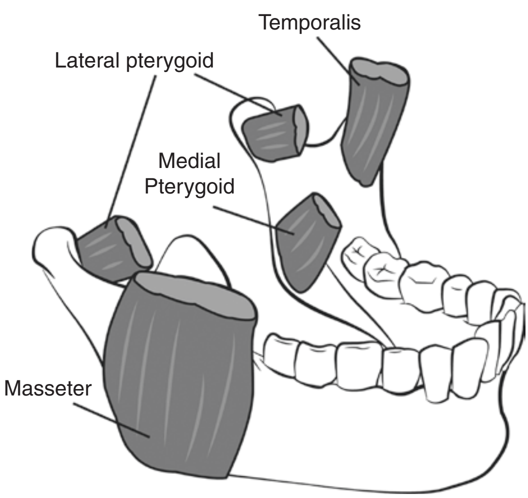


Fig. 2.12 Muscles influencing jaw opening and closing

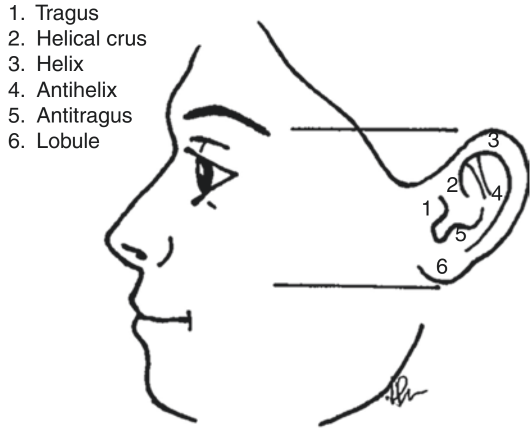


Fig. 2.13 Derivatives of Hillocks of His

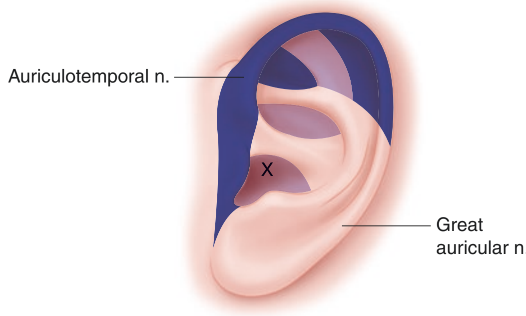


Fig. 2.14 Cutaneous innervation of the ear

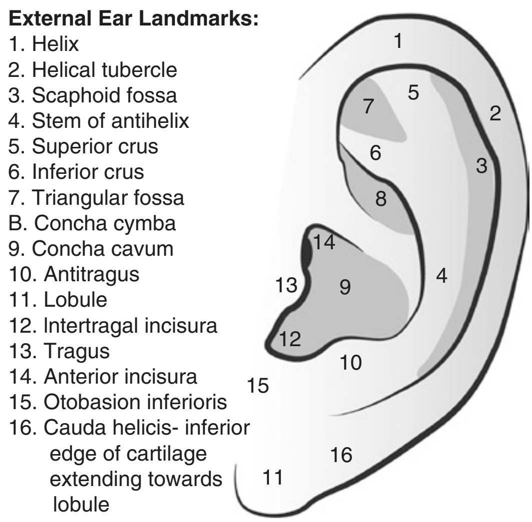


Fig. 2.15 External landmarks of the ear

  - *4–6 from second arch*
- – Cutaneous innervation (Fig. 2.14)
  Anterior: superior 1/3-Auriculotemporal (V3)
  Anterior: inferior 2/3- Great Auricular (C2/C3)
  Concha/EAC: Arnold's nerve (CN X)
  Posterior: superior 1/3 Lesser Occipital
- Arterial anatomy of the face (see Fig. 2.16 Branches of the internal maxillary artery, Table 2.10)

Venous system: connected to pterygoid plexus and cavernous sinus, valveless so allows for antero and retrograde flow

Table 2.10 Branches of the internal maxillary artery

Deep auricular
Anterior tympanic
Middle meningeal
Accessory meningeal
Inferior alveolar
Mylohoid
Deep temporal (post and ant)
Pterygoid
Masseteric
Buccinator
Posterior superior alveolar
Infraorbital
Descending palatine
Sphenopalatine
Pharyngeal

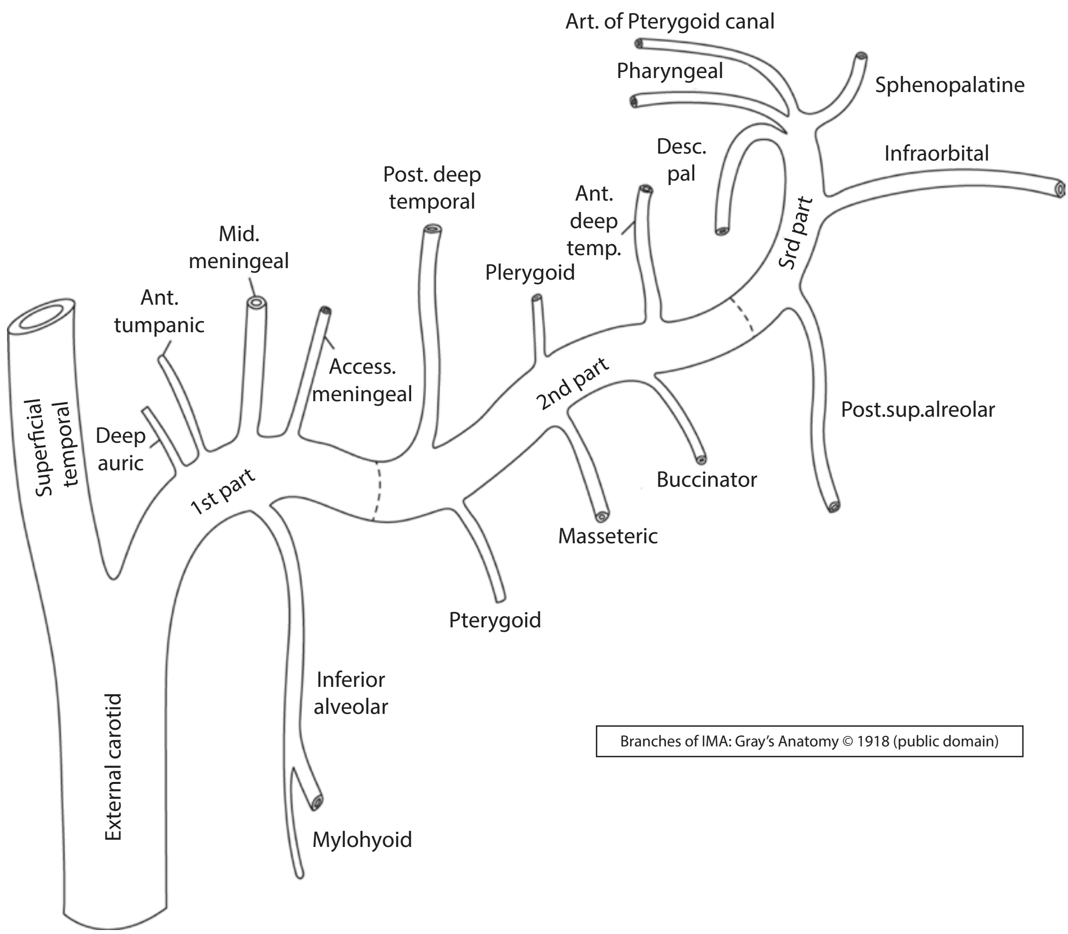


Fig. 2.16 Branches of the internal maxillary artery

- External carotid branches
  - *S*uperior Thyroid
  - *A*scending pharyngeal (posterior)
  - *L*ingual
  - *F*acial (“external maxillary” in old texts)
  - *O*ccipital (posterior)
  - *P*osterior auricular (posterior)
  - *M*axillary (“internal maxillary” in old texts)
  - *S*uperficial temporal (terminal branch)

Questions

1. What is the cellular mechanism of keloid formation?
2. How does edema inhibit wound healing?
3. How does diabetes interfere with wound healing?
4. How does nicotine affect wound healing?
5. What is the most reliable landmark to identify position of main trunk of facial nerve as it emerges from SM foramen?
6. During a facial animation case, where would you find the massetic in?
7. What are the soft tissue layers in temple and relation to temporal branch of facial nerve?
8. Position of ant/post ethmoid artery identifies level of what structure on other side of medial orbital wall?
9. What provides nasal tip innervation?
10. What are the anatomic reasons for aesthetic appearance of Asian eyelid?
11. What is the significance of Whitnall’s tubercle? What bone is Whitnall’s tubercle on?
12. Where would you expect to find the supratrochlear artery when designing PMFF?
13. What is the maximum tensile strength of a scar after 1 year?
14. Two weeks after surgery, a patient asks what vitamins could she take to help wound healing? How would they improve wound healing?
15. What is the wound classification for a dog bite? OSR?
16. Medial canthal tendon surrounds what structure?
17. What cells are found in the inflammatory phase?
18. What layer of skin is responsible for the majority of its strength?
19. What structure makes up marionette lines?
20. Name the facial muscles with superficial innervation by the facial nerve.
21. Which muscles contribute to the modiolus
22. How far does the ear project off the mastoid?
23. Name the four vertical buttresses of the face
24. What bones make up the bony orbit
25. The inferior oblique muscle is found between what two fat pads?
26. What does the gray line represent?

Answers

1. Proliferation of myofibroblasts extending past the wound boundaries
2. Decreased diffusion of O2 and growth factors get increased protein deposition which impedes healing
3. Microangiopathic interference which leads to decreased O2
4. Vasoconstriction via tx A2; CO binds to hgb thereby decreased O2 delivery
5. TM suture line
6. Subzygomatic triangle
   Frontal branch, zygoma, TMJ
   3 cm anterior to tragus
   1 cm inferior to zygomatic arch
   1.5 cm deep to SMAS
7. (Superficial) Skin, TPF/superficial layer of deep temporal fascia (contains temporal br of VII), deep layer of deep temporal fascia, temporalis muscle, periosteum
8. Cribiform plate
9. V1
10. Levator aponeurosis and orbital septum fuse below superior border of tarsal plate
    Allows orbital fat to lie anterior to tarsal plate giving fuller appearance
    Prevents levator/dermal attachment
11. Attachment of lateral canthal tendon and Lockwood’s ligament; zygomatic bone
12. 1.7–2.2 cm off midline. Supraorbital artery 1.5 cm lateral to the supratrochlear artery.
13. 80% of original skin

14. During collagen formation, the hydroxylation of proline and lysine residues depends on the presence of oxygen, vitamin C, and iron. Deficiencies of oxygen and vitamin C, in particular, result in underhydroxylated collagen which interferes with cross-linking.
15. Dog bite: class III contaminated; OSR class II: clean contaminated
16. Ant/post limbs surround lacrimal fossa and sac
17. Neutrophils within first 6 hours, macrophages by day 2–4
18. Reticular layer—has more compact collagen and more elastin.
    Papillary layer has looser tissue
19. Mandibular osteocutaneous ligaments
20. Mentalis, levator anguli oris, buccinator
21. Orbicularis oris, zygomaticus major, levator anguli oris, buccinator, risorius, depressor anguli oris, levator labii superioris all contribute to the modiolus
    NOT zygomaticus minor
22. 20–30 degrees
23. Nasomaxillary, zygomaticomaxillary, pterygomaxillary, mandible condyle/ramus
24. Frontal, zygomatic, maxillary, sphenoid, lacrimal, palatine, ethmoid
25. Medial and central
26. Pretarsal orbicularis that is behind the lash line (called the muscle of Riolan)

Additional Resources

1. Pitzer GB, Patel KG. Proper care of early wounds to optimize healing and prevent complications. Facial Plast Surg Clin North Am. 2011;19:491–504.
2. Leach J. Proper handling of soft tissue in the acute phase. Arch Facial Plast Surg. 2001;17(4):227–38. Review
3. van der Eerden PA, Lohuis PJ, Hart AA, et al. Secondary intention healing after excision of nonmelanoma skin cancer of the head and neck: statistical evaluation of prognostic values of wound characteristics and final cosmetic results. Plast Reconstr Surg. 2008;122(6):1747–55.
4. Zitelli JA. Wound healing by secondary intention: a cosmetic appraisal. J Am Acad Dermatol. 1983;9:407–15.
5. Ortega G, Rhee DS, Panpandria DJ, et al. An evaluation of surgical site infections by wound classification system using the ACS-NSQIP. Surg Res. 2012;174(1):33–8.
6. Frodel JL. Wound healing. In: Papel ID, et al., editors. Facial plastic and reconstruction surgery. St. Louis: Mosby; 1992. p. 14–21.
7. Powell N, Humphreys B. Proportions of the aesthetic face. Stratton, NY: Thieme Medical Publishers, Inc.; 1984. p. 24.
8. Baker SR, Graney DO. Chapter 18: anatomy of the face. In: Otolaryngology head and neck surgery. 1, 3rd ed. 1997.
9. Gray's anatomy (p. 457–459, 1425–1446).
10. Zide BM, Jelks GW. Surgical anatomy of the orbit. The eyelids. New York: Raven Press; 1985.
11. Toriumi DM, Mueller RA, Grosch T, Bhattacharyya TK, Larrabee WF Jr. Vascular anatomy of the nose and the external rhinoplasty approach. Clinical Trial Journal Article Arch Otolaryngol Head Neck Surg 1996;122(1):24–34.
12. Dutton JJ. Surgical anatomy of the eyelids. Atlas of ophthalmic surgery: oculoplastic, lacrimal, and orbital surgery. 1992;2(2):4–21. St. Louis: Mosby Year Book.
13. Zide BM, Jelks GW. Surgical anatomy of the orbit. Plast Reconstr Surg. 1984;74(2):301–5.
14. Frodel JL Jr. Wound healing. In: Papel ID, Nachlas NE, editors. Facial plastic and reconstructive surgery. St. Louis: Mosby Year Book; 1992. p. 14–21.
15. Schwab ME, Caroni P. Oligodendrocytes and CNS myelin are nonpermissive substrates for neurite growth and fibroblast spreading in vitro. J Neurosci. 1988;8(7):2381–93.
16. Powell N, Humphreys B. Proportions of the aesthetic face. Stratton: Thieme Medical Publishers, Inc. p. 11.
17. Toriumi DM, Larrabee WF Jr. Skin grafts and flaps. In: Papel ID, Nachlas NE, editors. Facial plastic and reconstructive surgery. St. Louis: Mosby Year Book; 1992. p. 31–44.
18. Larrabee WF Jr, Makielski KH, Cupp C. Facelift anatomy. Facial Plast Surg Clin North Am. 1993;1(2):135–53.
19. Whetzel TP, Mathes SJ. Arterial anatomy of the face: an analysis of vascular territories and perforating cutaneous vessels. Plast Reconstr Surg. 1992;89(4):591–603. discussion 604–605
20. Fairbanks DN. Pocket guide to antimicrobial therapy in otolaryngology—head and neck surgery. 13th ed. © 2007 AAO-HNS Foundation.

Evidence-Based Medicine

3

Brandyn Dunn, Giriraj K. Sharma, Jacob O. Boeckmann, and Brian J.-F. Wong

The practice of evidence-based medicine (EBM) involves the application of current, best-available clinical evidence from systematic research in health care decision-making for individual patients [1].

Five Steps of Evidence-Based Medicine [2]

1. *Asking an answerable clinical question:* A focused, structured clinical question directly relevant to a problem related to a patient or population [3].
   (a) Formulation of a well-constructed clinical question typically involves using the *PICO model* to define four key elements [4, 5].
      (i) *P* = Patient/problem.
      (ii) *I* = Intervention (a treatment, diagnostic test, or prognostic factor).
      (iii) *C* = Comparison intervention (if necessary).
      (iv) *O* = Outcomes from the intervention or test.
2. *Systematic retrieval of the best evidence available* [6]:
   (a) Generally, there are three categories of resources: background information (i.e., textbook), primary sources (i.e., primary literature), and secondary sources (i.e., guidelines, systematic reviews).
3. *Critical appraisal of evidence*: systematic evaluation of the identified resources based on the strength of the evidence. A hierarchical system of classifying evidence, known as the *levels of evidence*, ranks study design types according to the strength of the results and probability of bias [5].

Level of Evidence for Therapeutic Studies [5]:

Ia Systematic review of RCTs (with homogeneity*)
Ib Individual RCT (well-designed, narrow confidence interval)
Ic All-or-none study[†]
IIa Systematic review of cohort studies (with homogeneity*)
IIb Individual cohort study
Poorly designed RCT (e.g. <80% follow-up)
IIc “Outcomes” research, ecological studies.
IIIa Systematic review of case-control studies

B. Dunn (✉) · B. J.-F. Wong
Department of Otolaryngology Head and Neck Surgery, University of California Irvine, Orange, CA, USA
e-mail: bsdunn@uci.edu

G. K. Sharma
Otolaryngology – Head and Neck Surgery, University of California, Irvine, Orange, CA, USA

J. O. Boeckmann
Pacific Coast Facial Plastic Surgery, Laguna Niguel, CA, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_3

IIIb Individual case-control study (well-designed)
IV Individual case series
Poorly designed cohort and case-control studies
V Expert opinion, without critical appraisal (e.g., argument from physiology, bench research, or first principles)

* The homogeneity of a systematic review indicates the lack of variations in the directions and degrees of results between individual studies.

† Category Ic is met when all patients died before the treatment became available, but now some survive because of it. Or, when some patients died before the treatment became available, but now none die who receive it.

Clinical recommendations are developed based on the strength of supporting evidence. (Fig. 3.1) [5, 7].

Grades of recommendation [5]:

A. Consistent level I studies
B. Consistent level II or III studies
Extrapolations from level I studies
C. Level IV studies
Extrapolations from level II or III studies
D. Level V studies
Inconsistent or inconclusive studies of any level

4. *Application of results in practice*: evidence is integrated into clinical practice
5. *Evaluation of performance*: critical analysis is applied to evaluate the usefulness of the EBM process.

Study Design

- *Systematic review:* appraisal and synthesis of primary research papers using a rigorous and clearly documented methodology in both the search strategy and the selection of studies [8]. (https://libguides.library.qut.edu.au/systematicreviews)
- *Meta-analysis:* a quantitative statistical analysis of several separate but similar experiments or studies in order to test the pooled data for statistical significance [9]. (Merriam-Webster)

Individual clinical research studies can be divided into two categories: *experimental studies* and *observational studies*. This differentiation is based on whether the investigators assigned the exposures (e.g., treatments) or whether they observed standard clinical practice. (Fig. 3.2) [10].

Experimental Study investigator manipulates the exposure, that is, he or she allocates subjects to the intervention or exposure.

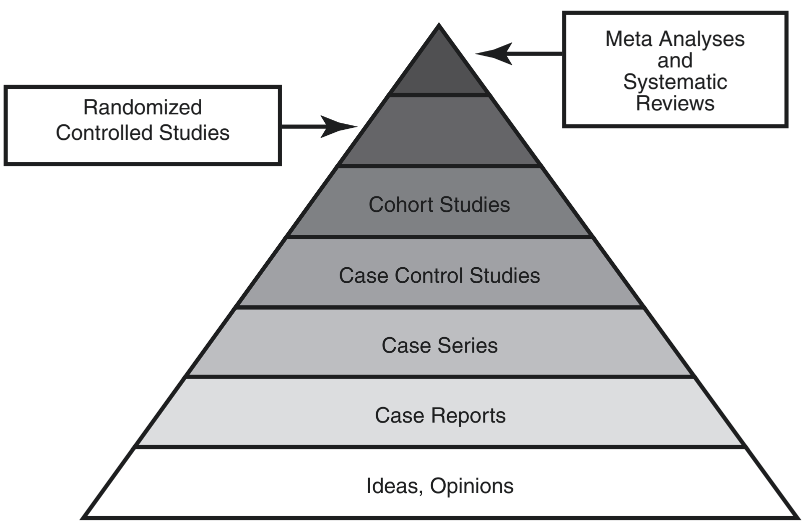


Fig. 3.1 Evidence pyramid. Each ascending level of the pyramid represents a different type of study design with corresponding increases in rigor of design, quality, and reliability of the evidence and decreases in bias from confounding variables

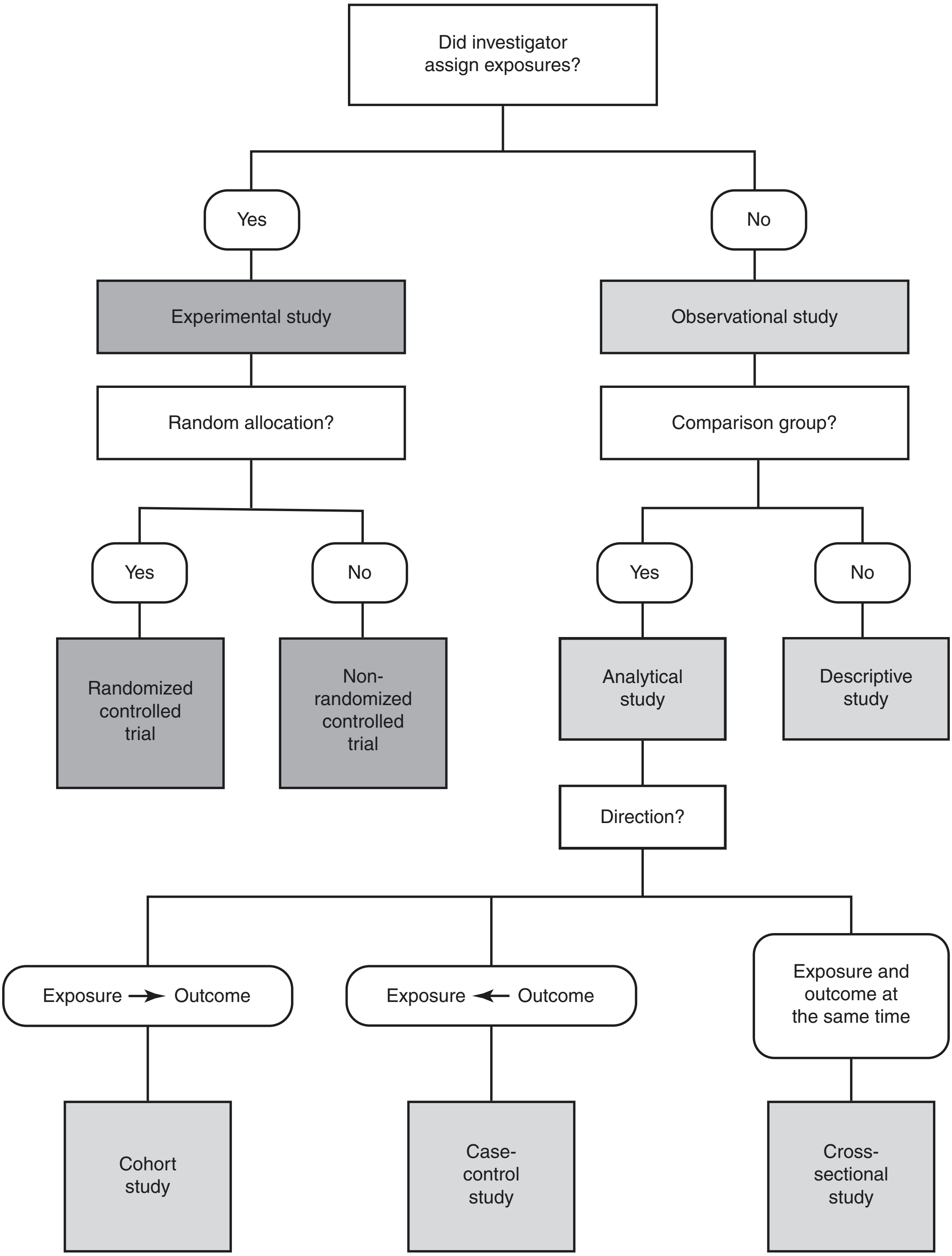


Fig. 3.2 Study design categorization

- *Randomized controlled trials:* (gold standard) subjects are randomly allocated into two or more different groups (often a control group and experimental group) and prospectively followed.
  - Advantages of RCTs over observational studies include (1) unbiased distribution of confounders, (2) easier to blind/mask, (3) randomization facilitates statistical analysis, and (4) clearly defined subject populations. RCTs typically have greater time and cost requirements, and are subject to volunteer bias.
  - *Expertise-based RCTs* randomize subjects to clinicians with expertise in intervention A or clinicians with expertise in intervention B [11]. In expertise-based RCTs, clinicians only perform the intervention, which they specialize in.

Blinded Experiment information about the experiment is masked from the researchers and study participants to reduce or eliminate bias [12].

Observational Study investigator simply measures the exposure or treatments of the desired study groups.

- *Cross-sectional study:* A descriptive study, which entails the analysis of data from a population at one specific point in time.
- *Cohort study:* An observational study where one or more samples (cohorts) who do not have a disease/outcome are followed prospectively. Correlation between initial exposure characteristics (risk factors) and disease/outcome is measured to determine an absolute risk of contraction.
- *Case-control study:* A study that compares samples with and without a disease/outcome of interest. Retrospective analysis is performed to identify factors that may contribute to development of the disease/outcome.
- *Case series:* A descriptive study that follows a group of subjects with a known exposure or who received a similar treatment.
  - Elements of a good *case series*: define study population, inclusion/exclusion criteria, intervention/co-intervention, and primary outcome. Only descriptive statistics should be used. A statement of external validity of the obtained data should be provided, including patient characteristics and completeness of follow-up [13].

Sources of Bias [14]

Bias is the difference between a population mean of the measurements or test results and an accepted reference or true value.

Precision is the statistical variation of a specific test independent of the reference or true value.

Major sources of clinical bias are highlighted below, categorized by the phase of a clinical research study:

Study Design

- *Design bias*: Researcher fails to account for the inherent biases liable in most types of experiments.
- *Selection bias*: Systematic differences between baseline characteristics of the groups being compared. Study subjects are not truly representative of the target population. Occurs when sampling is not random or if sample size is too small.
- *Channeling bias*: Patient assignment into a study cohort is dictated by prognostic factors or degree of illness. More likely to occur in nonrandomized controlled trials.

Study Implementation (Information Bias)

- *Interviewer bias*: A systematic difference in how information is solicited, recorded, or interpreted between different
- *Recall bias*: Systematic differences in the accuracy or completeness of reported information between subjects in cases and control groups.

- *Performance bias*: Systematic differences between groups in the care that is provided or in exposure to factors other than the intervention-of-interest.

Completion of the Study

- *Citation or reporting bias:* Systematic differences between reported and unreported findings. Example: researchers or trial sponsors may be unwilling to publish unfavorable results.
- *Confounding:* An observed association is due to an independent variable (exposure), a dependent variable (outcome of interest), and a third extraneous variable that correlates with both the dependent and independent variable. Pre-trial study design is the preferred method to control for confounding.

Questions

1. A study was designed to assess the impact of sun exposure on skin damage in baseball players. During a game, one team wore sunscreen while the other team did not. After the game, skin from players on both teams was analyzed for texture and burns. What type of study is this?
   (a) Case control study
   (b) Cohort study
   (c) Randomized controlled trial
   (d) Cross-sectional study
2. A study was designed to assess the effect of tamoxifen on the risk of breast cancer in women. In a blinded manner, women were allocated into two treatment groups: tamoxifen or placebo drug. All subjects were followed over a 10-year period of time and assessed for incidence of breast cancer. What type of study is this?
   (a) Case control study
   (b) Cohort study
   (c) Randomized controlled trial
   (d) Cross-sectional study
3. A systematic review of cohort studies classifies as what level of evidence?
   (a) Ia
   (b) Ib
   (c) IIa
   (d) IIb
   (e) III
4. A single, well-designed case series classifies as what level of evidence?
   (a) IIa
   (b) IIb
   (c) III
   (d) IV
   (e) V
5. What grade of recommendation is assigned to information extrapolated from a level I therapeutic study?
   (a) A
   (b) B
   (c) C
   (d) D
6. A report that systematically combines data from selected studies and computes a weighted statistical parameter to estimate an overall, combined effect:
   (a) Randomized controlled trial
   (b) Systematic review
   (c) Observational study
   (d) Meta-analysis
7. In a study of risk factors for breast cancer, women with breast cancer reported higher usage of oral contraceptives than women without breast cancer. Such a finding may be compromised by:
   (a) Reporting bias
   (b) Confounding
   (c) Recall bias
   (d) Selection bias
8. During systematic retrieval of the best-available evidence, background sources are best used to:
   (a) Find systematic reviews
   (b) Find randomized controlled trials
   (c) Find general information about a disease/treatment
   (d) Find expert opinion
9. What is the correct order of evidence, from lowest to highest, according to the evidence pyramid?
   (a) Case series, cohort studies, case-control studies, RCT, meta-analyses
   (b) Case-control studies, case series, cohort studies, meta-analyses, RCT

(c) Case series, case-control studies, cohort studies, meta-analyses, RCT
(d) Case series, case-control studies, cohort studies, RCT, meta-analyses

Answers

1. (b)
2. (c)
3. (c)
4. (d)
5. (b)
6. (d)
7. (c)
8. (c)
9. (d)

References

1. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS. Evidence based medicine: what it is and what it isn't. BMJ. 1996;312(7023):71–2.
2. Evidence-Based Medicine Working Group. Evidence-based medicine. A new approach to teaching the practice of medicine. JAMA. 1992;268(17): 2420–5.
3. Burns PB, Chung KC. Developing good clinical questions and finding the best evidence to answer those questions. Plast Reconstr Surg. 2010;126(2): 613–8.
4. Richardson WS, Wilson MC, Nishikawa J, Hayward RS. The well-built clinical question: a key to evidence-based decisions. ACP J Club. 1995;123(3):A12–3.
5. Center for Evidence Based Medicine. http://www.cebm.net. Accessed 28 Jan 2015.
6. McCarthy CM, Collins ED, Pusic AL. Where do we find the best evidence? Plast Reconstr Surg. 2008;122(6):1942–7.
7. American Society of Plastic Surgeons. ASPS Scale for Grading Recommendations. http://www.plasticsurgery.org/Documents/medical-professionals/health-policy/evidence-practice/ASPS-Scale-for-Grading-Recommendations.pdf. Accessed 4 Feb 2015.
8. QUT Library Guide: Systematic Reviews. https://libguides.library.qut.edu.au/systematicreviews. Accessed 14 Dec 2018.
9. Meta-analysis. 2018. In Merriam-Webster.com. Retrived December 14, 2018, from https://www.merriam-webster.com/dictionary/meta-analysis.
10. Hennekens CHBJ. Epidemiology in medicine. Boston: Little, Brown and Company; 1987.
11. Devereaux PJ, Bhandari M, Clarke M, et al. Need for expertise based randomised controlled trials. BMJ. 2005;330(7482):88.
12. Boutron I, Estellat C, Guittet L, et al. Methods of blinding in reports of randomized controlled trials assessing pharmacologic treatments: a systematic review. PLoS Med. 2006;3(10):e425.
13. Kooistra B, Dijkman B, Einhorn TA, Bhandari M. How to design a good case series. J Bone Joint Surg Am. 2009;91 Suppl 3:21–6.
14. Pannucci CJ, Wilkins EG. Identifying and avoiding bias in research. Plast Reconstr Surg. 2010;126(2):619–25.

4 General Head and Neck

Taylor Robinson Pollei and Nathan Johnson

Gross Anatomy

Arterial Divided into three arterial plexuses: deep facial, subdermal, and subcutaneous plexus, all connected by small musculocutaneous perforating arteries.

- *Lateral face*—(lateral to the nasolabial region): *External carotid* → perforators of the transverse facial, submental, and posterior auricular arteries.
- *Anterior face*—Superiorly: *Internal carotid* → ophthalmic (eyes, upper 2/3 of nose, central forehead). Inferiorly: *External carotid* → facial → inferior/superior labial and angular.

Venous Mostly coursing parallel and opposite to arterial flow. Connections to pterygoid plexus/cavernous sinus present with valveless, bidirectional flow distally (e.g., angular vein) = "danger triangle." Supratrochlear/supraorbital → angular (joins superior labial) → facial → common facial → *internal/external jugular veins* ← superficial temporal.

T. R. Pollei
Facial Plastic & Reconstructive Surgery, Williams Plastic Surgery Specialists, Latham, NY, USA

N. Johnson (✉)
Ear Nose and Throat Specialty Care, Minneapolis, MN, USA
e-mail: njohnson@entsc.com

Innervation

Facial Nerve

- The facial nerve is composed of approximately 10,000 neurons, 7000 of which are myelinated and innervate the muscles of facial expression.
- The remaining 3000 fibers are somatosensory and secretomotor, and are known as the Nervus Intermedius.
- Extratemporal → posterior digastric and stylohyoid muscle branches → enters parotid gland.
  - Travels deep to SMAS, typically entering muscles from deep side. The muscles not innervated on their deep side include the levator anguli oris, mentalis, and buccinator.
- *Temporal br.* → frontalis and upper orbicularis oculi muscle (OOM). Runs underneath the temporoparietal fascia over the zygomatic arch and divides into 2–4 branches.
- *Zygomatic br.* → lateral aspect of lower OOM, enters the muscle from deep.
- *Buccal br.* → procerus, medial lower OOM, nasal muscles, cheek, upper orbicularis oris.
- *Marginal mandibular br.* → lower orbicularis oris, lip depressors. Relationship to mandible angle is dependent on degree of neck flexion/extension and age.
- Nervus Intermedius

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_4

  - Superior salivary nucleus (pons) → nervus intermedius → geniculate ganglion. The nerve diverges in the geniculate ganglion
    Greater superficial petrosal nerve (GSPN) and chorda tympani.
  - GSPN synapses in the sphenopalatine ganglion, the post-ganglionic fibers travel via the zygomaticotemporal nerve (V2)to lacrimal gland and nasal mucosa
  - The chorda tympani joins the mandibular nerve (V3) to form the lingual nerve, synapses in the submandibular ganglion, and then provides parasympathetic innervation to the sublingual and submandibular gland.
- The parotid gland receives parasympathetic innervation via the glossopharyngeal nerve
  - Inferior salivatory nucleus → glossopharyngeal nerve → Jacobson's nerve → lesser petrosal nerve → otic ganglion → *postganglionic fibers* → Auriculotemporal nerve → parotid gland
- Damage to the facial nerve in the middle ear due to trauma or iatrogenic injury may result in misdirected parasympathetic nerve fibers reaching the lacrimal gland as well as the salivary glands. This causes profuse lacrimation with eating, also known as Bogorad's syndrome.
- Damage to the auriculotemporal nerve (parotidectomy) can result in "gustatory sweating," also known as Frey's Syndrome. The post-ganglionic parasympathetic secreto-motor fibers destined for the parotid become misdirected to join the sympathetic fibers that normally innervate sweat glands and vasculature of the skin of the cheek and scalp. This aberrant innervation results in erythema and sweating of the skin around the ear, cheek, and neck in response to gustatory stimuli.

Sensory and Autonomic

- *CN V1* (ophthalmic) → lacrimal, frontal (supraorbital, supratrochlear), nasociliary nerves
- *CN V2* (maxillary) → zygomaticotemporal, zygomaticofacial, infraorbital nerves
- *CN V3* (mandibular) → auriculotemporal, buccal, lingual, inferior alveolar/mental nerves
- Sympathetic—postganglionic = from superior cervical ganglion → vasoconstriction
- Parasympathetic—parotid = inferior salivary nucleus, submandibular and sublingual = superior salivary nucleus.

Lymphatics

Divided into 3–5 lymph node (LN) "vessels" running laterally from midline

- *Above the brow* → preauricular and deep parotid LNs
- *Lateral eyelids* → parotid LNs
- *Medial canthus* → buccinator, parotid and submandibular LNs (follows along with the angular vein)
- *External nose and cheeks* → submandibular LNs
- *Lateral lower lip and upper lip* → *ipsilateral* submandibular LNs (and some parotid drainage)
- *Central lower lip and chin* → *bilateral* submental LNs

Tissue Layers/Fascial Compartments

Scalp Layers Skin → SubQ → galea aponeurotica → loose areolar tissue → pericranium

Temporal Region Layers Skin → SubQ → superficial temporal fascia (aka temporoparietal fascia, includes superficial temporal a. & v. and joins with the galea superiorly) → loose areolar tissue → superficial layer of the deep temporal fascia → superficial fat pad → deep layer of the deep temporal fascia → deep fat pad → temporalis muscle → pericranium

Face and Neck

Superficial cervical fascia contiguous superiorly with the *SMAS*; travels from the clavicle up to the zygoma and becomes superiorly the galea. Envelopes mimetic muscles.

Deep cervical fascia

1. Superficial layer (investing): envelops salivary glands, masseter, and SCM
2. Middle layer (pretracheal): muscular division = straps, visceral division = pharynx, larynx, trachea, thyroid, buccopharyngeal fascia
3. Deep layer (prevertebral): cervical vertebrae, paraspinal musculature

Carotid sheath consists of all three layers, jugular vein, carotid artery, CN X.

Structure

Buttresses

- *Horizontal*: frontal, zygomatic, maxillary, mandibular
- *Vertical*: nasomaxillary, zygomaticomaxillary, pterygomaxillary, mandible condyle/ramus

Orbit bones (clockwise) frontal, ethmoid, lacrimal, palatine, maxillary, sphenoid, zygomatic

Musculature

- CN 3 – superior, medial and inferior rectus, inferior oblique, levator palpebrae superioris
- CN 4 (trochlear) – superior oblique
- CN 6 (abducens) – Lateral rectus
- CN 5 – *Masticatory muscles*: temporalis, masseter, medial and lateral pterygoid
- CN 7 – *Facial animation muscles*:
- *Face: Upper 1/3* – frontalis, corrugator supercilii, orbicularis oculi, procerus, depressor supercilii
- *Face: Middle 1/3* – orbicularis oris, buccinator, zygomaticus major/minor, levator labii, levator anguli oris, risorius, nasalis, depressor septi
- *Face: Lower 1/3* – depressor anguli oris, depressor labii inferioris, mentalis, platysma

Specialized Structures

Eye – Conjunctiva → cornea (sclera posteriorly) → Anterior chamber (aqueous humor) → iris → ciliary muscle, suspensory ligaments and lens → vitreous chamber (and body/fluid)

Ethnic Anatomic Considerations

Eyelid morphology—absence of an upper eyelid crease (e.g., Asian eyelid) is a result of:

1. Fusion of the orbital septum to the levator aponeurosis below the superior tarsal border
2. Protrusion of the preaponeurotic fat pad and a thick subQ fat layer at the tarsal border
3. Insertion of the levator into the orbicularis muscle and lid skin is closer to the lid margin

Nasal Morphology

- Platyrrhine (broad and flat) = thick skin, low radix, short dorsum, bulbous tip, nostril flaring
- Mesorrhine = low radix, variable dorsal projection, rounded and underprojected tip
- Leptorrhine (tall and thin) = thin skin, long and high dorsum, projected tip, narrow ala

Micro-anatomy/Pathology of Head and Neck Structures

Skin

- *Epidermis*—stratum corneum → lucidum → granulosum → spinosum → basale (basal layer) = location of melanocytes, Merkel cells
- *Dermis*—90% of skin thickness. Papillary layer (vascular network) → reticular layer (structure, elasticity) contains hair follicles, sweat (apocrine/eccrine) glands, sebaceous glands, nerve endings, collagen/elastin. Basement membrane = type IV collagen

Thyroid Central follicle interstitial space (colloid) surrounded by simple cuboidal epithelium as the secretory cells. Intermixed with C-cells (parafollicular cells) that secrete calcitonin

Parathyroid Embedded in the thyroid capsule, contains (1) chief (principle) cells, which are small and oxyphilic and secrete PTH and (2) oxyphil cells—have no secretory function

Salivary Based on the secretory acinus, which is comprised of either mucous- or serous-secreting cells → intercalated ducts (simple cuboidal epithelium that is myoepithelial cell lined) → striated ducts (site of active water resorption and ion exchange) → interlobular ducts. Sublingual = ↑ mucin; parotid = ↑ serous; submandibular = mixed

Lymphoid Tonsils are lined by squamous cells and contain lymphoid follicles below the surface containing germinal centers (similar to lymph nodes). Partially encapsulated with deep crypts present

Embryologic Development

Branchial Apparatus

1. Mandibular arch: *Meckel's cartilage*—malleus head and neck, incus body and short process, anterior malleal ligament, mandible
   - *Nerve*: CN V3 → masticatory muscles, tensor tympani, tensor veli palatini, mylohyoid, anterior digastric muscle
   - *Artery*: Maxillary artery
   - Hillocks of His (6). First three hillocks derived from the first branchial arch:
   - 1 (tragus), 2 (helical crus), 3 (helix)
   - Pouch: Eustachian tube, middle ear/mastoid, inner layer of tympanic membrane
2. Hyoid arch: *Reichert's cartilage*—manubrium of malleus, long and lenticular process of incus, stapes (not footplate) styloid process, stylohyoid ligament, lesser cornu and superior ½ of hyoid
   - *Nerve*: CN VII → facial animation, stapedius, stylohyoid, posterior digastric
   - *Artery*: Stapedial artery
   - Hillocks of His (6). Hillocks 4–6 derived from the second branchial arch:
   - 4 (antihelix), 5 (antitragus), 6 (lobule)
   - Pouch: supratonsillar fossa, palatine tonsils, middle ear
   - Groove: external auditory canal, outer layer of tympanic membrane
3. *Cartilage*—greater cornu and inferior ½ of hyoid bone
   - *Nerve*: CN IX—stylopharyngeus muscle, superior and middle constrictors
   - *Artery*: common and internal carotid arteries
   - Pouch: thymus, inferior parathyroids
4. Aortic arch: *Cartilage*—thyroid and cuneiform
   - *Nerve*: Superior laryngeal nerve (cricothyroid and inferior pharyngeal constrictors
   - *Artery*: Aorta (left); subclavian artery (right)
   - Pouch: Parafollicular cells of thyroid, superior parathyroids
5. and 6. *Cartilage*: Cricoid, arytenoid, and corniculate
   - *Nerve*: Recurrent laryngeal nerve (remaining intrinsic laryngeal muscles)
   - *Artery*: Ductus arteriosus and pulmonary artery

Physiology

Mastication/Deglutition *Oral Phase*—Mastication prepares food to become bolus with salivary mixture. Next, tongue compresses bolus against the palate and posterior pharynx with tongue elevation. Hyoid elevates and bolus is propelled toward the vallecula.

Pharyngeal Phase—Reflexive (CN IX and X) activated elevation of the larynx/hyoid bone, elevation of the soft palate, contraction of the superior constrictor, and pushing of the bolus posteriorly by the tongue base. Next, the multiple levels of laryngeal closure occur: epiglottis, AE folds, and arytenoids, false cords, and true cords. Lastly, the pharyngeal constrictors contract and the cricopharyngeus opens, triggering the *esophageal phase*.

Speech (Articulation) Speech sounds produced by a coordination of lip, tongue, and mandibular musculature activity.

Phonation The production of voice, dependent on vocal properties: position, vibratory capacity, length, and tension.

Lacrimation Spontaneous, reflexive, or emotion-based tear production.

Nerve Pathway Superior salivary nucleus → parasympathetic fibers of the GSPN → IAC → middle cranial fossa → joins sympathetic greater petrosal nerve → pterygopalatine ganglion → lacrimal gland (and nasal mucosal glands)

Lacrimal System Lacrimal gland (aqueous component) tear secretion is spread across the surface of the eye. Tarsal glands contribute the lipid component and conjunctival glands secrete mucous component. Lacrimal fluid is drawn into the punctum by capillary action → lacrimal canaliculi → lacrimal sac → nasolacrimal duct → inferior meatus

Testing

Imaging

CT scan Ideal for osseous visualization. Temporal bone = 0.5–0.625 mm thick slices. Max-face = 1.25 mm slices; neck = 3 mm slices. Axial images with coronal and sagittal recons. Volume rendering with 3-D reconstruction models. Contrast for delineating soft-tissue abscesses and cellulitis.

MRI Ideal for soft-tissue (skull base, neural tissue, cartilage) visualization with multiplanar images, even without contrast. Gadolinium contrast—risk of nephrogenic systemic fibrosis. Cons = motion artifact, pulsation artifact, metallic object interaction, greater cost, longer scan time. Most use a 1.5–3 T field strength.

Ultrasound Good soft-tissue and fluid resolution, noninvasive, inexpensive, often requires "specialized training" for interpretation. Combined with Doppler for flow testing.

Cineradiography Functional fluoroscopy, useful to delineate anatomic abnormalities in addition to functional (mastication, deglutition, esophageal) abnormalities.

Bone scan (bone scintigraphy) Nuclear scan utilizing T-99 m, for functional testing of bone lesions by highlighting areas of increased metabolic activity. Less expensive than FDG-PET.

DEXA (Aka—bone density scan) low-dose X-ray for bone density testing, structural, inexpensive.

Angiography The gold standard for evaluating vascular injury and vascular anatomy, but lengthy, expensive, and invasive.

CT angiography Screening tool for penetrating neck trauma or blunt trauma. Quick and noninvasive, easily accessible. Requires iodinated contrast.

Radiogram

FDG-PET Best technique for metabolic function testing. Poor spatial resolution, which can be enhanced with hybrid scanning techniques (with MRI or CT).

Functional

Rhinomanometry Measures transnasal airflow from the nares to nasopharynx, allowing for pressure changes and therefore resistance to be elucidated. Assesses turbulence vs. laminar flow. Clinically applicable in assessing degree of nasal obstruction. Use of decongestant spray attempts to separate structural/anatomic from functional/mucosal causes of obstruction.

Schirmer test Schirmer test I is performed placing a strip of filter paper inside lower lid (± topical anesthetic) and checked at 5 min. <10 mm of moisture on filter paper strip after 5 min is abnormal. Studies have shown the use of topical anesthesia to be more reliable in the diagnosis of dry eye than without anesthesia. The Schirmer test II is performed by irritating the nasal mucosa with a cotton-tipped applicator prior to measuring tear production, which is mainly used for measuring the reflex tear secretion of the lacrimal gland.

Assessment and Management

Differential Diagnosis Formation for Head and Neck Masses/Disorders

KITTENS Method …

K = Congenital, I = Infectious and Iatrogenic, T = Toxins and Trauma, E = Endocrine, N = Neoplastic, S = Systemic

Also, remember important info: Age, associated symptoms, symptom/sign characteristics

Management Algorithm

Management options can be arranged from most conservative and least invasive to most aggressive and high risk. Stepwise approach vs. combined approach may be useful.

1. Do nothing
2. Lifestyle: diet, exercise, sleep, habit changes
3. Therapy: physical/occupational therapy, stretching, massage, heat, ice
4. External applications: dressings, external splints
5. Medication: topical vs. systemic, taken orally, IM, IV, etc.
6. Surgery: prophylactic, diagnostic, and/or therapeutic
7. Ancillary treatments: chemotherapy, radiation therapy, cryotherapy, etc.
8. Complementary and alternative treatments

Photography

Standardized pre-, intra-, and postoperative photography help achieve consistency, facilitate comparisons, and demonstrate anatomic detail. Keys include:

- *Photographic Consent*—Use of photos for nontreatment purposes (presentations, lectures, print/Internet media, etc.) requires consent. Consent not required for treatment purposes.
- *Room setup*—appropriate flashes, background, camera mount
- *Patient preparation*—remove glasses, jewelry, place hair back, avoid excessive makeup
- Positioning—*Frankfort horizontal plane* = top of tragus (superior EAC) to the infraorbital rim (estimated by junction of lower lid and cheek skin). In some cases to avoid submental laxity, the *natural horizontal facial line* may be preferred. Oblique facial views should align either (1) nasal tip with far cheek, (2) nasal dorsum overlying the far medial eye, or (3) medial canthus aligned with the lateral oral commissure. Lateral views should be void of over- or under-rotation. Avoid head tilting by keeping ear lobes symmetric. Include from the top of the hairline to the sternal notch.
- *Views/Series*—Standard procedures have standard views. *Uniformity is key*. Utilize perspective/reference photos as needed. Typically with a 105 mm lens at 1 m distance
- *Camera Terminology*—*Aperture* = the size of the iris of the lens, determines the amount of light hitting the camera film/sensor. Measured in an "*f-stop,*" which is a fraction, and therefore increases as the aperture decreases. Smaller aperture = greater field depth.
  – *Shutter speed* = How long the iris is open and therefore how long the sensor is exposed to light.
  – *Depth of field* = (aka—focal range) The distance between the closest and farthest in-focus areas of a photograph. The smaller the aperture (↑ *f-stop*) = the greater the depth of field.
  – *Focal length* = the distance in millimeters from the optical center of the lens to the focal point, which is located on the sensor. Shorter focal length = larger field of view. Longer focal length = narrower field of view.
  – *Lenses*: Normal—When the focal length approaches the diagonal measurement (43.27 mm) of a rectangular 35 mm × 24 mm pane, the standard size that was previously

film and is now a sensor. Wide angle—shorter focal length and shorter lens but a wider field of view. Telephoto—longer focal length and a longer lens, leaving a narrower field of view. Ideal lens for facial plastic surgery is between 90 and 105 mm – designed for near focusing and produce the most realistic facial proportions.

– *Resolution*: A measurement of the pixel count of an image, given per inch or total. Increase resolution for print media (>300 ppi) and decreases for Internet use (72–150 ppi).
– *Zoom*: Optical zoom—changes the amount of the scene hitting the sensor, thus allowing for more detail that can be enlarged without issue. Digital zoom—interpolates data in the scene to fit on the sensor, mimicking zoom without gaining detail. Can result in blurry, pixelated images.
– *Lighting*: Camera-mounted flash is limited, studio lighting preferable for photodocumentation. Quarter light system most common – two lights placed at 45 degrees from the patient to produce consistent and reproducible light with minimal shadow effect. Patient is placed 12–18 inches in front of backdrop to minimize background shadow

Facial Analysis

Facial Subunits (Table 4.1)

Forehead Extends from the hairline (or the superior extent of the frontalis in receding hairlines) down to the superior orbital rim. Contains a continuation of the scalp layers, with the frontalis contained between divided galeal layers.

Periorbital region Includes the upper and lower lids, medial and lateral canthal regions, and the globe

Cheek Extends from the preauricular crease anterior to the nasolabial fold; from the zygomatic arch/inferior orbital rim down to the inferior border of the mandible.

Table 4.1 Soft-tissue landmarks

Trichion (Tn)	Forehead/hairline junction at midline (~ upper edge of frontalis)
Glabella (G)	Prominent forehead/brow junction at midline
Radix (R)/ Nasion (N)	Root of the nose, corresponding with soft-tissue nasion
Rhinion (Rh)	Junction of the nasal bone and upper lateral cartilage
Supratip (Su)	Gentle soft-tissue break between nasal dorsum and tip
Nasal tip (Tp)	Leading edge of nasal profile
Subnasale (Sn)	Soft-tissue point at the junction of the columella and upper lip
Vermillion (Vm)	Mucocutaneous junction of the upper lip and lower lip
Stomion (St)	Midpoint of the embrasure of lips when closed
Mentolabial sulcus (Ms)	Point of greatest depth above chin
Pogonion (Pg)	Most prominent soft-tissue point of chin
Menton (M)	Soft-tissue point at the inferior-most border of the chin at midline

Nose 9 subunits: dorsum, paired sidewalls, tip, paired ala, paired soft tissue triangles, and the columella

Perioral region and chin From the subnasale and nasolabial folds to the menton, between lateral commissure bilaterally.

Neck Key area of rejuvenation is the cervicomental angle, with superior neck skin behaving more like cheek skin, and lower neck skin more like chest skin (Table 4.2).

Skin Tension Lines

Relaxed Skin Tension Lines Direction of greatest elasticity, traveling *perpendicular to the facial musculature*. The exception is on the eyelids where the rigid tarsal plate overrides the orbicularis oculi pull. They tend to correspond with the direction in which rhytides form. The long axis of incisions and their scars need to line up with the RSTLs to close with minimal tension and avoid scar widening (Tables 4.3 and 4.4).

Table 4.2 Hairline evaluation

Classification	Description
Type I	Minimal or no recession of the hairline
Type II	Areas of recession at the frontotemporal hair line
Type III	Deep symmetrical recession at the temples that are bare or only sparsely covered. Anterior hairline at midline is receding
Type IV	Hair loss is primarily from the vertex, continued recession of the frontotemporal hairline
Type V	Vertex hair loss region is separated from the frontotemporal region but is less distinct; the band of hair across the crown is narrow
Type VI	Frontotemporal and vertex bald regions are joined together
Type VII	Most severe form, a narrow band of hair remaining in a horseshoe shape

Hamilton-Norwood Classification. Based on anterior and vertex degree of recession

Table 4.3 Skin analysis: Fitzpatrick's classification of skin types

Type	Skin color and features	Tanning ability
I	White skin, blue eyes, blond/red hair	Always burns, does not tan
II	White skin, blue eye	Easily burns, tans poorly
III	Darker white skin	Mild burn, average tan
IV	Brown skin	Occasionally burns, tans easily
V	Dark brown skin	Rarely burns, tans very easily
VI	Black skin	No burns, dark tan

Table 4.4 Glogau photoaging classification

Type	Severity	Typical age range	Characteristics
I	Mild	Late 20s to 30s	Little wrinkling, no keratosis, requires little or no makeup
II	Moderate	30s to 40s	Early wrinkling with facial motion, early actinic keratosis
III	Advanced	50s or older	Persistent wrinkling, discoloration with telangiectasias, visible actinic keratosis
IV	Severe	60s to 70s	Generalized wrinkling, actinic keratosis with or without malignancy

Lines of Maximal Extensibility Tend to run in the direction of the mimetic musculature.

Langer Lines Typically form in the direction of rhytides. Can be perpendicular to RSTLs and are based more on the direction of skin pull and the resulting rhytides.

Overall, scars are least conspicuous when placed in creases, and creases tend to occur perpendicular to muscle action.

Facial Divisions

Vertical 5ths Based on the width of one eye, which should equal 1/5 of the facial width. Helical rim → lateral canthus → medial canthus/nasal ala → contralateral medial canthus/nasal ala → lateral canthus → helical rim.

Horizontal 3rds Trichion → *glabella* → subnasale → menton.

Horizontal lower face In the absence of a defined trichion/hairline, the lower face can be divided with 43% from *nasion* → subnasale, and 57% from subnasale → menton.

Blepharoptosis

Anatomy Levator aponeurosis originates from the lesser wing of the sphenoid and inserts on the orbicularis oculi, dermis, and tarsal plate. CN 3 innervation providing 10–12 mm of lid elevation. Attaches to the orbital septum ~2.5 mm above the tarsal plate.

True lid ptosis = intrinsic drooping vs. pseudoptosis which is secondary to other issue giving the impression of ptosis.

Congenital Ptosis Developmental dysgenesis of the levator muscle, presents shortly after birth and is not progressive. Absent eyelid crease.

Acquired Ptosis

- *Myogenic* (typically *senile ptosis*) = the most common type. The levator attachments to the tarsus stretch and dehisce.

- Traumatic = second most common. Allow for ~6 months of recovery and healing before repair.
- Neurogenic = CN 3 palsy, Horner's syndrome, myasthenia gravis.
- Mechanical = severe upper lid dermatochalasis and excessive weight, growths, etc.

Diagnosis and Testing Degree of ptosis is measured by the amount of lid descent over the upper limbus; mild = 1–2 mm, moderate = 3 mm, severe = >4 mm. Levator function is measured by the amount of excursion with lid opening; good = >10 mm, fair = 5–10 mm, poor = 0.5 mm. Margin reflex distance one (MRD_1) measurement is required as well. See blepharoplasty chapter for specifics on MRD measurements.

Treatment Options Levator aponeurosis advancement done via external approach, external levator resection, frontalis suspension, or Mueller muscle/ conjunctival resection.

Dental Anatomy

- Pediatric dentition: 20 teeth; 4 incisors, 2 canines, 4 M per arch. Lettered A-J (R → L maxilla) and K-T (L → R mandible).
- Adult dentition: 32 teeth; 4 incisors, 2 canines, 4 premolars, 6 molars per arch. Numbered 1–16 (R → L maxilla), 17–32 (L → R mandible).

Terminology Mesial vs. distal, buccal vs. lingual. Overbite = amount of vertical incisor overlap. Overjet = amount of horizontal overlap of incisal edges. Crossbite = horizontal malalignment of teeth, either anterior or posterior. Open bite = occlusal surfaces not in contact when in centric occlusion (when condyle is in natural resting position).

Occlusion Refers to tooth relationship to one another. Angle classification:

- Class I—Mesiobuccal cusp of the maxillary first molar fits into the buccal groove of the mandibular first molar. *Class I occlusion does not = normal occlusion*.
- Class II—Mandibular molar is distally positioned … "overbite."
- Class III—Mandibular molar is mesially positioned … "underbite."

Cephalometric Evaluation

Allows for standardized measurements from lateral cephalograms to determine the relationship between the skull base, maxilla, and mandible. Evaluate dentofacial proportions and the diagnostic, anatomic basis for the deformity. Surgery planned based on aesthetic evaluation, not cephalometrics. Utilizes hard tissue/bone landmarks with the "hinge "point of the condyle and relationship to the sella.

Most common abnormalities:

1. *Maxillary excess* – vertical excess of middle third, convex profile typically a class II occlusion and lip incompetence. Treatment = LeFort I osteotomy with impaction.
2. *Maxillary deficiency* – deficiency of infraorbital/paranasal sinuses.
   Treatment = LeFort I osteotomy with expansion ± bone grafting.
3. *Mandibular excess* - (prognathism) a prominent lower 1/3 with a class III occlusion.
   Treatment = maxillary advancement ± mandibular setback.
4. *Mandibular deficiency* – (retrognathism) deficient lower 1/3 with a class II occlusion. Treatment = mandibular advancement.
5. *Microgenia* – Abnormal small size of chin, usually with a normal occlusion (cosmetic issue without a functional problem). Treatment = alloplastic implants

Basic Surgical Principles

General Principles

Alimentation/Nutrition

Malnutrition Complete H&P may show >12% weight loss, alcohol abuse, advanced-stage H&N

cancer, fat/muscle wasting, vitamin deficiency stigmata. Labs = albumin <3.0 g/dL: 1–2 months window. Transferrin <150 mg/dL—7 day half-life. Pre-albumin = t ½ of 3–5 days. Average adult requires 30–35 kcal/kg/day. Protein supplementation vital.

Nutrition delivery Oral supplementation ideal if possible.

Enteral feeds: NG tube best option, temporary (2–4 weeks), but risk of obstruction, nasal/sinus inflammation, esophagitis. Gastrostomy tube better tolerated long term. May be placed either percutaneously vs. open.

Parenteral nutrition: Rapid, not GI tract dependent. TPN must be given via central line. Indications for TPN = severe protein malnutrition, defunct GI tract, refractory chyle leak.

Wound Healing

Three phases of wound healing:

1. *Inflammatory Phase* (days 1–6)

   Initial *vasoconstriction* for 5–10 min followed by *coagulation* via platelet aggregation and fibrin accumulation. Histamine, serotonin, and NO-mediated vasodilation and increased vascular permeability allow for immune cells signaled by platelet products, complement, etc., to enter tissues. Predominant cell types = neutrophils (24–48 h) for early phagocytosis and inflammatory product production. *Macrophages* (48–96 h) for growth factor secretion and continued cleanup. Predominant cell up to fibroblasts, *Most critical to wound healing*. Lymphocytes' (5–7 days) questionable role, possibly for remodeling.
2. *Fibroproliferative Phase* (day 4–week 3)

   Fibroblasts move into the wound on day 3, and are the dominant cell at day 7. Collagen synthesis peaks from day 5 to week 3. Initial HA, dermatan, and chondroitin production are followed by collagen. Tensile strength increases from day 5 and on. Angiogenesis occurs with early VEGF production. Epithelialization begins.
3. *Maturation/Remodeling Phase* (week 3–1 year)

   After 3–5 weeks, collagen breakdown and synthesis are balanced. Early type III collagen is replaced by type I collagen, eventually hitting a normal 4:1 ratio. Reaches peak tensile strength (80%) at 60 days.

   Other important processes/factors/cells:
   - Epithelialization—requires mobilization of epithelial cells, migration, and mitosis followed by differentiation.
   - Contraction—a result of myofibroblast contractile forces. Appears at day 3, max at days 10–21, disappear as contraction is complete.

Types of Wound Healing—*Primary*: closed within hours of creation by reapproximating edges of wound. *Secondary*: wound allowed to heal on its own by granulation, contraction, and epithelialization. *Delayed primary*: subacute/chronic converted to acute wound by debridement then closed.

Factors that decrease/affect wound healing.

- *Genetic*: predisposition to keloid or hypertrophic scarring, collagen abnormalities, skin pigmentation, sebaceous quality, wound location, age.
- *Systemic*: delayed healing with diabetes, atherosclerosis, renal failure, immunodeficiency, smoking, vitamin deficiency, hypothyroidism.
- Vitamins required for wound healing = vitamin A (increases tensile strength and epithelialization), vitamin C (needed in collagen synthesis), vitamin E (cell membrane-stabilizing antioxidant), and zinc (enzyme cofactor).
- *Local*: infection ($>10^5$) decreases oxygen, lowers pH, slows angiogenesis, increases edema. Radiation, free radicals, denervation.

Hemostasis

Conceptual components of hemostasis

1. Blood vessel: Immediate vasoconstriction, damaged vessel wall presents factors that induce platelet aggregation.
2. Platelets: vWF and other factors assist the exposed collagen fibers in inducing platelet

adhesion, aggregation, shape change, and secretion of additional coagulation factor stimulators.
3. Coagulation system: Cascade set in motion by activated lipoproteins/enzymes that results in thrombin-activating fibrin and plug formation.

Anticoagulants

- *Unfractionated heparin*—inactivates factors II, IX, X, XI, and XII. t½ = 60 min. Monitored by aPTT.
- *Low-molecular-weight heparins*—(Lovenox) decreased nonspecific tissue binding, inactivates factors II and X. Increased t½, no lab test monitoring. Reversed with protamine.
- *Warfarin*—Inactivates Vit. K-dependent cofactors II, VII, IX, X, and proteins C and S. t½ = 20–60 h. Monitored by PT/INR. Reversible with vitamin K and/or FFP.
- *Factor Xa inhibitors*—(Xarelto). T1/2 = 5–9 hours. No effect on PT, PTT, or clotting time.

Preoperative Medications

Preoperative antimicrobial coverage should be given 30–60 min before incision.

- *Skin incisions*—cover for *S. aureus*: cefazolin, clindamycin, or vancomycin if PCN allergy
- *Mucosal incisions*—cover for anaerobes: clindamycin or ampicillin/sulbactam
- *Major aerodigestive surgery*—*S. aureus, Pseudomonas*, and anaerobe coverage: clindamycin + gentamicin, or cefazolin + metronidazole
- *CSF contamination*—aggressive *S. aureus, Pseudomonas*, and anaerobe coverage: vancomycin + ceftazidime + metronidazole

Wound Dressings

Moist wound healing = ~30% faster reepithelialization, plus ↑ local growth factors, ↑ fibroblast, and keratinocyte migration to wound. Oxygen at normal concentrations is required. Growth factor levels are based on oxygen tension. Ideal dressing properties: conform to wound, wick secretions, gentle compression (hemostasis), easy application/removal. Many pros and cons based on whether the dressing is natural (cellulose-based dressings) vs. synthetic, medicated vs. plain, occlusive vs. semipermeable. Skin-equivalent dressings typically contain a biocompatible structural scaffold/matrix with or without cultured keratinocytes or fibroblasts. Usually expensive, mostly for chronic wounds, burns.

Adjunctive Therapies

Hyperbaric oxygen Breathing 100% O_2 while under elevated atmospheric pressure allows for complete hemoglobin saturation and additional O_2 to dissolve in the plasma. Boosts oxygen tension in compromised areas (chronic wounds, ulcers, tenuous flaps/grafts), thus increasing angiogenesis and fibroblast proliferation.

Leeches Direct venous removal plus secretion of *hirudin*, collagenase, hyaluronidase, and factor X inhibitor, anticoagulant enzymes. Feed for 20 min–2 h, 10–15 ml of blood leeched, with additional oozing secondary to anticoagulants. Used for flap venous congestion. Prophylaxis with fluoroquinolone or third-gen cephalosporin due to *Aeromonas hydrophila* infection risk.

Intralesional steroid injection Decreases local fibroblast proliferation and collagen synthesis, thus favoring net collagen degradation. Useful for postoperative swelling, keloid, or hypertrophic scarring. Therapeutic effect lasts for ~6 weeks, decreased dose needed for earlier injection. Concern for subcutaneous atrophy, up to 4% in keloid/hypertrophic scar injections.

Complications

Tobacco Morbidity

Increased *tissue ischemia* due to (1) nicotine-induced vasoconstriction via ↑ thromboxane A_2 and platelet aggregation stimulation, (2) inhaled carbon monoxide binds to hemoglobin resulting in carboxyhemoglobin formation, thus decreasing oxygen delivery.

Additional intraoperative and postoperative pulmonary, cardiovascular, and cerebrovascular complications occur due to ciliary paralysis, thrombogenesis, leukocyte dysfunction, and microvascular injury. *Infection* rate is elevated. Wounds heal slower. Risk of skin flap necrosis increases. Suggest tobacco discontinuation for 4 weeks before and 4 weeks after elective surgery. Patient management should include identification of tobacco product use, provision of counseling and smoking cessation information, and selection of preoperative candidates based on tobacco use.

Complication Management

Arguably the most important step to preventing complications is patient selection, and the most important step to dealing with complications is patient education and management of expectations.

Complication management necessitates careful patient selection, estimation of operative risks, and patient-adapted selection of procedures. Preoperatively, the problem belongs to the patient. Postoperatively, the problem belongs to the surgeon.

Increasing frequency of obesity, aging population, and multimorbidity patients require more complication prevention discussions. Age alone is not an independent risk factor, but medical complications are more likely to be present in an advanced age population.

Informed Consent

Informed consent is not a signed piece of paper; it is a discussion of the risks and benefits involved, any alternative treatments, and the risks and benefits of doing nothing. For consent to be valid the patient must (1) be competent to take the particular decision, (2) have received sufficient information to make a decision, (3) not be acting under duress.

Universal Protocol

Intended to prevent wrong person, wrong procedure, wrong site surgery in hospitals and outpatient settings. Consists of three steps: (1) A preoperative/pre-procedure verification process. (2) Marking the operative/procedure site. (3) A "Time Out" (final verification) which is performed immediately before starting the operation/procedure.

Standard Precautions

A set of infection control practices used to prevent transmission of diseases that can be acquired by contact with blood, body fluids, non-intact skin, and mucous membranes. Treat each patient as if a potential infection source. Also, protects patients from the physician as a vector.

Questions

1. The buccal branch of the facial nerve innervates which muscles of facial animation?
2. Which is the most correct regarding the lymphatic drainage of (1) the upper lip/lateral lower lip and (2) the central lower lip/chin?
   (a) No difference, they both drain to ipsilateral submandibular lymph nodes
   (b) Upper lip/lateral lower lip drains ipsilaterally (submandibular nodes) and central lower lip/chin drains bilaterally (submental nodes)
   (c) No submental lymph node drainage is present from these areas
3. What are the soft-tissue layers in the temporal region? What layer does the temporal branch of the facial nerve reside in?
4. Which fascial layer of the neck is contiguous with the SMAS in the face?
   (a) Superficial cervical fascia
   (b) Deep layer of the superficial cervical fascia
   (c) Muscular division of the deep cervical fascia
   (d) Investing layer of the deep cervical fascia
5. What are the four vertical buttresses and four horizontal buttresses of the face?
6. What additional muscles does the trigeminal nerve, branch V3 innervate besides the muscles of mastication?
7. What additional muscles does the facial nerve innervate besides the muscles of facial animation?

8. In which ganglion are the cell bodies for the postganglionic parasympathetic fibers of the lacrimation nerve pathway found?
   (a) Lacrimal ganglion
   (b) Greater petrosal ganglion
   (c) Otic ganglion
   (d) Pterygopalatine ganglion
9. Name two major differences between the bone scan (bone scintigraphy) and DEXA scan (bone density scan):
10. What is the definition of "depth of field" in photography?
    (a) Distance in millimeters from the optical center of the lens to the focal point, which is located on the sensor
    (b) Distance between the closest and farthest in-focus areas of a photograph
    (c) Distance between the optical center of the lens and the object to be photographed
11. Which type of zoom, *optical zoom* or *digital zoom*, changes the amount of the scene hitting the sensor, thus allowing for more detail that can be enlarged without distortion or pixelation?
12. Typically history alone can diagnose congenital blepharoptosis, but if history is equivocal, which of the following will you see with downward gaze?
    (a) No change in the position of the affected upper eyelid
    (b) Lagophthalmos of the eyelid due to levator fibrosis
    (c) Additional descent of the affected upper eyelid, matching the gaze
    (d) None of the above
13. Which of the following descriptions and treatment options best fit the cephalometric diagnosis of "mandibular deficiency?"
    (a) Retrognathism—deficient lower 1/3 with a class II occlusion. Treatment = mandibular advancement.
    (b) Prognathism—a prominent lower 1/3 with a class III occlusion. Treatment = maxillary advancement ± mandibular setback.
    (c) Deficiency of infraorbital/paranasal sinuses. Treatment = LeFort I osteotomy with expansion ± bone grafting.
    (d) Vertical excess of middle third, convex profile typically a class II occlusion, and lip incompetence. Treatment = LeFort I osteotomy with impaction.
14. List the three phases of wound healing, their duration, and the predominant cell type/processes found in each:
15. How does smoking affect wound healing on a cellular level by inciting tissue ischemia?
16. Which muscles of facial expression are innervated on their superficial surface?
17. Describe Bogorad's syndrome, or crocodile tears
18. What is the neurotransmitter responsible for Frey's Syndrome?
19. In Frey's syndrome, damage to which somatosensory nerve can result in misdirected parasympathetic secretomotor innervation to the skin of the cheek anterior to the ear?
    - Greater superficial petrosal
    - Zygomaticofacial
    - Auriculotemporal
    - Zygomaticotemporal
    - Lesser petrosal nerve
20. What are the anatomical differences between the East Asian and Caucasian upper lid?
21. What are the five main blood vessels that supply the scalp?
22. What is the most commonly infected deep neck space?
    (a) Retropharyngeal
    (b) Carotid
    (c) Submandibular
    (d) Parapharyngeal
    (e) Danger space
23. What is the most common source of deep neck space infections?
    (a) Respiratory
    (b) Odontogenic
    (c) Foreign body
    (d) Penetrating trauma
    (e) Peritonsillar abscess
24. What are the layers of the pharynx in order starting at the mucosa advancing toward the cervical spine?
25. Why is the danger space so named?
26. True or False: It is appropriate to treat lingual abscesses with needle aspiration and

multi-antibiotic therapy as opposed to open incision and drainage with antibiotic therapy.

27. Which salivary gland produces the most mucinous saliva?
28. Which craniofacial syndromes are associated with branchial arch anomalies?
29. What is the difference between type I and type II first branchial arch anomalies?
30. What is the course of second branchial cleft cyst?
31. What is the course of a third branchial cleft cyst?
32. Which branchial arch gives rise the Hillocks of His I-III? What does each hillock differentiate into?
33. What age does the human ear reach approximately 90% of its adult size?
    (a) 3 years
    (b) 4 years
    (c) 5 years
    (d) 6 years
    (e) 7 years
34. What is the primary collagen type in early wound healing? Late wound healing?
35. How can you be sure your patient has stopped smoking prior to performing an elective operative procedure?
36. Which of the following statements (one or more) are false regarding the pogonion?
    (a) It describes the most anterior soft tissue aspect of the chin
    (b) It is one of the points that defines the cervicomental angle
    (c) It is the most inferior and posterior aspect of the mandible
    (d) The pogonion represents the most inferior soft tissue aspect of the chin
37. Aesthetic subunits of the face are used for
    (a) Guide margin width for excision of malignancy
    (b) Have no bearing on reconstructive options after an excision
    (c) Helping to orient scars, makes scars less conspicuously after reconstruction
    (d) Defining the lines of maximal extensibility
38. What is considered the ideal focal length of a lens used for photodocumentation in facial plastic surgery?
    (a) 18 mm
    (b) 35 mm
    (c) 55 mm
    (d) 105 mm
    (e) 18–55 mm

Answers

1. Procerus, medial, and lower aspect of the orbicularis oculi, nasalis, partial innervation of the zygomaticus major/minor, upper orbicularis oris
2. (b) Upper lip/lateral lower lip drains ipsilaterally (submandibular nodes) and central lower lip/chin drains bilaterally (submental nodes)
3. Skin → SubQ → superficial temporal fascia → loose areolar tissue → superficial layer of the deep temporal fascia → superficial fat pad → deep layer of the deep temporal fascia → deep fat pad → temporalis muscle → pericranium

   On the under surface of the superficial temporal fascia (temporoparietal fascia)
4. (a) Superficial cervical fascia
5. Vertical: nasomaxillary, zygomaticomaxillary, pterygomaxillary, mandible condyle/ramus

   Horizontal: frontal, zygomatic, maxillary, mandibular
6. Tensor tympani, tensor veli palatini, mylohyoid, anterior digastric muscle
7. Stapedius, stylohyoid, posterior digastric muscle
8. (d) Pterygopalatine ganglion
9. Bone scan = functional test vs. DEXA = structural test only

   Bone scan = nuclear based on radioactive particles vs. X-ray based

   Bone scan = more expensive vs. less inexpensive
10. (b) Distance between the closest and farthest in-focus areas of a photograph
11. Optical zoom
12. (b) Lagophthalmos of the eyelid due to levator fibrosis

13. (a) Retrognathism—deficient lower 1/3 with a class II occlusion. Treatment = mandibular advancement.
14. Inflammatory = days 1–6, macrophages
    Fibroproliferative = day 4–week 3, fibroblasts
    Maturation/remodeling = week 3–1 year, fibroblast collagen synthesis
15. (a) Nicotine-induced vasoconstriction via ↑ thromboxane A2 and platelet aggregation stimulation
    (b) Inhaled carbon monoxide binds to hemoglobin resulting in carboxyhemoglobin formation, thus decreasing oxygen delivery
16. Buccinator
    Levator anguli oris
    Mentalis
17. Parasympathetic secretomotor fibers intended for the salivary glands become misdirected in the geniculate ganglion to pass through the greater superficial petrosal nerve instead of the chorda tympani. This causes profuse lacrimation to occur as a result of a gustatory stimulus.
18. Acetylcholine
19. Auriculotemporal
20. East Asian upper eyelids lack a distinct crease due to
    (a) Fusion of the orbital septum to the levator aponeurosis below the superior tarsal border
    (b) Protrusion of the preaponeurotic fat pad and a thick subQ fat layer at the tarsal border
    (c) Insertion of the levator into the orbicularis muscle and lid skin is closer to the lid margin
21. Supratrochlear, supraorbital, superficial temporal, occipital, posterior auricular artery
22. (d) Parapharyngeal
23. (b) Odontogenic
24. Mucosa → Buccopharyngeal fascia → Retropharyngeal space → Alar fascia → Danger space → Prevertebral fascia → Spine
25. Composed of loose areolar tissue that offers little resistance to the spread of infection bilaterally into the mediastinum
26. True
27. Sublingual
    The most serous? Parotid
28. Branchial-oto-renal, Goldenhar, Treacher-Collins, Nager, Miller, Wildervanck, Bixler, Mobius, Orofaciodigital syndromes types I-VIII
29. Type I lesions typically track medial to the facial nerve presenting as a mass, sinus, or fistula near the angle of the mandible. They contain ectoderm only with no cartilage or adnexal structures.
    Type II anomalies appear as a duplication of the external auditory canal with a epithelial lining, adnexal structures, and cartilage.
30. Anterior neck neck mass just anterior and medial to SCM muscle → Along carotid sheath → Between external and internal carotid arteries → superficial to CN IX and XII → Opens into tonsillar fossa
31. Anterior neck mass (similar or lower location as 2nd BCC) → Deep to carotids → Deep to CN IX, superficial to CN XII, superficial to superior laryngeal nerve → Pierces thyrohyoid membrane → Opens into apex of pyriform sinus
32. First branchial arch. (1) Tragus, (2) root of the helix, (3) helical crus
33. (c) 5 years
34. Early – type III collagen
    Late – type I collagen
35. Urine nicotine test strip (nicotine stays positive in urine 1–3 days)
36. (c) It is the most inferior and posterior aspect of the mandible
    (d) The pogonion represents the most inferior soft tissue aspect of the chin
37. (c) Helping to orient scars, makes scars less conspicuously after reconstruction
38. (d) 105 mm

Legal, Ethical, and Information Technology Issues in FPRS

5

Brandyn Dunn and Andrew Kroeker

Legal Issues

Informed Consent

- Four components must be included:
  - *Capacity*: assess the patient's ability to make a medical decision
  - *Disclosure*: provide necessary information to make an autonomous decision.
  - *Competency*: assess the patient's ability to understand information provided
  - *Voluntary consent*: make a decision, free from coercion, manipulation, or other outside force
- Documentation and/or discussion must include:
  - *Diagnosis*
  - *Nature/reason of proposed treatment*
  - *Probability of success*
  - *Risks, benefits, complications, and side effects of intervention*
  - *Alternatives to treatment, potential consequences if advice is not followed*
- Photography Consent
  - *Confidentiality*: treated with same safeguards as other portions of the medical record, encompassed within HIPAA guidelines
  - *Copyright*: images are "owned" by the physician, but must have explicit written permission from the patient, and patient must understand that the photos are used to promote the physician's practice.
  - Use of "*before and after*" images:
    - Should be taken in similar lighting and background conditions
    - Use of these images vary by state statutes
  - Computer modeling: patients should sign an acknowledgment of the limitation of software
- Consent of a minor
  - "*Consent by proxy*" via parents until age 18, but recommendations are to procure adolescent children's *assent* to procedure
  - *Exceptions*: legally emancipated children, specific issues related to sexually transmitted diseases and pregnancy testing/procedures

Standard of care defined as the care that the average, careful, and prudent practitioner of one's specialty would be expected to meet under the same circumstances.

B. Dunn (✉)
Department of Otolaryngology Head and Neck Surgery, University of California, Irvine, Orange, CA, USA
e-mail: bsdunn@uci.edu

A. Kroeker
Department of Otolaryngology – Head and Neck Surgery, University of Michigan Health System, Ann Arbor, MI, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_5

Affirmative duty legal concept under which the physician is required to volunteer information to the patient, rather than wait for a question.

Liability

- *Liability*: legal responsibilities for ones actions or omissions.
- "*Prudent person*": legal concept in which physician has *no liability* as long as a *reasonable and prudent person* in that patient's position would have *accepted the treatment had he or she been adequately informed of all significant risks*
- *Vicarious liability*: secondary liability, the employer is responsible for the actions or omissions of their employees, as long as they are working within the confines of their job.

Record-Keeping and Protected Health Information (PHI)

- Important to maintain accurate, timely, and secure medical records
- Never alter the medical record
- Correcting inaccuracies in the medical record:
  - Never change or erase what is already present
  - Should include a narrative entry into the medical record explaining the error and describing the correct information, time, and date of the correction
  - The original information should be viewable

Duty to finish care, regardless of ability to pay care for that patient is the responsibility of the doctor regardless of whether the patient can meet financial obligations, until no further care is required

- Higher percentage of trauma patients will not pay bill and do not carry insurance; however, there is a "duty to treat" under the Emergency Medical Treatment and Active Labor Act of 1986

Medical Negligence/Malpractice

- Four general elements to prove in medical negligence/malpractice case
  1. *Physician duty to care for the patient*: ethics of doctor–patient relationship
  2. *Breach of duty*: physician did not perform to the level of the accepted *standard of care*
  3. *Causal relationship*: violation of standard of care must have led directly to bad outcome
  4. *Damage*: a loss/injury must have occurred to the patient that can be compensated

Legal Issues Associated with Treating "The Unhappy Patient"

- Continue to treat the patient until they find care elsewhere
- Be courteous and kind to the patient, instruct office staff to do the same in all interactions
- Promptly make copies of all patient records upon request
- Notify your malpractice insurance carrier, even if only an "occurrence" and not yet a "claim"
- *Never alter* any aspect of the medical record
- Respond promptly to the state medical board, if inquiries are made

If a Case Goes to Trial…

- Notify malpractice insurance carrier as soon as a claim is received
- Hire a lawyer for the state medical board (interview multiple attorneys, talk to physicians they have represented). The insurance carrier will hire one for the court case, but investigate the lawyer and assure they have a good track record, work well with others, etc.
- Research the case extensively; perform a "dress rehearsal"
- Assist in finding experts, texts, and journal articles to support your case
- Research the plaintiff's expert witness

- Give testimony at your attorney's office, not your office
- Insist on reviewing the trial strategy with your attorney
- If insurance company decides to settle…
  - Settlements go into the National Physician Databank
  - You have a right to refuse, but understand the specifics of the policy as it relates to your own liability
  - If settlement is above policy limit, hire your own attorney to demand the insurance company settle within the limits of the policy.

Termination of Doctor–Patient Relationship

- Determination that no further treatment is necessary
- Referral to another physician with patient consent
- Sufficient notice to patient must be given to obtain care elsewhere
- If these are not met, *regardless of patient ability to meet financial obligations*, can be considered "*abandonment*" (unilateral termination without effort to find competent and equivalent care)

Ethical Issues

Ethical Principles

- *Autonomy*: create conditions necessary for autonomous choice and to honor the patient's preference in accepting or not accepting medical care.
- *Beneficence*: act in the patient's best interest.
  - May conflict with autonomy or what is best for society
- *Non-maleficence*: "Do no harm." Must be balanced against beneficence; if the benefits outweigh the risks, a patient may make an informed decision to proceed
- *Justice*: To treat persons fairly and equitably. This does not always imply equally (e.g., triage).

Doctor–Patient Relationship

- *Physician duty*: perform to the level of the accepted standard of care.
- Privacy and trust: fundamental component of the relationship that allows for full disclosure and honest communication necessary for patient care
  - *Exception*: patient threatening harm to self or others

Discussion of medical errors straightforward component of the underlying ethic of the doctor–patient relationship. More subtly in "errors" that do not cause a measurable effect.

Conflicts of Interest

- Physician must manage personal (religious and political philosophies) and professional (financial and relationship to pharmaceutical industry) conflicts of interest in the doctor–patient relationship to avoid unethical behavior.

Information Technology Issues

IT Legal and Security Issues

Protected Health Information (PHI) includes names, dates relating to a patient (i.e., birthdates, dates of medical treatment, admission and discharge dates, and dates of death), telephone numbers, addresses (including city, county, or zip code), fax numbers and other contact information, social security numbers, medical records numbers, photographs, finger and voice prints, and any other unique identifying number.

The penalty for breaking HIPAA is tiered:

1. Individual did not know (and by exercising reasonable diligence would not have known) that he/she violated HIPAA → \$100 per violation, with annual max of \$25K for repeat violations.
2. Violation due to reasonable cause and not due to willful neglect → \$1000 per violation, \$100k max, possible imprisonment of 1 year

3. HIPAA violation to willful neglect, but violation is corrected within 30 days → $10k per violation, $250k max, possible imprisonment of 5 year
4. HIPAA violation to willful neglect, but violation is NOT corrected within 30 days → $50k per violation, $1.5 million max, possible imprisonment of 10 years

http://www.ama-assn.org/ama/pub/physician-resources/solutions-managing-your-practice/coding-billing-insurance/hipaahealth-insurance-portability-accountability-act/hipaa-violations-enforcement.page?

Online Ethics

- AMA guidelines for health information websites, area that require quality standards
  1. *Content*: disclosure, authorship, and attribution should always be provided, independent expert review
  2. *Advertising and sponsorship*: must be easily distinguishable from editorial content
  3. *Privacy and confidentiality*: must state explicit policies, ensure patient privacy
  4. *E-commerce*: must function efficiently and securely
  – AMA Guidelines for Truthful Advertising of Physician Services, 1992
    1. Advertisements should not contain material false claims or misrepresentation of material fact
    2. Advertisements should not contain material implied false claims implied misrepresentation of material fact
    3. There should not be knowing omissions of material fact from advertisements
    4. Physicians should be able to substantiate material objective claims and representations make in an advertisement
  – Additional criteria for websites recommended:
    The language on the web should be easily understandable
    The viewpoint should be from that of the patient
    Claims should not be overly exaggerated
    Claims should not be directed to the anxiety, fear, or hope of patients
    Advertisements should be identifiable as advertisements
- Other areas, if listed on the website, which a surgeon must be able to substantiate:
  – Licensure, board certification, memberships, years of experience, subspecialization/unique skills,
  – Financial conflicts should be disclosed on the surgeon's website
  – Fees and cost listed shall not be misleading
  – Success rates, if listed, must be explained
  – Images: should accurately depict the results of services, should be only of the surgeon's own patients, and should have full written consent for display in a public setting

Social Media

Social media is playing a larger role in marketing and patient education. Roughly half of plastic surgeons routinely use social media. In a survey of the American Society of Plastic Surgeons, most respondents (64.6%) stated that social media had no effect on their practice, whereas 33.8% reported a positive impact and 1.5% reported a negative impact. (Vardanian - Plast Reconstr Surg. 2013 May;131(5):1184–93)

An older article in the compendium from 2009 (Guesh II – J Med Ethics 2009:35: 584–586) reviewed the role of social media in medicine. The following is a summary of recommendations:

- Do not invite a patient to become an online friend.
- Avoid accepting friendship with patients on social media.
- Be thoughtful when using social media (twitter, Facebook) for personal use.
- Use conservative privacy settings.
- Respect patient's privacy and comply with privacy laws (e.g. HIPAA).
- Consent patients specifically for use of photography in social media.

Questions

1. Prior to obtaining an informed consent for a cosmetic rhinoplasty, you hear the patient's boyfriend whisper to the patient, "You are beautiful, but after this nose job you will be perfect." Which component of informed consent is compromised by this comment?
   (a) Capacity
   (b) Voluntary
   (c) Competency
   (d) Disclosure
2. When documenting informed consent in electronic medical records, all of the following components are necessary to document except for?
   (a) Diagnosis
   (b) Reason for treatment
   (c) Details of the procedure
   (d) Risk, benefit, alternatives
3. True or False. When consenting a minor, it is legally required for the patient to agree to the procedure.
   (a) True
   (b) False
4. In what situation is it ethical to terminate a doctor–patient relationship?
   (a) Patient went bankrupt and can no longer make payments on current payment plan
   (b) Physician moves across the country without making prior arrangements for his/her patients to see another physician
   (c) A patient request to be friends with you on a social media website
   (d) Physician notifies patients that he is getting ready to retire and will be selling his practice to a new facial plastics surgeon.
5. What is the definition of "standard of care?"
6. True or false. A calculation mistake made by a medical assistant in your clinic results in a patient receiving 10 times the expected concentration of Botox. This results in significant bilateral brow ptosis. As the treating physician, you are medically liable for this mistake.
   (a) True
   (b) False
7. Based on the prior question, which medical legal issue is being highlighted?
   (a) Standard of Care
   (b) Affirmative Duty
   (c) Vicarious Liability
   (d) Prudent Person
8. Name and describe the four fundamental ethical principles.

Answers

1. (b) Voluntary
2. (c) Details of the procedure
3. (b) False
4. (d) Physician notifies patients that he is getting ready to retire and will be selling his practice to a new facial plastics surgeon.
5. The care that the average, careful, and prudent practitioner of one's specialty would be expected to meet under the same circumstances
6. (a) True
7. (c) Vicarious Liability
8. 1. Autonomy: create conditions necessary for autonomous choice and to honor the patient's preference in accepting or not accepting medical care.
   2. Beneficence: act in the patient's best interest
   3. Non-maleficence: "Do no harm."
   4. Justice: To treat persons fairly and equitably

6 Psychosocial Aspects of FPRS

Brandyn Dunn and Anita Konka

The psychological aspects of plastic surgery as discussed below pertain to elective cosmetic surgery patients. Craniofacial, trauma, and reconstructive psychological factors are discussed later in this chapter.

Plastic Surgeon's Role

- Plastic surgeon's take on a psychosocial role of understanding and identifying the motivations of the cosmetic patient as well as managing expectations
  - External motivators: fear of age discrimination, coercion by family, friends, coworkers, avoidance of ethnic prejudice
  - Internal motivators: diminish unpleasant feelings associated with depression, shame, social anxiety, reduce psychosocial distress
  - Desire to address a specific feature that a patient dislikes or finds distracting
- DeVries key competencies for the modern plastic surgeon's successful fulfillment of his or her role

1. The influence of gender

   Women: predisposed to desire appearance of youth and health/active strategy to cope with transitions of life

   Men may be more interested in combatting age or altering features to maintain or achieve a strong, powerful look
2. Ruling out psychopathology
3. Including the patient's social world
4. Coping, stigma, and psychosocial history
5. Follow-up and a referral team

Body Image

- Definition: internal representation of the self's outer appearance
- Tridimensional conception of one's own body involving *interpersonal, environmental, and temporal factors.*
- *Dissatisfaction with body image is considered a major motivation for patients seeking cosmetic surgery* although overall body image dissatisfaction is not significantly different between plastic surgery patients and the general population.
  - However, cosmetic surgery patients had *significantly greater dissatisfaction with specific bodily feature*

B. Dunn (✉)
Department of Otolaryngology Head and Neck Surgery, University of California, Irvine, Orange, CA, USA
e-mail: bsdunn@uci.edu

A. Konka
Otolaryngology – Head and Neck Surgery, University of South Florida, Tampa, FL, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_6

Body Image: 4 Stages of Development

- *Stage I (Neonate)*: learn and develop feelings of self based on attention, love, and approval in their environment.
  - Foundation of a positive body image.
- *Stage II (Grade School)*: leave home environment and encounters outside perception with positive and negative feedback.
  - Influences personal confidence and patterns of thinking regarding body image
- *Stage III (Adolescence and Puberty)*: vulnerability and sensitivity to opinions and impressions formed by peers.
  - Foundation of negative pattern of body image.
- *Stage IV (Senescence)*: loss of the youthful appearance and change in perception of own body image

Body Dysmorphic Disorder (BDD)

- BDD involves a preoccupation or obsession with physical features that is far beyond what is considered normal.
- Associated with depression
- Often exhibit surgically addictive behaviors (insatiable desire for surgery)
- Only body image-related diagnostic category addressed in the DSM-V (Table 6.1).
- DSM V criteria also account for clinical impressions of whether the individual has good, poor, or absent (delusional) insight regarding their BDD beliefs.
- *Patients will focus on an average of five different body parts over the course of the disorder* (Table 6.2).
- *6–16% met diagnostic criteria for body dysmorphic disorder (BDD).*
  - *Type-changing patients* (i.e., *rhinoplasty, breast augmentation/reduction) showed significantly greater number of BDD symptoms.*
- These are dangerous patients for the aesthetic surgeon, as they usually cannot be satisfied unless BDD is treated successfully by mental health professional
- Patients rarely comprehend limitations of surgery (delusional fixation)

Table 6.1 Body Dysmoprhic Disorder (DSM V)

1. Preoccupation with one or more perceived defects or flaws in physical appearance that are not observable by or appear slight to others
2. During the course of the disorder, the individual has performed repetitive behaviors (e.g., mirror checking, excessive grooming, skin picking, or reassurance seeking) or mental acts (e.g., comparing his or her appearance with that of others) in response to the appearance concerns
3. The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning
4. The appearance preoccupation cannot be better explained by concerns with body fat or weight in an individual whose symptoms meet diagnostic criteria for an eating disorder

Table 6.2 10 Most perceived facial defects in patients with BDD

Body part	% of patients with concern
Skin	73
Hair	56
Nose	37
Eyes	20
Teeth	20
Ugly face	14
Lips	12
Chin	11
Eyebrows	11
Ears	9

Identifying Problem Patients

- The ideal patient has legitimate concerns, realistic expectations, is secure, well-informed, and understands the limitations of surgery
- *Tip-offs to "possible trouble"*
  - Obsessive-compulsive behavior
  - Excessive doctor shopping
  - Urgent demand for surgery without due consideration
  - Extreme flattery

- History of litigation
- Poor hygiene
- Motivation based in interpersonal issues
- Unreasonable or unrealistic expectations
- SIMON (rhinoplasty)
  Single, immature, male, over-expectant, narcissistic

Patient Selection and Assessment Tools

- Initial consultation should be given ample time to obtain a comprehensive history including full medical, including psychiatric history (patient may willingly omit details), complete medication history, social history, smoking history as well as information about the patient's social network, family, and significant others.
- Assess patient agenda during consultation
  - Psychological agendas (i.e., surgery as means to improve an emotional state)
  - Interpersonal agendas (i.e., improve a relationships)
  - Social agendas (i.e., employability, social status)
- *Psychosocial assessment tools include validated instruments*
  - *Multidimensional Body-Self Relations Questionnaire:* Measures body image
  - *Body Dysmorphic Disorder Examination Self-Report*: Measures body image dissatisfaction and the symptoms of BDD
- Photo documentation
  - Digital imaging can be used to assess perceptual dimensions of body image

Outcomes

- Factors associated with poor psychosocial outcome:
  - Being young, being male, having unrealistic expectations of the procedure, previous unsatisfactory cosmetic surgery, minimal deformity, motivation based on relationship issues, and a history of depression, anxiety, or personality disorder.
  - Body dysmorphic disorder was also recognized by some studies as a predictor of poor outcome

Anxiety Disorders in Plastic Surgery

- Surgery may induce a complex set of psychological symptoms, possibly reflecting additive effects of previous traumas or maladaptive coping patterns→ increased levels of anxiety
- Patients who are less anxious during perioperative period report less emotional distress, fewer defensive behaviors, and are more likely to be satisfied with surgical outcomes
- Anxiety related postoperative reactions include
  - Panic attack
    Discrete period of intense fear or discomfort, accompanied by at least four somatic or cognitive symptoms
  - PTSD
    Extreme anxiety following exposure to traumatic stressors involving direct person experiences
    Lasts >4 weeks
  - Acute Stress Disorder
    Immediate response to traumatic or stressful event that lasts at least 2 days; does not persist beyond 4 weeks after trauma or crisis.
    Sx: inability to concentrate, avoiding pleasurable activities, recurrent images/dreams/flashbacks of trauma. "Reliving the trauma of surgery"
- Engage mental health professional early in patient with significant anxiety disorders

Special Circumstances: The Craniofacial, Reconstructive, and Adolescent Patient

- Facial attractiveness has a strong effect on interpersonal impressions.
- Facial deformities caused by trauma, congenital disabilities, and postsurgical sequela (reconstruction/scar revision) present with significant adverse functional consequences

- *Patients with abnormal facial features were rated as significantly less honest, less employable, less trustworthy, less optimistic, less effective, less capable, less intelligent, less popular, and less attractive when compared to "normalized" patients*. (Rankin and Borah 2001)
- Sarwer 1998: Case-control study comparing 24 patients with craniofacial anomalies with age and gender-matched controls
  - Craniofacial patients had greater dissatisfaction with facial appearance
  - *Craniofacial patients reported lower self-esteem, quality of life*

Adolescent Patients

- Patients can present with congenital, aesthetic, or functional concerns
- Unique challenges of adolescent patients:
  - *Timing of operation* (awake, old enough, has comprehension, emotionally and intellectually mature)
    - Physical/functional impairment (otoplasty, cleft deformity): proceed with parent/guardian approval
    - Traumatic deformities (rhinoplasty): should be delayed until child matures
  - *Parent/guardian as decision-makers*
- Adolescents may benefit from plastic surgery particularly aesthetic breast surgery, in manner not accounted for by "natural" development.

Questions

1. How many stages are involved in the development of body image throughout life?
   (a) 4
   (b) 3
   (c) 6
   (d) 10

   Body image develops during (1) infancy/childhood (2) school-age (3) adolescence/puberty (4) senescence
2. Compared to the general population cosmetic surgery patients report
   (a) Higher dissatisfaction with body image
   (b) Lower prevalence of BDD
   (c) Higher prevalence of BDD, but lower dissatisfaction with body image
   (d) Higher prevalence of BDD

   Cosmetic surgery patients do not demonstrate a significantly different assessment of body image, but do have a higher prevalence of BDD (6–16% by DSM criteria)
3. Postoperatively, the majority of cosmetic surgery patients meet diagnostic criteria for clinical anxiety
   (a) True
   (b) False

   The majority of cosmetic surgery patients have a positive outcome. While a minority may be unsatisfied with surgical outcomes, >50% experience anxiety postoperatively. However, only a small proportion of this subgroup will meet diagnostic criteria for clinical anxiety disorder.
4. A patient with mild nasal deformity, who hates his nose and meets criteria for BDD, will stop obsessing if the deformity is corrected
   (a) True
   (b) False

   Patients with BDD have delusional fixations that inhibit acceptance of a good surgical result and may demanding additional surgery or revisions. If the patient is satisfied with surgical outcome, he is nonetheless likely to move on to obsessing over another facial feature.
5. Compared with "normalized" patients, those craniofacial deformities are evaluated as:
   (a) Less honest
   (b) Less employable
   (c) Less intelligent
   (d) All of the above

Answers

1. (a)
2. (d)
3. (b)
4. (b)
5. (d)

Part II

Reconstruction

7 Flaps and Grafts

Jennings Boyette, Philip K. Robb Jr.,
and Irene A. Kim

The Reconstructive Ladder

- Secondary healing
- Primary closure
- Delayed primary closure
- Skin grafts (split vs. full thickness)
- Local tissue transfer (random flaps)
- Regional/axial tissue transfer
- Free tissue transfer

Grafts

Autogenous grafts are always preferred over synthetic or cadaveric grafts to minimize complications.

Graft Types

Skin Grafts

Skin grafts can be split thickness (STSG), full thickness (FTSG), or dermal grafts. Generally speaking, the thinner the graft, the more likely it will survive—as there is less biologic load to support and decreased nutrient requirement. Graft survival is dependent upon the recipient bed for vascularization. The recipient bed should be free of necrotic debris, hemostatic, sterile, and immobile for optimal take. Grafts placed over tissues with high bacterial counts (>10^5 organisms/gram of tissue) may not survive. Grafts need direct contact for vascularization; therefore, do not place directly over bone, cartilage, or tendon. Bolsters may be used to prevent shearing and fluid accumulation under the graft. Grafts may survive if *<1 cm²* based on nutrition from wound edges alone.

- *Contracture of grafts*: *STSG > FTSG > composite grafts*.
  - Primary contraction—immediately after harvest
  - Secondary contraction—during healing phase
- *STSG has less primary contraction and more secondary contraction than FTSG*
- *Perioperative corticosteroids have been shown to increase graft survival*.

J. Boyette
Otolaryngology, University of Arkansas for Medical Sciences, Little Rock, AR, USA

P. K. RobbJr.
Robb Facial Plastic Surgery, Alpharetta, GA, USA

I. A. Kim (✉)
Head & Neck Surgery – Facial Plastic & Reconstructive Surgery, UCLA, Santa Monica, CA, USA
e-mail: iakim@mednet.ucla.edu

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_7

Stages of Graft Vascularization

- *Imbibition*—first 48 h, wound nourishes the graft via diffusion
- *Inosculation*—48–72 h, capillary anastomoses form
- *Neovascularization*—greater than 1 week, new capillary budding into tissue

Split-Thickness Skin Graft (STSG) Contains entire epidermis and variable amount of dermis. Thin STSG: 0.008–0.012 in.; medium STSG: 0.012–0.018 in.; thick STSG: 0.018–0.030 inch

- *Advantages*: Large surface area coverage, reliable, can mesh to increase surface area, no donor-site closure
- *Disadvantages*: Increased contracture, color/texture mismatch, hypertrophic scarring, fragile, donor defect left to heal secondarily. Postop *dermabrasion* is also helpful to smooth transitions
- *Uses*: Large scalp defects, high-risk recurrence sites, donor-site coverage (e.g., forearm/fibula)
- *Donor sites*: thigh, scalp, buttocks, lateral hip

Full-Thickness Skin Grafts (FTSG) Contain entire epidermis and dermis

- *Advantages*: Better color/texture match, less contracture, multiple donor site options, more durable, less donor site morbidity, no need for equipment (i.e., dermatoma)
- *Disadvantages*: High graft failure rate, slow revascularization, more resistant to trauma. Must close donor site due to no residual adnexal remnants
- *Uses*: Nasal, ear, eyelid defect
- *Donor sites*: Postauricular, preauricular, upper eyelid, pretrichial, supraclavicular skin

Dermal Grafts (1–2.5 mm) Contain dermis after deepithelialization

- *Advantages*: Reliable, resistant to infection, can be buried under skin, little resorption, capable of reepithelialization
- *Disadvantages*: Donor-site contour defect
- *Uses*: Intraoral coverage, vessel coverage, tissue bulk/contour (larger volume restoration of facial defects following parotidectomy)
- *Donor sites*: thigh, lower abdominal areas

Cartilage Grafts

Cartilage is classified into three types:

1. *Elastic*: contains elastic and collagen fibers (ear, epiglottis, Eustachian tube)
2. *Hyaline*: most common, contains type II collagen (trachea, bronchi, articulating surfaces of bones, nasal septum)
3. *Fibrocartilage*: cartilage with fibrous bundles of cartilage; more dense than hyaline cartilage (TMJ, pubic symphysis, tendon/ligament attachments)

   Vascularized by diffusion.

- *Advantages*: Ease of harvest, malleable but maintains structural integrity, minimal resorption, can be harvested as a composite graft
- *Disadvantages*: Need for soft tissue coverage, potential donor-site morbidity
- *Uses*: Nasal/orbital reconstruction, rhinoplasty, otoplasty
- *Donor sites*: Nasal septum, auricle, rib

Bone Grafts

May be autogenous, allograft, or synthetic. Autogenous bone consists of *cortical bone* (outer layer, provides strength) and *cancellous bone* (inner layer, contains osteocytes and growth factors).

Biologic Principles of Bone Grafting

- *Osteoconduction*—bone grafting material creates a scaffold which facilitates new bone formation (characteristic of all bone grafts)
- *Osteoinduction*—stimulation of osteoprogenitor cells to promote new bone growth (autogenous grafts and some allografts)
- *Osteogenesis*—new bone growth secondary to osteoblast from bone graft material (autogenous grafts only)
- *Advantages*: maintains structural support, minimal warping, abundant supply

- *Disadvantages*: requires bone-to-bone contact and immobility to minimize resorption, needs soft tissue coverage, potential donor-site morbidity
- *Uses*: mandible, midface, nasal, orbital reconstruction
- *Donor sites*: iliac crest, calvarium, rib

Fat Grafts

Fat grafting has become increasingly important as an adjunctive and primary tool in reconstruction and cosmetic procedures. The key to successful fat grafting is attention to technique. Survival of transplanted adipocytes is dependent upon *inosculation*. Harvesting is typically performed using the Coleman technique which utilizes low-pressure liposuction harvesting, centrifuge refinement, and small aliquot placement in multiple tissue planes. This increases the surface area of contact between the newly grafted fat and host tissue to promote survival.

- *Advantages*: abundant supply, natural contour, permanent
- *Disadvantages*: potential for irregular contours, variable take rate (up to 60% resorption)
- *Uses*: facial augmentation of deep and superficial fat compartments
- *Donor sites*: abdomen, flanks, medial/lateral thigh

Allopath

Biocompatibility is a major consideration when using alloplastic materials. Infection and extrusion are common. The degree of material inertness, the surface texture, the degree of tissue coverage, and fixation to the local tissues all affect an implant's long-term viability. Nasal implants are particularly prone to extrusion.

- *Expanded polutetrafluoroethylene EPTFE* (*Gore-Tex*): sheets can be used for lip, cheek, nasal augmentation, solid shapes available for skeletal augmentation. Initially used in vascular surgery and hernia repair
  – 10 to 30 μm pore diameter allow for soft tissue ingrowth and fixation. Minimal tissue inflammation and infection risk. Easier to remove.
- *Acellular dermis* (*Alloderm*): used for soft-tissue augmentation, dermal replacement following burns, gingival grafts and as slings. Serves as collagen matrix for host tissue ingrowth
  – dermal and epidermal cells and MHC molecules removed via freezing/drying process which minimized immunologic reactions. Prone to resorption over time
- *Porous polyethylene* (*Medpore*): skeletal augmentation, orbital floor implants, microtia; carvable; resistant to deformation secondary to external forces
  – 125 to 250 μm pore diameter allows for vascular and soft-tissue ingrowth; minimal tissue reactivity; difficult to remove
- *Silicone* (*Silastic*): solid implants used for augmentation of chin or malar region, lip and nasal augmentation; carvable, placed in subperiosteal pocket; may cause underlying bony resorption (2–5 mm at chin)
  – nonporous, no vascular or tissue ingrowth. Exhibits micromotion resulting in thick fibrous capsule
- *Methylmethacrylate*: used for cranial defects, can be custom molded in situ; exothermic reaction during curing
- *Polydioxanone plate* (*PDS*): used for orbital fracture repair, septal cartilage repair
  – 0.15 to 0.55 mm thickness. Perforated and unperforated plates available, degraded by hydrolysis and completely metabolized by 25 weeks

Flaps

Classification of Flaps

Flaps may be classified by their *blood supply, composition, location, or movement*. Cutaneous, fasciocutaneous, musculocutaneous, and composite flaps differ in the composition of tissue transferred. Local flaps originate adjacent to the defect.

Regional flaps are transferred within the same anatomic area but not adjacent to the defect, and distant flaps come from another area of the body.

Local flaps can be classified by their blood supply: *axial*, *random*, and *pedicled* flaps. *Axial flaps* are dependent upon a named blood vessel for the majority of their blood supply. These vessels typically run parallel to the linear axis of the flap in subcutaneous tissue. Distally, these flaps have some degree of random pattern vascularity. *Random pattern flaps* are based upon the interconnecting subdermal plexuses and do not have an associated named vessel. These constitute the majority of flaps utilized for facial reconstruction. Regional and free flaps are based upon an angiosome, which is an anatomic unit of tissue (consisting of skin, subcutaneous tissue, fascia, muscle, and bone) fed by a larger source artery (*pedicle*) and drained by associated veins. Adjacent angiosomes are connected through *choke vessels*, which consist of venous and arterial vessels. Under normal physiologic conditions, choke vessels are of small caliber but can dilate and provide perfusion to an adjacent angiosome in setting of decreased blood flow.

Vascularization of the skin occurs through either *musculocutaneous* or *septocutaneous* arteries, which then terminate into the subdermal and dermal vascular plexuses. Under normal circumstances the blood flow to the skin is 10x more than its nutritional requirement. Blood flow is regulated by capillary networks in the reticular dermis and AV shunts in papillary dermis. Local hypoxemia, excessive body temperature, and increased metabolic demand result in vasodilation and increased cutaneous circulation. Extreme cold, nicotine, and sympathetic release of norepinephrine result in the contraction of capillary networks and decreased cutaneous circulation.

Survival of a random pattern flap is dependent upon the *capillary perfusion pressure*. Increasing flap length results in less perfusion pressure to withstand the critical closing pressure exerted on the end arterioles. Venous congestion or lymphedema can also result in decreased perfusion pressure and resultant flap compromise/necrosis. Neovascularization is important for long-term viability and integration with the surrounding tissue. The majority of revascularization involves direct ingrowth of recipient vessels into the flap. New capillaries grow at a mean rate of 0.2 mm/day and can reach distances up to 2–5 mm. Immediately following flap transfer, a fibrin layer forms between the flap and adjacent tissue. Angiogenic growth factors are released, vascular endothelial cells dilate, and increased basement membrane permeability allows outgrowth of capillary loops for new capillary bed formation with preexisting flap vessels. This process is complete by 7 days.

Well-oxygenated blood directly correlates with skin flap survival, as local tissue oxygen tension influences angiogenesis, collagen deposition, and protection against infection. Active smokers have higher flap necrosis and complication rates than nonsmokers or ex-smokers.

Nicotine compromises wound healing by:

- Vasoconstrictive effects on cutaneous blood vessels
- Releases catecholamines from adrenal medulla➔ increase in heart rate, blood pressure, and *oxygen demand*
- Increases platelet adhesiveness
- Impairs collagen deposition via inhibition of fibroblast proliferation

Carbon Monoxide

- Binds to Hgb 200x more than O2➔ shifts oxyhemoglobin dissociation curve to left➔ cellular hypoxia

With any flap, impairment of arterial vascularization results in rapid flap loss (<24 h). Most common complication and reason for flap loss (free, regional, and random) is venous congestion. Congestion leads to stasis and clotting. Thrombus propagation ➔ microcirculation failure ➔ sludging ➔ ultimately causes irreversible cell death (the “no reflow” phenomenon). In free flaps, reversing this process should occur in less than 6 h to reliably reverse ischemia.

Several therapies exist to attempt reversal of venous stasis including:

- Aspirin (antiplatelet)
- Nitropaste (vasodilator)

- Leeches (*Hirudo medicinalis*)—suck blood and inject hirudin; need coverage for *Aeromonas hydrophila* (ciprofloxacin 500 mg q day)

Hyperbaric Oxygen

Improves flap viability of at-risk or ischemic flaps. One hundred percent oxygen is delivered at 2–2.5 atm of pressure.

- Increased dissolved plasma oxygen concentration
- Accelerated neovascularization.
- Increases blood flow, oxygen carrying ability, and delivery to the tissue
- Reduces oxidative stress secondary to increased free radical scavengers

Flap Delay

Flap delay: (1) *improves blood flow,* (2) *conditions the tissue to ischemia, and* (3) *closes AV shunts* by exploiting the angiosome to improve tissue viability and vascularity. Flap elevation results in local decreased perfusion pressure, denervation, and a relative ischemic state to the skin. When sympathetic nerves are divided, catecholamines are released and reuptake is impaired resulting in a temporary "hyperadrenergic state." Vasconstriction results in reduced total blood flow, increased free oxygen radial production, and inflammation. If perfusion pressure drops below critical closing pressure of the arterioles in the subdermal plexus, flap necrosis occurs. Return of blood flow begins by 12 hours and is maximum at 24 hours as norepinephrine is depleted. During this time, reperfusion injury is possible secondary to oxygen free radical production from the enzyme *xanthine oxidase*, which further places further stress on the flap.

Ischemia of the tissue results in increased longitudinal blood flow, development of vascular collaterals, and reorientation of vascular channels. The dilation of existing vessels at the level of the "choke" vessel during the delay is an important. The increase in caliber of the choke vessel is secondary to both hyperplasia and hypertrophy of the vessel and thought to be a permanent change following the delay period. Flap delay is typically performed 2 weeks prior to definitive transfer and has resulted in increased flap survival at the time of tissue transfer.

Tissue Expansion

A major reconstruction modality developed over the past 30 years, which is useful for a variety of reconstruction challenges. Tissue expansion has become a reliable method of reconstruction when soft tissue is of limited supply, such as in the scalp, pediatric patient, burn injury, and breast reconstruction.

Tissue expanders exploit the biomechanical properties of creep and stress relaxation.

- *Creep*—increase in skin length over time due to constant tension
- *Mechanical creep*—rapid, intraoperative tissue expansion due to disruption of elastin and realignment of collagen fibers to a parallel position, and displacement of interstitial fluid and ground substance. *No net increase* in surface area occurs or new skin formation.
- *Biologic creep*—long-term tissue expansion secondary to physiologic and histologic changes in the skin. *Net increase* in surface area occurs.
- *Stress relaxation* refers to a decrease in tension on the tissue over time after applying constant strain.
- *Advantages*: *similar skin color, texture and thickness, minimal donor-site morbidity, improved survival* versus *random-pattern flaps* (expanded flaps have enhanced survival rates like delayed flaps secondary to increased vascularity)
- *Disadvantages*: *need for multiple procedures, temporary cosmetic deformity, prolonged period of expansion*

Tissue Changes with Expansion

- *Epidermis* → thickens slightly
- *Dermis* → thins dramatically (30–50%)
- *Skeletal muscle atrophy* (but no functional alteration)
- *Fat atrophy*
- *Vascular*—increase in number and caliber of vessels (stimulus for vascular proliferation)

When choosing the size and shape of the tissue expander, the base of the expander should be *2.5–3x* the area of reconstruction and its shape should be based on the site of expansion. Rectangle expanders provide the greatest gain in surface area. The expander is placed in the subgaleal plane in the scalp and in the subcutaneous plane in the face and neck to reduce potential neurovascular injury.

Complications of Tissue Expansion

Can expect complications in up to *10%* of patients.

Major *infection (commonly around ear or in scalp), exposure/extrusion—most common (through incision or thinned intact skin), flap ischemia*

Minor *pain with expansion, seroma, hematoma, dryness/erythema/ hyperpigmentation of expanded skin, alopecia, stiffness, striae, neuropraxia*

Early infections may require premature expander removal. If antibiotics sufficiently control the infection, may complete expansion in a delayed fashion.

Local Flaps

Advancement Flaps

Flap moves in one, straight vector. Sliding flap in an area with increased skin laxity. Advancing border of the flap is immediately adjacent to margin of defect. *Random blood supply*. Two types of movement to consider to achieve optimal outcome and minimal surrounding tissue distortion. *Primary* movement refers to the sliding/advancement of the tissue toward the defect. *Secondary* movement refers to the displacement of the surrounding skin toward the center of the defect from wound closure tension. Secondary movement results in unfavorable distortion of surrounding tissue, e.g., vermillion, nostril, brow, eyelid, and lobule attachment Dissected in the subcutaneous plane with wide undermining minimizes secondary movement. May be *unipedicled* or *bipedicled*. *V-Y* (used in lengthening procedures) or Y → V. Unipedicled flaps are typically designed with a 1:3 defect-to-flap length. Burrow's triangles are frequently excised on either side of the flap to prevent standing cone deformities. Bipedicled flaps include O-T, A-T, and H-plasty and have the advantage of disturbing tension between two flaps, which lessens tissue distortion and increases perfusion

Rotational Flaps

Rotational flaps rotate immediate adjacent tissue skin into defect site. Ideal for triangular defects. The arc of rotation is typically less than 90° to the axis of the defect. Standing cone deformities develop at the base of rotational flaps (consider excising Burrow's triangle).

- The length of the *arc should be ~4× the width* of the base of the defect.
- Conversion of the defect to a *2*:*1* (*height*:*width*) triangular defect can reduce standing cone deformity.
- Point of greatest tension at distal border of the flap

More arc length → less tension. Inferiorly based flaps have better lymphatic drainage. Undermining around the base of the flap may facilitate more of an advancement movement, which will decrease wound closure tension. Rotational flaps are commonly used to repair large defects (cervicofacial advancement-rotation flap, Fig. 7.1) and scalp defects.

Cervicofacial Advancement Rotation Flap

Curvilinear flap is combination advancement-rotation flap utilized to reconstruct larger medial cheek defects taking advantage of cheek/jowl laxity. Scars are placed at the borders of the cheek subunit. The flap may be based anteriorly or posteriorly along the preauricular region. Unaffected skin must be resected to remove standing cone deformities, either at the melolabial crease or the temple.

- Extend flap to subciliary line of lower eyelid and affix the flap to the infraorbital rim periosteum.

- Extend the lateral brow incision *higher* than the palpebral fissure to support the eyelid.
- Typically elevated in subcutaneous plane although, large flaps may be elevated in the sub-SMAS plane for increased blood supply. Care must be taken with sub-SMAS elevation to avoid injury to the facial nerve.

Transposition Flaps

Transposition flaps are pivotal flaps based off of a random blood supply; only the base of the flap may be adjacent with the defect. Thus, the flap moves over normal tissue in order to inset into the defect. *Rhombic* and *bilobe* flaps are the most common.

Area of *greatest tension is at base of secondary donor site*. This is the most commonly used flap type in the face. The larger the angle of rotation, the more extensive the standing cutaneous deformity and the less length a flap can reach. Pivoting a flap at 45° reduces the length by 5%, 90° reduces flap length by 15%, and 180° by 40%. Prone to trap-door deformities if rounded (ex: bilobe).

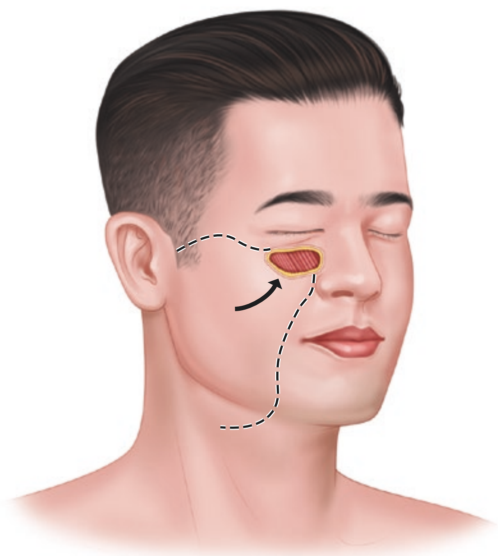


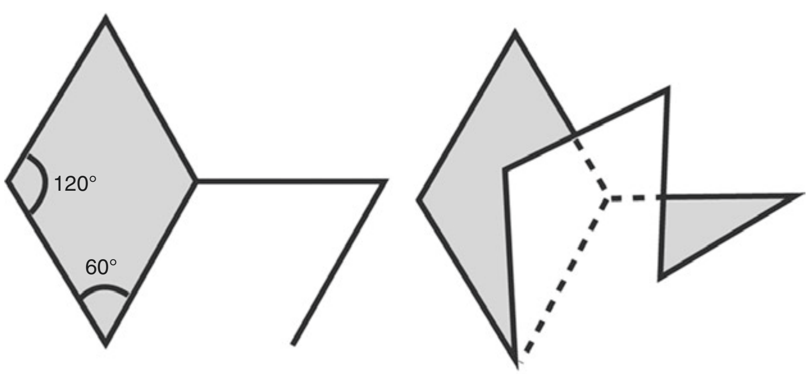
Fig. 7.1 Cervicofacial advancement-rotation flap designed to reconstruct a medial cheek defect

Rhombic Flap

The prototypical rhombic flap is transposed to reconstruct defects with internal angles of 60° and 120° and equal limb lengths—referred to as a *Limberg flap*. Conversion of a defect to this rhombic configuration is necessary which can lead to excessive amount of discarded normal tissue. Each defect has four possible flaps that can be created in order to take advantage of lines of maximum skin extensibility and placement in RSTLs. Standing cone deformities commonly occur at the medial base of the flap. Rhombic flaps can also be modified for circular defects (“note” flaps) or performed as bilateral opposing flaps to close larger defects.

- *Point of greatest tension* → at the point where the flap is designed extending from the defect and in the vector running parallel to this border of the defect (see Fig. 7.2).

Fig. 7.2 Limberg rhombic flap. Note the angles of design, 120° and 60°



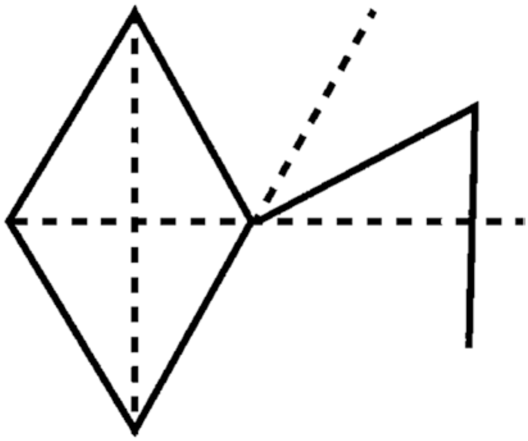


Fig. 7.3 Duformentel modification of a rhombic flap. The design places the border of the flap between a line drawn in the standard Limberg flap and a line drawn extending parallel from the defect border

Dufourmentel modification Modified design which allows for reconstruction of rhombus-shaped defects with more acute internal angles 60–90° (*more square shaped*).

This design places the border of the flap between a line drawn in the standard Limberg fashion and a line drawn extending parallel from the defect border (see Fig. 7.3). It pivots less than a traditional Limberg flap. This results in better distribution of closing tension and improved blood supply.

Webster modification Also known as a 30° rhombic flap, the Webster design calls for performing a W-plasty (*30° angle*) at the base of the defect instead of converting it to a 60° Limberg rhomboid. This limits the extent of the resection of normal tissue. The flap is then designed extending out from the defect at a similar bisected angle as the Duformentel, but with a triangular flap created by a *30° angle* (see Fig. 7.4). This smaller angled flap requires more secondary movement from the adjacent tissue to close the defect and therefore may disturb surrounding structures.

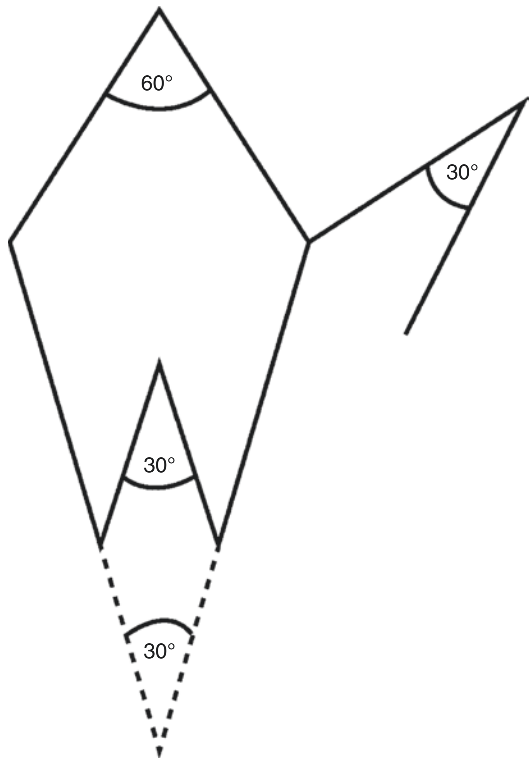


Fig. 7.4 Webster modification of a rhombic flap. A W-plasty is performed at the base of the defect, and a smaller angled flap is designed

Bilobe

The *bilobe flap* is a double-transposition flap with a single base based upon random or axial blood supply. The bilobe is advantageous because it recruits skin that is not immediately adjacent to the defect. The vector of tension is also moved perpendicular, which can avoid distortion of adjacent structures (such as elevation of the nasal ala). Bilobe flaps work well for caudal 1/3 nasal defects and nasal tip defects less than or equal to 1.5–2.0 cm. Curvilinear incisions are *prone to trap door deformities*. *Pincushioning* may be lessened by designing the flap with angular corners; postoperatively, it may be improved with steroid injections and/or scar revision.

- *Zitelli's modification*: Changed the total arc of rotation from 180° to 90°; thus each lobe moves 45°. This reduces tension and tendency for standing cone deformity.

Bilobe flap design: The classic small bilobe flap is used to repair a circular defect. A pivot point for the first flap is determined where the

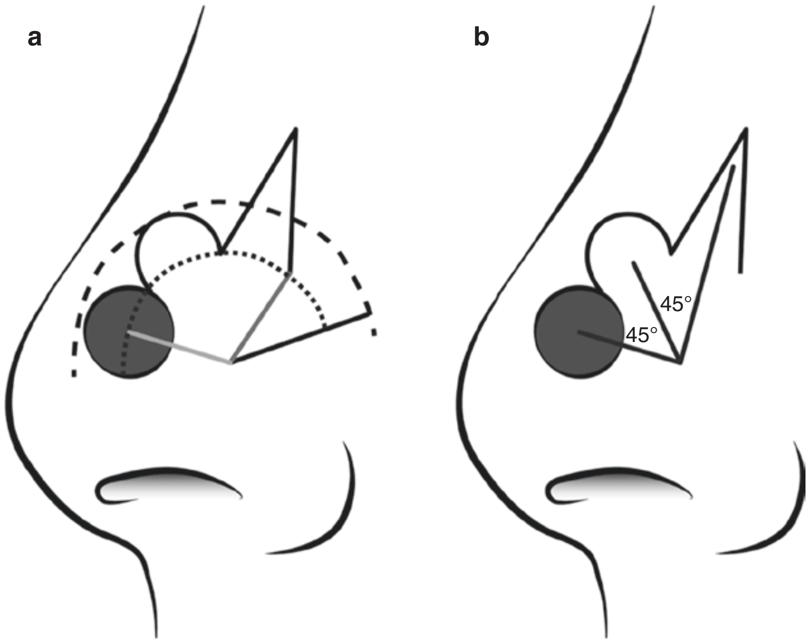


Fig. 7.5 **(a–b)** Bilobed transposition flap for nasal reconstruction. Each lobe is placed at 45° creating a 90° total arc of tissue movement

laxity and vectors of tension will be ideal. The second lobe is often designed to place the scar at the border of a subunit. The pivot point is placed from the defect edge at a distance of at least half of the defect diameter (see Fig. 7.5). The impending standing cone is removed as a triangle at the base of the circle.

At 90° to the pivot point, two semicircular arcs are created. A suture placed through the pivot point is particularly helpful during design.

- *First arc* is based upon length: pivot point → far edge of defect
- *Second arc* is based upon length: pivot point → center of defect

The first lobe flap extends to the top arc and the flap is as wide as the defect. The second lobe is twice as tall as the first, but half as wide and is more triangular in shape (see Fig. 7.5). For nasal reconstruction flap elevation occurs in the subnasal-SMAS plane. Wide undermining is crucial to ensure appropriate mobilization and minimal tension at closure. The second lobe donor site is closed first, followed by inset of the first lobe into the primary defect, before finalizing the inset and closure of the first lobe donor site.

Interpolated Flaps

Interpolated flaps pass over intact skin (noncontiguous donor site) to cover a defect. These are often axial blood supply flaps but can be random with the most notable of these the paramedian forehead flap and the nasolabial flap.

Paramedian Forehead Flap Flap based off the supratrochlear vascular bundle. This is the workhorse for any nasal defect and is capable of reconstructing the entire external or internal lining. In general, paramedian forehead flaps are used for *nasal defects >2 cm*. Preoperatively assess forehead height → flap can be extended obliquely to avoid hair-bearing skin (however, lateral extensions wider than 3 cm may elevate brow), or hair follicles can be cauterized or removed.

- Supratrochlear pedicle is found *1.7–2.2 cm from midline*.
- Three-dimensional templates will assist in flap design. Careful assessment of rotational forces on pedicle and ensuring sufficient pedicle length is key.
- Artery runs under the orbicularis oculi, pierces the corrugator, and passes through the frontalis muscle from deep to superficial

- Pedicle base widths around *1.2–1.5 cm* allow for better flap mobility and geometry.
- Extending incisions below brow and division of the corrugator muscles will allow for further length if needed.
- The distal 2 cm of the flap can be thinned of muscle and subcutaneous fat for better contouring.
- *Subperiosteal dissection* is usually performed above the orbital rim in order to avoid pedicle injury.
- The donor site is closed primarily with bilateral advancement flaps or left for secondary intention healing (usually if >4 cm).
- Pedicle division and inset typically performed after 3–4 weeks or longer in cases of smokers, poorly controlled diabetics, and vasculopathies.
- Can be performed as single-, two-, or three-stage procedure.

Melolabial (or nasolabial) flaps designed as interpolation flaps are commonly used for nasal alar and columellar reconstruction. The flap is based off supply from distal facial and angular arteries into the subdermal plexus and fibro-adipose layer. Therefore, it can be based superiorly or inferiorly depending upon the reconstructive requirements. The flap donor site places the scar in the melolabial crease.

- Reconstruct the entire ala by completing the subunit resection, but *leave 1–2 mm of lateral alar tissue* at the nasofacial junction to preserve the alar-facial sulcus
- An exact template is made of the defect.
- Auricular cartilage graft often needed for external valve support and to contour the overlying flap.
- Flap pedicle takedown and inset at 3–4 weeks postoperatively.

Island Flaps

Type of interpolation flap in which a skin bridge is de-epithealialized and tunneled under and intact skin bridge. Island flaps rely on blood supply through the subcutaneous tissue as the cutaneous circumference of the flap is incised. These flaps are best suited to areas where there is abundant fat and subcutaneous tissue—allowing for creation of subcutaneous pedicle for flap mobility. The cheek and upper lip are common locations where these flaps are employed..

Reconstructive Options Based Upon Location

Optimal reconstruction of facial defects is dependent upon several factors including the size and depth of the defect, aesthetic subunits, disease process, and underlying patient morbidities. In general, the *aesthetic regions* and their individual subunits should guide reconstruction—using similar skin quality and placing scars in the aesthetic borders. Placing incisions parallel to *relaxed skin tissue lines* will minimize visibility of scars and reduce wound tension. *Secondary tissue movement* is the displacement of the surrounding tissue in response to the closure of the primary defect, and may distort adjacent structures such as eyelids, lips, and nostril position.

Nasal Reconstruction

Reconstruction of the nose presents several challenges as it has a very specific three-dimensional structure that extends off the surface of the face and is formed from multiple distinct layers. The principle of replacing “like with like” (skin, cartilage, mucosa) is particularly applicable to nasal reconstruction. The nose is divided into nine subunits: tip, columella, dorsum, paired sidewalls, two alae, and two soft-tissue triangles. The aesthetic subunits of the nose form highlights and shadows between natural transition zones and should be considered individually when assessing the defect. *When >50% of the subunit is involved, it is better to resect the remaining subunit and repair the entire area as a whole*. When multiple subunits are involved, a combination of flaps may be required in order to optimally reconstruct the individual subunits.

Reconstruction of Nasal Skin

The skin of the nose is thick, rigid, and sebaceous over the nasal tip and alae as compared to the dorsum and sidewalls where the skin is thinner and more mobile. Incisions in subunit boundaries or in the midline heal with less perceptible scars.

Defect-Based Approaches: (Table 7.1)

Dorsum

Skin only defects can be repaired with primary closure, FTSG, or local flaps. Primary closure can be performed vertically or horizontally as long as no distortion occurs of the tip or ala. Closure can be facilitated by dorsum hump removal in the setting of a prominent osseocartilaginous hump.

- *Bilobed flap*—may be bilateral to split the tension
- *Single/double transposition flap*
- *Sliding dorsal nasal flap* (*Rieger flap*)—for larger defects, can reach to the supratip region (Fig. 7.6). Rotation flap based on branches of angular artery for single-stage reconstruction of <2.5 cm defects of the tip, sidewall, and dorsum.

Nasal Sidewall

Secondary intention can be appropriate for defects adjacent to the medial canthus in areas of concavity. Primary closure and FTSG are other options for defects <1.0 cm. Larger defects may require glabellar flap reconstruction.

- *Transposition flap*
- *Superior based melolabial flap*
- *Paramedian forehead flap*

Tip

The skin of the tip is thick, sebaceous, and tightly adherent to the underlying cartilage framework. Small defects can be repaired with FTSG with acceptable cosmesis. Grafts should be chosen to match nasal skin thickness, color, and texture as close as possible to the nasal skin. Deeper defect repair can be delayed 2–3 weeks to allow granulation for optimal contouring.

Table 7.1 General considerations to guide reconstruction of nasal defect based upon size

Defect size	Nasal tip	Nasal ala	Nasal dorsum
<1 cm	FTSG Bilobe	FTSG Bilobe	Bilobe Transposition flaps Advancement flap
1–1.5 cm	Bilobe	V-Y (at alar groove) Melolabial interpolation	Bilobe Transposition flaps Advancement flap
>1.5 cm	Paramedian forehead	Paramedian forehead	Dorsal nasal (up to 2.5 cm) Paramedian forehead

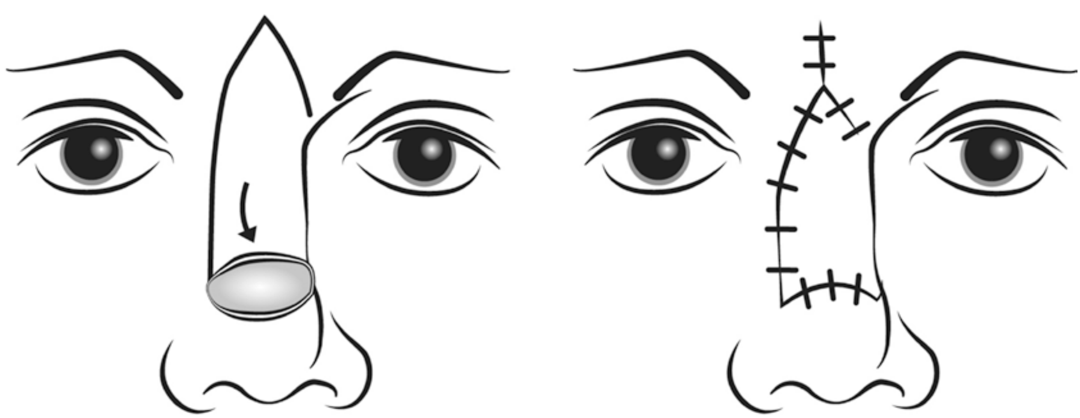


Fig. 7.6 Sliding dorsal nasal flap. This flap makes use of laxity in the glabellar region, and is best for dorsal defects supra-tip and upwards

- *Skin graft*—full-thickness or perichondrial composite graft from conchal bowl. Dermabrasion can improve final appearance.
- *Bilobed transposition flap*—distributes the tension horizontally to avoid rotation of the tip
- *Melolabial flap*
- *Paramedian forehead flap*-best for medium to large (>2 cm) defects

Ala

Small defects <1.0 cm of the central ala can be left to heal by secondary intention or FTSG reconstruction. The remaining defects are repaired with local flaps and cartilage grafting as determined by the defect. Full-thickness defects can be repaired using the contralateral ala as a template. Auricular or septal cartilage graft is placed along the inferior aspect of the margin to prevent alar retraction. Preservation of the alar base is recommended as it is very difficult to recreate without risks of blunting.

- *FTSG*
- *Bilobe transposition flap*
- *Melolabial interpolation flap*
- *Paramedian forehead flap*

Columella

Can be reconstructed with flaps or grafts. Skin-only defects can be left to heal by secondary intention or FTSG from the auricle. With loss of the medial crura, composite auricular grafts or a columellar struts with soft tissue covering is necessary to maintain postoperative tip support.

- *Full-thickness helical rim composite graft*
- *Double-upper lip transposition flaps*
- *Melolabial interpolation flap*
- *Paramedian forehead flap*— Can extend vertical incision into the hairline for increased flap reach.

Bone and Cartilage Reconstruction

Lateral defects can be repaired with free septal flaps, bony perpendicular plate of ethmoid, free conchal grafts, or septal hinge flaps. Midline cartilaginous defects can be reconstructed with cantilever bone grafts (secured to the radix), dorsal onlay grafts, chondromucosal composite septal flaps, and/or costal-chondral grafts.

Intranasal Mucosal Lining Flaps

Intranasal mucosal flaps offer thin, supple, high vascular tissue which can be utilized with primary cartilage grafts for internal reconstruction of a full-thickness nasal defect. The main limitation with these grafts is their limited dimension, therefore, they are most useful for smaller defects. As the defect size increases, distal perfusion is decreased which can result in distal flap necrosis, stenosis and disruption of the native intranasal anatomy.

Bipedicled Mucosal Advancement flaps (Bucket Handle Flap)

- *Alar margin/vestibular defects up to 1.5 cm* → Blood supply from septum and alar base. Intercartilaginous incision is made and flap advanced inferiorly. Donor site repaired with skin graft or contralateral septal flap.

Ipsilateral Septal Mucoperichondrial Flap (Hinge Flap)

- *Lower nasal vault defects* → Blood supply is derived from a septal branch originating off the superior labial artery. Therefore, 1 cm of mucosa should remain attached to the caudal septum at the hinge point. Flap can be as large as 3 × 4 cm.

Contralateral Septal Mucoperichondrial Hinge Flap

- *Ala and sidewall defects* → Blood supply is based on the superior ethmoid vessels. An ipsilateral mucosal flap hinged caudally is formed first and used for alar reconstruction. Septal cartilage is resected or a window is created and the contralateral mucoperichondrial flap is used to reconstruct the contralateral sidewall mucosal defect. The septal cartilage can be used as a nonanatomic support graft for the lateral sidewall reconstruction.

Septal Chondromucosal Composite Flap (Pivot Flap)

- *Tip or dorsal mucosal defect/subtotal central defect/total nasal defect*→
- Full-thickness mucosa, mucoperichondrium, and cartilage incisions are made to create a rectangular or trapezoidal shaped flap which is hinged caudally. The composite flap is pivoted forward and the cartilage is secured in place while the mucosa can be reflected laterally to line the nostrils. Often a wedge of cartilage only is removed at the posterior septal angle to facilitate in flap rotation. The degree of rotation depends upon the necessary regions for reconstruction. For significant movements an initial delay procedure with mucosal cuts only is sometimes employed. Septal lining care line upper and midvault but not frequently long enough for complete internal lining).

Other Options for Intranasal Lining

- *Composite Chrondrocutaneous Grafts*—useful for small full-thickness nasal reconstruction, such as for the soft triangle or alar rim. Prone to graft loss. General recommendations are for grafts *no larger than 1 cm*^{2}. Postoperative cooling with ice water may improve flap survival. Consider placing "windows" in the cartilage to allow for neovascularization to the cutaneous portion
- *Inferior turbinate mucosal flaps, pedicled anteriorly*—requires second-stage takedown
- *Folded paramedian forehead flap for alar mucosal reconstruction*
- *Pre-laminated forehead flap formed with skin graft prior to nasal inset*.
- *Radial forearm free flap*- total internal lining defect

Total Nasal Reconstruction

A multilayered reconstruction is required, to include intranasal lining, structural architecture, and overlying skin cover. Traditionally this has been accomplished with a *radial forearm fasciocutaneous free flap* to provide the intranasal lining. In successive stages, the free flap is thinned and sculpted around a rib cartilage framework (which may incorporate calvarial bone grafts), followed by a paramedian forehead flap. Double-paramedian forehead flaps have also been utilized—one for the intranasal lining and another for the overlying skin. Pre-laminated constructs built on the forehead prior to turning the forehead flap down are another option. Many elderly or cancer patients may instead opt for a *nasal prosthesis*.

Cheek Reconstruction

The cheek is the largest facial aesthetic subunit. Its convex curvature highlights even subtle imperfections and scars. It can be further subdivided into medial, zygomatic, lateral, and buccal subunits. The skin of the medial and buccal subunit is thick and freely mobile, unlike the zygomatic and lateral subunits which have firm attachments to the underlying fascia (zygomatic cutaneous ligament, a.k.a. "McGregor's patch" and parotidomasseteric ligaments). Incisions are best placed along boundaries of the aesthetic subunits when possible. Care must be taken to avoid distortion of landmarks (eyelid, lip, nasal ala) from wound closure tension. When cheek–nose deformities area encountered, separate techniques should be used to avoid blunting of the nasofacial groove. Dermal-periosteal sutures are needed to prevent blunting at the junction of the subunits. When underlying bony deficiencies are present (partial maxillecomties), split calvarial bone grafts and reconstruction plates are needed for maintenance of postoperative contours.

Medial Cheek

Medial cheek lesions can frequently be closed directly along the medial boundary of the cheek subunit (cheek–nose junction or melolabial crease) via an advancement flap or utilizing an island pedicle flap. Deep anchoring sutures to the underlying periosteum minimize skin tension. A small intervening skin excision is sometimes needed to facilitate placement along the subunit boundary. For defects too large for direct closure or simple advancement flaps, cervicofacial advancement flaps provide sufficient recruitment of tissue for closure

Lateral Cheek

Most lateral cheek lesions can be closed primarily or with an advancement flap similar to a rhytidectomy skin flap. Larger defects may require transposition flaps or cervicofacial rotation advancement flap repair.

Buccal Subunit

Central cheek defects many can be closed primarily along RSTL after undermining or with transposition flaps (rhomboid, bilobe, note flap). Elevation is deep to the subdermal plexus. Larger superficial defects can be closed with cervicofacial advancement flaps, while full-thickness defects require more complex reconstruction with regional or free flaps.

Zygomatic Subunit

Careful elevation in the subcutaneous plane is necessary to avoid injury to the frontal branch of the facial nerve in this region. Transposition flaps are good options for small to large defects and can be designed to reduce vertical tension upon the eyelid and canthus. Suspension sutures are used when defects approach the eyelid to avoid ectropion. For larger defects, cheek rotation-advancement flaps can provide coverage.

Eyelid Reconstruction

Eyelid reconstruction presents several challenges, as all aspects and layers of the lid must be carefully and precisely reconstructed in order to protect the eye. Reconstructive strategies are defined by the size of the defect, as well as by which lamellae of the eyelid are involved, and the location of these defects (canthal, lid margin, upper versus lower eyelid). The upper and lower eyelid is divided into superficial and deep layers based upon the anterior lamella (skin and orbicularis oculi) and posterior lamella (conjunctiva, tarsus, and retractors). Intraoperative recognition of excessive tension causing eyelid malposition is imperative to prevent lagophthalmos and ectropion. The surgeon must be prepared to adjust tension at the lateral canthal tendon via a tarsal strip procedure (or apply Frost sutures) as needed during the operation.

Superficial Defects Not Involving the Lid Margin

Small defects (<0.5 cm) of the upper and lower eyelid which do not involve the lid margin may be amenable to heal by secondary intention. For defects <50% anterior lamella, rectangular advancement flaps can be utilized. Upper eyelid defects should be oriented parallel to the natural eyelid crease. Horizontally placed elliptical incisions can be employed in areas of excess skin redundancy (such as in the setting of upper lid dermatochalasis).

For lower eyelid defects, direct closure may be possible in small defects; however, orientation of elliptical excisions on the eyelids should often be oriented vertically (perpendicular to the RSTLs) in order to prevent tension retracting the lid margin. Rotation skin-muscle flaps, FTSG, and laterally based upper eyelid skin transposition flaps are useful for defects >0.5 cm. Skin grafts can be harvested from the contralateral eyelid or postauricular skin. A blepharoplasty may need to be performed on the opposite eye to maintain symmetry, unless there is a concern for future skin grafting needs.

Full-Thickness Defects

Full-thickness defects must be reconstructed in multiple layers for optimal function and appearance. Careful approximation of the *gray line* is a priority in order to avoid notching of the lid margin. Small defects, usually up to 1/3rd of the lid margin, may be repaired with direct closure. Usually this entails transforming the defect into a pentagonal shape in order to remove the eventual standing cone deformity. The tarsus must be sutured to an exact vertical alignment. Vertical mattress suture at the margin with slight eversion is recommended. Larger defects up to 50% upper lid and 75% lower lid with intact lateral canthus can be repaired with semicircular advancement flaps and tarsoconjunctival flaps. When a combined cheek/lower eyelid defect is present, the anterior lamella is best repaired with a cheek rotation-advancement flap and a tarsal-conjunctival or nasal-septal mucochondrial composite graft is used for poster lamella reconstruction. Other options for conjunctival reconstruction include buccal mucosal or hard plate grafts.

<25%	Direct closure
25–50%	Direct closure with lateral cantholysis
25–50%	Tarsal rotation flap and skin-muscle flap or skin graft
25–75%	Tarsoconjunctival graft and skin-muscle flap
33–66%	Tenzel semicircular flap with periosteal flap
50–100%	Cutler-Beard flap

Tenzel Semicircular Flap

Defects 1/3rd–2/3rd of the Horizontal Length of Eyelid

The Tenzel semicircular flap makes use of redundancy of lateral canthal skin laxity. Lateral canthotomy and cantholysis are performed. The flap is undermined in the submuscular plane and rotated to the lateral border of the defect and is sutured primarily. The lateral canthal angle must be recreated at the lateral orbital rim. For larger defects where there is minimal tarsal plate remaining, consider placing an ear cartilage graft for structural support.

Hughes Tarsoconjunctival Flap

Defects 50–100% of the *LOWER* Eyelid

This two-stage flap reconstructs the posterior lamella with a superiorly pedicled tarsus and conjunctiva hinge flap (Fig. 7.7). The anterior lamellar skin is reconstructed either with full-thickness skin grafts or a local flap. The flap is divided and inset in approximately 4 weeks.

Cutler-Beard Bridge Flap

Large Defects (>50%) of the *UPPER* Eyelid

With this procedure, a muscle and skin advancement flap from the lower eyelid is advanced underneath the “bridge” of intact lower eyelid tarsal plate and lid margin. Therefore a graft for the upper eyelid posterior lamella is commonly employed (hard palate mucosa, free tarsoconjunctival graft from contralateral eyelid). The flap is divided and inset approximately 4 weeks later.

Medial Canthal Defects

The lacrimal drainage system and medial canthal tendon should be inspected to ensure integrity. Lacrimal intubation with Silastic stents for 2–6 months or endoscopic DCR at the time of repair can be performed to ensure long-term patency of the drainage system. If the medial canthal tendon has been damaged, repair should be performed to restore the posterior limb to the lacrimal crest to ensure proper lid apposition to the globe. When a small skin-only defect is present, FTSG, primary closure, or healing by secondary intention are options for reconstruction. The natural concavity of the medial canthus predisposes this area to webbing, therefore, linear closures should be avoided in this area. Considerations for closure include z-plasty or transposition flaps. For larger defects, FTSG, rotation flaps, glabellar flaps, and/or paramedian forehead flap repair is indicated.

Lip Reconstruction

The upper and lower lips constitute the focal point of the lower face. The upper lip has two lateral subunits, a central subunit, vermillion border, red lip, and cupids bow. The subunits of the lower lip include the red lip, white lip, vermillion, and labiomental crease. Reconstructive goals include maintenance of oral competence, adequate stomal access, maintenance of mobility, and alignment of anatomical subunits. An adequate stomal opening with neuromuscular movement and sensation is important for oral competence. Proper lip contour is achieved with precise reconstruction of the vermillion border, and a layered closure of skin, muscle, and mucosa.

Vermillion-Only Defect

- *Mucosal advancement flaps* → Both small and large vermillion defects can be covered by utilizing red mucosa from the oral lip. The new lip may be darker in color than the native vermillion. The flap is raised deep to the salivary glands, but over the surface of the orbicularis

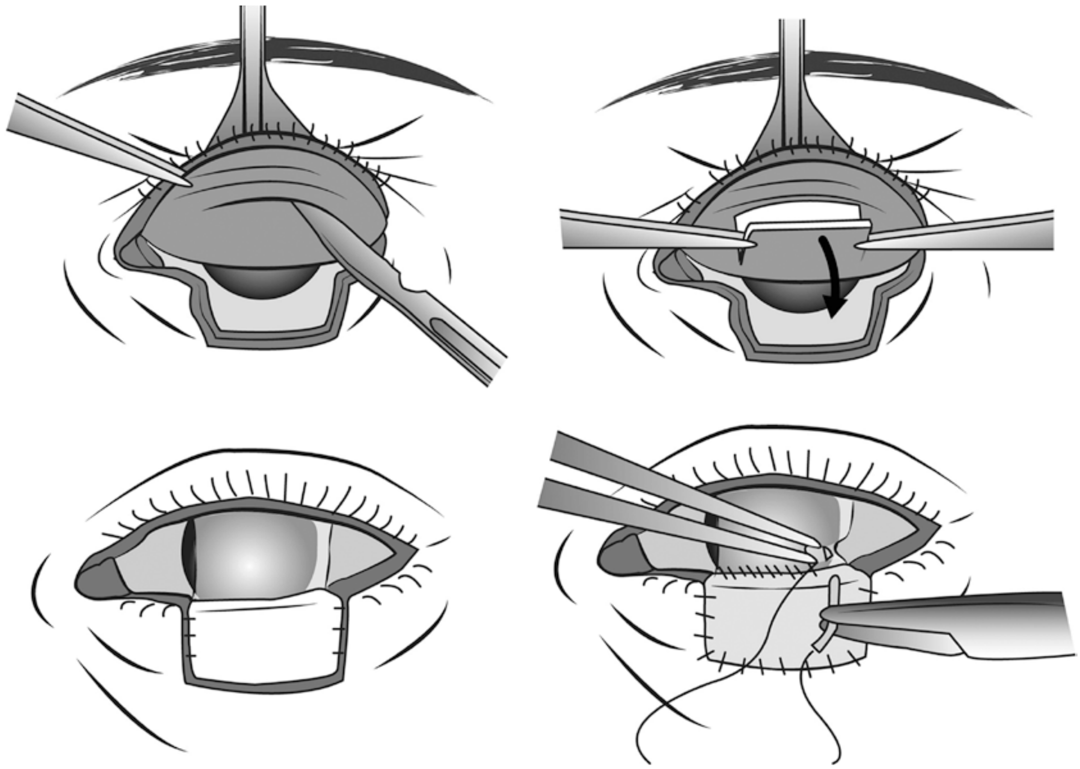


Fig. 7.7 A superiorly pedicled Hughes tarsoconjunctival flap is inset into the lower eyelid defect while its outer surface is skin grafted. The pedicle is divided at around 4 weeks

muscle. When local advancement is not possible, a pedicled flap from the ventral tongue can be utilized with pedicle division 10–14 days later.

- *Mucosal V-Y flap* → Small-volume deficiencies can be addressed with a V-shaped intraoral incision. An island flap is formed and moved in a vertical direction. Y-closure results in bulking of the tissue at the end of the incision.
- *Mucosal cross-lip* → For wider defects with volume loss, a cross-lip vermillion flap can be placed and the pedicle divided in 3–4 weeks' time. The flap is raised over the surface of the orbicularis muscle. Unfortunately this procedure requires the lips to be tethered together.

Cutaneous-Only Defects

Small defects of the white lip can be closed with vertical primary closure or with local skin flaps.

- *A-T Flaps*- useful for defects adjacent to labiomental crease, vermillion border
- *Transposition flaps*—work well for lateral defects using skin recruited from the melolabial region.
- *Rotational flaps*—consider for larger defects of the upper lip under the alar base. The arc of the flap is designed to follow the normal melolabial crease.
- *Advancement flaps*-bilateral flaps useful for central lower lip defects. For upper lip defects, incisions can be placed at philtral ridge, alar base, and vermillion.

Full-Thickness Defects

Full-thickness defects of the upper and lower lip are approached differently. The lower lip has more tissue elasticity and primary closure can often be performed even with defects involving up to 1/3 of the lower lip. Full-thickness upper lip defects are best repaired with entire subunit repair.

Up to 33% of the lip → wedge resection with primary closure

33–50% → cross-lip (Abbe or Estlander) flap, bilateral advancement flap

>50% → Karapandzic, Gillies, Bernard-Burrow, regional/free flap

Wedge Resection

Care should be taken to reapproximate the vermillion exactly. The V-excision should not cross the labiomental crease. Consider a *W-plasty* at the distal end of the excision to avoid crossing the mental crease. Three-layer wound closure of the mucosa, muscle, and skin is necessary.

Stair-Step Lower Lip Advancement Flap

For larger lower lip defects approaching the size recommended for a cross-lip flap, a stair-step flap is a good option to close the defect in one stage (Fig. 7.8). It also has the advantage of preserving the central chin subunit as incisions are placed outside of the mental crease.

Cross-Lip Flaps (Abbe-Sabatini and Estlander)

- Vertical height of flap = vertical height of defect
- Width of flap = ½ of defect width
- Labial artery typically courses *between the intraoral mucosa and the orbicularis muscle* at approximately the level of the vermillion border

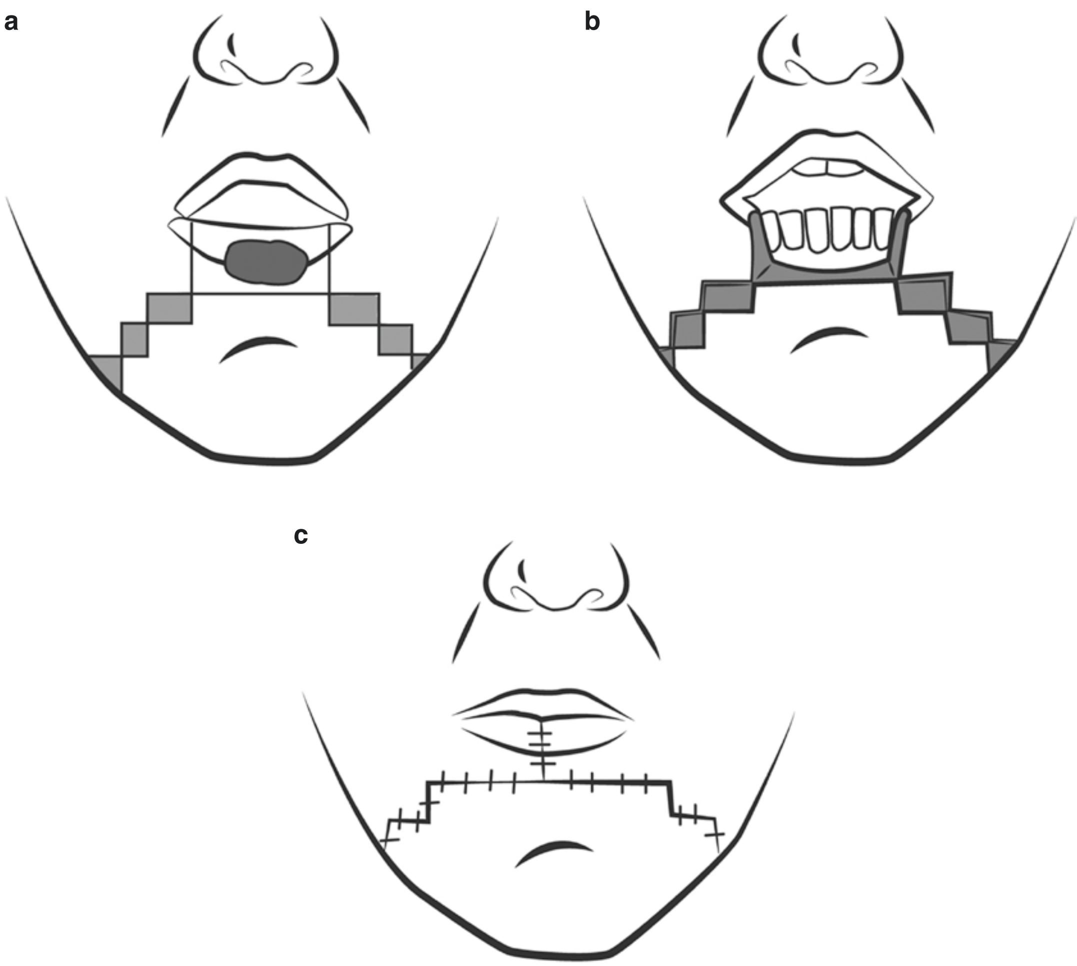


Fig. 7.8 (**a**–**b**) Stair-step flap lower lip reconstruction. This preserves the central subunit

The *Abbe cross-lip flap* is a two-staged, full-thickness flap based upon the labial artery. The arterial supply to the flap can be pedicled medially or laterally, depending upon the ease of flap rotation. Consider basing the pedicle on the side which will result in less limitation to mouth opening during the time between stages. Larger central lower lip defects can also be reconstructed with double-Abbe flaps based on either side of the philtrum.

Estlander flaps are similar flaps used to reconstruct a lateral lip defect and are performed in one stage. However, Estlander flaps have a tendency to result in blunting of the commissure that may require a commissuroplasty.

Karapandzic Flap

Larger lip defects over 50% of the lip can be reconstructed with Karapandzic flaps. The incisions are designed to follow the curvatures of the melolabial creases and can be designed with asymmetric lengths for defects which are not centrally located. The skin and intraoral mucosa are incised while leaving the central muscular layer intact to allow for dynamic oral closure.

- Advantage: motor/sensory nerve preservation
- Disadvantage: *Microstomia* is a common result with defects involving >2/3 lower lip. Stretching appliances may be of benefit postoperatively.

Gilles Fan Flap

For similarly large defects, a "fan" flap can be utilized to recruit from the laxity around the lips (Fig. 7.9). This is a *orbicularis oris myocutaneous flap* with its pedicle based around the commissure. A Z-plasty at the lateral aspect of the flap is often utilized to aid in flap movement and inset. Nakajima modified this flap to dissect and preserve the neurovascular supply from below in order to innervate the flap.

- Advantage: defect repair up to 80% upper/lower lip
- Disadvantage: delayed muscle function. Possible oral commissure blunting.

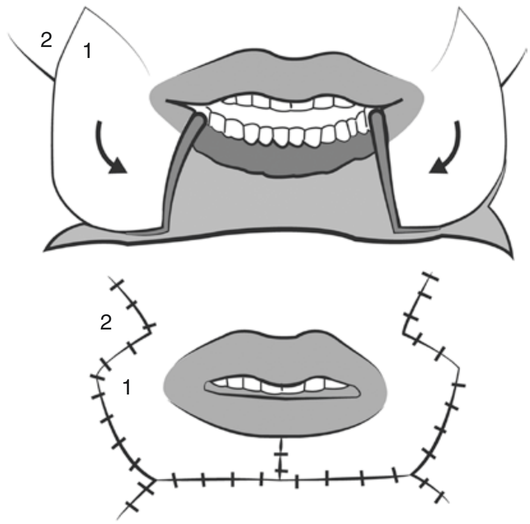


Fig. 7.9 Gillies fan flap design for a lower lip defect. This flap is best for larger defects

Civelek B, et al. *2006, Otolaryngol Head Neck Surg*: Postoperative EMG findings indicate that neurotization of non-innervated flaps (Gillies) occurs and that the lip ultimately may work as well as an innervated flap (Karapandzic or Nakajima).

Bernard-Burrow (and Webster Modification)

The Bernard-Burrow is a classic flap which employs *bilateral cheek advancement flaps* facilitated by excising "Burrow's triangles" of skin (Fig. 7.10). *Webster* modified this design to remove skin-only triangles along the melolabial folds and paramental regions, which better preserves the subunits and preserves some motor function.

- Advantage: repair central lower lip defects involving >2/3 lip
- Disadvantage: nonanatomic replacement of lower lip tissue (lacks functioning orbicularis oris) increased tension at closure susceptible to breakdown

Total Lip Reconstruction with Free Flap

Microvascular reconstruction provides the advantage of dual-layer reconstruction (and possibly

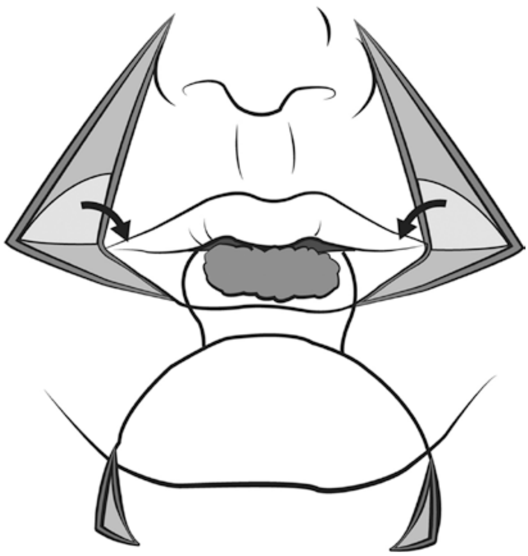


Fig. 7.10 Bernard-Burrow lip reconstruction. Triangles are removed at the melolabial and paramental regions to facilitate flap mobilization and inset

bony reconstruction) of large defects, but at the cost of a large adynamic segment that is often hypopigmented in comparison to the surrounding skin. Radial forearm flap folded over a *palmaris tendon sling* is a well-described technique for reconstructing the entire lower lip.

Forehead and Temple

Closure of forehead defects should maintain the hairline without distortion or paralysis of the brow. RSTL are horizontally oriented along the central forehead. Care is taken when raising flaps to avoid damage to the underlying sensory and motor nerves. Skin grafts typically avoided due to poor color and thickness match, but can be used temporarily in the setting of unknown margins or high risk of recurrence. If periosteum is present, healing by secondary intention is possible.

Midline and Paramedian: Recruit horizontal forehead laxity

- Primary closure—vertically oriented, ± W-plasty
- Rotational flaps along hairline
- Bilateral advancement flaps—along RSTL, avoids eyebrow distortion

Lateral Forehead: Avoid brow elevation, utilize RSTLs

- Horizontally oriented closure with W-plasties at each end—useful for small defects or horizontal defects
- Horizontally oriented unilateral advancement flap or H-plasty—defects close to brow
- A-T closure

Temple

- Rotational flaps
- Rhombic flaps

Scalp

The scalp consists of five layers: skin, subcutaneous fat, galea aponeurosis, loose connective tissue, and pericranium. Blood supply to the scalp is abundant and arranged in a radial pattern. The scalp receives contributions from the supratrochlear, supraorbital, superficial temporal, postauricular, and occipital artery which allows for the design of numerous random pattern flaps. Bone exposure frequently limits the application of skin grafts. However, healing by secondary intention can heal quite well if there is intact pericranium. Tight attachments between the galea and scalp skin significantly reduce tissue mobility. This makes closure of even small defects challenging. Rotation flaps are therefore the main workhorse for scalp defects. *Galeal relaxing incisions* can provide a modest stretching of the tissue. These incisions are made *perpendicular* to the direction of closure and are spaced approximately 2 cm apart. When insufficient tissue is available for reconstruction, tissue expansion or free flap reconstruction is necessary for closure

- Less than 3 cm defect → primary closure with wide undermining
- Greater than 3 cm defect → flap reconstruction or graft

Useful Flaps

- *Rotational Flap* (*Large*) → length of the flap border is 6× (instead of 4×) the defect width, back-cut often necessary. Secondary defect allowed to granulate or covered with skin graft

- *O to S* → useful for large defects (up to 9–10 cm, but best for around 5 cm), ideal for vertex defects
- *Orticochea flaps* → advantage is use of hair-bearing skin and the ability to restore the hairline for very large defects. Utilizes 2–4 individual transposition flaps, essentially elevating and mobilizing the entire scalp

Alternatives

Tissue Expansion Best if performed prior to removal of lesion. Expander shape chosen depending upon defect shape. Expander base should be *2.5x size of defect.* Placed *between galea and pericranium.* Inflation begins 2 weeks after placement and continued weekly until circumference of tissue 2-3x size of defect. Expansion should continue until patient experiences discomfort or blanching is noted.

Temporoparietal Fascia Flap Can be raised with overlying skin to provide hair-bearing reconstruction. Purely fascial flaps can be used to cover exposed bone with skin grafts applied to the vascularized fascia. TPFFs can be harvested as large as 14 × 17 cm. The vascular pedicle can be variable; therefore, may use a Doppler to map out the pedicle in advance of elevation.

- Disadvantages: tedious dissection (flap must be carefully separated from subcutaneous layer), temporary vs. permanent alopecia in area of scalp incision and flap elevation)

Allografts For large areas (10 cm or greater) of exposed calvarial bone, dermal regeneration templates (Integra) can be applied directly to the bone to create a recipient wound bed for delayed skin grafting (3–4 weeks). Burr the outer table of the calvarium to expose diploic blood supply prior to placing dermal regeneration template.

Free Flaps Latissimus dorsi free flap provides a thin sheet of muscle that approximates the thickness of all layers of the scalp. Skin grafts are used to cover the muscle. *Can cover defects up to 20 × 40 cm in size.*

Auricular Reconstruction

The multiple convexities and concavities of the elastic cartilage framework and thin, inelastic skin envelope make auricular reconstruction one of the most difficult challenges in facial reconstruction. All reconstructive attempts should be taken to preserve the size, location, orientation, and contours of the auricle. The concave surface areas of the ear (conchal bowl, cymba cartilage) are less critical to the overall appearance. Therefore, skin-only defects in this area can often be left to heal by secondary intention or with FTSG with acceptable cosmetic results.

Conchal Bowl Reconstruction

Cutaneous reconstruction alone results in successful aesthetic outcomes. Absence of conchal bowl cartilage is inconsequential.

- *FTSG*—intact posterior framework with or without cavum cartilage.
- *Postauricular skin flap*— two-stage island pedicle flap to reconstruction anterior concha; first-stage island pedicle flap from postauricular skin used to repair anterior defect and second-stage flap division with primary closure and FTSG repair of donor site.
- *Postauricular myocutaneous flap*—single-stage transposition skin-muscle flap
- *"Revolving door" postauricular flap*—single-stage flap utilizing postauricular skin as an island pedicle for anterior conchal reconstruction. The remaining conchal bowl is repaired with a postauricular skin flap.

Helical Rim Reconstruction

Goal is for restoration of convexity and soft transitions. Care should be taken to avoid "notching" of helical rim to avoid unnatural contour highlights.

- *FTSG*—skin-only defects with intact perichodrium
- *Helical rim advancement*—single-staged repair for <2 cm defects utilizing the adjacent helix and skin. May need to remove small amount of adjacent cartilage for optimal contouring

- *Tubed postauricular pedicle flap*—two-staged procedure for larger defects (2-4 cm).A postauricular bipedicled skin flap raised to contour the anterior helical deformity. Often combined with contralateral conchal cartilage graft for optimal contour.

Full-Thickness Helical Rim and Antihelix Reconstruction

- *Primary closure*— <25% auricle defects; star-shaped excision necessary to prevent cartilage bulking and wound closure tension
- *Postauricular flap*—staged repair utilizing a postauricular skin flap to cover a contralateral auricular or costal cartilage auricular reconstruction graft.
- *Contralateral auricular composite graft*—skin and cartilage donated from opposite ear, half the size of the defect. Donor site closed primarily.
- *Temporoparietal fascia flap*—used for extensive reconstruction. Typically used for coverage of a costal cartilage graft.

Lobule Reconstruction

Primary closure is useful for small defects. Larger defects require tissue reconstruction for the loss of skin and subcutaneous tissue. Postauricular skin flaps are useful for larger defects with or without contralateral auricular cartilage grafts.

Total Auricular Reconstruction

Auricular reconstruction is a complex task with numerous approaches designed to restore balance to face. Autologous costal cartilage grafts primary source of grafting material. Auricular prosthesis attached to osseointegrated implants are alternatives to autologous reconstruction. Tissue engineering of an auricular framework is under investigation but challenges remain in terms of tissue rejection and structural support

- *Brent technique* is a four-stage technique; performed ~3 months
  1. costal cartilage framework inserted into subcutaneous pocket
  2. lobule transposition
  3. elevation of framework and postauricular closure with advancement flap and/or FTSG
  4. tragus creation
- *Nagata Technique*
  1. costal cartilage framework with tragal component inserted into subcutaneous pocket and lobule is transposed
  2. Elevation of cartilage framework, temporoparietofacial flap used to cover posterior cartilage graft. Performed 6 months after first stage

Regional Flaps

Utilized when defect cannot be repaired with less-complex reconstructive options or patient not candidate for free flap repair. Consists of non-adjacent donor tissue (fasciocutaneous, fascial, myofascial, myocutaneous, osteomyocutaneous) with pedicled, axial blood supply.

- *Advantages*: shorter operative time, increased vascular reliability, decreased need for specialized microvascular training and postoperative monitoring
- *Disadvantages*: potential for increased donor site morbidity, decreased arc of rotation, increased risk of distal necrosis, less customizable for complex head and neck defects, bulky

Common Regional Flaps

Pectoralis Major Flap

Myocutaneous axial based off the *pectoral branch of the thoracoacromial artery*, *lateral thoracic artery* (supplies the inferolateral 1/5th). Can sacrifice lateral thoracic in order to maximize arc of rotation. Motor innervation is typically transected to promote muscle atrophy and avoid involuntary muscle contraction of the reconstruction site. Maximal flap size 10×20 cm. Skin paddles are generally positioned inferio-medial.

- *Advantages*: Donor site out of radiation field, large pedicle, easy dissection, good perforators/reliable blood supply

- *Disadvantages*: functional defect of pectoralis major, female—postoperative chest wall disfiguration and abnormal breast position, bulky, heavy body hair can be of concern for pharyngeal defects
- *Innervation*: lateral and medial pectoral nerves
- *Common usage*: oral cavity and oropharyngeal defects, great vessel coverage, pharyngoesophageal defect following total laryngectomy
- Assess preoperatively for *Poland syndrome*➔ rare birth defect characterized by unilateral underdevelopment or absence of pectoralis muscle as well as ipsilateral hypoplasia of arm and hand
- Also evaluate for previous chest wall surgery, trauma, intravenous access ports

Trapezius Flap

Versatile based off the *transverse cervical arteries*, *occipital*, suprascapular, paraspinous perforators. Maximal flap size: ~25 cm, but 8 × 12 cm is average size for skin paddle. Can be designed with three different skin paddle configurations:

1. *Superior flap*—Paraspinous perforations, some contribution from occipital artery
   - Reliable for defects of posterolateral neck that do not extend across midline
   - Blood supply unaffected by prior radical neck dissection
2. *Lateral flap*—Transverse cervical artery
   - Least reliable of the three flaps (must be careful before elevating if a neck dissection has been performed)
   - For external defects of lateral and anterior neck, oral cavity/oropharyngeal mucosal defects
3. *Lower flap*—Transverse cervical artery, dorsal scapular artery
   - Most versatile of the three flaps given arc of rotation
   - Thin, pliable
   - May mobilize entire muscle by pedicling on transverse cervical artery and vein
   - Can reach from midline of neck to lateral skull base or cheek

- *Advantages*: Can provide thin tissue for pharyngeal and carotid defects.
- *Disadvantages*: variable vascular supply, *transverse cervical vessels may be injured in patients who have undergone previous neck dissection.* Limited rotational reach, possible shoulder dysfunction, Longer OR time with positioning changes
- *Innervation*: spinal accessory nerve
- *Common usage*: inferior 2/3 facial defects, neck, temporal fossa defects

Latissimus Dorsi Flap

Myocutaneous axial based off the *thoracodorsal artery* (10–15 cm pedicle length). Broad thin muscle with maximum size 20 × 35 cm (tennis racket flap)

- *Advantages*: Long vascular pedicle, reliable blood supply, can typically close donor site primarily, no breast deformity, relatively hairless flap
- *Disadvantages*: Lateral decubitus position required for harvesting, bulky flap, large dissection; results in shoulder weakness and dysfunction, large STSG if not able to close primarily
- *Innervation*: thoracodorsal nerve
- *Common usage*: oral cavity, oropharyngeal, carotid coverage, face/neck defects

Deltopectoral Flap

Fasciocutaneous flap random based off the 1–4th perforators from *internal mammory artery* (*IMA*); random distal to deltopectoral groove. Maximum flap size 12 × 22 cm.

- *Advantages*: Donor site out of radiation field, Ease of dissection, good arc of rotation possible (45–135°)
- *Disadvantages*: Tenuous blood supply distally, contraindicated if s/p CABG, pacemaker, mastectomy, unreliable for defects that cause tissue stretch, possible requirement of split-thickness skin graft
- *Innervation*: sensory: supraclavicular nerves C3, C4 and intercostals T2–T4

- *Common usage*: cutaneous cervical defects, hypopharyngeal defects

Submental Island

Fasciocutaneous axial based off the *submental artery*; can ligate common facial vein to extend flap reach. Maximum flap size: 4×4 cm. In horizontal direction, limits of harvest at mandibular angles. In vertical direction, harvest limited by laxity of anterior cervical skin

- *Advantages*: Ease of access—neck is exposed in ablative portion
- *Disadvantages*: Cancerous nodes may involve pedicle, can be in radiation field, generally contraindicated if level 1 nodal disease present, can blunt cervicomental angle, potential injury to marginal mandibular branch of facial nerve
- *Innervation*: mental nerve
- *Common usage*: intraoral and facial cutaneous defects

Free Tissue Transfer (Table 7.2)

Most important contribution in last two decades for reconstruction of complex head and neck defects. Properly designed and executed flaps provide superior alternative to regional flaps. Ideal donor site characteristics include: long, large caliber anatomically consistent vascular pedicle, minimal donor site morbidity, ability for simultaneous two-team approach, and customizable tissue characteristics (e.g., motor/sensory innervation, ability to accept osseointegrated implants).

Fasciocutaneous and Musculocutaneous Free Flaps

Radial Forearm Free Flap

Versatile flap for reconstruction of the head and neck that can be harvested as a fasciocutaneous, myofascial, myocutaneous, osteomyocutaneous, or osteomyofascial flap. The radial forearm free flap is ideally suited for smaller defects or those requiring a thin flap for a three-dimensional shape. A preoperative *Allen's testing* must be performed to ensure the integrity of the palmar arch. A segment of the radius (up to 6–12 cm) can be harvested with the flap; however, the thickness of the bone graft is limited to *40%* of the cross-sectional area of the radius. The majority of surgeons recommend prophylactic internal fixation of the donor radius to minimize postoperative fracture, wrist immobility, and decreased grip strength. The palmaris longus tendon can be harvested and used as a sling (e.g., lower lip reconstruction). The skin paddle can also be made sensate via the antebrachial cutaneous nerve.

- *Advantages*: ease of harvest, thin, pliable flap that can be folded to fit the defect, potential

Table 7.2 Commonly referenced free flaps for head and neck reconstruction with their blood supply and sensory nerves

Flap	Artery	Vein	Nerve
Radial forearm	Radial a.	Venae comitantes or cephalic v.	Antebrachial cutaneous
Lateral arm	Post. radial collateral	Post. radial collateral	Post. cutaneous n.
Temporoparietal fascia	Superficial temporal a.	Superficial temporal v.	None
Anterolateral thigh	Descending branch from lateral circumflex femoral	Venae comitantes	Lateral femoral cutaneous
Rectus abdominis	Deep inferior epigastric	Deep inferior epigastric	Intercostal nerves
Latissimus	Subscapular a.	Subscapular v.	Thoracodorsal
Fibula	Peroneal a.	Peroneal v.	Lateral sural cutaneous
Scapula	Circumflex scapular a. Angular a. (tip)	Circumflex scapular v.	None
Iliac crest	Deep circumflex iliac	Deep circumflex iliac	None
Jejunum	Branch of sup. mesenteric a.	Branch of sup. mesenteric v.	None
Gracilis	Adductor a.	Venae comitantes	Obturator n. (motor)

for osseous component, long pedicle, potential sensate skin paddle
- *Disadvantages*: requires skin graft to donor site, concerns for hand morbidity, donor site appearance, potential flexor carpi radialis exposure. Unable to accept osseointegrated implants.

Anterolateral Thigh Free Flap

The anterolateral thigh (ALT) free flap has become the workhorse for head and neck reconstruction as it has many ideal properties. Can be harvested as myocutaneous or fasciocutaneous flap with skin paddles as large as 20 × 15 cm. Can include vastus lateralis for chimeric flaps as well as fascia lata for tissue suspension. Therefore it has many applications: oral cavity, glossectomy, oropharyngeal, and skull base defects. The flap perforators can be musculocutaneous or septocutaneous and can be identified along a line drawn from the anterior superior iliac spine to the lateral edge of the patella.

- *Advantages*: thicker than an RFFF but can be better contoured than a rectus flap, long pedicle, pedicle usually not affected by peripheral vascular disease, donor site closes primarily, possibility for chimeric flaps based on individual perforators
- *Disadvantages*: variable location of the pedicle and perforators, poor skin color match, potential for bulkiness in obese patients

Rectus Abdominus Free Flap

Reliable flap for head and neck reconstruction. Can be harvested as myocutaneous or muscle-only flap. The often ample subcutaneous fat in this region resists atrophy (unlike the rectus muscle) and may be advantageous for very-large-volume reconstruction. Uses for the rectus include total glossectomy, skull base defects. The use of a synthetic mesh for closure of the anterior rectus sheath is controversial. Proponents of mesh place it over the area of the rectus sheath that was removed with the flap, believing it to maintain the position of the remaining abdominal wall musculature and to help prevent hernias or other abdominal complications. Others believe that transfer of a single rectus abdominis muscle does not affect the postoperative function of the abdominal wall; they instead directly approximate the residual anterior fascial margins without mesh.

- *Advantages*: reliable pedicle, large bulk
- *Disadvantages*: large bulk, potential for ventral hernia, poor skin color match

Latissimus Dorsi Free Flap

The latissimus dorsi as a free flap instead of as a pedicled flap allows for more flexibility of placement and less strain on the vascular pedicle. It is often used as a muscle-only flap for total scalp reconstruction in conjunction with skin grafts

- *Advantages*: large surface area, thin (~4 mm muscle), possibility for two skin paddles
- *Disadvantages*: lateral decubitus positioning for harvest, small vessel caliber

Gracilis Muscle Flap

Myofacial flap useful for dynamic facial reanimation and wound coverage. Can provide wound coverage up to 6 × 20 cm in size. In head and neck, main indication is dynamic midface and lip reanimation for patients with long-standing facial paralysis. Typically performed as the second stage for facial reanimation following cross facial nerve grafting. Cross-facial nerve growth typically takes 6–9 months. A positive *Tinel sign* (tapping on preauricular nerve stump results in paresthesias) ensures cross-facial nerve growth prior to harvest.

- *Advantages*: ease of harvest, simple anatomy
- *Disadvantages*: potential for bulk. Can be trimmed during harvest for appropriate size

Osteocutaneous and Osteomusculocutaneous Flaps

Fibular Free Flap

The fibular free flap offers a long segment of bone for reconstruction (up to 25 cm) and therefore is ideal for mandibular reconstruction. However, the height of the bone is small, which may limit placement of dental implants. This can be overcome by using a "double-barreled" flap. The distal and proximal ends of the fibular should be preserved both proximally and distally (*6–8 cm*) to prevent injury to the peroneal nerve proximally and to support the ankle joint distally. A preoperative *MRA* or angiography can assist in ensuring viability of peroneal vessels, ruling out atherosclerotic disease (relative contraindication), and ruling out *peroneal arteria magna*, a condition in which the peroneal artery is sole blood supply to foot-(absolute contraindication). Skin paddles are designed between the middle and distal portion of the fibula to optimize viability. A cuff of soleus muscle is harvested with the flap to protect perforating vessels.

- *Advantages*: long segment of bone, ideal for total mandibular reconstruction, long high-caliber vascular pedicle, ability to support dental implants, ability to perform two-team approach
- *Disadvantages*: limited vertical height of bone, small viable skin paddle, need for skin graft closure, potential leg morbidity, pedicle often affected by peripheral vascular disease

Iliac Crest Free Flap

Less commonly used flap. The iliac crest can be used for hemimandibular defects with contouring cuts along the outer cortex. The bone can be contoured to match the symphyseal curvature by making osteotomies, resulting in wedge-shaped openings. Extension to the anterior inferior iliac spine can be included if condylar reconstruction is needed. The bone is a good caliber for osseointegrated dental implants. Nearly 16 cm of bone can be harvested. For internal and external oral cavity defects, only a small internal skin paddle can be obtained. Inner, mucosal, reconstruction can be performed with this flap if the internal oblique is included and skin grafted for the mucosal side.

- *Advantages*: good bone stock for dental implants
- *Disadvantages*: hernia formation at donor site is possible, short pedicle, difficult to harvest in obese patient

Scapular Free Flap

Donor site of choice for complex 3-D defects of the head/neck. Ideal for defects requiring significant bulk of tissue for reconstruction, as well as through-through defects of the oral cavity. This flap can provide a moderate (up to 14 cm) amount of bone from the lateral segment of the scapula, with additional bone stock available from the scapular tip via the angular artery. The scapular tip bone is also a good match for palatal and maxillary reconstruction. The advantage of this flap is that *two skin paddles* can be designed off of the circumflex scapular artery, and the arterial anatomy allows for a significant amount of mobility for the inset of these two flaps (great for dual-layer oral cavity reconstruction with mandibular reconstruction).

- *Advantages*: ability to reconstruction complex 3-D defects, primary closure of donor site, supports osseointegrated implants
- *Disadvantages*: lateral decubitus positioning, potential donor-site morbidity

Jejunum Free Flap

The main application of a free jejunum transfer is reconstruction of the hypopharynx and cervical esophagus. The diameter is a good match for the esophagus. It is vascularized via branches from the superior mesenteric artery. The distal end of the jejunum should be oriented distally to the esophagus upon inset in order to ensure antegrade peristalsis. A segment of omentum can be included in the flap to be skin grafted for external coverage. An external monitoring flap is commonly used.

- *Advantages*: already a tube, so only two anastomotic suture lines
- *Disadvantages*: intraabdominal complications from laparotomy and bowel resection, excess mucus production and wet voice with TEP, swallowing dysfunction from stenosis

Critical Times of Free Flap Warm Ischemia

- Jejunal flaps: 2 hours
- Skeletal flaps (fibula, etc.): 6 hours
- Cutaneous flaps: 8 hours

Postoperative Flap Anticoagulation

- No consensus on ideal regimen
- Aspirin (325 mg qd) and subcutaneous heparin (5000 U SC daily) common choices
- One study has shown that a regimen of aspirin (325 mg qd) + subcutaneous heparin (5000 U SC BID) can be effective; no increase in postoperative hematoma compared to other anticoagulation agents
- Postoperative anticoagulation choice has shown no statistically significant effect on incidence of free flap complications

Postoperative Flap Monitoring

- Flap monitoring includes:
  - Clinical observation
  - Handheld doppler
  - Surface temperature probe
  - Pinprick
  - Implantable doppler
- Some recommend hourly flap checks for the first 24 hours then q4h flap checks for the next 48 hours.
- For buried flaps (pharyngoesophageal reconstruction), can consider *monitoring segment* or flap *segment exteriorization*
- Implantable dopplers have been used with mixed success; they can be reliable and allow for rapid recognition of complications with return to the operating room if necessary. False positives do occur.
  - Can improve reliability of monitoring flap by avoidance of making it too small or damaging one of its perforators
- Flap failure rate (5–15%)
- Problems arise usually within the first 24–72 hours
- Flap salvage attempts typically most successful within 24 hours of initial operation.
- Studies *show up to 77% salvage rate* of free flaps in the operating room when problems are identified using conventional monitoring techniques. Buried flaps have significantly lower salvage rates.
- Disa JJ, et al. 1999: buried flaps had higher loss rate, usually later ~7 days due to infection or fistula. Conventional monitoring works great for nonburied flaps; but recommend implantable monitoring or monitor paddle for buried flaps.

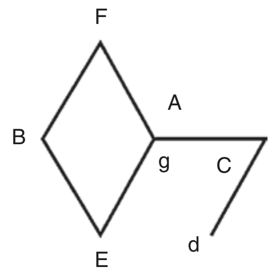


Fig. 7.11 Traditional rhombic flap prior to flap elevation and inset

Questions

1. Which free grafts typically exhibit the most postoperative contraction?
   (a) Thin split-thickness skin grafts
   (b) Perichondrial composite grafts
   (c) Full-thickness skin grafts
   (d) Thick split-thickness skin grafts
2. At 24 h postoperatively, a patient's nasal skin graft site appears to be necrotic. What medication could have been used perioperatively, which may have increased the chances of graft survival?
   (a) Nitroglycerin paste
   (b) Intravenous acetaminophen
   (c) Topical vitamin E
   (d) Dexamethasone
   (e) Bevacizumab
3. A rhombic flap is planned for reconstruction of a 3 cm cheek defect. According to the figure notations on the image below (Fig. 7.11), where would the vector of tension be greatest?
   (a) A → B
   (b) B → C
   (c) C → F
   (d) D → A
   (e) B → D
4. Dufourmentel and Zitelli are both known for modifying well-known transposition flap

designs. What do their modifications have in common?
(a) Both design modifications employ an M-plasty to close the defect
(b) Both design modifications lessen the arc of pivot for the flap
(c) Both design modifications are intended for smaller defects
(d) Both design modifications are intended to reduce "pin-cushioning"

5. A 33-year-old woman presents with a contour defect of the right cheek after removal of a hemangioma as a child. You decide to try a free dermal fat graft for her problem. How much volume loss should the counsel to the patient to expect postoperatively?
(a) 0–5%
(b) 10%
(c) 30%
(d) 75%
(e) 90%

6. A patient presents with a 1.5 cm skin-only defect involving the left nasal sidewall centered just medial to the medial canthus. Which flap would NOT be a reasonable option for reconstruction?
(a) Glabellar transposition flap
(b) Cheek rotation flap from below
(c) Inferior and medially based bilobe flap
(d) Melolabial island pedicle flap
(e) Double-opposing transposition flaps

7. You are repairing a 40% lower lip defect with a cross-lip flap. Which one of the following statements is correct?
(a) The width of the flap should equal the width of the defect.
(b) The labial artery is usually located between the orbicularis oris muscle and the labial mucosa.
(c) Estlander flaps can be used to create a sharp, acutely angled oral commissure.
(d) The vertical height of the flap is designed to be half the height of the defect.

8. A 70-year-old patient presents with a large squamous cell carcinoma of the scalp. Upon clearance of the tumor, a 20 cm scalp vertex defect remains with exposed cranial bone. Which reconstructive option would you recommend?
(a) Application of split-thickness skin graft from the lateral thigh.
(b) to S rotational flap closure
(c) Temporoparietal fascia flap covered with split-thickness skin graft
(d) Split-thickness skin graft over a latissimus dorsi free flap
(e) Radial forearm fasciocutaneous free flap

9. Which technique can result in reduced morbidity of radial forearm free flaps?
(a) Always use a monitor paddle.
(b) Perform rigid internal fixation when harvesting radial bone.
(c) Utilize ulnar arterial bypass grafts.
(d) Remove splint on postoperative day 1 for early mobilization.
(e) Always spare palmaris longus tendon.

10. Your head and neck surgery colleagues have just finished resection of a 5 cm buccal space and mandibular malignancy, and you are asked to assist in the reconstruction of the defect that encompasses the buccal mucosa, 3 cm of the angle of the mandible, and the overlying cheek skin. Which option would you recommend for this composite defect?
(a) Osteocutaneous radial forearm free flap
(b) Fibular free flap
(c) Rib-pectoralis major osteomusculocutaneous regional flap
(d) Split calvarial bone graft with a cervicofacial advancement-rotation flap
(e) Scapular osteocutaneous free flap

11. Which of the following scenarios would be best served with vascularized bony reconstruction?
(a) 6-cm posterior defect from mandibular condyle to angle
(b) 4-cm posterior mandibular defect of mandibular condyle and ramus
(c) 5-cm lateral defect of mandibular body
(d) 5-cm anterior composite defect of mandible and floor of mouth
(e) Both a and b

12. The survival of an axial flap corresponds mostly with:
    (a) Length of the axial vessel
    (b) Reliable subdermal blood supply
    (c) Maximum length-to-width axial ratio of 3:1
    (d) Maximum length-to-width axial ratio of 4:1
13. Which maneuver mostly likely improves flap advancement in a V-Y flap?
    (a) Increasing the width of the flap
    (b) Increasing the length of the flap
    (c) Increasing the depth of the pivot plane
    (d) Anchoring the subcutaneous tissues with deep dermal sutures
    (e) Designing the flap so that it is based on its pedicle, allowing for maximal movement
14. One month following reconstruction of a nasal tip defect with a full-thickness skin graft, which of the following will most likely NOT be necessary?
    (a) Chemical peels
    (b) Laser resurfacing
    (c) Dermabrasion
    (d) Steroid injection
15. Which of these locations is ideal for a full-thickness skin graft?
    (a) Forehead
    (b) Preauricular area
    (c) Nasal tip
    (d) Cheek
    (e) Neck
16. A 50-year-old man previously underwent a scalp melanoma excision with a skin graft. He is now undergoing scalp expansion with a tissue expander to help excise non-hair bearing skin. He has been doing well and has nearly met his expansion goals. Recently, he presented to the office with mild dehiscence and implant exposure. No evidence of infection is present. What is the best course of action at this time?
    (a) Immediately cease tissue expansion and allow the patient's wound to close secondarily
    (b) Abort tissue expansion and remove the implant
    (c) Proceed with inflation while carefully monitoring for signs of infection
    (d) Proceed with inflation only after attempting to close the dehiscent areas
    (e) Admit the patient for a wound washout and intravenous antibiotic therapy
17. A 65-year-old man, longtime smoker, undergoes treatment for a lower lip squamous cell carcinoma. The lesion involves greater than 2/3 of the lower lip, sparing the bilateral commissures. Which of the following is the best reconstructive option?
    (a) Primary closure
    (b) Abbe flap
    (c) Estlander flap
    (d) Abbe-Estlander flap
    (e) Bernard-Burrow flap
    (f) Free tissue transfer with palmaris tendon (i.e., radial forearm flap)
18. An elderly man previously underwent Mohs surgery years ago for a nasal basal cell carcinoma, ending up with a through and through defect. He brings with him operative notes which state that a septal hinge flap was used for the inner nasal lining and a paramedian flap for the external skin. Unfortunately, the patient appears to have a recurrence of his cancer, and the dermatological surgeon believes the defect will again involve all layers of his nose. Which of the following would be best to reconstruct the mucosal lining?
    (a) Split-thickness skin graft
    (b) Full-thickness skin graft
    (c) Nasal skin "turn in" flap
    (d) Inferior turbinate mucosal flap
    (e) Temporoparietal fascia flap
19. What percentage of the lower eyelid can be resected and still be closed primarily with a lateral canthotomy and cantholysis?
    (a) 10
    (b) 15
    (c) 25
    (d) 50
    (e) 75
20. During scalp reconstruction, which types of flaps are typically utilized?
    (a) Advancement (v-y)
    (b) Hinge

(c) Transposition (rhomboid)
(d) Rotation
(e) Interpolation

21. Which of the following best describe random skin flaps that have undergone tissue expansion?
(a) They are not as likely to survive as long as delayed flaps
(b) They are not reliable if expansion has been completed in <6 weeks
(c) They are likely to survive as long as or longer than nonexpanded flaps
(d) They generally have compromised vascularity secondary to chronic pressure
(e) They generally have compromised vascularity secondary to thinned, atrophic tissues

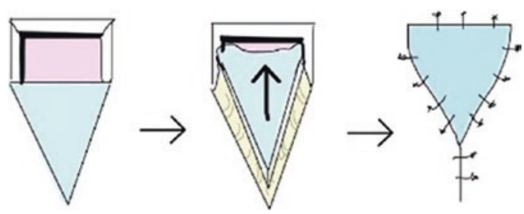
22. You are performing a reconstruction of a posterolateral neck defect and are considering your surgical options. The patient states that he did have a neck dissection on the same side many years ago. Which of the flaps would be most at risk?
(a) Pectoralis
(b) Submental
(c) Deltopectoral
(d) Trapezius
(e) Sternocleidomastoid

23. In the immediate postoperative period following a reconstructive free tissue transfer, when do most failures occur, and what is the average salvage rate on reexploration?
(a) First 2–12 hours, 75%
(b) First 12–24 hours, 75%
(c) First 12–24 hours, 50%
(d) First 24–72 hours, 50%
(e) First 72–96 hours, 25%

24. What is an absolute contraindication to use of the fibular free flap?
(a) Diabetes mellitus with neuropathy
(b) History of previous lower extremity fracture
(c) Peroneal arteria magna
(d) Severe atherosclerotic disease
(e) Impaired ambulation or use of walker/cane

25. A 35-year-old woman with a history of extensive sun damage and use of tanning booths developed a nasal dorsum and tip basal cell carcinoma. She underwent Mohs surgery and now has a 2.0 cm defect. Her reconstructive surgeon decides to proceed with a dorsal nasal flap to reconstruct this. Which of the following can be associated with this flap and must be discussed with the patient before proceeding?
(a) Nasal tip ptosis
(b) Widening of the nasal midvault
(c) Nasal tip rotation
(d) Alar retraction
(e) Both c and d

26. What type of flap is depicted in this diagram? *(JPEG image also attached)*
(a) Rotational flap
(b) Transposition flap
(c) Advancement flap
(d) Interposition flap
(e) Interpolated flap



27. A 25-year-old man is seen in the emergency room after being in a car accident and injuring his auricle. He appears to have a right sided full-thickness helical defect measuring 1.2 cm. What is the best treatment plan?
(a) Close the defect primarily
(b) Helical rim advancement flap
(c) Tubed postauricular pedicle flap
(d) Reconstruction with costal cartilage, temporoparietal flap, and skin graft
(e) Allow wound to heal by secondary intention

28. What does the "reverse flow flap" involve in a submental artery island flap?
(a) Ligation of proximal facial artery
(b) Ligation of common facial vein
(c) Ligation of external jugular vein
(d) Ligation of angular artery
(e) Ligation of retromandibular vein

29. A 65-year-old man undergoes a composite resection for squamous cell carcinoma and is reconstructed with a fibular free flap. The surgeon states that he is considering "double barreling." What would this accomplish?
    (a) Help preserve mandibular height
    (b) Reliably provide two paddles—external facial skin and oral mucosal lining
    (c) Provide improved venous outflow with two venous anastomoses
    (d) Help the thicker paddle withstand the effects of postoperative radiation
    (e) None of the above
30. Which of the following maneuvers is accurate during elevation of the supraclavicular island artery flap?
    (a) Raising the flap in a subfascial plane from a proximal to distal direction and then transitioning to a subperiosteal dissection over the clavicle into the fossa
    (b) Raising the flap in a subfascial plane from a distal to proximal direction and then transitioning to a subperiosteal dissection over the clavicle into the fossa
    (c) Raising the flap in a subfascial plane from a proximal to direction and then transitioning to a suprafascial dissection over the clavicle into the fossa
    (d) Raising the flap in a suprafascial plane from a distal to proximal direction and then transitioning to a supraperiosteal dissection over the clavicle into the fossa
    (e) Raising the flap in a suprafascial plane from a distal to proximal direction and then transitioning to a subperiosteal dissection over the clavicle into the fossa
31. Select the correct answer from the following when generally comparing cortical thicknesses among osseous free flap options:
    (a) Iliac > Fibula > Scapula
    (b) Fibula > Radius > Scapula
    (c) Scapula > Fibula > Radius
    (d) Scapula > Iliac > Fibula
    (e) Iliac > Radius > Fibula
32. Terminal angles of a cutaneous defect should be roughly ____ degrees or less to avoid cutaneous deformities.
    (a) 15
    (b) 20
    (c) 30
    (d) 45
    (e) 60
33. Which of the following locations would provide the ideal donor site skin for reconstruction of a nasal tip defect?
    (a) Supraclavicular
    (b) Preauricular
    (c) Postauricular
    (d) Forehead
    (e) Upper eyelid
34. You are designing a scalp rotation flap. Which of the following accurately describes the proper design to allow for best primary defect coverage?
    (a) Rotation arc 2× the defect diameter
    (b) Rotation arc 3× the defect diameter
    (c) Rotation arc 4× the defect diameter
    (d) Rotation arc 5× the defect diameter
    (e) Rotation arc 6× the defect diameter
35. Which of the following lip reconstructive techniques has the highest tendency for producing a rounded commissure?
    (a) Abbe flap
    (b) Estlander flap
    (c) Bernard-Burrow Webster flap
    (d) Gillies flap
    (e) Karapandzic flap

Answers

1. (a)
2. (d)
3. (d)
4. (b)
5. (c)
6. (d)
7. (b)
8. (d)
9. (b)
10. (e)
11. (d)
12. (a)
13. (c)
14. (d)
15. (e)
16. (c)

17. (e)
18. (d)
19. (d)
20. (d)
21. (c)
22. (d)
23. (d)
24. (c)
25. (e)
26. (c)
27. (a)
28. (a)
29. (a)
30. (b)
31. (b)
32. (c)
33. (a)
34. (e)
35. (b)

Additional Resources

1. Baker SR. Local cutaneous flaps. Otolaryngol Clin N Am. 1994;27(1):136–59.
2. Baker SR, editor. Local flaps in facial reconstruction. 2nd ed. Philadelphia: Elsevier; 2007.
3. Civelek B, Celebioglu S, et al. Denervated or innervated flaps for lower lip reconstruction? Are they really different to get a good result? Otolaryngol Head Neck Surg. 2006;134(4):613–7.
4. Coleman SC, Burkey BB, Day TA, et al. Increasing use of the scapula osteocutaneous free flap. Laryngoscope. 2000;110(9):1419–24.
5. Davis RE, et al. Autologous free dermal fat graft. Arch Otolaryngol Head Neck Surg. 1995;121(1):95–100.
6. Disa JJ, Cordeiro PG, Hidalgo DA. Efficacy of conventional monitoring techniques in free tissue transfer: an 11-year experience in 750 consecutive cases. Plast Reconstr Surg. 1999;104(1):97–101.
7. Fann PC, et al. Pharmacologic and surgical enhancement of composite graft survival. Arch Otolaryngol Head Neck Surg. 1993;119(3):313–9.
8. Fisher E, Frodel JL. Facial plastic and reconstructive surgery. 2nd ed. New York: Thieme; 2001. Chapter 2, Wound healing. p. 15–25.
9. Kaplan B, Moy RL. Flaps and grafts for facial reconstruction. Dermatol Surg. 1995;21(5):431–40.
10. Patel KG, Sykes JM. Concepts in local flap design and classification. Operat Tech Otolaryngol. 2011;22:13–23.
11. Richardson D, Fisher SE, Vaughan ED, Brown JS. Radial forearm flap donor site complications. Plast Reconstr Surg. 1997;99(1):109–15.
12. Suryadevara AC, Moe KS. Reconstruction of eyelid defects. Facial Plast Surg Clin North Am. 2009;17:419–28.
13. Urken ML. Composite free flaps in oromandibular reconstruction. Arch Otolaryngol Head Neck Surg. 1991;117(7):724–32.
14. Weber SM, Baker SR. Management of cutaneous nasal defects. Facial Plast Surg Clin North Am. 2009;17:395–417.
15. Weerda H. Reconstructive facial plastic surgery. 1st ed. New York: Thieme; 2001.
16. Dinehart SM. The rhombic bilobed flap for nasal reconstruction. Dermatol Surg. 2001;27(5):501–4.
17. Kinsella JB, Rassekh CH, Hokanson JA, Wassmuth ZD, Calhoun KH. Smoking increases facial skin flap complications. Ann Otol Rhinol Laryngol. 1999;108(2):139–42.
18. Hoffmann JF, Papel, et al., editors. Facial plastic and reconstructive surgery. 3rd ed. Thieme; 2016. Chapter 56, Musculocutaneous flaps. p. 1120–2.
19. Annino DJ, Shu RS, Gold DR, Papel, et al. Facial plastic and reconstructive surgery. 3rd ed. Thieme; 2016. Chapter 55, Tissue expansion in reconstruction of the head and neck. p. 1113–5.
20. Urken ML. Atlas of regional and free flaps for head and neck reconstruction. 2nd ed. Baltimore: Lippincott Williams & Wilkins; 2012. Chapter 1, Pectoralis Major. p. 39–76.
21. Pryor SG, Moore EJ, Kasperbauer JL. Implantable doppler flow system: experience with 24 microvascular free-flap operations. Otolaryngol Head Neck Surg. 2006;135(5):714–8.
22. Disa JJ, Cordeiro PG, Hidalgo DA. Efficacy of conventional monitoring techniques in free tissue transfer: an 11-year experience in 750 consecutive caes. Plast Reconstr Surg. 1999;104(1):97–101.
23. Blackwell KE. Unsurpassed reliability of free flaps for head and neck reconstruction. Arch Otolaryngol Head Neck Surg. 1999;125(3):295–9.
24. Cho BC, Shin DP, Byun JS, Park JW, Baik BS. Monitoring flap for buried free tissue transfer: its importance and reliability. Plast Reconstr Surg. 2002;110(5):1249–58.
25. Chien W, Varvares MA, Hadlock T, Cheney M, Deschler DG. Effects of aspirin and low-dose heparin in head and neck reconstruction using microvascular free flaps. Laryngoscope. 2005;115(6):973–6.
26. Ashjia P, Chen CM, Pusic A, Disa J, Cordeiro P, Mehrara BJ. The effect of postoperative anticoagulation on microvascular thrombosis. Ann Plast Surg. 2007;59(1):36–40.

8 Facial Paralysis and Facial Reanimation

Eric W. Cerrati and Jon-Paul Pepper

Extratemporal Anatomy of the Facial Nerve

Main Trunk Location [1]

- 5.5 ± 2.1 mm from posterior belly of digastric muscle
- 6.9 ± 1.8 mm from tragal pointer
- 10.9 ± 1.7 mm from external auditory canal
- *2.5 ± 0.4 mm from tympanomastoid suture* (*most reliable landmark due to its proximity and bony origin*)

Proximal to the pes anserinus, the extratemporal nerve gives off three branches innervating the posterior auricular muscle, the posterior belly of the digastric muscle, and the stylohoid muscle.

Davis Classification of Facial Nerve Branching Patterns (Percentages According to Original Paper)

- Type 1: absence of an anastomosis between the temporofacial division and cervicofacial division (13%)
- Type 2: anastomosis among the branches of the temporofacial division only (20%)
- Type 3: single anastomosis among the branches of the temporofacial division and cervico-facial division (28%)
- Type 4: combination of type II and III (24%)
- Type 5: double anastomosis between the temporofacial division and cervicofacial division (9%)
- Type 6: complex multiple anastomosis between the two divisions, where the buccal branch receives many anastomotic fibers from the cervicofacial division and the mandibular branch (6%)

Temporal (Frontal) Branch

Pitanguy and Ramos [2] described a line starting from a point 0.5 cm below the tragus that extended in the direction of the brow, passing 1.5 cm above the lateral extension of the eyebrow. This description has been referred to as "Pitanguy's line." With this approximation, the nerve lies 2–3 cm from the lateral orbital rim at

E. W. Cerrati (✉)
Division of Otolaryngology – Head and Neck Surgery, University of Utah, Salt Lake City, UT, USA

J.-P. Pepper
Department of Otolaryngology – Head and Neck Surgery, Stanford University, Palo Alto, CA, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_8

the level of the lateral canthus. Note that the frontal branch is on the deep surface of the temporoparietal fascia when above the zygomatic arch. At the level of the arch, the nerve is relatively deep and adjacent to the periosteum.

Relationship of Frontal Branch to the Superficial Temporal Artery

The frontal branch of the superficial temporal artery at the level of the lateral border of the frontalis is a common point of entry of the temporal branch of the facial nerve into the muscle. The nerve rami are usually anterior to the vessel (~90%) as it branches from the superficial temporal artery.

Buccal and Zygomatic Branches

The buccal and zygomatic branches of the facial nerve were found by Davis et al. [3] to divide 2.0 cm beyond the anterior edge of the parotid gland before supplying the midface muscles. The nerves are located in close proximity to the retaining ligaments (zygomaticocutaneous and masseteric).

Marginal Mandibular Branch

The marginal mandibular branch exits the anterior caudal margin of the parotid and remains deep to the parotidomasseteric fascia and deep cervical investing fascia. When the nerve travels inferior to the mandible, it runs across the surface of the posterior digastric muscle and submandibular gland. Staying deep to the platysma and the investing layer of the deep fascia, it runs superficial to the facial vessels as it rises above the mandibular border.

Relationship of Marginal Mandibular Branch to Facial Artery and Vein

Dingman and Grabb [4]: Posterior to the facial artery, the mandibular branch was found to pass *superior to* the lower border of the mandible in 81% of the specimens. In the other 19%, one or more rami of the mandibular branch formed a downward arc, whose lowest point extended up to 1.0 cm *below* the inferior border of the mandible. Anterior to the facial artery, *all* of the rami of the mandibular branch were found to be above the lower border of the mandible.

Cervical Branch

The cervical branch exits the caudal edge of the parotid just anterior to the angle of the mandible and perforates the deep cervical fascia. Chowdhry et al. [5] found the mean branching point of the nerve to lie 1.74 cm from the angle of the mandible along a trajectory that is perpendicular to a line from the mastoid to mentum.

Etiologies of Facial Paralysis, See Table 8.1 [6]

Sunderland Classification of Nerve Injury, Table 8.2 [8]

Systems Used to Classify Severity of Injury in Patient with Facial Weakness or Paralysis

House-Brackmann Scale (Table 8.3): a global facial function grading system that is used for prognosis for recovery following Bell's palsy [9]. It is not descriptive of branch by branch dysfunction, which is most important for facial nerve reanimation surgery, given the importance of the buccal branch for normal smile.

Table 8.1 Etiologies of facial paralysis

Category	Includes	Annual incidence	Percentage
Idiopathic	Bell's palsy	75,396	49.6
Infectious	Lyme disease, otitis media	23,222	15.3
Neoplastic	Acoustic neuroma, parotid malignancy	20,508	13.5
Neurologic	Stroke, Guillan Barre	20,508	13.5
Traumatic	Temporal bone fracture, birth trauma	12,365	8.1
Total		*151,999*	

Table 8.2 Sunderland classification of nerve injury

Degree of injury	Pathophysiology	Prognosis	Time to recovery
1st degree	Conduction block without axonal degeneration	Excellent	3 weeks
2nd degree	Arteriole compression, causing ischemic axonal injury, or inflammation such as from a viral infection	Good	3 weeks–3 months
3rd degree	Endoneurial injury	Moderate	2–4 months
4th degree	Perineurial injury	Poor	Uncertain
5th degree	Epineurial injury	Poor	Uncertain

Note that recovery times are based on intratemporal facial nerve injury, with a presumed rate of axonal regeneration of approximately 1 mm per day [7]

Sunnybrook Facial Grading System, see Fig. 8.1 [10]:

There are also three-dimensional image capture and analysis programs that provide quantitative measures, such as the FACIAL CLIMA system [11].

Bell's Palsy: Acute Idiopathic Facial Paralysis [6, 12, 13]

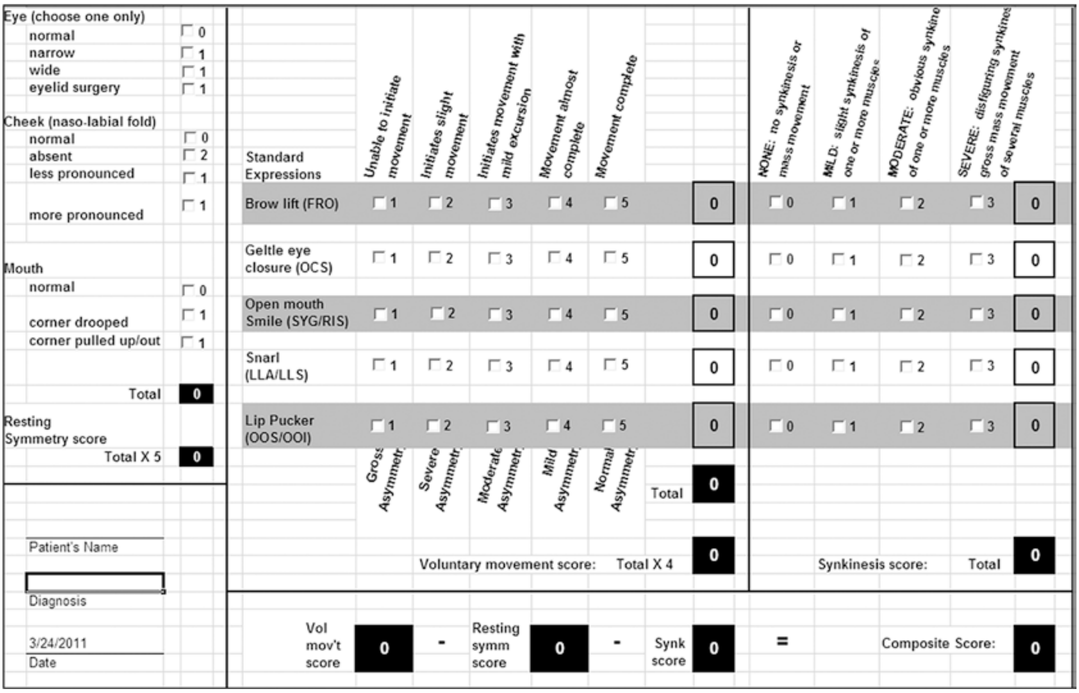
1. The most common cause of facial paralysis
2. "Acute" means symptoms develop over 72 h time frame
3. A diagnosis of exclusion (i.e., no other causative factors or ominous signs)
4. Self-limited
5. Overall, has a good prognosis (see below)
6. Represents 50–70% of all facial nerve paralysis per annum
7. Bell's palsy is an acute idiopathic facial nerve paralysis thought to have a viral cause.
8. Other causes of facial paralysis excluded by clinical examination and history: tumor, stroke, Lyme disease, autoimmune disease, multiple sclerosis, HIV, trauma, otitis media, others

Table 8.3 House-Brackmann scale

Grade	Function	Description
I	Normal function	Normal facial function in all areas
II	Slight dysfunction	Gross: slight weakness noticeable on close inspection; may have very slight synkinesis At rest: normal symmetry and tone Motion: forehead—moderate to good function; eye—complete closure with minimum effort; mouth—slight asymmetry
III	Moderate dysfunction	Gross: obvious but not disfiguring difference between two sides; noticeable but not severe synkinesis, contracture, and/or hemi-facial spasm At rest: normal symmetry and tone Motion: forehead—slight to moderate movement; eye—complete closure with effort; mouth—slightly weak with maximum effort
IV	Moderate severe dysfunction	Gross: obvious weakness and/or disfiguring asymmetry At rest: normal symmetry and tone Motion: forehead—none; *eye—incomplete closure*; mouth—asymmetric with maximum effort
V	Severe dysfunction	Gross: only barely perceptible motion *At rest: asymmetry* Motion: forehead—none; eye—incomplete closure; mouth—slight movement
VI	Total paralysis	No movement

Bell's Palsy Epidemiology and Prognosis

- Incidence ranges from 11.5 to 40.2 per 100,000
- More common in ages 15–45, diabetics, immunocompromised, during pregnancy, and in those with a concomitant viral upper respiratory tract infection
  - Slightly greater occurrence in men older than 40 years and in women younger than 20

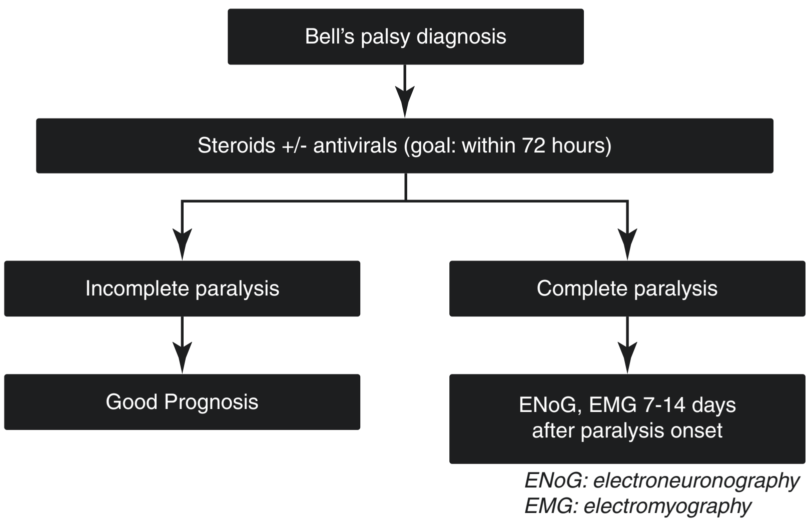

Eye (choose one only)
normal 0
narrow 1
wide 1
eyelid surgery 1
Cheek (naso-labial fold)
normal 0
absent 2
less pronounced 1
more pronounced 1
Mouth
normal 0
corner drooped 1
corner pulled up/out 1
Total 0
Resting Symmetry score Total X 5 0
Patient's Name
Diagnosis
3/24/2011
Date

Standard Expressions	Unable to initiate movement	Initiates slight movement	Initiates movement with mild excursion	Movement almost complete	Movement complete		NONE: no synkinesis or mass movement	MILD: slight synkinesis of one or more muscles	MODERATE: obvious synkinesis of one or more muscles	SEVERE: disfiguring synkinesis gross mass movement of several muscles	
Brow lift (FRO)	1	2	3	4	5	0	0	1	2	3	0
Geltle eye closure (OCS)	1	2	3	4	5	0	0	1	2	3	0
Open mouth Smile (SYG/RIS)	1	2	3	4	5	0	0	1	2	3	0
Snarl (LLA/LLS)	1	2	3	4	5	0	0	1	2	3	0
Lip Pucker (OOS/OOI)	1	2	3	4	5	0	0	1	2	3	0
	Gross Asymmetry	Severe Asymmetry	Moderate Asymmetry	Mild Asymmetry	Normal Asymmetry	Total 0					

Voluntary movement score: Total X 4 0
Synkinesis score: Total 0
Vol mov't score 0 - Resting symm score 0 - Synk score 0 = Composite Score: 0

Fig. 8.1 Sunnybrook facial grading system. Note the inclusion of synkinesis in the grading scale

Fig. 8.2 Workup and treatment algorithm for Bell's palsy



- In general, approximately one-third of patients present with complete paralysis
- Only 61% of patients with complete paralysis will have complete recovery
- Nearly all patients with incomplete paralysis (paresis) will fully recover
- Up to 94% with *paresis* will recover to complete function within 4 months
- Synkinesis occurs in ~20% patients
- Of those with complete paralysis, prognosis is worse
- In a large series of *untreated* patients, approximately 70% recover function with *watchful waiting alone* [14]

Bell's palsy workup and treatment algorithm, note that use of antivirals is per practitioner preference, Fig. 8.2 [15]:

Electrodiagnostic Testing When Complete Paralysis Is Present

ENoG: measures compound muscle action potentials after applying bipolar stimulation to the mastoid process region (main trunk of facial nerve)

1. Performed between day 3 and day 14 after symptom onset. Earlier evidence of conduction block (i.e., earlier Wallerian degeneration) likely indicative of higher grade of injury.
2. Fisch criteria: >90% degeneration. The amplitude of the compound muscle action potential on the paralyzed side is 10% (or less) of the amplitude of the compound muscle action potential on the contralateral (intact) side. Used as a criterion for possible facial nerve decompression [16].

EMG (volitional and resting): motor unit action potentials

1. Typically done on days 10–14 post injury. A confirmatory test for ENOG in the acute setting.
2. Resting: fibrillation potentials and positive sharp waves are indicative of denervation.
3. Volitional EMG: polyphasic action potentials are indicative of early re-innervation.
4. EMG can be used to follow patients for subclinical volitional responses in re-innervating muscle [17].
5. *EMG is a significant tool for determining the timing of reinnervation surgery*.

When to Image (MRI with Contrast Preferred) a Patient with Presumed Bell's Palsy

- A second episode
- No improvement in symptoms after 3 months
- Associated second cranial neuropathy
- Isolated branches of facial nerve affected
- Progression from paresis to paralysis

Paralysis Has Significant Implications on Patients' Lives

- Healthy individuals are willing to sacrifice 8 years of life and undergo a surgery associated with 21% mortality to correct facial paralysis [18]
- Ultimate goal of reanimation treatment is to restore resting tone/bulk and allow symmetric, spontaneous and dynamic function.

Facial Paralysis (Any Etiology), Management Concepts

1. Early corneal protection (first and foremost priority)
2. Timely neurodiagnostics (see above)
3. Possible surgical intervention

Corneal Protection

- Lubricating eye drops, tape, moisture chamber
- Platinum weight
- Palpebral spring
- Botulinum toxin of levator palpebrae superioris
- Temporary tarsorrhaphy

Lower lid management: corneal coverage with upper lid weight *without* lower lid suspension is significantly less effective than upper lid loading in combination with a procedure to support the lower lid [19, 20]. Lower lid tightening procedures are indicated for patients with paralytic ectropion who have pooling of tears, epiphora, and eversion of the lacrimal punctum.

Options for treating the lower lid include:

- Lateral canthopexy
- Formal lateral canthoplasty
- Lower lid wedge excision
- Sub-orbicularis oculi fat pad (SOOF) lift for very ptotic midface

Eyelid Procedures

- *Platinum (or gold) weight* – weights are commonly 0.8–1.2 g, incision is made in supratarsal crease, and weight is secured to the tarsal plate centered over the medial limbus
- *Palpebral spring* – technically difficult procedure and contraindicated in a patient that may require future MRI scans, spring places a downward force on the upper eyelid that increases with the elevation of the upper lid (levator function) resulting in a greater degree

of reflex blinking, often requires minor adjustments after placement
- *Tarsorrhaphy* – multiple techniques exist in which the eyelids are partially sewn together to narrow the opening, may be temporary or permanent
- *Canthopexy* vs. *Canthoplasty* – canthopexy uses sutures to tighten and stabilize the lower lateral canthal tendon and muscle without reconstruction; canthoplasty (aka tarsal strip) involves detaching the lateral canthal tendon to remove a portion of it and reconstructing it
- *Wedge excision* – alternative procedure for lower lid tightening, careful reapproximation of anterior and posterior lamella is critical for proper healing, may excise up to 25% of width without cantholysis
- *SOOF lift* – the fat pad can be isolated between the preperiosteal plane and the orbicularis muscle while carefully preserving the infraorbital nerve, fat pad is sewn to the orbital rim periosteum to alleviate the gravitational forces on the paretic midface and lower eyelid

Synkinesis

- *Definition*: unintentional motion of one area of the face produced during intentional movement of another following facial nerve paresis or paralysis [21]
- Socially and functionally debilitating, sometimes painful
- Poor prognosis for spontaneous resolution of synkinesis
- Location of injury is thought to be a significant predictor of future synkinesis

Incidence of Synkinesis, See Table 8.4

Natural History of Synkinesis

- Synkinesis can follow any type of significant facial nerve paresis or paralysis (see below)
- Often begins 3–4 months following injury, but has been reported as early as 6 weeks post injury
- Prognosis for spontaneous improvement of facial synkinesis once it begins is poor [24, 25]

Table 8.4 Relative incidence of synkinesis based on location and grade of injury

Population	Incidence of synkinesis (%)
Following surgical repair of intratemporal facial nerve, humans [22]	100
Following surgical repair of extratemporal facial nerve, humans [23]	69
Bell's palsy	18.3–55

Three proposed mechanisms:

1. Aberrant regeneration of axons (dominant theory)
2. Ephaptic transmission due to demyelination
3. Facial nucleus somatotopic changes and excitability threshold changes

Regenerating facial nerve axons may have a large number of collaterals after injury. A large number of these collaterals appear to stay intact over the long term, thereby disrupting the somatotopic organization of the facial nucleus for long periods of time [26].

Management of facial synkinesis:

1. Physical therapy/biofeedback
2. Neuromuscular facial retraining
3. Chemodenervation with botulinum toxin injections

Both physical therapy and neuromuscular retraining have been demonstrated to be effective based on large retrospective cohort studies [27, 28]. Chemodenervation has been validated by double-blind randomized controlled trial [29].

Surgical Management of Chronic Facial Paralysis

In general, a combination of static and dynamic techniques may be employed. The surgical approach is tailored to patient's desire, age, prognosis, prior radiation treatment, etc.

For dynamic reinnervation surgery, the best and most predictable results are when it is performed within 2 years of the palsy. Patients with paralysis who have undergone serial clinical and/

or electromyographic testing that has failed to show any functional recovery by 6 months should be considered for a reinnervation procedure before significant motor endplate atrophy occurs.

Static Resuspension

1. Direct brow lift
2. Midface lift
3. Functional septorhinoplasty or suspension of external nasal valve
4. Deep plane rhytidectomy
5. Static sling with fascia lata or allograft from zygomatic/temporal region to modiolus and/or lateral upper lip musculature

Note: The modiolus is the point of convergence of the zygomaticus muscles, buccinator muscle, orbicularis oris muscle, and the risorius muscle. It is usually 1 cm lateral to the oral commissure in most individuals [30].

Dynamic Reinnervation or Reanimation

1. Primary nerve repair
2. Nerve grafts
3. Hypoglossal nerve transfer via interposition graft
4. Facial nerve transposition to hypoglossal nerve, end to side coaptation
5. Nerve to masseter transfer
6. Cross facial nerve graft
7. Temporalis tendon transposition
8. Innervated free muscle transfer

Primary Nerve Repair

- Primary tension-free neurorrhaphy of fresh nerve endings is the gold standard.
- Epinerial repair with the minimum number of 10-0 sutures to provide tension-free coaptation is best.

Nerve Grafts

- Commonly used donor nerves include great auricular nerve, sural nerve, lateral antebrachial cutaneous nerve and the anterior division of the medial antebracheal cutaneous nerve.
- The use of a nerve graft produces better clinical results than suturing the two stumps together under tension.
- While it is classically taught that nerve polarity is important in cable grafting, this has been shown not to be the case in several animal studies.

Hypoglossal Nerve Transfer

- Excellent for tone, volitional movement may be limited or synkinetic
- End to side transfer is superior to the use of an interposition graft, due to the need for axons to traverse two sites of coaptation in an interposition graft [31].

Nerve to Masseter Transfer

- Usually coapted to buccal branch for zygomaticus complex reinnervation
- Masseteric nerve is identified in the “subzygomatic triangle” as it exits the infratemporal fossa through the mandibular notch to course anteroinferiorly along the deep part of the masseteric muscle. The triangle which is formed by the zygomatic arch superiorly, the TMJ posteriorly and the frontal branch of the facial nerve inferiorly and anteriorly.
- Popularity is increasing due to ease of dissection, adjacent location, sufficient length and caliber, minimal to no donor site morbidity, and rapid functional recovery.
- The nerve to masseter may be used to drive the nerve to the gracilis in single-stage gracilis-free muscle transfer for facial reanimation.
- High axonal input, *onset to function is most rapid of any re-innervation technique*.

Cross Facial Nerve Graft (CFNG)

- Most commonly, the sural nerve is used due to its reliable anatomy, easy of dissection, minimal morbidity, comparable caliber, and available length.
- In general, axonal ingrowth through this long (~14 cm) avascular nerve graft can be uncertain. Although direct nerve coaptation to a paralyzed facial nerve branch has been performed with success, the failure rate of direct nerve coaptation is significant.
- CFNG is the only technique to restore spontaneous emotional expression.

- Given the long regenerative period of the CFNG, the "babysitter" concept was introduced. Hypoglossal and/or masseteric nerves are used to salvage the paretic facial musculature until the CFNG is ready for substitution so that the contralateral facial nerve synchronizes the facial expression. Alternatively, the CFNG can be connected end-to-side with one of the babysitter nerves as described by the concept of neural supercharge.
- "Tinel's sign" is the tingling sensations produced by transcutaneous percussion of the nerve graft signifying that the nerve has regenerated to the distal end and is ready for transfer.

Gracilis-Free Tissue Transfer

- Neurovascular pedicle is composed of a single arterial branch arising from the adductor branch of the profunda femoris, two venae comitantes, and the obturator nerve.
- May be powered by a cross-facial nerve graft (two-stage procedure) or by a nerve to masseter transfer (single stage procedure). Additionally, the neural supercharge concept can be applied as a two-stage procedure by using both a massteric nerve transfer and a CFNG.
- In general, commissure movement is greater with the use of the nerve to masseter.
- The cross facial nerve graft reestablishes spontaneous movement. Nerve to masseter is reported to provide spontaneous smile in roughly 50% of patients in one case series [32].
- Facial reanimation using the free gracilis muscle transfer improves objective facial symmetry and patient quality of life [33–35].
- Other free functional microvascular transfer muscles include latissimus, pectoralis minor and sternohyoid.

Temporalis Tendon Transposition

- Temporalis muscle originates in the temporal fossa, passes under the zygomatic arch, and inserts on the coronoid process of the mandible. It receives its blood supply from the deep temporal artery and is innervated by the deep temporal nerves that arise from the mandibular division of the trigeminal nerve.
- Excellent for patient with dense paralysis or significant atrophy
- Performed in single surgery, no need for multistage surgery [36–38]
- Through either an external incision at the nasolabial fold or an intraoral incision, the coronoid process is osteotomized to allow the temporalis tendon to be released and reattached to the orbicularis oris muscle.
- During the dissection, the muscle glide planes must be preserved to allow dynamic excursion. These planes include the buccal fat space and region underneath the zygomatic arch.
- If additional length is needed to reach the orbicularis oris muscle, either a fascia graft can be used (most commonly from fascia lata) or a temporalis tendon lengthening myoplasty can be performed.
- In the myoplasty procedure, the temporalis muscle is exposed through a scalp incision and the posterior third of the muscle is released by elevating it from its periosteal attachments. This allows the muscle fibers to be redistributed inferiorly yielding an additional 4–5 cm of length.

Temporalis Muscle Transfer

- Procedure has largely been replaced by the tendon transfer, which results in better cosmetic and functional outcomes.
- Through a facelift incision, the central third of the temporalis muscle is released on 3 sides leaving its base attached. The released strip of muscle is then flipped over the zygomatic arch and tunneled toward the oral commissure to be sutured to the orbicularis oris.
- Besides from the nonphysiologic muscle contraction, patients were left with hollowing of the temporal region and a bulge over the zygomatic arch.

Questions

1. Which branch of the facial nerve is responsible for a normal, spontaneous blink?
   (a) Buccal
   (b) Temporal
   (c) Zygomatic
   (d) Marginal Mandibular

2. Which of the following is *not* an indication for imaging in a patient with acute onset facial paralysis?
   (a) A second episode
   (b) No improvement in symptoms after 3 months
   (c) Numbness of the conchal bowl
   (d) Associated second cranial neuropathy
   (e) Isolated branches of facial nerve affected
3. Which of the following muscles is *not* a contributor to the modiolus in most individuals?
   (a) Buccinator
   (b) Depressor anguli oris
   (c) Zygomaticus muscles
   (d) Risorius
   (e) Orbicularis oris
4. What time period is the upper limit that is considered "acute onset" for a clinical diagnosis of acute idiopathic facial nerve paralysis (Bell's palsy)?
   (a) 5 days
   (b) 4 days
   (c) 3 days
   (d) 2 days
   (e) 1 day
5. What is the most reliable marker for the anatomic location of the main trunk of the facial nerve after it emerges from the stylomastoid foramen?
   (a) Posterior belly of digastric muscle
   (b) Tragal pointer
   (c) Tympanomastoid suture
   (d) External auditory canal
6. All of the following are true regarding the masseteric nerve except:
   (a) Associated with the fastest recovery of facial reanimation
   (b) Located in the submalar triangle
   (c) Harvest leaves the patient with minimal to no morbidity
   (d) Can be used to innervated a free muscle transfer
7. A 35-year-old male had a radical parotidectomy with facial nerve sacrifice 2 years ago. He is now free of cancer but is search for facial reanimation procedure. What is the most effective way to give the patient involuntary emotion?
   (a) Temporalis tendon transfer
   (b) Gracilis free flap innervated by ipsilateral masseteric nerve
   (c) 5–7 transfer for upper division and 12–7 transposition for lower division
   (d) Gracilis free flap innervated by CFNG
8. Which of the following facial nerve branching patterns is most common?
   (a) No anastomoses between the upper and lower divisions
   (b) 1 anastomosis between the upper and lower divisions
   (c) Anastomoses among the upper division only
   (d) 2 anastomoses between the upper and lower divisions
   (e) Multiple complex anastomoses especially involving the buccal branch
9. What is the exam finding that indicates nerve regeneration has reached the end of a nerve?
   (a) Tinel's sign
   (b) Tyndell's sign
   (c) Trousseau sign
   (d) Troisier's sign
   (e) Traube's sign
10. During a temporalis tendon transfer procedure or gracilis free flap, where is the proper insertion site around the mouth?
    (a) Region of the nasolabial fold
    (b) Modiolus
    (c) As close to the lip margin as possible
    (d) None of the above
11. Which of the following is false regarding the frontal branch of the facial nerve?
    (a) The nerve's path can be estimated by Pitanguy's line.
    (b) The nerve pierces the orbicularis muscle 1.5 cm above the lateral eyebrow.
    (c) The nerve lies anterior to the artery.
    (d) The nerve runs on the deep surface of the temporoparietal fascia.
12. The most common cause of epiphora in a patient with facial paralysis is:
    (a) The inferior and lateral deplacement of the inferior punctum along with lid eversion
    (b) The inability to fully close the eye and blink

(c) Increased lacrimation as a result of increased corneal exposure and lacrimal gland ptosis
(d) Lacrimal sac obstruction

13. Which of the following is true regarding Bell's phenomenon?
(a) It is a pathologic finding.
(b) The eye moves upward and inward during eye closure.
(c) All normal babies exhibit the finding.
(d) It is usually present in forcibly closed eyelids.

14. Which cranial nerve assists with eye closure in a patient with facial paralysis?
(a) CN III
(b) CN IV
(c) CN V
(d) CN VI

15. You are called into the operating room by the trauma service after they have identified that the patient, who was a victim of an assault, has a lacerated lower division of the facial nerve. The best course of action is to:
(a) Freshen the edges and perform a direct epinerial repair with minimal sutures
(b) Freshen the edges and perform a direct perinerial repair with several sutures
(c) Tag the two ends and plan for a repair in the future
(d) Reapproximate the nerve endings with tissue glue

16. All of the following are reasons to support the "babysitter" concept except
(a) Facial muscles are innervated immediately to avoid atrophy
(b) CFNG can still be used for spontaneous movement and added neural input
(c) Patients can benefit from a quicker return of facial movement
(d) Patients can use the initial procedure as a trial and can re-evaluate the treatment plan later

17. What is the time limit that will allow the surgeon to utilize the nerve stimulator intraoperatively to assist in identification of distal branches of a severed facial nerve?
(a) 12 hours
(b) 24 hours
(c) 36 hours
(d) 48 hours
(e) 72 hours

18. Which of the following is not a border of the subzygomatic triangle?
(a) External auditory canal
(b) Frontal branch of the facial nerve
(c) Zygomatic arch
(d) Temporomandibular joint

19. All of the following are true regarding Bell's palsy except:
(a) Nearly all patients with incomplete paralysis will have complete recovery
(b) It is the most common case of facial paralysis
(c) Synkinesis occurs in 50% of patients
(d) It is a diagnosis of exclusion
(e) Classic MRI findings include perigeniculate enhancement

20. A 47-year-old female is postop day #5 from acoustic neuroma resection via a retrosigmoid approach. The neurologist states that the facial nerve is intact but signal was lost during the resection. On physical exam, the patient has complete ipsilateral facial paralysis. The best course of action regarding corneal protection is to:
(a) Eye care only with artificial tears, lacrilube, and nighttime taping
(b) Placement of gold weight
(c) Tarsal strip procedure
(d) Combined tarsal strip and gold weight

21. According to a new synkinesis patient, Botox is no longer effective in providing relief; however, she states the last physician used Dysport and it worked well for her. How can this need for switching neurotoxins be explained?
(a) The last physician to use Botox was either injecting in the wrong location or used an insufficient amount.
(b) The patient read incorrect information on the internet and likely requested the change herself.
(c) The last Botox bottle was likely from a defective batch.
(d) The patient developed toxin-blocking antibodies to Botox.

22. Which of the following is true regarding facelifting procedures for facial symmetry in paralysis patients?
    (a) SMAS plication is the preferred method.
    (b) The deep plane technique is superior to all others.
    (c) SMASectomy and imbrication is the preferred method.
    (d) Deep plane and SMASectomy are equally effective.
23. While performing a temporalis tendon transfer procedure, you realize the tendon is 1.5 cm too short to attach to the ideal location. What is your best option?
    (a) Attach the tendon to the nearby tissue
    (b) Perform a myoplasty procedure
    (c) Harvest fascia lata and use it as an extension
    (d) Stretch the tendon/muscle and attach under tension
24. All of the following are true regarding sural nerve harvest except:
    (a) It can be harvested via making stair-step incisions or endoscopically with one incision
    (b) It is located through a small incision posterosuperior to the lateral malleolus
    (c) The nerve is located posterior to the vein
    (d) The nerve does not branch until it reaches the lateral malleolus
    (e) Morbidity is minimal with a small numb area over lateral foot
25. The gracilis free flap is attempting to mimic the motion of what muscle?
    (a) Orbicularis oris
    (b) Risorius
    (c) Zygomatic major
    (d) Levator anguli oris

Answers

1. (a)
2. (c)
3. (b)
4. (c)
5. (c)
6. (b)
7. (d)
8. (b)
9. (a)
10. (c)
11. (b)
12. (a)
13. (d)
14. (a)
15. (a)
16. (d)
17. (e)
18. (a)
19. (c)
20. (b)
21. (d)
22. (b)
23. (c)
24. (c)
25. (c)

References

1. Rea PM, McGarry G, Shaw-Dunn J. The precision of four commonly used surgical landmarks for locating the facial nerve in anterograde parotidectomy in humans. Ann Anat. 2010;192(1):27–32.
2. Pitanguy I, Ramos AS. The frontal branch of the facial nerve: the importance of its variations in face lifting. Plast Reconstr Surg. 1966;38(4):352–6.
3. Davis RA, et al. Surgical anatomy of the facial nerve and parotid gland based upon a study of 350 cervicofacial halves. Surg Gynecol Obstet. 1956; 102(4):385.
4. 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. 1962;29(3):266–72.
5. Chowdhry S, Yoder EM, Cooperman RD, et al. Locating the cervical motor branch of the facial nerve: anatomy and clinical application. Plast Reconstr Surg. 2010;126:875–9.
6. Bleicher JN, et al. A survey of facial paralysis: etiology and incidence. Ear Nose Throat J. 1996;75(6): 355–8.
7. Sanders F, Whitteridge D. Conduction velocity and myelin thickness in regenerating nerve fibres. J Physiol. 1946;105(2):152–74.
8. Sunderland S, Williams HB. Nerve injuries and their repair: a critical appraisal. Plast Reconstr Surg. 1992;89(6):1170.
9. House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg. 1985;93(2):146.
10. Ross BG, Fradet G, Nedzelski JM. Development of a sensitive clinical facial grading system. Otolaryngol Head Neck Surg. 1996;114(3):380–6.

11. Hontanilla B, Aubá C. Automatic three-dimensional quantitative analysis for evaluation of facial movement. J Plast Reconstr Aesthet Surg. 2008;61(1):18–30.
12. May M, Blumenthal F, Klein SR. Acute Bell's palsy: prognostic value of evoked electromyography, maximal stimulation, and other electrical tests. Otol Neurotol. 1983;5(1):1–7.
13. Sullivan FM, et al. Early treatment with prednisolone or acyclovir in Bell's palsy. N Engl J Med. 2007;357(16):1598–607.
14. Peitersen E. Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. Acta Otolaryngol Suppl. 2002;549:4–30.
15. Baugh RF, et al. Clinical practice guideline Bell's palsy. Otolaryngol Head Neck Surg. 2013;149(3 Suppl):S1–27.
16. Djalilian HR. Bell's palsy. N Engl J Med. 2005;352(4):416–8. author reply 416–8
17. El-Kashlan H, Kileny P. Outpatient facial nerve evoked electromyography testing. Curr Opin Otolaryngol Head Neck Surg. 1998;6(5):334–7.
18. Sinno H, Thibaudeau S, Izadpanah A, et al. Utility outcome scores for unilateral facial paralysis. Ann Plast Surg. 2012;69(4):435–8.
19. Chepeha DB, et al. Prospective evaluation of eyelid function with gold weight implant and lower eyelid shortening for facial paralysis. Arch Otolaryngol Head Neck Surg. 2001;127(3):299–303.
20. Pepper JP, Kim JC. A surgical algorithm for lower eyelid resuspension in facial nerve paralysis. Oper Tech Otolaryngol. 2012;23(4):248–52.
21. Crumley RL. Mechanisms of synkinesis. Laryngoscope. 1979;89(11):1847–54.
22. Blomstedt GC, et al. Recovery of the sutured facial nerve after removal of acoustic neuroma in patients with neurofibromatosis-2. Neurosurgery. 1994;35(3):364–9.
23. Yamada H, et al. Facial synkinesis after experimental compression of the facial nerve comparing intratemporal and extratemporal lesions. Laryngoscope. 2010;120(5):1022–7.
24. Husseman J, Mehta RP. Management of synkinesis. Facial Plast Surg. 2008;24(2):242–9.
25. Pepper JP, Kim JC, Massry GG. Selective chemodenervation with botulinum toxin in facial nerve disorders. Oper Tech Otolaryngol. 2012;23(4):297–305.
26. Choi D, Raisman G. Somatotopic organization of the facial nucleus is disrupted after lesioning and regeneration of the facial nerve: the histological representation of synkinesis. Neurosurgery. 2002;50(2):355–63.
27. Beurskens CH, Heymans PG. Physiotherapy in patients with facial nerve paresis: description of outcomes. Am J Otolaryngol. 2004;25(6):394–400.
28. Beurskens CH, Heymans PG. Mime therapy improves facial symmetry in people with long-term facial nerve paresis: a randomised controlled trial. Aust J Physiother. 2006;52(3):177–83.
29. Borodic G, et al. Botulinum toxin for aberrant facial nerve regeneration: double-blind, placebo-controlled trial using subjective endpoints. Plast Reconstr Surg. 2005;116(1):36–43.
30. Pepper JP, Baker SR. Local flaps: cheek and lip reconstruction. JAMA Facial Plast Surg. 2013;15(5):374–82.
31. Bertelli JA, et al. Long interpositional nerve graft consistently induces incomplete motor and sensory recovery in the rat. An experimental model to test nerve repair. J Neurosci Methods. 2004;134(1):75–80.
32. Manktelow RT, et al. Smile reconstruction in adults with free muscle transfer innervated by the masseter motor nerve: effectiveness and cerebral adaptation. Plast Reconstr Surg. 2006;118(4):885–99.
33. Lindsay RW, Robinson M, Hadlock TA. Comprehensive facial rehabilitation improves function in people with facial paralysis: a 5-year experience at the Massachusetts Eye and Ear Infirmary. Phys Ther. 2010;90(3):391–7.
34. Lindsay RW, et al. The success of free gracilis muscle transfer to restore smile in patients with nonflaccid facial paralysis. Ann Plast Surg. 2014;73:177–82.
35. Bhama PK, et al. Objective outcomes analysis following microvascular gracilis transfer for facial reanimation: a review of 10 years' experience. JAMA Facial Plast Surg. 2014;16(2):85–92.
36. Byrne PJ, et al. Temporalis tendon transfer as part of a comprehensive approach to facial reanimation. Arch Facial Plast Surg. 2007;9(4):234–41.
37. Boahene KD, et al. Minimally invasive temporalis tendon transposition. Arch Facial Plast Surg. 2011;13(1):8–13.
38. Boahene K. Principles and biomechanics of muscle tendon unit transfer: application in temporalis muscle tendon transposition for smile improvement in facial paralysis. Laryngoscope. 2013;123(2):350–5.

Additional Resources

Adour KK, Byl FM, Hilsinger RL Jr, et al. The true nature of Bell's palsy: analysis of 1,000 consecutive patients. Laryngoscope. 1978;88:787–801.

Bain JR, Hason Y, Veltri KL, et al. Clinical application of sensory protection of denervated muscle: case report. J Neurosurg. 2008;109:955–61.

Bascom DA, Schaitkin BM, May M, et al. Facial nerve repair: a retrospective review. Facial Plast Surg. 2000;16:309–13.

Bénateau H, et al. Anatomic study of the tendinous insertion lamina of the temporalis muscle. Surg Radiol Anat. 2004;26(4):281–4.

Collar RM, Byrne PJ, Boahene KD. The subzygomatic triangle: rapid, minimally invasive identification of the masseteric nerve for facial reanimation. Plast Reconstr Surg. 2013;132(1):183–8.

De Diego JI, Prim MP, Madero R, et al. Seasonal patterns of idiopathic facial paralysis: a 16-year study. Otolaryngol Head Neck Surg. 1999;120:269–71.

Kim J. Neural reanimation advances and new technologies. Facial Plast Surg Clin North Am. 2016;24(1):71–84.

Labbe D, Huault M. Lengthening temporalis myoplasty and lip reanimation. Plast Reconstr Surg. 2000;105(4):1289–97. discussion 1298

Labbe D, Bussu F, Iodice A. A comprehensive approach to long-standing facial paralysis based on lengthening temporalis myoplasty. Acta Otorhinolaryngol Ital. 2012;32(3):145.

Nakatsuka H, Takamatsu K, Koshimune M, et al. Experimental study of polarity in reversing cable nerve grafts. J Reconstr Microsurg. 2002;18(6):509–15.

Nduka C, Hallam MJ, Labbe D. Refinements in smile reanimation: 10-year experience with the lengthening temporalis myoplasty. J Plast Reconstr Aesthet Surg. 2012;65(7):851–6.

Peitersen E. The natural history of Bell's palsy. Am J Otol. 1982;4:107–11.

Terzis JK. "Babysitters": an exciting new concept in facial reanimation. The facial nerve. In: Castro D, editor. Proceedings of the Sixth International Symposium of the Facial Nerve. Rio de Janeiro (Brazil); 1988.

Yamamoto Y, Sekido M, Furukawa H, et al. Surgical rehabilitation of reversible facial palsy: facial-hypoglossal network system based on neural signal augmentation/neural supercharge concept. J Plast Reconstr Aesthet Surg. 2007;60(3):223–31.

9 Mohs Surgery

Sim Choroomi and David Sarcu

Introduction

Mohs micrographic surgery (MMS) is a stepwise surgical technique of skin cancer extirpation named after Dr Frederik Mohs, from the University of Wisconsin, who conceptualized the technique in the 1930s. His technique was later modified by Stegman and Tomovich to today's current method.

MMS combines surgical excision with real-time histopathology analysis of margins. The components of the procedure include surgical excision, histopathological examination, precise tumor mapping, and confirmation of tumor clearance, followed by reconstruction and wound management.

MMS is considered to have the most optimal cure rates for cutaneous BCC and SCC compared with other treatment modalities. From a practical perspective, MMS is best indicated for select cases of skin cancers, particularly in the head and neck region where recurrence rates are highest and aesthetic reconstruction is often complex.

S. Choroomi
Facial Plastic Surgery, The Lasky Clinic, Beverly Hills, CA, USA

D. Sarcu (✉)
Facial Plastic and Reconstructive Surgery, Advocate Medical Group, Aurora, IL, USA
e-mail: david.sarcu@advocatehealth.com

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_9

Overview of Skin Cancers

Skin cancer is the most common form of malignant neoplasm in the United States, with an estimate one in five people having a lifetime chance of developing the disease. With over three million cases diagnosed annually, there has been a dramatic increase in the number of cases being diagnosed in recent times. The lesions with the highest incidence include basal cell carcinoma (BCC), squamous cell carcinoma (SCC), and malignant melanoma, with the following incidence:

- BCC: 80%
- SCC: 15%
- Melanoma: 5%
- Others < 1%

Etiology of Skin Cancers

- UV light (UVB wavelengths 280–320 nm most damaging, synergistic with UVA bulbs from tanning beds)—causes mutation of tumor suppressor genes
- H-zone of face (Fig. 9.1)—high risk and recurrence rate in this region

Fig. 9.1 H-zone of the face. The *gray-shaded area* highlights the high-risk "H-zone" of the face. The "H-zone" includes the nasolabial folds, floor of the nose, columella, preauricular region, and inner/outer canthus of the eye

Risk Factors

- Fair complexion—melanin protects skin from UV radiation effects:
  - Fitzpatrick type I or II skin
    Light hair
    Blue/green eyes
    Inability to tan
- Other risk factors:
  - History of multiple/severe sunburn
  - Celtic ancestry
  - Radiation therapy
  - Burns
  - Ulcers and/or chronic inflammation (Marjolin's ulcers)
  - Occupational exposure to oils, tar, arsenic
  - Immunosuppression (particularly transplant patients)
  - Genetic disorders
    Albinism—variable inheritance
    Xeroderma pigmentosa—autosomal recessive
    Gorlin's syndrome (nevoid BCC syndrome)—autosomal dominant

Basal Cell Carcinoma (BCC)

Occur mainly in the head and neck and sun-exposed areas. Malignant, slow growing, rarely metastasizes (<0.5% rate). Locally invasive and may have perineural involvement.

Pathology

Tumor cells originate in the epidermis; the cells appear histologically similar to the basal layer of the epidermis without intracellular bridges. The diagnostic histological features, common for all types of BCC, are basaloid cells with a thin pale cytoplasm surrounding round or oval nuclei with a rough granulated chromatin pattern. The peripheral borderline cell layers are characterized by palisade arrangement.

Subtypes of BCC

- *Nodular*—Most common. Lesions are discrete, dome-shaped with a "pearly" surface and may have telangiectasia. The borders may be rolled and central necrosis may occur. Histology shows islands and sheets of tumor cells. Subtypes: Pigmented and cystic nodular BCC. Pigmented nodular lesions may resemble nevi and melanoma.
- *Superficial*—Multicentric epithelial pink-red patches with scaly appearance; more common on the trunk and extremities. Normal skin may exist between adjacent lesions.
- *Morpheaform/Sclerosing*—Most aggressive form. Occurs frequently in the head and neck with plaque-like appearance and ill-defined borders. Insidious and infiltrating growth pattern with extensions to deep dermis and therefore has higher recurrence rates.

Squamous Cell Carcinoma (SCC)

Occurs most frequently in the head and neck; also on sun-exposed extremities. More aggressive and likely to metastasize than BCC (2–5%);

therefore, the draining lymph nodes must be assessed. May arise from preexisting lesions, namely, actinic keratosis, ulcers, chronic inflammation, or in situ SCC. Clinical changes are noted to occur when malignant transformation occurs—typically increased size, bleeding, and ulceration. In situ SCC may progress to SCC with a 30% risk over time.

Pathology

Macroscopically, the lesions appear as superficial crusting, erythematous with a granular friable base. Whether occurring de novo or from a preexisting lesion, histologically SCC demonstrates irregular masses of epidermal cells penetrating the basement membrane to reach the dermis. Keratinization may occur in well-defined low-grade lesions and less so in high-grade SCC.

Subtypes of SCC

- *Verrucous*—Uncommon on skin of the head and neck (more common on mucosal surfaces). Cauliflower-like, white lesions, minimal atypia
- *Spindle*—Anaplastic cells, little keratinization, spindle cells intermingled with collagen
- *Adenosquamous* cell carcinoma—Tubular and adenoid appearance
- *Basaloid squamous*—Aggressive, high-grade lesion

Keratoacanthoma (KA)—Though technically not a subtype of SCC, KAs are thought to be a low-grade SCC. They typically rapidly grow in sun-exposed area, into a nodule with central ulceration and keratin plug. They may spontaneously involute. Surgical excision is needed to confirm histopathology and margins.

Melanoma

The most deadly skin cancer; although only 5% of skin cancers, it causes 75% of deaths from skin cancer. Incidence is increasing, occurring in 20:100,000. Risks are mainly due to fair complexion and amount of sun exposure, with 10% of melanoma occurring in familial melanoma syndromes.

Pathology

Melanoma most commonly occurs on the skin, although oral, anogenital mucosa, esophagus, meninges, and the eye occur more rarely. Lesions arise at dermo-epidermal junction.

Two growth patterns occur: radial and vertical. *Radial* growth usually occurs first, spreading horizontally within the epidermis/superficial dermis with low capacity to metastasize. *Vertical* growth represents spread deeper into dermal layers. Cellular maturation decreases, cells become smaller, and clinical nodule develops. Extent (and timing) of vertical growth determines metastatic potential.

Subtypes of Invasive Melanoma

- *Superficial spreading*—Most common, accounting for about 70% of all cases. Flat, slightly elevated with variable pigment. Radial and superficial growth pattern.
- *Nodular*—Smooth, single-colored (brown or black) elevated nodule, minimal radial growth, may ulcerate. Thickest of all melanoma subtypes, correlation between thickness and prognosis.
- *Lentigo maligna melanoma (LMM)*—Slow, radial growth pattern. Typically arises from long-standing pigmented lesions on chronic skin damaged skin. May be hypopigmented. Lentigo maligna (LM) is a form of melanoma in situ (noninvasive).
- *Acral lentigo*—More common in darker-skinned individuals on palms and soles of feet. Aggressive growth pattern.
- *Desmoplastic*—Uncommon, unremarkable plaque or nodule which can be easily misdiagnosed early on. *Neurotropic*, locally aggressive but lower incidence of nodal metastasis.

Diagnosis—Full-thickness excisional biopsy with 1–2 mm margin for suspicious lesions or multiple punch biopsies of larger lesions. Shave biopsies should not be performed. Lymph nodes must be clinically examined as well as full skin exam as well as staging scans.

Nonsurgical Treatment of BCC and SCC Lesions

All therapies aim to remove or destroy primary tumor + some adjacent normal tissue:

- Curettage—lesion and small margin removed by feel
  - For <1 cm low-risk BCC.
  - If the subcutaneous tissue is reached, then surgical excision should be performed.
- Topical therapy
  - Imiquimod 5% cream—induces cytokines related to cell-mediated immune response, such as interferon alpha. Used five to seven times per week for a period of 6 weeks on superficial BCCs. Local skin reactions are common but well tolerated. The initial clearance rate at 12 weeks posttreatment is about 95%. Recurrence rate up to 20%.
  - 5-Fluorouracil (5-FU) cream is used for the treatment of superficial BCCs and actinic keratosis. Treat with 5% 5-FU cream twice daily for up to 12 weeks. Treatment is stopped sooner if the lesion is clinically resolved.
- Cryosurgery
  - Treatment of choice for isolated, well-defined actinic keratoses
  - Need temp < −50 °C to cause cell death, leaves an open wound
  - Useful when there is underlying cartilage as it is spared
  - Best for low-risk tumors with well-defined borders. Leaves scarring and hypopigmentation
- Radiation therapy
  - For wide-field tumors
  - For high-risk malignancies in poor operative candidates
  - High cure rate but lengthy treatment and radiation side effects
- Photodynamic therapy
  - A selective noninvasive therapy for non-hyperkeratotic actinic keratoses, in situ squamous cell carcinoma, and superficial and thin nodular basal cell carcinomas
  - Photosensitizing drug (porphyrin, 5-ALA) applied either topically, IV, or oral
  - Activate drug by exposure to laser or LED light
  - May be used for palliation of advanced lesions

Surgical Excision of Lesions

Numerous factors are taken into account for each lesion, as seen in Table 9.1. Excision margins for SCC and BCC are generally based on the size and histology of the lesion, with higher-grade lesions requiring a wider margin (Table 9.2). Location of the lesion may have some influence on margins taken, given potential functional outcomes post excision; however, priority should be on tumor clearance.

Table 9.1 Surgical treatment—determining factors for surgical treatment of cutaneous malignancy

Patient factors
Age General health Wound care post-op
Disease factors
Type of skin tumor Size and location of lesion Histopathological subtype and grade of lesion Primary tumor vs recurrent Presence of perineural involvement Prior treatment of lesion Presence of lymph nodes
Institutional factors
Physicians skills and competence with modalities of treatment Access and cost of resources Cosmetic and functional outcomes Cure rates for different modalities Access to adjunctive treatments

Table 9.2 Surgical margins for standard excision

	Margin (mm)
BCC	
Lesion <1 cm	4–5
Lesion >1 cm	5–10
Lesion >2 cm	15–20
SCC	
Low-risk lesions	4–5
High-risk lesions	6–10

Mohs

The concept of Mohs micrographic surgery (MMS) is a stepwise microscopic control of peripheral and deep margins during the procedure, to allow for maximal cure rates with minimal removal of normal skin.

To implement these two factors—tumor removal and normal skin preservation—every step of the MMS process needs to be meticulous, from the doctor's assessment to the technician's preparation of histopathology slides.

Given the variety of pathologies, particularly in cases of recurrent disease and aggressive tumors, MMS allows the surgeon to search deceiving lesions which initially may present as only the "tip of the iceberg." Some cases will have lesions with clinically easy-to-identify borders, while others may require peripheral and deep margins to be "chased" until clear.

A tailored approach is taken in MMS, with there being a relative importance of normal tissue preservation, depending on the patient's age, skin laxity, location of the tumor, and other factors (Table 9.3). A cheek lesion on an elderly male with lax skin will certainly be treated differently to a periorbital lesion in a young female, for example.

Tumors Treated by MMS

1. BCC
2. SCC
3. Melanoma in situ (MIS)—with reported 5-year recurrence rates of 0–7%, being superior to traditional surgical excision

Table 9.3 Indications for MMS in non-melanoma skin cancer

Tumor characteristics

Facial tumors, particularly the H-zone, periorbital lesions, ears
Recurrent lesions
Size >1 cm on the neck or face
Large lesions (>2 cm) on the body
Aggressive histopathology subtypes
  Morpheaform, micronodular, and infiltrating BCC
  High-grade, poorly differentiated, or infiltrating SCC
  Spindle-cell SCC
Poorly defined borders clinically
Lesions with perineural invasion
Mucosal lesions—oral cavity, paranasal sinuses

Background skin characteristics
Radiation-induced tumors Tumors developed in an old scar or ulcer (Marjolin's) Previous positive margins/reconstruction
Patient factors
Tumors in patients with familial tendency—xeroderma pigmentosa, BCC syndromes Immunocompromised patients

4. Other rare malignant skin lesions (such as dermatofibrosarcoma protuberans). Long-term follow-up necessary as recurrences have occurred after 5 years

Importance of H-Zone of the Face and Embryonic Fusion Planes

Of particular importance for a subgroup of difficult and recurrent BCC is understanding that some lesions have silent, subclinical contiguous extension, which tends to follow the "path of least resistance." Extensions of these lesions have an affinity for the fascial planes, periosteum, perichondrium, dermis, nerve sheaths, blood vessels, tarsal plates, and embryonic fusion plane sites.

An example is a BCC which spreads from the skin of the pinna to the perichondrium and then deeper to the periosteum and can travel down the external auditory canal.

The mode of spread at sites of embryonic fusion planes is perpendicular to the skin surface and has three important sites in the head and neck:

1. Preauricular at tragus
2. Along the columella
3. Junction of the nasal ala and nasolabial folds

BCCs located in fusion lines tend to infiltrate deeper and have a higher recurrence rate.

The high-risk H-zone (Fig. 9.1) incorporates these sites and the Mohs surgeon must be aware of subtle spread in these sites. The ala-nasolabial fold region may spread down a plane of the nasal septum, and lesions of the inner canthus may have deep extension to the ethmoid sinuses. To maximize cure and minimize normal skin loss, Mohs is indicated in this region, as reconstruction for function and cosmesis are imperative in these sites.

Pre-op Assessment Prior to MMS

- Full medical and surgical history
- Photo-documentation of lesion
- Consultations with other specialties as needed—Head and neck, oculoplastics, reconstructive
- Smoking/alcohol history
- Allergies—medicines, dressings, tape
- Presence of implants/defibrillators (use of cautery)
- Social history—important for post-op wound care
- Psych input for larger lesions and potential defects

MMS Procedure

MMS is stepwise, where the surgeon serves as his or her own histopathologist (Fig. 9.2). This requires specialized training, staff, and resources. The procedure is typically carried out in an outpatient setting over the course of a day, under local anesthesia, though sedation or general anesthesia may be used in some cases:

- The area is prepped with betadine or other antiseptic agents.
- Tumor boundary is marked with a pen.
- Local anesthesia, typically 1% lidocaine with epinephrine, is infiltrated.
- Obvious clinical tumor is removed, centrally debulked, and analyzed. The boundary however is the key for surgical excision.
- The initial boundary is incised with a scalpel at 45° to the skin with a 1–2 mm margin. This allows for orientation. A single, smooth peripheral cut is made circumferentially around the lesion.
- The area is marked with ink or is nicked with a blade, usually at 3, 6, 9, and 12 o'clock to orientate the lesion.
- A blade or scissor is then used on the deep layer, as it is then lifted and dissected carefully *parallel* to the base of the plane of the tumor.
- A Mohs "map" is drawn by the surgeon (such as quadrants in Fig. 9.2) based on the orientation of the specimen.
- The specimen is transferred to the histology lab.
- The open wound is dressed.

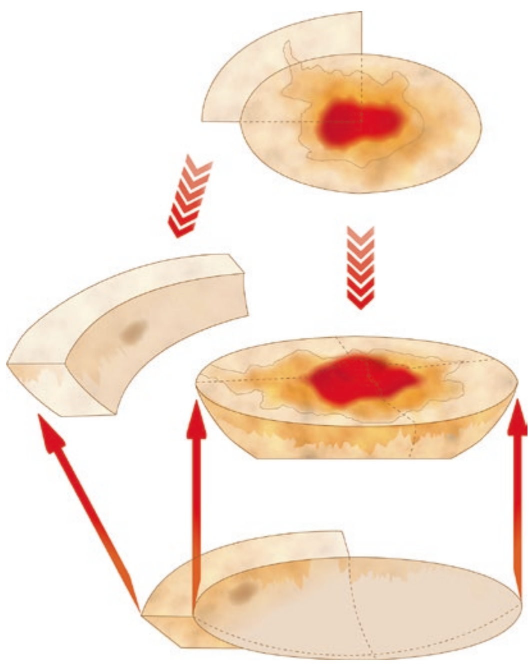


Fig. 9.2 Mohs technique. This figure depicts the principles of Mohs excision, the Mohs map (made of four quadrants here), and re-excising one positive quadrant to achieve clear margins. (By Amir M. Badiee, Beeware Graphics)

Processing/Interpretation

- The surgeon is the histopathologist, being unique to the Mohs process. Critical information is not lost in time between two physicians; rather, the surgeon has a clear intimate understanding of the lesion.
- Also unique to Mohs is the specimen is not cut vertically (e.g., it is not "bread-loafed" as in typical histology slides), but rather is processed horizontally. It is estimated that only 1% of a lesion is evaluated in standard vertical sectioning histopathology.
- The histo-technician has key steps to orientate, embed, fix, freeze, section, mount, and stain the specimen. Each step is critical for accurate evaluation. Orientation is always maintained with the lesion flattened and kept parallel to the deep margin.
- Sectioning and staining are performed in-house and fixed to glass slides and stained with H + E for evaluation.
- Horizontal sections are evaluated to ensure edges are clear.
- If an area is positive, the surgeon goes back to the "map" made, and more tumor is removed from only this area, thus maximally preserving normal skin in other segments (Fig. 9.2).
- The process is repeated until all the margins are clear.
- The defect can be reconstructed immediately or staged.
- Staged reconstruction calls for wound care and dressings (as per leaving a wound to heal by secondary intension) and is typically performed in a matter of days.

Variations in the Mohs Technique

- Single section method—for shallow lesions, highly beveled specimen, usually left for secondary healing
- Wide excision Mohs—wider margin taken on higher-risk lesions to prioritize tissue processing speed

Deep and Bone Involvement

Bone involvement is also seen with difficult cancers in certain sites, such as a desmoplastic SCC of the scalp or a high-grade BCC over the maxillary sinus. In addition, an SCC or BCC with perineural invasion can gain access to the bony canal through which the nerves pass.

Although bony invasion is infrequent, when it does occur, the following strategies are used to control its growth: (1) establish the diagnosis and mode of invasion of the tumor histologically; (2) obtain imaging to assess both local and regional (namely, lymph node) involvements; and (3) refer to a related subspecialist for wide excision and reconstruction as indicated.

Even when bone is exposed during tumor surgery, healing by second intention is possible. Other options include coverage with an acellular dermal matrix or a split-thickness skin graft. These three methods of wound management may be preferred over more complex flaps that may hide and bury residual tumor.

Cure Rates and Outcomes of MMS

MMS is widely accepted as offering the highest cure rates of all modalities of treatment for skin cancers (Tables 9.4 and 9.5). The resources needed and escalating cost of medical care do lead to a shift from evidence-based practice to cost-effective care. Certainly not all skin cancers need to be treated by MMS, and many may be successfully treated with satisfactory oncological

Table 9.4 Reported cure rates with MMS

BCC	99% primary 90–93% recurrent
SCC	92–99% primary 90% recurrent
SCC in situ	90%
Keratoacanthomas	98%
Melanoma in situ	98%
Merkel cell carcinoma	84%

Table 9.5 Five-year cure rate for primary BCC and SCC per modality of treatment

Treatment modality	5-year cure rate	
	BCC %	SCC %
Standard surgical excision	90	92
Electrodissection and curettage	92	96
Radiation	91	90
Cryotherapy	92	n/a
All non-MOHS	91	92
Mohs surgery	99	97

and cosmetic outcomes with standard excision or otherwise.

One large RCT comparing facial BCCs, randomized to be treated with either Mohs or standard excision, did report a lower recurrence rate at follow-up with Mohs, although figures were not statistically significant. The cost of Mohs was significantly higher, being almost twice as costly. The tables below outline the effectiveness of Mohs.

Considerations for Reconstruction of Mohs Defects

All factors already discussed, including both patient factors (namely, age, skin type) and lesion factors (size, location, and need for surveillance), must be taken into consideration. Reconstruction can be performed immediately or delayed.

The following reconstructive ladder approach can be applied to all defects:

- Secondary healing
- Primary closure
- Grafts—split skin, full thickness, composite
- Flaps—local, distal, regional/axial
- Free tissue transfer

Most Mohs defects will be either left to heal via secondary healing or closed using primary closure, grafts, or local tissue transfer. Anticipation of future forces of scar contracture may require structural reinforcement prior to skin closure (i.e., cartilage reinforcement of nasal sidewall or tip reconstructions or lamella reinforcement during eyelid reconstruction).

Secondary Intention Healing and Wound Care

Secondary intention healing may result in excellent cosmetic results in areas that are concave, such as the temple and ala-nasolabial groove. Particularly for recurrent tumors in which flaps or grafts may hinder surveillance of the treatment site, secondary intention will allow monitoring and allow for delayed reconstruction if need be.

Patients must be willing to meticulously cleanse and monitor the area over a 2- to 8-week healing period. Typically, wound care regimens involve cleansing the wound daily with a saline solution or soapy water, applying a layer of ointment, and then covering with a nonadherent dressing. Wound care after cutaneous reconstruction is similar to that of secondary intention healing, although limited to 1–2 weeks.

Primary Closure

For small lesions, primary closure is typically undertaken. Consideration of relaxed skin tension lines (RSTL) and minimizing tension, particularly in critical functional and aesthetic areas like the periorbital region, is of high importance. RSTL result from the orientation of collagen intrinsic to facial skin and manifest in the aging face as creases and rhytids.

Mohs defects ready for primary closure should be fashioned and orientated to sit parallel to the facial RSTL so that any undermining performed will be perpendicular to this and therefore tension will be distributed in the lines of maximal extensibility (LME).

Adjacent Tissue Transfer

When primary closure cannot be accomplished, defects are often able to close using local tissue transfer (random flaps) or regional flaps (i.e., forehead flap). These techniques are discussed in detail in the chapter Flaps and Grafts; examples are in Table 9.6.

Table 9.6 Examples of flaps often used for Mohs reconstructions

Nose	Bilobe, forehead rotation flap
Lip	Abbe-Estlander, Karapandzic, melolabial, vermillion advancement
Cheek	Rhomboid, cervicofacial rotation advancement

Challenges in Mohs Surgery

Excisional

- Patient skin characters—thickness, color, contour, and severely sun-damaged skin
- Sites of complex tissue components—muscle, cartilage, bone
- Fatty tissue—requires intense freezing, may lead to freeze artifacts
- Excessive use of cautery—may lead to artifacts in histological analysis

Lab Processing + Interpretation

- Improper cutting—too thick/thin or folded specimen.
- Poor-quality staining with H&E may compromise tumor cell recognition.
- Inability to recognize small tumor foci.

Complications and Pitfalls of Mohs

Complications are overall uncommon. As with all surgeries, the risk of bleeding, nerve damage, infection, pain, and allergic reaction exist. Reconstructive complications include hematoma, wound dehiscence, skin necrosis, poor cosmetic or functional outcome, as well as tumor recurrence adjacent or deep to reconstruction.

Thorough preoperative evaluation of anticoagulants, use of cautery intra-op, and post-op bandaging will minimize bleeding risks. The patient should be counselled pre-op about any possible major sensory or motor nerve loss, particularly for lesions in the temporal region.

Infection rates post MMS are <1%, and postoperative antibiotics should be considered for cases adjacent to the nares or mouth, as well as in cases of multiple or large reconstruction.

Future Trends

Continuous innovation in this field is occurring given the rise of skin carcinomas. The use of perioperative confocal scanning laser microscopy and digital technology allowing for telepathology confirmation is being explored. New immunohistochemistry stains are being discovered and produced to enhance the specificity and sensitivity of pathologic tissue analysis which will aim to further improve both detection and cure rates.

Questions

1. A 29-year-old patient presents with a lesion on the right cheek with pearly raised edges with telangiectasias. On further questioning, he reports having a cyst of his jaw operated on 3 years ago. He reports having a history of four basal cell carcinomas on his body. What is the most likely inheritance of his condition?
   (a) Autosomal recessive
   (b) Autosomal dominant
   (c) Sex-linked recessive
   (d) Non-Mendelian
2. Which of the following is a risk factor for squamous cell carcinoma only?
   (a) Previous burn
   (b) Light-colored eyes
   (c) Immunosuppression
   (d) Actinic keratosis
3. Which of the following is the most common type of basal cell carcinoma?
   (a) Nodular
   (b) Superficial
   (c) Morpheaform/sclerosing
   (d) Desmoplastic
4. Which of the following is the most common type of melanoma in situ?
   (a) Superficial spreading
   (b) Nodular
   (c) Lentigo maligna
   (d) Desmoplastic

5. Which of the following is the most aggressive subtype of squamous cell carcinoma?
   (a) Verrucous
   (b) Spindle
   (c) Adenosquamous
   (d) Basaloid squamous
6. Which of the following biopsy techniques is contraindicated if melanoma is suspected?
   (a) Incisional
   (b) Excisional
   (c) Shave
   (d) Wide local excision
7. Which of the following is *not* an accepted therapy for basal cell carcinoma or squamous cell carcinoma?
   (a) Cryosurgery
   (b) Radiation
   (c) 5-ALA
   (d) Brachytherapy
8. Which of the following is *not* an indication for Mohs micrographic surgery in non-melanoma skin cancer?
   (a) Size >1 cm on the neck or face
   (b) Perineural invasion
   (c) Mucosal lesion
   (d) Lesion in submental crease
   (e) Dermatofibrosarcoma protuberans
9. What is an acceptable margin range for a low-grade basal cell carcinoma 1.5 cm in size?
   (a) 2–4 mm
   (b) 5–10 mm
   (c) 11–15 mm
   (d) 16–20 mm
10. Which of the following areas is *not* a part of the H-zone?
   (a) Preauricular
   (b) Postauricular
   (c) Medial canthus
   (d) Forehead
11. What is the direction of spread of skin cancers at the embryonic fusion plate with respect to the skin surface?
   (a) Perpendicular
   (b) Parallel
   (c) Random
   (d) Radially
12. Which of the following lesions has the highest concern for recurrence?
   (a) 1 cm basal cell carcinoma of the columella
   (b) 1 cm squamous cell carcinoma in situ left cheek
   (c) 0.5 cm depth melanoma right neck
   (d) 1.5 squamous cell carcinoma with ulceration
13. What is the plane of sectioning during Mohs micrographic surgery?
   (a) Perpendicular
   (b) Parallel
   (c) 45 degree angle
   (d) 90 degree angle
14. In which of the following lesions are the margins hardest to assess with Mohs micrographic surgery?
   (a) Basal cell carcinoma
   (b) Squamous cell carcinoma
   (c) Melanoma
   (d) Dermatofibrosarcoma protuberans
15. Which of the following has the lowest cure rate with Mohs micrographic surgery?
   (a) Melanoma in situ
   (b) Keratoacanthoma
   (c) Squamous cell carcinoma
   (d) Merkel cell carcinoma
16. Which of the following areas could potentially be allowed to heal by secondary intention with acceptable cosmesis?
   (a) Alar facial groove
   (b) Forehead
   (c) Lateral canthus
   (d) Tragus
17. Which of the following patients would be best suited by treatment with primary radiation?
   (a) 1.5 cm squamous cell carcinoma of left auricular helix in 80-year-old male with poorly controlled CHF
   (b) 2 cm squamous cell carcinoma of right medial canthus in 45-year-old female with hypertension

(c) 1 cm basal cell carcinoma of left postauricular region in 67-year-old factory worker
(d) 2 cm squamous cell carcinoma left cheek with left facial nerve weakness

18. Which of the following is a risk associated with cryosurgery in the treatment of skin cancers?
(a) Allergic reaction
(b) Hypopigmentation
(c) Hyperpigmentation
(d) Infection

19. Which skin lesion is imiquimod traditionally used for?
(a) Basal cell carcinoma
(b) Squamous cell carcinoma
(c) Melanoma
(d) Rhabdomyosarcoma

20. What is the lifetime risk of progression to invasive squamous cell carcinoma for in situ squamous cell carcinoma?
(a) 20%
(b) 30%
(c) 40%
(d) 50%

21. Which lesion is characterized histologically by keratin pearls?
(a) Basal cell carcinoma
(b) Squamous cell carcinoma
(c) Melanoma
(d) Angiosarcoma

22. Which of the following characteristics of melanoma is most important in determining metastatic potential?
(a) Radial growth
(b) Vertical growth
(c) Ulceration
(d) Subtype

23. Which of the following is more common on the lower lip as compared to the upper lip?
(a) Basal cell carcinoma
(b) Squamous cell carcinoma
(c) Melanoma
(d) Angiosarcoma

24. Which form of radiation causes mutation of tumor suppressor genes leading to many skin cancers?
(a) UVA
(b) UVB
(c) Gamma rays
(d) X-rays

25. Which of the following pathologies is an indication for Mohs micrographic surgery?
(a) Angiosarcoma
(b) Rhabdomyosarcoma
(c) Ulcerated melanoma
(d) Sclerosing basal cell carcinoma

Answers

1. (b)
2. (d)
3. (a)
4. (c)
5. (d)
6. (c)
7. (d)
8. (d)
9. (b)
10. (d)
11. (a)
12. (a)
13. (b),
14. (c)
15. (d)
16. (a)
17. (a)
18. (b)
19. (a)
20. (b)
21. (b)
22. (b)
23. (b)
24. (b)
25. (d)

Additional Resources

1. Becker GD, et al. Secondary intention healing of exposed scalp and forehead bone after Mohs surgery. Otolaryngol Head Neck Surg. 1999;121:751–4.
2. Kwon S, Miller S. Mohs surgery for melanoma in situ. Dermatol Clin. 2011;29:175–83.
3. Narayanan K, Hadid OH, Barnes EA. Mohs micrographic surgery versus surgical excision for periocular basal cell carcinoma. Cochrane Database Syst Rev. 2012;(2).
4. Smeets NW, et al. Surgical excision vs Mohs' micrographic surgery for basal-cell carcinoma of the face: randomised control trial. Lancet. 2004;364:1766–72.
5. Swanson NA, et al. Mohs surgery: techniques, indications and applications in head and neck surgery. Head Neck Surg. 1983;6:683–92.

Neoplasm and Reconstruction

10

Jeffrey D. Markey and Jon Robitschek

Cutaneous Malignancies (Tables 10.1, 10.2, 10.3, and 10.4)

Mohs Micrographic Surgery (MMS): indicated for BCC and/or SCC cutaneous malignancies in setting of high-risk tumors, recurrent tumors, or areas requiring tissue preservation (eyes, nose, ears, lips).

Merkel Cell Carcinoma (MCC) Rare and highly aggressive (55% nodal involvement) dermal tumor of neuroendocrine origin (mechanoreceptor). Eighty percent secondary to Merkel cell polyomavirus (MCV). Challenging pathologic diagnosis relying on immunohistochemistry (IHC)/electron microscopy (EM) (dendritic core granules). IHC similar to small cell lung carcinoma—neuron-specific enolase (NSE), amine precursor uptake and decarboxylation (APUD). IHC for neurofilament protein is unique to MCC. Over half occur in the head and neck (most common in periorbital sites followed by the cheek/forehead). *Risk factors (RF)*: UV exposure, history of SCC, immunosuppression. *Symptoms (SX):* Painless, singular, rapidly enlarging, purple nodule on sun-exposed skin. *Treatment (TX)*: WLE with 2 cm margins, SLNB encouraged for clinically negative necks, neck dissection for clinically positive disease, and radiotherapy for high-risk tumors. Typical systemic therapy includes cyclophosphamide/doxorubicin.

Cutaneous Angiosarcoma Rare, multifocal, highly aggressive vascular tumor, 2:1 ratio of men to women, half occur in the head and neck. *RF*: Radiotherapy, foreign body (bone wax, Dacron), chronic lymphedema (increased risk among mastectomy patients). *SX*: Painless, rapidly enlarging bruise or blue/purple nodule on the scalp/face. *TX*: Multimodality: WLE with 2–3 cm margins, radiotherapy (80% local control rate), and systemic therapy (doxorubicin).

Microcystic Adnexal Carcinoma (MAC) Rare, locally invasive, low-grade tumor of sweat gland origin with high recurrence rate favoring whites (90% of cases). *RF*: Prior radiotherapy, UV exposure. *SX*: Indolent, yellow, indurated plaque, favoring the upper lip (reported 80% perineural invasion). *TX*: Mohs excision with adjuvant radiotherapy in select cases.

Sebaceous Gland Carcinoma Locally invasive tumor originating predominately from meibomian glands (minority from glands of Zeis) of the upper > lower eyelid; W > M. Common delay in diagnosis with noted foamy cytoplasm, pagetoid

J. D. Markey (✉)
Otolaryngology – Head and Neck Surgery, New York University Langone Health, New York, NY, USA
e-mail: Jeffrey.markey@nyulangone.org

J. Robitschek
Otolaryngology, Brooke Army Medical Center, Fort Sam Houston, TX, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_10

Table 10.1 Basal cell carcinoma, squamous cell carcinoma, and melanoma

	Basal cell carcinoma	Squamous cell carcinoma	Melanoma
General	80% of all cutaneous malignancies; 70% in H&N; nasal tip/ala most common site. Peripheral growth pattern	15% of all cutaneous malignancies; 2:1 M:F; 90% with p53 mutation. Vertical growth	5% of all cutaneous malignancies, 20% occur in H&N; two phases: radial and vertical
Risk factors	Acute, intense, intermittent UV exposure; IS	Cumulative UV exposure. Fitzpatrick types I and II, IS, HPV, chronic infection/inflammation (Marjolin ulcer)	Acute, intense, intermittent UV on typically unexposed skin; Fitzpatrick I and II; high mole prevalence; living near equator/high elevation
Precursor lesion	None	Actinic keratosis	Dysplastic nevus, cellular blue nevus, congenital/acquired nevus
Histologic subtypes	1. Nodular: 70%, often H&N. Pigmented, round, pearly, flesh-colored, telangiectasias 2. Superficial: 20%, often the trunk and shoulders. Erythematous, well-circumscribed, whitish, and scaly (mistaken for psoriasis/fungal/eczema) 3. Infiltrative: aggressive, dermal infiltration, poorly defined borders 4. Micronodular: deceptive and more common dermal spread, without ulceration 5. Morpheaform: white/yellow, waxy, flat, sclerotic plaque, no ulceration. Aggressive, worst prognosis	1. SCC in situ (CIS/Bowen disease): hyper-/parakeratosis, cellular pleomorphism 2. Well-differentiated: abundant cytoplasm, keratin pearls, limited nuclear atypia 3. Moderately differentiated: intermediate histology 4. Poorly differentiated: limited keratinization, increased nuclear-to-cytoplasm ratio, severe nuclear atypia	1. Superficial spreading: 70%, progression of prior stable, dysplastic nevus 2. Nodular: 10–15%, often the trunk, high risk 2/2 limited radial growth phase. Worst prognosis 3. Lentigo maligna melanoma: 10–15%, on chronically sun-damaged skin: face, arms; progression from lentigo maligna (in situ). Best prognosis 4. Mucosal lentiginous: 3%, GI, GU, respiratory mucosa; older pts; aggressive. Hard palate most common location 5. Acral lentiginous: rare, 1:1 W:B, often soles/palms, subungal; high risk 2/2 limited radial growth 6. Desmoplastic: rare, highly variable clinical presentation; often PNI, comparatively low met rate
Others	Basosquamous: BCC and SCC features, rare, aggressive spread	Adenoid (acantholytic): pseudoglandular histology, aggressive spread Spindle cell: rare, spindle-shaped epithelial cells, clinically aggressive	
High-risk characteristics	Recurrent, located on the eyelids, nose, ear, upper lip, scalp, IS, h/o XRT, perineural invasion	Recurrent, located on the eyelid, lip, ear, scalp; >2 cm diameter, >2 mm depth, IV Clark level; PNI; poorly differentiated	(Three staging systems: TNM, Breslow, Clark); Perform SNLB if >1 mm depth, Breslow >Stage I if <40 years, +margin, ulceration, elevated mitotic rate, ALI, recurrent
Surgical treatment	3–4 mm margins; wider for non-nodular/superficial subtypes. Basosquamous—MMS candidate	Non-high risk: 4–6 mm margins. High risk: 7 mm margins vs. MMS	Excisional biopsy for suspicious lesions, shave, curettage, laser excision contraindicated WLE: Tis/T1, 1 cm; T2/T3, 2 cm; T4, 2–3 cm; scalp, 3 cm

Table 10.1 (continued)

	Basal cell carcinoma	Squamous cell carcinoma	Melanoma
Additional treatment	Low risk (trunk/extremities): cryotherapy, electrodessication, topical 5-FU XRT—poor surgical candidates, improved LRC w/ PNI, nodal dz Chemo: high risk, mets, positive margins; oral 5-FU, EGFR inhibitors, platinum/taxanes/5-FU systemic therapy	Low risk (trunk/extremities): cryotherapy, electrodessication, topical 5-FU XRT—poor surgical candidates, improved LRC w/ PNI, nodal dz Chemo: high risk, mets, positive margins; oral 5-FU, EGFR inhibitors, platinum/taxanes/5-FU systemic therapy	Sentinel node biopsy for 0.76–3.99 mm lesions, if positive (15–20%) proceed with ND T4NO: no benefit w/ elective ND N1 neck: I–V ND, <30% 10-year survival. Chemo (advanced stage): traditional agents: dacarbazine, temozolomide; interferon alpha2b, IL-2 (NK and T cell activation), GM-CSF, vemurafenib (BRAF inhibitor). XRT—isolated brain mets

Legend: *UV* ultraviolet, *IS* immunosuppression, *PNI* perineural invasion, *MMS* Mohs micrographic surgery, *SNLB* sentinel lymph node biopsy, *ALI* angiolymphatic invasion

Table 10.2 BCC and SCC TMN staging

Tis: Carcinoma in situ (limited to the epidermis)	N1: single ipsilateral LN ≤3 cm
T1: <2 cm with <2 high-risk factors[a]	N2a: single ipsilateral LN >3 and <6 cm
T2: >2 cm or any size with >2 high-risk factors[a]	2b multiple ipsilateral LN <6 cm
T3: Facial bone invasion	2c contralateral LN <6 cm
T4: Skeletal bone invasion, skull base invasion	N3: any LN >6 cm
Stage 0	Tis, N0, M0
Stage I	T1, N0, M0
Stage II	T2, N0, M0
Stage III	T3, N0, M0; T1–3, N1, M0
Stage IV	T1–3, N2, M0; T4; any T, any N, M1

[a]cancer classification

Table 10.4 Melanoma TMN staging and 5-year survival

Stage 0	Tis, N0, M0	100%
Stage IA	T1a, N0, M	95%
Stage IB	T1b, N0, M0; T2a, N0, M0	90%
Stage IIA	T2b, N0, M0; T3a, N0, M0	75%
Stage IIB	T3b, N0, M0; T4a, N0, M0	65%
Stage IIC	T4b, N0, M0	45%
Stage III	Any T, N 1–3, M0	
Stage IIIA	T1–4a, N1a, M0; T1–4a, N2a, M0	65%
Stage IIIB	T1–4b, N1/2a/c, M0; pT1–4a, N1/2b, M0	30–50%
Stage IIIC	T1–4b, N1/2b, M0; any T, N3, M0	25%
Stage IV	Any T/N + M	10–20%

Table 10.3 Various melanoma classification schemes

Classic Breslow	Clark levels	T classification
Stage I: ≤0.75 mm	Level I—Above basement membrane (in situ)	Tis in situ
Stage II: 0.76–1.50 mm	Level II—Extends into papillary dermis	T1: ≤1 mm (a/b, w/o-w/ ulceration)
Stage III: 1.51–2.25 mm	Level III—Papillary/reticular dermis interface	T2: 1.01–2.0 mm (a/b)
Stage IV: 2.26–3.0 mm	Level IV—Into reticular dermis	T3: 2.01–4.0 mm (a/b)
Stage V: >3.0 mm	Level V—Invasion of subcutaneous tissue	T4: >4.0 mm (a/b)

spread on histology (fat/oil red O stain). Locally invasive with 10–20% LN metastatic rate. *SX*: Firm, painless nodule with loss of cilia on the upper eyelid. *TX*: WLE/Mohs with orbital exenteration in select cases. *Muir-Torre syndrome*—AD, combination of visceral malignancy and sebaceous gland carcinoma.

Atypical Fibroxanthoma Uncommon, spindle/giant cell tumor, appearing in elderly patients with sun-damaged skin as a locally aggressive, rapidly enlarging tumor that strongly favors the head and neck; low risk of metastasis. Considered a cutaneous equivalent of malignant fibrous histiocytoma with a 1:1 ratio of men to women. *RF*: UV exposure, elderly, prior radiotherapy, immunosuppression. *SX*: Nodular, dermal, ulcerative, singular lesion on sun-exposed skin. *TX*: WLE/Mohs primary treatment.

Dermatofibrosarcoma Protuberans (DFSP) Rare, low-/intermediate-grade dermal sarcoma thought to arise from fibroblasts/histiocytes (CD34 positive), locally invasive with high recurrence rate. Presents between ages 20 and 50, 1:1 M:F without racial predilection. *RF*: Local trauma, burn/surgical scar. *SX*: Indolent, painless violaceous plaque on the trunk with <20% occurring in the head and neck. *TX*: WLE with 2–3 cm margins (including fascia); unresectable/metastatic cases—imatinib mesylate (tyrosine kinase inhibitor).

Oral Cavity and Oropharyngeal Squamous Cell Carcinoma (SCC)

Oral Cavity SCC

More prevalent among men and blacks. *RF*: Tobacco, alcohol, betel nut quid, lower socioeconomic status. Citrus fruit- and beta-carotene vegetable-rich diet is protective. *TX*: Primary surgical excision (5 mm margins) typical first-line therapy. Radiotherapy offers similar outcomes in lower-stage disease, consider for poor surgical candidates. LN metastasis is the most significant prognostic indicator.

Anatomic Subsites

- Oral Tongue: Most common; risk of LN metastasis directly related to tumor depth (>2–4 mm).
- Lip: SCC higher prevalence on the lower lip with a better prognosis compared to the upper lip; BCC favors the upper lip.
- Floor of the Mouth: Locally invasive with a high risk of occult LN metastasis.
- Buccal: Relatively high recurrence rate w/ functional reconstruction required (skin graft, free tissue transfer) to prevent postoperative cicatricial trismus.
- Retromolar Trigone: High recurrence rate/risk of mandibular invasion (edentulous more at risk than patients with healthy dentition).
- Hard Palate: Rare; bony resection indicated in most cases, low incidence of occult LN metastasis (Table 10.5).

Surgical Management of the N0 Neck Selective neck dissection (levels I–III) generally indicated for Stage I tumors with >2 mm tissue invasion and all Stages II–IV. Caveats include:

1. Lower lip (T1 → observation; T2 → level I neck dissection)
2. Include perifacial LN dissection in buccal tumors
3. All stages of retromolar trigone/lower alveolar ridge → neck dissection

Adjuvant Therapy Considerations Indications for postoperative RT include perineural/lymphovascular invasion on primary tumor and/or positive or close margins, bone invasion, and

Table 10.5 Oral cavity and oropharyngeal SCC TMN staging

Tis carcinoma in situ	N1 single ipsilateral LN ≤3 cm
T1: ≤2 cm	
T2: >2 cm but ≤4 cm	N2a single ipsilateral LN >3 and <6 cm
T3: ≥4 cm	2b multiple ipsilateral LN <6 cm
T4a: Invasion to extrinsic muscle, cortical bone, skin	2c contralateral LN <6 cm
b: Invasion to pterygoid plates, masticator space, skull, or ICA encasement	N3 any LN >6 cm
Stage 0	Tis, N0, M0
Stage I	T1, N0, M0
Stage II	T2, N0, M0
Stage III	T3, N0, M0; T1–3, N1, M0
Stage IVa	T1–3, N2, M0; T4aN0–2, MO
Stage IVb	Any T, N3, Mo; T4b; any T, any N, M1

metastatic LN. Concurrent chemotherapy is considered in advanced-stage disease (Stage III/IV), extracapsular spread (ECS), and positive margins.

Oropharyngeal SCC

- *RF*: Tobacco, ETOH; human papillomavirus (HPV) for tonsil, base of the tongue.
- HPV: Serotypes 6 and 11, low malignant potential; serotypes 31 and 33, intermediate malignant potential; serotypes 16 and 18, worst malignant potential. Viral oncoproteins E6 and E7 promote degradation of tumor suppressor proteins p53 and Rb, respectively.

Anatomic Subsite Considerations

- Soft Palate: Typically undergoes nonsurgical management given postsurgical functional deficit.
- Tonsil: HPV cases with 85% HPV 16; younger, nonsmoker, more responsive to organ preservation therapy.
- Base of the Tongue: Most aggressive subsite; 20–40% risk of LN metastasis.
  – Low-stage disease → single-modality treatment (surgery vs. RT).
  – Advanced-stage disease → organ preservation therapy (concomitant—RT/chemotherapy) is generally favored as first-line therapy w/ exception of bony mandible invasion where composite resection is indicated (Tables 10.5, and 10.6).

Reconstructive Ladder (Fig. 10.1)

Secondary Intention A bed of granulation tissue is formed, and the wound heals from deep to superficial with contraction of wound edges. Surgical pearls: maintain a moist wound surface with petrolatum-based ointment to avoid fluid

Table 10.6 Management of the neck

N0/N1 neck → RT or selective ND (II–IV)	N2/N3 → RT/CHX or modified radical ND (I–V)

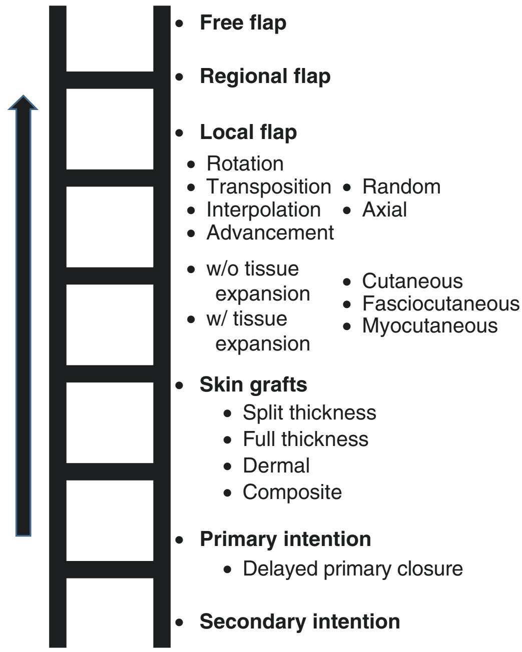


Fig. 10.1 Reconstructive ladder

and electrolyte loss and avoid eschar, debride necrotic tissue, drain fluid collections, and maintain fresh and open wound edges.

Primary Closure Surgical pearls: maintain tension-free suture line via subcutaneous and dermal suture layers, skin edge eversion (cutaneous resections should be performed with scalpel beveled away from wound), adequate undermining.

Skin Grafts Utilized as an alternative to local flaps or to offer temporary wound coverage and/or tumor surveillance. Primary limitation is color mismatch, thickness (men > women; adults > newborn), and texture match to bordering tissue. Graft survival depends on adequate recipient blood supply and patient risk factors (smoking, vascular disease, history of radiation) (see Table 10.7). Graft failure often secondary to underlying hematoma (most common), seroma, infection, graft mobility, wound tension.

Split-Thickness Skin Graft (STSG) Includes the epidermis and portion of the dermis. Donor sites include the abdominal wall, thigh, buttocks, and scalp (potential for repeated harvesting Q7

Table 10.7 Physiology of graft survival

Plasmatic imbibition	First 24–48 h → fibrin layer allows adherence of the graft to the wound
Inosculation	After 48 h → anastomoses form between graft and recipient blood vessels
Neovascularization	2–6 days → vascular ingrowth of recipient blood vessels into the graft

days without risk of alopecia). Variable thicknesses from thin (0.007–0.011 in.) and medium (0.012–0.016 in.) to thick (0.017–0.020 in.).

Advantages—Lower metabolic rate, shorter revascularization time, can cover large surface area, able to be meshed for greater wound coverage.

Disadvantages—Poor color/texture/thickness match, limited durability, increased contraction vs. FTSG.

Full-Thickness Skin Graft (FTSG) Includes the epidermis and entire dermis (w/ hair follicles). Donor sites: post-/preauricular, upper eyelid, supraclavicular skin, Burrow's triangle. Useful in nasal, auricular, eyelid defects.

Advantages—Better color/texture/thickness match, durable, decreased wound contraction vs. STSG.

Disadvantages—Higher metabolic rate, longer revascularization time, decreased uptake % vs. STSG, donor site morbidity.

Composite Graft Skin and cartilage w/ vascularization occurring via plasmatic imbibition, inosculation and then ingrowth of capillaries from graft edges by third day following transfer. Donor sites: concha, helical root, antihelix. Useful in nasal sill, ala, contralateral auricular defects.

Advantages—Provides both wound support and coverage.

Disadvantages—Contraction, graft size limited to ~1 cm in size, higher failure rate, donor site morbidity.

Dermal, Dermal/Fat Grafts Deepithelialized FTSG w/ or w/o underlying fat; useful in supplying bulk to facial defects. Should be <1.5 cm to avoid excessive resorption. Allograft materials (e.g., AlloDerm) may also be used in the same manner.

Tissue Expanders Gradually stretch local tissue for coverage of a planned defect. Process occurs through mechanical and biological creep. Biologic creep seen in long-term tissue expansion; actual increase in mitotic activity that results in new tissue growth.

Mechanical creep seen in short-term tissue expansion; realignment of collagen fibers to allow for increased tissue mobility.

Physiologic observations include:

1. Subtle thickening of the epidermis with increased fibroblast activity
2. Thinning of the dermis and adipose tissue layers (40–50%)
3. Myofibroblast hyperplasia with vascular proliferation
4. Creation of a fibrous capsule (facilitating wound contracture and flap thickening)

Local Flaps Multiple classification schema including:

- *Perfusion*: random, supplied via subdermal plexus; axial, supplied via named vessel.
- *Tissue:* cutaneous, skin and SQ (e.g., local facial flap); fasciocutaneous, skin, SQ, deep fascia (e.g., galea O-Z flap); myocutaneous, skin, SQ, muscle (e.g., pectoralis major flap).
- *Mode of transfer:* pivotal, advancement, hinge.

Pivotal Flaps Four main types: rotational, transposition, interpolated, island. Rotational: arc of rotation no more than 30°, flap radius 2.5–3× lesion diameter. Transposition: flap mobilized and transposed over incomplete tissue bridge, e.g., bilobed, rhombic flap (see Chap. 7). Interpolated: flap mobilized and transposed over complete tissue bridge, staged procedure (e.g., PMFF). Island: flap mobilized and transposed under complete tissue bridge with deepithelialization or while maintaining subcutaneous blood supply.

Advancement Flaps Four main types: unipedicle, bipedicle, V-Y/Y-V, and island advancement (similar to island pivotal flap but with linear movement vector). Linear tension vector, random blood supply with favored site being the forehead/brow. Dog ear/standing cutaneous deformity occurs with terminal angles >30° necessitating conversion to W-plasty or Burrow triangle excision.

Hinge Flaps Pedicle based on one border of the primary defect, dissected in the subcutaneous plane and turned over onto defect like page in a book; "trap door," "turn in." Dissection proceeds from end toward base of flap becoming progressively deeper to preserve blood supply.

Commonly Tested Local Flaps (See Chap. 7 for More Information)

Bilobed Flap Transposition flap commonly used for nasal defects <1.5 cm diameter, >0.5 cm from alar margin. Zitelli modification (90° arc of rotation) improved upon original 180° design.

Rhombic Flap Full-thickness transposition flaps based on a random blood supply for rhombic defects with angles of 60° and 120°. Initially described by Limberg with subsequent Dufourmental and Webster modifications. Favored sites include the cheek, lip, temple, and nose, with the forehead being less favorable. Designed for transfer within lines of maximal extensibility (perpendicular to relaxed skin tension lines).

Z-Plasty Flap used to lengthen scars, reorient wound tension, provide camouflage of scars within relaxed skin tension lines. Designed as double transposition of opposing triangular flaps. Facial Z-plasties should have limbs no longer than 0.5 cm; if greater than 0.5 cm, multiple Z-plasties should be used. Larger Z-plasties can be utilized on the neck. Limbs drawn at angles from the scar. Increased angle increases length of scar. 30° produces 25% increase in scar length. 45° increases scar length 50%. 60° increases scar length 75%. Optimal angle ranges 45–60° as >60° results in significant tension on wound.

Melolabial Flap One- (transposition) or two-stage (interpolated) flap favored for nasal alar/lobule defects. Inferiorly based, axial flap via angular artery pedicle; superiorly based, local blood supply.

Paramedian Forehead Flap (PMFF) One-/two-/three-stage axial flap used mostly for nasal defects. Based on the supratrochlear artery (identified at supraorbital rim 1.7–2.2 cm from midline) which runs superficial to the corrugator muscle (divided during elevation) and deep to the orbicularis, pierces frontalis muscle 2 cm superior to rim. Minimum base width: 1 cm. Depth of elevation varies: *pedicle* → subgaleal w/ frontalis vs. subperiosteal for enhanced rotation; *skin paddle* → subcutaneous vs. subgaleal (latter favored if small vessel disease or planned intermediate stages). Three varieties:

- *Paramedian*: Reputed to have increased dermal vascularity (despite similar viability) given center position over pedicle with a *less* desirable scar line than the midline flap
- *Midline*: Offers a longer skin paddle with a more favorable scar line
- *Single Stage*: Limited to young, healthy patients without small vessel disease; requires deepithelialization and tunneling under glabella

Cervicofacial Flap Shared elements of rotation and advancement for closure of sizeable cheek defects. Superior/medial tension vector with recommended acute angle at the inferomedial tip. Avoid primary excision of Burrow's triangle if tenuous blood supply. Primary pitfall involves inadequate suspension to the zygomatic or orbital periosteum.

Complications and Management

Flap failure Clinical presentation: venous failure, 3× more common, blue, warm, edematous with pin prick returning dark blood in <1 s; arterial failure, cool, pale, non-blanching, minimal blood return in >2 s. *RF*: Tobacco use, prior/

concurrent radiotherapy, prior local surgery, collagen/vascular disease, diabetes mellitus, and poor flap design. Interventions include relaxation of suture line, hyperbaric oxygen (pre-/postop), and leech therapy for venous congestion. Hirudin is a leech salivary peptide resulting in thrombin inhibition and vasodilation. If used, must control for infection risk from *Aeromonas hydrophila* (GNR, beta-lactamase-resistant) with aminoglycoside/fluoroquinolone considered first-line therapy.

Trapdoor deformity Elevated and bulging deformity found in “C”-, “V”-, and “U”-shaped scars, often following curvilinear flaps. Risk factors include oversized flaps with excess subcutaneous fat, lymphedema (inferiorly based better than superiorly based), and a lack of contact inhibition resulting in increased wound contraction in scar confines. Avoided by wide undermining beyond the base of the flap as well as squaring the corners of the flap and defect to convert a round flap into a rectangular one. Management includes surgical debulking or steroid injection.

Flap Delay

Physiologic phenomenon where flap viability is significantly improved following a 2-week delay from making initial incisions to tissue mobilization. Large percentage of neurovascular supply must be eliminated. Beneficial effects can last up to 6 weeks. Theories for etiology include:

- Loss of sympathetic tone leading to norepinephrine depletion in 24–48 h leading to change in small vessel vasoactivity and increased perfusion
- Release of vasodilators following local ischemia and inflammation leading to “choke vessel” increased caliber and dilation
- Development of vascular collaterals and reorientation of major vascular channels to distal flap

Soft-Tissue Reconstruction: Regional Flaps and Free Tissue Transfer

Regional Flaps (Table 10.8)

Free Tissue Transfer

Radial Forearm (RFF) Fasciocutaneous/osteocutaneous workhorse for reconstruction of intraoral and skull base defects as well as partial or total pharyngeal reconstruction. *Pedicle*: Radial artery and associated venae comitantes/cephalic vein which are located in the lateral intermuscular septum (flexor carpi radialis medially, brachioradialis laterally). Long vascular pedicle.

Innervation: Lateral antebrachial cutaneous nerve (associated with cephalic vein). Perform Allen test preoperatively to assess candidacy for radial a. sacrifice: compress ulnar and radial artery, release ulnar artery, and assess thenar perfusion (based on radial artery → deep palmar arch; ulnar artery → superficial palmar arch). Contraindications include positive Allen test, presence of AV fistula, and history of hand surgery (relative).

Surgical Pearls: Prudence with ulnar dissection to avoid transection of superficial ulnar vascular pedicle. Radial bone harvest (up to 12 cm and 40% of cortical diameter), distal perforators preserved, oblique (keel) cuts. Intraoperative plating following harvest reduces risk of iatrogenic radius fracture. Donor site-specific complications include compartment syndrome, ischemia of the first finger and thumb, and tendon exposure. Palmaris longus (medial to the flexor carpi radialis) is present in 85% of patients, and the tendon can be employed for support for midface/lip reconstruction. Donor site requires skin graft for closure.

Lateral Arm Fasciocutaneous flap with possible composite muscle (triceps) and osseous (humerus, 1 cm width) component; favored in partial glos-

Table 10.8 Fasciocutaneous, myocutaneous, and myofascial regional flaps

Flap	Artery / Nerve	Reconstruction sites	Advantages / Disadvantages	Pearls
Pectoralis major (PM)	Thoracoacromial a.	Oral cavity, oropharynx, neck	One stage, large cutaneous component, carotid coverage, single surgical position	Ligate lateral thoracic artery for increased rotation, not indicated in patients with Poland syndrome (underdeveloped muscle)
	Medial/lateral pectoral n.		Bulk, breast distortion, deficit in shoulder adduction/rotation	
Latissimus dorsi (LD)	Thoracodorsal a.	Oral cavity, oropharynx, neck	Sizeable bulk	Ligate circumflex scapular → rotation
	Thoracodorsal n.		Bulk, have to change surgical position	
Temporalis (TP)	Anterior and posterior deep temporal a.	Orbit, cheek, dynamic facial reanimation	Offers dynamic motion	Identify Pitanguy's line to avoid frontal branch (CN VII)
	Trigeminal n.		Short rotation arc, no cutaneous component	
Trapezius (TZ)	Transverse cervical, dorsal scapular a.	Lower face, neck, oropharynx	Thin, pliable, long rotation arc	DSA and TCA may both serve as pedicle; if neither dominant, DSA inferior skin, TCA superior skin for island flaps
	CN XI n.		CN XI loss, surgical positioning, donor site morbidity	
Supraclavicular artery island flap (SCAIF)	Supraclavicular a.	Face, oral cavity, pharynx, neck, auricle, temporal bone, lateral SB	Quick harvest, reliable, versatile	Contraindicated with hx of ND where pedicle may be compromised
	Supraclavicular n.		Minimal bulk	
Sternocleidomastoid (SCM)	Occipital, superior thyroid a, thyrocervical trunk	Oral cavity, oropharynx, neck	Easily accessible, single stage	Preserve at least two pedicled vessels for use
	CN XI n.		Limited use if substantial nodal disease	
Temporoparietal fascial flap (TPF)	Superficial temporal a.	Orbit, auricle, temporal bone	Thin, pliable, simple dissection	Can be used as free flap
	n/a		Minimal bulk, donor site alopecia	
Deltopectoral (DP)	Second/third internal mammary a.	Oropharynx, neck	Reliable anatomy, no change in surgical position	Can consider flap delay if smoker, pedicle may be sacrificed if prior pec flap
	Supraclavicular n.		Two stage, distal flap with random blood supply	

sal/pharyngeal defects. *Pedicle*: Posterior radial collateral artery (terminal brachial branch with negligible risk of distal ischemia) and cephalic vein. Pedicle located in lateral intermuscular septum (bordered by triceps/brachioradialis). Shorter available pedicle length compared to radial forearm. *Innervation*: Posterior cutaneous nerve of the forearm.

Surgical Pearls: Varying tissue thickness is favorable in base of tongue reconstruction (thinner skin → proximal flap; thicker skin → distal flap). Radial nerve at risk given its close proximity to the proximal lateral intermuscular septum.

Rectus Versatile myofascial/cutaneous flap offering tissue bulk for closure of skull base and cutaneous defects. *Pedicle*: Deep inferior (more commonly used) or superior epigastric artery. *Innervation*: Intercostal nerve, but limited availability for reinnervation.

Surgical Pearls: Long-term tissue bulk derived from vascularized adipose tissue around the periumbilical region. Postoperative hernia avoided by preserving posterior fascial integrity during muscle elevation at and below arcuate line (pedicle perforates rectus sheath).

Lateral Thigh Sizeable fasciocutaneous flap often employed for total or partial glossal/pharyngeal and facial defects. *Pedicle*: Profunda femoris artery perforators (primarily third perforator) and venae comitantes located in intermuscular septum (bordered by biceps femoris and vastus lateralis). *Innervation*: Lateral femoral cutaneous nerve. Contraindications include prior thigh surgery, obesity (relative), vascular disease, and diabetes mellitus.

Surgical Pearls: Allows for primary donor site closure. Considerable anatomic variability as to relative location and position of the third perforator. Use caution when harvesting second perforator which is a principal blood supply to the femur.

Anterolateral Thigh Generous fasciocutaneous flap with similar indications as lateral thigh free flap. *Pedicle*: (>85%) Descending branch of lateral circumflex femoral artery located in intermuscular septum (bordered by the rectus femoris and vastus lateralis), (<5%) transverse branch of lateral circumflex femoral artery, and venae comitantes. *Innervation*: Lateral femoral cutaneous nerve.

Surgical Pearls: Perforators located within 5 cm above and below the midpoint of a line drawn from lateral patella to superior iliac spine. Can denude flap of overlying adipocutaneous tissue for thin vascularized fascial flap: often employed for scalp reconstruction with STSG coverage.

Jejunal Free Flap Employed for hypopharyngeal reconstruction. *Pedicle*: Segmental arcades based off superior mesenteric artery. *Innervation*: None, but peristalsis resumes with revascularization (must be placed anterograde). Detractors include classic "wet voice," stenosis, and limited utility of tracheal puncture. Able to harvest up to 25 cm in length. Disadvantages include need for multiple anastomosis, limited ischemia time (2 h), and laparotomy required.

Free Flap Failure Classically described as either venous thrombosis (significantly more common, appearing at 48–72 h) or arterial thrombosis (presenting in the first 24 h). Hypercoagulability, venous stasis, and endothelial injury (Virchow's triad) are encountered with free tissue transfer. First-line treatment is urgent operative intervention with leeches offered as second-line therapy in cases of venous congestion. Early detection is paramount with multiple options (often surgeon specific) ranging from implantable or cutaneous Doppler, pin prick test, Doppler ultrasound, and near-infrared spectrophotoscopy. Flap salvage rate after take back averages 50%. Use of venous couplers shown to enjoy similar rates of success. No consensus regarding standardized anticoagulation protocol. Based on surgeon, training experience, personal outcomes, and anecdotal evidence. Aspirin, heparin, low-molecular-weight heparin, and dextran all used with comparable rates of free flap failure, hematoma, and thrombosis.

Bony Reconstruction

Calvarial bone graft Employed in nasal reconstruction; taking advantage of the lower comparative resorption rates of membranous bone over endochondral bone. Mesenchymal in origin; membranous bone found in the cranium (*excluding sphenoid*) and mandible with a thicker cortex than endochondral bone. Split calvarial grafts utilize outer cortical layer, leaving behind the diploic and inner layer. Harvested from parietal bone above the temporal line (below temporal line → outer/diploic layers thinner and greater curvature). Exercise caution to avoid midline and the sagittal sinus with most common complication being seroma/hematoma of the scalp.

Iliac bone graft Employed for mandibular, nasal, maxillary reconstruction. Enable harvest of cortical, cancellous, and tricortical bone. Approached by oblique incision along iliac ridge 1 cm inferior to iliac margin at widest point of iliac crest centered over anterior superior iliac spine. Skin is retracted and fascia overlying bone is divided prior to bony harvest. Complications include gait changes, inguinal hernia, chronic pain, lateral thigh numbness, and seroma/hematoma.

Free Tissue Transfer

Fibula Osteocutaneous workhorse, offering 25 cm of bone stock for maxillary/mandibular defects as well as a fasciocutaneous paddle for two-dimensional reconstruction. Skin paddle may be split based on two different perforators for reconstruction of composite defects of the oral cavity. Allows for segmental osteotomies and osteointegrated implants. *Pedicle*: Peroneal artery via combination of endosteal/periosteal perfusion (permitting multiple osteotomies to optimize contour) and associated venae comitantes. *Innervation*: Derived from lateral sural cutaneous nerve. Preoperative vascular assessment essential: check pedal pulses (dorsalis pedis, tibialis posterior) and investigate for vascular disease (edema, color, ulceration). Gold standard imaging is lower extremity angiography with Doppler, CTA, and MRA as alternatives. Contraindications include peroneus magnus (vascular abnormality with a dominant peroneus artery supplying the leg—must identify three-vessel runoff preoperatively), significant venous stasis, diabetes mellitus, and history of leg trauma.

Surgical Pearls: Must employ diligent preoperative planning to identify preference for anterior vs. posterior vessel geography, intraoral vs. extraoral skin paddle position. Proximal bone cut → 6 cm of fibula preserved (avoids peroneal injury). Distal bone cut → 6–8 cm of bone (maintain ankle integrity). Potential loss of great toe flexion secondary to flexor hallucis longus dissection. Can be transferred with sensate skin paddle (lateral sural cutaneous nerve).

Iliac Crest Composite flap offering cutaneous and inferior oblique muscle component with up to 16 cm of curved cancellous bone (anterior to posterior iliac spine) for mandibular reconstruction. *Pedicle*: Deep circumflex iliac artery/vein. *Innervation*: 12th thoracic nerve; lateral cutaneous branch.

Surgical Pearls: Inferior oblique elevated off transversus abdominis with pedicle on muscular deep surface. Primary detractors include donor site morbidity (sensory deficits most prevalent), bulky muscle component, and limited rotation arc for the skin paddle.

Scapula Versatile with three possible skin paddles (latissimus/scapular/parascapular) and osseous component (lateral border—10 cm; 14 cm with scapula tip) for mandibular and oral composite defects. *Pedicle*: Circumflex scapular artery and its terminal cutaneous branches (transverse → scapular; descending → parascapular). *Innervation*: None.

Surgical Pearls: Prone or lateral decubitus surgical positioning, thin bone, and functional morbidity at donor site are principal detractors.

Site-Specific Reconstructive Highlights

Lip

Upper: Includes paired upper lateral cutaneous subunits (boundaries: alar base superiorly, melolabial fold laterally, vermillion border inferiorly), philtral subunit (boundaries: philtral ridges, columella, cupid's bow), and red lip (boundaries: vermillion, stomion). *Blood supply*: Superior labial artery running between orbicularis oris and mucosal tissue layers. *Innervation*: Infraorbital nerve (V2) (Table 10.9).

Lower: Includes lower subunit (boundaries: vermillion, labiomental crease), chin (boundaries: labiomental crease, inferior margin), red lip (stomion, vermillion). *Blood supply*: Inferior labial artery running between orbicularis oris and mucosal tissue layers. *Innervation*: Mental nerve (V3) (Table 10.10).

Ear

Reconstructive options divided into central, upper 1/3rd, middle 1/3rd, lower 1/3rd, and subtotal/total defects. Blood supply: Posterior auricular and superficial temporal arteries. Innervation: Great auricular (C2, C3), lesser occipital (C2, C3), Arnolds (CN V), auriculotemporal (V3) nerves (Table 10.11).

Table 10.9 Full-thickness upper lip defects

<1/3	Wedge excision, primary closure, local flap (advancement/transposition)
1/3 → 2/3 (incl. medial lip)	*Perialar crescentic advancement*: Innervated flap, can be offered in conjunction with Abbe for central defect *Abbe*: Design flap using full width of defect (as opposed to ½ in the lower lip)
1/3 → 2/3 (incl. commissure)	*Estlander*: Use full width of defect in flap design
>2/3	*Karapandzic*, *Abbe*, *melolabial flap*

Table 10.10 Full-thickness lower lip defects

<1/3	Wedge excision, primary closure, local (advancement/transposition) flap
1/3 → 2/3 (incl. medial lip)	*Abbe flap*: Two stage, full thickness, cross lip, ½ width of defect from the upper lip, based on superior labial artery, orbicularis muscle direction maintained, divided at 2–3 weeks *Karapandzic*: Modification of Gillies (a full-thickness, rotation/advancement flap); partial thickness w/ preservation of neurovascular pedicle; limited by microstomia
1/3 → 2/3 (incl. commissure)	*Estlander*: Single stage, cross lip, ½ width of defect, muscle direction of orbicularis maintained *Gillies/Karapandzic* *McGregor*: Modification of Gillies, requires vermillion mucosal flap; fan-shaped flap w/ arc of rotation at commissure; orbicularis muscle direction rotated 90°
>2/3	*Bernard-Burrow-Webster*: Full thickness, horizontal advancement, triangles with skin only to maintain neurovascular bundle; vermillion derived from tongue/mucosa *Bilateral abbe*, *melolabial*, *regional* (SCM, deltopectoral), *free flap*

Table 10.11 Auricle reconstruction options by subsite

Central	FTSG, superiorly based preauricular, posteriorly postauricular flaps
Upper 1/3rd	Wedge closure (<1.5 cm), chondrocutaneous (cc) advancement (1.5–2.5 cm), interpolated tubed flaps (ITF) (>2.5 cm), TPF w/ cartilage grafts (total)
Middle 1/3rd	Wedge closure (<1.5 cm), cc advancement (1.5–2.5 cm), ITF (>2.5 cm), postauricular interpolated advancement w/ cartilage grafts (>2.5 cm)
Lower 1/3rd	Primary closure (<50%), composite grafts, anterior/posterior rotational flaps w/wo cartilage grafts
Subtotal/ total	Avulsion: microvascular reattachment, gross reattachment w/ leeches, pocket method, cartilage replacement w/ TPF flap, Baudet method; congenital: costal cartilage, Medpor w/ TPF

Eyelid

Reconstruction must replace missing layers with matching tissue. Conjunctival reconstruction: conjunctival rotation, advancement flaps, cross-lid flaps, hard palate mucosa, Alloderm. Tarsus: advancement flaps, cross-lid flaps, cartilage grafting. Skin: medial and lateral rotation/advancement flaps, cross-lid flaps, STSG/FTSG. *Blood supply*: Upper lid: superior lateral and medial palpebral, dorsal nasal arteries. Lower lid: inferior lateral and medial palpebral, transverse facial, angular arteries. *Innervation*: Upper lid: lacrimal, supraorbital, supratrochlear, infratrochlear nerves. Lower lid: zygomaticofacial, infraorbital, infratrochlear nerves.

Reconstruction

Upper lid: <25% direct closure; 25–50% direct closure with lateral cantholysis. 33–66% Tenzel semicircular flap w/ periosteal flap. 50–100% Cutler-Beard flap.

Lower lid: <25% direct closure, 25–50% direct closure with lateral cantholysis, 33–66% Tenzel semicircular flap, 50–75% semicircular flap w/ periosteal flap, 50–100% Hughes tarsoconjunctival flap.

Mandible

Segmental loss of the mandible has major impact on mastication, swallowing, speech, airway protection, and facial aesthetics. Primary reconstruction with free tissue transfer has revolutionized mandibular reconstruction offering the patient immediate, durable, functional, and aesthetic reconstruction of composite defects. Patient-related factors include presence of significant comorbidities, desire for dental implants, disease biology, and patient's motivation.

Broad defect variability: marginal mandibulectomy (mandibular continuity intact; lingual, buccal, or alveolar rim resected) vs. segmental mandibulectomy (mandibular continuity disrupted; incl. partial, hemi-, or total). Considerations: durability (+/− adjuvant XRT), airway patency, intra- vs. postoperative dental implants, aerodigestive tract lining, restoration of upper aerodigestive function (speaking, swallowing, mastication), aesthetics. *Blood supply*: Endosteal, inferior alveolar artery; periosteal, via contiguous muscle/ST. *Innervation*: Inferior alveolar nerve.

Segmental Mandibular Reconstruction

Non-bony reconstruction: poor surgical candidate, palliative surgery, staged reconstruction. Bridged fixation with plate +/− musculo(+/− cutaneous) pectoralis rotational flap, myomucosal intraoral flap.

Non-vascularized bone grafts: small defects <6 cm, little to no aerodigestive lining or external cover deficit, absence of microvascular capability. Corticocancellous iliac crest, pretibial, costal within rigid construct (plates, crib). Vs. microvascular reconstruction decreased bony union in serial comparisons (69% vs. 96%).

Vascularized bone grafts: loss of ST lining or cover in poor surgical candidate, palliative surgery, staged reconstruction. Options include pectoralis rotational flap with the rib, scapula-trapezius rotational flap, latissimus rotational flap with the rib, and SCM rotational flap with the clavicle.

Microvascular: provides concurrent bony, lining, or cover reconstruction as needed, robust blood supply, endosteal dental implant candidacy, improved bony union and decreased fistula rates. Osteocutaneous radial forearm FF (OCRFFF): predictable anatomy, up to 12 cm bone stock, minimize pathologic donor site fractures via prophylactic plating, capable of dental implants if "double barreled." Fibula FF (FFF): requires preoperative imaging, up to 26 cm bone stock, accommodates multiple osteotomies for contour (≥2 cm segments) larger caliber than OCRFFF, capable of dental implants. Scapular tip FF (STFF): provides ample myocutaneous ST within subscapular system, more limited bone stock vs. OCRFFF/FFF (<12 cm defects), capable of dental implants. Iliac crest FF (ICFF): provides ample bone stock (up to 15 × 6 cm), shortest pedicle length (4–7 cm), significant donor site morbidity, capable of dental implants.

Table 10.12 Flap of choice based on location and size (diameter and length)

<270° circumference and above thoracic inlet	RFF, LTF, ALTF; jejunal free flap w/ divided antimesenteric border[a]
>270° circumference, above thoracic inlet and <10 cm in length	Tubed—RFF, LTF, ALTF; jejunal free flap
>270° circumference, above thoracic inlet and >10 cm in length	Tubed—LTF; jejunal free flap

Hypopharynx (Table 10.12)

Questions

1. A patient presents with a diagnosis of microcystic adnexal carcinoma (MAC) following a punch biopsy of a preauricular cutaneous lesion. Which of the following statements is *not* correct regarding the typical clinical presentation/behavior of MAC?
   (a) Low-grade tumor arising from the sweat glands.
   (b) More common among Caucasians.
   (c) Low risk of perineural invasion.
   (d) Risks factors include prior history of radiotherapy.
2. A patient presents with a 1.5 cm diameter, ulcerative, pigmented scalp lesion. On physical exam, a prominent lymph node is palpated at level II. Following excisional biopsy, WLE, and regional neck dissection, a diagnosis of melanoma is made with a tumor thickness of 2.7 mm and two positive LNs. What is the correct stage for the patient?
   (a) T2bN2bM0—Stage IIIB
   (b) T2bN2aM0—Stage IIIC
   (c) T3bN2aM0—Stage IIIB
   (d) T3bN2bM0—Stage IIIC
3. Of the following treatment options for a Stage I BCC, which has the highest recurrence rate?
   (a) Surgical excision
   (b) Radiation therapy
   (c) Cryotherapy
   (d) Mohs micrographic surgery
4. Following a rhombic flap closure of a cutaneous cheek defect, a pin-cushion (trapdoor) deformity is noted. Which of the following statements is *not* correct regarding this complication?
   (a) Complication is more prevalent with superior-based flaps.
   (b) Occurs secondary to inadequate undermining.
   (c) Higher incidence in geometric flaps compared to curvilinear flaps.
   (d) Treatment includes surgical debulking and/or steroid injection.
5. For a patient who has undergone a re-excision of a cutaneous SCC, which finding is associated with the highest metastatic rate?
   (a) Previously treated lesion (recurrence)
   (b) Perineural invasion
   (c) Poor differentiated histology
   (d) >4 mm tumor thickness
6. A 65-year-old heavy smoker, diabetic, with a large, near-total, cutaneous dorsal/tip nasal defect presents for closure following a Mohs resection for SCCa. On exam you note a low anterior hairline. Which is the *best* choice in reconstructive options?
   (a) Paramedian forehead flap—two stage
   (b) Midline forehead flap—two stage
   (c) Paramedian forehead flap—three stage
   (d) Midline forehead flap—three stage
7. Which of the following is *not* an arterial blood supply for the trapezius musculocutaneous flap?
   (a) Occipital artery
   (b) Paraspinal perforators
   (c) Dorsal scapular artery
   (d) Thoracodorsal artery
8. A patient is undergoing reconstruction of a pharyngeal defect with a lateral thigh free flap. When harvesting the flap, which vessel (if ligated) is most likely to cause postoperative ischemia of the femur?
   (a) Third perforator of the profunda femoris
   (b) Second perforator of the profunda femoris
   (c) Septocutaneous perforator of the lateral circumflex femoral artery
   (d) Musculocutaneous perforator of the lateral circumflex femoral artery

9. Which of the following statements regarding lip anatomy is *incorrect*?
   (a) The upper lip is longer than the lower lip.
   (b) Paresis of marginal mandibular nerve results in ipsilateral lip depression.
   (c) Mentalis contraction leads to lip protrusion.
   (d) The upper lip has three separate subunits.
10. A patient is noted to have persistent venous congestion following a RFF flap for an oral cavity defect. Unable to return to the operating room, leech therapy is started. After 24 h, the patient develops signs of cellulitis at the graft site. Which antibiotic is most appropriate?
    (a) Ciprofloxacin
    (b) Augmentin
    (c) Mupirocin
    (d) Cephalexin
11. What is the most common subsite of the head and neck to suffer a basal cell carcinoma?
    (a) Superior helix
    (b) Lower eyelid
    (c) Nasal tip/ala
    (d) Lower lip
12. A 48 M is diagnosed with a right superior helix melanoma at the site of prior dysplastic nevus. The patient is immunocompetent, has extensive history of sun exposure, and suffers no clinical nodal masses. What is the most likely subtype?
    (a) Desmoplastic
    (b) Superficial spreading
    (c) Nodular
    (d) Lentigo maligna
13. A melanoma histopathological specimen shows a pigmented, ulcerated lesion, 0.83 mm in thickness, invading into but not beyond the papillary dermis. What are the Breslow, Clarke, and T stages?
    (a) I, I, T2a
    (b) II, I, T1b
    (c) II, II, T1b
    (d) II, III, T2b
14. Which of the following, in isolation, is NOT an indication for sentinel lymph node biopsy following head and neck melanoma diagnosis?
    (a) Current immunosuppression
    (b) Ulceration
    (c) Stage T2 or above
    (d) Presence of angiolymphatic invasion
15. Which of the following cutaneous malignancies has a viral etiology in the majority of cases?
    (a) Sebaceous carcinoma
    (b) Microcystic adnexal carcinoma
    (c) Atypical fibroxanthoma
    (d) Merkel cell carcinoma
16. The majority of head and neck sebaceous carcinomas arise from which facial structure?
    (a) Meibomian glands
    (b) Preorbital follicular units
    (c) Areas of local chronic inflammation
    (d) Nasolacrimal duct
17. Which is the most important predictor of regional nodal disease in oral tongue SCC?
    (a) Lesion diameter
    (b) Dorsal vs. ventral tongue location
    (c) Depth of invasion
    (d) Histologic grade
18. HPV infection is a common etiologic factor in oropharyngeal SCC. Which serotype below is most prone to oncogenesis?
    (a) 11
    (b) 16
    (c) 6
    (d) 31
19. Uptake failure following full-thickness skin graft is most often secondary to which complication?
    (a) Local fluid collection underlying graft
    (b) Graft mobility
    (c) Excessive graft thickness
    (d) Recipient site devascularization
20. Which of the following is NOT an advantage of STSGs vs. FTSGs?
    (a) Better color match
    (b) Lower metabolic rate
    (c) Shorter revascularization time
    (d) More easily able to be meshed
21. Dermal fat grafts should be less than which diameter (cm) to maximize graft uptake?

(a) 0.5 cm
(b) 1 cm
(c) 1.5 cm
(d) 2 cm

22. Which of the following is NOT true of tissue undergoing active tissue expansion?
(a) Epidermal thinning
(b) Dermal thinning
(c) Myofibroblast hyperplasia with vascular proliferation
(d) Creation of a fibrous capsule

23. A posterior scalp O-to-Z flap is performed. This local flap is named based on which flap characteristic?
(a) Perfusion
(b) Tissue movement vector
(c) Flap composition

24. The bilobed flap should be avoided in nasal reconstruction if the defect diameter is wider than which of the following?
(a) 0.5 cm
(b) 1.0 cm
(c) 1.5 cm
(d) 2 cm

25. Trapdoor deformities are NOT commonly seen in which of the following scenarios?
(a) Oversized flaps with excess subcutaneous fat
(b) Obstruction of lymph drainage
(c) Lack of contact inhibition at wound margins
(d) The base of a local flap due to excessive rotation

26. Which of the following is NOT a proposed mechanism for the increased success rates of local flaps following flap delay?
(a) "Choke vessel" dilation
(b) Increase in axial pedicle diameter
(c) Local norepinephrine depletion
(d) Reorientation of vascular channels

27. The frontal branch of the facial nerve is at risk when harvesting a temporoparietal fascia flap. What is the course of the frontal branch?
(a) Follows a line from the EAC to the superolateral orbital rim
(b) Follows a line from 0.5 cm below the tragus to 1.5 cm above the lateral brow
(c) Travels subcutaneously superior to the zygomatic arch
(d) Crosses the zygomatic arch in the anterior third

28. The fibula free flap should be harvested at which point below the head of the fibula to avoid the peroneal nerve?
(a) 4 cm
(b) 6 cm
(c) 8 cm
(d) 10 cm

29. The lateral thigh free flap is pedicled on which artery(ies)?
(a) Lateral descending circumflex femoral artery
(b) Internal iliac artery
(c) Popliteal artery
(d) Profundus femoris perforators

30. The supraclavicular island flap is pedicled on the supraclavicular artery from the thyrocervical trunk. Which of the following does NOT stem from the thyrocervical trunk?
(a) Transverse cervical artery
(b) Ascending cervical artery
(c) Thoracoacromial artery
(d) Inferior thyroid artery

31. Which of the following is NOT an appropriate method for reconstruction of a 2.5 cm defect of the middle third of the auricular helix?
(a) Composite cartilage graft
(b) Interpolated tube flap
(c) Buried two-stage postauricular advancement flap
(d) Superior and inferior chondrocutaneous sliding advancement flaps

32. What is the most appropriate method for reconstruction of a defect encompassing 75% of the upper lid?
(a) Tenzel semicircular flap
(b) Direct closure with lateral cantholysis
(c) Cutler-Beard flap
(d) Hughes tarsoconjunctival flap

33. An ipsilateral fibula flap with skin paddle is harvested for reconstruction of an oral composite defect. Given the geometry of the flap, which direction does the pedicle extend from the flap once the inset is complete?

(a) Anterior
(b) Posterior

34. Which free flap is most able to undergo neurorrhaphy as well as microvascular reanastomosis?
    (a) Scapula
    (b) Jejunum
    (c) Anterolateral thigh
    (d) Rectus abdominis
35. What is the maximal defect length able to be reconstructed by a non-vascularized bone graft?
    (a) 2 cm
    (b) 4 cm
    (c) 6 cm
    (d) 8 cm
36. On average, which osteocutaneous free flap provides the most length regarding bone stock?
    (a) Radial forearm
    (b) Fibular
    (c) Scapular
    (d) Iliac crest
37. One is able to harvest which maximal length of the jejunum when performing pharyngeal/esophageal free flap reconstruction?
    (a) 12 cm
    (b) 18 cm
    (c) 25 cm
    (d) 30 cm

Answers

1. (c)
2. (d)
3. (c)
4. (c)
5. (b)
6. (d)
7. (d)
8. (b)
9. (b)
10. (a)
11. (c)
12. (b)
13. (c)
14. (a)
15. (d)
16. (a)
17. (c)
18. (b)
19. (a)
20. (a)
21. (c)
22. (a)
23. (b)
24. (c)
25. (d)
26. (b)
27. (b)
28. (b)
29. (d)
30. (c)
31. (a)
32. (c)
33. (a)
34. (c)
35. (c)
36. (b)
37. (c)

Additional Resources

1. Ardehali B, Morritt AN, Jain A. Systematic review: anastomotic microvascular device. J Plast Reconstr Aesthet Surg. 2014;67(6):752–5.
2. Arganbright J, Tsue T, Girod D, Militsakh O, Sykes K, Markey J, Shnayder Y. Outcomes of the osteocutaneous radial forearm free flap for mandibular reconstruction. JAMA Otolaryngol Head Neck Surg. 2013;139(2):168–72.
3. Bader RS, Harris JE. Basal cell carcinoma. Medscape (276624). 2014.
4. Baker et al. Local flaps in facial reconstruction. 2007.
5. Barton BM, Riley CA, Fitzpatrick JC. Postoperative anticoagulation after free flap reconstruction for head and neck cancer: a systematic review. Laryngoscope. 2017. ePub ahead of print.
6. Cheney ML, Gliklich RE. The use of calvarial bone in nasal reconstruction. Arch Otolaryngol Head Neck Surg. 1995;121:643–8.
7. Dhar PK, et al. Identification of risk factors for specific subsites within the oral and oropharyngeal region—a study of 647 cancer patients. Indian J Cancer. 2000;37(2–3):114–22.
8. Foster RD, Anthony JP, Sharma A, Pogrel MA. Vascularized bone flaps versus nonvascularized bone grafts for mandibular reconstruction: an outcome analysis of primary bony union and endosseous implant success. Head Neck. 1999;21:66–71.

9. Grin CM, et al. Accuracy in the clinical diagnosis of malignant melanoma. Arch Dermatol. 1990;126:763–6.
10. Lai A, Cheney M. Temporoparietal fascial flap in orbital reconstruction. Arch Facial Plast Surg. 2000;2:196–201.
11. Lawrence N, Cottel WI. Squamous cell carcinoma of skin with perineural invasion. J Am Acad Dermatol. 1994;31(1):30–3.
12. Markey J, Knott PD, Fritz MA, Seth R. Recent advances in head and neck free tissue transfer. Curr Opin Otolaryngol Head Neck Surg. 2015;23(4):297–301.
13. McCarn KE, Park SS. Lip reconstruction. Facial Plast Surg Clin North Am. 2005;13:301–14.
14. McLeod GR, Black RJ. Malignant melanoma: cutaneous and mucosal. In: Paparella MM, Shumrick DA, editors. Otolaryngology, vol. 3. Philadelphia: W. B. Saunders Company; 1991. p. 2617–25.
15. Medina JE. Malignant melanoma of the head and neck. Otolaryngol Clin N Am. 1993;26(1):73–85.
16. Monroe MM, Meyers AD. Cutaneous squamous cell carcinoma. Medscape (1965430). 2015.
17. Papel et al. Facial plastic and reconstructive surgery. 2009.
18. Smith RB, Sniezek JC, Weed DT, Wax MK. Microvascular surgery subcommittee of the American Academy of Otolaryngology-Head and Neck Surgery. Utilization of free tissue transfer in head and neck surgery. Otolaryngol Head Neck Surg. 2007;137(2):182–91.
19. Weber RC, Kearns DB, Smith RJH. Split calvarium cranioplasty. Arch Otolaryngol Head Neck Surg. 1987;113(1):84–9.

Scar Revision and Dermabrasion

11

Sagar Patel and James A. Owusu

Histology of the Skin

The *epidermis* is avascular and composed mostly of keratinocytes that mature and migrate from the basal layer to the surface where it is lost through desquamation. Other cells are melanocytes, Merkel cells, and Langerhans cells. The layers of the epidermis are:

- *Stratum corneum*: dead and dying cells filled with mature keratin eventually lost through desquamation.
- *Stratum lucidum*: well defined in only thick skin, i.e., palms of hands and soles of the feet. It is the transition zone between the strata granulosum and corneum.
- *Stratum granulosum*: composed of cells with dense keratohyalin granules (dark pigmentation) connected with tight desmosomes creating a watertight barrier that prevents fluid loss.
- *Stratum spinosum*: divided cells from the stratum basale accumulate desmosomes on their outer surface to form this prickly layer.
- *Stratum basale*: composed of germinal cells necessary for regenerating the layers of the epidermis. It is separated from the dermis by a thin basement membrane.

Mnemonic: Come, Let's Get Sun Burned

The *dermis* is vascular and composed mostly of fibroblast that secretes collagen, elastin, and ground substance and is responsible for thermoregulation:

- *Papillary dermis*: composed of loose connective tissue with vascular network responsible for thermoregulation and supporting the avascular epidermis.
- *Reticular dermis*: composed of dense connective tissue with glands and hair follicles. The papillary dermis has smaller collagen bundles, higher cellularity, and higher density of vascular elements compared to the reticular dermis.

Skin Lines

- *Relaxed skin tension lines* (*RSTLs*): lines of intrinsic skin tension produced by the action of underlying muscles. They run perpendicular to the underlying muscle fibers. Skin

S. Patel (✉)
Facial Plastic Surgery, Sagar Patel Aesthetics, Beverly Hills, CA, USA
e-mail: appointments@spafacialplastics.com

J. A. Owusu
Otolaryngology – Head and Neck Surgery, University of Michigan, Ann Arbor, MI, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_11

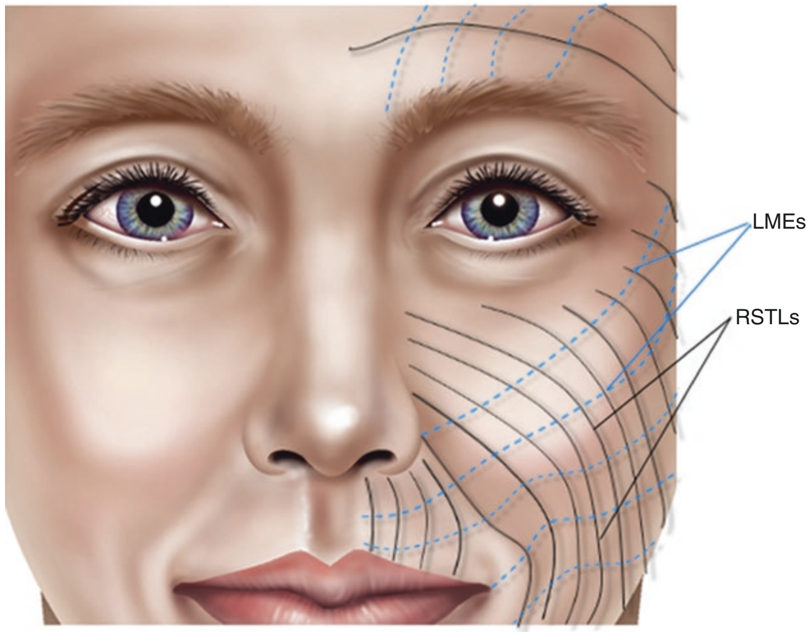


Fig. 11.1 Relaxed skin tension lines and lines of maximum extensibility. Ideal scars are parallel or fall within RSTLs

incisions should ideally be placed in or parallel to RSTLs (Fig. 11.1).
- *Lines of maximum extensibility* (*LMEs*): perpendicular to RSTLs, skin is most extensible in the direction of LMEs (Fig. 11.1).
- *Langer lines*: derived from cadaveric studies corresponding to alignment of collagen fibers in the skin.

Wound Healing Phases

- *Inflammatory* (0–7 days): vasoconstriction and activation of the coagulation cascade, histamine release, vasodilation with infiltration of neutrophils, monocytes, fibroblasts, endothelial cells, PMNs, and macrophages.
- *Proliferative* (24 h to 6 weeks): reepithelialization begins in this phase with differentiation of basal cells and separation from the basement membrane. Fibroblasts produce *type III collagen* starting around day 3. Neovascularization and wound contraction occur in this phase. Wound contraction is mediated by myofibroblasts that become oriented along lines of tension and pull collagen fibers together contracting the wound.
- *Remodeling/maturation* (2 weeks to 18 months): type III collagen is replaced by *type I collagen*. Collagen becomes cross-linked increasing the tensile strength of the wound. Maturation is affected by several factors including age. Maturation occurs faster in adults compared to children. Scars eventually achieve a tensile strength that is approximately 80% the tensile strength of normal skin. At maturation collagen remodeling continues, and there is a balance between collagen synthesis and collagen breakdown by collagenases and metalloproteinases. Collagen becomes increasingly organized and water is resorbed from the wound.

Factors Affecting Wound Healing

- Moisturization with a hydrophobic ointment during the initial stages of healing inhibits bacterial growth and promotes reepithelialization.
- Microangiopathic disease including diabetes reduces blood flow and oxygen delivery and impairs wound healing.
- Nicotine causes vasoconstriction that can lead to poor wound healing (Table 11.1).

Table 11.1 Hypertrophic scars and keloids

Keloid	Hypertrophic scar
Scars extend beyond the confines of the wound margin	Scars stay within the confines of the wound margin
Does not regress	May regress with time
50–100% recurrence rate following excision	Most do not recur following excision
Disorganized thickened collagen fibers	Fibroblastic nodules with fine randomly organized collagen fibers; fibroblasts with increased contractility and TGF-beta production
Reduced gap junction intracellular communication with very low expression of connexin (Cx43) leading to inability to receive inhibitory growth signals	Reduced gap junction intracellular communication with decreased expression of connexin with decreased ability to receive inhibitory growth signal

Management of Keloids and Hypertrophic Scars

- Excision with post-op steroid injection (decreases fibroblast and glycosaminoglycan production, reduces inflammation).
- Topical imiquimod (5%) may improve recurrence rate for keloids after excision as an adjuvant therapy resulting in a best case report of 15% recurrence. Side effects include itching, burning, pain, blisters, infection, and hyperpigmentation. Imiquimod is an immune modifier that induces NK cells, macrophages, Langerhans cells, and the release of multiple cytokines including IFN-alpha2b. It inhibits fibroblast collagen production and glycosaminoglycan production.
- Silicone gel improves the appearance of scars through its hydrating effect.
- Radiotherapy at a dose of 5 Gy improves keloids by destroying fibroblast which are not replaced.

Scar Revision

The ideal scar is flat, narrow, level with the surrounding skin, good color match, within or parallel to relaxed skin tension lines, or on the border of a facial subunit. Scar revision may be considered for scars that are widened, perpendicular to RSTLs, and webbed; interrupt facial subunits; or distort facial features, hypertrophic, depressed, or atrophic.

Timing of Scar Revision

Scars that are parallel to RSTLs will continue to improve over 6–12 months. It is best to allow complete scar maturation, i.e., 6–12 months prior to revision. Scars with unfavorable location or that disrupt facial features may be revised sooner.

Scar Revision Techniques

- *Fusiform excision* is ideal for scars in good position. The superficial aspect of the scar is excised in a fusiform shape with end angles <30°. The deeper aspect of the scar is preserved to prevent depression. An M-plasty can be performed at the end of the excision to shorten the overall length of the incision and minimize excision of normal skin (Fig. 11.2).
- *Serial excision* is useful for wide scars with surrounding elastic skin that cannot be completely excised in one stage. The scar is excised in multiple stages 6–8 weeks apart. This technique relies on biologic creep and is an alternative to tissue expansion.
- *Tissue expansion* stretches the tissue adjacent to the scar creating adequate skin to completely excise the scar. Adjacent tissue has the advantage of providing the best color and tex-

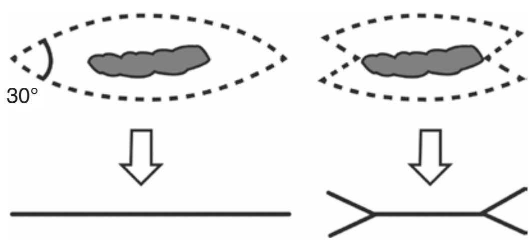


Fig. 11.2 M-plasty reduces the length of the overall incision length and the amount of normal skin excised

ture match. The thickness of the epidermis is preserved through epidermal mitotic activity and keratinocyte proliferation, while the thickness of the dermis is reduced.

- *Shave excision* is ideal for scars with raised or uneven edges or standing cutaneous deformity. The lesion is excised tangential to the skin and the wound is allowed to heal by secondary intention.
- *Scar repositioning*: Scars close to RSTLs or facial aesthetic unit boundaries, or hairline, can be repositioned by excising the intervening normal skin.
- *Scar irregularization* camouflages scars making them less perceptible to the eye.
- *Z-plasty* breaks up and lengthens long contracted scars and is used to realign distorted facial features and to reorient scars in a more favorable position. The degree of lengthening is dependent on the angles used to design the flaps. Classic Z-plasty involves transposition of 60° equilateral triangles that lengthens the scar by 75%. Thirty degree and 45° angles lengthen the flaps by 25% and 50%, respectively (Fig. 11.3, Table 11.2).
- *Z-plasty variations*: Combinations of Z-plasty allow longitudinal lengthening scars without creating excessively angulated or elongated flaps. Multiple Z-plasty can be combined in parallel to reorient and elongate a long scar. Mirror images of two Z-plasty can be combined to create a double-opposing Z-plasty. The double-opposing Z-plasty is particularly useful in areas with impaired skin vascularity as the large central flap is less prone to necrosis (Fig. 11.4).
- *W-plasty*—Consecutive triangular excisions irregularize the scar without increasing its length. The width of each flap should be 5–7 mm. The deeper aspect of the scar is preserved to avoid creating a depressed scar. Dermabrasion 6–8 weeks following excision may be indicated to improve appearance. It is ideal for curvilinear scars (Fig. 11.5).
- *Geometric broken lines* use irregularly irregular pattern of scar excision using geometric figures with a width of 5–7 mm. Again the deeper aspect of the scar is preserved. The irregular pattern makes the resulting scar less perceptible compared to "W"-plasty and does not lengthen the scar. Scars may be further improved with dermabrasion. It is ideal for scars that cross RSTLs. This is the best scar irregularization technique (Fig. 11.6).

Skin Resurfacing

- *Dermabrasion* uses controlled abrasion of the skin down to the depth of the papillary to superficial reticular dermis to improve scar appearance. The depth of the papillary dermis is characterized by pinpoint bleeding; parallel white lines characterize the superficial reticu-

Table 11.2 Z-plasty configuration parameters

Z-plasty angle	Rotation	Lengthening
30°	45°	25%
45°	60°	50%
60°	90°	75%

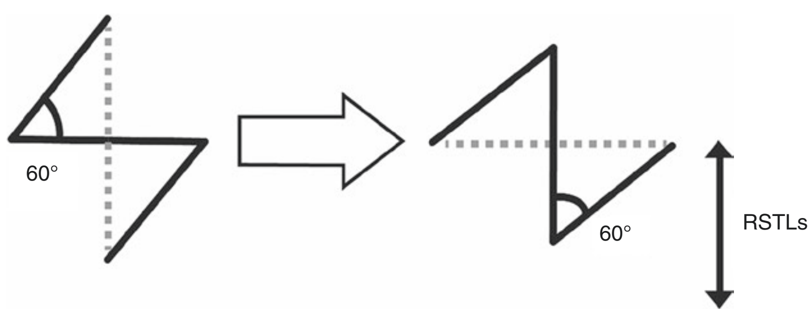


Fig. 11.3 Z-plasty is ideal for lengthening and repositioning scar into a more favorable orientation

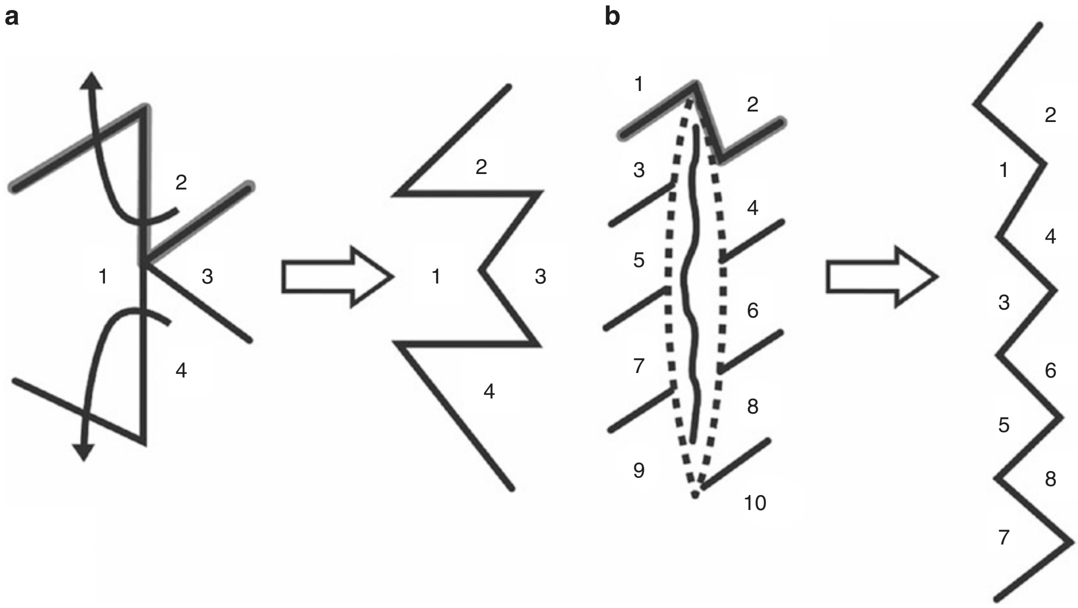


Fig. 11.4 (**a**) Double-opposing Z-plasty creates a large central flap particularly useful in areas with compromised vascularity. (**b**) Multiple Z-plasty in parallel is useful for reorienting long scars

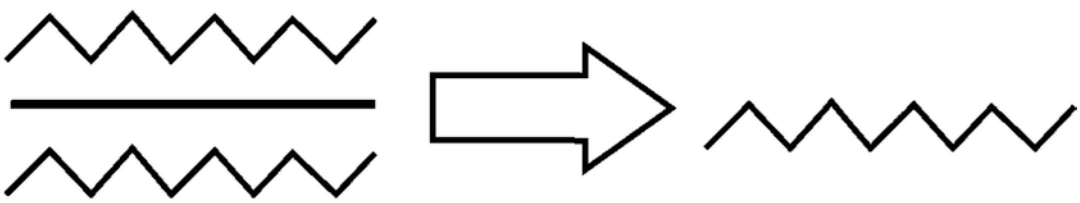


Fig. 11.5 Running W-plasty is ideal for curvilinear scars; the limbs of the flaps are oriented parallel to RSTLs

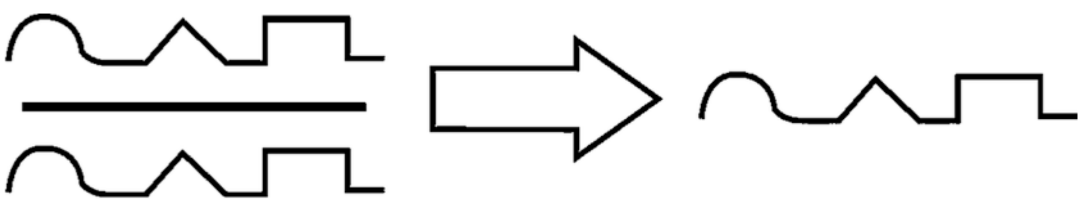


Fig. 11.6 Geometric broken lines—the width of each flap should be between 5 and 7 mm. The irregular pattern makes the scar less predictable compared to running W-plasty

lar dermis. Abrasion deep into the reticular dermis may lead to increased scaring. Ideally, dermabrasion is performed 6–8 weeks following injury when fibroblast activity is high. Dermabrasion improves scars by increasing collagen organization. Dermabrasion upregulates expression of the glycoprotein tenascin in the papillary dermis and expression of alpha 6 and beta 4 integrins in the stratum spinosum:

– *Indications*: Acne scars, traumatic scars, early operative scars, fine lines and wrinkles, photodamage.
– *Contraindications*: Recent use of isotretinoin (within 6–12 months), active herpes breakout. Isotretinoin causes apoptosis of

pilosebaceous units, which are responsible for regenerating the epithelium following dermabrasion.
  - *Precautions*: Perioperative antivirals (acyclovir or valacyclovir) reduce the risk of herpetic outbreak. Use face shield and PPE to avoid contact with aerosolized blood products.
  - *Pre-procedure* evaluation may include bleeding time, platelets, and inquire about recent isotretinoin use or herpes infection and use of anticoagulants.
  - *Technique*: Dermabrasion is performed using powered diamond or wire brush fraises. Wire fraises are used at a lower RPM compared to diamond fraises. Abrasion is carried down to the level of the papillary dermis characterized by punctate bleeding. Injuring the reticular dermis results in scarring.
  - *Post-procedure*: Antiviral should be continued for 5–10 days following procedure. Wound moisturization with hydrophobic ointment inhibits bacterial growth and promotes reepithelialization.
- *Laser resurfacing*: Both ablative and non-ablative lasers are effective for improving facial scars. Ablative lasers cause vaporization of water-containing tissue resulting in epidermal regeneration and reorientation of collagen fibers. Results from laser resurfacing are comparable to those of dermabrasion. The three main lasers are used in scar revision:
- *Pulsed dye laser* (585 nm) is a non-ablative laser with hemoglobin chromophore. It penetrates the epithelium without causing de-epithelialization. Softens scars by breaking collagen disulfide bonds and reorienting collagen and inhibiting fibroblast. Also improves erythema through elimination of blood vessels. Multiple treatments spaced 6–8 weeks may be needed to achieve optimal results.
- *CO_2* (10,600 nm) is an ablative laser with water chromophore causing superficial ablation resulting in de-epithelialization. CO_2 has a deeper depth of penetration than Er:YAG.
- *Er:YAG* (2940 nm) is an ablative laser with water chromophore causing de-epithelialization. It produces minimal thermal injury with faster reepithelialization. It is ideal for atrophic scars.

Complications of Skin Resurfacing

- *Hyperpigmentation* is common following dermabrasion and most resolve without treatment within 3–6 months. It is exacerbated by sun exposure and estrogen use. Persistent hyperpigmentation can be improved with chemical peel. Patients should be counseled to use sunscreen and avoid sun exposure following skin resurfacing.
- *Hypopigmentation* is also commonly seen following resurfacing. It is more common with laser resurfacing than dermabrasion. It may present late, i.e., 6–12 months following laser resurfacing, and can be permanent. Bleaching creams may be used to blend in the surrounding areas. Hypopigmentation following dermabrasion usually resolves within 6–19 weeks without intervention.
- *Infection*—De-epithelialized skin is susceptible to bacterial and fungal infection. Most bacterial infections are caused by *Staphylococcus*, S*treptococcus*, and *Pseudomonas*. Bacterial infections present with discolored crusting and malodorous drainage 2–5 days following the resurfacing. Fungal infections are mostly caused by candida and present with erythematous erosions. Fungal infections can be treated with acetic acid soaks and fluconazole.
- *Herpes reactivation* can occur following resurfacing. A 5–10 course of antivirals (acyclovir of valacyclovir) starting a day before the procedure decreases risk of herpes breakout. Patients who develop a breakout while on oral antivirals may need IV antivirals.
- *Milia* result from epithelial inclusions during reepithelialization. May be treated with gentle abrasion, unroofing, or controlled cauterization.
- *Delayed healing*: Reepithelialization is usually completed in 5–7 days. It is considered delayed if not completed within 14 days. This may result from patient comorbidities and

poor wound care. Treatment is reversing the underlying cause.

- *Erythema* is common following resurfacing. It generally decreases and ultimately resolves with time. It may be due to a reaction from topical antibiotic ointment. Persistent erythema can be treated with topical steroid cream.

Depressed scars are commonly seen as pitting scars associated with acne. These scars can be improved with a combination of techniques including excision followed by dermabrasion or laser resurfacing. Scars can also be improved with filler/fat injection. Subcision is performed using a needle, the connection between the skin and deeper scar tissue is released, and the depressed area is filled with fat or filler. Filler options include autologous fat, calcium hydroxyapatite, and hyaluronic acid derivatives.

Adjunctive Techniques

Steroids can be helpful in the management of hypertrophic scars and keloids. The most common form used is injectable triamcinolone acetonide (Kenalog) in concentrations between 10 and 40 mg/ml. Topical steroids may be used to diminish erythema 2–3 weeks following skin resurfacing. Dermal and subcutaneous atrophy may occur if an excess amount of steroid is used.

Silicone sheets and gels may be used alone or in combination with other techniques to improve the appearance of hypertrophic scars and keloids. The exact mechanism of improvement is unknown but is believed to be partially due to increased hydration of the scar.

Mechanical forces: Massage of scars following complete epithelialization decreases adherence to deeper structures and improves the appearance.

Cosmetics can conceal scars using a foundation or diminish the appearance of scars using the color theory. The color theory uses opposite shades of a color to tone down its appearance. Examples: A red scar is camouflage with green concealer, blue or purple scar with yellow concealer, and brown and yellow tones neutralized with purple concealer.

Questions

1. The best technique to lengthen a contracted scar is:
   (a) “W”-plasty
   (b) Geometric broken lines
   (c) “Z”-plasty
   (d) Fusiform excision with “M”-plasty
2. An 18-year-old lady undergoes revision of a facial scar with geometric broken line excision. The ideal time to perform dermabrasion is:
   (a) Immediately following closure
   (b) 6–8 weeks following the procedure
   (c) 6–12 months following the procedure
   (d) 2 weeks following the procedure
3. Which of the following lasers is the best option of improving a hyperemic scar?
   (a) Er:YAG
   (b) CO_2
   (c) KTP
   (d) Pulsed dye
4. Which of the following is a contraindication for skin resurfacing?
   (a) Isotretinoin
   (b) Tretinoin
   (c) Hydroquinone
   (d) Triamcinolone
5. Two weeks following dermabrasion, a patient presents with concerns about persistent redness at the site. On exam reepithelialization is complete and there are no signs of infection. What is the best management option?
   (a) Treat with topical steroid cream
   (b) Antibiotics to reduce the inflammation
   (c) Reassurance
   (d) Triamcinolone injection
6. A patient with hyperemic scar following revision is interested in camouflaging the scar appearance. The best concealer tone for this patient is:
   (a) Dark brown
   (b) Yellow
   (c) Blue
   (d) Green

7. Which of the following techniques is specific to the management of depressed scars?
   (a) Autologous fat grafting
   (b) Subcision
   (c) Er:YAG ablative resurfacing
   (d) Intralesional steroids
8. Which of the following is the most common complication of intralesional steroid injection for severe cystic acne?
   (a) Dermal atrophy
   (b) Epidermal necrosis
   (c) Hypervascularity
   (d) Worsening acne
   (e) Systemic steroid response
9. Which is the preferred method of treatment for ice-pick-type acne scars?
   (a) Dermabrasion
   (b) Excision
   (c) Dermaplaning
   (d) PRP injections
   (e) Chemical peel
10. Scar pigmentation originates in what epidermal layer?
   (a) Stratum corneum
   (b) Stratum lucidum
   (c) Stratum granulosum
   (d) Stratum spinosum
   (e) Stratum basale
11. Which region demonstrates the highest rate of complications in tissue expansion?
   (a) Cheek/neck
   (b) Scalp
   (c) Eyelid
   (d) Radial forearm
12. Which of the following is the most common complication of tissue expansion?
   (a) Accidental expander rupture
   (b) Infection
   (c) Exposure
   (d) Inadequate expansion
13. Dermabrasion is contraindicated in patients with HIV?
   (a) True
   (b) False
14. When compared to a diamond tip fraise, the use of a wire tip requires a higher RPM?
   (a) True
   (b) False
15. A geometric broken line closure should be constructed to include irregularly irregular pattern?
   (a) True
   (b) False
16. What is the ideal width of each pattern constructed into a geometric broken line closure
   (a) 2–3 mm
   (b) 3–5 mm
   (c) 5–7 mm
   (d) 1 cm
17. A Z-plasty with a 60° transposition results in a rotation and lengthening of?
   (a) 45° and 75% increase in length
   (b) 60° and 25% increase in length
   (c) 90° and 75% increase in length
   (d) 45° and 50% increase in length
   (e) 90° and 60% increase in length
18. Which of the following regions has the lowest tendency for keloid formation?
   (a) Chest
   (b) Nose
   (c) Earlobe
   (d) Back
19. Which of the following is an appropriate total radiation dose for adjuvant treatment of a keloid?
   (a) 1 Gy.
   (b) 70 Gy.
   (c) 12 Gy.
   (d) 3 Gy.
   (e) Radiation carries too high a carcinogenesis risk to be an acceptable treatment for keloids.
20. Which of the following has no proven benefit in aesthetic improvement of a scar?
   (a) Sunscreen
   (b) Silicone gel
   (c) Silicone sheets
   (d) Antibacterial ointment
   (e) Onion extract (Mederma)
21. Which technique is ideal for curvilinear scars?
   (a) Z-plasty
   (b) M-plasty
   (c) Geometric broken line
   (d) Dermabrasion
   (e) Running W-plasty

22. Langer lines are determined by:
    (a) The orientation of the underlying musculature
    (b) The orientation of hair follicles
    (c) The alignment of collagen fibers in the skin
    (d) A contour map of the face
23. Dermabrasion should be carried to the depth of:
    (a) Basement membrane
    (b) Stratum basale
    (c) Papillary dermis
    (d) Reticular dermis
24. Initial fibroblastic wound healing and contraction occurs via the deposition of?
    (a) Hyaluronic acid
    (b) Vascular buds
    (c) Type I collagen
    (d) Type III collagen
    (e) Type IV collagen
25. Pin cushion deformity results from:
    (a) Lymphatic obstruction
    (b) Poor vascularity
    (c) Curvilinear scars
    (d) Excess tension during closure

Answers

1. (a)—Z-plasty reorients and elongates scars; W-plasty and geometric broken line irregularize scars without lengthening. A "M"-plasty reduces the length of the overall incision and the normal skin excised.
2. (b)—Dermabrasion should ideally be performed 6–8 weeks following scar revision when fibroblast activity is high.
3. (d)—Pulsed dye laser has a hemoglobin chromophore and is ideal for treating hyperemic scars.
4. (a)—Isotretinoin causes apoptosis of pilosebaceous units that regenerates epithelium following skin resurfacing. Resurfacing procedures should be delayed for 6–12 months following use of isotretinoin.
5. (c)—Erythema is expected following skin resurfacing and resolves with time. Persistent erythema can be improved with topical steroid cream.
6. (d)—A red scar is best camouflaged with a green concealer.
7. (d)—Steroids can cause dermal atrophy making a scar appear more depressed.
8. (a)—Steroids can result in dermal atrophy which results in an ice-pick-type scar.
9. (b)—Excision of the scar provides the best cosmetic result.
10. (d)—Melanocytes sit in the stratum basale.
11. (a)—The chin/neck is an area where the expanders tend to fall secondary to gravity, and this creates a higher rate of exposure.
12. (c)
13. (a) Use of PPE is always recommended; however historical literature regularly cites HIV as a contraindication.
14. (b)—The use of a wire fraise should necessitate a lower RPM.
15. (a)—The purpose of the GBLC is to irregularize the scar without increasing the length. Though the width of each flap should be 5–7 mm in length, there should be no pattern.
16. (c)—Though the pattern is designed irregularly, the base of each geometric shape should be between 5 and 7 mm.
17. (c)-(see Table 11.2).
18. (b)—The ear has a higher tendency to form a keloid than the central face.
19. (c)—Though there is no optimal consensus, it is generally believed 6–15 Gy in multiple fractionations are required.
20. (e)
21. (e)—The limbs of the flaps should be oriented parallel to the RSTLs.
22. (c)—The alignment of the collagen fibers was determined by cadaveric studies.
23. (d)—Dermabrasion should be carried until punctate bleeding is seen from the highly vascular reticular dermis.
24. (d)—Type III collagen is later converted to type I collagen in the remodeling/maturation phase that begins at week 2.
25. (a)—Lymphatic obstruction after local flaps can lead to a pincushion deformity. These are often repaired using running Z-plasty technique.

Additional Resources

1. Alster TS. Improvement of erythematous/hypertrophic scars by the 585 nm pulsed dye laser. Ann Plast Surg. 1994;32:186–90.
2. Atiyeh BS, Costagliola M, Hayek SN. Keloid or hypertrophic scar? The controversy: review of the literature. Ann Plast Surg. 2005;54:676–80.
3. Baker SR, Swanson NA. Tissue expansion of the head and neck. Indications, technique, and complications. Arch Otolaryngol Head Neck Surg. 1990;116:1147–53.
4. Berman B, Villa A. Imiquimod 5% cream for keloid management. Dermatol Surg. 2003;29(10):1050–1.
5. Bradley DT, Park SS. Scar revision via resurfacing. Facial Plast Surg. 2001;17(4):253–62.
6. De Benito J, Fernandez I, Nanda V. Treatment of depressed scars with a dissecting cannula and an autologous fat graft. Aesthet Plast Surg. 1999;23(5):367–70.
7. Fagien S. Human-derived and new synthetic injectable materials for soft-tissue augmentation: current status and role in cosmetic surgery. Plast Reconstr Surg. 2000;105(7):2526–8.
8. Friedman PM, Mafong EA, Kauvar AN, Geronemus RG. Safety data of injectable nonanimal stabilized hyaluronic acid gel for soft tissue augmentation. Dermatol Surg. 2002;28(6):491–4.
9. Furnas DW, Fischer GW. The Z-plasty: biomechanics and mathematics. Br J Plast Surg. 1971;24:144–60.
10. Fulton JE. Silicone gel sheeting for the prevention and management of evolving hypertrophic and keloid scars. Dermatol Surg. 1995;21:947–51.
11. Gold MH. Dermabrasion in dermatology. Am J Clin Dermatol. 2003;4(7):467–71.
12. Gold MH, Foster TD, Adair MA, Burlison K, Lewis T. Prevention of hypertrophic scars and keloids by the prophylactic use of topical silicone gel sheets following a surgical procedure in an office setting. Dermatol Surg. 2001;27:641–4.
13. Gunter JP. Camouflaging scars in the head and neck area. J Otolaryngol. 1978;7(1):75–87.
14. Harmon CB, Zelickson BD, Roenigk RK, Wayner EA, Hoffstrom B. Dermabrasive scar revision. Immunohistochemical and ultrastructural evaluation. Dermatol Surg. 1995;21(6):503–8.
15. Hudson DA. Some thoughts on choosing a Z-plasty: the Z made simple. Plast Reconstr Surg. 2000;106(3):665–71.
16. Leventhal D, Furr M, Reiter D. Treatment of keloid and hypertrophic scars: a meta-analysis and review of the literature. Arch Facial Plast Surg. 2006;8:362–8.
17. Lindsey WH, Davis PT. A 15-year experience. Arch Otolaryngol Head Neck Surg. 1997;123:397–400.
18. Lu F, Gao J, Ogawa R, Hyuakusoku H. Variations in gap junctional intercellular communication and connexin expression in fibroblasts derived from keloid and hypertrophic scars. Plast Reconstr Surg. 2007;119(3):844–51.
19. Nehal KS, Levine VJ, Ross B, Ashinoff R. Comparison of high-energy pulsed carbon dioxide laser resurfacing and dermabrasion in the revision of surgical scars. Dermatol Surg. 1998;24:647–50.
20. Stal S, Hamilton S, Spira M. Surgical treatment of acne scars. Clin Plast Surg. 1987;14(2):261–76.
21. Sproat JE, Dalcin A, Weitauer N, Roberts RS. Hypertrophic sternal scars, silicone gel sheet versus kenalog injection treatment. Plast Reconstr Surg. 1992;90:988–92.
22. Stucker FJ, Shaw GY. Keloids and hypertrophic scar. In: Papel ID, Nachlas NE, editors. Facial plastic and reconstructive surgery. St. Louis: Mosby Year Book; 1992. p. 53–5.
23. Thomas JR, Prendiville S. Update in scar revision. Facial Plast Surg Clin North Am. 2002;10(1):103–11.
24. Wolfe D, Davidson TM. Scar revision. Arch Otolaryngol Head Neck Surg. 1991;117:200–4.
25. Younai S, et al. Role of growth factors in scar contraction: an in vitro analysis. Ann Plast Surg. 1996;36:495–501.

12 Trauma

James Teng and John Michael Harbison

Trauma Evaluation

Primary survey – ABCs (airway, breathing, circulation).

Acute life-threatening injuries in facial trauma patients (most to least common) [1]:

- *Cerebral trauma (hematoma most common)*
- Hemorrhagic shock
- Airway compromise (the midface falls back posteriorly with pterygoid plate fractures)
- Pulmonary injury
- Cervical spine injury (very low incidence)

- Basic airway management: Clear all debris/blood/secretions → Jaw thrust (*caution: can cause up to 5 mm of distraction in patients with C5/6 instability*).
- Indications for airway intervention: Apnea/inadequate oxygenation, GCS <9, unstable midface trauma, laryngeal injuries, aspiration risk, large flail segment.
- Types of airway intervention: Oral airway, LMA, nasopharyngeal intubation (good for spontaneous ventilating patient, contraindicated in skull base fx), oral intubation (minimize C-spine manipulation), tracheostomy, cricothyroidotomy.
- Hemorrhagic shock: Indicated by hypotension/tachycardia → Initial treatment is 2L crystalloid bolus (20–30% stays intravascular) in adults (20 ml/kg in children) → colloid bolus → consider PRBCs if patient is still unstable after 2L crystalloid bolus [2]:
  - 3:1 crystalloid solution replacement for volume of estimated blood loss
  - PRBCs indicated for >30% blood volume loss
  - Correct acid/base/electrolytes

Secondary survey – complete physical exam, allergies, medications, past medical history, last meal, details of injury, tetanus status.

Screening C-spine, chest, and pelvic x-rays.

Type/screen, CBC, electrolytes, coagulation status, UA, pregnancy test, drug screen. Consider cardiac enzymes, ABG, and serum amylase.

Soft Tissue Injuries

Lacerations

- Low risk of infection in the face and scalp due to abundant vascularity. Wounds <24 hours old can be closed primarily with low risk of infection. Wounds >6 hours old or grossly contaminated should be irrigated. Contaminated

J. Teng (✉)
Santa Cruz ENT, Santa Cruz, CA, USA

J. M. Harbison
Otolaryngology-Head and Neck Surgery, University of California-Irvine, Omaha, NE, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_12

wounds should be repaired in delayed fashion. High velocity/shotgun GSW should be closed in delayed fashion, treated with debridement, drainage, and packing [3].

- Preserve all facial tissue unless obvious necrosis. Utilize atraumatic techniques. Freshen edges as appropriate. Layered closure removes dead space and facilitates soft tissue alignment. Undermine as needed to reduce tension at dermal subcutaneous junction. Evert wound edges.
- Antiseptics have harmful effects on wound healing – should only be used on intact skin around wound. Chlorhexidine better than iodine or alcohol. Hydrogen peroxide harmful to fibroblasts – shouldn't be used for cleaning open wounds.
- Antibiotics should be given for bite wounds, infected wounds, smoking, alcoholism, diabetes, coronary artery disease, immunosuppression/immunocompromised, prosthetic valves, or prosthetic joints. Cover skin flora (gram-positives), and include coverage for mixed flora/anaerobes (penicillin, clindamycin) in cases of oropharyngeal exposure.
- Tetanus risk from contaminated wounds, devitalized tissue, deep wounds, avulsion, stellate lacerations:
  - Low tetanus risk: Tetanus toxoid (Td) if <3 doses of vaccination or uncertain vaccination history; Td if three doses of vaccination and last dose >10 years ago
  - High tetanus risk: Td and tetanus immunoglobulin (TIG) if <3 doses or uncertain vaccination history; Td only if three doses and last dose >5 years ago

Bite Wounds

- Animal bites: Cat bites more likely to become infected than dog bites (*Pasteurella*, *Staphylococcus aureus*, *Streptococcus viridans*). Low rabies risk if animal is a known pet without exposure to virus (no prophylaxis required). If rabies history unsure, quarantine and observe animal. If rabies suspected, kill animal and examine brain tissue. Rabies treatment: immunoglobulin on day 0, vaccine on days 0, 3, 7, 14, 28.
- Human bites: More likely than animal bites to become infected (*Eikenella*, *Bacteroides*, Hep B, Hep C, HSV, HIV). Treat with broad-spectrum abx; cover aerobes and anaerobes.

Facial Nerve

- Management depends on degree, mechanism, and location of injury.
- Temporal bone fractures: Incomplete or delayed paralysis can be managed with initial observation. Complete paralysis → ENoG after 72 hours → if >90% degeneration surgical exploration recommended. For immediate, complete paralysis in the presence of temporal bone fracture, immediate exploration is advocated if patient is otherwise stable.
- Penetrating trauma: High likelihood of nerve transection if paralysis is present. If extratemporal lesion proximal (*lateral to lateral canthus*), explore within 72 hours so that nerve is identifiable with stimulation. Distal lesions (*medial to lateral canthus*) → observation with higher likelihood of recovery.

Parotid Gland

Most risk from penetrating injuries occurring below line from the tragus to upper lip. Inspect for facial nerve injury and salivary leak. Buccal branch runs with the parotid duct, most at risk for injury. Risk of damage to the parotid duct stratified by regions:

- Region A – posterior to masseter, low risk of facial nerve injury, close wound in layers.
- Region B – area over masseter, highest risk of ductal injury; cannulate duct with stent and anastomose duct over stent or perform superficial parotidectomy.
- Region C – anterior to masseter, anastomosis not possible; create new duct opening.

Eyelid

- Traumatic lacerations to the eyelid can create a series of problems, including telecanthus, ptosis, and nasolacrimal disruption. It is important to evaluate all of these at time of repair. The general order is to repair posterior medial canthal tendon first → nasolacrimal system second → marginal eyelid lacerations → peripheral lacerations → ptosis repair at a later date.
- Medial canthus injury: Disruption of the posterior tendon results in telecanthus, and repair/attachment to lacrimal crest should be done with microplating or transcanthal wires.
- Marginal lacerations should be meticulously repaired to align lid margin. Topical and injected local anesthetic as well as corneal protectors should be used. Slight eversion with vertical mattress suture helps prevent notching. Primary repair of the tarsus may be necessary at this time.
- Peripheral lacerations should be repaired only after margin alignment is satisfactory [4].
- Primary closure can be performed with defects up to ¼ of the lid length. >¼ lid length involvement may require skin graft. Lateral canthotomy may add up to 10 mm of additional lid length.

Nasolacrimal Duct

- Anatomy: Superior and inferior puncta 5 mm lateral to medial canthus. A 2 mm vertical limb → 8 mm horizontal limb → common canaliculus → lacrimal sac sitting in lacrimal fossa → emptying into the inferior meatus.
- Lacerations through the medial eyelid should raise suspicion for ductal injury. Symptoms (epiphora) may not present initially especially in the setting of multiple injuries. Before closing the laceration, the puncta and proximal duct should be cannulated to identify proximal segment. If a complete laceration exists, identifying the distal segment can be difficult. Cannulation of the opposing lid affords ability to flush air or dye into the system to locate distal end. Once identified, a silastic or silicone stent is placed and reanastomosis is performed. Tarsal anchoring sutures aid is decreasing tension on repair.

Facial Basics

Bone Healing

Bone healing occurs in several stages:

- Inflammatory phase (immediate): hematoma, inflammatory response, granulation tissue formation; osteoclasts remove necrotic bone.
- Soft callus (2–3 weeks): differentiation into osteoblasts, fibroblasts, and chondrocytes; granulation replaced by vessels, ECM, cartilage.
- Hard callus (3–4 months): endochondral ossification, woven bone formed.
- Remodeling (months–years): woven bone replaced by lamellar bone.

Fracture Fixation

- Splinting: Applying rigid device to the fracture (i.e., MMF) helps to reduce fracture segments, but there will always be some movement at the fracture site.
- Compression: Can be achieved with plates or lag screws. Works by compressing two pieces of bone together, creating a friction and a “preload” on the bone. As long as functional forces do not exceed “preload,” the bone will not move.

Surgical Approaches to Facial Skeleton

Mandible

- Intraoral: Used to access parasymphyseal, body, and mandibular angle. Relatively safe; be sure to identify mental nerve and resuspend the mentalis muscle.

- Submandibular: Used to access the mandibular body and angle, often used for complex comminuted fractures or in the setting of an edentulous, atrophic mandible. Care must be taken to avoid the marginal branch of the facial nerve.
- Submental: Provides access to the anterior body, parasymphyseal, and symphyseal regions. Relatively safe approach, leaves external scar.
- Retromandibular: Access to the entire ramus and up to the subcondylar region. Risk of external scar and facial nerve injury.
- Preauricular: Provides access similar to the retromandibular.
- Facelift: Similar access to retromandibular and preauricular incisions with a more cosmetically favorable incision.

Orbit

- Subciliary: Lower lid blepharoplasty incision, direct or stair-step incision to orbital rim/floor. Risk of visible incision, soft tissue descent, lower lid malposition → resuspend soft tissues; place Frost stitch.
- Transconjunctival: Incision at inferior edge of the tarsus, followed by dissection through the septum (retroseptal) or anterior to the septum (preseptal). Orbital fat enters field with retroseptal approach. May need to add lateral canthotomy and cantholysis to improve exposure to orbital floor and lateral rim. No visible incision.
- Transcaruncular: Incision through caruncle to access medial orbital wall → dissection plane between Horner's muscle and medial orbital septum (Horner's muscle inserts onto posterior lacrimal crest, orbital septum along posterior surface of Horner's muscle). Risk of inferior oblique muscle disinsertion [5]. *Inferior fornix incision* extends exposure to orbital floor → requires disinsertion of Lockwood ligament and inferior oblique muscle (need to reconstruct when closing).
- Lateral brow: Incision along lateral eyebrow, provides access to superior orbital rim and zygomaticofrontal suture. No significant neurovascular structures are involved with this approach. Incision hidden within brow.
- Upper blepharoplasty**:** More cosmetically appealing access to the superolateral orbital rim.

Maxilla

- Vestibular/sublabial: Access to inferior orbital rim, zygomaticomaxillary suture, and majority of anterior midface. Infraorbital nerve located 7–10 mm below infraorbital rim, just medial to the zygomaticomaxillary suture. Incisions can be combined with intranasal incisions for midfacial degloving. Nasal support needs to be re-established for adequate projection and tip support. If incision extends bilaterally, nasal base must be resuspended and upper lip advanced with V-Y flap.
- Zygoma: Isolated zygomatic arch fractures can be approached via two incisions:
  1. Gilles: Incision within temporal scalp, 2.5 cm superior and anterior to root of the helix → dissection through superficial temporal fascia and deep temporal fascia → elevator dissection between deep temporal fascia and temporalis muscle until zygomatic arch
  2. Keen: Intraoral incision at ZM suture → subperiosteal dissection up to arch

Bicoronal Approach

Provides access to superior facial skeleton, ZMC complex, nasal bones, and NOE region. Incision placed 1 cm behind hairline and extends to root of the helix. Raise flap in supraperiosteal plane until supraorbital rim. Pericranial flap can be raised for facial reconstruction. If supraorbital nerve bundle exits from the foramen, use osteotome to release bundle and expose superior orbit.

Lateral to temporal line, dissection is along the deep temporal fascia (frontal branch of CN VII travels within superficial temporal fascia a.k.a. temporoparietal fascia). Avoid trauma to temporal fat pad to prevent temporal wasting. Periosteum over zygomatic arch is incised to expose fracture for reduction and plating.

Endoscopic Browlift Approach

Used for anterior table fractures → vertical working incision and adjacent endoscope incision → subperiosteal elevation to expose fracture and surrounding bone. Minimal risk with approach and cosmesis.

Facial Fractures

Nasal Fractures

Most common facial fracture. Despite the high frequency, controversy still exists on proper management of nasal fractures. Classically, these are best treated with closed nasal reduction within 7–10 days.

While closed reduction does achieve adequate results a majority of the time, open treatment has been shown to get better results. For complex fractures, those involving a largely deviated septum, or those outside of a 2-week window, open reduction +/− osteotomies should be considered. Furthermore, if there is significant septal deformity, releasing the ULCs as well as raising mucoperichondrial flaps can release deformational forces and allow proper realignment [6].

Osteotomies are avoided by many during the acute time period due to fear of instability; however, it has been shown to be an effective maneuver for achieving symmetry (often only need to osteotomize one side, due to trauma already breaking the first).

In the setting of severely comminuted nasal bones, fixation should always be attempted first with miniplating systems. If there are not enough fragments remaining, calvarial (or iliac) bone grafting should be done with the initial repair. This should not be placed over any remaining bone to avoid over-augmentation.

Zygomaticomaxillary Complex (ZMC) Fractures

Combination of fractures at the ZF suture, ZM suture, zygomatic arch, infraorbital rim, and zygomaticosphenoid suture. Strongest bone is ZF buttress; weakest bone is orbital floor. Nondisplaced fractures can be treated conservatively. If zygoma is displaced or comminuted, then ORIF is needed. Malar eminence typically located 2 cm inferior to lateral canthus. Malar flattening can occur due to posterior inferior rotation → reduce fracture segment with anterior superior rotation.

Zingg classification of zygoma fractures:

- A – one component of tetrapod fracture
- B – injury to all four supporting structures
- C – comminution of zygomatic bone

Isolated zygomatic arch fractures:

- Medially displaced arch – approach with either Keen incision or Gilles incision
- Laterally displaced arch – approach with coronal incision

ZMC fractures typically treated with 2- or 3-point fixation using ZF suture, ZM suture, and infraorbital rim. Expose all fractures and reduce zygoma before plating. If zygoma is inferomedially displaced, the zygoma can be reduced with elevator or Carroll-Girard screw (screw placed through small skin incision into body of zygoma). Ensure ZS suture is adequately reduced before plating:

- ZF suture plated first.
- Thickest plates at lateral buttress (1.5–2 mm); thinnest plates at infraorbital rim and ZF suture (1–1.5 mm).
- Zygomatic arch is plated when buttresses are comminuted or when arch is not anatomically reduced (*arch is plated first before ZF suture*); arch has straight configuration, not curved [7].

Cheek numbness commonly due to damage to infraorbital nerve, less commonly zygomaticotemporal and zygomaticofacial nerves. Diplopia results from trauma to extraocular muscles, muscle entrapment, or nerve damage. Enophthalmos – displacement of 1 ml of soft tissue or increase in 1 ml of orbital volume produces 1 mm of enophthalmos (3 mm enophthalmos becomes perceptible). Reduction of medially impacted zygoma can unmask enophthalmos caused by orbital floor/wall fractures.

Naso-Orbito-Ethmoid Fractures

Involves rigid bones (nasofrontal process of maxilla, origin of nasal bones, internal angular process of frontal bone) and fragile bones (ethmoid labyrinth and orbital floor/wall).

Medial canthal tendon → extension of tarsal plates. Receives contributions from preseptal/pretarsal orbicularis oculi, Whitnall's superior suspensory ligament, and Lockwood's inferior suspensory ligament. Three heads: superior limb attaches to frontal process of maxilla, posterior limb attaches to lacrimal fossa, and anterior limb fans out to attach to nasal bones.

Normal intercanthal distance 30–35 mm (intercanthal distance = ½ interpupillary distance). Pathologic posterolateral movement of lacrimal bone from fracture may cause telecanthus → almost impossible to correct telecanthus in delayed fashion if not repaired well initially.

Fracture classification:

- Type 1: single non-comminuted central fragment without MCT disruption
- Type 2: comminution of central fragment without MCT disruption
- Type 3: comminution of central fragment with MCT disruption

Approaches: Bicoronal, transconjunctival, vestibular/sublabial, open sky, or existing lacerations.

Treatment: medial orbital wall must be reconstructed prior to MCL:

- Type 1:
  - Nondisplaced → observation
  - Displaced → 2-point fixation (frontal bone to central fragment, maxilla to central fragment); exposure requires above (bicoronal) and below (sublabial).
- Type 2: more extensive exposure required to locate the MCT and central segment of bone. Treat with ORIF +/− transnasal wiring:
  - Transnasal wiring: Drill holes are placed above and below the tendinous insertion, and 26–28 gauge wire is passed through the drill holes. Transnasal wires are then passed *posterior and superior to the lacrimal fossa* on the contralateral side and secured to a screw or plate, reducing the central bone segment. Drilling must always be done below the frontoethmoid suture to avoid damage to the cribriform plate. Lastly, microplates are used as in type 1 fractures to get adequate reduction [8].
- Type 3: wide exposure required to locate MCT, treat with transnasal wiring, reconstruct MCT, and ORIF if fracture segments large enough to handle microplates. Bone grafts may be required for tissue loss.
- Cantilevered calvarial bone grafts used to establish nasal projection after loss of nasal bone structure. Calvarial bone graft length determined intraoperatively based on patient anatomy. Donor site easily accessible from bicoronal incision. Graft secured with screws or miniplate. Care must be taken to establish the proper nasofrontal angle. Drilling a groove into the glabella may facilitate a more secure graft and cosmetically favorable angle.
- Nasolacrimal duct injuries addressed at the time of fracture repair. Silastic stents placed to maintain patency.

Excessive nasoethmoid width can persist due to:

- Failure to reduce medial orbital rims.
- Unrecognized lateral displacement of zygoma (zygoma position sets up reduction of medial maxilla).
- Failure to reconstruct MCT, MCT stripped from bone during procedure.
- Scar tissue thickening may mimic telecanthus.
- Suturing of canthal ligament from inside coronal incision approach rather than externally at eyelid commissure.

Frontal Sinus Fractures

Development: not present at birth, starts at age 2, radiographically detectable at age 6, reaches adult size at ages 12–15. Unilateral in 10%, rudimentary in 5%, absent in 4% of population.

Frontal sinus closely associated with orbit, frontal lobe, and cribriform plate. Anterior table is thick, and posterior table is thin. Sinus drains posteromedially into the nasofrontal duct.

Frontal sinus fractures involve the anterior table only in 50% and anterior and posterior table in 50%. Isolated posterior table fractures rare. Structures to consider when evaluating frontal sinus fractures: anterior table, posterior table, nasofrontal duct. Other injuries to consider: dural tears, CSF leak, cerebral trauma.

Classification [9]:

- Type 1: isolated anterior table fx
- Type 2: comminuted anterior table fx +/− NOE, orbital rim fx
- Type 3: anterior + posterior table fx
- Type 4: anterior + posterior table fx + CSF leak/dural injury
- Type 5: anterior + posterior table fx, CSF leak, tissue loss, severe disruption

Treatment:

- Observation – anterior table fx (type 1) without displacement or deformity
- Endoscopic – anterior table fx (type 1) with deformity:
  – Fx camouflage easier and more efficacious compared to endoscopic reduction. Wait 3–4 months for edema to subside before repairing fracture. Subperiosteal dissection is similar to endoscopic browlift approach. Porous polyethylene implant carved, inserted over defect, secured with one or two screws. Implant is palpable, but has no discomfort, contour deformity, or mobility [10].
- ORIF – anterior table fractures (types 1, 2) with no nasofrontal duct injury
- Obliteration – anterior + posterior table fx (types 2, 3) with nasofrontal duct injury:
  – Bicoronal incision, remove all mucosa from frontal sinus, pack frontal sinus and outflow tract with fat, finish with ORIF of frontal sinus
- Cranialization – CSF leak and extensive injury (types 4, 5):
  – Similar approach as obliteration, remove posterior table fragments, place pericranial flap to separate sinus from nasal cavity
- CSF leak – send fluid for tau transferrin to diagnose, can look for "halo" sign (bloody drainage on gauze will have the appearance of halo or target if CSF present). Can manage conservatively initially with head of bed elevation, stool softeners, lumbar drain. Antibiotics administration debated (Fig. 12.1).

Orbital Fractures

- Anatomy: The orbit is a four-sided pyramid with many bony contributions. The orbital apex houses the optic nerve, while the superior orbital fissure contains CNs III, IV, V1, and VI and the inferior orbital fissure CN V2. The superior aspect of the orbit is formed largely by frontal bone, while laterally it is the zygoma and greater wing of sphenoid. Medially are the ethmoid and lacrimal bones, and inferiorly are the maxilla, palate, and zygoma.
- Hydraulic Theory: Direct force to the orbit increases orbital pressure which is transmitted to the point of greatest weakness resulting in inferior displacement of the orbital floor contents into the maxillary sinus. Medial wall and floor most fragile.
- Buckling Theory: Direct force applied to inferior rim transmits anterior to posterior force resulting in fracture of orbital floor. Can be associated with soft tissue injury.
- Fracture Classification: Important to identify all the structures involved, displacement, size, and location of orbital defects. Orbital floor fracture patterns determined by location of force transduction:
  – Type 1 fracture pattern – force applied to rim, no herniation of orbital contents

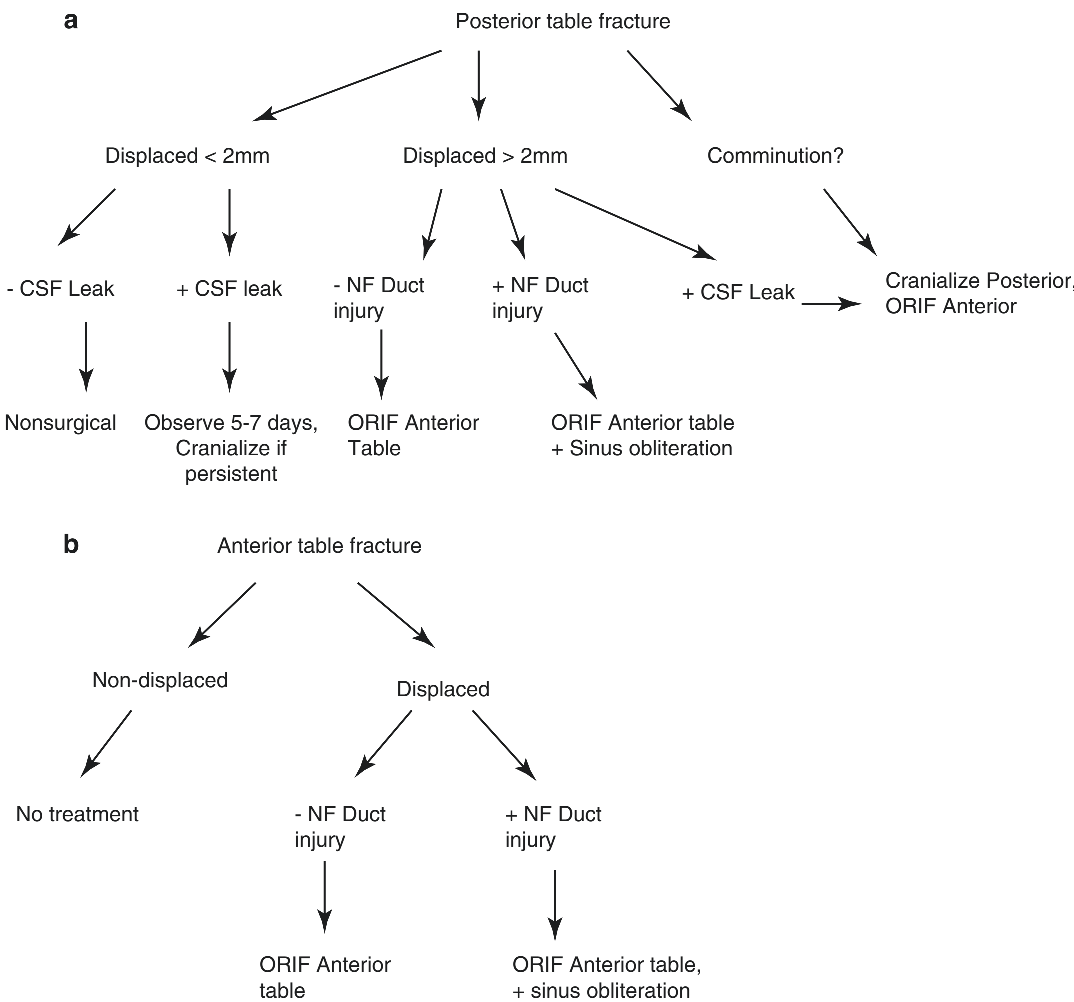


Fig. 12.1 (**a**, **b**) Management of frontal sinus injuries. Note that degrees of involvement of the nasofrontal duct, posterior table, and anterior table all influence management

  – Type 2 fracture pattern – force applied to globe, larger defect, typically involves medial wall and orbital floor as well as herniation of contents
- Evaluation: Extraocular mobility (forced duction), pupil reactivity, visual acuity, ocular pressure. Comorbid injuries to globe are common; obtain ophthalmology consultation. Emergent issues include severely elevated intraocular pressure (due to retrobulbar hematoma, emphysema, or severe shift in orbital contents) resulting in vision loss/ischemic damage to the retina and optic nerve:
  – Traumatic optic neuropathy (2–5% of severe facial trauma) – vision loss, diminished color perception, APD. Ophtho consult. Consider high-dose corticosteroids, osmotic diuresis, and nerve decompression. For bony nerve impingement, consider urgent decompression.
  – Oculocardiac reflex – vagal stimulation causes nausea, vomiting, bradycardia, and heart block.
  – Diplopia may be due to entrapment (especially in peds) or enophthalmos. Entrapment associated with severe pain, decreased ocular mobility.

– Enophthalmos >2 mm clinically noticeable – occurs with fracture defect >2 cm^2, >2 ml of herniation, or change in shape of orbit from conical to round.

Treatment is typically more conservative in adults and more aggressive in peds. Approaches include existing lacerations, subciliary, transconjunctival +/− lateral canthotomy and cantholysis, or transcaruncular +/− inferior fornix extension. No matter the approach, once the orbital rim is reached, it is important to dissect and elevate the periosteum/septum to help retract orbital contents. The contents of the fracture are elevated completely with ribbon retractor, and the floor is reconstructed. Implant materials include split calvarial bone graft, titanium mesh, porous polyethylene, and Silastic sheets.

Recommendations for repair of isolated orbital floor fractures [11]:

- Immediate repair
  – Oculocardiac reflex
  – White eye fx (entrapment and diplopia in peds)
  – Significant asymmetry (>50% floor involvement +/− soft tissue distortion)
- Within 2 weeks
  – Diplopia without improvement
  – >50% floor involvement without diplopia
  – Infraorbital nerve hypesthesia (according to some case series)
- Observation
  – Minimum diplopia
  – Good motility
  – No significant enophthalmos

Complications include dry eye, vision loss, ectropion, lid malposition, diplopia, and enophthalmos.

Post-op diplopia more likely in older patients (28 years vs 46 years) and delayed reconstruction (>2 weeks). Sex, location of defect, and reconstruction materials have no effect on post-op diplopia [12]. Early surgery likely minimizes fibrosis and contracture.

Delayed enophthalmos typically from enlargement of orbital volume → rounding of soft tissues, loss of ligamentous support, globe moves posteriorly, lateral/inferior displacement of lateral rim. Enlargement most common at (1) the inferior orbital fissure, followed by (2) medial orbit and (3) the greater wing of the sphenoid [13]. *Fat atrophy is not a major cause of late enophthalmos* [14]. Secondary orbital reconstruction less likely to resolve persistent diplopia.

Surgical contraindications: hyphema, open globe, retinal tears/detachment, monocular, frozen globe.

Maxillary/Le Fort Fractures

Large air-filled sinus cavities predispose the midface to fracture with far less force than adjacent facial bones. All Le Fort fx involve pterygoid plate fx. Fracture patterns:

- Le Fort I: horizontal fx between maxilla and palate, involving nasomaxillary (medial) and zygomaticomaxillary (lateral) buttresses. Seen in 30% of Le Fort fx.
- Le Fort II: involves nasal bones, frontal process of maxilla, lacrimal bone, orbital floor, infraorbital rim, and lateral maxillary sinus. Seen in 60% of Le Fort fx.
- Le Fort III: craniofacial disjunction, involves nasofrontal suture, frontal process of maxilla, lacrimal bones, lateral orbital wall, and ZF suture.
- Palatal and alveolar ridge fractures may be seen in combination with the abovementioned fx patterns.

Early intervention associated with earlier return to function, less infection, decreased scarring, and fewer overall complications. Delay repair in patients with GCS <6, intracranial hemorrhage, or midline cerebral shifts.

Oral intubation allows manipulation of the midface, but may interfere with occlusion. Nasal intubation allows occlusion, but can make nasal and maxillary reduction difficult. Tracheostomy an option for maintaining safe airway while allowing occlusion and MMF.

Multiple approaches used to access the midface. Disimpact and reduce the midface with Rowe forceps. Establish occlusion with MMF. Duration of MMF dependent on degree of fracture and stability achieved with repair. Bone graft may be needed for tissue loss. Low-profile titanium plates used for fixation of fx. Direct plating of palatal fx establishes width, but MMF required to prevent rotation of alveolus.

Titanium mesh can be used to reconstruct large defects in bone along the maxilla [15]. Fibrous capsules form around the implant. Increased thickness of the capsule correlates with increased risk of implant complications: coarse plates form thinnest capsule; smooth plates form the thickest capsule. Neo-mucosa forms over titanium mesh – shows evidence of ciliated respiratory epithelium, goblet cells, chronic inflammation, and squamous metaplasia.

Anterior open bite deformity results when the pterygoid buttress descends inferiorly and posteriorly after midface fx. Persistence of anterior open bite after surgery is due to early release from MMF, late fracture reduction, or placing elastic traction on posterior dentition only.

Class II occlusion (overbite) persists if an anteriorly displaced and impacted maxilla is placed in MMF. The TMJ can slide forward to allow the mandible to accommodate MMF, but slides back after the MMF is released.

Mandible Fractures

Symphysis, parasymphysis, body, angle, ramus, condyle.

Dental numbering (adults) – 8 teeth per quadrant (3 molar, 2 premolar, 1 canine, 2 incisors):

- Right maxillary third molar (1) → Left maxillary third molar (16)
- Left mandibular third molar (17) → Right mandibular third molar (32)

Occlusion:

- Class I (normal occlusion): first maxillary mesiobuccal cusp sits in first mandibular mesiobuccal groove.
- Class II (overbite): first maxillary mesiobuccal cusp anterior to first mandibular mesiobuccal groove, retrognathia.
- Class III (underbite): first maxillary mesiobuccal cusp posterior to first mandibular mesiobuccal groove, prognathia.
- Open bite: normal relationship of molars; anterior dentition doesn't contact.
- Cross bite: upper dentition fits into wrong side of lower dentition.

Evaluation: ABCs, C-spine status, intraoral exam (occlusion, missing dentition, laceration/open fractures, stability of the mandible), facial lacerations, lower face deviation, condylar head through EACs. Abx for open fractures. Teeth in fracture site don't necessarily need to be pulled unless carious or fractured through root. Removal leaves entry point for infection and point of weak fixation. Timely treatment important but rarely an emergency (classic teaching was within 24 hours, but increasing date and practice trends point to repair within 6–10 days without increased risk of complication).

Fracture Classification

Largely based on the location of the fracture (see Fig. 12.2).

- Fracture favorability: Determined by direction of fracture line and muscle force on mandibular segment. Masseter and temporalis muscles (elevators) can create horizontally unfavorable force of angle fractures. Lateral pterygoid muscles (protrusor) and medial pterygoid muscles (elevator) can pull apart body and symphyseal fractures due to vertically unfavorable forces.
- Treatment considerations: Take into consideration location of fracture, comminuted vs simple, open vs closed, favorable vs unfavorable fractures. Fractures treated with MMF +/− ORIF depending on the abovementioned factors.
- Closed treatment (MMF) for comminuted fractures that are minimally displaced/nondisplaced. External pin recommended for severe

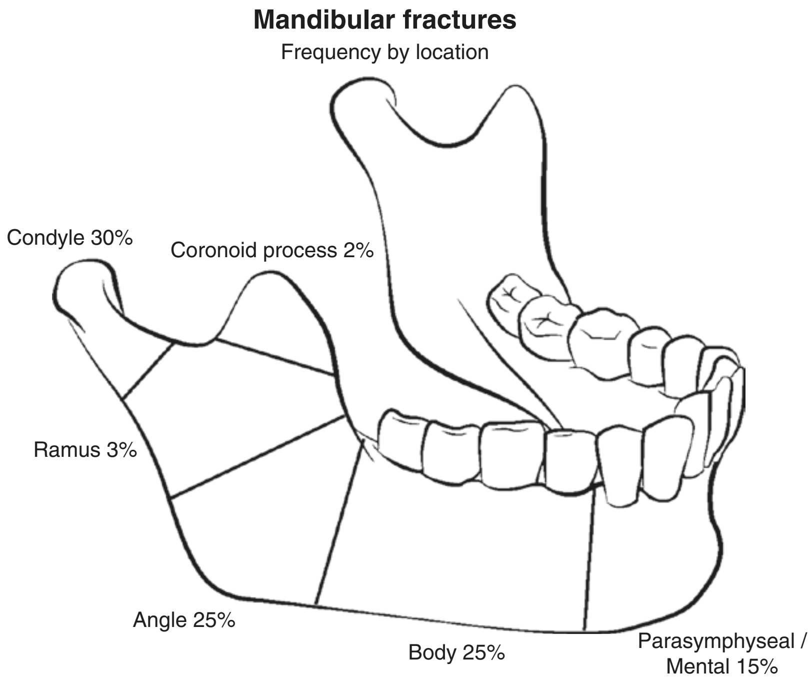


Fig. 12.2 Prevalence and location of mandible fractures. Accurate diagnosis of fracture(s) is important to determine proper management

comminution and soft tissue disruption. Closed treatment avoids removing too many displaced fragments and minimizes risk of devitalizing tissue from open approaches in setting of severe soft tissue damage [16]. External pins and gunshot wounds increase the risk of non-occlusal complications. The number of fragments in fracture has no impact on development of non-occlusal complications. Number of fragments, mechanism of injury, and treatment method don't have any impact on development of malocclusion. Can be removed 2–6 weeks post placement depending upon fracture and age: condyle/subcondylar, 2 weeks; body/angle: 4 weeks; symphyseal/parasymphyseal, 6 weeks.

- Subcondylar fractures typically treated conservatively with MMF and early mobilization. Indications for ORIF include condylar neck/subcondylar displacement (>15°) and ramus height instability/loss (>5%), bilateral fractures, and foreign body in TMJ. Indications for MMF include good range of motion, good occlusion, minimal pain, or isolated condylar head fx. Under similar conditions, ORIF should be performed over MMF. Approaches include retromandibular, preauricular, and endoscopic. Compression plates should be used for ORIF, as adaptation plates are less favorable. Risks of ORIF include scars and temporary paralysis from external approaches. MMF risks include pain, malocclusion, asymmetry, and limited mobility [17]. Bilateral fractures at risk for airway compromise (consider tracheotomy/intubation). Present as anterior bite deformity. Need early movement to prevent TMJ ankylosis.
- Body and parasymphyseal fractures: Tend to be vertically unfavorable. Generally best treated with ORIF through intraoral incisions. Severely comminuted, greenstick, or unstable patients can be treated with closed reduction [16]. Intraoral incision is made leaving cuff of gingival soft tissues, dissection subperiosteally to expose fracture and mental nerves. A 2.3 or 2.7 mm compression plate is placed inferiorly with ≥2 bicortical screws on each side. Next, a tension band (2.0 mm plate) is placed superiorly over the fracture (two monocortical screws on either side). For oblique fractures a bicortical lag screw can be used for fixation.

- Angle fractures can be repaired in several ways. Nondisplaced or favorable fractures can be treated with 6 weeks of MMF. Champy plate can be placed with intraoral incision. This utilizes compressional forces; the plate is placed after MMF and is located along the oblique ridge. Alternatively, two miniplates can be placed inferiorly and superiorly over the fracture (frequently need percutaneous screw placement). Severe fractures may require an external approach. Highest rate of complications due to location and vector forces from the masseter.
- Symphyseal fractures: Look for coexisting condylar/subcondylar fractures due to mechanism of injury. Anterior-posterior force transmitted to condyles resulting in fracture. Can be managed with tension/compression plates or lag screw placement.
- Ramus fractures: Rare due to overlying protection from masseter muscle. ORIF indicated for displaced or multiple fragments.
- Complications: Chin/lip hypesthesia most common complication secondary to mental nerve injury. Osteomyelitis from unstable segments, loss hardware, infected tooth root in fracture site, poor oral hygiene. Treatment involves removal of unstable hardware and infected teeth and debridement of nonviable bone and IV abx. Malunion, nonunion (fibrous union), exposed hardware, aseptic necrosis of condylar head, TMJ ankylosis, dental injury.

Panfacial Fractures

Anatomy: See Fig. 12.3.

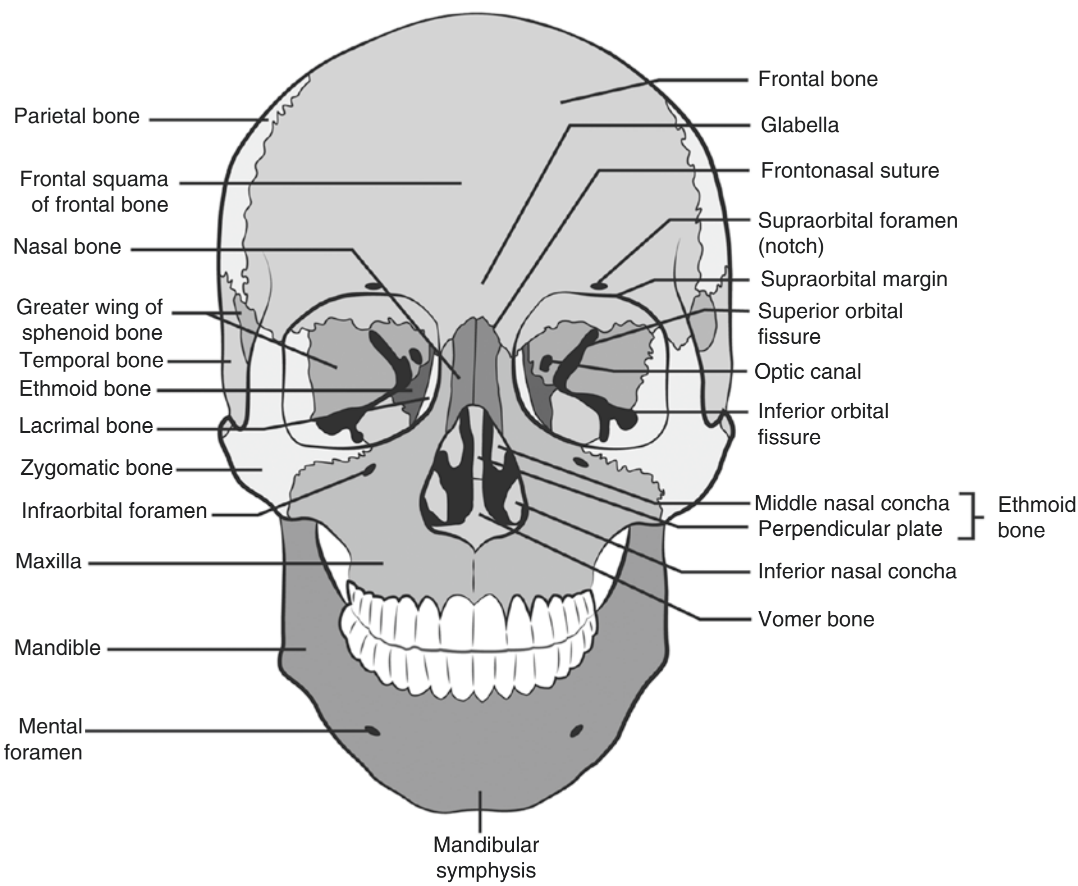


Fig. 12.3 Facial skeleton anatomy. Complex relationship of the facial bones forms the vertical and horizontal buttresses for stability

Four vertical buttresses: Nasomaxillary, vertical portion of the mandible, zygomaticomaxillary, pterygomaxillary.

Three horizontal buttresses: Frontal bar, infraorbital rim/nasal bones, maxillary alveolus/hard palate.

Establishment of the facial width is one of the primary goals in repairing panfacial fx. If untreated, the face becomes spherical due to increased width and decreased projection.

The sequence of repair varies depending on the text (i.e., known to unknown, superior to inferior, inferior to superior, inside out, outside in). Manson et al. [13] divide the face into upper and lower halves at the Le Fort I level and sequence the lower face and upper face starting at the most stable structure in each half:

- Lower face composed of occlusal unit (teeth, palate, alveolar processes) and mandibular unit (condyle, ramus, angle, body, symphysis):
  - Reduction sequence: palate → vertical mandible → horizontal mandible
- Upper face composed of upper face (frontal bone, supraorbital rim, orbital roof, frontal sinus) and upper midface (zygoma, nasoethmoid, lateral and inferior orbit):
  - Reduction sequence: cranial base and orbital roof → frontal sinus and frontal bone → nasoethmoid → zygoma

Pediatric Facial Fractures

Pediatric facial fx differ significantly from adult facial fx due to anatomic and physiologic differences, developmental and functional impact of treatment, and different treatment algorithms.

Anatomy: The midface is much more protected in peds than in adults. Facial trauma less common in peds due to increased cranial to facial volume (8:1 at birth, 2.5:1 in adults), retruded position of the face relative to the skull, flexible suture lines, lack of sinus pneumatization, and increased mandibular density from tooth buds. The orbit reaches adult size at age 5. The mandible undergoes lingual bone absorption and labial bone deposition and increases in size during puberty. Primary dentition erupts by age 3, and permanent teeth erupt starting at age 6, becoming stable for surgical intervention by age 14.

Bone healing in peds is much faster due to greater osteogenic potential in the periosteum. Faster fracture healing rates result in *fracture union at 3 weeks* (compared to 6 weeks in adults).

Mandible fx: Irregular fx lines due to tooth buds and crypts. Condylar head/neck fx most common. Conservative management favored due to high osteogenic potential, presence of primary and secondary dentition, and potential adverse effects of ORIF on craniofacial growth:

- Nonsurgical management (ORIF, PT, pain control): used for condylar fx and nondisplaced/greenstick fx of body and ramus, has advantage of decreased immobilization, muscle atrophy, ankyloses, and improved oral hygiene [18]. Not indicated for significantly displaced fx.
- Surgical management for body, angle, and parasymphyseal fx:
  - Closed reduction: interdental wires if dentition stable, occlusal splints if dentition unstable.
  - ORIF for displaced fx, decreases immobilization time and improves dental hygiene. Use of titanium plates is controversial because of potential growth impediment from periosteal stripping and removal of growth stimulus, possible dental trauma from screw tips (avoid bicortical screws), and need for secondary surgeries to remove plates. Absorbable plates (poly-L-lactic acid, polyglycolic acid) are bulkier and less malleable and require pre-drilling and pre-tapping, but don't require secondary surgeries for plate removal.

Orbital floor fractures are most common in peds, but there is an increased risk of orbital roof fractures in children <10 years due to decreased sinus pneumatization. Peds orbital trauma presents with less swelling, but more likely to have serious sequela of diplopia and EOM limitation. Greenstick/trapdoor fx more likely in children → entrapment of inferior rectus → muscle necrosis and fibrosis → diplopia. Peds orbital fx are managed more conservatively – surgery reserved for enophthalmos, entrapment, and oculocardiac symptoms only [19]. Repair should be undertaken <7 days, but trapdoor fx are true surgical emergencies and should be repaired <24 hours [20].

Nasal trauma is common in this population. The nasal bones have not fused yet, and the cartilage is relatively larger and more pliable than adults. Conservative reduction is recommended and there should always be a high index of suspicion for septal hematoma.

Questions

1. When evaluating a patient in the ED with complex laceration to the right cheek and orbital area, you note the patient has a full-thickness eyelid laceration through the upper lid, extending medially, as well as MRD1 of 1. Which is of least concern when repairing at bedside?
   (a) Meticulous alignment of the tarsus
   (b) Evaluating for corneal integrity
   (c) Correction of ptosis by reapproximating lid retractors
   (d) Evaluating and stenting lacrimal duct
2. When evaluating the same patient in the ED, you note another knife wound to the right cheek, approximately 2 cm anterior to the tragus and just below the zygomatic arch. Facial motion is intact throughout. Upon massage, you note some saliva within the wound. Next step in management?
   (a) Allow to granulate in, place on abx.
   (b) Cannulate the duct and perform anastomosis.
   (c) Close the parotid parenchyma and layered wound closure, abx.
   (d) Close skin only, ENOG after 72 hours, repair facial nerve if needed.
3. A 7-year-old male presents to the emergency room after falling off a bicycle. Physical examination showed trismus secondary to pain and tenderness over the left condyle. His dentition is intact and stable. CT scan showed only a minimally displaced left subcondylar fracture and a nondisplaced right parasymphyseal fracture. What is the best treatment plan?
   (a) MMF only for 3 weeks
   (b) MMF and ORIF for 3 weeks
   (c) MMF only for 6 weeks
   (d) MMF and ORIF for 6 weeks
4. A 9-year-old female presents to the emergency room after a MVC. Examination of the mandible showed multiple absent and unstable teeth as well as a mucosal laceration over the right mandibular gingiva. CT scan showed a nondisplaced fracture over the right mandibular parasymphysis. What is the best treatment option?
   (a) Interdental wiring
   (b) Occlusal splints with circum-mandibular wiring
   (c) MMF with drop wires
   (d) ORIF with titanium plates
5. Compared to adult orbital fractures, pediatric orbital fractures have more of the following signs and symptoms *except*:
   (a) Greenstick fracture on CT
   (b) Entrapment with diplopia
   (c) Periorbital edema
   (d) Clinically detectable enophthalmos
6. A 6-year-old male presents to the emergency room after being struck in the eye by a baseball. He had no loss of consciousness. He is resting comfortably without nausea. His visual acuity is normal. He has 3 mm of enophthalmos on exam. CT shows a defect over 60% of the orbital floor. Which part of the history and physical is the indication for surgical intervention?
   (a) Enophthalmos of 3 mm
   (b) Orbital floor defect 60%
   (c) Oculocardiac symptoms
   (d) None of the above
7. All of the following are characteristics of the pediatric facial skeleton EXCEPT:
   (a) Decreased sinus pneumatization
   (b) Increased cranial volume to facial volume
   (c) Decreased osteogenic potential
   (d) Increased mandibular angle
8. Which of the following statements regarding ORIF and pediatric mandible growth is true?

(a) The symphysis is the growth center of the mandible.
(b) Load-bearing potential from titanium plates doesn't impact growth potential.
(c) Periosteal stripping has no impact on mandibular growth.
(d) Titanium plate migration may occur if not removed at a second surgery.

9. A 25-year-old male presents with a gunshot wound through the right mandibular body. CT scan shows multiple comminuted fracture segments. Which of the following confers the highest risk for non-occlusal complications?
(a) Number of bone fragments
(b) Closed treatment with external pins
(c) Open treatment with MMF
(d) Open treatment with rigid internal fixation

10. A 58-year-old male suffered a severely displaced left ZMC fracture after a MVC. On questioning, he complains of new visual disturbances since the trauma. Which of the following symptoms is NOT associated with traumatic optic neuropathy?
(a) Vision loss
(b) Diminished color perception
(c) Afferent pupillary defect
(d) Diplopia

11. When treating a ZMC fracture with displacement of the arch and comminution of the ZF buttress, ZM buttress, and infraorbital rim, which location should be plated first?
(a) Zygomatic arch
(b) Zygomaticofrontal buttress
(c) Zygomaticomaxillary buttress
(d) Infraorbital rim

12. What is the most common life-threatening injury associated with facial fractures?
(a) Cervical spine injury
(b) Cerebral trauma
(c) Airway compromise
(d) Hemorrhagic shock

13. A 42-year-old female involved in an MVC suffered a frontal sinus fracture. CT scan shows comminuted anterior table fracture and nondisplaced posterior table fracture. After visualizing the frontal sinus via a bicoronal approach, a dural tear and CSF leak are identified. What is the best treatment option?
(a) Terminate the procedure, place a lumbar drain, and start antibiotics.
(b) ORIF of the anterior table and place a lumbar drain.
(c) Remove the posterior table, cranialization, and ORIF of the anterior table.
(d) Remove the frontal sinus mucosa and obliterate the frontal sinus with fat.

14. A 35-year-old male undergoes a transcaruncular approach to repair an isolated medial orbital wall fracture. Repair is performed by placing a porous polyethylene implant over the defect after elevating the orbital contents out of the defect. The incision is extended into the inferior fornix in order to insert the implant. The surgery is completed without complication, but the patient complains of diplopia on downward gaze after surgery. What is the most likely reason for diplopia?
(a) Change in orbit shape from conical to round
(b) Entrapment of medial rectus muscle
(c) Occult orbital floor fracture
(d) Disinsertion of the inferior oblique muscle

15. A 20-year-old male presents after blunt trauma to the jaw. He has minimal pain and no trismus. His dentition appears intact. CT scan shows a left condylar head fracture that is comminuted. What is the best treatment option?
(a) Soft diet
(b) MMF
(c) ORIF with compression plate
(d) ORIF with adaptation plate

16. A 20-year-old male presents after blunt trauma to the jaw. He has minimal pain and mild trismus. His dentition appears intact. CT scan shows a minimally displaced left subcondylar fracture. Considering the long-term complications of condylar fracture treatment, which of the following is the best treatment option?
(a) Soft diet
(b) MMF

(c) ORIF with compression plate
(d) ORIF with adaptation plate

17. When determining the timing of pure orbital fracture repair, which of the following clinical scenarios could be repaired 1–2 weeks after trauma?
    (a) An adult with intractable nausea and vomiting
    (b) A pediatric patient with motility restriction but without ecchymosis or edema
    (c) An adult with progressive infraorbital hypesthesia
    (d) A pediatric patient with minimal diplopia and good ocular motility
18. A combined transcaruncular and inferior fornix approach is used to repair a combined medial orbital wall and orbital floor fracture. Which of the following statements is correct?
    (a) Dissection is between Horner's muscle and orbital septum, and the inferior oblique muscle and Lockwood's ligament are disinserted.
    (b) Dissection is between the inferior oblique muscle and Lockwood's ligament, and Horner's muscle and the orbital septum are disinserted.
    (c) Dissection is between Horner's muscle and inferior oblique muscle, and the orbital septum and Lockwood's ligament are disinserted.
    (d) Dissection is between the orbital septum and Lockwood's ligament, and Horner's muscle and the inferior oblique muscle are disinserted.
19. A 50-year-old male presents 1 week after trauma with an orbital blowout fracture. He has diplopia on exam. CT scan shows an orbital floor defect involving approximately 60% of the floor. He undergoes immediate reconstruction with a Medpor implant. Which of the following is the biggest risk factor for postoperative diplopia?
    (a) Gender
    (b) Age
    (c) Location of defect
    (d) Reconstruction material
20. Which of the following does NOT cause late enophthalmos after orbital trauma?
    (a) Enlarged posterior orbit volume
    (b) Lateral displacement of the lateral orbital rim
    (c) Loss of ligamentous support
    (d) Fat atrophy
21. A 44-year-old woman presents 5 years after a right ZMC fracture. She underwent initial reconstructive surgery but presents today with persistent enophthalmos and diplopia. In addition, her physical exam is notable for hypoglobus and superior sulcus deformity. After undergoing revision surgery involving osteotomies and repositioning of the ZMC, which of the following is NOT expected to improve?
    (a) Diplopia
    (b) Enophthalmos
    (c) Hypoglobus
    (d) Superior sulcus
22. After performing an endoscopic approach to a displaced anterior table frontal sinus fracture with placement of a Medpor implant, what is the most likely complaint in the postoperative period?
    (a) Implant mobility
    (b) Discomfort at the implant site
    (c) Persistent contour deformity
    (d) Implant palpability
23. True or false? Reconstruction of a large anterior maxillary wall defect with titanium mesh prevents soft tissue ingrowth with respiratory mucosa.
    (a) True
    (b) False
24. A patient is released from MMF 4 weeks after undergoing ORIF and MMF for panfacial fractures, including bilateral Le Fort I and II fractures. Shortly thereafter, he complains of his incisors not contacting when he bites. All of the following are possible reasons for his complaint EXCEPT:
    (a) Late release from MMF
    (b) Late fracture reduction
    (c) Placing elastic traction on posterior dentition

(d) Placing an anteriorly displaced and impacted maxilla on MMF

25. Telecanthus after treating nasoethmoid fractures can be due to all of the following EXCEPT:
    (a) Medially displaced zygoma fractures
    (b) Anteriorly positioned transnasal wires
    (c) Hematoma and scarring at the canthal area
    (d) Resuspending the medial canthal tendon via the coronal incision

Answers

1. (c)
2. (b)
3. (a)
4. (b)
5. (c)
6. (a)
7. (c)
8. (d)
9. (b)
10. (d)
11. (a)
12. (b)
13. (c)
14. (d)
15. (b)
16. (d)
17. (c)
18. (a)
19. (b)
20. (d)
21. (a)
22. (d)
23. (b)
24. (a)
25. (a)

References

1. Tung TC, Tseng WS, Chen CT, Lai JP, Chen YR. Acute life threatening injuries in facial fracture patients: a review of 1025 patients. J Trauma. 2000;49(3):420–4.
2. Papel ID. Facial plastic and reconstructive surgery. 3rd ed. New York: Thieme Medical Publishers, Inc.; 2002.
3. Leach J. Proper handling of soft tissue in the acute phase. Facial Plast Surg. 2001;17(4):227–38.
4. Sykes JM, Dugan FM Jr. Evaluation and management of eyelid trauma. Facial Plast Surg. 1994;10(2):157–71. NY: Thieme
5. Goldberg RA, Mancini R, Demer JL. The transcaruncular approach: surgical anatomy and technique. Arch Facial Plast Surg. 2007;9(6):443–7.
6. Staffel JG. Optimizing treatment of nasal fractures. Laryngoscope. 2002;112(10):1709–19.
7. Hollier LH, Thorton J, Pazmino P, Stal S. The management of orbitozygomatic fractures. Plast Reconstr Surg. 2003;111(7):2386–92.
8. Markowitz BL, Manson PN, Sargent L, Vander Kolk CA, Yaremchuk M, Glassman D, 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. 1991;87(5):843–53.
9. Manolidis S. Frontal sinus injuries: associated injuries and surgical management of 93 patients. J Oral Maxillofac Surg. 2004;62(7):882–91.
10. Kim KK, et al. Endoscopic repair of anterior table frontal sinus fractures with medpor implant. Otolaryngol Head Neck Surg. 2007;136(4):568–72.
11. Burnstine MA. Clinical recommendations for repair of isolated orbital floor fractures. Ophthalmology. 2002;109(7):1207–11.
12. Hosal BM, Beatty RL. Diplopia and enophthalmos after surgical repair of blowout fracture. Orbit. 2002;21(1):27–33.
13. Manson PN, et al. Subunit principles in midface fractures: the importance of sagittal buttresses, soft-tissue reductions, and sequencing treatment of segmental fractures. Plast Reconstr Surg. 1999;103(4):1287–306.
14. Kellman RM, Bersani T. Delayed and secondary repair of posttraumatic enophthalmos and orbital deformities. Facial Plast Surg Clin North Am. 2002;10(3):311–23.
15. Schubert W, et al. Incorporation of titanium mesh in orbital and midface reconstruction. Plast Reconstr Surg. 2002;110(4):1022–30.
16. Ellis E 3rd, Muniz O, Anand K. Treatment of considerations for comminuted mandibular fractures. J Oral Maxillofac Surg. 2003;61(8):861–70.
17. Brandt MT, Hag RH. Open versus closed reduction of adult mandibular condyle fractures: a review of the literature regarding the evolution of current thoughts on management. J Oral Maxillofac Surg. 2003;61(11):1324–32.
18. Smith DM, et al. 215 mandible fractures in 120 children: demographics, treatment, outcomes, and early growth data. Plast Reconstr Surg. 2013;131(6):1348–58.
19. Losee JE, et al. Pediatric orbital fractures: classification, management, and early follow-up. Plast Reconstr Surg. 2008;122(3):886–97.
20. Gerbino G, et al. Surgical management of orbital trapdoor fracture in a pediatric population. J Oral Maxillofac Surg. 2010;68(6):13010–1316.

Part III

Congenital Malformations

13 Cleft Lip and Palate

Dana K. Petersen and Christian P. Conderman

Introduction

Cleft lip with or without palate (CL ± P) and cleft palate in isolation (CPO) constitute the *most common congenital defects of the face* (second most common overall developmental defect following clubfoot). Despite similarities and overlapping characteristics, CL ± P and CPO should be considered as individual disorders with different embryologic, etiologic, and epidemiologic factors. Most current evidence points to CL ± P and CP being multifactorial in nature with innumerable contributions of both genetic and environmental factors. Additionally, patients with these conditions can have concomitant health issues including otologic disease (e.g., OME, ETD, rAOM, cholesteatoma), nutritional deficits, speech and language problems, dental deformities, sleep apnea, concurrent facial growth deformities, and psychosocial difficulties. Given the complexity inherent to these disorders and their treatment, a comprehensive management plan necessitates a multidisciplinary team approach including the otolaryngologist, plastic surgeon, speech-language pathologist, geneticist, pediatrician, nutritionist, orthodontist, and orthognathic specialist. Antepartum diagnosis is now a standard practice in most communities, and early diagnosis allows for parental counseling and enrollment into a cleft treatment program.

Epidemiology

- Distribution of clefts: 50% cleft lip and palate (CL ± P); 30% isolated cleft palate (CPO) and 20% cleft lip (CL) alone.
- CL ± P—in North America 0.2–2.3 cases per 1000 population and varies by ethnic groupings—3.6/1000 in Native Americans; 2.1 per 1000 in Asians; 1 in 1000 in Caucasians; 0.41/1000 in African-Americans:
  – Males/females:1.5:1
- CPO—0.1–1.1 cases per 1000; females/males, 2:1; females more likely to develop cleft palate (due to later closure of palatal shelves during development).
- CL—distribution 6:3:1 for left-sided CL, right-sided CL, and bilateral CL, respectively:
  – Right-sided CL more commonly associated with syndromes
- CL ± P associated with syndromes (<20% of total cases)—most commonly associated syndrome is van der Woude (see below):

D. K. Petersen (✉)
Head and Neck Surgery, Marin General Hospital, Greenbrae, CA, USA
e-mail: Dana.Petersen@Maringeneral.org

C. P. Conderman
Otolaryngology-Head and Neck Surgery, Division of Facial Plastic and Reconstructive Surgery, Orinda, CA, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_13

- Approximately 85% of bilateral cleft lips and 70% of unilateral CL associated with cleft palate
- CPO more commonly associated with syndromes than CL ± P—most commonly associated with autosomal dominant syndromes with microdeletion or addition at chromosome 22q11.2 (DiGeorge, velocardiofacial, conotruncal anomaly face syndrome):
  - 22q11.2 syndromes have incidence of 1 in 4000 live births.
  - CPO associated with syndromes 55% of the time.
- Approximately 30–65% of facial clefts appear to be associated with named syndromes.
- Recurrence risks: Table 13.1.

Etiology

Environmental

- Multifactorial for most non-syndromic cases of clefting.
- Maternal health—Both prepregnancy DM (though not gestational diabetes) and maternal obesity are known to increase risk. Meta-analysis has determined maternal age is not a factor:
  - Alcohol consumption during pregnancy, although definitive relationship with in utero alcohol consumption remains unclear.
  - Smoking, particularly high levels, are consistently associated with increased risk for clefting in numerous studies. Risk appears stronger for CL ± P than CPO.
  - Poor folic acid and vitamin intake, folate antagonists associated with increased risk of clefting (folate supplementation may play protective role though evidence more convincing for protective effect in neural tube defect).
- Use of valproic acid, phenytoin, retinoic acids, dioxin, thalidomide, corticosteroids.

Table 13.1 Risk of cleft lip with or without palate and cleft palate in isolation in subsequent children based on prior history of cleft disorders in relatives

Percentage likelihood of next child having cleft defect		
	CL ± P	Cleft palate
No family Hx of CL ± P or cleft palate	0.10%	0.04%
Parents normal, first child is affected		
No affected relatives	4%	2%
Affected relatives	4%	7%
Parents normal, two affected children	9%	1%
Parents normal, two affected relatives	9%	10%
One parent affected, no children affected	4%	6%
One parent affected, one child affected	17%	15%

Table adapted from *KJ Lee's Essential Otolaryngology*, and Bailey's 5th edition, Ch. 103, Comprehensive Cleft Care

Syndromes/Associated Disorders

- *Van der Woude Syndrome* (*VWS*)—most common syndrome associated with CL ± P; autosomal dominant (AD) inheritance, lower lip pits/sinus tracts, hypodontia; mutations of *interferon regulatory factor gene* (*IRF6*) felt to contribute to this disorder.
- *Pierre Robin Sequence* (*PRS*)*—micrognathia, glossoptosis, and CP*:
  - Micrognathia prevents tongue descent; retrodisplacement of the tongue → abnormal palatal development with clefting (characteristically U-shaped) or high-arched palate and respiratory distress especially in the supine position; most commonly *associated with Stickler syndrome* (14–34% of cases).
  - *Children with PRS should undergo routine ophthalmologic evaluation in the first year of life* to rule out concurrent eye issues.
- *Stickler syndrome*—autosomal dominant with variable expression; SNHL, characteristically contains Pierre Robin sequence, ocular abnormalities, and arthropathies. Ocular anomalies include early (first decade) myopia that can be severe, retinal abnormalities, and glaucoma. Arthropathies relate to connective tissue disorders—marfanoid features, hyperextensible joints, kyphosis, and scoliosis.

- *22q12 deletion*: most frequent interstitial deletion known to be causally associated with clefting in humans:
  – Associated with wide phenotypic spectrum including DiGeorge syndrome, velocardiofacial syndrome (VCFS).
  – Palatal anomalies; most commonly VPI (29–50% of cases).
  – Also associated with cardiovascular malformations, immune deficiencies, and neurodevelopmental disabilities.
  – Additional features: elongated, flattened midface with malar hypoplasia, broad nasal base, low-set ears, thickened helical rim, micrognathia, microcephaly, dysphagia, pharyngeal hypotonia, middle ear disease, CHL, and chronic suppurative OM.
  – Adenoidectomy and operative pharyngeal procedures should be performed carefully as incidence of VPI is high following surgery; *medial course of the carotid artery* may contribute to increased risk of carotid injury during pharyngeal surgery.
  – *Dx: FISH testing*.
- *Velocardiofacial syndrome* (*Shprintzen* syndrome)—deletion at 22q (same locus as *DiGeorge* syndrome and overlap exists between the two syndromes); affected children have clefts, cardiac anomalies, and characteristic facies (as above):
  – Patients with clefts and the following associated findings should be tested for DGS/VCF deletion: any cardiovascular malformation, short stature, microcephaly, developmental delay, immune deficiency, hx/Fhx of psychiatric disease, and facial dysmorphism.
- *Ectrodactyly-ectodermal dysplasia-clefting syndrome*: lobster claw anomaly of all four extremities, typically have bilateral CL with CP; but unilateral CLP and CP alone reported; ocular findings: related to absence of lacrimal puncta → epiphora, blepharitis, keratoconjunctivitis, corneal ulcers.
- *Popliteal pterygium syndrome*—*IRF-6* gene disturbance; similar to VWS with CL ± P and presence of popliteal pterygium/webbing, syndactyly, genital abnormalities, intraoral adhesions (syngnathia), pyramidal skin on hallux, ankyloblepharon.
- Other disorders associated w/ clefts: Down's (trisomy 21), oculo-auriculo-vertebral, hemifacial microsomia/Goldenhar (constellation of first, second branchial arch abnormalities), Kabuki (CP, arched eyebrows, long palpebral fissures, flat nose), Treacher Collins (AD, colobomas, low-set ears), orofacial-digital syndrome (x-linked, oral clefting, mandibular hypoplasia).

Embryology

The development of the lip and palate occurs sequentially rather than coincidentally and varying degrees of clefting can occur; the degree of clefting is based on the point in fetal development when the fusion process is interrupted.

Lip Development

Beginning at the end of the fourth embryological week, five structures surround the primitive stomodeum and will ultimately give rise to structures of the face, nose, and lips. These include the frontonasal prominence and the paired neural crest-derived facial prominences (maxillary and mandibular prominences), which appear from the first pair of pharyngeal arches.

Frontonasal Prominence (FNP) Mesenchymal proliferation ventral to developing forebrain:

- FNP → nasal dorsum, forehead, contributes partially to the septum (downgrowth from posterosuperior aspect of FNP; other contributions from fused medial nasal processes)

Nasal Placodes Ectodermal thickenings at ventrolateral aspect of FNP; placodes invaginate (at fifth week) to form nasal pits → primitive nasal cavity internally and medial (MNP) and lateral nasal processes (LNP):

- LNP—forms nasal alae and sidewall; nasolacrimal groove between LNP and maxillary process (failure of fusion → oblique cleft [rare]).
- MNP—right and left MNPs fuse to form *intermaxillary segment* (sixth week; failure of fusion → midline cleft, associated with holoprosencephaly); fusion forms the philtrum, medial upper lip, nasal tip, and columella:
  – Intermaxillary segment → primary palate (see below)

Maxillary Prominences (MP) First arch derivative; sixth to seventh weeks; MPs grow medially and ultimately fuse with MNPs (mid-fifth week) to give rise to the upper lip; MPs → lateral upper lip; also have significant contribution to secondary palate (see below):

- NOSE: Formed from five facial prominences FNP = bridge/dorsum; fused MNPs = tip and columella, and LNPs = nasal alae and sidewall
- UPPER LIP: MNPs = medial upper lip, philtrum; MPs = lateral upper lip (from philtral column laterally); NO contribution of LNPs to formation of the upper lip; development usually complete by the end of the seventh week
- *Lack of fusion between maxillary process and medial nasal process → CL.*

Palate Development

Weeks 5–12 (palatal fusion completed later in females) MPs grow and push the MNPs medially → MNPs fuse not only at surface giving rise to the lip but also at deeper levels to form the premaxilla, alveolus, and primary palate; once primary palate fully developed, the secondary palate begins to develop; during the sixth week of embryonic development, palatine shelves are directed obliquely downward on either side of the tongue; seventh week → palatine shelves migrate inferomedially to lie horizontally above the tongue; palatal fusion occurs from anterior to posterior and simultaneously fuses with nasal septum which descends from above (derived from FNP); at the eighth week, the tongue begins to withdraw/descend from its position between lateral maxillary prominences (which form secondary palate).

Primary Palate Derived from intermaxillary segment after fusion of MNPs = central lip (philtrum), columella, nasal tip, premaxilla, central maxillary alveolar arch, which accommodates four anterior teeth (medial and lateral incisors) and hard palate anterior to incisive foramen.

- *Lack of fusion between maxillary process and medial nasal processes/intermaxillary segment → primary CP*

Secondary Palate Part of the hard palate posterior to incisive foramen as well as lateral hard and soft palate; formed by fusion of palatine shelves (from maxillary prominences); makes up the majority of palate including the hard palate posterior to incisive foramen and all of the soft palate; development begins after completion of primary palate.

- *Lack of fusion in the midline between two palatine shelves* (*from lateral maxillary processes*) *→ secondary CP*

Fusion of alveolus *between canine (cuspid) and lateral incisor*, i.e., *alveolar defect* between these teeth and clefts in this location can affect development and eruption of adult dentition.

Types of Clefts/Classification

Cleft Lip

- *Complete unilateral CL*—the entire vertical thickness of the upper lip and often associated with alveolar cleft because the lip and primary palate share the same embryologic origin; implies separation of medial and lateral lip with absence of all layers—skin, muscle, mucosa, and alveolar bone with extension to the nasal sill/floor.
- *Incomplete unilateral CL*—can contain muscle fibers within the cleft, residual fibers

known as *Simonart's band* (bridge or bar of lip tissue of variable size that usually consists of skin only, although some histological studies have shown that some muscle fibers lie within band). The band, which is more frequently observed in UCL than BCL, is located at the base of the nostril and associated with a higher frequency of maxillary lateral incisor development in the maxillary process.

- *Bilateral CL*—prolabium of complete bilateral clefts is devoid of muscular fibers and is attached at columella only (tenuous blood supply), although may have muscle fibers in incomplete clefts; the premaxilla can be horizontally oriented and may require molding/ manipulation of cleft components prior to definitive repair.
- *Microform CL*—diastasis of orbicularis fibers with presence of alveolus, skin, and mucosa.

Cleft Palate

Unilateral or bilateral and extent may be classified as complete or incomplete. CP is classified according to location relative to the incisive foramen:

- *Clefts of primary palate* occur *anterior* to incisive foramen.
- *Clefts of secondary palate* occur *posterior* to incisive foramen:
  - Unilateral cleft of secondary palate is defined as one in which the palatal process of the maxilla on one side is fused with the nasal septum.
  - Bilateral cleft of the secondary palate has no point of fusion between the maxilla and the nasal septum.
  - Cleft palate in isolation (CPO) usually involves the secondary palate only and has varying degrees of severity.
- *Complete CP*—palate involves both the primary and secondary palate and includes one or both sides of the premaxilla/alveolar arch and frequently involves cleft lip.
- *Incomplete CP*—clefting of *secondary palate only*.
- *Submucous cleft palate* (SMCP)—least severe incomplete cleft; bifid uvula, thinning of central soft palate with translucent appearance (zona pellucida), and palpable notch in posterior aspect of the hard palate; patients at high risk for VPI as levator veli palatini has similar configuration to what is seen in true cleft palate with central diastasis and abnormal longitudinal orientation of muscle fibers → Furlow palatoplasty may be effective Tx (see below).
- *Veau classification of cleft palate*:
  - I—Soft palate only
  - II—Hard and soft palate
  - *III*—Unilateral complete cleft
  - *IV*—Bilateral complete cleft

Muscular and Vascular Anatomy

Lip Divided into red (convex) and white lip (concave) separated by mucocutaneous junction/ vermilion border:

- *White roll*—epithelium just above vermilion border, reflects ambient light and is critical landmark in cleft lip reconstruction.
- *Vermilion*—dry portion of lip's red mucous membrane, lacks pilosebaceous units, salivary glands, eccrine glands.
- *Superior labial artery*—major blood supply to the upper lip; normally anastomoses in the midline; courses on undersurface of the orbicularis muscle:
  - In unilateral cleft—aberrant vascular supply on lateral aspect of cleft is better developed than on medial side; artery courses along margin of cleft, anastomosing with either angular or lateral nasal artery at base of the nose.
  - Incomplete cleft—thin, terminal branch of superior labial artery crosses bridge.
  - Bilateral clefts—artery is underdeveloped in prolabial segment which primarily derives its blood supply from the septal, columellar, and premaxillary vessels.
- *Orbicularis oris*—principal muscle of the lip; not a true sphincter as superficial and deep parts of muscle arise separately from the

modiolus; critical role in lip function and appearance; reestablishment of muscular sphincter is critical in achieving appropriate surgical outcome; superficial layer arises from the dermis and passes obliquely to insert into the mucous membrane lining the inner surface of the lips; deep fibers arise from the maxilla and mandible:

- Orbicularis in CL: Muscle is hypoplastic and has abnormal attachments/insertion in CL—no muscle crosses the cleft in complete clefts; the skin bridge (Simonart's band) in incomplete clefts also contains no functional muscle (muscle fibers do not cross gap unless skin bridge at least 1/3 total lip height); muscle is more hypoplastic on medial side of cleft; abnormal insertion at alar base and anterior nasal spine; fibers parallel cleft margin.

Palate

Muscular Anatomy of the Palate and Pharynx

- *Tensor veli palatini* (TVP): *V3*; tenses palate and opens ET during swallowing; from base of internal pterygoid plate and lateral aspect of torus tubarius; terminates in tendon/aponeurosis after winding around hamulus.
- *Levator veli palatini* (*LVP*): *pharyngeal plexus* (derived from IX, X, XI) origin: petrous bone and medial torus, fibers occupy central 40–50% of the soft palate and fuse with contralateral LVP; palatal sling causes superior and posterior motion of the soft palate; causes elevation of the soft palate during deglutition.
- *Musculus uvulae*: pharyngeal plexus; adds bulk to dorsal surface of the soft palate.
- *Palatoglossus*: pharyngeal plexus; forms anterior pillar; lowers and positions velum, elevates the tongue.
- *Palatopharyngeus*: pharyngeal plexus; forms posterior tonsillar pillar; constricts pharyngeal isthmus *narrows VP orifice*, superior heads (2) clasp LVP as it enters the velum; fibers intermingle with SPC at posterolateral pharyngeal wall, action: retrodisplacement and downward motion of the velum; antagonist to LVP and position of the velum during normal function is the net result of action of LVP and palatopharyngeus; stretches posterior free margin of the velum; acts with SPC for medial movement of lateral pharyngeal wall below the level of the hard palate.
- *Superior pharyngeal constrictor* (*SPC*): pharyngeal plexus; *medial movement of lateral pharyngeal walls*; relative activity of SPC may contribute to different patterns of VP closure (coronal, minimal SPC fxn; sagittal, high degree of SPC activity during closure; circular, moderate); *main component of Passavant's ridge*.

In CP, VP muscles can be hypoplastic and generally have abnormal course and attachment:

- TVP—normally attached to posterior hard palate; in CP, aponeurosis is incomplete with abnormal attachment at posterior hard palate, results in ETD.
- LVP—in CP and SMCP has abnormal longitudinal orientation with *abnormal insertion to posterior aspect of hard palate, SPC, and TVP aponeurosis*; abnormal LVP appears to be primarily responsible for VP dysfunction.

Arterial Supply of Palate

- Hard palate—Greater palatine artery with some anterior contributions from arterial branches in incisive canal (anterior palatine artery)
- Soft palate—Ascending palatine (facial artery), greater palatine (internal maxillary), ascending pharyngeal branch, and contribution of the lesser palatine (branch of the greater palatine)

Treatment

Table 13.2 provides a general outline for timing of intervention in patients with cleft lip and palate.

Table 13.2 Timing of intervention in patients with CL ± P and CPO

Timing of intervention in patients with cleft deformities	
Presurgical orthopedics/ intervention	1–2 weeks
Speech eval—feeding	
Cleft lip repair	3 months
Primary rhinoplasty	
Tympanostomy tube placement	
Palatoplasty/palate repair	9–18 months
T-tube/long-term PE tube placement	
Speech evaluation	3–4 years old (as early as 2 years)
Velopharyngeal work-up/ surgery	4–6 years old
Repair of alveolus	6–8 years old
Intermediate rhinoplasty	7–10 years old
Definitive septorhinoplasty	16–19 years old
Orthognathic surgery	

Table adapted from Park Facial Plastic Surgery: Essential Guide

Rule of tens 10 weeks old, hemoglobin 10 g/dL, weight 10 lb; partially based on anesthetic safety; incomplete clefts have less urgency to repair as Simonart's band tends to hold alveolar segments in place as growth occurs.

Oral intake can be compromised as a result of inability to feed (sucking mechanism impaired—child is unable to form a seal due to incomplete muscular sphincter and/or escape of air through cleft palate); goal should be weight gain of 0.5–1 ounce/day; *Mead-Johnson, Haberman, or Pigeon* nipple can be used to facilitate feeding; feeds should be done in upright position, as cleft patients tend to swallow more air and require frequent burping.

Presurgical Infant Orthopedics (PSIO)

Preoperative manipulation of alveolar segments in complete CL ± P is performed particularly in wide clefts to facilitate closure; most important in bilateral complete cleft lip in which control of premaxillary segment is critical in achieving satisfactory surgical outcome; ideally, PSIO should be started in the first 1–2 weeks of life.

Techniques

- *Taping*—requires family compliance to properly apply tape across cleft: doesn't address nasal deformity.
- *Head bonnet*—applies pressure across the premaxilla; removable and easier than taping.
- *Latham* appliance—mold with pins used to create more favorable alveolar position.
- *NAM* (*nasoalveolar molding*)—molding and repositioning of alveolar processes, nasal cartilages, and lengthening of deficient columella to create lasting aesthetic outcome and reduce need or minimize the extent of secondary surgical revision procedures; significantly improves nasal symmetry over surgery alone; once alveolar segments in close proximity, nasal stent is added to mold distorted nasal cartilages; the alar cartilage is lifted by intranasal stent to achieve normal elevation and symmetry:
  - *NAM in bilateral cleft patients* used to lengthen columella, reposition nasal cartilages toward the tip, and align the alveolar segments; first stage in bilateral molding consists of retracting and straightening the everted premaxilla into space between two lateral alveolar segments; second stage involves nasal stent incorporated into anterior rim of molding plate.

Gingivoperiosteoplasty—closure of soft-tissue alveolar segments; possible if there is close approximation of the alveolar cleft segments; use of NAM and presurgical approximation of alveolar defect provides the surgeon with the option to perform GPP at the time of lip closure.

Lip adhesion procedure in which cleft segments are united via myomucosal flaps, essentially creating an incomplete cleft; contributes to molding of the alveolar segment and reducing cleft width; secondary procedure then converts the lip adhesion to a formal lip repair; approximation of the orbicularis oris creates muscle pull on the maxillary alveolar segments, which is critical for molding; alternative for those unable to tolerate NAM or other orthopedic devices.

Cleft Lip Repair

Goals: creation of symmetrical nasal tip and alar bases, Cupid's bow, and lip fullness without loss of normal philtral contour with fullness of labial mucosa that is equivalent on both sides.

Techniques

Goals: restoration/reconstruction of normal lip anatomy and function by reestablishing continuity of the orbicularis; secondary goals—closure of the nasal floor and correction of nasal tip asymmetry.

Straight-line repair: poor orbicularis closure may result in lip shortening, tissue waste, and unappealing scars with potential for scar contracture; largely a historical procedure.

Geometric closures, modified Z-plasties, quadrangular flaps, triangular flaps—designed to decrease the amount of lip shortening that may occur and to improve orbicularis oris muscle function.

Millard Rotation-Advancement Flap most commonly used technique in the USA:

- Incorporates two opposing flaps with the medial lip rotated downward and lateral lip advanced medially to restore the lip integrity:
  - Must be designed to correct vertical height discrepancy from Cupid's bow to the columella.
  - Difference accounts for length and design of rotation flap and back-cut.
- Technique (Fig. 13.1): Methylene blue used for skin marking as outlined below; rotation flap incision is made first (point 3 → 5 with back-cut to x as needed) and is taken through the entire thickness of the orbicularis and oral mucosa to allow for complete release; c-flap remains and may be used to add additional height to columella; the advancement flap starts with incision from point 8 → 9 and this incision is then continued along the alar base; the gingivolabial sulcus is incised and incisions are taken down to face of the maxilla; bilateral undermining assists in closure; first deep stitch reapproximates the orbicularis at back-cut and second stitch at vermilion; mucosa, muscle, and skin closure follows.
- *Advantages*: flexible—can be applied to a wide variety of cleft lips; allows continuous modifications during the design, incisions, and execution of the repair; minimal discarding of tissue; good nasal access; camouflage of suture line along philtral column.
- *Disadvantages*: requires experienced surgeon; possible excessive tension—may cause constriction of maxillary growth; extensive undermining required; vertical scar contracture with possibility of vermilion notching if improperly designed; tendency toward small nostril.

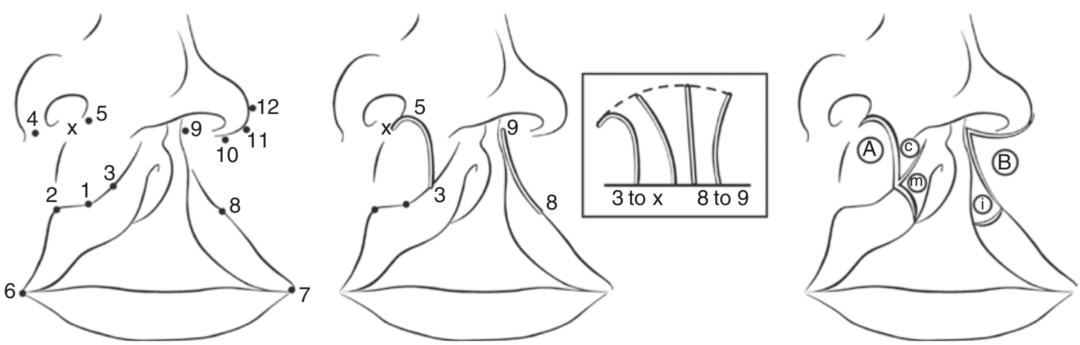


Fig. 13.1 *Left*: Anatomical landmarks used in the Millard rotation-advancement flap technique for repair of unilateral cleft lip deformity. *Middle*: The length of the rotation flap (non-cleft side) and advancement flap (cleft side) is equivalent in length (see *inset*). *Right*: Following incision, the flaps are advanced and rotated (A and B), while C contributes to repair of the nasal sill. (After Sykes and Tollefsen Management of Cleft Lip Deformity, depicting Millard Rotation-Advancement Flap)

Tennison-Randall Repair second most commonly used technique:

- Modified Z-plasty with a lateral, inferiorly based triangular flap
- *Advantages*—rebuilds the floor of the nostril; adds length to the medial lip element; preserves Cupid's bow; adds needed tissue volume in the lower one third of the lip
- *Disadvantages*—violation of normal philtral column on the non-cleft side, creates more prominent scarring across philtrum; requires exact presurgical measurement and lacks flexibility in surgical application

Bilateral Cleft Repair (Fig. 13.2)

The premaxilla can be quite protrusive and may be "locked out" from palatine segments requiring pre-op manipulation to achieve an appropriate repair via PSIO or lip adhesion → gives three segments best configuration to achieve subsequent repair; reestablishing continuity of the orbicularis is critical for optimal functional and aesthetic result; single-stage repair of bilateral deformity usually provides best chance of symmetrical outcome; staged repair of the bilateral cleft tends to create an asymmetrical result due to disparities of the two sides in terms of facial growth following

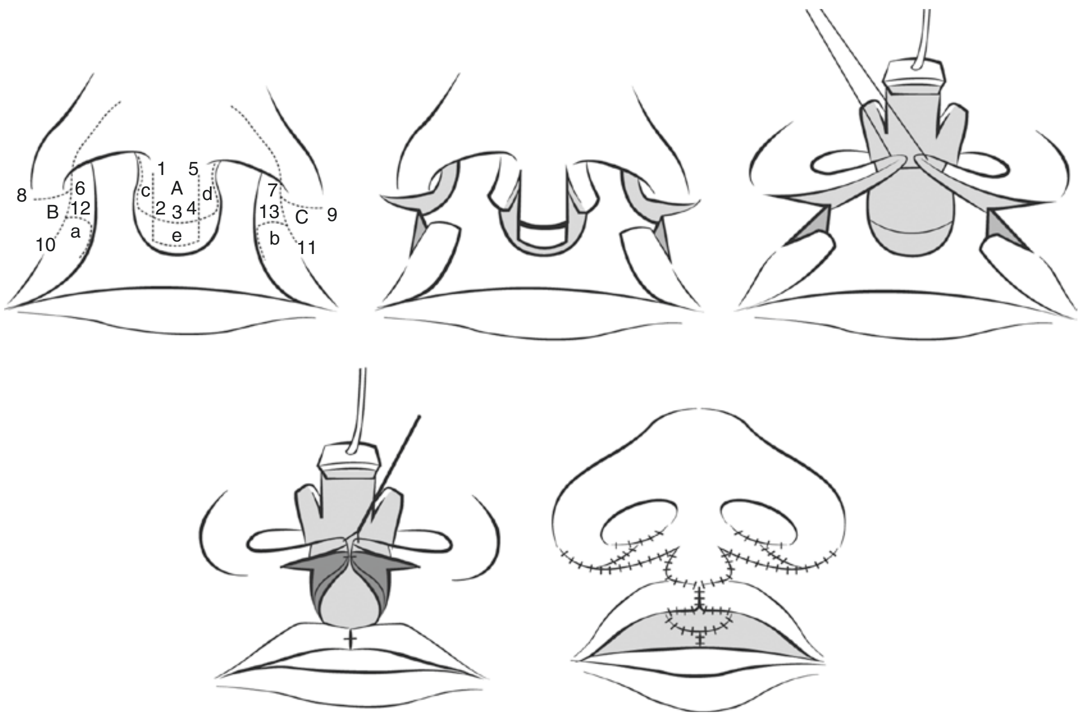


Fig. 13.2 *Top left*: Anatomical landmarks and incisions used for repair of the bilateral cleft lip deformity. *Top middle*: Incisions made in prolabial and lateral flaps in anticipation of flap advancement. *Top right*: De-epithelialized tips of lateral flaps advanced and sutured in place medially to the premaxillary periosteum. *Bottom left*: Tips of lateral flaps sutured medially and reconstruction of orbicularis muscle sling. *Bottom right*: Prolabium and fork flaps sutured in place. (After Seibert Surgical Repair of the Bilateral Cleft Lip Deformity)

"revascularization" of repaired lip; asymmetry is oftentimes difficult to correct at a later stage, and staged repair precludes muscle from being advanced across the prolabial segment.

Cleft Nasal Deformity

Unilateral Cleft Nasal Deformity (Fig. 13.3)

- Abnormal insertion of the orbicularis muscle at anterior nasal spine and alar base on cleft side.
- Cleft alar base displaced *inferiorly, laterally, and posteriorly* due to maxillary insufficiency and action of the orbicularis.
- Septum: caudal and inferior septum deviated to non-cleft side (away from the cleft); upper and posterior portions of the septum deviate to the cleft side.
- Columella is shortened on the cleft side and the nasal tip is deflected to the cleft side.
- LLC has normal size with a poorly projecting dome, alar flattening, and a horizontal nasal shape secondary to a short medial crus and long lateral crus.
- Deficiency and displacement of maxillary segment with lack of piriform aperture.
- Alar-facial groove is often absent and the ala attaches to the face at an obtuse angle. Absence of the nasal floor and sill commonly seen with relative stenosis of the internal nostril on the cleft side.
- Internal nasal valve collapse may occur due to the relative flaccidity of the ULC on the cleft side.
- External valve collapse also possible due to introversion of cleft ala.
- *Bilateral Cleft Nasal Deformity*—similar defects to those seen in unilateral CL nasal deformity, but present on both sides to varying degrees (if asymmetry of CL exists):
- Poorly projected tip with wide alar base.
- Alar bases positioned similarly to unilateral CL due to pull of abnormally inserting orbicularis oris and bilateral absence of maxillary segment → posterior, lateral, inferior displacement.
- Extremely short columella and the medial crura are displaced into the prolabium.
- Prolabium and premaxilla can be markedly displaced anteriorly with poor overall blood supply.
- Nasal tip is broad and flattened and the nasal domes are separated resulting in a bifid appearance.
- Depending on presence/absence of CP, the septum may not articulate with palatine shelves in bilateral complete CP; unilateral complete CP → the septum attaches to non-cleft side.

Fig. 13.3 Anatomical findings associated with unilateral cleft lip nasal deformity. (After Dutton, Bumsted Management of the cleft Lip Nasal deformity)

Treatment of Nasal Deformity

Correction begins as early as 1 week after birth with NAM (see above) → *positions CL and LLC for primary repair*; primary repair done at the time of lip repair; intermediate repair usually occurs at ages 7–11 years after alveolar bone grafting; definitive septorhinoplasty follows completion of growth after adolescence; staged nasal repair allows smaller corrections with each procedure:

- Goals: reconstruction of the nasal floor and sill, repositioning of alar base, columellar lengthening on cleft side, correction of deformed ala, provision of adequate nasal tip support and symmetry, straightening of nasal dorsum, attainment of adequate nasal airway

Primary Rhinoplasty

- Does not affect nasal or midfacial growth
- Performed at the time of Millard rotation-advancement procedure
- Cleft LLC separated from overlying skin-soft-tissue envelope via alar base (laterally, the alar base is separated from its attachment to displaced maxilla and abnormal orbicularis insertion) and columellar incisions with intercrural dissection
- Alar base sutured in more medial position after reconstruction of the nasal floor (mucoperiosteal flaps from lateral nasal wall and mucoperichondrial flap from the septum)
- LLC sutured in a more anatomic position and secured via internal and external bolster

Intermediate Rhinoplasty

- Most secondary deformities can be repaired at age 7–8 years when lower nasal complex can be addressed.
- Alveolar bone grafting usually precedes intermediate rhinoplasty as eruption of lateral incisor and canine, following grafting, provides adequate base for nasal repair.
- Nasolabial fistula also corrected prior to intermediate rhinoplasty to release abnormal pull on alar base.
- Judicious septal surgery at the time of intermediate rhinoplasty does not appear to interfere with subsequent growth of the face or nose.
- Repositioning of the caudal septum in the midline and reconstruction of the nasal tip and alar cartilages are appropriate at this stage.
- Performed via open technique for adequate visualization and accurate repair.
- Technical points (although these may be delayed depending on severity of deformity at the time of intermediate rhinoplasty)—dissecting pocket between the medial crura; freeing the caudal septum from abnormal attachment with suture fixation to anterior nasal spine; repositioning abnormal dome in more medial and superior location (lateral crural steal); V-Y advancement from the upper lip to lengthen cleft-side columella.

Definitive Septorhinoplasty

- Definitive correction includes septoplasty, osteotomies, and dorsal revision performed after completion of nasal growth.
- Complete and aggressive restructuring of internal and external anatomy can be performed.
- Done via open approach with or without alotomy.
- Graft materials: usually from the septum, via separate hemitransfixion approach with correction of bony and cartilaginous deviation.
- Columellar strut with fixation of LLCs enhances nasal projection with more advancement of cleft-side LLC.
- Repositioning of LLC may be necessary with suturing to ULC.
- Tip graft can improve tip definition.
- Unilateral spreader on cleft side can be used to address INV collapse.

Cleft Palate Repair Techniques

Goals of CP repair are to restore the integrity of the levator sling and close and lengthen the palate. All techniques rely upon adequate flap mobilization, atraumatic tissue techniques, and multilayer closure of oral and nasal mucosa and musculature.

Most will proceed with repair once the child is 10 kg around 10–12 months of age. Development of normal speech is the primary concern and dictates timing of repair. Improved speech outcomes are seen with early closure of a palatal defect (repair before 18 months):

- *Von Langenbeck*—bipedicled flaps with anterior and posterior blood supply; incisions placed along cleft margin and adjacent to alveolus; useful in narrow clefts and incomplete clefts; Medial fracture of the hamulus is

sometimes needed to assist with closure. Disadvantages: no palate lengthening; leaving anterior pedicle → decreased flap mobility; vascular pedicle (~1 cm medial to upper second molar) not visualized (Fig. 13.4).
- *Two-Flap Palatoplasty* (*Bardach*)—commonly used for repair of complete unilateral and bilateral CP, incisions extend along cleft margin, posterior to alveolus, and around last molar tooth; great care taken not to damage tooth buds during flap elevation; posteriorly based pedicle (greater palatine artery); combined with vomer flaps (advanced laterally and sutured to lateral undermined nasal mucosa for nasal closure); does not lengthen soft palate; dissection in space of Ernst (posterior to vascular pedicle) or fracturing of the hamulus allows additional mobilization of flaps (Fig. 13.5).
- *Furlow* (*Double-Opposing Z-Plasty*)—used for closure of the soft palate and in SMCP, lengthens and thickens the soft palate while reorienting LVP; also useful in surgery for VPD (see below); one flap is muscle/mucosa, while the other is mucosa alone; can be used for soft palate closure in conjunction with two-flap hard palate defect although the soft palate should be addressed before undermining flaps (Fig. 13.6).
- *V-Y Pushback/Three-Flap*—used for clefts of secondary palate/complete clefts; incisions similar to bipedicled technique, three-layer closure of nasal mucosa, muscle, with V-Y palatal lengthening of palate; subperiosteal release of flap and release of muscle allow repositioning of musculature in transverse orientation; lengthens palate; preserves mucosa over primary palate with bilateral posteriorly based unipedicled flaps (Fig. 13.7).

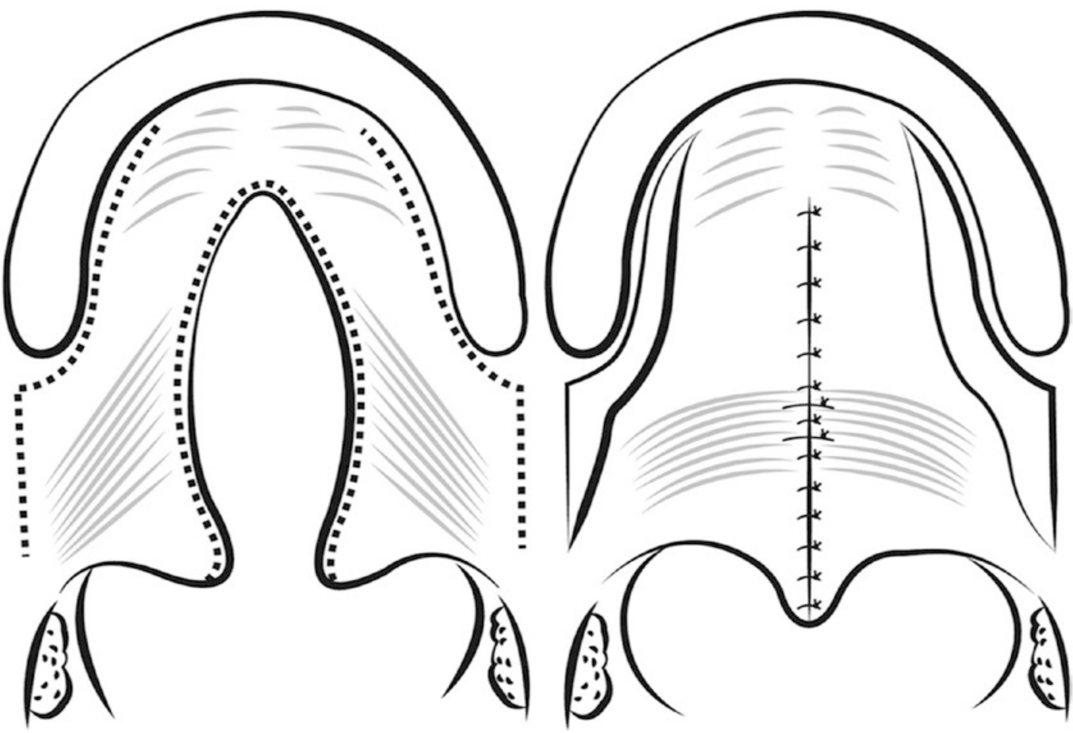


Fig. 13.4 Von Langenbeck palatoplasty. *Left image*: Incision design for right and left bipedicled flaps. *Right image*: Right and left flaps advanced for closure of cleft palate with residual exposed underlying hard palate laterally. Underlying levator sling is reconstituted with appropriate orientation of muscle fibers. (After Strong Management of the Cleft Palate Facial Plastic Surgery Clinics Feb 2001)

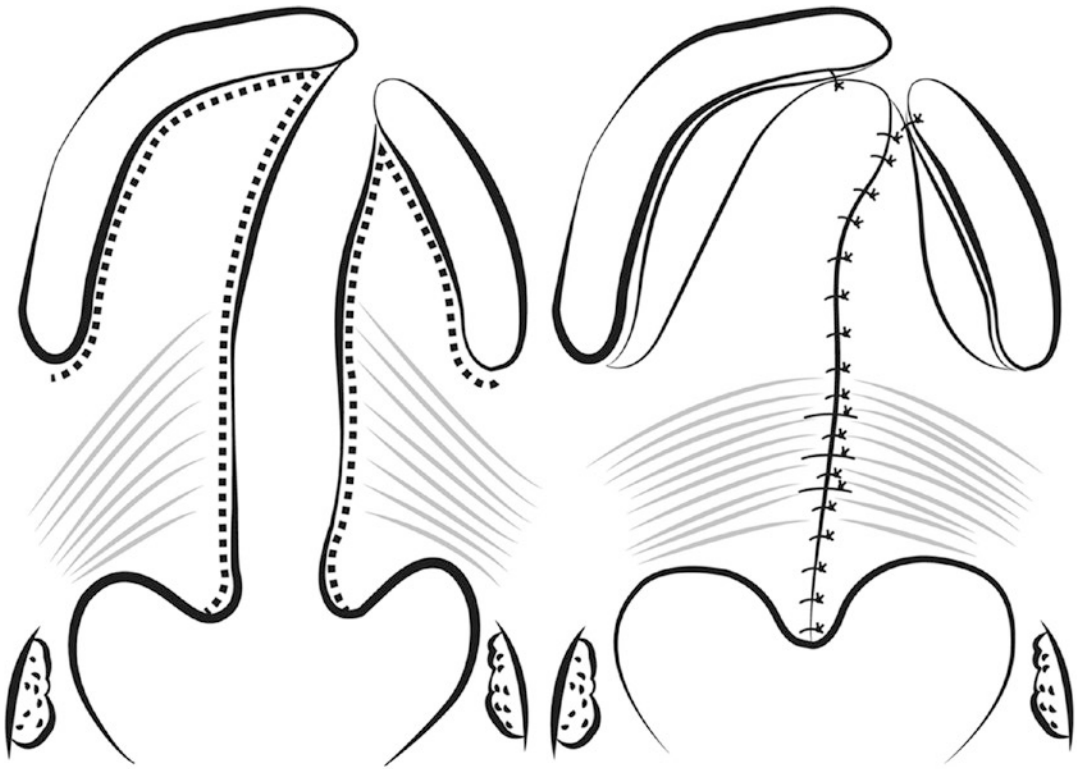


Fig. 13.5 Two-flap palatoplasty. *Left image*: Incision design for posteriorly based pedicle (off greater palatine vessels). *Right image*: Following elevation, mobilization and medialization of flaps for closure of cleft palate. Levator sling reconstituted in appropriate orientation. (After Strong Management of the Cleft Palate Facial Plastic Surgery Clinics Feb 2001)

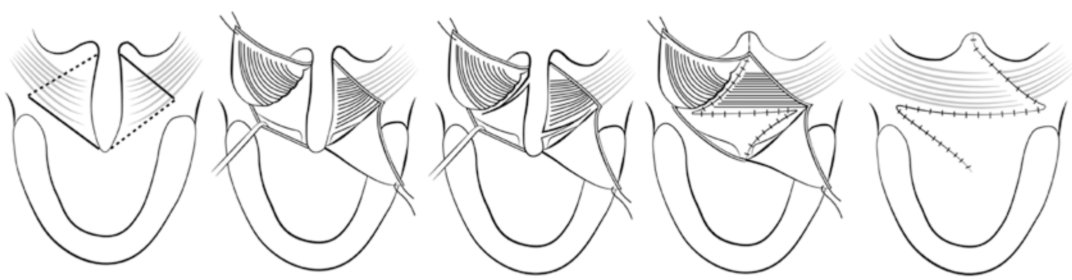


Fig. 13.6 Furlow (double-opposing Z-plasty) palatoplasty. *Leftmost image*: Incision design of opposing Z-plasties with cleft as central limb. Second image from *left*: Elevation of flaps—anteriorly based flaps are mucosa-only, while posteriorly based flaps are mucosa and palatal musculature. *Center image*: Incisions made through nasal mucosa. Second image from *Right*: Nasal flaps transposed. *Rightmost image*: Transposition and closure of oral flaps. (After Furlow Cleft Palate Repair by Double Opposing Z-plasty)

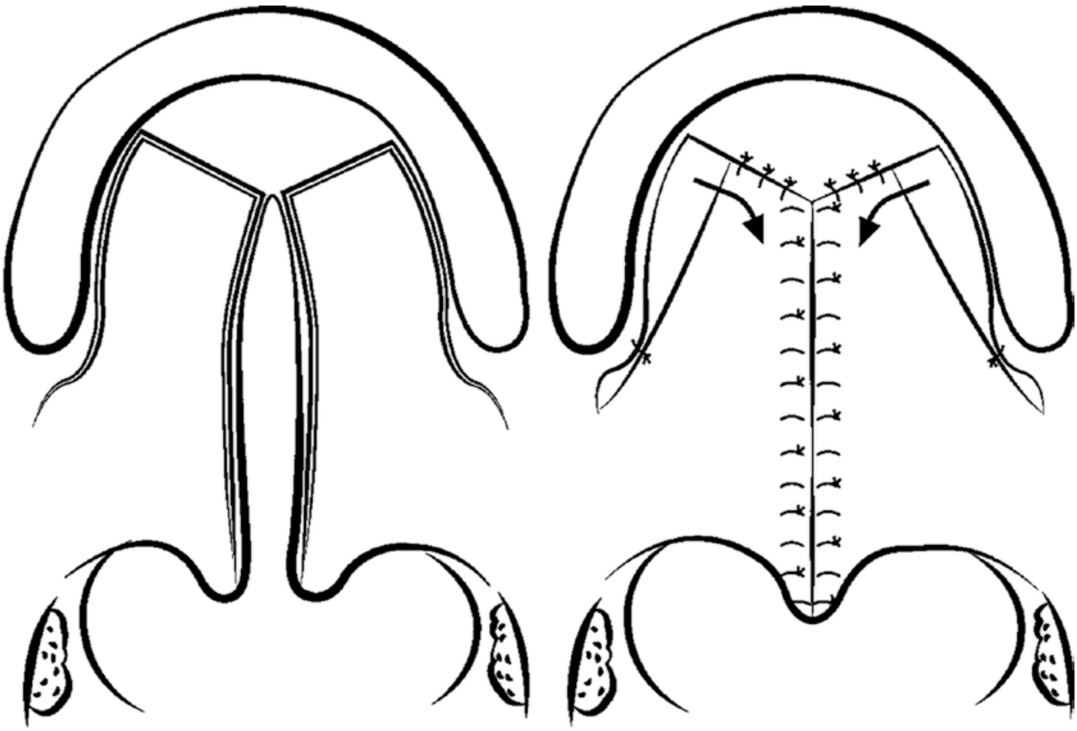


Fig. 13.7 V-Y (three-flap) palatoplasty. *Left image*: Incision design with right and left posteriorly pedicled flaps. *Right image*: Elevation, mobilization, medialization, and posterior advancement of flaps result in closure of defect and posterior lengthening of palate. (After Strong Management of the Cleft Palate Facial Plastic Surgery Clinics Feb 2001)

Complications of Palate Repair

- Velopharyngeal Dysfunction—most common complication following palatoplasty; lower rates seen with double-opposing Z-plasty than with other methods; abnormal orientation and insertion of LVP must be corrected to achieve appropriate functioning of the soft palate following CP repair.
- Fistulization can occur at varying rates depending on type and severity of cleft; sites of fistulization typically are the anterior hard palate and the junction of the hard and soft palate (*most common*).
- Airway problems following palatoplasty can occur as a result of tongue and FOM swelling (commonly due to relative hypoperfusion due to Dingman retractor or other retractor-exerted pressures → released every 30–45 min to decrease risk of post-op swelling); most likely to occur in children with Pierre Robin sequence.
- Bleeding—minimized with infiltration of epinephrine solution and appropriate tissue handling; exposed areas should be covered after repair with absorbable hemostatic material (e.g., Avitene, Surgicel) to promote healing and hemostasis.

Velopharyngeal Dysfunction

Velopharyngeal Inadequacy—inability to fully close VP valve during speech or swallowing:

- VP *Insufficiency*—structural, seen with cleft palate
- VP *Incompetence*—neurologic dysfunction
- Causes: previously repaired (inadequate lengthening of the velum at the time of primary palatoplasty and abnormal function of the levator musculature and cicatricial contracture of the velum) or unrepaired CP is the

most common cause of VPI; SMCP, neurogenic VPI, iatrogenic VPI following maxillary resection, UPPP, or adenoidectomy.
- Dx: speech evaluation/assessment of nasal/non-nasal phonemes; mirror exam—fogging during speech when held under nares; nasal endoscopy during phonation provides visualization of the velum, assessment of lateral pharyngeal walls and posterior pharynx during attempted VP closure; video fluoroscopy.
- Configuration of VP closure: can impact choice of surgical intervention; coronal (most common) > sagittal > circular:
  – Coronal—closure mainly due to action of the velum in anterior-to-posterior direction.
  – Sagittal—closure due mainly to medial motion of lateral pharyngeal walls.
  – Circular (± contribution by Passavant's ridge)—closure relies on combination of both A-P and medial/lateral motion for complete closure.

Treatment of VPI

Treatment decision made jointly by surgeon, SLP, and dentist/prosthodontist.

Speech Therapy

- Initial treatment indicated for children with mild VPD → aggressive speech Tx for 6 months, failure to improve with 6–12 months of speech therapy may indicate need for surgical intervention; with improvement → long-term aggressive speech therapy focused mainly on articulation.
- Not successful in remediating structural defects and surgical tx should not be delayed in these cases, e.g., children with VPD and nasal regurgitation secondary to obvious palatal abnormality such as a partial soft palate cleft or SMCP.
- Children with good articulation may be candidates for surgery if no VP closure demonstrated on nasal endoscopy with appropriately articulated phonemes.

Surgical Therapy

Surgery preferred for long-term management in children. Patients with structural causes of VPD or persistent VPD after speech therapy should be considered for surgical intervention:

- *Intravelar veloplasty*—reorientation of inappropriately oriented LVP sling with three-layer closure and reapproximation of (attenuated) muscle fibers in the midline.
- *Furlow* (*double-opposing Z-plasty*) *palatoplasty*—increasingly popular, reorients LVP, lengthens and thickens palate, and provides bulk in posterior VP; if VPD persists, sphincter pharyngoplasty can be performed; also used for primary closure of CP, repair of SMCP, or as a secondary procedure if the LVP longitudinally oriented; *sagittal* closure with VP gap of greater than 9 mm.
- *Pharyngeal flap*: permanent, central, passive obturator; brings posterior wall tissue to center of VP; relies on adequate function of lateral pharyngeal port for full velopharyngeal closure; superiorly based flap more common. Indicated for poor A-P velar motion with good lateral pharyngeal wall motion:
  – Most common complications: bleeding, airway obstruction (first 24 h following surgery); late complication: OSA, cor pulmonale, flap breakdown, nasal obstruction, and aspiration (associated with post-op hemorrhage). Consider pre-op sleep study in patients at risk for OSA.
  – VCFS—*pre-op angiography* should be obtained to evaluate course of carotid arteries as these patients can have a medial retropharyngeal course leading to significant intraoperative hemorrhage if unrecognized.
- *Sphincter pharyngoplasty* (dynamic sphincter pharyngoplasty, orticochea): narrows VP valve, decreased risk of postoperative airway obstruction; bilateral myomucosal flaps elevated from lateral pharyngeal wall/posterior tonsillar pillar wall and sutured to posterior NP wall to obturate the posterior and lateral portions of the VP; presence of adenoids may limit superior extent of flap placement → low

adenoidectomy ~6 weeks prior to sphincter pharyngoplasty; inset of flaps should be as high as possible (at or above the level of anticipated closure) to maximize function and minimize residual VPD and passive obstruction while in supine position (snoring common after sphincter pharyngoplasty); appropriate for correction of *coronal* and *circular* patterns of closure as long as the soft palate has adequate length.
- *Posterior wall augmentation*: filler or graft provides bulk near Passavant's ridge to aid in VP closure; suitable technique in child with small posterior midline gap.

Prosthetic Therapy

- Obturators—can be used if patient is poor candidate for surgical repair:
  - Palatal lift prosthesis pushes palate superiorly and posteriorly to contact pharyngeal wall.
  - Speech bulb prosthesis—with movement, walls of VP contact obturator.

Treatment of Alveolus/Bone Grafting

Alveolar bone grafting should occur during the period of mixed dentition; performed to restore normal architecture of maxillary arch and closure of oronasal fistula, if present (done prior to eruption of adult canine); secondary bone grafting allows eruption of permanent teeth (specifically lateral incisor and the canine) and provides base for subsequent correction of nasal deformity, orthognathic and orthodontic tx (including endosteal dental implantation).

Fresh, autogenous, cancellous bone is an ideal source because it supplies living, immunocompatible osteocytes that integrate fully with maxillary bone and are indispensable for osteogenesis. Grafting of alveolar bone defect has become standard of care at most cleft centers. Presence of bone in cleft creates odontotrophic milieu facilitating eruption and movement of teeth into cleft → dental aesthetics are enhanced by improvement of dental and gingival alignment.

Timing of Alveolar Repair

- Dental age/stage of dentofacial development more important than chronological age.
- Done as early as possible without causing deleterious effects on growth of the maxilla or damage to tooth buds.
- Root of permanent canine provides a guide to timing of therapy: should be formed 1/3–1/2 of definitive length at the time of graft placement (usually occurs sometime btw age 7 and 11 years old).
- Primary grafting: usually done before or during palatoplasty; has potential to inhibit midface growth, less common.
- Secondary grafting: performed after CP repair; *early* (if done <2 years old, may cause growth disturbances), *intermediate* (favored, during mixed dentition), or *late*:
  - Late grafting performed after exposure of cementum can lead to root resorption and ankylosis of teeth, predisposing to tooth loss and complicating later orthodontic management; graft resorption is also increased in grafts placed after eruption of teeth adjacent to the cleft.

Source of Grafting Material

- Allogeneic bone (treated homologous bone).
- Alloplastic materials—calcium phosphate based, eliminates donor-site morbidity.
- Autologous bone grafts considered gold standard because it provides osteogenic cells and osteoinductive factors; rapid incorporation, better dental support, resistance to infection; cancellous bone is preferred over cortical bone as it is more resistant to infection and undergoes less resorption.
- Iliac crest: major source of grafting material but postoperative gait disturbances can be seen.

- Alternative sources: cranium/calvarium, tibia, mandibular symphysis, rib (used for primary grafting in Rosenstein protocol).
- Bone morphogenetic proteins (BMPs)—recombinant BMP-2 in a soluble collagen matrix applied to alveolar cleft defect:
  - May be useful in skeletally mature patients with unilateral alveolar clefting; improved bone growth as compared to control group in this population
  - When performed in mixed dentition results are similar to autologous grafts

Correction of Secondary Defects

Secondary defects following correction of unilateral or bilateral cleft lip and palate are generally the rule rather than the exception. Age at the time of correction is key in decision-making process and largely based on severity of deformity; psychosocial effects must be considered; secondary procedures to correct speech or swallowing/feeding difficulty should be performed regardless of age; most commonly—preschool age (4–5 years old) or early adolescence; cessation of facial growth is another critical factor in timing of repair.

Lip—assessment in repose and during dynamic function; secondary defects classified as major/minor based on anticipated repair, i.e., if primary repair needs to be taken down to correct secondary defect; *secondary deformity of vermilion and Cupid's bow are most common* following primary CL repair.

Deformities of Vermilion—misalignment or peaked appearance of vermilion may be due to short vertical dimension of the lip; mild deformities common in early period following rotation advancement; massage may be useful early on; if persistent for >1 year → surgical tx:

- *1–2 mm vermilion misalignment → diamond-shaped excision* of scar and closure which increases length of scar and brings vermilion border down.
- >3 *mm*—problem is likely related to *inadequate rotation at the time of initial repair*: in these cases the repair should be taken down and rotation advancement should again be performed.
- Misalignment of vermilion-white roll is noticeable even if less than 1 mm: in these cases, a small Z-plasty can be designed to correctly align these structures.
- Deficient vermilion may be due to overaggressive mucosal resection at the time of initial repair or due to inadequate muscle alignment at the time of primary repair.
- *Whistle deformity*—mild deformities corrected with V-Y advancement (V apex toward sulcus carried through vermilion and orbicularis, incision carried to mucocutaneous junction, V-shaped flap then advanced to augment vermilion); vermilion augmentation with grafting materials (e.g., autologous fat) also an option but may be limited in patients with significant scar contracture.
- Vermilion excess: imprecise alignment at initial repair, treated by direct excision and closure.

Other Secondary Deformities

- Mucosal deficiencies—can consider buccal mucosal graft, especially in sulcus.
- Short lip—Z-plasty for mild defects; if orbicularis discontinuity exists (dx—have patient pucker his/her lips → visible bulge adjacent to cleft repair), must take lip down and reapproximate muscle; medial dissection should not extend past midline to avoid obscuring natural philtral dimple.
- Tight lip—relative underprojection of the upper lip; especially following closure of wide cleft; if severe can consider *Abbe cross-lip flap*.
- Wide lip—results from excessive width of initial design and more common in bilateral cleft lip repair; corrected by excising excessive portion along prior scar; reconstructed philtrum has tendency to stretch over time and should not be made wider than 4–5 mm initially to avoid *post-cleft repair wide philtrum*.

Questions

1. What ethnic group is most at risk of developing cleft lip and palate? Least at risk?
2. How does gender affect the likelihood of cleft lip with or without palate? Cleft palate in isolation?
3. What is the likelihood of cleft palate in the next child in normal parents with one affected child (assuming no other relatives are affected)?
4. What are some of the issues facing children with Pierre Robin sequence? Why is ophthalmology referral warranted in these patients?
5. Why are children with Shprintzen syndrome at particular risk during pharyngeal surgery, especially during a pharyngeal flap? What should be ordered prior to surgery?
6. What gene plays a role in van der Woude syndrome and popliteal pterygium syndrome? How do these two syndromes differ?
7. Describe the adult derivatives of the frontonasal prominence, nasal placode, and maxillary prominence. Failure of fusion between what structures causes cleft lip? What is the cause of primary cleft palate? Secondary cleft palate?
8. How does the anatomy of the orbicularis muscle and superior labial artery differ from normal in a unilateral and bilateral cleft lip deformity?
9. Describe the unilateral and bilateral cleft nasal deformity.
10. What are the stages of rhinoplasty to correct the unilateral cleft nasal deformity?
11. Describe the steps in the Millard rotation-advancement repair of the unilateral cleft lip deformity.
12. What is the treatment for unilateral complete CP? Bilateral CP? Secondary cleft palate? Submucous cleft palate?
13. What is the most common complication following cleft palate repair?
14. How does velopharyngeal closure affect the choice of surgical repair in velopharyngeal dysfunction?
15. What is the purpose of alveolar bone grafting in the cleft deformity? Should this be done before or after intermediate rhinoplasty to correct the nasal deformity?
16. What are the three abnormal attachments of the levator veli palatini muscle in the cleft palate deformity?
17. When should primary repair of the cleft lip be performed? What is the “rule of 10s”?
18. When should repair of the cleft palate be performed? Why is the timing of repair important?
19. Describe nasoalveolar molding? What are alternatives to NAM?
20. Describe the arterial supply of the soft palate.
21. Why do infants with cleft lip and palate have difficulty feeding? What can be done to overcome some of these difficulties?
22. How is the secondary tight lip treated? What predisposes to this secondary defect?
23. What is the whistle deformity? How is it treated?
24. What developmental pathology occurs in the condition of bilateral cleft lip and palate?
25. To what structure do the tensor veli palatini muscles originate from?
26. An adolescent patient has undergone early repair of a unilateral cleft lip and palate without correction of the alveolar defect. What type of malocclusion will likely be demonstrated?
27. What are the most common speech problems manifesting through VPI?
28. What anatomic derivatives develop from the embryonic lateral nasal processes?
29. Most surgeons today rely on a superiorly based pharyngeal flap for correction of VPI. What are the main disadvantages of the inferiorly based pharyngeal flap also utilized in the correction of VPI?
30. What pharyngeal motion is ideal for correction of VPI with a pharyngeal flap?
31. Patients with poor posterior pharyngeal wall motion may benefit from a sphincter pharyngoplasty for correction of velopharyngeal dysfunction. What muscles are transpositioned to accomplish tightening of the central port in this procedure?

32. What percentage best represents the incidence of associated malformations in neonates with isolated cleft palate?
33. What side of the face is a cleft lip most common and what side is more often associated with syndromes?
34. In secondary palatal development, when does elevation of the palatal shelves begin and when is fusion complete?
35. What is the purpose of the C-flap in the Millard rotation flap for the repair of unilateral cleft lip?
36. Describe the purpose of the lip adhesion procedure and define the optimal timing of the technique.
37. What is the most common otological manifestation of cleft palate and what is the proposed cause of the condition?
38. What percentage of patients will develop velopharyngeal dysfunction following surgical repair of the cleft palate?
39. At what age is definitive treatment carried out following a lip adhesion procedure?
40. When performing surgical correction of a cleft palate, what muscle sling repair is fundamentally important to velopharyngeal function?
41. In syndromic CLP, specific mutations of candidate genes have been identified. Describe three of the genes that are known to be responsible in a number of cleft-related syndromes.
42. Describe the importance of orbicularis oris muscle approximation in the lip adhesion procedure.
43. What is a submucous cleft and what is the underlying cause of this abnormality?
44. With respect to bone grafting of the deficient alveolar segment, what is the most important guide for timing?
45. How is VPI initially managed prior to determining the need for surgical correction?
46. What are the three main signs for the existence of a submucous cleft palate?
47. What is the purpose of presurgical infant orthopedics (PSIO) and when should such management be initiated?
48. Describe the actions of the velopharyngeal muscles.
49. What is the main cosmetic disadvantage of the Tennison-Randall repair versus the Millard rotation-advancement flap?
50. What method of palatoplasty confers the lowest rates of velopharyngeal dysfunction?
51. Where is the most common site of postsurgical palatal fistualization?
52. What is the most common configuration of VP closure?

Answers

1. Native Americans are the ethnic population at highest risk for the development of cleft lip and palate. African-Americans have the least risk.

   Overall incidence: 3.6/1000 in Native Americans, 2.1 per 1000 in Asians, 1 in 1000 in Caucasians, 0.41/1000 in African-Americans.
2. Males are more likely to develop cleft lip with or without palate (1.5:1 males/females).

   Females are more commonly affected by isolated cleft palate (2:1 females/males). This is thought to be due to later fusion of the palatal shelves in females.
3. 2%; see Table 13.1.
4. Management of the airway is critical in patients with Pierre Robin sequence. Due to the retro-positioned nature of the tongue, airway collapse at the pharyngeal level is common. This can oftentimes be managed by prone or lateral positioning; however these children may need further surgical intervention that may include glossopexy, tracheotomy, or mandibular distraction osteogenesis.

   Due to the high occurrence/association of Pierre Robin sequence with Stickler syndrome, these children should be evaluated at an early age by ophthalmology to rule out any retinal or ocular pathology.
5. Shprintzen syndrome or velocardiofacial syndrome results from an interstitial deletion of chromosome 22. It is associated with a medial course of the carotid artery,

and significant bleeding can result during pharyngeal surgery if this goes unrecognized prior to surgery. Preoperative angiography or alternative radiographic means of identifying the course of the carotid artery should be sought prior to proceeding with surgery in this setting. Additionally, patients with VCF tend to be at higher risk of developing VPI following adenoidectomy.

6. Interferon regulatory factor gene (IRF6) is the gene thought to play a role in both syndromes.

   Van der Woude—most common syndrome associated with CL ± P; findings include lower lip pits/sinus tracts, hypodontia, and autosomal dominant inheritance.

   Popliteal pterygium syndrome—findings similar to van der Woude syndrome with lower lip pits and absent or missing teeth. Additional findings include syndactyly, genital abnormalities, intraoral adhesions (syngnathia), pyramidal skin on hallux, and ankyloblepharon.

7. Frontonasal prominence derivatives: nasal dorsum, forehead, and septum.

   Nasal placode: gives rise to the medial and lateral nasal processes. Structures derived from the lateral nasal processes include the nasal alae and sidewalls. The medial nasal processes fuse and form the intermaxillary segment that forms the primary palate. Failure of fusion of the medial nasal processes results in a midline cleft lip. Additional structures derived from the medial nasal processes are the philtrum, medial upper lip, nasal tip, and columella. The maxillary prominences give rise to the lateral upper lip.

   Failure of fusion between the medial nasal processes and maxillary processes gives rise to cleft lip. Failure of fusion between the intermaxillary segment and the maxillary processes causes primary cleft palate. Secondary cleft palate results from failure of fusion of the palatal shelves (derived from lateral maxillary processes) in the midline, posterior to the incisive foramen.

8. In the unilateral cleft lip deformity, the orbicularis muscle is abnormally attached to the alar base and anterior nasal spine resulting in abnormal displacement of these structures. In the bilateral deformity, the orbicularis muscle is absent in the prolabial segment, while the lateral insertion onto the alar base is similar to that seen in the unilateral cleft lip deformity.

   The anatomy of the superior labial artery in unilateral cleft lip deformity is altered in that it travels parallel to the lateral aspect of the cleft and arborizes with branches of the angular artery. The arterial supply in the central prolabial segment is tenuous and stems largely from the columellar, septal, and palatine blood supply.

9. Unilateral cleft nasal deformity: The cleft alar base is displaced *inferiorly, laterally, and posteriorly* due to maxillary insufficiency and action of the orbicularis (as above), the caudal and inferior septum is deviated to the non-cleft side, and the upper and posterior portions of the septum deviate to the cleft side. Additionally, the cleft side may have inferior turbinate hypertrophy.

   Bilateral cleft nasal deformity: characterized by a poorly projected nasal tip with a wide alar base, alar bases that are positioned similarly to unilateral CL due to pull of abnormally inserting orbicularis oris and bilateral absence of maxillary segment → posterior, lateral, inferior displacement; extremely short columella and the medial crura are displaced into the prolabium; the prolabium and premaxilla can be significantly anteriorly displaced with poor overall blood supply; nasal tip is broad and flattened and the nasal domes are separated resulting in a bifid appearance.

10. Primary, intermediate, and definitive—primary involves separation of LLC from overlying skin with advancement and positioning closer to non-cleft dome. This is usually secured by bolstering the LLC in position following undermining and repositioning. The intermediate stage is performed prior to completion of growth and leads to further refinements in anticipation of definitive septorhinoplasty, usually performed

after growth is complete to correct residual defects of the septum, and nasal configuration. See text above for further details.

11. Methylene blue is used for skin marking. The rotation flap incision is made first (point 3 → 5 with back-cut to x as needed) and is taken through the entire thickness of the orbicularis and oral mucosa to allow for complete release. The c-flap remains and may be used to add additional height to the columella. The advancement flap starts with incision from point 8 → 9 and this incision is then continued along the alar base. The gingivolabial sulcus is also incised and incisions are taken down to face of the maxilla. Bilateral undermining assists in closure, and the first deep stitch reapproximates the orbicularis muscle at the back-cut. The second stitch is placed at the vermilion. The mucosal, muscle, and skin closure follows.
12. See text for full discussion of these techniques.

    Unilateral and bilateral complete: two-flap palatoplasty.

    Secondary cleft palate: V-Y pushback/three-flap.

    Submucous cleft palate: Furlow double-opposing Z-plasty.
13. Velopharyngeal dysfunction
14. See text for full discussion of each technique.

    Coronal—sphincter pharyngoplasty is generally preferred.

    Sagittal closure—pharyngeal flap vs Furlow double-opposing Z-plasty is the preferred method.

    Circular—sphincter pharyngoplasty preferred method.
15. Alveolar bone grafting is performed to restore normal architecture of the maxillary arch and closure of oronasal fistulae (if present, repair should be done prior to eruption of adult canine tooth); secondary bone grafting allows eruption of permanent teeth (specifically the lateral incisor and canine) and provides a base for subsequent correction of nasal deformity, orthognathic and orthodontic treatment.
16. The levator veli palatini has abnormal insertions to the posterior aspect of the hard palate, superior pharyngeal constrictor, and tensor veli palatini aponeurosis. This is seen in clefts of the secondary palate and in patients with submucous clefting.
17. Cleft lip repair is usually done at approximately 3 months of age. It follows the rule of 10s—10 weeks old, hemoglobin 10 g/dL, weight 10 lb; partially based on anesthetic safety.
18. Development of normal speech is a primary concern that dictates the timing of repair (usually done at ~12 months). Repair before 18 months of age has resulted in improved speech outcomes.
19. Nasoalveolar molding (NAM) is a process that involves molding and repositioning of the alveolar processes, nasal cartilages, and lengthening of deficient columella to create lasting aesthetic outcome and reduces need or minimizes the extent of secondary surgical revision procedures.

    Alternatives include lip adhesion procedures, taping, and the Latham appliance.
20. The soft palate is supplied by the ascending palatine (facial artery), greater palatine (internal maxillary), ascending pharyngeal branch, and contribution of the lesser palatine (branch of the greater palatine).
21. Oral intake can be compromised as a result of inability to feed (sucking mechanism impaired—child is unable to form a seal due to incomplete muscular sphincter and/or escape of air through cleft palate). A variety of feeding devices are available to assist in feeding a patient with cleft lip and palate such as the Mead-Johnson, Haberman, or Pigeon to facilitate feeding. Feeding can also be done in the upright position. Additionally, cleft patients tend to swallow more air and may require frequent burping.
22. The secondary tight lip often results from correction of wide cleft lips, and during the healing process, the premorbid absence of tissue leads to an underprojected lip after repair. In severe cases, this can be treated with an Abbe cross-lip flap.
23. The whistle deformity is a secondary deformity that may result following the repair

of unilateral or bilateral cleft lip and palate repair. It is characterized by central deficiency of the vermilion that leads to a shortened vertical height and the characteristic appearance of the upper lip seen with the deformity. Mild deformities may be corrected with V-Y advancement (V apex toward sulcus carried through vermilion and orbicularis, incision carried to mucocutaneous junction, V-shaped flap then advanced to augment vermilion); vermilion augmentation with grafting materials (e.g., autologous fat) is also an option but may be limited in patients with significant scar contracture.

24. Hypoplasia of the bilateral maxillary processes results in insufficient tissue to fuse with the fused medial nasal process/prominence.
25. The tensor veli palatini muscles arise from the membranous wall of the Eustachian tube. Their tendons pass around the hamular processes of the medial pterygoid plate of the sphenoid and insert into the palatine aponeurosis.
26. The arch is deficient in all dimensions: anteroposterior, transverse, and vertical. This child will demonstrate a posterior crossbite of the maxillary dentition relative to the mandibular dentition.
27. Hypernasality and articulation errors (glottal stops and pharyngeal fricatives).
28. Nasal ala and sidewall. Also, the nasolacrimal groove is formed between the LNP and maxillary process.
29. Length limitation and inferior tethering below the plane of the velum. This tethering is contradictory to the motion required to accomplish sufficient closure of the central port.
30. Lateral wall motion is imperative in valve closure in those patients with whom a pharyngeal flap is considered. An ideal patient has effective sagittal or circular closure patterns.
31. The paired palatopharyngeus muscles. The myomucosal flaps are elevated from the posterior tonsillar pillars and rearranged to achieve partial closure of a large central port without creating lateral ports. This arrangement is contrary to that accomplished with a pharyngeal flap.
32. Approximately 55%. Though CPO occurs less commonly than cleft lip and palate, it has a higher incidence of associated anomalies due to its higher association with various syndromes (DiGeorge, velocardiofacial, conotruncal anomaly face syndrome).
33. The left side is the most common (6:3:1—left/right/bilateral). The right side is more frequently associated with genetic syndromes.
34. In normal human development of the secondary palate, initiation begins in the sixth week with elevation of the shelves. The process is complete at week 12 with fusion, which occurs in an anterior-to-posterior direction from the incisive foramen. Additionally, the secondary palate fuses anteriorly with the primary palate and dorsally with the nasal septum, structures that are both derived from the medial nasal processes.
35. One characteristic finding of UCL includes columellar shortening. The C-flap is intended to lengthen the columella.
36. The lip adhesion procedure involves approximating the direct edges of the cleft without changing landmarks or disturbing the orofacial tissues, which will inevitably be required for definite treatment. A lip adhesion procedure should be considered for wide complete and wide near-complete clefts when a definitive lip repair is technically difficult due to tension. It essentially converts a complete cleft into a partial cleft. It is most commonly performed at 2–4 weeks.
37. Persistent OME is extremely common in the cleft palate population, and the prevalence has been estimated to be as high as 80–95%. The main cause is believed to be due to the abnormal insertion of tensor veli palatini, which prevents normal opening of the Eustachian tube.
38. Ten to twenty percent will manifest VPI after surgical correction. Hypernasality and articulation errors are the common manifestations of this condition, which is surgically

repaired by pharyngeal flap or sphincter pharyngoplasty.

39. Although the timing remains controversial with multiple time frames followed (6–12 weeks, 3–4 months, and 5–6 months), the majority of cases are performed at an average of 5 months of age.
40. Approximation and repair of the levator veli palatini sling (intravelar veloplasty) is critical for optimum VP valve functioning.
41. T-box transcription factor-22 (TBX22), poliovirus receptor-like-1 (PVRL1), and interferon regulatory factor-6 (IRF6). These gene mutations are accountable for X-linked cleft palate, cleft lip/palate-ectodermal dysplasia syndrome, and van der Woude and popliteal pterygium syndrome, respectively. The genetic complexity of non-syndromic CLP has also been recognized by a large number of candidate genes and loci (including the above mentioned); however, mutations have been identified only in a small fraction of cases.
42. Muscle pull is essential for the molding effect on maxillary arch segments.
43. A submucous cleft of the soft palate is characterized by a midline deficiency and/or lack of muscle tissue. The defect is lined by overlying oral mucosa. This type of cleft is caused by an anomalous insertion of the levator veli palatini muscle. Instead of muscle fibers being oriented in a transverse manner and decussating in the midline, they instead insert onto the posterior aspect of the hard palate.
44. Dentofacial development is more important than chronological timing with care taken to graft early enough without affecting maxillary growth or the developing permanent dentition. Optimal timing occurs during the mixed dentition stage. Generally, the length of the developing root of the permanent canine tooth is a good indicator. It should be approximately 1/3–1/2 of the permanent root length (17 mm on average). Typically, this would occur between the ages of 7 and 9.
45. In patients without structural defects and/or functional issues (i.e., nasal regurgitation), initial management includes aggressive speech therapy for 6 months. Failure of articulation improvement within 6–12 months should indicate the need for surgical intervention.
46. (1) Zona pellucida—a blue discoloration due to levator veli palatini muscle diastasis, (2) a bifid uvula, and (3) a palpable bony notch at the edge of the hard palate.
47. PSIO involves nonoperative reshaping of the alveolar and nasal segments prior to surgery. The concept, in particular NAM, works on the principle that the nasal cartilage could be molded due to increased plasticity concurrent to increased levels of maternal estrogen, if treatment is initiated within 6 weeks of life. Ideally, treatment should begin in the first 1–2 weeks.
48. *Levator Veli Palatini*: palatal sling causes superior and posterior motion of the soft palate; elevation of the soft palate during deglutition.

    Tensor Veli Palatini: tenses palate and opens Eustachian tube.

    Palatopharyngeus: retrodisplacement and downward motion of the velum; antagonist to LVP.

    Superior Pharyngeal Constrictor: main component of Passavant's ridge; contributes to different patterns of VP closure, specifically sagittal.
49. The Z in the lip crosses the philtral column creating more prominent scarring. The scar is better camouflaged with the Millard technique.
50. Furlow (double-opposing Z-plasty). Technique lengthens the soft palate and reorients LVP. Also, commonly utilized in SMCP repair.
51. Junction of the hard and soft palate.
52. Coronal (most common) > sagittal > circular. Coronal closure pattern is due to action of the velum in an anterior-posterior direction. Choice of VPI repair with coronal closure is a sphincter pharyngoplasty.

Additional Resources

1. Allori AC, Marcus JR. Modern tenets for repair of bilateral cleft lip. Clin Plast Surg. 2014;41(2):179–87.
2. Breitsprecher L. The functional anatomy of the muscles of facial expression in humans with and without cleft lip and palate. A contribution to refine muscle reconstruction in primary cheilo- and rhinoplasties in patients with uni- and bilateral complete CLP. Ann Anat. 2002;184(1):27–34.
3. Coleman JR, Sykes JM. Cleft lip rhinoplasty. In: Papel, editor. Fac Plast Reconstr Surg; 2002. p. 830–43.
4. Coleman JR, Sykes JM. The embryology, classification, epidemiology and genetics of facial clefting. Fac Plast Clinics. 2001;9(1):1–14.
5. Davit A, Otteson T, Losee J. Comprehensive cleft care. Bailey's head and neck surgery: otolaryngology. 5th ed. Ch. 103.
6. De La Pedraja J, Erbella J, McDonald WS, Thaller S. Approaches to cleft lip and palate repair. J Craniofac Surg. 2000;11(6):562–71.
7. Dutton JM, Bumsted RM. Management of the cleft lip nasal deformity. Fac Plast Clinics. 2001;9(1):51–8.
8. Emery BE, Sykes JM, Senders CW. Diagnosis and treatment of secondary unilateral cleft lip deformities. Fac Plast Clinics. 1996;4(3):311–22.
9. Furlow LT Jr. Cleft palate repair by double opposing Z-plasty. Plast Reconstr Surg. 1986;78:724.
10. Gray SD, Pinborough-Zimmerman J. Diagnosis and treatment of velopharyngeal incompetence. Fac Plast Clinics. 1996;4(3):405–12.
11. Huang MH, Lee ST, Rajendra K. Anatomic basis of cleft palate and velopharyngeal surgery: implications from a fresh cadaveric study. Plast Reconstr Surg. 1998;101(3):613–27. discussion 628–9
12. Kohli SS, Kohli FS. A comprehensive review of the genetic basis of cleft lip and palate. J Oral Maxillofac Pathol. 2012;16(1):64–72.
13. Ling EH, Wang TD, Cook TA. Diagnosis and treatment of secondary bilateral cleft lip deformities. Fac Plast Clin. 1996;4(3):323–32.
14. Matsuo K, Hirose T, Otagiri T, Norose N. Repair of cleft lip with nonsurgical correction of nasal deformity in the early neonatal period. Plast Reconstr Surg. 1989;83:25–31.
15. Morales L, Blushke JS. Intermediate cleft rhinoplasty: philosophy, timing, and technique. Fac Plast Clinics. 1996;4(3):343–50.
16. Ness JA, Sykes JM. Basics of Millard rotation: advancement technique for repair of the unilateral cleft lip deformity. Fac Plast Surg. 1993;9:167.
17. Pruzansky S. Presurgical orthopaedics and bone grafting for infants with cleft lip and palate. Cleft Palate J. 1964;1:164–87.
18. Senders CW. Management of velopharyngeal competence. Fac Plast Clinics. 2001;9(1):27–36.
19. Senders CW. Presurgical orthopedics. Fac Plast Clin. 1996;4(3):333–42.
20. Siebert RW. Surgical repair of the bilateral cleft lip deformity. Fac Plast Clinics. 1996;4(3):351–60.
21. Smith DM, Losee JE. Cleft palate repair. Clin Plast Surg. 2014;41(2):189–210.
22. Stal S, Hollier L. Correction of secondary cleft lip deformities. Plast Reconstr Surg. 2002;109(5):1672–81.
23. Strong EB, Buckmiller LM. Management of the cleft palate. Fac Plast Clinics. 2001;9(1):15–25.
24. Sykes JM. Diagnosis and treatment of cleft lip and palate deformities. In: Papel, editor. Fac Plast Reconstr Surg; 2002. p. 813–29.
25. Sykes JM. Management of the cleft lip deformity. Fac Plast Clinics. 2001;9(1):37–50.
26. Sykes JM. Syndromes and congenital anomalies. In: Park SS, editor. Facial plastic surgery: the essential guide. New York: Thieme Publishers, Ch. 1. 2005.
27. Sykes JM, Tollefson TT. Management of the cleft lip deformity. Fac Plast Clin N Am. 2005;13(2):157–67.
28. Tatum SA, Ness JA. Alveolar bone grafting. Fac Plast Clin. 2016;4(3):377–92.
29. Thomas C, Mishra P. Open tip rhinoplasty along with the repair of cleft lip in cleft lip and palate cases. Br J Plast Surg. 2000;53(1):1–6.
30. Valnicek SM, Zuker RM, Halpern LM, Roy WL. Perioperative complications of superior pharyngeal flap surgery in children. Plast Reconstr Surg. 1994;93(5):954–8.
31. Van Hout WM, Mink van der Molen AB, Breugem CC, Koole R, Van Cann EM. Reconstruction of the alveolar cleft: can growth factor-aided tissue engineering replace autologous bone grafting? A literature review of systematic review of results obtained with bone morphogenetic protein-2. Clin Oral Investig. 2011;15(3):297–303.
32. Vyas RM, Warren SM. Unilateral cleft lip repair. Clin Plast Surg. 2014;41(2):165–78.
33. Wang T, Milzcuk. Cleft lip and palate Cummings otolaryngology-head & neck surgery. 6th ed. Ch. 187.
34. Watkins SE, Meyer RE, Strauss RP, Aylsworth AS. Classification, epidemiology, and genetics of orofacial clefts. Clin Plast Surg. 2014;41(2):149–64.

14 Craniofacial Anomalies

Eugenia Chu and Leslie Irvine

Embryology

- Embryonic period, first 8 weeks' gestation; fetal period, ninth week to birth
- Embryonic period divided into 23 stages based on Carnegie system
- Ectoderm: cutaneous and neural systems
- Endoderm: lining of epithelium of digestive and respiratory systems, secretory cells of the liver and pancreas
- Mesoderm: cardiovascular, connective tissues, bones, and muscles

Neural crest cells—migrate to form the craniofacial mesenchyme—form majority of facial structures except the retina, lens, epithelial tissues, vascular endothelia, and skeletal muscle. The face is first recognized at stage 9 (day 20): appearance of stomodeum (depression between the brain and pericardium, precursor to the mouth and pituitary gland), optic disc present.

Development of the face is a result of disappearance of grooves and bulges which enlarge with proliferating mesenchyme, instead of the migration and fusion of distinct processes.

Middle 1/3 of the face associated with changes in the forebrain.

Lateral and lower facial regions formed by maxillary and mandibular processes of the first pharyngeal arch (first pharyngeal arch: maxillary process, premaxilla, maxilla, zygoma; Meckel cartilage, mandible, incus, malleus).

Stage 11 embryo: stomodeum (surface ectoderm) invaginates→buccopharyngeal membrane (consists of ectoderm and endoderm) breaks down→primitive oral cavity.

Stages 12 and 13: third and fourth pharyngeal arches are well developed; the forebrain broadens to form frontal prominence; the nasal placode develops as ectodermal thickening of frontal prominence.

Stages 14 and 15: mesoderm surrounding nasal placodes leading to the formation of lateral nasal fold and medial nasal fold, the depression of the nasal placodes becomes olfactory pits; olfactory pits divide the frontal prominence into upper and lower parts:

- Upper part—primitive forehead
- Lower part—future external nose

Maxillary prominence develops medially and contacts medial and lateral nasal folds.

Two medial nasal folds form and give rise to the intermaxillary segment→philtrum upper lip and anterior portion maxilla.

E. Chu
Department of Otorhinolaryngology – Head and Neck Surgery, Georgetown University Hospital, Washington, DC, USA

L. Irvine (✉)
Private Practice, Manhattan Beach, CA, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_14

Nasolacrimal groove (future nasolacrimal duct): in between maxillary prominence and lateral nasal fold.

Controversial origin of NLD: some believe the nasolacrimal groove is completely eliminated and the NLD arises as separate entity vs. NLD is remnant of nasolacrimal groove.

Stages 17 and 18: formation of external nose begins; olfactory pits open into the roof of the mouth.

Primitive oral cavity forms the palatal shelves, and mesoderm forms between medial nasal folds and maxillary process forms the upper lip. Lateral nasal wall develops into the inferior turbinate, then middle, and superior.

Paranasal sinuses develop from lateral walls of nasal cavity later.

Stages 19–23: Mesoderm from second pharyngeal arch develops into facial muscles. Lateral to Meckel cartilage is where mandibular ossification occurs; the TMJ develops during this stage; mesoderm fills between nostrils which gives external nasal prominence and epineural ectoderm from the roof of nasal cavity, and this also gives rise to olfactory epithelium.

Sinuses in order of development: Maxillary, first to develop in utero, biphasic growth at 3, then at 7 years old.

Sphenoid: second to develop; evagination of nasal mucosa into sphenoid bone (age?). Begins in utero (third month of gestation), completes pneumatization by 10 years of age.

Ethmoid: most developed sinus at birth.

Frontal: develops after birth; 5–6 years old.

Embryology of the Skull

Mammalian skull has four bones: frontal, parietal, squamosal, anterior portion occipital bone.

Cranial bones→intramembranous ossification →mesenchyme→differentiate into osteoblasts and deposit ECM.

Cranial sutures: fibrous tissues uniting cranial bones; allow for expansion during brain growth, major growth center in the skull for first years of life (Fig. 14.1):

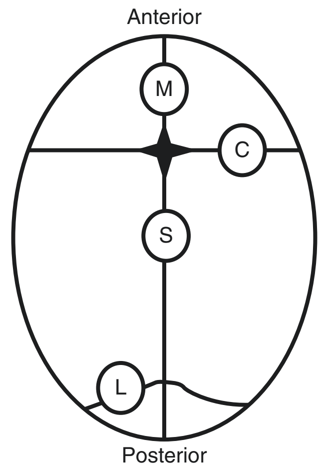


Fig. 14.1 Cranial sutures. M metopic, C coronal, S sagittal, L lambdoidal (shaped like the uppercase Greek lambda)

- Sagittal suture: btw paired parietal bones
- Metopic suture: btw paired frontal bones
- Coronal suture: btw paired frontal and sagittal suture
- Lambdoid suture: btw paired parietal and interparietal bone

Anatomy

Layers of scalp: mnemonic SCALP

S: skin
C: subcutaneous tissue
A: aponeurosis and muscle (galea) (SMAS is continuation of galea onto the face) (temporoparietal fascia is thin lateral extension of the galea aponeurosis)
L: loose areolar tissue
P: pericranium

Blood supply Main blood vessels run superficially in the galea→supratrochlear, superficial temporal artery, supraorbital and terminal branches of the occipital artery, postauricular artery (occipital and postauricular arteries run deeply before arising superficially near the scalp).

Dental Occlusion

Angle Classification for Occlusion

Class I: maxillary first molar slightly back to mandibular first molar; “mesiobuccal cusp of maxillary first molar is in line with buccal groove of mandibular first molar.”

Class II: maxillary first molar even with or anterior to mandibular first molar, “buccal groove of mandibular first molar distal to mesiobuccal cusp of maxillary first molar”; facial profile→ retrognathic.

Division I: maxillary central incisors normal or slightly protruded toward lips.

Division II: maxillary central incisions retruded toward the oral cavity.

Class III: maxillary first molar is toward back of mandibular first molar; “buccal groove of mandibular first molar is mesial to mesiobuccal cusp of maxillary first molar; facial profile→ prognathic.

Cross bite abnormal relationship of one arch of teeth to the opposing arch of teeth; maxillary incisors lingual to opposing mandibular incisors.

Overbite greater than 1/3 vertical overlap of maxillary teeth over the mandibular anterior teeth.

Overjet horizontal overlap between labial surface of mandibular anterior teeth and lingual surface of maxillary anterior teeth.

Orthognathic surgery
Vertical-subcondylar osteotomy: used for prognathism, intra- or extraoral approach; oblique osteotomy from sigmoid notch to mandibular angle, posterior to inferior alveolar nerve; proximal segments moved laterally; mandible setback.

Sagittal split osteotomy: used for prognathism or retrognathism, intraoral approach; horizontal osteotomy 5 mm above the lingula and second osteotomy vertically at the second molar; for prognathism, vertical piece of bone removed equal to desired distance of setback; wide surface area of contact and rapid healing; high risk of lower lip anesthesia; risk of retrognathia recurrence due to suprahyoid musculature pulling posteriorly.

Syndrome pattern of multiple anomalies, pathologically related and have common (suspected) cause.

Craniofacial syndrome affects facial features; affected patients tend to look alike, even though they share no familial relationship, e.g., Down’s syndrome.

Sequence anomaly or pattern of multiple anomalies that arise from a single known or prior anomaly; one anomaly occurs due to a preexisting anomaly, e.g., Pierre Robin sequence→U-shaped palate, glossoptosis, micrognathia; this sequence occurs due to interference in development of the mandible during gestation which causes the tongue to remain high in the oral cavity, which leads to disruption of the closure of the velum (causing palate defects) → ultimately causing upper airway obstruction. The sequence is caused by the inciting event during mandible development.

Association nonrandom occurrence pattern of multiple anomalies in two or more individuals that are not part of a syndrome or sequence; diagnosis of exclusion, pathogenesis of the association is unknown; the recurrence risk in association is no greater than the general population, e.g., VATER association (vertebral, anorectal, TE fistula, renal/limb anomalies).

Craniosynostosis

Premature fusion of one or more cranial suture lines

- Definitions:
  – Syn: joined/union
  – Plagio: slanting/inclining/oblique
  – Scapho: boat (boat-shaped)
  – Cephaly: head
  – Metopic: frontal/relating to the forehead
- Affects 1/2000–2500 births worldwide nonsyndromic (single suture)
- 1/30,000–100,000 syndromic (frequently multisutured)
- Etiology multifactorial: genetic, environmental
  – Genetic: Mutations in genes that affect mechanical force signaling pathways and cytokines that mediate cranial suture patency→*fibroblast growth factor receptor* (*FGFR*) and transforming growth factor (TGF-B)
  – FGFR 1,2,3 known to be involved in syndromic craniosynostosis
  – Environmental: multiple associations: paternal occupations with agriculture, forestry; maternal age; exposure to tobacco smoke
  – Medications: nitrofurantoin, warfarin use in pregnancy
  – Nutritional deficiencies: folic acid
- Malpositioned fetus and intrauterine constraint. Fused sutures typically cause restriction in skull growth, dysmorphic cranial vault, and facial asymmetry.
- Due to Virchow law, when suture line is fused prematurely, growth perpendicular to the affected suture is disrupted→compensatory growth enhanced parallel to affected suture.
- For example, synostosis of sagittal suture: growth proceeds in anterior to posterior direction→leads to scaphocephaly. Synostosis of coronal suture: leads to growth in mediolateral plane resulting in brachycephaly.
- Most common functional consequence reported: *increased intracranial pressure*
  – Higher ICPs seen in children with multiple suture involvement
- Indications for surgical intervention early in life: visual impairment, deafness, cognitive deficits.
- Condition usually occurs as isolated condition, but can manifest in association with syndrome.
- Sagittal synostosis most common type: seen in 40–55% nonsyndromic cases.
- Coronal synostosis: second most common (20–25%).
- Metopic synostosis: third most common (5–15%); metopic suture line fuses as early as 3–6 months and normally disappears (unlike other cranial sutures).
- Lambdoid synostosis: rare (0–5%).
- More than one suture affected in 5–15% cases.
- Surgical intervention is the only treatment for the condition.

Skull Deformities (Table 14.1, Fig. 14.2)

Scaphocephaly boat-shaped head, most common manifestation of sagittal synostosis; calvarial bone growth limited perpendicular to affected sagittal suture line→narrowing head transversely, brain grows anterior posterior→ leads to frontal bossing or occipital cupping.

Trigonocephaly the forehead appears more triangular; metopic synostosis causes metopic ridging in the midline forehead or combo of ridging, bitemporal narrowing, and hypotelorism.

Deformational plagiocephaly flattening of the head; most common type of plagiocephaly is positional, when the skull is subject to pressure usually due to supine positioning; congenital torticollis associated with condition; repositioning maneuvers/specialized pillows used to keep babies off affected side; when severe flattening occurs beyond 6 months, helmets used to improve cranial asymmetry when used before 12 months.

Anterior plagiocephaly unilateral coronal synostosis; affected frontal bone is underprojected, contralateral frontal bone protrudes anteriorly and can push orbit inferiorly, leading to orbital asymmetry or vertical dystopia; superior orbital

Table 14.1 Syndromic craniosynostoses chart

Syndrome	Genetics	Skull abnormality	Facial features	Related symptoms	Extremity features
Apert	FGRF2; autosomal dominant; majority sporadic	Turribrachycephaly (top of the skull is pointed or cone shaped), acrocephaly, bitemporal bulging, sagittal synostosis, bilateral coronal synostosis, bilateral lambdoid synostosis	Orbital hypoplasia, hypertelorism, proptosis, downslanting palpebral fissures, beaked nose, midface hypoplasia, mandibular prognathism, high-arched palate	Mental retardation, intracranial hypertension, cerebral atrophy, wide subarachnoid spaces	Pansyndactylies of hands and feet
Crouzon	FGFR2, FGFR3 (acanthosis nigricans); AD, sporadic, familial	Acrocephaly, coronal, lambdoid, basilar synchondroses/ synostoses	Shallow orbit, orbital proptosis, hypertelorism, strabismus, beaked nose, maxillary hypoplasia, mandibular prognathism, high-arched palate, enlarged sella turcica, jugular foramen stenosis	Mental retardation Hydrocephalus, syringomyelia, intracranial HTN, optic atrophy	Usually not affected
Pfeiffer	FGFR2, FGFR1, AD; sporadic, familial	*Type I*: Turribrachycephaly, bilateral coronal, frontosphenoidal synostoses *Type II*: Cloverleaf deformation *Type III*: Severe turribrachycephaly, bilateral coronal, frontosphenoidal, sagittal, metopic synostoses	Orbital proptosis, palpebral retraction, jugular foramen stenosis, midfacial hypoplasia, mandibular prognathism, high-arched palate, bifid uvula, aural atresia	Intracranial HTN, hydrocephalus, optic atrophy, mental retardation (types II–III), hearing loss, sleep apnea	Brachydactyly
Saethre-Chotzen	TWIST gene mut chr 7; AD, familial mostly; sporadic	Acrocephaly, plagiocephaly, scaphocephaly, unilateral coronal, b/l coronal, metopic, b/l lambdoid synostosis	Low-set frontal hairline, flat forehead, hypertelorism, eyelid ptosis, tear duct stenosis, high-arched palate, mandibular prognathism, angulated ears	Mental retardation, conductive hearing loss	Partial cutaneous syndactyly, some brachydactyly
Carpenter	RAB23 or MEGF8 mutation; AR	Sagittal and lambdoid synostosis	Downsloping palpebral fissures, low-set ears, short neck	Mental retardation (some), short height, obesity	Variable polysyndactyly, clinodactyly, brachydactyly
Muenke	FGFR3 mutation; AD	Unilateral or bilateral coronal synostosis	Variable hypertelorism, midface hypoplasia, low-set ears, hearing loss	Normal intelligence	Carpal and tarsal fusions

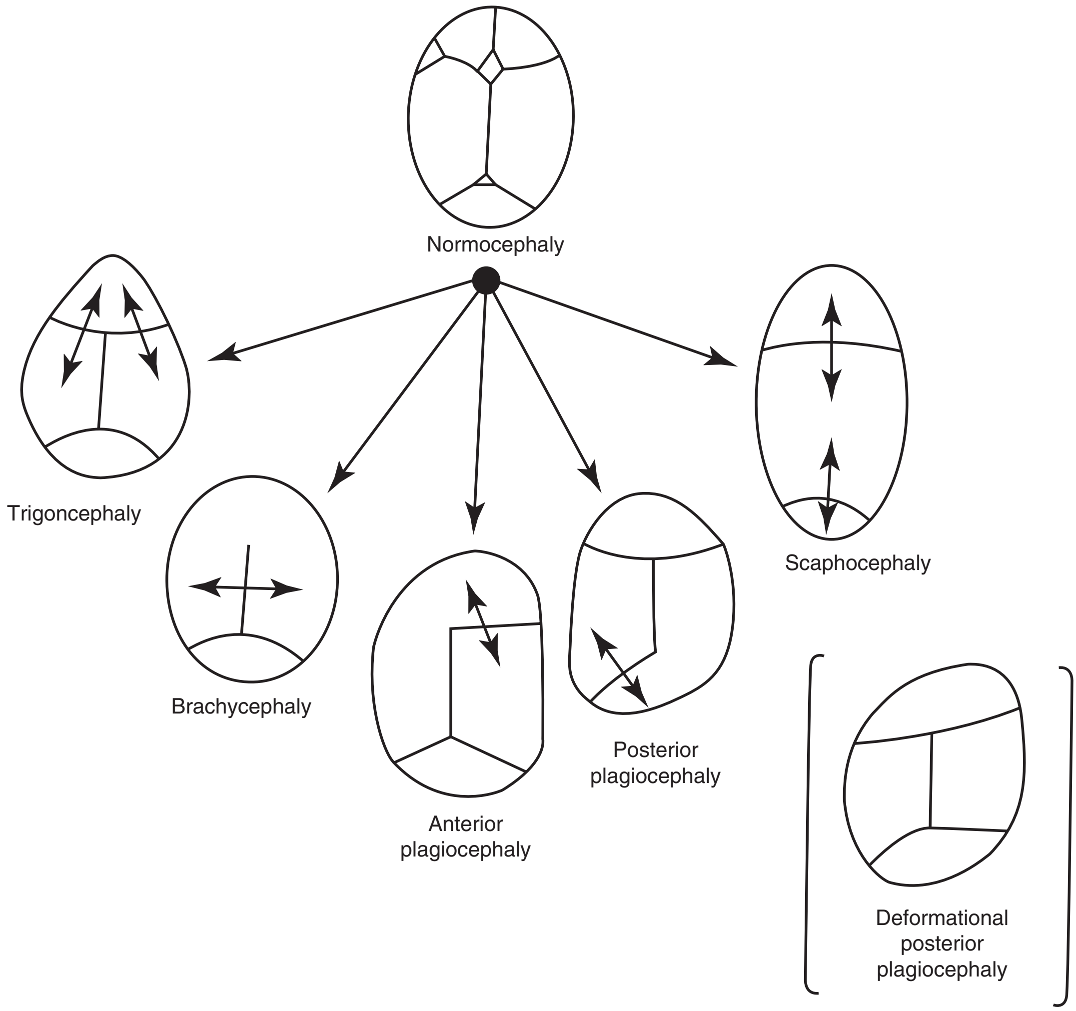


Fig. 14.2 Schematic illustration of suture changes in single-suture craniosynostosis. (With permission from Papel L, et al. *Facial Plastic and Reconstructive Surgery*. 3rd ed. Thieme: New York; 2009, p. 1030)

rim is elevated (harlequin eye); late-presenting synostosis can cause nasal deviation and mandibular displacement.

Posterior plagiocephaly unilateral occipital flattening can be caused by deformational plagiocephaly or lambdoid synostosis; also presents with ipsilateral mastoid bulging, posterior inferior displacement of ipsilateral ear; deformational plagiocephaly causing posterior plagiocephaly much more common than lambdoid synostosis.

Brachycephaly bilateral occipital flattening; short head deformation; may be caused by supine positioning or bilateral coronal synostosis.

Cloverleaf skull fusion of all cranial sutures, except metopic and squamosal; causes brain restriction in multiple planes→characteristic bulging occurs across two opened sutures giving cloverleaf shape; this is the most challenging craniosynostosis due to bony growth restriction, and limited intracranial volume can lead to increased ICP and hydrocephalus; impaired vision common due to proptosis and orbital hypoplasia. *Multidisciplinary team approach*: Craniosynostosis is often part of syndrome, and interdisciplinary team is involved to plan complicated care for these patients. Team usually involves plastic surgeon, pediatric otolaryngologist or facial plastic surgeon trained in craniofacial surgery, pediatric neurosurgeon, oral surgeon, pediatric anesthesiologist, pediatric intensivist, pediatric orthodontist, pediatric ophthalmologist, psychologist, geneticist, audiologist, speech pathologist, and general pediatrician.

Timing of surgical procedures depends on factors including age of patient at initial presentation, presence or absence of increased ICP, upper airway obstruction, severity of deformities, and surgeon preferences. Most recommend surgical correction by 3–12 months (publications vary on this range; 6 months is most commonly reported as optimal age); this allows for children younger than 1 year to completely re-ossify and relies on the malleability of the calvarium during this age and the tremendous brain growth that occurs during the first year.

The timing and sequence of synostosis surgery are usually determined by age. See Table 14.2.

Main goals for surgical treatment: excising fused suture and normalizing calvarial shape to increase intracranial volume→avert potential sequelae of increased ICP.

Surgical procedures include strip craniectomies and various cranial vault-remodeling techniques.

Delay in correcting craniosynostosis results→deformity of cranial base, facial asymmetry, dental malocclusion.

Distraction osteogenesis typically used for monobloc, posterior cranial vault, or midface distraction; performed by external or internal distracters; osteotomies are made with open approach, distracters placed, bone segment advanced slowly over days to weeks, by 1–3 mm/day to induce callous formation between distracted bones and induce new bone formation, regenerated bone strength 75% of normal bone; allows soft tissue to stretch over time; particularly useful for syndromic patients with brachycephaly where adequate advancement is difficult to achieve through conventional surgery.

Table 14.2 The timing and sequence of synostosis surgery are usually determined by age

Age	Procedure
3–6 months	Correction of craniosynostosis
1–2 years	Correction of syndactyly
4–5 years	Correction midface retrusion w/ distraction techniques
4–6 years	Correction hypertelorism and turricephaly
>17 years	Orthognathic surgery (Le Fort I or III in conjunction with mandibular osteotomy to correct malocclusion)

Surgical Treatment of Sagittal Synostosis

Simple strip craniectomy (suturectomy) this procedure largely abandoned unless done concomitant with spring assistance or postoperative molding helmets; recent authors advocate for use of minimally invasive suturectomy which decreases hospital stay and intraoperative bleeding and reoperations.

Complications of surgery: infection, optic nerve ischemia, seizures, bleeding, need for frequent blood transfusion, mortality rate 1.5–2%.

Endoscopic-assisted strip craniectomy with wedge craniectomy more modern technique; can be done in patients presenting at 3–6 months and for less severe deformities. Total cranial vault reconstruction is recommended at age 9–12 months for patients who present later than 6 months or have severe deformities. Two-stage procedure often advocated when occipital and frontotemporal deformities present, first surgery done at 3–6 months, second surgery at 12 months.

Pi procedure aka squeeze procedure; two parasagittal craniectomies performed with transverse craniectomy; used to shorten the skull in AP direction and squeeze the brain in the AP direction; bone grafting parasagittally adds to increased biparietal width. Postoperative molding helmeting is required unless spring utilized.

Fronto-orbital advancement with reshaping keystone procedure in trigonocephaly reconstruction in unilateral and bilateral coronal synostosis; procedure advances the unilateral or bilateral flattened superior orbital rim anteriorly and secures it in place; this construct, "bandeau," becomes the foundation of the remainder of the cranial vault; the supraorbital/supratrochlear neurovascular bundles are released from their bony notches; once the osteotomies are made, the bandeau is reshaped; for hypertelorism, the bandeau may be divided in the midline and small bone graft is placed; often bone grafts are placed as struts along supraorbital bar to reinforce and minimize relapse or secondary deformities.

Posterior synostotic plagiocephaly treatment lambdoid synostosis repair is done similar to coronal synostosis reconstruction; occipital bar is advanced posteriorly in the area of flattening, and the occipitoparietal region is reshaped to achieve symmetry posteriorly; bone flap is secured with absorbable plates and screws; protective helmet needed to prevent relapse due to supine positioning.

Absorbable plating material:

- Poly-L-lactic-polyglycolic copolymer (PLLA-PGA)
- Tessier clefting: classification system for rare craniofacial clefts.
- Clefts: 0 → 14.
- Cranial clefts: above the eyelids and orbits.
- Facial clefts: below the eyelids and orbits.
- Treacher Collins: Combination of #6, 7, 8 clefts.
- Hemifacial microsomia: Disruption of development of first and second branchial arches; due to environmental, maternal, and genetic factors. Associated with thalidomide, retinoic acid, maternal diabetes, several newly identified mutations.

Questions

1. The face is first recognized by the appearance of what embryologic structure?
   (a) Nasal placode
   (b) Maxillary process
   (c) Stomodeum
   (d) Palatal shelve
2. What paranasal sinus is known to develop first in utero?
   (a) Maxillary
   (b) Sphenoid
   (c) Ethmoid
   (d) Frontal
3. A patient presents with an overbite. Describe their angle classification with relationship to the maxillary molar to mandibular molar.
4. What gene mutation is most responsible for most syndromic craniosynostosis?
   (a) TWIST
   (b) FGFR
   (c) Myc
   (d) P53
   (e) Ret
5. At what age is repair of craniosynostosis best performed?
   (a) 1 month
   (b) 15 months
   (c) 6 months
   (d) 24 months
6. In what syndromic craniosynostosis is a cloverleaf skull deformity most likely to be observed?
   (a) Apert
   (b) Crouzon
   (c) Pfeiffer type I
   (d) Pfeiffer type II
7. Describe options for treatment management of sagittal synostosis. What is the significance of the "bandeau"?
8. The most common functional consequence of craniosynostosis is?
   (a) Increased intracranial pressure
   (b) Impaired vision
   (c) Mental retardation
   (d) Respiratory insufficiency
9. Posterior plagiocephaly is most commonly caused by?
   (a) Supine positioning
   (b) Lambdoid synostosis
   (c) Metopic synostosis
   (d) Sagittal synostosis
10. What skull deformity is most commonly seen in sagittal synostosis?
    (a) Anterior plagiocephaly
    (b) Brachycephaly
    (c) Trigonocephaly
    (d) Scaphocephaly
11. Describe indications for the use of distraction osteogenesis in the treatment of craniosynostosis.

12. The majority of orbital growth occurs between what ages?
    (a) 5 and 8 years
    (b) Birth and 3 years
    (c) 8 and 12 years
    (d) 3 and 5 years
13. Molding helmets should be used:
    (a) Between 2 and 8 months
    (b) At birth
    (c) Between 6 and 12 months
14. Which of the following is an advantage of using biodegradable implants for craniosynostosis surgery?
    (a) Assist in bone regeneration
    (b) Less risk of breakage of plates
    (c) Reduced risk of inflammation and subsequent revision surgeries
    (d) Reduced need for second operation to remove the material
    (e) A and D
    (f) A and C
15. Which of these is not an advantage of distraction osteogenesis?
    (a) Gradual expansion of facial soft tissue
    (b) Decreased risk of infection
    (c) Decreased dead space
    (d) Rapid results
16. When is the average time of onset of infection of poly-L-lactic-polyglycolic copolymer (PLLA-PGA) resorbable plates for cranial fixation?
    (a) 1 week
    (b) 2 weeks
    (c) 2 months
    (d) 1 year
17. Which of the following is the greatest risk of sagittal split osteotomy surgery?
    (a) Lower lip anesthesia
    (b) Marginal mandibular nerve injury
    (c) Osteonecrosis of intervening bone

Answers

1. (b)
2. (a)
3. Overbite is defined as greater than 1/3 vertical overlap of maxillary teeth over the mandibular anterior teeth
4. (b)
5. (c)
6. (d)
7. Surgical treatment of sagittal synostosis:
   - Simple Strip craniectomy (suturectomy)
   - Endoscopic assisted strip craniectomy with wedge craniectomy: more modernized technique compared to simple strip craniectomy
   - Pi Procedure: 2 parasagittal craniectomies are performed with a transverse craniectomy used to shorten the skull in the AP direction; additional bone grafting parasagitally adds to increased biparietal width
   - Fronto-orbital advancement with reshaping: keystone procedure in trigonocephaly reconstruction in unilateral and bilateral coronal synostosis, this procedure will advance the unilateral or bilateral flattened superior orbital rim anteriorly and secure it in place
   - § *The bandeau is the reconstruct which will become the foundation of the remainder of the cranial vault. The bandeau can be reshaped for hypertelorism with grafting
   - Posterior Synostotic Plagiocephaly treatment: an occipital bar is advanced posteriorly in the area of flattening; occipito-parietal region is reshaped to achieve symmetry posteriorly
8. (a)
9. (a)
10. (d)
11. Distraction osteogenesis is typically used for monobloc, posterior cranial vault or midface distraction. This procedure is particularly useful for syndromic patients with brachycephaly where adequate advancement can more technically challenging with traditional techniques.
12. (b)
13. (c)
14. (e)
15. (d)
16. (b)
17. (a)

15 Otoplasty

Gregory S. Dibelius and Colleen F. Perez

Basic Anatomy

The normal vertical position of the *auricle* lies between horizontal lines drawn from the upper rim of the orbit and nasal spine. The crus radix, or root of the helix, lies above the external auditory meatus and extends superiorly around the contour of the auricle as the helical rim. After completing its curvature superiorly, the helical rim descends to terminate at its cauda helicis or tail. The *antihelix* runs parallel to the curvature of the helical rim within the auricular structure, separated from it by the narrow *scaphoid fossa*. The antihelix diverges superiorly into a superior and inferior crus, between which lies the *triangular fossa*. The superior crus is characteristically wider and smoother than the inferior crus. Inferiorly the antihelix terminates above the lobule as the antitragus. Within the curvature of the antihelix lies the conchal portion of the auricle. The concha is composed of a superior *cymba concha* and an inferior *cavum concha*, approximately 8 mm deeper than the overlying *tragus* and *antitragus*. The *lobule* extends inferiorly from the cartilaginous framework and is itself devoid of cartilage (Fig. 15.1). The glenoid fossa of the mandible lies deep to the preauricular area, and temporomandibular joint problems may manifest as ear pain.

The auricular cartilage is enveloped within a thin, firm, adherent layer of the perichondrium which provides the vascular supply to the underlying cartilage. The anterior lateral surface of the cartilage is covered with a fine, thin skin that adheres closely to the cartilaginous framework. Subcutaneous tissue is very limited; however, a diffuse subdermal vascular plane exists that supports flap viability. The posterior surface of the cartilage framework is covered with a less adherent skin that contains two layers of fat and a

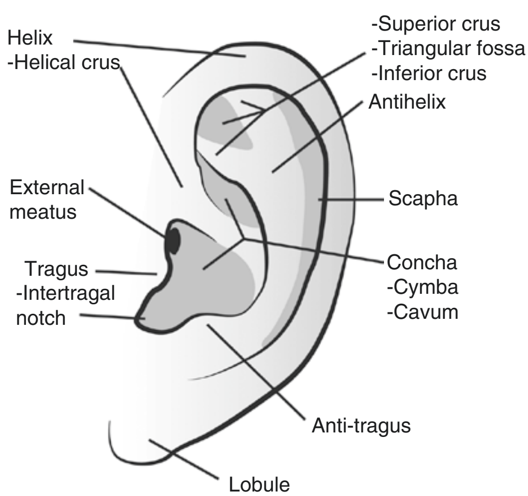


Fig. 15.1 External ear anatomy

G. S. Dibelius (✉)
Private Practice, New York, NY, USA
e-mail: drdibelius@rhinoplastymanhattan.com

C. F. Perez
Otolaryngology-Head and Neck Surgery, Naval Medical Center San Diego, San Diego, CA, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_15

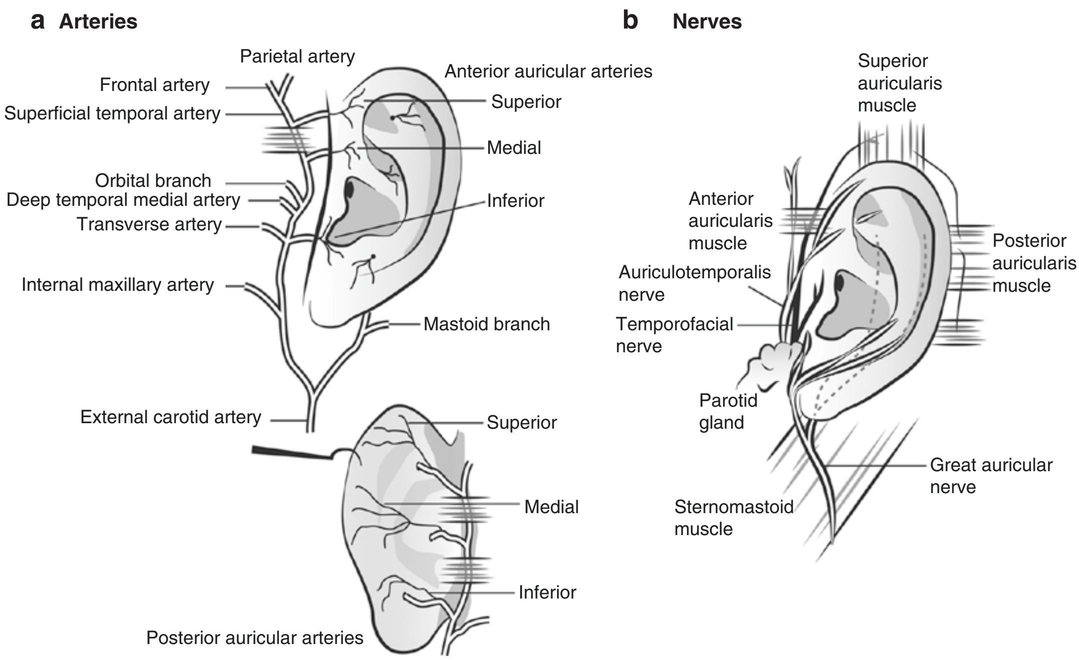


Fig. 15.2 (**a**) Arterial blood supply to the ear. (**b**) Nerve supply to the ear

larger subdermal plexus of arteries, veins, and nerves (Fig. 15.2a).

Arterial supply Two main branches of the *external carotid artery*—superficial temporal artery *(STA)* and the posterior auricular artery *(PAA)*. STA emerges from the parotid capsule 1 cm in front of the ear deep to the veins and below the anterior auricular muscle. It gives off the *superior, medial, and inferior branches* that supply the anterior and anterolateral surface of the auricle. The PAA supplies the posterior surface and travels parallel to the postauricular crease upward and crosses below the great auricular nerve and under the posterior auricular muscle. An extensive collateral system exists between the two systems.

Venous drainage Complementary veins into the external jugular vein.

Lymphatic drainage Preauricular and superficial cervical nodes.

Innervation Multiple sensory nerves supply the ear, including branches of the cervical plexus (lesser occipital, great auricular) and cranial nerves V (auriculotemporal), VII, IX (Jacobsen's nerve), and X (Arnold's nerve). The lesser occipital and great auricular nerves arise from Erb's point. The great auricular nerve travels 8 mm posterior to the postauricular crease and overlies the PAA.

Motor supply to the small vestigial auricular musculature is supplied by the facial nerve (Fig. 15.2b).

Regional nerve block anesthetic techniques must take into account the multiple innervation of the ear. The field block must encompass the areas supplied by the auriculotemporal, great auricular, and lesser occipital nerves. This can be accomplished by injecting anesthetic at the base of the auricle anteriorly and posteriorly. Supplemental anesthesia may be needed at the posterior wall of the external auditory meatus due to small branches supplied by the vagus nerve.

Structural support Auricular cartilage is a single piece of elastic fibrocartilage with a concave, convoluted anterior side and a smooth, posterior convex side. The cartilage thickness is uniform throughout. Extrinsic (anterior, posterior) and intrinsic ligaments attach the auricular cartilage to the skull and provide additional structural support. The lobule is suspended from the above cartilage.

Embryologic development of the ear

- Third week of intrauterine growth: Otic placode is present.
- Fifth week of gestation: The hillock theory postulates that three hillocks (hillocks 1, 2, and 3) arise from the first branchial (mandibular) arch and three (hillocks 4, 5, and 6) arise from the second branchial (hyoid) arch on either side of the first branchial cleft. Each hillock corresponds to an auricular landmark: (1) the tragus, (2) the helical crus, (3) the helix, (4) the antihelix, (5) the antitragus, and (6) the lobule. An alternate theory holds that only the tragus develops from the first branchial arch.
- The auricle emanates from the mesoderm of the first and second branchial arches.
- Week 12: The hillocks have fused. The concha derives from the ectoderm of the first branchial groove. There is a transient obstruction of the medial canal by proliferating epithelial cells to form a meatal plug that eventually dissolves, leaving a patent canal:
  – Lack of formation of the antihelix between the 12th and 16th weeks of embryonic life is a congenital hereditary *autosomal recessive* malformation that results in a prominent ear.
- Age 3: 85% of ear development complete.
- Ages 7–8: Nearly fully grown. Ear growth continues into adulthood, but width and distance of the ear from the scalp change little after age 10.
- The majority of auricular deformities are inherited in an *autosomal dominant* pattern.

Relationships and Angles

- *Concho-mastoid angle* is approximately 90° (Fig. 15.3).
- *Scapha-conchal angle* is a 90° angle formed as the *antihelical fold* (Fig. 15.3).
- *Auriculocephalic angle* is these two angles in combination with the curvature of the helix which set the auricle adjacent to the scalp at approximately 25–35°.
- *The helical rim* should be seen just lateral to the antihelix on the frontal view. The distance between the most lateral edge of the helix and the mastoid skin is around 2–2.5 cm. The distance between the scalp and the superior aspect of the helix is approximately 1 cm. Vertical height of the ear is approximately 59–63 mm and horizontal length is 33–36 mm.
- *Ear level (vertical position)* lies between horizontal lines drawn from the upper rim of the

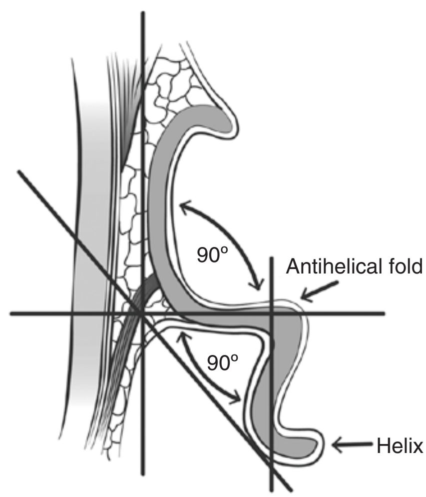


Fig. 15.3 Concho-mastoid and scapha-conchal angle

orbit and nasal spine. The superior limit should be either the lateral brow or the upper eyelid. The ratio of ear length to facial height declines from 55% in 4-year-olds to 51–53% in adults.

- *Ear inclination* can be measured in the sagittal plane with the head positioned in the Frankfort horizontal plane. It is determined by the angle formed by the longitudinal axis of the ear (connecting the highest and lowest points) and the true vertical. Ideal angle is 24.8°. The ear is typically not parallel to the nasal dorsum, and the angle between the axis of the ear and the bridge of the nose is 14.9°.

Otologic assessment Assess for hearing loss, tinnitus, vertigo, fullness, and otalgia in the history. Examine the external auditory canal and tympanic membrane with the otoscope and tuning fork tests. An audiogram may be indicated if hearing loss is suspected. Preoperative photography is important and should always be performed, including frontal, full right and left oblique, full right and left lateral, and close-up right and left lateral views.

Ear Deformities

Lop ear lid-like turning down of the helix characterized by reduced vertical height, reduced fossa triangularis, reduced scapha, and reduced or absent superior crus of the antihelix.

Stahl (satyr/vulcan) ear pointed ear characterized by an abnormal posterior antihelical crura extending to the helical rim, resulting in a pointed upper edge.

Shell ear characterized by underdeveloped helical rims.

Cauliflower ear deformity characterized by repeated trauma and often a history of auricular hematomas inadequately drained. Results from ischemia and necrosis of the underlying cartilage followed by fibrosis and tissue formation and scarring. Often seen in boxers or wrestlers.

Prominauris two major deformities account for most abnormalities of protruding ears (individually or in combination): (1) poorly developed antihelical fold and (2) overdevelopment of conchal cartilage.

Other deformities include a protruding earlobe, irregularities along the helix (including an unrolled margin of the helical rim), and an anteromedially displaced insertion of the postauricular muscle.

Otoplasty Surgical Principles

The age at which surgery is performed is a point of controversy. In many cases age 6 or older is preferred, when the cartilage has reached adequate consistency and maturity. Some surgeons advocate earlier repair, even younger than age 4, with documented success. Adults may have less flexibility of the auricular cartilage, as well as some degree of calcification, which will render it brittle and make the operation more difficult and less likely to succeed with suture techniques.

The contralateral normal ear should be used for symmetry in unilateral cases. Intraoperative measurements of the mastoid-helix distance should be made at the superior margin, the EAC, and the lobule.

Many techniques are described; however they can be categorized broadly as suture-only, cartilage-splitting, cartilage-weakening, or combination techniques.

Suture-Only Techniques

Mustarde method (concho-scaphal suturing) Suture technique used to treat the deficient antihelical fold by reconstruction of the conchoscaphalic angle. Commonly used in conjunction with the excisional or cartilage-weakening techniques. The surgery is approached through a postauricular incision. The scapha is folded back against the underlying scalp by pressing on the superior helical rim. The fold can be planned with the aid of through-and-through 25 gauge needles. The crest of the fold is marked, and marks parallel to the crest are made 7 mm apart to avoid excessive narrowing. The lateral marks are tattooed through the skin and cartilage with methylene blue. Sutures are placed perpendicularly across the antihelical fold so that when tightened, a natural-appearing antihelical fold is created. Nonabsorbable horizontal mattress sutures (4-0 Mersilene) are used. Keys to suture placement:

1. The suture must incorporate anterior perichondrium but not anterior dermis.
2. Three to four horizontal mattress sutures are placed *2 mm* apart (too close = tearing of cartilage, too far apart = buckling of cartilage).
3. The width of the mattress suture should be *1 cm* (superior-inferior axis).
4. Suture bites are *16 mm* (outer to inner bites).
5. The outer bite must be far enough away from the margin to prevent a vertical post-deformity.
6. The inferior suture is placed to allow retrodisplacement of the cauda helicis.

A pressure-type dressing is applied.

Furnas method (concha-mastoid suturing) Indicated for reducing excessive conchal cupping to close the gap between the concha and the mastoid prominence (Fig. 15.4). Markings: the floor

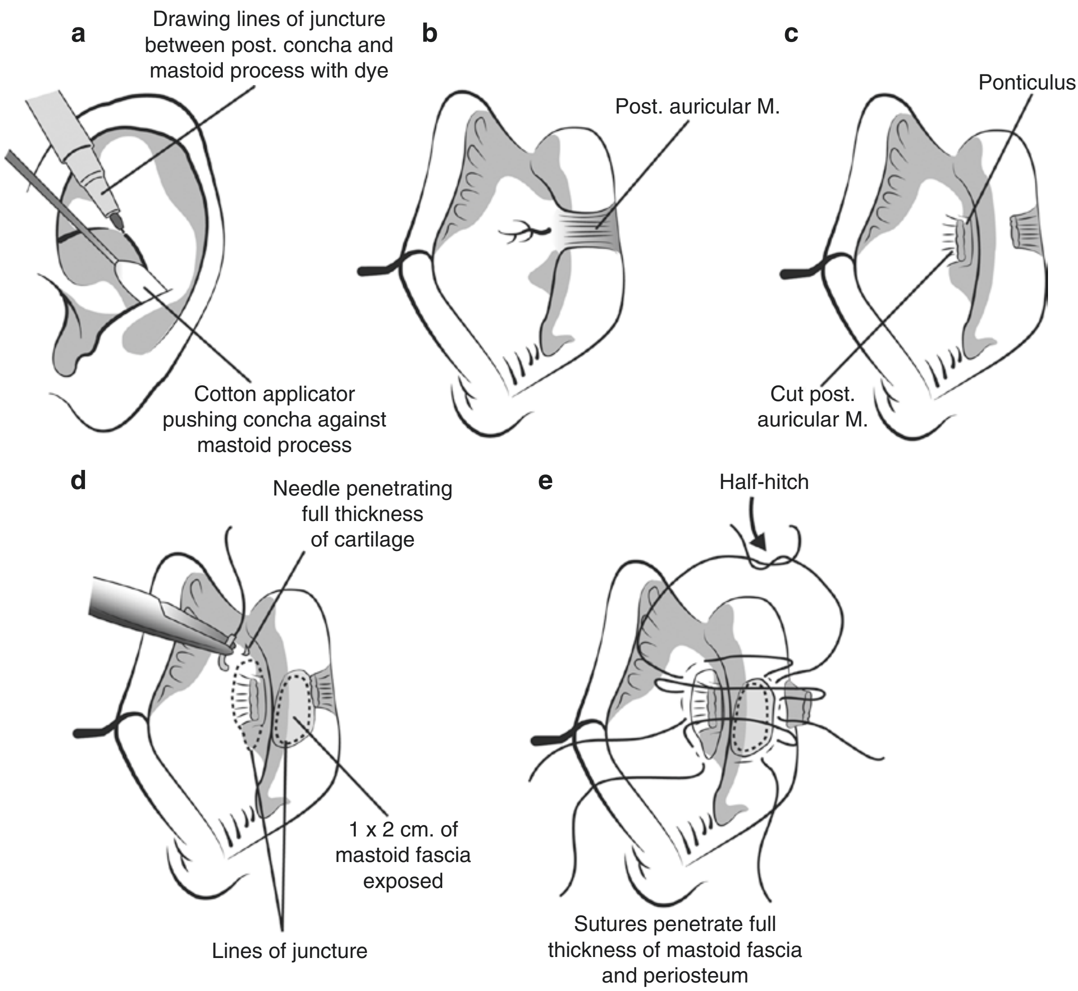


Fig. 15.4 Furnas method to reduce excessive conchal cupping (**a**) Line of juncture marked within conchal cup (**b**) post-auricular muscle (**c**) ponticulus (**d**) mastoid fascia exposed (**e**) suture placement

and posterior wall of the concha are pressed against the mastoid process, gradually reducing the depth of the concha to the point where the ear is no longer prominent. The line of juncture is marked within the conchal cup. An ellipse of postauricular skin is excised. A 1 × 2 cm area of deep fascia over the mastoid process is exposed. A horizontal mattress suture (4-0 clear monofilament nylon or Mersilene) is passed through the conchal cartilage posterior to the ponticulus, avoiding the skin on the anterior surface. The dye mark on the anterior surface of the concha serves as a guide for placement. The suture is passed through the mastoid fascia and into the periosteum. Three or more concha-mastoid sutures are required, and the final knots are not tied until after any corrections of the antihelix.

Inaccurate placement may displace the ear upward or downward or lead to inadequate correction (Fig.15.5). Sutures placed too far forward on the mastoid or too far back on the concha will decrease the diameter of the auditory canal.

Cartilage-Splitting Techniques

Antihelical fold creation 25 gauge needles and methylene blue tattooing are employed to demarcate the planned neoantihelix. A standard postauricular incision is made with planned elliptical skin excision. Wide posterior undermining is performed. A transcartilaginous incision is made 5 mm anterior to the tattoo marks at the apex of the neoantihelix. The incision then extends in a curvilinear fashion parallel to the helical rim through the cauda helicis. Triangular wedges are excised at the top and bottom of the incision. Anterior perichondrium is dissected free from the medial cartilage flap, and the exposed anterior cartilaginous surface is then burred with a diamond burr. The lateral cartilage is also beveled with the burr. The medial and lateral segments, which have been contoured to form the neoantihelical convexity, are overlapped. The skin is then closed and no cartilage sutures are required.

Correction of conchal hypertrophy (Davis technique) Markings: the height of the conchal wall that will remain is marked (8 mm of posterior conchal wall height), and the remainder of the conchal bowl to be excised is outlined (should be a “kidney bean” shape). An elliptical excision of postauricular skin is planned. The concha is exposed through the postauricular incision, and the cartilage is incised through the areas tattooed. A subperichondrial plane of dissection is developed and the cartilage removed, preserving 8 mm of posterior conchal wall. The ear is placed passively onto the mastoid surface, and the new projection is inspected. The underlying muscle and soft tissue are removed to the mastoid fascia. The ear is fixed in position with four mattress transfixion sutures of 3-0 silk, which go through-and-through and are tied over cotton dental rolls for compression. A cotton roll is placed in the ear canal to prevent stenosis. The postauricular incision is closed with absorbable suture, leaving a small opening inferiorly for drainage. The dressing is removed in 2 weeks.

Cartilage-Scoring Technique

Based on the principle that the natural tendency of cartilage is to bend in the direction opposite to the weakened side. The anterior surface of the cartilage is exposed through a 3.5 cm posterior parallel skin incision 1 cm from the helical rim (Fig. 15.6a). Very little undermining is done along the posterior surface of the cartilage. The antihelical cartilage is completely exposed up to its origin via a parallel transcartilaginous incision. The anterior skin and perichondrium are elevated through the transcartilaginous incision. Partial-thickness cartilage incisions are then made in a “fan-shape” such that the neoantihelix will curve both superiorly and posteriorly (Fig. 15.6b). In the middle third of the ear, the partial-thickness incisions converge at the upper portion of the conchal bowl. At this level a full-thickness incision is made to recapitulate the normal sharp demarcation here at the superior concha with careful attention paid toward maintaining proper conchal height. Plain gut suture can be used to maintain the position of the sharp fold of the concha but is otherwise not needed. If the lobule is prominent, the cauda helicis can be set back to the posterior aspect of the concha with a plain gut suture. Disruption of con-

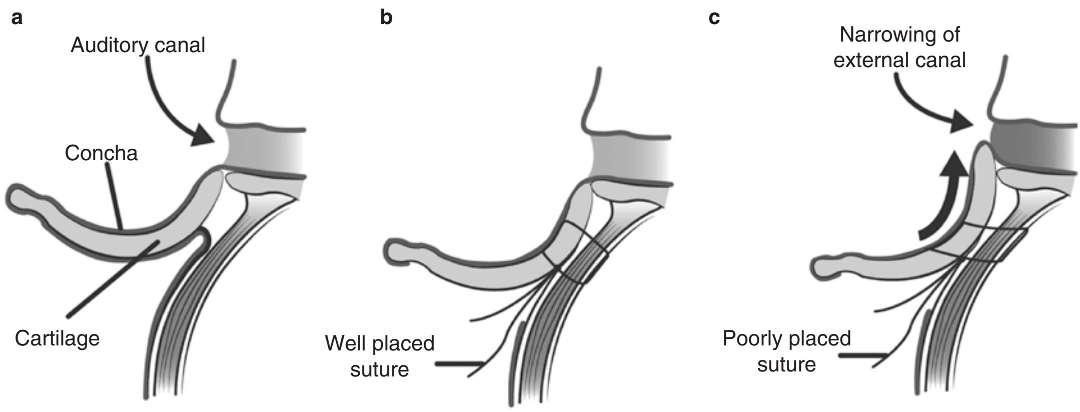


Fig. 15.5 Cross section showing (**a**) conchomastoid relationship prior to setback (**b**) proper suture placement (**c**) poorly placed sutures causing forward rotation of the concha and reduction in the diameter of the external auditory canal

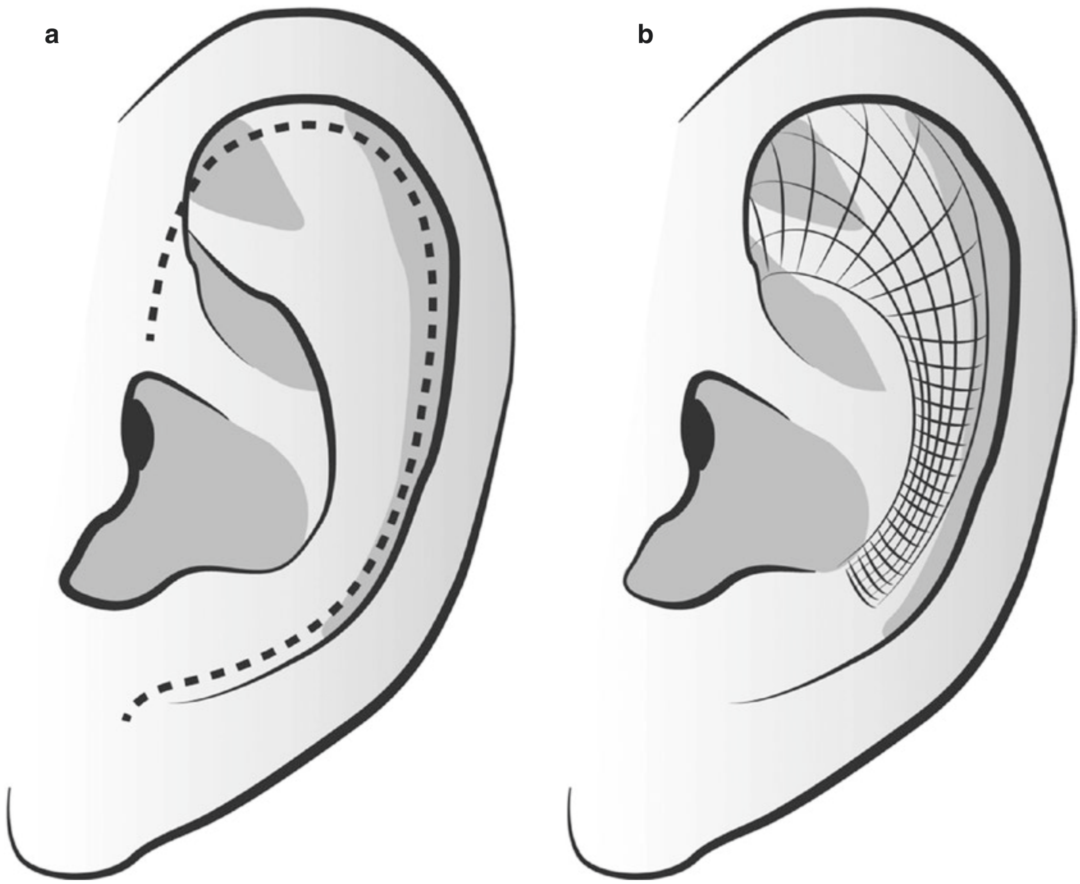


Fig. 15.6 (**a**) Location of cartilaginous incision between the helical rim and antihelix. The skin incision is performed posteriorly. (**b**) partial-thickness scoring incisions made in fan shape

tinuity with the upper helical margin with a small wedge excision prevents movement of the lobule from altering the position of the upper third of the auricle.

Correction of Lop Ear Deformity

Minor lop ear deformity can be treated with simple excision of the overhanging fold of cartilage.

In moderate and severe deformities, the upper third of the ear is deficient and tissue needs to be recruited for correction. In particular the superior crus and height of the upper third require reconstruction. To do this, a 180° rotation cartilage flap, created from the prominence of the present inferior antihelical cartilage, can be placed within a transcartilaginous incision placed at the level of the planned superior crus. The flap is inset to directly reconstruct the superior crus. The excess skin resulting from excision of the overhanging cartilage is sufficient to cover the increased vertical height after reconstruction of the superior crus. The superior crus, triangular and scaphoid fossae, and the height of the upper third are thus restored.

Correction of Protruding Earlobe

Prominent auricles are often accompanied by prominent lobules, and manipulations of the cartilaginous framework can sometimes accentuate lobular prominence. The etiology can be cartilaginous (flared cauda helicis) or soft tissue (overabundant fibrofatty tissues). The cauda helicis can be managed with sutures to the concha as described above or even with excision of the cauda helicis. The soft tissue of the lobule is managed with excisional techniques. Excisional techniques that place the incision close to the facial sulcus improve scar camouflage. A square-shaped excision near the lobular facial junction can be utilized to preserve the inferior lobule margin. In this technique, a square excision is performed, and the inferior flap is rotated into the geometric defect after deepithelialization of the inferior edge of the flap. Wedge excisions are a good option but will create a scar at the inferior margin of the lobule.

Iatrogenic Deformities and Complications

Complications after otoplasty are divided into early and late complications. Early complications are hematoma and infection, both of which may result in cartilage necrosis. Infections are typically pseudomonal or staphylococcal and appropriate antibiotic therapy should be instituted early. Serial debridement may become necessary. Hematoma can result in pressure necrosis of the cartilage and must be prevented with meticulous hemostasis, avoidance of anticoagulants, pressure dressings and drains, and prompt recognition and treatment. Unexpected pain is a heralding sign and warrants removal of dressings for inspection and treatment.

Late complications include suture extrusion and aesthetic deformities as well as keloidogenic or hypertrophic scarring. Suture extrusion results from excessive tension, poor placement, or infection. Hypertrophic scarring or keloid formation from the postauricular incision should be prevented with meticulous closure but can be treated with silastic gel and local injections of triamcinolone.

Iatrogenic Aesthetic Deformities

Deformities from inappropriate auricle/scalp relationship: undercorrection, re-protrusion, overcorrection deformity.

Deformities of the auricle: *telephone ear* (overcorrected middle third with prominent upper and lower thirds – aggressive conchal setback), *reverse telephone ear* (undercorrected middle third with prominent middle third – untreated conchal protrusion), *vertical post-deformity* (Mustarde sutures too close to auricular summit).

Auricular Reconstruction

Microtia

- *Grade 1*: the ear contains normal structural elements but is small, external auditory meatus usually present. Dysmorphic ear.
- *Grade 2*: the external ear is moderately anomalous. One or more anatomic subunits is deficient. Usually the lower half is normal. The auricle can be hook, S, or question mark shaped in appearance. Dysplastic ear.

- *Grade 3*: the external ear is a vertically oriented rudimentary structure with minimal cartilage; the lobule is present but anomalous. “Peanut-shaped” structure.
- *Grade 4*: anotia.

Auricular reconstruction with autogenous tissue is the method of choice for repair of severe deformities. Parents of children with microtia are often anxious to have the ear repaired early; however, it should be delayed until ages 6–8 or possibly later, when the rib cartilage is large enough and durable enough for sculpting. A contrasting philosophy applies to the treatment of grade 1 microtia, where conservative nonsurgical intervention (molding, taping) is undertaken as early as possible, while the ear cartilage is maximally pliable. Alloplastic frameworks (silicone, Medpor) have fallen out of favor. Newer techniques involving tissue engineering and prefabricated cartilaginous molds continue to advance, but sculpted autogenous rib cartilage is still the material of choice for surgical repair. The procedure is typically staged in order to have ideal conditions for healing and to minimize complications. An average of two to four stages is required depending on technique. Required steps regardless of approach include costal cartilage harvest, creation of the framework, skin coverage, lobule creation, elevation of the reconstructed auricle from the mastoid, and construction of a tragus. Stages are typically at least 3 months apart to allow for reestablishment of blood supply.

The classical microtia reconstruction operation is the four-stage procedure described by Brent. Stages include (1) cartilage implantation, (2) lobule transfer, (3) creation of postauricular sulcus, and (4) tragus construction.

In the first stage, rib cartilage is harvested from the contralateral chest generally to include ribs 6–8. Brent emphasized that preserving a minimal rim of the upper margin of the sixth rib cartilage can improve chest wall stability (Fig. 15.7). The eighth rib is used for the helical rim, so 8 or 9 cm of length is ideally obtained. The base plate of the framework is constructed from the synchondrosis between ribs 6 and 7. The cartilage is handled gently, kept in saline to prevent desiccation, and stripped of muscle and connective tissue while preserving the perichondrium. The cartilage is sculpted with scalpels and chisels in segments to recreate the framework (Fig. 15.8). Modifications are made in adult patients due to the calcified cartilage that often is carved as a single piece since the tissue will not bend without breaking.

A skin pocket is created at the site of the ear remnant as determined by preoperative measurements. Remnant cartilage is carefully removed and the new framework is implanted at the precise location for the new auricle. The vertical incision made for the pocket will be incorporated in the second stage during lobule transfer. Complete hemostasis within the pocket is achieved to prevent hematomas, and suction drains are placed under and behind the framework. Dressings to conform to the convolutions

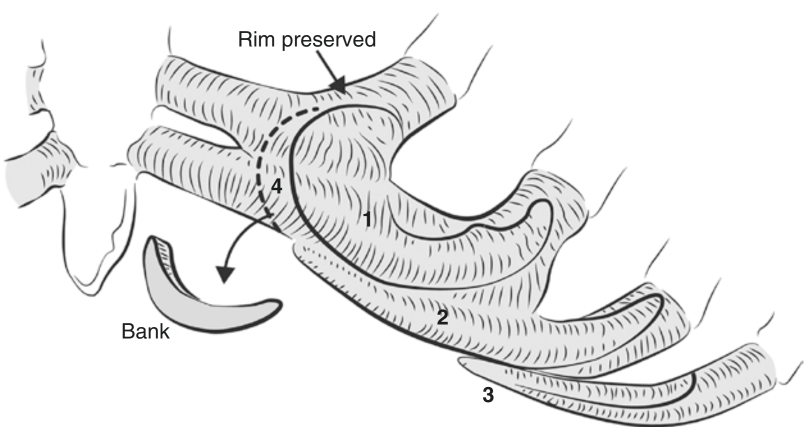


Fig. 15.7 Conchal cartilage ear reconstruction

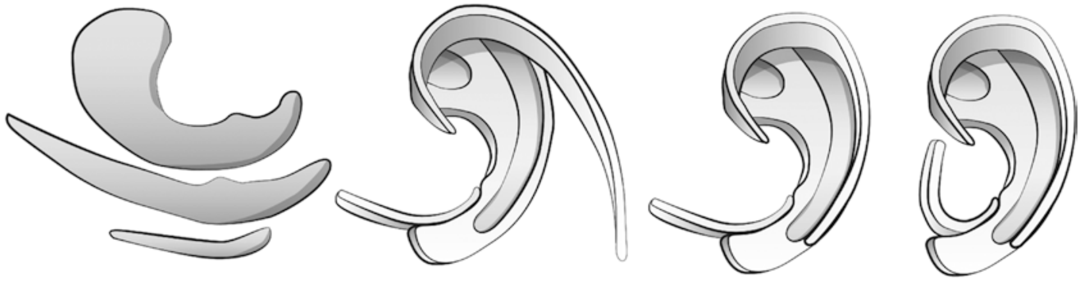


Fig. 15.8 Conchal cartilage carved to resemble normal ear framework

of the new auricle are placed along with a non-compressive, bulky head dressing. Often a piece of cartilage is banked in the skin pocket or at the rib harvest site to be used for ear projection when the framework is elevated.

The second stage is lobule transposition. A transposition flap is utilized to move the abnormally oriented lobule over the posteroinferior aspect of the cartilaginous framework. The tail of the framework is inserted into a pocket created in the lobule flap.

The third stage involves lifting the ear and creating a postauricular sulcus. An incision is made nearly circumferentially in the skin lateral to the framework, and the cartilage is elevated off the mastoid periosteum, preserving the connective tissue on the posterior surface. The banked cartilage is wedged into the postauricular sulcus to create projection, and this is covered with an occipitalis fascia turnover "book flap." A split-thickness skin graft is harvested from an inconspicuous location and secured in place and fastened with a bolster dressing left in place for 1 week.

The final stage of Brent's classical operation is tragus creation, which is created using an auricular composite graft from the contralateral "good" ear cymba concha. The graft is inserted into an incision just posterior to the planned neotragus. At this time debulking of the conchal bowl can help create bowl definition with the guidance of a facial nerve monitor. Brent has subsequently modified this technique to include rib cartilage tragus reconstruction during stage 1.

The two-stage technique is described by Nagata and contains numerous differences. This technique combines lobule transposition and tragus creation into the first stage. Sulcus elevation is the second stage. Patients are generally older (age 10) with adequate chest circumference (60 cm) to accommodate the larger amount of rib material harvested, which includes the ninth rib (antihelix, superior and inferior crura) and a piece of the fifth rib for conchal projection obtained at the second surgery. Given the extensive donor site harvest, emphasis is placed on preservation of posterior perichondrium for chest wall stability. The described technique typically uses ipsilateral rib cartilage and uses stainless steel wire sutures. A temporoparietal fascial flap is used at the second stage in place of the occipitalis fascia flap. There is an increased amount of manipulation and demand on the soft tissue flap in this technique, and the rate of tissue necrosis is reported to be as high as 14%.

Complications

Pneumothorax can result from pleural injury. Meticulous technique, intraoperative Valsalva, and postoperative chest X-ray are recommended. Hematoma, skin flap necrosis, infection, and graft exposure may lead to loss of the graft if intervention is not timely. Treatment of infection must cover pseudomonas and staphylococcal strains and serial debridement may be necessary. Scarring, resorption, and malposition can also occur.

Auricular Prostheses

The prosthesis is attached to a transcutaneous osseointegrated implant and requires replacement every 2–5 years for the life of the patient. The skin/implant interface is prone to irritation and requires meticulous hygiene. The use of a prosthesis will preclude future autogenous reconstruction since soft tissue and skin are removed. In general, autologous reconstruction is preferred in children when possible, whereas adult reconstruction is more suitable for prosthetic reconstruction.

Indications

- Failed autogenous reconstruction: Scarring, previously used TPF flap, propensity for hypertrophic or keloidogenic scarring, hesitance from patient after failed attempt.
- Severe soft tissue and/or skeletal hypoplasia: Skeletal or soft tissue deficits, common in hemifacial microsomia or other congenital maxillofacial anomalies, may limit the ability to implant a cartilaginous framework.
- Low or unfavorable hairline: Hair over the superior aspect can be treated with electrolysis, laser ablation, and excision/skin graft; however a prosthesis may be a superior option.
- Posttraumatic or postablative auricular defects: In this setting there is often preservation of the tragus, which allows the anterior border of the prosthesis to be hidden. Patients who have undergone resection for oncologic indications and treated with radiotherapy, which further complicates or precludes autogenous reconstruction.

External Auditory Canal Atresia

EAC atresia results from failure of canalization of the epithelial plug portion of the first branchial cleft. Persistence of the tympanic ring results in a bony atretic plate at the level of the tympanic membrane. Ossicular malformations may also exist, and patients typically have a conductive hearing loss or mixed hearing loss. Hearing assessment and amplification, typically with bone-anchored hearing aids (BAHA) should be instituted at a young age to facilitate language outcomes.

Realistic goals and expectations should be openly discussed with the patient and family. Surgical intervention on the first ear generally occurs around age 4–5 years with the goal to create a safe (preventing cholesteatoma or otitis media) and hearing ear. If microtia is present, microtia repair is typically performed prior to atresia repair.

The status of the middle ear is important for good surgical outcome. Atresia that is associated with microtia implies a malformation of the middle ear, mastoid, and VII nerve because the auricle is formed relatively early in development. Atresia in the setting of a normal auricle occurred later in development and is more likely to have favorable middle ear and mastoid features. CT scanning helps define the favorability of atresia repair and notes the relationship of the bony atretic plate to the facial nerve.

Surgical technique involves a postauricular incision, widening of the stenotic meatus by removing fibrous tissue, and deepithelializing the tympanic membrane. The posterior, superior, and inferior bony canal is widened until the first mastoid cells were encountered. The anterior canal is widened in cases where there was an anterior bony overhang. The bony canal is lined with split-thickness skin grafts. Meatoplasty is performed, and split-thickness skin grafts are grafted onto the margins of the meatoplasty to cover the lateral part of the ear canal. Ossicular reconstruction with a prosthesis is performed. The middle ear and ear canal are packed with absorbable packing. The EAC is debrided in 2–4 weeks. Once healing is complete, a postoperative audiogram should be performed. Patients may still require hearing amplification even after successful surgery.

Questions

1. The typical scapha-conchal angle is:
   (a) 15°
   (b) 30°
   (c) 90°
   (d) 120°
2. Which of the following is *false* regarding microtia repair?
   (a) Occipitalis or temporoparietal fascia flaps are used for coverage when the framework is elevated from the mastoid.
   (b) The Brent technique first stage imposes a higher soft tissue demand and thus has a higher rate of tissue necrosis.
   (c) In situ preservation of a superior margin of cartilage and as much posterior perichondrium as possible allows for reduced donor site morbidity.
   (d) Stainless steel sutures of the Nagata technique have been associated with suture extrusion and framework deformation.
3. Telephone ear deformity can best be described as:
   (a) Overcorrection of the middle third of the ear with a prominent helix and lobule
   (b) Pointed ear characterized by an abnormal folding of the skin and cartilage of the pinna
   (c) Underdevelopment of the helical rim
   (d) Thickening and loss of normal auricular landmarks caused by repetitive trauma
4. The Davis method for correction of auricular deformity is best selected to correct:
   (a) External auditory canal atresia
   (b) Hypertrophic posterior wall of the conchal bowl
   (c) Absence of the antihelical rim
   (d) Microtia
5. In the Mustarde suture otoplasty technique, the appropriate distance between consecutive mattress sutures is:
   (a) 1 mm
   (b) 2 mm
   (c) 3 mm
   (d) 4 mm
6. Which of the following is true regarding otoplasty techniques?
   (a) The Furnas technique is preferred because narrowing of the EAC is unlikely.
   (b) The Mustarde technique recreates the scapha-conchal angle.
   (c) Lop ear deformity involves excessive folding of the superior crura of the antihelix.
   (d) The inner and outer bites of the Mustarde suture are placed 12 mm apart.
7. The normal auriculocephalic angle is:
   (a) 15
   (b) 30
   (c) 50
   (d) 90
8. Which of the following is *false* regarding auricular reconstruction?
   (a) The use of osseointegrated auricular prosthesis precludes future autogenous reconstruction.
   (b) A prosthesis is indicated when soft tissues or skeletal are deficient in the temporal region.
   (c) A low-set hairline may indicate the use of a prosthesis.
   (d) Prosthesis camouflage is improved when the native tragus is absent.
9. A surgeon performs an otoplasty. Many months postoperatively the patient develops canal obstruction from cerumen impaction with associated conductive hearing loss requiring intermittent cerumen removal. Which error likely occurred?
   (a) Mustarde sutures were placed 4 mm apart.
   (b) Wedge excision of the lobule was excessive.
   (c) The conchal height was not preserved in the Davis technique.
   (d) Furnas sutures were placed anteriorly over the mastoid.
10. A 60-year-old patient presents for aging face surgery. On facial analysis, his upper third is characterized by good brow position with eyelid redundancy and moderate lid ptosis. His lower lids are normal. He has a mild tear trough deformity and prominent jowls. The

correct superior position of the helical rim is best described as:
(a) The lateral brow
(b) The upper eyelid
(c) The lateral canthus
(d) The lower eyelid

11. The degree of inclination of the auricle relative to the nasal dorsum is:
(a) 0 (parallel)
(b) 15
(c) 30
(d) 45

12. A patient calls the clinic to report that 2 days after undergoing otoplasty for prominent ears, she is having worsening pain of the operated ear that is severe and uncontrolled with routine postoperative pain medications. A postsurgical compression dressing with a headband was placed and has not been removed. Your advice to the patient is:
(a) Return to clinic urgently for dressing removal and inspection for a hematoma.
(b) Continue with postoperative pain medications and reassurance that the pain will improve in the upcoming days as the swelling resolves.
(c) Remove the dressing at home and start using antibiotic ear drops for likely otitis externa caused by trauma to the ear canal during the procedure.
(d) Maintain the head dressing since the pain is likely from overly tightened sutures that will loosen with time.

13. After a surgeon completes tying his Mustarde sutures during an otoplasty operation, he notices an aesthetic imbalance with relative protrusion of the lobule. The soft tissues of the lobule do not appear excessive. What can be done to correct this?
(a) Wedge excision
(b) Square excision with rotational flap
(c) Excision of cauda helicis
(d) Suture cauda helicis to posterior concha
(e) A or B
(f) C or D

14. What is the normal depth of the conchal bowl?
(a) 5 mm
(b) 6 mm
(c) 7 mm
(d) 8 mm

15. An alternative to the Hillock theory of development is challenging our traditional understanding of auricular development. In this theory, which structure derives from the first branchial arch?
(a) Tragus
(b) Helical root
(c) Cauda helicis
(d) Antitragus

16. In the two-stage technique for microtia repair described by Nagata, which of the following procedure(s) is(are) incorporated into the primary cartilage implantation stage?
(a) Tragus reconstruction
(b) Lobule transposition
(c) Auricular elevation
(d) A and B
(e) B and C

17. A novice otoplasty surgeon does not recognize the contribution of the protruding concha when planning his surgery for a prominent ear deformity. He proceeds with a Mustarde technique for creation of a neo-antihelical fold. The patient is unhappy postoperatively because of an aesthetic deformity. Which deformity is this likely to be?
(a) Telephone ear deformity
(b) Reverse telephone ear deformity
(c) Satyr ear deformity
(d) Vertical post-deformity

18. The lop ear deformity is comprised of the following anomalies *except*:
(a) Folding of helical rim
(b) Loss of vertical height
(c) Deficient inferior crura of the antihelix
(d) Deficient scapha

19. Chest wall stabilization following costal cartilage harvest for microtia repair in the Brent technique can be improved by:
(a) Individual harvest of each rib
(b) Preserving the superior margin of the sixth rib
(c) Limiting lateral extent of the harvest
(d) Partial-thickness harvest of floating rib

20. Which of the following is *false* regarding otoplasty for the prominent ear?

(a) The prominent lobule and prominent auricle are often associated abnormalities.
(b) Sutures should incorporate anterior perichondrium.
(c) Suture techniques have demonstrated unequivocal long-term inferiority to cartilage cutting techniques.
(d) Efficacy and safety of otoplasty techniques have been demonstrated in patients younger than 4 years old.

21. A regional field block is applied using local anesthetic for an auricular procedure. The patient still feels pain at the posterior EAC. Which nerve was likely not sufficiently blocked?
    (a) V3
    (b) Lesser occipital
    (c) IX
    (d) X
22. A 3-year-old child presents for otoplasty consultation. You tell the parents that his ear has reached what percentage of adult size at this time?
    (a) 50%
    (b) 70%
    (c) 85%
    (d) 95%
23. The antihelical fold defines the:
    (a) Concho-mastoid angle
    (b) Auriculocephalic angle
    (c) Scapha-conchal angle
    (d) Angle of inclination
24. The Stahl ear deformity results from:
    (a) Deficiency of antihelical superior crus
    (b) Inappropriate placement of preauricular incisions
    (c) Presence of an abnormal antihelical posterior crus
    (d) Vertical height deficiency of the upper third of the auricle
25. An 8-year-old child undergoes microtia repair with autologous costal cartilage graft. His hospital course after stage 1 is uneventful and he is discharged home. Ten days later he develops persistent drainage from the inferior aspect of the incision. Among the choices below, what is the most appropriate initial antibiotic therapy for this problem?
    (a) Clindamycin
    (b) Amoxicillin/clavulanate
    (c) Cephalexin + ciprofloxacin
    (d) Trimethoprim/sulfamethoxazole

Answers

1. (c)
2. (b)
3. (a)
4. (b)
5. (b)
6. (b)
7. (b)
8. (d)
9. (d)
10. (a)
11. (b)
12. (a)
13. (f)
14. (d)
15. (a)
16. (d)
17. (b)
18. (c)
19. (b)
20. (c)
21. (d)
22. (c)
23. (c)
24. (c)
25. (c)

Additional Resources

1. Azuara E. Aesthetic otoplasty with remodeling of the antihelix for the correction of the prominent ear: criteria and personal technique. Arch Facial Plast Surg. 2000;2(1):57–61.
2. Brent B. Technical advances in ear reconstruction with autologous rib cartilage grafts: personal experience with 1200 cases. Plast Reconstr Surg. 1999;104(2):319–34.
3. Caouett-Laberge L, Guay N, Bortoluzzi P, Belleville C. Otoplasty: anterior scoring technique and results in 500 cases. Plast Reconstr Surg. 2000;105(2):504–15.

4. Elsahy NI. Techniques for correction of lop ear. Plast Reconstr Surg. 1990;85(4):615–20.
5. Furnas DW. Correction of prominent ears by concha-mastoid sutures. Plast Reconstr Surg. 1968;42(3):189–93.
6. 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. 2004;114(5):1042–54. 89
7. Owsley TG. Otoplastic surgery for the protruding ear. Atlas Oral Maxillofac Surg Clin North Am. 2004;12(1):131–9.
8. Posnick JC, al-Qattan MM, Whitaker LA. Assessment of the preferred vertical position of the ear. Plast Reconstr Surg. 1993;91(7):1198–207.
9. Thorne CH, Brecht LE, Bradley JP. Auricular reconstruction: indications for autogenous and prosthetic techniques. Plast Reconstr Surg. 2001;107(5):1241–52.
10. Cheney ML, Varvares M, Nadol JB. The temporoparietal fascial flap in head and neck reconstruction. Arch Otolaryngol Head Neck Surg. 1993;19:618.
11. Cremers WR, Smeets JH. Acquired atresia of the external auditory canal: surgical treatment and results. Arch Otolaryngol Head Neck Surg. 1993;119(2):162–4.
12. Hochman M, Thomas JR. Reduction of hypertrophic earlobes. Laryngoscope. 1992;102(7):829–8.

16 Vascular Anomalies

Matthew Keller and Tristan B. Klosterman

Vascular anomalies or lesions consist of a variety of pathologies that affect the head and neck. They are relatively common and are not limited to pediatric population. Vascular lesions can be divided into *tumors* and *malformations* which are differentiated by clinical activities, course, histology, and treatment. This classification scheme is a product of the International Society for the Study of Vascular Anomalies (ISSVA) and helps differentiate lesions and to replace obsolete terms such *port-wine stains*, *stork bites*, *lymphangiomas*, *and cystic hygromas.*

Vascular tumors are true proliferating tumors, while malformations are anomalous growths (Fig. 16.1). Vascular tumors consist of infantile hemangiomas, congenital hemangiomas (rapidly involuting and non-involuting types), as well as tufted angiomas/kaposiform hemangioendotheliomas. Vascular malformations include venous, capillary, lymphatic, arteriovenous, and combined lesions (Table 16.1).

Vascular Tumors

Vascular tumors represent a unique subset of lesions that stem from an abnormal proliferation of endothelial cells and irregular vessel formation. These tumors exhibit pathologic cellular hyperplasia due to dysregulated angiogenesis. Infantile hemangiomas represent the most common of these tumors, but other vascular tumors can present including congenital hemangiomas, tufted angioma, hemangiopericytoma, pyogenic granuloma, kaposiform hemangioendothelioma (KHE), and angiosarcomas.

Infantile Hemangioma

Overview

Infantile hemangiomas affect up to 10% of children and represent both the most common benign pediatric tumor and pediatric head and neck neoplasm. Approximately 60% of infantile hemangiomas occur in the head and neck. They commonly occur in Caucasians, females (3:1 female: male), and premature infants (23%). The majority of these tumors occur sporadically. Most are solitary but multiple lesions can occur in up to 20%. These lesions are apparent at birth or appear within the first 2 weeks of life. One-third of these tumors present as a cutaneous mark (i.e., blanched area of vasoconstriction, telangiectatic

M. Keller
Otolaryngology/Head and Neck Surgery, Naval Medical Center San Diego, San Diego, CA, USA

T. B. Klosterman (✉)
Otolaryngology, Highland Hospital, Oakland, CA, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_16

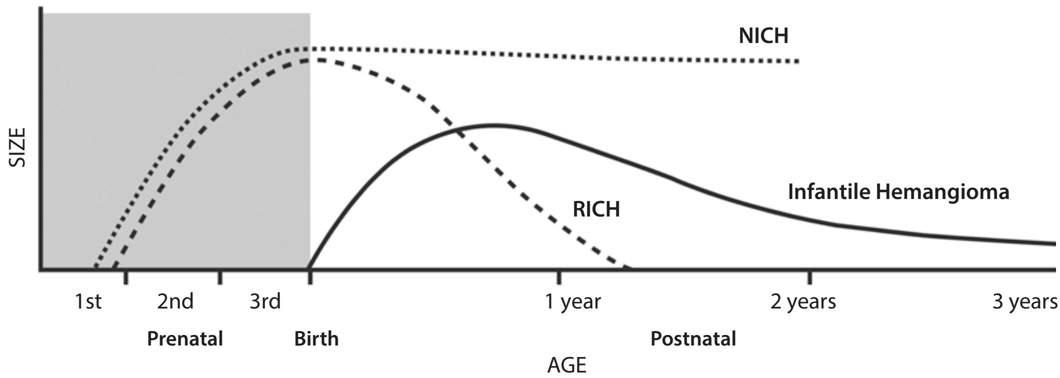


Fig. 16.1 Congenital and infantile hemangiomas: patterns of growth and involution [1]

Table 16.1 Characteristics of vascular anomalies

Type	Timing	Appearance	MRI characteristics	Therapy
Vascular tumors				
Infantile hemangioma	First weeks of life	*Proliferating* "Strawberry-like"	Well defined, lobulated	Propranolol
	Rapidly growing	Pulsatile	No perilesional edema	Laser therapy
	Regress by 7–10 years	Warm	Flow voids	Surgery
		Involuting	Homogenous enhancement	
		Grayish dark red Central ulceration		
Congenital hemangioma	Fully present at birth	Blue-red to purple	*Involuting phase*	
RICH (*rapidly involuting congenital hemangioma*)	Involute	Warm Central ulceration	Fatty replacement (high T1 signal)	*RICH*—no treatment
NICH (*non-involuting congenital hemangioma*)	Do not involute	Lighter colored raised plaques		*NICH*—embolization followed by surgery
Tufted angioma/kaposiform hemangioendothelioma	Variable	Hypertrichosis		Surgery Laser therapy
Vascular malformations				
Low flow				
Venous malformation	Present at birth (not always evident)	Compressible, blue, soft, nonpulsatile	Infiltrative, lobulated, fluid levels, phleboliths, no flow voids, no contrast enhancement	Sclerotherapy, laser Surgery
Lymphatic malformation	Grow proportionally (do not regress)	Soft, smooth, rubbery	Macrocystic (>2 cm) Microcystic (<2 cm)	Sclerotherapy Surgical debulking
Capillary malformation		Red skin discoloration	Thickened skin	Laser therapy Surgical debulking
High flow				
Arteriovenous malformation		Red pulsatile warm mass with thrill		Embolization, surgery

Modified from Wassef et al. [2]

macule, ecchymotic spot) which later undergo postnatal proliferation.

Pathology

Infantile hemangiomas arise as abnormal *endothelial cell proliferation* from hemangioma-promoting stem cells. Some studies suggest that these stem cells are “metastatic” maternal placenta cells in high-flow areas to the fetus. The hemangioma endothelial cells positively express glucose transporter protein-1 (*GLUT-1*), unlike the surrounding normal vascular endothelium. This serves as a useful marker when the diagnosis is in question. Clinically, these lesions are designated as *superficial*, *deep*, *or compound lesions*. Superficial lesions reside in the papillary dermis and are typically bright red, noncompressible, well demarcated, and elevated from the surrounding skin. Deep lesions are soft, ill-defined masses of the reticular dermis and subcutaneous tissue with an overlying bluish hue. Combined lesions exhibit both characteristics.

Classification

Infantile hemangiomas are divided into two major categories: *focal and segmental*. They most commonly present as *focal* tumors, localized to one cutaneous area. *Segmental* hemangiomas are less common and occur as plaque-like lesions in a geographic distribution over a specific cutaneous/dermatological segments’ region (e.g., CNV1, V2, frontonasal, V3 “beard distribution”). Sixty percent of patients with beard distribution hemangiomas have subglottic disease. *Disseminated* lesions (greater than five) can be associated with visceral (commonly liver and bowel) hemangiomas; thus those patients need ultrasound screening of the abdomen.

Characteristics

Infantile hemangiomas have a distinct natural history of proliferation followed by involution. The *proliferation phase* is characterized by a rapid growth phase usually beginning at 1–2 weeks and up through the first 8–10 *months of life* and complete by 1 year. This increase in angiogenesis is the result of upregulation of vascular endothelial growth factor (VEGF), matrix metalloproteinase (MMP)-2, and basic fibroblast growth factor (FGF). This further differentiates hemangiomas from vascular malformations which do not express these markers. The *involution phase* is frequently heralded by a grayish hue in the center of the lesion. Involution is the result of *apoptosis of the endothelial cells* which results in increased fibrosis and stromal cell formation. This phase has a highly variable and unpredictable time course, lasting anywhere from 2 to 10 years. In the past, regression stastistics were described using a standard age and percent (50% of children by the age of 5, 70% by age 7, and 90% by age 9) however, this is oversimplified as significant variability exists. Longer involution times are associated with a higher incidence of permanent sequela such as scarring, residual subcutaneous fibrofatty tissue, telangiectasia, or atrophy of the skin.

Complications

Many infantile hemangiomas resolve without functional or cosmetic consequences but up to 50% will leave residual tissue benefiting from surgical treatment. *Ulceration* is the most commonly noted complication and seen more often in segmented lesions and can occur in up to 5% of all lesions. Ulceration typically presents during the period of rapid proliferation and is prone to develop in areas of friction such as the nape of neck or anogenital area. Ulceration is typically painful and is associated with increased risk of infection, bleeding, and scarring. When present, topical wound care should be initiated with emollients and occlusive dressings. Depending on the extent and ability to control, excision may be immediately beneficial.

Functional issues can also arise depending upon the anatomical location of the lesion. *Visual impairment* can become an issue for expanding periorbital hemangiomas. Segmental hemangiomas in the cranial nerve V3 distribution (“beard distribution”) have a greater likelihood of a coexistent airway lesion (30–50%). Patients with *airway hemangiomas* present between the ages of 6 and 12 weeks with progressive stridor and hoarseness. Fifty percent of patients with airway hemangiomas have an associated cutaneous hemangioma. Classically, the term subglottic hemangioma has been used to describe airway lesions, but airway hemangiomas are not limited to the subglottis, and therefore the term should be avoided. Timely diagnosis and treatment are critical for these patients due to the ability of infantile hemangiomas to rapidly progress in size, which could result in respiratory failure. If suspected, the patient should undergo endoscopy. These lesions should not be biopsied, but the diagnosis should be confirmed with CT and MRI.

PHACES syndrome (posterior fossa malformations, hemangiomas of the cervicofacial region, arterial anomalies, cardiac anomalies, eye anomalies, sternal defects) should be considered in any infant with a large (>5 cm) or segmental hemangioma of the head and neck [3]. This condition has a marked female predominance (90%). Dandy-Walker malformation is the most common brain anomaly, but a number of other anomalies may be present. Cardiac anomalies are also common and can include coarctation of the aorta and congenital heart defects. Vascular anomalies can include aneurysms, anomalous branching of major cerebral and cervical vasculature, hypoplastic vessels, and stenosis. Eye findings can include optic nerve hypoplasia, microphthalmia, cataracts, and Horner’s syndrome. Hearing loss has been reported with PHACES, related to ipsilateral intracranial hemangiomas involving auditory structures. Infants with suspected PHACES should undergo MRI imaging of the head, echocardiogram, ophthalmology examination, and audiometric testing during infancy.

Rarely, a patient presents *with diffuse hemangiomatosis* (>5 cutaneous lesions), visceral hemangiomas, congestive heart failure, hepatosplenomegaly, and anemia. This requires aggressive medical intervention to minimize morbidity and mortality, which can be significant in these patients.

Treatment

Treatment for infantile hemangioma depends on multiple factors including location, size, age of the patient, and growth phase. Strong indications for treatment include hemorrhage, ulceration, functional disability, airway impairment, or congestive heart failure. Intervention is recommended in nearly all hemangiomas of the head and neck.

Observation

Up to 50% of hemangiomas involute without cosmetic or functional sequela. Unfortunately, the remaining lesions can cause complications such as airway obstruction, visual obstruction, or tissue distortion and loss. If an observation strategy is selected, the physician must follow the patient until the patient/family is satisfied with the end cosmetic and functional result.

Medical

Early medical therapy is now routinely recommended for the treatment of hemangiomas, especially those which could result in cosmetic disfigurement (i.e., nasal tip, eyelid). Other indications for treatment include (1) ulceration, (2) functional compromise, (3) visceral hemangiomas, and (4) segmental hemangiomas.

Propranolol has significantly changed the management of infantile hemangiomas since its serendipitous discovery in 2008 [4]. The mechanism of action of beta-blockers on hemangiomas is poorly understood. However, the inhibition of the VEGF signaling pathway interrupts angiogenesis, decreases endothelial cell migration, and may induce apoptosis. Multiple studies have shown equivocal data on apoptosis induction though B2 adrenergic receptors have been found on proliferation endothelial cells and blockade seems to reduce differentiation of local stem cells [5].

Effective dosing starts with 0.5–1 mg/kg/day in two or three divided doses which is increased by 0.5 mg/kg/day until a target dose of *2–3 mg/kg/day* is achieved. An EKG must be obtained prior to initiating therapy. It is important to monitor HR/BP for 2 hours after initial dose and after every dose increase. Hospitalization is reserved for patients under 8 weeks of age, preterm infants less than 48 weeks, and those with pulmonary or cardiac risk factors. Patients should be screened for contraindications such as cardiogenic shock, sinus bradycardia, hypotension, >first-degree heart block, congestive heart failure, asthma, PHACES (relative contraindication due to higher stroke risk), and diabetes (relative contraindication due to blunting of the hypoglycemic response) [3]. *Hemangeol* is an FDA-approved solution of propranolol hydrochloride that has seen widespread usage for infants and differs as it contains no alcohol, sugar, or parabens. *Topical beta-blockers* have been used with success for small, focal, and superficial lesions. Timolol in a 0.5% gel-forming solution applied twice a day for up to 3 months or more is the treatment of choice.

Corticosteroids had been the mainstay medical treatment for hemangiomas prior to the discovery of propranolol. Corticosteroids work by inhibiting angiogenesis through the downregulation of VEGF, MMP-1, IL-6, and other cytokines. They can be administered intralesionally for small, localized lesions or systemically for large or life-threatening hemangiomas. In patients who cannot tolerate beta-blockade, steroids remain a viable option. Intralesional injections are typically administered in 6-week dosing intervals throughout the proliferative phase. Corticosteroids have a ~85% response rate, with changes seen during the first week of therapy. Notably, if steroid usage is discontinued during the proliferative phase, rebound growth can occur in up to 30% of patients. Exact systemic dosing of prednisolone is controversial, but *2–3 mg/kg* appears to be optimal. Therefore, patients should be treated through 10–11 months of age and informed of potential risks including cushingoid facies, irritability, and temporary growth retardation. Care should be taken with periorbital injections as blindness is a potential risk.

Vincristine is a chemotherapeutic agent that has been used in refractory cases. It works by inhibiting microtubule formation. It has increasingly been used in *Kasabach-Merritt syndrome*. Kasabach-Merritt is a rare, life-threatening platelet-trapping, consumptive coagulopathy caused by vascular tumor trapping and destruction of platelets. Potential adverse consequences include peripheral neuropathy and electrolyte imbalances. Interferon alpha has also been used in the past due to its ability to inhibit angiogenesis but met with high failure rates and side effects including spastic diplegia.

Other therapies include topical imiquimod 5%, rapamycin, and VEGF inhibitors, but their use remains limited due to the significant efficacy of propranolol.

Lasers

Lasers remain a popular and effective tool in the treatment of vascular tumors. The yellow light *flashlamp-pumped pulsed dye laser* (*PDL*) at a 585 nm wavelength is the most common laser used in the treatment of the superficial components of IH (infantile hemangioma). The PDL is also effective for ulcerated hemangiomas, cosmetically sensitive (nasal tip) areas, hemangiomas with functional impact (periorbital), flat hemangiomas, or those with residual vascular pigmentation. Ulcerated hemangiomas may also respond to PDL with noted improvement in pain, rapid re-epithelialization, and accelerated involution. However, reservation should be taken in rapidly proliferating segmental hemangiomas as new ulceration is possible with aggressive PDL use. Treatment is typically performed every 4–6 weeks until involution. Recent studies have also shown benefit from the combination of PDL and propranolol. In general, PDL for deeper hemangioma components is of limited value due to the limited depth of penetration of the laser. These lesions may have continued proliferative growth of the deeper more subcutaneous components despite treatment with PDL. Nd:YAG has a greater depth of penetration than the FPDL and can be more successful in treating the deeper components. The laser emits at 1064 nm and can penetrate up to 4 mm, making it useful for aerodigestive lesions and subcutaneous/compound hemangiomas. KTP laser is absorbed at 532 nm and penetrates more deeply than the PDL but less than Nd:YAG. The CO_2 laser can be used for atrophic scarring, residual airway hemangioma, or those unresponsive to other treatments.

Surgery

Surgery is performed for correction of facial deformity, ulceration, obstruction (visual/airway), scarring, residual fibrofatty tissue, and unresponsiveness to medical treatment. Some areas (nose, lip, orbit) of the face have the potential for a severe cosmetic and/or functional deformity if left untreated. For these sensitive sites, the threshold for surgical intervention is therefore lower. The rate of involution can influence the ultimate cosmetic result. Late involution is associated with more residual findings such as epidermal atrophy, hypopigmentation, telangiectasias, and fibrofatty residual tissue. IHs are solid lesions with distinct surgical planes and minimal feeding vessels (unlike vascular malformations). Excision of facial tumors should be performed in a conservative fashion with a preference for primary closure. Often, they may serve as their own tissue expanders, facilitating closure. Serial excision is an option for large hemangiomas with an overall goal to minimize resultant scarring.

Congenital Hemangioma

Congenital hemangiomas represent a unique subset of vascular tumors which have distinct clinical entities from infantile hemangiomas. Unlike infantile hemangiomas, these lesions are *fully grown at birth* and do not demonstrate rapid neonatal proliferation. These lesions are *GLUT-1 negative*, and usually unifocal, but otherwise share common features (size, appearance, histology, and radiology) with infantile hemangiomas. These lesions are classified into two categories: *rapidly involuting congenital hemangiomas* (*RICH*) and *non-involuting congenital hemangiomas* (*NICH*).

Rapidly Involuting Congenital Hemangiomas (RICH)

These lesions are present to a full degree at birth and *involute* by 6–18 months old. They are GLUT-1 negative and stain positive for Wilms' tumor (WT1), a transcription factor activated during angiogenesis. On palpation, lesions are often warmer than surrounding skin with central ulceration present at birth or shortly after. They are high-flow lesions that have the potential to precipitate high-output cardiac failure in advanced cases. Involution starts days to weeks after birth, and in a small subset of patients, RICH involution is incomplete leaving a vascular plaque with coarse telangiectasias and bluish-white border. Many leave significant atrophy behind as they involute. Because RICH are self-limiting, treatment is not needed unless there are complications or significant residual tissue.

Non-involuting Congenital Hemangiomas (NICH)

These lesions are also present to a full degree at birth but *do not involute*; instead they enlarge proportionally with the child's growth. They are also GLUT-1 negative and stain positive for Wilms' tumor (WT1). On exam, they are well-circumscribed round to oval, plaque-like, or bossed soft tissue masses with color varying from pink to blue-red or purple. These lesions are also high flow which can be identified on ultrasound and radiology. There are two morphologic subtypes: *patch type* and *nodule/plaque type*. Treatment consists of surgical excision during the preschool age with possible preoperative embolization in selected cases.

Tufted Angioma (TA)

These are uncommon benign vascular tumors that are sometimes present at birth but can also develop later. They present as an infiltrated, firm, dusky red to violaceous plaque or nodule with overlying *hypertrichosis* (a key sign). Histology shows tightly packed capillary "vascular tufts," and they are GLUT-1 negative. These lesions are also often associated with Kasabach-Merritt syndrome.

Kaposiform Hemangioendothelioma (KHE)

These lesions are a continuation of a clinical spectrum increasing in disease severity from TA to kaposiform hemangioendothelioma (KHE). Over 50% of KHE are present before age 1 year though variable growth and regression patterns are common. Physical exam demonstrates a raised subcutaneous mass with purpuric bruised appearance. Other findings are mottled red to purple macules/patches/plaques, annular patterns, tenderness, hyperhidrosis, and hypertrichosis. KHE is very aggressive and invades the muscle, soft tissue, and bone. Histology shows hypercellular capillaries in reticular dermis, sometimes with dilated lymphatic vessels. Deeper spindle cells are associated with KHE, along with positivity for D2-40 (lymphatic marker), and like TA they are GLUT-1 negative. Visceral involvement is highly associated with Kasabach-Merritt phenomenon (platelet sequestration with severe thrombocytopenia, microangiopathic hemolytic anemia, and consumptive coagulopathy). This phenomenon is most often seen with rapidly enlarging lesions.

Treatment involves drug combinations/chemotherapy (sirolimus, vincristine) or surgery. Blood products should be avoided due to risk of exacerbating Kasabach-Merritt phenomenon. For superficial lesions, the flashlamp-pumped pulsed dye laser (585 nm) is most well proven. In the treatment of lesions with subcutaneous components, the Nd:YAG laser is the treatment of choice.

Vascular Malformations

These differ from hemangiomas in that vascular malformations are present at birth, grow proportionally with the child (though may enlarge later due to hormonal fluctuations and infections), and do not involute. Unlike vascular tumors, there is minimal to no endothelial turnover. They represent aberrant blood vessels due to disordered vasculogenesis which often enlarge by hypertrophy (ectasia) over time and do not regress. A sudden increase in size is a concerning feature which may suggest infection and require additional evaluation. Histopathology shows hypertrophied endothelium with *normal mitosis* (versus hemangiomas which have increased mitosis during the proliferative phase). Achieving cure is the goal but difficult to achieve in practice. Vascular malformations are categorized as either "low-flow," which includes lymphatic, venous, and capillary malformations, or "high-flow" arteriovenous malformations.

Low Flow

Capillary Malformation (CM)

Capillary malformations are sporadic lesions consisting of dilated capillary channels. The etiology is related to ectasia of vessels in the reticular and papillary dermis. They are present at birth as flat, blanchable, red or purple, macular lesions with irregular borders. They are painless and do not spontaneously bleed. They are typically located in the head and neck but can occur anywhere on the body. They typically have a unilateral or segmental distribution that respects the midline. They are classified as medial or lateral depending upon their location. Medial lesions are often "salmon patches (nape of neck)," "stork bite," or "angel kiss (forehead)" and usually lighten and fade by age five. Lateral lesions also known as port-wine stains, which do not regress and often follow cranial nerve V distributions. These venular capillary malformations (port-wine stains) may be associated with skin thickening and soft tissue hypertrophy that can be progressive over the life of the patient. This causes the lesion to become darker in color, as well as more raised and nodular. Nearly 30% are associated with cobblestoning, and a smaller portion can see dermal, fat, and deep tissue hypertrophy. This can also involve the muscle and bone in some cases. CMs can be associated with complex malformation syndromes, most commonly Sturge-Weber syndrome (CM along V1 distribution, leptomeningeal angiomatosis, choroid angioma, glaucoma, seizures).

Treatment options include the KTP laser and FPDL (585–600 nm) and have reported 50–70% response rates ranging from partial to complete resolution. Other options include sclerotherapy [6], major surgical excision [7], and compression therapy [8]. Soft tissue hypertrophies as result of chronic lesions are not responsive to laser therapy and can necessitate excision with rotational flaps or advanced surgical therapy.

Venous Malformation (VM)

VMs grow slowly in childhood and expand rapidly with hormonal changes (puberty) or after trauma. An important diagnostic principle is their enlargement with Valsalva, recumbent positioning, and compressibility with refilling. Histopathology shows disorganized and random pattern/network of venous channels and the stain negative for Wilms' tumor 1 (WT1). There is a familial autosomal dominant subtype that has been mapped to chromosome 9q. MRI is the imaging choice for diagnosis but can also be evaluated with ultrasound. Phleboliths are pathognomonic findings which are the result of localized thrombosis.

Complications include congestion, thrombosis, phlebitis, pain, and distal emboli (large lesions). In contrast to disseminated intravascular coagulation, local intravascular coagulation can occur in large lesions and necessitate anticoagulation and correction with recombinant factors. Treatment options include compression garments, surgery for deep lesions, laser for superficial lesions (Nd:YAG), sclerotherapy for small lesions, or preoperative adjunct for larger lesions. Preoperative sclerotherapy or judicious use of topical/intralesional thrombin often can significantly reduce surgical hemorrhage.

VM are a heterogenous group, and a subset is made up glomuvenous malformations. They comprise a distinct entity characterized by the presence of glomus cells. Unlike venous malformations, they are localized to the superficial components: epidermis, dermis, and subcutaneous fat. They can be painful and do not fully evacuate with compression. Treatment is similar with significant response to Nd:YAG and surgical excision.

Lymphatic Malformation (LM)

Lymphatic malformations are thin-walled dilated lymphatic spaces with fibrous interstitium lined by endothelial cells filled with lymphatic fluid. LMs are classified into either *macrocystic* (*≥1 cyst*, *≥2 cm*) or *microcystic* (*<2 cm*) or *mixed*. Macrocystic lesions have cystic components at least 2cm^3 in volume, while microcystic LMs have volumes <2cm^3. In general, macrocystic lesions are more amendable to treatment and have a better prognosis. Of all LMs, 80% are located in the head and neck region, but they can occur anywhere on the body. Formerly referred to as cystic hygromas and lymphangiomas, this terminology is a misnomer and poorly describes the lesions. Eighty percent of lymphatic malformations (LMs) are diagnosed by 1–2 years old, and 65% are present at birth. Symptoms include a soft, painless, compressible mass often with overlying normal skin. Unlike venous malformations, they are not dependent and not affected by Valsalva. These lesions can frequently be associated with dysphagia and dyspnea. A highly diagnostic feature is change in acute enlargement of these lesions with infections. They may become firm, swollen, enlarged, erythematous, and acutely painful.

Imaging of choice is MRI, and LM has characteristic T1 low-signal intensity, T2 high-signal intensity, rim/septal enhancement with gadolinium, and fluid-fluid levels. They may be associated with venous malformations as the lymphatic and venous systems develop concurrently.

There are staging systems that are helpful for research purposes but practically of limited clinical uses. Such systems like the *de Serres* staging system [9] relate the lesions in reference to the hyoid, retropharyngeal space, and mediastinum which can be helpful for anatomical classification. In general, those patients with infrahyoid, unilateral lesions have a better prognosis than those with bilateral and/or suprahyoid involvement.

Currently, no ideal treatment exists for LM. Medical management during periods of acute enlargement consists of antibiotics and corticosteroids. Definition treatment is delayed until resolution of the infection. Sclerotherapy is a common modality used for macrocystic LMs and deep malformations that are difficult to access surgically. Multiple agents are available and include OK-432, tetracycline, sodium tetradecyl sulfate, alcohol, and bleomycin. OK-432 was developed in Japan and is not currently FDA approved. It consists of a streptococcus culture treated and killed with penicillin, which incites a highly effective immune response when injected (delayed hypersensitivity reaction). *Bleomycin*, in particular, is becoming more popular because of ease of use and improved safety profile. Unlike other agents, it does not elicit a robust inflammatory response and can often be used more safely around the airway and orbits. There is a maximum lifetime dosage, and side effects include pulmonary fibrosis and scar hyperpigmentation. In general, macrocystic lesions are more responsive to sclerotherapy due in part to ease of diffuse injection. Complications include swelling, skin breakdown, neuropathy, and pain. In the setting of severe swelling, ICU monitoring is indicated, and care must be taken to ensure airway control.

Surgical intervention can be utilized for VM with functional compromise, cosmetic deformity, intractable pain, or microcystic VMs. Goals of treatment are complete eradication of the VM while *sparing all vital structures.* Incomplete resection is associated with high rates of recurrence. When surrounding soft tissue or bone involvement requires massive resection, local flap reconstruction or free flap reconstruction may be indicated. If a large LM is detected on prenatal imaging, EXIT procedure may be required for airway protection. The CO_2 laser may be used when there is extensive tongue or mucosal involvement by the LM. Tongue reduction surgery and staged debulking therapies may be appropriate. It is critical to have a multi-disciplinary team involved as both surgery and sclerotherapy are often indicated. It is worthwhile to include psychiatric care for children with large lesions due to long-term morbidity.

High Flow

Arteriovenous Malformation (AVM)

Arteriovenous malformations (AVMs) are formed by shunting between arterial and venous systems via anomalous capillary beds resulting in progressive expansion of capillary bed (nidus) with vessel hypertrophy. They may stay small and stable in childhood and then rapidly grow with puberty or following trauma. AVMs may be defined as either *focal* or *diffuse* depending on their distribution. They can also be staged based on *low* and *high* grade, with the former presenting later in life (second to fourth decades) and the latter at birth. *Schobinger* developed a system ranked I–IV based upon the natural progression of AVMs: quiescent, expansion (bruit, thrill, warm throbbing), destruction (ulcers, bleeding, bony changes), and systemic (congestive heart failure, left ventricular hypertrophy).

Symptoms include a warm, *pulsatile* intermittently growing lesion with skin discoloration and bruit. There are various diagnostic imaging options including pulsed Doppler, CTA, and MRA (digital angiography formerly used). Complications involve local tissue destruction, high-output cardiac failure, and hemorrhage. Treatment depends on location, size, and symptomology. Embolization is a primary therapy but may necessitate surgical excision for superficial or recalcitrant lesions. Preoperative embolization followed by surgery is generally recommended in these cases [10]. Identification and removal of the nidus is paramount as excision of surrounding dilated vessels alone will not result in a cure. Diffuse lesions may require staged approaches.

Vascular Malformation Imaging Recommendations [11, 12]

Diagnosis of a soft tissue vascular anomaly is primarily based on clinical examination. Imaging is usually reserved for therapeutic planning, lesions with unclear diagnosis, or deep tissue involvement. Ultrasound is a good initial imaging choice. MRI with fat-suppressed T2 and STIR sequences is useful in assessing the extent of a lesion; fat-suppressed T1 pre- and post-contrast is helpful in differentiating lymphatic from venous malformations. The use of skin marker is highly recommended. Contrast is invaluable for showing whether a mass exhibits enhancement, outlines its vascularity, defines the vascular supply and venous drainage pattern, and shows arteriovenous shunting, all of which are essential data for lesion characterization.

- High-flow lesions (AVM, infantile hemangioma in proliferation phase): Vessels appear as signal voids on spin-echo (SE) sequences and hyperintense foci on gradient-recalled echo (GRE) sequences.
- Low-flow lesions (LM, VM, CM): Hallmark is the lack of flow voids on SE or FSE sequences. Venous malformations exhibit phleboliths, while lymphatic lesions may show intra-cystic hemorrhage/layering.
- *Imaging Pitfalls:*
  - Low-signal-intensity structures resulting in pseudoflow voids caused by intralesional septations, intravascular thrombus, or phleboliths—these can be easily identified on contrast-enhanced and GRE sequences.
  - Dynamic time-resolved contrast-enhanced MRA permits estimation of the contrast arrival time, which is significantly longer in low-flow lesion.

Vascular Anomaly Syndromes

Kasabach-Merritt syndrome hemangioma, thrombocytopenia, and consumptive coagulopathy. Affects males more than females. Ten to thirty percent mortality if left untreated due to hemorrhage. Do not transfuse platelets unless active hemorrhage or planned surgical procedure.

PHACES syndrome posterior cranial fossa anomalies, cervicofacial hemangioma, arterial/carotid malformation, cardiac anomaly/aortic coarctation, eye anomalies, sternal pit.

Sturge-Weber syndrome unilateral port-wine stain in the V1 distribution (ophthalmic division) or V2 with associated ocular and CNS involvement. Can often extend to the leptomeninges. Can be bilateral and involved upper/lower eyelids. Workup includes CT (+/-MRI) for intracranial involvement.

Klippel-Trenaunay syndrome combination of cutaneous vascular malformations, lower extremity varicosities, and hypertrophy of soft tissue/long bones. Present at birth and often very aggressive. Classic feature is involvement of lateral thigh and knee.

Parkes-Weber syndrome arteriovenous and capillary malformation in association with skeletal/soft tissue hypertrophy.

Blue rubber bleb venous malformation with associated GI lesions heralded by lower GI bleeding.

Maffucci syndrome multiple hemangiomas associated with enchondromas beginning in adolescence. The skeletal lesions often degenerate into malignant tumors and involve the hands and feet. Pathologic fractures common in long bones.

Servelle-Martorell syndrome rare congenital angiodysplastic disease with capillary malformations similar to KTS but associated with progressive limb hypotrophy rather than overgrowth.

Proteus syndrome extremely rare disorder characterized by overgrowth of body parts. Forty percent have cutaneous vascular malformations. Can have parotid monomorphic adenomas.

Bannayan-Riley-Ruvalcaba syndrome PTEN mutation, skin lipomas, multiple hemangiomas, macrosomia.

Beckwith-Wiedemann syndrome macroglossia, gigantism (long limbs), ear pits, abdominal wall defects (omphalocele), *nevus flammeus*, neonatal hypoglycemia.

Capillary malformation-arteriovenous malformation syndrome multiple capillary malformations and AVMs. *RASA1* gene mutation.

Phakomatosis pigmentovascularis association of capillary malformation (port-wine stain) with extensive melanocytic lesions including dermal melanocytosis (Mongolian spots), nevus spilus, and nevus of Ota. This syndrome is often associated with glaucoma.

Questions

1. Which vascular lesion(s) stain positive for GLUT-1?
   (a) Congenital hemangiomas
   (b) Infantile hemangiomas
   (c) Kaposiform hemangioendotheliomas
   (d) Tufted angiomas
2. What does PHACES stand for?
   (a) Posterior fossa malformations, heart defects, arteriovenous malformations, capillary malformations, eye abnormalities, sarcopenia
   (b) Posterior fossa malformations, cervicofacial hemangioma, arterial/carotid malformation, cardiac anomaly, aortic coarctation, eye abnormalities, sternal pit
   (c) Posterior fossa malformations, cardiac defects, azotemia, cervical dysplasia, eye abnormalities, sternal pit
   (d) Posterior fossa malformations, heart defects, arteriovenous malformations, capillary malformations, eye abnormalities, sternal pit
3. You see a 10-day-old infant with a 2-cm erythematous blanchable macule on the bottom of his neck with irregular borders. He is otherwise healthy and there are no other skin lesions. What is the likely prognosis?
   (a) Grows with child over time
   (b) Gradually fades by age 1 year
   (c) Requires further imaging studies to determine the extent of disease
   (d) Will likely become raised and ulcerate prior to slowly fading by early childhood.
4. A 3-year-old child has a cutaneous capillary malformation on his lower posterior neck. He has a second erythematous macule lesion on his lateral upper calf and asymmetry of the lower limbs. What syndrome could this be?
   (a) Parkes-Weber syndrome
   (b) Maffucci syndrome
   (c) Servelle-Martorell syndrome
   (d) Klippel-Trenaunay syndrome
   (e) Beckwith-Wiedemann syndrome
5. How long does the proliferation phase generally last for infantile hemangiomas?
   (a) 8 to 12 months
   (b) 5 to 8 months
   (c) 2–3 years
   (d) 4–5 years
6. A parent brings in their 3-month-old child with swelling in the right cheek. It is compressible and seems to decrease in size when standing versus lying down. There are no skin changes and it is non-tender. The patient is otherwise completely healthy, and the parent is not sure if it was present at birth. What lesion is this likely based on physical exam alone?
   (a) Lymphatic malformation
   (b) Parotid tumor
   (c) Rapidly involuting congenital hemangioma (RICH)
   (d) Venous malformation
7. A 6-month-old child from a rural area has a very large, soft, erythematous patch along his jawline as well as ten scattered soft, red, raised, nonpulsatile lesions scattered along the chest and abdomen. The mother reports that the lesions presented shortly after birth but have not changed over the last month. On exam the child is noted to have expiratory stridor, lethargy, abdominal distention, and failure to thrive. Workup should include all the following except?
   (a) CBC
   (b) Microlaryngoscopy/bronchoscopy
   (c) Abdominal ultrasound
   (d) Urinalysis
8. What tests do you need to order prior to initiating propranolol therapy? What is a standard target dosing?
   (a) ECG, 5–6 mg/kg/day
   (b) ECG, 2–3 mg/kg/day
   (c) Echocardiogram and ECG, 5–6 mg/kg/day
   (d) Echocardiogram and ECG, 2–3 mg/kg/day

9. When is propranolol therapy effective for focal infantile hemangiomas?
   (a) 6 months total
   (b) During the proliferation phase (up to 8–12 months)
   (c) 2–3 years
   (d) Birth to 18 months
10. What radiologic sign is highly suggestive of venous malformations?
   (a) Flow voids
   (b) Enhancement on T2
   (c) Microcystic and macrocystic components
   (d) Phleboliths
11. Arteriovenous malformations are most rare in which area of the head and neck?
   (a) Oral tongue
   (b) Cheek
   (c) Ear
   (d) Larynx
12. Capillary malformations (port-wine stains) are best treated with what laser?
   (a) C02: 10600
   (b) Diode: 1450
   (c) Pulsed dye: 585
   (d) Erbium YAG: 2940
13. Indications for surgical excision of infantile hemangiomas include all the following except:
   (a) Rapidly growing at 6 months
   (b) Ulceration
   (c) No response to propranolol therapy at 12 months
   (d) Ocular lesion affecting the globe
14. Which of these following sclerotherapy agents are not commonly used for treatment of lymphatic malformations?
   (a) Bleomycin
   (b) OK-432
   (c) 98% ethanol
   (d) Vincristine
15. Congenital hemangiomas can be differentiated from infantile hemangiomas in all these ways except?
   (a) GLUT-1 negative
   (b) Always present at birth
   (c) Do not respond to propranolol
   (d) Surgery should not be considered for treatment.
16. The superficial bluish components of venous malformations are often best treated with this laser.
   (a) C02: 10600
   (b) Diode: 1450
   (c) Pulsed dye: 595
   (d) Nd:YAG: 1064
17. The mechanism of action of OK-432 (Picibanil) is thought to be?
   (a) Microtubule inhibition
   (b) DNA methylation
   (c) Delayed hypersensitivity reaction
   (d) Bacterial cell wall lysis
18. What radiologic finding is highly diagnostic for lymphatic malformations?
   (a) Flow voids
   (b) Cystic areas
   (c) Fluid-fluid levels
   (d) Intralesional enhancement
19. In a patient with a hemangioma where propranolol is contraindicated due to cardiac or pulmonary reasons, what systemic agent and dosing have proven a viable alternative?
   (a) Vincristine (5 mg/kg/day)
   (b) Prednisolone (2–3 mg/kg/day)
   (c) Prednisone (1–2 mg/kg/day)
   (d) Prednisolone (8 mg/kg/day)
20. What sclerotherapy agent has minimal postinjection inflammation and therefore may have enhanced safety around the airway?
   (a) Bleomycin
   (b) OK-432
   (c) 98% ethanol
   (d) Doxycycline
21. Pulsed dye laser (PDL) therapy for hemangiomas is indicated for all the following situations except:
   (a) Ulceration
   (b) Deep hemangiomas
   (c) Superficial hemangiomas
   (d) Residual vascular pigmentation following involution
22. A 3-year-old patient presents to the ER with fever, rhinorrhea, and painless rapid enlargement of the neck. Past medical history is unremarkable, and parents deny prior illnesses or developmental delays. The patient is admitted to the hospital and stabilized, and

imaging is performed to further characterize the neck swelling. MRI reveals a mixed microcystic lymphovenous malformation. Improved prognosis with these lesions is associated with:
   (a) Unilateral infrahyoid involvement
   (b) Unilateral suprahyoid involvement
   (c) Bilateral infrahyoid involvement
   (d) Bilateral suprahyoid involvement
   (e) Unilateral infra- and suprahyoid involvement
23. Vascular malformations when compared to vascular tumors are characterized by:
   (a) Increased endothelial cell turnover
   (b) Female predilection
   (c) Visibility at birth
   (d) Proportional growth with patient
24. Radiographic characterization of arteriovenous malformations is best obtained by:
   (a) Ultrasound
   (b) Contrast-enhanced CT
   (c) MRI
   (d) MRA
25. Which laser is matched appropriately to its absorption spectrum?
   (a) CO_2: 1060 nm
   (b) PDL: 585 nm
   (c) KTP: 585 nm
   (d) Nd:YAG: 10,600 nm

Answers

1. (b)
2. (b)
3. (b)
4. (d)
5. (b)
6. (d)
7. (d)
8. (b)
9. (b)
10. (d)
11. (d)
12. (c)
13. (a)
14. (d)
15. (d)
16. (d)
17. (c)
18. (c)
19. (b)
20. (a)
21. (b)
22. (a)
23. (d)
24. (d)
25. (b)

References

1. Mulliken JB, Enjolras O. Congenital hemangiomas and infantile hemangioma: missing links*1. J Am Acad Dermatol. 2004;50(6):875–82. https://doi.org/10.1016/s0190-9622(03)04277-4.
2. Wassef M, Blei F, Adams D, et al. Vascular anomalies classification: recommendations from the international society for the study of vascular anomalies. Pediatrics. 2015;136:e203.
3. Metry DW, Haggstrom AN, Drolet BA, et al. A prospective study of PHACE syndrome in infantile hemangiomas: demographic features, clinical findings, and complications. Am J Med Genet A. 2006;30.
4. Léauté-Labrèze C, Hoeger P, Mazereeuw-Hautier J, et al. A randomized, controlled trial of oral propranolol in infantile hemangioma. N Engl J Med. 2015;372:735–46.
5. Darrow DH, Greene AK, Mancini AJ, Nopper AJ. Diagnosis and management of infantile hemangioma. Pediatrics. 2015;136(4):e1060–104.
6. Sung HM, et al. Clinical experience of the Klippel-Trenaunay syndrome. Arch Plast Surg. 2015;42(5):552–8. https://doi.org/10.5999/aps.2015.42.5.552. Epub 2015 Sep 15
7. Jacob AG, et al. Klippel-Trénaunay syndrome: spectrum and management. Mayo Clin Proc. 1998;73(1):28–36.
8. Capraro PA, et al. Klippel-Trenaunay syndrome. Plast Reconstr Surg. 2002;109(6):2052–60. quiz 2061-2
9. De Serres LM, Sie KC, Richardson MA. Lymphatic malformations of the head and neck. A proposal for staging. Arch Otolaryngol Head Neck Surg. 1995;121(5):577–82.
10. Richter GT, Friedman AB. Hemangiomas and vascular malformations: current therapy and management. Int J Pediatr. 2012:1–10.
11. Dubois J, Alison M. Vascular anomalies: what a radiologist needs to know. Pediatr Radiol. 2010;40(6):895–905.
12. Legiehn GM, Heran MKS. A step-by-step practical approach to imaging diagnosis and interventional radiologic therapy in vascular malformations. Semin Intervent Radiol. 2010;27(2):209–31. https://doi.org/10.1055/s-0030-1253521.

Part IV

Aesthetic Surgery

17 Blepharoplasty

Benjamin P. Caughlin

Eyelid Anatomy [1–7]

Arterial supply Arcades supplied by the internal carotid artery (ICA) and external carotid artery (ECA). Within the ICA system, terminal branches of the ophthalmic artery at the medial eyelid anastomose with terminal branches of lacrimal artery at the lateral eyelid. Contribution from the ECA system includes branches from the facial, superficial temporal, and infraorbital arteries.

Venous outflow Facial vein is the main pathway; there are communications with the ophthalmic veins, cavernous sinus, and pterygoid plexus.

Lymphatic drainage Ipsilateral preauricular lymph nodes and submandibular lymph nodes. Vertical incisions at the region of the lateral canthus are associated with increased risk for prolonged/chronic lymphedema due to transection of the lymphatic channels that run horizontally.

Motor nerve innervation Orbicularis oculi innervated by frontal, zygomatic, and buccal branches of facial nerve (CN VII). Levator palpebrae superioris innervated by superior division of oculomotor nerve (CN III). Muller's muscle (superior tarsal muscle) innervated by sympathetic nervous system (fibers from superior cervical ganglion that cross the cavernous sinus)

Sensory nerve innervation Upper eyelid innervated by ophthalmic division of trigeminal nerve (CN V1) through the supraorbital, supratrochlear, and lacrimal branches. Lower eyelid innervated by ophthalmic (CN V1) and maxillary (CN V2) divisions of the trigeminal nerve

Orbicularis oculi muscle Oblique and circular muscle that requires medial canthal and lateral canthal anchoring points to convert the oblique circular contraction into a vertical vector to complete eye closure. Divided into orbital, palpebral (preseptal and pretarsal), and lacrimal components defined by underlying anatomy

Levator palpebrae superioris muscle Elevates the upper eyelid. It is often simply referred to as the levator.

Capsulopalpebral head/fascia Main retractor of the lower eyelid. The capsulopalpebral head originates as fascial tissue from the inferior rectus muscle, splits to envelope the inferior oblique muscle, and then reunites to form the capsulopalpebral fascia which inserts at the inferior tarsus. It is often referred to as the lower eyelid retractor.

Orbital septum In the upper eyelid, it extends from the periosteum at the superior orbital rim to

B. P. Caughlin (✉)
@manyfacesofchicago, Chicago, IL, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_17

join the levator aponeurosis. In the lower eyelid, it extends from the periosteum at the inferior orbital rim to join the capsulopalpebral fascia.

Muller's muscle Origin is undersurface of levator, and insertion is the superior tarsal plate.

Lamella Often categorized as the anterior lamella (the skin and orbicularis muscle) and posterior lamella (tarsus and conjunctiva).

Layers of the eyelid At the level of the tarsus, the layers from anterior to posterior include the skin, orbicularis oculi muscle, tarsus, and conjunctiva. At a more superior level in the upper eyelid and a more inferior level in the lower eyelid, the layers from anterior to posterior include the skin, orbicularis oculi muscle, orbital septum, orbital fat, levator (upper eyelid) or capsulopalpebral fascia (lower eyelid), and conjunctiva. This is a generalization and depends on the precise level.

Fat compartments At the upper eyelid, two fat compartments deep to the orbital septum are traditionally described. These are the medial (nasal) fat pad and the central (preaponeurotic) fat pad, which are separated by fascial connections to the trochlea of the superior oblique muscle. The central fat pad is larger in size and more yellow in appearance (compared to the paler medial fat pad). The more lateral region of the upper eyelid is occupied by the lacrimal gland. Some texts describe preaponeurotic fat protruding under the lacrimal gland and/or a third compartment. At the lower eyelid, three fat compartments are traditionally described, which include the medial (nasal), central (middle), and lateral fat pads. The inferior oblique muscle separates the medial and central fat pads, and a fascial sheath of the inferior oblique muscle extends laterally to separate the central and lateral fat pads.

Eyelid Crease [8]

Non-Asian eyelid At the upper eyelid, the orbital septum typically fuses with the levator aponeurosis approximately 10 mm superior to superior tarsal border. This, in combination with the levator attachment to the skin, defines the upper lid crease (Fig. 17.1). At the lower eyelid, the orbital septum fuses with the capsulopalpebral fascia approximately 4–5 mm inferior to the inferior tarsal border.

Asian eyelid At the upper eyelid, the orbital septum typically fuses to the levator aponeurosis inferior to the superior tarsal border. This inferior extension of the orbital septum prevents aponeurotic fibers of the levator from reaching the subcutaneous tissues of the eyelid and thus preventing a well-developed crease. Secondly, this is also associated with orbital fat extending more inferiorly, to the space anterior to the tarsus, resulting in a fuller appearance to the upper eyelid. The excess fat herniated can lead to epiblepharon (inward rotation of the lashes toward the globe). A similar phenomenon is observed in the lower eyelid, but the effect is less pronounced because the lower eyelid crease is generally less prominent in even the occidental population.

Ligaments of Orbital Support [9]

Whitnall's ligament (superior transverse ligament) It extends from superomedial orbital rim, running horizontally across the orbit, to the superolateral orbital wall (medial edge of frontal bone). It suspends the lacrimal gland, superior oblique tendon, and levator muscle. Also it functions as a fulcrum to facilitate reorientation of the force vector of the levator to a more superior-inferior direction.

Lockwood's ligament Formed by conjoining fascia of the inferior rectus and inferior oblique muscles with medial and lateral attachments to orbital rim. It functions as a suspensory sling for the orbit. Over time this ligament weakens and lowers, which contributes to dropping of the eyeball and pseudoherniation of the fat anteriorly.

Arcus marginalis Collections of connective tissue fibers forming a thickening at the region

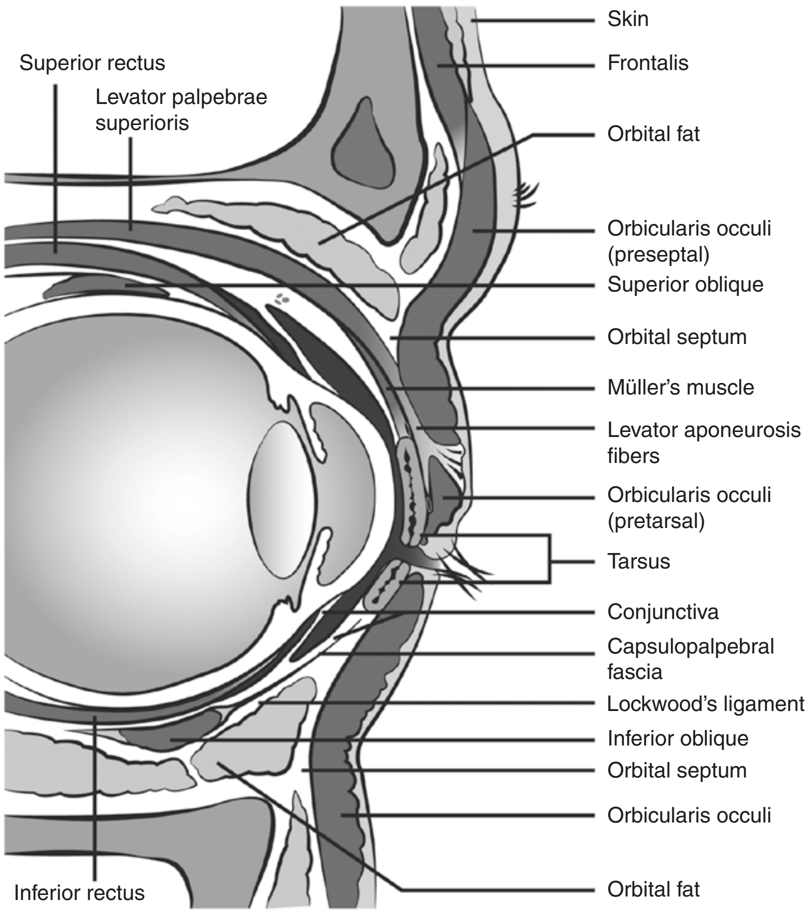


Fig. 17.1 Sagittal view of the orbit and lid anatomy to illustrate soft tissue layers and fat pad position

where the orbital septum fuses with the periosteum of the orbital rim

Orbicularis retaining ligament Attachment extending from the inferior orbital rim periosteum to the orbicularis oculi muscle. It is also described as extending through the orbicularis oculi muscle to attach with the dermis.

History, Assessment, and Analysis [10–17]

Relative contraindications to blepharoplasty Actinic changes, acne rosacea, keloids, herpes zoster, thyroid abnormalities, autoimmune diseases, smoking history, extensive history of dry eye syndrome, and acute angle glaucoma history. Generally, should wait approximately 6 months after LASIK or PRK (exact timing varies depending on the source).

Skin Loss of elastic fibers of the skin over time leads to dermatochalasis (redundant and excess skin).

Orbital septum Weakening of orbital septum leads to pseudoherniation of the fat; medially the orbital septum is more thin, and this is the main location of pseudoherniation.

Suspensory ligaments Weakens over time and the eye drops (senile enophthalmia) which causes pseudoherniation of the fat pushing the orbital septum outward and demarcating the orbital rim.

Orbicularis oculi muscle hypertrophy Leads to festoons (folds of orbicularis from medial to lateral canthi which can contain fat). Festoons are caused by excessive squinting and facial animation, it should not be mistaken for fat herniation as the treatment differs. Festoons are treated with orbicularis muscle stripping and microvulimizers.

Retro-orbicularis oculi fat pad and suborbicularis oculi fat pad Located between the orbital septum and the orbicularis muscle. Descent of the retro-orbicularis oculi fat (ROOF) over time contributes to increased fullness of the upper eyelid; it can be mistaken for orbital fat prolapse. Descent of the suborbicularis oculi fat (SOOF) contributes to hollowing of the infraorbital region.

Lacrimal system History of dry eye sensation is not a contradiction to blepharoplasty surgery but requires thorough evaluation and conservative skin excision. *Schirmer's test* is performed by placing a strip of filter paper extending out from inferior fornix and measuring the length of wetting. Normal wetting is typically considered to be 15 mm or greater after 5 minutes (without topical anesthesia); however the test has poor consistency, and some texts consider an even smaller length of wetting to be normal. *Phenol red thread test* is a similar concept but instead uses strips of special thread that becomes red in color when wet with tears; normal wetting is typically between 9 and 20 mm after a 15-second test.

Lid margin On neutral gaze, the upper eyelid margin should be at or just below the upper limbus (no lower than 2 mm beyond the limbus). The lower eyelid margin should be at or slightly above the lower limbus with no scleral show.

Upper eyelid crease In Caucasians, typically 4 mm above lid margin at medial canthus, 6–10 mm above lid margin at mid-pupillary line, and 5–6 mm above lid margin at lateral canthus. Women have a slightly higher lid crease compared to men.

Brow position In women, slightly above the orbital rim and more curved with the peak at the lateral limbus. In men, it tends to be at orbital rim and straight.

The brow and eyelid relationship Ideal to have a crisp upper lid sulcus inferior to the superior orbital rim, free of redundant skin and protuberant fat pads. Overly deep and hollow upper sulcus is equally best avoided. Brow position at or above the orbital rim prevents crowded appearance and maintains vertical distance from high point of brow to lid ciliary margin of the upper lid with minimal periorbital shadowing.

Brow ptosis Exaggerates and confounds aging of the upper eyelid complex. It is critical to evaluate the role for brow lift prior to proceeding with blepharoplasty. Brow ptosis can lead to suborbicularis fat protrusion giving the appearance of pseudoherniated fat. It may also lead to over-resection of the skin and lagophthalmos if not recognized prior to blepharoplasty.

Recognize lateral hooding There should be a smooth transition of the upper eyelid to lateral temporal skin with no hooding of the skin lateral to the orbital rim. Evaluate the role for brow lift.

Blepharoptosis Causes include myogenic, neurogenic, aponeurotic, mechanical, and pseudoptosis. Aponeurotic ptosis is the most common type of acquired ptosis. It is defined as dehiscence, stretching, or disinsertion of the levator aponeurosis, which can be due to age-related degeneration/thinning (involutional ptosis), repeated traction on the eyelid (e.g., using contact lenses and rubbing eyes), eye surgery, and trauma. It is associated with a high upper eyelid crease (>10 mm) and can lead to a superior visual field defect.

Levator function Difference in position of the upper eyelid margin from downward to upward gaze, >12 mm good, 6–11 mm fair, and 0–5 mm poor (these numbers vary depending on citation).

Margin reflex distance-1 (MRD-1) The distance from the corneal light reflex to the central upper lid margin while in primary gaze, typically 4–5 mm.

Margin reflex distance-2 (MRD-2) The distance from the corneal light reflex to the central lower lid margin while in primary gaze, typically 5 mm.

Globe-orbital rim relationship Assess relationship of anterior most projection of globe in relation to lid margin and malar eminence. If there is negative vector (most anterior projection of globe is anterior to the lid margin and the malar eminence), care should be taken to be conservative with skin and muscle resection. Lateral canthoplasty, lid tightening, infraorbital/midface augmentation, and/or midface lifting should be considered.

Eyelid cheek complex Ideal is smooth convex profile with no soft tissue contour irregularities and without definition of the inferior orbital rim.

Double-convexity deformity First convexity is created by a weakened lower eyelid orbital septum. Second convexity is associated with the weakened arcus marginalis and descended suborbicularis oculi and cheek fat. Medially this forms tear trough deformity and laterally hallowed eye deformity.

Malar fat pad A triangular soft tissue structure with the base at the nasolabial fold and the apex pointed superior-laterally to the malar eminence. Age-induced descent of this structure leads to an aged, rounded, and hollowed appearance to the lower eyelid and infraorbital area/rim. When pseudoherniation of the orbital fat happens in conjunction with ptosis and loss of soft tissue volume of the midface, the nasojugal groove is deepened and creates the tear trough deformity.

Tear trough deformity Descent of suborbicularis oculi fat and malar fat pad leading to soft tissue depression and skeletonization at the region of the medial inferior orbital rim

Barton tear trough triad [18] (1) herniation of orbital fat, (2) sharp demarcation of the orbit-cheek junction, and (3) retrusion of the lower orbital rim creating a negative vector orbit. The anatomy of the tear trough deformity demonstrates the muscular triangle formed by the orbicularis oculi, levator labii superioris, and levator labii alaeque nasi. It can be treated with intraorbital fat mobilization and septal reset to anterior inferior aspect of inferior orbital rim.

Orbicularis festoons/"cheek bags" Patient should be educated that this is a complex problem to treat; adjunct procedures to standard lower blepharoplasty should be considered, such as fat repositioning, lifting procedures, and laser resurfacing.

Lateral canthus Ideally located 1–2 mm above that of the medial canthus and at the level of the inferior edge of the pupil. This allows the tears from the superolateral-positioned lacrimal gland to bathe the eye and then run downward to the lacrimal drainage apparatus, located in the medial canthus. The lateral scleral triangle is larger, wider, and more pointed when compared to the medial scleral triangle. The sharpness of the lateral canthus can be lost and is a complication of blepharoplasty (rounded eye).

Lower lid laxity Identifying lower lid laxity preoperatively is important to establish risk for eyelid malposition and ectropion (complications of blepharoplasty). Poor punctal position can cause tear flow issues. Test for lower lid laxity with snap test and lid distraction test. Lower lid laxity should be treated (e.g., with lid-tightening procedure) if performing blepharoplasty to reduce risk of complications.

Snap test Grasp the lower lid between thumb and index fingers and pull the eyelid away from globe. Upon release, the eyelid should return (snap back) to original position immediately; the amount of time for return (or no return) relates to the degree of poor orbicularis oculi tone and lid laxity.

Lid distraction test The lower eyelid is pulled away from globe; the ability to move the lid margin greater than 6 mm is generally associated with significant lid laxity.

Preoperative photos Full face frontal, frontal close-up (eyes open, upward gazing, and eyes closed), and left and right oblique (and/or lateral) close-up with eyes open

Discussion of adjunct procedures Evaluate for and discuss indicated adjunct procedures such as brow elevation, lateral canthal position adjustment, correction of lower lid laxity, eyelid skin rejuvenation via resurfacing techniques, and ptosis correction.

Surgical Techniques

Upper Eyelid Approaches

Transcutaneous approach [19, 20] Patient is marked in upright position. Inferior marking is made at or near supratarsal crease (6–10 mm above lid margin at mid-pupillary line). The marking extends from superior punctum medially to lateral orbital rim laterally. Forceps are used, and a pinch test is performed to delineate redundant skin, and superior marking is noted at multiple points. A minimum of 10 mm of infrabrow/supratarsal skin should be preserved above superior marking to allow for complete eye closure and prevent lagophthalmos. Superior marking forms a gentle arc, and a small lateral extension beyond the lateral canthus is often required to address lateral hooding. More skin can typically be taken laterally. If a lower blepharoplasty is also being performed, a minimum of 5 mm should be left between the upper and lower blepharoplasty incisions laterally. The superior marking is longer laterally than the inferior marking and is tapered to join inferior marking medially. This leaves a lenticular or trapezoid shape. Avoid extension of incision lateral to orbital rim if possible, especially in males (unlikely to use cosmetics for camouflage). The medial edges of the superior and inferior markings join ending 6 mm from the angular vein with care taken not to extend past the medial canthus to prevent webbing. The incision begins through the dermis. Dissection is performed between the skin and orbicularis with curved scissors, and the premarked skin is removed. If orbicularis hypertrophy is present, then the predetermined amount of orbicularis muscle is removed. The medial and central fat pads can then be accessed by making a small incision through the septum. Conservative fat excision/reduction can be performed on the fat that bulges out while applying pressure on the globe. Excellent cautery should be performed at the excision site before allowing the remaining fat to retract back into its pocket. Skin incision is then approximated with suture.

Transconjunctival approach to upper lid medial fat pad [21] Useful for isolated upper lid medial fat herniation. Often utilized as a touch-up procedure after healing if a patient is unhappy with upper lid medial bulge of pseudoherniated fat. The upper lid is everted over a retractor, and pressure on globe helps identify fat bulging. An incision is made several millimeters superior to the tarsus, and dissection is carried to the fat pad. Only redundant fat is removed. No sutures required.

Lower Eyelid Approaches [22–27]

Pinch excision of lower lid skin This is worth mentioning, particularly after a midface lift. Lower lid skin is grasped just below the lash line with forceps, and small pinch of the skin is demarcated along the length of the lower lid. This is excised with curved scissors, and edges are reapproximated with glue or suture.

Transcutaneous skin only flap Incision is made approximately 2–4 mm below lash line, at first major rhytid, extending from the level of inferior punctum medially to lateral canthal angle laterally. Dissection is performed between the skin and orbicularis, skin flap is laid back down, and then only redundant overlapping skin is excised. Used on patients with dermatochalasis and no pseudoherniation of the fat or orbicularis hypertrophy.

Transcutaneous skin muscle flap Incision is the same as above. Dissection is performed between orbicularis and orbital septum. It is used on younger patients with robust orbicularis and lack of festoons. Submuscular compartment is accessed laterally while taking care to leave pretarsal orbicularis undisturbed (reduce ectropion risk). First, you ask the patient to look up and to open their mouth; this puts tension on the lower lid. Then, only the overlapping skin and muscle can be safely removed.

Fat removal The amount of fat to be removed is determined preoperatively. Develop plane by dissecting deep to the orbicularis muscle through preseptal fibers creating the muscle flap. Orbital septum is entered and fat is removed in lateral to medial direction. Small incision is made into each fat compartment, and with globe pressure, only redundant fat is removed with clamp and cautery. Careful hemostasis is obtained to prevent hematoma.

Transconjunctival approach Benefits include no external scar and maintaining orbicularis and orbital septum intact and less risk of lower lid malposition (e.g., ectropion and retraction). Ideal patient is young with smooth skin, with moderate fat pseudoherniation, and with no excess skin or orbicularis muscle hypertrophy. Incision is made through conjunctiva (e.g., halfway from fornix to lid margin, 1–2 mm below the tarsus) and carried through inferior retractors. Orbital fat can be approached anterior or posterior to the septum depending on where inferior retractors are incised. If plane of dissection is posterior to the septum, the fat compartments are approached directly. If dissection is carried out anterior to the septum, the fat compartments and septum are approached from an anterior direction between the orbicularis muscle and the orbital septum (Fig. 17.1). In this preseptal approach, the orbicularis is separated from the orbital septum (superficial to the septum, deep the orbicularis). The septum is incised from an anterior direction, and the fat is removed as indicated starting with the lateral compartment with caution to control bleeding. Lid is allowed to snap back into place, and no sutures are required. Consider postoperative or simultaneous chemical peel vs. pinch technique (removing 2–3 mm of only truly redundant skin below the ciliary margin) for those patients with dermatochalasis and superficial rhytides.

Fat Preservation (Repositioning/Mobilization) [28–30]

Repositioning Pseudoherniated fat is repositioned in a more favorable position posterior to the orbital rim (intraorbitally). Of note, the term “repositioning” has also been used in the literature in reference to “mobilization” techniques.

Mobilization The septum is incised, and the orbital fat is mobilized to a new position inferiorly over the orbital rim. This aids in camouflaging the skeletonized infraorbital rim/region that occurs with aging. Additionally suborbicularis oculi fat (SOOF) can be mobilized to a new position superiorly over the orbital rim. The ideal patient for this procedure has orbital fat pseudoherniated anterior to the lower eyelid margin and with a prominent inferior orbital rim associated with a hollowed-out appearance.

Mobilization steps Can be performed using either a transcutaneous or transconjunctival approach. Once the septum is reached, an incision is made along the attachment of the septum with the orbital periosteum (region of the arcus marginalis). This then provides access to the fat, which is released and draped over the orbital rim. If desired, a *septal reset* can be performed at this stage, such that the inferior most aspect of the orbital septum is sutured to the anterior aspect of the orbital rim at a more inferior position. For a prominent nasojugal groove (tear trough deformity), the fat can be mobilized (while still connected with a blood supply) and used to fill the depression. Placing the fat subperiosteally can aid in achieving a smoother contour. The fat can be held in position with temporary percutaneous sutures that are removed several days postoperatively.

Special Techniques

Skin/muscle suspension during lower lid blepharoplasty During a standard blepharoplasty, a lateral muscle suspension suture is placed with a buried knot between orbital periosteum and lateral orbicularis muscle.

Prophylactic lateral canthal anchoring [31–33] Helps improve/maintain proper eye shape and reduce postoperative complications such as lower lid malposition and scleral show. The ideal patient has lower eyelid laxity and/or scleral show. Generally, canthoplasty is a more extensive procedure that is suited for more severe laxity, as compared to canthopexy.

Canthopexy Anchoring without cantholysis

Suture is passed through the lateral canthal tendon and then secured to the periosteum at least 3–4 mm inside the lateral orbital rim (in line with the inferior edge of the pupil).

Canthoplasty Cantholysis and re-anchoring

Lysis of the lower lid tendon is performed at canthal angle. If desired, a lateral tarsal strip technique would be performed at this point. Small amount of lower lid margin can be trimmed to aid with advancement. Suture is passed through the tendon at the lower lid and/or tarsal plate and additionally passed through the tendon of the upper lid. This suture is then anchored to the periosteum similarly as with a canthopexy.

Orbital septal tightening [34, 35] Ideal patient has pseudoherniation of the fat due to weakened orbital septum. The orbital septum is exposed (not entered) by blunt muscle elevation and retraction. Gentle pressure on the globe delineates the amount of pseudoherniation and bulging. Electrocautery with a grid spray of 30 units is applied using Colorado tip cautery to the intact septum. A CO_2 laser can also be used, set at 7 W and 2 mm spot size in scanner mode. Energy is applied over the entire septum until shrinkage is obtained and a symmetric smooth contour is appreciated with gentle pressure on the globe.

Supratarsal crease creation/Asian blepharoplasty [8, 36, 37] As delineated in the anatomy section, it is the lack of dermal attachment of the levator aponeurosis because of the lower fusion of the septum with the levator that blunts the upper lid crease in the Asian patient. This low fusion also leads to preaponeurotic fat that occupies the space anterior to the tarsus leaving a fuller appearance to the upper eyelid. Depending on the patient's goals, this fat may need to be repositioned or removed which can debulk the eyelid fullness and allow for a sharpened upper lid crease. Removal of this redundant fat can also be used to treat epiblepharon which causes inward deviation of the lashes toward the globe. The supratarsal crease can then be created by securing the levator aponeurosis to the dermis at the superior tarsal border. Incision is planned 6–10 mm above the lid margin at the predetermined position of desired crease. Skin pinch is performed, and the location of the superior incisions determines the amount of the skin to be removed as in standard blepharoplasty. The skin is removed superficial to orbicularis followed by a thin strip of orbicularis removal along the inferior incision to allow for a deepened crease. The lower flap is then undermined ~2 mm inferiorly to expose the dermis for future fixation to levator. Orbital septum is identified and entered followed by removal or repositioning of predetermined fat to create the deepened sulcus. The levator aponeurosis and superior tarsus are exposed. The lid crease is created by fixation of the dermis from the undermined inferior incision flap to the levator fibers. A minimum of three sutures (midline, lateral limbus, medial limbus) are required.

Blepharoptosis Repair [38]

Frontalis sling Ideal patient has less than 4 mm of levator function due to congenital ptosis, external ophthalmoplegia, third nerve paralysis, or myasthenia gravis. Standard blepharoplasty incision ~8–10 mm above eyelid margin is made, two small stab incisions just above the eyebrow, and a third incision at the forehead that is 5 mm higher and centered between the brow incisions. Fascia (e.g., fascia lata or temporalis fascia) or other grafting material is sutured to anterior aspect of the upper third of tarsus at midline and at the points correlating with the medial and lateral limbus. The graft is passed through the incisions and pulled up from the forehead incision until eyelid margin is at the superior limbus. The end of the graft is secured to the frontalis muscle.

Levator resection and advancement Ideal patient has poor levator function but greater than 4 mm of function, which is due to congenital, myogenic, and aponeurotic ptosis. Standard blepharoplasty incision and blunt dissection through orbicularis pretarsal fibers inferior to orbital septum until lower border of levator aponeurosis is exposed. The septum is entered, and fat is retracted to expose the levator muscle aponeurosis. Levator and Muller's muscle are separated at the upper edge of the tarsus. Desired amount of levator is resected, and then levator is secured to the anterior-superior third of the tarsus. The eyelid is pulled downward to confirm height.

Muller muscle-conjunctival resection Ideal patient is ptosis that corrects with instillation of topical phenylephrine. Evert the eyelid over a retractor. Toothed forceps are used to pull conjunctiva and Muller's muscle from the levator. Predetermined amount of resection is performed. Approximately 8.5 mm of Muller's muscle resection produces similar results to 10% phenylephrine application.

Surgical Techniques to Repair Ectropion [39]

Manipulate tissues to determine which surgical maneuver (or combinations of maneuvers) will return the lower lid to a more natural position.

Lateral canthal tendon tightening [39] Treats lateral canthal tendon laxity. If preoperative repositioning of lateral canthus results in good aesthetics and function, then repositioning of the lower lid complex may be all that is required. It is accomplished by canthopexy vs. canthoplasty as described above.

Spacer graft insertion Treats scarring between the septum and deeper capsulopalpebral fascia. A spacer graft separates the capsulopalpebral fascia from the orbital septum and allows vertical elevation of the central lower eyelid margin to correct scleral show and retrodisplaced orbital fat. Graft material options include hard palate mucosal graft, alloplastic grafts, hyaluronic acid fillers, or nasal septal graft.

Cheek fat pad elevation Treats ectropion caused by descent of the malar fat pad

Subperiosteal midface elevation Treats lack of adequate lower eyelid skin, such as cases of prior over-resection. This can allow enough skin movement to possibly prevent free grafting. This technique is used to reposition the lower eyelid and all the midface structures back to anatomic position.

Complications [40–46]

Retrobulbar Hematoma

Cause Inadequate hemostasis often due to retraction of vessel into orbit

Symptoms Decreased acuity, amaurosis fugax, scintillating scotomas, diminished extraocular movements, loss of pupillary response, and scleral hematoma

Treatment Remove dressings, ophthalmology consult, stat OR setup while performing the following:

- Head elevation
- 20% mannitol (2 g/kg IV, 12 g over 3 min the rest over 30 min)
- Diamox 500 mg IV
- 95% O_2/5% CO_2 which dilates drainage vessels
- Solu-Medrol 100 mg IV.

- Betoptic (betaxolol HCL) glaucoma med: one drop immediately and then BID
- Surgical intervention: Surgical wound is opened and explored. If unsuccessful, then lateral canthotomy and cantholysis. Release Lockwood's, Whitnall's, and arcuate ligaments.

Lagophthalmos

Cause Likely from over-resection of the skin. If uncorrected, it can lead to corneal injury.

Treatment Initial step is massage. If persistent, consider full-thickness skin graft from contralateral eyelid, postauricular area, or supraclavicular area to reinstate adequate height to eyelid.

Webbing of Medial Upper Eyelid Incision

Cause Extension of medial incision past medial canthus

Treatment Massage; if persistent, consider surgical correction with Z, W, or V to Y plasty as indicated.

Conjunctivitis/Chemosis

Cause Likely from excessive cautery and disruption of orbital and eyelid lymphatics

Treatment Ice application for first 48 h postoperatively, and conservative treatment is recommended, which can take 2–5 months to resolve. It can require tarsorrhaphy suture or patching in severe cases.

Oversculpted/Hollowed Appearance

Cause Over-resection of the orbital fat

Treatment Best prevented by preoperative assessment and conservative fat removal. Surgically treated by injectables and/or fat mobilization/repositioning/transfer

Diplopia

Cause Most commonly from injury to inferior oblique muscle (separates the medial and central fat pads of the lower eyelid). The muscle should be identified, and injury should be prevented during fat manipulation.

Treatment 6 months of observation and ophthalmology consult is required if persistent.

Infections

Cause Unsterile technique

Treatment Rare due to high vascularity; it requires prompt recognition and treatment with antibiotics.

Suture Granuloma/Pyogenic Granuloma

Cause Increased incidence with transconjunctival approach to the lower eyelid

Treatment Topical corticosteroid; surgical removal if persistent

Tearing/Epiphora

Cause Consequence of lagophthalmos, lid margin integrity malposition, lacrimal gland disruption, and canalicular disruption

Treatment Assess lacrimal system and lid function and treat accordingly.

Lateral Canthal Rounding

Cause Results from malposition of lateral canthus and disruption of anchor function. Increased incidence with transcutaneous approach of the lower eyelid resulting in scarring and retraction

Treatment Canthopexy, canthoplasty, or lateral canthal anchoring

Entropion

Cause Likely from posterior lamellar retraction, scaring, lateral canthal tendon laxity, and midface decent

Treatment Conservative management first with taping of the eyelid, massage, and observation. If persistent, surgical intervention such as with canthoplication versus combined lateral tarsal strip with grafting and or wedge resection depending on the severity. The contribution from the malar fat pad descent can be corrected by repositioning of this structure and can assist to restore infraorbital and lower lid position.

Questions

1. Which statement is false?
   (a) In the Caucasian eyelid, the orbital septum of the upper lid attaches to the levator aponeurosis ~10 mm above the superior tarsal margin.
   (b) In the lower lid, the orbital septum attaches to the capsulopalpebral fascia ~5 mm below the lower tarsal margin.
   (c) Muller's muscle attaches to the orbital septum posterior to the levator aponeurosis.
   (d) The inferior orbital septum attaches to the periosteum of the inferior orbital rim inferiorly and to the capsulopalpebral fascia superiorly.
2. What muscles form the tear trough?
   (a) Nasalis, levator labii superioris, and levator labii alaeque nasi
   (b) Orbicularis oculi, levator labii superioris, and levator labii alaeque nasi
   (c) Zygomaticus minor, levator labii superioris, and levator labii alaeque nasi
   (d) Zygomaticus major, levator labii superioris, and levator labii alaeque nasi
3. In the Asian eyelid, what structure prevents aponeurotic fibers of the levator from reaching the subcutaneous tissues of the eyelid, thus preventing a crease?
   (a) Orbital septum
   (b) Levator aponeurosis
   (c) Preaponeurotic fat
   (d) Thick skin
4. Capsulopalpebral fascia is a fibrous extension of what structure?
   (a) The periosteum of inferior orbit
   (b) Inferior rectus muscle
   (c) Inferior oblique muscle
   (d) Orbital septum
   (e) Perichondrium of inferior tarsus
5. The best option to treat a tear trough deformity during blepharoplasty is:
   (a) Inferiorly reposition the pseudoherniated fat over the orbital rim
   (b) Excision of as much orbital fat as possible
   (c) Lower lid skin only blepharoplasty
   (d) Medial canthopexy
6. Retro-orbital hematoma is treated by surgical exploration, but on the way to the OR, what percentage of mannitol should be started?
   (a) 1% mannitol
   (b) 2% mannitol
   (c) 5% mannitol
   (d) 20% mannitol
7. Which of the following is not an indication for lateral canthoplasty?
   (a) Lateral canthal dystopia
   (b) Vertical lid laxity
   (c) Ectropion from cicatrix, atony, or paralysis
   (d) Lid retraction
8. What is the result of disinsertion of the lower eyelid retractors from the lower edge of the tarsal plate?

(a) Entropion
(b) Ectropion
(c) Diplopia
(d) Pseudoherniation of the orbital fat

9. In the lateral suspension technique using the transcutaneous skin muscle flap, the lateral suspension knot of the suture is placed:
   (a) Between the skin and lateral orbicularis muscle
   (b) Between the periosteum and the lateral orbicularis muscle at the lateral canthus
   (c) Between the periosteum and the lateral orbicularis muscle at the mid-inferior orbital rim
   (d) Between the periosteum and the orbital septum at the lateral canthus
10. Which statement is true?
   (a) Using electrocautery to reduce the orbital fat volume requires opening of the orbital septum.
   (b) Using CO_2 laser to reduce the orbital fat volume requires opening of the orbital septum.
   (c) When reducing pseudofat herniation during blepharoplasty with CO_2 laser and/or electrocautery, the orbital septum is not opened/entered.
11. Which muscle is most commonly injured during blepharoplasty that leads to diplopia?
   (a) Superior rectus
   (b) Superior oblique
   (c) Inferior rectus
   (d) Inferior oblique
12. Descent of the malar fat pad leads to all of the following except?
   (a) Rounding of the lower eyelid
   (b) Retracted appearance to the lower eyelid
   (c) Hollowed appearance to the infraorbital area
   (d) Shallow nasolabial fold
13. Select the false statement:
   (a) Canthopexy is anchoring the lateral canthal tendon to the inside of the orbital rim without lysis of the tendon.
   (b) Canthoplasty is anchoring the lateral canthal tendon after lysis of the tendon.
   (c) The anchoring stitch for lateral canthal anchoring is placed at the height of the superior limbus.
   (d) The ideal lateral scleral triangle is larger, wider, and more pointed than the medial scleral triangle.
14. When working up blepharoptosis, the normal margin reflex distance for the upper eyelid (MRD-1) is:
   (a) 1–2 mm
   (b) 4–5 mm
   (c) 5–10 mm
   (d) 10 mm or greater
15. Medical management for ectropion as a complication of a recent blepharoplasty includes all of the following except?
   (a) Taping
   (b) Massage
   (c) Traction on the lower eyelid in the inferior direction
   (d) Artificial tear ointment/drops
16. The lid distraction test is best described as:
   (a) Pulling the lower eyelid away from globe and determining how quickly the eyelid returns to original position
   (b) Determining if there is a change in visual field testing while pulling up the upper eyelid
   (c) Measuring how far the lower eyelid can be pulled away from globe
   (d) Measuring how far the lower eyelid can be pulled away from the upper eyelid
17. The snap test is best described as:
   (a) Pulling the lower eyelid away from globe and determining how quickly the eyelid returns to original position
   (b) Determining if there is a change in visual field testing while pulling up the upper eyelid
   (c) Measuring how far the lower eyelid can be pulled away from globe
   (d) Measuring how far the lower eyelid can be pulled away from the upper eyelid
18. The Schirmer's test is best described as:
   (a) Application of fluorescein dye to the eye followed by examination of the cornea with blue light

(b) A method to test for visual field defects
(c) Placing a strip of filter paper at the superior fornix and measuring the length of wetting
(d) Placing a strip of filter paper at inferior fornix and measuring the length of wetting

19. Which of the following describes the ideal brow position:
    (a) In men, slightly above the orbital rim. In women, at the level of the orbital rim
    (b) In men, at the orbital rim. In women, slightly above the orbital rim and more curved with the peak at the mid-pupillary line
    (c) In men, at the orbital rim. In women, slightly above the orbital rim and more curved with the peak at the lateral limbus
    (d) In men, at the orbital rim. In women, slightly above the orbital rim and more curved with the peak at the medial limbus
20. The most common type of acquired blepharoptosis is:
    (a) Mechanical
    (b) Aponeurotic
    (c) Myogenic
    (d) Neurogenic
21. The ideal position of the lateral canthus is:
    (a) 1–2 mm above that of the medial canthus
    (b) 2–4 mm above that of the medial canthus
    (c) At the superior edge of the pupil
    (d) Answer
22. Which of the following is false?
    (a) The fascial sheath of the inferior oblique muscle extends laterally to separate the central and lateral fat pads.
    (b) The inferior oblique muscle separates the medial and central fat pads.
    (c) The inferior rectus muscle separates the medial and central fat pads.
    (d) The inferior oblique muscle is innervated by cranial nerve III.
23. A common teaching is to maintain approximately how much eyelid skin above the superior incision of an upper eyelid blepharoplasty?
    (a) 1–3 mm
    (b) 3–5 mm
    (c) 5–7 mm
    (d) At least 10 mm
24. Which of the following is recommended to reduce the risk of webbing in an upper blepharoplasty?
    (a) Avoid extending the incisions medial to the medial limbus.
    (b) Avoid extending the incisions medial to the medial canthus.
    (c) Avoid using absorbable sutures for skin closure.
    (d) Avoid resecting any orbicularis oculi muscle.
25. Negative vector is best described as:
    (a) The lateral canthus is more superior than the medial canthus.
    (b) The medial canthus is more superior than the lateral canthus.
    (c) The most anterior projection of globe is posterior to the lid margin and to the malar eminence.
    (d) The most anterior projection of globe is anterior to the lid margin and to the malar eminence.

Answers

1. (c)
2. (b)
3. (a)
4. (b)
5. (a)
6. (d)
7. (b)
8. (a)
9. (b)
10. (c)
11. (d)
12. (d)
13. (c)
14. (b)

15. (c)
16. (c)
17. (a)
18. (d)
19. (c)
20. (b)
21. (a)
22. (c)
23. (d)
24. (b)
25. (d)

References

1. Friedland JA, Lalonde DH, Rohrich RJ. An evidence based approach to blepharoplasty. Plast Reconstr Surg. 2010;126:2222–9.
2. Goldberg RA, McCann JD, Flaschetti D, Ben Simon GJ. What causes eyelid bags? Analysis of 114 consecutive patients. Plast Reconstr Surg. 2005;115(5):1405–12.
3. Papel ID. Lower eyelid blepharoplasty for the senile lid. In: Papel ID, Nachlas NE, editors. Facial plastic and reconstructive surgery. St. Louis: Mosby Year Book; 1992. p. 179–83.
4. Pastorek NJ. Upper lid blepharoplasty. In: Papel ID, Nachlas NE, editors. Facial plastic and reconstructive surgery. St. Louis: Mosby Year Book; 1994. p. 158–68.
5. Putterman AM, Urist ML. Surgical anatomy of the orbital septum. Ann Ophthalmol. 1974;6:290–303.
6. Susan R, Carter MD. The International Ophthalmology Clinics; Advancing in Ophthalmic Plastic Surgery volume 42 (2) spring issue 2002. Ptosis in the elderly patient. Philadelphia: Lippincott Williams and Wilkens. p. 61–74.
7. Yousif NF, Sonderman P, Dzwierzynski WW, Larson DL. Anatomic considerations in transconjunctival blepharoplasty. Plast Reconstr Surg. 1995;96(6):1271–8.
8. Doxanas MT, Anderson RL. Oriental eyelids: an anatomic study. Arch Ophthalmol. 1984;102(8):1232–5.
9. Muzzaffer AR, Mendelson BC, Adams WP. Surgical anatomy of the ligamentous attachments of the lower lid and lateral canthus. Plast Reconstr Surg. 2002;110(3):873–84.
10. Becker BB, Berry FD. Eyelid level after lower lid blepharoplasty with skin excision. Arch Otolaryngol Head Neck Surg. 1992;118:959–62.
11. Brennan HG, Joseph BA. Delineating the cleft in upper blepharoplasty. Arch Otolaryngol Head Neck Surg. 1979;105(9):515–8.
12. Halverson EG, Husni NR, Pandya SN, Seckel BR. Optimal parameters for marking upper blepharoplasty incisions: a 10 year experience. Ann Plast Surg. 2006;56(5):569–72.
13. Holt JE, Holt GR. Blepharoplasty: indications and preoperative assessment. Arch Otolaryngol Head Neck Surg. 1985;111(6):394–7.
14. Karesh JW. Diagnosis and management of acquired blepharoptosis and dermatochalasis. In: Facial plastic surgery. New York, NY: Thieme; 1994. p. 185–201.
15. Lee JS, Park WJ, Shin MS, Song IC. Simplified anatomic method of double-eyelid operation: septodermal fixation technique. Plast Reconstr Surg. 1997;100(1):170–81.
16. Rees TD. Aesthetic surgery the neck and face. In: Rees TD, editor. Aesthetic plastic surgery. Philadelphia, PA: W. B. Saunders Company; 1980.
17. Rohrich RJ, Coberly DM, Fagien S, Stuzin JM. Current concepts in aesthetic upper blepharoplasty. Plast Reconstr Surg. 2004;113(3):e32–42.
18. Barton FE, Ha R, Awada M. Fat extrusion and septal reset in patients with the tear trough triad: a critical appraisal. Plast Reconstr Surg. 2004;113(7):2115–22.
19. Fagien S. Advanced rejuvenation upper blepharoplasty: enhancing aesthetics of the upper periorbita. Plast Reconstr Surg. 2002;110(1):278–92.
20. Pastorek N. Upper lid blepharoplasty. Facial Plast Surg Clin North Am. 1995;3(2):143–57.
21. Kamer FM, Mingrone MD. Experiences with transconjunctival upper blepharoplasty. Arch Facial Plast Surg. 2000;2(3):213–6.
22. De Castro CC. A critical analysis of the current surgical concepts for lower blepharoplasty. Plast Reconstr Surg. 2004;114(3):785–93.
23. Garcia RE, McCollough EG. Transcutaneous lower eyelid blepharoplasty with fat excision; a shift-resisting paradigm. Arch Facial Plast Surg. 2006;8(6):374–80.
24. Honrado CP, Pastorek NJ. Long-term results of lower-lid suspension blepharoplasty: a 30-year experience. Arch Facial Plast Surg. 2004;63(3):150–4.
25. Pastorek NJ. Blepharoplasty, SIPAC, AAO-HNS, 1994.
26. Perkins SW, Dyer WK, Simo F. Transconjunctival approach to lower eyelid blepharoplasty: experience and technique in 300 patients. Arch Otolaryngol Head Neck Surg. 1994;120(2):172–7.
27. Perkins SW, Batniji RK. Rejuvenation of the lower eyelid complex. Facial Plast Surg. 2005;21(4):279–85.
28. Baker SR. Orbital fat preservation in lower-lid blepharoplasty. Arch Facial Plast Surg. 1999;1:33–7.
29. Hamra ST. Arcus marginalis release and orbital fat preservation in midface Rejuvenation. Plast Reconstr Surg. 1995;96:354–62.
30. Persichetti P, DiLella F, Delfino S, Seuderi N. Adipose compartments of the upper eyelid: anatomy applied to blepharoplasty. Plast Reconstr Surg. 2004;113(1):379–80.
31. Glat PM, Jelks GW, Jelks EB, et al. Evolution of the lateral canthoplasty: techniques and indications. Plast Reconstr Surg. 1997;100(6):1396–405.

32. Jacobs SW. Prophylactic lateral canthopexy in lower blepharoplasties. Arch Facial Plast Surg. 2003;5(3):272–5.
33. McCord CD, Boswell CB, Hester TR. Lateral canthal anchoring. Plast Reconstr Surg. 2003;112(1):222–37.
34. Hamra ST. The role of septal reset in creating a youthful eyelid-cheek complex in facial rejuvenation. Plast Reconstr Surg. 2004;113(7):2124–41.
35. Prado A, Andrades P, Danilla S, Castillo P, Benitez S. Nonresective shrinkage of the septum and fat compartments of the upper and lower eyelids: a comparative study with carbon dioxide laser and Colorado needle. Plast Reconstr Surg. 2006;117(6):1725–35.
36. Fernandez LR. The East Asian eyelid: open technique. Clin Plast Surg. 1993;20(2):247–53.
37. McCurdy JA Jr. Upper blepharoplasty in the Asian patient: the "double eyelid" operation. Facial Plast Surg Clin North Am. 2005;13(1):47–64.
38. Shields M, Putterman A. Blepharoptosis correction. Curr Opin Otolaryngol Head Neck Surg. 2003;11(4):261–6.
39. Hirmand H, Codner MA, McCord CD, Hester TR, Nashai F. Prominent eye: operative management in lower lid and midfacial rejuvenation and the morphologic classification system. Plast Reconstr Surg. 2002;110(2):620–8.
40. Adamson PA, Constantinides MS. Complications of blepharoplasty. Facial Plast Surg Clin North Am. 1995;3(1):211–21.
41. Glavas LP. The diagnosis and management of blepharoplasty complications. Otolaryngol Clin N Am. 2005;38(5):1009–21.
42. McGraw BL, Adamson PA. Postblepharoplasty ectropion. Arch Otolaryngol Head Neck Surg. 1991;117:852–6.
43. Patipa M, Patel BCK, McLeish W, Anderson RL. Use of hard grafts for treatment of postsurgical lower eyelid retraction: a technical overview. J Craniomaxillofac Trauma. 1996;2(3):18–28.
44. Patipa M. Evaluation and management of lower eyelid retraction following cosmetic surgery. Plast Reconstr Surg. 2000;106(2):438–59.
45. Thornton WR. Combined approach to eyelid surgery in Graves' disease. Facial Plast Surg. 1994;10(2):172–6.
46. Wilkins RB, Byrd WA. Complications of blepharoplasty. Ophthal Plast Reconstr Surg. 1985;1(3):195–8.

Check for updates

18 Browplasty

Boris Paskhover and Virginia P. Teti

The facial plastic surgeon must appreciate the contribution of the brow position in conjunction with the upper eyelid skin redundancy when evaluating periorbital aging.

Anatomy

- *Subunits*: The forehead represents the upper third of the face, and the anatomic boundaries include the trichion or anterior hairline superiorly, the supraorbital rim and nasion inferiorly, and the temporal line laterally.
- *Vascularity*: Branches of the external and internal carotid provide vascularity to the forehead. Namely, the superficial temporal artery and subsequent zygomaticotemporal branches provide blood supply to the lateral forehead. Centrally, the internal carotid via the ophthalmic artery provides the supratrochlear and supraorbital branches, located approximately 1.7 cm and 2.7 cm from the midline, respectively.
- *Innervation*:
  - *Sensory*: All branches of the trigeminal nerve provide sensation to the forehead. Central forehead sensation is supplied by the ophthalmic (V1) division of the trigeminal nerve via the supraorbital and supratrochlear nerves. Laterally, the lacrimal branch of V1, the zygomaticofacial branch of V2, and the auriculotemporal branch of V3 provide sensation.
  - *Motor*: The temporal branch of the facial nerve provides all motor innervation. The temporal branch exits the parotid and courses superiorly from a point 1.5 cm below the external auditory canal, crossing the zygoma approximately 2.5 cm anterior to the auditory canal, and traverses superomedially toward to the frontalis muscle, approximately 2 cm lateral to the lateral orbital rim. The temporal or frontal branch of the facial nerve traverses within the temporoparietal fascia, prior to innervating the frontalis muscle from below. Endoscopically, the zygomaticotemporal vein, otherwise known as the "sentinel" vein, may serve as a landmark of the temporal branch of the facial nerve; the nerve typically runs immediately superficial to this vessel within 2–10 mm.
- *Layers*: The forehead layers are extensions of the scalp. The mnemonic SCALP reminds us of the surgical layers of the scalp from superfi-

B. Paskhover (✉)
Department of Otolaryngology, Division of Facial Plastics and Reconstructive Surgery, New York University Langone Medical Center, New York, NY, USA
e-mail: Boris.Paskhover@nyumc.org

V. P. Teti
Department of Otolaryngology, Naval Medical Center Portsmouth, Portsmouth, VA, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_18

cial to deep, namely, skin, subcutaneous tissue, aponeurosis of the galea and frontalis muscle, loose connective tissue, and the pericranium. Laterally, the galeal layer is continuous with the temporoparietal fascial layer (superficial temporal fascia), which is continuous with the superficial musculoaponeurotic system (SMAS) inferiorly. The deep temporal fascia is continuous with the periosteum of the forehead and parotidomasseteric fascia of the face.

- *Muscles*: The predominant muscle of the forehead is the anterior belly of the occipitofrontalis muscle. The vertical orientation of the frontalis muscle creates the transversely oriented relaxed skin tension lines (RSTL) of the forehead. The primary brow elevator is the frontalis muscle. Brow depressors include the paired corrugator supercilii, procerus, and orbicularis oculi muscles. The corrugator supercilii muscles form the glabellar vertical rhytids, and the procerus muscle forms the transverse glabellar rhytids. Lateral rhytids at the lateral canthus, otherwise known as crow's feet, appear secondary to orbicularis oculi contraction (Fig. 18.1)

Aesthetics of the Orbital Complex and Brow Positioning

The ideal brow shape and position vary depending on gender, age, culture, and current aesthetic trends (Fig. 18.2).

- *Women*:
  - The medial brow begins at a vertical line drawn through the medial canthus extending to the alar-facial junction with a club shape medially and a gradual taper laterally.

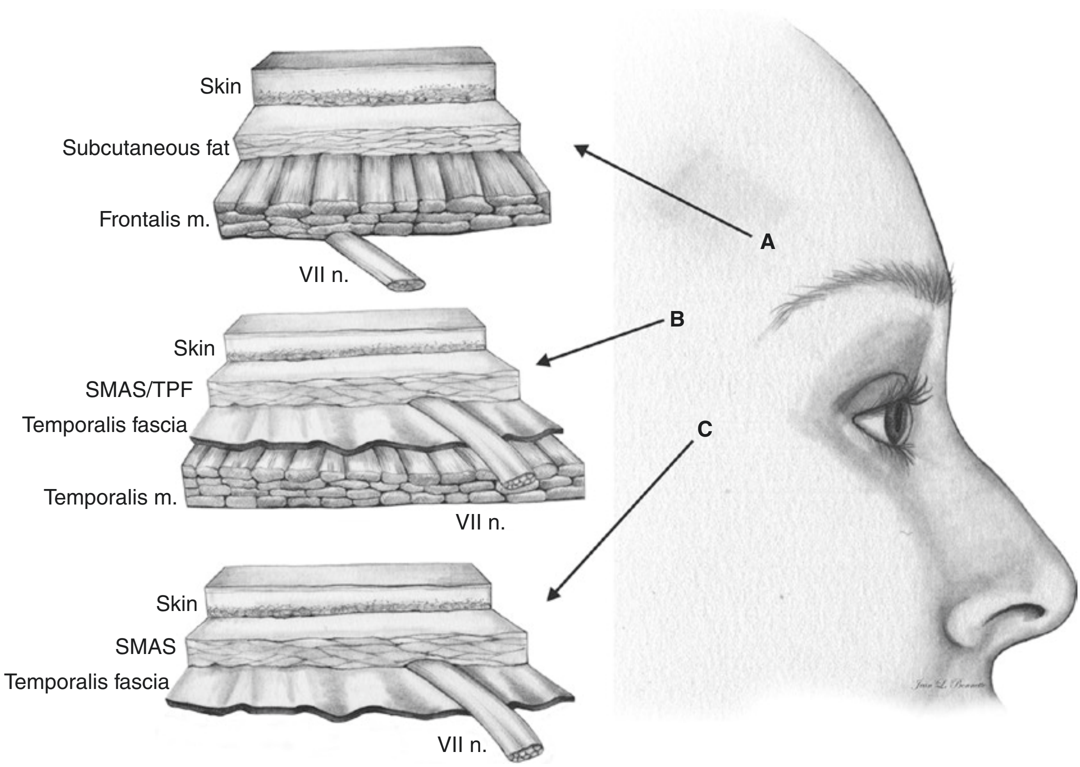


Fig. 18.1 Pathway of the frontal branch of the facial nerve. Note the frontal branch of the facial nerve traverses beneath the frontalis muscle in the forehead (*area* **a**); yet in the temple, the nerve is intimately associated with the temporoparietal fascial layer (*area* **b**) and within close proximity to the overlying skin; while traversing over the zygomatic arch (*area* **c**)

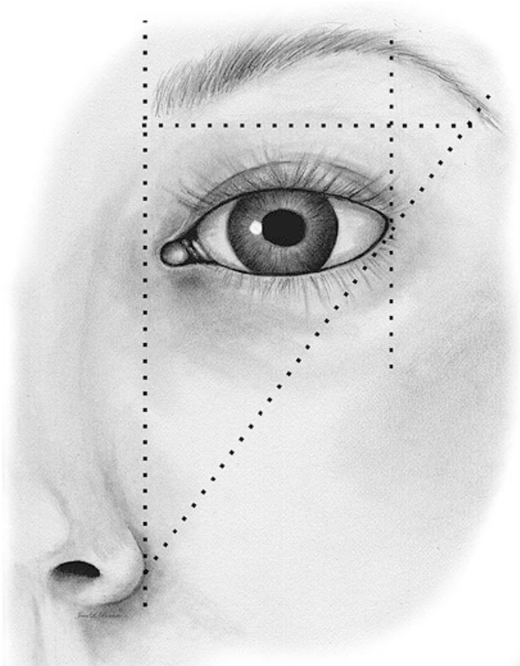


Fig. 18.2 The ideal female brow. Note the apex of the brow is tangent to the lateral canthus

– The apex of the brow typically arches above the lateral limbus, but some may prefer a slightly more lateral arch.
– The lateral brow ends along an oblique line drawn through the alar-facial junction and lateral canthus while resting on the same horizontal plan as the medial brow.
– The average height of the female brow from eyebrow to hairline is typically between 5 and 6 cm. Forehead heights greater than 7 cm may be aesthetically improved with forehead shortening maneuvers.

- *Men*:
  – The male brow shape should assume a more transverse orientation and rest along the orbital rim.
  – The male brow is thicker and flatter than the female brow.

Preoperative Analysis: Anatomic Considerations

Individuals with ptotic eyebrows often involuntarily attempt to elevate the brow via contraction of the frontalis muscle which should be noted. Only then may the surgeon accurately assess the actual position of the brow and the degree of upper eyelid dermatochalasis. Overaggressive resection of the brow or upper eyelid skin may result in further brow ptosis, short upper eyelid syndrome, and lagophthalmos. As a general rule, leaving 25 mm of the skin between the lid margin and brow prevents any issues with eye closure.

When determining which brow-lifting procedure to perform, one must consider specific factors of the patient:

- Location of frontal and temporal hairline along with patient's preferred hair style
- Quality of hair—alopecia, thinning, or abundant
- Forehead height relative to facial proportions
- Eyebrow aesthetics—shape, symmetry, quality, position, and mobility
- Degree of dermatochalasis of the upper eyelids, lateral canthal hooding, and medial fat pseudoherniation
- Presence of eyelid ptosis or eyelid lagophthalmos
- History of dry eyes, prior eye surgery or blepharoplasty, and thyroid disorders

Nearly all patients have some aspect of facial asymmetry which is important to be discussed with the patient. Passive and active asymmetries of the brow must be noted preoperatively. Dynamic eyebrow asymmetries are typically better addressed nonsurgically. Surgeons must take caution if attempting to correct a static brow asymmetry, as these subtle changes may alter the patient's unique facial characteristics.

Forehead bony contour should be noted. Women with frontal bossing or prominent supraorbital rims may appear masculinized. Bone reduction or alloplastic augmentation techniques may be considered.

In general, the brow elevation procedure is performed first before an upper lid blepharoplasty, if indicated. Often, after correction of the brow position, the amount of upper lid eyelid redundancy is significantly reduced, allowing the surgeon to accurately assess the need for blepharoplasty, thereby limiting the risk of lagophthalmos.

Similar to other facial plastic surgery procedures, accurate preoperative and postoperative measurements and photographic documentation are essential for introspective critique and medicolegal security. Typical views include the frontal full face, lateral, oblique, and frontal views with brows at rest, raised, and frowning.

Selection of Browplasty Techniques

A variety of procedures and incisions exist designed to improve the brow position from which the facial plastic surgeon must select based on individual anatomic and aesthetic considerations.

- A chemical brow lift is a nonsurgical therapeutic option used to elevate the temporal brow by using Botox (Allergan, Irvine, CA) to selectively denervate the temporal brow depressors, the lateral orbicularis muscle. Unopposed elevation by the frontalis muscle leads to brow elevation, typically providing 3–4 mm in vertical height (Fig. 18.3).

Surgical Techniques

In general, the techniques for surgical rejuvenation of the upper face may be considered within three categories: the open lift and modifications, the direct brow lift and modifications, and the endoscopic brow lift. The indications, advantages, and disadvantages are listed in the chart along with details of the surgical techniques described below (Table 18.1).

Coronal Forehead Lift

- The coronal lift remains a reliable method to address a forehead with short vertical height

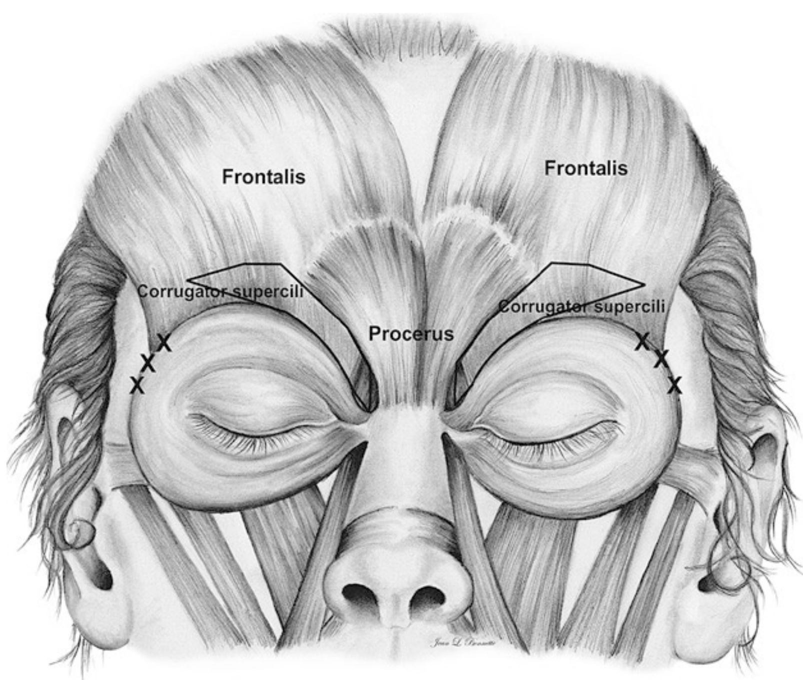


Fig. 18.3 Muscular anatomy of the brow and forehead. *X* indicates the lateral orbicularis oculi muscle and site for injection of Botox to elevate the temporal brow

Table 18.1 Surgical indications and considerations for brow lifting

Procedure	Indications	Advantages	Considerations and disadvantages
Browpexy, transblepharoplasty	Mild brow ptosis	Performed via upper lid blepharoplasty	Possible to control for brow asymmetry. Mainly used for lateral brow elevation
Direct brow lift	Correct brow asymmetry, unilateral brow ptosis, pale nonsebaceous skin	Most accurate brow elevation, preserves forehead sensation, favorable scar camouflage in patience with nonsebaceous skin	Visible scar, sharply defined suprabrow, only treats the brow with limited glabella exposure
Midforehead lift	Correct brow asymmetry, patients with thick horizontal forehead rhytids and androgenic baldness	Precise brow elevation, useful in patients with horizontal forehead creases, preserves the hairline	Visible scar, avoid in oily, thick skin
Endoscopic brow lift	Less invasive brow and forehead lift	Excellent scar camouflage, preserves hairline	Steep learning curve
Trichophytic forehead lift	High forehead	Reduces vertical height of high forehead, preserves hairline, great for patients that prefer bangs	Visible scar, prolonged hypesthesia of scalp hairline alopecia
Coronal forehead lift	Forehead and brow lift, low forehead	No visible scars, increases vertical height of low forehead	Elevates the hairline, vertically lengthens the upper third of the face, possible visible scar with very large incision, less precise manipulation of brow position

with significant brow ptosis and forehead rhytids.

- *Technique*: The surgical curvilinear incision is placed approximately 4–6 cm posterior to the hairline and designed in a beveled fashion to minimize follicle injury by paralleling them.
- A subgaleal dissection is performed until 1–2 cm above the supraorbital rim. Laterally, blunt dissection proceeds above the superficial layer of the deep temporal fascia. The flap is dissected over the supraorbital rim to release the arcus marginalis in a subperiosteal plane.
- Myoplasty of the corrugator and procerus muscles may be performed bluntly with care to avoid the supratrochlear artery and nerve traversing around and through the corrugator. The frontalis muscle is identified in the flap, and unipolar cautery may be used to incise the muscle and galea immediately deep to the horizontal forehead crease, with care to remain medial to the midpupillary line to avoid injury to the temporal branch of the facial nerve. Lastly, excision of frontalis or procerus muscle is commonly avoided to prevent contour irregularities, and many surgeons prefer botulinum toxin instead.
- The flap is advanced superiorly and posteriorly with an appropriate amount of redundant skin excised parallel to the hair follicles. The incision is closed in layers, and a suction drain may be used for 24 h.

• Disadvantages of this technique include the elevation of the anterior hairline, temporary or permanent hypesthesia or paresthesia posterior to the incision, and the risk for hematoma formation. Also, a rather large incision is made, and even in skilled hands, this can be noticeable especially when women's hair gets wet.
• Due to the distance between the incision and the brow, the coronal lift and its modifications are not routinely used to correct precise brow asymmetries.

Trichophytic Forehead Lift

- The pretrichial forehead lift can offer a means of shortening the vertical height of the forehead in patients with a high hairline. The procedure offers the benefit of reducing the redundant forehead skin without raising the anterior hairline (Fig. 18.4).
  - Technique: The incision is designed in an irregular fashion approximately two or three hair follicles behind the anterior hairline. The incision is beveled parallel to hair follicle shaft to allow ingrowth of hair and designed at the junction of the cephalad forehead and anterior hairline to optimize scar camouflage. The pretrichial incision is extended laterally in the temporal region posterior to the hairline.
  - The flap is elevated in a similar fashion to the coronal lift with similar options to perform myoplasty, if desired.
- Disadvantages include the visible scar despite meticulous closure and the broader area of anesthesia posterior to the incision.

Temporal Lift

- This lift is designed to address the lateral one-third of the brow and indicated primarily for female patients with appropriate medial brow position and isolated lateral brow ptosis.

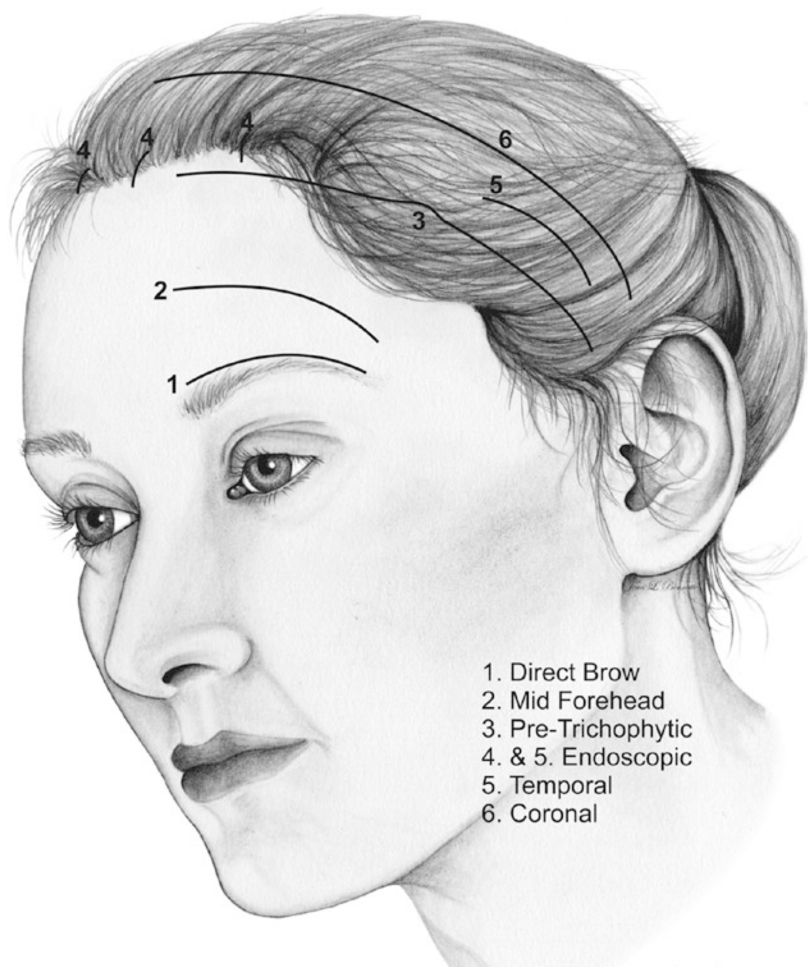


Fig. 18.4 Proposed incision placement for brow lift procedures in oblique view: *1*. Direct brow. *2*. Midforehead. *3*. Pre-trichophytic. *4*, *5*. Endoscopic port sites and temporal incision. *5*. Temporal lift. *6*. Coronal

  - *Technique*: The incision is typically 2 cm posterior to the temporal hairline, and dissection proceeds along the superficial layer of the deep temporal fascia. The dissection extends inferiorly to identify the lateral orbital rim with release of the arcus marginalis, conjoint tendon, and temporal line to enable lateral brow mobility. Use of the endoscope is commonly practiced to improve visualization of the zygomaticotemporal (sentinel) vein and adequate release of the arcus marginalis. Finally, lateral suspension is directed superiorly and posteriorly and secured with suture secured from the deep temporal fascia to the temporoparietal fascia. The scalp is closed in layers.

Midforehead Brow Lift

- The midforehead brow lift may be considered in males with deep horizontal rhytids and a receding anterior hairline.
  - *Technique*: An elliptical incision is designed around the rhytid with two-thirds of the ellipse above the incision and one-third below. Two different horizontal rhytids are selected to stagger the incisions; however, some surgeons prefer one continuous incision across the brow, often requiring an intentional irregularization to improve scar camouflage.
  - The overlying skin and subcutaneous tissue are excised until the frontalis muscle is exposed. Dissection proceeds in the subcutaneous plane until the orbicularis oculi muscle or supraorbital rim is exposed.

Direct Brow Lift

- The direct brow is commonly indicated to modify an asymmetric or ptotic brow especially in patients with unilateral facial paralysis. Technically, this method requires minimal undermining with relative low risk of damage to the supratrochlear and supraorbital neurovascular bundles. The control of the brow positioning is direct, yet it requires precise placement of permanent suspension sutures to the superior periosteum.
  - *Technique*: An ellipse of the skin/subcutaneous tissue is resected from the suprabrow area to elevate the brow approximately 4–8 mm above the opposite eyebrow. The inferior border of the skin ellipse is incised along the superior margin of the eyebrow parallel to the axis of the hair shafts, with great care to avoid injury to the hair follicles of the brow. The incision is beveled to maximize scar eversion. Alternatively, the ellipse of skin incision may be designed in a prominent midforehead rhytid in an attempt to camouflage the incision, utilizing the same surgical technique as described above.

Browpexy

- This procedure is typically indicated in women with mild-to-moderate lateral brow ptosis.
  - *Technique*: A standard blepharoplasty excision of the skin and orbicularis muscle is performed followed by dissection superiorly toward the brow in a submuscular orbicularis plane, extending above the superior orbital rim. Permanent suture is then used to grab submuscularly at the level of the infrabrow hairs and then passed through the periosteum above the supraorbital rim. With tightening of the suture, the eyebrow will be elevated. Precise placement and tension of each suture may allow modification of the eyebrow position based on the degree of elevation desired. The blepharoplasty procedure is then completed.
- Risk of this procedure involves potential injury to the supraorbital neurovascular bundle resulting in bleeding or forehead anesthesia. Additionally, due to the meticulous suspension suture placement, dimpling of the thinner eyelid skin, overcorrection with lagophthalmos, and inadequate correction of brow ptosis have been described. Preoperative botulinum toxin helps to prevent suture displacement postoperatively.

Endoscopic Brow Lift

- The indications for the endoscopic brow lift are similar to the coronal lift with the advantage of direct endoscopic visualization and magnification and perhaps improved identification of the supraorbital and supratrochlear neurovascular bundles. Key principles of endoscopic brow lift include subperiosteal dissection and adequate and *complete release of the arcus marginalis*.
  - *Technique*: Typical incision placement includes bilateral paramedian incisions tangent to the midbrow that are typically 2 cm in length and located 1–2 cm posterior to the anterior hairline. Additionally, two temporal incisions are marked in an elliptical fashion obliquely oriented from the alar-facial junction, through the lateral canthus, and parallel to and approximately 1–1.5 cm posterior to the hairline. These locations may vary depending on surgeon preference.

    Dissection: The central scalp and paramedian incisions are elevated anteriorly and posteriorly in a subperiosteal plane, typically in a blunt fashion, and dissections proceed to a level 1–2 cm above the supraorbital rim, as the neurovascular bundle and/or branches can emanate from a true foramen above the supraorbital rim in approximately 10% of cases. The endoscope is then introduced to allow improved visualization of the supratrochlear and supraorbital neurovascular bundles while releasing the arcus marginalis over the orbital rim and onto the nasion. Laterally, the temporal incisions are dissected similarly to the lateral brow lift as described above. Of note, the sentinel vein lies in close proximity to the temporal branch of the facial nerve, and cautery should be applied with great care at the deep aspect of the vessel to avoid overlying nerve injury. The temporal line is divided from lateral to medial to connect the two planes. Transverse releasing incisions are made through the periosteum along the supraorbital margin to allow appropriate brow mobility and release.

    Fixation: Two commonly used methods of fixation include sutures secured through cortical bone tunnels and cortical anchors (Endotine Coapt System, Palo Alto, CA). Sclafani et al. reported that a minimum of 6 weeks is required for adhesion between the cranium and overlying periosteum. Additionally, Thomas et al. demonstrated in the rabbit model that in 8 weeks the biomechanical strength of the dissected subperiosteal versus subgaleal flaps was similar to matched, undissected controls.
  - The temporal incisions are closed as described in the temporal lift, and staples are utilized to close the midline and paramedian anterior scalp incisions.

Complications

- Substantial heterogeneity exists in the reporting of paresthesia in the literature (specifically, nerve distribution, severity, and duration of paresthesia). Paresthesia/dysesthesia most frequently occurs with anterior hairline and mid-forehead techniques.
- The anterior hairline approach in the subcutaneous plane demonstrated the overall highest probability of alopecia.
- Infection or abscess formation is unlikely to occur in any of the techniques studied (0.2–0.4% incidence).
- Loss of fixation.
- Asymmetry.
- Alopecia.

Questions

Browplasty Review Questions

1. Which of the following techniques will best be suited for a patient with a long forehead who also desires their hairline lowered?
   (a) Midforehead lift.
   (b) Direct brow lift
   (c) Endoscopic brow lift
   (d) Pretrichial lift

   (e) Coronal lift
2. While performing a midforehead lift, indicate the proper plane of dissection.
   (a) Subcutaneous
   (b) Subfrontalis
   (c) Subgaleal
   (d) Subperiosteal
3. A patient undergoes botulinum toxin injections into her bilateral corrugators and procerus, which of the following activities can she still enjoy immediately post-procedure?
   (a) Face down massage
   (b) Swimming with her goggles on
   (c) Skiing with her ski mask
   (d) Swimming without goggles
4. A younger patient presents to your office with mild-to-moderate lateral hooding of her upper eyelids secondary to lateral brow ptosis. She is reluctant to undergo surgical correction of her brows. To improve her condition, you offer her:
   (a) Reassurance. Gently tell the patient that no intervention is warranted.
   (b) Botulinum toxin A—to forehead, 20 units
   (c) Botulinum toxin A—to procerus, corrugator supercilii m, 20 units
   (d) Botulinum toxin A—to lateral orbicularis oculi m, 10 units
5. A 63-year-old female presents to your office with moderate lateral brow ptosis and desires an upper blepharoplasty without "going to sleep." What procedure can you safely offer?
   (a) Trichophytic lift
   (b) Coronal lift
   (c) Endoscopic temporal lift
   (d) Direct brow lift
   (e) Transblepharoplasty brow lift
6. While performing a temporal brow lift, you encounter significant bleeding in the vicinity of the sentinel vein. How do you manage?
   (a) Wide bipolar cautery on a low setting
   (b) Unipolar cautery
   (c) Hold pressure, consider using Afrin-soaked pledgets and reassess, and consider minimal cautery.
   (d) Place several figure 8 sutures through the area.
7. Which of the following techniques will allow the most accurate eyebrow lift with the greatest elevation per millimeter of tissue excised and is optimal in the facial paralysis patient?
   (a) Direct eyebrow lift
   (b) Midforehead lift
   (c) Temporal lift
   (d) Endoscopic lift
8. A dark haired patient presents having undergone a previous coronal brow lift with a main complaint that her incision is very visible whenever she is swimming. The thin incision has not widened or scarred significantly. Which of the following options are not reasonable?
   (a) Dermatographia
   (b) Hair transplantation to the incision line
   (c) Scar excision
9. A 60-year-male presents requesting improvement of his brow position; he has a Norwood V hair pattern. Which of the following options for rejuvenation should be avoided?
   (a) Direct brow lift
   (b) Transblepharoplasty brow lift
   (c) Coronal lift
   (d) Chemical Brow lift
   (e) Midforehead brow lift
10. A patient undergoes an endoscopic brow lift with endotines placed; after the dressing is placed, you notice that the brow is now lower on the left. What is your next step?
   (a) Leave it alone; the dressing is just applying pressure downward.
   (b) Remove the dressings and inspect the incisions; they must have opened.
   (c) Gently check to see if the scalp is still engaged on the endotines; if not, carefully grasp the scalp and reposition.
   (d) Leave it alone; the asymmetry is expected with the Endotine approach.
11. During drilling for the cortical tunnels for suture-based fixation during endoscopic brow lift, the surgeon inadvertently allows it to travel deeper than expected, and significant bleeding is noted. What is your next step?
   (a) Emergent CT head
   (b) Bone wax

   (c) Cautery
   (d) No intervention is needed; it will stop on its own.

12. During the coronal brow lift, in which area should the dissection not proceed?
   (a) Anteriorly in the subgaleal plane
   (b) Posteriorly in the subgaleal plane
   (c) Anterior laterally on the deep temporal fascia
   (d) Within 2 cm of the brow in the subperiosteal plane
   (e) All of the above answers are appropriate dissection planes.
13. A patient undergoes an endoscopic brow lift with endotines placed; after the dressing is placed, you notice that the brows are slightly too high bilaterally. What is your next step?
   (a) Leave it alone; the dressing is just applying pressure upward.
   (b) Remove the dressings and inspect once again.
   (c) Grasp the scalp and reposition it to a lower position.
   (d) Leave it alone since a slight overcorrection is beneficial with the use of endotines.
14. A patient undergoes a trichophytic brow lift, and approximately 3 cm of the skin is excised. You are now having a hard time closing the incision without significant tension. What is your next step?
   (a) Use towel clips to apply mechanical creep and assist in closure.
   (b) Dissect further anteriorly down to the orbit if needed.
   (c) Dissect posteriorly in a subgaleal plane and perform galeotomies if needed.
   (d) Close under tension.
15. A patient undergoes an endoscopic brow lift with endotines placed, and they noted they can still feel the endotines at 3 weeks. What is your next step?
   (a) Reassurance, they will take several months to fully dissolve.
   (b) Tell the patient that they must be removed, and schedule the OR.
   (c) Inject the area with steroids.
   (d) Tell the patient that they are permanent, and this is normal to always feel them postoperatively.
16. A patient undergoes a transblepharoplasty brow lift, and at 1 week postoperatively, they note that they were having an argument with their significant other and all of a sudden felt their eyebrows get heavy on one side. Patient has noted unilateral brow ptosis. What is your next step?
   (a) Leave it alone; it will improve over time.
   (b) Perform botulinum toxin injections to help elevate the unilateral brow.
   (c) Perform an open brow lift since the patient is clearly not a candidate for browpexy procedures.
   (d) Tell the patient that the sutures fixating her brow must have come loose and a small revision procedure is needed.
17. A patient undergoes a transblepharoplasty brow lift, and at 1 week postoperatively, they note that they were having an argument with their significant other and all of a sudden felt their eyebrow get heavy on one side. Patient has noted unilateral brow ptosis. What would have prevented this complication?
   (a) Using preoperative botulinum toxin to prevent forehead movement in the healing period.
   (b) Wider undermining
   (c) Perform an open brow lift since the patient is clearly not a candidate for browpexy procedures.
   (d) None of the above
18. Which of the following is not an appropriate characteristic of the female brow?
   (a) The medial brow begins at a vertical line drawn through the medial canthus.
   (b) A club shape medially and a gradual taper laterally
   (c) The apex of the brow typically arches above the lateral limbus.
   (d) The lateral brow ends along an oblique line drawn through the alar-facial junction and lateral canthus while resting on the same horizontal plane as the medial brow.

(e) All of the above are female brow characteristics.

19. Which of the following incisions is not typically made during an endoscopic brow lift?
    (a) Temporal incisions
    (b) Vertically oriented parasagittal incisions
    (c) Horizontally oriented parasagittal incisions
    (d) All of the above are typically made.
20. What is the major benefit of not releasing the arcus marginalis during some brow-lifting procedures?
    (a) Faster healing
    (b) Maintains the patient's exact brow shape
    (c) Improves the amount of lifting possible
    (d) All of the above
21. What is the major downside of not releasing the arcus marginalis during some brow-lifting procedures?
    (a) Incisional dehiscence
    (b) Paresthesias
    (c) Limits the amount of lift possible
    (d) All of the above
22. A patient undergoes an endoscopic brow lift and notes diffuse hair loss postoperatively. What is your diagnosis?
    (a) Normal post-operative occurrence
    (b) The incisions were closed poorly and caused the hair loss.
    (c) The patient is likely losing their hair due to unrelated causes.
    (d) The patient is likely experiencing diffuse alopecia areata secondary to surgical stress.
23. Which component of the coronal brow lift closure is the most vital for ensuring the incision does not widen postoperatively?
    (a) Skin edge to skin edge closure
    (b) Galea to galea closure
    (c) Periosteum to periosteum closure
    (d) They equally contribute.
24. A patient undergoes an endoscopic brow lift that went smoothly without any issues. In the PACU, the patient is noted to have some right brow paresis. What do you do?
    (a) Urgent take back to the OR for re-anastomosis.
    (b) Possible local anesthetic effect, wait for resolution.
    (c) Neither
25. A patient undergoes an endoscopic brow lift that went smoothly without any issues. Three weeks postoperatively, the patient is noted to have some right brow paresis. What do you do?
    (a) Urgent take back to the OR for re-anastomosis.
    (b) Possible local anesthetic effect, wait for resolution.
    (c) Possible intraoperative facial nerve injury, take back to the OR for exploration and repair.
    (d) Possible intraoperative facial nerve injury, reassure the patient that there is a good chance it will improve over the next few weeks.

Answers

1. (d)
2. (a)
3. (d)
4. (d)
5. (e)
6. (c)
7. (a)
8. (c)
9. (c)
10. (c)
11. (a)
12. (b)
13. (d)
14. (c)
15. (a)
16. (d)
17. (a)
18. (e)
19. (c)
20. (b)
21. (c)
22. (e)
23. (b)
24. (b)
25. (d)

Disclaimer The views expressed in this article are those of the author and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, nor the U.S. Government.

Additional Resources

1. Byun S, Mukovozov I, Farrokhyar F, Thoma A. Complications of browlift techniques: a systematic review. Aesthet Surg J. 2013;33(2):189–200.
2. Byun S, Mukovozov I, Farrokhyar F, Thoma A. Complications of browlift techniques: a systematic review. Aesthet Plast Surg. 2013;33(2):189–200.
3. Chaiet SR, Williams EF. Understanding midfacial rejuvenation in the 21st century. Facial Plast Surg. 2013;29:40–5.
4. Cook TA, Brownrigg PJ, Wang TD, Quatela VC. The versatile midforehead browlift. Arch Otolaryngol Head Neck Surg. 1989;115:163–8.
5. Dyer WK, Yung RT. Botulinum toxin-assisted brow lift. Facial Plast Surg Clin. 2000;8(3):343–54.
6. Frodel JL, Maretette LJ. The coronal approach. Arch Otolaryngol Head Neck Surg. 1993;119:201–7.
7. Hamamoto AA, Liu TW, Wong BJ. Identifying ideal brow vector position: empirical analysis of three brow archetypes. Facial Plast Surg. 2013;29:76–82.
8. Henderson JL, Larrabee WF. Analysis of the upper face and selection of rejuvenation techniques. Otolaryngol Clin N Am. 2007;40:255–65.
9. Koch RJ, Troell RJ, Goode RL. Contemporary management of the aging brow and forehead. Laryngoscope. 1997;107:710–5.
10. Maas CS. Botulinum neurotoxins and injectable fillers: minimally invasive management of the aging upper face. Otolaryngol Clin N Am. 2007;40:283–90.
11. McKinney P, Mossie RD, Zukoski ML. Criteria for the forehead lift. Aesthet Plast Surg. 1991;15(2):141–7.
12. Sclafani AP, Fozo MS, Romo T III, et al. Strength and histological characteristics of periosteal fixation to bone after elevation. Arch Facial Plast Surg. 2003;5:63–6.
13. Thomas JR, Lee AS, Patel AB. Brow lift: subgaleal vs subperiosteal flap adherence in the rabbit model. Arch Facial Plast Surg. 2007;9:101–5.

19 Cheiloplasty

Yuan F. Liu, Jared C. Inman, Farhad Ardeshirpour, and Nikita Gupta

Anatomy

Limits

- Upper: nasal base
- Lower: mental crease
- Lateral: melolabial and labiomandibular creases

Tissue Layers (Fig. 19.1)

- Lip skin: contains hair, sebaceous glands, and eccrine glands
- Orbicularis oris muscle: sphincteric arrangement with decussation at commissures, modiolus where it is joined by buccinators, levator anguli oris, depressor anguli oris, zygomaticus major, risorius, platysma, and levator labii superioris
- Mucosa: relatively few salivary glands compared to the rest of the oral cavity

Arterial supply *superior/inferior labial artery* (courses horizontally in submucosal plane, deep to orbicularis oris, at about the level of anterior vermillion line; medially, upper division may rise slightly; both may have tortuous path through the muscle)

External carotid → facial artery→ labial artery

Venous outflow not well defined. Typically venae comitantes

- *Superior/inferior labial vein* → *facial vein* → *internal jugular* (*or* joins anterior branch of *retromandibular vein* to form *common facial vein*, which drains into *internal jugular*). Upper lip may have deep venous system separate from arterial.

Innervation upper lip, *infraorbital nerve* → *maxillary branch of trigeminal nerve (V2)*. Lower lip: *mental nerve* (emerges from mental foramen usually apical to second premolar or between apices of lower premolars) → *inferior alveolar nerve* → *mandibular branch of trigeminal nerve* (*V3*)

Y. F. Liu
Otolaryngology – Head and Neck Surgery, Loma Linda University Medical Center, Loma Linda, CA, USA

J. C. Inman
Otolaryngology/Head and Neck Surgery, Loma Linda University, Loma Linda, CA, USA

F. Ardeshirpour
Adresh Facial Plastic Surgery, Los Angeles, CA, USA

N. Gupta (✉)
Department of Otolaryngology-Head and Neck Surgery, Division of Facial Plastic and Reconstructive Surgery, University of Kentucky Chandler Medical Center, Lexington, KY, USA
e-mail: Nikita.Gupta@uky.edu

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_19

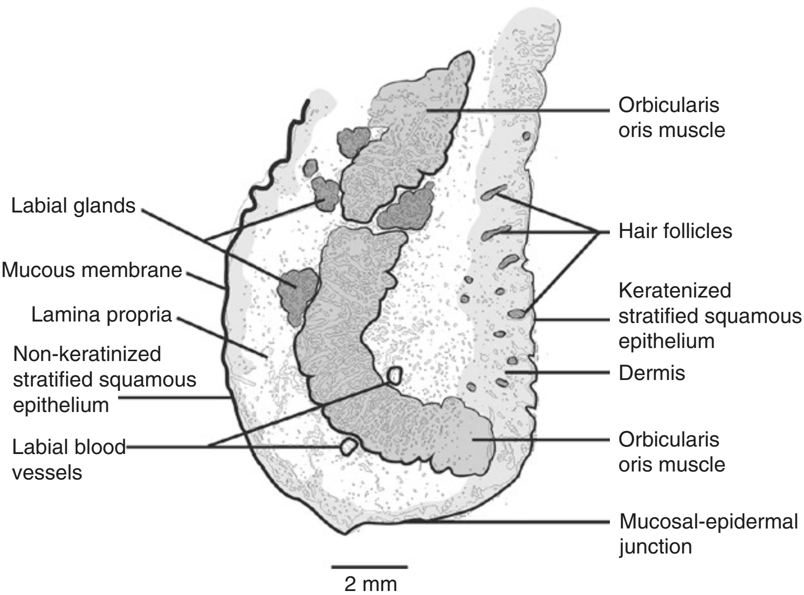


Fig. 19.1 Layers of the lip and structures contained in each layer

Histology Numerous septations and chambers are present within the upper and lower lips (lower > upper lip). These can explain congenital lip pits and post-lip augmentation complications particularly in the lower lip, such as nodule formation, rupture of the skin, or abscess formation. Increased septations found in women and patients > age 65.

Relaxed skin lines course radially, perpendicular to underlying orbicularis oris muscle

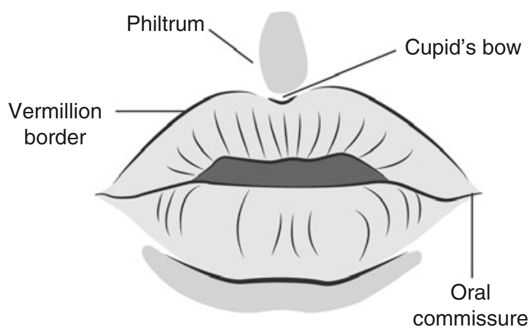


Fig. 19.2 Borders of the lip

Vermillion

- *External demarcation*: anterior vermillion (or mucocutaneous) line at transition to lip skin
- *Internal demarcation:* posterior vermillion (or wet) line, which separates dry and wet vermillion and is the innermost line of contact with lips closed
- Red color due to lack of keratinization and underlying capillary plexus
- Mostly devoid of hair follicles, sweat glands, and sebaceous glands

Philtrum embryologically derived vertical ridges of no functional, only aesthetic significance (wide/smooth philtrum associated with fetal alcohol syndrome)

Cupid's bow two high points of upper lip anterior vermillion line with V-shaped depression in between (Fig. 19.2)

White roll/line raised line of the skin at transition from anterior vermillion line to lip skin. Possibly functions as excess skin to allow complex movements of the lip

Embryology

Upper lip paired maxillary prominence fuse with paired medial nasal swellings. Gives rise to philtral ridges and Cupid's bow

Lower lip paired mandibular prominence fuse together. Less elastic than upper lip due to fewer fusion planes

Analysis and Physical Exam

Key aesthetic feature of the lower face. No ideal standard of beauty, and it varies for different ethnicities and perspectives. However, there are some proportions and features which may contribute to attractiveness. African Americans and Asians tend to have larger lips in all dimensions with increased projection.

Frontal View

- Distance between menton and subnasale = 1/3 distance from menton to hairline
- Distance between menton and subnasale = distance from subnasale to glabella
- Upper lip height = lower lip height = 1/3 distance from subnasale to menton (Fig. 19.3)

Lateral View

- The upper lip should fall 4 mm, and lower lip 2 mm, behind line tangent to pogonion and nasal tip inferior break (Fig. 19.4).
- Or the upper lip should lie 3.5 mm, and lower lip 2.2 mm, anterior to line from subnasale to pogonion.

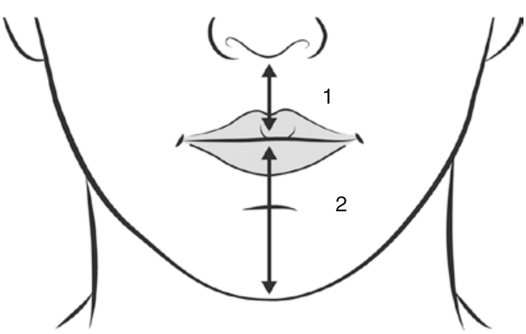


Fig. 19.3 Ratio of the upper lip to the lower lip. The distance from the subnasale to the junction of upper and lower lips is about half of the distance from the junction to the menton

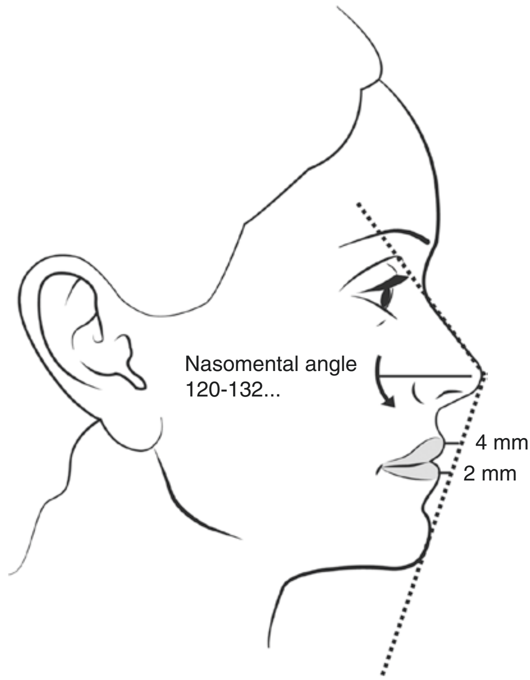


Fig. 19.4 Amount of upper and lower lip protrusion. The upper lip falls about 4 mm, and the lower lip 2 mm, behind the line drawn from the nasal tip to the pogonion

Aging

- Lip size increases in puberty due to muscular and glandular hypertrophy and then gradually decreases thereafter.
- White roll flattens.
- Blunting of Cupid's bow.
- Decreased vermillion show.
- Loss of projection.
- Downturning of commissures.
- Rhytids develop in vermillion and lip skin.

Exam should consist of all factors mentioned in aging, along with dental occlusion, philtrum definition, dental show, condition of epithelium, chin position, and size of the chin in proportion to lips. Must evaluate the teeth to assess the cause of lip over- or under-projection as well as other maxillary/mandibular protrusions or deficiencies

Volume the lower lip has about 50% greater volume than upper lip.

Fig. 19.5 Subnasal lip lift. (Photo courtesy of Jeffrey Spiegel, M.D)

Smoking changes increased risk in deep rhytids perpendicular to vermillion border, discoloration, and increased risk of oral cavity malignancy

Sun damage changes red, scaly lesions may appear called actinic cheilitis which are premalignant lesions.

Treatments

Treatment of the Aging Lip

Rhytid Treatments

Development of cutaneous perioral rhytids is accelerated by sun exposure, cigarette smoking, and frequent puckering (e.g., smoking, straws).

Botulinum toxin A (Botox) injected immediately beneath the skin in the middle of rhytids. Helps more with hyper-dynamic rather than static rhytids. Also creates lip eversion and fullness. Narrow therapeutic window: functional motor impairment versus cosmetic relaxation [1]

Fillers see below for details. May be combined with Botox for longer-lasting results

Resurfacing dermabrasion, chemical peels, and CO_2 laser (for deep rhytids). CO_2 is ideal laser as it creates a deep injury with subsequent remodeling, but prolonged erythema.

Lip Augmentation (See Below)

Subnasal lip lift Bullhorn cutaneous excision under nasal base leaving muscle intact. Lifts the upper lip, increases dental show, and everts the red lip. Can be combined with augmentation for additional effect (Fig. 19.5).

Dermal flap A strip of the skin on the vermilion border is deepithelialized and buried to address lengthening of the upper lip, lowering of the vermilion border, and blunting of the Cupid's bow. Minimally improves upper lip rhytids. Results in scar at vermilion border. Can be combined with augmentation as well [2].

Augmentation Cheiloplasty

Involves either increase in vertical height and/or projection of the lip. Augmentation can be achieved with a number of different methods including autologous material, injectable fillers, tissue advancement, alloplastic implants, and surgery. For increasing vertical height, material is placed submucosally in the inferior edge of upper or superior edge of the lower lip. For more projection, material is placed in the same plane anteriorly. Lip augmentation should be uniform, with emphasis on accentuating the central lip. Excessive lateral augmentation results in an unnatural look and should be avoided.

Autologous Materials

- *Fat*: unpredictable resorption rate, generally 20–50%. Has become more reliable with newer harvest and injection techniques, but fat persistence in the lips remains lower than fat augmentation elsewhere in the face. Decreased survival in the lips has been shown in post-traumatic cases, and intramuscular injection is recommended to combat this. Risks of liposuction apply [3].
- *Dermis-fat*: donor fat and dermis excised instead of liposuctioned. Much more predictable results than fat injection. Smooth, long lasting, versatile [4]. Risk of cysts if all the epidermis is not removed
- *Temporalis fascia*: gives thin, minimal augmentation [5]
- *SMAS*: easily performed with concurrent rhytidectomy, swelling up to 3–4 weeks postop, variable survival, up to 5 years [5, 6]
- *Tendon* (e.g., palmaris longus tendon): long lasting. Maintains lip mobility [7]

Fillers injected just below the dermis

Injectable fillers have gained popularity for lip augmentation. Although the majority of fillers used are off-label by the FDA, their availability and reliability have made these products a first-line treatment option for patients needing volume enhancement. Although a number of injectable fillers are on the market, hyaluronic acid fillers remain the preferred product of choice. Augmentation can be performed along the vermillion, dry red lip, peristomal junction, commissure, and philtrum depending upon contouring needs.

- *Bovine* (Zyplast, Zyderm) *or porcine collagen* (Permacol, Evolence): generally nonreactive. Newer versions decreased risk of hypersensitivity. Lasts 2–6 months. Now largely historical and no longer available in the United States (historical).
- *Hyaluronic acid* (Juvéderm, Restylane, Perlane, Belotero): preferred material for augmentation. Malleable. Can be injected along philtrum, white roll, rhytids, and vermillion. Generally nonreactive, but carrier components may elicit allergic reaction which is self-resolving. Lasts 6–12 months. It is commonly used and well tolerated and has high satisfaction rate. Reversible with hyaluronidase injection [8, 9].
- *Polyacrylamide gel* (Royamid, Aquamid): high rate of granuloma formation and infectious complications. Permanent [10]. Non-FDA approved.
- *Calcium hydroxyapatite* (Radiesse): firm, lumpy. Can cause nodules and stiffness, which limits its usefulness in the lip. Lasts 1–2 years. Not recommended for lip augmentation.
- *Polymethylmethacrylate* (Bellafill formerly Artefil): polymethylmethacrylate particles stimulate neocollagenesis. Palpable. Permanent. Can cause granulomas or chronic infection. Not recommended for lip augmentation [11–13].
- *Silicone* (BioPlastique, Silikon 1000): placed using microdroplet technique due to tendency to lump. Permanent. Long-term risk of foreign body reaction. Can migrate. Not FDA approved and no longer recommended in the United States.

Aesthetic complication*s* “trout pout,” “duck lip,” and “chimp lip”—all due to overinjection of agent

Techniques recommend infraorbital and/or mental nerve blocks in addition to local anesthetic (topical or within the filler), as lip injections are very painful. Inject deep to the dermis and focus on even distribution to avoid lumps or irregularities. Discuss patient-specific goals and obtain a full history of prior procedures.

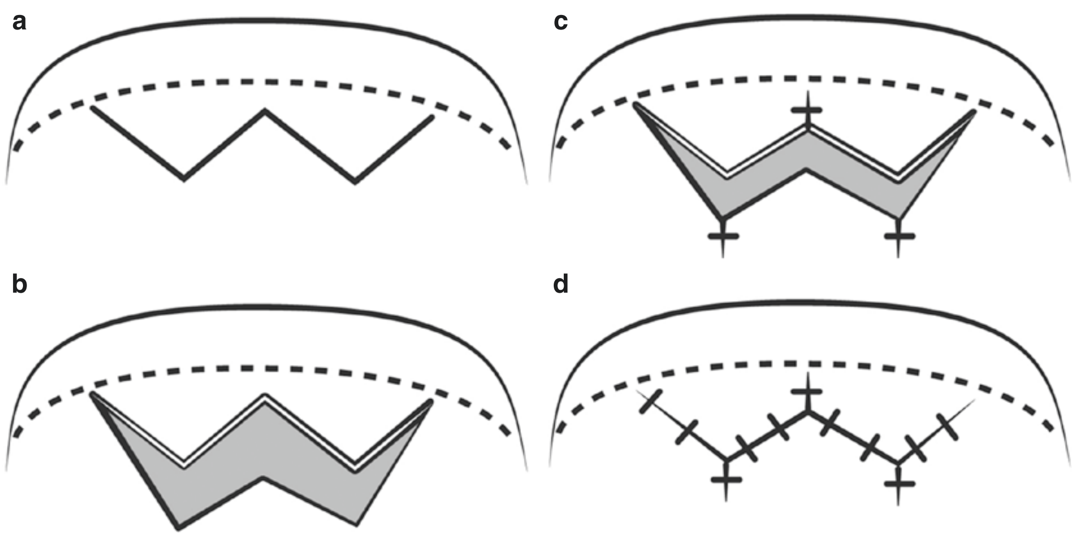


Fig. 19.6 (**a**–**d**) W-plasty. Consists of two V-to-Y advancements to increase the height of the lip by sacrificing width

Implants placed in submucosal tunnel using tendon passer through commissures. Important to avoid placing implant incisions directly at the commissures or across the vermilion due to risk of scarring

- *Acellular human dermal matrix* (AlloDerm, Cymetra): estimate size with dry instead of rehydrated state of AlloDerm due to compression in vivo. Injectable form shorter lasting than implantable sheets. Lasting 6–12 months versus partial permanent integration (approx. 20% of original volume).
- *Expanded polytetrafluoroethylene* (dPTFE, Gore-Tex, SoftForm): does not resorb but develops capsule fibrosis that may tighten or increase firmness of the lip. Patients can feel material in the lip. Extrusion possible, especially in the upper lip due to motion. Permanent. Not favored in most practices due to higher complication rate comparatively.
- *Silicone implants*: easily placed, easily reversible, most commonly used.

V-Y advancement technique involves recruitment of oral mucosa to achieve augmentation. Incision made down to orbicularis oris. Place apex of V inward. Multiple Vs (W-plasty) can be used in series to advance wider area of tissue. May have less predictable results. Useful for patients with very thin lips. Scarring not significant. Complications include potential "fish mouth" deformity (protrusion of the philtrum), lowering of the upper lip, and minimal loss of animation [14]. Prolonged swelling. Transient loss of sensation and this may take some time to resolve (6–9 months). Very bold and distinctive appearance postoperatively (Fig. 19.6).

Double Y-V mucosal plasty Modification of the above technique where the mucosal surface is incised with a horizontal line with horizontal Vs pointed laterally to advance over the horizontal line (>–< becomes ><). Good results in patients <40 years old while patients >40 required additional augmentation as well. Similar complication pattern as above (numbness and edema), but with decreased protrusion of philtrum [15]

Vermillion advancement excision of ellipses of cutaneous skin next to vermillion. The skin excised down to fascia right above the muscle but not through fascia. No undermining of edges. Can be used to better define Cupid's bow. Will maintain white roll

Reduction Cheiloplasty

The goal of lip reduction is to achieve balance between the upper and lower lips in harmony with the face, with preservation of lip competence. Prior to intervention, one must rule out other causes of macrocheilia (lymphatic malformation, dentoalveolar changes, Melkersson-Rosenthal syndrome) and ensure integrity of supporting musculature.

Vermillion reduction resect equal amount of tissue on either side of wet line. May excise the muscle along with the mucosa. Preserve lower lip height at the level of lower incisors [16]. Useful for patients with previous silicone injection to correct over augmentation as silicone is difficult to remove. Patients with large intralabial gap in repose (normal intralabial space 0–3 mm) may not be good candidates due to potential further enlargement.

"Bikini lip reduction" excision of "bikini top"-shaped area (infinity loop) from the upper lip and triangular area (with point down) from the lower lip. Designed to maintain upper to lower lip volume proportion (Fig. 19.7) [17]

Complications of Lip Procedures

- Bleeding.
- Infection: consider prophylactic antiviral for history of herpes simplex cold sores.
- Incision dehiscence: after surgery, rest lips for 2 weeks. May feel tightness with smiling for 6–8 weeks. Avoid smoking.
- Paresthesia: may last up to 6 months.
- Asymmetry: may be treated with steroids.
- Accentuation of pre-op defects: identify these pre-op defects.
- Implant extrusion: remove exposed portion. Large extrusions may require complete removal. Steroid injections may be used for tightness from scar tissue formation in extrusion pocket.
- Lip nodules: may inject steroid or incise if appear fluid filled.
- Minor defects: can be corrected with permanent cosmetics or dermal pigmentation.

Questions

1. Which lip is more prone to complications of lip augmentation and why?
   (a) Upper—contains fewer chambers and septations compared to the lower lip
   (b) Lower—contains fewer chambers and septations compared to the upper lip
   (c) Upper—contains more chambers and septations compared to the lower lip
   (d) Lower—contains more chambers and septations compared to the upper lip
2. Higher numbers of chambers and septations in the lip are found in which group?
   (a) Patients under age 65
   (b) Women
   (c) Smokers
3. Which filler requires test treatment prior to use in lip augmentation?
   (a) Zyplast
   (b) Cymetra
   (c) Belotero
   (d) Restylane

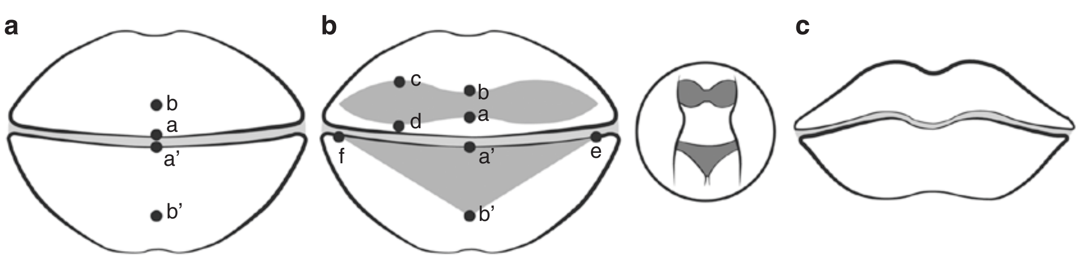


Fig. 19.7 (**a**–**c**) "Bikini" lip reduction design

4. Which represents the correct lip 1:2 ratio?
   (a) Upper lip: upper lip vermilion
   (b) Lower lip volume: upper lip volume
   (c) Subnasale to junction of upper and lower lips: menton to junction of upper and lower lips
5. On frontal view, the distance between menton and subnasale equals the distance between
   (a) Trichion to nasal tip
   (b) Tragus to lateral canthus
   (c) Nasal tip to pogonion
   (d) Subnasale to glabella
6. Which of the following is true about the profile view?
   (a) Upper lip should be 2 mm behind a line from nasal tip to pogonion.
   (b) Upper lip should project more than the lower lip.
   (c) Lower lip should lie 3.5 mm anterior to a line from subnasale to pogonion.
7. Which of the following is not an age-related change of the upper lip?
   (a) Shortening of the upper lip skin
   (b) Vertical rhytids
   (c) Blunting of Cupid's bow
   (d) Decreased vermillion show
8. What is the level of the labial arteries?
   (a) Deep to orbicularis oris and superficial to mucosa
   (b) Anterior to orbicularis oris
   (c) Subcutaneous
9. How is CO2 laser used in lip rejuvenation?
   (a) Stimulates collagen production in the dermis of red lip
   (b) Induces collagen remodeling in cutaneous upper lip
   (c) Shrinks the upper lip to induce clinically evidence vermilion eversion
   (d) Applied to upper and lower red lip to reduce wrinkling
10. Exam should consist of the following except:
    (a) Dental occlusion
    (b) Dental show
    (c) Chin position
    (d) All of the above
11. The V-Y plasty for lip augmentation improves fullness in the medial area of the lip but also results in
    (a) Shortening of the upper lip skin
    (b) Blunting of the Cupid's bow
    (c) Eversion of the corner vermilion
    (d) Large protrusion of the philtrum
12. The double Y-V flap for lip augmentation without additional filler is best suited for patients with
    (a) Age <40
    (b) Poor muscle tone
    (c) Age >40
    (d) Vertical rhytids
13. Lip augmentation with SMAS graft during concurrent rhytidectomy is a useful technique. Patients were primarily concerned with postoperative
    (a) Dermoid cysts
    (b) Need for additional augmentation at 6 months
    (c) Swelling for up to 3–4 weeks
    (d) Feeling of foreign material in the lip
14. The following are true regarding injectable silicone for lip augmentation except:
    (a) It has a tendency to lump.
    (b) Histologically a capsule surrounds each particle associated with moderate infiltration with lymphocytes.
    (c) This use is not FDA approved.
    (d) There is unpredictable resorption.
15. Removal of injectable silicone from the lip is best done by
    (a) Liposuction technique
    (b) Excisions of mucosa to reduce augmentation effect (reduction cheiloplasty)
    (c) Hyaluronidase injection
    (d) Open treatment with direct removal
16. A downside of dermal fat graft for lip augmentation includes
    (a) Cyst development
    (b) Permanent augmentation
    (c) High cost
    (d) Reliable
17. Bullhorn cutaneous excision at the alar base provides all of the following except
    (a) Shortening of the upper lip
    (b) Decreased visibility of the upper teeth
    (c) Eversion of the red lip
    (d) d. A visible incision
18. Dermal flaps for the aging lip are least effective at addressing which of the following?

(a) Cupid's bow blunting
(b) Vertical rhytids
(c) Reduction of the red lip
(d) Lengthened upper lip

19. Calcium hydroxylapatite injection in the lip is contraindicated due to risk of
(a) Lip nodule formation
(b) Keloid formation
(c) Calcification
(d) Pain on injection

20. Fat grafting for successful lip augmentation includes all of the following except:
(a) Superficial injection
(b) Use of intermediate layer after centrifugation
(c) Intramuscular injection
(d) Overcorrection of approximately 30–50% due to resorption

21. What effect does Botox have when injected into the lips?
(a) No cosmetic effect
(b) Reduces perioral rhytids
(c) Increases lip fullness and eversion
(d) b and c above

22. Lip augmentation with filler should focus on all of the following except
(a) Lateral corners for eversion
(b) Increased projection
(c) Medial lip
(d) Increased vertical height

23. PMMA was previously used for lip augmentation but is avoided because of
(a) Inducing neocollagenesis
(b) High rate of resorption
(c) Granuloma formation

24. Lip augmentation with filler is best done using all of the following techniques except:
(a) Nerve and local field block with injectable anesthetic agent
(b) Hyaluronic acid to vermilion border, white roll, and philtrum
(c) Radiesse deep to the dermis
(d) Pretreatment for patients with history of herpetic cold sores

25. Disadvantages to PTFE (Gore-Tex) lip implants include all of the following except:
(a) Resorption
(b) Capsule fibrosis that may tighten soft tissue or increase firmness
(c) High risk of displacement
(d) Infection

Answers

1. (d)
2. (b)
3. (a)
4. (c)
5. (d)
6. (b)
7. (a)
8. (a)
9. (b)
10. (d)
11. (d)
12. (a)
13. (c)
14. (d)
15. (b)
16. (a)
17. (b)
18. (b)
19. (a)
20. (a)
21. (d)
22. (a)
23. (c)
24. (c)
25. (a)

References

1. Semchyshyn N, Sengelmann RD. Botulinum toxin A treatment of perioral rhytides. Dermatol Surg. 2003;29(5):490–5. discussion 495
2. Guerrissi JO. Surgical treatment of the senile upper lip. Plast Reconstr Surg. 2000;106(4):938–40.
3. Botti G, Villedieu R. Augmentation cheiloplasty by using mucomuscular flaps. Aesthet Plast Surg. 1995;19(1):69–74.
4. Niechajev I. Lip enhancement: surgical alternatives and histologic aspects. Plast Reconstr Surg. 2000;105(3):1173–83; discussion 1187
5. Recupero WD, McCollough EG. Comparison of lip enhancement using autologous superficial musculo-aponeurotic system tissue and postauricular fascia in conjunction with lip advancement. Arch Facial Plast Surg. 2010;12(5):342–8.
6. Leaf N, Firouz JS. Lip augmentation with superficial musculoaponeurotic system grafts: report of 103 cases. Plast Reconstr Surg. 2002;109(1):319–26; discussion 327-328. 25

7. Trussler AP, Kawamoto HK, Wasson KL, et al. Upper lip augmentation: palmaris longus tendon as an autologous filler. Plast Reconstr Surg. 2008;121(3):1024–32.
8. Philipp-Dormston WG, Hilton S, Nathan M. A prospective, open-label, multicenter, observational, postmarket study of the use of a 15 mg/mL hyaluronic acid dermal filler in the lips. J Cosmet Dermatol. 2014;13(2):125–34.
9. Glogau RG, Bank D, Brandt F, et al. A randomized, evaluator-blinded, controlled study of the effectiveness and safety of small gel particle hyaluronic acid for lip augmentation. Dermatol Surg. 2012;38(7 Pt 2):1180–92.
10. Buck DW 2nd, Alam M, Kim JY. Injectable fillers for facial rejuvenation: a review. J Plast Reconstr Aesthet Surg. 2009;62(1):11–8.
11. Salles AG, Lotierzo PH, Gemperli R, et al. Complications after polymethylmethacrylate injections: report of 32 cases. Plast Reconstr Surg. 2008;121(5):1811–20.
12. Bagal A, Dahiya R, Tsai V, Adamson PA. Clinical experience with polymethylmethacrylate microspheres (Artecoll) for soft-tissue augmentation: a retrospective review. Arch Facial Plast Surg. 2007;9(4):275–80.
13. Solomon P, Sklar M, Zener R. Facial soft tissue augmentation with Artecoll((R)): a review of eight years of clinical experience in 153 patients. Can J Plast Surg. 2012;20(1):28–32.
14. Aiache AE. Augmentation cheiloplasty. Plast Reconstr Surg. 1991;88(2):222–6.
15. Lassus C. Surgical vermillion augmentation: different possibilities. Aesthet Plast Surg. 1992;16(2):123–7.
16. Niamtu J 3rd. Lip reduction surgery (reduction cheiloplasty). Facial Plast Surg Clin North Am. 2010;18(1):79–97.
17. Fanous N, Brousseau VJ, Yoskovitch A. The ‘bikini lip reduction’: a detailed approach to hypertrophic lips. Can J Plast Surg. 2007;15(4):205.

Additional Resources

Al-Hoqali RA, Abdel Mequid EM. The lip: a histologic and analytical approach of relevance to aesthetic plastic surgery. J Craniofac Surg. 2009;20(3):726–32.

Byrne PJ, Hilger PA. Lip augmentation. Arch Facial Plast Surg. 2004;20(1):31–8.

Kontis TC. Contemporary review of injectable facial fillers. JAMA Facial Plast Surg. 2013;15(1):58–64.

Matarasso S, Carruthers JD, Jewell ML. Consensus recommendations for soft tissue augmentation with nonanimal stabilized hyaluronic acid (Restylane). Plast Reconstr Surg. 2006;117(3):15–25.

Mojalial A, Shipkov C, Braye F, Breton P, Foyatler JL. Influence of the recipient cite on the outcomes of fat grafting in facial reconstructive surgery. Plast Reconstr Surg. 2009;124(2):471–83.

20 Chemical Peels

David M. Lieberman and Sudeep Roy

Skin Analysis

Cross-Sectional Anatomy of the Skin

Epidermis (0.05–0.1 mm in Depth):

Five Layers (From Superficial to Deep)

- Stratum corneum, stratum lucida (only in friction-prone skin, i.e., palms), stratum granulosum, stratum spinosum, and stratum basale (mitotically active layer)

Epidermal Cell Types

- Keratinocytes – most abundant cell type; 28-day cycle migrating from basal layer to stratum corneum
- Melanocytes – interspersed between basal cells; neural crest-derived and pigment-producing (melanin helps protect mitotically active basal keratinocytes from UV-induced DNA damage); two types of melanin: eumelanin (brown/black, prevalent in darker skin types and more effective in UV protection) and pheomelanin (yellow/red, prevalent in light skin types and less effective in UV protection)
- Merkel cells – mechanoreceptors located in areas of high tactile sensitivity; present in the basal layer; + for CK20 (important marker in Merkel cell carcinoma)
- Langerhans cells – antigen-presenting cell; found in stratum spinosum; contains intracytoplasmic tennis racket-shaped organelles known as Birbeck granules
- Fibroblasts – produce collagen and elastin

Dermis:

- Papillary dermis: thin layer with loosely arranged collagen bundles and abundant fibrocytes; has upward-facing papillae that interdigitate with epidermal rete ridges to resist shearing forces, papillae lost in scars which explains decreased wound strength compared to normal skin
- Reticular dermis: contains larger, densely packed collagen bundles and fewer fibrocytes; continuous with fibrous septae of underlying subcutaneous fat

Pilosebaceous unit: contains hair, hair follicle, sebaceous gland, sensory end organ, and arrector pili muscle

- Mostly within reticular dermis
- Contain multipotent stem cells found at the bulge of hair follicle (area beneath sebaceous gland where arrector pili muscle attaches)

D. M. Lieberman
Lieberman & Parikh Facial Plastic Surgery, Palo Alto, CA, USA

S. Roy (✉)
Refined Facial Plastic Surgery, Los Gatos, CA, USA
e-mail: sudeep.roy@refinedmd.com

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_20

capable of regenerating all parts of pilosebaceous unit and interfollicular skin

Skin changes with aging: intrinsic and extrinsic factors

- Intrinsic factors: aging ➔ alter skin collagen with generalized atrophy
- Extrinsic factors: smoking, UV light ➔ generation of free radicals leads to DNA changes; upregulation of AP-1 (transcription factor) causes activation of metalloproteinases (i.e., collagenase and gelatinase) which hasten collagen degradation; decrease in capillary and arteriolar blood flow in the skin causing thinning and wrinkling; increase in glycosaminoglycans (GAGs)

Histologic changes: skin regeneration slows, epidermis thins, and outer stratum corneum becomes less organized; rete ridges and dermal papillae less pronounced (flattening of dermal-epidermal junction); dermis thins with diminished and abnormal collagen and elastin bundles (solar elastosis); actinic damage and uneven melanin distribution.

Clinical changes/indications for chemical peeling: fine and coarse rhytids, uneven skin texture, cutaneous lesions, loss of elasticity, and uneven pigmentation (dyschromias)

*acne scarring additional indication

Chemical peels: removal of superficial layers of the skin

- Re-epithelialization occurs from adnexal structure within deep reticular dermis as well as surrounding epidermis.
- Dermal remodeling following inflammatory response from tissue injury.
- Increased volume of papillary dermis.
- Re-organization of reticular dermal collagen.
- Restoration of normal cellular polarity.
- Correction of cellular atypia and melanocytic hypertrophy and hyperplasia.

*The face is the optimal location for resurfacing given high density of adnexal structures.

Wound healing as it relates to chemical peeling:

Three stages:

- Inflammatory phase
  - Vasodilation, cytokine response, and complement cascade
  - ***Polymorphonuclear leukocytes first to appear.
  - ***Macrophages have the most important role.
  - Immediate dusky erythema
  - Lasts approximately 12 h
  - Coagulation: seen as the epidermis separates
    (a) Treat with debriding soaks, compresses, and occlusive emollients.
    (b) Consider 0.25% acetic acid soaks to prophylaxis against bacterial infection including *Pseudomonas*.
- Proliferative phase
  - Angiogenesis
  - Re-epithelialization: begins 1–3 days after peeling
    (a) Lasts approximately 10 days
    (b) Epidermal migration from wound margins and adnexal epithelium
    (c) Collagen synthesis
- Maturation phase
  - Continues for 3–4 months
  - Collagen maturation—bundles reorient into mature formation.
  - Neovascular dermis regresses (30 days for superficial peel, 60 days for medium-depth peels, 90 days for deep peels).

Patient Selection

Fitzpatrick skin-type classification scheme (Table 20.1) useful when stratifying risk of performing chemical peels:

Table 20.1 Fitzpatrick classification

Skin type	Skin color	Tanning response
Type I	White, freckled	Always burn, never tan
Type II	White	Usually burn, tan with difficulty
Type III	White, olive	Sometimes mild burn, tan average
Type IV	Brown	Rarely burn, tan with ease
Type V	Dark brown	Very rarely burn, tan very easily
Type VI	Black	No burn, tan very easily

Originally created to classify patients prior to a study on the effects of oral photochemotherapy (PUVA) on psoriasis [1]

Table 20.2 Glogau classification [2]

Group	Age (years)	Findings
I: Mild	28–35	Minimal wrinkles, no keratosis, minimal acne scarring, little makeup
II: Moderate	35–50	Early wrinkling with motion, early keratosis, mild scarring, some makeup
III: Advanced	50–65	Wrinkling at rest, actinic keratosis, telangiectasis, moderate scarring, always wears makeup
IV: Severe	60–80	Severe wrinkling, actinic keratosis and skin cancers, severe acne scarring, makeup cakes on

Risk Level

- Types I–II: lower risk for posttreatment dyschromias
- Types III–IV: higher risk of posttreatment dyschromias

Glogau skin classification scheme (Table 20.2) useful in rating patient skin quality and severity of photodamage:

- Photoaging Type I: not suitable for deep peels
- Photoaging Type IV: not suitable for superficial peels

Potential Contraindications

- Darker Fitzpatrick skin types (III–VI)
- Skin infection
- History of keloids
- History of herpetic infections
- History of diabetes mellitus or prior facial irradiation
- Unrealistic patient expectations
- Telangiectasias
- Anticipation of inadequate photo protection
- Significant hepatorenal disease
- HIV
- Immunosuppression
- Poorly treated emotional instability or mental illness
- Ehlers-Danlos syndrome
- Scleroderma or collagen vascular disease
- Accutane/isotretinoin treatment (within 6–12 months)
  - Induces sebaceous gland atrophy and impairs keratinization ➔ higher risk of scarring
- Breastfeeding
- Prior radiation therapy
  - Delayed re-epithelialization

The deeper the peel, the more significant the result *but* the higher the risk and downtime. The experience of the physician in managing patient characteristics and expectations will aid in achieving optimal results.

Levels of Frosting

1. Erythema with streaky surface whitening
2. White-coated frosting with erythema showing through (papillary dermis)
3. Solid white opaque frosting with little or no background erythema (penetration through papillary dermis into reticular dermis)

Classification of Chemical Peels (Fig. 20.1)

Superficial Chemical Peel

- Removal of either the stratum corneum (light superficial peel) only or entire epidermis (superficial peel)
- Indicated for mild photoaging, actinic keratosis, solar lentigines, pigmentary dyschromias, and acne with associated post-inflammatory erythema
- Typically done in a series of treatments to achieve best outcome
- Does *not* improve dermal issues such as deeper rhytides

TCA 10–20%

- TCA causes precipitation of epidermal proteins.
- Light frost, exfoliation of superficial half of the epidermis
  - Preparation: wash and degrease skin
  - Endpoint: level I frost (erythema and streaky white frost)
  - Frosting no desired in Fitz IV–VI due to pigmentation risk and scarring
  - Redness and peeling for 1–3 days
  - Moisturizer and sunscreen posttreatment
  - Typically used for early wrinkle treatment, mild photodamage

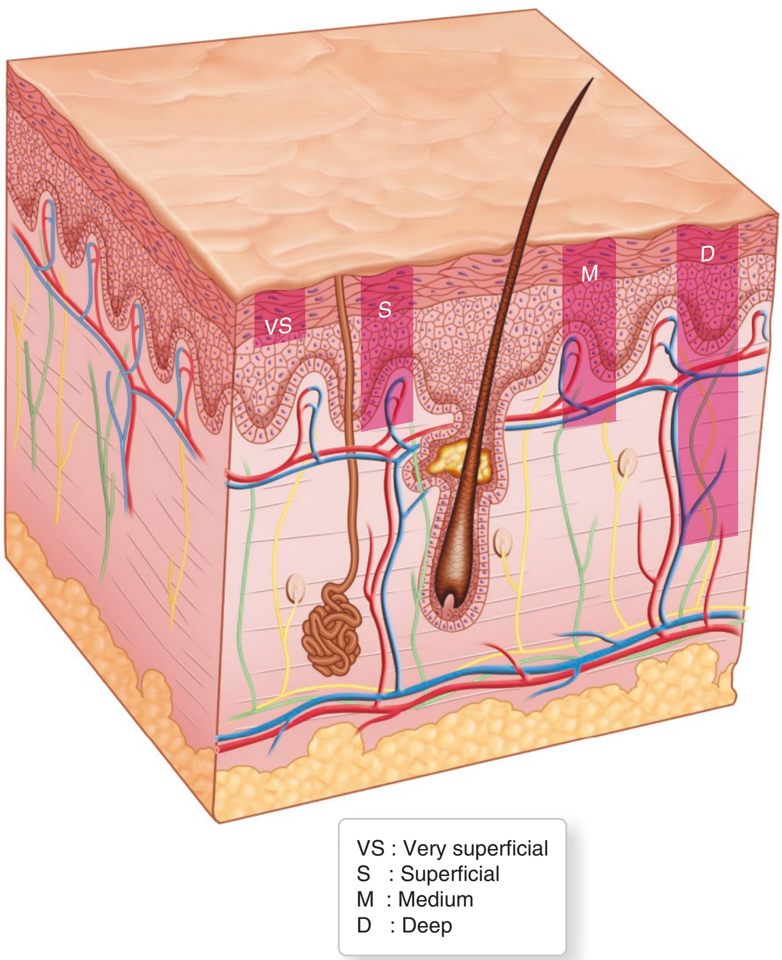


Fig. 20.1 Classification of chemical peels

Table 20.3 The Jessner's formula

Resorcinol	14 g
Salicylic acid	14 g
Lactic acid (85%)	14 mL
Ethanol	To make 100 mL

Jessner's Solution (Table 20.3)

- Resorcinol 14 g, salicylic acid 14 g, lactic acid 14 g, and ethanol to make 100 ml
- Keratolytic agent: removes the stratum corneum and destroys portions of the epidermis
- Thought to break intercellular bridges between keratinocytes (effective in preparation for a medium-depth peel)
- Often used for acne treatment on every other week schedule
- Resorcinol linked to increased risk of postinflammatory hyperpigmentation and cardiotoxicity; modified Jessner's solution: salicylic acid 14 g, citric acid 8 g, lactic acid 17 ml, and ethanol to make 100 ml

Alpha Hydroxy Acid (AHA)

- Common agents: glycolic acid 40–70% (30–40% Fitzpatrick skin type V or VI, <50% type IV), lactic acid
- Occurs naturally in food (glycolic acid, sugarcane; lactic acid, sour milk)
- Popularized as a home-use agent
- Theory: cause desquamation due to diminished corneocyte cohesion above the granular layer in the epidermis [3]; increases dermal thickness
- *Glycolic acid: must be rinsed off with water or neutralized with 5% sodium bicarbonate after 2–4 min*
- Typically used for wrinkle treatment and reduction of benign keratosis

Thirty Percent Salicylic Acid (β-Hydroxy Acid) Peels

- Breaks intercellular lipid bonds in the stratum corneum.
- Anti-inflammatory, antimicrobial properties, and noncomedogenic effects.
- Higher strength (30%) indicated for early-to-moderate photodamaged skin.
- Half face control study: 30% salicylic vs. 70% unbuffered glycolic acid [4].
  - Thirty percent salicylic acid does not contain resorcinol (found in Jessner's) eliminating risk of cardiotoxicity and has proven efficacy.
- Typically only 2–3 peels needed in sequence.
- Good for acne vulgaris, rosacea, melasma, hyperpigmentation, mild photoaging, and textural roughness.
- Ideal for darker skin types with PIH due to acne.
- *Self-limiting peel*. No neutralization needed as penetration stops once the vehicle (hydroethanolic) volatilizes (5 mins).
- Typical downtime 1–4 days.
- Risk: *salicylism* (early Sx: tinnitus, nausea and vomiting, disorientation, CNS effects); peel should be limited to relatively small surface areas to reduce risk.

Medium-Depth Peel

- Destruction of the epidermis with inflammation/damage *extending into the papillary dermis and upper reticular dermis*.
- Typically single treatment (i.e., not done in series); wait at least 1 year before subsequent treatment if desired.
- *Traditional agent: 50% TCA. No longer used due to risk profile of TCA treatment in strengths >50%, including increased risk of scarring.*
- *Agents: 35% TCA solution. Start with Jessner's vs. 70% glycolic acid vs. solid CO_2 (dry ice) to improve TCA penetration.*
- *Common treatment protocols*:
  - Jessner's solution to break up epidermal barrier and then 35% TCA for deeper treatment [5]
  - *Indications*: mild-to-moderate photoaging, dyschromias, melasma, lentigines, epidermal growths, rhytides, and actinic keratosis
  - Typically use mild/moderate sedation or deeper anesthesia
  - *Pretreatment*:

Retinoic acid to accelerate epidermal regeneration

Topical bleaching agent for high risk of post-inflammatory hyperpigmentation (PIH)

– *Protocol*:

Skin is washed and degreased (essential to optimize even and complete peel penetration).

TCA dose is determined by amount of solution used (dampness of the gauze or cotton tip applicator and number of passes).

Peel endpoint: *Level II frosting* (white coat with erythema showing through) or *level III frosting* (solid white frost without visible erythema—indicates dermal penetration)

More sensitive areas (eyelid skin) or areas more prone to scarring (bony prominences such as the zygomatic arch and inferior border of the mandible) should be treated up to a level II frost only.

Eyelid skin: treat to within 2–3 mm of the lid margin, cotton tip applicator should be damp without excess solution, and dry tears as they form since they can wick peel solution into the eye through capillary action (if chemical gets into the eye, flush with mineral oil).

Postoperative treatment varies by center. Considerations include 0.25% acetic acid compresses, emollient, and gentle cleansers; antiviral treatment starts 24 h before the peel and continues until epidermal regeneration is complete (approximately 7 days).

Peeling occurs over 4–7 days. Pink appearance can last 2–4 weeks.

Deep Peel

- Inflammation *extending through the papillary dermis into the reticular dermis*
- Results in new collagen formation

Table 20.4 Baker-Gordon phenol peel

3 mL USP liquid phenol 88%
2 mL tap water
8 drops liquid soap (Septisol)
3 drops croton oil

- Candid discussion with patient about risks and downtime before proceeding
- *Indications*: moderate-to-severe photodamaged skin (Glogau III/IV)

Baker-Gordon Deep Chemical Peel

(Table 20.4)

- Gold standard for deep chemical peels.
- Formula needs to be mixed close to the time of application.
- Conscious sedation or general anesthesia is required. Local blocks are recommended.
- Treat cosmetic subunits q15 minute intervals. Total treatment time 60–90 min to minimize systemic concentrations of phenol.
  - Occluded: application of a waterproof zinc oxide tape applied to each subunit after application

    Increases penetration into the midreticular dermis

    Risks include hyper- and hypopigmentation, a glossy and hypopigmented appearance to the skin (“alabaster skin”), and scarring
  - Unoccluded: penetration not as deep, therefore reducing the efficacy and risk profile
- Full-strength phenol causes immediate coagulation of keratin proteins, thereby blocking further penetration.
  - Phenol alone does not peel when less than 35% and peels only lightly up to 88%, but only with croton oil.
  - Phenol: cardiotoxic, hepatotoxic, and nephrotoxic.
- In the Baker-Gordon formula, the 88% *phenol* is diluted to 50–55% resulting in *keratolysis* as well as *keratocoagulation* and increasing penetration.
- Soap surfactant (*Hibiclens or Septisol*) reduces skin tension and allows more even penetration.

- *Croton oil*: vesicant epidermolytic agent that enhances phenol absorption.
  - Peel depth increases with increasing concentrations or multiple applications of croton oil.
  - Primary agent responsible for the peel.
- Pain occurs for 20s following application and returns 20 min later. Can last 6–8 h.
- Biosynthetic dressing (Vigilon or Flexzan) for first 24 h.

Histology Study Following Baker-Gordon Phenol Peel [6]

- Facial skin specimens taken after face-lifts performed years after the initial peel (1.5–20 years later)
- Dermal changes:
  - New 2–3 mm dermal strip seen with thin, compact parallel collagen bundles arranged horizontally
  - Abundance of elastin fibers
  - Less ground substance and inflammatory infiltrate compared to unpeeled skin
  - Absence of telangiectasias (normal-appearing blood vessels)
  - Normal number of melanocytes still present (creates hypopigmentation not depigmentation)
- Epidermal changes:
  - Return of polarity
  - Improved order of layers
  - Fewer cytologic irregularities (actinic keratosis, lentigo)
  - More uniform basement membrane

Hetter Peel (Table 20.5) [7]

- Modified the Baker-Gordon formula based on different phenol and croton oil concentrations to obtain robust results and decrease risks:
  - 0.25–0.5% croton oil: heals within 7 days
  - 0.6–1.0% croton oil: heals within 9–10 days
  - >1% croton oil: healing time longer than 10 days and some risk of hypopigmentation
  - >2% croton oil: consistent hypopigmentation and delayed healing

    Standard concentrations: 25% phenol, 1.05% croton oil

    Croton oil is added with a dropper. Standard drop size *0.04 mL*

Table 20.5 Hetter formula tables

Medium-light peel formula	4 cc phenol 88 % 6 cc water 16 drops Septisol 1 drop croton oil	33 % phenol 0.35 % croton oil
Very light peel formula *Used for the eyelids and neck*	3 cc of above mixture plus 2 cc phenol 88 % 5 cc water	27.5 % phenol 0.105 % croton oil
Medium-heavy peel formula *Not for lids, temples, preauricular, or neck*	4 cc phenol 88 % 6 cc water 16 drops Septisol 2 drops croton oil	33 % phenol 0.7 % croton oil
Heavy peel formula *Not for lids, temples, preauricular, or neck*	4 cc phenol 88 % 6 cc water 16 drops Septisol 3 drops croton oil	33 % phenol 1.1 % croton oil
Heaviest peel formula *Used for perioral rhytides or heavy, desiccated, pale skin*	3 cc phenol 88 % 2 cc water 8 drops Septisol 3 drops croton oil	50 % phenol 2.1 % croton oil

Postoperative Care

Upon completion of the chemical peel, a moisturizing emollient should be applied. Biosynthetic dressings such as Vigilon can be used for initial 48 hours followed by emollient until re-epithelialization is complete (superficial peels, 5 days; medium peels, 7–10 days; deep peels, 10–12 days). Frequent follow-up is imperative. Posttreatment erythema can last up to 6 weeks to 3 months and may be managed with 2.5% topical hydrocortisone. Sun avoidance for the initial 6–12 weeks following treatment is advised; and sunscreen can be started approximately 3 weeks post-peel (for deep peels, close to 6 weeks). Gentle, mineral-based powder makeup can be started 2 weeks post-peel to conceal erythema but should be discontinued if irritation is noted.

Cardiac Arrhythmias in Phenol Face Peeling [8]

- Arrhythmias correlated with procedure duration and surface area treated (dosage)
  - 80% excreted by the kidney
- Tachycardia, premature contractions, and ventricular tachycardia OR AFib
  - Protocol to limit toxicity:
    - Preoperative hydration (~1 L).
    - Intraoperative hydration (~1 L).
    - Staging the treatment to limit systemic phenol levels (allow for 15 min between application of each subunit).
    - Continuous cardiac monitoring.
    - Oxygen supplementation.
    - Aborting procedure for signs of EKG abnormalities (PVC, PAC).
    - Consider forced diuresis with furosemide 20 mg IV 10 min prior to procedure.
    - Consider prophylactic lidocaine hydrochloride 75 mg IV prior to procedure to protect myocardium and decrease chance of phenol-induced arrhythmias.
    - Preoperative cardiac clearance if appropriate.
      (a) Other side effects of phenol: stimulation and then depression of the CNS and decrease in BP and UOP
- *Water* dilution of the solution can *increase absorption*. Flushing or removing the solution should be done with *mineral oil*.

Prolonged Post-peel Erythema *PPPE* [9]

- Characterized by erythema, pruritus, burning, stinging, and irregular skin texture.
- Likely the result of an inflammatory reaction.
- Can begin as early as 2 weeks post-peel.
- Study: retrospective review of 236 nonoccluded Baker-Gordon peels.
  - Eleven percent developed PPPE.
  - Only factor that correlated with developing PPPE was a history of tape allergy.
  - Of the 27 patients: 15 resolved within 7 weeks, 25 within 4 months, and final 2 by 1 year.
  - *Intrinsic risk factors*: sensitivity to peeling agent, preexisting condition (rosacea, SLE, eczema, atopy), and contact dermatitis.
  - *Extrinsic risk factors*: sensitized by pretreatment (Retin-A, glycolic acid), aggressive peel technique or preparation, and infection.
- *Potential complications*: post-inflammatory hyperpigmentation, textural changes, and scarring:
  - Treatment sequence: avoidance of irritants ➔ emollients ➔ topical steroids ➔ acetic acid soaks ➔ systemic steroids ➔ dermatology consult
  - Can try Vitamin C serum as well which may help decrease erythema
- If scarring is evident, add intralesional steroids, silicone sheeting, and pulsed dye laser.

Specific Conditions/Concerns

- *Post-radiation*-treated skin: higher risk of post-peel breakdown if the skin is not healthy. Evaluate the quality of the skin and presence of hair (suggests sufficient health to support pilosebaceous units for regeneration of the epidermis).
- *Herpes simplex*: pretreatment history increases risk of posttreatment recurrence. Treatment with antiviral agents is necessary.
  - Acyclovir/valacyclovir: inhibit viral replication within the epidermal cell. Therefore therapy should be continued until full re-epithelialization (7–10 days for medium depth, 14 days for deep peel [5]).
- *Melasma*: large symmetric macules on the cheeks, forehead, upper lip, nose, and chin.
  - Etiology: genetic predisposition, exposure to UV radiation, pregnancy, oral contracep-

tives, thyroid dysfunction, cosmetics, and phototoxic and anti-seizure drugs [3]
  – Pre- and post-treat with hydroquinone 4–8%, retinoic acid, and sunscreen

Relevant Medications

- *Hydroquinone*: blocks tyrosinase from developing melanin precursors and therefore reduces new pigment formation
  – Reduces new pigment as the epidermis regenerates
- *Azelaic acid and kojic acid*: non-hydroquinone bleaching agents. Azelaic used if acne is an issue
- Topical vitamin C (ascorbic acid)
  – Promotes collagen synthesis and has antioxidant capabilities

Topical Tretinoin

- *Tretinoin mechanism of action*: binding to nuclear receptors exposing DNA-binding sites, thereby changing gene expression (decreasing MMPs such as collagenase, gelatinase, and stromatolysis) [10]
- *Weiss et al*. (1988) [11]: *Double-blind vehicle-controlled study*
  – Tretinoin versus vehicle: forearm and face.
  – Global improvement in tretinoin group: fine wrinkles, coarse wrinkles, tactile roughness, facial lentigines, and solar freckling.
    One hundred percent showed improvement by 12 weeks.
    Main adverse reaction, dermatitis. Typically subsided with time, reduced frequency of application, and topical steroids.
  – Biopsy results: tretinoin-treated areas showed increase in epidermal thickness, increase in granular layer thickness, increase in mitotic figures, improved stratum corneum organization (more compact and homogenous appearance), reduced melanocytic hypertrophy and hyperplasia, and reduced epidermal atypia.
    Dermal increase in collagen may take longer treatment period (>1 year).
- *Kang et al*. (1997) [12]: *Topical tretinoin and photoaging*
  – Tactile smoothening shortly after starting tretinoin due to compaction of the stratum corneum and increased hyaluronic acid with associated water retention.
    This epidermal change is transient—therefore long-term wrinkle reduction is due to dermal changes.
  – Topical tretinoin increases collagen in the papillary dermis.
    Dermis contains 85% type I collagen and 10% type III collagen.
    Decrease of mature collagen in photoaged skin (a cause of wrinkling) and restoration seen with retinoic acid.
    UV radiation upregulates breakdown of collagen (collagenase) causing reduction of collagen and wrinkle formation.
    When human skin is treated with tretinoin, AP-1 DNA binding is reduced by 70%, thereby preventing collagen breakdown [10].

Complications of Chemical Peeling

Intraoperative: incorrect peel medication and solution misplacement during application

- Ensure the solution has not expired.
- Saline, bicarbonate, and mineral oil should be immediately available.

Postoperative

- Infection: the best prevention is appropriate use of soaks and debridement of necrotic material.
  – *Streptococcus*, *Staphylococcus*, *Escherichia coli*, and *Pseudomonas*
    Bacterial infection signs: delayed wound healing, ulcerations, necrotic material accumulation, purulence, and odor

- Herpes simplex: due to reactivation of HSV
  Increases risk of postoperative scarring. Patients with pretreatment history of HSV should be treated appropriately.
  (a) Acyclovir 400 mg TID starting 24 h before the peel and continuing for 7–14 days
  (b) Valtrex 1000 mg BID starting 24 h before the peel and continuing for 7–14 days
  (c) Acyclovir topical ointment to supplement oral treatment during active infection
- Fungal infection
  Acetic acid soaks
  Antifungal treatment (oral, topical)

Post-inflammatory hyperpigmentation (PIH)

- –Acquired excess of melanin production.
- –Reduce risk with pre/post-peel treatment lightening agents combined with retinoic acid/AHAs to promote exfoliation of the epidermis in Fitz III-IV patients.
- –Consider single dose of oral corticosteroid (prednisone 20–60 mg) following medium-depth and deep peels in patients with history of PIH.
  Hypopigmentation
  Lines of demarcation

Questions

1. Brown skin that rarely burns and tans easily is classified as:
   (a) Fitzpatrick type II
   (b) Fitzpatrick type III
   (c) Fitzpatrick type IV
   (d) Fitzpatrick type V
   (e) Fitzpatrick type VI
2. Seventy percent glycolic acid can penetrate to a depth similar to:
   (a) 10% TCA
   (b) 35% TCA
   (c) Baker-Gordon peel
   (d) Microdermabrasion with aluminum oxide
   A. Jessner's solution
3. Best neutralizing agent for glycolic acid is:
   (a) Normal saline
   (b) Lactated ringer
   (c) Dilute sodium bicarbonate
   (d) Dilute acetic acid
   (e) Mineral oil
4. When applied Baker-Gordon peel, which facial subunits are generally not treated?
   (a) Forehead
   (b) Upper eyelid
   (c) Lower eyelid
   (d) Cheek
   (e) Perioral
5. After a phenol peel, re-epithelialization generally occurs by:
   (a) 3 days
   (b) 5 days
   (c) 10 days
   (d) 21 days
   (e) 30 days
6. Which is least important in preoperative evaluation of a patient prior to phenol peel?
   (a) Hepatic status
   (b) Respiratory status
   (c) Cardiac status
   (d) Renal status
   (e) History of isotretinoin use
7. Which statement is FALSE regarding rhytidectomy in conjunction with chemical peeling?
   (a) TCA is the preferred peeling agent in this setting.
   (b) Chemical peeling can be done in areas that have not been undermined during rhytidectomy.
   (c) Chemical peeling in conjunction with deep plane rhytidectomy is safe.
   (d) Chemical peeling after rhytidectomy with a long skin flap can be done simultaneously as long as a superficial peeling agent is used.
   (e) It is appropriate to wait 6 months after rhytidectomy to perform chemical peeling.
8. Which of the following is an absolute contraindication to chemical peeling?
   (a) Fitzpatrick type IV

(b) Fitzpatrick type VI
(c) History of herpes infection
(d) Telangiectasias
(e) Recent isotretinoin use

9. Which of the following medications would not help in the treatment of post-inflammatory hyperpigmentation?
(a) Hydroquinone
(b) Doxycycline
(c) Azelaic acid
(d) Tretinoin
(e) Kojic acid

10. Which statement is true regarding post-phenol peel hypopigmentation?
(a) It is usually permanent.
(b) Usually resolves with topical hydroquinone
(c) Usually seen in the first 2 weeks after peel
(d) Occurs as frequently as hyperpigmentation
(e) It is usually temporary.

11. Regarding the composition of Baker-Gordon phenol peel, which is incorrect?
(a) Phenol 88% 3 ml
(b) Septicol 8 drops
(c) Croton oil 3 drops
(d) Distilled water, 8 ml
(e) Should be prepared fresh after each use

12. Which of the following measures will increase the toxic effects of phenol?
(a) Extending the time of application for full-face peel
(b) Intravenous hydration
(c) Cleaning and degreasing face prior to use
(d) Decreasing concentration of phenol
(e) Administration of oxygen

13. Hyperpigmentation as a side effect of chemical peeling will be increased by:
(a) Kojic acid
(b) Sun avoidance
(c) Estrogen
(d) Tretinoin
(e) Hydroquinone

14. Which cells have the most important role in wound healing process?
(a) Macrophages
(b) Leukocytes
(c) Platelets
(d) Neutrophils
(e) Monocytes

15. Which is false regarding trichloroacetic acid?
(a) No systemic toxicity
(b) Low cost
(c) Neutralization not needed
(d) Unstable, needs to be mixed fresh prior to each use
(e) Depth of peel correlates with concentration of solution.

16. Which of the following cells produces collagen?
(a) Keratinocytes
(b) Langerhans cells
(c) Fibroblasts
(d) Merkel cells
(e) Melanocytes

17. Which is true regarding the use of Jessner's solution prior to 35% TCA in a medium-depth chemical peel?
(a) Reduces risk of prolonged post-peel erythema
(b) Keratocoagulant which removes the stratum corneum and destroys portions of the epidermis
(c) Keratolytic agent which removes the stratum corneum and destroys portions of the epidermis
(d) Surfactant that reduces skin tension and allows more even penetration
(e) Causes precipitation of epidermal proteins

18. In a deep peel, depth of peel is increased by:
(a) Higher concentration of phenol
(b) Jessner's solution before deep peel
(c) Leaving post-peel skin unoccluded
(d) Higher concentration of croton oil
(e) Lower concentration of croton oil

19. The following is not a result of a Baker-Gordon peel:
    (a) New, horizontally oriented collagen in the reticular dermis
    (b) Less ground substance
    (c) Less cytologic atypia
    (d) Increased inflammatory infiltrate
    (e) Normal number of melanocytes
20. Appropriate sequence for medium-depth peel could be?
    (a) Pretreatment with tretinoin and hydroquinone ➔ antiviral treatment ➔ peel procedure ➔ emollients ➔ steroid cream
    (b) Pretreatment with steroid cream and emollients ➔ antiviral treatment ➔ peel procedure ➔ start tretinoin and hydroquinone
    (c) Pretreatment with tretinoin and hydroquinone ➔ peel procedure ➔ anti-viral treatment ➔ emollients ➔ steroid cream
    (d) Antiviral treatment ➔ peel procedure ➔ start tretinoin and hydroquinone ➔ emollients ➔ steroid cream
    (e) Pretreatment with isotretinoin and hydroquinone ➔ antiviral treatment ➔ peel procedure ➔ emollients ➔ steroid cream
21. The following is not a means to reduce phenol cardiotoxicity
    (a) Preoperative hydration
    (b) Oxygen supplementation
    (c) Forced diuresis with furosemide 20 mg IV 10 minutes after procedure
    (d) Prophylactic lidocaine hydrochloride 75 mg IV prior to procedure
    (e) Staging treatment by waiting 15 min between application of each subunit
22. During a Baker-Gordon peel, signs of cardiotoxicity become evident. A reasonable first step would be?
    (a) Neutralize the peel with 5% sodium bicarbonate
    (b) Flush the peeled skin with distilled water
    (c) Flush the peeled skin with normal saline
    (d) Flush the peeled skin with mineral oil
    (e) Start CPR
23. Which of the following statements is false regarding tropical tretinoin?
    (a) Binds to nuclear receptors and reduces AP-1 binding to DNA
    (b) Main adverse reaction is dermatitis.
    (c) Permanent epidermal changes leading to long-term wrinkle reduction
    (d) Results in tactile smoothening of the skin shortly after starting treatment
    (e) Reduces metalloproteinases in the skin
24. Prolonged post-peel erythema is correlated with:
    (a) Previous resurfacing
    (b) History of skin dermatitis
    (c) Exogenous estrogen use
    (d) Tobacco use
    (e) Tape allergy
25. Which of the following peels is described as self-limited, requiring no neutralization?
    (a) 35% TCA
    (b) 30% salicylic acid
    (c) 50% glycolic acid
    (d) Baker-Gordon Peel
    (e) Hetter peel

Answers

1. (c)
2. (b)
3. (c)
4. (b)
5. (c)
6. (b)
7. (d)
8. (e)
9. (b)
10. (a)
11. (d)
12. (d)
13. (c)
14. (a)
15. (d)
16. (c)
17. (c)
18. (d)
19. (d)
20. (a)
21. (c)
22. (d)
23. (c)
24. (e)
25. (b)

References

1. Fitzpatrick TB. The validity and practicality of sun-reactive skin types I through VI. Arch Dermatol. 1988;124(6):869–71.
2. Glogau RG, Matarasso SL. Chemical face peeling: patient and peeling agent selection. Facial Plast Surg. 1995;11(1):1–8.
3. Clark CP 3rd. Office-based skin care and superficial peels: the scientific rationale. Plast Reconstr Surg. 1999;104(3):854–64.
4. Kligman D, Kligman AM. Salicylic acid peels for the treatment of photoaging. Dermatol Surg. 1998;24(3):325–8.
5. Monheit GD. The Jessner's + TCA peel: a medium-depth chemical peel. J Dermatol Surg Oncol. 1989;15(9):945–50.
6. Kligman AM, Baker TJ, Gordon HL. Long-term histologic follow-up of phenol face peels. Plast Reconstr Surg. 1985;75(5):652–9.
7. Hetter GP. An examination of the phenol-croton oil peel: part IV. Face peel results with different concentrations of phenol and croton oil. Plast Reconstr Surg. 2000;105(3):1061–83. discussion 1084-7
8. Botta SA, Straith RE, Goodwin HH. Cardiac arrhythmias in phenol face peeling: a suggested protocol for prevention. Aesthet Plast Surg. 1988;12(2):115–7.
9. Maloney BP, Millman B, Monheit G, McCollough EG. The etiology of prolonged erythema after chemical peel. Dermatol Surg. 1998;24(3):337–41.
10. Fisher GJ, Datta SC, Talwar HS, Wang ZQ, Varani J, Kang S, Voorhees JJ. Molecular basis of sun-induced premature skin ageing and retinoid antagonism. Nature. 1996;379(6563):335–9.
11. Weiss JS, Ellis CN, Headington JT, Tincoff T, Hamilton TA, Voorhees JJ. Topical tretinoin improves photoaged skin. A double-blind vehicle-controlled study. JAMA. 1988;259(4):527–32.
12. Kang S, Fisher GJ, Voorhees JJ. Photoaging and topical tretinoin: therapy, pathogenesis, and prevention. Arch Dermatol. 1997;133(10):1280–4.

Additional Resources

Been MJ, Harmych BM, Mangat DS. Dermabrasion, chemical peels, and dermaceuticals. In: Ira D, editor. Papel's facial plastic and reconstructive surgery. 4th ed. New York: Thieme; 2016.

Fedok FG, Paul J. Carniol's minimally invasive and office-based procedures in facial plastic surgery. New York: Thieme; 2014.

21 Fillers, Botulinum Toxin, Mid-facial Implants, and Tissue Expansion

Yuna Larrabee and Nate Jowett

Anatomy

Soft-Tissue Envelope of the Face

- The muscles of facial expression (Table 21.1), together with the temporoparietal fascia, form the superficial musculoaponeurotic system (SMAS).
- Motor innervation is provided solely by the facial nerve; muscles are innervated from their undersurface except the following deep muscles: levator anguli oris, buccinator, and mentalis.
- Fasciocutaneous retaining ligaments connect the SMAS to the overlying dermis throughout the face:
  - Strong over the forehead, eyelids, nose, lips, and chin
  - Intermediate strength over the medial cheek and neck
  - Loose over the lateral cheek and temple
- Osteocutaneous retaining ligaments (very strong) exist between the dermis and periosteum overlying the zygoma and body of the mandible.

Y. Larrabee (✉)
Otolaryngology, Massachusetts Eye and Ear Infirmary, Quincy, MA, USA

N. Jowett
Otology and Laryngology, Massachusetts Eye and Ear Hospital/Harvard Medical School, Boston, MA, USA

Facial Aesthetic Units and Subunits

- Forehead—Midline (1), lateral/temporal (2), brow (2)
- Eye—Lower lid (1), upper lid (1), medial canthus (1), lateral canthus (1)
- Nose—Dorsum (1), sidewall (2), tip (1), columella (1), soft-tissue triangle (2), ala (2)
- Cheek—Infraorbital (1), buccal (1), zygomatic (1), lateral (1)
- Lips—Upper lateral lip (2), philtrum (1), lower lip (1)
- Chin
- Other muscles of expression supplied by facial nerve: anterior/superior/posterior auricular muscles (move auricle in respective directions), occipitalis (moves scalp backward)

Blood Supply

- External carotid artery
  - Facial artery → submental, angular, and inferior and superior labial branches
  - Internal maxillary artery → inferior orbital, anterior and deep temporal, and mental branch of inferior alveolar branches
  - Superficial temporal artery → transverse facial, frontal, middle temporal, and anterior auricular branches

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_21

Table 21.1 Major muscles of facial expression

Muscle	Origin	Insertion	Action/effect	Associated rhytides	Associated expressions
Frontalis (Fn)	Galea aponeurotica near coronal suture	Frontal bone (superciliary ridge) and skin of brow Interdigitates with CS (deep) and Pr and OOc (superficial)	Elevates eyebrows and forehead skin	Horizontal forehead	Surprise, interest, excitement Sadness
Corrugator supercilii (CS)	Frontal bone (medial superciliary ridge)	Skin of medial brow (passes between pars orbitalis and pars palpebralis of OOc)	Pulls eyebrows medially (major) and inferiorly (minor)	Vertical glabellar	Contempt, disgust, anger, pain
Procerus (Pr)	Fascia overlying upper lateral cartilages and nasal bone	Skin between eyebrows Interdigitates with Fn (deep)	Pulls medial brows inferiorly (major) and medially (minor)	Transverse radix nasi Nasal bridge	Pain, anger, aggression
Nasalis [compressor naris (CN), dilator naris (DN), depressor septi (DS)]	CN: Maxilla, superolateral to incisive fossa DN: Maxilla, superior to incisive fossa, medial to CN DS: Maxilla, incisive fossa	CN: Opposing muscle and procerus aponeurosis DN: Lower alar cartilage DS: Base of caudal septum	CN: Narrows nostrils, depresses nasal cartilages DN: Dilates nares DS: Narrows nostril, pulls ala inferiorly	DS: horizontal philtral	DN: Anger, aggression
Orbicularis oculi (OOc)—Pars orbitalis	Nasal and superomedial orbital portions of frontal bone, inferomedial orbital portions of maxilla Medial palpebral ligament	Lateral palpebral raphe (*not* lateral canthal tendon) Skin of brow Superior aspect interdigitates with Fn and CS (deep)	Forced eye closure, brow depression	Lateral canthal (crow's feet) (major)	Happiness (Duchenne smile)
OOc—Pars palpebralis preseptalis	Superficial head: MCT Deep head: Fascia overlying lacrimal sac	Lateral palpebral raphe (*not* lateral canthal tendon)	Gentle eye closure (sleeping, blinking), lacrimal sac pump	Lateral canthal (minor)	Happiness (Duchenne smile)
OOc—Pars palpebralis pretarsalis	Superficial head: MCT Deep head: Posterior lacrimal crest and fascia overlying lacrimal sac	Lateral canthal tendon (~7 mm from lateral orbital tubercle and ~7–10 mm below zygomaticofrontal suture)			
Buccinator (Bn)	Pterygomandibular raphe, alveolar processes of maxilla and mandible	Modiolus	Pulls angle of mouth posteriorly (whistling, smirk, suckling, mastication)	Lateral oral commissure	Annoyance, contempt, disapproval, sarcasm

Table 21.1 (continued)

Muscle	Origin	Insertion	Action/effect	Associated rhytides	Associated expressions
Levator labii superioris alaeque nasi (LLSAN)	Superior aspect of frontal process of maxilla at level of medial canthal tendon	Lower lateral cartilage Skin of superomedial NLF, ala, and Cupid's bow Skin, vermillion, and OOr fibers of medial upper hemi-lip	Elevates upper lip, nasal ala, skin of nasal sidewall, NLF, and cheek, with indirect upward displacement of lower lid (snarl, nostril flaring)	NLF (superomedial) Nasal sidewall Lid/cheek junction	Anger, disgust, contempt, aversion
Levator labii superioris (LLS)	Inferior orbital rim (zygomatic and maxillary bones), just superior to infraorbital foramen	Skin of NLF (lateral to nasal ala) and Cupid's bow Skin, vermillion, and OOr fibers of central upper hemi-lip	Elevates central upper hemi-lip, NLF (superolaterally), and cheek with indirect upward displacement of lower lid (sneer, smile)	NLF (lateral to nasal ala) Lid/cheek junction	Disgust, contempt, aversion
Zygomaticus minor (Zm)	Temporal process of zygoma, just posterior to zygomaticomaxillary suture	Skin of NLF (middle aspect) and malar fat pad Skin, vermillion, and OOr fibers of central upper hemi-lip	Deepens and retracts middle aspect of NLF Everts and draws upper lip posteriorly and superiorly (crying)	NLF (middle aspect)	Sadness
Levator anguli oris (LAO)	Canine fossa of maxilla, inferior to infraorbital foramen	Modiolus	Pulls angle of mouth directly upward (uncomfortable smile)	NLF (middle aspect) (indirect)	Discomfort, embarrassment
Zygomaticus major (ZM)	Temporal process of zygoma, just inferior to zygomaticotemporal suture	Modiolus Occasional interdigitation with DAO and Rs and insertions into skin of lower NLF	Draws angle of mouth superiorly, posteriorly, and laterally (smile, laugh)	NLF (lower aspect)	Happiness, joy
Risorius (Rs) (variable expression)	Parotidomasseteric fascia	Modiolus	Draws angle of mouth posteriorly and laterally (smug smile)	NLF (lower-lateralmost aspect)	Contempt, smugness, happiness
Orbicularis oris (OOr)	Modiolus Alveolar processes of maxilla and mandible	Decussation in midline with contralateral side Skin of philtral columns and vermillion	Deep fibers: appose lips to teeth Superficial fibers: closure and protrusion (pucker, purse)	Vertical perioral (lipstick)	Pouting, anger
Depressor anguli oris (DAO)	Oblique line of mandible, inferior to mental foramen	Modiolus Skin of labiomandibular crease	Curves angle of mouth inferiorly, lengthening and deepening lower NLF (frown)	Marionette lines	Sadness, grief, aversion, disgust

(continued)

Table 21.1 (continued)

Muscle	Origin	Insertion	Action/effect	Associated rhytides	Associated expressions
Depressor labii inferioris (DLI)	Oblique line of mandible, inferior and medial to mental foramen	Skin, vermillion, and OOr fibers of central lower hemi-lip	Draws lower lip inferiorly and laterally, partially everts	Horizontal labiomandibular	Anger, aversion, irony
Mentalis (Mn)	Vertical line adjacent to midline on mental protuberance of mandible, inferior to level of teeth roots and mental foramen	Skin of the chin Medial fibers interdigitate with paired muscle (when absent, results in cleft of the chin)	Raises soft-tissue prominence of chin, indirectly elevating and everting the lower and upper lips (pouting)	Labiomental sulcus, chin dimples	Sadness, grief, anger, doubt, disdain
Platysma (Pt) [pars modiolaris (PMo), pars labialis (PL), pars mandibularis (PMn)]	Superficial fascia overlying pectoral and deltoid muscles	PMo: Modiolus PL: Skin, vermillion, and OOr fibers of central lower hemi-lip PMn: Mandible, below oblique line	Widens neck; deepens suprasternal notch PMo: Pulls angle of mouth inferiorly and laterally PL: Pulls central lower hemi-lip inferiorly and laterally, partially everts PMn: Pulls jaw inferiorly	Longitudinal neck bands Horizontal neck rhytides	Fear, horror, aversion, pain, anger, disgust, sadness

- Internal carotid artery
  – Ophthalmic artery → dorsal nasal, supraorbital, supratrochlear, medial palpebral branches
  – Lacrimal branch of ophthalmic artery → lacrimal, zygomaticofacial, lateral palpebral branches
- Rich external–internal carotid anastomoses throughout the face give rise to a risk of retrograde embolic spread of filler to retinal and CNS vascular supply.

Skin Innervation

- Ophthalmic branches (V1): Supratrochlear (medial brow/forehead/scalp), supraorbital (central hemi-brow/forehead/scalp), palpebral branch of lacrimal (lateral upper lid), infratrochlear (medial upper lid, nasal dorsum, conjunctiva, caruncle), external nasal branch of anterior ethmoidal (nasal tip)
- Maxillary branches (V2): Infraorbital (medial lower lid/cheek, upper lip), zygomaticofacial (lateral lower lid), zygomaticotemporal (medial zygoma, hairless temple)
- Mandibular branches (V3): Mental (mentum and lower lip), buccal (lateral cheek), auriculotemporal (lateral zygoma/scalp, hair-bearing temple, superior helix, and concha)
- VII: Sensory auricular branch (posterior wall of external auditory meatus)
- X: Auricular branch or "Arnold's nerve" (external auditory meatus)
- Cervical plexus (C2): Greater occipital nerve (posterior scalp to vertex), lesser occipital nerve (post-auricular)
- Cervical plexus (C2, C3): Great auricular nerve (lobule and peri-lobule skin, inferior helix and concha, posterior auricle), transverse cervical nerve (anterior neck)
- Cervical plexus (C3, C4): Medial/middle/lateral supraclavicular nerves (sternal notch, supraclavicular regions, skin of upper chest and shoulders)

Regional Variation in Skin Thickness (Full Thickness)

3–3.5 mm	Nasal tip, chin, forehead, cheek
2–3 mm	Lower eyelid, lips, philtrum, submentum
1.5–2 mm	Neck, nasal dorsum
1–1.5 mm	Upper eyelid, posterior concha

Fat Compartments of the Face

The subcutaneous fat was originally thought to be a large confluent mass of fat; however, recent studies have shown that the fat of the face and neck is organized into distinct anatomic compartments (Fig. 21.1). These compartments are separated by fascial septae that originate from the superficial cervical fascia and insert into the dermis. Perforating vessels travel through the septae to supply the skin in distinct angiosomes.

These fat compartments age differently causing noticeable contour differences through both volume and position changes (volume loss and ligament laxity/tissue descent).

The Aging Face

Signs and Symptoms

- Skin: Thinning of epidermis, fragility, loss of turgor, decreased elasticity, increased laxity, rhytides, folds, ptosis, hyperpigmentation
- Other: Fat atrophy and ptosis, temporal wasting, bone resorption

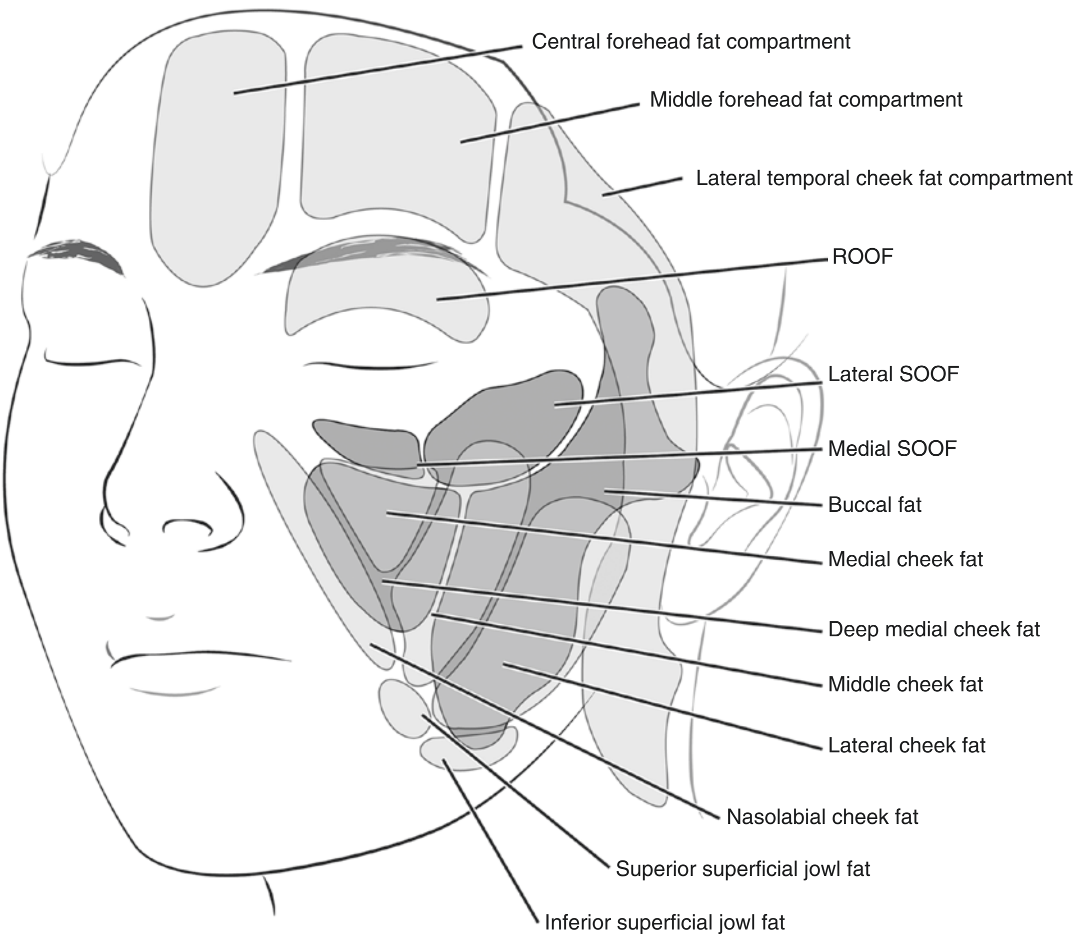


Fig. 21.1 Facial fat compartments. The fat of the face and neck is organized into distinct anatomic compartments. *Light gray* superficial fat compartments, *dark gray* deep fat compartments

Intrinsic Factors: Chronologic Aging

- Accumulation of cellular damage from reactive oxygen species (ROS) generation through mitochondrial oxidative metabolism over time → increased matrix metalloproteinase (MMP) expression → dermal extracellular matrix (ECM) breakdown with loss of skin elasticity → accumulated damage from imperfect repair
- Replicative senescence of epidermal keratinocytes → decreased rate of epidermal turnover → thinning of epidermis → fragile skin
- Replicative senescence of dermal fibroblasts → thinning of basement membrane, flattening of rete pegs, decreased ECM turnover → thinning of dermis → skin laxity, wrinkling
- Marked decrease in epidermal glycosaminoglycans (GAGs), especially hyaluronic acid (HA) → loss of skin moisture (HA binds and retains water)
- Decrease in dermal GAG content (esp. HA) → loss of dermal turgidity, laxity, wrinkling
- Weakening of retaining ligaments over time potentiated by gravity → ptosis
- Volume loss of facial adipose tissue, muscle, and bone (partial resorption of maxilla and mandible leading to decreased soft-tissue projection) all occur with age

Extrinsic Factors

- Photoaging is the superposition of ultraviolet radiation (UVR) damage on intrinsic aging.
- UVA (penetrates deeply, ~30% reaches deep dermis) → ROS generation → upregulation of MMP and downregulation of procollagen expression → dermal ECM breakdown with loss of skin elasticity → accumulated damage from imperfect repair.
- UVB (absorbed superficially, results in sunburn, only 10% reaches superficial dermis) → neutrophil influx and release of neutrophil elastase together with upregulation of elastin promoter in upper dermal fibroblasts → breakdown of ECM with accumulation of disorganized (dysfunctional) elastin and associated proteoglycans in ECM of superficial dermis (solar elastosis).
- UVB → TP53 mutation and p16 deletion in keratinocytes → unregulated growth → actinic keratoses and carcinomas.
- Solar elastosis is more pronounced between wrinkles (likely from relative shielding of wrinkle base from UV exposure) leading to deepening of wrinkles.
- UVR also alters HA homeostasis, compounding intrinsic loss of HA.
- Erythema ab igne (EAI)—Clinical hyperpigmentation and solar elastosis in skin exposed to chronic heat/infrared radiation.
  - Mechanism: Thought to result from neutrophil influx, release of neutrophil-derived proteolytic enzymes, and subsequent fibroblast-mediated imperfect repair
- Other accelerants of skin aging: smoking, alcohol, chronic disease, and poor nutrition.

Age-Related Pigmentation Changes

- Melanocyte counts decrease by ~8–20% per decade (sun-exposed and sun-protected areas).
- Density of melanocytes remains ~2× higher in sun-exposed areas.
- Paradoxical increase in hyperpigmentation with age due to UVR-induced upregulation of tyrosine kinase activity in surviving melanocytes and UVR-induced abnormal pigment retention by keratinocytes (solar lentigines).

Repetitive Use of Facial Muscles Over Time

- SMAS connected to the dermis via fasciocutaneous retaining ligaments throughout the face.
- Repeated/habitual contraction of underlying SMAS produces and determines orientation of folds, wrinkles, and relaxed skin tension lines (RSTLs) (except lids due to rigid tarsal plates).
  - Mechanism: Muscle shortens without corresponding shortening of overlying skin → temporary wrinkle at right angles to vector of muscle contraction → skin adaptation and increasing laxity over time leads to presence of wrinkles/folds at rest

Mechanisms Underlying Major Facial Folds

- Nasolabial fold: Elevator muscles of the lip send muscular projections to the dermis along path of NLF. Dynamically present in youth; present at rest with age.
- Labiomandibular (melomental/marionette line and jowl lines): Weakening of the fasciocutaneous ligaments along the anterior border of the masseter over time results in ptosis of the premasseteric space.
  - Gravity pulls the contents medially and inferiorly resulting in marionette lines.
  - Jowl lines are the continuation of the marionette lines inferior to the strong mandibular osteocutaneous retaining ligament (same mechanism).
  - Appear only with age.
  - DAO also believed to contribute to Marionette lines.

Botulinum Toxin

- Exotoxin produced by *Clostridium botulinum* (Gram-positive, motile, anaerobic rod).
- Nine known toxin types (A, B, C_1, C_2, D, E, F, G, H)—A, B, E, F, and H are neurotoxic in humans.
- A and B are commercially available for clinical use (Table 21.2).
- Mechanism: Enters distal neuron through receptor-mediated endocytosis and cleaves SNAP-25 (type A) or VAMP (type B), preventing docking and fusion of synaptic vesicles to presynaptic membrane (necessary for acetylcholine release into the neuromuscular junction).
- Onset: Peak effect seen at 7–14 days (type B having a slightly faster onset than type A).
- Duration of effect: Striated muscle paralysis lasts 3–4 months (type A lasts slightly longer than type B); autonomic blockade lasts 6–9 months.

Table 21.2 Fat compartments of the face. The layers of facial fat are organized into distinct compartments that contain important structures and constitute noteworthy surgical landmarks

Region	Superficial fat compartments	Notes
Forehead	Central, middle temporal, lateral temporal-cheek	Supratrochlear a. is the boundary between central and middle temporal; lateral temporal-cheek is the most lateral compartment and spans from forehead to neck
Orbital	Infraorbital fat (malar mound)	Superior margin is at lid/cheek junction. Above junction, little fat is present; below junction, fat covers orbicularis oculi
Nasolabial	Nasolabial	Most medial; lower border of zygomaticus major m. adheres to compartment; angular a. perforators run in NL septum
Cheek	Medial, middle, lateral temporal-cheek	Facial v. found at deep surface of medial cheek fat; where medial and middle compartments meet is parotidomasseteric ligaments; lateral cheek septum is the first transition zone in face-lift
Cheek	Jowl fat compartment (superior/inferior)	Adherent to depressor anguli oris (DAO) medial boundary is lip depressor; inferior boundary is a membranous fusion with platysma (mandibular retaining ligament)
Region	Deep fat compartments	Notes
Cheek	Deep medial cheek	Deep to medial and middle superficial cheek fat; potential space between deep medial cheek fat and maxilla periosteum (Ristow's space) can be used for fat transfer, located medial to zygomatic major. Important for central cheek prominence. Inadequate volume results in anterior flattening
Cheek	Buccal	Lies adjacent to deep medial cheek fat in masticator space. Extends from edge of upper mandible into the temple affects the jowl prominence. Facial nerve and parotid duct travel through this compartment
Orbit	SOOF-medial and lateral compartment	Lies along orbital rim and zygomatic arch immediately above periosteum, medial compartment smaller than lateral. V2 travels through medial compartment; zygomaticofacial travels through lateral compartment. Lateral SOOF is important for cheek projection

- Recovery of function
  - Growth factor secretion from denervated muscle results in active axonal sprouting.
  - Original nerve terminals recover function by production of new SNAP-25/VAMP.
- Cosmetic indications
  - FDA approved: Glabellar (4 U into each of 5 sites in total) and lateral canthal lines (4 U into each of 3 sites per side)
  - Off-label: Forehead rhytids, brow elevation (lateral OOr injection), palpebral fissure widening, vertical perioral and Marionette lines, NLFs, "gummy" smile, platysmal bands, hyper prominent masseter muscle, synkinesis
- Loco-regional complications: Paralytic lagophthalmos and exposure keratopathy (excessive OOr weakening), lid ptosis (migration of toxin to lid elevators), facial ptosis (e.g., brow), facial asymmetry, dry eye (lacrimal gland inhibition).
- Systemic complications (rare): Spread of toxin outside injection area causing dysphagia or respiratory paralysis and possibly death, hours or weeks later (FDA black box warning 2009).
- Contraindications: Known hypersensitivity, egg allergy, active infection at injection site.
- Relative contraindications: Neuromuscular disease, pregnancy and lactation (effects unknown), concurrent use of aminoglycosides or muscle relaxants (potentiates effect).
- Antibodies and subsequent resistance is rare but may develop with repeated injections over time (delay repeat injection as much as possible); type switching (A to B) is sometimes necessary.

Injectable Volumizing Fillers

Indications

- Fine lines—superficial papillary dermis injection
- Moderate-to-severe lines and wrinkles—mid and deep dermis injection
- Facial augmentation (e.g., orbital rim, tear trough, temple, mandible, malar/submalar, chin)—subcutaneous, supra- and preperiosteal injection

Ideal Filler

- Biocompatible, hypoallergenic, noninflammatory, nonmigratory, non-carcinogenic, nontoxic, resistant to infection, consistent and predictable response, natural feel, inexpensive, long-lasting, retains shape over time, short and painless injection, requires no recovery time, long shelf life, easily stored (no refrigeration)

Classification and Properties (Tables 21.3 and 21.4)

- Biologic—Tissue-derived (auto-, allo-, or xenograft) or cell culture-derived
  - Collagen, micronized connective tissue, hyaluronic acid (HA), autologous tissue.
  - Collagen and HA preparations differ in concentration (higher concentration for mid-deep dermis) and degree of cross-linking (delays degradation).
  - HA preparations also differ in particle size (larger particles → more augmentation) and carrier gel characteristics (gel viscosity, presence of lidocaine).
  - Elastic modulus (G') of filler/carrier combination (measurement of hardness) is a prime determinant of appropriate placement.

    High G' products should be placed in deeper lines (e.g., NLF and marionette lines).

    Low G' products should be used in areas requiring a soft agent (e.g., body of lip, tear trough).
- Synthetic—Chemically synthesized biocompatible compounds.
  - Bioabsorbable (calcium hydroxylapatite, polylactic acid, methylcellulose).
  - Permanent (PMMA: polymethyl methacrylate).

Table 21.3 Current FDA-approved botulinum toxins

Type	Brand	FDA-approved cosmetic uses	Dose ratio (to BOTOX®)	Notes
OnabotulinumtoxinA	BOTOX®	Glabellar lines Lateral canthal lines	1:1	Original and most commonly used commercial toxin Freeze-dried powder (50 or 100 units per vial) Storage: −5 °C or 2–8 °C, 2 years (24 h once reconstituted)
AbobotulinumtoxinA	Dysport®, Reloxin®	Glabellar lines	1:2–1:4	Possible higher diffusion (greater spread of effect) vs. BOTOX® Freeze-dried powder (300 or 500 units per vial) Storage: 2–8 °C, 2 years (4 h once reconstituted)
IncobotulinumtoxinA	Xeomin®	Glabellar lines	1:1–1:1.3	Possible reduced risk of antibody formation as compared to BOTOX® Freeze-dried powder (50 or 100 units per vial) Storage: −20 to −10 °C or 2–8 °C, 3 years (24 h once reconstituted)
RimabotulinumtoxinB	MYOBLOC® (NeuroBloc®)	None	1:50–1:100	Faster onset, shorter duration of action (vs. Type A) Greater area of diffusion, more painful injection (acidic pH) Higher protein content, more frequent antibody formation Available in 2500, 5000, or 10,000 U liquid vials Storage: 2–8 °C, 21 month (4 h once diluted)

Table 21.4 FDA-approved biologic injectable fillers and fat

Source	Brands	Uses and duration	Notes
Collagen			
Bovine	Zyderm® Zyplast®	Fine-mod lines, folds, scars ~2–4 months	*Requires skin testing 6 and 2 weeks prior to first injection* Storage: Refrigerator, 3 years 96%/4% Type I/III
Recombinant human	CosmoDerm® CosmoPlast®		Storage: Refrigerator, 3 years 93/7% Type I/III
Micronized connective tissue			
Human cadaveric dermis	Cymetra®	Depressed scars ~3–12 months	Thick paste, difficult to inject Avoid periocular, forehead, glabellar injection (risk of emboli) Avoid if allergic to polysorbate 20 or specified antibiotics Storage: Room temperature, 18 months
Human cadaveric fascia	Fascian®	Previously used for mod-severe wrinkles	Inflammation and nodules result from dermal injection *Off-market, no longer available*

(continued)

Table 21.4 (continued)

Source	Brands	Uses and duration	Notes
Hyaluronic acid			
Rooster coon	Hylaform	Mod-severe wrinkles/folds ~6 months	Higher incidence of skin reaction vs. bacterial source Superficial injections can result in bluish tint (Tyndall effect), especially perioral and orbital rim Storage: Room temperature, 2 years Effect reversible with hyaluronidase injection (big advantage)
Bacterial cultures of equine streptococci	Restylane/ Perlane® Juvéderm® (various forms) Belotero® Captique® Hydrelle/ Elevess® Puragen®	Fine lines (Captique®) Mod-severe wrinkles/folds Facial augmentation (Juvéderm Voluma) ~6 months (up to 24 months)	No risk of nodule formation; all areas of face may be injected Superficial injections can result in bluish tint (Tyndall effect), especially perioral and orbital rim Storage: Room temperature, 2 years Effect reversible with hyaluronidase injection (big advantage)
Autologous tissue			
Fat	N/A	Facial augmentation Years	Unpredictable result, ~30–50% initial resorption rate, often uneven resulting in lumpiness ***High risk of embolic sequelae, especially with periocular and forehead injections
Fibroblasts	LaViv®	Mod-severe NLF At least 6 months	Requires punch biopsy harvest (behind ear), 4–6 weeks' culture time, and then three treatment sessions at 3–6-week intervals *Inject into superficial papillary dermis* Avoid if allergic to gentamicin, amphotericin, DMSA, bovine serum

  – Although still often mentioned in many textbooks as an option, injection of liquid silicone anywhere in the body is not FDA approved and is considered unethical by most experts due to high risk of complications (silicone granulomas, induration, nodules, fibrosis, ulceration, migration).
- Contraindications: Documented hypersensitivity to active compound or carrier components, active infection at site of injection, bleeding disorders.

Technique and Plane of Injection

- Needle gauge selection dependent on filler viscosity (30 G for low viscosity agents, 27 G for thicker agents)
- Serial puncture: Superficial dermis (fine lines) and mid-papillary dermis (moderate wrinkles)
- Linear threading: Subcutaneous or deep dermis injection requiring a linear path (e.g., lip augmentation, deep wrinkles such as nasolabial folds)
- Fan: Subcutaneous requiring a large area (e.g., malar/submalar augmentation)
- Cross-hatching: Recommended for PLLA (Sculptra®) injections (deep dermis, preperiosteal space injection)

Complications of Injectable Fillers

- Minor/moderate—Edema, erythema, ecchymosis, asymmetry, hypersensitivity reaction, granuloma, nodule formation, infection

- Major—Thrombotic events
  - Mechanism: Anterograde or retrograde embolic spread of filler through inadvertent intravascular injection or high-pressure transudation
  - Local—Vascular compromise leading to soft-tissue necrosis
  - Regional—Central retinal artery occlusion (CRAO), cavernous sinus thrombosis, cerebral ischemic events, death
  - Presentation of CRAO: Sudden loss of vision, retinal whitening, cherry-red spot in central macula
- Risk reduction—Use of blunt tip needles, large cannulas, epinephrine at injection site, low-pressure injections, multiple passes, small volumes on each pass (avoid large boluses), inject on withdrawal, use of ice immediately after filler injection, careful observation following injection, crash cart, and ready protocol for management of embolic/thrombotic events
- Management of filler-related embolic/thrombotic events—stop injection, hyaluronidase injection (regardless of filler used), 2% topical nitroglycerin paste onto injection site, massage, warm compress, glucocorticoid (oral or IV), enoxaparin and aspirin (if not contraindicated), sildenafil and hyperbaric oxygen, and prophylactic antibiotic and antiviral therapy

Nonsurgical Rhinoplasty

- Nasal fillers can be used for augmentation and correction of postrhinoplasty asymmetries.
- Deep radixes or dorsal humps can be camouflaged with dorsal injection.
- Short noses can be elongated and broad noses narrowed by linear threading along midline nasal dorsum.
- Pollybeak deformity can be treated by augmenting dorsum and tip.
- Saddle nose and inverted V deformity can be addressed by augmenting soft tissue overlying cartilage.
- Alar or columellar retraction can be augmented.
- Tip depressions or bossae can be filled.
- Functional rhinoplasty with injection of CaHA into internal nasal valve.

Technique

- HA most commonly used followed by CaHA.
- Small volumes—dorsum and tip can be altered with 0.1 mL of filler.
- Avoid large volumes to prevent Tyndall effect or pressure necrosis.
- Avoid high-pressure injection.
- Linear threading is the most common injection technique (useful for correction of dorsal contour). Serial puncture or single puncture for smaller injections of tip or columella.

Complications of Nasal Fillers

- Pressure necrosis and embolism.
- CaHA has increased risk of complications compared to HA.
- If blanching occurs, stop injection and treat with hyaluronidase. Visual loss suggests retinal embolism and should be treated seriously (see ocular complications below).
- Risk reduction: Avoid over-injection, especially at tip where skin is thinnest. Aspiration prior to injection to prevent intra-arterial injection. Knowledge of vasculature (angular artery at nasal ala, supraorbital and supratrochlear arteries superiorly, lateral nasal arteries at lateral nasal walls). Risk reduced with use of cannulas over needles.
  - Filler most safely placed in avascular deep supra-periosteal plane below nasal SMAS. Caution in patients with prior surgical treatments of the nose

Ocular Complications from Filler Injections

- Etiology: intravascular injection and retrograde embolization of filler. Arterial pressure

can be overcome with injection, and rapidly injected filler may find less resistance proximally (retrograde embolization) rather than in the higher resistance narrower distal vessel. Arterial pressure can carry embolus from proximal ophthalmic artery to distal retinal arteries.
- A review through 1/2015 identified 98 cases of ocular complications from soft-tissue fillers (Beleznay et al. Dermatol Surg 2015 Oct;41(10):1097–117).
- High-risk sites: glabella (38.8%), nasal region (25.5%), nasolabial fold (13.3%), and forehead (12.2%).
- Autologous fat (47.9%) was the most common filler type to cause this complication, followed by hyaluronic acid (23.5%).
- Most common symptoms: immediate vision loss and ocular pain.
  – Management: stop injection. Contact ophthalmologist or oculoplastics. Treat injected area with hyaluronidase. Consider retrobulbar injection of hyaluronidase. Reduce intraocular pressure with ocular massage, anterior chamber paracentesis, IV mannitol, and acetazolamide. Monitor patient's neuro status for CNS complications.
- Most cases of vision loss did not recover. If perfusion to eye is not restored within 90 minutes, damage from retinal ischemia is irreversible.
- No treatments were found to be consistently successful in treating blindness.

ATX-101 (Deoxycholic Acid, Kybella®) (Georgesen and Lipner, J. Cosmet Dermatol. 2017) (Shamban, PRS Global Open, 2016)

- First pharmaceutical therapy FDA approved for reduction of submental fat.
- Deoxycholic acid: endogenous secondary bile acid, solubilizes dietary fat.
- ATX-101 is a synthetic form of deoxycholic acid and is a lipolytic agent.
- When injected into subcutaneous fat, causes focal adipolysis. After cell destruction, macrophage infiltrate clears cell membrane fragments and free lipids and recruits fibroblasts, stimulating neocollagenesis.
- Target tissue: Preplatysmal fat.

Indication

- Improvement in appearance of moderate-to-severe convexity or fullness associated with submental fat

Technique

- Dosage: 0.2 mL injections spaced 1 cm apart. Up to 50 injections in a single treatment. Up to 6 treatments in 1-month intervals.
- Safe administration: avoidance of marginal mandibular nerve, avoidance of platysma, use of a 1 cm injection grid to mark injection sites (grid provided with product).
- Injection technique: pinch preplatysmal fat between two fingers (isolating it from underlying structures), and inject perpendicular to skin until needle midway into preplatysmal fat.
- Storage: 20–25 degrees C (68–77 degrees F).
- Contraindications: infection at injection sites.

Complications

- Marginal mandibular nerve injury, dysphagia, submental hematoma/bruising
  – In one study, marginal mandibular nerve paresis occurred in 4.3% of patients lasting from 7 to 61 days.
  – To prevent marginal mandibular nerve injury, avoid injections above a line drawn 1–1.5 cm below the inferior mandibular border.
- Adverse reactions: edema/swelling, hematoma, pain, numbness, erythema, induration

Mid-facial Implants

Indication

- Augmentation of bony or soft-tissue deficiencies of malar eminence, submalar region,

premaxilla, mandible, or nose (discussed in other chapters)
- Causes: Congenital, syndromic, traumatic, HIV-related, or age-related volume loss

Ideal Implant

- In addition to factors listed for ideal facial fillers, easily shaped without compromising implant integrity, able to permanently retain its form, tapered margins that blend into adjacent bony areas, sterilizable without degradation

Materials

- All induce capsule formation to varying degrees (stabilized implant).
- Significant tissue ingrowth stabilizes implant but complicates removal/switching.
- Risk of extrusion decreases with increasing depth of placement.
- Polymers (Table 21.5).
- Calcium ceramics
  – Calcium hydroxylapatite (mixed with blood and microfibrillar collagen) moldable paste (disadvantages: brittle, low tensile strength, difficult to fix in position).
  – Calcium phosphate (Norian CRS Bone Cement®, BoneSource®, Mimix®) moldable putty, hardens in 5–10 min.
  – Nova Bone® (45% sodium oxide, 45% silica dioxide, 5% calcium, 5% phosphate) is an osteoconductive material.
- Biologic materials (auto-, homo-, or xenografts): Bone, cartilage, fat, and dermis may be used but are limited by donor site morbidity and resorption.
- Injectable fillers are increasingly being used for facial volume augmentation over implants.

Preoperative Analysis: Mid-face

- Assess bony malar structure and adequacy of submalar soft-tissue volume.

Table 21.5 FDA-approved synthetic injectable fillers

Compound	Brands	Uses and duration	Notes
Calcium hydroxyapatite (CaHA)	Radiesse®	Mod-severe wrinkles/folds Facial augmentation (off-label) Nasal contour defects (off-label) ~6–12 months (up to 18 months)	Avoid superficial and avoid lip injection (risk of nodules) Non-osteogenic microspheres enzymatically degraded over time to Ca^{2+} and PO_4^{-3}, acts as scaffold for fibroblast deposition of new collagen Storage: Room temperature, 2 years
Poly-L-lactic acid (PLLA)	Sculptra®	Severe folds/wrinkles Facial augmentation (esp. for concavities) Lipoatrophy in HIV patients ~2 years	Avoid superficial, nose, lip injections (risk of nodules) Inject into deep dermis (at dermal/SQ interface) using cross-hatch technique; SQ, preperiosteal injection also feasible Delayed onset of effect, typically requires 3–5 injections Storage: Room temperature, 2 years (72 h reconstituted)
Polymethyl methacrylate (PMMA) and bovine collagen	Bellafill® (previously Artefill ® Artecoll®)	Mod-severe NLF and acne scars Facial augmentation (off-label) Permanent	*Requires allergy skin test* Avoid superficial, nose, lip injections (risk of nodules) PMMA microspheres are contained within bovine collagen, trigger fibroblast deposition of new collagen Storage: Refrigerator, 12 months

- Type I: Primary malar hypoplasia with adequate submalar soft tissue
  Treat with malar shell implant that projects cheek laterally.
- Type II: Normal malar skeleton, submalar soft-tissue deficiency (most common)
  Treat with submalar implant that projects anteriorly (and/or rhytidectomy).
- Type III: Bony malar hypoplasia and submalar soft-tissue deficiency
  Treat with combined malar and submalar implants.

Surgical Technique

- Approaches: Intraoral (most common), subciliary (via lower blepharoplasty), transconjunctival, rhytidectomy, zygomaticotemporal, transcoronal.
- Outlines areas requiring augmentation with the patient awake and sitting up.
- Intraoral approach
  - Incision in gingivobuccal sulcus—use a wide elevator directed superolaterally along bone up to malar prominence, oblique elevation of soft tissue off maxillary buttress, and malar eminence.
  - Submalar space created by elevating soft tissues above masseter below malar eminence.
  - For routine malar implant, no need to identify/dissect infraorbital nerve as the medial border of the implant is lateral to the infraorbital foramen.
- Implant should fit easily into pocket.
- Secure implant using screws and internal or temporary transcutaneous sutures.
- Avoid dead space behind implant; ensure margins blend into adjacent areas, with no palpable step-offs.

Soft-Tissue Expansion

Indications (Table 21.6)

- When adequate tissue adjacent to large cutaneous defect is unavailable
  - Falls between regional flap and free tissue transfer on reconstructive ladder
- Works best with a bony support underlying the balloon (scalp and forehead are ideal)

Table 21.6 Polymers used in semisolid and solid facial implants

Material	Advantages	Disadvantages
Silicone	Induces strong capsule without tissue ingrowth Nontoxic, nonallergenic	Strong inflammatory reaction Unstable placement and/or inadequate soft-tissue coverage results in ongoing inflammation +/− seroma If placed too superficially, may result in capsular contraction and implant deformity, or extrusion
Porous polyethylene (MEDPOR®)	Minimal inflammatory reaction Porosity permits extensive fibrous tissue ingrowth which prevents migration	Difficult to sculpt Fibrous ingrowth makes removal difficult
Expanded polytetrafluoroethylene (ePTFE, GoreTex®)	Minimal capsule formation and tissue ingrowth (best for soft-tissue augmentation) Easily removed if necessary	Soft (unsuitable for bony augmentation)
Polymethyl methacrylate (PMMA)	Can be custom-made intraoperatively (supplied as powdered mixture, which hardens in 10 min by an exothermic reaction—must use irrigation to cool)	Very rigid and hard, difficult to shape Difficult placement through small openings Thermal injury due to exothermic reaction
Mesh polymers (Dacron®, Mersilene®, Marlex®, Supramid®)	Extremely pliable, easy to shape Promotes significant fibrous tissue ingrowth	Extremely difficult to remove Supramid elicits foreign body reaction causing implant degradation and resorption over time

Internal Tissue Expanders (Balloon)

- Shapes: Round, rectangular, crescent
- Sizes: 100–2000 cc
  - General guideline is to choose a base size that is 2–2.5× the width of the defect over flat areas and 2.5–3× the width of the defect over curved areas.
- Types of filling valves:
  - External remote—Port outside of patient, avoids percutaneous injections (better for children), risk of infection and tube obstruction
  - Integrated—Less dissection required, risk of implant rupture
  - Internal remote—Port connected to balloon by silicone tubing tunneled subcutaneously away from expander; requires more dissection, risk of valve overturn, migration, and tube obstruction

External Tissue Expanders

- Limited tissue expansion may also be achieved using external devices as simple as externally applied adhesive tape to induce skin stretch and subsequent biologic creep (success has been reported in the preoperative expansion of radial forearm skin to obtain primary closure of a subsequent radial forearm free-flap donor site)

Mechanism and Tissue Response to Expansion (Table 21.7)

- Soft tissue tolerates controlled and gradual expansion better than rapid expansion.
- Tissue expanded flaps may be considered a type of delayed flap, as they demonstrate enhancements in vasculature commonly seen with delay phenomenon.
- Mechanisms
  - Mechanical creep: Displacement of interstitial fluid and ground substance, with realignment of collagen fibers and fragmentation of elastin. The underlying process behind stress relaxation of tissue (decrease in the recoil force of skin when held under stretch)
  - Biological creep: Cell proliferation and upregulated ECM synthesis occurring in response to stretch that functions to restore resting tension of the stretched tissue toward baseline, necessitating an increase in surface area
- Rapid intraoperative tissue expansion (ITE): Mechanical creep + enhanced undermining and recruitment of surrounding tissues.
- Long-term tissue expansion: Biologic and mechanical creep.

Table 21.7 Uses, advantages, disadvantages, and complications of head and neck tissue expansion

Uses	Advantages	Disadvantages	Complications
Common: Staged repair of scalp defects, hair replacement, auricular reconstruction Other: Cheek and neck Distant free-flap donor sites (prior to flap harvest to allow for primary closure)	Sensate tissue Excellent color and texture match for surrounding tissue Avoids free flaps Allows for hair-bearing skin	High risk of complications (inexperienced 25–40%, experienced 3–7%) Time and expense: 2 operations and 4–8 weeks of expansion, up to 20 injections Cosmetically unsightly (during expansion) Discomfort/pain with expansion Requires repeated visits, patient compliance, patient motivation	Hematoma/seroma Infection, extrusion Mechanical failure (leakage, rupture, valve or tubing obstruction) Skin necrosis Nerve injury Vascular compromise Muscle atrophy Fat atrophy Bone resorption Insufficient expansion Flap failure (following expansion)

Implant Surgery

- Placement plane
  - Scalp: Between the galea and pericranium.
  - Forehead: Between the frontalis and pericranium.
  - Face: Sub-SMAS is possible; supra-SMAS is likely safer (lessen risk of facial nerve branch transection/direct nerve compression).
  - Neck: Subcutaneous placement, care with neck placement (avoid pressure over carotid sinus).
- Wide undermining is required; balloon must lie flat without buckling.
- Select appropriate location for remote port; suture edges (if possible) to prevent overturn.
- Slight filling of balloon at the time of insertion is warranted to obliterate dead space and aid in hemostasis, but must avoid tension on wound edges on closure.
- Permanent suture use for all layers of closure (implant placement surgery) to lessen risk of wound dehiscence with subsequent expansion.

Inflation

- Begins 2 weeks after placement.
- Use sterile saline and sterile technique; a small (23-gauge) needle is best.
- Inject saline until overlying skin becomes firm, within the tolerance of patient comfort.
- Do not blanch skin; if blanching occurs, withdraw saline until color restored.
- Patient can expect discomfort for 12–48 h following each injection.
- Number of injections, volume per injection, and length of expansion vary with patient, implant size, and location (5–7 days between injections; 4–8 weeks for expansion is typical).
- Endpoints—implant reaches capacity or skin expansion adequate for desired flap.

Explant Surgery

- Brief over-inflation of balloon immediately prior to removal is useful for flap closure (provides additional tissue surface area through mechanical creep and disrupts the capsule). A dense fibrous capsule forms around expander balloon (lined by macrophages, surrounded by fibroblasts, highly vascular).
  - Contributes to contracture, shrinkage, thickening, and decreased pliability of flap after balloon removal (some experts advise against expansion of forehead tissue for nasal reconstruction for this reason).
  - Capsule may be cautiously excised at the time of implant removal.

Complications

- Expander exposure or extrusion; most common
  - Typically through an incision line but can occur anywhere.
  - If early exposure, may need to abort expansion. If toward completion, may be able to be completed.
  - Higher risk in thin areas or previous radiation. Slow inflation may decrease risk.
- Infection
  - Sterile technique important
- Ischemia or necrosis
  - Rare, may be due to aggressive inflation techniques.
  - Risk factors include smoking history, diabetes, significant scarring, and radiation.
- Neuropraxia
  - Uncommon, typically temporary

Questions

1. Name two osteocutaneous ligaments of the face.
2. Which of the following muscles is innervated from its superficial surface?
   (a) Levator labii superioris
   (b) Levator anguli oris
   (c) Orbicularis oris
   (d) Depressor labii inferioris
   (e) Depressor anguli oris
3. Which of the following is not a brow depressor?
   (a) Corrugator supercilii
   (b) Procerus
   (c) Nasalis
   (d) Orbicularis oculi

4. Which of the following does not form the modiolus?
   (a) Zygomaticus minor
   (b) Zygomaticus major
   (c) Levator anguli oris
   (d) Buccinator
   (e) Risorius
   (f) Depressor anguli oris
5. Discuss the anatomy of the nasolabial fold.
6. Discuss the origin of marionette lines and jowling.
7. Of the following regions, which has the thinnest covering of skin?
   (a) Upper lid
   (b) Lower lid
   (c) Nasal dorsum
   (d) Nasal tip
   (e) Neck
8. Discuss intrinsic and extrinsic factors and mechanisms believed to result in changes to the skin seen with age.
9. Discuss the mechanism of botulinum toxin.
10. Which of the following is not an FDA-approved usage of Botox?
    (a) Strabismus and blepharospasm
    (b) Primary axillary hyperhidrosis
    (c) Glabellar lines
    (d) Lateral canthal lines
    (e) Perioral lines
11. Name eight properties of an ideal volumizing filler.
12. The duration of effect of injectable calcium hydroxyapatite (Radiesse®) for moderate-to-severe wrinkles is:
    (a) 2–4 months
    (b) 4–6 months
    (c) 6–12 months
    (d) Permanent
13. Which of the following (in isolation) is inappropriate for facial augmentation?
    (a) Collagen
    (b) Hyaluronic acid
    (c) Calcium hydroxyapatite
    (d) PLLA
    (e) PMMA
14. Which of the following facial implants results in the least tissue ingrowth?
    (a) Porous polyethylene
    (b) ePTFE
    (c) Dacron®
    (d) Mersilene®
15. Discuss mechanical vs. biologic creep.
16. List six complications of tissue expanders.
17. Discuss the effects of long-term tissue expansion on the epidermis, dermis, subcutaneous fat, and dermal vascularity.
18. What is the rate of resorption for dermal grafts?
    (a) 10%
    (b) 20%
    (c) 40%
    (d) 80%
19. What is the most common complication of tissue expanders?
    (a) Pain
    (b) Bone resorption
    (c) Implant exposure
    (d) Infection
20. Which of the following is true of hyaluronic acid fillers?
    (a) Requires pre-injection skin testing.
    (b) Hyaluronic acid is identical in all species.
    (c) They are allogenic.
    (d) Treat dynamic rhytids.
21. Which area of the head and neck has the highest rate of complications for tissue expanders?
    (a) Eyelid
    (b) Cheek/neck
    (c) Scalp
    (d) Forehead
22. How is skin testing performed prior to bovine collagen (Zyderm and Zyplast) injection?
    (a) Two skin tests administered 2 weeks apart
    (b) One skin test 4 weeks prior to procedure
    (c) No skin tests necessary
    (d) One skin test 2 weeks prior to procedure
23. What is the Tyndall effect?
    (a) Bluish tint from superficial injection of a filler
    (b) Erythema from local skin reaction to a filler
    (c) Edema from inflammatory reaction to a filler
    (d) Blanching effect from intravascular injection of a filler

24. What is the composition of Radiesse?
    (a) Polymer of glycosaminoglycan chains
    (b) Polymethyl methacrylate
    (c) Bovine collagen
    (d) Calcium hydroxyapatite particles
    (e) Poly-L-lactic acid
25. Which of the following is NOT a technique used to deliver filler?
    (a) Serial puncture technique
    (b) Linear threading technique
    (c) Jump puncture technique
    (d) Fan technique
    (e) Cross-hatching technique
26. A patient on which antibiotic treatment cannot receive BOTOX® injections?
    (a) Amoxicillin
    (b) Clindamycin
    (c) Gentamycin
    (d) Azithromycin
27. What is NOT a clinically evident skin change in tissue undergoing expansion?
    (a) Dryness
    (b) Acne
    (c) Erythema
    (d) Hyperpigmentation
28. Compared to local flaps, expanded flaps (ones made after tissue expansion):
    (a) Are more prone to infection
    (b) Have decreased vascularity
    (c) Have an enhanced survival rate
    (d) Have less contracture
29. In general, the base of a tissue expander should be _____ times the base of area to be reconstructed.
    (a) 1–1.5
    (b) 1.5–2
    (c) 2.5–3
    (d) 3.5–4
30. After the tissue expander has been placed, when does inflation usually begin?
    (a) In 2 weeks
    (b) In 2 days
    (c) In 4 weeks
    (d) In 6 weeks
31. How long does it usually take for full expansion to be reached when inflating a tissue expander?
    (a) 2–4 weeks
    (b) 4–6 weeks
    (c) 6–8 weeks
    (d) 8–10 weeks
32. During tissue expansion, exposure of the expander balloon is noticed. What should be done?
    (a) If it is early in the course of expansion, continue slow and gradual expansion.
    (b) If it is early in the course of expansion, the procedure should be aborted.
    (c) If it is late in the course of expansion, the procedure should be aborted.
33. An infection is noted around a tissue expander. What is the next step?
    (a) If it is early in the course of expansion, continue slow and gradual expansion.
    (b) Complete a course of antibiotic therapy and remove the tissue expander.
    (c) Complete a course of antibiotic therapy and continue expansion.
34. What is NOT a technique to help prevent nodule formation with injection of poly-L-lactic acid?
    (a) Slow injection while withdrawing needle
    (b) Subdermal injection
    (c) Postinjection massage
    (d) Longer latency between reconstitution and injection
35. A patient was injected with poly-L-lactic acid 3 days ago into the nasolabial fold. She returns to the office and says that the nasolabial fold has returned. The next step should be:
    (a) Injection with additional poly-L-lactic acid
    (b) Injection with hyaluronic acid
    (c) Injection with calcium hydroxylapatite
    (d) Reassurance
36. Per the manufacturer's recommendations, how should poly-L-lactic acid be injected to correct nasolabial folds?
    (a) Linear threading technique
    (b) Cross-hatching technique
    (c) Serial puncture technique
    (d) Fan technique
37. Where should calcium hydroxylapatite NOT be injected?
    (a) Lips
    (b) Nasolabial folds

(c) Marionette lines
(d) Malar eminence

38. What can be done to decrease the risk of developing antibodies to BOTOX®?
    (a) Limit total amount of Botox to less than 200 U per session.
    (b) Limit total amount of Botox to less than 100 U per session.
    (c) Minimum of 2 months between Botox injections.
    (d) Minimum of 4 months between Botox injections.
39. When malar augmentation is performed with rhytidectomy, which procedure is performed first?
    (a) Cannot place a malar implant in the same setting as rhytidectomy due to risk of implant extrusion.
    (b) Perform malar implant first.
    (c) Perform rhytidectomy first.
40. When performing fat injections, which technique is advised?
    (a) Use a larger syringe and cannula to inject depots of fat.
    (b) Apply significant positive pressure to expel fat. The extra positive pressure helps to elevate the dermis.
    (c) Inject small amounts of fat with minimal positive pressure.
    (d) Inject fat combined with tumescence for better post-operative pain control.
41. Fat augmentation should not be performed in which layer?
    (a) Intradermal
    (b) Intramuscular
    (c) Superficial to periosteum
42. The complication of a granuloma formation after filler injection can best be managed by:
    (a) Injection of 5-fluorouracil
    (b) Injection of steroid
    (c) Massage
    (d) Watchful waiting
43. What is the typical injection schedule for deoxycholic acid (Kybella)?
    (a) One time treatment
    (b) Up to two treatments 1 week apart
    (c) Up to three treatments in 2-week intervals
    (d) Up to 6 treatments in 1-month intervals
44. What is a potential complication of deoxycholic acid (Kybella) injection?
    (a) Dysphagia
    (b) Dysphonia
    (c) Diplopia
    (d) Dyspnea

Answers

1. Zygomatic and mandibular
2. (b)
3. (c)
4. (a)
5. The lip elevators (LLSAN, LLS, zygomaticus minor, sometimes zygomaticus major) send muscle fibers that attach directly to the dermis along the path of the NLF. Although present and necessary for dynamic expression in young and old, the fold deepens and is present at rest with age.
6. Marionettes and jowl lines appear only with age. Weakening of the fasciocutaneous ligaments along the anterior border of the masseter with age leads to ptosis of the premasseteric space. The ptosis ends (gets "hung-up") at the strong mandibular osteocutaneous ligament. Additionally, marionette lines are also believed to result from repetitive DAO action over time.
7. (a)
8. Intrinsic: Accumulation of cellular damage from reactive oxygen species (ROS) generation through mitochondrial oxidative metabolism over time, replicative senescence of epidermal keratinocytes and dermal fibroblasts, decreases in GAGs (esp. HA), weakening of retaining ligaments, volume loss

   Extrinsic: Photoaging (UVA affects primarily dermis; UVB affects primarily epidermis; solar elastosis), nutrition, alcohol, smoking, chronic disease
9. Enters distal neuron through receptor-mediated endocytosis and cleaves SNAP-25 (type A) or VAMP (type B), preventing docking and fusion of synaptic vesicles to presynaptic membrane (necessary for acetylcholine release into the neuromuscular junction
10. (e)
11. Biocompatible, hypoallergenic, noninflammatory, nonmigratory, non-carcinogenic,

nontoxic, resistant to infection, consistent and predictable response, natural feel, inexpensive, long-lasting, retains shape over time, short and painless injection, requires no recovery time, long shelf life, easily stored (no refrigeration)

12. (c)
13. (a)
14. (b)
15. Mechanical creep: Displacement of interstitial fluid and ground substance, with realignment of collagen fibers and fragmentation of elastin. The underlying process behind stress relaxation of tissue (decrease in the recoil force of skin when held under stretch)

    Biological creep: Cell proliferation and upregulated ECM synthesis occurring in response to stretch that functions to restore resting tension of the stretched tissue toward baseline, necessitating an increase in surface area
16. (a) Hematoma/seroma
    (b) Infection, extrusion
    (c) Mechanical failure (leakage, rupture, valve or tubing obstruction)
    (d) Skin necrosis
    (e) Nerve injury
    (f) Vascular compromise
    (g) Muscle atrophy
    (h) Fat atrophy
    (i) Bone resorption
    (j) Insufficient expansion
    (k) Flap failure (following expansion)
17. See Table 21.8.
18. (b)
19. Implant exposure. Higher risk in area of scar, atrophy, and irradiation. However, majority in uninjured, well-vascularized skin
20. (b) Hyaluronic acid has the unique property of being identical in all species; thus, its derivatives should not be antigenic across species. Hyaluronic acid is xenogenic (animal and non-animal based).
21. (b) The incidence of complications is highest in the cheek and neck (69%), forehead (50%), eyelid (33%), and scalp (17%). Tension and shearing are generated by the neck of jaw motion. Avoid subcutaneous pocket over the mandibular border or clavicle. Gravitational pooling of saline is difficult to control and can cause the implant to migrate inferiorly.

    Reference: Antonyshyn et al. 1988
22. (a) Two skin tests are administered 2 weeks apart, the second test being 4 weeks prior to the procedure (Eppley and Dadvand, 2006).
23. (a) The principle of the Tyndall effect is that different wavelengths scatter depending on

Table 21.8 Tissue response to long-term expansion

Epidermis	Dermis	Fat	Vascular plexus	Other tissues
Surface area increases without thinning of overlying epidermis Increased mitotic activity within expanded skin Epidermal height is maintained and/or enhanced, stratified organization preserved, basal layer thickened Hyperpigmentation occurs secondary to melanocyte upregulation of melanin expression Return to normal histologic appearance typically occurs within 1–2 years following expansion	Thinning occurs (30–50%), the degree of which is related to the rate of expansion Upregulation of fibroblast-mediated collagen synthesis Altered three-dimensional arrangement of ECM with fragmentation of elastic fibers Number of hair follicles remains fixed; density decreases in expanded skin	Most intolerant of expansion Thinning of adipose tissue (up to 50%) occurs with flattening and fibrous degeneration of adipocytes Some reversal of fat loss may occur following completion of expansion, but majority is permanent	Tissue expansion upregulates VEGF expression and subsequent angiogenesis Expanded skin flaps are more vascularized (in both number and caliber of vessels), are more resistant to infection, and have a higher survival rate than non-expanded skin Rate of survival of random-expanded tissue flaps is similar to that obtained for random delayed flaps	Muscle: Poor tolerance for expansion (thinning, atrophy, necrosis, weakness) Nerve: Tolerate gradual expansion (up to 30%); however, some loss of function is expected Bone: Resorption is rare; calvarial bone resorption has been described; special care should be taken in pediatric patients

the size of the substance they encounter. Blue light is scattered about ten times more than red light when passing through very small particles. Filler in a superficial placement beneath the skin scatters more light of shorter wavelength and has a bluish discoloration.

24. (d) Hyaluronic acid fillers are composed of glycosaminoglycan chains. Bovine collagen is in Zyderm. Artecoll is a suspension of PMMA microspheres. Poly-L-lactic acid is Sculptra.
25. (c)
26. (c) Aminoglycoside antibiotics may interfere with neuromuscular transmission and potentiate effect of Botox. It is recommended not to inject Botox in patients undergoing aminoglycoside treatment.
27. (b) Dryness, erythema, and occasionally hyperpigmentation are common noticeable skin changes while undergoing tissue expansion.
28. (c) Tissue expansion increased vascularity, and the enhanced blood supply allows for elevation of extensive local flaps, which are more resistant to infection. Expanded flaps have an enhanced survival rate similar to delayed flaps. Expanded flaps develop a dense fibrous capsule around the expander balloon—this leads to contracture of the flap after inset and may cause flap to become thickened and undesirable for areas that require thin and pliable flaps such as nasal reconstruction with a forehead flap.
29. (c) The base should be 2.5–3 times the area to be reconstructed.
30. (a)
31. (c)
32. (b) If exposure occurs near the completion of expansion (as is most common), inflation can be completed or shortened.
33. (c) If infection occurs early in the course of expansion, removal of the expander may be required. If antibiotic therapy controls the infection. Then expansion process can continue to completion after some delay.
34. (a) Daines and Williams (JAMA Facial Plast Surg, 2013) write that nodule formation is lower with subdermal injection, postinjection massage, a higher reconstitution volume (off-label 8:1 dilution), and reconstitution 48 hours prior to injection.
35. (d) Poly-L-lactic acid (Sculptra) provides gradual improvement. Initial injection should be an undercorrection, and after the initial swelling has resolved, the initial contour depression can appear to recur. Patients should be seen 3 weeks after injection to determine if additional injections are required. Typically three sessions are required for optimal correction.
36. (b)
37. (a) Increased risk of nodule formation when calcium hydroxylapatite (Radiesse) is injected into the lips. This could be due to the very mobile nature of lips.
38. (b) To prevent antibody formation to Botox (and thus resistance to treatment with Botox due to neutralizing effect of antibodies), it is recommended to limit the treatment dose to 100 U per session and to wait a minimum of 3 months in between injections.
39. (b) Binder (1990, Ann Plast Surg). Submalar augmentation performed before rhytidectomy in patients with deficient bone structure returns youthful contour. It can also enhance and increase the result from rhytidectomy, reducing the need for multiple facelift procedures and avoiding stretched out appearance.
40. (c) Placing fat in extremely small amounts with each pass using gentle pressure maximizes fat survival, integration, and anchoring. Small amounts of fat maximize surface area for vascularization. After harvest, fat is centrifuged to remove the nonviable layers of oil and tumescence (Coleman, 2001, Clinics in plastic surgery).
41. (a) injection of fat into the intradermal layer can cause unsightly visible isolated deposits of fat (Coleman, 2001, Clinics in plastic surgery).
42. (b) Intralesional steroids remain mainstay of granuloma treatment. Granulomas typically appear late, months to years after injection, and remain localized to injection site (Winslow, 2009, Fac Plast Sug).

43. (d) Patients can be treated at 1-month intervals repeated up to six times. However, majority of patients experience satisfactory results with four or less treatments (Georgesen and Lipner, J. Cosmet Dermatol. 2017).

44. (a) Dysphagia. Dysphagia related to volume of injection is a potential complication (Georgesen and Lipner, J. Cosmet Dermatol. 2017).

22 Genioplasty

Christian Paquet and Matthew D. Johnson

Overview

The chin is a prominent structure of the lower face and plays a vital role in facial perception, communication, and overall facial symmetry. If not appropriately proportioned with the rest of the facial features, it can often distract from other more salient attributes. The mandible is not a structure in isolation, but has a significant impact on facial shape, nasal projection, occlusion, and neck aesthetics. A well-defined jawline and appropriate chin proportion provide the patient with balance and harmony of the lower face. A strong chin is classically associated with confidence and assertiveness, while a weak chin is associated with negative traits, such as indecisiveness and shyness. Genioplasty refers to surgically altering the chin shape and position. Anytime this is considered, the mandible must be evaluated in three dimensions. In addition, it is vital to consider the patient's dental occlusion during their evaluation and exam, as orthognathic surgery may be necessary in addition or as an alternative. Cephalometry may be useful in determining the bony configuration and position of the maxilla and mandible. Genioplasty can involve either a chin augmentation or reduction procedure and is performed through either a submental or transoral approach. Chin augmentation typically involves either bony osteotomies and subsequent sliding genioplasty or the use of alloplastic implants. Techniques for chin reduction include bony osteotomies and sliding genioplasty or direct chin reduction through the use of bony reduction and fibro-fatty tissue excision.

Definitions

- *Genioplasty* – surgical repair of the chin, whereby its shape or size is altered, recontoured, or repositioned through the use of varying techniques
- *Mentoplasty* – augmentation and contouring of the chin with implant materials
- *Retrognathia* – a condition in which the mandible is normal in size, but abnormally positioned posteriorly relative to the facial plane; typically associated with *Class II malocclusion*
- *Micrognathia* – hypoplastic, underdeveloped mandible; typically associated with *Class II malocclusion*
- *Microgenia* – normal mandible with underdeveloped chin which is retruded and deficient; typically retains normal dental occlusion (Class I occlusion)

C. Paquet (✉)
Biltmore ENT, Facial Plastics & Allergy, Phoenix, AZ, USA
e-mail: info@paquetplasticsurgery.com

M. D. Johnson
Department of Surgery/Division of Otolaryngology – Head and Neck, Southern Illinois University, Springfield, IL, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_22

- *Prognathia* – a condition in which the mandible protrudes anteriorly relative to the facial plane; typically associated with *Class III malocclusion*
- *Witch's chin deformity* – prominent submental crease, lack of boney projection, and ptosis of soft tissues of the chin pad
- *Labial incompetence* – inability to achieve a relaxed, consistent lip-together rest posture resulting in mentalis muscle strain/dimpling

Anatomy

Muscles

Mentalis Muscle

- *Origin*: anterior mandible/mentum
- *Insertion*: soft tissues and skin of chin
- *Innervation*: mandibular branch CN VII
- *Function*: elevates and wrinkles the skin overlying the chin; protrudes the lower lip; produces lower lip "pout"

Depressor Labii Inferioris

- *Origin*: oblique line of mandible between symphysis and mental foramen
- *Insertion*: skin of the lower lip
- *Innervation*: mandibular branch CN VII
- *Function*: depresses and draws the lower lip laterally

Platysma

- *Origin*: skin of the upper lateral chest and lower neck
- *Insertion*: inferior border of mandible and skin over lower face and angle of mouth
- *Innervation*: cervical branch of CN VII
- *Function*: depresses and wrinkles the skin of the lower face and mouth; aids in forced depression of the mandible

Geniohyoid

- *Origin*: inferior mental spine of mandible (posterior surface of symphysis)
- *Insertion*: hyoid bone
- *Innervation*: C1 via hypoglossal nerve
- *Function*: elevates the hyoid, depresses the mandible

Mylohyoid

- *Origin*: mylohyoid line on internal aspect of mandible
- *Insertion*: body of hyoid bone
- *Innervation*: mylohyoid nerve (branch of V3)
- *Function*: elevates the floor of mouth, elevates the tongue and hyoid, depresses the mandible

Digastric Muscle

- *Origin:* anterior belly on digastric fossa of symphysis, posterior belly on mastoid notch
- *Insertion*: intermediate tendon (hyoid)
- *Innervation*: anterior belly (V3) posterior belly (VII)
- *Function*: elevates hyoid, depresses mandible

Physical Exam

After a detailed consultation with the patient to identify their concerns and goals, the preoperative consult should include a complete history and physical exam, including a dental history. Standard facial photographs should be obtained.

Of note, the physical exam should also include an evaluation of the occlusion, chin position and projection, labiomental crease, hyoid position, nose, lips, and facial proportions. If a skeletal/dental abnormality exists, the patient may require an orthodontic evaluation for possible orthognathic surgery.

While not commonly obtained, additional evaluations include a lateral soft tissue study, lateral cephalometric study, anteroposterior (AP) skull radiography, and occlusal panoramic radiograph.

Occlusion

Prior to evaluation of chin size and position, dental occlusion should be evaluated and the patient's occlusal class established. When assessing occlusion, observe for significant variation between the patient's centric occlusion and centric relation; occlusion should be assessed in centric relation with the condyle seated in the temporomandibular joint in the most superior position.

Overbite describes the overlapping of the maxillary incisors in relation to the mandibular incisors. Normal overbite is around 3–5 mm or 25–30% the height of the mandibular incisor. *Overjet* describes the horizontal (anteroposterior) extent that the maxillary incisors project beyond the mandibular incisors.

Patients with malocclusion often have an abnormal chin profile and position. A retrodisplaced chin may be associated with Class II malocclusion, and an anteriorly displaced chin may be associated with Class III malocclusion. *Patients who have malocclusion should be evaluated and offered orthognathic surgery prior to consideration of genioplasty or mentoplasty*. Patients who are not interested in orthognathic surgery may still be considered candidates for genioplasty or mentoplasty.

Angle's Classification (1899) (Fig. 22.1)

1. Class I is orthognathic, neutroclusion.
   (a) First molars contact normally; mesiobuccal cusp of first maxillary molar sits in mesiobuccal groove of first mandibular molar.
2. Class II is retrognathic, distoclusion.
   (a) First mandibular molar is displaced posteriorly with respect to the first maxillary molar (cusp sits anterior to the groove). Maxillary canine is anterior to embrasure formed by mandibular first bicuspid and canine.
3. Class III is prognathic, mesioclusion.
   (a) First mandibular molar is displaced anteriorly with respect to the first maxillary molar (cusp sits posterior to the groove).

Chin

Normal soft tissue thickness of the chin pad is 8–11 mm. It is important to identify the structural anatomy of the chin. Chin abnormalities occur in three dimensions; therefore, assessment must include all three planes: vertical plane (frontal and lateral views), transverse plane (frontal view), and anteroposterior plane (lateral view). Most deformities occur in the anteroposterior plane Figs. 22.2 and 22.3 assessment and facial divisions.

There are multiple methods for assessing the projection of the chin. A common measurement for assessing adequate chin projection is its relationship to the lower lip. A man's chin should approach, but not exceed, a line from the lower vermillion border in repose. A woman' chin, however, is ideally situated 2–3 mm behind this

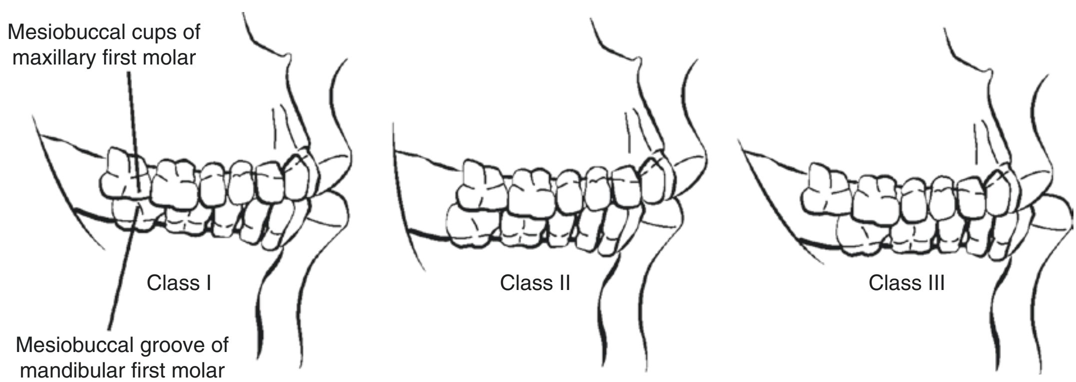


Fig. 22.1 Occlusal classes

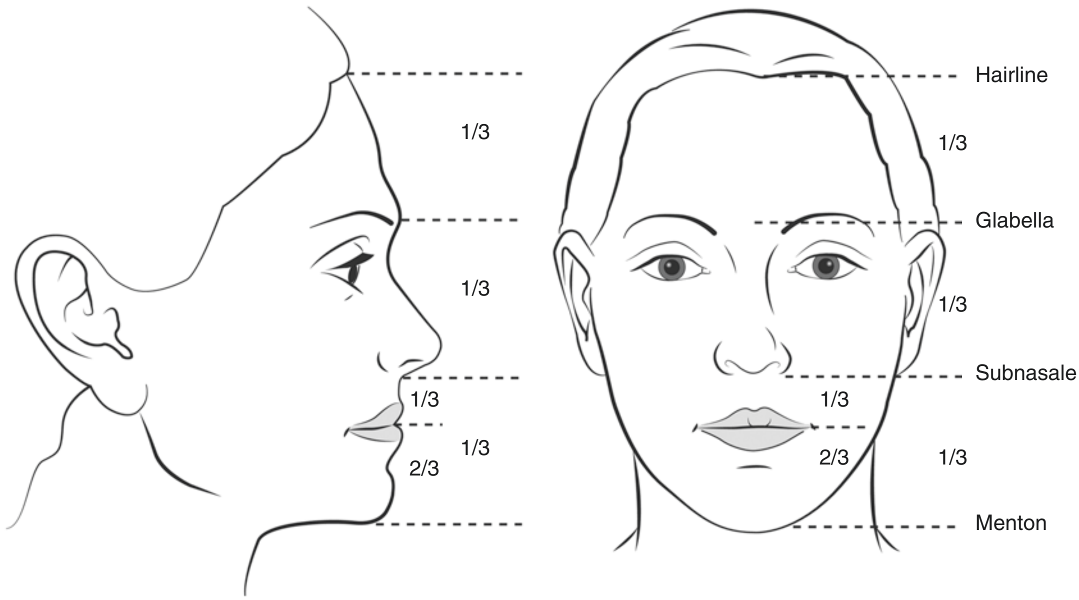


Fig. 22.2 Horizontal thirds of the face in anterior and lateral views

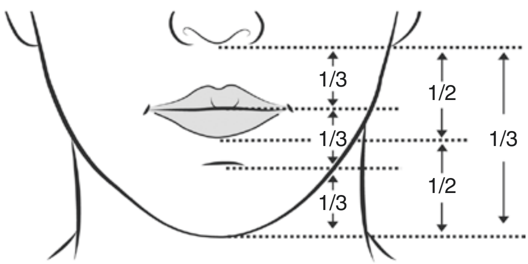


Fig. 22.3 Horizontal divisions of the lower third on anterior view

line. However, no single method provides comprehensive evaluation but allows a reference for assessing the chin and soft tissue of the lower face:

- Chin should approach a line drawn perpendicular to the Frankfort horizontal line intersecting the vermillion border of lower lip.
- Pogonion should lie tangent to an oblique line drawn through the most anterior aspects of the upper and lower lips (Fig. 22.4).
- A line drawn perpendicular to the Frankfort horizontal line through the subnasale has a consistent relationship with the upper lip vermillion (touches the line), lower lip vermillion (2 mm posterior to line), and pogonion (4 mm posterior to line) (Fig. 22.4).
- *Steiner* – line drawn from pogonion to inflection point of columella; the upper and lower lips should touch this line (Figs. 22.5 and 22.6).
- *Ricketts* – line is placed tangent to the tip of the nose and pogonion; the upper lip is ~4 mm behind this line and the lower lip ~2 mm behind (Figs. 22.5 and 22.6).
- *Burstone* – line is placed tangent to the subnasale and pogonion; the upper lip extends ~3.5 mm beyond this line and the lower lip ~2.2 mm beyond this line (Figs. 22.5 and 22.6).
- *Gonzalez-Ulloa Zero Meridian* –line drawn perpendicular to the Frankfort horizontal line through the nasion → pogonion should be within 5 mm.
- *Legan's Angle* – angle defined by glabella-to-subnasale line intersection with subnasale-to-pogonion line (normal 8–16 degrees).
- *Merrifield Z-Angle* – angle defined by Frankfort horizontal line intersection with pogonion-to-upper lip vermillion border line (normal 75–85 degrees).

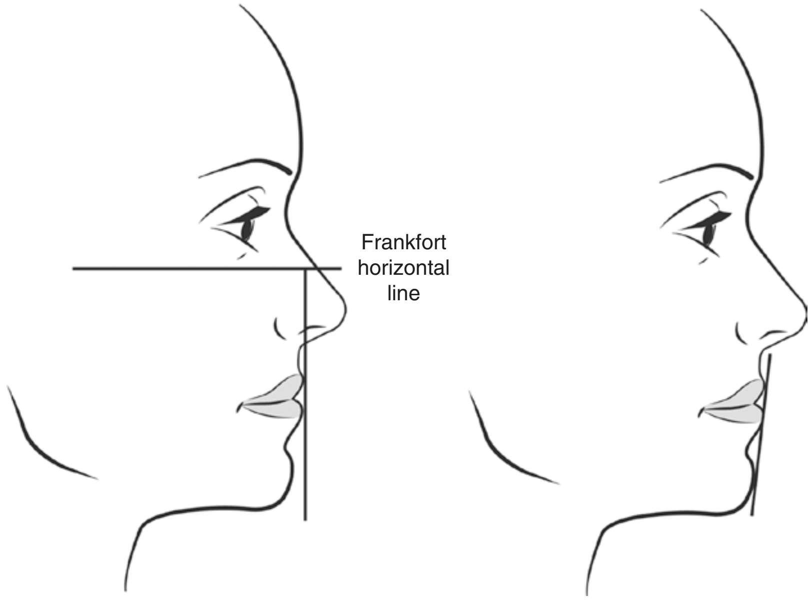


Fig. 22.4 Chin position. *Left*, a line is dropped perpendicular to the Frankfort horizontal line through the subnasale where pogonion is ~4 mm behind line. *Right*, oblique line touching most anterior projection of upper and lower lips should lie in tangent to pogonion

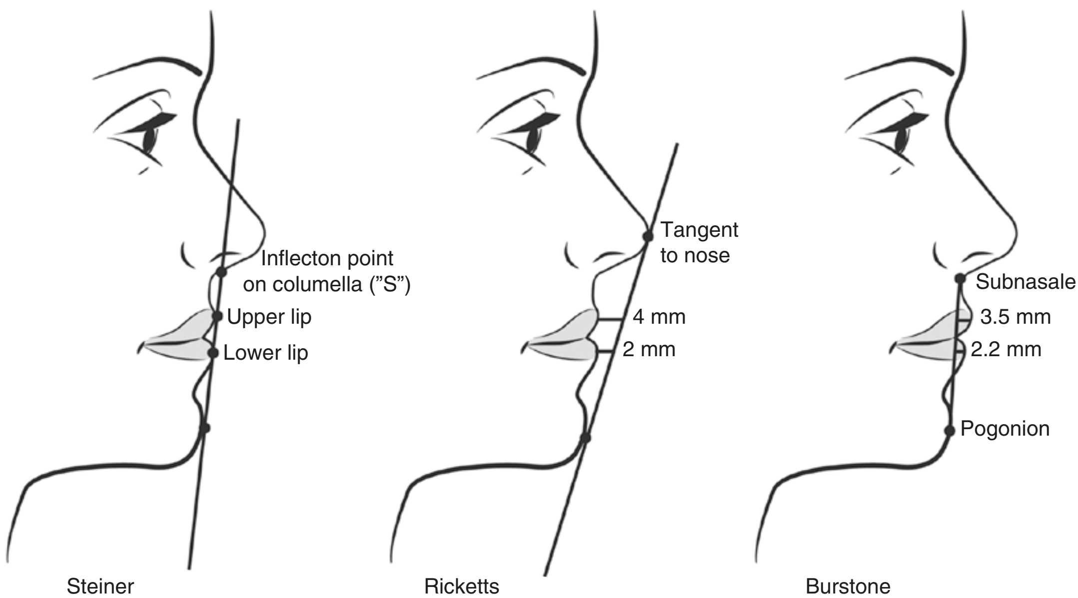


Fig. 22.5 Chin position. Steiner, Ricketts, and Burstone methods

Fig. 22.6 Assessment of chin position on lateral cephalogram

Merrifield's Z-angle	Formed by a line from soft tissue chin (Po) tangent to most procumbent lip Z-angle should be 80 +/- 5 degrees	
Gonzales-Ulloa-Steven's	Line perpendicular to Frankfurt horizontal line passing through soft tissue nasion Chin should be tanget to this line	
Ricketts law of lip relationships	Line from tip of nose to chin Aesthetically pleasing profiles have upper lip 4mm behind and lower lip 2 mm behind	
Holdeqay's H-line and H-angle	Line tangent to the chin and upper lip that forms an angle with a line between the nasion and basion of 7-9 degrees	
Zimmer's aesthetic plane	Line from anterior nasal spine to Down's "B" point Nose, lips, and chin are almost identical in thickness to this plane Nose ratio of 5:1 to any other soft tissue structure	
Reidel's plane	Lips and chin generally fall on a straight line	
Steiner's aesthetic plane	Plane from middle of columella, midway between cureves of upper lip and nasal tip Lips should fall on this line	

Methods of Vertical Analysis

- Golden Proportion
  - Ratio of upper lip to lower lip and chin equals 1:1.618; the upper lip is ~62% the height of the lower lip and chin.
- Vertical Thirds – Trichion-to-Glabella = Glabella-to-Subnasale = Subnasale-to-Menton
- Subnasale to Menton = lower 1/3 of face
  - Further subdivided:
    Subnasale to stomion = 1/3
    Stomion to menton = 2/3

Other Associated Features

- Labiomental Fold
  - Marks the junction of the mentalis with the orbicularis oculi. If muscle overlap occurs, the fold is indistinct. If the orbicularis is thin or narrow, the fold is high.
  - The depth and height of the labiomental fold should be carefully evaluated. Powell and Humphrey suggest "ideal" labiomental depth should 4 mm from a vertical line drawn between the labrale inferius and soft tissue pogonion. Males can tolerate a deeper fold (up to 6 mm) than females.
  - Advancing the chin anteriorly or superiorly will deepen the labiomental crease. In contrast, inferior or posterior movements soften the labiomental crease.
  - High labiomental fold – poor candidate for augmentation secondary enlargement of the entire lower face.
  - Low labiomental fold – good candidate for augmentation. Augmentation enlarges chin only.
- Lip
  - The ideal lip relationship is one in which the upper lip projection is 2 mm beyond the lower lip and the lower lip projection is 2 mm beyond the chin.
  - The position of the lower lip determines the degree of chin augmentation needed. Advancement beyond the lower lip leaves the patient with a distracting, artificial appearance.
- Interlabial Distance
  - Normally 1–5mm with relaxed lip. Lower lip contributes more to closure of the interlabial gap than the upper lip.
  - An increased interlabial distance requires a greater degree of contraction from mentalis muscle and characteristic chin dimpling.
  - Increased distance can result in *labial incompetence*. Characteristic finding in patients with anterior open bite, vertical maxillary excess, long face syndrome, Down syndrome, severe microgenia, and bimaxillary protrusion.
- Nose
  - The relationship of the chin to the nose is instrumental in the appropriate preoperative assessment for both chin augmentation and aesthetic rhinoplasty. The size of one will influence the perceived appearance of the other.
  - The perception of nasal projection can be altered by adjusting chin position and vice versa. Augmenting the chin will give the appearance of decreased nasal projection, and a reducing the chin will give the appearance of increased nasal projection.
- Neck
  - An aesthetically attractive neck is characterized by a sharply defined cervical mental angle with a well-defined jawline that is in harmony with the upper two thirds of the face. The chin is the keystone structure linking the face and neck. Augmentation may be enhances with soft tissue procedures (liposuction, platysmal plication, rhytidectomy).
  - Ideal cervical mental angle is 105°–120°.
  - The hyoid position is important to consider prior to approaching chin augmentation procedures that are performed in conjunction with neck.
  - A favorable hyoid position may benefit from the placement of an alloplastic implants alone when combined with submentoplasty.

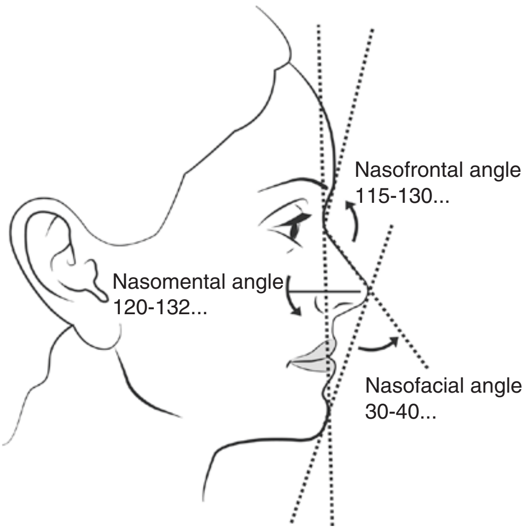


Fig. 22.7 Nasofrontal angle (defined by glabella-to-nasion line intersecting with nasion-to-tip line), nasofacial angle (defined by glabella-to-pogonion line intersecting with nasion-to-tip line), and nasomental angle (defined by nasion-to-tip line intersecting with tip-to-pogonion line) provide the boundaries of the aesthetic triangle of Powell and Humphreys

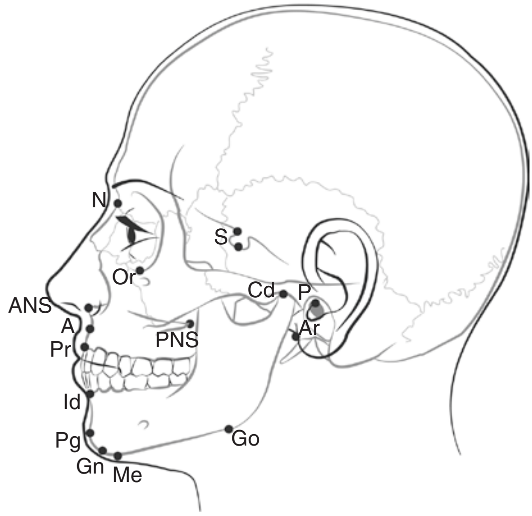


Fig. 22.8 Cephalogram points. Point A, subspinale or most concave point on maxilla; ANS, anterior nasal spine; Ar, articulare; Point B (not shown), supramentale or most concave point on mandible; Cd, condylion (alternatively denoted Co); Gn, gnathion; Go, gonion; Id, inferior prosthion; Me, menton; N, nasion; Or, orbitale; P, porion; Pg, pogonion; PNS, posterior nasal spine; Pr, prosthion; S, sellion

- An unfavorable low set hyoid position may benefit more with a sliding genioplasty to provide a sharper cervical mental angle and a better outcome secondary to advancement of muscular insertions and hyoid elevation.

Powell and Humphrey's triangle (Fig. 22.7)

Cephalometric Imaging

- Cephalometric assessment of maxillary and mandibular position assists in relating the position of the chin to that of the upper and lower jaws and, thus, occlusion. These should be obtained when patients are undergoing sliding genioplasty or when there is concern for abnormal bony anatomy.
- *Cephalometric Points*: Fig. 22.8
  - N – nasion: most anterior point on frontonasal suture
  - S – sella: midpoint of sella turcica
  - A – subspinale: position of deepest concavity on anterior profile of maxilla
  - B – supramentale: position of deepest concavity on anterior profile of mandibular symphysis in the outer contour of the mandibular alveolar process
  - Me – menton: lowest most point on the mandible
  - Pg – pogonion: most anterior point on the bony chin in the midline
  - Gn – gnathion: midpoint between the (Pg) and the (Me)
  - Go – gonion: the midpoint at the angle of the mandible, the most posterior inferior point of the mandible
- Common Reference Angles: Fig. 22.9
  - S-N-A: sella-to-nasion-to-subspinale angle: anteroposterior position of maxilla, 81° (±3), and mean of 82°.
  - S-N-B: sella-to-nasion-to-supramentale angle: anteroposterior position of maxilla, 79°(±3), mean of 80°.
  - These angles provide information regarding the lateral relationship between the anterior skull base, maxilla, and mandible.

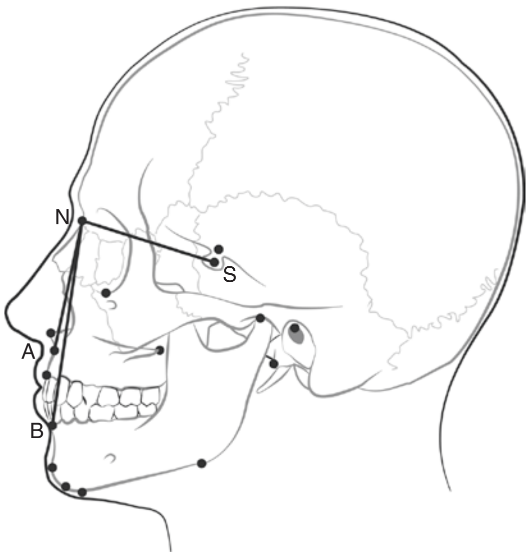


Fig. 22.9 Lateral cephalogram angles. SNA and SNB

– A-N-B: difference between SNA and SNB
  If greater than 4°, skeletal Class II malocclusion.
  If greater than 4° and SNA is greater than normal, Class II malocclusion is due to maxillary protrusion.
  If greater than 4° and SNA is less than normal, mandibular retrognathia is probable.

Surgery

Genioplasty or mentoplasty can be performed through an external (submental) or intraoral (gingivobuccal) approach.

Submental allows for placement and stabilization of the utilizing an incision 2 mm behind the submental crease.

- Advantages: no division of mentalis muscle, ability to perform adjuvant neck procedures (submental liposuction, platysmal plication)
- Disadvantage: external scar

Transoral gingivobuccal sulcus incision is made inside the lower lip extending laterally to the canine/cuspid region. The mentalis muscle is traversed; subperiosteal elevation is performed exposing the symphyseal region. Placement is on the pogonion.

- Advantages: no external scar
- Disadvantages: larger incision, intraoral contamination, difficult to stabilize implant internally, potential chin ptosis
- The surgeon must ensure accurate re-approximation of the mentalis muscle to prevent superior malposition of the implant, chin pad ptosis, and lower lip malposition/dysfunction (witch's chin deformity).

Alloplastic Mentoplasty

Alloplastic implants remain the most popular technique for chin augmentation. Alloplastic implants primarily affect the chin in the anteroposterior (horizontal) dimension. The procedure can be performed under local anesthesia in the office or in an outpatient surgery setting. While the most commonly used alloplastic chin implant is made of pliable silicone, there are a wide variety of implant materials available, which are delineated below (Table 22.1).

The surgical approach may be either submental or intraoral. Care must be taken to ensure symmetric placement of the implant both in the vertical plane and when aligning the implant in the midline at the symphysis. Placement can be in the subperiosteal or supraperiosteal plane. Subperiosteal placement is associated with improved fixation. The subperiosteal dissection should only be as big as necessary to accept the implant.

It is important to stabilize implant at time of placement to avoid displacement. Some surgeons advocate supraperiosteal placement over the symphysis and subperiosteal placement laterally to improve fixation to limit erosion. Other options to secure the implant include sutures or screws. Screw fixation prevents movement and allows for precise contouring for desired augmentation, elimination of dead space between the implant and jawline, and improved skeleton-implant transitions.

Table 22.1 Alloplastic implants

Implants	Advantages	Disadvantages
Silastic (most commonly used)	Essentially inert Firm Easily sculpted	Rare foreign body reaction and rejection Infection risk Bone resorption below implant Extrusion when superficial
Polytetrafluoro-ethylene (Gore-Tex)	Perforated implant Pore size 10–30 μm Some soft tissue ingrowth Less tendency for migration	Some tissue ingrowth
Polyethylene (MEDPOR)	Firmer implant Pore size 125–250 μm More extensive fibrous ingrowth	More soft tissue adherence Difficult to remove
Supramid (organopolymer related to nylon and Dacron)	Stable, reliable Infiltrated by host tissues Slightly more resistant to infection	Extrusion after infection Foreign body response Possible resorption Does not give firm support
Proplast (highly porous Teflon polymer and vitreous carbon fibers)	Tissue reactivity and ingrowth Firm Easy to shape	Less resistant to infection and extrusion

Final augmentation stabilizes 2 years following placement at 80% thickness (10 mm implant yields 8 mm soft tissue increase). This is due to small amount of settling of the implant, bone resorption, and soft tissue contraction. Sherris et al. studied bony erosion following silastic mandibular implant placement and found no statistical difference in mandibular resorption with supraperiosteal versus subperiosteal placement.

Disadvantages to alloplastic mentoplasty include the limited ability to correct for asymmetries or vertical excess, the potential for infection/extrusion, soft tissue distortion, implant migration, and deepening of the labiomental crease. Care must be taken in patients with preoperative labial incompetence who demonstrated chin dimpling and strain due to increased risks of resorption or displacement.

Osseous (Sliding) Genioplasty

Osseous genioplasty is the second most commonly performed osteotomy of the facial skeleton for both reconstructive and aesthetic reasons (osteotomies in rhinoplasty being the first). While most surgeons agree that alloplastic implantation is technically easier to perform, osseous genioplasty remains a more versatile procedure, able to address deformities in all three dimensions.

Indications for osseous genioplasty include correction of vertical height excess/deficiency, extreme microgenia, hemifacial atrophy, mandibular asymmetry, and failed alloplastic chin augmentation.

The horizontal osteotomy is generally placed 5 mm below the mental foramen. If no vertical change is desired, the osteotomy is made parallel to the occlusal plane. This provides for anteroposterior movement only. The osteotomy is carried as far posteriorly as possible (ideally to gonial notch) to ensure a natural look with adequate skeletal volume advancement. Failure to complete the osteotomy at the lingual cortex may result in a mandibular fracture, nerve injury, irregular inferior mandibular border, and/or altered aesthetics. The upper limit of bony advancement is generally considered 8–10 mm; greater than this amount requires either stair-step osteotomies or release of muscular attachments.

Vertical height changes can be achieved by employing angled osteotomies, interposition grafting, and wedge resections. Vertical augmentation of lower face height can be performed with an interposition graft using autologous grafting material (generally a bone graft). Vertical reduction is generally accomplished using a wedge osteoectomy of mandibular bone utilizing two parallel horizontal osteotomies with removal of the intervening segment.

Stabilization of the bony segment can be performed by various techniques including unicortical or bicortical wires, adaptation plates, pre-bent chin plates, lag screws, and absorbable plating materials.

The major disadvantages of the osseous genioplasty approach are increased surgical time, longer healing time, injury to dental roots, the potential for unnatural bony contours, step-off deformities, or notching.

Correction of Witch's Chin Deformity

An overlooked ptotic chin pad can lead to an unfavorable depression in the submental crease and create an appearance of meanness and anger. Corrected through submental approach with elevation of skin over ptotic tissue pad. Subperiosteal pocket is created along inferior border of mandible which enables placement of alloplastic implant and/or tissue removal. Mentalis is advanced inferiorly and posteriorly over platysma and secured with sutures.

Postoperative Care and Recovery

Pressure dressings are placed postoperatively and left in place for 3–5 days to minimize the chance of hematoma and assist with soft tissue reattachment after degloving. Patients are generally placed on a 1-week course of antibiotics postoperatively. Patients are followed for varying periods of time.

Complications

Complications are generally minor and can be avoided by appropriate patient selection and technical execution. The surgeon must take care to avoid complications such as an infection, tooth injury, nerve damage, mandibular fracture, and undesirable aesthetic outcomes. Chin implant removal from any approach can result in chin pad configuration changes that are challenging to fix. Some authors recommend placing a smaller implant at the time of removal to prevent pocket contraction or osseous genioplasty to limit postoperative contour irregularity.

Bony resorption after alloplastic mentoplasty occurs under the implant with mean resorption volumes reported as 0.1 mm/year. Etiologies include improper implant placement (too high over cortical bone), increased pressure secondary to oversized implant, subperiosteal placement, gender, age, implant characteristics, and labial incompetence. The majority of the time this is of no cosmetic concern, but in cases of severe resorption, implant removal is recommended. Osseous genioplasty may be required for correction following implant removal.

Undesirable aesthetic outcomes can be the result of soft tissue changes and wound contracture, pre-existing asymmetries, implant malposition, or overcorrection. Chin ptosis can result from an inferior redistribution of the soft tissues of the chin. This can often occur if the mentalis muscle is not re-approximated, leaving an appearance of submental skin redundancy, flattening of the labiomental fold, excessive show of the mandibular teeth, and lip incompetence when severe.

Pre-existing or undiagnosed asymmetries may become more apparent after augmentation. Careful examination preoperatively and disclosure of these asymmetries is important so the surgeon is not blamed postoperatively. Maintaining appropriate implant orientation intraoperatively with markings and measurements minimizes the risk of iatrogenic asymmetry. Malposition may also occur due to capsular contraction and movement; therefore, care is taken to fix the implant to the mandible. Revision surgery should generally not be undertaken earlier than 6 months postoperatively (with rare exceptions).

While infection is quite unusual (<1%), it is always a concern. Meticulous attention to sterile technique can minimize the majority of surgical infections. This includes minimal handling of the implant, appropriate antibiotic coverage, and appropriate oral hygiene postoperatively. Porous implants (due to tissue ingrowth) and the intraoral approach generally have a slightly higher incidence of infection. If infection does occur at any point, the implant should be removed, the infection treated, and re-implantation performed at a later date.

Tooth injury can result from direct damage to the tooth roots or from compromised pulpal blood flow. Therefore, during osseous genioplasty, it is recommended that osteotomies be placed at least 5 mm below the tooth roots.

The majority of neurosensory disturbances are temporary secondary to neuropraxia. Nerve compression from capsule contraction and implant malposition should be corrected. Nerve damage can be avoided by intimate knowledge of the anatomy and attention to detail. If a bony sliding genioplasty is to be performed, it is important to expose the mental foramen bilaterally. The foramen is located below the second premolar at the approximate vertical midpoint of the mandibular height. In the edentulous patient, the nerve may be located more superficially due to alveolar recession. If nerve transection occurs, immediate repair is indicated

Mandibular complications include nonunion, malunion, avascular necrosis of the mobilized segments, bony resorption under the implant, and fractures. To minimize the risk of necrosis, maintenance of a wide soft tissue pedicle with attached periosteum to the symphysis is important. Care is also taken to only expose the amount of bone necessary for augmentation. Bone deposition and osseous remodeling occurs in a somewhat consistent and predictable pattern. There is approximately ~10% resorption rate of bone that is moved. Mandibular fractures can occur with osseous genioplasty, and they are generally due to an incomplete osteotomy through the buccal and lingual cortices. The fracture occurs during the mobilization of the inferior segment and often extends through the mandibular body, angle, or ramus. Management involves completion of the osteotomy, followed by routine mandibular fracture postoperative care.

Questions

1. What is the ratio of soft tissue change to bony genioplasty change?
   (a) 1:1
   (b) 0.6:1
   (c) 0.8:1
   (d) 1.2:1
2. What is the ratio of soft tissue change to alloplastic chin implant change?
   (a) 1:1
   (b) 0.6:1
   (c) 0.8:1
   (d) 1.2:1
3. Moving the chin forward or superiorly will have what effect on the labiomental crease?
   (a) Soften the crease
   (b) Elevate the crease
   (c) Deepen the crease
   (d) Lower the crease
4. Where should the osteotomy cut for a genioplasty be made?
   (a) 10 mm below the mental foramen
   (b) Directly below the apical tooth roots of the mandibular teeth
   (c) At the level of the labiomental crease
   (d) 5 mm below the mental foramen
5. Postoperatively, a patient who underwent genioplasty has lower lip incompetence and incisor show. This is prevented by:
   (a) Watertight closure
   (b) Limiting osseous advancement of ≤10 mm
   (c) Resuspending the mentalis muscle in the midline
   (d) Preservation of the lateral mental neurovascular bundles
6. Recessive chin is most often associated with all of the following EXCEPT:
   (a) Horizontal midline shift
   (b) Recessive procumbent lower lip
   (c) Deep labiomental fold
   (d) Diminished to normal lower facial height
7. Preoperative evaluation of a patient for chin surgery. What radiographic study do you order?
   (a) Anteroposterior skull radiograph.
   (b) Lateral cephalogram.
   (c) Orthopantomograph.
   (d) No radiologic tests are necessary.
8. A woman has a long chin and blunted labiomental fold. What is the best method to improve the aesthetics of her chin?
   (a) Mentoplasty with silastic implant and wafer
   (b) Sliding osseous genioplasty
   (c) Recontouring of the mentum by burring down
   (d) Acute-angle osteotomy genioplasty

9. The relationship of the pogonion to a line drawn perpendicular to the Frankfort horizontal line through the subnasale?
   (a) It is located at or just behind the line.
   (b) It is located 2 mm behind the line.
   (c) It is located 4 mm behind the line.
   (d) It participates in the determination of the facial plane.
10. Normal chin pad soft tissue thickness is?
   (a) 2–5 mm
   (b) 4–7 mm
   (c) 6–9 mm
   (d) 8–11 mm
11. All of the following are true of alloplastic chin augmentation except?
   (a) Patient satisfaction rate ranges from 85% to 90%.
   (b) Bony resorption is common and a significant aesthetic issue requiring removal.
   (c) Infection rates are equal between intraoral and cutaneous approaches.
   (d) Silastic, Gore-Tex, and Mersilene mesh are all examples of alloplastic implants.
12. The mental nerve is typically located?
   (a) Below the mandibular canine
   (b) Below the first mandibular premolar
   (c) Below the second mandibular premolar
   (d) Below the first mandibular molar
13. All of the following are examples of commonly used alloplastic chin augmentation materials except?
   (a) Gore-Tex
   (b) MEDPOR
   (c) Silastic
   (d) Titanium
14. 3–12% is the incidence of postoperative?
   (a) Chin hypoesthesia or dysesthesia
   (b) Wound infection
   (c) Lower lip retraction
   (d) Hematoma
15. All of the following are features of silicone implants except?
   (a) Silicone is highly biocompatible, with minimal to no clinical toxicity.
   (b) A fibrous capsule commonly grows around a silicone implant.
   (c) The porous nature of silicone allows for soft tissue ingrowth.
   (d) Silicone maintains its shape and strength even at high temperatures.
16. All of the following are innervated by the facial nerve except?
   (a) Mentalis
   (b) Mylohyoid
   (c) Platysma
   (d) Depressor labii inferioris
17. What is the relationship of the maxillary and mandibular canines in Angle Class II malocclusion?
   (a) The maxillary canine occludes with the distal half of the mandibular canine and the mesial half of the mandibular first premolar.
   (b) The distal surface of the mandibular canine is posterior to the mesial surface of the maxillary canine.
   (c) The distal surface of the mandibular canine is mesial to the mesial surface of the maxillary canine.
18. A patient who had an alloplastic chin implant placed several years prior presents with an acute infection in the region of the mentum. What is the appropriate management?
   (a) Surgical removal of the implant with re-implantation several weeks/months later
   (b) Admission for IV antibiotic coverage including treatment for MRSA
   (c) Trial of an outpatient course of oral antibiotics with MRSA coverage
   (d) Surgical removal of the implant, washout, and immediate placement of a new implant
19. All of the following are appropriate techniques for alloplastic implant fixation except?
   (a) Suture fixation to the periosteum
   (b) Screw fixation to the underlying mandible
   (c) Placement of the implant in a carefully dissected narrow pocket
   (d) All of the above
20. In cephalometric analysis, an ANB angle of $<1^\circ$ indicates what?
   (a) Normal occlusion
   (b) Class II skeletal jaw relationship

(c) Class III skeletal jaw relationship
(d) Crossbite

21. Technical maneuvers associated with successful submental placement and long-term stability of alloplastic implants include all the following except?
    (a) Wide subperiosteal exposure
    (b) No division of the mentalis muscle
    (c) Screw fixation of the implant
    (d) Postoperative use of chin strap
22. A patient undergoes supraperiostal placement of an extended Silastic implant via an intraoral approach, which was secured with a 6 mm titanium screw to the symphysis. The patient returned for follow-up POD7 complaining of left lower lip numbness. She has been wearing her chin strap and denies fever, drainage, asymmetry, or pain. Examination of the patient 3 months later is significant for continued left-sided lower lip numbness and asymmetry. This complication could have been avoided by all the following except:
    (a) Submental approach with wide exposure
    (b) Subperiosteal placement of the implant
    (c) Lateral fixation of implant to mandible
23. A patient presents for improvement in his chin weakness. Physical examination reveals Class II occlusion with a 5 mm of overjet, vertical deficiency, and micrognathia. Which of the following techniques is best suited to improve this patients concern?
    (a) Silastic extended implant
    (b) Sliding genioplasty
    (c) Sagittal mandibular split with advancement
    (d) Expanded polytetrafluoroethylene (e-PTFE) implant
24. Advantages of the extended "anatomical" implants over standard alloplastic implants include all the following except:
    (a) Correction of prejowl sulcus depressions
    (b) Correction vertical height deficiencies
    (c) Prevention of "button chin" deformities
    (d) Improved mandible contour
25. A 52-year-old female presents requesting improvement in her neck profile. Physical exam is significant for a blunted cervicomental angle with submental adipose fullness, skin laxity, platysma banding, retrognathia with a low labiomental fold, lack of a lateral jawline definition, and a low set hyoid position. Which procedures would optimize the appearance of the neck in this patient?
    (a) Submental liposuction, sliding genioplasty
    (b) Platysmaplasty, submental liposuction, alloplastic implant
    (c) Platysmaplasty, submental liposuction, sliding genioplasty
    (d) Submental liposuction, alloplastic implant

Answers

1. (a)
2. (c)
3. (c)
4. (d)
5. (c)
6. (a)
7. (b)
8. (d)
9. (c)
10. (d)
11. (b)
12. (c)
13. (d)
14. (a)
15. (c)
16. (b)
17. (b)
18. (a)
19. (d)
20. (c)
21. (a)
22. (d)
23. (c)
24. (b)
25. (c)

Additional Resources

1. Adams JS. Grafts and implants in nasal and chin augmentation: a rational approach to material selection. Otolaryngol Clin N Am. 1987;20(4):913–30.
2. Chang EW, Lam SM, Karen M, Donlevy JL. Sliding genioplasty for correction of chin abnormalities. Arch Facial Plast Surg. 2001;3(1):8–15.
3. Cheney ML. Facial surgery: plastic and reconstructive. 1st ed. Baltimore: Williams and Wilkins; 1997.
4. Choe KS, Stucki-mccormick SU. Chin augmentation. Facial Plast Surg. 2000;16(1):45–54.
5. Edwards RC, Kiely KD, Eppley BL. Resorbable fixation techniques for genioplasty. J Oral Maxillofac Surg. 2000;58(3):269–72.
6. Glasgold AI, Glasgold MJ. Intraoperative custom contouring of the mandible. Arch Otolaryngol Head Neck Surg. 1994;120(2):180–4.
7. Henderson JL, Larrabee WF, Krieger BD. Photographic standards for facial plastic surgery. Arch Facial Plast Surg. 2005;7:331–3.
8. Hoenig J. Sliding osteotomy genioplasty for facial aesthetic balance: 10 years of experience. Aesthet Plast Surg. 2007;31:384–91.
9. Kim SK, Han JJ, Kim JT. Classification and treatment of prominent mandibular angle. Aesthet Plast Surg. 2001;25(5):382–7.
10. Papel ID. Facial plastic and reconstructive surgery. 2nd ed. New York: Thieme Medical Publishers, Inc.; 2002.
11. Pearson DC, Sherris DA. Resorption beneath silastic mandibular implants: effects of placement and pressure. Arch Facial Plast Surg. 1999;1:261–4.
12. Saleh HA, Lohuis PJ, Vuyk HD. Bone resorption after alloplastic augmentation of the mandible. Clin Otolaryngol. 2002;27(2):129–32.
13. Sykes J, Frodel JL. Genioplasty. Op Tech Otolaryngol. 1995;6:319.
14. Wang J, Gui L, Xu Q, Cai J. The sagittal curving osteotomy: a modified technique for advancement genioplasty. Plast Reconstr Aesthet Surg. 2007;60:119–24.
15. Wider TM, Spiro SA, Wolfe SA. Simultaneous osseous genioplasty and meloplasty. Plast Reconstr Surg. 1997;99(5):1273–81.
16. White JB, Dufresne CR. Management and avoidance of complications in chin augmentation. Aesthet Surg J. 2011;31(6):634–42.
17. Goldsmith D, Horowitz A, Orentlicher G. Facial skeletal augmentation using custom facial implants. Atlas Oral Maxillofac Surg Clin North Am. 2012;20(1):119–34.
18. Yaremchuk MJ. Improving aesthetic outcomes after alloplastic chin augmentation. Plast Reconstr Surg. 2003;112(5):1422–32.

23 Hair Restoration

Michelle G. Arnold and Scott R. Owen

Embryology

Hair development begins around the 10th week of gestation and is completed by the 18th week [1].

Ectoderm epithelial placode invaginates into the dermis to form the components of the intrinsic hair follicle and melanocytes.

Mesoderm Forms supportive/accessory structures of the follicular subunit, including the arrector pili muscle, follicular sheath, dermal papilla, and feeding vessels.

Anatomy

All terminal hair follicles are predetermined to grow long, thick hair.

M. G. Arnold
Otolaryngology – Department of Head and Neck Surgery, Naval Medical Center San Diego, San Diego, CA, USA

S. R. Owen (✉)
Facial Plastic and Reconstructive Surgery, Department of Otolaryngology, University of Iowa Hospitals and Clinics, Iowa City, IA, USA
e-mail: scott-owen@uiowa.edu

Hair Follicle Distribution at Birth [1]

- Total: 5,000,000
- Total head: 1,000,000
- Total scalp: 100,000*
- Density: 1 fu/mm^2
- *Scalp layers*: (pneumonic, SCALP) *s*kin, loose *c*onnective tissue, epicranial *a*poneurosis (galea), *l*oose areolar tissue, and *p*eriosteum
- *Vascular supply*: Preauricular region by supratrochlear, supraorbital, and superficial temporal arteries. Postauricular region by posterior auricular and occipital arteries
- *Venous outflow*: Anterior by supratrochlear and supraorbital veins which communicate with the ophthalmic vein and cavernous sinus and numerous emissary veins to intracranial venous sinuses
- *Hair follicle*: Consists of three segments: infundibulum, isthmus, and bulb. The Hair bulb, site of hair shaft formation, is comprised of papilla and surrounding epidermal cells at the base of each follicle. Melanocytes in hair bulb confer hair color.

Follicular unit Anatomic unit of hair, consisting of a group of 1–4 terminal hairs in the scalp, with or without several vellus hairs, a sebaceous gland and duct, neurovascular plexus, and arrector pili muscle

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_23

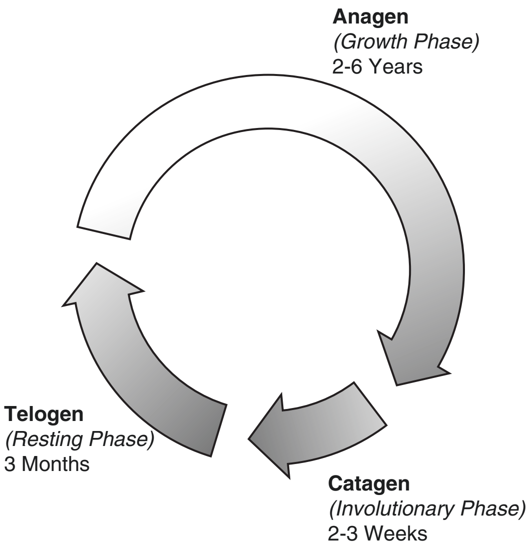


Fig. 23.1 Cyclical follicular unit extraction (FUE) hair growth cycle

Hair Growth Cycle (Fig. 23.1)

Anagen Active phase, lasts 3–4 years, 90% of hairs in this phase

Catagen Involution phase, lasts 2–3 weeks

Telogen Resting phase, lasts 3 months, 10% of hairs in this phase

- Scalp hair grows ~1 mm every 3 days.
- Each day, up to 100 hairs in telogen are shed, and the same number enters anagen.
- The duration of anagen determines the length of the hair.
- The volume of the hair bulb determines the diameter.

Etiology of Hair Loss

Affects 65–85% of men and 30–40% of women, progressing with age [2].

Androgenic alopecia (AGA) most common cause of hair loss, also known as male or female pattern hair loss (MPA, FPHL). Multiple pattern subtypes are described, mediated by scalp follicles with various genetic potentials for androgen inhibition. Pattern hair loss is probably multifactorial and may be inherited as an autosomal dominant trait with variable penetrance.

- *Histopathophysiology*: AGA is characterized by a decrease in the number of terminal hairs and increase in the number of miniaturized vellus hairs. *Miniaturized* hairs of various lengths and diameters are the hallmark of AGA, a result of binding of dihydrotestosterone to androgen receptors in susceptible hair follicles. The hormone-receptor complex then activates the genes responsible for gradual transformation of large terminal follicles to *miniaturized* follicles. Affected hairs cycle more quickly due to the decreased length of *anagen phase*.
- Mediated by the peripheral conversion of *testosterone* to *dihydrotestosterone* by organs containing *5-alpha-reductase*, *type II* (hair follicles, adrenal glands, testes, and prostate), resulting in the gradual conversion of terminal hairs to vellus hairs.
- Young men and women with AGA have higher levels of 5-α-reductase, more androgen receptors, and lower levels of cytochrome P-450 aromatase which converts testosterone to estradiol in hair follicles in the frontal and occipital scalp regions. Various clinical patterns of AGA may reflect quantitative differences in the levels of 5-α-reductase, the number of androgen receptors, and the levels of aromatase in specific regions of the scalp at various ages.
- Incidence [3–5]:
  - Men
    18–29 years: 16%
    30–39 years: 30%
    40–49 years: 50%
    70–79 years: 80%
  - Women
    70 years: 40%

Alopecia areata A nonscarring alopecia, autoimmune-like disorder, which results in patchy areas of hair loss, usually resolving with time. *Exclamation point hairs* which are narrower along the length of the strand closer to the base produce a characteristic *exclamation point* appearance. Patients may report that areas of hair loss may be painful or tingle. Treatment includes observation as the problem often spontaneously regresses. Consider injection of corticosteroids (non-FDA approved).

Telogen effluvium A form of nonscarring alopecia characterized by diffuse synchronized entry into telogen. Presents as diffuse thinning, generally lagging 3–4 months behind an inciting physiologic stressor such as sudden weight loss, pregnancy, iron or protein deficiency, hypothyroidism, general anesthesia, hormonal fluctuation, or chronic illness. Generally, recovery is spontaneous and occurs within 6 months with correction of insult. Treatment usually is limited to reassurance. Any reversible cause of hair shedding should be corrected. Although topical minoxidil is not proven to promote recovery of the hair in telogen effluvium, it has a theoretical benefit and is well tolerated.

Anagen effluvium A reversible condition of hair loss as a result of exposure to an offending agent such as antineoplastic chemotherapeutic agents, radiation, or toxic chemicals. Onset is within 2–6 weeks of exposure and should be reversible within a few months after cessation of the agent.

Scarring alopecias Inflammation resulting in destruction of stem cells and associated sebaceous gland. Follicle is replaced with scar tissue resulting in permanent hair loss. Ultimately, punch biopsy with histologic evaluation is the best method to confirm the presence of a fibrosing/scarring process with loss of hair follicles. Scarring alopecias include *lichen planopilaris*, *central centrifugal cicatricial alopecia*, *pseudopelade* (*Brocq*), *tufted folliculitis*, and systemic processes such as *scleroderma* and *discoid lupus erythematosus*. Surgical correction of alopecia should not be performed if the patient has any active scalp disease or inflammation, and test grafts should be considered to prevent graft loss from an active scarring process.

Triangular alopecia Rare, usually appears in childhood as a focal patch of loss that may be complete or leave fine vellus hairs behind. It appears as a triangular patch of vellus hairs in a frontotemporal distribution, pointing toward the vertex. Affected individuals are typically healthy. Hair transplantation is a successful treatment option.

Behavioral Causes of Alopecia

- *Traction alopecia*: Alopecia from mechanical stress on hair. More common in African Americans or patients with tightly braided hairstyles. Can also be seen with very long hair with weight stress on follicle. Slow gradual process that develops over a period of years. *Fringe sign*: A fringe of preserved hair along the marginal hairline on physical exam. Once stable, can consider hair transplant options.
- *Trichotillomania*: An obsessive-compulsive disorder characterized by incessant hair pulling, which can result in patches of baldness. Patchy alopecia with broken hairs of different lengths. More common in women, usually begins in adolescence, and can involve all hair-bearing areas, not just the scalp. Once behavior is controlled, hair transplantation can restore permanent hair growth.

Drugs Medications associated with hair loss include warfarin, isotretinoin, fibrates (gemfibrozil), antidepressants such as SSRIs (sertraline, Prozac) and TCA (amitriptyline), male androgenic hormones, steroids, and anti-inflammatory medications (naproxen, indomethacin, and methotrexate).

Infectious causes Dermatophytes, ringworm (tinea capitis), and syphilis can appear as scattered "moth-eaten" alopecia patches.

Inflammatory causes Seborrheic dermatitis, psoriasis, and pityriasis amiantacea.

Other systemic causes Thyroid disorders, radiation, autoimmune disorders, granulomatous disorders, developmental/hereditary causes, and poisons such as arsenic, lead, thallium, and boric acid.

Classification Systems

Norwood Classification for Hair Loss in Men (Fig. 23.2) [6]

- *Class I*: adolescent or juvenile hairline. Hairline generally rests on the upper brow crease. Not considered balding.
- *Class II*: Progression to adult or mature hairline that sits 1.5 cm above the upper brow crease, with some temporal recession. Also does not represent balding.
- *Class III*: Earliest stage of male hair loss, characterized by a deepening temporal recession.
- *Class III Vertex*: Early hair loss in the crown (vertex).
- *Class IV* is characterized by further frontal hair loss and enlargement of vertex, but there is still a solid band of hair across the top separating the front and vertex.
- *Class V*: The bald areas in the front and crown continue to enlarge, and the bridge of the hair separating the two areas begins to break down.
- *Class VI* occurs when the connecting bridge of hair disappears leaving a single large bald area on the front and top of the scalp. The hair on the sides of the scalp remains relatively high.
- *Class VII* patients have extensive hair loss with only a wreath of hair remaining in the back and sides of the scalp.

Ludwig Classification of Female Pattern Baldness (Fig. 23.3) [7]

- Stage I: Perceptible hair loss with thinning on the crown, but preservation of the anterior hairline
- Stage II: Significant widening of the midline part and noticeably decreased volume
- Stage III: Diffuse thinning

Evaluation

A through *history and physical* is essential Ludwig Classification: prior to intervention to determine the etiology of the patient's alopecia. History should be focused on pattern of hair loss, timeline of progression, and prior management. A family history should include both male and female pattern hair loss on both sides. As with all cosmetic procedures, the patients' goals and motivations should be addressed, and the patient should be counseled regarding realistic expectations. This is particularly relevant for younger patients who may wish to restore a youthful hairline that will look unnatural in middle age. Goals should be to reproduce an appropriate, mature hairline [1].

Physical exam is important. Density should be evaluated both at recipient and donor sites. Hair and skin color, as well as texture, are important factors in transplantation. Skin laxity should be evaluated, as, in rare circumstances, a tissue expander may need to be employed.

- *Hair pull test*: 50–100 hairs pulled at once; >5 telogen hairs are considered abnormal.
- *Hair loss count*: patient wears a shower cap and counts shed hairs, >100/day abnormal.
- *Hair window*: used to evaluate growth, small patch shaved and hair growth measured. <0.4 mm/day is abnormal.

Laboratory Testing

- Not routinely used in men with MPA.
- May consider TFTs and serum iron with ferritin if history warrants.
- Female patients should be evaluated for hyperandrogenism, with serum androgen levels and evaluation for polycystic ovary syndrome, androgen-producing tumors, or congenital adrenal hyperplasia.
  - Lower threshold for laboratory evaluations of TFTs, zinc levels, prolactin, antinuclear antibodies, and syphilis titers
- Tissue biopsy is considered in uncertain diagnosis and is needed to diagnose scarring alopecia, chronic telogen effluvium, and diffuse alopecia areata.

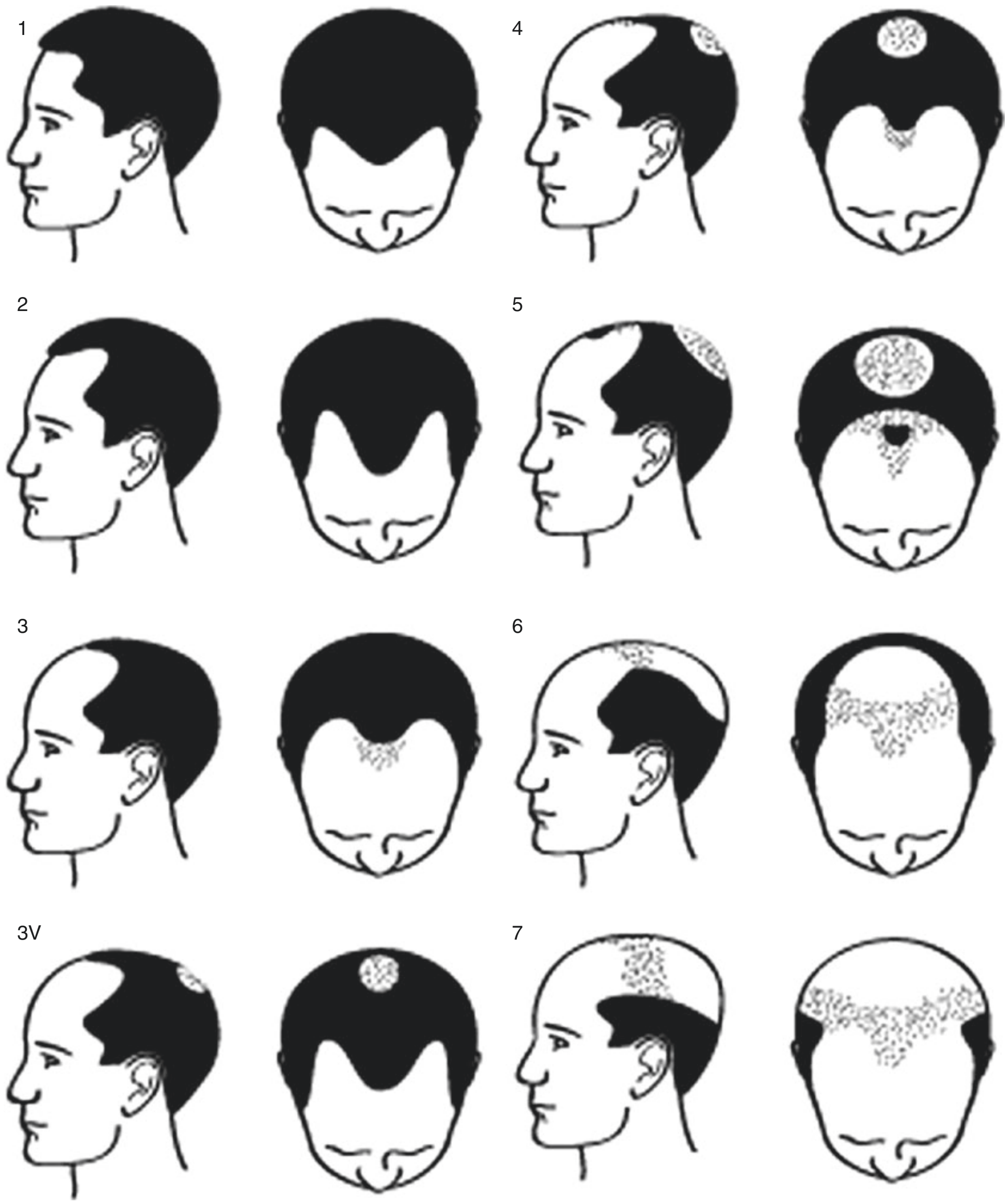


Fig. 23.2 Norwood classification of male pattern alopecia

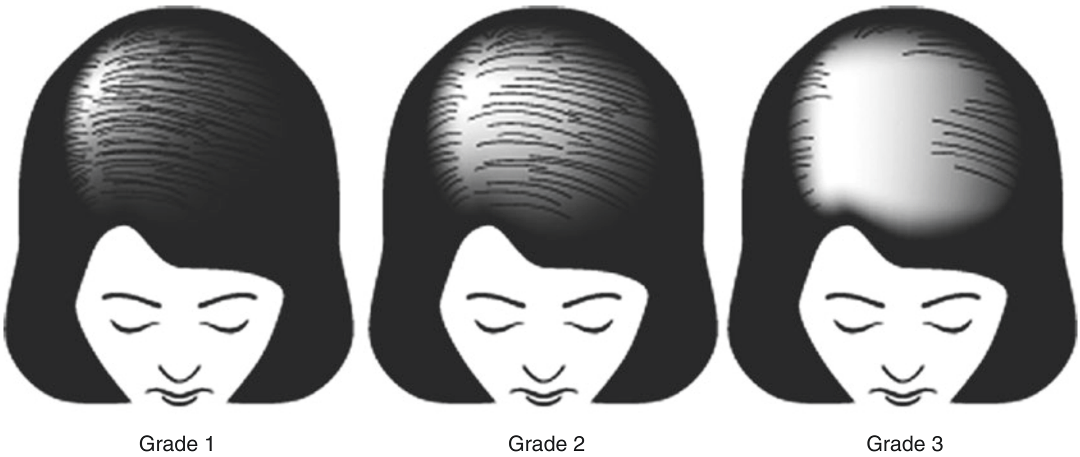


Fig. 23.3 Ludwig classification of hair loss in women

Medical Treatment

Minoxidil (Rogaine [McNeil-PCC])

- First drug approved by the FDA for AGA.
- A piperidinopyrimidine derivative functions as a vasodilator (calcium channel blocker) initially used for cardiac purposes. Side effect of hypertrichosis noted in 70% patients with oral minoxidil. Exact mechanism is unknown.
- Effects include increasing follicle size and inducing anagen, lengthening this phase and shortening latent period.
- Currently topical 2–5% minoxidil shows hair growth with continuous use in 4–6 months. Discontinuation leads to loss of gained hair over 3–4 months.
- Used to decrease posttransplant shedding period after surgery, shortening time for hair grafts to regrow.
- The American Hair Loss Association recommends it as a second drug to be added after finasteride or for patients who fail finasteride treatment. Although not considered a first-line therapy, it does provide effective treatment for a small percentage of its users.
- In addition to its effectiveness in AGA, minoxidil may promote hair growth in patients with alopecia areata, congenital hypotrichosis, and loose anagen syndrome.

Finasteride (Propecia [Merck & Co., Inc.])

- Type II 5-α-reductase inhibitor, competitive antagonists of testosterone.
- Finasteride rapidly lowers serum and scalp DHT levels by >60%. It has no affinity for the androgen receptor and therefore does not interfere with the actions of testosterone.
- Forty-eight percent experience regrowth of hair, and 42% experienced no further hair loss [8]. Increases the hair growth rate, thickness, and, to a limited degree, hair count when compared to placebo.
- *Not effective in women without evidence of hyperandrogenism*, *contraindicated in child-bearing years*.
- Side effects are reported in 3.8% of men taking finasteride (compared to 2.1% on placebo). These include decreased libido, erectile dysfunction, ejaculation disorders, orgasm disorders, and decrease in sperm quality. These typically resolve within 3 months of discontinuation of medication.

Medical Treatment for Hair Loss in Women

- *Minoxidil* appears to be more effective in female patients than in men. It is recommended for women to only use the 2% formulation and not the 5%.

- Spironolactone (Aldactone) is an antiandrogen that decreases production of androgens in the adrenal glands and ovaries.
- *Cyproterone acetate* – antiandrogen and progestogen (producing progesterone-like effects) used to treat hirsutism and precocious puberty. It can be used to treat AGA in women.
- *Cimetidine* (*Tagamet*) also has a weak antiandrogenic effect. High doses are needed to achieve hair-raising results; therefore it is not recommended for men due to possible feminizing effects and sexual side effect.
- *Low androgen index oral contraceptives* can be used to treat hair loss (high androgen index oral contraceptives can trigger hair loss).
- *Topical ketoconazole*, a topical antifungal agent, has antiandrogenic effects and can be used to help treat hair loss.

Surgical Treatment

Hair Transplantation

Principles

- Hair from the occipital and parietal scalp is more resistant to androgen effects than frontal and vertex follicular units.
- Hair transplants maintain the characteristics of the donor site.

Evolution of the field from hair plug grafts to follicular units. Initially 15–20 hair plug grafts, to half and quarter grafts (8–10 and 3–5 hairs, respectively), to where in the late 1980s, minigrafts (3–5 hairs) and micrografts (1–2 hairs) came to the forefront.

Considerations

1. Classification of baldness: Norwood classification.
2. Classification of hair quality: density, texture, curl, and color.
3. Color similarity between the hair and skin: Generally, similar hair and skin color leads to better result.
4. Future hair loss expectation: family history, can be maternal or paternal, warn pt of uncertainty.
5. Age of patient: <20 generally discouraged, too difficult to predict future alopecia.
6. Motivation, expectations, and desires of patient.
7. Contraindications include active autoimmune process, poor health, or lidocaine allergy.

Hairline Placement

General guide for anterior hairline: 7.5–9.5 cm above the mid-glabella. Frontotemporal angle is most critical to design. Hairline should gently slope backward as it approaches the frontotemporal recession, which is located along a vertical line above the lateral canthus. Hairline should exist in an irregular fashion, scattered up to 1 cm in front of the area where the eye actually perceives the hairline.

Donor Site Harvest

Grafts can be harvested by excising a strip of the scalp (follicular unit grafting) or by individual punch extraction (follicular unit extraction).

Determining the number of grafts needed Each square centimeter yields approximately 100 follicular units (80–120 per sq. cm) depending on the donor density. If desiring 1000 grafts/session, can harvest a donor strip 1.5 × 6.5 cm donor strip (or 1 × 10 cm if tight scalp).

Goal is redistribution of hair follicles, with 50% of original density restoration to areas of loss.

Follicular Unit Grafting (FUG)

Donor strip harvesting Donor strip marked out just above the external occipital protuberance. Donor strip hairs are trimmed to 4 mm. Incision should be angled to parallel hairs to *minimize transection* of hair shafts. Strip is excised in a subcutaneous plane just below the hair follicles. For best donor site scar results and depending on scalp laxity and/or prior hair transplant surgery, the donor strip can typically be 8–15 mm in width. Donor site can be closed with a *trichophytic* closure in which the inferior edge of the donor site defect is de-epithelialized and overlapped with the upper lip of the superior edge, thus permitting hair growth through the scar.

Grafts must then be prepared under microscopic visualization for transplantation. The single strip is subdivided into slivers, each 1–2 follicular units wide, from which the individual follicular units can then be dissected. Because of the small size, these grafts can be placed into tiny recipient sites. Grafts are kept in *chilled saline* until time of implantation; desiccation may result in graft loss.

Follicular Unit Extraction

Follicular unit extraction involves harvest of individual follicular units, using a motorized punch for extraction. This allows diffuse graft harvest and trades a linear strip harvest scar with small punctate scars at harvest sites. These are theoretically less detectable and allow patients to have less postoperative discomfort and a shorter recovery time. The trade-off is a more time-consuming and technically demanding harvest.

Donor site hair needs to be shaved down to approx. 2 mm in length for efficient graft harvesting. To obtain the same number of follicular units with FUE (compared to FUG), a much larger donor area is required. Punch must be angled along the axis of the hair shafts in the follicular unit to prevent transection of the graft.

Transection rates initially reported at 30% are now down as low as 2–5.5%. Powered punch FUE devices with a 1.0 mm punch attached to a motor with a rotation capability of 700–1500 rotations per minute statistically lowered the transection rate ($P = 0.003$) and harvesting time ($P = 0.001$) when compared to manual harvest, thought to prevent greater torsional forces that generate more distortion of the follicular unit axis.

Indications for FUE include inadequate laxity for strip harvest site closure, minimal hair loss, history of poor scar formation, and patients requiring limited recovery time [9].

Automated Harvest

Labor-intensive harvest and advances in technology have spawned a market for automated systems to improve harvest time/accuracy. Several robotic options have emerged, such as the ARTAS (Restoration Robotics System, Inc., San Jose, CA). These systems use computer guidance and a single operator to allow for rapid harvest of follicular units. Harvest rate is listed as 400–600 grafts per hour. The literature reports a low graft transection rate as well, at approximately 1.59% [10].

Robotic harvest systems require extensive staff training, an individual “pilot” to operate the machine, and a willing patient. Patients must be able to sit still for the 90–120-minute graft harvest. While significantly shorter than manual FUE, strip harvest remains faster. Cost is the limiting factor, requiring an investment in the realm of $240,000, or adding approximately $1 per graft harvest [10].

Graft Insertion

Graft transplantation involves meticulous planning and evaluation of a patient’s natural hair whorls and angle patterns. Grafts will continue to grow at the angle in which they are placed, so recipient sites are made mimicking the native hair direction. Recipient sites are created using specialized blades or needles that create a precise depth and width to the incision. Follicular units are then placed meticulously with fine forceps to limit trauma to the graft.

Generally, densities between 20 and 30 follicular units per sq. cm produce cosmetically favorable results. Diminished graft survival has been shown when placed at densities >30 follicular units per sq. cm, and transplantation may be staged if increased density is desired.

Rapid transplantation is essential for maximization of graft survival. General rule of thumb: 92% of grafts survive if transplanted within 6 hours, decreasing by 1% every subsequent hour [11]. Care must be taken to prevent desiccation of grafts or traumatic insertion, both of which decrease graft survival.

Graft extrusion may occur from placement of grafts in too high of a density or from too large a recipient incision.

Ingrown hairs may result from too deep of graft placement. Most surgeons advocate 1–10% of graft elevated above the donor scalp to prevent ingrown hairs. Intervention is unroofing and extracting hair from the scalp.

Special Considerations

- Eyebrows: Best results attained when recipient sites are oriented in a horizontal/lateral direction. Angle of growth away from the skin should be minimal, so hairs grow flat along the surface rather than projecting out from the brow.
- Neolashes: Aesthetic and functional goal is to have the neolashes grow away from the leading edge of the eyelid to prevent corneal irritation. Patients often need a curler to direct the hairs in the proper direction of growth.

Postoperative Expectations of FUG and FUE

All hair will temporarily enter *telogen phase* and fall out. New hair will generate from the follicle in 3–4 months. This interval can be shortened by as much as 6 weeks by using minoxidil starting the tenth day after the procedure. Patients are advised that it can take as long as 12 months before the final results are apparent. Sequential stages of transplantation can be done in 3–6 months.

Scalp Reductions

Discrete sites of alopecia may be treated with excision and rotation of surrounding hair-bearing scalp. Ideal candidates are patients with a balding vertex or an area of traumatic or iatrogenic patch of alopecia.

In this technique, the scalp is excised to galea, and wide undermining of the surrounding tissue is performed. Excisions are largely performed in the axis of the lines of minimum tension (*Langer's lines*), and many patterns are described. Scalp elasticity should be assessed preoperatively to ensure adequate closure, but even so most patients require serial excisions. Progression of alopecia is sited as a major drawback to this technique and limits its employment by most surgeons, though it is a useful technique in the properly selected patient.

Tissue expansion involves the surgical placement under the galea of one or more balloon expanders with progressive stretching of surrounding hair-bearing skin over a period of several months. Though rarely used for cosmetic scalp reduction surgery, tissue expansion is still a valuable technique when dealing with areas of scarring, such as from burns or prior surgery, or for certain hairline advancement procedures in women.

Tissue extension involves the surgical placement of a playing card-sized elastic device with a row of hooks on each end. The hooks are seated into the galea along the opposite sides of the scalp reduction incision. Over a span of 30 days, the contractive forces of the extender further loosen the surrounding scalp (inducing mechanical creep, rather than the expansion-induced biologic creep), permitting the removal of a greater amount of bald tissue than what would normally be expected. Subsequently, a second scalp reduction is carried out, during which the extender is removed. Advantages include achieving maximal reduction and improved wound closure tension.

Galea fixation in alopecia reduction refers to the suturing of the galea to the central pericranium with the effect of drawing the wound margins into close approximation so the scalp can be closed without tension.

Once excised, the remaining bald area after one or multiple scalp reductions can be filled in with transplants.

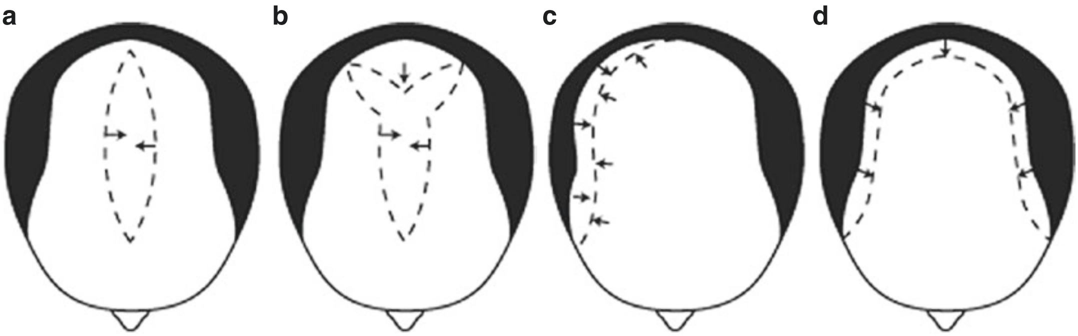


Fig. 23.4 The four basic types of alopecia reduction: (**a**) midline reduction, (**b**) Mercedes reduction, (**c**) paramedian reduction, and (**d**) circumferential reduction

The four basic types of alopecia reduction are (a) midline reduction, (b) Mercedes reduction, (c) paramedian reduction, and (d) circumferential reduction (Fig. 23.4).

Scalp Flaps

Previously mainstay treatment. Flap procedures are rarely performed today with the ability of modern hair-grafting techniques to create such a natural-appearing result.

Flap procedures offer the advantage of rapid reconstruction; no disruption of blood supply to follicles which prevents postoperative telogen phase. Patients do not need to wait for graft growth or necessarily trim hair preoperatively. Results are not as natural as with other techniques, however. The ideal candidate is generally a candidate with alopecia restricted to the frontal area, elderly with a stable hairline, and those desiring immediate results without multiple procedures. May also be a useful technique for large scalp defects secondary to cancer or trauma.

Juri flap axial flap following the posterior branch of the superficial temporal artery proximally with random pattern at most distal point. Flap can be 3–4 cm wide (up to 5–6 cm if expanded first) and 25 cm long, allowing for the entire frontal hairline to be bridged with one flap. It can recreate an entire frontal hairline. Although the direction of hair growth is primarily posterior (instead of the natural anterior direction), the density achieved is unsurpassed; it has been estimated that a single flap provides over 10,000 hairs. For further coverage behind the flap, a second flap from the contralateral side and/or hair grafting can be performed. Base of flap starts 4 cm above the helical crus and spans a distance of 4 cm at an angle of 30°–40° from the horizontal. Mild distal flap necrosis may occur, but extensive flap loss is rare if the flap is raised in a twice-delayed staged fashion (raise distal flap and replace first stage, 1 week later raise proximal flap and replace, and then raise the entire flap and rotate 1 week later) (Fig. 23.5).

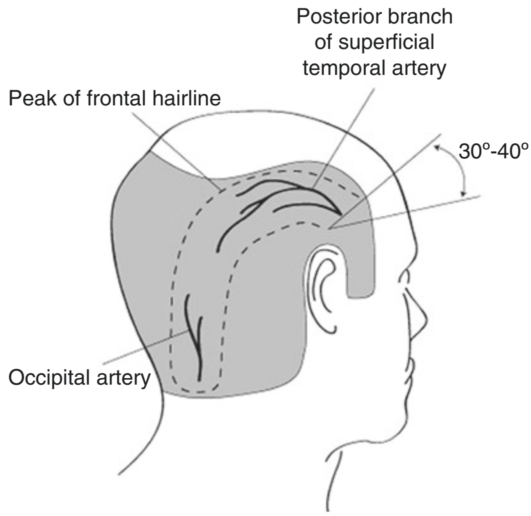


Fig. 23.5 Juri flap design

Dardou flap random flap, blood supply less predictable with potential of early telogen or tip necrosis.

Disadvantages Hairlines are abrupt, donor and recipient site scarring often requires the hair to be worn long, and it is reasonably limited to patients who will not progress to a class 4 or greater hair loss pattern as advancement of hair loss can lead to visible donor site scars or areas of baldness behind the flap.

Commonly Cited Complications of Surgical Hair Restoration

1. Large graft size: See visible grafts with unnatural tufts of hair at graft site, "doll hair." Frequently seen in the past with large 4–5 mm plugs, much improved with follicular unit grafting.
2. Poorly designed hairline: Frequently visible if designed without adequate temporal contour or too low of an anterior hairline. Another pitfall in younger patients is creation of a youthful hairline that will look unnatural as the patient ages.
3. Poorly designed graft direction: surgeons should recreate natural parts and whorls when placing grafts.
4. Unrealistic coverage: *remember the first region to bald is where you should be most hesitant to transplant*.
5. Scarring in recipient area: donor incisions should be no larger than 1.7 mm (size of 18 G needle).
6. Ridging: problem with larger grafts where the transplanted area results in a ridge demarcating it from the surrounding bald scalp.
7. Linear donor scarring: Seen if strip harvest is poorly designed; consider trichophytic, tension-free closure.
8. Low or depleted donor supply: donor hair limited by low density, fine hair caliber, poor scalp mobility, and scarring.
9. "Pitting": depression of the skin around a group of hairs from graft placement. To minimize pit formation, a small cuff of the skin from the graft is left sticking above the surface of the surrounding scalp.
10. "Slot deformity" from scalp reductions: divergent directions of hair growth creating an unnatural appearance from a midline scalp reduction.
11. Bleeding, edema, infection, poor hair growth, and AV fistula in the occipital region.

Alternative Treatments

Many alternatives exist to medical or surgical intervention to alopecia. Patients may prefer camouflaging techniques during hair styling, wigs, or hair extensions. Commercial products exist that may darken the scalp or increase the thickness of existing hair making areas of alopecia less noticeable. Multiple light and herbal therapies have been proposed but lack sufficient conclusive efficacy data [12].

Future Directions

The field of hair restoration remains an evolving field with high demand for quick, inexpensive, and effective treatments that minimize patient recovery time and provide lasting, meaningful results.

The use of platelet-rich plasma (PRP) injections has been a controversial treatment for alopecia. Proponents cite an increased hair density and follicle thickness, but most studies fail to show statistical significance. This remains a controversial technique in hair restoration [13].

Scalp "threading" with insertion of grids of absorbable polydioxanone (PDO) filaments has been reported to improve hair density in androgenic alopecia. Thread is inserted intradermally under sterile conditions along bald areas of the scalp. Mechanism is speculated to mimic the effects of microneedling, with increased expression of hair growth genes, upregulation of PDGF, and activation of stem cells around the implants [12].

The generation of hair follicles from stem cells, particularly for burn patients, remains a promising frontier, but without current clinical application [14].

Questions

1. Which patient is likely to have the best result with hair transplantation?
   (a) Black hair on Fitzpatrick type 1
   (b) Dark brown hair on Fitz type 1
   (c) Red head on Fitz type 1
   (d) Gray hair on Fitz type 1
2. In a normal scalp, most hair follicles are in which stage of the hair cycle?
   (a) Anagen
   (b) Catagen
   (c) Telogen
3. Hair loss becomes visible when the amount of hair reduction reaches _%.
   (a) 30%
   (b) 40%
   (c) 50%
   (d) 60%
4. Which of the following is true about the Juri flap?
   (a) It is an axial flap based on the superficial temporal artery.
   (b) It is a random-based flap.
   (c) It is good for balding in the region of the vertex.
   (d) The flap is limited to a length of 10 cm.
5. A 40-year-old female comes in requesting hair transplantation. She is concerned about the thinning of her hair on the top of her head. Examination shows areas of significant thinning with miniaturization of hairs. She is otherwise healthy and not taking any medications. She is a good candidate for hair transplantation.
   (a) True
   (b) False
6. A 40-year-old female comes in requesting hair transplantation. She is concerned about the thinning of her hair on the top of her head. Examination shows patches of hair loss with exclamation point appearing hairs. She is otherwise healthy and not taking any medications. She is a good candidate for hair transplantation.
   (a) True
   (b) False
7. Based on the Norwood classification of hair loss in men, which stage represents the earliest stage of balding?
   (a) I
   (b) II
   (c) III
   (d) IV
   (e) V
8. Slot deformity is a complication of which type of hair restoration surgery?
   (a) Scalp flap surgery.
   (b) Scalp reduction surgery
   (c) Follicular unit grafting
   (d) Follicular unit extraction

Answers

1. (d) Like curliness, light-colored hair on white skin and/or black hair on black skin almost always ensure that a good result will ensue. This correlation exists because light scalp does not project through light hair and dark scalp does not likewise project through dark hair. Conversely, a light scalp projects significantly through dark hair, making the patient appear balder than he or she may actually be.
2. (a) 90–95% of hairs are growing (anagen phase), less than 1% are in the involution (catagen) phase, and 5–10% are in the resting (telogen) phase.
3. (c) The eye cannot perceive hair loss until it exceeds 50%. Because of this redundancy, there is no reason to restore more than 50% of the hair, especially in view of the fact that the balding individual has less total hair volume.
4. (a) It is an axial-based flap based on the posterior branch of the superficial temporal artery used to reconstruct the anterior hairline and can be 25 cm in length.

5. (a) True. Miniaturization is characteristic of androgenic hair loss. Would also consider trial of minoxidil.
6. (b) False. This is alopecia areata which is autoimmune and typically self-limited. Treatment includes observation with reassurance, possibly steroid injections. Biopsy could be performed if unsure of the cause.
7. (c) Class III represents the earliest stage of balding in men.
8. (b) "Slot deformity" from scalp reductions is the unnatural appearance of hair from a midline scalp reduction characterized by divergent directions of hair growth where the hair was brought together.

References

1. Rousso DE, Stough DB, Rutherford KD. Hair restoration. In: Papel ID, editor. Facial plastic and reconstructive surgery. New York: Thieme Publishers; 2016. p. 334–50.
2. Norwood O. Alopecia. Classification and incidence. In: Stough DB, Haber RS, editor. Hair replacement. Mosby: St. Louis; 1996. p. 13–20.
3. Stough D, Stenn K, Haber R, Parsley WM, Vogel JE, Whiting DA, Washenik K. Psychological effect, pathophysiology, and management of androgenetic alopecia in men. Mayo Clin Proc. 2005;80:1316–22.
4. Rhodes T, Girman CJ, Savin RC, et al. Prevalence of male pattern hair loss in 18-49 year old men. Dermatol Surg. 1998;24(12):1330–2.
5. Birch MP, Messenger JF, Messenger AG. Hair density, hair diameter and the prevalence of female pattern hair loss. Br J Dermatol. 2001;144(2):297–304.
6. Norwood OT. Male pattern baldness: classification and incidence. South Med J. 1975;11(68):1359–65.
7. Ludwig E. Classification of the types of androgenic alopecia (common baldness) occurring in the female sex. Br J Dermatol. 1977;3(97):247–54.
8. Merk. How propecia works; clinical study. Available at: http://www.propecia.com/finaseride/propecia/consumer/about_propecia/propecia.jsp?WT.svl=2. Accessed 21 May 2017.
9. Dua A, Dua K. Follicular unit extraction hair transplantation. J Cutan Aesthet Surg. 2010;2(3):76–81.
10. Avram MR, Watkins SA. Robot follicular unit extraction in hair transplantation. Dermatol Surg. 2014;40:1319–27.
11. Limmer R. Micrograft survival. In: HR Stough D, editor. Hair replacement. Mosby: St Louis; 1996. p. 147–9.
12. Bharti J, Sidharth S, Patil P, Dhurat R. Scalp threading as a novel, effective and safe method of hair restoration. J Eur Acad Dermatol Venereol. 2017 Nov;31(11):e492-e493.
13. Giordano S, Romeo M, Lankinen P. Platelet-rich plasma for androgenetic alopecia: does it work? Evidence from meta-analysis. J Cosmet Dermatol. 2017;16:374–81.
14. Mahjour SB, Ghaffarpasand F, Wang H. Hair follicle regeneration in skin grafts: current concepts and future perspectives. Tissue Eng Part B Rev. 2012;1(18):15–23.

Additional Resources

Bernstein RM, Rassman WR, Rashid N, Shiell RC. The art of repair in surgical hair restoration part I: basic repair strategies. Dermatol Surg. 2002;28:783–94.

Bernstein RM, et al. Standardizing the classification and description of follicular unit transplantation and mini-micrografting techniques. Dermatol Surg. 1998;24:957–63.

Bouhanna P. Multifactorial classification of male and female androgenetic alopecia. Dermatol Surg. 2000;26:555–61.

Brandy DA. An evaluation system to enhance patient selection for alopecia reducing surgery. Dermatol Surg. 2002;28:808–16.

Cash TF. The psychosocial consequences of androgenetic alopecia: a review of the research literature. Br J Dermatol. 1999;141:398–405.

Epstein JS. The treatment of female pattern hair loss and other applications of surgical hair restoration in women. Facial Plast Surg Clin North Am. 2004;12:241–7.

Epstein JS. Evolution of techniques in hair transplantation: a 12 year perspective. Facial Plast Surg. 2007;23(1):51–9.

Epstein SS. Follicular-unit hair grafting. Arch Facial Plast Surg. 2003;5:439–44.

Fiedler VC, Galan A. Androgenetic alopecia. Facial Plast Surg Clin North Am. 1994;2(2):101–9.

Gandelman M, Epstein JS. Hair transplantation to the eyebrow, eyelashes, and other parts of the body. Facial Plast Surg Clin North Am. 2004;12:253–61.

Irwig MS, Kolukula S. Persistent sexual side effects of finasteride for male pattern hair loss. J Sex Med. 2011;8(6):1747–53.

Konior RJ, Kridel RWH. Tissue expansion in scalp surgery. Facial Plast Surg Clin North Am. 1994;2(2):203–17.

Lam SM, Hempstead BR, Williams EF. A philosophy and strategy for surgical hair restoration: a 10-year experience. Dermatol Surg. 2002;28(11):1035–42. Discussion, 1042. Review.

Lee TS, Minton TJ. An update on hair restoration therapy. Curr Opin Otolaryngol Head Neck Surg. 2009;17:287–94.

Orentreich N, Durr NP. Biology of scalp hair growth. Clin Plast Surg. 1982;9(2):197–205.

Orentreich N. Punch grafts. In: Rees TD, editor. Aesthetic plastic surgery. 30th ed. Philadelphia: W. B. Saunders; 1980. p. 865–74.

Price VH. Treatment of hair loss. N Engl J Med. 1999;341:964–73.

Saraswat A. Minoxidil vs Finasteride in the treatment of men with androgenic alopecia. Arch Dermatol. 2007;139:1219–21.

Seery GE. Galea fixation in alopecia reduction surgery. Dermatol Surg. 2001;27:931–6.

Seery GE. Hair transplantation: management of donor area. Dermatol Surg. 2002a;28:136–42.

Seery GE. Surgical anatomy of the scalp. Dermatol Surg. 2002b;28:581–7.

Shapiro R. Principles and techniques used to create a natural hairline in surgical hair restoration. Facial Plast Surg Clin North Am. 2004;12(2):201–17. Review

Swerdloff J, Kabaker S. The state of the art. Donor site harvest, graft yield estimation, and recipient site preparation for follicular-unit hair transplantation. Arch Facial Plast Surg. 1999;1:49–52.

Unger MG. Scalp reduction. Facial Plast Surg Clin North Am. 1994;2(2):163–81.

Whiting DA. Possible mechanisms of miniaturization during androgenetic alopecia or pattern hair loss. J Am Acad Dermatol. 2001;45(3 Suppl):S81–6.

24 Lasers in Aesthetic Surgery

Jennifer Goodrich

Science of Lasers

Laser is an acronym for "Light Amplification by the Stimulated Emission of Radiation." Light from the laser is within the electromagnetic spectrum of energy (Fig. 24.1) and is usually contained within visible light spectrum to infrared wavelengths. Laser light differs from natural or other artificial light in three main ways:

1. Monochromatic: light emitted from the laser is of the same wavelength, whereas light from sun or artificial light is a culmination of several different colors/wavelengths. If you passed sunlight through a prism, you would have separation of various colors, whereas light from a laser passed through a prism would not change.

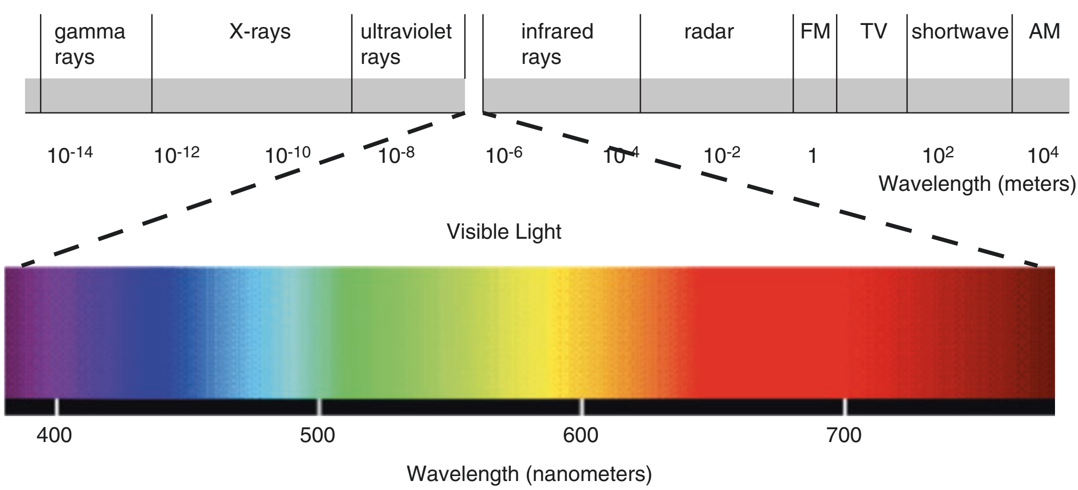


Fig. 24.1 Electromagnetic spectrum

J. Goodrich (✉)
Walter Reed National Military Medical Center, Bethesda, MD, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_24

2. Coherence: all waves are in phase with each other, meaning the peaks and troughs of each wave are in alignment.
3. Collimation: light from laser has a narrow diameter without divergence, whereas light from sun (or flashlight) will diffuse/spread out. This allows the laser beam to maintain its energy throughout the duration of its travel, regardless of the distance it must travel.

Essentially, a laser is composed of a laser chamber containing the lasing medium, two mirrors and an external pump or excitation source. The lasing medium, for which most lasers are named (i.e., PDL, CO2, etc.) is the source of electrons needed to yield a laser beam. The lasing media can be either liquid (dye), solid (crystal or diode), or gas (CO2).

The external pump applies energy to the lasing medium, which causes the electrons within the medium to transition to an unstable, excited state. As these electrons return to a more stable state, they release energy in the form of photons. These photons traverse through the lasing chamber, being reflected between the two mirrors at either end. As these photons hit other atoms within the medium, they stimulate these atoms to undergo the same change (stimulated emission). When the surgeon is ready, firing the laser opens the lasing chamber and allows for escape of the laser beam.

Tissue Interaction

When laser light interfaces with the skin, it is either absorbed, reflected, scattered, or transmitted. All lasers have a specific target chromophore, based on the wavelength of the laser (see below). The main chromophores in skin are: hemoglobin, melanin, and water, with tattoo ink being an exogenous chromophore. When the target molecule absorbs the light from the laser, the energy from the laser is transferred to the target, which causes its destruction.

About 4–6% of the light from the laser beam is reflected off of the skin's surface. This is the key reason why appropriate eye protection should be worn by all personnel in the room. Reflection of the light can be minimized by keeping the handpiece at a 90° angle, or perpendicular to the skin.

Light can also be scattered, usually due to collagen fibers in the dermis. This will reduce the amount of light or energy that reaches the target molecule, which is sometimes deep to the skin (blood vessel, tattoo ink, hair follicle, etc.). The amount of scattering is inversely proportional to the wavelength of the laser, that is, the shorter the wavelength, the more likely it is to scatter, and the less the depth of penetration. Stated another way, due to increased scattering with shorter wavelengths, those lasers with shorter wavelengths cannot penetrate effectively deep into the skin and are used more for superficial lesions. Scattering defocuses the laser spot, which also leads to more collateral damage (damage to surrounding structures that are not the target molecule). Selecting the largest spot size possible on the laser will actually limit scattering and improve energy delivery and increase depth of penetration, all due to reduction in scattering.

Lastly, laser light can be transmitted through structures to reach deeper molecules. An example would be light from the laser being transmitted through the skin to reach its deeper target molecule, such as hemoglobin.

Variables

Selective photothermolysis is a key concept in understanding lasers, stating that laser energy can be absorbed by a defined target chromophore, which leads to that target's complete destruction without causing significant damage to surrounding tissues. Often times, the challenge with using lasers is that while melanin (the pigment in skin) may not be the target chromophore, it is absorbed, to a variable degree by many of the common lasers. This key concept will be discussed later.

There are certain variables that need to be controlled or addressed in order to ensure the appropriate chromophore is targeted for destruction:

1. Wavelength: the specific wavelength of the laser needs to be within the absorption maximum of the target chromophore, or at least lie within the absorption spectrum of that molecule (see Fig. 24.2).
2. Pulse Duration: (also called the pulse width of the laser beam) must be equal to or shorter than the thermal relaxation time (TRT) of the chromophore. The TRT is the time needed for the target chromophore to dissolve two-thirds of its peak temperature (Table 24.1). This time needed is directly related to the size of the chromophore, so larger molecules or larger blood vessels will need longer pulse durations.
3. Energy Density: the energy density (J/cm^2), or fluence, must be high enough to destroy the chromophore within the defined pulse duration. The spot size of the laser beam is inversely proportional to energy density, such that decreasing the spot size will increase the density by a factor of 2.

Table 24.1 Thermal Relaxation Time (TRT) of common target chromophores

Target	Size, um	TRT
Tattoo ink	0.5–4	10 ns
Melanosome	0.5–1	1 us
Erythrocyte	7	2 us
Blood vessel	50–200	1–20 ms
Hair follicle	200	10–100 ms

Epidermal Cooling

In many instances, the target chromophore is DEEP to the skin surface, so the laser light needs to be transmitted through the skin to its target without losing much energy and without damaging the epidermis. The skin needs to be cooled so it is not damaged by the transmission of laser energy through it. There are a variety of options for cooling (applying ice, cool air, cryogen spray, sapphire tip, etc.). Damage to the epidermis will result in blistering and potential for scarring and pigment changes.

Common Lasers

There are a variety of lasers commonly used for aesthetic purposes (see Table 24.2).

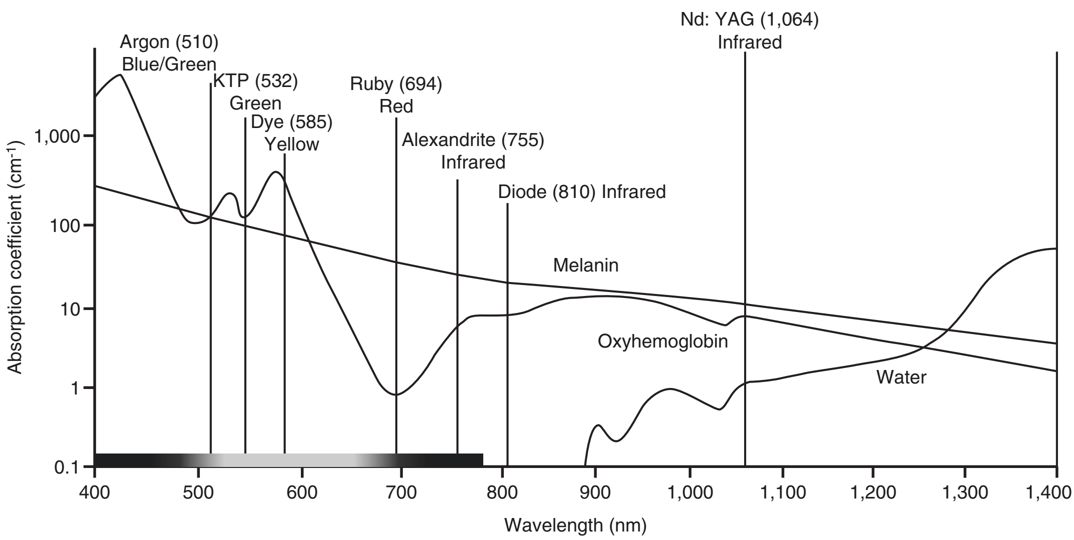


Fig. 24.2 Absorption spectra for various common chromophores

Table 24.2 Table of commonly used lasers in the clinical setting

Laser	Wavelength/Color	Target	Application
Argon	510/Blue	Melanin/tattoo ink	Tattoo ink
KTP	532/Green	Hemoglobin/melanin	Vascular lesions, tattoo ink
PDL	585–595/Yellow	Hemoglobin	Vascular lesions, photofacial, rosacea
Ruby	694/Red	Melanin	Brown spots, tattoo ink
Alexandrite	755	Melanin/Hemoglobin	Vascular lesions, tattoo ink
Er:YAG	2940	Water	Resurfacing
Nd:YAG	1064	Hemoglobin	Vascular lesions with deeper penetration
CO2	10,600	Water	Resurfacing

- PDL has become the workhorse for treatment of port wine stains (PWS) and hemangiomas.
- Alexandrite can be used for resistant PWS, not completely cleared with PDL.
- Nd:YAG is particularly useful in patients with dark skin because it is not well absorbed by melanin, thus limiting risk of hypopigmentation. It is useful for treatment of vascular lesions, tattoo removal, and hair removal in darker skinned (Fitzpatrick 4–6) patients. It also penetrates the skin deeper than PDL or Alexandrite and can be used to treat larger or deeper (4–6 mm below skin) blood vessels.
- Both Er:YAG and CO2 lasers are used for resurfacing (scar revision or facial rejuvenation). There is a more coagulative property to the CO2 laser. Head-to-head outcome comparisons favor CO2 laser; however, with multiple passes of Er:YAG, the results are equivocal with less side effects than CO2.
- Intense Pulsed Light (IPL): is not a true laser, but it can be utilized in similar clinical settings as a laser. IPL contains many wavelengths of light, and thus can have many different targets. It is usually utilized to target vascular lesions and photo aging.

Preoperative Evaluation

It is important to understand the goals the patient wishes to obtain, in order to decide (1) if lasers are an appropriate treatment and (2) which laser to use. The following are important considerations during consultation:

- History of prior skin cancers, skin treatments, laser or dermabrasion procedures.
- History of scar formation/keloid formation.
- History of HIV or Hepatitis or HSV outbreaks.
- History of scleroderma or prior radiation treatment as these will impact healing and are contraindications.
- Isotretinoin (Accutane) use: impairs adnexal structures, which is the source of re-epithelialization following resurfacing procedures. Patients should wait 6–12 months after discontinuing isotretinoin before undergoing resurfacing procedures.
- Consider discontinuing blood-thinning medications prior to resurfacing procedures.
- Smokers have decreased healing capabilities and will have prolonged postoperative course.
- Skin type: Fitzpatrick types 1–2 are ideal candidates for all laser treatments. Patients with Fitzpatrick levels higher than 2 or 3 are at increased risk for scarring and permanent hypopigmentation.
- Concerning skin lesions should be biopsied first to rule out malignancy prior to laser treatment.
- Patients with active skin infections or broken down epidermis should not undergo laser treatment in that setting.
- Contraindications: consumption of gold (used for treatment of RA – can induce chrysiasis – a cutaneous hyperpigmentation), isotretinoin use (relative contraindication, would wait 6–12 months after discontinuing prior to laser treatment), collagen/vascular disorder, inability

to care for the wound posttreatment, active skin infection or skin breakdown (exception would be ulceration due to hemangiomas – these can be treated successfully with PDL), vitiligo, psoriasis, pregnancy, history of radiation.

Laser Treatment Preparation

- Patients undergoing resurfacing procedure, or any laser procedure that violates the epidermis, should receive pre-procedural antivirals (acyclovir 400 mg tid, or valacyclovir 500 mg bid) starting day prior to procedure and continue for total of 7 days, especially in patients with history of HSV outbreaks. Patients should also be placed on antibiotics to cover skin flora, if they are undergoing a laser treatment which violates the epidermis (resurfacing/scar revision) starting day of procedure.
- Patients should be counseled that their skin needs to be as light as possible, so they need to avoid sun exposure prior to treatment, so that the increased melanin from the sun exposure does not interfere with the laser treatment.
- All personnel during the treatment, including the patient, needs to wear appropriate eye protection. Staff should wear appropriate goggles that block the specific wavelength of laser used. Patients should either wear laser aid eye covers, or impervious metal goggles or metal eye contacts to prevent eye injury.
- Level of anesthesia depends on type of laser used and patient comfort level (see below in clinical applications for specific recommendations).

Complications

- Pain: usually the majority of the pain encountered is during the laser procedure, so once the treatment is done, the pain should be tolerable.
- Post-inflammatory hyperpigmentation (PIH): can occur in up to 30% of patients and is usually transient, but can take several weeks or months to resolve. PIH is more common to occur in patients with Fitzpatrick skin type 3 and above. Strict sun precautions pre- and posttreatment will help prevent its occurrence. PIH can be treated with topical steroids or lightening creams (hydroquinone), or gentle chemical peeling.
- Hypopigmentation: while less common than PIH, it is a permanent and more difficult problem to treat. Ensure patient's skin is as light as possible (strict sun avoidance pre- and posttreatment) and avoid over-aggressive laser treatment (better to treat the patient over several treatments versus trying for perfection with one aggressive treatment).
- Scarring: this occurs due to aggressive treatment. These can be very difficult to treat, so advocate for more sessions at lower settings versus more aggressive fewer treatments. Scarring under the eye with an aggressive ablative laser could lead to ectropion.
- Infection: can be bacterial, fungal, or HSV outbreak. Prophylaxis with antibiotics and antivirals as indicated is the key. Ensure patients are cleaning and dressing wounds and abiding by post-op instructions.
- Milia: common complication, oftentimes exacerbated by occlusive dressings.
- Eye injury: completely avoidable complication that should never happen if all members involved in the treatment are wearing appropriate protection. For patients who need treatment in the periocular area, metal laser approved contact lenses should be inserted.
- Swelling/bruising: not complications but expected post-procedural sequelae. All patients undergoing any form of laser treatment can expect swelling in the area. Patients who are receiving laser treatments targeting blood vessels (PWS, etc.) may see purpura/bruising, which is an expected outcome from the treatment.
- Prolonged erythema: Resolves with time but can persist up to a year.

Clinical Applications

Vascular malformations/lesions PWS and hemangiomas are best treated with PDL, which is the workhorse laser for vascular lesions. Since melanin also absorbs light from PDL, it is important to ensure the epidermis is as light as possible so that the melanin in the skin does not compete for the laser energy. When treating or targeting more superficial vessels, the patient will notice purpura. Blistering may occur if the fluences are too high or the epidermis is not appropriately cooled. Patients with resistant PWS may be treated with Alexandrite laser or Nd:YAG.

Telangiectasias can also be treated with any of the vascular lasers: PDL, KTP, IPL. These lasers are also used to treat rosacea and to reduce redness/erythema in scars.

Skin resurfacing the Er:YAG and CO2 laser are the main lasers used for skin resurfacing/rejuvenation. They are considered ablative lasers, in that they remove epidermis and portions of superficial dermis. Studies have shown that CO2 laser is more efficacious than Er:YAG; however, with multiple passes of the Er:YAG, you can achieve the same result as CO2, and with fewer side effects. Many practitioners are using fractional resurfacing techniques in lieu of fully ablative techniques. The benefit of fractional resurfacing is that only a portion or "fraction" of the skin is treated, thus leaving normal skin in between treated areas, which allows for more rapid regeneration of skin from the undisturbed skin appendages and lessens downtime and potential for scarring. Post-procedural wound care is of utmost importance, as the epidermis is violated and patients are at risk for infection and scarring. Post-procedural wound care usually entails keeping wound clean and covered with an occlusive ointment, such as petroleum jelly or antibiotic ointment. Strict sun avoidance is mandatory to prevent dyspigmentation and scarring.

Non-ablative lasers are also used to help improve quality of the skin. The non-ablative lasers do NOT destroy the epidermis, but deposit the heat underneath the epidermis. These lasers have the benefit of no wound to care for; however, the improvement in skin quality/texture is not as significant as the ablative lasers. Examples of non-ablative lasers include: infrared laser (1550 nm), PDL and IPL (both of which are used in photo facials).

Scar treatment Many of the same lasers used for skin resurfacing can be used for treatment of scars. Lasers can help scars blend into surrounding skin, reduce erythema/redness associated with scar and can be used to treat keloids and contracted scars. Both ablative and non-ablative lasers can be used for treatment of scars. As above, the non-ablative lasers have the benefit of not causing epidermal damage, so there is no wound to care for. Non-ablative lasers are certainly helpful in treating patients with history of keloid, to help PREVENT keloid from forming (such as post-procedural scar). Ablative lasers are particularly helpful for raised scars or keloids, as the epidermal injury allows for topical application of steroids to further augment the benefit of the laser. Vascular lasers (PDL, KTP, IPL) can also be utilized to reduce the erythema associated with scars. Anesthesia in some form should be given depending on size and location of scar. Oftentimes, topical anesthesia is sufficient; however, topical anesthesia may cause some vasoconstriction, which will negatively affect the use of associated vascular lasers. If scar is in a sensitive area, or is of a large size, some patients may benefit from general anesthesia.

Tattoo Removal Quality-switched (Q-switch) nanosecond lasers are the mainstay for tattoo removal. Q-switch lasers allow for more precise pigment targeting than initial lasers. With the advent of picosecond lasers, these are quickly becoming the go-to laser for tattoo removal, since picosecond laser allows for precise targeting of the pigment without collateral damage and has been shown to clear the tattoo faster. Treatment of tattoos can be frustrating, as the tattoo industry is not regulated, and the ink used and particle sizes of pigment are variable. It usually takes several treatments to clear a tattoo, with dark blue/black pigments being the easiest to clear. Depending on the colors in the tattoo, various lasers should be used:

- Ruby (694 nm): dark ink
- Alexandrite (755 nm): green/dark ink
- Nd:YAG (1064 nm): blue/black ink
- KTP (532 nm): red/orange ink

When treating patients with darker skin (Fitzpatrick 3 and above), Nd:YAG should be used for treatment of tattoos, as Nd:YAG is not well absorbed by epidermal melanin, and the laser's depth of penetration is 4–6 mm into the skin, so there is less risk of hypopigmentation.

Laser Hair Removal The term "removal" is a misnomer, as the laser will reduce the density of hair and increase the interval of hair regrowth. About 15–30% of hair is permanently destroyed at each session, so multiple sessions are typically required. Current lasers are used to target the germinal cells in the follicle, which are non-melanin containing structures. Since the germinal cells have no absorption for lasers, the surgeon is reliant upon diffusion of the laser heat to destroy the germinal cells. This results in a longer hair-free period, as opposed to targeting the follicle itself. Lasers used for hair "removal" are long pulsed ruby, Alexandrite, diode and Nd:YAG. Nd:YAG is most useful in patients with darker skin, so as not to cause dyspigmentation.

Non-laser Technologies

There has been a rise in the interest in nonsurgical options for facial rejuvenation (Botox, fillers, lasers, etc.). These technologies can be offered to patients who are not yet surgical candidates (too young, not ready for downtime) or as adjuncts to surgery itself.

- Microneedling: addresses improving quality of skin. Tiny needles create microscopic holes in the epidermis, even down to papillary dermis, which stimulates growth factors and promotes collagen and elastin production. Additionally, following microneedling, topical serums, such as growth factors, can be applied, thus allowing penetration of skin barrier and allowing for more effective treatment. This technology is particularly useful for acne scarring and decreasing pore size. Several treatments may be necessary, with starting recommendation of four treatments spaced 3 weeks apart. This technology has low complication profile (minimal risk of dyspigmentation or scarring).
- Radiofrequency (RF) devices: these devices generate an electrical current, which causes heat destruction of collagen, which will lead to collagen remodeling and skin tightening. There are both monopolar and bipolar devices. It is difficult to reach adequate depth with bipolar devices, as they usually only reach epidermis, and as such are often combined with other techniques. It takes 3–6 months, even up to 1 year for the final result to be established. The skin tightening result should last 3–5 years, assuming there are no changes in patient's body habitus. There are both transcutaneous as well as minimally invasive (probes underneath the skin) applications. It is important to protect the epidermis, as heat is generated with these devices and can cause damage to epidermis, if it is not properly cooled/protected.
- Ultrasound: used for similar application as RF devices. Transducers deliver the ultrasonic energy to a focal point, causing heat destruction and tissue coagulation of the target. Ultrasound devices for skin tightening utilize Microfocused Ultrasound (MFU), as opposed to High Ultrasound (HIUS) that is used to target tumors or destroy fat. The energy density of the MFU devices is lower, and transducers emitting various frequencies can be used to target: dermis (1.5 mm), deep dermis (3 mm), or SMAS/subcutaneous tissues (4.5–5 mm), while still protecting the epidermis. The SMAS can also be targeted for tightening in addition to dermis. The thermal injury also affects collagen, which causes it to contract and become thicker and more organized. Ultrasound devices tend to cause more discomfort during the procedure than RF devices.

Questions

1. Match wavelength with laser:

CO2	1064 nm
Nd:YAG	532 nm
Er:YAG	10,600 nm
KTP	2940 nm

2. Which laser is the best choice to treat a red tattoo?
   (a) KTP
   (b) Ruby
   (c) Alexandrite
   (d) Nd:YAG
3. Which is NOT a contraindication to laser treatment?
   (a) Active skin infection
   (b) Gold consumption
   (c) Dark-skinned patient
   (d) Current isotretinoin use
4. Decreasing the spot size will ____________ the energy density?
   (a) Increase
   (b) Decrease
5. Longer wavelength lasers, in general, will have a ____________ depth of penetration?
   (a) Shorter
   (b) Deeper
6. Which is the best laser to use for dark-skinned patients?
   (a) KTP
   (b) PDL
   (c) Alexandrite
   (d) Nd:YAG
7. What is the difference between an ablative and non-ablative laser?
8. If patient has no prior history of HSV outbreak, you do not need to pretreat them with antivirals.
   (a) True
   (b) False
9. Which is more common following laser treatment: hypopigmentation or hyperpigmentation?
10. Which laser has the higher absorption: CO2 or Er:YAG.
11. It is NOT necessary to wear eye protection when using any of the lasers.
    (a) True
    (b) False
12. What is the chromophore of the CO2 laser?
13. Which of the following is NOT a use for PDL?
    (a) Hemangiomas
    (b) Port wine stain
    (c) Photofacial
    (d) Skin resurfacing
14. Microneedling is NOT indicated for which of the following?
    (a) Acne scarring
    (b) Diminishing pore size
    (c) Improving fine lines and wrinkles
    (d) Treating deep rhytids
15. What should you do if a patient has blistering following laser treatment?
16. Is IPL a laser?
17. How does laser light differ from sunlight?
18. What are the four different tissue interactions with laser light?
19. Prior to commencing with skin resurfacing, what medications should the patient begin?
20. How many months should you wait after a patient has stopped taking isotretinol before resurfacing procedure?
21. The target of laser hair removal is the hair follicle.
    (a) True
    (b) False
22. Scars may be treated with same lasers used for resurfacing.
    (a) True
    (b) False
23. Which laser is best used for red or orange-colored tattoos?
24. How can hypopigmentation following laser procedure be treated?
25. How can hyperpigmentation following laser procedure be treated?

Answers

1. See Table 24.2
2. (a)
3. (c)
4. (a)

5. (b)
6. Nd:YAG
7. Ablative will cause injury/damage to epidermis
8. False
9. Hyperpigmentation
10. CO2
11. False
12. Water
13. (d)
14. (d)
15. Patient should keep blister covered with occlusive dressing and petroleum ointment applied, and avoid sun,
16. No, IPL is an Intense Pulsed Light with many different wavelengths. Filters can be applied to narrow the wavelength bands
17. Laser light is monochromatic (single wavelength), coherent (in phase) and does not diverge
18. Absorption, transmitted, scattering, reflected
19. It is important to ensure patient is on antivirals the day before the procedure and starts antibiotics the day of the procedure
20. At least 6 months
21. (b) False. The target is the germinal center, which is not a direct target of the laser, but relies on heat diffusion to destroy the germinal center. This allows for longer lasting results,
22. (a),
23. KTP
24. Avoidance
25. Lightening creams (hydroquinone), gentle chemical peeling

Additional Resources

1. Allemann IB, Kaufman J. Laser principles. Curr Probl Dermatol. 2011;42:7–23.
2. Gan SD, Graber EM. Laser hair removal: a review. Dermatol Surg. 2013;39:823–38.
3. Torbeck R, Bankowski R, Henize S, Saedi N. Lasers in tattoo and pigmentation control: role of the PicoSure ® laser system. Med Devices Evidence Res. 2016;9:63–7.
4. Alexiades-Armenakas MR, Dover JS, Arndt KA. The spectrum of laser skin resurfacing: nonablative, fractional and ablative laser resurfacing. J Am Acad Dermatol. 2008;58(5):719–37.
5. Fabi SG. Noninvasive skin tightening: focus on new ultrasound techniques. Clin Cosmet Investig Dermatol. 2015;8:47–52.
6. Ramaut L, et al. Microneedling: where do we stand now? A systemic review of the literature. J Plast Reconstr Aesthet Surg. 2017; https://doi.org/10.1016/j.bjps.2017.06.006.

Liposuction

25

Andrew D. Beckler and Mehdi Sina

Anatomy

Sensory Nerves

Cervical Plexus (C2–4)

- Greater occipital n. (C2).
- Lesser occipital n. (C2, 3).
- Great auricular n. (C2, 3).
- Transverse cervical n. (C2, 3).
- Supraclavicular n. (C3, 4).

Motor Nerves

CN VII (Facial Nerve)

Innervates the muscles of facial expression via five terminal motor branches: *temporal*, *zygomatic*, *buccal*, *marginal mandibular*, *cervical*. These branches are found at four different depths in the face and neck.

Motor nerve function to the mimetic muscles of the first three layers is supplied through the deep surface of the muscle whereas the deepest layer of muscles, including the *buccinator*, *levator anguli oris*, *and mentalis*, receive their *innervation* via *the superficial surface* of the muscle (Table 25.1).

A. D. Beckler
Department of Head and Neck Surgery, UCLA, Los Angeles, CA, USA

M. Sina (✉)
Pearlman Aesthetic Surgery, New York, NY, USA

Vascular Supply

The blood supply to the lower face and neck is provided by branches of the external carotid artery including the superior/inferior labial and angular arteries, superficial temporal artery, transverse facial, submental, and posterior auricular arteries. Venous drainage is carried by the internal, external, and anterior jugular veins as well as the veins that run with their corresponding arteries. The majority of the neurovascular supply lies superficial to the subcutaneous fat in the subdermis.

Neck

Anterior compartment—bounded by the inferior border of the mandible, the medial border of the SCM and the sternum.

Posterior compartment—bounded by the posterior border of the SCM, the anterior border of the trapezius, and the middle third of the clavicle.

Superficial cervical fascia is deep to the skin and envelopes the platysma muscle. The *deep cervical fascia* invests the neck structures and is divided into *three layers*: the *investing layer* surrounds the SCM and trapezius; the *visceral (pretracheal) layer* is limited to the anterior neck and surrounds the trachea, esophagus, infrahyoid strap muscles, and the *prevertebral*

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_25

Table 25.1 Mimetic muscles. The mimetic muscles are supplied by the facial nerve (CN VII) and are involved in facial movements and the expression of human emotions

Muscle layer	Mimetic muscles	Innervation
1 (Superficial)	Depressor anguli oris, zygomaticus minor, and orbicularis oculi	Deep surface of muscle
2	Depressor labii inferioris, risorius, platysma, zygomaticus major, and levator labii superioris alaeque nasi	Deep surface of muscle
3	Orbicularis oris and levator labii superioris	Deep surface of muscle
[a]4 (deep)	[a]Mentalis, levator anguli oris, and buccinator (MLB = mnemonic for these muscles)	[a]Superficial surface of muscle

[a]Muscles that are innervated along their superficial surface

layer invests the vertebral column and the prevertebral muscles.

Introduction to Liposuction

Physiology of Adipose Tissue

Fat in the fetus is thought to develop from mesenchymal derived fibroblasts and appears in month 4 of gestation. The number of fat cells triples during year 1 of life and increases again during puberty. Adipose tissue is composed of adipocytes (with variable fat content) surrounded by a connective tissue matrix (collagen, elastin) along with macrophages, pericytes, fibroblasts, and mast cells. Weight gain is initially through lipid accumulation and *adipocyte hypertrophy*. As weight gain continues, however, immature precursors form new fat cells—this *adipocyte hyperplasia* is thought to be permanent and resistant to future weight loss. Weight loss is associated with decreased lipid cellular concentration, but the number of adipocytes remains constant.

Fat deposits in the face and neck can be attributed to genetics, hormonal imbalance, poor diet, and lack of exercise. Facial fat compartments are isolated and as a result they do not respond to diet and exercise like other fat deposits in the body, and they are the first to hypertrophy with an increase in weight. Since the number of fat cells in the adult is relatively stable, the *goal of liposuction* is to *permanently remove facial/neck fat cells* by suction-assisted avulsion and facilitate *contraction of the subdermis* (including the subcutaneous tunnel network) during the healing process.

Patient Selection and Analysis

The *ideal candidate* for liposuction is a younger patient *with average weight, excess fat deposits on exam, good skin elasticity and muscle tone*. Women are generally better candidates than males as they have thinner, less sebaceous skin that allows for improved postoperative contraction. As patients age, the ability of the skin to contour to the neck is diminished due to breakdown of elastic fibers and collagen and progressive muscle laxity. Patients with significant skin aging, prominent platysmal bands, and muscular ptosis may be better suited for rhytidectomy with plastymaplasty. Illouz recommends measuring the distance between the earlobe and menton at rest and while pulling the skin away from the face. Patients with measurement differences <15% are considered ideal liposuction candidates, while differences >20% are better served with rhytidectomy. As with any elective surgical procedure, it is critical that the patient has realistic expectations of the anticipated outcome.

The facial plastic surgeon needs to elicit information from the patient regarding their past medical history, past surgical history, medications, allergies, tobacco/alcohol use, history of easy bleeding, and problems with anesthesia. Specific questions regarding prior neck surgery, trauma, history of radiation, or history of collagen, vascular, or systemic diseases that would impede wound healing should be identified. Additionally, patients that are overweight and who have generalized fat deposition in multiple tissue layers are difficult to correct with liposuction alone and may benefit from a weight loss program. Planned weight loss should occur at least 6 months prior to surgical intervention. Failure to recognize these important considerations can lead to complications, poor results, and patient dissatisfaction.

Physical Exam

Characteristics of a youthful, aesthetically pleasing neck: *distinct border of inferior mandible with no jowl, visible thyroid cartilage, subhyoid depression, visible anterior border of SCM, and a cervicomental angle between 105 and 120°*. Definition of the cervicomental angle can be affected by a low/anterior hyoid, retrusive chin, prominent digastrics, ptotic submandibular glands, and skin elasticity/tone. The Dedo classification system provides a useful reference for the surgeon to highlight certain physical exam findings as well as the aesthetic procedures that are commonly used to address them (Table 25.2). The surgeon must carefully consider these when evaluating a patient for surgery.

The hyoid and chin position are critical to analyze as the underlying bony anatomy ultimately determines the appearance of the neck. The position of the hyoid is a critical factor in determining the cervicomental angle. A low hyoid position is difficult to correct and can lead to disappointing results. The *ideal chin projection* is defined as 3 mm posterior to the nose-lip-chin plane, which is the line extending from a point one-half the distance of the ideal nasal length through the upper and lower lip vermillion. Alternatively, it has been defined as one in which the anterior border of the chin closely approximates a vertical line from the vermillion border of the lower lip. Patients with retrognathia or microgenia may benefit from chin augmentation in addition to suction-assisted lipectomy for an optimal outcome.

Table 25.2 Dedo classification of cervical abnormalities. The Dedo classification is used to assess the types of neck deformities as well as the best surgical techniques to address them

Dedo classification		
Class	Deformity	Proposed technique
1	Minimal deformity, well-defined cervicomental angle, no fat accumulation, good platysma tone, younger patient (<40)	N/A
2	Skin laxity, no fat accumulation, good platysma tone	Rhytidectomy
3	Fat accumulation	Suction-assisted lipectomy
4	Platysma banding	Platysmaplasty
5	Congenital/acquired retrognathia	Mentoplasty, genioplasty
6	Low hyoid	Difficult to optimize cervicomental angle

The submandibular glands and anterior digastric muscles should also be inspected for their role in obscuring the cervicomental angle. Digastric prominence, submandibular gland ptosis and/or hypertrophy should be distinguished from fat deposits as correction of these deformities is optimally achieved with reduction techniques through an anterior neck approach.

Patients with platysmal banding should be cautioned regarding liposuction. Removal of fat can exacerbate or unmask platysmal banding resulting in *cobra neck deformity*. In this patient population, a platysmaplasty is often required to produce an improved neck contour.

Patients with good skin elasticity, no platysmal banding, and localized fat deposits stand to benefit the most from this procedure.

Photo Documentation

Standard facial photographs should be obtained preoperatively for documentation and to use for photo manipulation to demonstrate anticipated postoperative results. Photographic views should include anterior, lateral, oblique, and neck flexion. Additional photographs with the patient smiling and grimacing document the form and function of the platysma.

Patient Counseling

Skin irregularities and asymmetries should be noted and discussed with the patient preoperatively. The expected postoperative course should also be discussed with the patient including bruising and discoloration, which can last for 1–3 weeks, as well as edema and induration, which can last for weeks to months. Less commonly, patients can experience pigment changes related to hemosiderin deposits. Patients commonly wear a facial sling continuously for a week to help limit swelling and then intermittently for up to 4 weeks after surgery. Irregularities are usually transient and are treated conservatively with reassurance, gentle massage, and occasionally with steroid injections. Temporary numbness and tingling is also possible. Activity is usually restricted for 2–3 weeks to prevent additional swelling, decrease the chance of bleeding, and allow the soft-tissue envelope to adhere to the subcutaneous tissues. Results are unpredictable and vary by patient and may take up to 6 months to be evident.

Incisions, Approaches, Techniques

Incisions can be placed in the following areas: *submentum*, *infralobular crease*, *nasal vestibule*, *pretragal*, *postlobular*, *and temporal hairline*. Incisions must be made large enough (4–8 mm) to avoid friction burns and skin excoriation. It is important to use a carefully designed and evenly distributed tunnel system to avoid irregularities and bulges.

Facial regions typically treated with liposuction include submentum, lateral neck, jowls, and buccal region. Liposuction may treat nasolabial folds and lower jowls, although results are less predictable. Treatment of the mid-face is risky because of the natural tendency for fat atrophy in this region. Areas of excess skin laxity and tissue descent are not good areas to treat with this procedure.

Equipment

- Cannulas: 1, 2, and 3 mm (most precise) up to 4 and 6 mm (used judiciously for larger fat deposits).
- Fat cells are sucked up through the perforated cannula and avulsed.
- High vacuum pressure can cause cell lysis directly.
- Low vacuum pressure is used if fat transfer is going to be performed to decrease trauma to adipocytes and increase the yield of viable cells (usually with Luer-Lock aspiration cannula).
- Negative pressure generated by electrical machines is 1 atm = 960 mmHg; handheld syringe is 700 mmHg initially, then drops to 600 mmHg.

Description of Procedure

Patient marked preoperatively for important landmarks (SCM, hyoid, angle of mandible) and areas to be treated in a seated, upright position (tissue can become distorted when supine).

Approaches

Closed is often used when suction lipectomy is performed as an isolated procedure.

Open is common when the procedure is performed along with a facelift and larger incisions are used (an electric vacuum must be used for the open technique because there is no seal present to allow for use of handheld syringes).

Tumescence of treatment area with hypotonic saline solution mixed with local anesthetic (causes hydrodissection and facilitates fat aspiration); additionally, local anesthetic is used for nerve blocks along planned incisions and throughout the treatment area.

Incisions are made based on the areas to be treated and scissor dissection is carried out to establish the appropriate plane; the cannula is then used for pretunneling before attaching the vacuum to facilitate passage of the cannula during the procedure.

Perforated rigid blunt-tipped cannula (1, 2, and 3 mm—best control/precision) attached to a suction device inserted through skin incision and passed back and forth through fatty deposits through subcutaneous tunnels just deep to the dermal–subcutaneous interface; fenestra of the cannula is *pointed away from the dermis* to avoid injury.

Superficial tunnel is used by tenting skin away from deep tissues; the left hand is the "smart" hand and is used to guide the cannula, direct fat into the fenestra, and maintain the correct plane; the right hand is the "motor." Care is taken to avoid perforation of the platysma, which could put the patient at risk of injury to the underlying neurovascular structures and promote bleeding.

Treatment extends to SCM laterally and hyoid/sternum inferiorly in a radially directed fan-shaped pattern. Areas of greatest fat density can be treated with larger cannulas to aid in fat removal; more distant sites are treated more conservatively to help achieve a feathering/blending effect.

Care should be taken when removing the cannula through incision site to avoid skin injury (simply pinching the suction tube temporarily will stop the vacuum to allow safe withdrawal and reinsertion).

Use of multidirectional crisscross approach generates extensive overlapping tunnel network that facilitates contour enhancement (Fig. 25.1). Avoidance of fat overexcision is accomplished by frequent checking of progress and the *pinch-and-roll technique*, which involves gently pinching the skin between thumb and forefinger and rolling it—a sufficient amount of fat is excised when a thin layer of fat is felt between the layers.

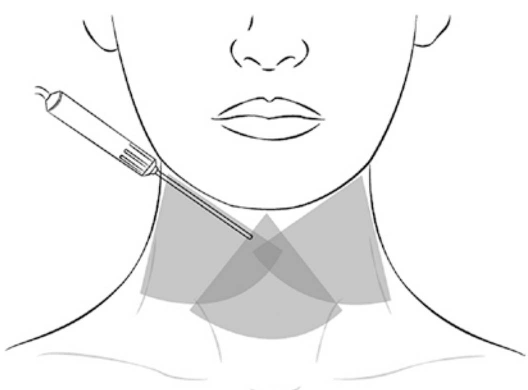


Fig. 25.1 Radial fan-shaped tunnels are used to perform suction-assisted lipectomy. Multiple access points and overlapping treatment areas help to blend and feather transition zones

Extraction volumes vary per person, but usually require 10–100 cc.

Sometimes, fat deep to the platysma needs to be excised to improve the cervicomental angle either using careful liposuction or direct excision via an open approach.

At the conclusion of the procedure, the sites are irrigated and hemostasis is achieved, drains are usually not needed, but a compression dressing is used to help prevent edema/hematoma.

Tumescent Liposuction

Potential lidocaine toxicity is most important consideration with tumescent anesthesia. Absorptive pharmokinetics of dilute lidocaine in fat differ from dermally injected lidocaine. Lidocaine is highly lipid soluble and strongly lipophilic. When combined with dilute epinephrine (1:1,000,000), lidocaine concentrations 0.1% or less, can be used up to a maximum acceptable lidocaine dose ranging from 35 to 55 mg/kg.

Epinephrine is the key ingredient to improve hemostasis, lower peak lidocaine serum levels, and prolong anesthesia. Increased cardiac output may increase hepatic metabolism. Maximum epinephrine dose should not exceed 50 μg/kg. The addition of sodium bicarbonate (10 mEq/1 L) lowers the pH and eliminates the discomfort associated with injection.

Complications

Transient irregularities are common and should be discussed with patients preoperatively. There is less than 1% chance of infection, hematomas, seromas, and sialoceles. Skin sagging and dermal scarring can result in longer-lasting irregularities. Asymmetry may result from uneven aspiration from poor technique. Depressions can develop that require treatment with filler. Rarely, patients may experience weakness of the marginal mandibular branch of the facial nerve or hypoesthesia.

Bulges, asymmetries, and dimples that persist after 6 months can often be treated with additional suction lipectomy, fat injection, or subcutaneous steroid injection. Treatment that is too aggressive can result in hollowing, concavities, or unmasking of platysmal banding.

Lidocaine Toxicity

Ostad et al. demonstrated peak plasma concentrations <5 μg/mL over 24 h period with no evidence of lidocaine toxicity with a lidocaine dose of 55 mg/kg. Main elimination route is hepatic metabolism via cytochrome P450 (CYP450) and renal excretion. Common medications that inhibit CYP450 include benzodiazepines, antidepressants, azole antifungals, macrolide antibiotics, calcium channel blockers, HIV protease inhibitors, as well as herbal supplements. Therefore, one should discontinue all medications that can impair hepatic metabolism 2 weeks prior to the procedure or limit maximum lidocaine dosing to 35 mg/kg.

Autologous Fat Transfer

Since the introduction of liposuction, autologous transfer of harvested fat has been used for facial rejuvenation, traumatic deformities, lipodystrophy, hemifacial atrophy, rhytides, malar and chin augmentation, post-liposuction irregularities, etc. Knowledge of adipocyte physiology and optimal harvesting techniques has led to the use of cannulas no larger than 3 mm—common technique uses 3–10 cc syringe with 14-G needle or blunt micro-cannula for aspiration of fat. The harvested fat is then separated by allowing it to settle over time, by filtering it with gauze, or with light centrifugation. Minimizing trauma to the fat cells helps to improve yields. Extra fat can be frozen and used for up to 2 years. The recipient area is anesthetized with care not to distort the tissue. Fat is injected in a retrograde fashion from deep to superficial with a 1 ml syringe using a cannula or needle. It is done using minimal positive pressure with multiple passes to achieve a natural and smooth appearance. It is usually injected into the subcutaneous plane with slight overcorrection. After injection, the area can be molded for further refinement and contouring. May require multiple treatments for optimal results.

Additional Considerations

When used alone, suction lipectomy is most effective in creating *contour changes* in the cervicomental angle and jowl regions. Less predictable results are achieved when treating the nasolabial folds and lower jowl fat pads. Patients over 40 may not possess enough skin elasticity to allow for adequate contraction after liposuction alone; therefore, rhytidectomy may be necessary to achieve optimal results.

Suction lipectomy can also be used as an adjunctive procedure for further refinement along with chin implant, rhytidectomy, or platysmaplasty. For example, pretunneling followed by closed liposuction is done in the neck and lower face. After reduction of cervicofacial fat collections, undermining of facial flaps is facilitated by the atraumatic subcutaneous tunnels created from suction lipectomy, which only requires division of the subcutaneous bands to complete the dissection. Once the flaps are raised, open liposuction may be carried out using a spatula-shaped cannula tip to further refine the lower face and neck.

New innovations in the field of liposuction of the face and neck include the introduction of smaller and varying types of cannulas, tumescence of the areas to be treated, use of ultrasound, and the liposhaver.

In ultrasound-assisted liposuction, ultrasonic energy is used either internal or external to the fatty deposit to disrupt fat cells and facilitate aspiration. Some studies of body liposuction with ultrasonographic assistance also report less swelling and less bruising postoperatively. The mechanism by which ultrasound energy works is its conversion mechanical vibrations resulting in a cavitational effect that results in liquefaction of fat. Limitations of this technique include excess heat generated by the ultrasound device at the skin incision site and the potential heat-related complications generated at more distant subdermal sites. In an animal-based study, high-amplitude ultrasonic energy caused histologic neural injury as well as neuropraxias. The facial plastic surgeon should be cautious when considering use of internal ultrasonography-assisted liposuction.

The liposhaver (similar to the nasal polyp shaver used in endoscopic sinus surgery) is a guarded, motorized blade that removes fat via sharp excision and requires minimal suction pressure. It is thought to be less traumatic because of the excision-versus-avulsion principle resulting in less postoperative bruising.

Noninvasive Treatment Options

Deoxycholic acid, also known as Kybella® (Allergan, Irvine CA), is a bile acid that emulsifies fat, which is then absorbed in the intestine. It is FDA approved for reducing moderate to severe submental fat. It is injected into subcutaneous fat using an area-adjusted dose of 2 mg/cm^2. A single treatment consists of up to a maximum of 50 injections, 0.2 mL each (up to a total of 10 mL), spaced 1 cm apart. Up to 6 single treatments may be administered at intervals no less than 1 month apart.

Cryolipolysis, or CoolSculpting®, is a noninvasive treatment used to selectively destroy fat cells. After an assessment of the dimensions and shape of the fatty bulge to be treated, an applicator of the appropriate size and curvature is chosen. The area of concern is marked to identify the site for applicator placement. A gel pad is placed to protect the skin. The applicator is applied and the bulge is vacuumed into the hollow of the applicator. The temperature inside the applicator drops, and as it does so, the area numbs.

It works by controlled cooling of fat deposits to a temperature of +5 to −10 °C for 60 min. Patients sometimes experience discomfort from the vacuum's pull on their tissue, but this resolves within minutes, once the area is numb. Currently, a small-volume vacuum applicator, CoolMini applicator, is used in the treatment of the submental area. In a recent study, patient surveys showed that submental cryolipolysis is well-tolerated, produced visible improvement in the neck cryolipolysis contour, and generated high patient satisfaction. Cryolipolysis of submental fat has FDA approval. Final results can be seen by 3–5 months with each treatment session reducing the fat volume by 20–25%.

Questions

1. Inconspicuous cervicofacial liposuction incisions may be placed where?
   (a) Submentum
   (b) Infralobular crease
   (c) Nasal vestibule
   (d) Temporal hairline
   (e) All of the above
2. All of the following are modes of fat cell mobilization, except:
   (a) Thermal
   (b) Mechanical disruption
   (c) Ultrasound
   (d) Tumescence
3. How long should a patient wait after planned weight loss prior to cervicofacial liposuction?
   (a) 1 month
   (b) 3 months
   (c) 6 months
   (d) 12 months
4. True or false: women are better candidates than men for cervicofacial liposuction as a secondary procedure?
5. What is the most important anamonic factor when evaluating a patient for cervicofacial liposuction?
   (a) Position of hyoid bone
   (b) Skin tone
   (c) Position of the chin
   (d) Position of submandibular glands
6. Potential problems with use of subcutaneous ultrasonic energy include:
   (a) Excess heat generated by the ultrasound device at the skin incision site
   (b) More swelling
   (c) Increased rates of hematoma formation
   (d) Functional evidence of nerve injury caused by low-amplitude ultrasound energy

7. When compared to standard liposuction techniques, the “liposhaver”:
   (a) Causes more soft tissue trauma
   (b) Does not require high suction pressure
   (c) Is less precise
   (d) Requires less experience
8. Important facial landmarks to be marked preoperatively include all of the following, except:
   (a) Hyoid
   (b) Sternocleidomastoid
   (c) Digastric
   (d) Angle of mandible
9. When using a tumescent solution, how much time should elapse to allow for adequate vasoconstriction and anesthesia to take effect?
   (a) 5 min
   (b) 10 min
   (c) 15 min
   (d) 30 min
10. Overaggressive fat liposuction in the cervicomental region may result in:
    (a) Unmasking platysmal banding
    (b) Hollowing
    (c) Masculinized appearance
    (d) All of the above
11. The recommended minimum incision length to avoid friction burns in cervicofacial liposuction is?
    (a) 2 mm
    (b) 4 mm
    (c) 6 mm
    (d) 8 mm
12. What is the largest cannula diameters suggested in cervicofacial liposuction procedures?
    (a) 4 mm
    (b) 6 mm
    (c) 8 mm
    (d) 10 mm
13. Common postoperative changes include all of the following, except:
    (a) Bruising and discoloration for up to a month
    (b) Numbness and tingling
    (c) Marginal mandibular nerve weakness
    (d) Skin irregularities
14. True or false: infections occur in less than 1% of patients?
15. True or false: preoperative antibiotics are required prior to cervicofacial liposuction?
16. All of the following are potential complications of triamcinolone injection, except:
    (a) Epidermal thinning
    (b) Dermal dimpling
    (c) Spider telangiectasias
17. Treatment of submental fat deposits of deoxycholic acid must be spaced at least how long apart?
    (a) 2 weeks
    (b) 4 weeks
    (c) 6 weeks
    (d) 8 weeks
18. Which of the following statements about cryolipolysis is true?
    (a) It is not FDA approved for the treatment of submental fat
    (b) It has poor patient satisfaction scores
    (c) It cools fat to as low as −10 °C
    (d) Frostbite to overlying skin is not uncommon
19. A female patient with prominent bands, good skin elasticity, no appreciable skin excess and no lipodystrophy would benefit most from what procedure?
    (a) Neck lift
    (b) Suction-assisted lipectomy with platysmaplasty
    (c) Platysmaplasty
    (d) Injection of bands with neurotoxin
20. Which of the following facial mimetic muscles receives facial nerve innervation from its superficial surface?
    (a) Depressor anguli oris
    (b) Mentalis
    (c) Platysma
    (d) Zygomaticus major
21. Negative pressure levels generated by electric vacuums and handheld syringes used in suction-assisted lipectomy are:
    (a) 960 mmHg/700 mmHg
    (b) 900 mmHg/660 mmHg
    (c) 1.5 atm/600 mmHg
    (d) 1 atm/650 mmHg

22. Techniques for harvesting autologous fat for transfer include all of the following, except:
    (a) Tissue filter
    (b) Centrifuge
    (c) Cell sorter
    (d) Separation in syringe
23. A 58-year-old female patient comes to your office to consult with you regarding her concerns about the appearance of her neck. She has moderate jowling, fat deposits in the submental and submandibular regions, skin laxity, and no obvious platysmal banding. What procedures would you recommend for her facial rejuvenation?
    (a) Rhytidectomy and liposuction
    (b) Liposuction and platysmaplasty
    (c) Rhytidectomy and platysmaplasty
    (d) Rhytidectomy, liposuction, and platysmaplasty
24. The volume of fat typically extracted during cervicofacial liposuction is approximately:
    (a) 5–50 cm^3
    (b) 10–100 cm^3
    (c) 50–200 cm^3
    (d) 75–300 cm^3
25. The primary limitation of using tumescent solution in cervicofacial liposuctions is:
    (a) Large fluid shifts
    (b) Lidocaine toxicity
    (c) Higher chance of postoperative asymmetry
    (d) Greatly increased operative time

Answers

1. (e)
2. (a)
3. (c)
4. False (women are better candidates for cervicofacial liposuction as a primary procedure because of thinner and less sebaceous skin)
5. (a)
6. (a)
7. (b)
8. (c)
9. (c)
10. (d)
11. (b)
12. (b)
13. (c)
14. (a)
15. (b)
16. (a)
17. (b)
18. (c)
19. (d)
20. (b)
21. (a)
22. (c)
23. (d)
24. (b)
25. (c)

26 Rhinoplasty

Gregory Kelts and Jacob O. Boeckmann

Anatomy

Arterial supply *internal carotid* (the nasal lining → ant. and post. ethmoidal arteries) (terminal branch of the ophthalmic), *external carotid artery* (external skin envelope—facial artery → angular artery and superior labial artery), and also sphenopalatine and greater palatine arteries.

Venous outflow corresponds to artery distribution. Directly connected to cavernous sinus due to lack of valves → intracranial extension of infections.

Innervation ophthalmic division of V1 and maxillary division of V2.

Skin/soft tissue envelope the skin covering the nose is of variable thickness—thick over radix, thin at rhinion, and thickening again toward the tip (more sebaceous glands are present).

The *nasal lining* → squamous epithelium lining the vestibule before transitioning to psuedostratified columnar epithelium. The nasal *SMAS* of the nose connects the nasal muscles together [1].

Paired nasal bones superiorly attached to the frontal bone, laterally attached to the lacrimal bone, and ascending process of the maxilla.

Upper lateral cartilage Two trapezoidal cartilages fused to the cartilaginous septum superiorly. Cephalic edge travels under the caudal border of the nasal bone for a variable distance (*keystone area*), and is attached to the lateral edge of the pyriform aperture by loose ligaments.

Lower lateral cartilage (Fig. 26.1) Consists of medial, middle/intermediate, and lateral crura.

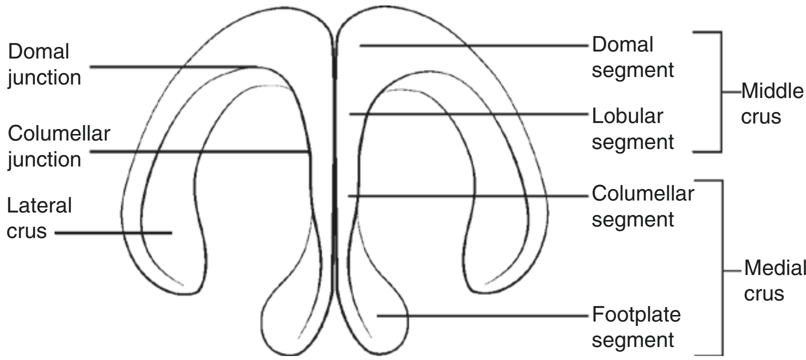
- *Medial crura*: two components, footplate segment and columellar segment. *Columellar-lobule junction*: distinct entity where the paired vertically oriented medial crura diverge at the middle crura, serving as breakpoint of double break. Middle crura-lobular and domal segments: *Domal junction*: transition from middle crus to lateral crus, critical landmark for nasal tip. Ideal domal anatomy has a convex domal segment next to a concave lateral crura [2]. The *distance between tip defining points* should be *6–8 mm* for females and *8–10 mm* for males. The medial crural foundation is supported by the attachments to the caudal septum.

G. Kelts (✉)
Otolaryngology, HealthPartners Specialty Center, Minneapolis, MN, USA

J. O. Boeckmann
Pacific Coast Facial Plastic Surgery, Laguna Niguel, CA, USA

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_26

Fig. 26.1 Lower lateral cartilage anatomy



- *Lateral crura*: primary component of lobule, influences shape, size, and position. Normal position when angled 45° or more from the midline directed at the lateral canthus. *Normal angulation of lateral crura* is 15° [2].

Sesamoid/accessory cartilages located at the lateral aspect of the lateral crura in fibrofatty tissues—support the ala.

Major Tip Support (1) size, shape, thickness, and resilience of the lower lateral cartilage; (2) upper lateral cartilage attachment to the cephalic margin of the alar cartilage; and (3) medial crural footplate attachment to the caudal septum

Minor Tip Support anterior septal angle, skin of nasal tip, membranous septum, caudal septum, nasal spine, paradomal ligament, sesamoid cartilage

Nasal Valves

External nasal valve alar sidewall, medial crus of alar cartilage, nasal spine, and floor of the nose. The dilator nasalis muscle opens during inspiration. Assessed on basal view → alar collapse on inspiration.

Internal nasal valve septum, caudal end of upper lateral cartilage, nasal floor, and head of the inferior turbinate. Site of maximal airway resistance. Airflow velocity increases at areas of narrowing, which increases transmural pressure resulting in inspiratory collapse (Bernoulli principle). A larger cross-sectional area remains patent due to decreased transmural pressure. The valve angle should be *10–15°*. Patients with short nasal bones can be predisposed to internal nasal valve collapse due to the collapse of long upper lateral cartilage during inspiration. Airflow measured by classic rhinomanometry or acoustic rhinometry (measures sound reflection secondary to nasal volume). Widespread application is limited.

Analysis

Careful nasal analysis is the essential first step for a successful outcome [3]. If the surgeon fails to preoperatively identify a specific anatomic anomaly, long-term results will be compromised. The ideal nose is one that is balanced with other facial features of the patient. The ideal nose does not draw attention to itself, but instead draws attention to the eyes. Proper analysis begins with a thorough history to identify the patient's perceived complaint, motivation for surgery, and surgical expectation following treatment. The surgeon must determine if the patient's expectations are reasonable, realistic, and obtainable. Men typically are more resistant to changing their external appearance than women. It is during the history that the rhinoplasty surgeon evaluates the patient's psychiatric status. The majority of individuals seeking rhinoplasty are psychiatrically normal, but one must always be cautious when dealing with someone with body dysmorphic disorder. In addition, preoperative high levels of anxiety can be a risk factor for postoperative mild depression and must be considered in the consultation process [4].

Standard photo documentation for all patients consists of frontal view, left and right lateral, left and right oblique, and base view. All photographs should be taken with the patient in the Frankfort horizontal position (line from EAC to infraorbital rim).

Frontal View

Frontal view begins with assessment of the vertical thirds and horizontal fifths. The vertical thirds consist of trichion to glabella, glabella to subnasale, and subnasale to menton. The horizontal fifths are divided with the width of the nasal base equal to the intercanthal distance. Ideal nasal length should be two-thirds the length of the midfacial height.

Symmetry, the *brow-tip aesthetic line*, contour abnormalities, and tip definition are all assessed on frontal view. The brow-tip aesthetic lines follow a path from the supraorbital rim to the tip-defining points and should be symmetric. The nasal width is also assessed on frontal view. The *bony base* should be 75–80% the width of the alar base. The *alar base* should approximate the intercanthal distance (half the interpupillary distance). Asymmetry or a wide bony base may require osteotomies to correct. *Tip definition* should be assessed. A well-defined tip is associated with a *double break* appearance (supratip: junction of nasal tip and dorsum, infratip: junction of infratip lobule and columella). *Bulbosity of the tip* should be noted as well as the underlying cause (thick skin, widened lateral ala crura, divergent interdomal angle, wide domal angle (Fig. 26.2), lateral crural cephalic positioning). The nasal rim should have a *gull-in-flight* configuration with the columella hanging just below the alar rims. Excessive nostril may be present in the setting of over-rotation.

The width of the bony base at the level of the maxilla is compared to the intercanthal distance to establish if the nasal dorsum needs to be narrowed. If the width is larger than the intercanthal distance, lateral osteotomies are recommended.

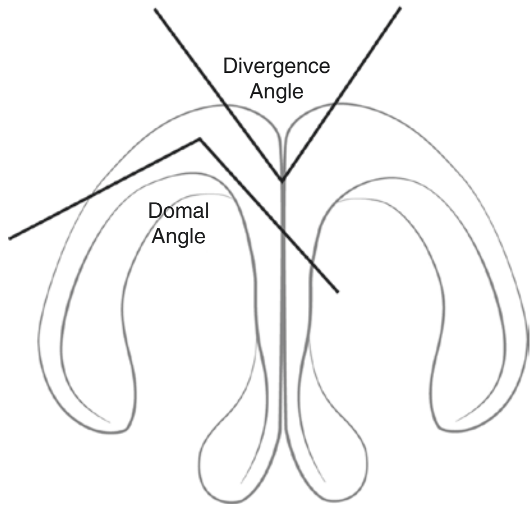


Fig. 26.2 Lower lateral cartilage angle of divergence and domal angle

Lateral View

Often one of the most concerning to the patient, the lateral view is a 2D analysis of the profile. *Projection, rotation, columellar show*, the *dorsal contour, nasal length*, and *chin position* are assessed on lateral view. *Projection* is the distance to the nasal tip from the facial plane. There are several methods to evaluate it:

1. Crumely-Lanzer method: 3–4–5 triangle.
2. Simon method: Columella to vermillion: subnasalae/tip = 1:1 ratio
3. Goode method: projection/length ratio 0.55–0.60

Rotation is the position of the tip-defining point along an arc in relation to the porion (EAC). Most often measured using the *nasolabial angle* (females, *95–115°*; males 90–95°). Shorter patients can tolerate more rotation, while taller patients need less.

The dorsum is then inspected for contour, height, and length. The *nasofrontal angle* (angle of line from *glabella* to nasion and nasion to tip defining point) should be *115–130°*. The deepest point of the nose (*nasion*) is ideally positioned at the *supratarsal crease*. It ideally lines *4–6 mm* posterior to glabella and *11 mm* anterior from the supratarsal crease. A shallow angle results in a longer nose, while an acute angle gives the nose a short appearance. The nasion may be positioned at the upper lash line instead of the supratarsal crease. One then examines the dorsal height. For females, the ideal dorsum should lie 1–2 mm posterior to a line drawn from the nasion to the tip. A slight supratip break is a sign of a well-defined tip in females. The male dorsum should be straight without a supratip break.

Chin position evaluated relative to the *Gonzales-Ulloa* line (dropping a line from the nasion perpendicular to the Frankfort horizontal line). The chin should be within *2 mm* of this line. Alternatively, one can drop a perpendicular line from the lower lip. Males should meet or closely approximate this line. Inadequate projection may be due to *microgenia*, *micrognathia*, or *retrognathia* and should be managed accordingly.

The alar–columellar relationship is important with *2–4 mm* of columella show. Excessive show can be due to an over-projected caudal septum, overdeveloped medial crura, or alar retraction. Inadequate show can be the result of a hanging alar lobule or a retracted columella.

Base View

When viewed from the base, the nose should resemble an isosceles triangle. The lobule should be one-third the height of the triangle with the columella making up the remaining two-thirds (columella:lobule ratio 2:1). The nostrils should be pear/teardrop shaped with the widest portion at the base. The ala base should equal the *intercanthal width*.

Ethnic Variations

Variations exist in different ethnic groups that must be recognized to ensure balanced approached to rhinoplasty. African-Americans, Asians, and Latinos typically have a wider nasal base, weak cartilage, thick skin, and an under-projected dorsum. The Middle Eastern patients frequently have strong alar cartilages with an over-projected dorsum.

Physical Exam

The skin thickness evaluation is critical: *thick skin* → inelastic and requires aggressive techniques for refinement. Thin skin → small imperfections can result in postoperative contour deformities.

Palpation of the nasal framework is crucial when examining the nose, as the strength of the lower lateral cartilages has a significant impact on the chosen technique. *Tip recoil* → inherent strength of the nose. Palpation of the nasal spine → relationship between posterior septal angle and nasal spine. If significant cartilage is needed, such as in revision cases, the septum is palpated to ensure sufficient grafting material is available.

An intranasal exam: should include assessment of the septum, turbinates, mucosa, and internal nasal valve. A Cottle maneuver → internal nasal valve collapse. Lateral displacement of the upper lateral cartilage with a fine tip q-tip improves airflow when internal valve incompetence is present. In the setting of a *septal perforation*, one must elicit a history to determine the causes (previous surgery, cocaine abuse, Wegener's/autoimmune disease, medication abuse). If the diagnosis is unclear, proceed with a biopsy to rule out an autoimmune disorder.

An overactive *depressor septi muscle* can result in lowering of the tip with rounding.

Nasal Obstruction can be from reversible or irreversible causes.

Reversible nasal obstruction typically the result of mucosal abnormalities from infectious etiologies (viral, bacterial, fungal) or medically induced (rhinitis medicamentosa). *Irreversible obstruction* can be from a number of anatomic issues such as upper lateral cartilage collapse, dorsal septal deflections, synechiae, and turbinate hypertrophy.

The rhinoplasty surgeon needs to carefully evaluate the relationship of the caudal and dorsal septum, as deviations in these segments may require more extensive correction to eliminate obstruction. Posterior deflections and turbinate hypertrophy of significant size can result in posterior nasal obstruction but are less common causes.

Incisions, Approaches, and Techniques

Incisions

1. Transfixion/hemitransfixion—between septum and medial crura of LLC. Provides access to the caudal septum bilaterally.
2. Intercartilaginous—between ULC and LLC
3. Marginal incision—at the inferior/caudal border of the LLC
4. Intracartilaginous incision—going through the LLC transnasally
5. Rim incision—nasal rim
6. Killian incision—unilateral septal incision
7. Columellar—inverted V placed at the narrowest portion of columella. Meets marginal incision at a right angle. Care must be taken to avoid placing below the medial crural footplates and to avoid injury to the underlying medial crura to avoid postoperative notching.

Approaches

1. Closed: delivery versus nondelivery
   (a) Nondelivery approaches are used for conservative tip refinement. Accessed from transcartilaginous or intercartilaginous incisions. Transcartilaginous approach is preferred due to its simplicity. Used when favorable anatomy, medium skin thickness, tip symmetry, minimal bulbosity.
   (b) Delivery approach is used for more significant tip deformities: performed through intercartilaginous and marginal incision to develop a chondrocutaneous flap. More exposure than nondelivery, but more aggressive.

Table 26.1 Advantages of endonasal rhinoplasty

Decreased need for surgical dissection	Shorter operative times
Decreased risk of scarring, unfavorable healing	Elimination of visible external scar
Reduced postoperative edema	Quicker return to normal appearance
Ability to make exacting changes in situ	

2. Open rhinoplasty: indicated for nasal tip asymmetry, anatomically complex (revision, trauma, cleft noses), significant projection/rotation abnormalities, severe bulbosity, teaching.

Endonasal rhinoplasty advantages see Table 26.1.

Major disadvantage is exposure and potential for asymmetric resection.

Advantages of Open Rhinoplasty

1. Direct exposure, inspection, and assessment of the osseocartilaginous framework
2. Precise modification and stabilization of the abnormality (tip and dorsum modification, graft placement, osteotomies)
3. Excellent tool for training purposes

Disadvantages of Open Rhinoplasty

1. Transcolumellar scar and columellar flap necrosis
2. Extensive dissection of skin off the osseocartilaginous framework with increased scarring
3. Increased operative time
4. Postoperative nasal tip edema and numbness

Techniques

The tripod concept, described by Anderson [5], provides an understanding of the dynamics of tip rhinoplasty. The anatomy of the two alar cartilages forms a functional tripod that provides tip support. Projection is preserved when major and minor tip support mechanisms are left largely. Control of projection and rotation are critical for successful rhinoplasty and a number of techniques may be utilized depending on the situation (see Tables 26.2 and 26.3). A more detailed discuss will follow.

Tip

The tip is considered one of the most challenging aspects of rhinoplasty. Unlike altering the dorsum, which is a 2D undertaking, the nasal tip is a 3D structure with complex anatomy that must be contoured appropriately for favorable light reflexes and shadowing [6]. Frequently, the patient presents with a desire to narrow the nasal tip. In the past, emphasis on tip reduction resulted in long-term sequelae of pinching and distortion. With increased understanding of these maneuvers, augmentation techniques have been implemented to provide stability against long-term forces of contraction.

Table 26.2 Techniques utilized to modify nasal tip projection

Methods to increase projection	Methods to decrease projection
Tip graft	High, partial, or full transfixion incision
Plumping graft	Lateral crural overlay (also increases rotation)
Premaxillary graft	Nasal spine reduction
Transdomal suture	Vertical dome division with excision of excess medial crura with suture reapproximation
Septocolumellar sutures (buried)	
Columellar strut (maintains projection)	
Caudal extension graft	
Illusion of projection with supratip break	
Lateral crural steal (also increases rotation)	

Table 26.3 Techniques utilized to modify nasal tip rotation

Increasing rotation	Decreased rotation
Lowering nasal dorsum	Transfixion incision
Shortening caudal septum	Shorten medial crura
Cephalic resection of lower lateral cartilage	Caudal extension graft
Vertical dome division	Double layer tip graft
Lateral crural overlay	Reconstruction of L-strut with extended spreader grafts
Plumping graft to nasolabial angle	

Base

When approaching the nasal tip, one must take into account a number of factors to ensure long-term stability. A *stable base* is critical for maintaining the desired projection, nasolabial angle, and alar–columellar relationship. A number of maneuvers are available to the rhinoplasty surgeon, which enable a firm foundation for the lower third of the nose. The *columellar strut graft* is a commonly used graft for maintenance of structural support, columellar shape, and projection of the nasal tip. It is typically 20 mm long, 2.5 mm wide, and 1.5 mm thick and is useful for those patients with short medial crura but an appropriate alar–columellar relationship. The surgeon must ensure placement is just below domes to avoid a distorted tip. In addition, preserving a small amount of soft tissue at the nasal spine helps prevent postoperative clicking or graft displacement with movement.

The *tongue-in-groove* technique is a useful maneuver in the patient with excessive columellar show and a prominent caudal septum [7]. The crura are fixated with a horizontal mattress suture to the caudal end of the septum allowing the surgeon to achieve precise rotation and projection depending upon placement. This technique also provides some length to the upper lip, which is advantageous for those patients with short upper lips. A tongue-in-groove set back may also allow for decrease in nasal tip projection. When there is deficiency of the caudal septum or if significant tip stability is needed, the *caudal septal extension graft* provides the strongest stable foundation nasal tip support. This graft can be placed in an end-to-end or end-to-side position, shaped depending upon the patient's need. This graft significantly strengthens the tip and is particularly useful for controlling projection, rotation, and the columellar-lobular angle.

Finally, when augmentation of the premaxilla is needed (e.g., cleft nasal deformity), a *premaxillary graft or alar base graft* consisting of bone, cartilage, fat, or alloplast material can be used.

Tip Refinement

Once the foundation is stabilized, the surgeon can focus on refinement of the tip. This typically requires a combination of reduction and augmentation techniques with the specific techniques dictated by the individual anatomy. The majority of primary rhinoplasty patients present with the complaint of too large a tip. This may be the result of tip bulbosity, broadness, bifidity, cephalic malpositioning, soft tissue fullness, or a combination of these factors. Traditional approaches have relied on excessive reduction of the alar cartilage, which results in long-term collapse with aesthetic and functional distortion of the tip. Cartilage sparring techniques have been introduced and have replaced a number of older techniques, improving long-term results. It is critical that the rhinoplasty surgeon be well versed in a variety of techniques to optimize patient outcomes. When dealing with a wide nasal tip, a number of techniques are available including excision, suturing, morselizing, and incision techniques.

Excision techniques can be classified as *complete strip and interrupted strip techniques*. The *interrupted strip technique*, popularized by Goldman, was introduced as a means of increasing tip projection and narrowing the nasal tip. It involved *vertical dome division* 2–3 mm from the dome with advancement and transdomal suturing of the medial cartilage strips. The lateral strips were left unattached, which weakened the tripod, and predisposed patients, especially those with thin skin, to a number of complications including alar notching, a tent-pole appearance, bossae, and a pinched tip deformity. Thus, many surgeons have abandoned this technique and if vertical lobule division is utilized for tip refinement, reconstruction of the lateral crura is performed for maintenance of a lateral crural stability. This interrupted strip method remains useful for those patients needing deprojection and increased rotation. In contrast to the Goldman method, which attempted to achieve increased projection, vertical lobule division combined with *lateral crural overlay* and suture fixation of the lateral crural segments has been utilized as a powerful technique for patients with severe under-rotation and over-projection [8].

The *complete strip method* remains a reliable technique for those patients requiring tip refinement from overdeveloped lower lateral cartilage [9]. When performing a cephalic trim, it is critical to preserve a minimum of *6–8 mm* of lateral crura to avoid pinching and lateral wall collapse. This can be performed in combination with the *lateral crural steal technique* to increase projection while narrowing the domes [10, 11]. Alternatively, a cartilage-sparing technique can be performed with regard to a limited cephalic trim, in which a conservative paradomal trim is completed at the site of the neodomes after a lateral crural steal maneuver. Paradomal trim allows for increased lateral crural conservation.

Suture techniques are utilized to complete the tip refinement and consist of a number of sutures, depending on the clinical situation [12]. *Domal creation sutures* (*transdomal sutures*) are useful when additional tip definition and narrowing is needed. These are horizontal mattress sutures through the domal segment at the notch that reduce the intradomal distance and increase tip projection. Care is taken for precise placement to avoid too sharp a tip. The surgeon must also be aware of the position of the lateral crus, as this suture can result in descent of the caudal margin of the lateral crus and concavity of this segment. If these remain unrecognized, a pinched tip and notching of the ala can result. *Alar rim grafts* are useful in this setting as they provide support to the alar margin and soften the transition from the tip complex to the alar lobule [13, 14]. They are typically 12–15 mm in length and 2–3 mm in length and placed in a precise subcutaneous pocket at the alar margin. When convexity of the lateral crura is an issue, *lateral crura convexity control sutures* (*Gruber sutures*) can be placed as a horizontal mattress suture at the site of convexity [15]. When these are ineffective, *lateral crural strut grafts* may be needed for optimal contouring [16, 17]. *Interdomal sutures* (*dome-*

binding sutures) are useful for controlling tip width at domes and infralobule. These are placed at angle of divergence 2–3 mm from the caudal edge. If placed too tight, a pointed tip appearance will result. Finally, in those patients with strong lateral crura who require additional refinement of the supratip after cephalic trim, a *lateral crural spanning suture/alar-spanning suture* can be placed to eliminate supratip dead space and refinement of the supratip [18]. It is a horizontal mattress suture placed cephalic to domes at mid-portion of the lateral crus incorporating the caudal septum (see Fig. 26.3).

In certain situations (revision noses, inadequate medial crura, thick-skinned patients), tip sutures are not able to achieve the desired definition and contour and must be combined with other grafts. *Onlay tip grafts* (*cap or Peck grafts*) are occasionally needed to camouflage irregularities or achieve increased projection and tip contour. These typically consist of crushed or morselized cartilage placed over the caudal margin of the domes to maintain tip aesthetics and to provide a smooth transition from the tip to the alar lobule. These grafts are contraindicated in thin-skinned patients as these grafts will become visible with time with skin contraction. *Shield grafts* (*aka Sheen graft*) are rarely useful in primary rhinoplasty and are most commonly used in the revision patient needing tip projection, improved contour of the infratip lobule, or derotation of an over-rotated nose [19]. The graft is positioned over the medial crura from the tip to the medial crural footplates. Again, this graft is best suited for thick-skinned patients as it has a tendency to become visible in thin-skinned patients or when not appropriately contoured.

Patients with cephalic malpositioning of the lateral crura require special consideration for tip refinement. The normal orientation of the lateral crura is 45° or more off-midline and toward the lateral canthi. Cephalic malposition is described as lateral crura 30° from midline and directed toward the medial canthi. Cephalic malpositioning can create a "parentheses" appearance on frontal view. This frequently leads to a deficiency in the alar lobule following cephalic trim if this deformity is not recognized and appropriate

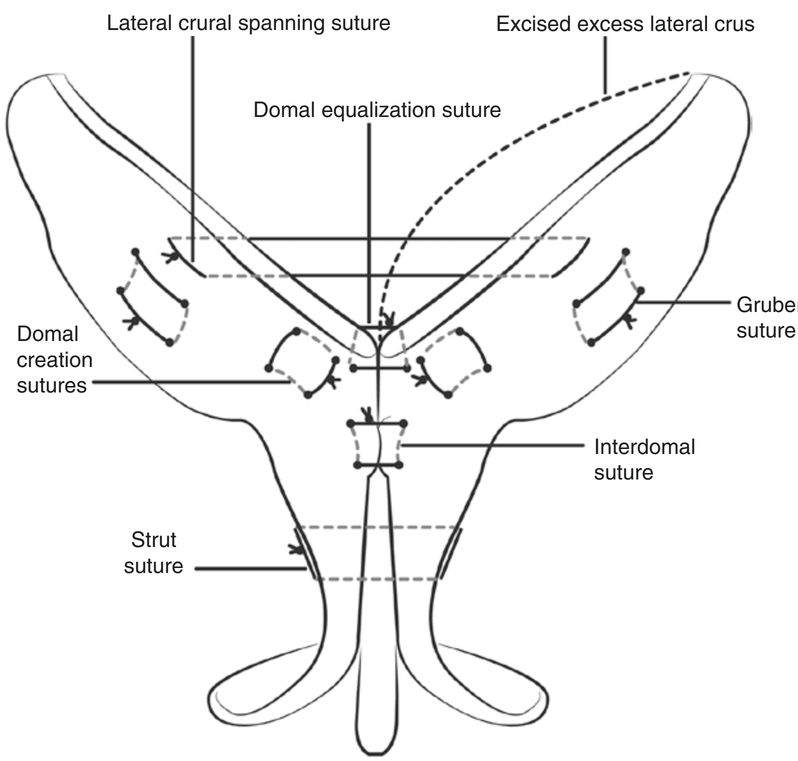


Fig. 26.3 Lower lateral cartilage suture techniques

maneuvers taken [20]. For excessive convexity of the lateral crura, a cephalic trim with lateral crura strut grafting and repositioning can produce a more favorable tip contour and prevent lateral wall collapse. If cephalic malpositioning is not contributing to tip bulbosity, repositioning can be avoided and the lateral wall can be supported with alar rim grafting.

Final tip definition should assess the width of the ala. The width of the ala should be equal to the intercanthal width and when excessive, reduction should be considered. Increased alar width of excessive show can be congenital for secondary to placement of alar rim grafts. *Weir excisions* are useful for the correction of alar flare. They are fusiform excisions of the lateral nasal base and must be performed carefully to avoid blunting of the nasofacial angle, nostril width is not affected by this maneuver. Nostril *sill excisions* are internal reductions, which reduce the nostril sill. They are trapezoidal excisions inside nasal base and have the added advantage of limited risk of alar-facial blunting (Fig. 26.4). This should be the final maneuver in rhinoplasty [21].

Middle Third

Correction of the middle vault is commonly required in primary and revision rhinoplasty. The two most common complaints that arise from the middle vault are nasal obstruction and dorsal over-projection. In many cases, the aesthetic result of the dorsum is the determining factor for a successful rhinoplasty. Therefore, the rhinoplasty surgeon must thoroughly evaluate this area for form and function in order to ensure a happy patient and surgeon.

Dorsal Reduction

When patients desire dorsal "hump" removal, a number of factors are considered. This must be individualized to the patient and one must take into account the patient's sex, ethnicity, anatomy, and aesthetic goals. The surgeon must first establish the desired nasion and tip position to determine the ideal dorsal height. Failure to establish these points can put the patient at risk of an over-resected dorsum and functional nasal obstruction. The brow-tip aesthetic lines are evaluated to determine if modification is needed.

Whenever reduction is indicated, it is important to perform *prior to septoplasty* in order to preserve an adequate 10 mm dorsal strut. Traditional reduction techniques were performed in a composite manner using sharp dissection with an osteotome to resect the osseocartilaginous hump en bloc. Unfortunately, this technique can lead to significant complications when poorly performed including inverted V deformities, keystone instability, and contour abnormalities. Contemporary techniques have been developed that focus on a component reduction of the osseocartilaginous hump that respects the anatomic, aesthetic, and functional relationship of the middle vault [22]. Component reduction is performed by sharply excising the upper lateral cartilages from the septum after the creation of submucoperichondrial tunnels. When dissecting the mid-vault, care must be taken to preserve the mucosal

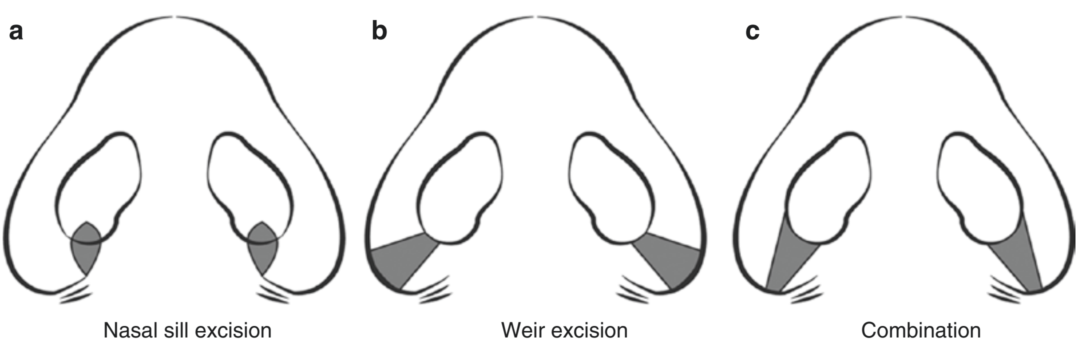


Fig. 26.4 Alar reduction techniques. (**a**–**c**) Different alar reduction techniques

attachment of the upper lateral cartilage to the septum to minimize the risk of scarring and resultant nasal valve stenosis. The cartilaginous and bony dorsum is then taken down incrementally with sharp dissection and an osteotome until the desired dorsal height is achieved.

Spreader grafts are routinely placed in primary and revision rhinoplasty to provide functional and aesthetic correction. Spreader grafts restore the brow-tip aesthetic line, correct midvault asymmetry, prevent inverted V deformities, increase the cross-sectional diameter of the internal nasal valve, and provide structural support to the L-strut. These grafts are frequently harvested from septal cartilage and are 1.5–2.5 cm in length, 1.5 mm in height, with the width dependent upon the amount of midvault correction needed. *Autospreaders* (*turn-in flaps*) can be used in select patients undergoing a significant hump reduction with long upper lateral cartilages [23]. Grafts are secured to the septum and upper lateral cartilage using horizontal mattress suture fixation to complete the reconstitution of the midvault. The "*butterfly*" *conchal cartilage graft* has also been described, which takes advantage of the native curvature of the conchal cartilage to increase the cross-sectional area of the area [24]. It has been shown to be an effective treatment of internal nasal valve collapse without compromising aesthetics in the properly chosen patient. The concern with this graft is that it can become noticeable in the supratip region because of excessive bulk or the cephalic edge showing. Unfortunately, the learning curve is steep for those surgeons not trained in this technique.

For those patients with minimal valve obstruction or who are not candidates for spreader graft placement, an *upper lateral cartilage spanning suture* (Fig. 26.5) has been described to increase the cross-sectional area of the internal nasal valve [25].

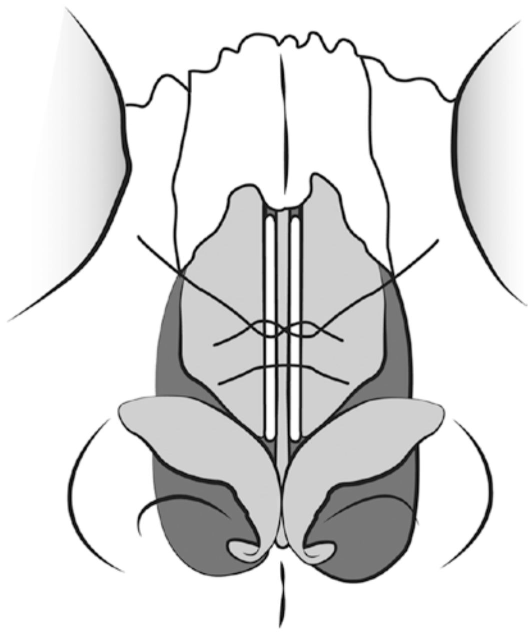


Fig. 26.5 Upper lateral cartilage spanning suture used for improving cross-sectional diameter of the internal nasal valve

Dorsal Augmentation

Dorsal augmentation can be indicated for functional and aesthetic reasons and can be achieved with a variety of grafting material. It is not uncommon for the Asian or platyrrhine rhinoplasty patient to request dorsal augmentation for a more "western" nose. This patient population typically has thicker skin with weak cartilage support. Alloplast grafts are popular in the Asian culture for dorsal augmentation. Meticulous placement is indicated for alloplast implants and special care must be taken to ensure there is no tension over the implant or contact with the dermis. Despite care placement, these grafts remain at a higher risk of infection and long-term extrusion.

For mild dorsal irregularities or revision cases, autogenous grafts are favored. *Fascia* can be used to provide a smooth dorsum in those thin-skinned patients at risk of minor contour abnormalities. When mild to moderate correction is needed in a patient with normal or thicker skin, dorsal onlay grafts or diced cartilage/fascia grafts can be used for contouring. *Dorsal onlay grafts* are useful for correction of minor dorsal deformities and are typically morselized to minimize irregularity. *Diced cartilage/fascia grafts* (aka Turkish delight) are cylinder-shaped graft consisting of diced cartilage wrapped in temporalis fascia useful for correction of secondary dorsal depressions, primary excessive hump reduction, saddle nose deformities, or ethnic rhinoplasty [26–28].

When structural support is needed, a more robust grafting material must be obtained and frequently requires the harvesting of osseocartilaginous rib, calvarial bone, or irradiated homograft costal cartilage. Autogenous costal cartilage is the preferred source due to its availability, ease of carving, and ease of fixation. It provides excellent structural and functional support to the middle vault.

Bony Vault

Management of the bony vault is critical for a successful surgical outcome. Many patients present with complaints of a dorsal hump or a wide nose. A "balanced approach" is recommended to maintain a strong dorsal profile and a natural result. It is critical to *establish the ideal nasion point*, which will influence the nasofacial angle and determine if radix reduction or augmentation is needed. A shallow nasofrontal angle gives the appearance of a longer nose, while an acute/deep nasofrontal angle contributes to a short nose. The nasion can then be compared to the tip to establish the ideal dorsal height.

Reduction of the radix can be challenging, as an over-projected radix can be the result of soft tissue (adipose, procerus muscle) or bone. Soft tissue reduction can be managed with excision or in extreme cases an endoscopic forehead lift with central release. Bony reduction is achieved with rasps, osteotomes, or powered reduction.

Augmentation is achieved with a *radix graft* consisting of fascia, or diced cartilage in fascia [29]. It can be a single or layered graft. Solid cartilage grafts are not recommended due to the tendency to become visible with time. Repositions radix more cephalic and anterior. Decreases the amount of dorsal reduction required. Can soften an "angry" appearance.

Osteotomies

Osteotomies are frequently required for those undergoing aesthetic rhinoplasty and have been traditionally classified according to their location. The bony nasal base should approximate 80% of the alar width. Techniques vary among surgeons for performing osteotomies and include percutaneous, endonasal, and intraoral techniques (Fig. 26.6).

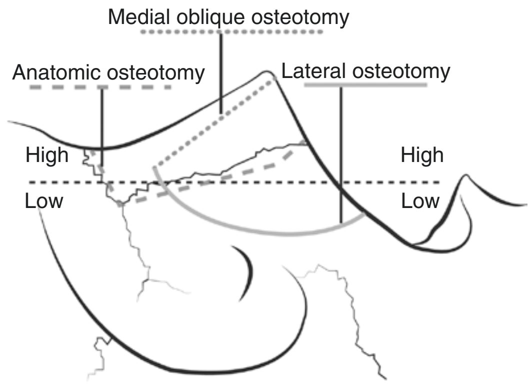


Fig. 26.6 Osteotomies

Percutaneous osteotomies can be performed along the traditional location of osteotomies or along the junction of the nasal bones with the frontal bone and the maxilla ("anatomic osteotomies") [30, 31]. Those in favor of the percutaneous approach state the advantage of periosteal preservation, reduced lateral wall collapse, less airway compromise, and reduced bleeding, ecchymosis, and edema. Opponents site the potential risk of a visible external scar and an irregular osteotomy due to the postage stamp technique.

Indications for osteotomies are: (1) to close an open roof deformity, (2) narrow the bony nasal base, and (3) to straighten a deviated nose.

Lateral Osteotomies utilizing a *low–high osteotomy* is traditionally performed when a *greenstick* fracture of the lateral nasal bone is indicated for narrowing. It begins at the pyriform aperture *above the attachment of the inferior turbinate* to prevent airway compromise (preserving Webster's triangle). The osteotomy is carried cephalically along the nasal process of the maxilla and ends at the nasal base at the level of the medial canthus.

When complete mobilization of the nasal bones is required, a *low–low osteotomy* can be considered. The lateral osteotomies can be connected to a *medial–oblique* or *transverse osteot-*

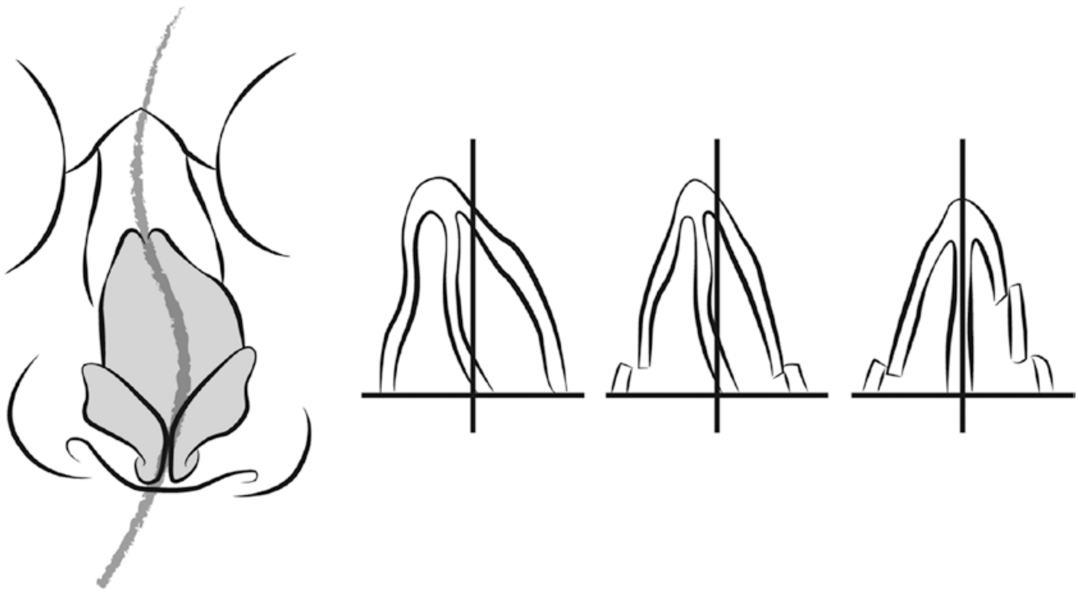


Fig. 26.7 Utilization of intermediate osteotomies to correct a crooked nose

omy. Medial–oblique osteotomies are useful for those patients with a wide bony dorsum to achieve full mobilization and sufficient narrowing. It is performed using a curved osteotome angled laterally 15–20° off midline.

Intermediate osteotomies (*double osteotomies*) (Fig. 26.7) are useful for narrow extremely wide noses (bilateral intermediate and bilateral lateral) or to correct crooked noses (unilateral intermediate on the longer nasal bone and bilateral lateral ± transverse) such as in the setting of significant nasal bone asymmetry, convexity, or trauma. This consists of an osteotomy along the inferior nasal bone parallel and combined with a low–low osteotomy. It is performed prior to the inferior osteotomy. *Anatomic osteotomies* are performed percutaneously along the nasal bone suture lines. This allows for precise manipulation of the nasal bones for correction of minor bony nasal vault asymmetry in a stable, controlled manner [30].

Finally, osteotome size is important when performing lateral osteotomies. Becker studied nasal bone thickness along osteotomy lines and discovered that the bone thickness is 2.4–2.29 mm thick in males and females, respectively [32]. When a 2.5-mm osteotome was used, mucosal tearing occurred in 4% of patients. In contrast, the mucosal tearing incidence increased from 34% to 95% when using 3.0 and 4.0 mm osteotomes, respectively. Therefore, to minimize ecchymosis and edema some surgeons recommend *osteotomes no larger than 3–4 mm in size*.

Surgical correction of the bony vault can be challenging and when performed incorrectly, a number of complications can result including rocker deformities, collapsed nasal side walls, open roof deformity, irregularities, step-offs, persistently wide bony vault, and bony dorsum over-resection.

Septum

The cosmetic and reconstructive rhinoplasty surgeon must be prepared and trained to manage septal deformities that arise among rhinoplasty patients. These may be congenital or posttraumatic and can be challenging to even the most experienced rhinoplasty surgeon. From an aesthetic perspective, septal deflections can result in a twisted nasal appearance, under-projection, columellar deformity, and tip ptosis. From a functional perspective, deflections can be present anywhere along the bony or cartilaginous septum, which can result in static or dynamic airway obstruction of the internal and external nasal valves. If structural integrity of the dorsal and caudal L-strut is disrupted or weakened, a number of undesirable complications will arise including loss of tip support, saddle nose deformities, and valve collapse. Therefore, a 1 cm L-strut must be left intact to minimize postoperative morbidity.

Caudal Septal Deflection

The rhinoplasty surgeon must be component and comfortable treating the caudal septum due to the critical role the caudal septum plays in form and function. When left untreated, columellar deformities, tip ptosis, twisted tip deformities, and external valve collapse are potential sequelae.

The *swinging door technique* was first described in the 1920s as a way to correct the caudal–septal deflection [33]. It involves release of the inferior attachments of the septum off the maxillary crest with or without excision of a wedge of inferior cartilage and repositioning the septum on the contralateral side. It is secured in place with a nonabsorbable suture through the anterior nasal spine. Some authors use *extended bilateral spreader grafts* placed on either side of septum to buttress and stabilize the septum. Other techniques include *scoring* or *morselizing* the concave side of the septum and attaching it to an ethmoid bone or cartilage *batten grafts* to maintain a straight position. The surgeon must take care not to weaken the caudal support with this maneuver by excessive scoring. For significant deformities, an *extracorporeal septoplasty* may be indicated to remove and reconstruct the offending cartilage [34]. It involves complete dissection of the cartilaginous septum with disarticulation from the perpendicular plate of the ethmoid, vomer, and maxillary crest. The septum is then reconstructed using various techniques and replaced into position. When performed by an experienced surgeon, results are reliable. Complications related to this technique include dorsal irregularities, keystone collapse, and the potential for saddling. A new modification of this technique includes preserving a portion of the dorsal cartilage at the keystone, which facilitates easier reconstruction.

Recently, *polydioxanone* (*PDS*)-perforated plates have been incorporated into septal techniques [35]. The original use was described in extracorporeal septoplasty but has been expanded for use for a number of applications. The PDS plate value is providing structural support in situations when the native cartilage is unable to provide appropriate form for function. In the case of extracorporeal septoplasty, the cartilage pieces can be secured to the PDS plate, which provides a scaffold to increase stability and provide a matrix for chondrocyte regeneration. Disadvantages to PDS are that it is resorbable, it can result in prolong edema, and it can increase the risk of infection. PDS plates are available in different thicknesses, with the perforated 0.15 mm plate being the most popular for rhinoplasty.

Finally, in the case of significant cartilage loss, the surgeon must seek material from elsewhere and frequently has to resort to costal cartilage.

Septal Perforation

Septal perforation can be the result of a number of conditions including iatrogenic, traumatic, infectious, autoimmune, inhalant drugs, and malignancy [36]. Therefore, it is prudent for the rhinoplasty surgeon to determine the etiology of the perforation prior to surgical intervention. When a biopsy is indicated, it is important to avoid biopsy from the superior aspect of the perforation to avoid enlarging the defect.

A number of complaints can be associated with anterior perforations including pain, crusting, epistaxis, obstruction, whistling, and saddling. When asymptomatic, repair is rarely indicated and treatment consists of medical management with humidification and antibiotic ointment to prevent crust buildup and inflammation. Temporary closure can also be achieved with a prosthetic *nasal button*. Unfortunately, patient compliance can be an issue and requires adherence to nasal hygiene maintenance. Complications of button use include discomfort, crusting, epistaxis, and enlargement of the perforation.

Surgical correction is indicated for failed medical management with significant symptoms [37]. *Bilateral mucoperichondrial flaps with an interposition graft* are the preferred flap choice for repair. Smaller perforations have a higher success rate for closure. Interpositional grafts used have included acellular dermal matrix and fascia lata. The endonasal approach can be utilized for anterior perforations less than 5 mm in size. For perforations 5 mm to 2 cm in size, the open approach is preferred as it provides unparalleled access for repair. For larger (>2 cm) perforations, other flaps must be utilized including: inferior turbinate flaps, sublabial mucosal flaps, and radial forearm free flaps. Whatever technique is chosen, a *tension-free closure* is critical to minimize a persistent perforation.

Special Circumstances

The crooked nose If minor asymmetry with or without a history of trauma, careful facial analysis is needed to identify potential facial asymmetries (e.g., midface flattening, pyriform aperture displacement, facial width asymmetry). When severe asymmetry is present, it frequently is the result of trauma. It can be managed with (1) camouflaging, (2) deconstruction/reconstruction, or (3) combination of techniques [38]. True correction of the crooked nose requires complete dissection and correction of the intrinsic deformities with reconstruction. Correcting the septum is the initial step (and can require extracorporeal techniques), followed by asymmetric spreaders for midvault and asymmetric osteotomies.

Lengthening the short nose A number of factors result in a shortened nose, or the appearance of a short nose. These include concavity of the dorsum, tip projection to nasal length ratio > 0.6:1, low, deep nasion, over-projected tip, obtuse nasolabial angle. *Treatment techniques* depend upon the causative factor and include *complete release of the mucoperichondrium*, *caudal septal extension grafts*, *tip grafts*, *radix grafting*, *interposition graft between upper and lower lateral cartilage*, *extended spreader grafting combined with columellar strut* [39].

The tension nose tip over-projection secondary to overdeveloped, long caudal septum. Typically associated with weak alar cartilage inferiorly displaced. Treatment involves dorsal septal reduction for deprojection (consider autospreader grafts) followed by stabilization with struts/caudal septal extension grafts for support [40]. Tongue-in-groove can also be a helpful technique. Reduction of the nose often leads to alar base flairing/widening that requires wedge excision of ala for symmetry. Can have internal and external nasal valve collapse.

Pediatric rhinoplasty deferred until age *15–16* if possible to limit growth disturbance of quandrangular cartilage. Exceptions to this rule include cleft rhinoplasty, which is discussed elsewhere.

Graft Materials in Rhinoplasty

Various materials are available to the rhinoplasty surgeon. These are categorized as autogenous, homogenous, and alloplastic. Autogenous grafting is the material of choice when available and can include cartilage, bone, and fascia (perichondrium, temporalis fascia).

Autogenous

Material of choice due to lower rate of tissue reaction, resorption, and infection [41].

Septal cartilage preferred as first line due to proximity, availability, firm, flexible, provides structural support. Can be limited in revision cases.

Conchal cartilage used in revision cases or when septal cartilage is limited. More malleable and curved limiting broad application of this graft. Harvested from preauricular or postauricular approach. *Composite grafts* are harvested from auricle and consist of skin and cartilage. These grafts are particularly useful for correction of significant alar retraction or vestibular stenosis [42]. Graft survival is maximized when the graft radix is no greater than 10 mm.

Costochondral donor site of choice when sufficient cartilage is needed for traumatic or revision cases. Solid, carvable, typically harvested from 6th to 7th rib [43]. Disadvantage: potential for warping, donor site discomfort, separate field, pneumothorax, and scarring. Pneumothorax risk can be minimized by careful dissection with preservation of the posterior perichondrium. Age should be taken into consideration when evaluating for costal cartilage, as increased age may predispose patient to increased ossification and less malleability for graft use. Needles can be used to evaluate degree of ossification before the skin incision is made, or even used in the office under local.

Bone-calvarial, rib, iliac crest, ethmoid. Used for upper one-third reconstruction but less favored due to lack of flexibility, fixation techniques required, and higher metabolic demand. Calvarial is the preferred choice as it is of membranous origin, therefore resists absorption unlike the iliac crest which is of endochondral origin [44]. Calvarial bone harvested from parietal bone above the temporal line [45]. Risks include sagittal sinus injury, csf leak, epidural/subdural hematoma, intercranial injury.

Fascia-perichondrium and temporalis fascia. Good for softening nasal contours and correction of minor irregularities. Little to no donor site morbidity. Can have potential for prolonged edema after placement.

Homologous

Derived from human cadavers. Potential risk of transmission of human pathogens.

Irradiated homograft costal cartilage obtained from young donors (no calcification) and screened for systemic disease, infection, cancer, drug abuse, syphilis, HIV, hepatitis and exposed to gamma radiation. Advantages: readily available, easily carve, low infection/extrusion rate, large quantity, no donor site. Disadvantages: potential for warping/absorption, infection [46, 47].

Acellularized dermal matrix (Alloderm) useful for camouflage or graft coverage [48]. Risk of absorption in first year.

Alloplast

The ideal synthetic should be noninflammatory, noncarcinogenic, nonallergic, have an adaptable shape, readily available, able to integrate into the host tissue, sterilizable, and resistant to mechanical strain. Unfortunately, no material has yet to meet all these requirements. Therefore, the patients own cartilage remains the best choice for augmentation. Unfortunately, situations arise when the patients' own tissue may not be available for reconstruction and alternative grafting material is needed. A number of alloplast materials have been used [49]. Popular due to ready availability, endless supply, adaptable shape, lack of donor morbidity. Disadvantage is that all alloplasts cause an inflammatory response upon placement and have risk of complication. The presence and size of pores influence the amount of tissue ingrowth and infection risk. No definitive evidence has proven a reduced risk of extrusion with presoaking the implant in antibacterial solution.

Expanded-porus polytetraflouoethylene (Gortex) has been used for >20 years in various surgical fields. Smaller pore size so there is less tissue in-growth (easier to remove). Higher infection risk with *revision cases*, *septal perforations*, *or when in contact with dermis* [50].

Porus high-density polyethylene (Medpor) larger pore size allows for more tissue ingrowth (harder to remove) and strength. *Increased infection risk* until ingrowth complete. So should be used with caution especially in revision cases, incomplete soft tissue covering, or thin skin [51].

Polydioxanone (PDS) available as perforated or unperforated plate. Recommend perforated plate to allow for blood supply preservation [52]. Degraded by hydrolysis by 25 weeks. Provides structural support to weakened cartilage and acts as scaffold/guide for regenerating chondrocytes.

Silicone used primarily in Asian population for dorsal augmentation. Fibrous capsule forms without tissue in-growth. Persistent risk of infection. Extrusion at higher risk when tension is over the implant. Need to place subperiosteal. When infection and extrusion, it is preferable to reconstruct immediately to limit tissue contracture [53].

Revision Rhinoplasty and Treatment of Rhinoplasty Complications

Knowledge of common rhinoplasty complications and their etiology is fundamental for all rhinoplasty surgeons. Although adherence to rhinoplasty principles will help prevent a number of these from happening, complication rates remain relatively stable in the literature with reported incidences of 8–15%. It is the surgeon's responsibility to have a detailed knowledge of the anatomic variants encountered, knowledge of surgical alternatives, and the result of healing over time to optimize patient outcomes [54]. Below is a list of common complications and their prevention/management strategies.

1. *Pollybeak deformity*—over-projection of the lower nasal dorsum. *Causes* include loss of tip support, inadequate cartilaginous hump removal at anterior septal angle, supratip scar formation. Treatment is directed to the cause and includes adequate lowering of the cartilaginous component, surgical defatting of supratip nasal SMAS, reestablishing appropriate tip support, or interval steroid injections with skin taping. The best treatment is avoidance.
2. *Saddle-nose deformity*—characterized by a loss of nasal dorsal height with nasal vault collapse. Most saddle-nose deformities are acquired resulting from an over-resected dorsum or septal L-strut. Other causes include Wegener's granulomatosis, relapsing polychondritis, cocaine abuse, and chronic nasal decongestants. For patients with no nasal airway obstruction and minor-to-moderate nasal dorsal saddling, onlay grafting techniques can be used. Moderate-to-severe saddle nose requires structural reconstruction of the dorsum, middle vault, and tip support with costal cartilage or other alloplast material. Contraindications include patients with malignancy, poorly controlled chronic medical conditions, active autoimmune disease, continued intranasal drug use, and patients who are poor candidates for rhinoplasty in general.
3. *Inverted V deformity*—inferiomedial collapse of the upper lateral cartilage secondary to inadequate support after dorsal hump reduction. Appearance is washed out with prominence of the caudal end of the nasal bones. Usually associated with internal nasal valve collapse. Corrected with spreader graft placement. Patients at risk have *short nasal bones*, *long upper lateral cartilage, and large dorsal hump reduction*.
4. *Over-resection of lateral crura*—common problem seen in revision rhinoplasty, results in pinched tip, supra-alar collapse, tip asymmetry, bossae, alar retraction. Prevention includes adequate preservation of lateral crura (6–8 mm) when performing cephalic trim. *Alar batten grafts* are the first-line treatment for supra-alar collapse with alar retraction [55]. These curvilinear grafts are placed at the precise point of lateral wall collapse and extend to the pyriform for lateral support. Special consideration must be made for those patients who are thin skinned due to the potential for fullness or a bulbous appearance following placement.
5. *Bossae deformity*—knuckling of the lower lateral cartilage in the dome resulting in visible or palpable nasal tip asymmetry. Predisposed with the *triad of thin skin*, *strong alar cartilage, and tip bifidity* after aggres-

sive cephalic trim in a young patient [56]. Treatment involves trimming or resection of knuckled cartilage, transdomal suturing, onlay camouflaging grafts, or reconstruction of the lateral crura.

6. *Alar retraction*—overzealous resection of lower lateral crus or in cephalic trim of cephalically oriented lower lateral cartilage. Recommended to preserve at least 7 mm of cartilage. Treated with alar rim grafting, lateral crural strut grafts, auricular composite grafts, or alar batten grafts [57].
7. *Hanging columella*—usually due to failed correction at original surgery. Correction involves resection of excessive caudal membraneous or caudal septum or tongue-in-groove technique [58].
8. *Retracted columella*—consequence of over-resection of caudal septum. Treatment involves columellar strut, plumping grafts, or caudal septal extension grafts.
9. *Vestibular stenosis*—due to excessive scarring in vestibule from collapsed cartilage, skin excision, or excision of lesions. Correction involves composite auricular grafting, z-plasty, injection with kenalog/5-fu, or stents.
10. *Open roof deformity*—results from inadequate lateral osteotomies or failure to perform osteotomies after dorsal reduction. Can give appearance of surface indentation with longitudinal shadowing. Corrected with spreader grafting and lateral osteotomies with infracture.
11. *Rocker deformity*—results from carrying the osteotomy too cephalic into the frontal bone. Results in lateral displacement of the superior segment with infracture. Corrected with transverse percutaneous osteotomy.

Questions

1. Cephalic malpositioned lower lateral cartilages contribute to all of the following deformities, except:
   (a) Parentheses-shaped tip deformity
   (b) Bulbous nasal tip
   (c) Excess fullness in the supratip
   (d) Nasal tip bossae
2. All of the following are techniques to address alar retraction in revision rhinoplasty, except:
   (a) Alar rim graft
   (b) Lateral crura repositioning
   (c) Composite auricular cartilage graft
   (d) V-Y columellar advancement
3. Flaring Sutures are designed to affect the nasal vault by which of the following methods?
   (a) Sutures placed in a horizontal mattress fashion across the caudal aspect of the of the upper lateral cartilage to increase the angle of the ULC in relation to the dorsal septum
   (b) Sutures placed between the lower lateral cartilages to bring the lower lateral cartilages together and improve their alignment
   (c) Sutures placed between the tip defining points of the lower lateral cartilages to widen the angle between the medial and lateral crura
   (d) Sutures placed in horizontal mattress fashion on each lower lateral cartilage to flatten then lower lateral cartilages
4. Performing lateral crural steal technique (LCS) as compared to lateral crural overlay (LCO) during an open septorhinoplasty would cause the following changes to tip projection and rotation?
   (a) LCS increase tip rotation and projection, LCO increase tip rotation and decrease tip projection
   (b) LCS decrease tip rotation and increase tip projection, LCO decrease tip rotation and decrease tip projection
   (c) LCS decrease tip rotation and projection, LCO increase tip rotation and projection
   (d) LCS increase tip rotation and decrease projection, LCO decrease tip rotation and increase projection
5. Which of the following best describes the arterial anatomy of the external nose?
   (a) External carotid artery via the facial and infraorbital branches and the internal carotid via the ethmoid and branches of the ophthalmic artery

(b) External carotid artery via the facial artery and the internal carotid artery via the infraorbital branches and the ethmoid branch of the ophthalmic artery
(c) External carotid artery via the lingual branch and infraorbital branches and the internal carotid via the infraorbital branches
(d) External carotid artery via the facial branch and the internal carotid artery via the infraorbital branches

6. A patient presents to clinic with the complaint of a big nose and desires rhinoplasty for a smaller, more well-defined tip. On exam she has noted tip bulbosity. Which of the following anatomic variations does NOT contribute to the appearance of tip bulbosity?
   (a) Thick skin soft tissue envelope
   (b) A divergent interdomal angle, wide domal angle
   (c) Cephalic malposition of the lateral crura
   (d) Short medial crura
7. The lower lateral cartilages contribute to how many major tip support mechanisms?
   (a) 0
   (b) 1
   (c) 2
   (d) 3
8. Surgeons can increase tip rotation by all of the following methods, except:
   (a) Cephalic trim of the lower lateral cartilages
   (b) Lateral crural overlay
   (c) Dorsal onlay graft
   (d) Vertical dome division
9. A patient presents to clinic for revision rhinoplasty consult 3 years after her previous primary rhinoplasty. She initially had dorsal hump reduction, spreader graft placement, and medial and lateral osteotomies. Her complaint today is that her nasal tip appears split with bumps on either side of the tip. All of the following factors predisposed this patient to this complication, except:
   (a) Thin skin
   (b) Strong alar cartilage
   (c) Tip bifidity
   (d) Placement of tip onlay graft
10. A patient presents with frequent nasal crusting and nasal obstruction. He has a distant history of cocaine abuse, but has not used this drug for 15 years. On examination, a 1 × 1 cm septal perforation is noted on the inferior aspect of his septum. Biopsies are negative for malignancy and Wegener's granulomatosis. He is self-admittedly noncompliant with medications. What is the best treatment plan?
    (a) Nasal irrigations, antibiotic ointment and frequent follow-up
    (b) Septal button placement
    (c) Elevation of bilateral mucoperichondrial flaps with placement of acellular dermis interposition graft through an endonasal approach
    (d) Elevation of bilateral mucoperichondrial flaps with placement of acellular dermis interposition graft through an open approach
    (e) Elevation of inferior turbinate flap in combination with an open approach
11. The correct lobule to tip ratio on lateral view is:
    (a) 2:1
    (b) 1:2
    (c) 1:1
    (d) 3:1
12. Which of the following is true regarding alar base reduction?
    (a) Weir incisions reduce the nostril circumference and reduce alar flare
    (b) Nasal sill incisions reduce the nostril circumference and reduce alar flare
    (c) Weir incisions reduce alar flare but do not change the nostril circumference
    (d) Nasal sill incisions reduce alar flare but do not change the nostril circumference
13. A 65-year-old female presents to clinic for evaluation for a rhinoplasty. Her biggest complaint is that her nose "looks like a witch's nose" and she has noticed more droop over the past few years. On examination you do notice decreased columellar-labial angle and ptosis of the nasal tip. All of the following maneuvers will improve tip support, except:

(a) Caudal septal extension graft
(b) Tongue-in-groove technique suturing the medial crural footplates to the caudal septum
(c) Lateral crural strut graft
(d) Placement of spreader grafts
(e) Reattaching the caudal septum to the nasal spine

14. In a patient that you wish to increase tip support and decrease columellar show, which maneuver would address both of these concerns?
    (a) Columellar strut graft
    (b) Spreader graft
    (c) Tongue–in-groove
    (d) Extended spreader graft
15. Which description of appropriate tip projection is correct?
    (a) Crumley-Lanzer method: the dorsum of the nose is the hypotenuse of a 3-4-5 right triangle
    (b) Simon method: the ratio of subnasale to tip as compared to the lateral crura is 1:1
    (c) Goode method: The ratio of the length of the nose to the projection is 0.45
    (d) Simon method: the ratio of the columella to vermillion compared to the subnasale to tip is 1:2
16. When performing lateral osteotomies, the superior exent is typically on the horizontal plane with the medial canthus. If one carried this osteotomy higher, the patient would be at risk for what complication?
    (a) Inverted V deformity
    (b) Saddle nose deformity
    (c) Rocker deformity
    (d) Polly beak deformity
17. All of the following maneuvers can improve airflow through the internal nasal valve, except:
    (a) Septoplasty
    (b) Inferior turbinoplasty
    (c) Alar strut graft
    (d) Spreader graft placement
    (e) Upper lateral cartilage flaring stitch
18. Chin position should be 2 mm from the Gonzales–Ulloa line for ideal aesthetics. This line is best defined as:
    (a) Line perpendicular to the Frankfort horizontal dropped straight down from the Nasion.
    (b) Line from the tip defining points of the nose to the supraorbital rim.
    (c) Vertical line from the medial canthus to the lateral margin of the ala.
19. When discussing the alloplastic materials polytetraflouethylene (Gortex) and porous polyethylene (Medpor), which of the following statements are true?
    (a) Medpor has smaller pore size, leading to less tissue in-growth, making them easier to remove.
    (b) Gortex has large pore size, leading to greater tissue in-growth and making them more difficult to remove.
    (c) Medpor has greater pore size, leading to greater strength and tissue in-growth.
    (d) Gortex is a new material in rhinoplasty having been used only for the last 10 years.
20. Cephalic trim is a complete strip technique that helps to refine the tip in patients with overdeveloped lower lateral cartilages. It is typically recommended to leave between 6 and 8 mm of residual width on the lower lateral cartilages. Which of the following is not a complication of over-resection of the lower lateral cartilages?
    (a) Internal nasal valve narrowing
    (b) Pinched tip
    (c) Bossae formation
    (d) Alar retraction
    (e) External valve collapse
21. Which of the following will be better addressed by an open rhinoplasty approach as compared to a closed rhinoplasty approach?
    (a) Cleft lip nasal deformity
    (b) Tip deformities
    (c) Tip bulbosity
    (d) Need for spreader grafts
    (e) Asymmetry of the bony vault requiring osteotomies
22. Which of the following statements is most accurate?
    (a) Onlay tip grafts: crushed cartilage, extending from the cranial margin of the

tip defining point, covering the tip defining points, providing increased projection
   (b) Shield grafts: positioned over the medial crura from the tip to the medial crural footplates, increasing tip projection
   (c) Diced cartilage/fascia grafts: diced cartilage wrapped in tubed fascia placed on the dorsum of the nose to provide structural support to the nose
   (d) Butterfly graft: extends over the caudal margin of the lower lateral cartilages to open the domal angle widening the external nasal valve

23. Advantages of percutaneous lateral osteotomies include all of the following, except:
   (a) Less airway compromise
   (b) Preserved periosteum
   (c) Smooth osteotomy line
   (d) Decreased bleeding
   (e) Decreased edema
24. Which of the following is a method to increase tip projection?
   (a) Premaxillary graft
   (b) Vertical dome division with excision of excess and suture reapproximating
   (c) Lateral crural overlay
   (d) Placement of extended spreader grafts
25. To accomplish an endonasal rhinoplasty with delivery approach one needs to perform which combination of incisions?
   (a) Transcartilaginous and intercartilaginous incisions
   (b) Mid-columellar and marginal
   (c) Intercartilaginous and hemitransfixion incisions
   (d) Intercartilaginous and marginal incisions
26. All of the following are useful in the correction of the bulbous tip, except:
   (a) Interdomal suture
   (b) Lateral crural overlay
   (c) Cephalic trim
   (d) Lateral crural repositioning
27. A patient presents with excessive columellar show. Describe the potential causes and the maneuvers for correction.
28. Correction of a short nose can include all of the following techniques, except:
   (a) Plumping graft to nasolabial angle
   (b) Radix graft
   (c) Dorsal augmentation
   (d) Caudal septal extension graft
   (e) Tip grafting
29. A patient presents to the clinic with the complaint of bilateral nasal obstruction. The patient states this started 3 months after her rhinoplasty and has gradually become worse with time. Upon further questioning, the patient states she had been unhappy with the appearance of her nose and underwent surgery to "remove the hump" and to refine the tip. On examination, you note the patient has a somewhat washed-out appearance with slight prominence of the caudal ends of the nasal bones. The tip is refined and appropriately projected. Improvement in airflow is noted with the Cottle maneuver bilaterally. All of the following preoperative findings would alert the operating surgeon that the patient is at risk for this complication, except:
   (a) Long upper lateral cartilage
   (b) Prominent dorsal hump
   (c) Thin skin
   (d) Short nasal bones
30. Describe the characteristics of the platyrrhine nose and the impact of nasal maneuvers for rhinoplasty.
31. A patient comes to your clinic with a history of redness, drainage, and swelling of the nasal tip for the last several weeks, which has not responded to topical antibiotic ointment. The patient has no recent trauma, infection, or procedures. Her past surgical history is significant for a rhinoplasty 5 years ago in Korea for dorsal augmentation. You strongly suspect an alloplast infection. Describe the most appropriate course of management of this patient. Describe the risk and benefits associated with surgical intervention with immediate versus delayed reconstruction.
32. A 20-year-old patient comes in for rhinoplasty evaluation complaining that her nose was too large. She was wishing for a smaller,

cuter nose. The patient is 5′6, 120 lbs. On examination, the patient has a mild dorsal hump, her nasal rotation is 90°, and she appears over-projected. She has normal skin thickness and strong tip support. It is otherwise unremarkable in appearance. Further facial analysis reveals that her chin lies 3 mm posterior to a vertical line drawn from the glabella to the pogonion and the patient has Class I occlusion. You are considering all of the following techniques, except:
   (a) Dorsal hump reduction
   (b) Columellar strut
   (c) Chin augmentation
   (d) Sagittal split osteotomy with sliding genioplasty
33. Calvarial bone graft is harvested from which site?
   (a) Temporal bone
   (b) Occipital bone
   (c) Parietal bone
   (d) Frontal bone
34. After performing lateral and medial osteotomies, the rhinoplasty surgeon attempts superior infracture of the bony segment and notes lateral displacement of the caudal segment. Correction of this deformity is achieved by:
   (a) Internal and external nasal splinting
   (b) Intermediate osteotomy
   (c) Percutaneous lateral osteotomy
   (d) Percutaneous transverse osteotomy
35. All of the following techniques are potentially useful in straightening a crooked nose, except:
   (a) Intermediate osteotomies
   (b) Spreader graft placement
   (c) Incomplete elevation of mucoperichondrial flaps
   (d) Swinging door technique
   (e) Scoring of cartilage
   (f) Extracorporeal septoplasty
36. Correction of a pollybeak deformity may include all of the following, except:
   (a) Columellar strut
   (b) Kenalog injection to the supratip
   (c) Resection of anterior septal angle
   (d) Dorsal onlay graft

Answers

1. Nasal tip bossae
2. V–Y columellar advancement
3. Sutures placed in a horizontal mattress fashion across the caudal aspect of the of the upper lateral cartilage to increase the angle of the ULC in relation to the dorsal septum
4. LCS increase tip rotation and projection, LCO increase tip rotation, decrease tip projection
5. External carotid artery via the facial and infraorbital branches and the internal carotid via the ethmoid and branches of the ophthalmic artery
6. Short medial crura
7. 3
8. Dorsal onlay graft
9. Placement of tip onlay graft
10. Elevation of bilateral mucoperichondrial flaps with placement of acellular dermis interposition graft through and open approach
11. 1:1
12. Weir incisions reduce alar flare but do not change the nostril circumference
13. Placement of spreader grafts
14. Tongue-in-groove
15. Crumley-Lanzer method: the dorsum of the nose is the hypotenuse of a 3-4-5 right triangle
16. Rocker deformity
17. Alar strut graft
18. Line perpendicular to the Frankfort horizontal dropped straight down from the nasion.
19. Medpor has greater pore size leading to greater strength and tissue in-growth
20. Internal nasal valve narrowing
21. Cleft lip nasal deformity
22. Shield grafts: positioned over the medial crura from the tip to the medial crural footplates, increasing tip projection
23. Smooth osteotomy line
24. Premaxillary graft
25. Intercartilaginous and marginal incisions
26. (d) Rohrich RJ, Adams WP. The Boxy nasal tip: classification and management based on alar cartilage suture techniques. Plast Reconstr Surg. 2001;107(7): 1849–1863.

27. The alar–rim columellar relationship is best assessed on lateral view. The ideal configuration is 2 mm of columellar on each side. When excessive, can be the result of (1) alar retraction, (2) hanging columella, or (3) both. For mild alar retraction (<2 mm), alar rim grafts can be used to lower or blunt the notched ala. For more significant alar retraction, conchal composite grafting is needed. Columellar can be the result of an overdeveloped caudal septum, membraneous septum. Treatment can include tongue-in-groove, caudal septal resection, membraneous septal resection. For combination deformities, surgical correction involves combined maneuvers for optimal correction.

    Gunter JP, Rohrich RJ, Friedman RM. Classification and correction of alar-columellar discrepancies in rhinoplasty. Plast Reconstr Surg.1996; 97(3):643–648.
28. (a)

    Naficy S, Baker SR. Lengthening the short nose. Arch Otolaryngol Head Neck Surg. 1998; 124(7): 809–13.
29. (c)
30. The platyrrhine nose is characterized by a broad, flat, under-projected tip, wide flaring alae, with an under-projected, wide dorsum. The nasal skin is typically thicker with a large amount of subcutaneous tissue while the cartilage and bony support is lacking. Frequently, there is premaxillary hypoplasia. Rhinoplasty principles remain the same and the surgeon should practice a balanced approach that is in harmony with the face. If significant augmentation is anticipated, one must take into consideration available grafting material as the thicker soft tissue envelope does not enable traditional reduction techniques for improvement in definition. Frequently, patients require additional grafting material such as conchal cartilage, costal cartilage, or alloplast to achieve the desired aesthetic changes. Dorsal augmentation, osteotomies, alar base excisions, and tip projection are all frequent maneuvers in this patient population.

    Romo T, Abraham MT. The Ethnic Nose. Facial Plast Surg. 2003; 19(3): 269–278.
31. Alloplast infection and extrusion is a known complication following rhinoplasty. Traditional wisdom has been to treat the patient with culture-directed antibiotics, graft removal, with delayed reconstruction following the resolution of edema. Unfortunately, this leaves the patient with an aesthetic deformity, enables scar contraction of the nasal skin and SMAS, and results in a more difficult reconstruction. An alternative technique has been proposed that involves immediate reconstruction with autogenous or irradiated homograft costal cartilage at the time of implant removal. This approach provides the patient with the benefit of immediate reconstruction and lack of aesthetic deformity and minimizes the risk of skin and SMAS contraction. Patients must be informed of the potential for wrapping, infection, absorption, extrusion, and need for further surgery.

    Clark JM, Cook TA. Immediate reconstruction of extruded alloplastic nasal implants with irradiated homograft costal cartilage. Laryngoscope. 2002; 112(6):968–974.
32. (d)
33. (c)
34. (d)
35. (c)
36. (d)

References

1. Letourneau A, Daniel RK. The superficial musculo-aponeurotic system of the nose. Plast Reconstr Surg. 1988;82(1):48–57.
2. Daniel RK. The nasal tip: anatomy and aesthetics. Plast Reconstr Surg. 1992;89(2):216–24.
3. Papel ID, Frodel JL, Holt RG. Facial plastic and reconstructive surgery. New York, NY: Thieme; 2008. p. 477–87.
4. Goin MK, Rees TD. A prospective study of patients' psychological reactions to rhinoplasty. Ann Plast Surg. 1991;27(3):210–5.

5. Anderson JR. A reasoned approach to nasal base surgery. Arch Otolaryngol Head Neck Surg. 1984;110:349–58.
6. Toriumi DM. New concepts in nasal tip contouring. Arch Facial Plast Surg. 2006;8(3):156–85.
7. Kridel RW, Scott BA, Foda HM. The tongue-in-groove technique in septorhinoplasty. A 10-year experience. Arch Facial Plast Surg. 1999;1(4):246056.
8. Adamson PA, McGraw-Wall BL, Morrow TA, Constantinides MS. Vertical dome division in open rhinoplasty. An update on indications, techniques, and results. Arch Otolaryngol Head Neck Surg. 1994;120(4):373–80.
9. Constantinides M, Liu E, Miller PJ, Adamson PA. Vertical lobule division in open rhinoplasty: maintaining an intact strip. Arch Facial Plast Surg. 2001;3:258–63.
10. Kridel RW, Konior RJ, Schumrick K, et al. Advances in nasal tip surgery: the lateral crural steal. Arch Otolaryngol Head Neck Surg. 1991;117:411–5.
11. Foda HM, Kridel RW. Lateral crural steal and lateral crural overlay: an objective evaluation. Arch Otolaryngol Head Neck Surg. 1999;125(12):1365–70.
12. Daniel RK. Rhinoplasty: open tip suture techniques: a 25-year experience. Facial Plast Surg. 2011;27:213–24.
13. Boahene KD, Hilger PA. Alar rim grafting in rhinoplasty: indications, techniques, and outcomes. Arch Facial Plast Surg. 2009;11(5):285–9.
14. Rohrich RJ, Raniere J Jr, Ha RY. The alar contour graft: correction and prevention of alar rim deformities in rhinoplasty. Plast Reconstr Surg. 2002;109(7):2495–505.
15. Gruber RP, Nahai F, Bordan NF, et al. Changing the convexity and concavity of nasal cartilages and cartilage grafts with horizontal mattress sutures. Part I. Experimental results. Plast Reconstr Surg. 2005;115:589. Part II Clinical results. Plast Reconst Surg. 2005;115:595.
16. Gruber RP, Peled A, Talley J. Mattress sutures to remove unwanted convexity and concavity of the nasal tip: 12-year follow up. Aesthet Surg J. 2015;35(1):20–7.
17. Gunter JP, Friedman RM. Lateral crural strut graft: technique and clinical applications in rhinoplasty. Plast Reconstr Surg. 1997;99(4):943–52.
18. Perkins SW, Sufyan AS. The alar-spanning suture: a useful tool in rhinoplasty to refine the nasal tip. Arch Facial Plast Surg. 2011;13(6):421–4.
19. Sheen JH. Tip graft: a 20 year retrospective. Plast Reconstr Surg. 1993;91(1):48–63.
20. Yeh CC, Williams EF. Cosmetic and functional effects of cephalic malposition of the lower lateral cartilages: a facial plastic surgical case study. Otolaryngol Clin N Am. 2009;42:539–46.
21. Kridel RWH, Castellano RD. A simplified approach to alar base reduction: a review of 124 patients over 20 years. Arch Facial Plast Surg. 2005;7:81–93.
22. Rohrich RJ, Muzaffar AR, Janis JE. Component dorsal hump reduction: the importance of maintaining dorsal aesthetic lines in rhinoplasty. Plast Reconstr Surg. 2004;114(5):1299–308. discussion 1309–12
23. Byrd HS, Meade RA, Gonyon DL Jr. Using the auto-spreader flap in primary rhinoplasty. Plast Reconstr Surg. 2007;119(6):1897–902.
24. Clark JM, Cook TA. The 'butterfly' graft in functional secondary rhinoplasty. Larygnoscope. 2002a;112(11):1917–25.
25. Park S. The flaring suture to augment the repair of the dysfunctional nasal valve. Plast Reconstr Surg. 1998;101(4):2230–2.
26. Daniel RK. Diced cartilage grafts in rhinoplasty surgery: current techniques and applications. Plast Reconstr Surg. 2008;122:1883–9.
27. Erol OO. The Turkish delight: a pliable graft for rhinoplasty. Plast Reconstr Surg. 2000;205(6):2229–41.
28. Daniel RK, Calvert JW. Diced cartilage grafts in rhinoplasty surgery. Plast Reconstr Surg. 2004;113(7):2156–71.
29. Becker DG, Pastorek NJ. The radix graft in cosmetic rhinoplasty. Arch Facial Plast Surg. 2001;3(2):115–9.
30. Cochran CS, Ducic Y, DeFatta RJ. Rethinking nasal osteotomies: an anatomic approach. Laryngoscope. 2007;117:662–7.
31. Goldfarb M, Gallups JM, Gerwin JM. Perforating osteotomies in rhinoplasty. Arch Otolaryngol Head Neck Surg. 1993;119(6):624–7.
32. Becker DG, McLaughlin RB Jr, Loevner LA, Mang A. The lateral osteotomy in rhinoplasty: clinical and radiographic rationale for osteotome selection. Plast Reconstr Surg. 2000;105(5):806–16.
33. Pastorek NJ, Becker DG. Treating the caudal septal deflection. Arch Facial Plast Surg. 2000;2(3):217–20.
34. Gubish W. Extracorporeal septoplasty for the markedly deviated septum. Arch Facial Plast Surg. 2005;7(4):230–9.
35. Boenisch M, Nolst Trenite GJ. Reconstructive septal surgery. Facial Plast Surg. 2006;22(4):249–54.
36. Watson D, Barkdull G. Surgical management of the septal perforation. Otolaryngol Clin N Am. 2009;42:483–93.
37. Romo T, Sclafani AP, Falk AN, Toffel PH. A graduated approach to the repair of nasal septal perforations. Plast Reconstr Surg. 1999;103(1):66–75.
38. Pontius AT, Leach JL. New techniques for management of the crooked nose. Arch Facial Plast Surg. 2004;6(4):263–6.
39. Naficy S, Baker SR. Lengthening the short nose. Arch Otolaryngol Head Neck Surg. 1998;124(7):809–13.
40. Johnson CM, Godin MS. The tension nose: open structure rhinoplasty approach. Plast Reconstr Surg. 1995;95(1):43–51.
41. Parker PJ. Grafts in rhinoplasty: alloplastic vs. autogenous. Arch Otolaryngol Head Neck Surg. 2000;126(4):558–61.
42. Constantian MB. Indications and use of composite grafts in 100 consecutive secondary and tertiary rhinoplasty patients: introduction of the axial orientation. Plast Reconstr Surg. 2002;110(4):1116–33.

43. Marin VP, Landecker A, Gunter JP. Harvesting rib cartilage grafts for secondary rhinoplasty. Plast Reconstr Surg. 2008;121(4):1442–8.
44. Cheney ML, Gliklich RE. The use of calvarial bone in nasal reconstruction. Arch Otolaryngol Head Neck Surg. 1995;121(6):643–8.
45. Frodel JL, Marentette LJ, Quatela VC, Weinstein GS. Calvarial bone graft harvest: techniques, considerations, and morbidity. Arch Otolaryngol Head Neck Surg. 1993;119(1):17–23.
46. Kridel RW, Ashoori F, Liu ES, Hart CG. Long-term use and follow-up of irradiated homologous costal cartilage grafts in the nose. Arch Facial Plast Surg. 2009;11(6):378–94.
47. Clark JM, Cook TA. Immediate reconstruction of extruded alloplastic nasal implants with irradiated homograft costal cartilage. Laryngoscope. 2002b;112(6):968–74.
48. Ferril GR, Wudel JM, Winkler AA. Management of complications from alloplastic implants in rhinoplasty. Curr Opin Otolaryngol Head Neck Surg. 2013;21(4):372–8.
49. Romo T, Kwak ES. Nasal grafts and implants in revision rhinoplasty. Facial Plast Surg Clin North Am. 2006;14(4):373–87.
50. Godin MS, Waldman SR, Johnson CM. Nasal augmentation using Gore-Tex: a 10-year experience. Arch Facial Plast Surg. 1999;1(2):118–21.
51. Winkler AA, Soler ZM, Leong PL, et al. Complications associated with alloplastic implants in rhinoplasty. Arch Facial Plast Surg. 2012;14(6):437–41.
52. Tweedie DJ, Lo S, Rowe-Jones JM. Reconstruction of the nasal septum using perforated and unperforated polydioxanone foil. Arch Facial Plast Surg. 2010;12(2):106–13.
53. Peled AV, Warren AG, Johnston PMM, Yaremchuk MK. The use of alloplastic materials in rhinoplasty surgery: a meta-analysis. Plast Reconstr Surg. 2008;121(3):85–92.
54. Davis RE, Bublik M. Common technical causes of the failed rhinoplatsy. Facial Plast Surg. 2012;28:380–9.
55. Toriumi DM, Josen J, Weinberger M, Tardy ME. Use of alar batten grafts for correction of nasal valve collapse. Arch Otolaryngol Head Neck Surg. 1997;123(8):802–8.
56. Gillman GS, Simons RL, Lee DJ. Nasal tip bossae in rhinoplasty. Etiology, predisposing factors, and management techniques. Arch Facial Plast Surg. 1999;1(2):83–9.
57. Gunter JP, Rohrich RJ, Friedman RM. Classification and correction of alar-columellar discrepancies in rhinoplasty. Plast Reconstr Surg. 1996;97(3):643–8.
58. Adamson PA, Tropper GJ, McGraw BL. The hanging columella. J Otolaryngol. 1990;19:319–23.

Rhytidectomy Essentials

27

David Shamouelian and Jordan P. Sand

Anatomy

Topographical analysis of the face.

Soft Tissue Landmarks

- Trichion—Anterior hairline in the midline
- Glabella—Most prominent point of the forehead on profile
- Nasion—The deepest depression at the root of the nose; typically corresponds to the naso-frontal suture
- Radix—Root of the nose, a region and not a point; part of an unbroken curve that begins at the superior orbital ridge and continues along the lateral nasal wall
- Rhinion—Osseocartilaginous junction on the nasal dorsum, area of thinnest skin over nasal dorsum
- Sellion—Soft tissue correlate of the nasofrontal suture line
- Supratip—Point cephalic to the tip
- Tip—Ideally, the most anterior projection of the nose on profile
- Subnasale—Junction of columella and upper lip
- Labrale superius—Vermilion border of upper lip
- Stomion—Central portion of interlabial gap
- Labrale inferius—Vermilion border of lower lip
- Mentolabial sulcus—Most posterior point between lower lip and chin
- Pogonion—Most anterior midline soft tissue point of chin
- Menton—Most inferior soft tissue point on chin
- Cervical point—The innermost point between the submental area and the neck

Skeletal Cephalometric Reference Points

- Sella: The midpoint of the hypophyseal fossa
- Orbitale: The most inferior point on the infra-orbital rim
- Porion: The most superior point on the external auditory meatus
- Condylion: The most superior point on the head of the mandibular condyle
- Articulare: The point of intersection of the posterior margin of the ascending mandibular ramus and the outer margin of the cranial base
- Anterior nasal spine
- Posterior nasal spine

D. Shamouelian
Otolaryngology – Head and Neck Surgery, University California, Irvine Medical Center, Orange, CA, USA

J. P. Sand (✉)
Spokane Center for Facial Plastic Surgery, Spokane, WA, USA
e-mail: info@sandplasticsurgery.com

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8_27

- Subspinale: The deepest point in the concavity of the premaxilla
- Prosthion: The lowest most anterior point on the alveolar portion of the premaxilla
- Infradentale: The highest most anterior point on the alveolar portion of the mandible
- Supramentale: The most posterior point in the outer contour of the mandibular alveolar process
- Pogonion: Most anterior point on the bony chin in the midline
- Gnathion: A point between the most anterior (pogonion) and inferior point (menton) on the chin
- Menton: The lowest point on the mandible

Aging Face

Microscopic Skin Changes

Intrinsic Processes Due to Senescence

- Thinning epidermis: impaired cell turnover resulting in slower wound healing and less effective desquamation.
- Flattening of the dermal–epidermal junction: increased skin fragility and impaired nutrient transfer between dermis and epidermis. Thinning occurs due to the flattening of the rete ridge pattern.
- Atrophic dermis: reduced numbers of fibroblasts with reduction in collagen and fragmentation of the dermal collagen matrix. Ratio of type III collagen to type I collagen increases.
- Subdermal adipose tissue atrophy

Extrinsic Processes Primarily Due to Photoaging (Others Include Gravity and Smoking)

- Elastosis: Accumulation of elastin material just below the dermal–epidermal junction
- Epidermal atrophy and loss of orderly differentiation
- Fragmentation of collagen and elastin fibers
- Reduction of melanocyte density but higher density of melanocytes in sun-exposed skin
- Reduction in Langerhans cells: permissive development of skin cancer

Alteration of Aesthetics with the Aging Face Syndrome

The basic shape of the face is a function of skin, soft tissues, and underlying skeletal support that changes with aging. Skin becomes wrinkled and loses elasticity. Subcutaneous tissues shift under the constant force of gravity while fat atrophies and bone demineralizes.

Upper Third of the Aging Face

Brow/Forehead

- Elongation of the upper third as the hairline recedes and the brow descends.
- *Brow ptosis and lateral brow hooding*: increased skin laxity, fat atrophy, gravity, and chronic downward contraction of the orbicularis oculi.
- *Crow's feet—lateral orbicularis oculi muscle is the most powerful brow depressor*
- *Deep furrows* of the forehead and glabella: repeated pull on the skin by frontalis (horizontal forehead rhytides), procerus (horizontal glabellar rhytides), and corrugator supercilii (vertical glabellar rhytides) muscles.
- *Increased brow prominence secondary to volume loss leads to masculine upper third appearance*

Periorbital Area

Periorbital aging may convey unintentional expression of disinterest, fatigue, anger, and sadness, which may not be consistent with actual feelings.

- Skeletal changes of the orbit alter the aging face with increased orbital width and change in shape/contour—increase in the height of the superior orbital rim medially and the inferior orbital rim laterally. This can contribute to hollowing of the eyes and flattening of the malar eminence.
- Aging, ptotic brow (see above) pushes skin over the upper eyelid, worsening eyelid cosmesis and potentially causing visual field deficits.
- Pseudoherniation of fat from orbital septum laxity results in lower "eye bags"

- *Excess eyelid skin* (*dermatochalasis*): loss of eyelid skin elasticity and brow descent (should not be mistaken for blepharoptosis or blepharochalasis).
- *Blepharochalasis*: thin, stretched, wrinkled upper eyelid skin secondary to recurrent eyelid edema. Lower eyelid is typically spared.
- *Blepharoptosis*: abnormal drooping of upper eyelid with reduction in MRD1; most commonly due to levator aponeurosis dehiscence.
- *Entropion*: inversion of the eyelid margin.
- *Ectropion*: eversion of eyelid margin due to weakening or stretching of the lateral canthal tendon and dehiscence of the orbicularis oculi.

Middle Third of the Aging Face

Midface

- Drooping of the orbicularis and malar skin–soft tissue complex leads to loss of youthful “cherubic” appearance and the Ogee curve.
- Inferomedial descent of midface fat secondary to deep medial cheek fat and malar volume loss.
  - Deepening and accentuation of the nasolabial fold.
  - Elongates lower eyelid–cheek junction
  - Tear trough deformity
- Simultaneously, the orbicularis oculi muscle stretches and descends inferolaterally.
  - Exposure of the inferior and lateral bony orbital rims, and loss of malar prominence.
  - *Tear trough deformity*, a hollowness developing in the area of transition between lower eyelid and cheek.
- Loss of midface skeletal support or congenitally deficit midface with resultant negative vector

Nose

- Thinning skin and subcutaneous tissue reveal and accentuate underlying cartilage and osseous structure (dorsal irregularities).
- *Loss of tip projection*: loss of tip support due to skin soft-tissue envelope changes.
- Weakening fibrous attachment at the scroll with separation of the upper lateral cartilages from the lower lateral cartilages.
- Stretching and weakening of interdomal ligaments resulting in a boxy tip and loss of tip definition.
- *Tip ptosis/loss of tip rotation*: due to midface osseous resorption with loss of maxillary and alveolar height.
- These changes may result in narrowing of the nasal valve and ultimately obstructive nasal breathing.

Lower Third of the Aging Face

Jowls

A central stigmata of the aging face: obscured fine osseous definition of the mandible due to fat and soft-tissue descent along the mandibular line or surrounding volume loss.

- Bordered anteriorly by the mandibular retaining ligament.
- Bone resorption of mandible exacerbates deformity.
- “Bulldog-looking” cheeks due to fullness of the jowls along the mandibular body lateral to the oral cavity.
- Attributed to accumulation of fat in the neck, gravity descent of surrounding fat pads, laxity of facial retaining ligaments, ptosis of the skin and mandibular septum, submandibular gland hypertrophy, and/or surrounding facial fat volume loss.

Neck

A youthful neck is characterized by thin soft tissue with apparent underlying structures (anterior border of the sternocleidomastoid muscle and thyroid cartilage), cervicomental angle 90–105°, and an absence of liposis, tissue sagging, and wrinkles. Hyoid position and jawline definition are critical structures for overall neck contour.

- *Platysmal bands*: Vertical bands extending from the submentum to the clavicle.
- *Turkey gobbler neck*: Platysmal dehiscence leads to subplatysmal soft tissue, submandibu-

lar gland, and fat descent into the submentum and lower midline neck.
- *Submandibular gland ptosis and hypertrophy*—can be exacerbated by mandibular bone resorption
- *Obtuse cercivomental angle*—can be secondary to low, inferior hyoid position, submental fat accumulation, or retrognathia/micrognathia.

Perioral Area

The youthful perioral area has significant fullness, exhibits minimal depth of the nasolabial folds, and lack of rhytides.

- *Upper lip elongation* with thinning of the red lip and vertical expansion of the white cutaneous portion. The vermillion becomes less prominent, the lateral commissures tend to droop, and the upper teeth are less visible.
- *Fine rhytides* radiate from the circumference of the oral cavity to the nose superiorly and the labiomental sulcus inferiorly.
- Lower teeth become more visible due to the effects of gravity on the lower lip.

Chin

- Ptosis of the soft tissues of the skin of the chin, also known as *Witch's chin deformity*: excess submental fullness and prominent submental crease due to premental fat ptosis and loss of mandibular osseous height and projection.

Earlobe

- Elongation of the earlobes is attributed to long-standing earring use and increased skin laxity.

Superficial Musculoaponeurotic System (SMAS) and the Retaining Ligaments of the Face

An understanding of the superficial muscular aponeurotic system (SMAS) is essential to facial surgery. The SMAS is a fibromuscular fascial layer that envelops and interconnects the mimetic muscles of the face. It maintains consistent relationships with vessels and nerves and serves as a guide to the depth of key neurovascular structures in the lower face. Facial nerve branches and the parotid duct are seen deep to the SMAS and superficial to the masseter and the buccal fat pad.

The SMAS is contiguous with the platysma inferiorly and the temporoparietal fascia superiorly. The SMAS invests the frontalis muscle and is continuous with the galeal aponeurosis. In the periorbital area, the SMAS interlocks with the orbicularis oculi. Medially, it has attachments to the zygomaticus major and minor as well as the dermis of the upper lip.

With sub-SMAS dissection of the cheek anteriorly, fascial condensations of the SMAS are encountered. These so-called ligaments support the soft tissues of the cheek. The major (osseocutaneous) retaining ligaments of the face include the zygomatico-cutaneous ligament (McGregor's patch) superiorly and the mandibular ligaments inferiorly. The other fascia–fascia retaining ligaments include the parotid and masseteric ligaments.

Superficial to this SMAS layer in the cheek lies the malar fat pad that becomes ptotic with age. A plane can be developed between the fibromuscular SMAS and malar cheek pad. This malar fat pad can be repositioned separately from the SMAS. The fibromuscular SMAS, if followed anteriorly, merges with the superficial layer of the orbicularis oris muscle. The orbicularis oculi muscle when elevated exposes the sub-orbicularis oculi fat (SOOF). Deep to this fatty layer along the orbital rim lie the insertions of the elevator muscles of the lip. There is a perforating branch of the transverse facial artery, which is encountered inferior to the malar eminence and before reaching the zygomatic major muscle. There are no branches of the facial nerve along the zygomatic prominence and infraorbital rim. As a result, this area can be used safely for suturing the malar fat pad. Because of the osseous insertions and prominence of the zygomatic major and other lip elevator muscles, it is safer to begin elevation above these muscles and elevate inferiorly toward the platysma. An inferior-to-superior elevation would more likely result in injury to a facial nerve branch as one may slide into a deeper plane given the more tenuous nature of the SMAS at the mid-cheek.

Facial Nerve Anatomy for the Rhytidectomy Surgeon

The facial nerve traverses anteriorly through the parotid gland after exiting the stylomastoid foramen. It branches into five branches: temporal (or frontal), zygomatic, buccal, marginal mandibular, and cervical branches. Within the parotid gland, the main trunk usually divides into a superior temporofacial and inferior cervicofacial branches. Familiarity with the anatomy of the facial nerve is critical to avoiding nerve injury. The frontal and marginal branches are the most commonly injured branches of the facial nerve in facelift.

The frontal branch of the facial nerve innervates the frontalis muscle and the orbicularis oculi muscle. This division of the facial nerve courses from the parotid gland, anterior to the superficial temporal artery, toward its final destination, where it pierces the undersurface of the frontalis muscle 1.5 cm above the lateral canthus. The frontal branch of the facial nerve lies deep to the superficial musculoaponeurotic system fascia (SMAS) and its continuation in the temporal region, the temporoparietal fascia. The course of the nerve may be approximated by a line drawn from a point 0.5 cm anterior to the tragus to a point 1.5 cm lateral to the lateral brow.

The danger zone of the frontal branch of the facial nerve has been further delineated—the region overlying the zygomatic arch between 1.8 cm anterior to the helical root and 2 cm posterior to the lateral canthus. As the nerve crosses over the zygomatic arch, it lies between the periosteum of the zygoma and the SMAS. Dissection in the region of the central third of the zygomatic arch requires caution to avoid injury to the nerve and should be carried out either subcutaneously or subperiosteally. This is relevant during a subperiosteal midfacelift approach that requires elevation of the periosteum from the anterior portion of the zygomatic arch.

The temporal branch is also particularly at risk when combining forehead- and facelifting. Above the zygomatic arch, the nerve travels in the temporoparietal fascia. To avoid injury when dissecting in the temple and lateral orbital areas, the plane of dissection for the lateral portion of the forehead lift is in the subgaleal plane below the temporoparietal fascia and over the superficial layer of the deep temporal fascia. The facelift dissection anterior to the hairline in the temple is in the superficial subcutaneous tissue plane. Therefore, there are two planes of dissection overlapping in the lateral orbital and temporal region. The temporal branch is located between these planes.

As the deep plane rhytidectomy transitions from a sub-SMAS plane in the inferior cheek to a supra-SMAS plane in the superior medial cheek, the zygomatic branch of the facial nerve is at risk for injury. This is because the shifting of the plane to a more superficial plane anterior to the zygomatic major muscle requires sharp dissection.

The subperiosteal approach for midfacelifting places the buccal branch of the facial nerve at risk. Releasing the periosteum off the inferior border of the zygoma requires dissection over the masseter muscle tendon that lies in proximity to the buccal nerve.

The marginal mandibular nerve is responsible for innervation of the depressor labii angularis, depressor anguli oris, mentalis muscle, and portions of the platysma. The “danger zone” for the marginal mandibular nerve is from the angle of the mandible to its crossing superficial to the facial artery and extends from the inferior border of the mandible to a line that is 3 cm parallel and inferior to the mandible. Below the angle of the mandible the branch is located immediately beneath the platysma. Atrophy or hypoplasia of the platysma muscle may result in little protection of the marginal mandibular nerve. Nerve injury can be avoided by remaining superficial to the platysma and SMAS. When elevating the SMAS laterally in the cheek, the nerve is encased in sub-SMAS fat after exiting the parotid tail. Blunt dissection under direct visualization should be performed in the region to anterior to the parotid to avoid injury. The marginal mandibular branch is at risk for injury while developing the posterior platysmal flap for correction of platysmal laxity. Therefore, the surgeon should only release the retaining ligaments necessary for adequate mobility to minimize facial nerve risk.

Rhytidectomy Preoperative Assessment

Patient Selection

- Understand patient's motivation for having plastic surgery.
- A patient with poor self-image or body dysmorphic disorder, whose motivation for surgery is to create personal happiness, is often dissatisfied with the surgical result.
- Warning signs—patient in the midst of a life crisis, an unhappy patient, a psychologically impaired patient (paranoia, psychosis, obsessive-compulsive disorder, borderline personality, SIMON syndrome), body dysmorphic disorder, "package of pictures" syndrome, etc.
- The patient's general health status, past medical history, medications, allergies, previous surgeries, and social habits need to be reviewed to ensure fitness for anesthesia and surgery:
  - Bleeding history, use of supplements/homeopathy, drug use, recent skin infections, isotretinoin use.
- Tobacco use is a contraindication for facial surgery. Refraining 2–4 weeks prior to and after surgery are essential due to deleterious effects on healing. Cigarette smoking is associated with a 12-fold increase in risk of flap necrosis (nicotine patches should also be avoided in the perioperative period).
- Table 27.1 reviews absolute contraindications for facelift surgery.

Physical Exam

- Evaluation includes inspection and palpation of facial structures.
- Facial aesthetic ideals and the changes associated with aging are recognized (see above).

Table 27.1 Absolute contraindications for rhytidectomy

Active smoking
Active vasculitides
Active autoimmune disease of facial vasculature
Active chemotherapy or immunosuppression
Full-course facial radiation exposure
Largely fluctuating weight
Uncontrolled hypertension
Medically unfit for anesthesia
Psychologically unprepared or unfit
Unrealistic expectations

Table 27.2 Favorable and unfavorable rhytidectomy candidates

Favorable features	Unfavorable features
Strong forward chin	Retrognathic or weak chin
Prominent cheek structure	Deep oral commissure grooves
Good facial bone structure	Thin skin
Fuller midface	Severely wrinkled and sun-damaged skin
Sharp cervicomental angle	Low hyoid with obtuse cervicomental angle
Shallow cheek/lip grooves	Deep cheek/lip grooves
Nonsmoker	Weak cheek bones
Good skin tone	Deficient midface tissues
Few wrinkles with minimal photoaging	Visible submandibular glands

- Facial asymmetry—due to bony skeleton, the preference for sleeping on one side of the face, and involuntary repetitive facial expressions.
- Fitzpatrick skin types—patient with darker skin may have a tendency for hypertrophic scar formation and pigmentation changes along incision lines.
- Patients with thinner faces tend to demonstrate better postoperative contour improvement when compared with patients with fuller faces. Table 27.2 reviews favorable and unfavorable physical exam features for rhytidectomy.

Photo Documentation

Before and after photographs should highlight the surgical difference by keeping all other elements the same—background, clothes, hairstyle and makeup, distance of subject from camera, positioning of head, and light source. Two-point light is preferable to ring flash. For lateral views, position the Frankfort horizontal plane parallel to the floor.

Facelift Standard Photographic Views

- Full-face front, neutral gaze
- Full-face front, smiling
- Lateral, neutral gaze
- Lateral, smiling
- Lateral, neck in flexion
- Oblique

Facial Surgery Perioperative Care and Anesthesia

Safe, consistent anesthesia instills a positive perception of the experience, elicits confidence, and prevents conditions that can significantly hinder the healing process.

- Stop nonsteroidal anti-inflammatory medications, herbal supplements, and vitamins (excluding multivitamins) at least 2 weeks before surgery; the use of aspirin is stopped at least 3 weeks before surgery. Table 27.3 reviews medications and supplements that impair blood clotting and may induce perioperative arrhythmia.
- Blood pressure management and control of postoperative nausea are key in the prevention of hematoma after rhytidectomy.
- Antibiotic prophylaxis

Table 27.3 Medications and supplements that impair blood clotting and may induce perioperative arrhythmia

Medication/ supplement	Example
Vitamin/health supplement	St John's wort, ginkgo biloba, vitamin E, Echinacea, ginseng, valerian, Ephedra, glucosamine, vitamin C (>2000 mg daily), Feverfew, goldenseal, fish oils (omega-3 fatty acids), garlic, licorice, Kava, Dong quai
Nonsteroidal anti-inflammatory drugs	Ibuprofen, indomethacin, naproxen, nabumetone
Anti-inflammatory medications	Toltetrin, dipyridamole, fenoprofen
All aspirin-containing medications	Alka-Seltzer, Excedrin

Patient Safety and Anesthesia/Airway Considerations

- Patient position with padded support to avoid ulnar/brachial neuropathy
- Deep vein thrombosis prophylaxis with sequential compression device and/or compression stockings
- Anesthesia/airway options
  - IV sedation with oxygen supplementation and oral airway
  - Deep sedation with laryngeal mask airway (LMA)
  - General anesthesia with endotracheal intubation

Rhytidectomy Techniques

Limited Facelift Procedures

Short Flap Rhytidectomy (AKA: Mini-Lift, "Weekend" Lift)

Selected in cases with the desire for a safe and sensible, yet subtle improvement in the signs of aging. Typically reserved for young patients with mild degree of skin laxity without significant midface ptosis or jowling.

- *Advantages*
  - Excellent safety record
  - Reduced operating time and expense
  - Rapid recovery
- *Drawbacks*
  - Limited and short-lived benefits

Technique Key Points

- Limited preauricular incision terminating just behind the lobule
- Short segment of skin undermining and minor SMAS treatment with plication or imbrications

Extended Rhytidectomy Procedures

This group includes several approaches with variable degree of tissue undermining. Selected in cases with more significant signs of aging affecting the face and neck, and where a more dramatic improvement is sought.

Subcutaneous Rhytidectomy (AKA: Long Flap Technique)

- Advantages:
  - Excellent safety record with low risk to facial nerve
- Drawbacks:
  - Limited improvement of the jowl
  - No correction of the midface
  - Greater risk of scarring as tension on subcutaneous, not deeper, structures

Technique Key Points

- Skin flap is elevated in the subcutaneous tissue plane.
- The extent of anterior dissection of the face is based on surgeon's preference and the degree of facial skin laxity.
- Dissection to the lateral bony orbital rim in the temple releases the skin from the underlying orbicularis oculi. This helps address the crow's feet and facilitates redistribution of the vertically advanced preauricular skin flap.
- The skin of the upper neck is also elevated in a preplatysmal plane. Dissection may continue forward in the neck to the midline.
- The SMAS can be plicated by folding it on itself using sutures to suspend it to the parotid fascia.

SMAS Techniques

- Advantages:
  - Striking improvement of the jowl
  - Longer-lasting results
- Drawbacks:
  - No corrective lift of the midface

Technique Key Points

- Elevation of a thick flap below the SMAS (*sub-SMAS dissection*) limited to the area over the parotid gland or extending anterior to the parotid gland
- The SMAS flap is then suspended in a posterior–superior vector
- SMAS dissection with mobilization of a composite flap of platysma and overlying soft tissue is favorable for correcting the jowl
- Plication/imbrication versus sub-SMAS dissection to address SMAS
- Use of multiple vectors for resuspension: more superiorly based vector in preauricular area to avoid windswept look; more posterolateral pull on lateral platysma/infra-auricular area to resuspend to mastoid fascia
- *Baker's Strip (Lateral) SMASectomy*
  - Removing a portion of the SMAS in the region directly overlying the anterior edge of the parotid gland. Usually, a 2–4-cm-wide strip of SMAS is excised, depending on laxity.
  - Excision of SMAS along a line extending from the angle of the mandible to the lateral malar eminence.
  - The vector in which the SMASectomy is performed is oriented parallel to the nasolabial fold, thereby producing improvement of the nasolabial fold.
  - The mobile anterior SMAS is then sutured securely to the fixed portion of the superficial fascia overlying the parotid in a posterior–superior fashion.

Hamra's Deep Plane Rhytidectomy

The SMAS represents a fascial extension of the platysma muscle beneath the cheek fat of the midface and thins prior to reaching the zygomatic major muscle. Traction placed on the SMAS, as in traditional SMAS rhytidectomy approaches, is not transmitted to the melolabial fold and as a result does not improve the fold. The SMAS is effectively anchored by the osseous attachments of the mimetic muscles it invests. Hence, the investments of the SMAS in the region of the midface must be released in order to move the SMAS and the overlying malar fat pad superiorly, thereby improving the melolabial fold and the appearance of the midface.

- The sub-SMAS dissection is advanced superiorly at the inferior cheek, sharply cutting through the SMAS peripherally.
- Essentially, the plane of dissection transitions from a sub-SMAS plane in the inferior cheek to a supra-SMAS plane in the superior medial cheek remaining just superficial to the zygomatic major and minor muscles.
- This releases the SMAS from its superomedial investments of the midface, permitting the SMAS and the overlying skin and subcutaneous fat pad to advance upward as a single tissue flap.
- The dissection extends medially and can proceed beyond the melolabial fold, totally releasing all SMAS attachments to the dermis of the upper lip, creating a thick musculocutaneous flap composed of skin, subcutaneous fat of the cheek, and the platysma.
- Shifting the dissection sharply to a more superficial plane from beneath the platysma of the lower cheek to a plane superficial to the SMAS superior and anterior to the zygomatic major muscle in the superomedial cheek places the zygomatic branch of the facial nerve at risk to injury.
- *Hamra's Composite Rhytidectomy*
  – Modified the deep plane rhytidectomy to incorporate the orbicularis oculi in the rhytidectomy flap, thereby connecting the facelift flap with a lower blepharoplasty dissection to achieve improvement in the malar crescents and resetting of the orbital septum.

Supra-SMAS Technique

- Advantages:
  – Corrects ptotic cheek fat of midface aging and mitigates the nasolabial fold
- Drawbacks:
  – Extended preauricular skin flap via lengthy anterior dissection, leading to large dead space where hematoma may accumulate
  – Does not provide as much improvement of the jowl compared with employing SMAS flap of the inferior cheek

Technique Key Points

- Subcutaneous tissue dissection extended anteriorly beyond the parotid gland toward the midface.
- However, this dissection is performed at a deeper level than the standard subcutaneous facelift plane and is immediately superficial to the SMAS and mimetic muscles of the face.
- The resulting thick cutaneous flap consisting of the cheek fat and the overlying attached facial skin.
- *Owsley's Extended Supra-SMAS Rhytidectomy*
  – The dissection is carried anteriorly to the upper lip to release all of the dermal attachments of the SMAS to the nasolabial crease.
  – The cutaneous flap is then suspended under considerable tension posterosuperiorly to the fascia overlying the zygoma and parotid gland.
  – The goal of this technique is to displace the cheek fat of the midface superiorly correcting the ptotic cheek fat, thus softening the nasolabial fold.

Subperiosteal Rhytidectomy

- Lifting of the midface tissues with a subperiosteal plane of dissection over the maxilla and zygoma.
- All midface soft tissues are lifted including skin, fat, and muscle.
  – Elevators of the lip, zygomatic major and minor, and orbicularis oculi
- Elevating the origin of the zygomatic musculature to a higher position on the zygoma improves elevation of the upper lip and oral commissure, resulting in an aesthetically pleasing shape to the mouth.

- Procedure also typically includes an endoscopic subperiosteal forehead lift.
- Tri-plane rhytidectomy combines subperiosteal dissection with sub-SMAS approach to achieve improvement in both midface and jowls.
- Advantages:
  - Overall safety with less risk of injury to the zygomatic branch of the facial nerve with the subperiosteal approach compared with the deep plane or extended supra-SMAS facelift
  - Rejuvenating the midface without placing unnatural tension on the skin in the temporal area
  - Enhancing malar eminence due to increased horizontal width of the face by displacing the origin of the zygomatic major muscle to a more superior and lateral position
  - No disruption of the blood supply to the skin of the midface
  - Does not require a long cheek flap extending anteriorly compared to the deep plane and extended supra-SMAS facelifts
- Drawbacks:
  - Protracted recovery time due to increased edema compared to other techniques
  - Subperiosteal midfacelifts do not significantly correct the jowl and have no influence on the upper neck
  - Increases the horizontal width of the face by displacing the origin of the zygomatic major muscle to a more superior and lateral position
  - Risk to the temporal branch of the facial nerve with elevation of the periosteum from the anterior portion of the zygomatic arch
  - Risk to the buccal branch of the facial nerve during dissection of the periosteum off the inferior zygoma requiring dissection over the masseter muscle tendon

Technique Key Points

- The key is to release the periosteum from the lateral and inferior bony orbital rim and from the entire zygoma and maxilla.
- Four surgical approaches used:
  - (1) Transtemporal usually using an endoscope

    Incision made behind the hairline in the temple.

    Endoscopic dissection deep to the temporoparietal fascia of the anterior lateral scalp, i.e., plane of dissection is immediately superficial to superficial layer of deep temporal fascia. Temporal branch is protected in a plane superficial to the plane of dissection.

    Transition made to a subperiosteal plane as dissection continues inferiorly over the zygoma and zygomatic arch.

    Subperiosteal dissection extended medially in the midface releasing the soft tissues from their attachment to the maxilla.

    Requires some dissection of the upper portion of the masseter muscle.

    Disadvantage: difficulty releasing the periosteum from the medial maxilla due to poor visualization, even when an endoscope is used.
  - (2) Transorbital through a lower eyelid or transconjunctival incision

    Advantage: direct access to the midface without the need for an endoscope.

    Subperiosteal dissection occurs under direct visualization.

    Midface soft-tissue suspension to the inferior bony orbital rim or to the anteroinferior temporalis fascia.

    Disadvantage: transient distortion of the lateral canthal area from bunching of redundant soft tissue and risk for lower eyelid malposition.
  - (3) Transoral through an upper gingival buccal incision

    Combined with transorbital or transtemporal incisions for a combined surgical approach.

    Allows the periosteum to be easily lifted from the inferior aspects of the maxilla and zygoma via oral access.

Advantage: ease of dissection and direct visualization without the need for an endoscope.

Disadvantage: risk of wound infection via bacteria in the oral cavity.

– (4) Combined using two more of the previously listed approaches

Midfacelift

The midfacelift can be performed alone or as an adjunct to rhytidectomy in treating the aging face. The various planes of dissection and surgical approaches to the midface are found in Tables 27.4 and 27.5, respectively.

Threadlift or Suture Lift

- A minimally invasive technique that has gained popularity due to shorter operating time and opportunity for performance of surgery in office-based setting. Marketed to patients that are willing to trade a more modest degree of cosmetic improvement for decreased morbidity.
- To counteract the descent and laxity of the facial tissues, *barbed sutures or cones* are placed under the skin of the neck and face
- Suspension of ptotic facial soft tissues via the subcutaneous plane
- Advantages:
  – Avoids incisions and undermining
  – Reduced recovery time
- Drawbacks:
  – Repositions the soft tissues in a superficial plane without addressing excess skin
  – Suture/cone *breakage* and *extrusion*, skin dimpling, superficial hemorrhages, mild asymmetry, ecchymosis, erythema, edema, and persistent pain
  – Not easily reversible
  – Revision rate as high as 20%
  – Can result in short-lived results with low patient satisfaction

Standard Rhytidectomy Incisions

Factors that influence incision design: gender, hairline, hairstyle, history of previous facelift surgery, and facial and cervical skin redundancy

Temporal Incision

- Extends above the auricle into the hair, curving upward and forward to end 1 or 2 cm above the level of the eyebrow
- Often a horizontal incision (*sideburn incision*) is added beneath the temporal tuft of hair to enable removal of vertically elevated facial skin without raising the temporal hairline to an abnormal height
- Preservation of temporal hair tuft is critical in all facelift patients and poorly designed incisions in this area can lead to significant temporal alopecia that is difficult to correct and may necessitate hair grafting.

Table 27.4 Midfacelift: Planes of dissection

	Advantages	Disadvantages
Subperiosteal	Avoids preauricular incision/skin flap Does not impair vascular supply of facial skin Avoids risk to zygomatic branch of facial nerve Lifting orbicularis oculi of lower eyelid Lifting of oral commissure	Increases horizontal width of face Risk to temporal and buccal branches of facial nerve and infraorbital nerve Greater postoperative edema May cause hypesthesia of malar eminence
Supra-SMAS	Less postoperative edema Does not reposition zygomatic musculature Avoids risk to temporal and buccal branches of facial nerve	Long preauricular cutaneous flap Reduces skin vascularity of face Risk to zygomatic branch of facial nerve Does not lift orbicularis oculi

Table 27.5 Surgical approaches to the midface

Surgical approach	Dissection plane	Advantages	Disadvantages
Transfacial	Supra-SMAS	Avoids risk to temporal and buccal branches of facial nerve Less post-op edema	Preauricular incision and long skin flap Requires some blind dissection Does not lift orbicularis oculi
Transtemporal	Subperiosteal	No preauricular incision Simultaneous lateral brow lift	Risk to temporal and buccal branches of facial nerve Requires endoscope Poor access to periosteal dissection of maxilla
Transorbital	Subperiosteal/supraperiosteal	Direct access for dissection and suspension of midface More vertical vector for suspension of midface soft tissues	Risk to buccal branch of facial nerve Risk to infraorbital nerve Risk of lower lid malposition
Transoral	Subperiosteal	Direct access for dissection of midface Ease in elevating periosteum of maxilla	Risk to buccal branch of facial nerve Does not provide access for suspension of midface tissues Greater risk of infection Risk to infraorbital nerve
Combined	Subperiosteal usually transoral combined with transtemporal or transorbital	Direct visualization of entire dissection Ease of suspension of midface tissues	More postoperative edema Greater risk of infection if transoral route used

Preauricular Incision

- Follows curve of the root of the helical crus to the level of the tragus.
- The incision may then continue posterior to the tragus or in the pretragal crease to the inferior aspect of the earlobe.
- Post-tragal incision should be used in women:
  - In men this can result in the tragus being covered with hair-bearing skin, although electrolysis and laser hair removal in this area can be used as adjunctive measures to remove unwanted hair.
- Advantages of the retro-tragal incision:
  - Incision is hidden behind the posterior border of the tragus.
  - Should be used in women.
- Disadvantages of the retro-tragal incision:
  - Obliteration of the pre-tragal crease and scar contraction that may pull the tragus forward, with an unnatural visibility of the external auditory canal.
  - If a retro-tragal incision is utilized, the pretragal hollow must be reestablished with a dermal stitch, recreating the normal preauricular contour.
- Unnatural contours in the peri-tragal area are prevented by thinning the portion of skin flap immediately anterior to the tragus and trimming the flap with a minor redundancy of skin covering the tragus.

Earlobe Incision

- Fashioned so the lobe is separated from the skin flap, with a 2 mm cuff of facial skin at its base
- Ensures the preservation of the sulcus between the lobe and the facial skin
- Complication: *satyr's ear/pixie ear deformity*
  - Due to excessive tension on the earlobe postoperatively causing the earlobe to gradually become pulled down or stretched down onto the face. This unnatural-

appearing earlobe is a giveaway for facelifting.
  - Litton stitch—mattress suture placed at cut edge of ear-lobe secured to underlying tissue can assist in minimizing risk of satyr's ear deformity.

Retroauricular Incision

- Can be positioned either 2–3 mm anterior or posterior to the postauricular sulcus (AKA cephaloauricular groove) on the medial surface of the auricle.
- If anteriorly placed, addresses the potential for some posterior migration of the retroauricular scar, so that the scar will remain hidden by the auricle.

Posterior Scalp Incision

- Extends from the retroauricular incision across the postauricular sulcus at the level of the tragus or where the helix meets the hairline to best conceal incision.
- The straight line from the auricle to the hair-bearing scalp can be altered with a small superiorly based triangular flap in the mastoid area.
- This dart of skin helps prevent straight-line scar contracture and hypertrophic scarring.
- The scalp incision is directed into the hair, curving slightly inferiorly and parallel to the postauricular hairline.
- The incision should not be made along the inferior border of the postauricular hairline as incisions designed along the hairline can become obvious, rendering patients unable to wear their hair up to due visible scarring in this location.
- In younger patients with only mild elastosis, the skin does not need to be elevated from the postauricular area and neck dissection may not be necessary. Therefore, the retroauricular incision may be limited only to a short vertical incision behind the ear with no posterior scalp incision.

Male Rhytidectomy

Special Considerations

- The approach to the male rhytidectomy patient is different due to fundamental differences in their motivation, expectations, support systems, and adaptability to changes in appearance compared to women.
- Anatomically, men have a higher degree of descent of the midface leading to increased hollowing and more accentuated nasolabial folds.

Incisions

Specific pattern and distribution of facial and temporal hair influence incision design. Additionally, males who wear their hair short or shave their head may need to be advised to grow their hair out to better camouflage the periauricular facelift incisions.

- *Temporal incision*—most important consideration is to minimize alterations in the hairline and maintain the temporal tuft of hair.
  - *Full growth of hair and no temporal recession*: no significant modification of the incision is necessary and the sideburn incision is not necessary.
  - *Thinning hair with temporal recession or significant male-pattern baldness*: requires modifications of the temporal incision.
    - Superior incision may be shortened to prevent it from extending into the area of recessed hair.
    - Alternatively, a *sideburn incision (see above)* with a horizontal extension, inferior to the temporal hairline, helps maintain the temporal tuft and prevents its recession or elevation. It also permits bearded hair to be moved superiorly to recreate the sideburn.
- *Preauricular incision*—A balance between preserving the non-hair-bearing skin in the preauricular area and maintaining the proper location of sideburns

- *Preauricular incision placed just posterior to sideburn*
  - Curvilinear incision following the root of the helical crus and then transitioning into the pretragal crease
  - Potential for more conspicuous scar
- *Incision in the preauricular sulcus with a posttragal extension*:
  - Less obvious scar, but may move hair-bearing skin onto the tragus, eliminating the natural area of hairlessness in the preauricular area
- *Earlobe incision*—A 2-mm cuff of tissue should be preserved around the earlobe, preventing displacement of hair-bearing skin into the preauricular sulcus, creating issues when shaving.
- *Retroauricular incision*—Standard incision (see above) used for women may result in transfer of hair-bearing skin onto the posterior aspect of the ear, necessitating shaving
  - Optimal to place the incision *within* the postauricular sulcus (AKA cephaloauricular groove) although posterior migration of the incision may occur with this design and the possibility of a visible postauricular incision should be discussed with the patient at the time of the preoperative visit.
- *Posterior scalp incision*—incision (see above) used for women with extension of the posterior incision into the hairline may result in displacement of the hairline as well as contour irregularities.
  - To reduce posterior and superior hairline displacement and stair stepping in the male patient, to place the incision in a pretrichial position along the inferior aspect of the hairline.
  - More inferiorly located incisions reduce the height of the hair-bearing flap behind the ear and the need for shaving in the superior postauricular sulcus.

Male Rhytidectomy Pearls

- Deep plane technique is the procedure of choice for men; however, facelift should be tailored to the patient's needs.
- Hair follicles in the midface and neck skin of men carry a more robust subdermal plexus, with the potential for increased bleeding:
  - More difficult to develop appropriate plane of dissection.
  - Higher rate of hematoma formation in men.
  - Allow for more subcutaneous fat on the undersurface of the flap to preserve hair follicles.
- Before any trimming, skin should be redraped and the postauricular hairline reestablished to minimize step deformities.
- Superior advancement of the skin flaps often results in redundant tissue and standing cone deformities at the temporal tuft:
  - The redundant skin inferior to the temporal tuft is excised and relationship between the sideburns and the temporal hairline is preserved.
- To prevent the shift of bearded skin into the hairless preauricular area noted with the pretragal incision, the hair follicles on the undersurface of the flap can be excised or cauterized

Adjunctive Procedures and Treatment

The Aging Neck

The presentation and etiology of the aging neck are described at the beginning of this chapter (Table 27.6). Ellenbogen and Karlin describe youthful attributes of the neck, while (Table 27.7) Dedo offers a classification schema for the aging neck. Table 27.8 describes various approaches for the management of the aging neck.

Table 27.6 Ellenbogen and Karlin—visual criteria for the aging neck

(5) Youthful neck features
Distinct, smooth inferior mandibular border with no jowl overhang
Small subhyoid depression at the cervicomental angle
Visible thyroid cartilage convexity
Distinct anterior border of the sternocleidomastoid muscle from mastoid to sternum
Cervicomental angle between 105° and 120°

Fat Transfer

- Fat transfer can be used as an adjunct to rhytidectomy or primary treatment to restore age-related facial contour changes.

Attractive, Youthful, Heart-Shaped Facial Features

- Strong, well-projected chin with a straight jawline
- A slight hollowing of the buccal area
- A full anterior and lateral cheek

Aged, Hollowed, Square-Shaped facial Features

- Loss of volume in periorbital region with a hollowed-out, cachectic appearance
- Loss of volume in the midface and temple accentuates the squaring of the upper face
- Jowl and buccal fat descent with gravity
- Volume loss in the chin and perioral region, especially at the prejowl sulcus
- Lateral jawline volume loss
- Lower face jowls promote a squaring of lower face appearance

Areas of Fat Augmentation

- Periorbital areas, nasojugal groove, malar eminence, buccal area, prejowl sulcus

Other Adjunctive Treatments

- Skin resurfacing with laser treatments or chemical peel
- Forehead-lift
- Blepharoplasty
- Rhinoplasty
- Midfacial implants and/or chin augmentation

Table 27.7 Dedo aging neck classification

Class	Description
Class I (youthful neck)	Well-defined cervicomental angle, good platysmal tone, and no submental fat
Class IIs	Simple skin laxity only
Class III	Skin laxity with excess of submental fat
Class IV	Appreciable platysmal banding
Class V (bone structure)	Retrognathic/hypoplastic mandible
Class VI (bone structure)	Low hyoid bone

Rhytidectomy Complications

Parotid Injury

- More common with sub-SMAS dissection.
- If gland parenchyma is violated, it should be cauterized to seal ductules.
- Injury may result in *sialocele* or *fistula formation*:
  - Treated with drainage, pressure dressing, and anticholinergic agent (glycopyrrolate)

Table 27.8 Rejuvenation of the aging neck

Excess skin	Subcutaneous fat	Subplatysmal fat	Low hyoid	Platysmal banding	Deficient chin
Direct excision or Lower facelift with or without platysmal tightening or Extended platysmaplasty with suspension suture or Energy-based skin-tightening technology	Suction-assisted lipectomy or Laser-assisted liposuction or Energy-based fat destruction or Judicious direct excision	Direct excision of subplatysmal fat with platysmal tightening (+/−) Digastric muscle plication and/or reduction if indicated (+/−) Submandibular gland excision	Suspension suture	Midline platysmal plication Botulinum toxin A treatment of platysmal bands (nonsurgical)	Osseous or alloplastic genioplasty or Chin augmentation using fat grafting or Orthognathic surgery if indicated

  - Botulinum toxin injection.
  - Large sialocele greater than 3 cm and persistent salivary fistulas should be treated with closed suction drainage.
- *Parotid duct injury*
  - May occur along the anterior border of the masseter on a line from the external auditory canal to the upper lip.
  - Treated with retrograde cannulation and repair under magnification.
  - Catheter removed after 2 weeks.

Facial Nerve Branch Injury

- 0.3–2.6% risk
- Spectrum of mild paresis to complete paralysis
- See section on "Facial Nerve Anatomy for the Rhytidectomy Surgeon"
- If the nerve branch is inadvertently injured, it is repaired with 10-0 nylon perineural (if possible) or epineural sutures placed under magnification.
- Assymetry due to injury of frontal branch may be addressed with neuromodulator (Botox) on the contralateral side for mild cases, or may require ipsilateral brow lift for gross asymmetry after permanent injury.
- Marginal mandibular nerve "pseudoparalysis"—Injury to the cervical branch can result in transient lip depressor dysfunction. The platysma and depressor anguli oris cofunction as lip depressors. Can be distinguished from marginal mandibular nerve injury by having patient evert lower lip. If marginal nerve intact, lip eversion occurs from functioning mentalis muscle.

Auricular Nerve and Jugular Vein Injury

- The great auricular nerve is the most frequently injured nerve in rhytidectomy, 1–7% risk:
  - Originating from second and third cervical nerves (C2 and C3).
  - Supplies sensation to the upper lateral neck and ear lobule.
  - Emerges at the posterior border of the SCM at Erb's point approximately 6–6.5 cm inferior to the external auditory canal. It circumscribes this border, and ascends in the neck in a sub-platysmal plane.
  - Distally it imparts a small postauricular branch and penetrates the parotid gland to provide its sensory innervation.
- Care is taken to avoid exposing the fascia overlying the sternocleidomastoid muscle and risking injury to the great auricular nerve and accompanying external jugular vein, which is located anterior to the nerve.
- If injured, perform direct repair of nerve and suture ligature of vein.

Spinal Accessory Nerve Injury

- May be injured with extensive elevation of the postauricular skin flap into the posterior superior cervical triangle.
- Injury is very rare although it may be injured during dissection posterior to the SCM as it lies in a fairly superficial location (with coverage only by a thin layer of subcutaneous fat and fascia) where it exits from the posterior border of the sternocleidomastoid muscle within 1–2 cm superior to *Erb's point*.
- Injury is associated with constant, deep pain, limitation of shoulder abduction and cosmetic defect resulting from unilateral trapezius atrophy.
- Management includes conservative measures including analgesics, physical therapy, electrophysiologic studies, and consideration of nerve exploration with repair.

Hematoma

- Typically occurs in first 24 h after surgery
- Most common postoperative complication 3–8%
- Associated with hypertension, post-op nausea/vomiting, coughing, agitation, poorly controlled pain, use of anticoagulants
- More common in men
- Warning signs include increasing facial edema, bruising, and pain out of proportion to procedure
- Important early detection and treatment within a few hours to avoid skin flap necrosis and subsequent scarring:

- Drain placement does not reduce risks of hematoma but may limit seroma formation and ecchymosis.
- No statistical significant reduction with the use of tissue sealants.

- *Minor hematomas* (2–10 mL)
  - Most commonly in the infra-auricular and postauricular region of the neck and amenable to evacuation in the office followed by placement of compression dressing with reexamination in 24 h
- *Major hematoma* (actively expanding)
  - 1.9–3.6% require immediate evacuation and control of hemorrhage in the OR
- *Seroma formation*
  - Usually presents 5–7 days postoperatively
  - Treated with percutaneous aspiration and placement of pressure dressing
- *Postoperative vacuum drains*—decrease seroma formation but have not been shown to have statistical impact on hematoma formation
- *Fibrin glue*—improved flap adherence with controversial reduction in hematoma or seroma formation
  - Disadvantage: Increased cost

Infection

- Unusual following rhytidectomy (<1%)
- *Staphylococcus* and *Streptococcus* species

Skin Flap Necrosis

- Etiology
  - Untreated hematoma (vascular congestion and arterial compromise)
  - Ischemia due to tobacco use *(risk increases 12-fold)*; stop tobacco at least 2 weeks before and after rhytidectomy
  - Subdermal plexus injury during dissection
  - Closure of incisions with excessive tension
- Most common at the postauricular region where skin flap is thinnest, wound tension is greatest, and blood supply is most tenuous *Partial-thickness injury* (superficial epidermolysis) heals with limited sign of scarring and/or hypopigmentation
- Full-thickness injury may lead to hypertrophic scar
- Conservative management is mainstay with topical application of nitropaste or DMSO two to three times daily, hyperbaric oxygen, and local wound care
  - Area of concern is allowed to declare fully into eschar acting as a biological dressing, with conservative serial excision
  - Judicious use of steroid injection

Scarring

- Usually due to inappropriate incision placement or excessive wound closure tension
- *Hypertrophic scar*
  - Most commonly in the retroauricular incision where skin is thinnest at the mastoid with even moderate closure tension results in scar thickening
  - Responds well to serial intralesional injections of triamcinolone
- *Satyr's ear/pixie ear*
  - Downward traction and displacement of the earlobe with obliteration of the sulcus between earlobe and cheek.
  - Due to poorly placed earlobe incision.
  - Mild deformity can be repaired with V-Y advancement at 6 months, although this can leave a visible suture line in the infra-auricular area.
  - Severe deformity should be repaired in a delayed fashion and requires undermining of peri-auricular cheek skin, scar excision, and advancement of infra-auricular skin.

Hair Loss

- Up to 8% of rhytidectomy with hair loss
- Follicle damage due to electrocautery, excess traction/tension on skip flaps
- *Telogen effluvium* (up to 8% of rhytidectomy)
  - Regrowth occurs within 4–6 months
  - Can treat with minoxidil to shorten duration
- If persistent after 12 months, consider micrografting
- *Distortion of natural hairline*
  - Insufficient anterior pivoting of postauricular flap to realign hairline

- *Inadvertent elevation/elimination of temporal hair tuft or hairline*
  - Can be prevented with the use of horizontal sideburn incision below the level of the tuft
- *Stair stepping deformity* of the postauricular hairline with marked advancement of the cervical skin
  - More noticeable with short hair and certain hairstyles

Pigmentary Changes

- Post-inflammatory hyperpigmentation
  - More commonly affects Fitzpatrick skin types IV–VI
  - May persist for months but eventually dissipates
  - Aggravated with sun exposure
  - May consider treatment with hydroquinone or other bleaching cream
- Hypopigmentation
  - Affects all Fitzpatrick skin types
  - Excessive skin tension on incision closure
  - Dermal electrocautery
  - Areas of partial- or full-thickness necrosis
- Telangiectasias
  - Patients with premorbid tendency for telangiectasias have higher occurrence postoperatively in areas of skin undermining
  - Usually resolve spontaneously in 1–2 months following rhytidectomy

    If persistent, can be treated with vascular laser (e.g., KTP or diolite lasers, both 532 nm)

Contour Deformity

- *Cobra deformity*—hollow depression above the hyoid with overly aggressive liposculpting of submentum and neck
  - Worse when platysma muscles are not sutured at midline
- Nodules and skin puckering
  - May be due to organization of localized hematomas not aspirated or unrecognized contour deformities of the underlying deeper tissue layers prior to skin redraping
  - Small deformities resolve over months with faster recovery with massage and steroid injection
  - Dermal filler or autologous fat injection can be used to smooth irregularities

Systemic Complications

- 0.1–0.5% risk
- DVT, PE, need for blood transfusion, major anesthetic complication, death
- Events more likely with general anesthesia compared with local anesthesia with IV sedation

Depression

- Short-term depression can occur in up to 30% of women after rhytidectomy
- More likely in patients with preexisting depression
- Duration usually 1 month after surgery
- Reassurance/emotional support and occasional short course of antidepressant may be warranted

Rhytidectomy Multiple Choice Questions

1. Most anterior point on the bony chin in the midline is the:
   (a) Porion
   (b) Prosthion
   (c) Pogonion
   (d) Gnathion
   (e) Menton
2. Which of the following intrinsic changes due to senescence is FALSE?
   (a) Thinning epidermis
   (b) Widening of the dermal–epidermal junction
   (c) Atrophy of the dermis
   (d) Atrophy of the subdermal adipose tissue
3. Which of the following processes is caused by *photoaging*?
   (a) Increased overall density of melanocytes
   (b) Infiltration of Langerhans cells into sun-exposed skin
   (c) Accumulation of elastin material in the subdermal tissues
   (d) Fragmentation of collagen and elastin fibers
4. Periorbital changes due to aging include:
   (a) Increased orbital width

(b) Increase in height of the superior orbital rim laterally
(c) Decrease in height of the superior orbital rim medially
(d) Inversion of the eyelid margin
(e) (a), (b), (c)
(f) All of the above

5. The “Witch’s chin” aging deformity is in part the result of:
(a) The lower teeth becoming more visible from gravitational effects
(b) An overprojecting of the mandible
(c) Restoration of mandibular height with dentures
(d) A prominence of premental fat

6. The major osseocutaneous retaining ligaments of the face are the:
(a) Parotid ligaments
(b) Masseteric ligaments
(c) Zygomatic ligaments
(d) Mandibular ligaments
(e) (a), (c)
(f) (c), (d)
(g) (a), (d)
(h) All of the above

7. The danger zone for the frontal branch of the facial nerve over the zygoma is:
(a) 1.2 cm anterior the helical root and 2.2 cm posterior to the lateral canthus
(b) 1.0 cm anterior the helical root and 1.6 cm posterior to the lateral canthus
(c) 1.8 cm anterior the helical root and 2 cm posterior to the lateral canthus
(d) 1.5 cm anterior the helical root and 2.5 cm posterior to the lateral canthus
(e) Cannot be predicted

8. A patient is scheduled for a facelift consultation. During the consultation it is identified that the patient is an active smoker. Your discussion as to why you will not perform his procedure until he or she has stopped smoking for 2–4 weeks includes:
(a) A fivefold increased risk of flap necrosis
(b) A sevenfold increased risk of flap necrosis
(c) A tenfold increased risk of flap necrosis
(d) A 12-fold increased risk of flap necrosis
(e) A 17-fold increased risk of flap necrosis

9. During facelift consultation and examination, you identify several physical features that are favorable for facelift. Which of the following is an unfavorable feature?
(a) Sharp cervicomental angle
(b) Thin skin with good tone
(c) Full midface
(d) Shallow cheek grooves
(e) Prominent cheeks

10. What is a safe plane of dissection above the zygomatic arch to avoid the frontal branch of the facial nerve?
(a) Just superficial to the temporoparietal fascia
(b) Subperiosteal
(c) Just superficial to the deep temporal fascia
(d) Subcutaneous
(e) (b), (c)
(f) (b), (c), (d)
(g) There is no safe plane

11. Which branch of the facial nerve is at risk during a subperiosteal cheek lift?
(a) Temporal
(b) Zygomatic
(c) Buccal
(d) Marginal
(e) Cervical
(f) There is no risk to the facial nerve during a subperiosteal approach

12. Which procedure can help prevent a cobra deformity from developing in a neck lift?
(a) Completion of aggressive lipocontouring
(b) Performing a corset platysmaplasty
(c) Resection of the digastric musculature
(d) Aspiration of all localized hematomas

13. Skin flap necrosis can be avoided by:
(a) Having a patient stop smoking 1 week prior to surgery
(b) Performing a deep plane facelift
(c) Treating all hematomas immediately
(d) Closure of the wounds with no tension
(e) (b), (c), (d)
(f) All the above

14. The most common complication following facelift is hematoma. Which of the following

can be done to prevent hematoma formation?
(a) Postoperative vacuum drains under skin flaps
(b) Routine use of fibrin glue on skin flaps
(c) Aggressive treatment of post-operative pain, nausea and vomiting
(d) Have patient stop smoking 4 weeks prior to surgery
(e) All the above

15. What is the most common nerve injured during rhytidectomy?
(a) Marginal branch of facial nerve
(b) Frontal branch of facial nerve
(c) Great auricular nerve
(d) Spinal accessory nerve
(e) Zygomatic branch of facial nerve

16. A 57-year-old female patient is being seen in the clinic for consideration for neck lift. It is noted that she has skin laxity with moderate platysmal banding. Which Dedo aging neck classification does she fall into?
(a) I
(b) II
(c) III
(d) VI
(e) V
(f) VI

17. Visual criteria for the aging neck include all of the following, except:
(a) Small subhyoid depression at cervicomental angle
(b) Visible thyroid cartilage concavity
(c) Distinct anterior border of the SCM muscle
(d) Cervicomental angle between 105° and 120°
(e) Smooth inferior mandibular border with no jowl overhang

18. A disadvantage to the supra-SMAS approach to the midfacelift is:
(a) Increasing the horizontal width of the face
(b) Lifting the oral commissure
(c) Reduction of skin vascularity of the face
(d) Greater postoperative edema
(e) Hypesthesia of the malar eminence

19. A 35-year-old patient comes in because she is dissatisfied with the appearance of her neck. You classify her neck as a Dedo class VI. What is the best surgical treatment for this patient?
(a) Direct excision of subplatysmal fat
(b) Suction-assisted lipectomy of subcutaneous fat
(c) Botulinum toxin injection of platysmal banding
(d) Suture suspension of the hyoid bone
(e) Chin augmentation using fat grafting

20. Management of a post-facelift sialocele includes which of the following:
(a) Pressure dressing
(b) Botulinum toxin
(c) Incision and drainage
(d) Revision facelift
(e) (a), (b), (c)

21. Which of the following medications can be used to shorten postoperative telogen effluvium after facelift?
(a) Botulinum toxin
(b) Finasteride
(c) Minoxidil
(d) Herbal supplementation
(e) Baby shampoo

22. Which of the following is not part of the standard photographic views of a facelift patient?
(a) Oblique, neutral gaze
(b) Lateral, neutral gaze
(c) Lateral, neck in flexion
(d) Full-face front, smiling
(e) Oblique, smiling
(f) Full-face front, neutral gaze

23. The SMAS is contiguous with:
(a) The galea
(b) The temporoparietal fascia
(c) The superficial layer of the deep temporal fascia
(d) The platysma
(e) The parotidomasseteric fascia
(f) A,B
(g) A,B,D
(h) A,B,C,E
(i) B,D

24. A cosmetically pleasing mentocervical angle is:
    (a) 60°
    (b) 70°
    (c) 85°
    (d) 100°
    (e) 110°
25. What is McGregor's patch?
    (a) Parotid cutaneous ligaments
    (b) Masseteric cutaneous ligaments
    (c) Zygomatic cutaneous ligaments
    (d) Mandibular cutaneous ligaments

Answers

1. (c)
2. (b)
3. (d)
4. (a)
5. (d)
6. (f)
7. (c)
8. (d)
9. (b)
10. (f)
11. (c)
12. (b)
13. (e)
14. (c)
15. (c)
16. (d)
17. (b)
18. (c)
19. (d)
20. (e)
21. (c)
22. (e)
23. (g)
24. (d)
25. (c)

Index

A
AbobotulinumtoxinA, 307
Acellular dermis (Alloderm), 77
Acellular human dermal matrix, 280
Acellularized dermal matrix, 385
Acral lentigo, 123
Adipocyte hyperplasia, 362
Adipocyte hypertrophy, 362
Adipose tissue, physiology of, 362
Advancement flaps, 80, 139
Aesthetic surgery, lasers
  Alexandrite, 354
  complications, 355
  epidermal cooling, 353
  intense pulsed light, 354
  laser hair removal, 357
  Nd:YAG, 354
  non-laser technologies, 357
  PDL, 354
  preoperative evaluation, 354, 355
  scar treatment, 356
  science, 351, 352
  skin resurfacing, 356
  tattoo removal, 356, 357
  tissue interaction, 352
  treatment preparation, 355
  variables, 352, 353
  vascular malformations/lesions, 356
Aging face, 396
  age-related pigmentation changes, 304
  botulinum toxin, 305, 306
  cheiloplasty, 277, 278
  chronologic aging, 304
  extrinsic factors, 304
  facial muscles over time, repetitive use of, 304
  major facial folds, mechanisms, 305
  periorbital changes due to, 412
  rhytidectomy essentials
    alteration of aesthetics with, 396
    brow/forehead, 396
    chin, 398
    earlobes, 398
    jowls, 397
    microscopic skin changes, 396
    midface, 397
    neck, 397, 398
    perioral area, 398
    periorbital area, 396, 397
  signs and symptoms, 303
Aging lip, 278
Aging neck
  rejuvenation of, 409
  rhytidectomy, 408
Airway hemangiomas, 234
Alar base, 373
Alar–columellar relationship, 374
Alar reduction techniques, 379
Alar retraction, 387
Alar–rim columellar relationship, 392
Alar rim grafts, 377
Alar strut graft, 389
Alexandrite, 354
Alloderm, 385
Alloplast graft materials, 385, 386
Alloplastic chin augmentation, 333
Alloplastic implants, 333, 334
Alloplastic materials polytetraflouethylene, 389
Alloplastic mentoplasty, 329, 330
Alloplast infection, 392
Alopecia areata, 338
Alopecia reduction, 346
Alpha-hydroxy acid (AHA), superficial chemical peel, 289
Anagen, 338
Anagen effluvium, 339
Anatomical implants, 334
Anatomic osteotomies, 382
Androgenic alopecia (AGA), 338
Angle Class II malocclusion, 333
Angle's classification, 323
Anterior compartment, 361
Anterior digastric muscles, 363
Anterior plagiocephaly, 208
Arrhythmia, 401
Arterial supply, 371
Arteriovenous malformations (AVMs), 239
Articulare, 395
Atrophic dermis, 396

© The Author(s) 2021
B. J.-F. Wong et al. (eds.), *Facial Plastic and Reconstructive Surgery*,
https://doi.org/10.1007/978-3-030-45920-8

Atrophy, 399
ATX-101, 310
Atypical fibroxanthoma, 135, 136
Augmentation cheiloplasty, 278, 279
  autologous materials, 279
  fillers, 279
  implants, 280
  vermillion advancement, 280
Auricular cartilage, 215
Auricular nerve and jugular vein injury, 410
Auricular reconstruction, 94
  ALT free flap, 98
  Brent technique, 95
  deltopectoral flap, 96, 97
  free tissue transfer, 97
  gracilis muscle flap, 98
  latissimus dorsi free flap, 96, 98
  Nagata technique, 95
  pectoralis major flap, 95–96
  radial forearm free flap, 97, 98
  rectus abdominus free flap, 98
  regional flaps, 95
  submental island, 97
  tissue engineering, 95
  Trapezius flap, 96
Autogenous graft materials, 384, 385
Autologous fat transfer, 366
Automated harvest, 344
Autospreaders, 380
Azelaic acid, 293

B

Baker-Gordon deep chemical peel, 290–291, 294, 295
Baker's strip (Lateral) SMASectomy, 402
Bannayan-Riley-Ruvalcaba syndrome, 240
Barbed sutures, 405
Base, rhinoplasty, 376, 377
Batten grafts, 383
Beckwith-Wiedemann syndrome, 240
Bernoulli principle, 372
Bikini lip reduction, 281
Bilateral cheek advancement flaps, 92, 93
Bilateral cleft repair, 189–190
Bilateral mucoperichondrial flaps, 384
Bilateral nasal obstruction, 390
Bilobe flap, 82, 83, 139
Biological creep, 318
Biologic injectable fillers, 307–308
Bipedicled mucosal advancement flaps, 86
Blepharochalasis, 397
Blepharoptosis, 54–55, 397
Blood supply, 299, 302, 361
Blunted labiomental fold, 332
Body dysmorphic disorder (BDD), 70
Bogorad's syndrome, 48, 59
Bone and cartilage reconstruction
  bucket handle flap, 86
  contralateral septal mucoperichondrial hinge flap, 86
  hinge flap, 86
  intranasal mucosal flaps, 86
  pivot flap, 87
Bony base, 373
Bone-calvarial, 385
Bony chin, 412
Bone grafts, 76–77
Bony genioplasty, 332
Bony reconstruction
  calvarial bone graft, 143
  free tissue transfer
    fibula, 143
    iliac crest, 143
    scapula, 143
  iliac bone graft, 143
Bony vault, 381
Bossae deformity, 386
Botulinum toxin A (Botox®), 278, 283, 305–307, 315
Bovine, 279
Bovine collagen, 315
Brachycephaly, 210
Branchial apparatus, 50
Brown skin, 294
Browplasty
  browpexy, 269
  chemical brow lift, 266
  complications, 270
  coronal forehead lift, 266, 267
  direct brow lift, 269
  endoscopic brow lift, 270
  men, 265
  midforehead brow lift, 269
  preoperative analysis, 265, 266
  surgical indications, 267
  temporal lift, 268, 269
  trichophytic forehead lift, 268
  women, 264, 265
Brow ptosis, 396
Brow-tip aesthetic line, 373
Bulbosity of tip, 373
Bullhorn cutaneous excision, 282
Burstone, 324
Butterfly conchal cartilage graft, 380

C

Calcium hydroxyapatite (CaHA), 279, 283, 311, 315
Calcium phosphate, 311
Calvarial bone graft, 391
Canthopexy vs. canthoplasty, 112
Cap grafts, 378
Cardiac arrhythmias, phenol face peeling, 292
Cartilage grafts, 76
Catagen, 338
Caudal septal deflection, 383
Caudal septal extension graft, 376
Cauliflower ear, 218
Cephalic malpositioned lower lateral cartilages, 387
Cephalic trim, 389
Cephalogram points, 328
Cephalometric analysis, 333

Cephalometry, 321
Cervical plexus, 302
 liposuction and, 361
Cervicofacial advancement rotation flap, 80, 81, 139
Cervicofacial liposuction, 367–369
Cheek reconstruction
 buccal subunit, 88
 cheek-nose deformities, 87
 dermal-periosteal sutures, 87
 incisions, 87
 lateral cheek lesions, 88
 medial cheek lesions, 87
 zygomatic subunit, 88
Cheiloplasty
 aging, 277, 278
 analysis and physical exam, 277
 anatomy
  arterial supplyrs, 275
  innervation, 275
  limits, 275
  tissue layers, 275
  venous outflow, 275
 augmentation, 278, 279
  autologous materials, 279
  fillers, 279
  implants, 280
  vermillion advancement, 280
 Cupid's bow, 276
 embryology, 276
 frontal view, 277
 histology, 276
 lateral view, 277
 philtrum, 276
 reduction, 281
  bikini lip reduction, 281
  complications, lip procedures, 281
  vermillion, 281
 relaxed skin lines, 276
 treatment
  lip augmentation, 278
  rhytid, 278
 vermillion, 276
 white roll/line, 276
Chemical peels
 classification of, 288
 complications of, 293, 294
 deep peel, 290
  Baker-Gordon deep chemical peel, 290, 291
  Hetter's peel, 291
 dermis, 285
 epidermal cell types, 285
 herpes simplex, 292
 histologic changes, 286
 inflammatory phase, 286
 layers, 285
 maturation phase, 286
 medium-depth peel, 289, 290
 melasma, 292, 293
 patient selection, 286, 287
 phenol face peeling, cardiac arrhythmias in, 292
 post operative care, 291, 292
 post-radiation, 292
 proliferative phase, 286
 prolonged postpeel erythema, 292
 skin analysis, 285
 skin changes with aging, 286
 superficial layers, 288
  alpha-hydroxy acid, 289
  Jessner's solution, 289
  removal of, 286
  salicylic acid, 289
  TCA, 288
 tretinoin, 293
Chin, 323–325, 332, 389
 assessment of, 326
 rhytidectomy, 398
 surgery, 332
Chromophores, 353
Chronologic aging, 304
Chrysiasis, 354
Cimetidine, 343
Circumferential reduction, 346
Class III skeletal jaw relationship, 334
Cleft lip and palate
 alveolar bone grafting, 196, 197
 arterial supply of palate, 186
 bilateral CL, 185
 bilateral cleft nasal deformity, 190
 bilateral cleft repair, 189–190
 cleft palate in isolation, 181
 complete CP, 185
 complete unilateral CL, 184
 comprehensive management plan, 181
 ectrodactyly-ectodermal dysplasia-clefting syndrome, 183
 embryology, 183
 environmental etiology, 182
 epidemiology, 181–182
 frontonasal prominence, 183
 gingivoperiosteoplasty, 187
 head bonnet, 187
 incomplete CP, 185
 incomplete unilateral CL, 184
 intermediate rhinoplasty, 191
 latham appliance, 187
 lip adhesion procedure, 187
 lip development, 183
 lip repair, 188
 maxillary prominences, 184
 microform CL, 185
 Millard rotation-advancement flap, 188
 muscular and vascular anatomy, 185–186
 NAM, 187
 nasal deformity treatment, 190–191
 nasal placodes, 183
 orbicularis oris, 185, 186
 with or without palate (CL ± P), 181
 palate development, 184
 Pierre Robin Sequence (PRS), 182
 popliteal pterygium syndrome, 183

Cleft lip and palate (*cont.*)
presurgical infant orthopedics (PSIO), 187
of primary palate, 185
primary rhinoplasty, 191
prosthetic therapy, 196
22q12 deletion, 183
repair techniques
complications, 194
Furlow (double-opposing Z-Plasty), 192, 193
two-flap palatoplasty (Bardach), 192, 193
Von Langenbeck, 191, 192
V-Y pushback/three flap, 192, 194
secondary defects correction, 197
of secondary palate, 185
septorhinoplasty, 191
Stickler syndrome, 182
submucous cleft palate, 185
taping, 187
Tennison-Randall repair, 189
treatment, 186–187
unilateral cleft nasal deformity, 190
Van der Woude syndrome, 182
Veau classification, 185
velocardiofacial syndrome, 183
velopharyngeal dysfunction, 195
causes, 194
configuration of VP closure, 195
speech therapy, 195
surgical therapy, 195, 196
treatment, 195
Cleft lip nasal deformity, 389, 391
Cobra neck deformity, 363, 412, 413
Cocaine, 11–12, 21, 23
Coherence, 352
Collagen, 295
Collimation, 352
Columellar-lobule junction, 371
Columellar strut graft, 376
Complete strip method, 377
Composite grafts, 87, 385
Conchal bowl reconstruction, 94
Conchal cartilage, 384
Conchal hypertrophy correction, 220
Concha-mastoid suturing, 219, 220
Concho-scaphal suturing, 218, 219
Condylion, 395
Contour deformity, 412
Contraction of subdermis, 362
Contralateral septal mucoperichondrial hinge flap, 86
CoolMini applicator, 367
CoolSculpting®, 367
Cosmetic rhinoplasty, 382
Costochondral, 385
Craniofacial anomalies
anatomy, 206
blood supply, 206
dental occlusion
class I, 207
class II, 207
class III, 207
craniosynostosis, 207–208
cross bite, 207
orthognathic surgery, 207
overbite, 207
overjet, 207
sagittal split osteotomy, 207
upper airway obstruction, 207
VATER association, 207
vertical-subcondylar osteotomy, 207
sagittal synostosis
endoscopic assisted strip craniectomy with wedge craniectomy, 211
fronto-orbital advancement with reshaping, 211, 212
Pi procedure, 211
posterior synostotic plagiocephaly treatment, 212
suturectomy, 211
skull deformities, 209
anterior plagiocephaly, 208
brachycephaly, 210
cloverleaf skull, 210, 211
deformational plagiocephaly, 208
distraction osteogenesis, 211
posterior plagiocephaly, 210
scaphocephaly, 208
timing of surgical procedures, 211
trigonocephaly, 208
skull embryology, 206
Craniosynostosis, 207, 208
Crooked nose, 382, 391
rhinoplasty, 384
Cross-hatching technique, 316
Croton oil, 291
Crumley-Lanzer method, 389
Cryolipolysis, 367, 368
Cryosurgery, 124
Cupid's bow, 276
Cutaneous angiosarcoma, 133
Cutler-Beard bridge flap, 89
Cyproterone acetate, 343

D

Dantrolene, 22
Dardou flap, 347
Dedo aging neck classification, 409, 414
Dedo classification of cervical abnormalities, 363
Deep cervical fascia, 361
Deep chemical peel, 290
Baker-Gordon deep chemical peel, 290, 291
Hetter's peel, 291
Deep plane rhytidectomy, 399, 402, 403
Deformational plagiocephaly, 208
Deoxycholic acid (Kybella) injection, 317, 367
Depressions, 365
Depressor labii inferioris, 322
Depressor septi muscle, 374
Dermal flaps, 278, 282
Dermal grafts, 76
Dermatochalasis, 397

Dermatofibrosarcoma protuberans (DFSP), 136
Dermis, 151, 285, 318
  chemical peels, 285
Dermis-fat, 279
Diced cartilage/fascia grafts, 380
Digastric muscle, 322
Distraction osteogenesis, 211
Domal creation sutures, 377
Domal junction, 371
Donor site harvest, 343
Donor strip harvesting, 343, 344
Dorsal augmentation, 380, 381
Dorsal onlay grafts, 380, 391
Dorsal reduction, 379, 380
Double break appearance, 373
Double osteotomies, 382
Double Y-V mucosal plasty, 280, 282
Dufourmentel modification, 82
Dysphagia, 320

E
Ear
  anatomy, 215–217
  arterial supply, 216
  embryologic development, 217
  innervation, 216
  lymphatic drainage, 216
  otoplasty (*See* Otoplasty)
  reconstruction, 144
  relationships and angles, 217–218
  structural support, 216, 217
  venous drainage, 216
Earlobe, rhytidectomy, 398, 406, 407
E-commerce, 66
Ectrodactyly-ectodermal dysplasia-clefting syndrome, 183
Ectropion, 397
Elastic modulus (G') of filler, 306
Electromagnetic spectrum, 351
Ellenbogen and Karlin—visual criteria for the aging neck, 408
Embryology, 205–206
Emergency Medical Treatment and Active Labor Act of 1986, 64
Endonasal rhinoplasty, 375
Entropion, 397
Epidermal cooling, 353
Epidermal thinning, 368
Epidermis, 151, 318
Epinephrine, 365
Erythema ab igne (EAI), 304
Estlander flaps, 92
Ethical issues
  autonomy, 65
  beneficence, 65
  doctor–patient relationship, 65
  justice, 65
  medical errors, 65
  non-maleficence, 65
Evidence-based medicine (EBM)
  application of results in practice, 42
  asking an answerable clinical question, 41
  channeling bias, 44
  citation/reporting bias, 45
  clinical recommendations, 42
  confounding, 45
  critical appraisal of evidence, 41
  design bias, 44
  evaluation of performance, 42
  experimental study, 42, 44
  information bias, 44–45
  levels of evidence, 41, 42
  meta-analysis, 42
  observational study, 44
  precision, 44
  selection bias, 44
  systematic retrieval of best evidence, 41
  systematic review, 42
Excess eyelid skin, 397
Expanded polutetrafluoroethylene (EPTFE), 77, 280, 315
Expanded-porus polytetraflouoethylene, 385
Extended bilateral spreader grafts, 383
External carotid artery, 299
External demarcation, 276
External nasal valve, 372
External nose, arterial anatomy of, 387
External pump, 352
Extracorporeal septoplasty, 383
Eye injury, 355
Eyelid
  anatomy
    arterial supply, 247
    capsulopalpebral head/fascia, 247
    fat compartments, 248
    lamella, 248
    layers, 248
    levator palpebrae superioris muscle, 247
    lymphatic drainage, 247
    motor nerve innervation, 247
    Muller's muscle, 248
    orbicularis oculi muscle, 247
    orbital septum, 247
    sensory nerve innervation, 247
    venous outflow, 247
  Asian eyelid, 248
  Barton tear trough triad, 251
  blepharoplasty, contraindications, 249
  blepharoptosis repair, 250, 254, 255
  brow and eyelid relationship, 250
  brow position, 250
  brow ptosis, 250
  canthopexy, 254
  canthoplasty, 254
  complications
    conjunctivitis/chemosis, 256
    diplopia, 256
    entropion, 257–260
    infections, 256
    lagophthalmos, 256

Eyelid (*cont.*)
lateral canthal rounding, 257
oversculpted/hollowed appearance, 256
retrobulbar hematoma, 255–256
suture granuloma/pyogenic granuloma, 256
tearing/epiphora, 256
webbing of medial upper eyelid incision, 256
double-convexity deformity, 251
ectropion repair
cheek fat pad elevation, 255
lateral canthal tendon tightening, 255
spacer graft insertion, 255
subperiosteal midface elevation, 255
fat preseveration (repositioning/mobilization), 253
globe-orbital rim relationship, 251
lacrimal system, 250
lateral canthus, 251
levator function, 250
lid distraction test, 251
lid margin, 250
ligaments of orbital support
arcus marginalis, 248
Lockwood's ligament, 248
orbicularis retaining ligament, 249
Whitnall's ligament, 248
loss of elastic fibers of skin, 249
lower eyelid surgical approaches, 252, 253
lower lid blepharoplasty, 254
lower lid laxity, 251
Malar fat pad, 251
margin reflex distance-1 (MRD-1), 250
margin reflex distance-2 (MRD-2), 251
non-Asian eyelid, 248
orbicularis festoons/cheek bags, 251
orbicularis oculi muscle hypertrophy, 249
orbital septal tightening, 254
orbital septum, 249
preoperative photos, 252
prophylactic lateral canthal anchoring, 254
recognize lateral hooding, 250
retro-orbicularis oculi fat, 250
snap test, 251
suborbicularis oculi fat, 250
supratarsal crease creation/asian blepharoplasty, 254
suspensory ligaments, 249
tear trough deformity, 251
upper eyelid crease, 250
upper eyelid surgical approaches
transconjunctival approach, 252
transcutaneous approach, 252
Eyelid reconstruction, 145
Cutler-Beard bridge flap, 89
full-thickness defects, 88, 89
Hughes tarsoconjunctival flap, 89, 90
intraoperative recognition, 88
medial canthal defects, 89
superficial defects, 88
Tenzel semicircular flap, 89
upper and lower eyelid, 88

F
Face
fat compartments of, 303, 305
horizontal thirds of, 324
retaining ligaments of, 398
soft tissue envelope of, 299
Facial aesthetic units and subunits, 299
Facial aging, 362
Facial artery, 299
Facial augmentation, 315
Facial expression, major muscles of, 300–302
Facial fat compartments, 303
Facial implants, 315
Facial nerve, 48, 333, 362, 413
danger zone, frontal branch, 413
innervation, 368
rhytidectomy surgeon, 399, 400
Facial nerve branch injury, rhytidectomy, 410
Facial paralysis and reanimation
Bell's palsy, 109–111
buccal and zygomatic branches, 108
cervical branch, 108
corneal protection, 111
cross facial nerve graft, 113, 114
Davis classification, 107–108
electrodiagnostic testing, 111
etiologies, 108
eyelid procedures, 111–117
gracilis-free tissue transfer, 114
House-Brackmann scale, 108, 109
hypoglossal nerve transfer, 113
main trunk location, 107
marginal mandibular branch, 108
nerve grafts, 113
nerve injury, Sunderland classification, 108–111
nerve to masseter transfer, 113
primary nerve repair, 113
static resuspension, 113
Sunnybrook facial grading system, 109, 110
superficial temporal artery, 108
surgical management, 112, 113
synkinesis
definition, 112
natural history of, 112
prognosis, 112
temporal (frontal) branch, 107–108
temporalis muscle transfer, 114
temporalis tendon transposition, 114
Facial rejuvenation, 357, 366
Facial trauma
acute life-threatening injuries, 161
airway management, 161
angle fractures, 172
anterior table frontal sinus fracture, 176
body and parasymphyseal fractures, 171
bone healing, 163
cerebral trauma, 175, 177, 213
closed treatment (MMF), 170
complications, 172

- condylar fracture treatment, 175
- fracture classification, 170–172
- fracture favorability, 170
- fracture fixation, 163
- frontal sinus fractures, 167, 175
- hemorrhagic shock, 161
- large anterior maxillary wall defect, 176, 177
- mandible fractures, 170
- maxillary/Le Fort fractures, 169–170
- nasoethmoid fractures, 177
- naso-orbito-ethmoid fractures, 166
- non-occlusal complications, 175
- pan-facial fractures, 172–173, 176, 177
- pediatric facial fractures, 173–177, 213
- primary survey, 161
- ramus fractures, 172
- soft tissue injuries
  - bite wounds, 162
  - eyelid, 163
  - facial nerve, 162
  - lacerations, 161, 162
  - nasolacrimal duct, 163
  - parotid gland, 162
- subcondylar fractures, 171
- surgical approaches
  - bicoronal approach, 164, 165
  - endoscopic browlift approach, 165
  - mandible, 163–164
  - maxilla, 164
  - orbit, 164
- symphyseal fractures, 172
- transcaruncular and inferior fornix approach, 175, 176
- treatment, 170
- ZMC fractures, 165–166, 175, 176

Fascia, 380
Fascia-perichondrium, 385
Fat augmentation, 279, 317
Fat grafting, 77, 283
Fat transfer, rhytidectomy, 409
Female pattern baldness, 340
Fibroblasts, 285
Fillers, augmentation cheiloplasty, 279
Finasteride, hair restoration, 342
Fine rhytides, 398
Fitzpatrick classification, 30, 287
Flap delay, 79, 140
Flaps
- advancement flaps, 80
- arterial vascularization results, 78
- bilobe flap, 82, 83
- cervicofacial advancement rotation flap, 80, 81
- classification, 77
- delay, 79
- hyperbaric oxygen, 79
- interpolated flaps, 83, 84
- ischemic flaps, 79
- island flaps, 84
- local flaps, 77, 78
- melolabial/nasolabial flaps, 84
- necrosis and complication rates, 78
- random pattern flaps, 78
- rhombic flaps, 81, 82
- rotational flaps, 80, 81
- tissue expansion, 79, 80
- transposition flaps, 81
- venous stasis, 78

Flaring sutures, 387
Follicular unit extraction (FUE), 337, 338, 344, 345
Follicular unit grafting (FUG), 343–345
Forehead anatomy, 263–264
Forehead and temple reconstruction, 93
Frankfort horizontal line, 324, 333
Frey’s syndrome, 48, 59
Frontal sinus fractures, 167
Full-thickness skin grafts (FTSG), 75, 76
Furlow (double-opposing Z-plasty) palatoplasty, 195

G

Galea fixation, 345
Geniohyoid, 322
Genioplasty, 321
- anatomy
  - depressor labii inferioris, 322
  - digastric muscle, 322
  - geniohyoid, 322
  - mentalis muscle, 322
  - mylohyoid, 322
  - platsysma, 322
- Angle’s classification, 323
- cephalometric imaging, 328
- chin, 323, 324
- complications, 331, 332
- interlabial distance, 327
- labiomental fold, 327
- lip, 327
- neck, 327, 328
- nose, 327
- occlusion, 322, 323
- physical exam, 322
- post-operative care and recovery, 331
- surgery, 329
  - submental, 329
  - transoral, 329
- vertical analysis, methods of, 327

Gentamycin, 316
Gilles fan flap, 92
Gingivoperiosteoplasty, 187
Glabella, 373, 395
Glogau skin classification scheme, 287
Glycolic acid, 294
Gnathion, 396
Gonzales-Ulloa line, 324, 374
Gore-Tex, 77, 385, 389
Graft extrusion, 345
Graft insertion, 344, 345

Grafts
alloplasts, 77
bone grafts, 76–77
cartilage grafts, 76
dermal grafts, 76
fat grafts, 77
full-thickness skin grafts, 75, 76
split-thickness skin graft, 75, 76
vascularization, 76
Granuloma formation, 317
Great auricular nerve, 410, 414
Gruber sutures, 377
Gull-in-flight configuration, 373

H
Hair cycle, 348
Hair follicle, 337
Hairline placement, 343
Hair loss, 348
alopecia areata, 339
anagen effluvium, 339
androgenic alopecia, 338
rhytidectomy, 411, 412
scarring alopecias, 339
telogen effluvium, 339
triangular alopecia, 339
in women, 342, 343
Hair restoration, 337
alopecia, behavioral causes of
traction alopecia, 339
trichotillomania, 339
classification systems
Ludwig Classification of female pattern baldness, 340
Norwood Classification for hair loss, 340
complications, 347
drugs, 339
evaluation, 340
follicular unit, 337
hair follicle, 337
hair growth cycle, 338
hair loss, etiology of
alopecia areata, 339
anagen effluvium, 339
androgenic alopecia, 338
scarring alopecias, 339
telogen effluvium, 339
triangular alopecia, 339
infectious causes, 339
inflammatory causes, 339
laboratory testing, 340
medical treatment
finasteride, 342
hair loss, women, 342, 343
minoxidil, 342
physical exam, 340
scalp layers, 337
surgical treatment
automated harvest, 344
donor site harvest, 343
FUE, 344, 345
FUG, 343–345
graft insertion, 344, 345
hairline placement, 343
hair transplantation, 343
scalp flaps, 346, 347
scalp reductions, 345, 346
vascular supply, 337
venous outflow, 337
Hair transplantation, 343, 348
Hamra's composite rhytidectomy, 403
Hamra's deep plane rhytidectomy, 402, 403
Hanging columella, 387
Harvesting autologous fat, 369
Head and neck
adjunctive therapies, 57
aesthetic subunits, 60
alimentation/nutrition, 55–56
arterial plexuses, 47
blepharoptosis, 54–55
Bogorad's syndrome, 59
branchial cleft cyst, 60
buttresses, 49
camera terminology, 52, 53
cephalometric diagnosis, 59
cephalometric evaluation, 55
complications
informed consent, 58
management, 58
standard precautions, 58
tobacco morbidity, 57–58
universal protocol, 58
congenital blepharoptosis, 59
craniofacial syndromes, 60
deep cervical fascia, 49
deep neck space infections, 59
dental anatomy, 55
differential diagnosis formation, 52
embryologic development, 50
eyelid morphology, 49
facial divisions, 54
facial nerve, 47, 48
facial plastic surgery, 60
facial subunits, 53
Fitzpatrick's classification, skin types, 54
Frey's syndrome, 59
Glogau photoaging classification, 54
hairline evaluation, 54
hemostasis, 56–57
horizontal buttresses, 58
imaging, 51, 59
lacrimation, 51
lymphatics, 48
management algorithm, 52
mastication/deglutition, 50
micro anatomy/pathology, 49–52
musculature, 49
nasal morphology, 49
orbit bones, 49

phonation, 51
photographic consent, 52
preoperative medications, 57
pterygopalatine ganglion, 59, 60
rhinomanometry, 51
salivary gland, 60
Schirmer test, 51, 52
sensory and autonomic, 48
skin tension lines, 53–54
soft-tissue landmarks, 53
soft-tissue layers, 58
specialized structures, 49
speech (articulation), 50
superficial cervical fascia, 48, 58
tissue expansion, 313
tissue layers/fascial compartments, 48
type I and type II first branchial arch anomalies, 60, 61
venous, 47
wound dressings, 57
wound healing, 56, 59, 60
Helical rim reconstruction, 94–95
Hematoma, 410, 411, 413
Herpes simplex, chemical peels, 292
Hetter formula, 291
Hinge flaps, 86, 139
Homologous graft materials, 385
Horizontal osteotomy, 330
House-Brackmann scale, 108, 109
Hughes tarsoconjunctival flap, 89, 90
Hyaluronic acid fillers, 279, 315, 319
Hydroquinone, 293
Hyperpigmentation, 295, 358, 359
Hypertrophic scar, 411
Hypertrophy, 398
Hypopigmentation, 358, 412
Hypoplasia, 399

I

Ideal chin projection, 363
Implants, cheiloplasty augmentation, 280
Incisions
male rhytidectomy, 407, 408
rhinoplasty, 375
IncobotulinumtoxinA, 307
Inconspicuous cervicofacial liposuction incisions, 367
Infantile hemangioma
characteristics, 233
complications, 233, 234
disseminated lesions, 233
focal tumors, 233
medical therapy, 234, 235
corticosteroids, 235
hemangeol, 235
lasers, 235, 236
propranolol, 234, 235
topical beta blockers, 235
vincristine, 235
observation, 234
overview, 231
pathology, 233
surgery, 236
treatment, 234
Information technology (IT) issues
AMA guidelines, 66
legal and security issues, 65–66
online ethics, 66
social media, 66
Informed consent, 63–64
Infradentale, 396
Infraorbital nerve, 275
Injectable volumizing fillers
ATX-101, 310
classification and properties, 306, 308
complications, 308, 309
ideal filler, 306
indications, 306
nasal fillers, complications of, 309
nonsurgical rhinoplasty, 309
ocular complications from, 309, 310
technique and plane of injection, 308
Injection of steroid, 317
Intense pulsed light (IPL), 354, 358
Intercartilaginous and marginal incisions, 390
Interdomal sutures, 377
Interlabial distance, 327
Intermediate osteotomies, 382
Internal carotid artery, 302
Internal demarcation, 276
Internal maxillary artery, 299
Internal nasal valve
narrowing, 389
rhinoplasty, 372
Internal tissue expanders, 313
International Society for the Study of Vascular Anomalies (ISSVA), 231
Interpolated flaps, 83, 84
Interposition graft, 384
Intravelar veloplasty, 195
Inverted V deformity, 386
Ipsilateral septal mucoperichondrial flap, 86
Irradiated homograft costal cartilage, 385
Irreversible obstruction, 374
Ischemic flaps, 79
Island flaps, 84
Isotretinoin, 155–157, 159, 287, 294–296, 339, 354, 358, 400
Isotretinol, 358

J

Jessner's formula, 289, 295
Jump puncture technique, 316
Juri flap, 346, 348

K

Karapandzic flap, 92
Kasabach-Merritt syndrome, 235, 240
Kasposiform hemangioendothelioma (KHE), 237

Keratinocytes, 285
Keystone area, rhinoplasty, 371
Klippel-Trenaunay syndrome, 240
Kojic acid, 293
Kybella®, 367

L
Labial arteries, 282
Labial incompetence, 322
Labiomental fold, 327
Labrale inferius, 395
Labrale superius, 395
Langerhans cells, 285
Langer lines, 152
Laser hair removal, 357
Lasers, aesthetic surgery
  Alexandrite, 354
  in clinical setting, 354
  complications, 355
  epidermal cooling, 353
  intense pulsed light, 354
  laser hair removal, 357
  Nd:YAG, 354
  non-laser technologies, 357
  PDL, 354
  preoperative evaluation, 354, 355
  scar treatment, 356
  science, 351, 352
  skin resurfacing, 356
  tattoo removal, 356, 357
  tissue interaction, 352
  treatment preparation, 355
  variables, 352, 353
  vascular malformations/lesions, 356
Lateral brow hooding, 396
Lateral cephalogram, 329, 332
Lateral crura, 372
Lateral crura convexity control sutures, 377
Lateral crural overlay, 377
Lateral crural steal technique (LCS), 377, 387
Lateral crural strut grafts, 377
Legal issues
  affirmative duty, 64
  doctor–patient relationship termination, 65
  financial obligations, 64
  informed consent, 63–64
  insurance, 64
  liability, 64
  medical negligence/malpractice, 64
  PHI, 64
  record keeping, 64
  standard of care, 63
  treating unhappy patient, 64
Legan's angle, 324
Lentigo maligna melanoma (LMM), 123
Levator anguli oris, 314
Lidocaine toxicity, 365, 366
Limberg flap, 81
Lines of maximum extensibility (LMEs), 152
Lip, 316, 327
  borders of, 276
  layers of, 276
Lip augmentation, 278, 281–283
  complications of, 281
  V-Y plasty for, 282
Lip elevators, 317
Liposhaver, 367, 368
Liposuction, 369
  adipose tissue, physiology of, 362
  approaches, techniques, 364
  autologous fat transfer, 366
  blood supply and, 361
  closed approaches, 364
  complications, 365, 366
  equipment, 364
  incisions, 364
  lidocaine toxicity, 366
  liposhaver, 367
  multidirectional crisscross approach, 365
  non-invasive treatment options, 367
  open approaches, 364
  patient counseling, 363, 364
  patient selection and analysis, 362, 363
  photo documentation, 363
  physical exam, 363
  procedure, 364
  suction, 366
  superficial tunnel, 365
  tumescent liposuction, 365
  ultrasound assisted liposuction, 366
Lip reconstruction, 144
  advancement flaps, 90
  A-T flaps, 90
  Bernard-Burrow, 92
  cross-lip flaps, 91–92
  full thickness defects, 90
  Gilles fan flap, 92
  goals, 89
  Karapandzic flap, 92
  mucosal advancement flaps, 89
  mucosal cross-lip, 90
  mucosal V-Y flap, 90
  rotational flaps, 90
  stair-step lower lip advancement flap, 91
  total lip reconstruction with free flap, 92
  transposition flaps, 90
  wedge resection, 91
Lip rejuvenation, 282
Lobule reconstruction, 95
Local flaps, 138
Lop ear, 218
Lower lateral cartilage, 371, 372, 388
Lower lip, 277
Low–high osteotomy, 381
Low-low osteotomy, 381
Ludwig classification, 340, 342
Lymphatic malformations (LMs), 238, 239

M
Mallampati classification, 5
Malposition, 331
Mandible fractures, 170
Mandible reconstruction, 145
Mandibular branches, 302
Mandibular fractures, 332
Marginal mandibular nerve, 399
Marionettes and jowl lines, 317
Maxillary branches, 302
Maxillary/Le Fort fractures, 169–170
McGregor's patch, 415
Mechanical creep, 318
Medial crura, rhinoplasty, 371
Medial-oblique/transverse osteotomy., 382
Medium-depth peel, 289, 290, 296
Medpor, 176, 385, 389
Melanocytes, 285
Melanoma
  classification scheme, 135
  TMN staging & 5-year survival, 135
Melasma, chemical peels, 292
Melolabial/nasolabial flaps, 84, 139
Mental nerve, 333
Mentalis, 322, 331
Mentocervical angle, 415
Mentolabial sulcus, 395
Menton, 396
Mentoplasty, 321
Mercedes reduction, 346
Merkel cell carcinoma (MCC), 133, 285
Merrifield Z-angle, 324
Methylmethacrylate, 77
Microcystic adnexal carcinoma (MAC), 133
Microfocused ultrasound (MFU), 357
Microgenia, 321, 374
Micrognathia, 321, 374
Microneedling, 357, 358
Midface
  lift, 399, 405
  rhytidectomy essentials, 397
  surgical approaches to, 406
Mid-facial implants
  ideal implant, 311
  indication, 310
  materials, 311
  preoperative analysis, 311, 312
  surgical technique, 312
Midline reduction, 346
Milia, 355
Millard rotation-advancement flap, 188
Mimetic muscles, 362
Miniaturization, 349
Minoxidil, 339, 342, 414
Mohs micrographic surgery (MMS), skin cancers
  adjacent tissue transfer, 128, 129
  basal cell carcinoma, 122, 125, 126
  complications, 129
  components, 121
  cure rates and outcomes, 127, 128
  deep and bone involvement, 127
  etiology, 121–122
  excisional, 129
  incidence, 121
  lab processing + interpretation, 129
  melanoma, 123–124
  melanoma in situ (MIS), 125
  nonsurgical treatment, 124
  normal skin preservation, 125
  pre-op assessment, 126
  preoperative evaluation, 129
  primary closure, 128
  procedure, 126
  processing/interpretation, 127
  reconstruction, 128
  risk factors, 122
  secondary intention healing, 128
  single section method, 127
  squamous cell carcinoma, 122–123, 125
  surgical excision of lesions, 124, 125
  tumor removal, 125
  wide excision, 127
  wound care, 128
Motor nerves, 361
Mucosal advancement flaps, 89
Muir-Torre syndrome, 135
Mylohyoid, 322, 333
Myofibroblasts, 26

N
Narcotics, 23
Nasal button, 383
Nasal crusting, 388
Nasal fillers, complications of, 309
Nasalis, 314
Nasal lining, 371
Nasal obstruction, 388
Nasal reconstruction, 84
  ala, 86
  columella, 86
  defect-based approaches, 85–86
  dorsum, 85
  nasal sidewall, 85
  principle, 84
  skin reconstruction, 84
  subunits, 84
  tip, 85, 86
Nasal tip bossae, 391
Nasal tip projection, 376
Nasal tip rotation, 376
Nasal valves, 372
Nasion, 395
Nasofrontal angle, 328, 373
Nasolabial angle, 373
Naso-orbito-ethmoid fractures, 166
Nd:YAG, 354

Neck
  genioplasty, 327, 328
  rhytidectomy, 397, 398
Neovascularization, 26
Neurotoxin, 368
Non-involuting (NICH), 236
Non-laser technologies, 357
Nonsurgical rhinoplasty, 309
Norwood classification of hair loss, 340, 348
Norwood classification of male-pattern alopecia, 341
Nose
  genioplasty, 327
  rhytidectomy essentials, 397

O
Occlusal classes, 323
Occlusion, genioplasty, 322, 323
OnabotulinumtoxinA, 307
Onlay tip grafts, 378
Open roof deformity, 387
Ophthalmic artery, 302
Ophthalmic branches, 302
Oral cavity squamous cell carcinoma (SCC)
  adjuvant therapy, 136
  anatomic subsites, 136
  radiotherapy, 136
  surgical management of N0 neck, 136
Orbicularis oris muscle, 275
Orbital fractures, 167–169
Oropharyngeal squamous cell carcinoma (SCC), 137
Osseocutaneous, 413
Osseous (sliding) genioplasty, 330, 331
Osteoconduction, 76
Osteocutaneous and osteomusculocutaneous flaps
  fibular free flap, 98, 99
  free flap warm ischemia, 99
  iliac crest free flap, 99
  jejunum free flap, 99
  postoperative flap anticoagulation, 100
  postoperative flap monitoring, 100–105
  scapular free flap, 99
Osteogenesis, 76
Osteoinduction, 76
Osteotomies, 381, 382
  rhinoplasty, 381, 382
Otoplasty
  auricular reconstruction
    auricular prostheses, 225
    external auditory canal atresia, 225
    microtia, 222–224
  cartilage-scoring techniques, 220, 221
  cartilage-splitting techniques
    antihelical fold creation, 220
    Davis technique, 220
  deformities, 218
  Furnas method, 219–221
  iatrogenic aesthetic deformities, 222
  iatrogenic deformities and complications, 222
  lop ear deformity correction, 221, 222
  mustarde method, 218, 219
  protruding earlobe correction, 222
  surgical principles, 218–222
Overaggressive fat liposuction, 368
Overresection of lateral crura, 386
Owsley's extended supra-SMAS rhytidectomy, 403

P
Paired nasal bones, rhinoplasty, 371
Papillary dermis, 151, 285
Paramedian forehead flap (PMFF), 83, 139
Paramedian reduction, 346
Parkes-Weber syndrome, 240
Parotid duct injury, 409, 410
Patient counseling, liposuction, 363, 364
Peck grafts, 378
Pediatric rhinoplasty, 384
Percutaneous osteotomies, 381
Perioral area, rhytidectomy, 398
Periorbital area, rhytidectomy, 396, 397
PHACES syndrome, 234
Pharyngeal flap, 195
Phenol cardiotoxicity, 296
Phenol face peeling, 292, 294
Phenol peel hypopigmentation, 295
Phenol red thread test, 250
Phenol, toxic effects of, 295
Philtrum, cheiloplasty, 276
Photoaging, 293, 304, 396, 412
Photo documentation, 363
Photodynamic therapy, 124
Photothermolysis, 352
PICO model, 41
Pierre Robin Sequence (PRS), 182
Pilosebaceous unit, 285
Pinch-and-roll technique, 365
Pincushioning, 82
Piperidinopyrimidine, 342
Pivot flap, 87, 138
Platsysma, 322
Platyrrhine nose, 390, 392
Platysmal bands, 363, 397
Platysmaplasty, 363, 369
Pogonion, 324, 395, 396
Pollybeak deformity, 386, 391
Polyacrylamide gel, 279
Polydioxanone (PDS), 386
Polydioxanone (PDS)-perforated plates, 77, 383
Poly-L-lactic acid (PLLA), 311, 316, 319
Polymers, 312
Polymethyl methacrylate (PMMA), 279, 283, 311
Popliteal pterygium syndrome, 183
Porcine collagen, 279
Porion, 395
Porous polyethylene (Medpore), 77, 389
Porus high-density polyethylene, 385
Posinflammatory hyperpigmentation, 412
Posterior compartment, 361
Posterior plagiocephaly, 210

Posterior scalp incision, 407
Posterior wall augmentation, 196
Post-facelift sialocele, 414
Post-inflammatory hyperpigmentation (PIH), 295, 355
Post-peel erythema, 296
Preauricular incision, 406
Premaxillary graft/alar base graft, 377, 390
Presurgical infant orthopedics (PSIO), 187
Pretracheal layer, 361
Prevertebral layer, 361–362
Prognathia, 322
Projection, 373
Prolonged post-peel erythema (PPPE), 292
Prominauris, 218
Prosthion, 396
Protected Health Information (PHI), 64, 65
Proteus syndrome, 240
Psychosocial aspects
  anxiety disorders in plastic surgery, 71
  body dysmorphic disorder, 70
  body image, 69–70
  craniofacial, reconstructive, and adolescent patient, 71–72
  patient selection and assessment tools, 71
  patients problem identification, 70–71
  plastic surgeon's role, 69
  psychosocial outcome, 71
PTFE (GoreTex) lip implants, 283

Q
Quality-switched (Q-switch) nanosecond lasers, 356

R
Radial fan-shaped tunnels, 365
Radial forearm fasciocutaneous free flap, 87
Radiesse, 316
Radiofrequency (RF) devices, 357
Radix graft, 381, 395
Ramus fractures, 172
Random pattern flaps, 78
Rapidly involuting (RICH), 236
Reassurance, 316
Recessive chin, 332
Reconstructive ladder, 137
  composite graft, 138
  dermal, dermal/fat grafts, 138
  full-thickness skin graft, 138
  graft survival, 138
  primary closure, 137
  secondary intention, 137
  split-thickness skin graft, 137, 138
Reconstructive rhinoplasty, 382
Reduction cheiloplasty, 281
  bikini lip reduction, 281
  complications, lip procedures, 281
  vermillion, 281
Rejuvenation of aging neck, 409
Relaxed skin tension lines (RSTLs), 151
Resurfacing, 278, 354
Reticular dermis, 151, 285
Retracted columella, 387
Retroauricular incision, 407
Retrognathia, 321, 374
Reversible nasal obstruction, 374
Rhinion, 395
Rhinoplasty
  advantages, 375
  analysis, 372, 373
  anatomy, 371, 372
  approaches, 375
  base, 376, 377
  base view, 374
  bony vault, 381
  caudal septal deflection, 383
  complications, revision rhinoplasty and treatment, 386, 387
  crooked nose, 384
  disadvantage, 375
  dorsal augmentation, 380, 381
  dorsal reduction, 379, 380
  endonasal rhinoplasty advantages, 375
  ethnic variations, 374
  evaluation, 390
  frontal view, 373
  graft materials
    alloplast, 385, 386
    autogenous, 384, 385
    homologous, 385
  incisions, 375
  lateral view, 373, 374
  lengthening short nose, 384
  major tip support, 372
  middle third, 379
  minor tip support, 372
  nasal valves, 372
  osteotomies, 381, 382
  pediatric, 384
  physical exam, 374, 375
  septal perforation, 383, 384
  septum, 382, 383
  tension nose, 384
  tip refinement, 376–379
  tripod concept, 375
Rhombic flaps, 81, 82, 139
Rhytidectomy, 317, 369
  absolute contraindications for, 400
  aging face
    alteration of aesthetics with, 396
    brow/forehead, 396
    chin, 398
    earlobes, 398
    jowls, 397
    microscopic skin changes, 396
    midface, 397
    neck, 397, 398
    perioral area, 398
    periorbital area, 396, 397
  anatomy, 395

Rhytidectomy (*cont.*)
- Baker's Strip (lateral) SMASectomy, 402
- complications
  - auricular nerve and jugular vein injury, 410
  - contour deformity, 412
  - depression, 412
  - facial nerve branch injury, 410
  - hair loss, 411, 412
  - hematoma, 410, 411
  - infection, 411
  - parotid duct injury, 409, 410
  - pigmentary changes, 412
  - scarring, 411
  - seroma formation, 411
  - skin flap necrosis, 411
  - spinal accessory nerve injury, 410
  - systemic, 412
- extended rhytidectomy procedures, 402
- facial surgery perioperative care and anesthesia, 401
- favorable and unfavorable, 400
- Hamra's composite rhytidectomy, 403
- Hamra's deep plane rhytidectomy, 402, 403
- incision, 405
  - earlobe, 406, 407
  - posterior scalp, 407
  - preauricular, 406
  - retroauricular, 407
  - temporal, 405
- limited facelift procedures, 401
- male
  - advantages, 408
  - incisions, 407, 408
- midfacelift, 405
- nerve injured during, 414
- nose, 397
- Owsley's extended supra-SMAS rhytidectomy, 403
- preoperative assessment
  - facelift standard photographic views, 401
  - patient selection, 400
  - photo documentation, 400, 401
  - physical exam, 400
- procedures and treatment
  - aging neck, 408
  - fat transfer, 409
- skeletal cephalometric reference points, 395
- SMAS techniques, 402
- soft tissue, 395
- subcutaneous rhytidectomy, 402
- subperiosteal, 403–405
- superficial muscular aponeurotic system and retaining ligaments of face, 398, 399
- surgeon, facial nerve anatomy for, 399, 400
- threadlift or suture lift, 405
- threadlift/suture lift, 405

Ricketts, 324
RimabotulinumtoxinB, 307
Rocker deformity, 387, 389, 391
Rotational flaps, 80, 81

S

Saddle-nose deformity, 386
Salicylic acid, superficial chemical peel, 289
Salicylism, 289
Satyr's ear/pixie ear deformity, 406, 411
Scalp flaps, 346, 347
Scalp layers, hair restoration, 337
Scalp reconstruction
- allografts, 94
- blood supply, 93
- free flaps, 94
- galeal relaxing incisions, 93
- orticochea flaps, 94
- rotational flap, 93
- small defects, 93
- temporoparietal fascia flap, 94
- tissue expansion, 94

Scalp reductions, hair restoration, 345, 346
Scaphocephaly, 208
Scar revision and dermabrasion
- abrasion, 156
- collagen organization, 155
- complications
  - bacterial and fungal infection, 156
  - delayed healing, 156
  - depressed scars, 157
  - erythema, 157
  - hyperpigmentation, 156
  - hypopigmentation, 156
  - Milia, 156
- contraindications, 155
- cosmetics, 157
- dermis, 151
- epidermis, 151
- fusiform excision, 153
- geometric broken lines, 154, 155
- hypertrophic scars and keloids, 153
- indications, 155
- keloids and hypertrophic scars management, 153–157
- laser resurfacing, 156
- mechanical forces, 157
- post-procedure, 156
- precautions, 156
- pre-procedure evaluation, 156
- scar irregularization, 154
- scar repositioning, 154
- serial excision, 153
- shave excision, 154
- silicone sheets and gels, 157
- skin lines, 151, 152
- steroids, 157
- tissue expansion, 153
- wire fraises, 156
- wound healing, 152
- W-plasty, 154, 155
- Z plasty, 154, 155

Scarring alopecias, 339
Scarring, rhytidectomy, 411
Scar treatment, 356
Schirmer's test, 250

Sebaceous gland carcinoma, 133
Secondary tissue movement, 84
Sedation-analgesia
  airway difficulty, 22, 23
  airway management, 5
  alpha adrenergic blocker, 21
  antagonist medications, 22
  ASA classification chart, 4, 5
  availability of personnel, 7
  barbiturates, 15, 16
  basics, 13
  benzocaine, 21
  benzodiazepines, 14
  in cardiac or respiratory depression, 3
  cardiovascular complications, 22
  cardiovascular support, 22
  clonidine, 16
  cocaine, 11–12, 21, 23
  deep sedation, 4
  depth of sedation, 3
  dexmedetomidine, 16, 23
  emergency equipment, 7
  fentanyl/versed conscious sedation, 19, 20, 22
  general anesthesia, 4
  hemodynamics, 6, 7
  hepatic degradation, 14, 15
  intravenous access, 8
  ketamine-induced laryngospasm, 23
  level of responsiveness, 3, 6, 22
  lidocaine, 20, 21
  local anesthesia, 9–11, 21
  malignant hyperthermia, 17, 18, 21, 22
  mentoplasty, 20
  minimal sedation, 3
  moderate (conscious) sedation, 4
  monitoring, 6
  narcotics, 23
  nitroglycerin, 21
  operating room fires, 18
  opioids/narcotics, 13, 14, 23
  oral evaluation, 5
  oxygenation, 6
  patient evaluation, 5
  patients with advanced age and hepatic dysfunction, 22
  in pediatric patients, 16, 17
  phase II and phase III signs, 21
  pre-procedure checklist, 6
  pre-procedure preparation, 6
  propofol, 15
  pulmonary ventilation, 6
  recovery/discharge, 8
  reflexive withdrawal, 22
  regional anesthesia, 12, 13
  regional nerve blocks, 20
  rescue medications, 22
  reversal agents, 8, 22
  STOPBANG scoring, 5
  supplemental O2, 7
  surgical fire, 23
  total intravenous anesthesia, 16
  toxicity and management, 9, 11
  training of personnel, 7
  uses, 3, 7, 8
  with local and regional anesthesia, 3
Sellion, 395
Semisolid and solid facial implants, 312
Senescence, 396
Septal cartilage, 384
Septal chondromucosal composite flap, 87
Septal perforation, 374, 383, 384
Septoplasty, 379
Septorhinoplasty, 191
Septum, rhinoplasty, 382, 383
Seroma formation, 411
Servelle-Martorell syndrome, 240
Sesamoid/accessory cartilages, 372
Shallow nasofrontal angle, 381
Sheen graft, 378
Shell ears, 218
Shield grafts, 378
Short flap rhytidectomy, 401
Short nose, lengthening, 384
Shprintzen syndrome, 183
Sideburn incision, 405
Silastic implant, 334
Silicone implants, 279, 280, 282, 333, 386
Simonart's band, 185
Skin flap necrosis, 413
Skin innervation, 302
Skin resurfacing, 356, 358
Skin/soft tissue envelope, 371
Sliding dorsal nasal flap (Rieger flap), 85
Slot deformity, 348, 349
Smooth osteotomy line, 390
Soft tissue envelope, 299
Soft-tissue expansion
  complications, 314
  explant surgery, 314
  implant surgery, 314
  indications, 312
  inflation, 314
  internal tissue expanders (Balloon), 313
  mechanism and tissue response, 313
Soft-tissue reconstruction
  anterolateral thigh, 142
  free flap failure, 142
  jejunal free flap, 142
  lateral arm, 140, 142
  lateral thigh, 142
  radial forearm, 140
  rectus, 142
  regional flaps, 140, 141
Soft tissue rhytidectomy, 395
Solar elastosis, 304
Sphincter pharyngoplasty, 195
Spinal accessory nerve injury, 410
Spironolactone, 343
Split-thickness skin graft (STSG), 75, 76
Spreader grafts, 380

Stable base, 376
Stahl (satyr/vulcan) ear, 218
Steiner, 324
Stickler syndrome, 182
Stomion, 395
Stratum basale, 151
Stratum corneum, 151
Stratum granulosum, 151
Stratum lucidum, 151
Stratum spinosum, 151
Sturge-Weber syndrome, 240
Subcutaneous rhytidectomy, 402
Subcutaneous ultrasonic energy, 367
Submandibular gland ptosis, 398
Submental surgery, 329
Subnasal lip lift, 278
Subnasale, 395
Sub-orbicularis oculi fat pad (SOOF) lift, 112
Subperiosteal rhytidectomy, 403–405
Subspinale, 396
Suction-assisted lipectomy, 366, 368
Superficial cervical fascia, 361
Superficial chemical peel, 288
  alpha-hydroxy acid, 289
  Jessner's solution, 289
  salicylic acid, 289
  TCA, 288
Superficial muscular aponeurotic system (SMAS), 279, 282, 304, 398, 399, 402, 414
Superficial surface, 361
Superficial temporal artery, 299
Superficial tunnel, 365
Superior/inferior labial artery, 275
Supramentale, 396
Supra-SMAS approach, 414
Supra-SMAS technique, 403
Supratarsal crease, 374
Supratip, 395
Swinging door technique, 383
Symphyseal fractures, 172
Synthetic injectable fillers, 311

T
Target chromophores, 353
Tarsorrhaphy, 112
Tattoo removal, 356, 357
Tear trough deformity, 397
Telangiectasias, 356, 412
Telogen, 338, 414
Temporal incision, rhytidectomy, 405
Temporalis fascia, 279, 385
Tendon, cheiloplast, 279
Tennison-Randall Repair, 189
Tension-free closure, 384
Tension nose, 384
Tenzel semicircular flap, 89
Thermal relaxation time (TRT), 353
Threadlift/suture lift, 405
Tip definition, 373
Tip, rhinoplasty, 376–379
Tissue expanders, 138, 345
  complications of, 315
Tissue extension, 345
Tissue interaction, 352
Tissue response, 318
Tongue-in-groove technique, 376
Topical ketoconazole, 343
Total nasal reconstruction, 87
Traction alopecia, 339
Transdomal sutures, 377
Transposition flaps, 81
Trapdoor deformity, 140
Trapezius flap, 96
Tretinoin, chemical peels, 293, 296
Triamcinolone injection, 368
Triangular alopecia, 339
Trichion, 395
Trichloroacetic acid, 295
Trichotillomania, 339
Trigonocephaly, 208
Tripod concept, rhinoplasty, 375
Tufted angioma (TA), 236–237
Tumescent liposuction, 365
Turkey gobbler neck, 397
Turn-in flaps, 380
Tyndall effect, 315

U
Ultrasound assisted liposuction, 366
Upper lateral cartilage, 380
  rhinoplasty, 371
  spanning suture, 380
Upper lip, 276
Upper lip elongation, 398

V
Van der Woude syndrome (VWS), 182
Vascular anomalies, see Vascular tumors; Vascular malformations
Vascular lasers, 356
Vascular malformations, 356
  arteriovenous malformations, 239
  Bannayan-Riley-Ruvalcaba syndrome, 240
  Beckwith-Wiedemann syndrome, 240
  Blue rubber bleb, 240
  capillary malformation, 237, 238
  capillary malformation-arteriovenous malformation syndrome, 240
  histopathology, 237
  imaging recommendations, 239, 240
  Kasabach-Merritt syndrome, 240
  Klippel-Trenaunay syndrome, 240
  lymphatic malformations, 238, 239
  Maffucci syndrome, 240
  Parkes-Weber syndrome, 240
  PHACES syndrome, 240
  phakomatosis pigmentovascularis, 241

Proteus syndrome, 240
Servelle-Martorell syndrome, 240
Sturge-Weber syndrome, 240
venous malformation, 238
Vascular plexus, 318
Vascular supply, hair restoration, 337
Vascular tumors, 231
congenital hemangioma
NICH, 236
RICH, 236
infantile hemangioma
characteristics, 233
complications, 233, 234
disseminated lesions, 233
focal tumors, 233
medical therapy, 234–236
observation, 234
overview, 231
pathology, 233
segmental hemangiomas, 233
treatment, 234
kasposiform hemangioendothelioma, 237
pathologic cellular hyperplasia, 231
tufted angioma, 236
Vasodilation, 25
Velocardiofacial syndrome, 183
Venous outflow
hair restoration, 337
rhinoplasty, 371
Vermillion, 276, 281
Vertical analysis, methods of, 327
Vertical dome division, 377
Vertical thirds, 327
Vestibular stenosis, 387
Visual criteria for aging neck, 414
Visual impairment, 233
V-Y advancement, cheiloplasty, 280
V-Y columellar advancement, 391
V-Y plasty for lip augmentation, 282

W
Webster modification, 82
Weir excisions, 379
White roll/line, 276
Wide subperiosteal exposure, 334
Witch's chin aging deformity, 322, 331, 413
Wound healing, 286, 295
aesthetic appearance of asian eyelid, 39
ant/post ethmoid artery, 39
bony orbit, 39, 40
classification for dog bite, 39, 40
clinical practice, 26
diabetes, 39
edema, 39
facial nerve, main trunk position, 39
Fitzpatrick scale, 30
growth factors, 27
histology, 29–30
hyperbaric oxygen, 27
inferior oblique muscle, 39, 40
inflammatory phase, 25
keloid formation, 39
local, 26
marionette lines, 39, 40
medial canthal tendon, 39, 40
medical comorbidities, 26
medications, 27
modiolus, 39, 40
nasal tip innervation, 39
neutrophils, 39, 40
nicotine affect, 39
phases and characteristics, 25–26
PMFF, 39
pretarsal orbicularis, 39, 40
proliferative phase, 25, 26
remodeling/maturation, 26
skin layers, 39, 40
soft tissue layers, facial nerve, 39
subzygomatic triangle, 39
surgical anatomy, 31, 33
buccal, 32
cutaneous innervation, 31, 32
embryology, 30
facial proportions, 30, 31
facial skeleton, 30
marginal mandibular, 32
motor nerves, 31, 32
scalp, 33
SMAS, 32
superficial landmarks, 30, 31
superficial layer of deep temporal fascia, 33–38
zygomatic, 31
surgical techniques, 28–29
absorbable suture types, 28
non-absorbable suture types, 28
technical, 27
tensile strength of scar, 39
traumatic wounds, 27–40
vacumm-assisted closure, 27
vertical buttresses of face, 39, 40
vitamins, 39
Whitnalls tubercle, 39
W-plasty, 280

Z
Zitelli's modification, 82, 83
Z-plasty, 139
Zygomaticomaxillary complex (ZMC) fractures, 165–166
Zygomaticus minor, 315