Facial Plastic and Reconstructive Surgery

A Comprehensive Study Guide

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

Second Edition

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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
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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.

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Part I

Basic Principles, Perioperative Management, and Miscellaneous Topics

Analgesia and Conscious Sedation

1

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.

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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

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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

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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 P*C*N 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*

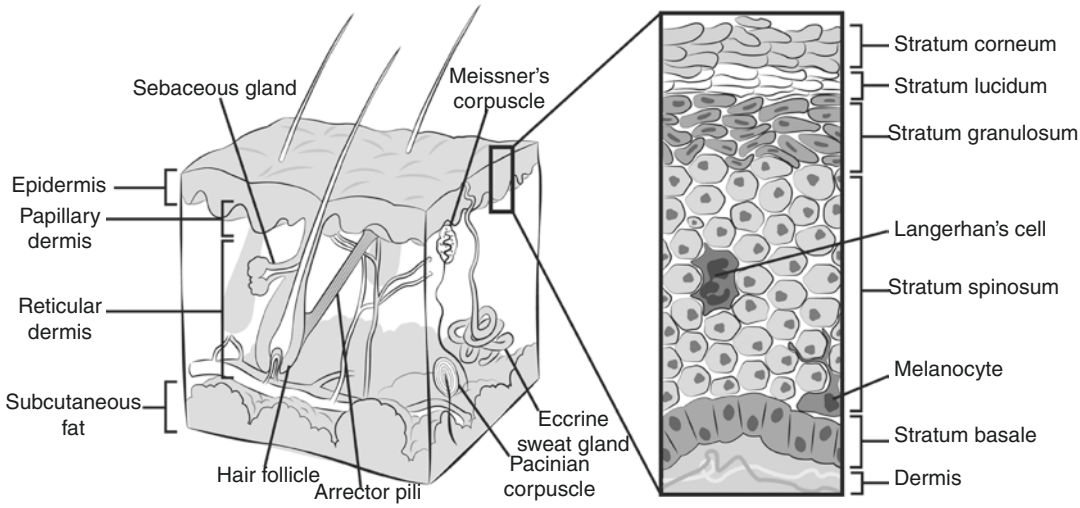

Fig. 2.1 Cross-section of skin (*left*) with cellular component of epidermis (*right*)

  - 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

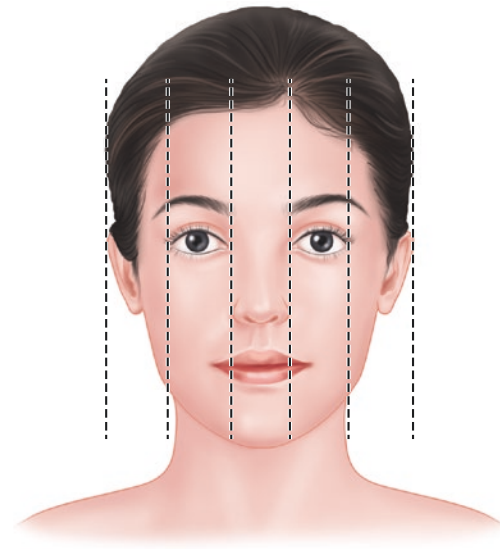

Fig. 2.2 Facial proportions- Vertical

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)

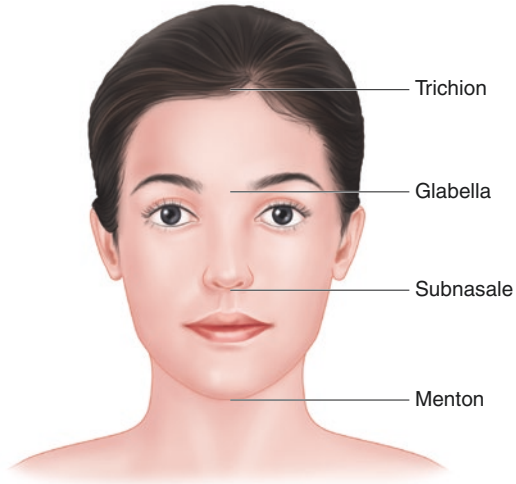

Fig. 2.3 Facial proportions- Horizontal

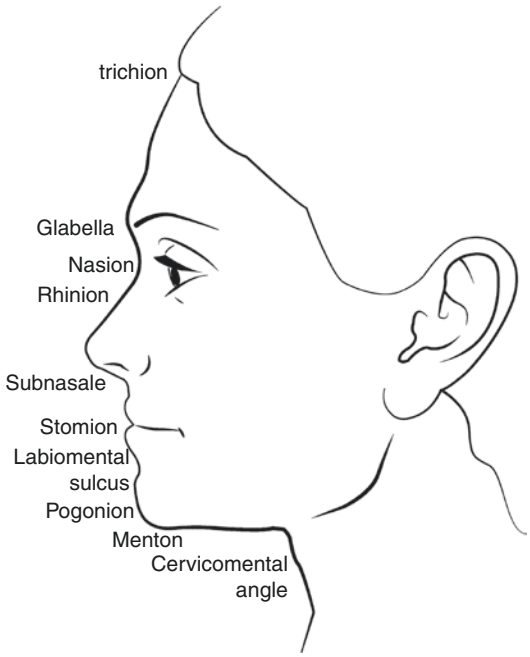

Fig. 2.4 Superficial landmarks (profile view)

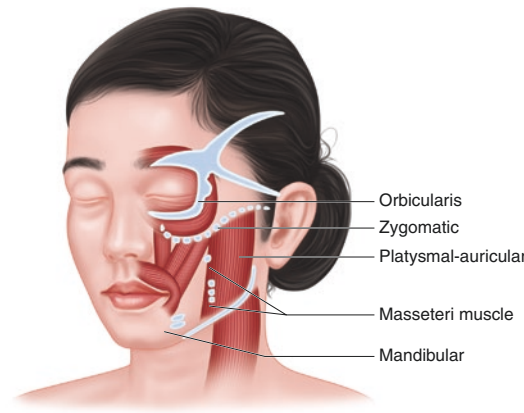

Fig. 2.5 Skin-retaining ligaments of the face

Frontal bar
Infraorbital/Zygomatic process
Maxillary alveolus
Mandible

– Buttresses (4)
Nasomaxillary
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:

    Pretarsal, preseptal } 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

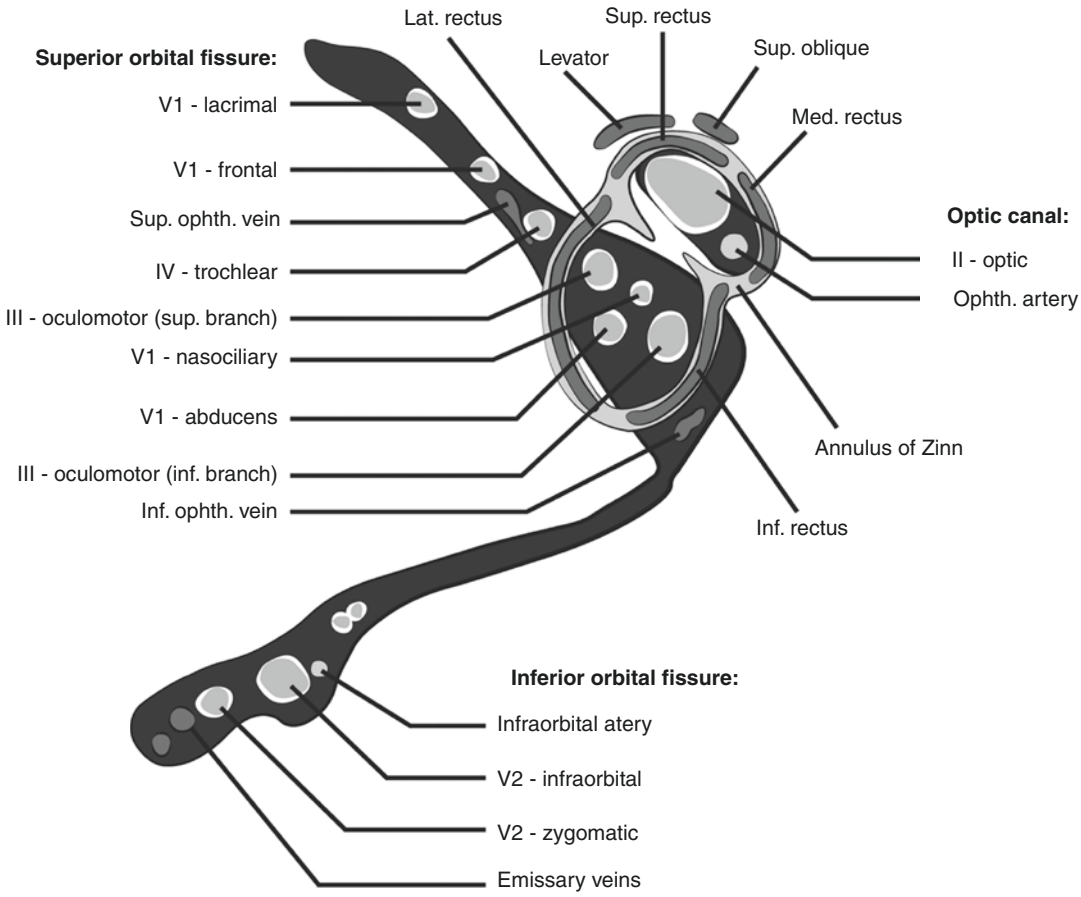

Fig. 2.6 Right orbital fissure contents

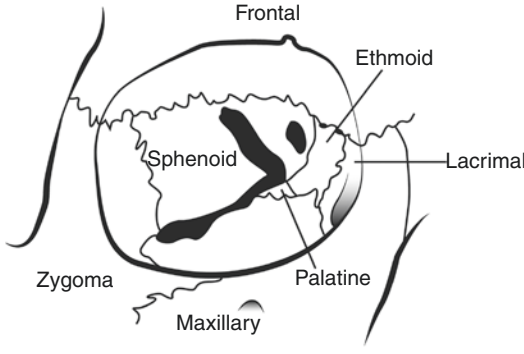

Fig. 2.7 Bone composition of the orbit (right side)

- 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)

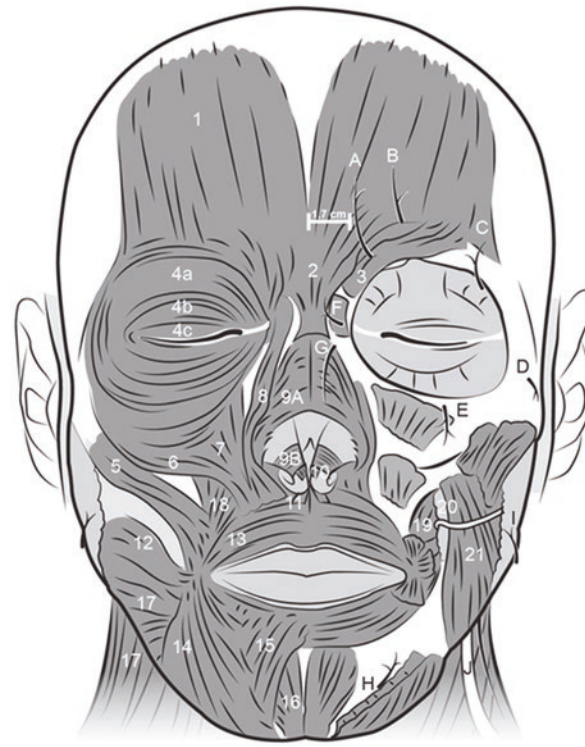

Fig. 2.8 Muscles of facial expression, facial innervation

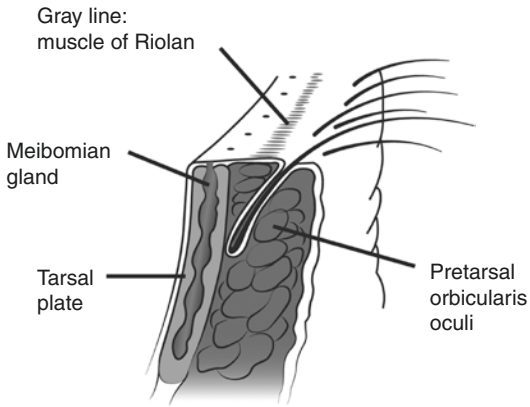

Fig. 2.9 Cross-section of lower eyelid

- 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)

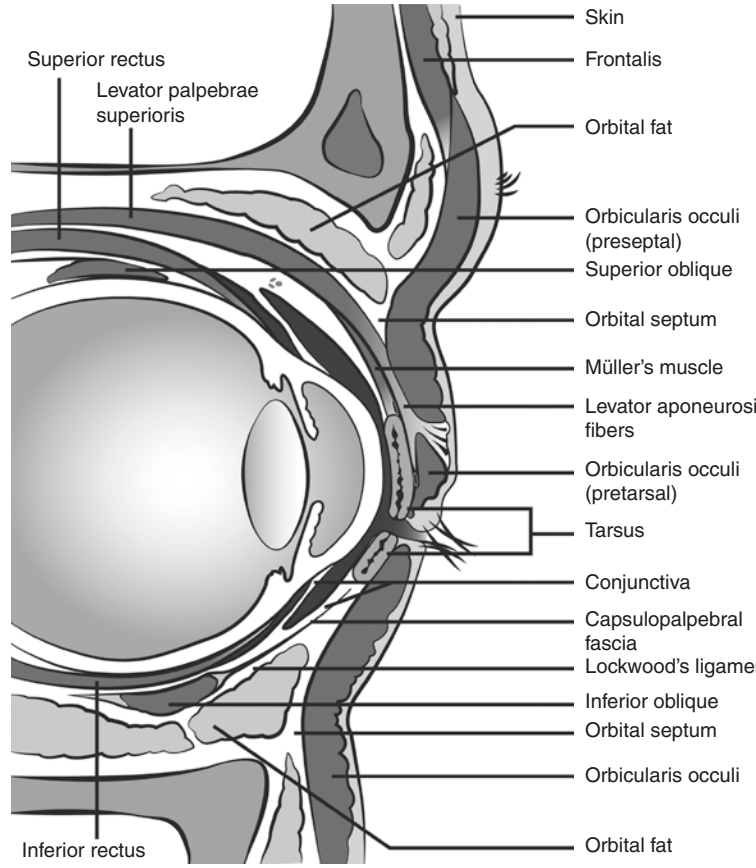

Fig. 2.10 Cross-sectional view of orbital anatomy

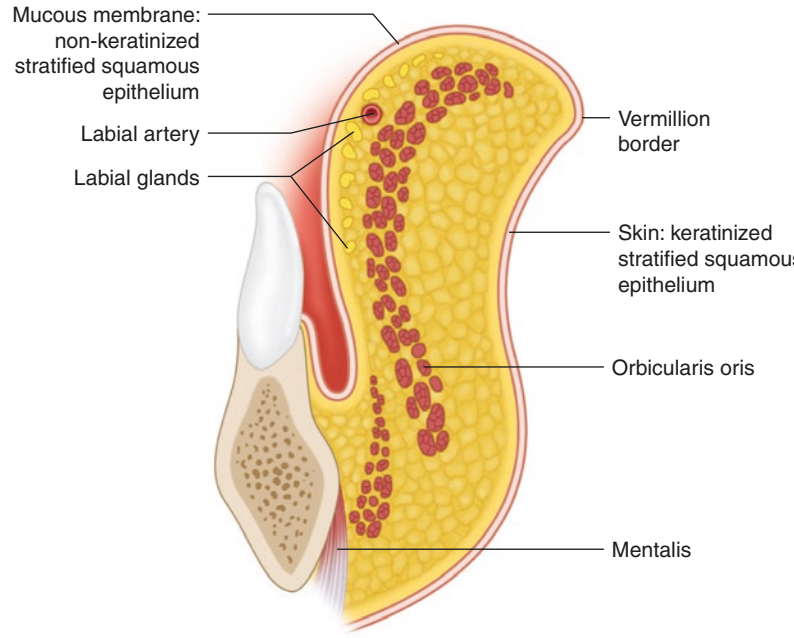

Fig. 2.11 Cross-section of lower lip

- 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*

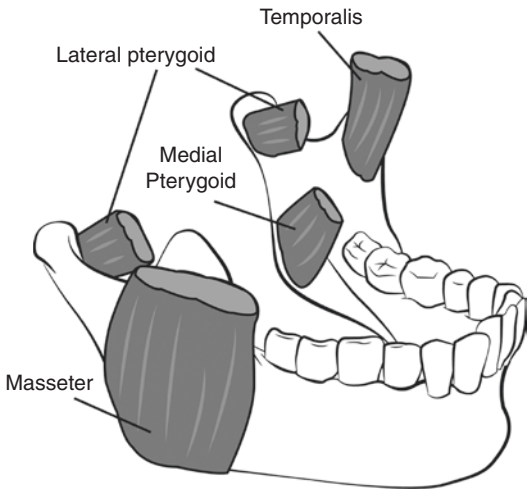

Fig. 2.12 Muscles influencing jaw opening and closing

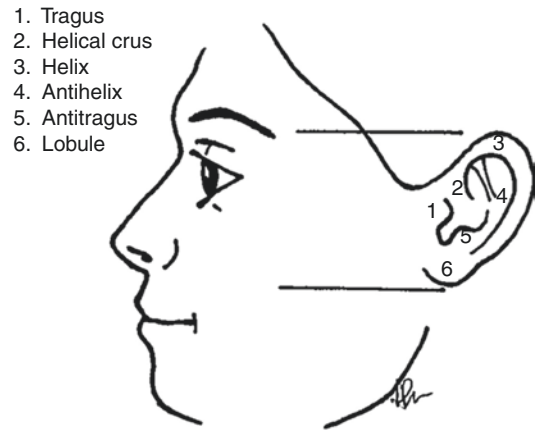

Fig. 2.13 Derivatives of Hillocks of His

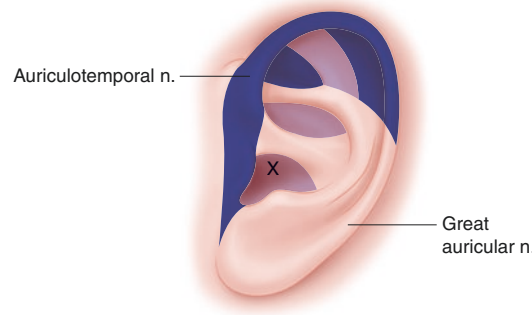

Fig. 2.14 Cutaneous innervation of the ear

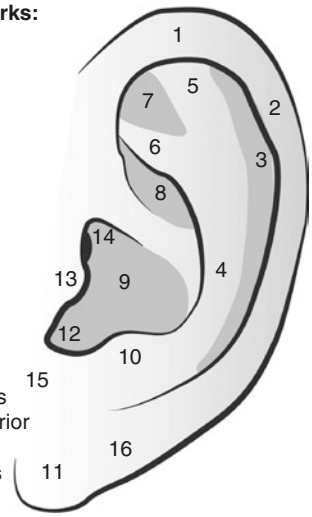

Fig. 2.15 External landmarks of the ear

- *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

*D*eep auricular
*A*nterior tympanic
*M*iddle meningeal
*A*ccessory meningeal
*I*nferior alveolar
*M*ylohoid
*D*eep temporal (post and ant)
*P*terygoid
*M*asseteric
*B*uccinator
*P*osterior superior alveolar
*I*nfraorbital
*D*escending palatine
*S*phenopalatine
*P*haryngeal

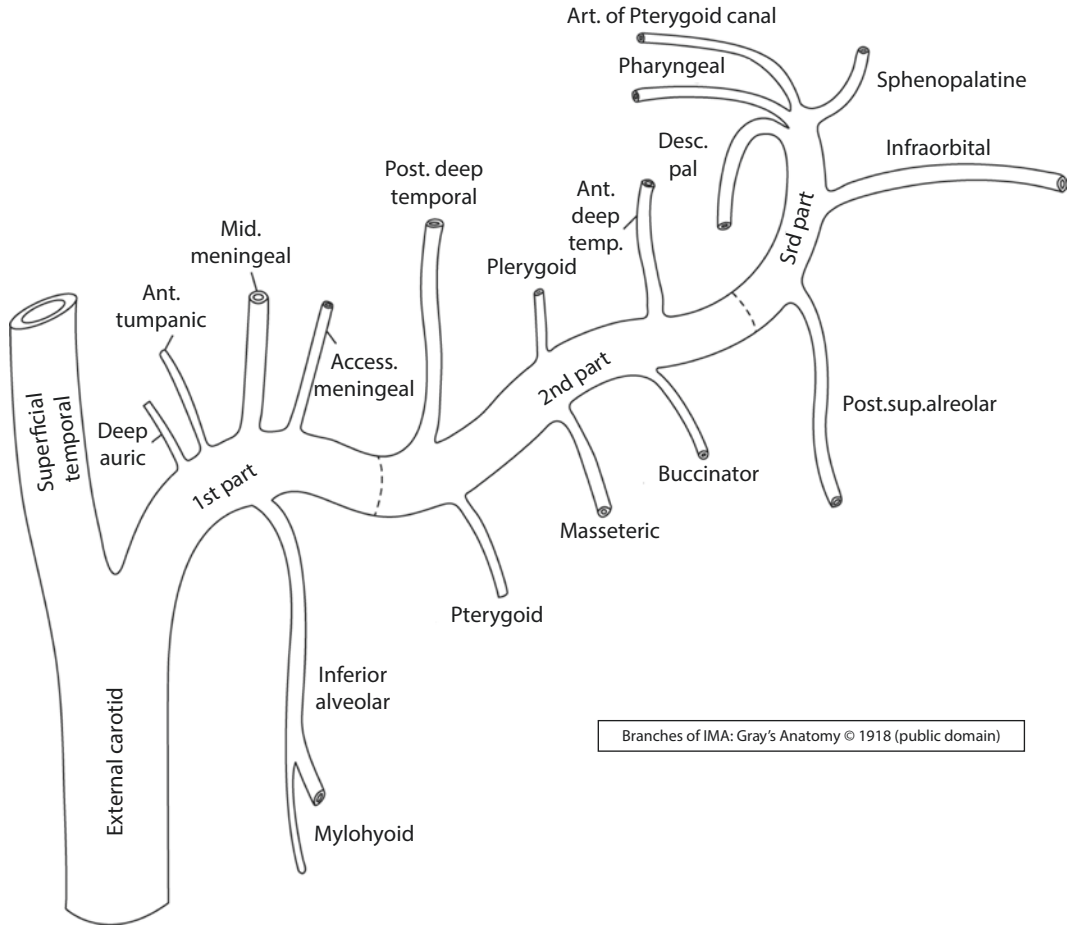

Fig. 2.16 Branches of the internal maxillary artery

- 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