Oral Board Review for Oral and Maxillofacial Surgery

Robert Reti
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To my wife Allison for her patience, understanding, and making my dreams come true. My son Ezra for giving me an understanding of what truly matters in this world. My grandmother Lili Friedman, a Holocaust survivor and toughest human I have ever met. My mother Marianne for giving up so much so I may achieve my dreams. My sister Susanne for always being a better person than I could hope to ever be. Drs. Richard Sorbera and William Gilmore for your patience, guidance, and instilling humility. To all my colleagues, dental students, residents, and fellows that I have been privileged to work with and learn from, I will forever be indebted to you.

Robert Reti, DDS, FACS

To the MOST HIGH: Thank you for giving me the opportunity to be a part of this most noble profession and surrounding me with multiple counselors that have been instrumental in my career progression.

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Damian Findlay, DMD, MD, FACS

Preface

Congratulations on completing the grueling gauntlet of Oral and Maxillofacial Surgery residency. Now it is time to take the exam to become a diplomate. Preparation for the ABOMS oral certifying exam can be a daunting task. You will not be the first surgeon to ask, where do I start? Why am I subjecting myself to this?

First, you need to understand that all training programs are unique. There are strengths and weaknesses of every program. Everyone has a gripe about deficiencies in their program, but there is no bad training program. The board is not meant to compare your training versus another person's training. The exam is standardized to ensure a fair exam. The purpose of the exam is not to humiliate you. It is an exam, which looks to examine your competency as a surgeon. It tests your knowledge of basic principles and assesses if you practice safe and evidence based oral and maxillofacial surgery. Board certification is a message to the community that a group of your colleagues have tested your knowledge and they are in agreement that you have achieved mastery of the basic knowledge as it relates to practicing oral and maxillofacial surgery.

The book is structured to provide a comprehensive review of various topics and to take you through a series of cases similar to those that you may see on the board exam. This book is not meant to replace other textbooks or board review courses. The purpose of this book is to both streamline your preparation with the salient didactic information and to be used as an adjunct to other resources including board review courses. There is so much information out there and you have a limited amount of time. The key to studying passed on from surgeon to surgeon is first; learn what you need to know (the basics), then what is nice to know, and, if time permits, what is nuts (impractical) to know.

We are not board examiners and this book is only to be offered for professional development and as an adjunct to studying. To our knowledge, no course or textbook is endorsed or sponsored by the American Association of Oral and Maxillofacial Surgeons or the American Board of Oral and Maxillofacial Surgery. However, we are young surgeons that have recently gone through the process and successfully passed the exam. We feel that we have put together a text that will be a major part of the equation of your pass-

ing the exam. You are juggling work life, family life, and your well-deserved free time. Our hope is to ease the anxiety of the diplomatic candidate and be instrumental in your successfully passing this exam.

St. Louis, MO, USA — Robert Reti
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Part I

Dentoalveolar, TMJ, Maxillofacial Infections, and Implantology

Dentoalveolar

1

Michael Barbick, David Chang, Robert Reti, and Anthony Kramer

Dentoalveolar

Impacted tooth – a tooth that cannot or will not erupt into its normal functioning position.

Management of Impacted Third Molars

Background

- Frequency of impacted third molars ~25% in most studies when present [1].
- Third molars are the most common teeth to be missing, followed by second premolars and maxillary lateral incisors.
- Third molars are the most likely teeth to be impacted followed by maxillary canines, mandibular premolars, maxillary premolars, and second molars.
- Third molar agenesis is reported between 10% and 41% [2].
- African Americans develop third molars faster than Caucasians.

Development of Third Molars

- Age 6–9 – follicles become visible on radiography.
- Age 9 – molar germ visible.
- Age 11– cusp mineralization, located anterior border of ramus.
- Age 14 – crown formation is done.
- Age 15 – tooth uprighting as roots form.
- Age 16 – 50% of root formed. Anterior border of ramus resorbs as mandible lengthens.
- Age 18 – root completely formed with an open apex.
- Age 24 – 95% of molars in final tooth position.
- Age 25 – little change in tooth positioning, but minor changes may occur past this age.

Theories of Tooth Impaction

1. Differential growth rate of roots causes under- or overrotation leading to impaction.
2. Arch length: Impacted third molars are larger than erupted third molars.
3. Ectopic position: abnormal germ position puts teeth in contact with a denser external oblique ridge.

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4. Late mineralization: tooth growth lags behind maturation of jaws due to decreased influence of resorption of jaw.
5. Attrition: softer diet leads to less attrition retaining mesiodistal space.

Classification for Mandibular Third Molars

Pell and Gregory based on radiographic review (Fig. 1.1).

Classes A–C based on relation to second molar occlusal plane.

A: Third molar occlusal plane in line or nearly in line with adjacent second molar.

B: Third molar occlusal plane between occlusal plane and cervical junction of the second molar.

C: Third molar occlusal plane below cervical junction second molar.

Classes 1–3 based on relation to the anterior border of ascending ramus.

1: Mesiodistal diameter of crown anterior to ascending ramus.

2: Half of crown covered by ramus.

3: Tooth is completely located within ramus.

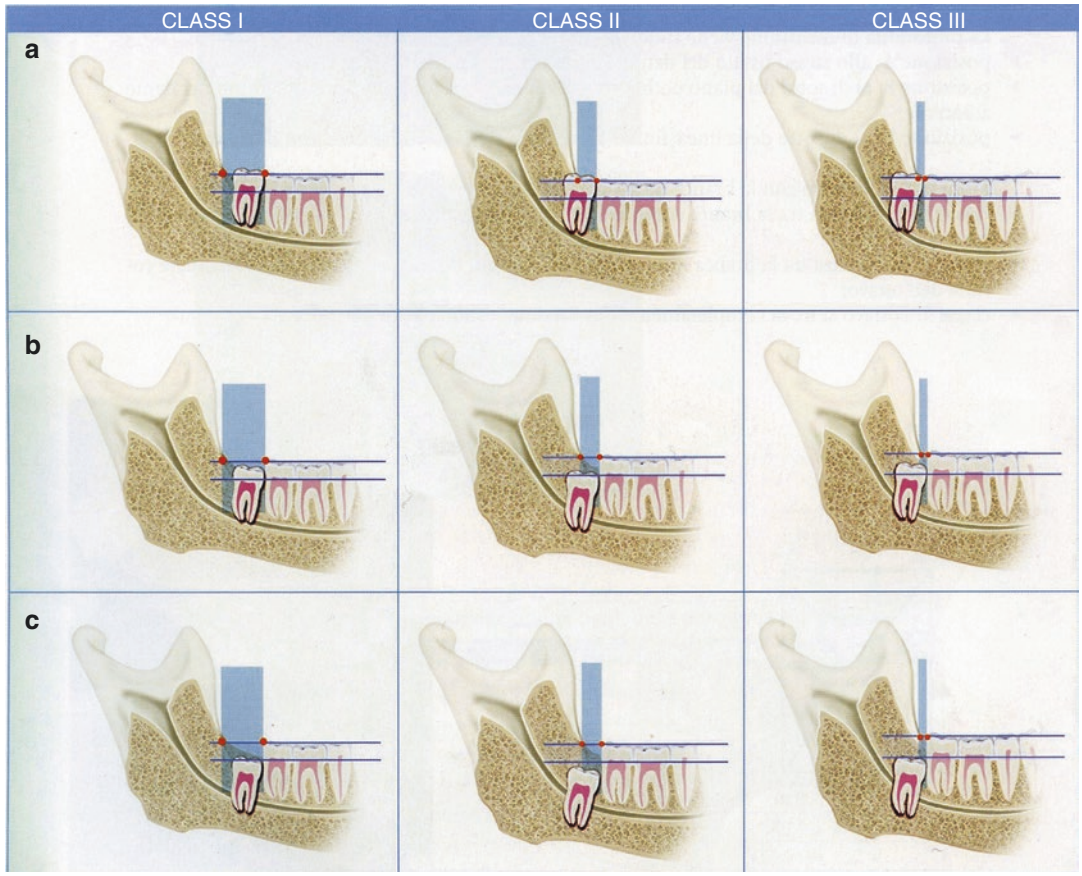

Fig. 1.1 Pell and Gregory classification. (Reprinted with Permission from Mantovani et al. [13])

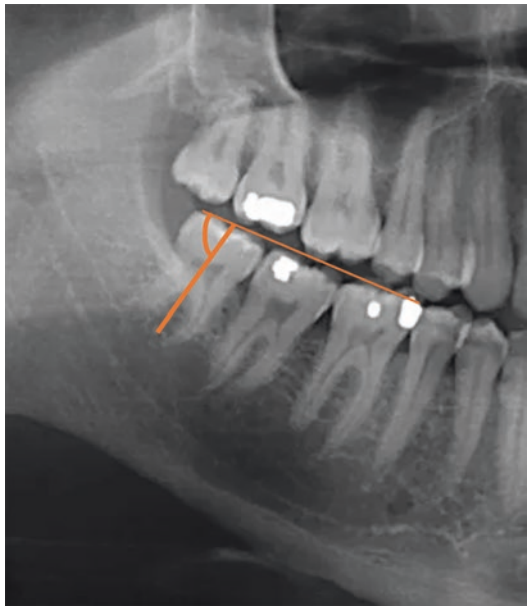

Fig. 1.2 Winter's classification is based on the angle between the occlusal plane and the longitudinal axis of the third molar

Winter's Classification [3] (Fig. 1.2):

- Most commonly used classification system.
- Angle between the occlusal plane and the longitudinal axis of the third molar.
- <0° = inverted, rare. As is buccoangular and linguoangular.
- 0° and 30° horizontal impactions, ~10% of impactions.
- 31° and 60° mesioangular impactions, ~45% of impactions.
- 61° and 90° vertical impactions, ~40% of impactions.
- >90° distoangular impactions, ~5%.

Indication for Third Molar Removal

Pericoronitis
- Most common reason over the age of 20.
- Associated with inflammation of the operculum (a dense fibrous flap of tissue).
- May be treated symptomatically with irrigation and antibiotics.
- Removal of third molars is the most predictable treatment.

Orthodontic Needs
- Dental crowding? No consensus if third molars associated with mandibular incisor crowding.
- Interference with orthodontic treatment may prevent distalization of other teeth.
- Orthognathic: surgeon may require third molars out 6–12 months prior to surgery to aid in bone fill. This allows an increase in bony area for fixation and may prevent bad mandibular splits.

Pericoronal Pathology
- Most common pericoronal pathology is a dentigerous cyst or an enlarged follicle. A pericoronal radiolucency >3 mm is suggestive of a dentigerous cyst.

Caries
- Inability to clean adequately, most commonly on cervical of second molar.
- Inability to isolate third molar for restorative measures.
- Access to distal caries on second molar.

Fracture
- Presence of third molars creates an area of lowered fracture resistance.
- Approximately 3× increase in angle fractures with third molar present due to disruption in cortical bone.
- Special consideration in patients in contact sports.
- Presence may complicate repair of angle fracture reduction.

Unexplained Pain
- 5% of third molars removed for this reason and often "cures" pain for reasons unclear.

Overlying Prosthesis
- Third molars are unpredictable in future position or eruption. There is evidence for pathology to develop if left in place under prosthesis.

- Up to clinician to remove or to watch but generally recommended to be removed unless 2 mm bone is surrounding the crown.

Periodontal Disease
- Consider removal if >5 mm pocketing with bleeding on probing or >1 mm attachment loss on adjacent second molar (may lead to future progression of periodontitis).
- Presence of third molars associated with elevated levels of periodontitis of adjacent teeth.

Pertinent Anatomy

Inferior Alveolar Nerve
- Provides sensation to mandibular lower teeth up to midline, chin, and lower lip.
- Injury incidence reported approximately 1% after third molar extraction.
- Generally the nerve is located buccal and apical to impacted third molars.

Lingual Nerve [4]
- Lies on average 2.8 mm below the crest and 2.5 mm medial to lingual cortex.
- 0.6–2.0% reported incidence of lingual nerve damage during third molar surgery.
- In 4.6–21.0%, the lingual nerve is at or above the crest of the bone.
- 22% reported at lingual plate of bone.
- Turns toward tongue at region of first and second molars.

Workup

CC/HPI: Reason for removal, is the patient experiencing pain, in progress or planning on orthodontic/orthognathic treatment? etc.

PMHx:

Age of the Patient
Advanced age:

- Increased healing time and increased risk of morbidity.
- Bone becomes more sclerotic and less elastic, which makes the removal complicated. This leads to the need for more bone removal.
- Patients who are 25 years and younger have decreased risk of complications and improved recovery after surgery.

Inadequate age:
- Unable to predict impaction when there is minimal tooth formation.
- Ideally remove tooth when there is ½–2/3 development of root formation.

Compromised Medical Status
- Benefits must outweigh the risks.
- A history of cardiopulmonary disease, immunocompromised states, or coagulopathies could prevent a safe extraction (may be best to watch until symptomatic).
- Consider treating patients in a more controlled setting should an extraction be indicated.

Anesthetic History
- Problems with induction or awakening.
- History of nausea or vomiting.

Temporomandibular Disorder (TMD)
- History of clicking, pain, or locking of joint.
- Difficulty of opening for long periods of time or limited opening.

Smoker
- Delayed healing.
- Higher risk of dry socket.
- Reactive airway for anesthesia.

Exam
General: Examine distress and anxiety (can help determine if sedative/anesthesia required for procedure). Also degree of difficulty will aid in decision.

Head and Neck Exam
- Customary head and neck exam is part of workup. Examine anesthesia considerations such as large neck, retrognathia, and limited range of motion of neck.

- TMJ documentation for clicks, cracks, or pops.
- Maximal incisal opening, ease of access for surgical procedure, and anesthesia considerations.
- Tongue size: large tongues can fall back and cause airway obstruction.
- General oral health. Could be higher risk for postoperative infection and delayed healing.
- Pathology or infection around third molar site? Pericoronitis.
- Are the third molars visible? Guide to difficulty of extraction.
- Mallampati score.
- Tonsil grade.
- Periodontal probing distal to second molars (>4 mm indication with bleeding on probing (BOP) could be sign of periodontal disease).
- Teeth are in good functional position and without disease. Direction of general dentist to have them removed based on an algorithmic approach to treatment is inappropriate. Surgeon should conduct a separate evaluation that deems this is a necessary procedure.

Radiographic Exam

Orthopantogram:

- For overall survey of third molars. Most studies on risk based on orthopantogram. Able to classify difficulty of extraction.
- Root morphology:
  - Ideally removal when two-thirds roots are developed.
  - Fused roots easier than flared roots.
  - Direction and severity of curved roots increase difficulty and can change the path of tooth extraction.
  - Periodontal ligament space, wider makes for easier extraction.
- Follicular sac – if present, the wider the sac, the less the bone required for removal and the easier to identify tooth crown.
- Density of bone:
  - Higher density leads to increased difficulty.
  - Patients >35 years of age regularly have increased density appreciated.
  - Higher likelihood of fracture due to lack of expansion and increased force required for removal.
- Radiographic signs on orthopantogram described by Rood and Shehab, aka Rood's Criteria, describing intimacy of Inferior Alveolar Nerve (IAN) with roots of mandibular third molar [5].
  1. Darkening of root
  2. Deflection of root
  3. Narrowing of root
  4. Bifid root apex
  5. Diversion of canal
  6. Narrowing of canal
  7. Interruption in white line of canal

CBCT

- Not routine, but can be selected if pathology is suspected or if there is intimacy with anatomical structures.
- Most software can convert imagery into a reformatted orthopantogram.

Treatment

* The approaches in this book are just a guide. On the board exam you are asked how *YOU* would perform the procedure. The key is to practice articulating what you do every day in a concise and efficient fashion. Provided is just a guide on how to articulate the procedure. Practice verbalizing your technique.

Surgical Approach to Extraction of Third Molars

- Local anesthetic with vasoconstrictor is administered (via, e.g., IAN, lingual and buccal nerve blocks, PSA, and greater palatine).
- A 15 blade is used to make a sharp incision to outline a full thickness mucoperiosteal flap. This flap is outlined from the mesiobuccal of the second molar and carried sulcular around the second molar with a distobuccal releasing incision.
- A periosteal elevator is used to carefully reflect a full thickness flap exposing the bony mandible/maxillae.
- Using a fissure bur under copious irrigation the bone surrounding the crown is carefully removed exposing the tooth and a buccal

trough is created. (Bone in the maxilla is thin and can usually be removed with a molt/periosteal elevator).
- Once the crown is uncovered, the tooth is luxated (if appropriate)/tooth is sectioned parallel to the mesiobuccal groove two-thirds through the width of the tooth to prevent cortical perforation.
- The tooth crown and fragments are carefully removed.
- The granulation tissue and follicle are curetted free of the socket.
- Bone rasp is used to smoothen the bony lip of the osteotomy.
- Saline irrigation is used to cleanse the socket. (Distilled water is hypotonic and leads to cell death).
- Visual inspection to ensure IAN/sinus not compromised.
- Reapproximate tissue with resorbable suture (e.g., 3-0 chromic gut).

Complications

- *Alveolar Osteitis* – Incidence reported between 1% and 30%, range due to subjective criteria [6]. Commonly seen at days 3–7 after extraction. Current theory is increased fibrinolytic activity leading to break down of clot. Symptoms include referred pain to ear, eye, and temple region, foul odor, extreme tenderness to palpation. Risk factors: tobacco smoke, increasing age, pericoronitis, birth control, female gender, inexperience of surgeon leading to traumatic extraction, inadequate irrigation, and increased medical comorbidities. Some evidence show that chlorhexidine can reduce incidence. Treatment is commonly with iodoform gauze or gel foam coated with eugenol (commercial pastes are also available) that acts by inhibiting neural transmission. Review panorex and/or CT scan prior to application as should not be used when IAN exposed due to neurotoxic effects of eugenol. If concerned for IAN exposure, consider the use of topical lidocaine in place of eugenol.
- *Root Fracture* – Can be left in place if (1) non-infected, (2) small, i.e., <2 mm, (3) and risk of surgery would outweigh benefits.
- *Bleeding* – Bleeding report as a result of third molar surgery ranges from 0.2% to 5.8% [7]. Must always rule out coagulopathies such as hemophilia or Von Willebrand disease. In general, postoperative bleeding from dental extractions reported to be at about 1% and about 7% when taking oral anticoagulation therapy such as a vitamin K antagonist. Patients on vitamin K antagonist need not stop it if INR <4. Hemostatic agents are listed in Table 1.1.

Table 1.1 Hemostatic agents

Surgical packing for tooth extraction sites	
Product	Properties
Absorbable gelatin sponge, e.g., Gelfoam®	Matrix for blood clot formation Gelatin made from purified porcine skin May cause excessive granuloma or fibrosis
Microfibrillar collagen, e.g., Avitene®	Mechanically broken down bovine collagen Aggregates platelets onto fibrils and acts as a matrix for blood clot formation
Chitosan dressing, e.g., HemCon® and ChitoFlex®	Polysaccharide from shellfish Positively charged to attract erythrocytes Acts as a scaffold for clotting New dental formulation dissolves in 48 hrs
Thrombin	Promotes clot formation through activated bovine prothrombin Activates factor IIA Acts as serine protease converting fibrinogen to fibrin
Oxidized regenerated methylcellulose, e.g., Surgicel®	Binds platelets Negative pH is bacteriostatic and precipitates fibrin More efficient at hemostasis than gelatin sponge Can be packed into socket to aid in pressure hemostasis Does cause some prolonged healing Be cautious when using in lower third molar sockets as Surgicel creates an acidic milieu which can be toxic to the inferior alveolar nerve
Cross-linked collagen, e.g., Collaplug® or Collatape	Promotes platelet aggregation

Table 1.1 (continued)

Surgical packing for tooth extraction sites	
Product	Properties
Tanin, e.g., found in teabag	Serves as a vasoconstrictor
Aminocaproic acid mouth rinse	Stabilizes clot by inhibiting plasmin
Tranexamic acid 5% mouth rinse	Antifibrinolytic, inhibits conversion of plasminogen into plasmin

- *Displacement of Root into Sinus* – Most commonly the palatal root of maxillary first molar; take PA to verify position. Several local measures should be made: (1) suctioning into sinus, (2) pack sinus with xeroform gauze and pull in one stroke (often root will attach to gauze), (3) perform antral lavage, (4) have patient block opposite nostril and blow nose to force into socket, (5) enlarge opening and explore. If attempts fail, fragments 3 mm or less that are non-infected may be left in place and patient be informed. Roots >3mm or those that presented with an infection/peri-apical pathology should be removed via a Caldwell-Luc approach is indicated.
- *Oral Antral Communications (OAC)* – Most small OACs will heal by themselves. Openings of 3–6 mm can be managed by placing gel foam and closing with a figure-of-eight suture technique. OAC >6 mm may require tension-free primary closure, excision of the fistulous tract, and inversion into the sinus. Consider treating larger OACs with a buccal fat pad closure, buccal finger flap, or tongue flap. Sinus precautions for 2 weeks (decongestants, antibiotics that cover sinus flora, no heavy nose blowing, saline nasal spray).
- *Displacement of Root or Tooth into Submandibular/Sublingual Space* – Lingual cortex thins out in the more posterior region. Displacement is often inferior to mylohyoid muscle. First attempts should be to "milk" root back through cortical hole via manipulation. An attempt at a lingual flap extended anteriorly to premolar with an incision to detach the mylohyoid muscle to gain access and visualization of crown. This can be difficult due to bleeding. Allow 6 weeks for fibrosis. Get a CT scan to localize the root. Patient may require a transcutaneous approach via a submandibular incision for retrieval.
- *Displacement into Infratemporal Space* – Likely due to lack of retractor protection with excessive force and poor visualization. Position most commonly lateral and inferior to pterygoid plate. May attempt to manipulate the tooth back manually into incision by placing finger high into vestibule near the plates and applying manual pressure. If good access and lighting, may attempt to extend incision and retrieve with hemostat. If primary efforts fail, allow 4–6 weeks to allow for fibrosis. Obtain a CT scan and use a spinal needle to identify, dissect along needle length. Needle-guided fluoroscopy may also be used. It also has been reported to perform a hemicoronal incision to gain access to infratemporal space. If no functional deficit and asymptomatic, may elect to leave in place.
- *Displacement into IAN Canal* – Retrieval attempts may lead to nerve damage, and single attempt with suction should be attempted. If root is not infected and no neurological abnormalities, consider leaving in place. If sensory complications, must retrieve. CT scan should be taken to ensure whether in canal space versus medial to mandible. IAN root retrieval may be attempted by unroofing the extraction site, lateral window intraoral, or via submandibular incision.
- *Aspiration of Foreign Object* – Heimlich maneuver may be attempted while patient is in beach chair position. If under anesthesia, deepen the level of sedation and attempt visualization and removal with Magill forceps. Cord pressure may help move objects caudally past the cords. If no respiratory distress, likely ingested, obtain abdominal and chest radiography to rule out. Always presume aspiration and place patient on right side in Trendelenburg. Continue monitoring and watch out for signs of hypoxia and respiratory distress. Refer to emergency room for removal.

Management of Impacted Maxillary Canines

Background

- Maxillary canines are the second most frequently impacted teeth (third molars are the most commonly impacted teeth).
- Maxillary impacted canine incidence of approximately 2% and mandibular 0.4% [8].
- 2:1 female to male ratio.
- Canines normally erupt between 11 and 12 years of age.
- Maxillary canine erupts along the lateral incisor, closing the diastema.
- Labial impacted canines thought largely due to arch length discrepancies.
- Palatal impacted canines more often seen in patients with peg laterals or missing laterals.
- Two theories:
  1. Genetic theory – genetic disposition or dental anomalies.
  2. Guidance theory- as the maxillary canines erupt along the lateral incisors, malformation or lack of the lateral incisor leads to failure of canine to erupt.
- Other possible causes are trauma, pathology, genetics, and malposition tooth germ.

Workup

Review CC/HPI and pertinent medical history.

Head and Neck Exam

- Pay attention to bulging of tissue for location of the impaction. Expect canine bulge on buccal by age of 10. Normally will have eruption by 1 year later.
- Look at the overall gingival health and quality of gingiva (thick vs. thin/presence of gingivitis). Quantify the amount of keratinized tissue. This may influence surgical approach as keratinized tissue is desirable for long-term periodontal health.
- Lateral incisor presence and position. Is the incisor compromised in size or shape? The canine will erupt along the lateral incisor.
- Examine airway for tonsil and adenoid size in children for planned sedation. It is normal to see large tonsils in children.
- Mobile teeth – aspiration risk or damage during procedure.
- Is the patient in active orthodontic treatment? (i.e., Are the brackets on the teeth? Is there an arch wire in place? Is the patient ready for treatment?) Where will the chain be secured? Has adequate space been made for canine (average 7.5 mm mesial-distal space at contact area)?

Localization/Radiography

- CBCTs.
- Clark's rule/shift rule (SLOB) – same lingual, opposite buccal.
- Orthopantogram – if horizontal, larger and out of focus likely palatal impaction. Labial impactions appear vertical in position.
- Orthopantogram and occlusal film (vertical parallax method).
- Examine root development. If vertical position and non-complete root development, can expect some movement. If apex closed, do not expect much potential for further eruption.
- Is there resorption of permanent central or lateral incisor? This requires exposure of permanent canine to correct path and reduce resorption.
- If canine crown is overlapping less than half of the root of lateral incisor, there is 91% chance of normalization of eruption path (if not this drops to 64%) [9].

Orthodontic Considerations

- Create room for the canine before exposing and orthodontically erupting the tooth.
- Stabilize teeth in arch with full thickness passive wire to allow anchorage to pull teeth through tissue.
- Average mesial-distal dimension maxillary canine is 7.5 mm.
- The more perpendicular the canine is to the lateral incisor, the more likely it should be extracted as opposed to being exposed and brought into occlusion.

Treatment Options

1. *Interceptive*
   - Extract primary canine before age 11 if not palpable on buccal. Expect 91% success

for eruption if crown distal to midline lateral incisor. Success drops to 64% if mesial to midline of lateral incisor [9].

2. *Apically Positioned Flap*
   - For labial impactions and not for displaced mesial or distal.
   - Maintains keratinized gingiva.
   - Use if less than 3 mm of keratinized gingiva is expected after an open window technique.
   - Do not use for canines high in alveolus as the thick palatal bone can push the canine buccally and create a dehiscence in the tissue. High labial impactions should be treated with a closed technique.
   - Technique:
     – Flap outline in the mesial distal width of tooth.
     – Remove bone over surgical margin and remove follicular remnants.
     – Reposition flap apically at cervical margin.
     – Tooth surface etched for 30 seconds with phosphoric acid 30% (know what you use and its requirement), then irrigate thoroughly.
     – Place primer if part of a step system and lightly puff air to make even spread.
     – Bonding agent of choice is, for example, glass ionomer (fluoride is released and can work in partially wet environment).
     – Suture keratinized tissue apically around CEJ of tooth.
3. *Open Exposure Technique (Window Technique)*
   - Crown is uncovered and left exposed. Some surgeons use electrosurgery/steel to expose tooth based on radiography. Other surgeons open a palatal flap, expose tooth, and create window after identification.
   - Ortho bracket may or may not be placed at the time of surgery.
     – Window of overlying gingival is removed or reflected.
     – Tooth may spontaneously erupt, or the site can be packed with periodontal packing open with or without bracketing (speak to orthodontist preference).
     – Packing normally left for 2–3 weeks.
4. *Closed Techniques*
   - Used when teeth are not in a position to allow for repositioning of the flap after crown is exposed.
   - Palatal impaction that is not close to the alveolar ridge.
   - Technique:
     – Local with vasoconstrictor.
     – Full thickness flap raised and the impacted tooth is exposed to level CEJ.
     – Tooth surface etched for 30 seconds with phosphoric acid 30% (know what you use and its requirement), then irrigate thoroughly. Acid etching can also aid in hemostasis.
     – Ensure adequate hemostasis; some surgeons opt to place retraction cord soaked with vasoconstrictor. This cord must not be forgotten.
     – Place primer if part of a step system and lightly puff air to make even spread.
     – Bonding agent of choice: e.g., glass ionomer (fluoride is released and can work in partially wet environment) with placement of bracket and chain.
     – Test chain to ensure there is a good bond.
     – The chain is then secured to a bracket or arch wire with a suture or wire.
     – The wire or chain may need to pass through the flap through a small incision.
     – Flap is sutured back into place.
   - Ortho can begin traction after ~1 week of tissue healing.

Complications

- *Failure of Eruption* – Re-explore the area and check for ankylosis. May percuss tooth, and if a metallic higher pitched sound heard, this could be an indication of ankylosis. Luxation of tooth may aid in mobility as this can cause bleeding and inflammation to encourage movement. May present as intrusion or teeth within arch. May consider segmental osteotomy or corticotomies to aid in movement. Consider other options including tooth

removal, space maintenance for dental implant, bicuspid substitution, or autotransplantation.
- *Resorption of Tooth* – Can occur from over-aggressive exposure (removal of bone below the CEJ). Halt orthodontic movement and re-evaluate treatment plan. Consider other options including tooth removal, space maintenance for dental implant, bicuspid substitution, or autotransplantation.
- *Lack of Attached Gingiva* – Due to poor quality of gingival mucosa, over-aggressive tissue removal and inappropriate treatment selection such as an open window happen. A connective tissue graft in future may aid in correction.
- *Bracket Detachment* – Can be due to ankylosis, disruption in path of guidance, over-aggressive movement, poor bonding, or outdated material. Re-exposure of tooth, check for ankylosis. Consider reattachment with removal of old bonding material and slight roughening of tooth (consider polish with pumice, diamond bur, or gentle painting with a carbide bur) to aid attachment or convert into open technique if a closed technique was used. Do not use ligature.

Pearls of Wisdom
- Least desirable way to obtain attachment of an impacted crown is to place a wire ligature around it. It will result in loss of periodontal attachment and root resorption (no longer recommended).
- Do not remove bone past CEJ, as it leads to root resorption, ankylosis, and periodontal disease. Over-exposure of tooth to beyond the CEJ further causes a junctional epithelium that will be severed irreparably. This leads to a longer clinical crown and poor periodontal condition months after exposure.
- A gold mesh chain is preferred for a light cured bonding agent as it allows penetration of the light.
- General consensus now on luxating tooth is not to. Luxation may actually initiate cervical root resorption and ankylosis, causing failure of orthodontic traction attempt.
- Place bracket as close to the incisal edge as possible for best mechanical traction.
- Remove excessive lag on chain as this is an annoyance to the patient and increases the chance of debonding of the bracket.

Impacted Canine Case

- *A healthy 14-year-old male presents for exposure of maxillary canine from the orthodontist. What would you like to know? What do you evaluate?*
  I would like to know the medical history and about previous surgeries requiring anesthesia. I would like to measure the adequacy of the mesial-distal space to ensure correct amount of room for orthodontic interception. I would assess the overall gingival health and the amount of keratinized gingiva. Is there a palpable bulge? I would conduct an airway exam (including tonsil size, Mallampati, etc.) and assess the entire dentition (loose teeth, any other missing or impacted teeth to suggest syndrome).
- *There is 9 mm of space between teeth # 5 and 7. There is 4 mm of keratinized gingiva. Airway evaluation shows grade 2 tonsils with a Mallampati score of 2. No history of anesthesia. No other missing or impacted teeth. Healthy gingivae, no signs of bleeding. What imaging would you like?*
  CBCT (Fig. 1.3).
- *What do you see on the CBCT?*
  Maxillary impacted tooth # 6 proximal to the floor of the nose with buccal positioning. No enlarged follicle or pathology appreciated. Resorption of the tooth root of #7 noted.
- *How would you want to treat this?*
  I would want to approach this with a closed technique.
- *Could you not do an apically positioned flap?*
  No, it would not be feasible in this situation as the tooth is too high up the alveolus. I would not have adequate area to secure the flap. Additionally, it is mesially positioned, so it would expose the bone over the lateral incisor.

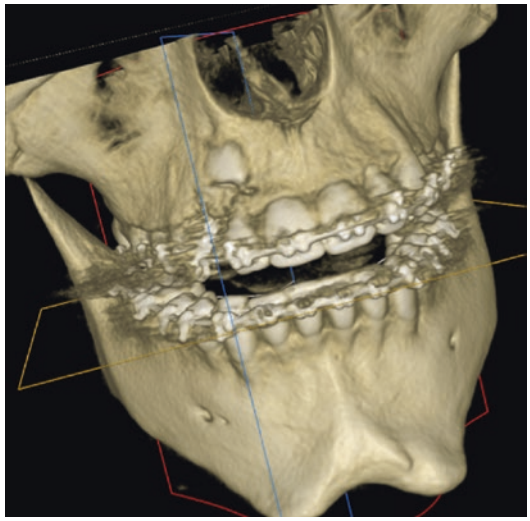

Fig. 1.3 Cone beam CT. (Image courtesy of Dr. Robert Reti)

- *What you would talk to the orthodontist about?*
  I would discuss whether a bracket and ligation is desired and timing with the orthodontist.
  (Some orthodontists want you to wait until there is enough room. Creating arch space for the canine. Enquire as to whether they would like a chain/bracket attached.)
- *What would you want to do about tooth #7?*
  There is apical resorption. I would explain to the patient and the parents the guarded prognosis of #7 and recommend vitality testing. During traction, the orthodontist would need to plan for the vector to prevent further damage.
- *How would you expose this tooth?*
  I would administer local anesthetic with a vasoconstrictor and allow 5 minutes for hemostasis. Reflect a flap with wide exposure to allow retraction of the flap. Identify the tooth and remove adequate buccal bone and follicle to the level of the CEJ. I would place retraction cord soaked in local anesthetic with vasoconstrictor around the osteotomy of the tooth. Etch and prime the tooth. I would bond a gold mesh bracket with glass ionomer light curing bonding agent (to the center and closer to the incisal edge). I would test to ensure adequate fixation by placing traction on the chain. I would secure the chain to a bracketed tooth (or arch wire) and remove excessive chain links. I would remove packing and ensure adequate hemostasis. I would replace the flap with 3-0 chromic gut sutures.

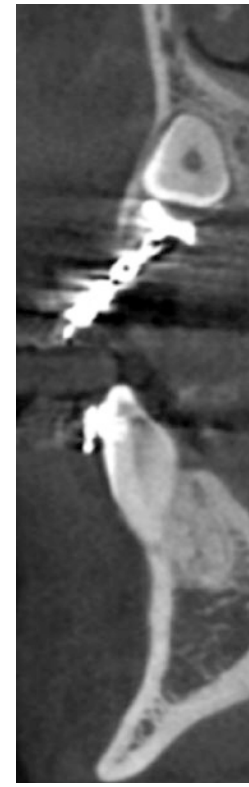

Fig. 1.4 Cone beam CT: sagittal view. (Courtesy of Dr. Robert Reti)

- *What kind of bonding agent would you use?*
  Glass ionomer.
- *Why?*
  It can work in a wet field and release fluoride to prevent decay.
- *How do you apply it?*
  Press firm, light cure for 20–40 seconds.
- *When should the patient see the orthodontist after application?*
  Within 1 week of activation.
- *After multiple attempts, you're unable to bond the bracket to the tooth, what's your alternative?*
  Convert into an open window technique, pack site and have the orthodontist place bracket in one week (High risk for thin or lack of keratinized gingiva).
- *Six weeks later, your orthodontic referral asks you to re-evaluate the area as he has not had*

any movement. You take a CBCT and you see this (Fig. 1.4). *What is going on?*
The chain has debonded from the tooth.

- *What could have been the reasons and why?*
  Wet field, obstruction in path, old material used, and tooth is ankylosed.
- *What would you like to do?*
  I would discuss with the orthodontist and patient the need for re-exposure and bracketing. It would be prudent to also discuss the possibility that the tooth may be ankylosed and another treatment plan should be considered if no movement occurs after reattachment. Alternative treatments such as removal with space maintenance for future dental implant placement, autotransplant, or bicuspid substitution if failure of a second attempt or noted ankylosis.
- *Would you put a cervical ligature to grasp the tooth?*
  No, as this causes external resorption of the tooth and is no longer practiced.

Impacted Third Molar Case

- *A 18-year-old female present with history of waxing and waning pain of her lower jaw. Her dentist placed her on a course of antibiotics, which resolved her discomfort. She is healthy and has no allergies and no surgical history. She was referred by her general dentist for third molar extraction. What would you like to do next* (Fig. 1.5)*?*
  I would review the medical/surgical history and follow-up with questions; for example, does she play any contact sports and how often does she have these flare ups? Which antibiotics has she used to treat this issue? Does she have any TMD symptoms? I would conduct a complete head and neck exam. This would include evaluating her maximal incisal opening, the overlying tissue of the region of the third molars, probing depths on the distal of the second molars, overall hygiene, and condition of the adjacent teeth.
- *She plays field hockey for her high school team. No joint complaints. The last flare up was 3 months ago, but she doesn't recall the frequency. She was prescribed penicillin VK by her dentist. The evaluation shows erythema distal to teeth 17 and 32 with a probing of 6 mm distal to 32 and 5 mm of #17. The adjacent teeth are in good condition.*
- *Can you describe what you see on the orthopantogram?*
  Teeth 1, 16, 17, and 32 are full bony impacted wisdom teeth. All teeth appear two-thirds in development. No intrabony pathology is appreciated. Teeth 17 and 32 have enlarged follicular sacs. This is a diagnostically unacceptable radiograph, as it is missing the complete head of the condyles and ramus. It does show the

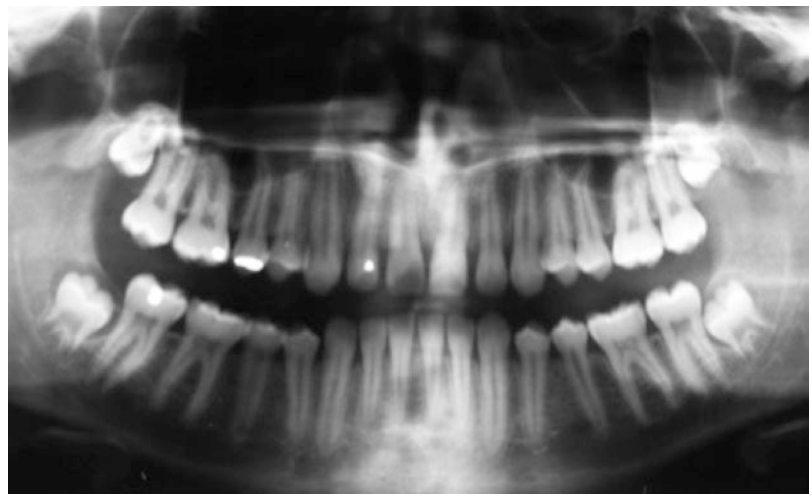

Fig. 1.5 Orthopantogram for case. (Image courtesy of Dr. Damian Findlay)

third molars in question and other vital structures. However, I would recommend taking a second orthopantogram, which includes the temporomandibular joint anatomy.

- *Why do you think she is having pain that comes and goes?*
  She likely has bouts of pericoronitis.
- *Would you remove these third molars or watch them?*
  As she has a history of pain, clinical signs of pericoronitis, periodontal probing greater than 5 mm, and a history of contact sports, I would recommend removal at this time.
- *If there was no history of pain, would you still consider removal?*
  Yes, to optimize the periodontal health of her second molars. There is also evidence that patients under 25 have a much lower rate of complications and improved recovery time. As she is 18 years old, mandibular growth has likely ceased; therefore, I would not foresee adequate arch space with further eruption. Also, it is unlikely that the teeth will change position due to her age.
- *What would your sequence be? How would you remove tooth #1?*
  I would extract the mandibular thirds first followed by the maxillary thirds to prevent blood entering the field from above. After achieving adequate local anesthesia, I would make a sharp crestal flap from the mesial of tooth #2 extending onto the maxillary tuberosity. A periosteal elevator would be used to carefully elevate the flap. I would remove the bone covering the crown to allow for adequate visualization up to the CEJ of tooth #1. I would apply pressure apically with my retractor to the flap in the region apical and distal to tooth #1. I would carefully luxate tooth #1 to ensure mobility and retrieve with a forcep. I would smoothen the bone with a rasp, curette the socket from granulation or dental follicle, and irrigate the site with copious irrigation. I would then reapproximate the tissue with 3-0 chromic gut suture to ensure primary closure.
- *You look at your tooth and notice one of the roots is incomplete, comparing the adjacent roots and your radiography it would be fair to estimate 1 mm of root remains, what would you do next?*
  I would explore the socket and see if the root is mobile or easily visualized. If it is easily accessed, I would attempt retrieval. If there is confirmation that the root tip has been displaced into the sinus or infratemporal fossa, then I would consider leaving the root (if the root is less than 3 mm, non-infected) to prevent damage to proximal vital structures. I would make a note in the chart as well as inform the patient. I would get baseline imaging at 6 months and then at one year.
- *You are paged to the ED for evaluation of a 56-year-old healthy female with mild dysphagia 3 hours s/p extraction of teeth 17 and 18 with complaint of difficulty breathing and swallowing. Her oxygen saturation is stable at 98% on room air. Exam reveals a well-developed well-nourished female in no acute distress with stable vital signs. Oral exam reveals a somewhat firm sublingual swelling adjacent to the extraction site #17. Past medical history is significant for high blood pressure that is treated with Lisinopril. NKDA. What do you want to do next?*
  Review a complete past medical history and further clarification for potential familial history of coagulopathies or recent street drug use.
- *This is all negative, now what?*
  Obtain a CT of the neck with contrast. I would also order CBC and coagulation studies.
- *CBC shows white count in high normal range and coagulation studies come back as normal. The CT scan shows the area of concern* (Fig. 1.6). *What is the most likely diagnosis?*
  Sublingual hematoma secondary to violation of the lingual cortex during extraction.
- *How would you manage this?*
  If stable, then watchful waiting is appropriate with serial CT neck with contrast scans every 6 hours to evaluate for expansion. If no expansion, no surgical intervention would be required. If actively expanding, I would intubate for airway protection. Transfer to the operating room where a lingual flap would be raised for evacuation of the hematoma and identification of bleeding source. If the source

cannot be identified, interventional radiology should be consulted to aid in identification and possible embolization.

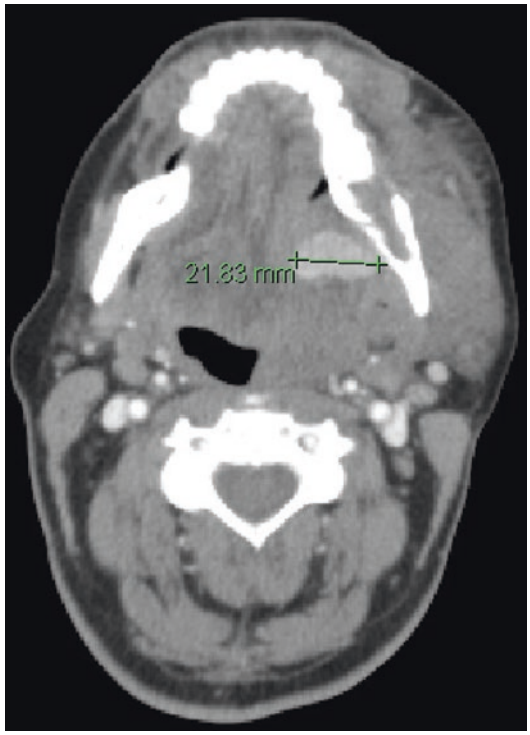

Fig. 1.6 Complication of tooth extraction. (Image Courtesy of Dr. Robert Reti)

Trigeminal Nerve Injury

Nerve Terminology

Endoneurium – connective tissue sheath that surrounds group of fibers forming a fascicle.

Perineurium – connective tissue surrounding a bundle of fascicles within a nerve.

Epineurium – outermost layer of a peripheral nerve, surrounding multiple fascicles and blood vessels (Fig. 1.7).

Classification of Nerve Injuries

- The rate of permanent injury to the lingual nerve from third molar surgery ranges between 0.04% and 0.6%, whereas for the IAN it ranges between 0.1% and 1% [4, 5, 10].
- The incidence of persistent nerve impairment of the IAN 1 year after BSSO surgery is reported to be 33% [11].
- IAN has a greater chance of spontaneous recovery (bony canal acts as a conduit for guided regeneration).
- Seddon in 1942 classified severity of nerve motor nerve injuries based on histology (Dr. Cohen actually first used the terms neuropraxia, axonotmesis, and neurotmesis).
- Seddon classification of peripheral nerve damage is the most commonly used for catego-

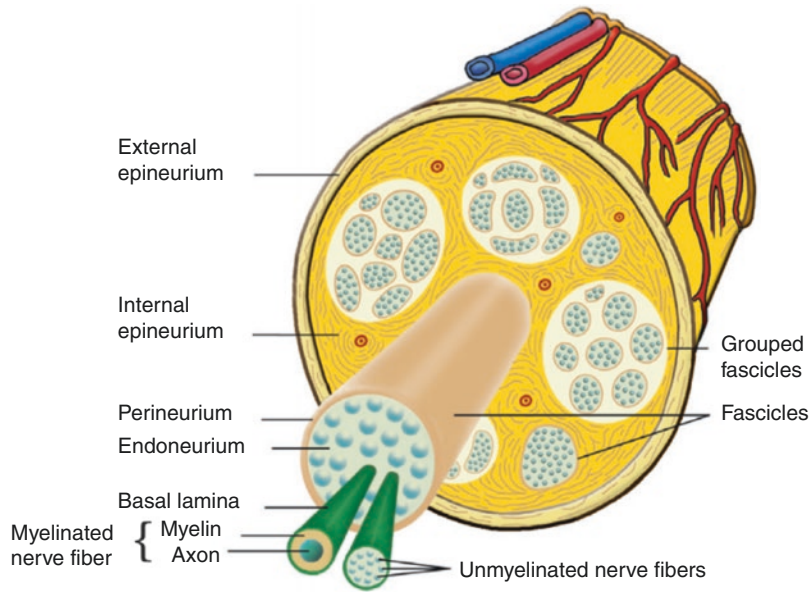

Fig. 1.7 Nerve anatomy. (Reprinted with permission from Winn [14])

rized into three groups: neuropraxias, axonotmesis, or neurotmesis. Sunderland's classification basis the injury on level of anatomic injury and expands on Seddon's classification to five degrees. The classification system aids in directing treatment (Table 1.2).
- Nerve recovery progresses slowly at 1 mm/day or 1 in/month.
- More recently the Medical Research Scale developed for brachial plexus injury monitoring has been modified to allow comparison between studies (Table 1.3).
- Simple to use, only three assessments, all of which can be done with a cotton plier: 1) pain (deep or superficial), 2) touch, and 3) 2-point discrimination (Table 1.4).

Pain Terms

- Allodynia – pain from a non-painful stimulus.
- Anesthesia – absence of any sensation.
- Anesthesia dolorosa – deafferentation pain is pain felt in an area, which is completely anesthetic to touch.
- Hyperalgesia – increased response to stimulation that is normally painful.
- Hyperpathia – prolonged pain following a repetitive noxious stimulus that lingers beyond expected duration.
- Hypoalgesia – diminished response to a normally painful stimulus.
- Hypoesthesia – decreased sensitivity to stimulation.

Table 1.2 Seddon and Sunderland classification

Classification of nerve injury				
Seddon	Sunderland	Description	Treatment	Outcomes
Neuropraxia	1	Temporary disturbance in nerve conduction, resembles common effect of anesthesia Axon continuity preserved Caused by nerve trunk traction or compression Type I (<24 hours): Mild manipulation, compression, traction Type II (<1 week): Moderate manipulation, compression, traction Type III (<1–2 monthos): Severe manipulation, compression, traction	No treatment	Spontaneous recovery, hours to 3 months
Axonotmesis	2	Loss of axonal continuity Endoneurium and perineurium intact Forceful compression or nerve traction greater than 25 g Wallerian degeneration distal to injury	Surgery only if foreign body	Possible spontaneous recovery takes 2–4 months
	3	Endoneurium and axonal loss of continuity Perineurium intact Severe crush, puncture, chemical, thermal trauma, or needle track injury	Microsurgery if no improvement for 3–4 months	Possible spontaneous recovery takes 3–4 months, unlikely complete
	4	Loss of endoneurium, perineurium Epineurium intact Extreme crush, thermal injury, intraneural local anesthetic injection, or caustic substances	Microsurgery if no improvement for 3–4 months	
Neurotmesis	5	Complete transection, interruption with or without neuroma formation	Requires microsurgery	No spontaneous recovery
Neuroma	6	Mackinnon used sixth degree to describe a neuroma in continuity	Requires microsurgery	No spontaneous recovery

Table 1.3 Modified Medical Research Council Scale

Modified Medical Research Council Scale	
Grade	Description
S0	No sensation
S1	Deep cutaneous pain in an autonomous zone
S2	Some superficial pain and touch sensation
S2+	Superficial pain and touch plus hyperesthesia
S3	Superficial pain and touch sensation without hyperesthesia and static 2 point discrimination >15 mm (useful sensory function)
S3+	Same as S3 with good stimulus localization and static 2 point discrimination 7–15 mm
S4	Same as S3 but static 2 point discrimination 2–6 mm (complete sensory recovery)

Table 1.4 Normal two-point discrimination distances

Test area	Average normal threshold distance (mm)	Upper normal limit (mm)
Upper lip (skin)	4.5	8.0
Upper lip (mucosal)	3.0	6.0
Lower lip (mucosal)	3.5	6.5
Lower lip (skin)	5.0	9.0
Chin	9.0	18.0
Tongue (tip)	3.0	4.5
Tongue (dorsum)	5.0	12.0

- Paresthesia – abnormal sensation, whether spontaneous or evoked and is not unpleasant.
- Tinel sign – tingling or "pins and needles" sensation elicited upon tapping on the distribution of the nerve. Thought originally to be an effect of regenerating nerves and also may be misinterpreted as neuroma formation.
- Wallerian degeneration – distal degeneration of the axon and its myelin sheath after injury may result from passive wasting of the distal axonal fragment due to lack of nutrient supply.

Workup

Patient assessment

CC:/HPI:/PMHx:

- Date of injury, type of procedure, type of pain (VAS), changes in taste (parageusia), characteristics of "numbness" progression (better or worse), interference in daily life (e.g., lipstick, shaving, and tooth brushing), speech, chewing, intimacy, and consult with previous surgeon.
- If no insulting surgery, concern for pathology such as metastatic tumor, osteomyelitis, central nervous origin, etc.
- Time frame needs to state first 3 months critical for intervention, by 12 months distal nerve tissue too damaged and ganglion cell death decreases likelihood of recovery.
- Patient to be followed every 2–4 weeks, if no improvement in subsequent visit, after 3 months do not expect improvement in future visits.
- When performing neurosensory testing, always test the affected side and compare to contralateral non-injured side if available.

Neurosensory Testing (NST)

- The area of altered sensation is delineated with the marching needle technique. A 27-gauge needle is marched in 1–2 mm increments from unaltered side to altered side until patient denotes sharp to dull in all directions, and then is marked with a skin pen.
- Compare normal side to abnormal side during NST testing. A score of 8 or less correct responses on the abnormal side is considered impaired (scale out of 10).

Level A – Spatiotemporal Perception

- A-α and A-β fibers.
- Moving brush stroke identification – cotton swab, Semmes-Weinstein monofilament, or camel brush hair.
- Two-point discrimination – caliper and Boley Gauge, cotton pliers or Disk-Criminator®. In general, the normal range for the IAN is 4 mm

and 3 mm for lingual nerve. Anything greater than 6.5 mm is considered abnormal.
- Stimulus localization – touch patient with tip of a cotton stick and ask patient to localize the stimulated area with their finger. 1–3 mm off examiner point is considered normal.
- If testing judged normal, no further testing required.

Level B – Static Light Touch

- A-β fibers.
- Touch skin with end of a cotton tip applicator; if able to detect, then normal, and if can feel only when skin indented, this is an increased threshold which is abnormal.
- Semmes-Weinstein monofilaments or von Frey hairs – touch skin to just create bend in filament on the normal side. Compare this value of fiber on the abnormal side.

Level C – Nociception

- A-δ and C fibers.
- 27-gauge needle without indentation of skin should evoke painful response.
- May discriminate A-δ with heated gutta percha vs. cold from ethyl chloride for C fibers.

Diagnostic Nerve Blocks – if patient complains of altered sensation and abnormal NST, a nerve block may be given to establish if pain is from injured peripheral nerve or central source. If no relief given, likelihood of nerve repair relieving pain is unlikely.

Nerve Repair
- Preoperative imaging such as CBCT or an orthopantogram can give insight into foreign body, retained root, hardware, or bony damage. They do not give information on the nerve integrity.
- Indications for nerve repair:
  - Observed nerve transection
  - Complete postoperative anesthesia
  - Persistent anesthesia >1 month without improvement
  - Presence or development of dysesthesia
  - Paresthesia without improvement >3 months
  - Foreign body in canal
  - Patient unable to tolerate hypoesthesia
- External neurolysis (decompression) – first step to microsurgical repair, involves exposing nerve from soft tissue bed without disruption of epineurium. May be only surgical maneuver required if mild sensory disturbances and without neuroma.
- Neuroma excision – resect 3 mm proximal and distal. Examine fascicles under magnification for opacity and architecture and check for scarification by pressing on the nerve with micro forceps. Adequacy may also be tested by frozen sections 1 mm cross-section biopsies.
- Direct neurorrhaphy – 4–6 circumferential epineural sutures with 7.0–9.0 non-ophthalmic nylon sutures. Lingual nerve gap of 1 cm and IAN gap of 5 mm possible for direct repair (without need for interpositional graft). Minimal tension of 25 g or less to prevent axonal gapping and prevent axon downgrowth to the distal nerve.
- Nerve grafting – requires 25% longer graft than defect due to shrinkage. Sites include sural, greater auricular and the antebrachial cutaneous nerves, dorsal cutaneous branch of the ulnar nerve, medial antebrachial cutaneous, superficial branch of the radial, and other nerves of the cervical plexus (Table 1.5). Most common autografts are sural and greater auricular due to ease of harvest and minimal postoperative morbidity. Important to orient the nerve graft in a functional direction, proximal-proximal, and distal-distal.
- Processed allograft AxoGen Avance® is a non-immunogenic alternative that provides a scaffold for nerve tissue to grow. Provides unlimited length and no donor site morbidity.
- Entubulation – best for gaps <10 mm. Polyglycolic acid conduits start to break down in 3 months and are resorbed by 8 months. Vein and artery grafts have mixed success but have been used. Collagen type I tubes available in 1–3 cm length.

Table 1.5 Nerve anatomy

Nerve anatomy				
Nerve	Fascicles	Diameter (mm)	Length harvest	Morbidity
IAN	18–21 in third molar region and 12 in the area of mental foramen	2.4	N/A	N/A
Lingual	15–18	3.2	N/A	N/A
Sural	11–12	2.1	20–30 cm Found below and posterior to lateral malleolus between the gastrocnemius tendons	Anesthesia of heel and lateral foot, temporary gait disturbance
Greater Auricular	8–9	1.5	2–4 cm Found by drawing line that bisects a line from the mastoid process to mandible	Anesthesia lateral neck, posterior mandible, and earlobe; smaller diameter may require cable graft

Prognosis

- Overall success rate of around 50%.
- 70% of patients with painful neuromas are helped regardless of surgical technique.
- All patients require sensory education after nerve repair.
- Hypoesthetic nerve injuries have a higher success rate than hyperesthetic injuries.
- Delays >6 months have poorer outcomes.

Coronectomy [12]

- Partial tooth removal leaving roots behind to prevent inadvertent IAN damage.
- Contradictions include:
  1. Horizontal impaction with tooth along length of nerve, risk sectioning higher than complete removal.
  2. Inability to access or removal all enamel layer.
  3. Infection of roots.
  4. Plan for distalization of second molar.
  5. Mobility of roots.
- Surgical technique requires removal of all enamel and root remnant 3 mm below the alveolar crestal bone.
- Antibiotics are of surgeon's preference and get rid of the similar results with and without.
- Primary closure has not shown increased success.
- Roots migrate about 30% of the time and can be appreciated in first 3 months as an apparent radiolucency below the roots with coronal migration.
- It should be noted that other techniques to reduce nerve damage include orthodontic extrusion (if nerve does not perforate roots) and sequential coronal reduction.

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Temporomandibular Joint Dysfunction

2

Rishad Shaikh, Damian Findlay, and Robert Reti

Anatomy

- The temporomandibular joint (TMJ) is a ginglymoarthrodial joint with translational movement in the superior joint space and rotational movement in the inferior joint space (Fig. 2.1).
- The capsular ligament or joint capsule is a functional ligament that surrounds the joint (attaching to the temporal bone and surrounds the condylar head/neck circumferentially).
- The capsular ligament is lined by the synovium, which functions to provide nutrition and immunosurveillance and lubricates the joint.
- The other two functional ligaments are the collateral ligaments and the temporomandibular ligaments.
- The accessory ligaments are the sphenomandibular and the stylomandibular ligaments.
- The articular disk is composed of fibrocartilage. It has three zones (anterior band, intermediate band, and posterior band). Posterior to the disk are the retrodiscal tissues, which are highly vascular and innervated.
- Primary joint movement is determined by the muscles of mastication (masseter, lateral pterygoid, medial pterygoid, and temporalis) and the inframandibular accessory muscles serve to impact mandibular function secondarily.
- The vascular supply of the TMJ is primarily from branches of the superficial temporal, maxillary, and masseteric arteries.
- The nerve supply of the TMJ is predominantly from branches of the auriculotemporal with contributions from the masseteric and posterior deep temporal nerve.

Myofascial Pain Dysfunction (MPD)

Definition – non-articular TMJ disorder that manifests itself as dull regional masticatory myalgia that worsens with function and can lead to a decreased range of motion. It can involve the muscles of mastication and any combination of the supramandibular and inframandibular muscle groups. This is the most common TMJ disorder.

Etiologies

- Parafunctional habits such as bruxism, nail biting, clenching, or gum chewing.
- Life stressors.
- Apertognathia and/or overjet greater than 6 mm.
- Lack of posterior dentition leading to muscle hyperactivity.

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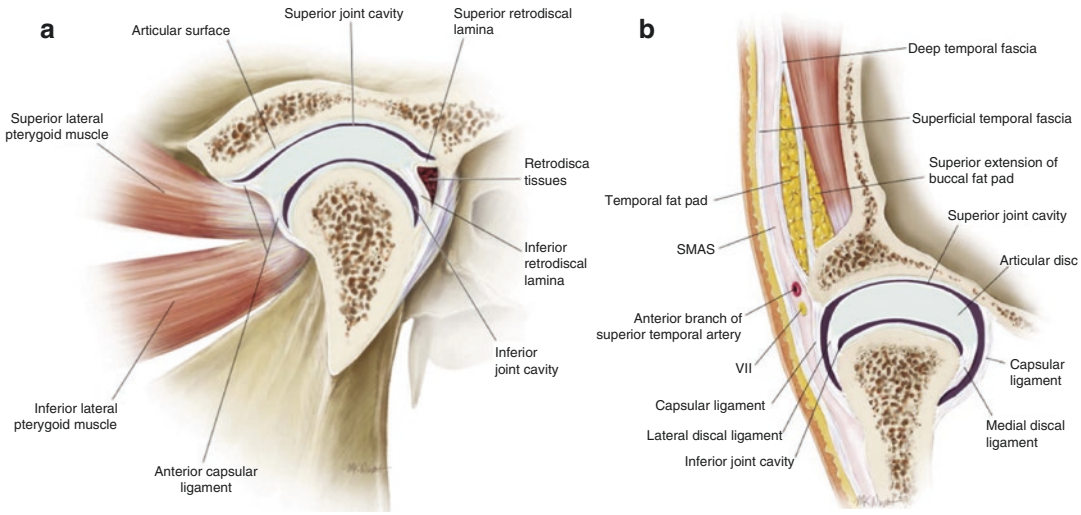

Fig. 2.1 Temporomandibular joint anatomy. (**a**) Lateral view. (**b**) Sagittal view. (Reprinted with Permission from Kadenami and Patel [9])

Clinical Manifestations

- Jaw pain with tenderness of the muscles of mastication and/or supramandibular and infrahyoid muscles.
- May see wear facets of the dentition.
- Scalloping of the tongue.
- Morsicatio buccarum, laborium, or linguarum.
- Patients may complain of sore teeth.
- Decreased range of motion.
- Buccal exostoses (Wolff's law states that bone responds to the pressure exerted on it by an osteoblastic response).
- Patients often will complain of pain on the side of their face as opposed to pointing to the preauricular region. Pain is usually diffuse and involves the muscles of mastication (DDx of MPD-trigeminal neuralgia, atypical facial pain, fibromyalgia).

Treatments

- NSAIDS to reduce pain and inflammation (e.g., ibuprofen 600 mg QID × 2 weeks, naproxen 500 mg BID × 2 weeks, Mobic 7.5–15 mg daily for 2 weeks)
- Occlusal appliances
- Soft diet
- Muscle relaxants:
  - Cyclobenzaprine 5–10 mg daily QHS, baclofen 5–10 mg TID (Some advocate for prescribing these medications TID. There is concern for dizziness/sedation, which is why some surgeons choose to prescribe it QHS.)
- Warm compresses
- Occlusal equilibration
- Trigger point injections
- Botox intramuscular injections
- Replacing the posterior dentition
- Physical therapy

Degenerative Joint Disease

Definition – a chronic inflammatory arthritis within the TMJ resulting in degradation of articular cartilage with remodeling of the subchondral bone.

Non-inflammatory Degenerative Joint Disease (aka Osteoarthritis)

- Due to an imbalance between catabolic and anabolic processes. This leads to expression of catabolic cytokines (TNF-alpha, IL-1, IL-6), initiating liberation of collagenases and proteases that result in degradation of the articular

cartilage. Osteoarthritis of the TMJ can be preceded by internal derangement and trauma and can also develop in patients that have had orthognathic surgery.

Inflammatory Arthritis

- Joint destruction due to an inflammatory arthritic process (e.g., rheumatoid arthritis (RA), juvenile rheumatoid arthritis, psoriatic arthritis, gout, pseudogout, ankylosing spondylitis, reactive arthritis).

Treatment

- Depends on the extent and the level of life disruption. May include medications, physical therapy, or steroids or disease-modifying drugs.
- For mild cases failing conservative treatments, consider arthrocentesis and arthroscopic procedures.
- More advanced cases may require arthroplasty or total joint replacement.

Internal Derangement of the Temporomandibular Joint

Definition – disorder of the TMJ in which the articular disk is in an abnormal position as it relates to the condyle and fossa when the teeth are in occlusion. Malposition of the disk may lead to pain, instability, decreased range of motion, and abnormal mobility of the mandible.

Etiologies

- Trauma
- Joint laxity
- Parafunctional habits
- Altered joint lubrication system
- Anchored disk phenomenon (disk adhesion to articular fossa)
- MPD

Diagnosis

- Look for decreased maximal incisal opening (MIO), deviation, deflection, palpable clicks (reciprocal), and crepitus. Patients often will complain of pain in the preauricular region as opposed to pointing to the side of the face.
- Diagnosis by MRI-T1 and T2. Disk is normally displaced in an anteromedial vector. Can see osseous changes and abnormal contours of the disk.
- Disk displacement with reduction – patient opens the mouth with an accompanying click that is produced when the condyle passes over the posterior portion of the disk. During opening, the disk returns to its normal anatomical position in relation to the fossa and condylar head. During closing, a second click can be appreciated as the condyle passes back over the thickened posterior portion of the disk.
- Disk displacement without reduction – patient attempts to open but the condyle cannot pass over the posterior band of the disk. May see deflection to the ipsilateral side and decreased excursion to the contralateral side. This results in limitation of opening.
- Wilkes classification classifies the degree of internal derangement and provides guidance in relation to treatment options (Table 2.1).

Table 2.1 Wilkes classification of internal derangement

Wilkes classification [1]			
Stages	Clinical findings	Radiographic findings	Surgical findings
Stage I	Painless clicking No pain or locking	Anterior disk displacement noted. Disk contour remains normal with no osseous changes	Normal disk noted and displaced anteromedially
Stage II	Occasional painful clicking with intermittent locking	Anterior disk displacement noted with reduction on opening Mild disk deformity with no osseous changes	The disk appears thickened and displaced anteromedially.

(continued)

Table 2.1 (continued)

Wilkes classification [1]			
Stages	Clinical findings	Radiographic findings	Surgical findings
Stage III	Frequent painful clicking with severe limitation in range of motion Joint tenderness noted	Anterior disk displacement noted without reduction Moderate disk deformity with no osseous changes	The disk is deformed and displaced anteromedially Adhesions may be appreciated
Stage IV	Restricted range of motion with chronic pain and joint crepitus	Anterior disk displacement noted without reduction Marked disk deformity with osseous changes	The disk is perforated with noted osseous changes of the condylar head and the fossa
Stage V	Joint pain and crepitus	Disk is displaced Marked disk deformity with severe osseous changes	The disk is perforated with noted severe osseous changes of the condylar head and the fossa

Treatment

- Conservative treatment as previously mentioned (if appropriate).
- Intra-articular injections with a local anesthetic/steroid mixture.
- Those unresponsive would benefit from arthrocentesis with or without arthroscopy, arthroplasty with repositioning, or meniscectomy with or without graft/replacement, or modified condylotomy.
- Postoperative management – physical therapy/range of motion exercises.

Disorders of Hypomobility and Hypermobility

Hypomobility can be due to intra-articular factors or extra-articular factors (pseudoankylosis).

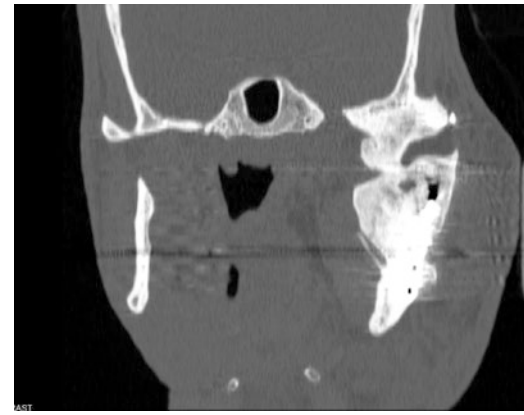

Fig. 2.2 Ankylotic mass extending from the medial aspect of the ramus to the mandibular fossa over a previously placed prosthetic temporomandibular joint. (Image courtesy of Dr. Damian Findlay)

Extra-Articular Causes

- Muscle fibrosis secondary to radiation, myofascial pain, tumors, infection, hysteric trismus, myositis ossificans.
- Fractures involving the condyle, zygomatic arch, or coronoid process.

True ankylosis (Fig. 2.2) – intra-articular fusion within the joint space resulting in hypomobility:

- Can be bony, fibrous, or fibro-osseous.
- Can be complete vs. incomplete.
- Can be caused by trauma, infection, otitis media, rheumatoid arthritis, psoriatic arthritis, prolonged immobilization, and previous TMJ or orthognathic surgery.
- Based on radiographic findings, two commonly accepted classifications have been adopted. Topazian based on three classes and Swahney has four classes (Table 2.2).

Workup for Ankylosis

- Clinical exam – decreased MIO, inability to appreciate translation of the condylar head.
- Orthopantogram – can see a radiodense mass, overall bony morphology, and coronoid hypertrophy.

Table 2.2 Sawhney and Topazian classifications of ankylosis

Classification of ankylosis [2]	
Sawhney (1986)	Topazian (1984)
Type 1 – flattened condylar head with close approximation to joint space Type 2 – flattened condyle close to glenoid fossa, bony fusion on outer aspect of articular surface. No fusion of the medial joint space Type 3 – bony block bridging the mandibular ramus and zygomatic arch Type 4 – wider bony block bridges the mandibular ramus and zygomatic arch, completely replacing the architecture of the joint	Stage 1 – only condyle involved Stage 2 – extends to sigmoid notch Stage 3 – entire condyle, sigmoid notch, and coronoid

- CT with contrast – defines the extent of the heterotopic bone/ankylotic mass. It also delineates the relationship of the mass to vital structures (foramen ovale, foramen spinosum, carotid canal, jugular foramen, pterygoid plexus). CT also aids in fabrication of a custom TMJ prosthesis in the setting of immediate reconstruction.

Treatment Options – requires excision of the mass with reconstruction. The goal of MIO is 35 mm and greater. In an adult, the reconstruction is more commonly achieved with a prosthetic joint, which is described later in text (other options include costochondral graft (CCG) or fibula free flap).

The Seven-Step KABAN Protocol [3]
Dr. Kaban described a protocol for the treatment of TMJ ankylosis in pediatric patients:

- Aggressive resection of the fibrous and/or bony ankylotic mass.
- Coronoidectomy on the affected side and measure MIO intraoperatively.
- Coronoidectomy on the contralateral side if you cannot achieve an MIO >35 mm and/or to the point of dislocation of the unaffected TMJ.
- Lining of the TMJ with a temporalis myofascial flap or the native disk (if salvageable).
- Reconstruction of the ramus condyle unit with either distraction osteogenesis (DO) (activate 2–4 days) or CCG and rigid fixation (10 days of MMF (Maxillary-Mandibular Fixation). If DO is used to reconstruct the ramus condyle unit, reshape the native bone narrowed and rounded. A corticotomy is then created distally to serve as transport disk. The distraction is set at 1 mm/day. Mobilization begins the day of the operation. In patients who undergo CCG reconstruction, mobilization begins after 10 days of MMF. DO takes advantage of the fibrocartilaginous cap that forms on the advancing front of the distracted bone heading toward the fossa.
- Early mobilization of the jaw.
- Aggressive physiotherapy.

Treatment Options for Fibrous Ankylosis
- Can be treated more conservatively.
  - Lysis of adhesions and fibrosis.
  - Diskectomy.

Postoperative Management
- Aggressive physical therapy is paramount in the treatment.
- Frequent follow-up.
- Consider radiation therapy (20 Gray in 10 fractions) to prevent recurrence and consider when using autogenous grafting, as the risk of recurrence is higher.

Costochondral Graft
- The CCG is commonly used in the growing child. It offers many advantages including ease of adaptation and remodeling, low morbidity at the harvest site, low rate of infections, and reduced relative cost. It does, however, increase operating time. In adults 12–17 cm of rib can be harvested and 7–10 cm in children within the borders of the lateral edge of the latissimus dorsi and costochondral junction.
- Ribs 4–7 may be harvested as they have a direct cartilaginous connection to the sternum. Rib 6 is the most commonly harvested as the incision falls in the inframammary crease creating a better cosmetic outcome (fusion of the rectus and pectoralis major forms an avascular plane.) It is common practice to harvest the right rib, as it is least likely to be confused with cardiogenic pain. Many advocate the rib

contralateral to the side of the defect to allow appropriate curvature of the harvested rib.

Rib Harvest Technique [4]

- A sharp incision is made in the inframammary crease (5 cm long).
- Dissection is carried through the subcutaneous tissue, fascia, and the plane between the pectoralis major and rectus abdominis.
- Two fingers are used to straddle the fifth and sixth intercostal space to prevent slipping of instruments. A sharp incision is cut through the periosteum down to the outer cortex of the rib.
- A molt periosteal now can be used to dissect in a subperiosteal plane around the rib. Some surgeons used the Doyen rib stripper, but its usage is known to be associated with parietal pleural tears.
- A sharp blade is used to make the cartilaginous incision. In children it is important to harvest no more than 3 cm (no less than 1 cm) to avoid overgrowth of the rib and to prevent separation of the cartilaginous cap.
- The rib is pulled laterally and a protected rib cutter is now used to section the length of desired rib.
- Check for pleural tears by filling the cavity with normal saline and have the anesthesiologist perform a Valsalva maneuver to check for bubble formation.
- The periosteal sleeve is now closed with 3-0 polyglactin (this may promote de novo regeneration of the missing rib in the child patient).
- The fascia between the rectus and pectoralis major is closed with a 3-0 resorbable suture, followed by subcutaneous tissue and finally skin.
- Post-operatively a chest X-ray is ordered to rule out a missed pneumothorax or hemothorax. The patient may return to normal activity post-op day 7, but any strenuous activity is withheld for 6 weeks.

Complications

- *Cartilaginous Cap Has Separated from Harvested Rib* – this is a highly debated question and the opinion of the authorities appears to be diverse. One approach is to drill a hole through the width of the rib and tie a non-resorbable suture to secure the cap. Another approach is to simply harvest the second rib above and start fresh. The rib directly above is preserved to prevent a cosmetic defect.
- *Pneumothorax* – occurs when air is trapped between the visceral and parietal pleural cavity. The condition develops when there is a one-way valve allowing air to enter and not escape. This condition can rapidly progress to respiratory insufficiency and cardiovascular collapse. Clinically the patients will have labored (tachypneic) breathing, chest pain, tachycardia, hyperresonance of chest wall on the affected side with diminished breath sounds. Late findings include cyanosis, distension of neck veins, tracheal deviation, and a decreased level of consciousness. Radiographically can appreciate tracheal deviation, loss of pleural lines, and loss of vascular markings (Fig. 2.3). Treatment firstly is 100% oxygen therapy to reduce the alveolar concentration of nitrogen, effectively increasing the difference in concentration of oxygen between tissue capillary and pneumothorax space, leading to rapid absorption by the surrounding vasculature. A pneumothorax 10% or less in size can be left to reabsorb and serial chest X-rays are indicated. If it does not resolve

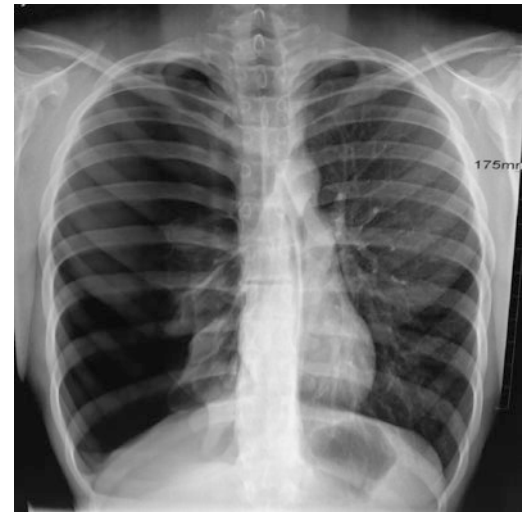

Fig. 2.3 Right sided pneumothorax. (Reprinted with permission from Fontaine and Page [5])

in 1 week, a tube thoracostomy is required. Estimation is provided by using a crude method by using a correlation that a 2.5-cm margin of gas peripheral to the collapsing lung corresponds to a pneumothorax of about 30%. Complete collapse of the lung is a 100% pneumothorax. If immediate pressure release is required, needle decompression can be done by placing an IV catheter at the second intercostal space along the mid-clavicular line and listen for rush of air. This procedure will normally buy time for tube thoracostomy. Tube thoracostomy requires a 2–3 cm incision that is marked at the fifth intercostal space just above the top of the sixth rib. Local anesthetic is infiltrated in the skin and tissues. A proximal end of a thoracotomy tub is clamped and advanced over the sixth rib, avoiding the neurovascular bundle on the inferior border of the fifth rib. The tube is placed on water-sealed suction drainage.

- *Pleural Tear* – air bubbles may be appreciated during the Valsalva maneuver indicative of a pleural tear. A suction catheter is placed into the wound and a purse string suture through the tear. The suction catheter is removed under suction while tightening the purse string simultaneously.

Hypermobility/Dislocation

Mandibular subluxation resulting in an inability to close from the patient's maximal incisal open position. This results in the condylar head being anterior to the articular eminence causing what is known as an open lock.

Etiologies

- Excessive yawning
- Excessive opening/prolonged opening (e.g., dental appointment)
- Seizure disorder
- Intubation
- Tardive dyskinesia
- Phenothiazine treatment – causes involuntary oromandibular movements
- Connective tissue disorders (e.g., Ehlers-Danlos and Marfan syndromes)

Acute Treatment

- Reduction by bimanual mandibular manipulation in a downward and posterior vector. Consider sedation beforehand.
- Wrap the head with a Barton bandage after reduction to limit jaw movements for a week (this allows stretched tissues to heal).

Treatment for Chronic Dislocation

- Noninvasive measures include intra-articular injections of a sclerosing agent such as alcohol or autogenous blood in the superior joint space.
- Botox has also been used in the lateral pterygoid.
- LeClerc/Dautrey procedures (zygomatic arch osteotomies), eminectomy, lengthening the articular eminence with a bone graft (calvarium, symphysis, ramus).

MRI Imaging of the Joint

- Ordering an MRI should be done with T1- and T2-weighted images in 3 mm serial cuts in the coronal, sagittal, and axial views for both open and closed mouths.
- A normal MRI will have the junction of the posterior band and the posterior attachment at the 12' O clock position in a closed mouth.

T1 Imaging (Fig. 2.4)

- Fat is bright and will appear white.
- Better for anatomy evaluation.
- The marrow fat in the condyle will have a high T1 signal intensity. An easy way to identify a T1-weighted image is if the condyle is white and the gyri of the brain do not show white banding or the orbits appear gray.
- On both T1- and T2-weighted image, the disk and cortical bone will appear black due to low proton density.
- Of note, in avascular necrosis, T1 marrow will be black and T2 will be bright due to necrosis.

T2 Imaging (Figs. 2.4 and 2.5a)

- Water is bright and fat is dark.
- Brain appears gray.

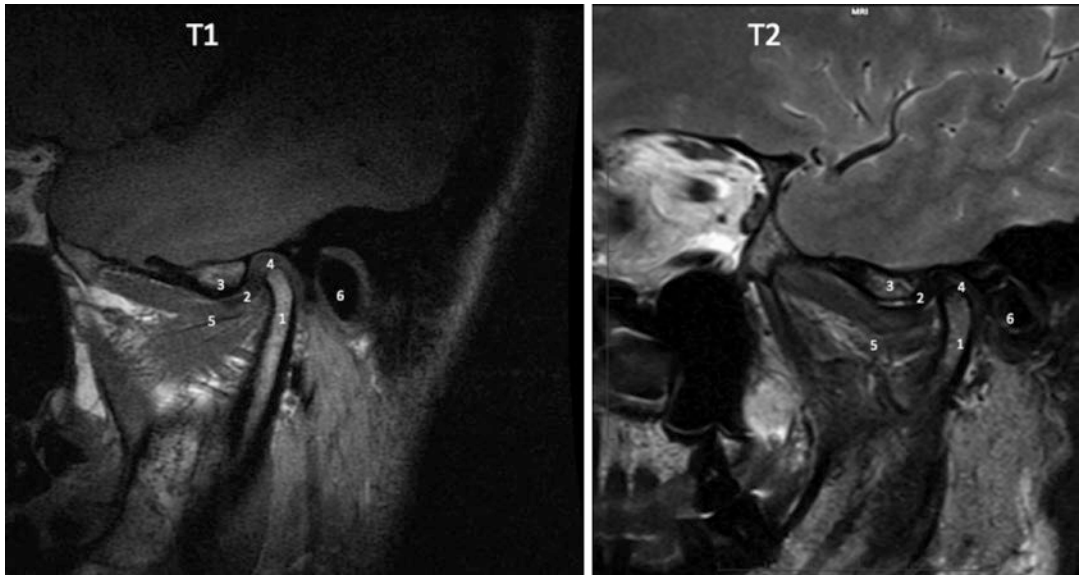

Fig. 2.4 (1) Condyle, (2) disk, (3) articular eminence, (4) posterior attachment, (5) lateral pterygoid, (6) auditory canal. (Image courtesy of Dr. Robert Reti)

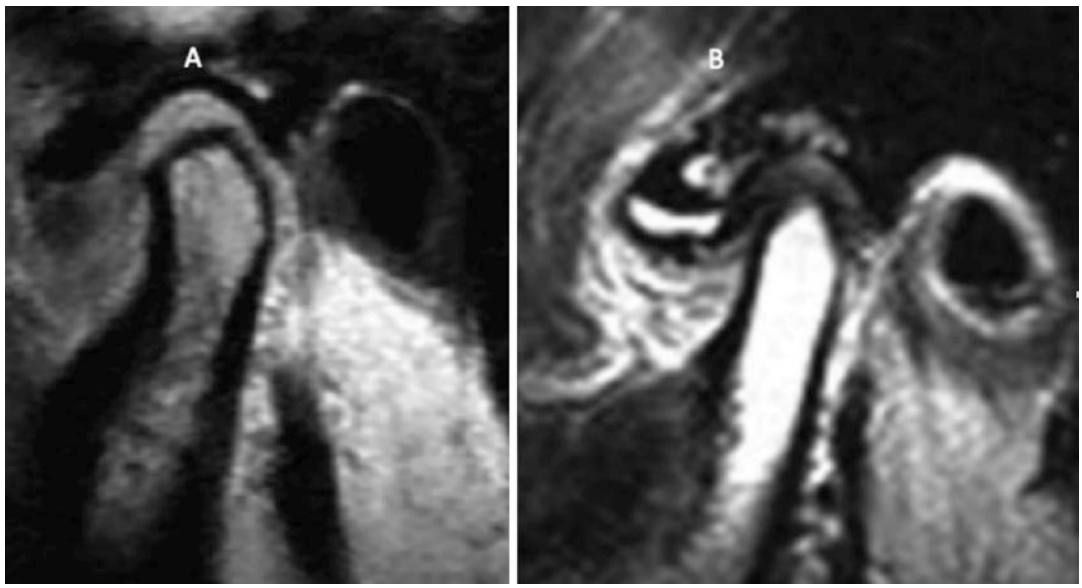

Fig. 2.5 (**a**) Example T1-weighted image sagittal view of TMJ in a closed mouth position with anterior disk position. (**b**) T2-weighted sagittal view of the TMJ in a closed mouth position with an effusion of the superior disk space. (Image courtesy of Dr. Robert Reti)

- Better to look for effusions and pathology (trauma/pathologies normally accompanied by edema) (Fig. 2.5b).
- The bone marrow is less bright (hence the condyle looks gray).
- Easy to identify whether you see a bright signal from gyri of the brain.

Approach to the Facial Pain/ Temporomandibular Joint Section

CC – Always ask the patient to expound on the chief complaint.

- *HPI* (HPI – Use the OLD CARTS acronym)
- *O*nset – when did the issue start? Was there a history of trauma?
- *L*ocation – where is the issue anatomically? For example, have the patient point to the region of discomfort.
- *D*uration – how long has the pain or decrease in opening been going on?
- *C*haracter – describe the character of the pain (throbbing, sharp, or dull).
- *A*ggravating/associated symptoms – is there anything that makes it worse? Do you have headaches, bruxism, clenching, gum chewing, nail biting, tinnitus, neck pain, or ear pain? Does your bed partner report any nocturnal bruxing? History of open or closed locks? Does your bite feel normal?
- *R*elieving – is there anything that makes the issue better?
- *T*iming – has this happened before? Any recent increase in life stressors? Does the pain improve during the day (nocturnal bruxism discomfort regularly improves during the day) or in the evening (arthritic joints tend to have more pain with continued function)?
- *S*everity – on a scale from 1 to 10, how severe are the symptoms? Is function impaired? Is there difficulty with mastication? Is there difficulty with speech due to pain? How has this affected your quality of life?

Physical Exam/Workup

- *Inspection* – look for gross asymmetry of the face (chin point and preauricular region). Look for signs of occlusal trauma such as wear facets, abfractive lesions, broken/worn restorations, exposed dentin, and missing teeth. Loss of posterior teeth can lead to MPD and overloading of the TMJ, which can cause intra-articular degenerative changes. Cheek bite marks or tongue scalloping. Look for premature contacts.
- *Palpate* – palpate the muscles of mastication (temporalis, masseter, pterygoids) and cervical musculature (SCM and trapezius). Palpate in the preauricular region to assess for masses and if movement of the condylar head can be appreciated. The condylar head may not move in cases of mechanical obstruction (i.e., ankylosis or neoplasm involving the TMJ).
- *Assess the Mandibular Gait* – check for maximal incisal opening (normal ~42–55 mm), lateral excursive (normal ~10–12 mm), and protrusive movements (normal ~8–11 mm). Look for deflection and deviation (possible signs of internal derangement) and the length of opening on occurrence.
- *Other Physical Exam Maneuvers*
  - Can you elicit Mahan's sign? Working side is loaded by placing tongue blades on the contralateral canines. If there is ipsilateral preauricular pain, then there is an internal derangement of the ipsilateral joint.
  - Joint auscultation to assess for clicks and crepitus.
- *Radiographic Imaging*
  - Orthopantogram – is it of diagnostic quality? Can both TMJs be visualized? Are the condyles in the fossae? Are there degenerative changes of the condylar head? Is there loss of joint spacing? Subcortical cysts? Chondromatosis (joint mice)? Subchondral eburnations (sclerosis)? Osteophytes? Are there radiodense changes or coronoid hypertrophy indicative of ankylosis? Is there condylar hyperplasia, hypoplasia, or agenesis? Are there any signs of hemiman-

dibular hypertrophy or condylar hyperplasia? Is there adequate penetration to visualize structures clearly? Look for fractures, third molars, caries, periodontitis, and sinus pathology.
- TMJ MRI – get T1- and T2-weighted (non-contrast) open and closed mouth views to assess disk position with function, disk integrity, and condition of the condyles. T1 gives better detail of joint anatomy. T2 useful for inflammatory changes and effusions. Look for the position of the disk and whether there is deformation. Remember in T1 fat is bright, T2 water is bright. Can look at the brain in T2 and note the brightness of the gyri and periorbital tissues.
- TMJ arthrogram – plain film of the TMJ that uses contrast. Good to visualize the position of the disk and good to assess for perforations.
- CT with contrast of the TMJs – look for ankylotic masses, neoplasms, mechanical obstruction, or infectious causes of trismus. Contrast helps to delineate the proximity of blood vessels to an ankylotic mass or if there is a collection that could be indicative of an infection (i.e., temporal space infection leading to trismus).

Arthrocentesis

Indications are for acute closed lock, previous surgery with continued discomfort, TMJ arthralgia, Wilke's classification 1, 2, and 3.

- Contraindications – ankylosis, overlying skin infection, and inability to appreciate the regional anatomy (i.e., obese patients).
- Can be done under local anesthesia or sedation.

Arthrocentesis Technique

- Use a marking pen to draw out the canthaltragal line (aka Holmlund-Hellsing line): First point is 10 mm ahead of the line and 2 mm below. Second point is 20 mm ahead (10 mm anterior to the first line) and 10 mm below. First point corresponds to the deepest point of the glenoid fossa and second point corresponds to the height of the articular eminence.
- Prepare skin with antiseptic solution.
- Use local anesthetic without epinephrine to anesthetize the area. This allows early evaluation if concern for traumatic versus anesthetic palsy of facial nerve. Additionally, if planning for diagnostic arthroscopy, epinephrine may mask erythema, rendering findings inaccurate.
- Manipulate the jaw to open the joint space.
- Insufflate superior disk space with a 27-gauge needle with lactated ringers.
- Using an 18-gauge needle, aim the needle at a 45-degree angle superiorly and anteriorly to reach the lateral aspect of the zygomatic arch, then walk the needle off the bone to enter the superior joint space. This will be your anterior port. (Joint entry with needle on average is 25 mm from skin.) Average superior joint space is around 3 cc.
- Place posterior port in similar manner with 18-gauge needle (of note a Shepard cannula can also be used which has an entry and exit port).
- Irrigate with lactated ringers (at least 100 ml). Lavaging the joint can break up adhesions, which can allow the disk to recapture into its premorbid position. This also irrigates out inflammatory mediators.
- Remove anterior port and inject a single agent or combination of steroid (Kenalog 40 mg/ml), hyaluronic acid (10 mg/ml), local anesthesia (bupivacaine 0.5% with 1:200 K epi), and morphine (10 mg/ml).
- Manipulate joint under anesthesia and check opening under sedation.
- Postoperative management includes aggressive range of motion exercises, NSAIDS, splints.

Disk Reposition Procedure

- Surgical procedure to manually reposition the disk into its premorbid position.
- Indications are failure of conservative therapy, Wilkes 2–5.

Preauricular Approach/Disk Reposition Technique

- Incision is marked in the preauricular crease (may consider the Al-Kayat extension to increase access).
- The incision is made through the skin and subcutaneous tissues for the entire length.
- Attention is then directed to the superior portion of the incision. Dissect through temporoparietal fascia (TP) and auricularis anterior muscle down to the temporalis fascia layer (which is recognized by the glistening white color). The TP fascia is attenuated in this region and not as thick as its superior counterpart (Galea). Remember the temporal branch of the facial nerve runs within the TP fascia anywhere from 8 mm to 35 mm (average 20 mm) from the bony anterior extent of the external auditory meatus. The remaining intervening tissues are dissected down to the level of the temporalis fascia using a nerve monitor/stimulator to avoid the course of the nerve (Fig. 2.6).
- Palpate the zygomatic arch. Incise through the attached periosteum. Dissect subperiosteally until you appreciate the joint capsule.
- Insufflate the joint with local anesthesia or saline. Make an incision into the joint capsule to enter superior joint space.
- Mobilize the disk. Assess for perforations (repair if small perforations/remove disk if the perforation is large). Disk can be plicated in a posterolateral vector to the disk capsule or temporalis fascia with non-resorbable suture or a Mitek® anchor.
- Close in layers.

Complications

Otitis Externa – Infection of external auditory canal. Patient will complain of pain on movement or pressure of ear. Otoscopy will reveal edematous EAC with possible discharge. Treatment includes topical fluoroquinolone otic products (to cover pseudomonas – most common bacteria implicated in otitis externa).

Otitis Media – inflammation of the middle ear structures. Patient will complain of ear pain, difficulty hearing, and fever. Otoscopy shows full or bulging tympanic membrane or possible purulence (if there is perforation of tympanic membrane). Treatment includes antibiotics such as amoxicillin. Consider consult with ENT for myringotomy tubes.

Broken Instrument During Arthroscopy/Arthrocentesis – if you are able to visualize the fragment, and having arthroscopic training, attempt removal arthroscopically. If you cannot visualize, then obtain radiographs in multiple planes for identification. If these failed or not trained, convert to an open approach.

TMJ Rupture/Hemotympanum – otoscopy to examine for TM rupture or hemotympanum. Consult ENT for intra-op examination if this is

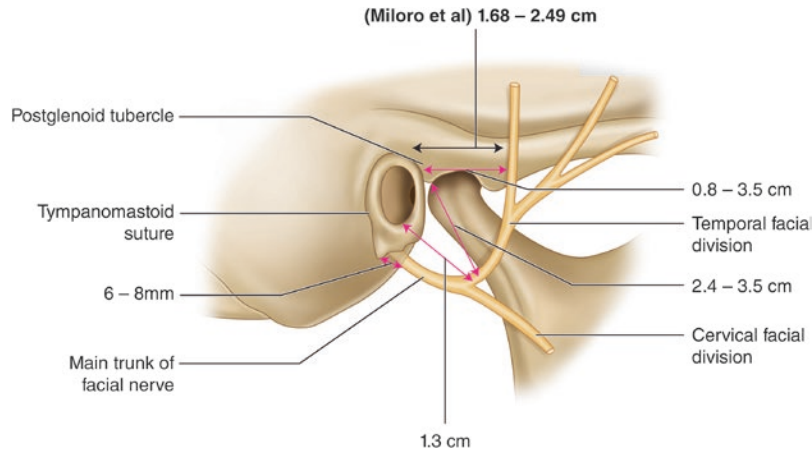

Fig. 2.6 Facial nerve as measured from the lowest point of the external bony auditory canal. Bifurcation of facial nerve: 1.5–2.8 cm. Facial nerve crossing zygomatic arch: 0.8–3.5 cm (average 20 mm). (Reprinted with permission from Miloro M, Redinger S, Peddington D, Kolodge T. JOMS 2007; 65(12):2466–2469)

noted. If EAC is damaged, place an antibiotic impregnated sponge dressing. This is sutured to maintain opening of the EAC, thereby, preventing stenosis. Some physicians will place on antibiotic-hydrocortisone suspension for 14 days post-op and monitor for granulation tissue formation. This granulation tissue can be removed with bipolar cautery or silver nitrate.

Violation of the Middle Cranial Fossa – keep in mind that the fossa is approximately only 0.9 mm thick. If a large perforation is noted, an intraoperative neurosurgical consult is recommended (as it may be able to be treated immediately). If you suspect CSF leak postoperatively, then obtain a CT scan/MRI. A tracer study should also be taken. (Neurosurgery should be consulted and should advise on the desired imaging.) The patient is placed on bed rest, with the head of the bed raised greater than 30 degrees. Some advocate administration of antibiotics such as cotrimoxazole as this is bactericidal and enters CSF. The overwhelming majority of small leaks spontaneously heal within 1 week.

Damage to the Temporal Branch of the Facial Nerve – the temporal branch of the facial nerve on average is 2 cm anterior to the bony external auditory canal. The classic study of Al-Kyat and Bramley identified a range of 0.8 cm to 3.5 cm [6]. The temporal branch of the facial nerve innervates the frontalis, orbicularis, and corrugator supercilii. Most injuries resolve in 3–6 months and, therefore, observation is warranted. Treatment should be reserved for those who are symptomatic. Ophthalmologic consult is indicated. Lubrication and taping of the eye at night are necessary to prevent keratoconjunctivitis. Physical therapy with electrical stimulation may aid in maintaining muscle tone while awaiting recovery. Gold weights implants can be placed in the upper eyelid for more permanent defects.

Auriculotemporal Nerve Syndrome (Frey Syndrome) – signs and symptoms include gustatory sweating, flushing, and warmth over the temporal and preauricular areas. It results when there is auriculotemporal nerve damage and occurs most commonly with arthroscopy. It usually is temporary and will resolve within 6 months.

**Author's Note*. The patient is to be evaluated with the Minor test (starch-iodine). A solution of 3 g iodine, 20 g castor oil, and 200 ml absolute alcohol is applied to both preauricular regions of the face. Gustatory sweating is elicited by having the patient chew on a lemon drop. A positive test is conversion of the yellow mixture to a dark blue. Case reports have shown that 16–80 IU of botulinum A subcutaneous injection has resulted in resolution within 1 week. Other treatments are application of scopolamine ointment (anticholinergic properties) and surgical transection of the innervation.

Bleeding During Condylotomy – during the condylotomy, the concern for bleeding is from the internal maxillary artery (IMA) and its branches. The IMA runs 3 mm medial from the mid-sigmoid notch and 20 mm below the condylar head. A commonly damaged vessel, as the cut is made through the sigmoid notch, is the masseteric artery.

Bleeding During Diskectomy – many times during a diskectomy, the bleeding may be also originating from the retrodiscal tissues or the lateral pterygoid muscle. The most commonly damaged vascular structure is the middle meningeal artery. It is found on average 31 mm medial the zygomatic arch and an average of 2.4 mm anterior from the height of the glenoid fossa. The first step to managing bleeding is to establish visualization. Attempt to identify any vessels for cauterization or ligation. If no obvious source, then apply firm pressure with a moistened gauze packed tightly into the wound. Additional hemostatic measures include thrombin-soaked gauze, flowable hemostatic agents, collagen sponges, or tissue adhesives. The inferior border of the mandible is then displaced superiorly to aid in pressure hemostasis (holding pressure for at least 5 minutes). Interventional radiology for embolization is warranted immediately if bleeding is not controlled by local hemostatic measures.

**Author's Note.* Some surgeons advocate carotid artery cut down for uncontrollable bleeding. Some question its efficacy due to contralateral circulation. In this approach, the neck incision is extended (a horizontal incision 5 cm in length) 2 cm below the inferior border of the

mandible, over the sternocleidomastoid muscle (SCM). The SCM is retracted posteriorly, and with blunt dissection parallel to the vessels, the carotid sheath should be identified. The SCM is carefully dissected from the sheath and the sheath is carefully entered. The internal jugular vein should be retracted posteriorly to reveal the common carotid. Dissection to the bifurcation aids in identification. The hypoglossal nerve will cross the arteries above this bifurcation and should be identified to prevent damage. Ligation should be above the facial branch, third of the anterior branches. Blood flow has been found to be reduced by 73%, when ligated at this position [7].

Total TMJ Joint Replacement

Total joint replacement (TJR) indications:

- Failed previous TMD surgeries
- Severe arthritic joint
- Loss of vertical mandibular height and occlusal relationship
- Pathology
- Ankylosis – either bony or fibrotic
- Condylar agenesis

Two Approved TJR Prosthetics in the USA

1. Biomet®
   - Stocked with multiple sizes
   - Chromium cobalt alloy for condylar component and ultra-high molecular weight polyethylene for fossa component.
   - Pseudotranslation possible (if unilateral placement due to push of contralateral TMJ).
   - Chromium cobalt mandibular prosthesis is offered in three sizes (45 mm, 50 mm, and 55 mm) and in three styles (standard, narrow, and offset).
   - Chromium cobalt may contain nickel (a consideration in those with a nickel allergy).
2. TMJ Concepts®
   - Custom made w/CT scan and stereolithography.
   - Pure titanium for condylar component.
   - Pure titanium with ultra-high molecular weight polyethylene for the fossa component.

Surgical Technique for TJR

- Requires a preauricular and submandibular/retromandibular approach.

Preauricular Approach

- Standard preauricular approach to joint capsule (see above).
- Make an incision in the periosteum of the lateral aspect of the condylar head, in a T shape fashion, to expose the lateral aspect of the condyle. Of note, the anatomy may be distorted due to an ankylotic mass and, therefore, recognizable anatomical landmarks should be used as a reference for the dissection.
- Dissect subperiosteally to expose the anterior and posterior regions of the condylar neck.
- Pack site and direct attention to the submandibular region.

Submandibular Approach

- Mark mandible 2 cm below inferior aspect of the mandible.
- Inject vasoconstrictor.
- Make an incision approximately 6 cm long.
- Dissect through skin and subcutaneous tissue to the level of the platysma.
- Undermine skin flap in all directions.
- Sharp dissection through platysma exposing superficial layer of the deep cervical fascia. Dissect through this layer with the aid of nerve stimulator/monitor testing for marginal mandibular nerve, which is within or deep to the fascia.
- (Don't forget Dingman and Grabb [8] study – 19% of the time, the marginal mandibular nerve passed below the inferior border of mandible until it crossed facial artery 1 cm below the inferior border of the mandible).
- Marginal mandibular nerve has two branches 61% of the time and 21% it is a single branch.
- Dissect out facial artery and vein; isolate and clamp and tie vessels.
  - Hayes-Martin maneuver – ligation of facial vein (posterior to facial artery) at the lower

border of the mandible aiding in reflection of the superficial layer of the deep cervical fascia preserving the marginal mandibular nerve.
- Divide the pterygomasseteric sling along the inferior border of the mandible (the most avascular portion of sling). Redirect attention to the preauricular region.

Condylar Resection (Condylectomy)
- Condyle retractors placed to isolate the neck of the condyle (may not be possible in large ankylotic masses).
- Resect exposed condyle (a minimum of 15 mm of clearance for condyle and fossa component) if additional condyle neck requires removal, may place bone clamp on inferior border and displace ramus superiorly, further exposing condyle neck into preauricular/endaural incision.
- Inadequate removal may lead to impingement of ramus remnant on fossa prosthesis when MMF placed.

Fossa Preparation
- Removal all soft tissues from tympanic plate to remnant articular eminence.
- TMJ Concepts® – if necessary, reproduce any fossa contouring noted on preoperative model. TMJ concepts will require verification of seating by using the fossa-seating tool.
- BIOMET® stock joint requires manipulation of a specially designed diamond rasp or burr to modify the articular eminence. This allows positioning of fossa component. The surgeon must choose the appropriate fossa from sizers to ensure tripod stability. Note, the articulating surface of the fossa component stays constant and the amount of screw hole positions over the arch increases with size.
- Secure fossa component (make sure to apply firm pressure with fossa seating tool from TMJ concepts).
- Place only two screws for securing the prosthesis to allow check for the range of motion/interferences and to avoid damage to bone stock if repositioning is required.

Condyle Component
- Place patient in MMF.
- Biomet®, choose correct mandibular component from the sizers.
- Contour bone of the lateral ramus (rarely needed with TMJ concepts due to it having a custom fit) to allow passive fit of BIOMET sizer.
- Secure with two screws at this time.

Final Screw Securement
- Cover open wound sites, enter oral cavity, and remove MMF. (Consider paralysis at this time for freedom of movement).
- Ensure ROM is at least 32–35 mm.
  - If cannot achieve ROM, perform coronoidectomy (first ipsilateral and if not bilateral).
  - Note: If this is an ankylotic case, a coronoidectomy is required per Kaban protocol.
- Place final screws, at least four screws for fossa component and six in ramus.
- Irrigate sites and close.
- Consider fat graft around fossa to prevent ankylosis.

Post-op
- Post-op radiographs to confirm position and alignment.
- Post-op exercises and soft diet.
- Consider physical therapy for 4–6 weeks.

TMJ Case

**Authors Note.* There are many ways to treat TMD. The goal is to explain the way you will be treating this patient. Do not deviate from your algorithm or explain how other surgeons would approach this. The goal of this portion is to guide you down a path to a surgical procedure. Be prepared to talk about open joint procedures, which could include disk manipulation or total joint replacement.

A 35-year-old female presents to your office with a chief complaint of diffuse, increasing left

side facial pain for 2 months. She reports morning headaches occasionally.

Medical History

- PMHx – asthma
- Rx – albuterol
- Allergy – NKDA
- PSurgHx – appendectomy
- PSocHx – attorney by profession, alcohol socially, denies tobacco and recreational drug use.
- *What do you want to know?*
  HPI (OLD CARTS) (*note above).
- *She has noticed that the pain started 3 months ago when she made partner at her law firm. She reports increased stress due to her promotion. She wakes up with morning headaches and her husband says that she grinds her teeth when she sleeps. What is your next step?*
  I would perform a physical exam – inspect, palpate, auscultate. Evaluate for facial asymmetries. Palpate muscles of mastication, denoting any pain. Palpate and auscultate TMJ for joint noises and crepitus. Evaluate maximal incisal opening and freedom of movement. I would note any irregular jaw movements and at what length of opening. Exam dentition for signs of attrition and compensatory hyperuption, overall dental condition, and premature contacts, and look for morsicatio buccarum and linguarum. I would try to elicit a Mahan sign.

Pertinent findings on clinical exam – Maximal incisal opening of 36 mm. No deflection or deviation noted. Anterior wear facets noted. Tenderness to palpation of the left masseter and temporalis muscles was appreciated. Prominent morsicatio buccarum (cheek biting) was also appreciated.

- *What imaging would you order?*
  I would order an orthopantogram.
- *Describe what you see on the orthopantogram* (Fig. 2.7).
  This is a diagnostic orthopantogram. Impacted third molars are present. Slight degenerative changes of both condylar heads noted. Even spacing of the TMJs noted. Condyloramal units appear congruent. No subcortical cysts noted. A full complement of teeth is appreciated. No other gross dental or bony pathology appreciated.
- *What is your diagnosis?*
  Myofascial pain disorder/early DJD.
- *How would you treat this patient?*
  Conservative therapy/non-surgical therapy would be the first-line treatment. This would include warm moist heat, NSAIDS/anti-inflammatories, soft diet, and an orthotic dental splint. A trial of low-dose anxiolytics may also be appropriate with this patient if the source of her parafunctional habits is stress related (e.g., alprazolam 0.25mg q8 hours). This also may warrant consultation with a PCP/psychologist for long-term care.

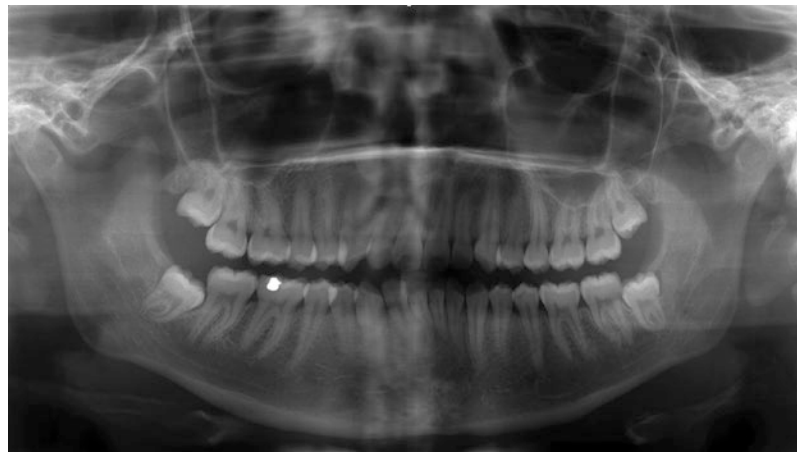

Fig. 2.7 Orthopantogram of TMJ case. (Image courtesy of Dr. Damian Findlay)

- *The patient returns 8 months later with complaint of a frequent clicking sound whenever she opens her jaw and now has limitation of opening. How do you proceed?*
  Review any recent events associated with recent changes in symptoms: trauma, dental work, changes in medications, etc. Conduct a new complete head and neck exam. Evaluate new maximal incisal opening, deviations/deflections, symmetry in lateral excursive movements, new myositis, palpable/audible clicks.
- *New pertinent findings* – maximal incisal opening of 26 mm with a left sided click. *Would you order any imaging?*
  An MRI would be beneficial at this point to examine the disk position and condition.
- *Does this patient have any contraindications for taking an MRI*?
  No. MRIs should not be taken with patients with implanted ferromagnetic metals. These include AICD and clips to treat aneurysms. Dental materials such as braces, dental implants, cobalt chromium prostheses, and amalgam fillings are not contraindications.
- *What do you see on this MRI* (Fig. 2.8)*?*
  This is a T1 image sagittal slice closed mouth image. The MRI shows the disk to be anteriorly displaced. The disk shows only mild deformity.
- *How do you proceed?*
  I would discuss the different treatment options for the patient including arthrocentesis and arthroscopy.

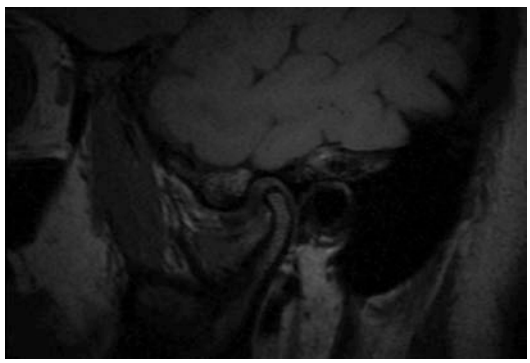

Fig. 2.8 MRI of TMJ case

- *You decided with the patient you would like to continue treatment with arthrocentesis. Can you describe the procedure?*
  See arthrocentesis procedure in text.
  Board Guidance Point – You may also consider arthroscopy for diagnosis purposes. Arthroscopic surgery of the TMJ is a subspecialty that few surgeons do. Unless you are trained on this procedure, it would be best not to mention it as your option of choice to treat the patient.
- *What fluid do you use? How much?*
  Lactated ringers.
  – 100 mL: probable minimum for therapeutic lavage
  – 200 mL: reduces protein, bradykinin, IL-6
  – 300–400 mL: no detectable protein, bradykinin

  Author's Note. Lactated ringers is a crystalloid solution containing calcium chloride, potassium chloride, and sodium lactate. It is close to human serum in makeup; therefore, it is best tolerated by the tissues.
- *What do you inject into the joint after?*
  One regiment is 2 cc mixture of 10 mg/ml hyaluronic acid and 10 mg/ml of morphine. (Some surgeons in place of HA will use a 2 cc mixture of Kenalog® 40 mg/ml and bupivacaine 0.5%).
  **Author's Note.* Recall the joint space is 2–3 cc. HA mimics the glycosaminoglycan made by synovial cells. It is a viscous, high molecular weight substance that lubricates and protects joint articulating surfaces by preventing phospholipid destruction. It has been found to be effective with degenerative joint disease and anterior disk displacement with and without reduction and provides prolonged pain relief.
- *You are seeing the patient for her 3-month follow-up. She reports that she has been compliant with her post-op physical therapy and soft diet. She reports a tremendous amount of pain and a decreased range of motion. Exam reveals severe left-sided preauricular tenderness with a maximal incisal opening of 20 mm. After reexamining the patient, you discuss the option of an arthrotomy* versus *a condylotomy.*

What are the options to manage a malposed disk during arthrotomy?
Once the disk is exposed, you may plicate the disk in a posterior lateral vector. The disk can be plicated to the temporalis fascia using a non-resorbable suture. The other option is a mini Mitek anchor placed into the most posterior, superior, lateral condylar neck. You may repair a disk perforation if it is less than 3 mm. Diskectomy should be considered if the disk appears fragmented, if the perforation is large, or if the disk is balled up.
**Author's Note.* Mitek anchors are cylindrical pins with wings measuring 1.8 mm in diameter and 5 mm in length. Body is a titanium alloy (90% titanium, 6% aluminum, and 4% vanadium) and the wings are nickel-titanium alloy (Nitinol). There are two strands of single O suture attached to any eyelet to allow anchoring of the disk.

- *The patient opts for a condylotomy. What is the rationale for it use?*
  The goal is to increase the joint space and unload the disk to relieve pain by allowing some condylar sag. It is accessed like an IVRO with medial pterygoid muscle release to allow for condylar sag. The posterior cut is made 6–8 mm from the posterior border and 10 mm from the sigmoid notch. The amount of sag is assessed by looking at the inferior border and the tip of the proximal segment. The goal is to achieve 3–4 mm of sag.
- *What is your post-op protocol for a condylotomy?*
  - Post-op orthopantogram
  - 7 days of IMF (unilateral) for 14–21 days (bilateral)
  - Elastics and physical therapy
  - Remove arch bars off in 7 weeks
- *Patient is lost to follow-up and returns 10 years later with inability to open. You get a CT and you see this* (Fig. 2.9). *What would you expect to see on exam?*
  Painful muscles of mastication as they continually attempt to open the ankylotic joint. With this you might also see masseteric hypertrophy. A firm preauricular swelling from the mass may be appreciated. On orthopantogram, you may see prominent antegonial notching and coronoid hypertrophy.
- *What imaging would you like?*
  A medical grade-contrast enhanced CT of the maxillofacial region. This scan will aid in identifying the extent of the mass and in identifying vascular structures like the external carotid branches. It can also be used if a custom CAD-CAM generated joint is part of the treatment plan.
- *What are the different types of ankylosis?*
  False ankylosis – limited movement of joint due to extra-articular fibrosis or pathological condition.
  True ankylosis – fibrous or bony fusion or intra-articular joint structures.
  Two Subtypes of True Ankylosis:
  - Fibrous ankylosis – fibrous adhesion between condyle and fossa.
  - Bony ankylosis – formed by a bony mass between articular surface and the condyle.
- *How would you treat this case?*
  I would offer resection of the ankylotic mass with reconstruction, using a TMJ prosthesis.
  Author's Note. Other options for reconstruction include a costochondral graft or fibula-free flap.

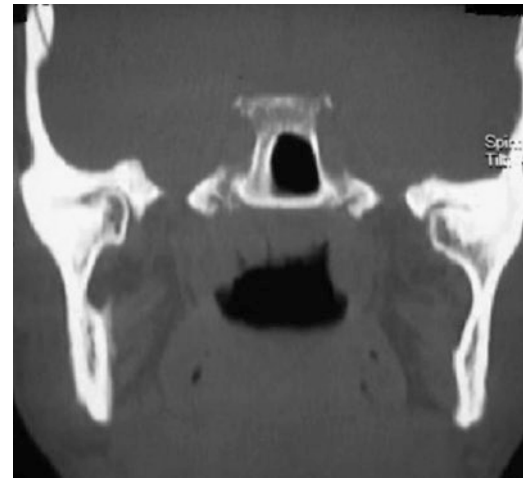

Fig. 2.9 Bilateral hyperdense masses noted in the condylar regions consistent with ankylosis. (Image courtesy of Dr. Damian Findlay)

- *What are indications for total joint replacement?*
  - Failed previous surgeries
  - Severe arthritic joint
  - Loss of vertical mandibular height and occlusal relationship
  - Pathology
  - Ankylosis – either bony or fibrotic
  - Condylar agenesis
- *What are the two approved TMJ TJR options in North America?*
  Biomet® and TMJ Concepts®
- *Describe briefly how to place a TMJ concepts or BIOMET stock joint?*
  See above surgical approach section.
- *You have completed a total joint replacement. On post-op day 2, you decide to round on the patient. The patient opens wide and now is stuck open. What are you concerned about and what do you do?*
  The concern is that the condylar head has slipped out of the fossa. This requires immediate return to the OR for manipulation. If unsuccessful, the incisions may need to be reopened to reposition the condylar component.
- *Patient after total joint replacement returns with a red and swollen preauricular region. What do you do?*
  Examine external auditory canal for concern of perforation. If a superficial infection is suspected, remove some sutures to establish drainage, obtain cultures, and prescribe a short course of oral antibiotics. If no improvement or recurrence, get a CT image with contrast to rule out deeper infection (a return to OR for aggressive debridement and/or removal of components may be indicated).
  **Author's Note.* In the event of an acute infection (5 days or less), open incisions, debride wound, and get a tissue sample for culture. The wound should be irrigated with antibiotic irrigation. The wound should be irrigated on a q 4 hour basis. Consider Infectious Disease consult for IV antibiotics via PICC line. For a late-stage infection (>1 month), remove hardware and debride surrounding soft tissue. Place an antibiotic-impregnated orthopedic spacer/tobramycin spacer to fight infection and prevent tissue in growth. An Infectious Disease consult is indicated (patient may require PICC line with 6 weeks of antibiotic infusion). Polyethylene components get discarded and all pure metal components can be cleaned and autoclaved. All screws get replaced. Of note, not all patients may require reconstruction if the occlusion, function, and range of motion are acceptable. Consider elastic training and physical therapy if the patient does not wish to undergo another alloplastic joint replacement.
- *How can you reduce the incidence of heterotopic bone formation?*
  Most cases occur 2–3 months after surgery. Most important it is to regain range of motion as soon as possible. Total radiation dose of 10 Gy to 20 Gy has been reported via fractionated daily doses in the immediate post-op phase. Wolford reported use of fat graft around the joint to decrease heterotopic bone formation.

  Indomethacin, a non-selective COX inhibitor, has been compared to radiation treatment in prevention of heterotopic bone formation in hip arthroplasty and found to be equally effective. It is given in 75 mg doses for 6 weeks (along with pantoprazole).

Mnemonic for External Carotid Branches

Some Anatomists Like Freaking Out Poor Maxillofacial Surgeons

- S: superior thyroid artery
- A: ascending pharyngeal artery
- L: lingual artery
- F: facial artery
- O: occipital artery
- P: posterior auricular artery
- M: maxillary artery
- S: superficial temporal artery

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3 Management of Maxillofacial Infections

Thomas R. Flynn, William Gilmore, Robert Reti, Trina Sengupta, and Damian Findlay

Odontogenic Infections

- Treatment of odontogenic infections is based on medical management, surgical treatment, and antibiotic therapy.
- Odontogenic infections are polymicrobial in nature and are normally composed of more anaerobic bacteria.
- Results from:
  - Dental caries
  - Dentoalveolar infections (infections of the pulp and periapical abscesses)
  - Gingivitis
  - Periodontitis
  - Perimplantitis
  - Pericoronitis

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- Factors that determine the spread include thickness of the cortical plate and the relationship of the adjacent muscle attachment with the apices of the offending teeth.
- Infections spread via hydrostatic pressure along the path of least resistance. These paths tend to be composed of loose connective areolar tissue that is enclosed by fascial layers.
- The cervical fascia is a fibrous connective tissue that envelops and divides the muscles of the neck and creates potential spaces.
- There is a synergistic interdependence between aerobic and anaerobic bacteria thought to be necessary for the development of an abscess.
- Respiration of aerobic bacteria -> depletes the local environment of oxygen -> creates an oxygen-poor and nutrient-rich habitat -> anaerobic bacteria growth increases -> anaerobes proliferate and secrete toxins and enzymes which results in tissue destruction.

Path of Third Molar Infection to Mediastinum

- Periapical abscess erodes through thinnest cortical plate (lingual) into the submandibular space.
- As the submandibular space is filled, the infection travels through the buccopharyngeal gap between the middle and superior pharyngeal constrictors to the lateral pharyngeal space.

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A direct connection to the lateral pharyngeal space is via spread directly around the posterior belly of the digastric muscle.
- There is no barrier between lateral pharyngeal space and retropharyngeal space.
- Retropharyngeal space fuses with alar fascia between C6 and T4 (Figs. 3.1 and 3.2).
- The infection normally enters danger space at the fusion of alar and prevertebral fascia.
- Danger space is continuous with posterior mediastinum.

Principles of Management of Odontogenic Infections

- Determine severity: anatomic location, rate of progression, and airway compromise.
- Evaluate host factors: evaluate immunocompetence and systemic reserve of the patient.
- Decide on setting: inpatient criteria – fever, dehydration, need for general anesthesia, deep space infection, or control of systemic disease.
- Treat surgically.
- Support medically.
- Choose and administer the appropriate antibiotic.

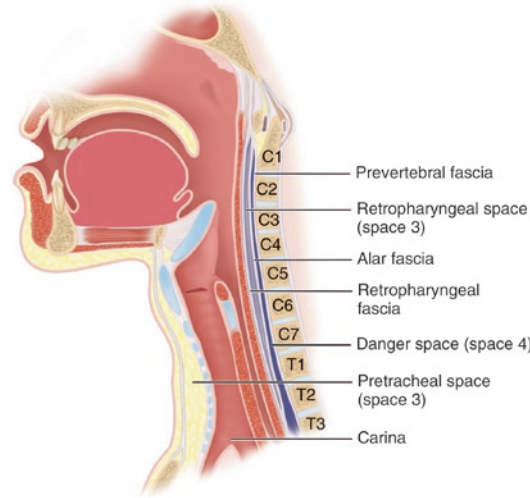

Fig. 3.2 Sagittal section of the neck demonstrating fascial spaces and danger spaces. (Reprinted with permission from Flynn [1])

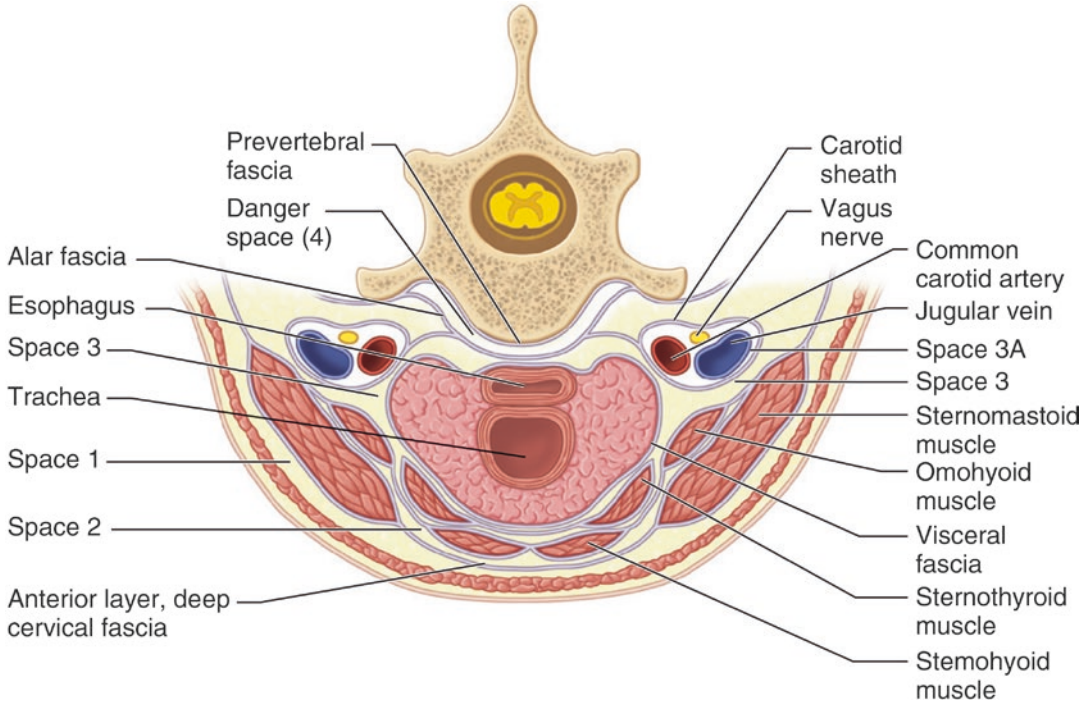

Fig. 3.1 Axial view demonstrating the fascial cervical spaces at the level of the sixth cervical vertebrae. (Reprinted with permission from Flynn [1])

Table 3.1 Characteristics of cellulitis vs. abscess [2]

Cellulitis vs. abscess		
	Cellulitis	Abscess
Duration	3–5 days	5–7 days
Palpation	Hard and very tender	Fluctuant and tender
Skin quality	Thick	Thin, shiny
Bacteria	Mixed	Anaerobic
Tissue fluid	Serosanguineous	Purulent
Size	Diffuse	Localizing

- Evaluate the patient frequently.
- Odontogenic infections pass through three stages: inoculation, cellulitis, and abscess (Table 3.1).
- Cellulitis is more severe than abscess as cellulitis continues to spread.
- Abscess formation is the beginning of localization.

Primary Fascial Spaces (See Table 3.2)
Primary fascial spaces are those that are directly adjacent to the origin of odontogenic infections. Infection spread by direct invasion from the source. These spaces include the following:

- Buccal
- Submandibular
- Canine
- Submental
- Vestibular
- Sublingual

Secondary Fascial Spaces (See Table 3.2)
Secondary Fascial space infections are those that become involved via spread of infection from the primary fascial spaces. These spaces include the following:

- Pterygomandibular
- Infratemporal
- Masseteric
- Masticator (see Fig. 3.3)
- Lateral pharyngeal (see Fig. 3.4)
- Retropharyngeal (see Fig. 3.5)
- Prevertebral

Workup for the Odontogenic Infection Patient

History of Present Illness

- Need to determine onset, duration, symptoms of infection, and any antibiotics previously prescribed.
- NPO (Nil Per Os (nothing by mouth)) status.
- Assessment of concerning signs:
  - Dysphagia – difficulty swallowing
  - Dysphonia – difficulty speaking
  - Dyspnea – difficulty breathing
  - Odynophagia – pain on swallowing
  - Mental status changes
  - Trismus
  - Fevers/chills

Past Medical History

- Important to assess if there are any disease processes that render the patient immunocompromised—e.g., HIV, DM, hepatitis, alcoholism, malignancy, chemotherapy, malnutrition, patients on steroids, or immunosuppressants
- IV drug users have a higher incidence of MRSA infection

Physical Exam

- *Vital Signs*
  - Temperature – elevated temperatures can be indicative of serious infection with systemic involvement. Normal oral adult temperature is on average 98.6 °F/37 °C. Rectal temperature tends to be 1 °F higher and axillary is 1–3 °F lower.
  - Heart rate – tachycardia can be indicative of systemic involvement. Each degree increase in °F tends to correlate with an increase of 10 BPM of heart rate.
  - Respiratory rate (normal 12–20 breaths/min) – elevated rate could suggest respiratory compromise or acid-base imbalance suggestive of SIRS.
  - Blood pressure – hypertension can be present secondary to pain. Hypotension can be seen in septic patients.

Table 3.2 Borders of the deep fascial spaces of the head and neck

Space	Anterior	Posterior	Superior	Inferior	Superficial or Medial*	Deep or Lateral*
Buccal	Corner of mouth	Masseter muscle Pterygomandibular space	Maxilla Infraorbital space	Mandible	Subcutaneous tissue and skin	Buccinator muscle
Infraorbital	Nasal cartilages	Buccal space	Quauratus labii superioris muscle	Oral mucosa	Quadratuc labii superiors muscle	Levator anguli oris muscle Maxilla
Submandibular	Anterior belly digastric muscle	Posterior belly digastric muscle Stylohyoid muscle Stylopharyngeus muscle	Inferior and medial surfaces of mandible	Digastric tendon	Platysma muscle Investing fascia	Mylohyoid muscle Hyoglossus muscle Superior constrictor muscles
Submental	Inferior border of mandible	Hyoid bone	Mylohyoid muscle	Investing fascia	Investing fascia	Anterior bellies of digastric muscles*
Sublingual	Lingual surface of mandible	Submandibular space	Oral mucosa	Mylohyoid muscle	Muscles of tongue*	Lingual surface of mandible*
Pterygomandibular	Buccal space	Parotid gland	Lateral pterygoid muscle	Inferior border of mandible	Medial pterygoid muscle*	Ascending ramus of mandible*
Submasseteric	Bucall space	Parotid gland	Zygomatic arch	Inferior border of mandible	Ascending ramus of mandible*	Masseter muuscle*
Lateral pharyngeal	Superior and middle pharyngeal constrictor muscles	Caroid sheath and scalene fascia	Skull base	Hyoid bone	Pharyngeal constrictors and retropharyngeal Space*	Medial pterygoid muscle*
Retropharyngeal	Superior and middle pharyngeal constrictor muscles	Alar fascia	Skull base	Fusion of alar and prevertebral fasciae at C6-T4		Carotid sheath and lateral pharyngeal space*
Pretracheal	Sternothyroid-thyrohyoid fascia	Retropharyngeal space	Thyroid cartilage	Superior mediastinum	Sternothyroid-thyrohyoid fascia	Visceral fascia over trachea and thyroid gland

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*Medial border

*Lateral border

  - Oxygen saturation – patients unable to maintain an oxygen saturation greater than 96 on room air may have airway compromise (if no underlying pulmonary disease).
- *Inspection (global view of the patient)*
  - Look for facial/cervical swelling and asymmetry.
  - Assess whether or not the patient has a toxic appearance such as pallor, sweat, (diaphoretic), shivering, lethargy, etc.
  - Assess whether the patient can tolerate their secretions (concern for airway swelling). Are they sitting in a tripod position to allow collection of saliva, open a constricted airway, and prevent dirtying clothes?

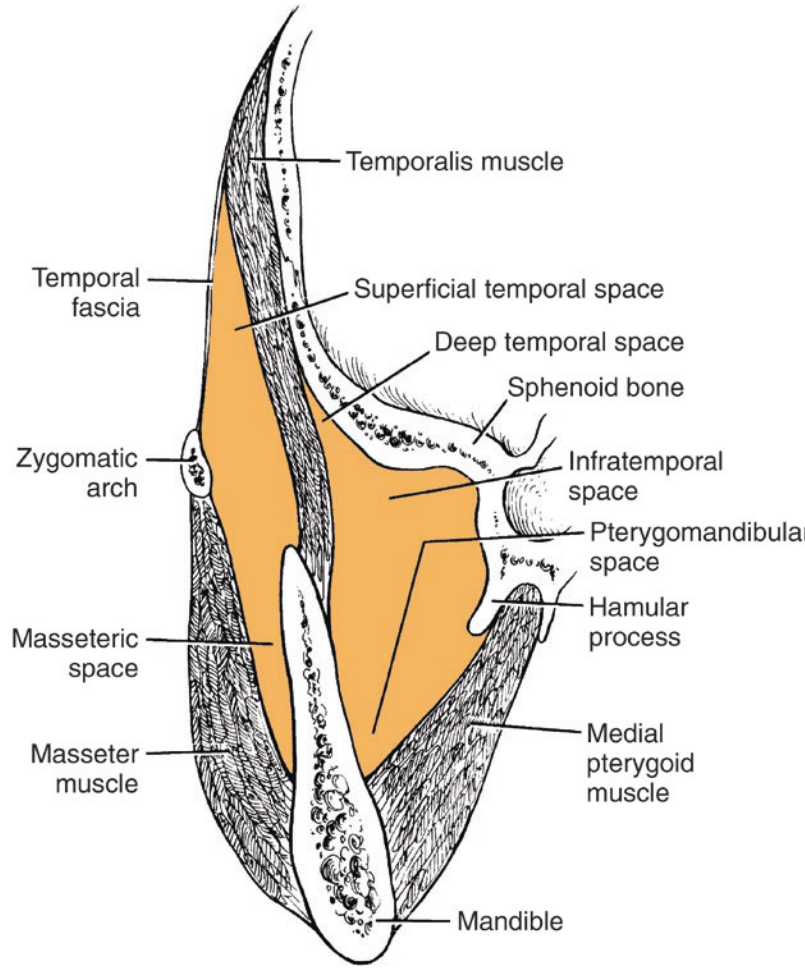

Fig. 3.3 The masticator space is made up of the temporal space, pterygomandibular space, and masseteric space. The pterygomandibular space lies between the medial aspect of the mandible and the medial pterygoid muscle. The masseteric space lies between the lateral body of the mandible and the masseter muscle. The temporal space is posterior and superior to the masseteric space and pterygomandibular space. It is bound by the temporalis fascia laterally and the skull medially. (Reprinted with permission from Flint et al. [4])

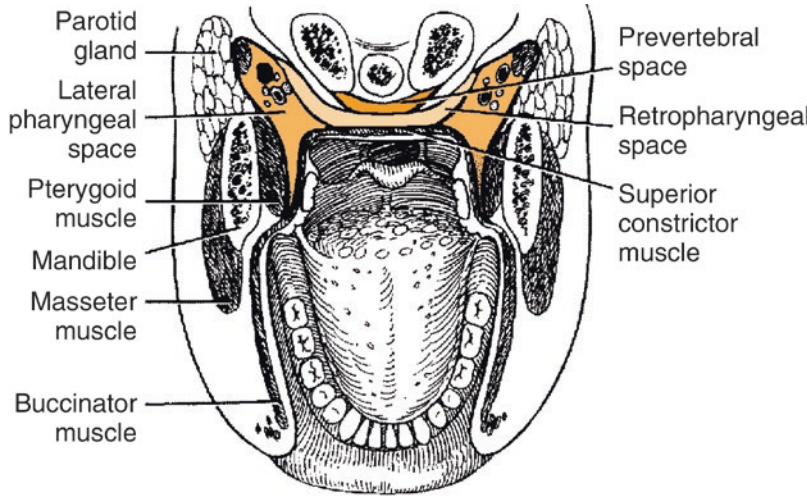

Fig. 3.4 The lateral pharyngeal space is shaped like an inverted cone with its base as the skull base and the apex at the hyoid bone. It is located between the medial pterygoid muscle laterally, the superior pharyngeal constrictor superiorly, the pterygomandibular raphe anteriorly, and the retropharyngeal space posteriorly. The styloid processes and its attachments divide the space into anterior compartments (containing muscles) and posterior compartments (containing the carotid sheath and cranial nerves). (Reprinted with permission from Flint et al. [4])

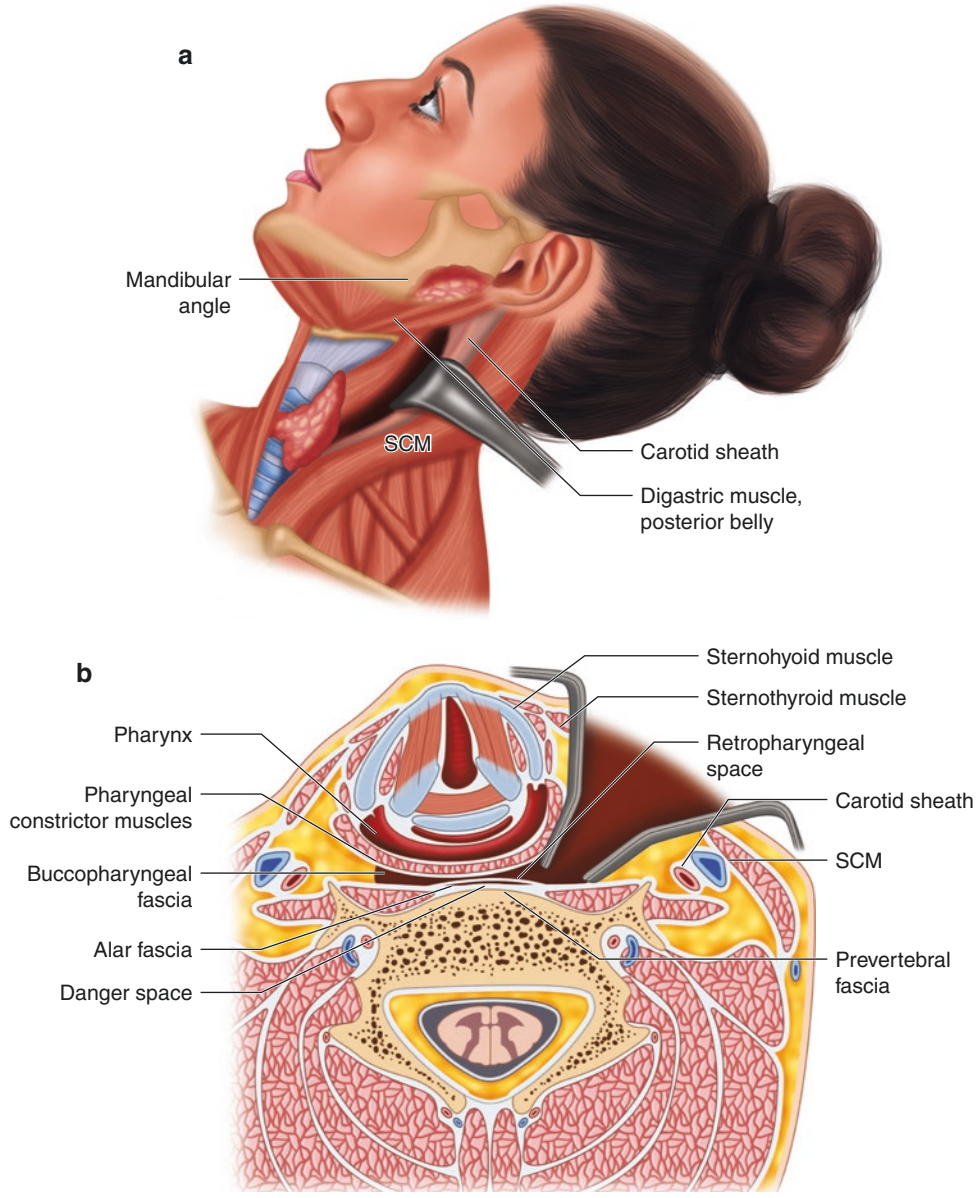

Fig. 3.5 (**a**) Retropharyngeal space is located posterior and medial to the lateral pharyngeal space. It is bounded superiorly by the base of skull, anteriorly by the superior pharyngeal muscle, posteriorly by the alar fascia, and extends inferiorly to the level of C7 or T1 (fusion of alar and buccopharyngeal fascia at the level of the posterior mediastinum). Involvement of the prevertebral space (danger space no. 4) may communicate to the region of the diaphragm. (**b**) Access to the retropharyngeal space. Incision is made parallel to the anterior border of the sternocleidomastoid (SCM) muscle inferior to the hyoid bone. The SCM and carotid sheath are retracted laterally. The loose connective tissue is bluntly dissected between the carotid sheath and the esophagus to gain access to the retrophrangyeal space. (Reprinted with permission from Kademani and Tiwana [5])

  - Signs of respiratory distress such as labored breathing (dyspnea) or noisy breathing (stridor) or inability to tolerate being in a supine position. Does the patient have hoarseness to their voice (dysphonia)? If outpatient setting, activate emergency response system for transfer. Does the airway need to be secured prior to imaging? Look for patient posturing to improve airway patency by aligning upper and lower airways (e.g., sniffing position).
- *Head and Neck Exam Assessment*
  - Measure maximal incisal opening. A concerning sign is opening less than 30 mm.
  - Palpate for tenderness, warmth, induration (firm), crepitus (sensation of crackling suggestive of gas), or fluctuance (fluid wave on bidigital palpation suggestive of pus). Note any parulis or fistula of skin.
  - Lymphadenopathy can aid in determination of origin.
  - Decreased mobility of neck. Normal flexion 70–90°, extension 55°, and rotation 70°. Nuchal rigidity may be a sign of retropharyngeal space infection.
  - Attempt to palpate if trachea is midline; in severe infection there can be deflection and markings for emergency cricothyrotomy/tracheostomy may be off-center.
  - Inability to palpate inferior border of mandible (indicative of submandibular space involvement).
  - Floor of the mouth and tongue elevation (indicative of sublingual space involvement). If the patient is able to extend tongue past vermillion border of the upper lip, there is less of a chance that the sublingual space is involved.
  - Deviation of uvula to the opposite side (indicative of the lateral pharyngeal/pterygomandibular/peritonsillar space involvement; also may be indicative of an oropharyngeal malignancy). Swelling of lateral neck between the sternocleidomastoid and mandibular angles, just above hyoid, is suggestive of lateral pharyngeal space involvement.
  - Look for carious, periodontally involved or abscessed teeth and their relation to the region of involvement.
  - Look for erythema and crepitus spreading to chest and neck for spread of infection/mediastinitis/necrotizing fasciitis. Consider serial markings of skin in area of erythema to monitor spread.
  - Look for use of accessory muscles of respiration.
  - Dimpling over zygomatic arch can be seen with temporal space involvement due to adherence of temporal fascia to periosteum.
  - Cranial exam to examine for intracranial extension.
- *Cardiopulmonary Exam*
  - Tachycardia may be appreciated in the setting of an infection due to an increase in sympathetic tone.
  - Pulmonary rales may be appreciated in the setting of acute respiratory distress syndrome secondary to sepsis.
  - Distant heart sounds, murmurs, and pericardial friction rub may be indicative of mediastinal spread.

Labs

- *Complete Blood Count*
  - Look for leukocytosis with a left shift. Leukopenia can also be seen in a serious infection. WBC count can also be trended to assess for resolution of an infection. A thrombocytosis can also be appreciated in the setting of infection (acute phase reactant).
  - Left shift/bandemia refers to the presence of immature white blood cells released into bloodstream denoting an acute infection.
- *Basic Metabolic Panel*
  - BUN/creatinine ratio can be used to assess the volume status of the patient. Patients may display prerenal (denoted by a BUN/CR ratio of greater than 20) or renal azotemia.
  - Renal baseline function is important to know as certain antibiotics are nephrotoxic

which may have implications on dosing. Creatinine levels are also necessary prior to obtaining CT with contrast due to the risk of contrast-associated nephropathy.
  - Hyperglycemia or hypoglycemia (if no oral intake) may be present in diabetics which may need to be treated. Blood sugar below 200 mg/dL is imperative for good infection control.
  - Electrolyte disturbances may also be present with long-term malnutrition.
- *C-Reactive Protein*
  - Marker of inflammation that rises in response to inflammation (acute phase reactant). Can be trended to assess for resolution of an infection.
- *Blood Cultures*
  - Routine culturing is not practiced. Should be reserved for those with signs of septicemia to prevent false-negative results.

Systemic Inflammatory Response Syndrome (SIRS)

SIRS is defined by having two or more of the following:

1. Fever >38 °C or <36 °C
2. Heart rate >90 beats per minute
3. Respiratory rate >20 breaths per minute or $PaCO_2$ <32 mm Hg
4. Abnormal WBC count (>12,000/mm^3 or <4000/mm^3 or >10% bands)

Sepsis

Sepsis – life-threatening organ dysfunction caused by a dysregulated host response to infection

- Organ dysfunction – based on sequential organ failure assessment (SOFA) scores. Points are given to abnormalities in cardiovascular, coagulation, pulmonary, liver, renal, and brain panels. A score of two or more denotes organ dysfunction.
- Infection – based on clinical signs (e.g., SIRS) and supportive microbiologic and radiological data.
- *Imaging*
  - *CT with contrast* – image must extend from the skull base to the thoracic inlet. 3 mm cuts in the neck and 5 mm below the hyoid. Contrast used to delineate collections manifested as ring-enhancing collections noted on CT. Fat stranding can also be appreciated. Also can assess for radiographic evidence of airway embarrassment and lymphadenopathy.
  - *Panorex* – plain scout film used to assess for causative teeth of the odontogenic infection. Can also see resorptive changes that could be indicative of apical periodontitis or osteomyelitis.
  - *Plain Neck Films* – screening for retropharyngeal and pretracheal spaces. Normal retropharyngeal tissue 7 mm at C-2; 14 mm for children and 22 mm for adults at C-6. Largely supplanted by CT.

Medical/Surgical Management

- Prompt medical management:
  - Intravenous fluids to address dehydration.
  - Initiate empiric antibiotic therapy. Change to specific antibiotic therapy once culture and sensitivity becomes available. The spread of an infection that is still in the cellulitis stage is aborted by prompt incision and drainage of all anatomic spaces affected by cellulitis or abscess. Cultures taken from cellulitis yield viable specimens for culture and sensitivity testing.
  - Analgesics
- Determine what spaces are involved.
- Admit to hospital for serious infection.
- Criteria for hospital admission:
  - Temperature >101 °F
  - Dehydration
  - Signs of airway embarrassment
  - Infection involving secondary fascial spaces
  - Need to control systemic disease that has implications on the infected patient
  - Need for general anesthesia

- Nursing orders:
  - Suction at the bedside
  - NPO
  - Monitor ins and outs
  - Q4 vital signs
  - Head of bed elevated to 30 degrees

Surgical Management

- Discuss securing a definitive airway with the anesthesia team before proceeding to the operating room. An awake fiberoptic intubation or an awake tracheotomy may be indicated.
- Consider needle decompression prior to intubation to prevent rupture of abscess upon intubation.
- Be prepared for emergency tracheotomy in the "cannot ventilate, cannot intubate" situation.
- Mark out emergency cricothyrotomy prior to intubation attempt. This may be deviated in serious infection.
- Attempt aspiration for sterile sample for culture and sensitivity.
- Make incision in healthy skin versus height of fluctuance to prevent scar contracture.
- Place an incision in a natural fold of skin in a gravity-dependent position.
- Bluntly dissect into the involved spaces to establish drainage. Attempt to follow lingual border of mandible to prevent damage to facial vessels.
- A through and through drain, passing from one skin or mucosal incision through the infected space to a separate incision, can be used to allow unidirectional flow of irrigation fluid, to provide two routes for drainage, and to keep the incisions away from the site of abscess formation.
- Irrigate copiously.
- Extract the offending teeth.
- Reassess the patient frequently after incision and drainage.
- Consider infectious diseases consultation.

Management of Orbital Infections

- Orbital infections can include different anatomic sites with varying clinical manifestations. A thorough clinical exam to evaluate visual acuity, pupillary reflexes, extraocular movement, and ophthalmoscopy is indicated to evaluate and distinguish the extent of infection.
- Orbital infections are rare sequelae of sinusitis, odontogenic infections, or orbital trauma and may have devastating consequences if they are not treated aggressively.

Classification of Orbital Infections (See Fig. 3.6) [3]

- Group 1: inflammatory edema (preseptal cellulitis)
- Group 2: orbital cellulitis
- Group 3: subperiosteal abscess
- Group 4: orbital abscess
- Group 5: cavernous sinus thrombosis

Review of Pertinent Anatomy

- The orbit is a cone-shaped structure.
- Surrounded by paranasal sinuses (frontal, ethmoid, and maxillary).
- Orbital septum (see Fig. 3.7): membranous sheet that extends from the periosteum of the infraorbital region to the tarsal plate and forms the anterior boundary of the orbital compartment.
- Lamina papyracea: separates ethmoid sinuses from the orbit. Nerves and vasculature within natural fenestrations are named Zuckerkandl's dehiscences.
- Most common route of infection to the orbit is by extension from the ethmoid sinuses.
- Superior and inferior ophthalmic veins drain blood directly into the cavernous sinus.
- Inferior orbital veins are valveless, and infections can pass readily from the orbit to intracranial structures.

Workup

- Usual review of medical history, systems, and duration/onset. Emphasis on symptoms of decreased vision and decreased color perception.
- Labs tests: complete blood cell count and blood cultures.
- Visual acuity (e.g., Snellen chart).

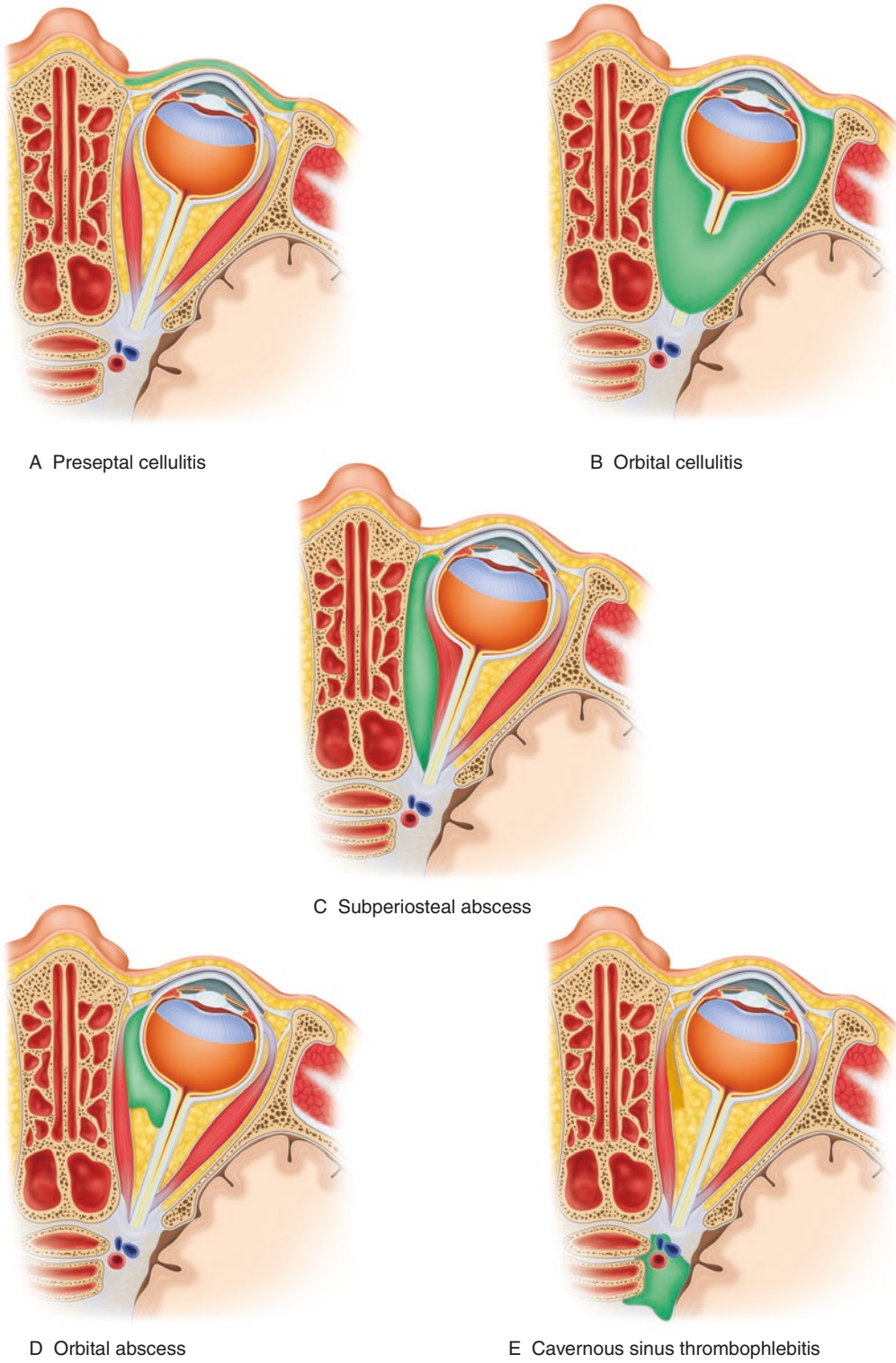

Fig. 3.6 Classification of orbital infections. (Reprinted with Permission from Chander J., Lagenbrunner D, Stevens E [3])

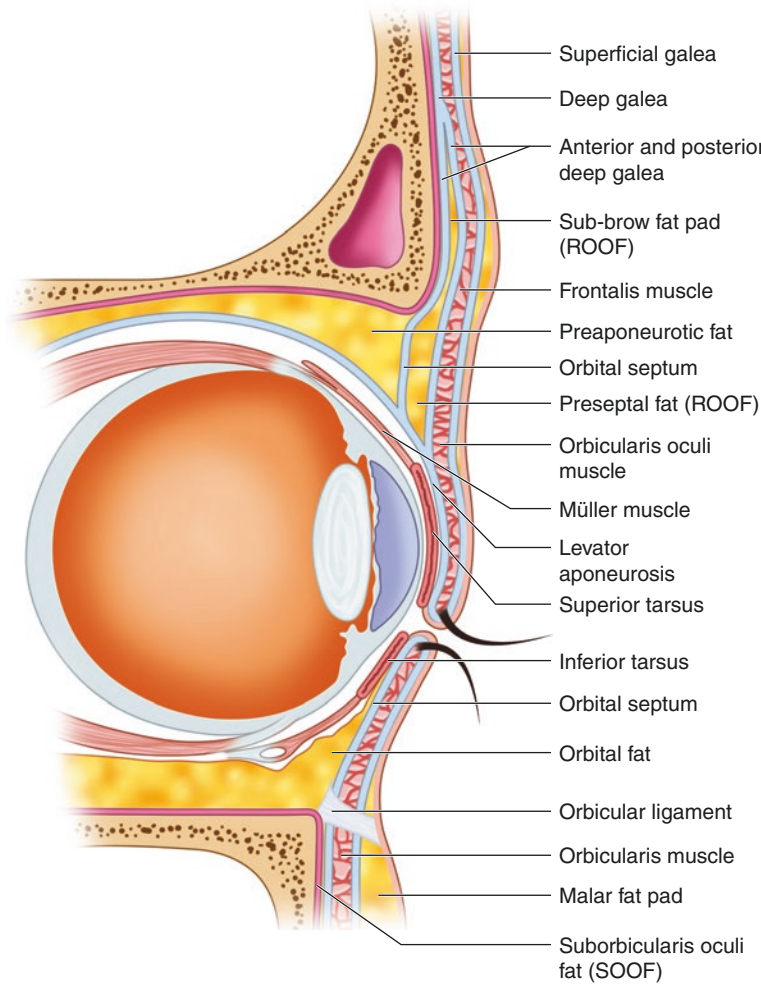

Fig. 3.7 Orbital septum. (Reprinted with permission from Tyers and Collin [6])

- Extra-ocular muscles movement, cardinal position of gaze.
- Pupillary examination, check afferent pupillary reflexes.
- Check ocular pressures.
- Fundoscopic exam to assess optic nerve involvement (papilledema – optic nerve swelling).
- Consider ophthalmology consultation.
- Posterior orbital involvement around the superior orbital fissure and optic foramen may result in orbital apex syndrome.

Radiography

- CT of orbits and/or sinuses with contrast, 3 mm cuts to help distinguish between preseptal and orbital cellulitis.
- Preseptal cellulitis radiographic features: diffuse soft tissue edema will be seen anterior to the septum on CT.
- Postseptal cellulitis radiographic features: intraconal fat stranding and edema of the extra-ocular muscles are seen. All these radiographic signs are posterior to the septum on CT.

- Postseptal abscess radiographic features: collection of purulent material (ring-enhancing lesion) between the bony walls of the orbit and the periorbita. Displacement of the globe away from the site of the abscess.

Preseptal Cellulitis

- Infection confined to the lids and periocular soft tissues anterior to the orbital septum.
- More common in children than in adults.
- Three primary sources: (1) paranasal sinusitis, (2) upper respiratory tract infection, (3) direct inoculation (e.g., chalazion or trauma to area).
- Bacteria implicated: *Staphylococcus aureus, Streptococcus pneumoniae, Haemophilus influenzae,* other streptococci, and anaerobes [3].
- Clinical manifestations – ocular pain, eyelid swelling, erythema. Chemosis can occur in severe cases.

Treatment

- Antibiotic treatment should be tailored to cover community-acquired MRSA, such as clindamycin or TMP/SMX.
- Serial ocular exams looking for deteriorating or improving symptoms.
- Keep head of bed elevated to prevent worsening edema due to gravity, which may confound exam findings.
- If abscess is defined, drainage via transcutaneous, transconjunctival, or transnasal endoscopic approach through ethmoid sinus.

Postseptal/Orbital Cellulitis/Abscess

- Reflects true involvement of the orbital contents (retroseptal).
  - Fat and ocular muscle involvement.
- More common in young children.
- Blood cultures can be positive in children. Rarely positive in adults.
- Most common cause is rhinosinusitis.
  - Ethmoid sinusitis and pansinusitis can also lead to subperiosteal orbital abscess or orbital cellulitis.
- Other potential causes:
  - Ophthalmic surgery: strabismus surgery, blepharoplasty, retinal surgery.
  - Orbital trauma.
  - Dacryocystitis.
  - Odontogenic infection.
  - Fungal rhinosinusitis.
- Clinical features that distinguish from preseptal cellulitis include ophthalmoplegia, decreased visual acuity, proptosis, eye pain (ophthalmalgia), changes in visual acuity, superior orbital fissure syndrome, orbital apex syndrome.
- Usually abscess located medially or superomedially causing the eye to be fixed looking "down and out."
- Most common bacteria involved are *Staphylococcus aureus* and *Streptococci*. Mucor and Aspergillus (fungi) can cause life-threatening invasive infections and are more common in poorly controlled diabetics [3].

Treatment

- Uncomplicated orbital cellulitis– may attempt conservative treatment with antibiotics. All patients with orbital abscesses should have immediate surgical drainage.
- Broad-spectrum regimen aimed at *S. aureus, S. pneumoniae,* and other streptococci and gram-negative bacilli.
- Initial treatment includes a combined antibiotic therapy; may narrow and tailor antibiotic treatment with culture results.
- When intracranial extension is suspected, include coverage for anaerobes and request neurosurgical consultation.
- Contrast-enhanced CT or magnetic resonance venogram (MRV) may be diagnostic.
- Common antibiotics regimens include vancomycin, ampicillin–sulbactam, metronidazole, ceftriaxone, piperacillin–tazobactam, or levofloxacin.
- Indications for surgical intervention:
  - Poor response to antibiotic treatment (24–28 hours).
  - Worsening visual acuity or pupillary changes.
  - Large abscess (>10 mm).
- Surgical approaches:
  - Common approach is transconjunctival which may include transcaruncular or lateral canthotomy extensions.

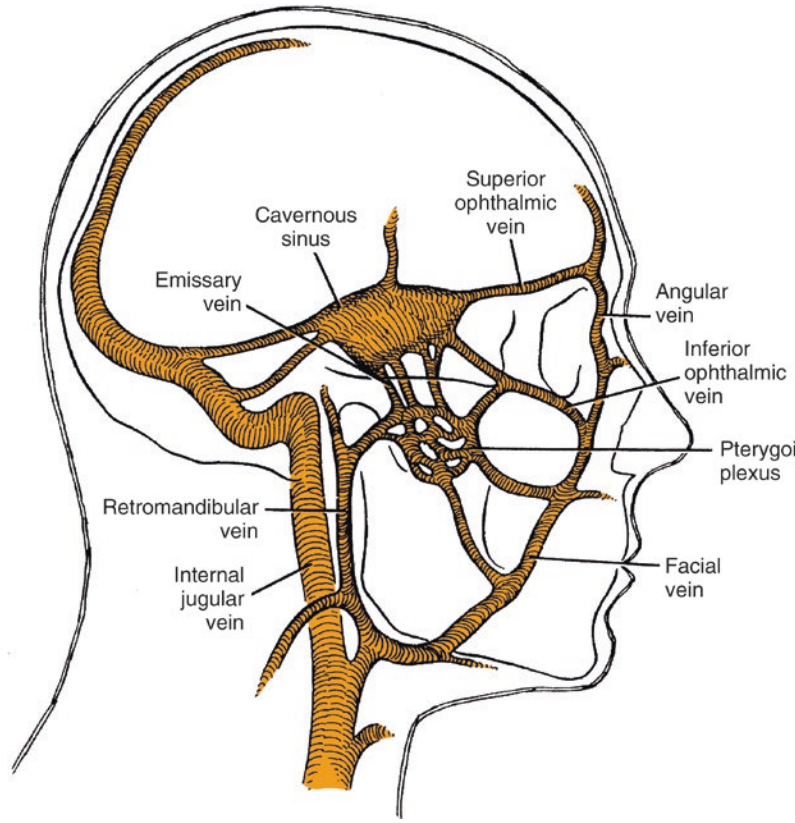

Fig. 3.8 Spread to cavernous sinus from an odontogenic source may travel via the inferior or superior ophthalmic vein or via the emissary veins to the pterygoid plexus. (Reprinted with permission from Flint et al. [4])

  - Endoscopic sinus surgery is indicated in patients with severe destructive rhinosinusitis.
- Some clinicians initiate high-dose steroid 24 hours after antibiotic therapy has begun. Steroid therapy is used to prevent ocular complications due to increased intraorbital pressure.
- Emergency lateral canthotomy and cantholysis may be required if signs of optic nerve involvement.

Cavernous Sinus Thrombosis (CST)

- Cavernous sinus thrombosis is a vascular thrombosis in the cavernous sinus with inflammation of its anatomic structures.
- Most common etiology is from contiguous spread of infection from the sinuses and very uncommon from dental abscesses (see Fig. 3.8).
- *Staphylococcus aureus* is the most common pathogen.

Anatomy of the Cavernous Sinus (See Fig. 3.9)

- Bilateral venous drainage for middle cranial fossa.
- Anteriorly bordered by the superior orbital fissure, receiving tributaries of the ophthalmic vein.
- Posterior border is the trigeminal ganglion.
- Superior and inferior ophthalmic veins, central retinal vein, and the middle meningeal vein drain into cavernous sinus.
- The cavernous sinus drains into the superior and inferior petrosal sinuses.
- Emissary veins drain from the sinus into the pterygoid plexus to the retromandibular vein.
- Nerves in sinus:

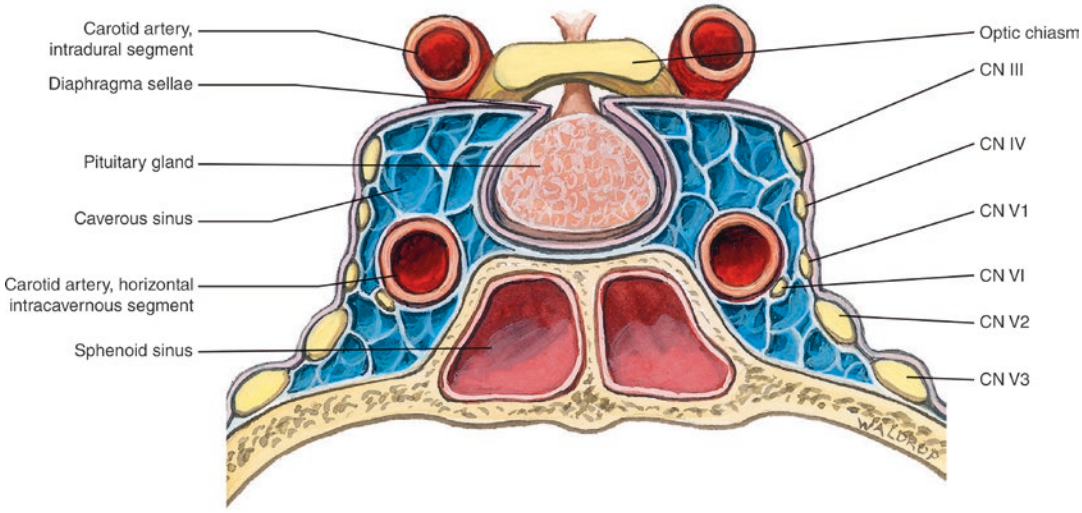

Fig. 3.9 Anatomy of the cavernous sinus. (Reprinted with permission from Dutton [7])

- Oculomotor (CN III)
- Trochlear (CN IV)
- Abducens (CN VI)
- Ophthalmic (CN V-1)
- Maxillary (CN V-2)

Presentation of Cavernous Sinus Thrombosis

- Aseptic causes – after surgery or after trauma.
- Infectious causes:
  - Sinusitis
  - Otitis
  - Facial furuncles
  - Erysipelas – superficial cellulitis of skin that is caused by β-hemolytic streptococci and group B streptococci.
- Symptoms: fever, headache, and diplopia common symptoms.
- Earliest neurological sign is lateral gaze palsy (CN 6). First easily assessed sign of CST, as it is the only cranial nerve traversing the interior of the sinus.
- Clinical signs include photophobia, proptosis, sepsis, lid edema, chemosis, dilated pupils, cranial nerve 3, 4, 6 palsies (ophthalmoplegia), and paresthesia of V1, V2. Dilatation of the retinal veins of the opposite eye may precede lateral gaze palsy CN 6 on the affected side. This is due to venous congestion in the cavernous sinus obstructing the venous outflow of the retinal veins on the unaffected side.
  - Pyrexia is seen with a "picket fence" pattern of high temperature spikes, suggestive of thrombophlebitis.
  - On fundoscopic exam, congested retinal veins on the opposite side are the earliest signs of cavernous sinus thrombosis.
- Clinical evidence of intracranial extension:
  - Nausea/vomiting
  - Altered mental status
  - Generalized sepsis
- Intracranial extension of infection may result in meningitis, encephalitis, blindness, brain abscess, pituitary infection, epidural and subdural empyemas, possible coma, and death.

Danger Triangle on Face leading to CST

- Triangular region formed by the corners of the mouth, medial cheeks, and bridge of the nose.
- Pathway is via retrograde flow through veins that are valveless.
  - Facial veins>angular vein>ophthalmic veins>cavernous sinus
  - Emissary veins connected to the pterygoid plexus (slower spread)
  - Internal jugular vein connecting to inferior petrosal sinus (complication of Lemierre's syndrome)

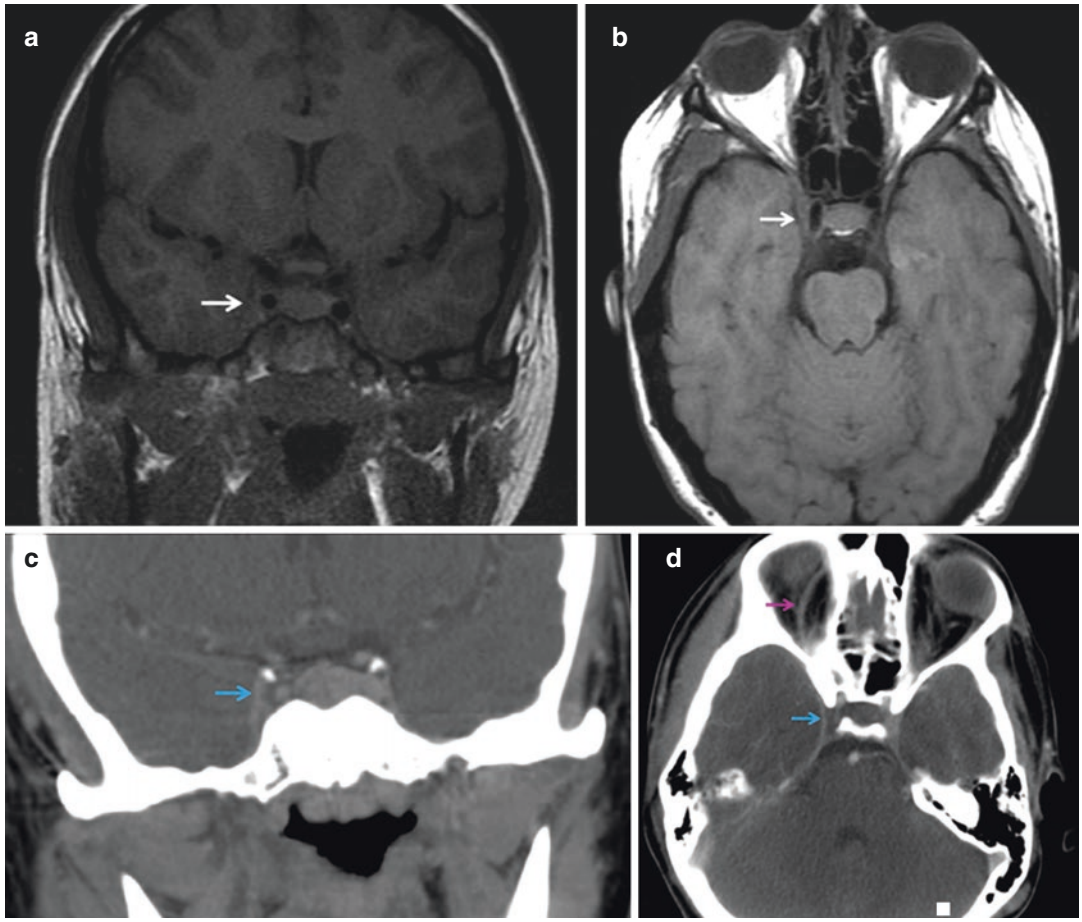

Fig. 3.10 Cavernous sinus thrombosis. (**a**) T1-weighted MRI at level of the cavernous sinus, thickening of the right cavernous sinus (arrow). (**b**) T1-weighted MRI at the level of orbits, thickening of the right cavernous sinus (arrow). (**c**) Soft tissue window coronal CT with areas of low attenuation within cavernous sinus representing thrombus (arrow). (**d**) Soft tissue window axial CT at level of orbits with mild enlargement of superior ophthalmic vein (superior arrow) and low attenuation region in the right cavernous sinus representing thrombus (inferior arrow). (Images courtesy of Dr. Gillian Lieberman and Dr. Christopher Aderman)

Radiographic Features of CST (see Fig. 3.10)

- Magnetic resonance venography (MRV):
  - Imaging modality of choice to examine venous anatomy and demonstration of decreased or absence of signal in area of thrombus
  - Venous wall thickening
  - Filling defect in cavernous sinus
  - Lateral bulging of cavernous sinus wall
  - Narrowing of the internal carotid artery within the cavernous sinus
- CT with and without IV contrast:
  - Expansion of cavernous sinus
  - Convexity of the normally concave lateral wall
  - Indirect signs: venous obstruction, dilation of the superior ophthalmic vein

Treatment of Cavernous Sinus Thrombosis

- Surgery
  - Directed at the primary source of infection and obtaining culture.
- Antibiotics:
  - Start with empiric antibiotic regimens with combination of (1) third- or fourth-generation cephalosporin that crosses the blood–brain barrier, such as ceftriaxone (third generation) or ceftazidime (fourth generation), (2) metronidazole, and (3) vancomycin (high likelihood of *S. aureus*) until culture results.

  - 6–8 weeks of therapy.
- Steroids (controversial):
  - May have benefits of decreasing orbital inflammation and cranial nerve edema.
- Anticoagulants (controversial):
  - Proposed benefit: cessation of the progression of the thrombus to other dural venous sinus and cerebral veins.
  - Early anticoagulation decreased morbidity (blindness, stroke, ophthalmoplegia, focal seizures, vascular steal syndrome).
  - Risk: intracranial and systemic hemorrhage. Many postmortem evaluations have shown venous hemorrhagic infarcts.

Mucormycosis (Zygomycosis)

- Mucormycosis is an opportunistic fungal infection (caused by fungi in the Mucorales family) that occurs in immunocompromised patients.
- Head and neck manifest as two forms: rhinocerebral and rhinomaxillary.
- Disease involves thrombosis, vascular invasion, ischemia, and infarctions.
- Black necrotic eschars in the oral cavity, palate, or face can be the early diagnostic signs.
- Fungi will enter the body through the nasal mucosa, lungs, or skin.
  - Cutaneous mucormycosis: injured tissue in the oral cavity can be a suitable port of entry.
  - Fungal hyphae preferentially invade the walls of blood vessels, producing thrombi and infarctions.
  - Progressive tissue ischemia and necrosis are the inevitable result.
- Rhizopus organisms have an enzyme, ketone reductase, which allows them to thrive in high glucose and acidic conditions.
  - Serum from individuals in diabetic ketoacidosis stimulates growth.
- Rhino-orbital cerebral and pulmonary mucormycosis are acquired by the inhalation of spores.
- The agents of mucormycosis are angioinvasive; infarction of tissues is a hallmark of invasive disease.

Oral Mucormycosis

- Most frequently seen as palatal ulcers – almost always necrotic, well-defined borders.
- May result from rapid lysis of the maxilla or other adjacent structures.
- Has been reported in the alveolar ridge, lips, cheeks, tongue, mandible (rare), and maxillary sinuses.
- Diagnosis by biopsy with histopathological evidence of fungal invasion showing broad non-septate hyphae with right angle branching.
- Fungal cultures may not reveal mucor, so biopsy is important for diagnosis.

Management/Treatment

- Surviving this condition largely depends on early identification and treatment.
- Elimination of predisposing factors for infection (e.g., hyperglycemia, metabolic acidosis, immunosuppressive drugs, and neutropenia).
- Several simultaneous approaches: surgical intervention and antifungal therapy.
- Antifungal treatment: systemic (IV), high-dose amphotericin B (5 mg/kg with the liposomal formulation).
  - Side effects: renal toxicity and high fevers and chills (shake and bake).
  - Monitor serum urea nitrogen, creatinine, and creatinine clearance.
  - Amphotericin mechanism of action – binds with the ergosterol of fungal membrane causing disruption and ion permeability.
- Posaconazole or isavuconazole can also be used for salvage therapy for patients who don't respond or cannot tolerate amphotericin B.
- Surgical debridement to limit aggressive spread of infection. Aggressive surgical debridement of involved tissues should be undertaken as soon as the diagnosis of any form of mucormycosis is suspected.
- Therapy should continue until there is a clinical resolution of signs/symptoms as well as resolution of radiographic signs of active disease.

Cervicofacial Necrotizing Fasciitis

- Aggressive bacterial infection leading to necrosis of the superficial fascial planes with concurrent systemic toxicity.
- High mortality rate due to sepsis.
- More common in diabetic, alcoholics, malnourished, malignancy, obese, and immunocompromised patients.
- Five distinct bacterial patterns are seen in necrotizing fasciitis:
  - Type I – mixed aerobic and anaerobic, most commonly seen.
  - Type II – *Streptococcus pyogenes* (group A beta-hemolytic streptococci), seen more often in otherwise healthy children.
  - Type III – *Staphylococcus aureus* (MRSA).
  - Type IV – clostridial, gas-producing bacterium.
  - Type V – *Klebsiella pneumonia* – may be highly resistant to antibiotics.
- Clinical signs:
  - Erythematous skin without demarcation that is tense, smooth, shiny, and painful.
  - Will have signs of sepsis including tachycardia, pyrexia, apathy, weakness, hypotension, etc.
  - Progression leads to vesicle and blister formation early, followed by dusky purple discoloration.
  - Skin may become anesthetic due to compression and destruction of the underlying sensory nerves.
  - Crepitus may be present due to gas production.
  - Drainage is described as "dishwater" due to the foul smell, low viscosity, and gray color. Product of colliquative necrosis.
- Radiographic signs:
  - CT scan would identify soft tissue emphysema and edema, possible gas bubbles.
- Laboratory studies:
  - Complete blood counts. Extreme leukocytosis and anemia secondary to bacterial hemolysis and bone marrow suppression.
  - Comprehensive metabolic panel. Hypocalcemia due to sequestration of calcium into regions of fat necrosis, elevated blood glucose, elevated blood urea nitrogen, and elevated creatinine.
  - Lactate levels will be increased.
- Treatment:
  - Secure a definitive airway.
  - Early recognition leading to surgical intervention via fasciotomy and necrotic tissue debridement (requires serial debridement). Muscle layers can be preserved, but all necrotic tissues and overlying skin must be removed.
  - Biopsies of the involved fascia to identify toxin-producing invasive streptococcal infection should be taken.
  - Gram-positive cocci invading fascia, without leukocytic infiltrate, indicates streptococcal toxin production.
  - Frozen sections show dense polymorphonuclear infiltrates in the dermal layers of skin; these may guide the removal of devitalized tissue.
  - Biopsies should be taken of adjacent normal looking tissue, not necrotic tissue.
  - Broad-spectrum empiric antibiotics (e.g., carbapenem plus vancomycin to cover all five types) with de-escalation according to culture and sensitivity results.
  - The wounds should be washed (consider hydrogen peroxide to aid in tissue debridement) and packed with antimicrobial-soaked gauze (e.g., povidone iodine) regularly.
  - Hyperbaric oxygen. Must start early in treatment. Must weigh against cost and risk, availability in hospital, and risk of transport. Some protocols for acute infection require two three dives per day.
  - Fluid, electrolyte, and blood replacement for volume repletion and hemolysis.
  - Secondary reconstruction may require locoregional flaps and skin grafts.

Mediastinitis

Life-threatening infection involving the mediastinum.

- Spread from an odontogenic source is via the danger space (also called Space 4) that is found between the alar and prevertebral fascia. This space extends from the base of the skull, through posterior mediastinum, to the level of the diaphragm. Its loose areolar tissue allows for rapid spread of infection. The infection normally enters this plane through the fusion of alar and prevertebral fascial layers between C6 and T4.
- Clinical signs:
  - Chest pain
  - Dyspnea
  - High fever
  - Tachypnea
  - Hypotension due to decreased venous return
- Radiographic signs:
  - Mediastinal widening and pulmonary congestion/effusions can be appreciated on chest radiographs.
  - CT may show location of collections, tissue emphysema, pericardial effusions, and decreased airway patency.
- Treatment:
  - Establish definitive airway.
  - Aggressive surgical source control including drainage of spaces (repeat drainage and debridement often required).
  - Cardiothoracic surgery consultation for open mediastinal drainage.
  - Broad-spectrum antibiotics.
  - HBO therapy may be indicated.

Osteomyelitis

In its strict definition, it is an inflammation of the medullary portion of bone. It frequently involves the cortical bone and periosteum, however.

- Bone marrow offers a path of lower resistance that allows for the spread along the medullary bone.
- Most often seen in the mandible, as the thin cortical bone of the maxillae does not easily confine the infectious process. The mandible is also not as well vascularized as the maxillae and, therefore, is more susceptible to osteomyelitis.
- Odontogenic mixed flora (primarily alpha-hemolytic *Streptococci* vs. *Staphylococcus aureus* seen in the axial skeleton.
- In general, grouped as (1) acute or chronic (1 month or greater), (2) suppurative (pus forming), or (3) non-suppurative.
- Symptoms: pain, trismus, paresthesia/anesthesia, anorexia, swelling over affected area, loose teeth, adenopathy, and malaise. Chronic disease may form a fistula or sinus tract.
- Imaging:
  - A scouting film like an orthopantogram may show odontogenic infection with or without sequestra.
  - CT scan can show the extent of lytic bone, keeping in mind 30% demineralization is required to appreciate changes.
  - Radionuclide imaging:

    Allows for earlier identification of osteomyelitic activity, as early as 3 days. Technetium-99, although non-specific, will aid in identifying areas of higher blood flow and osteoblastic activity. It is used in a three-phase scan and is typically ordered if osteomyelitis is suspected.

    Although technetium-99 scan alone is normally sufficient, a gallium-67 aids in ruling out osteomyelitis from malignancy and trauma. Gallium-67 identifies inflammatory changes, as it binds to granulocytes.
  - White blood cell tagging can be useful in detecting early infection when lytic processes are not appreciated on imaging.
  - PET CT scan with fluoride isotope is sensitive to areas of bone turnover with much greater resolution.
- Treatment
  - Patient should be treated with corticotomies (bur fenestration) or removal of the buccal bone (decortications) for decompression, plus removal of infected teeth or repair of a mobile fracture segment.
  - Infected bone marrow should be debrided until it bleeds. It is useful to send multiple culture and histopathological samples along the length of involved bone to see the extent of infection or changes in flora.

- Cultures of affected bone for microbiology and bone biopsies sent to pathology (to rule out neoplasia and identify fungi or actinomycosis in samples).
- Infectious diseases service should be consulted due to the need for long-term IV antibiotics with PICC line.
- Hyperbaric oxygen should be considered to aid in revascularization and for antimicrobial utility.

Case (Fig. 3.11)

- *You have been paged to the ED to evaluate a 40-year old male with dental pain and extensive left facial swelling. What would you like to know?*
  - HPI (history onset, duration, symptoms etc.)
  - Past medical history
  - Presence of dysphagia, dysphonia, odynophagia, fevers etc.
  - Vital signs
  - NPO status
- *The patient has a history of hypertension and takes lisinopril. He has no previous history of surgery. He smokes 1 pack per day for 15 years. Social use of alcohol. He has no known drug allergies. He has not had anything to eat or drink for 2 days as he says he cannot swallow. What would you like to do next*?
  - Physical exam:
  - Inspection

    Look for facial swelling

    Assess whether the patient can tolerate his secretions.

    Look for posturing to improve airway patency (e.g., sniffing position or tripod position)
  - Head and neck exam:

    Record temperature.

    Measure maximal incisal opening.

    Assess for lymphadenopathy.

    Look for spreading erythema, using marking pen to delineate extent.

    Is the inferior border palpable (indicative of submandibular space involvement)?

    Is the floor of the mouth elevated (indicative of sublingual space involvement)?

    Is there uvular deviation from the midline (indicative of lateral pharyngeal/pterygomandibular space involvement)?

    Look for carious or periodontally involved teeth and their relation to the region of involvement.

    Palpate the area of involvement to determine the character of the swelling (firm versus fluctuant).
  - Cardiopulmonary Exam

    Are the accessory muscles of respiration being used?

    Auscultate heart and lungs

    Tachycardia may be appreciated in the setting of an infection

    Are the heart sounds distant or muffled?

    Is there any heart murmur?

    Rales?

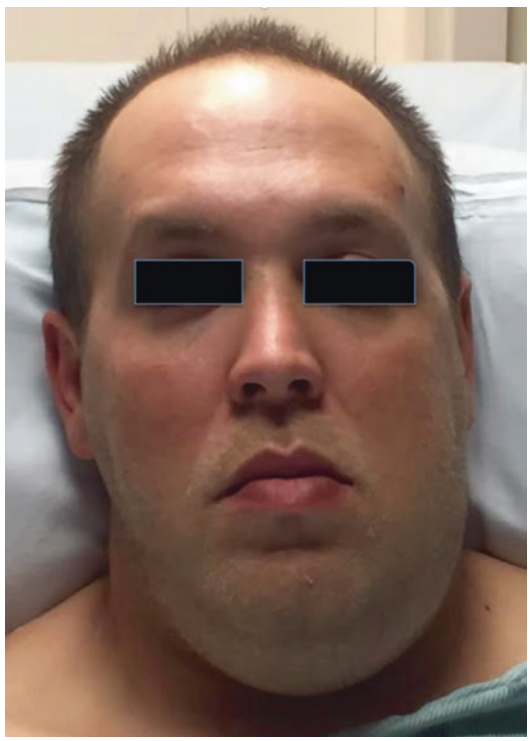

Fig. 3.11 Infection case photo. (Image courtesy of Dr. Damian Findlay)

- *What laboratory studies would you like to see?*
  - CBC, CRP, BMP, and obtain a blood culture.
- *Your patient is febrile to 104 degrees Fahrenheit (40 degrees Celsius) with a heart rate of 105 bpm. His maximum incisal opening is 25 mm. On exam, he has a toxic appearance. The inferior border is not palpable. Intraoral exam reveals fullness in the floor of the mouth and left palatoglossal arch. Extensive caries noted on #17 and #18 with tenderness to percussion. He has a WBC count of 17,000 with a left shift and he has BUN/creatinine ratio of 20:1. What imaging would you like to order?*
- CT with contrast-image must extend from the orbits to the clavicles. Contrast used to delineate encapsulated abscesses manifested as ring-enhancing collections noted on CT.

Your patient returns from CT. Below are his images (Figs. 3.12 and 3.13)*:*

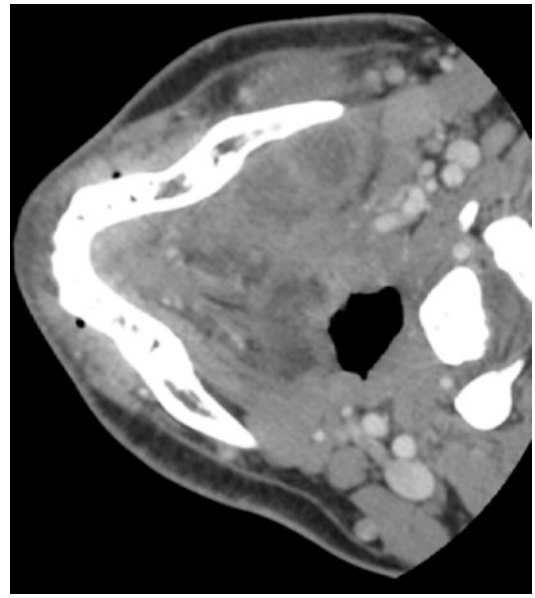

Fig. 3.12 CT scan with contrast at the level of the mandible. (Image courtesy of Dr. Damian Findlay)

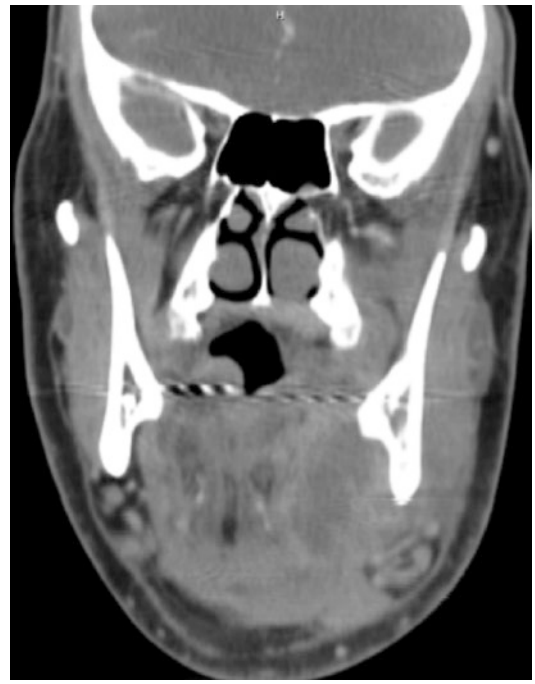

Fig. 3.13 CT scan with contrast at the level of the pterygoid plate. (Image courtesy of Dr. Damian Findlay)

- *What spaces are involved?*
  The left sublingual, submental, submandibular, and pterygomandibular spaces are involved (note the ring-enhancing areas in the aforementioned spaces).

- *How do you want to proceed?*
  - Admit to hospital
  - Intravenous fluids to address dehydration
  - Start empiric antibiotic therapy
  - Analgesics
  - Nursing orders
    - Suction at the bedside
    - NPO
    - Monitor ins and outs
    - Q 4 vital signs
  - Plan for prompt incision and drainage
- *How would you proceed with your drainage?*
  Mark out landmarks for cricothyrotomy and have surgical airway kit open and ready in case of emergent airway concerns. After securing a definitive airway, I would scrub and drape the patient in a normal sterile surgical fashion. I would then place a throat pack. At this time, I would attempt needle aspiration of the abscess to have a sterile sample for culture. I would palpate and mark a 3 cm incision in healthy skin under the area of the fluctuance, ensuring I was 2 cm below the inferior border of the mandible. I would

make a sharp incision using a 15 blade down to platysma. I would bluntly dissect with a hemostat toward the lingual border of the mandible. I would continue to dissect superiorly in a subperiosteal plane along the lingual border of the mandible traversing the sublingual and submandibular spaces. My other hand would be inside the mouth along the floor of the mouth for the purpose of appreciating the tip of the hemostat. I would then continue my dissection posteriorly along the lingual mandible to enter the pterygomandibular space. I would then redirect the hemostat along the lingual border deep to the lingual gingiva into the oral cavity adjacent to the involved teeth. This bimanual manipulation also would ensure entry to the correct spaces and also aid in placement of through and through drains. I would use penrose drains (another option is a perforated red rubber catheter or a Jackson-Pratt drain) and place them in the involved spaces in a through and through fashion. The drains would then be irrigated to ensure patency of drains and communication with drains in other spaces. The drains would then be secured with a non-resorbable suture. I would remove the offending dentition. I would then irrigate the oral cavity. I would remove the throat pack and place drain sponges/dressings.

- *What is the function of the drain?*
  They facilitate gravity-dependent drainage of fluids or purulence from the wound. They allow for also cleansing of the infected site via irrigation. Prevent closure of the mucosa which can result in reformation of abscess.
- *When would you remove the drains?*
  I would remove the drains when the drainage is nearly complete (without signs of gross purulence/mainly serosanguineous drainage). This would be typically between 3 and 7 days. Leaving the drain for an extended period of time may lead to secondary infection as drains are antigenic and allow for ingress of skin flora.
- *How does incision and drainage treat the patients?*
  Reduction in tissue tension improves local blood flow and increase host defenses to area. Decreases bacterial load and removes necrotic tissue.
- *What cultures would you order?*
  Aerobic, anaerobic, and fungal cultures with antibiotic sensitivities in addition to a gram stain.
- *What is gram stain?*
  A gram stain is a rapid test to aid in categorizing involved microorganisms into four broad groups: gram positive cocci, gram positive rods, gram negative cocci, or gram negative rods. This aids in tailoring early antibiotic therapy. It involves process of staining, decolorizing, and counterstaining the microorganism to detect a peptidoglycan in the cell wall found in gram positive bacteria.
- *What is Ludwig angina?*
  Rapidly progressing cellulitis that may not have yet formed abscesses. This infection involves the bilateral submental, sublingual, and submandibular spaces. Angina refers to the respiratory distress associated with airway obstruction.
- *The patient is 3 days s/p incision and drainage. On exam your patient appears toxic and is not tolerating his secretions. He spiked a fever this morning of 103 degrees Fahrenheit. On exam the uvula is deviated to the right. How do you proceed?*
  Rescan the patient. He may have developed a new collection in the previously drained spaces, inadequate drain placement, inadequate surgery/missed space or may have had extension into additional spaces.
- *What is going on?* (*see* Fig. 3.14)
  The patient's CT now shows a ring-enhancing abscess with the involvement of the lateral pharyngeal space with deviation of the airway. The patient should be taken back to the OR for incision and drainage. I would consider changing my antibiotic therapy based on culture results if not already tailored. (It may be wise in this instance to keep the patient intubated.)
- *You get a call from the ICU nurse that the patient is complaining of frequent nose bleeds. What might be going on?*

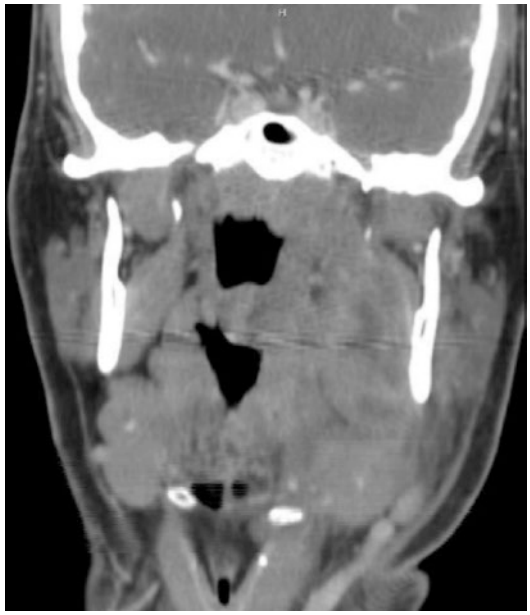

Fig. 3.14 Coronal CT scan with contrast at the level of mandibular ramus. (Image courtesy of Dr. Damian Findlay)

Possible carotid sheath involvement. Herald bleeds are intermittent bleeding episodes of the nose or the pharynx caused by erosion of the carotid artery or internal jugular vein. The erosion can be caused by placement of a rigid drain in the lateral pharyngeal space. Other signs can include palsies of nerves IX, X, or XII, Horner's syndrome, or an enlarging hematoma. If carotid sheath involvement is suspected, then order CTA of the head and neck and request vascular surgery consultation.

- *You order a CT scan, describe what you see?* (*see* Fig. 3.15)
  A ring-enhancing multilocular collection in the lateral pharyngeal space proximal to the bifurcation of the common carotid artery. Numerous intra-arterial calcifications are seen within the carotid vasculature.
- *What is the potential sequalae of this collection expanding?*
  Expansion of this infection collection could cause airway compromise and erosion of the carotid arteries. Extension into the internal carotid artery could result in a cerebrovascular accident.
- *What is your criteria for extubation?*

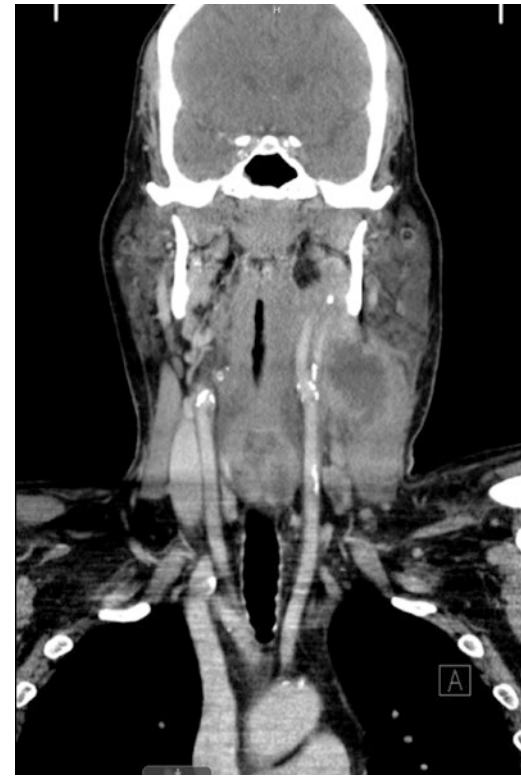

Fig. 3.15 Coronal CT with contrast level of carotid bifurcation. (Image courtesy of Dr. Damian Findlay)

Stable vital signs
Positive air leak test
Acceptable ventilatory readings (vital capacity >15 mg/kg, minute ventilation of 6–10 L/min, inspiratory pressures of >25 cm H_2O)
Normal arterial blood gases

Appendix 1. Bacteria Involved in Odontogenic Infections

Cell wall characteristics – gram negative vs. gram positive

Oxygen consumption: aerobic, anaerobic facultative

Aerobic: bacteria that utilize oxygen to generate ATP

- *Bacillus*
- *Pseudomonas*
- *Nocardia*
- *Mycobacteria*

Anaerobic: bacteria that can proliferate in low oxygen tension

- *Peptostreptococci* (also called *Anaerococcus* or *Parvimonas*)
- *Bacteroides*
- *Fusobacteria*
- *Prevotella*
- *Porphyromonas*
- *Actinomyces*
- *Actinobacillus*
- *Eikenella*
- *Veillonella*
- *Capnocytophaga*
- *Neisseria*

Facultative: bacteria that make ATP by aerobic respiration if oxygen is present but are capable of switching to fermentation or anaerobic respiration if oxygen is absent.

- *Staphylococcus*
- *Streptococcus*
- *Escherichia*
- *Listeria*
- *Lactobacillus*

Major Pathogens:

- *Viridans* group (facultative alpha-hemolytic streptococci)
- *Streptococcus milleri* group (subset of *Viridans* group)
  - *S. anginosus*
  - *S. intermedius*
  - *S. constellatus*
- *Peptostreptococcus* (anaerobic)
- *Prevotella* and *Porphyromonas* (anaerobic)
- *Fusobacteria* (anaerobic)

Appendix 2: Antibiotics

Beta-lactams

- Mechanism of action – beta-lactam antibiotics inhibit penicillin-binding proteins, which disrupt cell wall synthesis.
- Bactericidal.
- Penicillin G is no longer recommended for serious odontogenic infections due to the high number of bacteria involved in these infections that liberate beta lactamases.
- Penicillins (examples include penicillin, amoxicillin, dicloxacillin, oxacillin, nafcillin, piperacillin, ampicillin).
- Cephalosporins (examples include cephalexin, cefazolin, cefuroxime, ceftriaxone).
- Cephamycins (examples include cefoxitin and cefotetan).
- Carbapenems (examples include imipenem, meropenem, doripenem, ertapenem).
- Monobactams (e.g., aztreonam): treatment of gram negative, aerobic bacilli. Synergistic with aminoglycosides. No activity for anaerobic and gram positive bacteria.
- Beta-lactamase inhibitors – bind to the catalytic site of beta-lactamases to prevent hydrolysis of the beta-lactam (e.g., tazobactam, sulbactam, and clavulanate). Used in combination with a penicillin to cover bacteria that confer their resistance through liberation of beta lactamases. Examples include Unasyn® (ampicillin and sulbactam), Zosyn® (piperacillin and tazobactam), and Augmentin® (amoxicillin and clavulanate).

Macrolides

- Binds to the 50S ribosomal subunit and inhibits its bacterial protein synthesis. Examples include erythromycin, clarithromycin, and azithromycin.
- Bacteriostatic.
- Side effects – prolongation of the QT interval is of concern, especially when combined with other drugs that are metabolized by CYP3A4 in the liver microsomal system. Gastrointestinal side effects such as nausea and diarrhea are common.

Clindamycin (Cleocin®)

- Binds to the 50S ribosomal subunit and inhibits its bacterial protein synthesis. Its action is bacteriostatic or bactericidal depending on drug concentration, infection site, and organism.

- Part of the Lincosamides family.
- Good anaerobic coverage and excellent abscess and bone penetration.
- Does not cover *Eikenella corrodens*.
- *Pseudomembranous colitis* – common side effect
  - Treatment with cleocin and other antibacterial agents alters the normal flora of the large intestine that can lead to overgrowth of *C. difficile.*
  - *C. difficile* produces toxins A and B that cause disorganization of actin microfilaments leading to enterocyte cytoskeleton destruction and leaks between the tight junctions between enterocytes. Additionally, toxin A leads to proinflammatory cytokines that cause further intestinal mucosal damage.
  - Major side effect is antibiotic-associated colitis (AAC), which manifests itself as fever, abdominal pain, and foamy diarrhea that may be bloody.
  - First step is to discontinue use.
  - Stop any opiates or antidiarrheal medications.
  - Administration of fluids and correction of electrolytes.
  - Management includes discontinuing the offending antibiotic, fluid and electrolyte management, antibiotic treatment for *C. difficile*, and surgical evaluation as clinically indicated.
  - Antibiotic treatment is 10–14 days of metronidazole (500 mg three times daily) or oral vancomycin (125 mg four times daily). Fidaxomicin (200 mg PO BID) is now available, with decreased recurrence rates.
  - Antibiotic-associated colitis/fulminant colitis/toxic megacolon

    Usually occurs in elderly, extended hospital stay (greater than 2 weeks), exposure to IV antibiotics, and occurs in patients who underwent surgical procedures.

    See rapid spike in WBC to >20 cell/μL.

    Toxic megacolon manifests as abdominal pain, abdominal distension, fever, and extreme leukocytosis. The goal is to decompress the bowel within 24 hours or total colectomy is indicated.
  - Diagnosis of pseudomemnranous colitis is by assessing the presence of the *C. difficile* via. toxin assay from a stool sample via the cell cytotoxicity neutralization assay. Nucleic acid amplification tests and enzyme immunoassays to detect toxins A and B area also available.

Fluoroquinolones

- Bactericidal agents that inhibit bacterial enzymes (DNA gyrase and topoisomerase IV), which are involved in DNA replication. This inhibition results in damage to bacterial DNA and bacterial cell death.
- Fourth-generation fluoroquinolones have gram-positive and anaerobic coverage. Oral and IV bioavailability are equal. Examples include ofloxacin, ciprofloxacin, levofloxacin, moxifloxacin, and gemifloxacin.
- Side effects – chondrotoxicity, especially to growing cartilage. Avoid in children <18 years of age. Tendinitis/tendon rupture has been reported in patients over 60 years of age also. Many drug interactions via CYP3A4 which can worsen prolonged QT syndrome can lead to torsades des pointes and sudden cardiac death, fatigue, dizziness, skin rash, and diarrhea.

Dosages of Common Antibiotics

BETA-LACTAMS

- Pen VK
- 500 mg P.O. q6h
- Amoxicillin
- 500 mg P.O. q8h
- Amoxicillin–clavulanate (Augmentin®)
- 875 mg/125 mg P.O. q12h
- Pen G
- 4 million units IV q4h

- Ampicillin–sulbactam (Unasyn®)
- Dosage 1.5 or 3 g IV q6-8h

- Nafcillin (Unipen®)
- 500 mg IV q4h

- Dicloxacillin
- 250–500 mg P.O. q6h

- Piperacillin–tazobactam (Zosyn®)
- Dosage 1.5 or 3 g IV q6-8h

- Ertapenem (Invanz®)
- Dosage 1 g IV daily

- Cephalexin (Keflex®)
- 500 mg P.O. q6h
- Cefazolin (Ancef®)
- 1–2 g IV q8h

LINCOSAMIDES

- Clindamycin
- 600–900 mg IV q8h
- 300–450 mg P.O. q6h

MACROLIDES

- Erythromycin
- 250–500 mg P.O. q6h

- Clarithromycin (Biaxin®)
- 500 mg P.O. bid
- Azithromycin (Zmax®, Zithromycin®)
- Z-pack: 250 mg P.O. bid first day, 250 mg P.O. qday x 4 days
- May be prescribed as directed by doctor for a longer or shorter course.

FLUOROQUINOLONES

- Moxifloxacin (Avelox®)
- Oral 400 mg P.O. q24 hours
- IV: 400 mg P.O. q24 hours
- Levofloxacin (Levaquin®)
- 500 mg P.O. q24 hours

Antibiotic Prophylaxis

2007 American Heart Association now only recommends antibiotic prophylaxis in (Table 3.3) the following conditions:

1. Patients with prosthetic heart valves or who have had a heart valve repaired with prosthetic material.
2. A history of endocarditis.
3. A heart transplant with abnormal heart valve function.

Table 3.3 AHA guidelines for antibiotic prophylaxis for prevention of subacute bacterial endocarditis

Antibiotic regimen for dental procedures (30–60 minutes prior to procedure)			
Clinical scenario	Antibiotic	Adult	Pediatric
Not allergic to PCN and able to take oral fluids	Amoxicillin	2 g	50 mg/kg
Not allergic to PCN and unable to take PO	Ampicillin	2 g IM/IV	50 mg/kg IV/IM
PCN Allergic	Clindamycin Cephalexin Azithromycin Clarithromycin	600 mg 2 g 500 mg 500 mg	20 mg/kg 50 mg/kg 15 mg/kg 15 mg/kg
PCN allergic and unable to take PO	Clindamycin Cefazolin Cetriaxone	600 mg IV/IM 1 g IV/IM 1 g IV/IM	20 mg/kg IV/IM 50 mg/kg IV/IM 50 mg/kg IV/IM

4. Certain congenital heart defects including:
   - Cyanotic congenital heart disease (birth defects with oxygen levels lower than normal) that has not been fully repaired, including children who have had a surgical shunts and conduits.
   - A congenital heart defect that has been completely repaired with prosthetic material or a device for the first six months after the repair procedure.
   - Repaired congenital heart disease with residual defects, such as persisting leaks or abnormal flow at or adjacent to a prosthetic patch or prosthetic device.

2015 ADA guidelines for prosthetic joints: No association between dental procedures and prosthetic joint infections. No longer recommended, but clinicians may use their judgment after patient input in decision-making.

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Dental Implantology

4

Waldemar D. Polido, Akshay Vij, Robert Reti, Sherrill Fay, and Gregg W. Hosch

Dental Implant Basics

- An implant system can be divided into an endosseous part, a transmucosal section, and a prosthodontic interface.
- A dental implant is the surgical component that interfaces mechanically and biologically with the bone to support a dental prosthesis.
- Most commonly used implants are screw root forms that are threaded into a prepared osteotomy, reliant on threads for initial stability via mechanical retention.
- Implant body can be divided into a crest module, body, and an apex (see Fig. 4.1).
- Implants can be designed with the neck of the implant supra-crestal (tissue level), crestal (bone level), or sub-crestal.
- Supra-crestal (soft tissue level) implants are favored to reduce marginal bone loss or saucerization around implants when compared to butt-joint bone level implants, by moving the neck above the bone and preventing bacterial colonization of the microgap. With the advent

For this section it is important to know the implant system *you use*. Know the sizes, drilling sequence, surface modification, thread distance, and abutments. It is not uncommon to be told by the examiners that there is no financial barrier to implant therapy in these cases. Make sure that you offer a defendable treatment plan based on how you would manage a case and not based on what you think the examiners want to hear.

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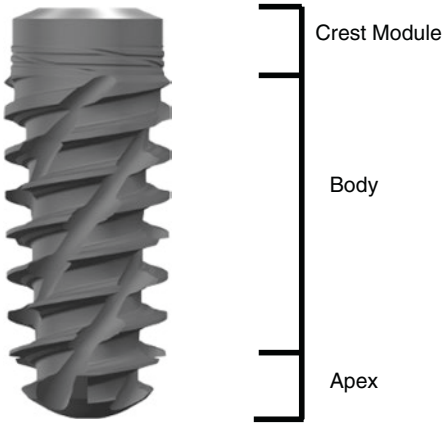

Fig. 4.1 Dental Implant. Straumann BLX Dental Implant. (Image Courtesy of Straumann)

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of platform switching, and internal conical connections, that can also be obtained with bone level or below the bone level implants.

Length

- Important for implant primary implant stability, and influences immediate loading. Once secondary implant stability has been achieved (osseointegration), length is not as important. (Side note: thread pitch, drilling sequence, and bone quality also play a great role in gaining stability prior to osseointegration) [1].
- Increases surface area of bone-implant interface.
- Most stress of implant at first 5 mm making diameter important in stress reduction.

Diameter

- Larger diameter implants increase the surface area of bone-implant interface.
- Disperses forces in poor bone, thereby reducing risk of overload.
- Reduces the magnitude of force to system when used as part of bridgework or cantilever.
- Increased diameter can allow for better emergence profile for larger crowns. In modern platform switch implants this is not the case, since the choice of prosthetic components is independent of the implant diameter.
- Increased diameter of crest module size decreases the risk of implant fracture and prosthetic component fracture.
- Concern for stress shielding from wide diameter implants leads to bone atrophy due to lack of strain transfer to the bone.
- Increased diameter implants require a larger drilling, thereby reducing bone thickness around the implant. There is a current trend to not use wide body implants (>5 mm diameter) in order to preserve more alveolar bone.
- Narrow (Reduced Diameter) implants are indicated in the anterior region of the maxilla or mandible. Narrow implants are more prone to implant fracture and internal connection damage, especially when placed in the posterior region. When placed in the posterior region, they shoud be splinted with other(s) implants. Modern alloys (TiZi) provide additional mechanical resistance to narrow implants, increasing survival rates.

Shape

Parallel wall:

- Provides increased surface area.

Tapered:

- Provides stability by creating pressure on cortical bone, which is good for poor bone quality sites.
- Allows compression in poor bone quality sites.
- Reduced apical width allows for placement in constricted sites.
- Reduced overall surface area increases with taper.

Implant Surface Modifications

- Hydroxyapatite (HA) – HA-coated implants are no longer used as the processing methods convert HA to tricalcium phosphate which is rapidly absorbed and is easily colonized with bacteria. Additionally, there have been problems with delamination of HA.
- Micro-rough surfaces 0.5–2.0 microns (minimally rough 0.5–1, intermediately rough 1.0–2.0, and rough 2.0–3.0 microns) create peaks and depressions in the implant to increase surface area. Roughened surfaces can be created by acid etching with such chemicals as sulfuric, hydrochloric, and hydrofluoric acids. Spraying the implant surfaces with titanium oxide, hydroxyapatite, and aluminum oxide is another option. Micro surface roughness causes an increased implant to bone surface area, clot retention, aids in earlier osseointegration, and leads to harder and stronger bone around implants by increasing mRNA expression of osteonectin and osteocalcin [2].
- Electrowetting – wettability of implants important to improve plasma protein adherence and mesenchymal cell adherence and differentiation. Many methods are available, but commonly fluoride and magnesium ions are used. Some manufacturers package implants in saline.

Crest Module

- Microthreads – preserves both bone and soft tissue around cervical portion of implant fixture by dissipating forces around crest. Can facilitate higher incidence of peri-implantitis

due to plaque retention if the implant is exposed to the oral cavity.
- Microgap – connection between implant and abutment.
- Anti-rotational component – platform of the crest module has an anti-rotational feature to retain prosthetic component. This can be a platform such as an external hex (external connection) or within the implant body itself (e.g., internal hex, Morse taper, octagon, internal grooves, or pins).
- External connection – connection to implant that is superior to coronal portion of implant creating a butt joint connection. Have higher prevalence of screw loosening, rotational misfit, and microbial penetration. Classic example is the External Hexagon connection.
- Internal connection – internal connection to implant, seen in most modern implants. Can have parallel walls (Internal Hexagon) or morse cone type (connical connection). Conical connection preferred vs. flat connection as it can disperse load and prevent microgap elongation on function with fluid invasion. Connical connections have Improved microbial seal, reduced screw loosening, increased joint strength, and increased platform switching abutment options.
- Platform switching – it is an horizontal offset between the implant connection and the cervical area of the abutment. This method can help to reduce crestal bone loss using a narrower restorative abutment compared to the crest module which leads to a more superior position of the epithelial attachment around the neck of the implant. This technique also medializes the implant abutment interface, which redirects stress from the crestal bone [3]. Inflammatory infiltrates are positioned away from the crestal bone leading to less bony destruction/loss [4, 5].

Materials
- Titanium is a metal that presents low-weight, high-strength/weight ratio, low modulus of elasticity, excellent corrosion resistance, excellent biocompatibility, and easy shaping and finishing.
- Most commonly used: Grade 4 pure titanium (cpTi), titanium-zirconium alloy, and titanium-6 aluminum-4 vanadium (Ti 6AL-4V).
- Biocompatibility due to surface dioxide layer that forms almost instantaneously upon exposure to air (2–10 nm by 1 second). Important role in corrosion resistance, biocompatibility, and osseointegration. This oxide layer is composed of titanium dioxide (TiO_2).
- Zirconia: Implants produced with zirconia are biocompatible, bioinert, radiopaque, and have a high resistance to corrosion flexion and fracture. They are typically considered "non-metallic" and are white in color.

Criteria for Implant Success
- Immobile when tested clinically.
- No radiographic evidence of peri-implant radiolucency.
- Vertical bone loss is less than 0.2 mm annually after the first year of service of implant.
- Implant performance is characterized by an absence of persistent or irreversible signs and symptoms of pain, infection, neuropathy, paresthesia, violation of mandibular canal.

(These aforementioned success criteria were based on radiography, clinical signs, and symptoms. There are other factors today that we would take into account to establish implant success. New parameters to take into account include esthetics, soft tissue integrity/appearance, patient satisfaction, and prosthodontic parameters) [6].

Important Numbers to Know About Implants
- Distance of 1.5 mm between implants and natural teeth to allow for lateral biologic width. Violation leads to bone loss around implants and adjacent structures.
- Normal bone loss is <1.5 mm for the first year and 0.2 mm per year after (These numbers do not take into account the prosthetic construct used. For example, an implant supported FPD where forces are evenly distributed may have less bone loss per implant in the entire construct. Platform switching has also lessened the microbial bioburden that contributes to overall bone loss).
- A minimum distance of 3 mm between two implants must be adhered as to maintain interproximal bone height which provides room for restorative components.
- Minimum Distance of 1 mm of bone between implant and buccal/lingual wall. In the aes-

thetic zone, 2 mm posterior to buccal wall is desired for emergence profile and to preserve the buccal bone.
- Minimum Distance of implant apex is 1 mm from nasal floor.
- Minimum Distance of implant apex is 2 mm above the inferior alveolar nerve.
- Implant body 5 mm in front of mental foramen.
- Head of bone level type implant should be 2–3 mm below gingival margin of planned crown to allow space for emergence profile.
- Each 0.25 mm increase in diameter yields a 10% increase in surface area.
- Classic integration timelines for smooth surface implants are 3 months for the mandible and 6 months for the maxillae. Modern implants surface treatments (SLA) can decrease time to as early as 6–8 weeks for conventional loading.
- Ensure that drills are sharp and use new drills often, especially for dense bone.
- Thermal necrosis during drilling occurs above temperatures of 47 °C. Keep RPM to 2000 or less and ensure pumping action during drilling to allow water to reach base of osteotomy.
- Minimal intra-arch space of 5 mm for cement retained and 8 mm for screw retained for single crowns. More inter-arch space may be needed for overdentures or fixed hybrid prosthetics.
- Minimal interarch clearance for a bar attachment is 12 mm.
- Implants in a growing child will lead to a submerged implant, that is more palatal/lingual, out of occlusion and deep into alveolus, secondary to facial and dentoalveolar growth adjacent to the implant. Implants should be placed after confirmation of growth cessation by following growth indices for 1 year such as hand-wrist or spine radiography. Some authors recommend a minimum age of 15 for females and 18 for males. Literature shows reports of adult patients with continous alveolar growth, leading to vertical defects around the implant area.
- Most implant drills have tapered tips of 0.5 mm beyond their established measurement.
- In comparison to a medical grade CT, cone beam computed tomography (CBCT) uses about 2% of radiation dose.
- 40–60% of expected bone loss occurs during the first 36 months after the tooth is extracted.
- Contact point to crest of bone with presence of papillae [7]:
  - 3 mm – 100%
  - 4 mm – 100%
  - 5 mm – 98%
  - 6 mm – 56%
  - 7 mm – 27%

Osseointegration

- Process of which there is a bone to alloplastic interface without the interposition of non-bone tissue, which is clinically asymptomatic and is maintained in bone during functional load (based on electron micrographic findings).
- Classic definition of osseointegration by Branemark: osseointegration is the direct, structural, and functional connection existing between ordered, living bone and the surface of a functionally loaded implant.
- Primary stability: mechanical stability achieved at the moment of implant placement. Depends on bone quality (density), shape of implant, and adequacy of surgical technique. Can be optimized when these three factors are considered and technique is adequate for existing type of bone and implant placed.
- Secondary stability: biological stability, achieved after bone healing (osseointegration). Influenced by bone quality, implant surface, overall health of patient, and loading protocols.
- Two mechanisms of osseointegration: (1) distance osteogenesis occurs from existing bone and blood supply and (2) contact osteogenesis (de novo bone formation) from osteogenic cells.

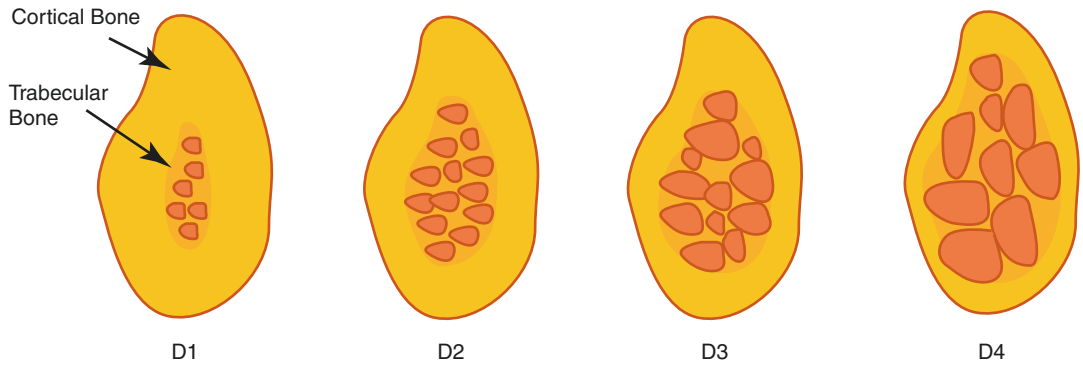
Bone Quality

- Implant survival is multifactorial but arch location plays a vital role. Most failures occur in softer bone.
- Bone density is directly correlated to bone strength.

- Lekholm and Zarb, in 1985, classified based on the ratio of cortical and cancellous bone using radiographs [8].
  - Type 1 bone is composed mostly of compact bone.
  - Type 2 is mostly a compact bone surrounded by a core of trabecular bone.
  - Type 3 is composed of thin layer of cortical bone surrounded mostly by trabecular bone.
  - Type 4 is composed of thin layer of cortical bone surrounded by a core of low-density trabecular bone.
- In 1998, Misch described bone densities in the edentulous maxilla and mandible based on macroscopic specimens based on cortical and trabecular bones. In 1999 Misch updated his classification to include bone density independent of region of jaw while taking into consideration Hounsfield units. Classes range from D1 to D4, with D1 being the most dense (see Fig. 4.2) [8].
- Misch classification: Bone elasticity increases from D1 to D4, leading to increased micro strain and implant mobility leading to failure. The cortical cancellous ratio decreases from D1 to D4.
- Crestal strain and stress transfer increase with decreasing bone density.
- As bone density decreases, it is prudent to treatment plan longer/wider implants with maximization of the number of implants and designs, which increase surface area.

Testing for Implant Stability

1. Insertion torque of an implant should ideally be 35 Ncm or more. Over Torquing >80 Ncm may impair implant healing.
2. Absence of clinical mobility with 500 g in any direction.



TYPE	Description	Location	Hounsfield CT
D1	Dense cortical bone	A. Mandible	>1250
D2	Thick cortical and course trabecular	A. Mandible P. Mandible A. Maxilla	850–1250
D3	Thin cortical compartment with dense trabecular	A. Maxillae P. Maxilla P. Mandible	350–850
D4	Fine trabecular, extremely thin cortical	P. Maxilla	150–350

Fig. 4.2 Misch bone density classification. (Modified from Torabinejad et al. [21])

3. Implant stability quotient (ISQ) – a resonance frequency analysis with a number between 1 and 100. High stability, >70 ISQ; medium stability, between 60 and 69 ISQ; and low stability, <60 ISQ.

Loading Protocols

1. Immediate loading – prosthesis is delivered up to 7 days after implant placement.
2. Early loading – prosthesis is delivered 6–12 weeks after implant placement. Some implant surfaces consider 8 weeks as conventional loading.
3. Conventional loading – prosthesis is delivered after osseointegration is achieved. Classic period is 3 months for mandible and 4–6 months for maxilla.

Pre-surgical Workup

CC/HPI: Patient's desires or concerns, prior prosthetic reconstructive efforts, expectations, how long has the patient been without teeth, and causes of tooth loss.

REMEMBER: the patients want teeth and not implants. Make sure that all surgical procedures follow a restoratively-driven plan, in order to ensure the best possible restorative outcome.

Medical History/Medication History/Surgical History

- Smoking – reduced success rate, about 6.5–20% lower than in nonsmokers [9].
- Diabetes – need longer healing times to reach stability [10, 11].
- Osteoporosis – studies have found similar rates of implant failure in patients suffering from osteoporosis vs patients with normal bone densities. Some weak evidence reduced bone healing and may consider longer healing times [12–14]. Higher risk for failure of bone grafting procedures in this patient population [15].
- Oral bisphosphonates – AAOMS recommends a drug holiday of 2 months, for patients taking oral bisphosphonates, prior to surgery. The bisphosphonate should be held until osseous healing has occurred [16].
- Avoid implants in patients using IV bisphosphonates or antiangiogenic drugs.
- IV bisphosphonates or antiangiogenic drugs for cancer.
- Denosumab – no studies to support discontinuation at this time [16].
- Radiation of the head and neck: consider HBO if necessary (>60 Gy); failure rates similar with the advent of newer radiation protocols [17].
- Parafunctional habit – consider wider diameter or stronger alloy implants. Judicious planning of designing load-sharing prosthetics, occlusal adjustments of prosthetics, and longer healing time for loading bearing bone formation may help counteract the destructive forces of parafunctional habits.
- TMD complaints –assess for placement and length of procedure.
- Debilitating disease – e.g., rheumatoid arthritis, scleroderma, or Parkinson's disease that may cause xerostomia due to medications and make dental care difficult to maintain. Consider home assistance and prosthetic type, fixed vs. removable.

Evaluation

- Head and neck exam as expected on all patients.
- Lip support/gingival display on repose and animation. Short upper lip, high smile line, or hyperanimation may reveal artificial teeth and flange.
- Width of remaining ridge. If edentulous a prosthesis with flange may be desirable vs. fixed crown and bridge to provide lip support and better esthetic outcome.
- Papillae position and gingival margins of adjacent teeth.
- Condition of the oral cavity and restorability of teeth to determine best prosthetic type.
- Palpation of muscles of mastication and observe for hypertrophy of masseter, concern for parafunctional habit. Assess for wear pattern of teeth, bruxism, or occlusal interferences.
- Occlusion – assess for angle classification, scissor bites, and cross bites and how these occlusions may affect implant success. May

create prosthesis design issues or cantilevers. May require orthognathic procedures.
- Inter-incisal opening ability to access site of implant.
- Periodontal health/oral hygiene. Periodontal probings to ensure healthy cervical margins of adjacent teeth. Higher failure rate in those with poor periodontal status and poor hygiene (should be controlled before dental implant placement).
- Gingival biotype: assess visibility of probe through gingival sulcus. Thick biotype associated with greater soft tissue stability, less recession, and is more resilient to oral flora.
- Keratinized tissue – 2 mm or more of keratinized gingivae reduces gingival inflammation, increases implant survivability, and reduces marginal bone loss.
- Interarch crown height space, ideal 8–12 mm for fixed restoration or 12 mm or more for bar connections.
- Ridge contour – bone loss may push prosthesis palatal/lingual if restored with implant which will lead to extensive ridge overlap or food trap. Bone grafting may be indicated.
- Articulated diagnostic models aid in planning with diagnostic wax-ups, stent fabrication, and easier measurements such as for prosthetic space.
- Photos of patient in repose, full smile, lateral views for implants in the aesthetic zone.

Radiography

- Overall use is to rule out pathology, assess bone quality, dental relationships, and proximity to vital structures.
- Periapical films – may use for initial evaluation, intraoperative assessment, and postoperative monitoring. However, periapical films lack reproducibility and it is often difficult to assess the proximity of vital structures (best indicated to observe crestal bone around adjacent teeth especially in the aesthetic zone).
- Orthopantogram – used as a generalized scout film that allows the visualization of vital structures, bone quality, and the presence of pathology. A major drawback is magnification. Vertical magnification is more uniform than horizontal magnification and can be overcome by radiographic markers of a known size.
- Cone Beam Computed tomography – allows for accurate assessment of distances to vital structures. Can view the height and width of ridge to plan for bone graft needs. Software allows for easier planning with dental implant database. Digital workflow improves collaboration and interaction with prosthodontic plan. Involves merging and superimposition of DICOM and STL files (created from intraoral or model scanning) data to create a fully guided stent for guided surgery. For the vast majority of implant cases, a CBCT is the indicated imaging modality.
- Hounsfield unit assessment gives objective measures of bone density in region and is based on medical CT imaging. CBCT imaging utilizes a gray value and is not directly correlated to Hounsfield units.

Implant Complications of Implant Placement

Failure to Integrate/Fibrous Connection Likely due to lack of primary stability, type IV bone, inadequate preparation of osteotomy (over-preparation of osteotomy, excessive torque when placing implants in type 1 bone, poor irrigation leading to bone necrosis and infection). Treatment is to remove the implant and assess the need for graft for future implant placement or if ridge allows preparing the site for a wider or longer implant. Soft tissues recession may require additional soft tissue graft or place implant in a secondary procedure.

Encroachment to IAN Canal Patient may express discomfort as though they experienced an electrical shock, or a rush of blood may come through the osteotomy site. Verify implant position with radiography (3D imaging preferred). The implant should be removed immediately if noted to encroach upon the nerve. In theory removal allows psychological therapy for the patient, pathway for escapement of debris and irritants, ease for future nerve repair, and takes pressure of the nerve (if not severed). No bone graft should be placed into the site. Steroid application to the injury site and high-dose steroids orally for a week may help reduce

neuropathy. NSAIDs such as ibuprofen 800 mg q 8 h for 3 weeks also have been recommended in the literature. The benefit of steroids and NSAIDs is questionable. Neurosensory testing is evaluated serially. If the patient has anesthesia/dysesthesia for 3 months or hypoesthesia for 4 months, then consider microneurosurgery. If no evidence of encroachment with patient complaining of a neurological disturbance, then one cannot rule out injection injury. Consider removing implant.

Sinus Penetration Implant penetration into maxillary sinus of 1–2 mm has been shown to be fully covered with sinus membrane and partially by bone in animal studies. No difference in stability is noted. Penetration of 3 mm or more showed exposure into the sinus cavity without any coverage.

Mandible Fracture Usually occurs late once implants are loaded but can also happen when placing implants in extremely atrophic mandibles. Recommended at least 6 mm in vertical height and width required for implant placement. If there is not enough bone stock, then a bone graft is indicated. Treatment follows basic trauma principles. Treatment of the edentulous mandible may require a large reconstruction plate with consideration for bone grafting.

Excessive Countersinking May cause excessive bone loss and difficulty with connections. May also result in loss of primary stability.

Peri-implantitis Infectious disease surrounding a load-bearing dental implant with features of bone loss and inflammation of the soft tissue. Associated with gram-negative anaerobes including *P. gingivalis, P. intermedia, and Aggregatibacter actinomycetemcomitans.* Symptoms include bleeding on probing, bone destruction, suppuration on probing, erythema, hyperplasia, probing depth >5 mm, mobility of implant, and swelling. Pain is normally only present in the setting of acute infection.

Adequate soft tissue management (plan to increase or maintain thick keratinized tissue around neck of implant) can reduce the chance for periimplantitis.

Treatments

1. Local debridement – exposure and cleaning with instrument softer than titanium. Consider rubber cup polisher with paste, plastic scalers, abrasive air powder treatment, and interdental brushes.
2. Decontamination – 40% citric acid with a pH of 1 for 60 seconds, chlorhexidine, tetracycline (50 mg/ml saline for 2 minutes), or application of local antibiotics (e.g.,tetracycline granules), Er:YAG or CO_2 laser or 3% H_2O_2.
3. Surgical – open flap combination of debridement and decontamination with allograft/autograft with membrane.
4. Removal of implant.

Sublingual Gland Injury/Sublingual Artery Palpate ridge or CBCT to visualize the sublingual fossa. Injury can be caused by perforation through the lingual cortical plate. Ranula or bleed can occur. Evaluate floor of mouth and be mindful of the airway. Sublingual artery bleed can be managed by exploration with cautery/ligation (Consider treatment in the hospital setting for airway protection). If ranula develops, consider removal of sublingual gland.

Bone Augmentation

Bone Grafting

- Heals by creeping substitution – a process by which osteoclasts resorb bone creating new vascular channels with osteoblastic bone formation resulting in new haversian systems. Laying down new bone and subsequent resorption of old bone.
- Osteogenic – transfer of osteocompetent cells for de novo bone formation, e.g., autografts.
- Osteoinduction – bone formation by stimulation of host mesenchymal cells to differentiate, e.g., allograft, bone morphogenic protein.
- Osteoconduction – providing scaffolding for new bone formation propagated by native bone. Does not contain proteins or cells, e.g., xenograft.

Bone Graft Materials

Autogenous – composed of tissue from the same person

- Osteogenic, osteoinductive, and osteoconductive.
- Gold standard.
- Disadvantage is a second surgical site.

Allogeneic – grafts taken from another individual of the same species but different genotype

- Osteoinductive and osteoconductive.
- Strict screening for infections, malignant neoplasm, degenerative bone disease, hepatitis B or C, STDs, autoimmune disease, or other diseases that may affect bone quality.
- Comes as a mineralized freeze-dried bone allograft (FDBA) or demineralized freeze-dried bone allograft (DFDBA). Both provide type 1 collagen which is the exclusive organic component of bone.
- Methods to decrease antigenicity – freeze-drying, irradiating, dry heating.

Xenograft – grafts taken from another species

- Osteoconductive.
- No organic component.
- Treated by sintering at 900 °C or high alkaline solution. Risk of prion transmission (e.g., bovine spongiform encephalopathy) is theoretical.
- Hydroxyapatite crystalline structure allows for ingrowth of vessels and migration of osteogenic cells.

Recombinant Human-Bone Morphogenetic Protein-2 (BMP)

- Part of transforming growth factor β superfamily.
- Recombinant DNA technology in Chinese ovarian hamster cells allows for transcription and collection of non-contaminated protein.
- Water soluble, requiring a collagen type 1 carrier (acellular collagen sponge) for slow release. Requires 15 minutes of absorption.
- Concentration of 1.5 mg/cc mixed with sterile water (do not substitute with normal saline as too hypertonic).
- Chemotactic for preosteoblasts and stem cells as well induces expression of VEGF by osteoblasts.
- Only on label use is currently for sinus augmentation or alveolar ridge reconstruction.
- Will have extensive edema due to influx of fluid and cells from the chemotactic and neovascularization activities of BMP.
- Allow healing of 6 months prior to implant placement.
- Contraindications: (1) pregnancy, (2) allergy to rhBMP or type I bovine collagen, (3) active infection at recipient site, (4) active or history of malignancy at site, and (5) skeletal immaturity.
- Postoperative steroids and icing of tissue may reduce the intensity of swelling.

Platelet-Rich Plasma (PRP)

- Platelet-derived growth factors act as a mitogen (encourages cell division) and encourage osteoid production and endothelial cell replication.
- PRP is a blood clot that is highly concentrated with platelets, about 1 million platelets/μL.
- Alpha granules in platelets secrete the growth factors that bind to transmembrane receptors to induce its effect, initiating a faster initial cellular response.
- Collection tube contains citrate dextrose as anticoagulant, which works by binding to calcium.
- The platelets are spun down either in two spins (separation spin followed with a concentration spin) or some manufacturers offer single spin units.
- Activated via the addition of $CaCl_2$ and thrombin.
- Utilized in soft and hard tissue grafting.

Platelet-Rich Fibrin (PRF)

- Platelet-rich fibrin (PRF) was developed as an improved formulation of the previously utilized platelet-rich plasma (PRP), to serve as a three-dimensional scaffold to biologically enhance healing.
- This new approach is based on the concepts that were introduced over a decade ago con-

sisting of a platelet concentrate without the use of anticoagulants.
- PRF is obtained simply by centrifugation without anticoagulants and is therefore strictly autologous.
- This fibrin matrix contains platelets and leukocytes as well as a variety of growth factors and cytokines including transforming growth factor-beta1 (TGF-β1), platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), interleukin (IL)-1β, IL-4, and IL-6.
- These factors act directly on promoting the proliferation and differentiation of osteoblasts, endothelial cells, chondrocytes, and various sources of fibroblasts.

Classification of Cawood and Howell

- Class 1: Dentate.
- Class 2: Immediately post-extraction.
- Class 3: Well-rounded ridge, adequate in height and width.
- Class 4: Knife-edged ridge, adequate in height and inadequate in width.
- Class 5: Flat ridge, inadequate in height and width.
- Class 6: Depressed ridge with varying degrees of basal bone loss that may be extensive but follows no predictable pattern.

Maxillary Sinus and Grafting

Anatomy

- Schneiderian membrane is 0.13–0.5 mm thick and is composed of respiratory epithelium.
- Paired sinuses with a mean size of 15 ml per sinus. Width ~2.5 cm; height ~3.75 cm.
- Sinus ostium is located in superior medial sinus wall (halfway in the A-P distance of the sinus just below the orbital floor). It is usually 25–35 mm above the antral floor. It opens up to the middle meatus via the infundibulum.
- Underwood's septa – fine bony projections from the floor of the maxillary sinus, which can cause two or more compartments and complications during sinus grafting. One septum is present in about 90% of patients.

Table 4.1 Sinus lift methods in relation to alveolar bone height

Technique consideration based on remaining alveolar ridge height (based on 10 mm length implant)	
≤4 mm	Lateral approach and delayed implant placement
>4 mm	Lateral approach/summer approach with simultaneous implant placement
6-8mm	Summer osteotome technique/internal lift (≤2 mm) with immediate implant placement
+10+	Placement of implant

- Vascular supply to maxillary sinus is from branches of the maxillary artery:
  1. Posterior superior dental artery
  2. Anterior superior dental arteries
  3. Greater palatine artery
  4. Lesser palatine arteries
  5. Lateral and posterior nasal branches of the sphenopalatine artery
- The venous flow occurs via the facial vein, the sphenopalatine vein, and the pterygoid plexus.

Preoperative Evaluation

- PMHx – recent upper respiratory tract infection, history of sinusitis or chronic sinus disease, sinus or nasal surgeries, otitis media, and smoking. Chronic steroid use may thin out membrane, making it more fragile during sinus procedures. Reduction of sinus volume may lead to worsening of sinus symptoms. Smoking has not been shown to reduce sinus lift viability, but does affect implants. If there is acute sinus issue, delay until resolves.
- Adequate inter-arch space.
- Adequate remaining alveolar bone (see Table 4.1).
- Discussion with restorative doctor for stent/surgical plan.
- For Summer's technique, Meniere's or Meniere's-like diseases are contraindications.
- CBCT/CT scan – rules out pathology and identifies remaining width and height of alveolar ridge, sinus topography including septae, air fluid levels, and presence of polyps.

- Antral pseudocyst/mucocele – should be removed/aspirated 6 months prior to lift and re-evaluated for recurrence. A relative contraindication.

Lateral Approach (Also Known as the Tatum's Technique)

1. Local anesthetic for hemostasis and insufflation.
2. Incision should be palatal to the alveolar ridge to reduce risk of postoperative fistula if no immediate implant is planned. A crestal flap should be utilized if an implant is planned at time of augmentation.
3. Ostectomy – thin out lateral sinus wall exposing sinus or window outline (quadrilateral ostectomy) to act as superior bony roof. The inferior extent should be 1 mm superior to the floor. Many surgeons now use piezosurgery to reduce perforation rate from 30% with conventional burs to about 7%.
4. Elevation of sinus membrane with piezosurgery with non-cutting blade along perimeter or sinus curettes. If patient is awake, asking them to inhale allows visualization of adherent membrane. Check for perforations.
5. Place graft material medially to ensure adequate bulk toward the medial aspect of the sinus cavity. In simultaneous implant placement, after lifting the sinus membrane and preparing the implants, a graft should be placed first, then implants, and then more graft material. Materials include autogenous (gold standard, good for larger grafts), allograft, xenograft, or alloplastic. Non-autogenous grafts have similar success rates, only a small percentage lower.
6. Placement of absorbable membrane at bony window.
7. Suture to watertight closure of flap.

Summer's Technique (Transalveolar, Vertical or Internal Approach)

1. Local anesthetic for hemostasis and insufflation.
2. Crestal incision to expose ridge.
3. Start osteotomy with 2 mm twist drill to 1 mm below sinus floor.
4. Guide pin placed and PA taken to ensure subsinus ideal position.
5. Osteotomies of different gauges are now malleted 2 mm higher than native bone using up to appropriate gauge of planned implant.
6. Test with Valsalva and hand mirror to evaluate sinus integrity.
7. Placement of autograft/allograft and work into sinus space created to dome sinus.
8. Placement of implant.
9. Repair incision with sutures.

Postoperative Management

- Sinus precautions: no nose blowing for 2 weeks, sneeze with mouth open, no pressure changes such as scuba diving and use of straws or wind instruments.
- Antibiotic with sinus coverage (e.g., amoxicillin 500 mg q 8 h × 7 days), oxymetazoline 0.05% q 12 h for 3 days, saline nasal spray PRN congestion, pseudoephedrine 30 mg q 6 h PRN congestion.
- Allow 6 months for graft consolidation.

Complications

Sinus Perforation If perforation is 2–3 mm, will likely self-repair by folding over or blood clot formation, consider collagen wound dressing. If perforation is 5–10 mm, consider bioabsorbable collagen membrane. If larger (10 mm >), assess possibility of using collagen membrane to completely cover graft. If not possible, abort surgery and return in 3 months. At this point, the sinus will be thicker in the area of the perforation.

Antral Septae Make two windows and treat as two compartments or osteotomize septum along sinus floor.

Bleeding Pack sinus with epinephrine-soaked gauze. Enlarge opening and attempt to visualize bleeder for cauterization. Clamping vessel may cause further damage and increased bleeding.

Infection/Acute Sinusitis Common sign is swelling over lateral window site with pain and localized tenderness. Antibiotic with respiratory flora coverage. If no spontaneous drainage, surgical drainage is indicated with consideration for graft removal.

Graft Exposure Gentle daily normal saline irrigation and chlorhexidine rinses.

Blockage Ostium Caused by overfill or migration of particles, infection, or inflammation. Assess extent of sinusitis with imaging. Place on steroids and antibiotics. If no improvement, consult with ENT.

Vertigo Usually resolves on its own. Attempt Epley maneuver. Anti-vertigo drugs like meclizine 50 mg PO BID for symptomatic treatment.

Harvesting Techniques of Autografts from Oral Cavity Sites (Please See Reconstruction Chapter for Other Bone Harvesting Techniques)

Autogenous bone grafts are contraindicated if metabolic bone disease, previous radiation treatment, local infection or pathology is present in donor site area.

Mandibular Symphysis

- Can be performed under local anesthesia.
- It is important to open recipient site first to ascertain appropriate graft size.
- Best to harvest lateral to midline, at least 5 mm below apex of canine. Can be done bilaterally, if larger graft is required. Preservation of anterior chin contour is recommended.
- Some authors recommend grafting the harvest site with allograft/xenograft to maintain chin contour.
- Allow 5 months for integration.
- Second graft can be taken no sooner than 10 months from initial harvest [18].

Mandibular Symphysis Surgical Approach

1. Infiltration of local with vasoconstrictor.
2. Incision from canine to canine, through mucosa 1 cm below mucogingival junction, then through mentalis muscle and periosteum.
3. Exposure of symphysis using periosteal elevator, do not completely remove the attached mentalis muscle.
4. Outline planned osteotomy with saw/bur of choice, ensuring to be 5 mm inferior to root tips and 5 mm from the inferior border, entering into cancellous layer. Preferred area to remove bone is below lateral incisor and canine, preserving the midline region.
5. Remove graft, a curved chisel or fine osteotome may aid in its harvest. May harvest additional cancellous bone with curettes.
6. For large grafts, place bone substitute to fill donor site.
7. Close in layers with 4-0 slow resorbing sutures deep and with 3-0 resorbable sutures for mucosa.
8. Pressure dressing over chin.

Ramus Graft

- Can be done under local anesthesia.
- Convenient to be done with third molar removal.
- Harvest the external oblique ridge. Provides mainly cortical bone with minimal marrow.
- Allow 5 months for integration.

Ramus Graft Surgical Technique

1. Local anesthetic with vasoconstrictor.
2. Open and prepare graft site to obtain graft size.
3. Sharp incision along the external oblique ridge from the level of the maxillary occlusal plane to the distal of the mandibular molar.
4. Periosteal elevator to reflect periosteum and temporalis tendon.
5. Outline graft site with saw/bur extending just into cancellous bone.
6. Make osteotomy using saw/bur, ending 5 mm distal to last molar in mandible.
7. Remove graft with periosteal or osteotome.
8. 3-0/4-0 resorbable suture to close. Collagen plug or dressing can be applied to area before closing.

Maxillary Tuberosity
- Older patients will have more fatty marrow.
- Ease of harvest and can be done under local anesthesia.
- Contraindicated if highly pneumatized sinus or sinus infection is present.
- Risk of sinus exposure if over aggressive harvesting.

Maxillary Tuberosity Technique
1. Local anesthetic to area.
2. 3-corner full thickness flap with a distal release.
3. Rongeur used to remove bone, or chisel to gain a thin segment of cancellous bone.
4. Close with 3-0 resorbable suture.

Complication of Block Grafts

Exposure of Block Graft Overall a poor prognosis, inform patient of this. Opening the wound and attempting to suture again will lead to increased microbiological load, large dehiscence, and possible flap necrosis. One protocol calls for chlorhexidine rinses for 4 weeks with debridement/reduction of the graft. Partial or complete survival of graft is low.

Screw Exposure Decreasing bone volume is expected up to 25%. The position of the screw will remain constant as the tissue collapse. Patient asked to keep screws clean with chlorhexidine mouth rinses and debridement with tooth brushing.

Membrane Exposure Titanium membranes commonly exposed and are to be treated with chlorhexidine gel (0.5%) or rinses (0.12%). Membranes of e-PTFE need complete removal with graft as the membrane is quickly vegetated with microorganisms. Resorbable membranes will break down quickly with resorption of the bone.

Mobility of Graft During implant placement if graft moves after screw removal, the graft is not properly integrated. Remove covering soft tissue, provoke bleeding, and fragment should be resecured with screws and allowed to heal further for 4 months.

Ridge Expansion Techniques

Interpositional Graft/Sandwich Graft

Commonly used in esthetic zone, anterior maxillae, but can be used in any part of edentulous ridge.

- Also known as sandwich graft, as bone is "sandwiched" between basal and osteotomized bone.
- Blood supply is maintained by pedicle on lingual/palatal.
- Vertical bone height of 5 mm can be expected, limitation is stretch in pedicle.
- Bring tissue with the osteotomized bone.
- The final position of the bone tends to be more palatal/lingual.

Technique
1. Local with vasoconstrictor.
2. Elevate flap with a horizontal component in vestibule, vertical limits at papillae of adjacent papillae.
3. Divergent wall osteotomies to allow for a free path of advancement.
4. While holding graft in maximal expansion, place a fixation plate.
5. Graft around the gaps of the osteotomy with bone graft of choice.
6. Close wound with suture of choice.
7. Allow 6 months for healing prior to implant placement.

Ridge Split Technique
- More often used on the maxillae than mandible.
- Can gain from 3–6 mm of horizontal bone.
- Adequate bone height of 10 mm should be available on maxillae.
- In mandibular procedure, ideally more than 12 mm above canal.

- Must ensure ridge in favorable position and not too medial and without concavities.
- Minimum 3 mm of width.
- After ridge split, implant should be more facially positioned, likely require custom abutments when restoring.
- Tapered implants best to allow for increased expansion.
- Consider implant with less depth to threads.

Split Ridge for Maxillae Technique

1. Local anesthesia with vasoconstrictor.
2. Midcrestal gingival incision.
3. Minimal reflection of mucoperiosteal flap, no greater than 5 mm.
4. Use guide to mark implant position if immediate placement planned with 2.0 drill. If peak of bone present, it should be reduced.
5. Piezotome or saw used to make osteotomy, ensure angulation parallel with residual ridge for even splitting of bone. Ensure cut 2 mm away from adjacent teeth if present.
6. Spatula osteotome used for separation of cortical plates with gradually wider osteotomes/chisels to expand ridge. If difficult to expand, may make vertical osteotomies on facial bone at end of osteotomy to aid expansion.
7. Accomplish implant preparation with implant osteotomies (allow for more expansion) or implant drill if planned at this time.
8. Graft gap of osteotomy with bone graft of choice. Cortical graft may aid in keeping plates separated. Placement of collagen membrane to cover site.
9. Reapproximate tissues.
10. Allow 6 months for healing.

Technique for Mandible

1. Local anesthesia with vasoconstrictor.
2. Midcrestal incision and reflection of flap for complete buccal exposure.
3. Osteotomies to create an outline of the bone window into cancellous bone.
4. Close flap and allow 5 weeks of healing.
5. Re-expose a limited flap.
6. Spatula/chisel osteotomes used to carefully create greenstick fracture and expand ridge.
7. Graft site with bone substitute of choice and place membrane and close with sutures.
8. Implants can be placed 6 months after initial surgery or at time of expansion.

Complications

Facial Plate Fracture No implant placement. Graft gap and stabilize facial plate with plate and screws. Alternately, no expansion, secure fragment with plates, screw, or wire, and reattempt graft in 6 months.

Distraction Osteogenesis for Implant Site Development

- Based on "tension-stress," brings bone and tissue.
- Defects 6–9 mm in height are often indicated for distraction.
- Hard to control vector some doctors will wrap wire between adjacent teeth to aid in vector control or use surgical guides. Tendency for transport segment to rotate palatal/lingual.
- Lingual portion of osteotomies should be lingually convergent to prevent lingual tipping.
- With expansion of the bony segments, a "regenerate" is formed.
- This regenerate has four distinct zones:
  1. Fibrous tissue zone – located centrally and is organized type I collagen.
  2. Extended bone formation – located on both sides of the fibrous tissue zone. Mesenchymal and osteoblasts synthesize early bone spicules.
  3. Zone of bone remodeling – osteoblastic and osteoclastic activity causing bone remodeling.
  4. Zone of mature bone – located at the edges of the osteotomies.

Phases of Distraction

1. Surgical procedure – ensure divergent walls to allow passive movement. Location of incision should be in attached gingiva, if possible, to encourage gingival growth on distraction. 1 mm is allowed between roots to prevent injury in the osteotomy.

2. Latency period (3–7 days) – mobilizing the trasnport segment too early will cause the regenerate to be formed with high levels of fibrous tissue and low bone density.
3. Activation Period:
   – *Rate* – activation best at 0.7–1.3 mm per day (recommendation on most distractors 1 mm/day). High distraction rates (>2 mm/day) leads to impaired angiogenic response and fibrous bone. 0.5 mm/day carries risk for premature ossification and failure of distraction.
   – *Rhythm* – number of distractions per day. Increasing rhythm to several cycles a day reduces soft tissue trauma and patient comfort. A rate of 0.25 mm at 4 rotations a day, or 0.5 mm with 2 rotations a day, has shown to improve regenerate quality.
4. Consolidation (3 months) – keep device on until seeing radiographic evidence of bone healing. This may be longer in older patients. Can place implants at time of device removal.

Complications

Immobility of Transport Segment Incomplete osteotomy or poor osteotomy design that leads to blocking of transported segment. Treatment is to retrace osteotomies.

Loss of Distractor May be due to poor bone stock. Options are to replace distractor or consideration of bone graft and segment fixation with plate.

Tissue Dehiscence Slow rate of distraction, allowing for short period of tissue healing. Consider smoothing edges of segment if there are sharp areas.

Resorption of Transport Segment Due to interruption of blood supply that is most likely due to over reflection of tissue. Allow for prolonged latency period before further distraction.

Inadequate Vector of Transported Segment This can be avoided using extraoral devices or dental wiring to aid in vector guidance. May also consider other ridge augmentation techniques to overcome the malpositioned regenerate.

Zygomatic Implants

- Good for large maxillary ablative defects, traumatic defects, severely atrophic maxillae, cleft palate unrepaired defects, and patient's refusal for sinus augmentation.
- About 97% success rate [19].
- Zygomatic implants are available in 30–52.5 mm fixture lengths, 4 mm diameter in apical 2/3- and 5-mm diameter in the alveolar 1/3 (45° tilted connection to correct for angulation).
- Frequently enter oral cavity on palate side, reducing tongue space and disrupting palatal contour of prosthesis.
- As implants pass through sinus, sinusitis may compromise survival and should be addressed prior to placement. Patients are at a higher risk of postoperative sinus infections.
- Healing time is 3–4 months.
- Implant can be immediately loaded if a torque of >40 Ncm is acheived.
- Placement should be in the premolar region and into the mid portion of the zygomatic body.
- Intrasinus technique – create a lateral sinus window to push sinus membrane from implant; some clinicians elect to bone graft around implant and sinus cavity.
- Extra sinus technique – allows more crestal emergence, reducing sinus complications, increases tongue space allowing for decreased risk for altered speech and increase space for hygiene access. Major disadvantage is mid portion of implant is in direct contact with soft tissue, which may cause exposure and perforation.
- Require cross stabilization due to long moment arm of zygomatic implant.

Implant Prosthetics

Implant Attachments

Implant Bar Attachment

- Used for retention and support for an implant supported over denture.
- Can be fabricated using casting or milling (CAD-CAM) process.

Fig. 4.3 Implant bar attachments. (Reprinted with permission from Rutkowski [20])

- Material options: titanium, soldered gold, non-precious metal, zirconia.
- Bars constitute an excellent anchorage system that provides greater retention, enabling better force balance by its splinting effect and it can also correct severe lack of parallelism.
- The retention elements or clips are interchangeable and can be reactivated.
- The main disadvantages of bar attachments are the need for a large prosthetic space. There is an increased risk of mucositis and hyperplasia due to an inadequate oral hygiene under the bar.
- A minimum of 12–14 mm of vertical restorative space is required for a bar retained overdenture.
- Bars need to be parallel to the rotation axis, be straight, and be positioned 1–2 mm above the alveolar crest to aid in hygiene.
- There are some different bar designs such as Ackermann bar (spherical shape), Dolder bar (ovoid or "U" shape), and Hader bar (keyhole shape). Also, there are implant-supported milled bar overdentures. They are bars with precision attachments and rigid anchorage, made by casting, electroerosion, or CAD-CAM.
- Types: Hader bar, Dolder bar, Round bar, Free form milled bar, Paris bar, Wrap-around bar, Montreal bar, Hybrid bar (Fig. 4.3).

Locator Attachment

- The male part consists of an implant screw-metallic abutment and the female part of a metallic cap is lined with nylon of different colors depending on their retention capacity, which is anchored to the denture.
- These attachments do not need a large prosthetic space and they can correct non-parallel implants up to 40°.
- A reported minimum space requirement for implant-supported overdentures with locator attachments is 8.5 mm of vertical restorative space and 9 mm of horizontal space.

"O" Ring or Ball Attachment

- Ball attachments are considered the simplest type of attachment for clinical application with tooth- or implant-supported overdentures.
- It has a screw-retained male abutment in the implant with a spherical shape on its occlusal portion and a prosthetic anchored female part that can be metallic or covered with nylon having a different retention range.
- These attachments do not need a great prosthetic space and they allow hinge and rotation dislodgements.

Magnetic Attachment

- They consist of one magnet attached to the denture and another to the implant. They constitute a simple and comfortable system for the patient as magnet attraction guides the denture insertion. On the other hand, they have a weaker lateral stability and retention in comparison with mechanical attachments as ball or bar devices.
- They are susceptible to corrosion by saliva, explaining why they are clinically less often used.

Screw-Retained Versus Cement-Retained Restorations (See Tables 4.2 and 4.3)

Anterior Posterior Spread (AP Spread)

- AP spread is defined as the distance between a line drawn between the distal sides of the posterior implants and a parallel line drawn

Table 4.2 Pros and cons of screw-retained restorations

Screw-retained restorations	
Pros	Cons
Ease of retrievability Low-profile retention Limited crown height space: Low-profile bar for overdentures Crown contour requirement Reduced moment loads No residual cement Splinting nonparallel implants As the screw is the weakest link, it can be designed as the point of failure to prevent mechanical overload	Risk of prosthetic screw loosening Fracture risk of prosthetic screws Device not sealed (bacterial growth) Passive casting requires much more accuracy Lack of axial occlusal loads Less aesthetic restorations Increased risk of porcelain fracture Access is often difficult Lack of progressive loading Increased cost Angulation problems, fixture determines screw access, which can lead to undesirable access hole position

Table 4.3 Pros and cons of cement-retained restorations

Cement-retained restorations	
Pros	Cons
Ease of splinting implants Better passive fit Easier correction of non-passive casting Progressive loading can be achieved Improved force direction of loads Optimal occlusal contacts Enhanced aesthetics Improved access to posterior regions Reduced fracture of components Reduced porcelain fracture More economical More aesthetic overall as no access hole	Risk of residual cement causing peri-implantitis and implant failure, especially if implant placed too deep into bone More difficult to retrieve if abutment becomes loose Need for more crown height space

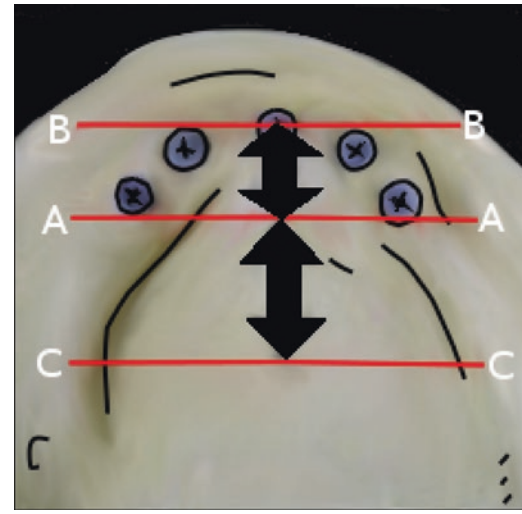


Fig. 4.4 AP spread. From line A to B is the A-P spread. Measure from line A to C, which is distal cantilever extension. (Courtesy of Erik Steenberg)

through the center of the most anterior implants (Fig. 4.4).

- The ideal AP spread is 1 cm when four or five implants have been placed.
- When the AP spread is 1 cm or more, Branemark and his colleagues concluded that the cantilever could be extended to, but not beyond, 20 mm or up to two times the AP spread.
- Others have recommended that the cantilever extension be limited to 1.5 times the AP spread. If the cantilever extension is excessive, the load delivered to the posterior implants is magnified and can lead to screw fractures and prosthesis or implant failure.
- It may be difficult to obtain implant arrangement with suitable AP spread in patients with square arch forms or significant anterior loop of the inferior alveolar nerve.
- In these patients, if a fixed prosthesis is desired, distal angling of the posterior implants may offer some theoretical biomechanical advantage. The distal angulation of the implants can be corrected with angled abutments (e.g., – tilted implant technique aka All-On-4®)

Restoration of Edentulous Maxillae and Mandible with Implant Prostheses

Implant-Assisted Overdentures

- The term implant-assisted prosthesis implies a system of shared support where implants provide retention and stability and the denture bearing areas providing support. Therefore, dentures must be extended properly to cover primary support areas with bilateral balanced occlusion.
- Implants should be positioned sufficiently anteriorly or else the denture will tip and rock around the implants during function.
- Several types of attachments have been used for implant-assisted overdentures.
- Selection is based on biomechanics, the quality of retention, stability and support provided, ease of fabrication, laboratory costs, the cost of maintenance, and the personal preference of the clinician.

Individual Attachment Systems

- Conventional ball-type attachment.
- Locator abutments.
- Mini-implants with O-ring attachments.
- Magnetic attachments.

Advantages
- Distribute occlusal forces in a favorable manner.
- Minimize risk of mechanical failures and implant loss secondary to implant overload.
- Exhibit less wear on the patrix and matrix of the system.
- Allow dentures to rotate freely.
- A high amount of implant divergence can be accommodated.
- Magnetic attachments do not lose retention over time.

Disadvantages
- Do not provide as much retention compared to bar-clip systems.
- Gradual loss of retention due to attachment fatigue and wear.
- Implants can be exposed to lateral torqueing forces.
- If implants are positioned improperly, it may be difficult to position denture teeth.
- High implant failure rates of mini-implants vs. conventional implants.
- Magnetic attachments do not provide as much retention as mechanical attachments.

Implant Connecting Bar-Clip Design
- Two implants splinted together with Hader-type bar can be used (Fig. 4.3).
- A Hader bar is round, allowing bar clips to freely rotate during occlusal function, resulting in less attachment fatigue of clips and wear of the bar compared to elliptical bar designs.
- The bar should be perpendicular to the midline and parallel to the plane of occlusion. ERA (extracoronal resilient attachment) may be added to the bar for increased retention.

Advantages
- Excellent support to resist incising forces.
- Implants share vertical and lateral forces.
- Superior retention and less maintenance compared to other systems.
- Provides better lip support compared to a fixed prosthesis.
- Can compensate for unfavorably positioned implants.

Disadvantages
- Added time and cost of fabrication.
- Harder to maintain oral hygiene.
- Tissue hypertrophy around implants and under the bar especially if the bar is designed to contact the underlying mucosa. This can lead to peri-implantitis.
- Implant-connecting bars are subject to wear and subsequent loss of retention of the denture.

Implant-Supported Overdentures

- With implant-supported overdentures, all of the forces of mastication are borne by implants.
- This can be achieved by fabricating implant-connecting bars with conventional or CAD/CAM methods.

- The prosthesis may be removable or fixed.
- The advantages/disadvantages of implant-supported removable prosthetics are as follows.

Advantages
- May be indicated when inferior alveolar nerve is exposed.
- Easier to maintain oral hygiene compared to a fixed prosthesis.
- Longer life span of the bar since there is no movement of denture base.

Disadvantages
- Requires placement of at least four implants in the mandible with at least 1 cm of AP spread to support posterior occlusal forces.
- The implant-connecting bars have to be bulkier and acrylic denture should be reinforced with metal substructure.
- Increased cost, time, and need for accuracy compared to implant-retained overdentures.

Implant-Supported Fixed Prosthesis

Advantages
- Psychological and psychosocial advantages of having a fixed prosthesis.
- Increased bite force.
- Patient prefers not having the palatal coverage in the denture.
- Improves phonetics, appreciation of temperature, and taste.

Disadvantages
- Needs five implants in the mandible or six in the maxilla, unless an All on 4®/tilted implant prosthesis is planned using angled implants.
- May require additional surgical procedures to augment alveolar ridge or maxillary sinus, or alveolectomy to provide interocclusal space.
- Significantly increases cost of prosthesis and need for accuracy.
- Sufficient interocclusal space is required to allow for fabrication of a rigid prosthesis and provide space beneath prosthesis to maintain oral hygiene.
- Challenge to maintain oral hygiene especially in elderly or debilitated patients.
- Esthetic limitations. Inability to provide adequate lip support due to insufficient denture flange.
- Needs sufficient AP spread to reduce distal cantilever or prosthesis.
- Unfavorably positioned implants can add significant cost and complexity to prosthesis.
- Complications include metal or zirconia framework fracture, fracture or delamination of veneering porcelain, separation of resin from metal framework, fracture and wear of denture teeth, fracture of implants and prosthetic screws.

Material Options
1. Hybrid prosthesis – denture teeth embedded in heat-cured acrylic resin supported by a rigid metal framework.
2. Metal-ceramic prosthesis – can be cast or milled. Acceptable aesthetics, tissue response is excellent, material is non-porous, little wear on occlusal surfaces. High cost.
3. Zirconia prosthesis – aesthetic, biocompatible. Produces less wear than porcelain surfaces; high strength, toughness, wear resistance, and acid resistance; minimal abrasiveness.

Guided Surgery

- Guided surgery is the surgical placement of dental implants using site and patient specific surgical templates developed with software programs that combine DICOM (cone beam computed tomography (CBCT)) and STL files (intraoral or bench scanners) or scanned stone models.
- Allow for virtual treatment planning based on the anatomy of the patient and the prosthetic plan. Surgical templates incorporate drill keys and metal sleeves and allow for the precise preparation of the osteotomy sites and positioning of implants. Guided surgery can be static or dynamic (navigation). Static guided implant surgery is more widely used. Dynamic (navigation) requires a specific equipment for it.
- CBCT should be taken with partial separation of teeth.

- In fully edentulous patients, the surgical template is secured with anchor pins or bone screws inserted into bone adjacent to the implant sites.
- Will require newly relined or a well-fitting denture.
- In partially edentulous patients, the surgical template can be retained with either anchor pins or bone screws or by the residual dentition.
- Fully guided surgery provides complete control of implant positioning, depth, and angulation. However, the size of the drill sleeves makes their use difficult in partially edentulous patients and posterior teeth.
- Semi-guided surgery allows for accommodation of first or maybe second drill and allows control of only the initial osteotomy.
- Computer-guided planning allows the clinician to scrutinize the potential implant sites, select implants of suitable length and diameter, and position them to be compatible with the prosthetic design.
- Provides a prediction of stability around the implant based on Hounsfield units around implant.
- Prefabricated temporary crowns, custom healing abutments, and even final custom abutments can be fabricated in the lab.
- These software programs are particularly valuable in the esthetic zone to identify thin layers of bone overlying the labial surface of the implant that are at risk of resorption.

Advantages

- Visualization of the potential implant sites in three dimensions in relation to the prosthesis.
- Allows of precise implant positioning including extraction sockets.
- Reduced risk of encroaching upon adjacent vital structures.
- Allows for the fabrication of prostheses and abutments before surgery and immediate loading.
- Allows for flapless surgery.
- Allows for increased communication between restoring dentist and surgeon.

Disadvantages

- Increased cost.
- Lack of interocclusal space especially in the posterior region.
- Lack of flexibility during surgery.
- It is technically demanding.
- Radiation exposure from CBCT scans is a concern. Radiation dose varies between machines.
- Scatter from metallic restorations may reduce accuracy.
- Limited mouth opening and mesiodistal space are contraindications.

Implant Case #1

- *A 48-year-old female presents with consultation for reconstruction of her upper left maxillae. She is healthy without any medication or*

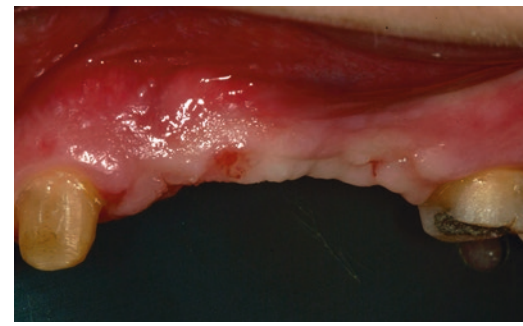


Fig. 4.5 Photo of case implant case #1. (Courtesy of Dr. Polido)

allergies. What do you see in the image focusing in the left maxilla? (Fig. 4.5)
I see a partially edentulous left maxillary ridge. It appears she is missing teeth 11 through 14. She has crowns placed on teeth 9 and 10. There is some gingival recession on tooth #10. Tooth #15 appears heavily restored. There is both height and width loss of the remaining ridge. There appears to be adequate keratinized gingivae.

- *What would you do next?*
  I would review how long the teeth have been missing and the mechanism. I would enquire about smoking and parafunctional habit history, what is her motivation for treatment, and how often does she have dental visits. I would inquire about her expectations on a prosthesis and whether she wants a fixed or removable solution, followed by a complete head and neck exam.
- *She has been missing these teeth for over 10 years. She had them extracted due to dental caries. She had worn a removable prosthesis but never liked it. She has always had difficulty chewing on her left side, but now she has the financial means to rehabilitate her maxilla. She is on a regular maintenance with her dentist every 6 months. She is a non-smoker and doesn't think she grinds or clenches. What specifically would you be looking for on a head and neck exam?*
  I would like to know the maximal incisal opening to gauge access to the surgical site. I would assess the interarch space, the width of the ridge and the amount of keratinized tissue to ensure implant long-term health. I would evaluate the gingival biotype diagnosed by probing into the sulcus of remaining teeth. I would also evaluate the overall health of the remaining dentition and whether there is any treatment work that would need to be completed, such as a dental prophylaxis or restorative treatment, prior to implant surgery.
- *What would you do next?*
  I would take a CBCT in my office and reformat for an orthopantogram.
- *What do you see* (Fig. 4.6)*?*
  A heavily restored dentition with a large spanning fixed prosthesis between teeth numbers 2 and 6. There is an implant at site #7. There is an edentulous maxillary ridge spanning from sites 11 through 14. There is sinus pneumatization of the upper left encroaching into the region of the second premolar. Tooth #19 has a slight overfill and leakage of material into the surrounding alveolus. Root canal therapy and full coverage restorations are noted on teeth 20 and 29. There are atrophic changes of the posterior mandible in the edentulous regions. There appears to be no intrabony pathology.
- *What next?*
  I would like to evaluate the cone beam slices of the edentulous left maxillary ridge.
- *What do you see in this image* (Fig. 4.7)*?*
  I see what appears to be a Cawood and Howell class III of the premolar region and class V of the molar region. The premolar region appears

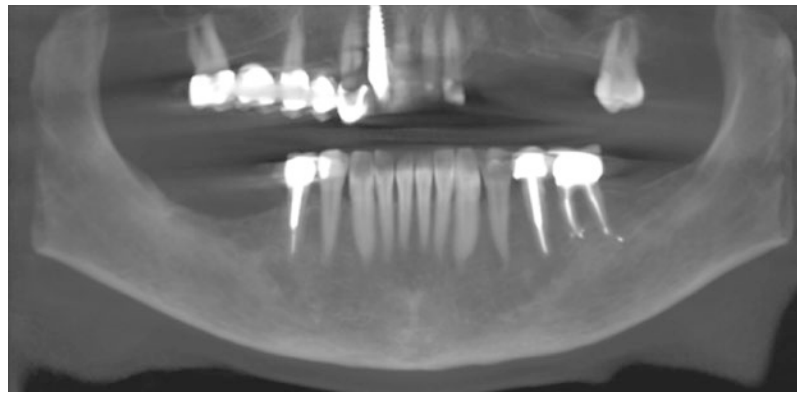


Fig. 4.6 Orthopantogram of implant case #1. (Courtesy of Dr. Robert Reti)

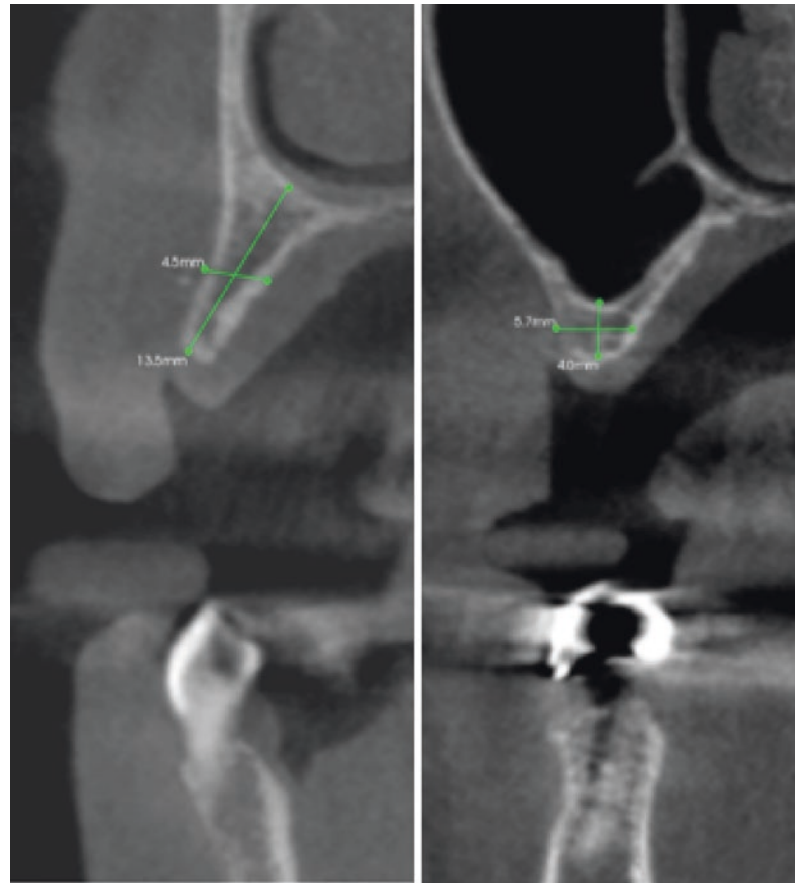


Fig. 4.7 CBCT of implant case #1

to have adequate height but a maximal width of 4.5 mm which would be inadequate for a regular diameter implant, which on average needs a minimum of 6 mm to allow a 1 mm buccal and lingual ridge. The molar site appears to have only 4 mm of height before the sinus cavity and just under 6 mm for width. Both sites are inadequate for a regular size implant placement.

- *Focusing on the premolar region, what are some options to create adequate width?*
  Options would include a veneer bone graft, split ridge procedure, cancellous graft, or a tunnel graft procedure.
- *What would you want to do and why?*
  I would offer a split ridge procedure as it is a procedure I am comfortable with and has good success in my hands. As well, it could potentially allow for immediate implant placement saving the patient healing time and reduced morbidity from a second graft site.
- *Describe how you would complete a split ridge procedure?*
  After infiltration with local anesthetic, I would make a sharp crestal incision down to the bone. I would carefully reflect a minimal flap to visualize the crestal bone. Using a piezotome I would make a corticotomy down to cancellous bone, staying 2 mm away from adjacent teeth not to disrupt them. I would carefully expand the bone with flat chisel osteotomes, keeping a dual finger guidance to help direct the slow careful expansion and mold the buccal bone. When appropriately expanded I would attempt to place my implants by under-preparing the apical portion of the osteotomy. I would graft around the implants and into the gap between the plates with a mixture of 1:1 allograft:xenograft. I would then place a resorbable membrane to contain the graft and close with 3-0 non-resorbable suture.

- *What implant system would you use and why?*
  I would use a tapered implant system. I would not want a system with a large aggressive thread pattern that would compromise the bone. The less aggressive threads would allow some further expansion upon placement. I would use a tapered implant, as it would aid in grasping the remaining apical bone that would be limited.
- *Your patient who has undergone a sinus floor elevation using the Summer's technique calls in 2 days after the procedure complaining of dealing with dizzy spells every morning. What do you want to know?*
  Inquire about constitutional symptoms (fever, chills, nausea, or cold-/flu-like symptoms), ear ache, or tinnitus. Has she ever experienced this before and if so has she ever been worked up for these symptoms? (rule in/out idiopathic endolymphatic hydrops?) Are the symptoms brought upon when she first wakes up from bed form a supine to upright position? How long do they last? Does she experience any nystagmus with these dizzy spells?
- *This has never happened before. She has no flu-like symptoms. She usually experiences a self-limiting dizzy spell from about 30 seconds when she gets up from bed. Her husband notes her eyes tend to shake when this happens. What next?*
  Benign paroxysmal positional vertigo is a known complication of sinus floor elevation and would be my working diagnosis. I would attempt to control her symptoms with antihistamines such as meclizine. I would reassure her that this likely would self-correct. If symptoms continue for a prolonged period, I would refer her to an ENT colleague for workup and canalith repositioning procedure (Epley maneuver).

Implant Case #2

- *A 78-year-old male presents with complaint of ill-fitting dentures. He takes simvastatin for hypercholesterolemia and an 81 mg ASA. His dentist has asked him to consult you for dental implants to help retain his denture. What would you like to do next?*
  I would perform a history and physical with some focused questions. Has he had a history of cancer? Use of any antiresorptive medications like bisphosphonates? Does he smoke? How long has he been missing his teeth for? What is his restorative goal and what motivates him to come in now? Is he having pain or discomfort when he wears his dentures?
- *He has no history of cancers or use of antiresorptive medications. He did smoke 1 pack per day in the past but had quit after his time in the service. He blames his edentulism due to "soft teeth" that runs in his family. His denture will not stay in any more even with denture adhesive. He reports recent pain with mastication. He complains of occasional "pins and needles" sensation in his lower lip and this is limiting his diet. What next?*
  I would like to do a head and neck exam. I would focus on if there is any keratinized tissue that remains. I would evaluate for the attachment of the genioglossus. I would press along the ridge to see if I can recreate the paresthesia. With his history of smoking, I would like to identify any concerning lesions.
- *There is no keratinized tissue remaining and no lesions appreciated. When you apply digital pressure on the mid mandible, it elicits a painful response from the patient. Would you like any imaging?*
  Yes, I would like a CBCT as I can use it for accurate measurements and can reformat it into an orthopantogram.
- *Ok, what do you see?* (Fig. 4.8)
  I see a severely resorbed mandible with ditching of the cortical bone. No fractures are appreciated or pathological changes.
- *How would you classify this mandible?*
  Cawood type 6.
- *Are there any other grading systems for edentulous mandibles?*
  Yes, there is the Luhr classification system that takes into account residual bone height. Class I, mild atrophy with a height between 15 and 20 mm. Class II, moderate atrophy with a height between 10 and 15 mm. Class III, severe atrophy with a height of less than 10 mm.

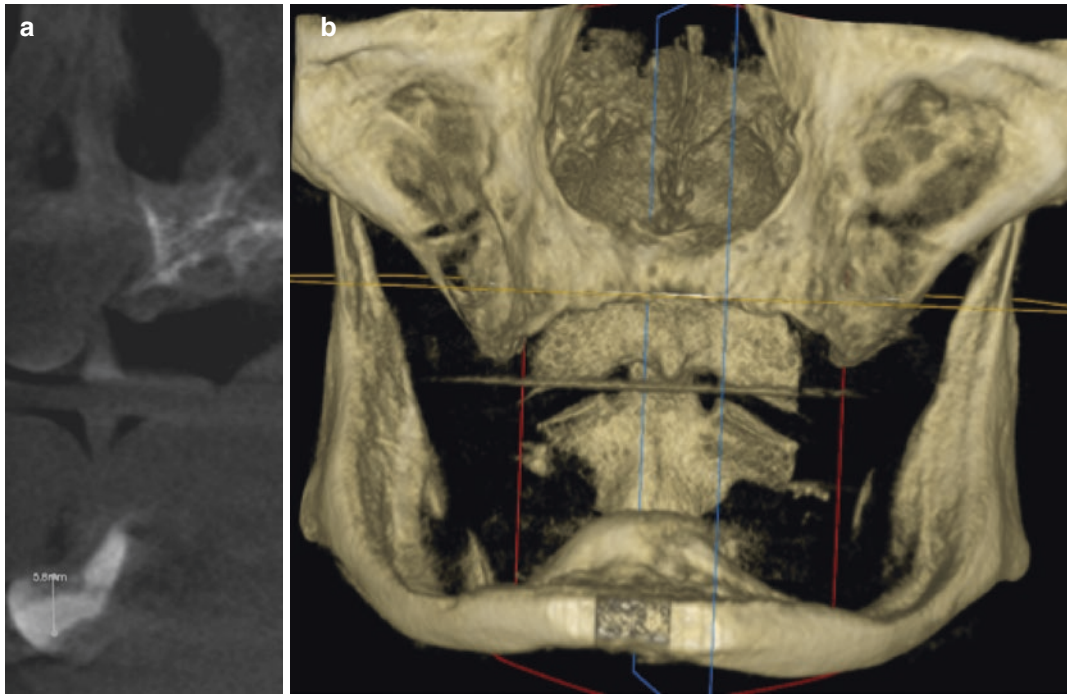


Fig. 4.8 (**a**) CBCT sagittal view of implant case #2. (**b**) 3D reformat of implant case #2. (Image Courtesy of Dr. Robert Reti)

- *What would your treatment plan be?*
  I would offer a "Tent Pole Procedure" with 6 implants.
- *Can you describe how you would do that briefly?*
  Ensuring no paralysis and with nerve monitoring. I would inject the tissue with anesthetic. I would outline a submental incision, not in the submental crease but 5 mm behind this to allow for the forward position of the tissue after grafting. I would carefully dissect down to the mandible in a layered fashion. I would dissect the buccal and occlusal tissues to the trigone regions along the mandible. I would identify the mental foramina on both sides. I would place my first two wide-bodied 15 mm in length implants, 5 mm in front of the identified mental foramina emergence. I would space the remaining implants evenly between the two distal implants. I would place cover screws at this time. I would protect the surgical site with saline-soaked gauze and harvest a posterior iliac graft. I would then return to the neck and graft around the implants and into the trigone regions. I would close in a layered fashion.
- *What would your postoperative instructions be?*
  I would recommend a soft diet for 3 months. No denture could be worn for the first 2 weeks. For the graft harvest, bed rest for the first 24 hours, assisted ambulation for the first week, and no physical activity for 6 weeks.
- *Patient complains of swelling on the floor of the mouth. Exam shows the tongue is slightly elevated, but no respiratory distress. This is the CT scan. What do you think is going on?* (Fig. 4.9)
  It appears the implant has perforated the lingual cortex. The implant may have damaged the sublingual gland causing what appears to be a possible plunging ranula. Damage to the muscle or vessel may have caused a bleed or hematoma in the area.
- *You aspirate the area and it comes out a clear thick fluid? What is your treatment?*

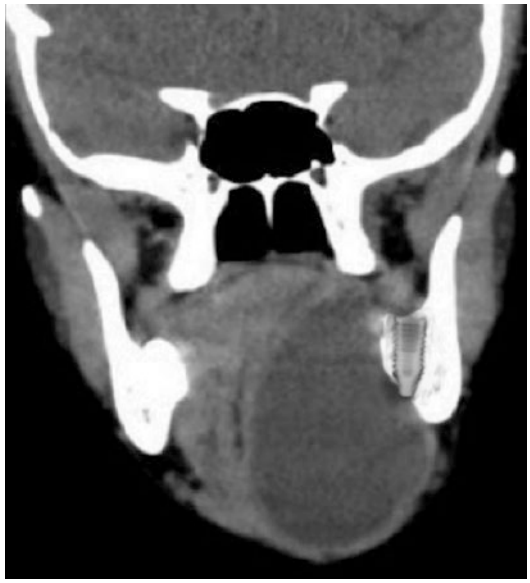


Fig. 4.9 Complication of implant case #2. (Image Courtesy of Erik Steenberg)

I would remove the implant and perform a transcervical removal of the ranula and the damaged gland.

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Part II

Orthognathic, OSA, Cosmetics, and Trauma

Orthognathic and Obstructive Sleep Apnea

5

Myron R. Tucker, Richard G. Burton, Aaron D. Figueroa, Vincent Carrao, Riddhi Patel, Bryan Weaver, Gregg A. Jacob, and Joseph W. Ivory

Indications for Orthognathic Surgery

When dentofacial deformities cannot be corrected by conventional orthodontic compensation, including growth modification and camouflage techniques as well as for treatment of OSA.

Anterior-Posterior Discrepancies

- Over jet 5 mm or more or a zero to negative value (normal 0–2 mm).
- Molar relationship discrepancy of 4 mm or more (normal is 0–1 mm).

Vertical Discrepancies

- Skeletal facial deformity of two or more standard deviations from published norms.
- Open bite: no vertical overlap of the anterior teeth or posterior open bite of 2 mm or more.
- Deep overbite with irritation of tissues.
- Supraeruption of dentoalveolar segment due to lack of occlusion.

Transverse Skeletal Discrepancies

- Presence of skeletal transverse discrepancy of two or more standard deviations from published norms.

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- Maxillary palatal cusp to mandibular fossa relationship that is 3 mm or greater for a unilateral or 4 mm or greater for a bilateral relationship. Of note, one must differentiate between dental tipping and skeletal deficiencies in this deformation.

Other Indications for Orthognathic Surgery

- 3 mm or greater of asymmetry in any vector with 3 mm associated occlusal asymmetry.
- Patients with severe class II and class III problems, anterior open bite, markedly increased overbite, and facial asymmetries.
- Once the skeleton and dentition are aligned, the surgery may lead to improved speech, esthetics, function, and social interactions, and alleviate temporomandibular joint dysfunction.
- Cleft lip and palate patients with maxillomandibular skeletal disharmony.
- Facial syndromes and congenital anomalies.

Presurgical Orthodontic Goals

Decompensation of Teeth

- Dental compensation is nature's attempt to camouflage a jaw deformity.
- Orthodontic decompensation aims to reverse the natural compensation of teeth and move them into their appropriate axial inclination within the upper or lower jaw so they are housed within the alveolus.
- Remember that the upper and lower incisor angulations drive the anterior- posterior position of the maxilla and mandible into their final position.
- The orthodontic decompensation will exaggerate the malocclusion and thus make the skeletal deformity more noticeable.
- In class II skeletal patients, the retroclined upper incisors should be aligned over the alveolar bases (proclined to 102 degrees to SN) to maximize the overjet while at the same time maintaining a normal angulation. Caution must be used so that the transverse position of the maxillary canines does not block out the mandibular incisors as the mandible is advanced surgically. The lower incisors are typically proclined in this population and the goal should be to have the lower incisors at 90–95 degrees to the mandibular plane. This may require removal of the first or second bicuspids or reproximation (stripping) if very significant crowding exists.
- In class III skeletal patients, the lower incisors are usually found to be retroclined and upper incisors proclined. The lower incisor dentition in these patients should be proclined so that the lower incisor is at 90–95 degrees to the mandibular plane. The upper incisors should be retracted to obtain and angle near 102 degrees to SN.

Arch Alignment and Leveling

- All teeth are aligned when gross crowding, spaces, or rotations are corrected.
- Adjustments for tooth size discrepancy (TSD) – calculated with Bolton's analysis (see below).
- Create divergence of roots adjacent to interdental osteotomy sites.
  - To perform interdental osteotomies, space should be created between the lateral incisors and the cuspids or between the cuspids and first bicuspids ideally.
- Lingual cusps of the mandibular posterior teeth should be 1 mm below the buccal cusps.
- Palatal cusps of the maxillary posterior teeth should be 1 mm below the buccal cusps. Plunger cusps create open bites post-op as the dentition relapses.

Bolton Analysis

- Determines the disproportion of the size of the permanent maxillary and mandibular teeth (tooth size discrepancy between the upper and lower teeth). Two ratios are calculated (overall ratio and anterior ratio).
- The overall ratio is calculated by taking the sum of the mesiodistal width 12 mandibular

teeth (first molar to first molar) divided by the sum of the mesiodistal width of the 12 maxillary teeth. According to Bolton, the overall ratio should be 91.3%. A ratio less than 91.3% indicates maxillary tooth excess.
- The anterior ratio is calculated by the sum of the mesiodistal widths of the anterior mandibular teeth divided by the sum of the mesiodistal widths of the anterior maxillary teeth (canine). According to Bolton, the overall ratio should be 77.2%. A ratio less than 77.2% indicates maxillary tooth excess.
- Tables are available in multiple orthodontic textbooks with calculated ratios easily obtainable.

Arch Coordination

- Teeth may not interfere with planned skeletal movement.
- Both dental arches should be reasonably compatible with one another at the time of surgery to allow maximum intercuspation post-surgically.
- Arch form must be changed to expand the constricted areas in the more tapered arch or change narrow arches into more rounded form.
- As a general rule, orthodontic expansion should be limited to 4–5 mm total, although this depends on angulation of the posterior teeth. In many cases, there is dental compensation (vertical position of maxillary posterior teeth) that can easily be corrected with orthodontic treatment up to 6–7 mm. However, if the position of the posterior teeth is angulated facially with narrow basal bone surgical expansion is indicated.
- For severe maxillary transverse discrepancy surgical correction greater than 5 mm, surgically assisted rapid palatal expansion (SARPE) or segmental osteotomy should be considered.

Final Presurgical Orthodontic Preparation

- At the conclusion of presurgical orthodontic goals, the patient should be in full dimension rectangular steel arch wire that fills the bracket slot.
- There should be absolutely no movement of the teeth for at least four weeks before taking presurgical models: either stone or virtual.
- The stabilizing wire must fit passively to be effective.
- Surgical hooks attached to the brackets or arch wires are usually necessary to facilitate maxillomandibular fixation and to provide a means of using postsurgical elastic guidance or traction.
- Fixtures attached to the brackets include ball hooks or K (Kobayashi) hooks (Fig. 5.1).
- They may distort or break during the surgery.
- Fixtures attached to the arch wire include crimped-on hook and soldered pins.
- The use of postsurgical elastics may activate the arch wire, possibly creating unwanted orthodontic movements.

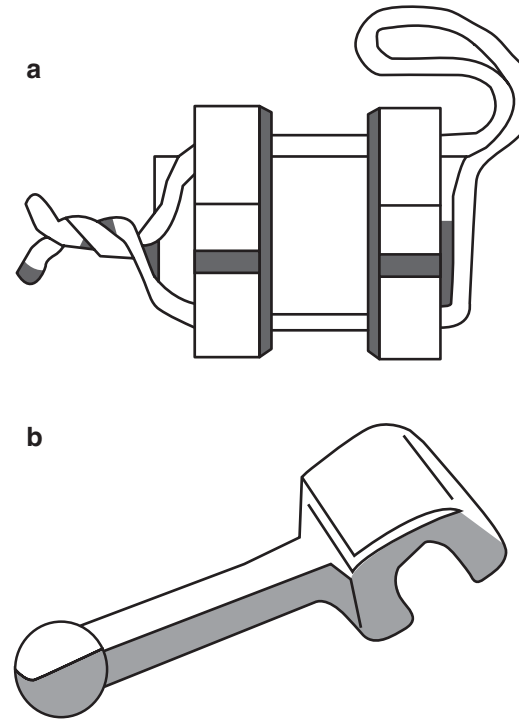

Fig. 5.1 A. Kobayashi hook B. Ball hook. (Image courtesy of Erik Steenberg)

Surgical Workup

Traditional Model Surgery

- Clinical data collection
  - Facial measurements
  - ROM/TMJ findings
  - Cranial nerve exam
- Photographic records
  - Extraoral front relaxed and smile (natural head position), lateral with lips in repose, ¾ view. If you are using the GALL line, then lateral with smile is needed, and ensure no hair is covering forehead.
- Facial measurements
  - Look for position of orbits
  - Position of ears
  - Alar width
  - Asymmetries and Cants.
- Inter-occlusal records or bite registration in centric relation (CR)
- Diagnostic casts that are mounted on an anatomic fully adjustable articulator in CR
- Facebow transfer to mount maxilla
- Radiographic exam

Virtual Surgical Planning

- Clinical data collection
- Photographic records
- Facial measurements
- Diagnostic casts/digital dental scan
- Centric relation record with fiducial markers
- Cone beam computed tomography (CBCT) or CT scan
- Final occlusion established by surgeon
- Data transfer to service center
- Planning specifics – order form (osteotomy design, order, and anticipated movements)

Radiography

Lateral Cephalogram

- Used to aid in making a skeletal diagnosis. Patient is held in adjusted natural head position.
- Lips relaxed, mandible in retruded contact position, and teeth lightly in occlusion.
- Assess the inclination of the various facial planes.
- Used to assess the AP position of the maxilla and mandible.
- May show asymmetries of condylar ramal height/size between the left and right (inferior borders do not coincide). Ensure radiograph taken correctly, verify with AP cephalometric.
- Can be used to assess the relationship of the maxilla to the cranial base.
- Can visualize the posterior airway space.
- Can assess growth by looking at the cervical vertebrae along with a growth chart.
- The goal of surgery is not to give patients cephalometric normal values, instead it is used as a guide to treat the clinical picture.

Orthopantogram

- Used to examine the dentition.
- Can assess the temporomandibular joint anatomy.
- Assess for presence of third molars.
- Assess for pathological lesions/entities.
- Can assess the inclination of roots (could be supplemented with periapical radiographs to view interdental osteotomy sites).
- Used to assess the position of the inferior alveolar canal and entry to ramus.
- Degree of sinus pneumatization.

Computed Tomography

- Used for gross facial asymmetries and for planning surgical treatment. Scan should be obtained at a high spatial resolution with no motion artifact.
- The patient's CT/CBCT scan is reoriented by the computer engineers to reflect their NHP for more accurate planning. To produce accurate CAD/CAM splints, occlusal surfaces from CT or CBCT scans are replaced with a high-resolution laser scan from stone models

or an intraoral scanner to create a composite model.
- CT/CBCT can be reformatted into orthopantogram. Also allows assessment for TMJ, third molar, interdental osteotomy site, and IAN nerve canal position.

Posteroanterior Cephalogram

- Will highlight significant facial asymmetry and can quantify excess or deficiency with tracing and measuring films. This film also allows the surgeon to further evaluate for skeletal cants of maxillae, mandible, and chin point.

Submental Vertex

- Evaluate for U or V shape mandible, may have combination. V shape favors IVRO for setback. U shape favors BSSO for setback.

Presurgery Records

- An ideal time to obtain presurgical records is 2 weeks prior to the planned surgery date. If custom plates (patient-specific plates) are to be used, then at least a month of lead time is needed to have time to plan and manufacture them.
- Don't take models until after the final rectangular orthodontic arch wire has been in place to be passive – ideally 4 weeks or more.

Dental Casts or Scanned in Dentition (STL Files)

- To be used in model surgery itself.
- Facilitates fabrication of occlusal wafer splints.

Facial Photographs

- Frontal full face with lips in repose.
- Frontal full face with animation.
- 45-degree oblique (three-quarter) with lips in repose.
- Right and left profile view in natural head position (NHP), in repose, full smile, and lips together.
- Additional – submental view.
  - To document mandibular and/or midface asymmetry.
  - To allow detailed analysis of nasal tip form in patients with abnormalities.

Intraoral Photographs

- Right, center, and left view with teeth in occlusion.
- Maxillary and mandibular occlusal view.

Facial Examination

- An examination of the face is performed with the patient in adjusted NHP.

Frontal View

Vertical Facial Proportions

- Facial thirds – The ideal face in both males and females is horizontally divided into equal thirds by horizontal lines at the hairline (Tr), glabella, the subnasale (Sn), and menton (Me') (Fig. 5.2).
- Upper third of the face, measured from trichion to glabella.
  - Deformities may indicate craniofacial deformity.
  - Assess eyebrow shape, position, and symmetry.
- Middle third of the face, measured from glabella to subnasale.
  - Includes eyes, nose, and cheeks.
  - Scleral show and flattening of cheek bones/paranasal region may indicate midface deficiency.

Fig. 5.2 Facial thirds. (Courtesy of Dr. Damian Findlay)

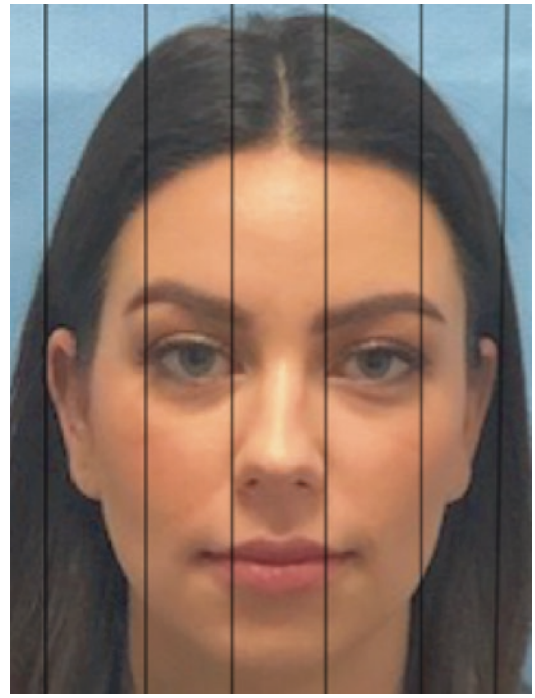

Fig. 5.3 Facial fifths. (Courtesy of Dr. Damian Findlay)

  - The nose, center of the lips, and middle of the chin should fall along the true vertical line.
  - The cheek bone-nasal base-lip contour line evaluates harmony of the structures of the midface with paranasal area and upper lip.
- Lower third of the face measured from subnasale to menton.
  - Lower third is further subdivided into upper one-third from subnasale to stomion superius (Sn-Sts) and lower two-thirds from stomion inferius to menton (Sti-Me').
- The ratio of middle third to lower third vertical height of face should be 5:6.
- Racial differences need to be considered.

Transverse Facial Proportions

- The "Rule of Fifths" – the face is divided sagittally into five symmetric and equal parts and each of the segments should equal the width of one eye (Fig. 5.3).
- The outer canthi should coincide with the gonial angles.
- The medial canthi should coincide with the alar bases of the nose.
- The inter-pupillary distance should coincide with the corners of the mouth.

Facial Symmetry

- Maxillary and mandibular dental midlines should be coincident to facial midline.
- Important midline structures are glabella (G), nasal bridge (NB), nasal tip (Pn), the midpoint of the philtrum of the upper lip (F), dental midline (DM), and the midpoint of the chin (Pog').
- Assess maxillary and mandible dental midlines in relation to each other.
- Mandibular dental midline in relation to the mid chin point should coincide.
- Smile and tooth-lip relationship.
- Upper lip should expose full crown length in males and with 2 mm gingival show in females.
- Normal tooth exposure on repose is 2–4 mm. Greater than 4 mm indicative of lip incompetence. Patient may display mentalis strain and have a tendency for mouth breathing.

- Excessive tooth display at rest may be from:
  - Short philtrum height. Look for this in the cleft population.
  - Vertical maxillary excess.
  - Excessive crown height/length.
  - Lingually tipped maxillary incisors.
- Inadequate incisor display may be from:
  - Excessive philtrum height; subnasale to upper lip is >22 mm.
  - Vertical maxillary deficiency.
  - Inadequate crown height.
  - Flared maxillary incisors.

Profile View

The Upper Third of the Face

- Supraorbital rim projects beyond the most anterior projection of the globe of the eye.
- Glabella should be coincident with the base of the nose.

The Middle Third of the Face

- Assess nose, cheeks, and paranasal area.
- Nose:
  - Lower nose projection can be affected by the anteroposterior (AP) position of the maxilla.
  - Alar base shape resembles isosceles triangle from worm's view.
- Cheek:
  - The lateral orbital rim lies 8-12 mm behind the globe.
  - The globe projects 0-2 mm ahead of infraorbital rim.
  - Malar eminence should be located 10-15 mm lateral and 15–20 mm inferior to the lateral canthus.
  - Cheekbone-nasal base-lip curve contour should be smooth, uninterrupted curve.
- Paranasal Area:
  - Paranasal deficiency represented by flatness of cheeks is often present in patients with maxillary deficiency.
  - The nasolabial angle is normally 100 degrees ±10 degrees; it is greater in females than in males.

The Lower Third of the Face

- Lower third face height: subnasale to soft tissue menton
  - Lower third divided into two portions:
    Subnasale to wet line of upper lip (1/3)
    Wet line of lip to soft tissue menton (2/3)
- Lips:
  - Subnasale-pogonion line (lower facial plane) – the upper lip should be 3 ± 1 mm and lower lip 2 ± 1 mm ahead of the line
  - Interlabial gap normally 0–3 mm in repose
  - Upper lip length: 20 +/− 2 mm in females, 22 +/− 2 mm in males
  - Lower lip length: 40 +/− 2 mm in females, 44 +/− 2 mm in males
- Labiomental Fold:
  - S-contour
  - Angle – 130°.
- Chin-Throat Angle:
  - Normally 110 degrees – provides chin definition.
  - Chin adiposity and hyoid bone position effect angle.

Cephalometric Analysis

Soft Tissue Analysis

Maxillary and Mandible AP Evaluation

- A vertical line perpendicular to constructed horizontal is drawn through soft tissue glabella (G').
- Pog' should be 1-4 mm behind this line.
- For maxillary AP position, Sn should be 6 ± 3 mm ahead of the line.
- For mandible AP position, Pog' should be 1-4 mm behind the line.

Nasolabial Angle

- Angle formed by tangent line to columella and upper lip. Normal range is 85–105 degrees.
- More acute in males and obtuse is more attractive in females.
- Acute in class III.
- Obtuse in class II.
- Influenced by lip support, lip thickness, lip strain, and magnitude of the overjet.

Lip Prominence

- A line drawn from subnasale (Sn) to soft tissue pogonion (Pog').
  - The perpendicular distance of upper lip ahead of this line should be 3 ± 1 mm, while lower lip should be 2 ± 1 mm.
  - The AP position of upper lip is an indication of soft tissue support by maxillary incisors.
- Another way to measure it is by using subnasale vertical (SnV) – a vertical line drawn from subnasale perpendicular to true horizontal line.
  - The upper lip should be 1-2 mm ahead of this line.
  - Lower lip should be on or just posterior to SnV.

Chin Prominence (Fig. 5.4)

- A line drawn through N′ perpendicular to FH is 0-degree meridian line.
  - Pog' 0 ± 2 mm ahead of 0-degree meridian and 3 ± 3 mm behind SnV (subnasale vertical).

Lower Lip-Chin-Throat Angle

- Angle formed by a line drawn from Li to Pog' and submental tangent line – 110 ± 8 degrees.
  - Acute in patients with mandible AP excess and/or macrogenia.
  - Obtuse in patients with mandible AP deficiency and/or microgenia.

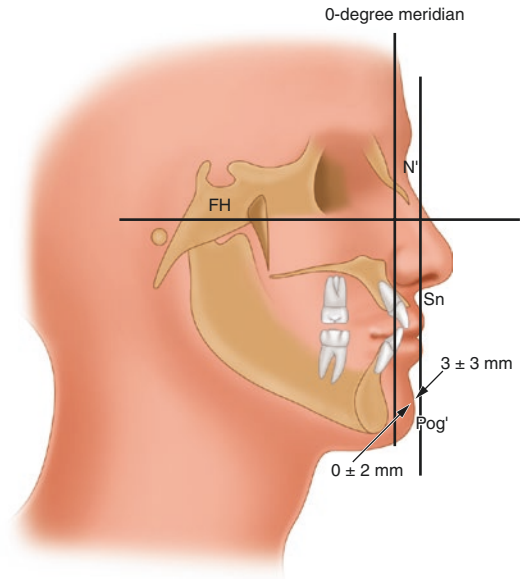

Fig. 5.4 Chin prominence can be evaluated in relation to Frankfort horizontal (FH). A line drawn perpendicular through FH through soft tissue nasion is known as (0-degree meridian); soft tissue pogonion should be 0 ± 2 mm ahead of this line. A line perpendicular through FH through subnasale is known as subnasale vertical. Soft tissue pogonion should be 3 ± 3 mm behind subnasale vertical. (Image courtesy of Erik Steenberg)

- Chin Throat Length
  - Measured from angle of the throat to Me' (normal value 42 ± 6 mm).
  - Helps differentiate between mandibular excess and maxillary deficiency.
- Facial Contour Angle
  - Formed by lines drawn from G' to Sn (upper facial plane) and from Sn through Pog' (lower facial plane).
  - Mean angulation is −12 degrees (Fig. 5.5).
  - Males tend to have a straighter profile (−11 ± 4 degrees), while females have slightly more convex profile (−13 ± 4 degrees).
  - Various facial deformities may produce the same facial contour angle.

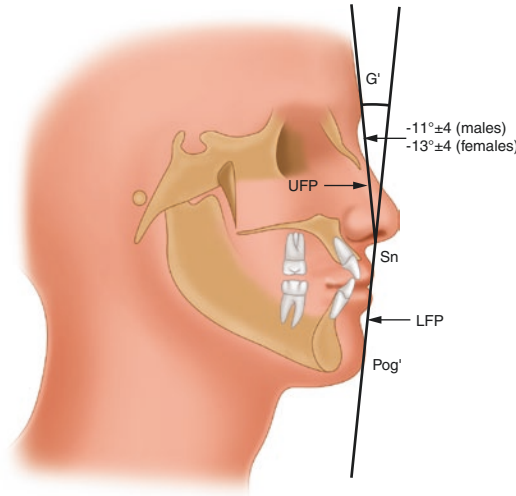

Fig. 5.5 The facial contour angle describes facial convexity or concavity. It is formed by the angle of the upper and lower facial planes. Averages are −13 ± 4 for females and 11 ± 4 for males. (Image courtesy of Erik Steenberg)

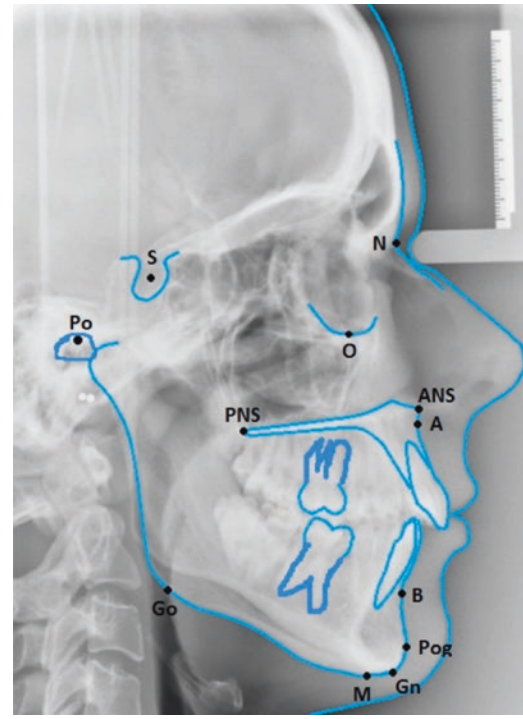

Fig. 5.6 Cephalometric hard tissue points

Skeletal Analysis

- Tracings of lateral cephalograms utilize stable hard and soft tissue points to aid in diagnosis, changes in growth, and treatment of dentofacial deformities (Fig. 5.6).

Assess Maxillary AP Position

- Steiner Analysis
  - Maxillary anteroposterior positions in relation to the anterior cranial base (S-N).
  - SNA of 82° is considered normal. An angle <82° indicates maxillary AP deficiency, while >82° indicates maxillary protrusion.
- Ricketts Analysis
  - Ricketts analysis uses maxillary depth.
  - Measures angle at the intersection of FH line and the NA line.
  - An angle of 90 ± 4 ° is ideal. An angle less than 86° indicates retrognathia, while an angle greater than 94° indicates prognathism.
- McNamara Analysis
  - Measures the distance from A point to Nasion perpendicular (a line that crosses N and is perpendicular to FH), normal range is 0-1 mm.
  - A negative number indicates retrognathia, while a positive number greater than 1 indicates prognathism.

Assess Mandibular AP Position

- Steiner Analysis
  - Mandibular anteroposterior positions in relation to the anterior cranial base (S-N).
  - SNB of 80° is considered normal. SNB <80° indicates mandibular AP deficiency and a greater angle indicates mandibular excess.
- Facial Angle (Downs Analysis)
  - Indicates relative AP position of mandible to cranium.

  - An angle formed by the intersection of the facial line, N-Pog' line and FH line.
  - Mean is 82–95°.
- McNamara Analysis
  - Measures the distance from Pog to N perpendicular (a line that crosses N and is perpendicular to FH).
  - An ideal number for mixed dentition is −8 to −6 mm, adult female is −4 to 0 mm, and adult male −2 to +2 mm.

Assess AP Maxillomandibular Relationship

- Steiner
  - Provides an idea of anteroposterior relationship between maxilla and mandible.
  - A normal maxillomandibular relationship is indicated by ANB of 2°.
  - In class III cases, angle is <2 or even negative. In class II cases, angle is >2.
- Wits Appraisal
  - Linear relationship between maxilla and mandible not influenced by cranium.
  - Points BO and AO are established by dropping perpendicular lines from A point and B point, respectively, onto the occlusal plane (OP) (Fig. 5.7).
  - The mean in male is BO 1 mm ahead of AO. In females, AO and BO coincide.
  - The measurement between AO and BO indicates the AP discrepancy between maxilla and mandible.
  - Small discrepancy between AO and BO indicates that the case can be treated orthodontically, while large discrepancy may indicate surgical correction.
- Mandibular Plane Angle (Steiner)
  - Formed between mandibular plane (Go-Gn) and anterior cranial base (S-N), normal value is 32 degrees.
  - An angle ≥39° is considered high, ≤ 28° is a low angle.

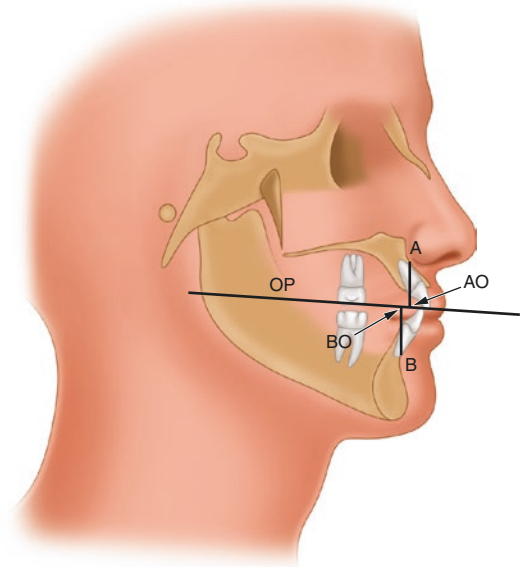

Fig. 5.7 Wits appraisal

  - Interprets the difference between anterior and posterior facial heights.
  - Increase in mandibular plane angle tends to have dolichocephaly, class II malocclusion, vertical maxillary excess, and apertognathia.
  - Decrease in angle is associated with brachycephaly, skeletal deep bite, and notched gonial angles.

Analysis of Dental Relationships

Maxillary Incisor Position

- Steiner
  - AP position of incisor to maxilla.
  - The axial inclination should be 22° to NA and the most anterior point of maxillary incisor should be 4 mm ahead of NA.
  - The facial surface of maxillary incisor should be 4-6 mm ahead of the vertical line through A point.

Mandibular Incisor Position

- Steiner
  - AP position of incisor to mandible (NB line).
  - Mandibular incisor angulation to NB line should be 25 ° and the most labial point of incisor should be 4 mm anterior to the line.

Occlusal Plane Angle

- Steiner Analysis
  - Angle between the occlusal plane (OP) and anterior cranial base (S-N) – 14°.
  - High-angle individuals have relatively long anterior facial height, while low-angle individuals have vertically short anterior facial height.

Chin Assessment

- Holdaway Ratio
  - Extend NB line to the inferior border of the mandible and compare the distance between L1 (incisal edge of mandibular incisor) and Pog from this line.
  - A ratio of 1:1 is ideal in males and 0.5–1 in females.
  - REMEMBER, this is only of value if lower incisors are in the proper position. Predictions for chin correction must be made with this in mind.

Growth Evaluation

- Skeletal age – serial hand-wrist radiographs.
- Deceleration of growth – serial cephalometric radiograph.
- Six stages of cervical vertebrae maturation on lateral cephalometric radiograph.
  - The peak of mandibular and craniofacial growth corresponds to the peak in statural height growth, which corresponds to stages 3 and 4 of cervical vertebral maturation.
  - As cervical vertebrae mature through six stages, they develop a concavity on the inferior border and assume a more rectangular shape in both boys and girls.

LeFort (LF) Osteotomy

The LeFort osteotomy has a long history and has become the standard of care when performing maxillary osteotomies to achieve a change in maxillary position. The maxillary position can be changed in any one of three dimensions or a combination thereof. When deciding on maxillary position, we must be sure to have the correct diagnosis, which ultimately will guide our surgical plan and produce the best surgical result for the patient. Maxillofacial deformities are always determined by the physical exam which then may or may not be supported by radiographic findings.

The following list of maxillary diagnoses includes important physical findings that exemplify the specific diagnosis.

Maxillary hyperplasia can be one of the following or a combination thereof:

Posterior vertical excess

- Open bite

Anterior vertical excess

- Excessive gingival show and/or excessive incisal show (resting and/or smiling)

Anterior posterior excess

- Acute nasolabial angle
- Excessive gingival show and/or excessive incisal show (resting and/or smiling)
- Maxillary protrusion

Maxillary hypoplasia can be one of the following or a combination thereof:

Posterior vertical deficiency

- Steep mandibular plane
- Posterior open bite

Anterior vertical deficiency

- Incisors not visualized at rest
- Minimal to no incisor show with smiling

Anterior posterior deficiency

- Midface deficiency
  - Poor piriform rim support (flat to concave maxillary appearance)
- Incisors not visualized at rest
- Minimal to no incisor show with smiling

LeFort Osteotomy Technique

- General anesthesia with nasal intubation.
- Place K wire at nasofrontal suture and take measurement to lower edge of maxillary incisor orthodontic bracket. This is an external vertical reference marker that can be used when complicated movements are to be done.
- Full-thickness maxillary vestibular incision from one zygomatic maxillary buttress to the other (leave at least 5 mm of nonkeratinized mucosa to facilitate closure).
- Bony exposure superiorly via this full thickness mucoperiosteal flap.
- Bluntly dissect to the nasal aperture and protect the nasal mucosa by lifting it up with a freer elevator, stay just shy of the infraorbital nerve and tunnel to the pterygoid plates bilaterally. The soft tissue posteriorly can be protected with a neuro-patty soaked in local anesthesia and a small retractor.
- Dissect anterior nasal spine free, dissect nasal floor off the palatal shelf using a freer.
- Bone markings can be used to measure subsequent maxillary movement (internal reference marks).
- Horizontal cut (or stepped cut) from posterior maxilla to piriform rim bilaterally.
- If impacting, use a caliper to measure bone to be removed and cut a wedge out.
- Lateral nasal wall osteotomies completed with guarded osteotome.
- Nasal septum separated with nasal-guarded osteotome.
- Pterygoid plate osteotomies with pterygoid osteotome (vector should be anterior and inferior).
- If multiple-piece LeFort planned, place vertical interdental osteotomies at this time (cut arch wire if not already segmentalized by the orthodontist).
- Induced hypotension (50–65 mm Hg MAP) is helpful to control bleeding. Patient must have an A-line.
- Down fracture with simple digital pressure or gently with Rowe maxillary disimpaction forceps or Tessier mobilizers.
- Check fractures and trim bony interferences of the septum and lateral walls.
  - Ensure complete mobility of the maxilla in three planes of space, should be able to passively move.
- If multi-piece is planned, cut palatal paramedian osteotomy just lateral to nasal septum. This region represents thin bone and thick palatal tissue, reducing the chance of palatal tear.
- Place splint securely to the dentition.
- Intermediate or final splint used to position maxilla with maxillomandibular fixation (MMF).
- Rotate the maxillomandibular complex up. Use a single finger to lift up complex to first point of contact. Grind as needed to achieve the correct vertical position. Do not force up or will pull condyles down out of fossae and bite will be open once you release the MMF.
- Plate maxilla into new position (plates should be at the piriform and zygomaticomaxillary buttresses).

Complications with LeFort Osteotomy

- *Bleeding* – sources include the pterygoid plexus, posterior superior alveolar artery, greater palatine artery, terminal branches of the maxillary artery. The internal maxillary artery is normally 25 mm superior to the base of the junction of the pterygoid plates in a normal maxilla. The pterygoid osteotome is 15 mm in height, leaving a 10 mm margin of safety. Treatment: attempt pressure packing with gauze and/or hemostatic agent. If no resolution, try to identify vessel for cautery. If bleeding continues consider an interventional radiology intraoperative consult for emboliza-

tion. With an extremely small maxilla, like in a cleft child or syndromic patient, a preoperative CT angiogram may be useful to identify these vessels.

- *Anterior Open Bite After MMF Release* – etiology: condyles not seated in fossa or area of premature bony contact. Remove fixation and check for bony interferences. Ensure passive condylar positioning into fossa. Replace fixation.
- *Dental Iatrogenic Injury* – recommend placement of osteotomies 5 mm above apices of roots. Observe, root canal therapy if symptomatic, or extract. Teeth may remain insensate up to around 5 years.
- *Cut Palatal Mucosa During Multi-Piece* – For a small tear, perform a simple suture repair and continue with the case. For large tear, consider replacing maxilla to original position. Do not attempt to raise flap of defect as it may further compromise blood supply and lead to avascular necrosis. The treatment in this case requires local irrigation and cover with a noncompressive splint. Formal closure is performed when revascularization is confirmed if the region has not closed spontaneously.
- *Vertical Posterior Maxillary Wall Fracture* – check globe for increased pressure. Fracture of the pyramidal process of the palatine bone can cause injury to vessels or contents of the globe. Intraoperatively redirect fracture with osteotome. Postoperatively check globe pressure, changes in visual acuity, proptosis of the globe, and pupillary response. Ophthalmology consult may be indicated. If pressures are high, may require CT orbit with contrast for evaluation or emergency lateral canthotomy.
- *Anterior Maxillary Wall Fracture* – attempt plating.
- *Midline Discrepancy* – etiology: error introduced into workup/mounting/or splint fabrication. Reposition maxilla by utilizing facial midline or dental midline or stable jaw.
- *Decrease in Maxillary Perfusion* – as denoted by poor capillary refill or purple gingiva. Replace and fixate maxilla to original position, check to ensure stents are not impinging tissue. Keep area clean to prevent infections with chlorhexidine and antibiotics until resolved. For severe delayed vascular compromise, consider hyperbaric oxygen.
- *Cut Endotracheal Tube* – tube transfer/reintubate. Consider tracheostomy or submental intubation.
- *Nosebleed (Postoperative)* – pack nose with nasal packing. If not controlled with packing, then return to the operating room to take maxilla down and control bleeding. Consider embolization.
- *Trigeminocardiac Reflex* – stop stretch on maxillae. Further anesthetize soft tissue to decrease sensitivity of CN V. Atropine or glycopyrrolate may be required to complete surgery.
- *Epiphora* – more common in high LeFort osteotomies due to damage of nasolacrimal system, nasoseptal deviation, or swelling. If no resolution after 6 weeks, CT scan to r/o source. May require dacryocystorhinostomy or nasoseptoplasty depending on etiology.
- *Pseudoaneurysm* – patient may complain of build-up pressure with release. Bleed may be early or late. CT angiogram with interventional radiology consult.
- *Nasal Septum Deviation or Buckling* – suture septum to the ANS to prevent deviation. Trim cartilaginous septum or maxillary crest for buckling.
- *Infection* – obtain imaging. Antibiotics, incision and drainage, debridement. If hardware is source of infection, then remove appropriate hardware, replace if clinically acceptable.
- *Hardware Failure* – replace hardware and consider more rigid fixation.

Surgically Assisted Rapid Palatal Expansion (SARPE)

While this section covers surgically assisted rapid palatal (or maxillary) expansion (SARPE or SARME), a brief overview of other related procedures to increase the maxillary transverse dimension is necessary for completeness.

Dental Tipping

For small transverse discrepancies up to 5 mm with healthy periodontium and upright teeth, the maxillary dentition may be tipped to facilitate expansion.

- Orthodontic rapid palatal expansion (RPE) takes advantage of growth potential in growing children and adolescents.
- A transpalatal, dental-borne, and/or microimplant borne (MARPE) orthopedic expander opens the midpalatal suture, tips teeth, and bends and remodels the alveolus.
- Older patients have more sutural resistance that results in less expansion and more dental tipping, lateral tooth displacement, and periodontal defects.
- Children have a 50% tipping and 50% expansion.
- Adolescents have 65% tipping and 35% expansion.
- High relapse (40–60% depending on age) with up to 50% overcorrection recommended.
- More widening occurs at the canines than molars (3:2).

Up to 18% of adult patients that present for orthodontic treatment have a transverse discrepancy >5 mm that cannot be corrected with orthodontics alone. After palatal suture fusion and for patients who failed RPE, two primary options for palatal expansion exist: SARPE or segmental (LF) osteotomy.

Segmental LeFort

- This is part of the definitive orthognathic surgery and treats multiple planes of occlusion and when more expansion is needed posteriorly versus anteriorly. Surgery includes interdental osteotomies to create multiple dental bearing segments that are independently mobilized, repositioned, and fixated. While this has the benefit of a single stage procedure, it is more complex, time consuming, has more minor and major complications, higher relapse (if >7 mm expansion), and less expansion potential than a SARPE.

SARPE Alone or Before Single-Piece LF

- This involves creating osteotomies in the maxilla and application of an expander for distraction osteogenesis. This may be performed alone or to obtain transverse expansion prior to a single-piece LF.
- Indications:
  - >7 mm expansion
  - Desire to avoid segmental maxillary surgery
  - Thin, delicate soft tissue with gingival recession in the bicuspid-canine region
  - Significant nasal stenosis
  - Level occlusal plane
  - Constricted V-shaped arch form

Benefits of SARPE

- Greater arch expansion
- May avoid extractions
- Better orthodontic alignment before definitive orthognathic surgery
- Improved periodontal health, esthetics, and buccal corridor

Segmental LF vs SARPE and Single-Piece LF

- For expansion >7 mm, SARPE stability (30% relapse at canine and molars) far exceeds segmental LF and RPE (50% relapse) (1).
- More expansion at the canines than the molars due to lateral nasal wall and palatine bone resistance.

Preoperative Preparation

- After determining a patient is appropriate for SARPE, the patient should stop nicotine at

least three weeks preoperatively and stop NSAIDs 1 week prior. Nutritional supplements that predispose patients to bleeding should be stopped at least two weeks preoperatively, and oral hygiene should be optimized.
- The orthodontist should tip teeth at the site of the interdental osteotomy with 3 mm between the apices, decompensate the curve of Wilson, stop all orthodontic forces, remove or segment the maxillary arch wire, provide the surgeon with the expander key, and preferably, apply the tooth or bone-borne expansion device (usually a Hyrax, Haas, or microimplant supported) prior to the OR.

SARPE Surgical Procedure

- Maxillary vestibular incision from one zygomaticomaxillary buttress to the other.
- Full thickness mucoperiosteal flap.
- Identify piriform rims, infra orbital nerve, and the pterygoid plates bilaterally.
- Bilateral maxillary osteotomies from the piriform rim to the pterygomaxillary junction that is parallel to the occlusal plane and 5 mm above dental apices.
- Release of nasal septum to avoid septal deviation during expansion (optional).
- Dissect mucosal tunnel between 8 and 9 with thin periosteal elevator to expose the facial cortex and root projections.
- Small fissure bur (#701) or sagittal safe blade to score the cortex at the osteotomy site between 8 and 9 from the alveolar crest to the nasal floor.
- Use a thin spatula osteotome between 8 and 9 and extend the osteotomy to the PNS.
  - Variation: stop midline osteotomy after 1.5 cm to avoid injury to the palatal tissue.
  - Variation: do not release the nasal septum, incise along the palatal midline, elevate two conservative midpalatal flaps, create two paramedian (2 mm lateral to the suture) osteotomies that connect posterior to the incisive foramen, suture palatal tissue closed, and cement expander.
- Perform an osteotomy of the anterior portion of the lateral nasal walls for 1.5 cm.
  - Variation: stop after 1.5 mm as the bone posteriorly is thin and offers minimal resistance to transverse expansion.
- Bilateral pterygoid plate osteotomies.
- Activate the distractor to allow passive expansion of 3–4 mm. Then, decrease the expansion to a total bony gap of 0.5–1.5 mm at the end of the procedure.
- Any perforations of palatal tissue should be closed without undermining.
- Close with an alar base cinch and V-Y closure.
- 5–7 days of latency.
- Rate and rhythm of 0.25 mm twice/day (0.5 mm/day).
- Palatal expansion must occur within 4 weeks of surgery. Allow 4 months of retention before removing the expander to get bone fill.

Complications of SARPE

- Complications similar to the LeFort procedure.
- *Periodontal Compromise Between the Central Incisors* – decrease the appliance back a few notches and reduce the rate of expansion.
- *Asymmetric Expansion* – this is the most common expansion complication and results from incomplete release of pterygomaxillary junction on one side. Half of the time, the asymmetry self-corrects. Others may require a segmental osteotomy to correct the asymmetry at least 4 weeks after the SARPE.
- *Inadequate Expansion* – inadequate surgical mobilization results in pain, dental tipping, periodontal breakdown, palatal tissue impingement by the expansion device, and post-orthodontic relapse. Treat with adequate mobilization and removal of bony interferences.

Mandibular Osteotomies

- There are various osteotomy techniques that have evolved over time. The most common osteotomy techniques are sagittal split osteotomy and the vertical ramus osteotomy.
- The workhorse of the group is the sagittal split procedure. This is followed by the vertical ramus osteotomy being the second most common technique performed.
- The mandibular position can be changed in any one of three dimensions or a combination thereof; however, it is important to realize that the final mandibular position should be governed by the maxillary position to get a functional and cosmetic result.
- When deciding on mandibular position, we must be sure to have the correct maxillary and mandibular diagnosis, which ultimately will guide our surgical plan and produce the best surgical result of the patient.
- Maxillofacial deformities are always determined by the physical exam, which may or may not be supported by radiographic findings.
- The following list of diagnoses includes important physical findings that exemplify the specific diagnosis:

Mandibular Hyperplasia

- Class 3 skeletal appearance

Mandibular Hypoplasia

- Class 2 skeletal appearance
- Micrognathia
- Short mandibular ramus
- Short mandibular body

Condylar Hyperplasia

- Mandibular asymmetry deviating the chin point to the contralateral side
- Posterior open bite on the ipsilateral side
- Maxillary cant on the ipsilateral side

Condylar Hypoplasia

- Mandibular asymmetry deviating the chin point to the ipsilateral side
- Posterior open bite on the contralateral side
- Maxillary cant on the contralateral side

Bilateral Sagittal Split Osteotomy (BSSO)

- The BSSO is the predominant workhorse procedure for various mandibular movements in orthognathic surgery.
- For mandibular setback, the BSSO is a good choice if the setback is less than 8 mm. While it is possible for larger setbacks, there is greater potential for bony interference and a posterior border step defect if the proximal end of the distal segment is passed beyond the posterior border of the proximal segment. Must always be cognizant of the posterior airway space during planned setbacks.
- BSSO can also be used for mandibular advancements. Advancements of 12 mm or more are unstable/more prone to relapse. Regardless of the vector of movement, the basic operation is the same with minor variations.
- Hunsuck modification – the medial osteotomy does not extend to the posterior ramus as opposed to the original Obwegeser medial cut. This modification allowed for a shorter split, less soft tissue stripping, and improved mandibular contour.
- Dal Pont modification – advanced the vertical osteotomy on the buccal cortex between the first and second molars. This allowed for greater advancement by allowing more bony contact surface area.
- Epker-Schendel modification – reduced stripping of the masseter and soft tissue of the medial ramus. Cut at inferior border to extend to lingual side, including entire inferior border in the proximal segment. This led to decreased postoperative swelling, hemorrhage, and reduced manipulation of the neurovascular

bundle. This also allows for easier repositioning of TMJ, reduction of relapse, and more blood flow to the proximal segment.

BSSO Surgical Technique

- Incision over the anterior border of the ramus into the mandibular vestibule to the second molar region, leave 5 mm of a non-keratinized tissue cuff to aid in closure.
- Dissection begins laterally with a full thickness mucoperiosteal flap over the body of the mandible proceeding toward the inferior border. Avoid aggressive stripping of the pterygomasseteric sling.
- The dissection proceeds up the anterior ramus to the coronoid process, and the fibers of the temporalis muscle are prudently freed.
- Medial dissection is completed subperiosteally above the lingula and the mandibular foramen to identify the entrance of the nerve. Dissection should be limited to prevent overstripping of the attached muscles, reducing blood supply to the mandibular segments. If difficulty identifying the lingula or nerve, then osteotomy should be done above the level of the mandibular occlusal plane (remember that the lingula usually is at the level of the occlusal plane).
- Make the medial corticotomy just above the lingula.
- Extend the osteotomy anteriorly through the ramus paralleling the buccal plate to the medial aspect of the second molar region.
- Drop a vertical corticotomy in the second molar region to the inferior border.
- Chisels are carefully used to slowly expand the corticotomies to split the mandible. Make sure the inferior alveolar nerve is in the distal segment.
- Medial pterygoid attachment must be released on the distal segment for advancement – best accomplished with a J stripper. In setbacks, the medial pterygoid must be dissected from the medial aspect of the proximal segment creating a pocket for distal segment positioning.
- Interim/final splint is placed and the mandible is moved to the new position.
- May use pickle fork or condylar seating instrument to push proximal segment posterior and superior. Also may evaluate alignment of inferior borders.
- Place fixation with plates or screws. L configuration for screws is more stable than a straight line. Plates allow for less nerve compression, less torque on condyle, and can be used to aid positioning of condyle.

Complications

- *Nerve Transection* – epineural repair with a 7-0 non-resorbable suture.
- *Bad Split (Buccal/Lingual Plate Fracture)* – complete osteotomies and set to planned occlusion. Fixate fractures with plates and screws as you would a traumatic fracture (single bicortical positional screw for a lingual plate fracture). Stabilize as planned to new position. If segment is small and attached to the periosteum, consider leaving it.
- *Subcondylar Fracture* – complete osteotomies. Set to planned occlusion. Fixation with plates and screws or consider IMF.
- *Infection* – imaging to review if hardware/screw loosening. Antibiotics, incision and drainage, debride, or hardware removal if appropriate.
- *Bleeding* – most likely due to damage of the inferior alveolar artery. Attempt packing as normally this will spontaneously correct. Important to stay in periosteal pocket to prevent retromandibular vein or facial artery damage.
- *Bite Discrepancy/Malocclusion* – remove fixation. Ensure that the condyles are seated in the fossae and replace fixation.
- *Condylar Sag* – manifests as unilateral malocclusion after removing IMF. Remove fixation, reposition condyle in the fossa, and reapply fixation (3).
- *Condylar Resorption* – associated with skeletal class 2 deformities with high mandibular planes (higher incidence in female patients)

that require large advancements. Treatment involves bite splint therapy and anti-inflammatory medications. More severe cases may warrant synovectomy, costochondral grafting, or TMJ replacement.
- *Non-union* – reoperation of the sites. Freshen bony margins and remove fibrous tissue. Apply more rigid fixation and consider bone grafting the sites. Consider period of IMF.

Bilateral Intraoral Vertical Ramus Osteotomy (BIVRO)

- When mandibular set back is considered, in addition to BSSO, the surgeon and patient also have the option of BIVRO.
- There are nuances of the BIVRO that need to be taken into account.
  - Best operation for V-shaped mandible (divergent ramus pattern).
  - Incidence of both short- and long-term paresthesia is less with BIVRO based on osteotomy location and design.
  - Also, when evaluating the anatomy of the mandible, a very thin mandibular ramus with little marrow space may be more suited for BIVRO over BSSO, given the increased risk of unfavorable split with the latter osteotomy in this circumstance.
  - This operation should also be considered with large mandibular asymmetrical prognathism. As the mandible is rotated, a vertical ramus osteotomy may reduce the incidence of segmental interference on the setback side.
  - Lastly, in patients who have symptomatic TMD preoperatively that have not been addressed either non-surgically or with TMJ surgery, an IVRO may be of benefit, given less potential pressure on the intracapsular tissues.
  - Some potential downsides to BIVRO are the need for a period of IMF.
  - If there is a large counterclockwise movement planned, the proximal end of the distal segment will rotate inferiorly lengthening the pterygomandibular sling and potentially pulling on the proximal segment. Accommodations are necessary if this is the case including possibly wiring the segments together to counteract this force.
  - Can create splaying of the proximal bony segment creating fullness in the region of the mandibular angle, more prominent with larger setbacks.

IVRO Technique

- Incision is made over anterior border of the ramus into the mandibular vestibule.
- Full-thickness mucoperiosteal flap is developed exposing the lateral ramus from the inferior border up to the sigmoid notch.
- A J stripper is used to release attachments of the inferior border.
- Bauer retractors are then inserted into the sigmoid notch and along the inferior border to provide retraction. Some surgeons use the Merrill-Levasseur retractor along the posterior border.
- Identify the antilingula as this marks the anterior limit of the osteotomy.
- Make vertical cut utilizing an oscillating saw blade beginning in the midramus region posterior to the antilingula to the inferior border. (If you cannot identify the antilingula, a cut 7–10 mm anterior to the posterior border provides safety.)
- The superior osteotomy is completed last as there is a risk for insult to the masseteric artery. This will provide quicker access upon completion of the osteotomy to manage the bleeding.
- On the medial aspect of the mandible, judiciously dissect a subperiosteal pocket to accept the overlapping segment.
- Manipulate the proximal segment laterally.
- Trim excess inferior portion of the bony proximal segment.
- Establish occlusion with MMF (6 weeks of MMF, then training elastics).
- If segments are passively positioned and in approximation, rigid fixation with two or three screws can be done easily through a transfacial trocar.

Complications

- *Infection* – antibiotics, incision and drainage, debridement as needed.
- *Bleeding* – injury to internal maxillary artery branches (most commonly the masseteric branch) at sigmoid notch. Direct pressure with gauze. Consider application of hemostatic agents. If can identify bleeding vessels, consider hemoclips. If uncontrolled, consider embolization. The masseteric artery on average is 8 mm above the sigmoid notch and 25 mm from the anterior border of ramus.
- *Displaced Proximal Segment* – distract mandible anteriorly and reposition. Evaluate for medial bony interferences and reduce as necessary.
- *Distraction of Condylar Segment* – may occur early in treatment or weeks after surgery. Aggressive elastic traction treatment should be attempted. May require revision and placement of fixation/wire placement.
- *Inadvertent Subcondylar Osteotomy* – place patient in 6-week of IMF and complete surgery after establishment of bony union.

Genioplasty

- Genioplasty is occasionally necessary to help balance the patient cosmetically.
- When the genial tubercles are included in the osteotomy, anteriorly repositioning the bone can help in opening up the lower airway in sleep apnea patients. This is usually referred to as anterior mandibular horizontal osteotomy with genioglossus advancement.
- Careful examination of each of the facial units is critical to obtain optimal outcomes both functionally and aesthetically.

Genioplasty Surgical Technique

- The incision should not be done in the depth of the vestibule but rather half of the distance between the vestibule and the wet line of the lower lip. Incision from canine to canine through the mucosa and submucosa.
- Incision is carried through the mentalis muscle down to the bony mandible.
- A subperiosteal dissection proceeds to the most inferior portion of the bony chin and laterally identifying the mental nerves and foramina bilaterally.
- Mark the midline of the chin with a hand-piece (other measured markings are occasionally used). A prefabricated cutting guide created with virtual surgical planning (VSP) can increase accuracy of the chin repositioning.
- Using a reciprocating saw to make a cut beneath the mental foramen (5 mm below the foramina and 5 mm below the apices of the teeth) at the inferior border of the mandible to a point at least 10 mm above the inferior border at the midline; this is done bilaterally. (Bone wedges or an osteoplasty can be performed for asymmetric movements.)
- Bony chin is mobilized and separated from the mandible and repositioned using a measured bone plate or fixation screws (a surgical guide can be used to position the bony chin if necessary).
- Closure should be accomplished in two layers. It is important to reapproximate the mentalis muscle to prevent ptosis of the chin. The mucosa is then closed with resorbable sutures.

Complications

- *Ptosis* – avoided by aggressive soft tissue dissection and ensure reapproximating the mentalis muscle. Reopen wound and reapproximate mentalis muscle. Placement of pressure dressing.
- *Malposition of Chin* – remove plate and realign.
- *Injured Root Apices* – observe or possible root canal in the future.
- *Nerve Injury* – ensure during dissection that osteotomies are 5–6 mm away from mental foramina. Epineural repair with 7-0 suture for observed transection.

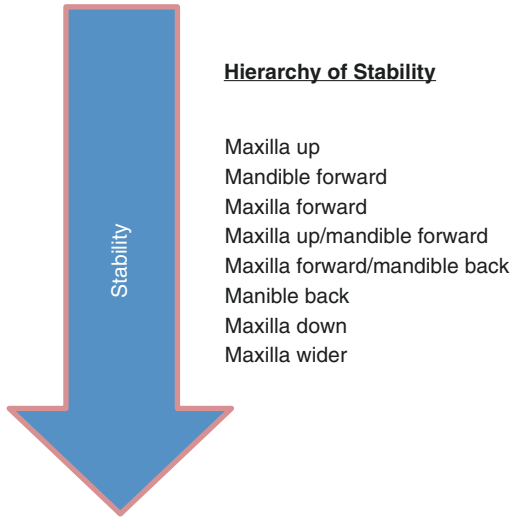

Fig. 5.8 Stability of orthognathic movements. It is important to note that this hierarchy deals primarily with changes and stability of the occlusion rather than close evaluation of skeletal changes. (Modified from Proffit et al. [2])

- *Malunion* – debride interposing fibrous tissue and reposition with new hardware. Consider adjunctive bone grafting.
- *Infection* – incision and drainage, antibiotics, debridement, or remove hardware.
- *Hardware Failure* – remove hardware and replace if no evidence of union.

Hierarchy of Stability

There is a hierarchy of stability as it relates to movements accomplished with orthognathic surgery. In 1996, Turvey, Profitt, and Phillips conducted a study that has become an established paradigm for the hierarchy of stability (Fig. 5.8). In 2007, the aforementioned authors wrote a paper that updated this hierarchy to include procedures where rigid fixation is used (2).

Orthognathic Surgery Considerations in Cleft Lip and Palate Patients (CLP)

Management of patients with orofacial clefts requires a multidisciplinary approach in order to provide optimal care. Listed below are members of the CLP team that are part and parcel to successful management of these patients.

- Patient care coordinator
- Pediatrics/primary care/geneticist
- Surgeon (oral and maxillofacial surgery, plastic surgery, or otolaryngology)
- Pediatric dentistry/general dentistry
- Orthodontist
- Speech pathology
- Audiology
- Psychology
- Social work

Orthognathic surgery is performed when growth is complete prior to secondary nasal revision. Contemporary orthognathic treatment of cleft patients is aimed at correcting the hypoplastic abnormality. A mandibular setback procedure should be avoided if the maxilla is hypoplastic. Orofacial cleft patients oftentimes can present with large anterior-posterior discrepancies. A decision must be made if the patient is a candidate for a traditional orthognathic procedure or if they require distraction osteogenesis. After careful evaluation of the physical and radiographic findings, an appropriate treatment plan can be formulated and surgery undertaken.

As it is not the topic of this section to review orthognathic surgery, only the important aspects involved in treating orofacial cleft patients will be reviewed. Specific areas to consider include the following:

- Intubation
- Incision design in the maxilla and vascularity
- Osteotomy and down fracture
- Management of residual oronasal fistulas and altered nasal anatomy
- Rigid fixation and bone grafting

Intubation

Oftentimes orofacial cleft patients may have previously undergone pharyngeal flap procedures in order to address velopharyngeal insufficiency. This must be recognized and discussed with the anesthesiologist. Different techniques can be

used to ensure atraumatic placement of the nasal endotracheal tube through a lateral port of the pharyngeal flap without trauma to the flap or the posterior pharyngeal wall.

- Red-rubber catheter placement and visualization in the posterior pharynx with which the nasal endotracheal tube is then guided through a lateral port of the flap.
- Endoscopic or fiberoptic guidance.
- Nasopharyngeal airways can be used to guide a bougie into the appropriate position, which the endotracheal tube is then passed over.

Incision Design in the Maxilla and Vascularity

The vascular supply to the maxilla and premaxilla is critical. Due to previous surgeries and scarring, it can be more fragile than in a non-cleft patient. Most of the blood supply to the mobilized maxilla is derived from the palatal tissues.

Alterations in incision design that can be considered include the following:

- Shortened circumvestibular incision leaving a large buccal soft tissue pedicle.
- An anterior midline pedicle can be left with a vertical incision at the midline and two lateral vestibular incisions.

It is ideal to attempt to preserve the descending palatine vessels if possible and care should be taken during separation of the lateral nasal walls and down fracture to avoid unnecessary trauma. Maxillary segmentation should be approached with caution. A compromise in the posterior occlusion and finishing with a slight posterior crossbite or edge-to-edge occlusion may be more desirable than the risk of avascular necrosis of a segment. If segmentation is planned to address a severe transverse discrepancy, the best location is the cleft site.

Remember, the cleft patient has no bone in the midline. The only transverse stability is from a well-healed alveolar cleft bone graft. Once that needs to be cut to achieve better arch form, a transpalatal device is needed (splint, Hyrax, TPA) to allow for the alveolus to heal again.

Osteotomy and Down Fracture

- The greatest areas of buttressing in the maxilla are the pyriform and pterygomaxillary buttresses. Care must be taken when completing the osteotomy to ensure careful separation in these areas.
- Failure to do so can lead to unfavorable fractures into the skull base or superiorly into the orbit. In addition, leaving an anterior pedicle in the incision design can make down fracture more difficult, making it even more crucial to ensure that the osteotomy is completed and buttresses weakened sufficiently.
- Too much pressure during down fracture can lead to fracture at a thin alveolar graft site and lead to transverse instability.

Management of Residual Oronasal Fistulas and Altered Nasal Anatomy

- Once down fracture is achieved, the nasal floor and nasal cavity must be examined for the following:
  - Residual oronasal fistulas
  - Septal deviation
  - Enlarged inferior turbinates
  - Alar support
- Closure of any residual fistulas should take place as well as any needed additional bone grafting.
- If significant septal deviation is noted, a septoplasty may be indicated.
- Inferior turbinectomy can be considered at this juncture as well if they are large or may impinge on the nasal floor.
- Time should be spent stretching the soft tissues to allow for planned vertical and anterior-posterior movements passively.

Rigid Fixation and Bone Grafting

- Soft tissues must be stretched to allow for planned vertical and anterior-posterior movements without tension.

- Bone contact is paramount to achieve union in this patient population. Bone grafting may be considered at buttress regions.
- This can be autogenous for large gaps or allogeneic for smaller gaps.
- Fixation should be achieved with the largest or strongest possible rigid fixation system to reduce relapse.

Final Considerations

With a small alveolar cleft graft, the area of the graft may be weak and prone to fracture during mobilization and down fracture of the segment. Consider a palatal splint preoperatively or plan for a final splint with holes to allow it to be wired into place to stabilize the mobile segments. If an alveolar cleft graft was not undertaken, then a palatal splint and/or occlusal splint will be necessary. In addition, the splint may assist in repositioning of the lesser segment to minimize the size of the cleft for bone grafting.

Cleft Distraction Osteogenesis

- Distraction osteogenesis is considered in patients with large anterior-posterior discrepancies that would not be possible to correct in a single orthognathic procedure or which would require unnecessary mandibular setback procedures.
- There are two commonly employed methods of distraction osteogenesis at the LeFort I level:
  - Rigid external maxillary distraction
  - Internal maxillary distraction

Rigid External Maxillary Distraction

- The orthodontist will start by fabricating an intra-oral splint that will attach to the orthodontic appliances with extra-oral extensions. The extension sits in the paranasal regions. These are used to deliver the distraction forces.
- The LeFort I osteotomy is completed in the same manner and consideration as described above.
- Adequate mobilization is critical.
- A cranial halo is placed with an external adjustable distraction screw system. A rigid down-rod is attached to the halo, which is used to attach wires to the extra-oral component of the orthodontic splint.
- Using the principles of distraction osteogenesis including a latency period of about 5–7 days, activation phase, and consolidation phase, advancement of the maxilla is undertaken.

Advantages:

- Adjustable vector of distraction throughout the activation phase.
- Does not require secondary surgery for removal; removal is complete in the office.

Disadvantages:

- Psychosocial aspects of wearing a large extra-oral appliance.
- Does not offer retention and requires the use of reverse pull headgear after removal.

Internal Maxillary Distraction

- Typically, a stereolithographic model is obtained preoperatively and the planned Lefort I osteotomy is marked on the model.
- The intra-oral appliances are then pre-bent and modified preoperatively to achieve the most appropriate distraction vector.
- The devices are sterilized and again the LeFort I osteotomy is completed as previously described.
- The intra-oral device is anchored superiorly in the region of the zygomatic buttress and inferiorly to the osteotomized segment via circumdental wires. The same principles of distraction are employed.

Advantages:

- Lack of a large extra-oral halo device.

- Reverse pull headgear is not necessary as the devices can be left in place after consolidation with removal of the activation arm.

Disadvantages:

- Unidirectional vector that cannot be altered after placement.
- A second surgery is required for removal of the device; this may provide an opportunity for placement of rigid internal fixation plates if desired.

Orthognathic Case

The patient is a 22-year-old male patient with no past medical history. He denies any medication use and has no allergies. The patient's chief complaint is, "It is hard for me to bite my food because of my underbite."

Describe your clinical workup for this patient.

Patient will need photos, dental models with CR records, and clinical measurements.

*Describe what you see in the photos below (*Fig. 5.9*).*

- In the frontal repose image, you can appreciate that the overall facial profile is dolichocephalic. The facial thirds appear congruent. The maxillary dental midline is coincident with facial midline. The mandibular dental midline is deviated to the left. There is an appropriate amount of incisor show at rest and the interlabial gap is within normal limits. There is increased scleral show and paranasal flatness. There is no appreciable asymmetry of the eyes or ears. The bigonial width appears to be less than the bizygomatic width.
- On the profile view, the patient appears to be prognathic. There is volume deficiency of the midface with a negative vector of the orbit. There is a break in the cheek bone-nasal base-lip curve contour. The nasolabial angle appears within normal limits.
- In the animation image, there is an appropriate amount of incisor show. The nasolabial folds deepen during animation, which suggests midface deficiency. There is no appreciable occlusal cant and the mandibular dental midline appears coincident with the mandibular skeletal midline.

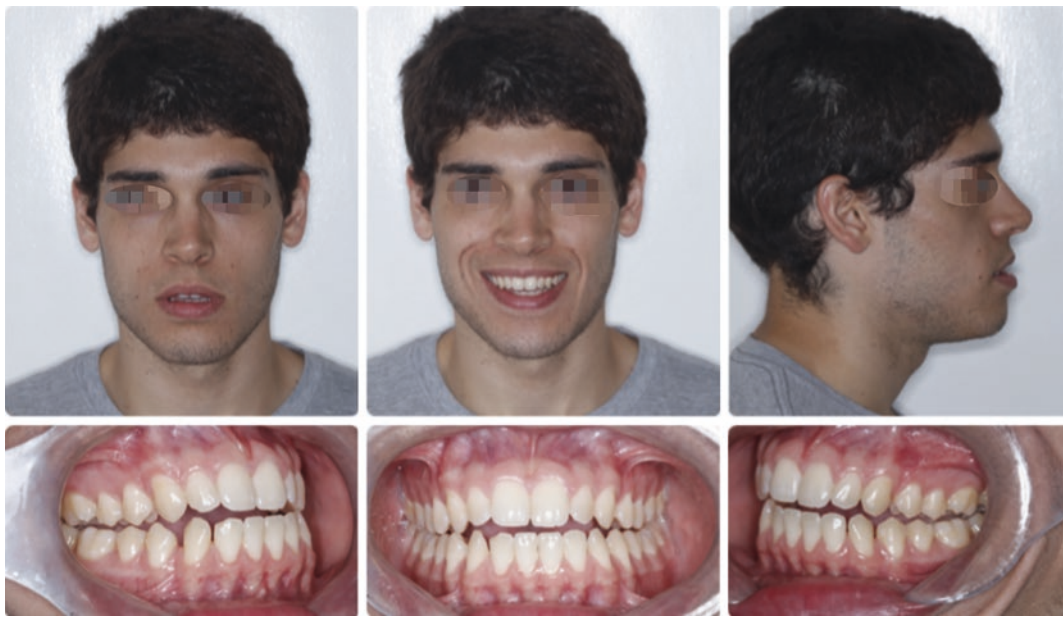

Fig. 5.9 Facial and intraoral photos of orthognathic case patient. (Courtesy of Dr. Gregg Jacobs)

- Intraorally, there is a class I cuspid and molar occlusion on the right and a class III on the left with a midline discrepancy. There appears to be a biplanar maxillary occlusal plane. There is a crossbite relationship (transverse discrepancy) on the left side. There appears to be excessive interdental spacing in the mandibular canine-premolar-canine region. There is appreciable apertognathia with mamelons present on the anterior teeth. The periodontium appears healthy and oral hygiene appears appropriate.

How would you like to proceed next?

- At this stage, you may request to see radiographs (orthopantogram, lateral cephalogram, and possibly a PA cephalogram if necessary).

Here you are provided with the orthopantogram and lateral and PA cephalogram. Describe what you see (refer to Figs. 5.10, 5.11, and 5.12 *below).*

- In the orthopantogram, you can appreciate a dental midline discrepancy. The condyles are seated within the fossae; however, the left condyle is seated slightly forward on the eminence indicating that in the image the patient is in centric occlusion and perhaps not centric relation. There are mild degenerative changes noted in the condylar heads. The maxillary sinuses appear well pneumatized. The inferior alveolar nerves are proximal to the inferior border in the second molar region. Third molars are present and erupted. There is no overt dental pathology noted (no radiographic evidence of periodontitis or caries). There are intracoronal restorations noted.
- The lateral cephalogram demonstrates a class III facial-skeletal malocclusion with nearly edge-to-edge anterior occlusion. The inferior borders are well aligned and there is slight flaring of the maxillary and mandibular incisors.
- The PA cephalogram reveals a true mandibular skeletal asymmetry to the left (mandibular skeletal midline is rotated to the left of the facial and maxillary midline).

What is your diagnosis?

This patient's skeletal diagnosis includes AP and vertical maxillary hypoplasia, mandibular prognathism, and mandibular asymmetry with dental compensation. There is a slight microgenia. The dental diagnoses include an angle class 3 malocclusion and apertognathia.

***Author's Note:** At this stage, you should be able to establish a diagnosis list even without a cephalometric tracing.

What is the next step?

You would suggest that this patient undergo presurgical orthodontics to level, align, and coordinate his arches and place teeth over basal bone in preparation for definitive orthognathic surgery.

What is your surgical treatment plan?

Given that the skeletal diagnosis involves both arches, prior to orthodontic preparation, you may suggest that this patient requires a LeFort I osteotomy for maxillary advancement, possibly in segments should a two-plane maxillary arch still be present, and a BSSO or IVRO for asymmetric mandibular setback.

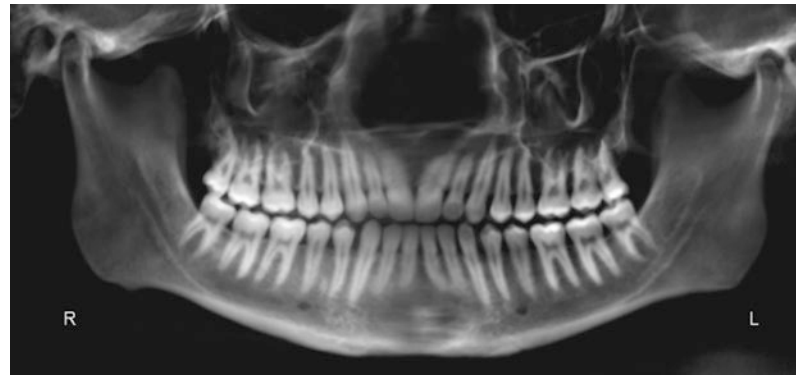

Fig. 5.10 Orthopantogram of orthognathic case patient. (Courtesy of Dr. Gregg Jacobs)

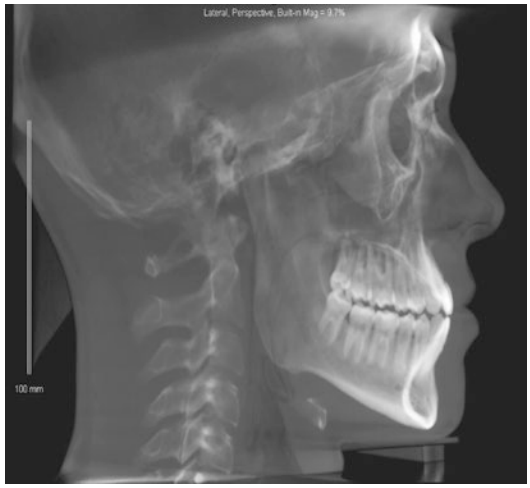

Fig. 5.11 Lateral cephalogram of orthognathic case patient. (Courtesy of Dr. Gregg Jacobs)

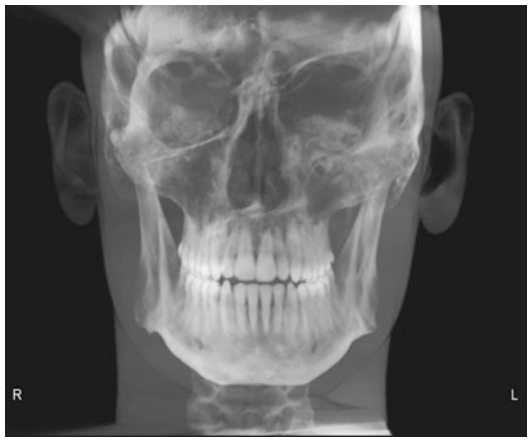

Fig. 5.12 PA cephalogram of orthognathic case patient. (Courtesy of Dr. Gregg Jacobs)

Patient has completed presurgical orthodontics and has surgical wires and hooks in place. Patient returns to your office and is now ready for surgery. Your final treatment plan is for a LeFort I osteotomy and a BSSO. Today is the day of surgery and the patient is in the preoperative holding area. What would you want to discuss with the anesthesiologist?

I would request a nasal intubation. In anticipation of blood loss, I would request a type and cross in addition to an accurate blood loss and fluid count. An arterial line would be important for accurate blood pressure intraoperatively. I will be requesting hypotensive anesthesia during down fracture of the maxilla. With a long operating time and high blood loss possibility, I would request placement of a Foley catheter. I would warn them about possible maxillomandibular fixation on waking up. Patient may require an overnight stay in the ICU or step down unit.

What is the difference between a type and screen and a type and cross?

Type and screen determines blood type, Rh factor, and antibodies in case a blood transfusion is required. A type and crossmatch tests determines the same information but matches a unit of blood to be prepared for immediate transfusion.

A patient returns to your office for a second opinion following orthognathic surgery with a posterior open bite on the right. You take a CBCT and you see this (Fig. 5.13). *What happened and how do you manage it?*

During seating of the proximal and distal segments, the mandibular condyle is torqued laterally

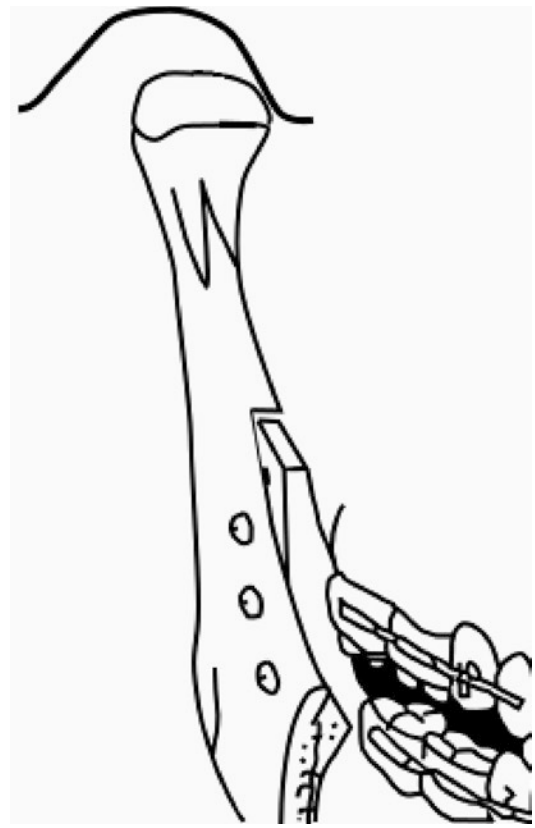

Fig. 5.13 Orthognathic complication. (Modified from Reyneke and Ferretti [3])

out of the fossa because of bony interferences along the ramus while fixation is taking place; once the IMF is released, the condyle will reseat itself into the fossa and the occlusion will deviate to that side.

This should be noticed immediately and corrected by removal of the plates and screws, reapplication of the IMF into the splint, and re-fixation with the condyle (proximal segment) seated appropriately within the fossa. The surgeon can avoid this issue by ensuring there are no bony interferences as the condyle is seated and the proximal segment is laid flush with the distal segment prior to plating. Very little pressure should be necessary to achieve flush seating. If this is not recognized and a maxillary osteotomy is performed after the mandible, the final occlusion may fit well into the splints but will be off from a facial perspective with the midline deviating toward the side of the condylar dislocation. This may, in severe cases, necessitate revision of both osteotomies. It is best to look for this at the time the IMF is released and addressed immediately. If a single jaw surgery is being performed and the deviation is small, then this may be able to be compensated for with postsurgical orthodontics.

Obstructive Sleep Apnea (OSA)

The surgeon needs to be familiar with the diagnosis, key points of the physical examination, questionnaires, analysis of diagnostic imaging, and the use of polysomnography. Treatment is multidisciplinary and consists of both nonsurgical and surgical modalities that should be familiar to the oral and maxillofacial surgeon.

Definitions of Basic Terms

- Obstructive Sleep Apnea – a sleep disorder characterized by obstructive apneas and hypopneas caused by collapse of the upper airway during sleep.
- Central Sleep Apnea – the absence of respiration associated with an absence of respiratory effort.
- Polysomnography (PSG) - a diagnostic test used for the evaluation of sleep disorders. Components include EEG, EOG, EMG, ECG, and pulse oximetry with or without a video recording of the subject. It measures several variables indicative of sleep apnea. The PSG is considered the gold standard in the diagnosis of sleep apnea.
- Apnea – a cessation of airflow at the nostrils and mouth for at least 10 seconds while sleeping.
- Hypopnea – a reduction of airflow resulting in a drop-in oxygen saturation followed by an arousal.
  - 50% reduction in airflow for 10 seconds with a 3% drop in oxygen saturation or
  - 30% reduction of airflow for 10 seconds with a 4% drop in saturation.
- Apnea Index – the apnea index is the average number of apneic events per hour.
- Hypopnea Index – the average number of hypopnea events per hour.
- Apnea/Hypopnea Index – the average number of apnea and hypopnea events per hour (Table 5.1).
- Respiratory Disturbance Index – the average number of apnea events, hypopnea events, and respiratory event related arousals (RERAs) per hour.
- RERA – an event that causes an arousal or a decrease in oxygen saturation, without qualifying as apnea or hypopnea.
- Cheyne Stokes Breathing – a breathing pattern marked by crescendo-decrescendo changes in airflow and respiratory effort that often ends with apnea (typical of central sleep apnea syndrome).
- Mueller's maneuver – inhalation with the nasal passages occluded and the mouth closed with an endoscope inserted through one nostril to observe the location of airway collapse.
- Fujita Classification – a classification system developed to indicate the level of obstruction identified by nasopharyngoscopy in conjunction with a Mueller's maneuver or during sleep-induced nasopharyngoscopy. It can be used as a

Table 5.1 AHI values

0–4	Normal
5–15	Mild OSA
15–30	Moderate OSA
>30	Severe OSA

guide as to what surgical interventions may be useful in alleviating the source of obstruction:
- Type I – upper pharynx to include the palate, uvula, and tonsils.
- Type II – upper and lower pharynx.
- Type III – lower pharynx to include the tongue base, lingual tonsils, and supraglottic region.

Functions of Sleep

- Sleep serves a variety of functions that are essential to human physiology. Secretion of growth hormone peaks during sleep, which is particularly important to growing children. During sleep, the body repairs damage, clears waste products, and revitalizes. It is crucial for proper brain function, as sleep promotes brain plasticity through synapse formation and maintenance.
- Conversely, those suffering from lack of sleep suffer from slow reaction time, difficulty in problem-solving, and problems with short-term memory as well as a host of systemic problems outlined below.
- There are two cycles of sleep (non-REM and REM sleep).
- The first cycle of sleep starts with wake, transitioning to N1, then to N2, then to N3, and finally to REM. As the cycles progress throughout the night, the percentage of REM sleep in each cycle gradually increases. Conversely, the percentage of stage N3 tends to decrease over the course of the night, with the largest amount of N3 in the first half of the night.
- It appears that REM sleep appears to be the most important in maintaining vitality, as interruption of REM sleep appears to have the greatest impact on health.

Stages of Normal Sleep

Non-rapid Eye Movement Sleep

- *N1* – (5–10% of sleep time) the lightest stage of sleep. Characterized by slow rolling eye movements and low–amplitude, mixed EEG frequencies.
- *N2* – (45–50% of sleep time) marks the first appearance of sleep spindles and K-complexes.
- *N3* – (10–20% of sleep time) deep sleep characterized by low-frequency, high-amplitude EEG waves. Tends to occur more in the beginning of the night and sleepers are difficult to arouse.

Rapid Eye Movement (REM) Sleep

- (18–20% of sleep time) Characterized by a low-voltage, mixed EEG pattern (saw-toothed waves), rapid eye movements, and muscle atonia.
- Consists of two phases:
  - *Phasic* – bursts of rapid eye movements, respiratory variability, and brief EMG activity.
  - *Tonic* – limited motor activity with few eye movements.
- REM sleep may be delayed or suppressed by alcohol, sedative-hypnotic drugs, barbiturates and other antiepileptic drugs, beta antagonists, monoamine oxidase inhibitors, selective serotonin reuptake inhibitors, and stimulants.

Pathophysiology of OSA

- OSA causes the patient to have an increased sympathetic tone, which leads to autonomic arousals.
- The hypoxia associated with OSA leads to sleep fragmentation and restriction.
- Hypoxia followed by oxygenation can also lead to production of free radicals and endothelial damage via hypoxia-reperfusion injury. There is also activation of PMNs and the release of inflammatory mediators that cause further damage.
- Over time, this leads to a chronic inflammatory state.
- OSA is much more common in males aged 18–60 years old.

Medical Conditions Associated with OSA (Table 5.2)

Table 5.2 Medical conditions associated with OSA

Cardiovascular	*Hypertension* *Arrhythmias* (sinus bradycardia most common, also PVCs and other more serious in advanced disease) *Congestive heart failure (right sided heart failure)* *Myocardial ischemia/infarction* (probably due to endothelial damage)
Neurological	*Stroke* (strong statistical correlation) *Parkinson's disease* (some PD patients show obstructive breathing patterns) *Seizure disorders*
Endocrine	*Diabetes and insulin resistance*
Psychiatric	*Cognitive function* (decreased reaction time, concentration, memory) *Depression* (strong evidence of overlap)
Pulmonary	*Pulmonary hypertension* (strong correlation)
Digestive	*Acid reflux*
Other	*Decreased wound healing* *Morning headaches* *Immune system impairment* *Secondary polycythemia* *Erectile dysfunction and impotence*

Diagnosis and Workup for Sleep Apnea

Health History

In patients with known OSA, the health history should focus on those medical comorbidities strongly associated with OSA. Many symptoms can be elicited from the STOP BANG (4) or Epworth Sleepiness (5) questionnaires and, therefore, they are useful screening tools.

STOP-BANG Questionnaire (Score out of 8)

STOP

Do you **S**NORE loudly (louder than talking or loud enough to be heard through closed doors)?

Do you often feel **T**IRED, fatigued, or sleepy during the daytime?

Has anyone **O**BSERVED you stop breathing during your sleep?

Do you have or are you being treated for high blood **P**RESSURE?

BANG

BMI more than 35 kg/m^2?

AGE over 50?

NECK circumference >16 inches (40 cm)?

GENDER: Male?

Interpretation

High risk of OSA: Yes 5–8.

Intermediate risk of OSA: Yes 3–4.

Low risk of OSA: Yes 0–2.

Epworth Sleepiness Scale

Age (Yrs.): _____.

Sex (Male = M, Female = F): _____.

Use the following scale to choose the **most appropriate number** for each situation:

0 = would **never** doze

1 = **slight chance** of dozing

2 = moderate chance of dozing

3 = high chance of dozing

It is important that you answer each question as best you can.

Chance of Dozing (0–3)

Sitting and reading - _____

Watching TV - _____

Sitting, inactive in a public place - _____

As a passenger in a car for an hour without a break - _____

Lying down to rest in the afternoon when circumstances permit - _____

Sitting and talking to someone - _____

Sitting quietly after a lunch without alcohol - _____

In a car, while stopped for a few minutes in the traffic - _____

Interpretation

0–5 Lower Normal Daytime Sleepiness

6–10 Higher Normal Daytime Sleepiness

11–24 Excessive Daytime Sleepiness

Pertinent Physical Exam Findings

Body Habitus

- Obesity (BMI >30 kg/m^2) has a strong causative correlation with OSA. Many obese patients have OSA, and weight loss can result in alleviation or resolution of symptoms. Neck and waist circumferences are more relevant than BMI alone.
- Obesity results in fat deposition in the uvula, tonsils, tongue, aryepiglottic folds, lateral pharyngeal wall, and between the medial and lateral pterygoids. The most frequent site of

collapse, as well as the area of greatest fat deposits, is the lateral pharyngeal wall.
- There is a strong correlation between OSA and neck greater than 16 inches in females and 17 inches in males.
- Lung volumes are reduced due to fat deposition and decreased compliance. There is decreased FRC and expiratory reserve volume due to displacement of the diaphragm into the chest by visceral fat. Ventilation is shifted to the upper portions of the lungs resulting in a worse V:Q mismatch and lower PaO_2.

Airway Examination

- Examination of the occlusion via clinical exam and lateral cephalogram is essential.
- A class II dentoskeletal malocclusion resulting from retrognathia can be associated with a narrow posterior airway space and OSA.
- Micrognathia can also be a source of obstruction in the OSA patient. Macroglossia can be caused by obesity as well as some genetic abnormalities (i.e., Down's syndrome) and can be a source of obstruction.
- Other sites of obstruction may be due to an elongated uvula/soft palate nasal septal deformities, enlarged turbinates, and tonsillar/adenoid hypertrophy.
- Evaluation should include Mallampati score (Table 5.3). For each increase in class, the odds ratio of having OSA increase by 2.5 (6).

Diagnostic Aids

Nasopharyngoscopy

- Nasopharyngoscopy is the use of a flexible fiberoptic laryngoscope to examine the lumen of the nasal passages, oropharynx, and vocal cords.

Table 5.3 Mallampati classification

Class I	Complete visualization of the soft palate, uvula, and fauces
Class II	Complete visualization of the uvula and soft palate
Class III	Only base of uvula visible
Class IV	Soft palate not visible

- When used in conjunction with a Mueller's maneuver, it can identify potential sites of obstruction and can be performed both in the supine and in the sitting positions.
- In experienced hands, it can easily be performed in an office setting with only topical or aerosolized local anesthetic.

Drug-Induced Sleep Endoscopy (DISE)

- DISE is nasopharyngoscopy performed on a patient that is undergoing sedative-induced sleep.
- Anesthetic agents blunt the negative pressure reflex, thus mimicking the conditions of REM sleep.
- It is usually performed in the operating room in a controlled environment where airway intervention can be safely performed if necessary.
- This technique can identify the areas of collapse in the pharynx and direct site-specific surgical intervention.

Polysomnography

- Full night, in-laboratory, attended polysomnography (PSG) is considered the gold standard for diagnosis of obstructive sleep apnea.
- Those patients who are diagnosed with OSA and elect CPAP therapy are brought back for another night of PSG during which their device is titrated.
- Split night PSG is similar except that PSG is performed during the first half of the night, and as OSA is diagnosed, a CPAP is titrated during the second part of the night.
- Despite the fact that it is considered the gold standard of OSA diagnosis, a negative result should be viewed with skepticism in patients that have a high suspicion of OSA.

Home Sleep Apnea Testing (HST)

- An alternative to in-laboratory PSG is home sleep apnea testing (HST). This has been tested and validated against standard PSG and has been shown to have high sensitivity and specificity in appropriate patients.

- It is useful in patients who have a high probability of having moderate to severe sleep apnea, patients who do not have associated medical comorbidities, or patients that are suspected of having components of central sleep apnea.
- It is limited in that it can only detect breathing-related sleep disorders and it often underestimates AHI.

Imaging

Cephalometric Analysis

- The lateral cephalogram is a valuable tool in assessing various aspects of the maxillofacial skeleton that are positively correlated with obstructive sleep apnea.
- Its advantages are that it is relatively inexpensive, easily obtained, and widely available.
- It is important to recognize that cephalograms are useful in identifying potential sources of obstruction but are not useful in predicting surgical success.

Concerning Cephalogram Signs (Fig. 5.14):

- Retrognathia – a posteriorly positioned mandible can be indicative of a narrow posterior airway space making the patient susceptible to airway collapse.
- Posterior Airway Space (PAS) – a measurement of the airway along a line that bisects B point and gonion through the posterior airway space. A PAS of less than 11 may indicate a base of tongue obstruction, making the candidate a poor candidate for UPPP.
- Increased dimension of the soft palate (P to PNS) – the length of the soft palate as measured from the posterior nasal spine to the tip of the soft palate. Normal measurement is 37 ± 3. Increased length is associated with OSA.
  - Position of the hyoid (H to MP) – the distance of the hyoid bone to the inferior border of the mandible along a line perpendicular to the mandibular plane angle. An inferiorly positioned hyoid (greater than 15 mm) is indicative of a longer airway and is correlated with UPPP failure.

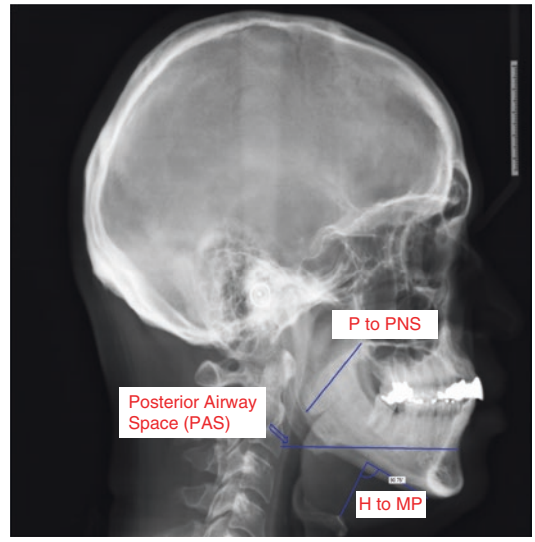

Fig. 5.14 Illustration of PAS, P to PNS, H to MP on lateral cephalogram. (Courtesy of Dr. Joseph Ivory)

Cone Beam Computed Tomography (Fig. 5.15)

- CBCT can be performed on OSA patients to identify the examination of the airway in sagittal, axial, and coronal planes.
- Certain programs allow volumetric, three-dimensional reconstruction of the airway.
- Most programs allow reconstruction of a panoramic radiograph, lateral cephalogram and PA cephalogram as well, making it useful for examination and planning of surgery.
- In addition, postoperative CBCT can be performed to examine the effects of global airway procedures if desired.

Magnetic Resonance Imaging

- Like DISE, MRI can be done under sedation to detect site of obstruction and plan surgery.
- It gives excellent anatomic detail of the soft tissues surrounding the airway and can detect fatty deposits around the pharynx.
- It can be useful in patients with obesity, craniofacial syndromes, and neuromuscular disorders.

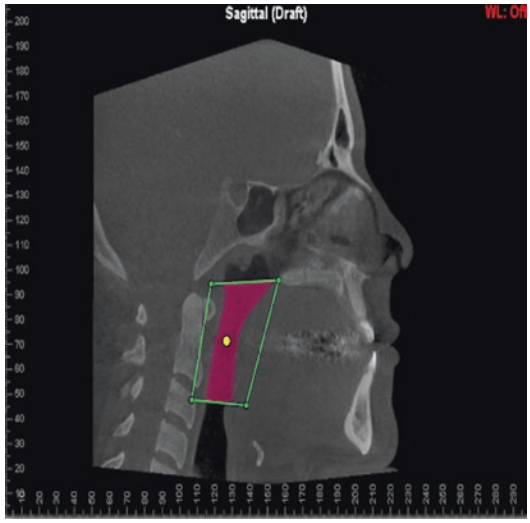

Fig. 5.15 Volumetric analysis of CBCT. (Courtesy of Dr. Joseph Ivory)

Nonsurgical Therapies

- Weight Reduction – can result in reduction in the severity of OSA, lower pressure requirements for CPAP, and in some cases offer resolution.
- Oral appliances can be used as a first-line therapy for patients who snore and have mild to moderate OSA. They can be used as second-line therapy for patients who have severe OSA after initiation of CPAP. Adverse effects of oral appliances can include backward movement of the maxillary anterior teeth and forward movement of the mandibular anterior teeth. Other adverse effects include bruxism, dry mouth, dental discomfort, excessive salivation (at the initiation of therapy), TMJ disease, and gingival irritation. All patients fitted for oral appliances need to be followed up both by their dentist and by a sleep physician. Dental follow-up should be every six months for the first year and afterward should be annual. The dentist needs to monitor for comfort, occlusal changes, adherence, and device deterioration and should question the patient about symptoms of OSA.

Surgical Therapies

- The Stanford Protocol was created as an attempt to standardize the surgical approach to OSA among surgeons and prevent excessive operations. While it is important to be familiar with the Stanford Protocol for historical purposes, it should not be followed as a rigid algorithm for all patients. The author of the protocol, Nelson B. Powell, was very clear about this fact. Powell himself recognized that there is no "cookbook" approach to the surgical treatment of OSA (7).
- Surgery should be addressed to the site of obstruction, which is why diagnosis and treatment planning are critical in identifying the site(s) of obstruction to prevent unnecessary surgery.
- The Stanford Protocol is divided into two phases. The first phase consists of soft and hard tissue procedures that address specific sites of obstruction. Those who fail Phase I treatments are either referred for CPAP or are offered MMA as a global airway surgery. The original protocol did not include nasal procedures, which are a more recent addition.
- Phase I (directed to site of obstruction) – 61% success
  - Nasal Obstruction – Septoplasty, turbinectomy, alar collapse, valve deformities, etc.
  - Retropalatal Obstruction – UPPP with tonsillectomy if present
  - Retrolingual Obstruction – Genioglossus advancement
- Phase II
  - Maxillomandibular advancement
  - Tracheostomy

Many patients presenting to the oral and maxillofacial surgeon for MMA have been previously treated according to the Stanford Protocol, and thus will have undergone significant soft tissue procedures that can affect MMA. It is important to at least be familiar with these procedures, though it is doubtful that the candidate will be required to give an in-depth discussion. They are listed and briefly explained below for familiarization.

Uvulopharyngopalatoplasty (UPPP)

- This surgery involves the reduction, tightening, and/or repositioning of the soft palate and related oropharyngeal structures as well as removal, reduction, or reconfiguration of the uvula.
- It is usually done in conjunction with a palatine tonsillectomy if present.
- Adenoidectomy in conjunction with a UPPP should be done with caution because it can lead to circumferential velopharyngeal scarring.
- Laser-assisted UPPP (LAUPPP), while once popular, has fallen out of favor because it can lead to abnormal palatal scarring leading to nasopharyngeal stenosis.
- The procedure has approximately a 50% success rate for OSA overall.
- Complications can include significant pain, postoperative bleeding up to 2 weeks out from surgery, symptomatic dysphagia, nasal regurgitation, velopharyngeal incompetence (VPI), and subjective globus sensation.
- Of particular interest to the oral and maxillofacial surgeon is that the scarring caused by UPPP and tonsillectomy can have a significant effect on the ability to move the maxilla forward during MMA. Additionally, these patients are at risk for postoperative VPI after MMA.

Tonsillectomy

- This can be done in patients with tonsillar hypertrophy as an isolated procedure or in conjunction with UPPP.
- An extra capsular tonsillectomy involves the removal of the entire palatine tonsils along with the surrounding fascia.
- An intracapsular tonsillectomy involves the removal of the bulk of the palatine tonsils. While an intracapsular tonsillectomy reduces the risk of postoperative bleeding and allows for a more rapid recovery, it does allow for tonsillar regrowth in 0.5–16.6% of patients.
- When done in isolation, it is frequently done in conjunction with adenoidectomy.

Hyoid Suspension

- In patients with a low-lying hyoid bone, a hyoid suspension may be performed in isolation or in conjunction with other procedures (such as UPPP).
- The procedure involves the advancement and stabilization of the hyoid bone to the thyroid cartilage or the inferior border of the mandible. This advances the hyoglossus muscle and increases the posterior airway space.
- The procedure can have the effect of stabilization of the base of the tongue and other pharyngeal musculature.
- Adverse effects include dysphagia, infection, rupture of the hyoid suspension sutures with relapse and voice changes.

Tongue Reduction

- Patients with retrolingual obstruction stemming from macroglossia may benefit from reduction in tongue volume. Reduction in tongue volume can open the posterior airway space and the scar formation can have the added benefit of stiffening the base of the tongue to prevent collapse.
- There are a variety of techniques available with multiple modalities including electrocautery, irrigating/suctioning bipolar instruments, and radiofrequency ablation.
- It may also be done in conjunction with lingual tonsillectomy.
- Adverse effects include hemorrhage, infection, airway compromise, dysphagia, neurosensory changes, and paresis. Radiofrequency techniques have fewer complications but are less effective.

Genioglossus Advancement

- This procedure can be used in isolation of MMA or in conjunction with it.
- It involves creating an osteotomy around the genial tubercle and advancing the bone, pulling the genioglossus forward and opening the posterior airway space.

- There have been several different techniques developed to include a traditional horizontal osteotomy or variations of a window technique (square or trephine).
- Complications of genioglossus advancement include infection, anesthesia or paresthesia of the chin and anterior teeth, swelling and bruising of the floor of the mouth, tooth injury, mental nerve damage, and mandible fracture.

Maxillomandibular Advancement/ Telegnathic Surgery

- The surgical technique for maxillomandibular advancement is identical to that for orthognathic surgery. However, there are additional considerations for MMA for the treatment of OSA that bear special attention and should be kept in mind.
- The patient may or may not have an existing dentoskeletal malocclusion (class II or class III malocclusion, cross bite, transverse discrepancies, etc.) but can be addressed simultaneously.
- Patients may or may not require orthodontic treatment prior to MMA.
- Patients who are going to receive telegnathic surgery need to be counseled that their facial profile may be significantly different after surgery.
- The surgical goal is to advance the mandible as far as possible, to maximize the volume of the hypopharynx. This can result in a bimaxillary protrusive profile that the patient may find unappealing.
- The maxillomandibular complex is brought forward and can be rotated in a counterclockwise fashion to maximize the anterior movement of the mandible. This puts significant soft tissue strain on the osteotomies, which in turn can lead to relapse. Additional measures should be taken to prevent relapse. The use of bicortical screws to reinforce osteotomies, heavier plates than in traditional orthognathic surgeries, and bone grafts in the mandible and the maxilla to stabilize the segments and prevent soft tissue ingrowth can help prevent relapse.
- Often, patients may present for MMA after having underwent UPPP with unsatisfactory results. It is important to obtain a thorough history and even ask for the operative report from the UPPP if available prior to going to surgery. Scarring from UPPP and other pharyngeal procedures can limit the amount of advancement that can be obtained during surgery.
- MMA following UPPP may result in VPI as evidenced by dysphonia while speaking and occasional nasal regurgitation. This is usually transient and can often be managed by a "watchful waiting" approach instead of rushing to surgery.
- If a patient has known hypopharyngeal obstruction, performing MMA prior to attempting a UPPP can make global airway procedures easier as well as spare the patient unnecessary pharyngeal procedures.
- Postoperatively, these patients need to be monitored in the ICU with continuous pulse oximetry. Often, the facial swelling of this procedure can be dramatic, but does not usually affect the pharynx.

Tracheostomy

- Historically, tracheostomy was performed on patients suffering from severe OSA. While a tracheostomy provides an almost 100% cure for OSA (as it completely bypasses the entire upper airway), it is generally not favored by patients.
- Tracheostomy still has a role in children with severe craniofacial malformations (i.e., Pierre-Robin syndrome) and may still be used for OSA patients whose comorbidities may preclude global airway procedures but who still require surgical intervention (e.g., morbidly obese patients with severe medical comorbidities).
- The cannula remains capped during the day and the patient can eat and drink normally. However, the site must be vigilantly maintained to prevent mucus plugging, stomal granulation tissue, bronchitis, and peristomal infections.

Case

The patient is a 42-year-old African-American male referred to your practice for the management of his obstructive sleep apnea. He has undergone previous treatment and was referred to your practice by a colleague in ENT. The patient is being consulted for telegnathic surgery.

Medical History

- PMHx – HTN, HLD
- PSHx – UPPP, tonsillectomy, hernia repair
- RX – lisinopril, labetalol, simvastatin
- ALL – NKDA
- SOCHx – denies tobacco, 1–2 drinks/week, no illicit drug use
- Vitals – BP, 140/89; HR, 82; R, 15; T, 37.2°C

- *What information do you want?*
  History of present illness including onset, symptoms, and previous treatment details.

HPI – The patient reports that he has a long history of snoring and that he was diagnosed with OSA approximately 10 years ago. His initial management included CPAP therapy and weight loss. His wife reports that he was still snoring loudly, choking, and waking up frequently whenever he tried to take the CPAP off. He was referred to an ENT surgeon and underwent a UPPP with tonsillectomy. He is still snoring loudly per his wife and is unable to tolerate CPAP therapy. His ENT sent him for a follow-up PSG and he remembers that his AHI was "about 50." He still has morning headaches and feels tired all day.

- *What do you want to do now?*
  Conduct a physical examination including inspection of body habitus, intranasal inspection, airway assessment, dental exam, and occlusion.

The patient is a muscular 42-year-old African American male. His current weight is 198 lbs and his BMI is 27 kg/m^2. He is orthognathic with submental lipomatosis and neck circumference of 18 inches. A nasal exam shows nares patent bilaterally, negative Cottle test, no evidence of polyps, and no evidence of turbinate hypertrophy. Intraorally, the patient has evidence of his previous UPPP with an MP I airway. He has class I molars and canines and the tongue appear to be of normal size.

- *Is there any other diagnostic procedures or imaging that you would like to obtain?*
  A nasopharyngoscopy with a Mueller's maneuver will show obstruction at the base of the tongue. Obtain a lateral cephalogram or CBCT (Fig. 5.16).
- *What cephalometric measurements would you evaluate?*
  SNA, SNB, P to PNS, PAS, H to MP

His measurements are as follows:

- SNA – 91
- SNB – 90
- P to PNS – 40
- H to MP – 27
- PAS – 5

* **Author's Note.** Requesting a CBC and an ECG would be required in such a patient given his history of severe OSA and medical comorbidities. If you request labs during this section, you will most likely be told that they are within normal limits. Do not think that you wasted your time. It is important to remember that this is not the medicine section of the board. While it is important that you know there are several medical comorbidities associated with OSA and that you should get appropriate labs, it is unlikely that the examiners are going to take you down the rabbit trail of chasing all of the patient's medical comorbidities.

- *Do you want any other information?*
  Results of the previous PSG.

The report shows an AHI of 49 with the lowest O_2 saturation of 84%. His ECG shows occasional PVCs and two runs of sinus bradycardia.

- *What is your assessment?*
  The patient is a 42-year-old, ASA 3, male with severe OSA due to obstruction in the hypopharynx.

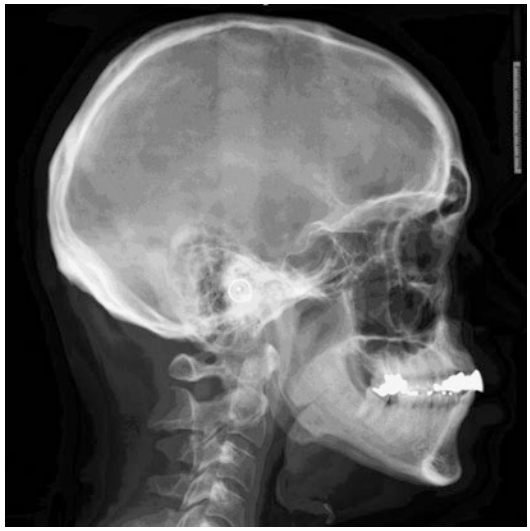

Fig. 5.16 Preoperative lateral cephalogram. (Courtesy of Dr. Joseph Ivory)

- *What is your plan?*
  The patient is a candidate for maxillomandibular advancement surgery which would include LeFort I advancement, mandibular advancement, and genioglossus advancement.
- *What is the amount of recommended advancement?*
  It is important to maximize the mandibular advancement to ensure the most gains in the volume of the posterior pharynx. The patient is class I, so the degree of mandibular advancement is directly related to the maxillary advancement. A maxillary advancement of about 10 mm with some impaction would be ideal. Counterclockwise rotation in this particular case would probably not be warranted because the patient already has a flat occlusal plane angle. This would give a mandibular advancement of about 10 mm coupled with a genioglossus advancement (cortex to cortex).

*__Author's Note.__ Be aware that you should remember that the patient had a UPPP prior to surgery. You should anticipate difficulty advancing the maxilla during your surgery due to scarring from the UPPP and consider making an alternative intermediate splint with a smaller advancement.

- *What are your considerations for hardware placement?*
  For this degree of advancement, it is important to utilize heavy, 2.0 plates on the piriform rims, shorter 2.0 L-plates for the zygomatic buttresses. Grafting may also be necessary to optimize bony contact in the LeFort osteotomy sites. The mandible is secured with bicortical screws in an inverted L with plates along the osteotomy with monocortical screws. The genioglossus advancement is secured with prebent chin plates and grafted with cortical wedges when necessary.
- *Intraoperatively, there is difficulty advancing the maxilla 10 mm as planned. What is the most likely etiology and how do you manage this?*
  This patient had a UPPP, and difficulty in advancing the maxilla the full 10 mm should be anticipated due to scarring. I would use the Rowe disimpaction forceps to stretch the maxilla to try to achieve the ideal advancement (anticipate reflex bradycardia when you do this). If you cannot achieve the ideal advancement, then the maxilla advanced as possible manually and should be fixated in this position. The osteotomized mandible is then set to the new position of the maxilla.

Appendix 1. Cephalometric Analysis

Measurement	Description	Normal values
SNA	Relation of the maxillae to the anterior cranial base	82 ± 2°
SNB	Relation of the mandible to the anterior cranial base	80 ± 2°
ANB	AP discrepancy between the maxillae and mandible in relation to the cranial base	0–2°
FH-NA	Maxillary depth AP position of the maxillae	90 ± 3°
FH-NB	Mandibular depth AP position of the mandible	88 ± 3°

(Continued)

Measurement	Description	Normal values
MPA (FH-MP)	A line from the menton through the gonion (mandibular plane) relative to the FH. Gives you the direction of the facial growth. Low angle of high angle	25 ± 5°
OPA (FH-occlusal)	Occlusal plane angulation: a line tangent to the buccal grooves of the mandibular second molar through the cusp tip of the premolars and the angle of this line relative to FH	8 ± 4°
U1:NA	Upper incisor angulation: long axis of the maxillary incisor to the NA line	22 ± 2°
U1:NA distance	Measurement from the labial tip of the upper incisor tip to the NA line should be 4 +/− 2 mm anterior to the NA line	4 ± 2 mm
L1:NB	Lower incisor angulation: long axis of the mandibular incisor to the NB line	20 ± 2°
L1:NB distance	Measurement from the labial surface of the lower incisor tip should be 4+/−2 mm from NB	4 ± 2 mm
L1:MP	A line from the lower incisor to the mandibular plane. Shows the inclination of the lower incisor	90–95°
N-ANS	N to anterior nasal spine, measure the middle facial height. It is measured perpendicular to FH	54 ± 3 mm
ANS-Me	Measures lower facial height. It is measured perpendicular to FH	65 ± 4 mm
Upper lip tooth	Amount of incisal show. This measurement is correlated with the upper lip length	1–4 mm
Wits appraisal	Expression of AP position between maxillae and mandible without taking into consideration the cranial base. Points BO and AO are established by dropping a line from A and B point to the occlusal plane	BO is 1 mm ahead in males and AO should coincide in females

Appendix 2. Chin Measurements

Measurement	Description	Normal values
Pog-NB = L1-NB	Pogonion projection: Most protrusive point of bony pogonion to the NB line. Mandibular balance achieved when the labial surface to the lower incisors and pogonion are in a ratio of 1:1 anterior to the NB line	4 ± 2 mm
Subnasale vertical	Distance from the soft tissue chin to a line perpendicular to FH through subnasale	3 ± 3 mm behind line
0° Meridian	Distance of soft tissue chin to a line perpendicular to FH through soft tissue nasion	Chin should be 0 ± 2 mm
E-line	A line drawn from the tip of the nose to the tip of the chin	Lower lip should be 2 mm behind it and upper lip 4 mm behind the line

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Cosmetic Surgery

6

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Facial Analysis

General Esthetics

Transverse Facial Fifths

- Divided into equal fifths based on intercanthal distance, the distance between the medial canthi. This should equal the width of an eye in the average person. Intercanthal distance = eye width = distance from lateral canthus to lateral projection of ear (Fig. 6.1).

Vertical Facial Thirds

- Equal thirds: trichion to glabella; glabella to subnasale; subnasale to menton.

Forehead

- Communicates with scalp.
  - 5 layers: **S**kin, **C**utaneous tissue, galea **A**poneurotica, **L**oose areolar tissue, and **P**ericranium.
- Four muscles contribute to its motion: frontalis, procerus, corrugator supercilii, and orbicularis oculi.

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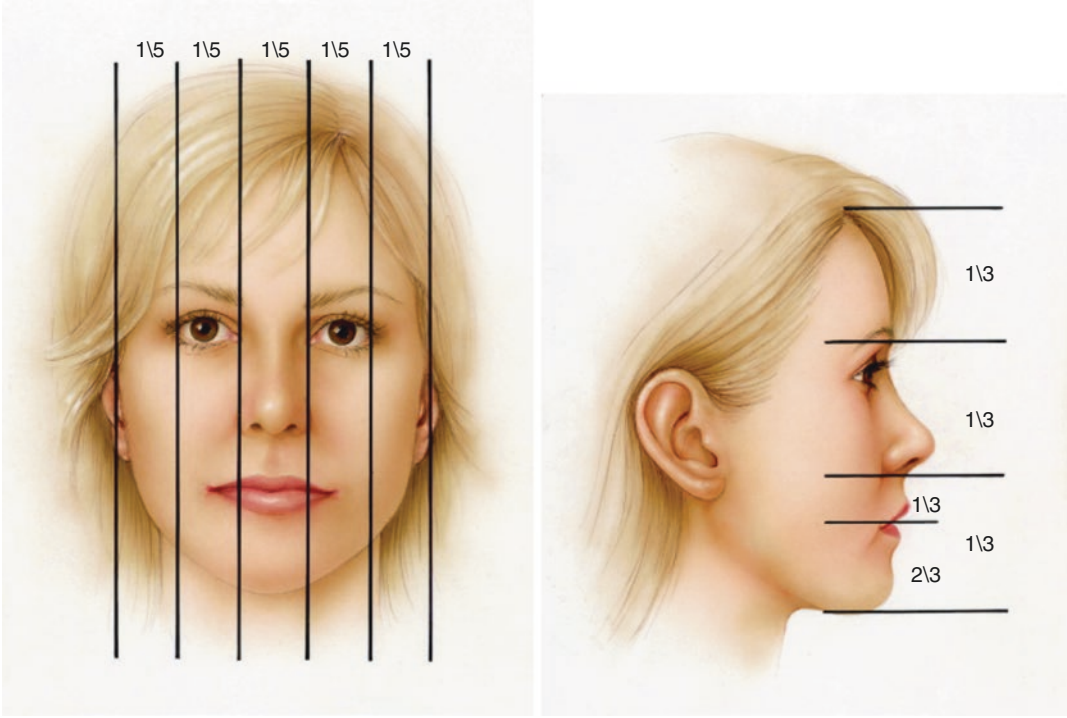

Fig. 6.1 Facial proportions. (Reprinted with permission from Azizzadeh B, Murphy MR, Johnson CM. Master Techniques in Facial Rejuvenation. WB Saunders Company; 2006)

  - These contribute to the dynamic wrinkles in the forehead often treated with neuromodulators (e.g., botulinum toxin).

Eyebrows

- Medially, it begins in the same vertical plane as the medial canthi about 1 cm superiorly.
- Apex should lie on a vertical line drawn on the lateral limbus.
  - The most lateral portions of the eyebrows meet in tandem with an oblique line drawn from the alar base to the lateral canthus (Fig. 6.2).
- The medial and lateral portions of the eyebrow should lie in the same horizontal plane.

Eyelids/Eye

- The upper eyelids cover a small portion of the iris and the lower lid should be within 1–2 mm of the iris in neutral gaze.
- Profile view, the cornea should be 12–16 mm anterior to the lateral orbital rim.

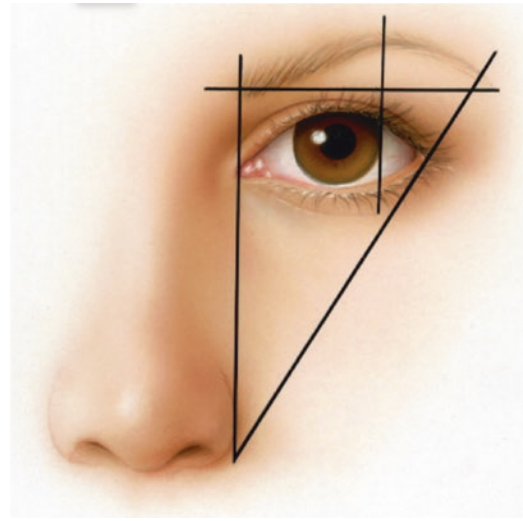

Fig. 6.2 Ideal female eyebrow position. (Reprinted with permission from Azizzadeh B, Murphy MR, Johnson CM. Master Techniques in Facial Rejuvenation. W B Saunders Company; 2006)

Nose/Midface

- Ideal anatomic relationships:
  - Nasofrontal angle: 115–135°.
  - Nasolabial angle: 95–110° in females, 90–95° in males.
  - Nasofacial angle: 30–40° (Fig. 6.3a). Angle formed from a vertical tangent to the glabella through the pogonion and intersecting the line formed through the nasal tip.
  - Nasomental angle: 120–132° (Fig. 6.3b). The angle formed by the tangent line from nasion to the nasal tip and the nasal tip to pogonion.
- Radix should be 4–9 mm anterior to corneal plane.
- Nasal projection:
  - Simons' method: length of the upper lip from the vermillion border to columella, and columella to tip ratio should be 1:1. (Fig. 6.4a) [1].
  - Goode method: ratio of radix-nasal tip (RT) and the line drawn from RT to the alar groove should be 0.55–0.6 RT. Retains nasofacial angle from 36 to 40° (Fig. 6.4b).
  - 3-4-5 triangle by Crumley and Lanswer. Hypotenuse is a line from nasion to nasal tip, projection is the smallest length (Alar crease to nasal tip). Nasal projection is 60% of nasal length (3:5) [1].
- Columellar show: 2–4 mm can be seen below the level of the alae when viewed in profile.
- Malar projection is ideally located at a point 1 cm lateral and 1.5 cm inferior to the lateral canthus.

Mouth/Chin (Lower Facial Third)

- Thirds: upper lip (stomion superioris) to nasal base = $\frac{1}{3}$, lower lip to chin = $\frac{2}{3}$ (1:2 ratio).
- Posture of lip can be procumbent (pushed out) or recumbent (pushed in).
- Lip position:
  - Determined from line drawn from subnasale to soft tissue pogonion. Upper lip should be 3.5 mm anterior and lower lip 2.2 mm anterior to this line.
  - E-line: A line between nasal tip and pogonion. Upper lip should be 4 mm and lower lip 2 mm behind this line.
- Ideal chin projection:
  - 0 Degree meridian: pogonion in vertical alignment with the nasion, perpendicular to the Frankfort horizontal line. Chin position should be within 2 mm ahead or behind this line.
  - Subnasale vertical: A line drawn perpendicular to Frankfort horizontal through subnasale. Chin position more than 6 mm behind this line is considered deficient. Chin position on or in front is considered excessive.

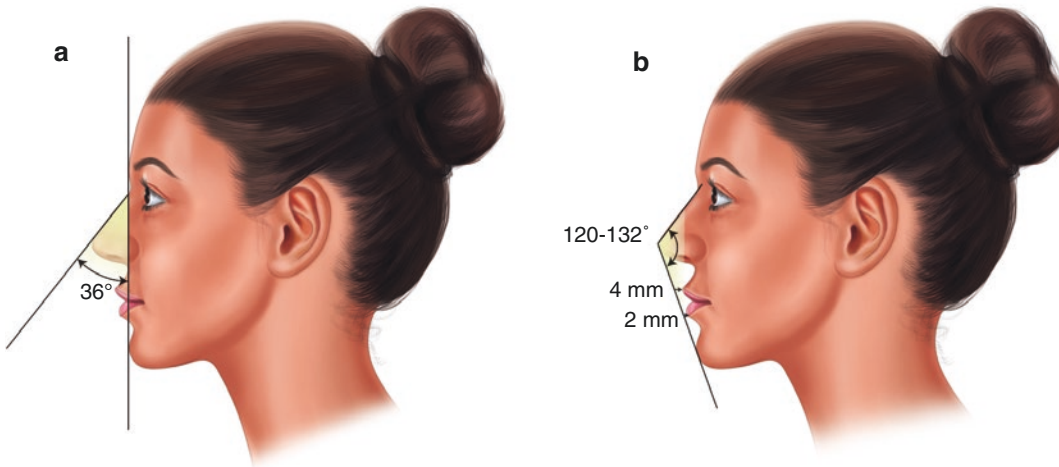

Fig. 6.3 (**a**) Nasofacial angle (**b**) Nasomental angle. (Reprinted with permission from Zimbler M. Cummings Otolaryngology 6th edition. Elsevier Books; 2015)

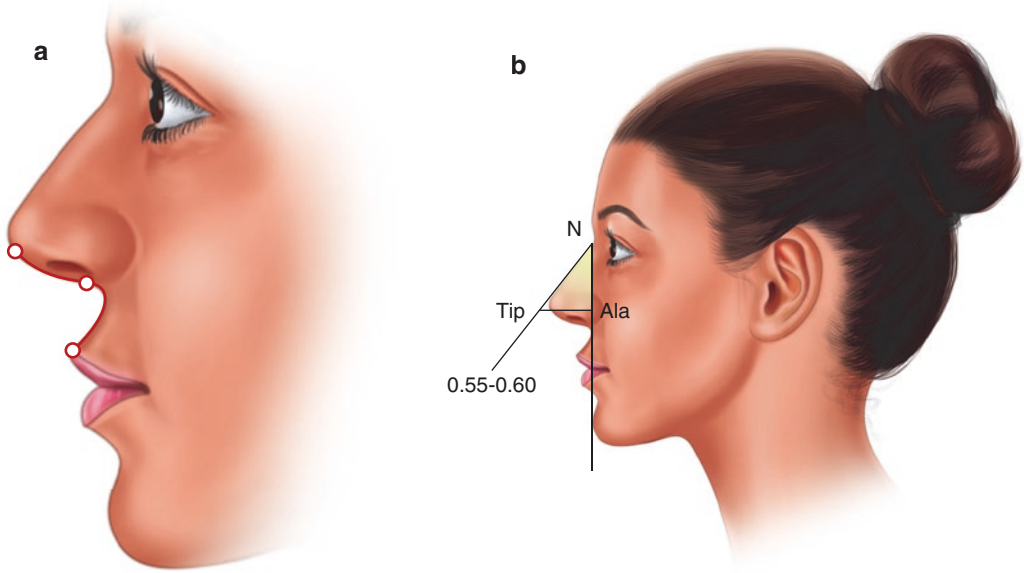

Fig. 6.4 Nasal tip projection. (**a**) Simons method (**b**) Goode method. (Reprinted with permission from Zimbler M. Cummings Otolaryngology 6th edition. Elsevier Books; 2015)

Skin Evaluation

Wrinkles (Rhytids)

- Dynamic – due to repetitive muscle movement
  - E.g., between eyebrows, forehead wrinkles, crow's feet
  - Rx: Neuromodulators (Botox)
- Static – due to skin elasticity loss
  - Nasolabial folds, mentolabial sulcus, along the cheeks, under the eyelids, and neck wrinkles
  - Rx: dermal fillers, chemical peels, lasers, and rhytidectomy

Glogau Classification of Photoaging – assesses patient's level of photoaging and categorizes the amount of wrinkling and discoloration into four categories (Table 6.1) [2].

Fitzpatrick Scale of Sun-Reactive Skin Type – evaluation of skin response to UV light and thus susceptibility to burn (Table 6.2).

Dedo Classification of Cervical Anomalies – classifies aging neck abnormalities based on the anatomic layers of the neck (Fig. 6.5a, b and Table 6.3) [3, 4].

- Position of hyoid is important in formation of the cervicomental angle that ideally is between 105 and 120°.

Rhinoplasty

Evaluation

- The first step is to identify the patient's chief complaint. Are the expectations realistic? Is there a functional component in addition to an aesthetic component?
- Standardized photographs are necessary for all rhinoplasty patients. Frontal, ¾, profile, and submental views are the minimum required photos.
- Is there a history of previous nasal trauma, surgery, and/or sinus disease? Computed tomography might be helpful in these situations.

Table 6.1 Glogau classification of photoaging

Type	Photoaging	Age	Wrinkles	Description
I	Early	20s–30s	Minimal	No age spots Mild pigment changes Little or no makeup use No keratoses
II	Moderate	30s–40s	During movement	Early brown "age spots" Skin pores more prominent Early skin texture changes Usually wears some foundation Keratoses palpable but not visible
III	Advanced	50s–60s	At rest	Telangiectasias and some dyschromia Visible brown "age spots" Prominent, small blood vessels Heavy foundation worn Advanced photoaging
IV	Severe	>60s	Everywhere	Yellow-gray skin tone Prior skin cancers Actinic keratoses"Caked on" makeup, cannot wear makeup as it cakes and cracks

Table 6.2 Fitzpatrick scale of sun-reactive skin type [2]

Skin type	Skin color	Response to ultraviolet light
I	White (very fair)	Always burns, never tans
II	White (fair)	Usually burns, tans with difficulty
III	White/olive (most common)	Occasional mild burn, tans on average
IV	White (light brown)	Rarely burns, tans easily
V	Dark brown	Very rarely burns, tans very easily
VI	Black	Never burns

- Characteristics of the soft tissue envelope of the nose.
  - Thick sebaceous overlying skin can make a rhinoplasty quite challenging by obscuring the underlying anatomical structures.
  - Thin skin will expose every underlying characteristics and flaws.
- Nasal complex:
  - Deviated, wide/narrow, does the nose look too big or too small (over-/under-rotated, over-/under-projected, etc.)?
  - Nasal tip: bulbous, round, triangular, trapezoidal, boxy, amorphous.
  - Nasal dorsum: C-shaped, reverse C-shaped, twisted, deviated, deflected, wide/narrow, inverted-V deformity.
  - Nasal complex deviation is often indicative of septal deviations. Asymmetrical nostrils are also telltale signs of caudal septal deviation. Any underlying cartilaginous or bony irregularities are noted.
- Cottle's test is performed in order to assess the integrity of the internal nasal valve. The test is performed by occluding one nostril and having the patient breathe in and out of the other nostril. After assessing patency, the cheek tissue is pulled laterally on the same side as the breathing nostril. If breathing significantly improves, the test is positive denoting collapse of the internal nasal valve.
  - A more reliable clinical diagnostic procedure is a modified Cottle's test. In the modified test, the wooden end of a cotton tip applicator is placed at the junction of the dorsal septum and upper lateral cartilages to stent out or expand the internal nasal valve angle.
- External valves are assessed by watching the patient breathe in and out forcefully. If the nostrils collapse during negative inspiration, then the lower lateral cartilages are weak and need augmentation during surgery.

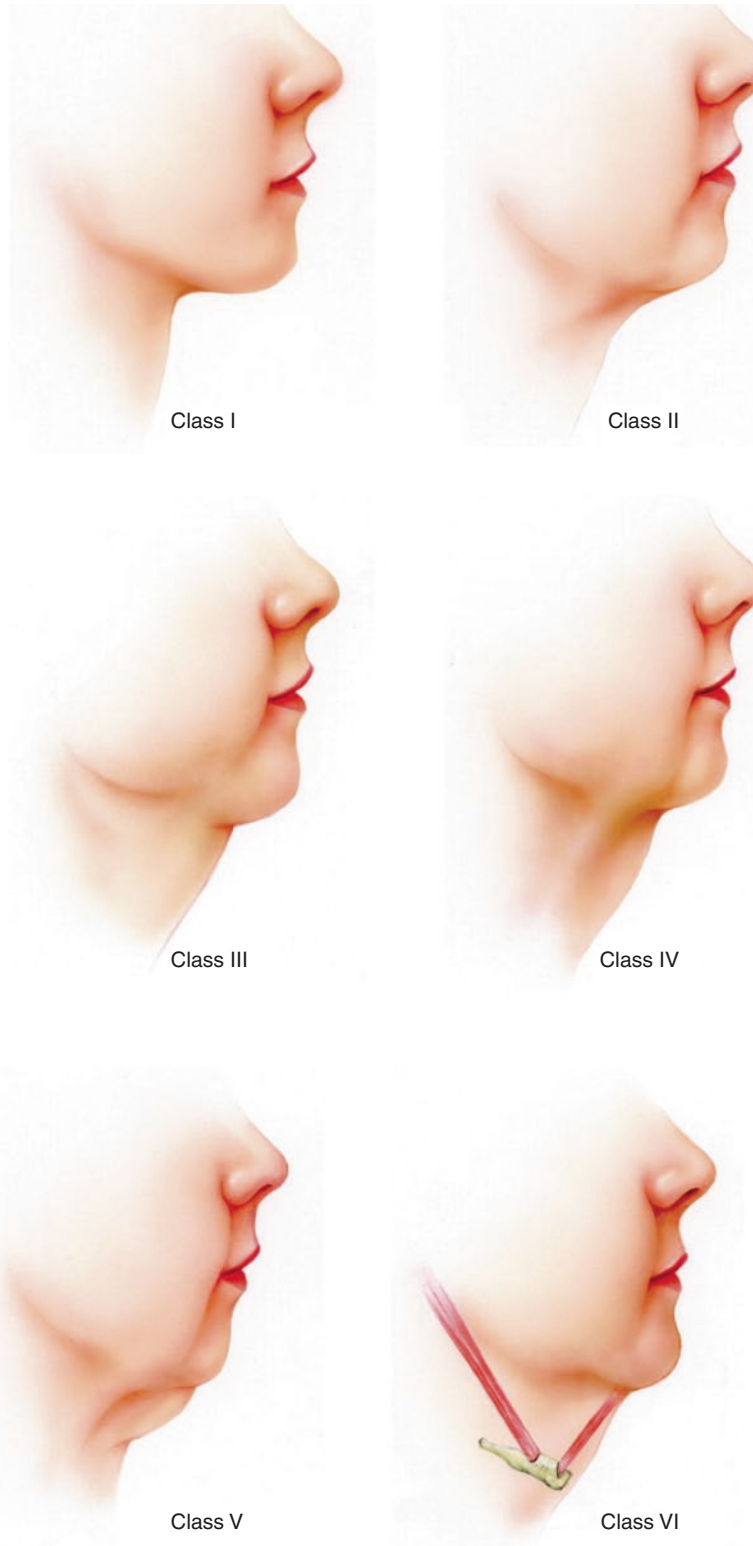

Fig. 6.5 Dedo neck classification system. (Reprinted with permission from Azizzadeh B, Murphy MR, Johnson CM. Master Techniques in Facial Rejuvenation. W B Saunders Company; 2006)

Table 6.3 Dedo classification of cervical anomalies

Class	Anatomical area involved	Deformity	Features
I	Normal	Minimal deformity	Well-defined cervicomental angle, good muscle tone, nominal submental fat
II	Skin	Turkey-gobbler	Lax skin – begins to hang like a curtain. No fat accumulation. No platysma weakness *Treatment: Cervicofacial rhytidectomy*
III	Fat	Jowling	Excessive submandibular/submental adipose *Treatment: submental lipectomy/liposuction +/− cervicofacial rhytidectomy*
IV	Muscle	Anterior platysmal banding	Have patient grimace with teeth clenched to evaluate *Treatment: resect platysma/suture together +/− cervicofacial rhytidectomy*
V	Bone	Microgenia/ retrognathia	*Treatment: consider chin implant or bony genioplasty* vs *orthognathic surgery +/− cervicofacial rhytidectomy*
VI	Bone	Low hyoid bone	Normal hyoid position is C3-C4 Lowered position precludes optimal outcome/requires more aggressive surgery Inform patient of limitations

- Endoscopic- or speculum-assisted anterior rhinoscopy is undertaken to evaluate for any septal deviation, turbinate hypertrophy, and patency of the nasal airway.

Pertinent Anatomy (Fig. 6.6)

It is imperative to use appropriate language when describing the topography of the nasal complex. Due to its orientation on the face, phrases such as anterior or posterior are not as descriptive; rather, terms such as cephalad, dorsal, or caudal are much more useful. A nose that is "up turned," similar to a *Miss Piggy* appearance, is called an over-rotated nose. A nose whose tip is ptotic and pointing toward the floor is called under-rotated. Conversely, a nose that is "too big," and projecting off the face, is called an over-projected nose. The cartoon character of *Pinocchio* has an over-projected nose.

There are four different tissue types in the nasal cavity: skin, mucosa, cartilage, and bone. The soft tissue envelope is thickest at nasal bridge and nasal tip and thinnest along the mid-dorsum region. There are two major components of the nose: the bony vault and the cartilaginous vault, components of which are listed below:

- Bony Vault
  - Paired nasal bones
  - Bony septum: vomer inferior, ethmoid superior
- Cartilaginous Vault
  - Cartilaginous septum
  - Paired upper lateral cartilages (ULC)
  - Paired lower lateral cartilages (LLC)
    Lateral crura of LLC
    Medial crura of LLC
- The caudal edge of the septum sits along the nasal crest of maxilla and attaches to the anterior nasal spine (ANS). Deflection off this crest can cause nostril asymmetry.
- Upper and lower lateral cartilages are attached to each other via the scroll area.
- The internal nasal valve is made up of the septum medially, the caudal end of the upper lateral cartilage laterally, and the anterior end of the inferior turbinate inferolaterally. The valve is typically about 10–15° in most Caucasian noses.
- The external nasal valves are the external perimeter of the nostrils (comprises LLC, nasal septum, and nasal floor). A weak LLC will cause collapse of the external valves upon forceful inspiration.
- It is also important to note that the two medial crura are attached to each other by transdomal ligaments that attach the medial crura to the caudal edge of the septum (transdomal ligaments are a major structural support mechanism of the nasal tip) (Table 6.4).

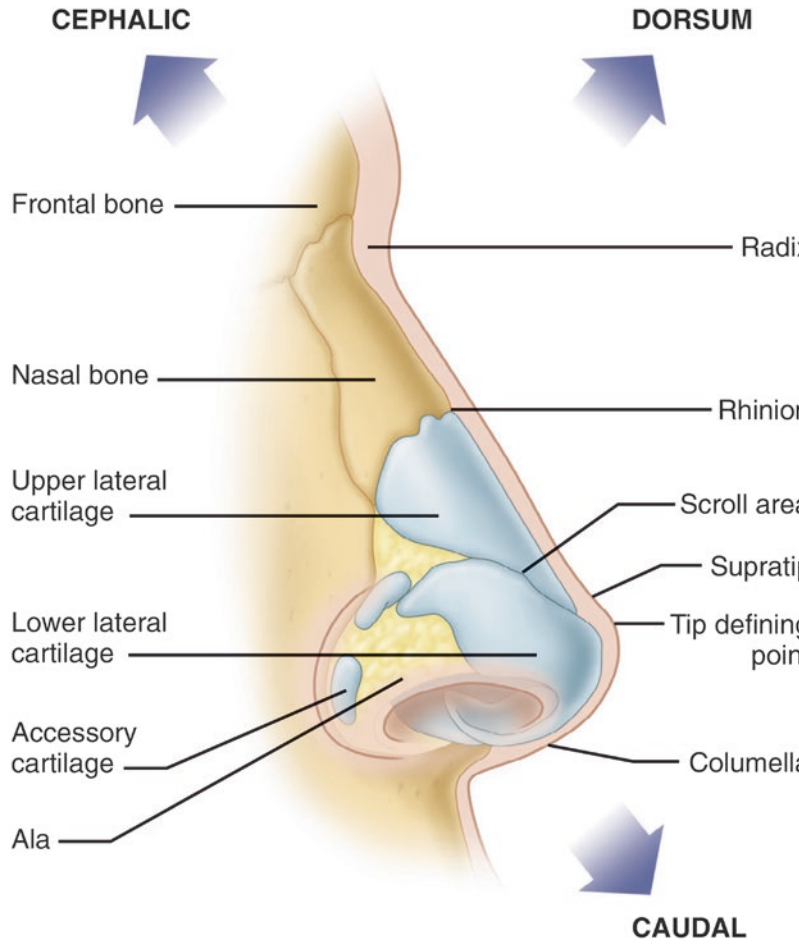

Fig. 6.6 Nasal anatomy. (Reprinted with permission from Bagheri S and Khan A. Clinical Review of Oral and Maxillofacial Surgery. Elsevier; 2014)

Table 6.4 Support mechanisms

Major tip support mechanisms	Minor tip support mechanisms
Size, shape, resilience of the medial and lateral crura Attachment of the medial crural footplates to the caudal margin of the quadrangular cartilage Connective tissue attachment of the upper and lower lateral cartilages (scroll region)	Interdomal ligament Dorsal cartilaginous septum Membranous septum Sesamoid complex Skin and subcutaneous fibrofatty tissue Nasal spine

Surgical Technique

There are essentially two methods to approach the nasal complex: open structure rhinoplasty and endonasal. There are inherent advantages and disadvantages to each technique (Table 6.5).

Table 6.5 Open vs. endonasal technique

Open technique	Endonasal technique
Longer operation Longer recovery External scar Prolonged tip swelling (due to transcolumellar incision) Greater access and visualization	Shorter procedure Shorter recovery No external scar Limited access, especially for structural grafting Preferred for "touch-up" revision surgery

Open Technique

1. Open structure rhinoplasty should begin with harvest of the septum. First, the nose is approached through a marginal incision that is connected to an inverted "V" transcolumellar incision. Once the nose is degloved in a

subperichondrial and subperiosteal fashion, attention is directed to the septum. Submucosal resection of the septum involves removal of cartilaginous septum for grafting purposes and to remove nasal deviation.
   - Must retain a 1 cm "L" strut to maintain support of the nasal complex (1 cm dorsal and 1 cm caudal septum).
   - Septum can be approached through the dorsal approach, Killian incision, and/or hemi- or complete transfixion approach. Once the septum is harvested, attention is directed back to the nose.
2. If a dorsal hump reduction is needed, removal of the cartilaginous and bony components must be performed incrementally to prevent over-resection.
3. Spreader grafts are indicated for augmentation of the internal valve, or if trying to straighten a crooked nose. Spreader grafts are harvested usually from the septum and placed between ULC and dorsal septum. For revision rhinoplasties with previously harvested septal cartilage, allograft rib cartilage or autologous rib/ear may be used. After placement of spreader grafts, lateral and medial osteotomies are frequently performed.
4. Lateral osteotomies involve fracturing of the frontal processes of maxilla and portions of the nasal bones in order to reduce nasal width, straighten a deviated nasal complex, or close minor open roof deformities. Medial osteotomy requires fracturing of the nasal bones in order to further narrow a nose or to prevent a "rocker deformity."
5. Nasal tip: Once the cephalad portion of the nose is addressed, attention is directed toward the tip.
   - A columellar strut graft is placed between the medial crura to provide tip support.
   - Alar batten grafts can be placed along the dorsal aspect of lateral crura to provide stability, especially in cases of external valve collapse.
   - Cephalic trim, if necessary, requires removal of cephalic portion of lateral crura to debulk the tip and rotate the nasal tip; it is imperative to leave about 7–8 mm of native lateral crus in place to maintain tip support.
   - Transdomal and intradomal suturing are performed to narrow the nasal tip and provide support.
   - Shield grafts, named for their "shield"-like shape, are secured to the dome in four corners for enhanced tip definition, to provide an increase or decrease in apparent tip rotation, and to increase tip projection (Table 6.6).

Postoperative Management

- If internal packing is used, systemic antibiotics must be administered for the duration of the intranasal packing. External dressing must be used in all cases in order to redrape the soft tissue envelope over the underlying cartilaginous and bony skeleton; this is a critical part of the procedure.
- Postoperative systemic decongestants are helpful, although patients are asked not to blow their nose for 7–10 days. Saline nasal rinses can be used as often as necessary.
- If an open structure rhinoplasty was performed, persistent tip edema occurs in all of the cases due to the transcolumellar incision; this edema will take months up to 1 year to resolve. Patients must be informed in advance about this.
- Regarding residual asymmetries, minor asymmetries should not undergo revision surgery for several months (up to a year); minor issues usually resolve with time, massaging, or steroid injections. However, major asymmetries, or significant residual deviation of the nasal complex, should undergo revision within a few months; major deviations will not self-correct.

Common Complications

- *Residual Hump* – very common occurrence; this is typically due to inadequate hump reduction. Requires revision surgery.

Table 6.6 Nasal tip augmentation

	Cephalic Trim	Cephalic Resection	Intradomal Sutures	Interdomal Suture	Columellar Strut	Shield Graft	Medial Crura Resection	Caudal Septal Resection
Increased tip rotation	✓	✓	✓		✓	✓		
Decreased tip rotation						✓	✓	
Narrow nasal tip	✓		✓	✓				
Decrease tip projection		✓					✓	✓
Increased tip projection			✓	✓	✓	✓		

- *Pollybeak Deformity* – fullness of the nasal supratip relative to the rest of the nose. Classified as cartilaginous (e.g., due to loss of nasal tip support) or soft tissue etiologies (e.g., scar tissue fills the supratip break). It is caused by inadequate dorsal septum removal and/or excessive bony dorsum removal, excessive dorsal septum resection, excessive alar cartilage removal, or excessive supratip scar removal. If soft tissue, may attempt intralesional steroid injection. Surgical revision dependent on etiology.
- *Saddle Nose Deformity* – loss of septal support and saddling of the nose; could occur due to large septal perforations and loss of structural support. Requires major reconstruction of the nose, typically requiring large cartilage and/or bone grafting.
- *Open Roof Deformity* – flat dorsum following large hump reduction due to failure to perform lateral osteotomy to close the "open roof." Requires revision surgery via lateral osteotomy.
- *Rocker Deformity* – green stick lateral osteotomy. This occurs when a lateral osteotomy is extended too cephalad along the medial canthal area where the bone can be quite thick. As an incomplete fracture occurs, the inferior aspect of the osteotomy "rocks" and the upper portion simply hinges or does not move at all. This deformity requires revision surgery in order to complete the cephalic portion of the lateral osteotomy.
- *Inverted V* – collapse of the upper lateral cartilages. Caudal edges of nasal bone can be seen through the non-supported skin. Treated most often with spreader grafts.
- *Keel Deformity* – the dorsum in cross section comes to a point rather than a rounded dome. Often treated with spread grafts and nasal osteotomies.

Pearls of Wisdom

- A minimum of 7–8 mm of lower lateral cartilage should remain after a cephalic trim to prevent pinching, alar retraction, external nasal valve collapse, and/or tip asymmetry.
- The supratip break is formed from the junction of the caudal edge of the lower lateral cartilages and the dorsal septum (anterior septal angle). For significant reduction of the cartilaginous septum, one should use the anterior septal angle as the starting point for hump reduction.
- There should be roughly 2–4 mm of the columella shown from the profile view. The amount of columella show is related to the amount of "hooding or retraction" of the alar rim or the amount of "hanging or retraction" of the columella.

- Tip defining points of the nose: supratip break, infratip break, domes of the lower lateral cartilages.
- Relative over projection of the nasal tip may be due to microgenia or midface deficiency. A rhinoplasty evaluation must also include consideration of the chin projection and midface projection.

Rhytidectomy (Face Lift)

A surgical procedure to rejuvenate the appearance of the face by the removal of excess skin and may include manipulation of the SMAS (superficial musculoaponeurotic system).

Anatomy

There are five layers of the face that includes the skin, subcutaneous tissue (superficial fat layer/superficial fascia/deep fat or areolar layer), musculoaponeurotic layer, retaining ligaments and spaces, and the deep fascia in the midface and periosteum in the scalp.

- The SMAS is the superficial fascia and incorporates muscle and fat of the face, temples, forehead, and neck.
  - Separates the superficial fat layer from the underlying deep fat and fascia.
  - Superficial to the facial nerve in the surgical area.
  - Over the parotid gland, it is thick and aponeurotic.
  - Over the facial mimetic muscles, it is thin and layered.
- Retaining ligaments of the face are osteocutaneous (tether skin to bone) and fasciocutaneous (SMAS to deep fascia).
  - Osteocutaneous ligaments: zygomatic, infraorbital, and mandibular ligaments.
  - Fasciocutaneous ligaments: parotid cutaneous and masseteric cutaneous ligaments.
- McKinney's point – where the greater auricular nerve passes over the center of the sternocleidomastoid muscle.
  - It is 6.5 cm inferior to the caudal most point of the bony external auditory meatus with the head turned 45 degrees in the opposite direction.
- McGregor's patch – zygomatic cutaneous ligaments found in the malar area, difficult area of dissection due to fibrous attachment and thickening of the subcutaneous layer. Risk of bleeding due to perforating branch of transverse facial artery.
- All muscles of facial expression are innervated on their deep surfaces except:
  - Levator anguli oris
  - Buccinator
  - Mentalis

Evaluation

- What is the patient's chief complaint?
- Complete medical history to include social and psychological evaluation. Past facial surgery/cosmetic surgery? Multiple cosmetic surgeries should raise concern for body dysmorphic disorder.
- Medications and supplements should be reviewed. It is important to identify products that can increase bleeding such as antiplatelet agents, anticoagulants, NSAIDS, high dose vitamin E, fish oil, ginseng, ginkgo biloba, and St. John's wort.
- Smoking/nicotine history? Recommended to stop using nicotine products 6 weeks before and 4 weeks after any surgery to reduce necrosis risk (3× incidence versus non-smokers). As nicotine supplements aids are widely available over the counter, question patients on such aids as patches and gums. Medications such as bupropion SR (Zyban ®) or varenicline tartrate (Chantix ®) can help aid in quitting prior to surgery.
- Realistic expectations? Will not erase all signs of aging. Will address lower third of face including neck laxity, jowling, mesolabial folds, and some nasolabial folds. Will not address wrinkles around mouth.
- Upper, middle, lower face evaluation. Platysmal dehiscence, jowling, descent of the

malar fat pads, nasolabial folds, marionette lines, etc.? Skeletal profile, e.g., retrognathia? Microgenia?
- Clinical photos (both smiling and at repose): frontal, right and left ¾ view, right and left profile views, submental vertex.

Superficial Plane Versus Deep Plane Face Lifts

- Superficial plane facelift
  - Substantially faster to perform; however, the appearance isn't as natural and has a limited duration.
  - Skin only, mini-lifts, SMAS plication, SMAS imbrication, SMASectomy, and thread lifts.
- Deep plane facelift
  - Deeper plane facelifts use the facial SMAS to achieve and maintain a consistent, predictable, natural, stable, and youthful appearance to the middle and lower thirds of the face.
  - Surgery takes longer to perform and care has to be taken when elevating the SMAS off the facial nerve.

Surgical Technique

- The exact locations, extensions, and depth vary from doctor to doctor and type of facelift (Fig. 6.7a, b).
- The typical incision design consists of a temporal hair tuft sparing incision, 45° hockey stick, or vertical incision design.
- The incision rests in the preauricular sulcus until the tragus of the ear is reached. At this point either an endaural incision (females) is made or one may choose to stay in the preauricular fold (men). The preauricular fold is preferred for men to prevent hair growth on the tragus.
- The inferior extension goes under the earlobe (a 2 mm cuff to prevent a pixie ear deformity) and then extends to the posterior auricular sulcus. Some surgeons prefer to carry the incision onto the conchal cartilage to prevent migration of the scar.
- At the point of the greatest width of the pinna, the incision turns posteriorly into the hair-bearing region of the scalp. The incisions in the hair are beveled as much as possible to allow ingrowth of hair into the scar.
- Short scar facelifts may only have a pretragal incision with minimal extension.
- Dissection and management of the SMAS varies depending on the type of facelift performed (Fig. 6.8). SMAS plication: The SMAS is folded on itself and sutured.

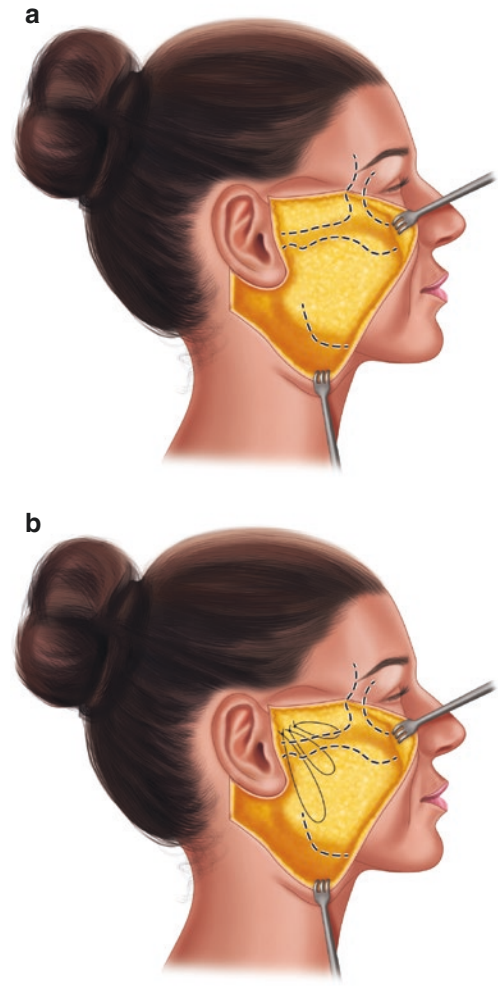

Fig.6.7 (**a**) Mini lifts: skin only. (**b**) Thread lift. (Courtesy of Dr. William T Evans DDS, MD, FACS)

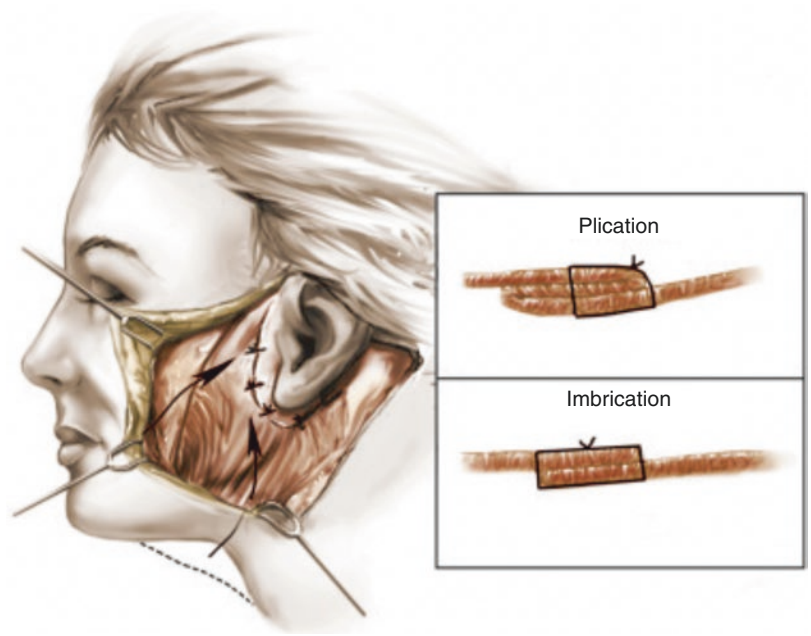

Fig. 6.8 Imbrication vs. plication facelift. (Reprinted with permission from Fisher E. Rhytidectomy. Oral and Maxillofacial Surgery, Third Edition. Elsevier; 2017)

Includes the preauricular and possibly the infrazygomatic SMAS.

SMAS imbrication: The SMAS is incised, overlapped, and sutured.

SMASectomy: A portion of the SMAS is excised from the malar eminence to the mandibular angle and the edges are sutured together.

Types of Facelifts

Superficial Plane Facelifts

- Skin lift: Utilizes a short flap or an extensive long flap and only a subcutaneous dissection is performed. Redundant soft tissues are repositioned by traction of the skin only. Looks tight, pulled, and unnatural. Rarely used.
- Mini lifts (S-lift, Feather Lift) (Fig. 6.7a): Redundant preauricular soft tissue is excised and incision edges are undermined for closure. Minimal undermining may be performed. The facial SMAS is plicated, purse stringed, or barbed sutures are utilized for elevation.
- Threadlift (Lifestyle lift, Quicklift, Lunchtime lift, etc.) (Fig. 6.7b): Redundant SMAS is elevated by sutures that are typically fixed to the preauricular deep temporal fascia.

Deeper Plane Facelifts

- SubSMAS: First a subcutaneous dissection is performed. Then infrazygomatic, preauricular, and infra-auricular platysmal incisions are made through the facial SMAS. The SMAS is then undermined to the anterior border of the parotid gland. Traction is placed on the SMAS and the excess is excised or folded, then sutured.
- Extended SubSMAS: Purported to improve facelift results in the area of the nasolabial fold. First a subcutaneous dissection is performed. Then infrazygomatic, preauricular, and infra-auricular platysmal incisions are made through the facial and neck SMAS. The SMAS is then undermined further than the subSMAS facelift, enough for passive mobilization. The retaining ligaments are interrupted. Traction is again placed on the SMAS and the excess is excised or folded, then sutured. A cervicoplasty is usually performed.

- Deep Plane: An extended SubSMAS facelift that has minimal subcutaneous dissection. The SMAS incision is from the malar eminence to the mandibular angle. Extreme traction is placed on the SMAS-skin flap in a superolateral direction, then sutured to the preparotid SMAS. A subcutaneous neck dissection is performed.
- Composite: A deep plane facelift with a subSMAS dissection below the central part of the malar fat pad that includes the pre-zygomatic SMAS and orbicularis oculi muscle.
- Extended Multiplanar Multivector: An extended subSMAS deeper plane facelift that includes a suborbicularis oculi muscle and sub SOOF dissection in order to interrupt the infraorbital osteocutaneous ligaments. In addition, a wedge of the orbital portion of the orbicularis oculi muscle is excised.

Combined Procedures

- The ideal age for a facelift patient is between 45 and 65. The patient's age can vary significantly depending upon genetics, environmental exposure, smoking, injury etc. The younger the patient, the quicker they will recover and longer they will benefit from the procedure. A facelift only addresses the midface and lower face.
- Multiple additional procedures may be performed simultaneously including brow lifts, upper and lower lid blepharoplasties, fat transfer, facial implants, and rhinoplasty.
- Laser skin resurfacing can only be performed simultaneously if the skin flap is of sufficient thickness to withstand the insult; however, this is reserved for experienced surgeons.

Postoperative Care

- Facelift dressing for first 48 hrs then nightly for 1 week.
- See after 24 hours for correct wound drape and rule out hematoma.
- Ice for first 24–48 hours.
- Hydrogen peroxide to clean wounds daily.
- Appropriate pain medication.
- Appropriate antibiotics (topical and oral).
- Avoid aspirin, ibuprofen, vitamin E, herbal and homeopathic medications.
- No alcohol for a minimum of 7 days postoperatively.
- No smoking during healing process.
- Sleep on back with two pillows for 2 weeks.
- Shampoo hair after 48 hours.
- Soft foods initially for comfort.
- Sunblock SPF $\geq$ 30.

Post-op Complications and Management of Face Lift Surgery

- *Hematoma* – two types: major and minor, both pose a risk for skin necrosis. Minor hematoma is usually less than 10 cc and is often not appreciated until bandages are removed. Treatment requires needle aspiration or manual expression. Excessive facial pain and excessive edema are major signs of a major (expanding) hematoma. Major hematomas require operative setting to identify causative vessel.
- *Pixie Ear Deformity* – inferior traction of earlobe due to pull of skin. Avoided by leaving cuff of tissue around earlobe. Surgical treatments include undermining the skin and reinforcing the SMAS or a triangular wedge (V-Y closure) is removed and the lobe is reattached in a superior and posterior position.
- *Necrosis* – most affected areas are the mastoid and post-auricular regions due to thin skin thickness and distance from vascular supply. Cleanse area with hydrogen peroxide and maintain moisture, e.g., trolamine salicylate (Biafine ®). Some clinicians recommend nitropaste to encourage vasodilatation. Hyperbaric oxygen may be used to encourage wound healing and revascularization in large affected areas.
- *Unaesthetic Scar* – steroid injections such as triamcinolone 3 mg every 6 weeks for 3 months. Overuse may cause dermal atrophy, depression, and spider telangiectasia. Carbon dioxide laser resurfacing and microneedling

may help reduce visibility of scar. Scar revision surgery may also be considered.
- *Facial Nerve Damage* – temporal and marginal mandibular branch (most common motor nerve damaged) can be affected. Most commonly this is transient and only a matter of time until nerve function returns. May also consider neurotoxin to the unaffected side (to help mask difference in animation) or referral for facial reanimation consultation. Damage to frontal branch may impair orbicularis oculi and require globe protection such as eye patches, temporary tarsorrhaphy, or gold weight implantation to upper eyelid.
- *Sensory Nerve Damage* – greater auricular nerve (most common nerve injured) injury reported around 1–7%. Most injuries resolve in 6 months. Patient may complain of anesthesia, paresthesia, or dysesthesia in the inferior portions of the ear lobule, ear, and the sternocleidomastoid region. If neuroma, suspected MRI may help identify for early intervention. Gabapentin and tricyclic antidepressant therapy may help alleviate pain.
- *Infection* – incision and drainage with cultures and sensitivities.
- *Hair Loss* – tension alopecia can be avoided by adequate wound support without excessive tension. May be due to telogen effluvium, reversible hair loss due to stress, allow 6 months for observation and consider steroid injections. Permanent alopecia may be treated with topical minoxidil (Rogaine ®), hair follicle transplant, PRP injections, local flap, or resection with primary closure.
- *Hyperpigmentation* – usually resolves in 6 months. Patient may apply 4% hydroquinone or kojic acid cream to the affected area.

Platysmaplasty

A platysmaplasty is a surgical procedure that rejuvenates the central submental area of the neck. It is performed through a submental incision. A platysmaplasty rejuvenates a sagging neck by removing excess platysma and by tightening the remaining edges thus improving the angle between the chin and the neck.

- An isolated cervicoplasty/platysmaplasty is typically reserved for patients under 40 years old.
- In an older patient, it should be done in combination with a facelift.
- The results are often enhanced by doing a chin and/or cheek implants at the same time when indicated.

The anterior neck boundaries are as follows:

- Inferior border of the mandible superiorly
- Suprasternal notch inferiorly
- Anterior border of the sternocleidomastoid laterally

The layered anatomy of the neck is:

- Skin
- Superficial fat layer, that is removed via liposuction, open lipectomy or deoxycholic acid injection (Kybella®)
- Superficial cervical fascia (SMAS) that contains the platysma muscle
- Deep areolar fat
- Deep cervical fascia
- Cervical muscles

Characteristics of a youthful attractive neck:

- Cervical-submental angle of 115 degrees +/− 10
- No folds or bands
- Distinct inferior border of mandible
- Distinct edges of SCM
- Appropriate length of neck
- Skin free of aging stigmata

Evaluation

- Dedo classification and the etiology of facial laxity.
- Skin condition – skin firmness important for success. Skin with more elastic integrity will do better after recontouring.
- Fat location – using pinch and roll technique. Pre-platysmal fat should be differentiated from subplatysmal fat that is firmer.

Liposuction alone will not treat subplatysmal fat. Another method is to pinch the skin and ask patient to grimace, if the fullness is gone, then the fat is under the platysma.
- Integrity of platysma – have patient clench teeth to evaluate the midline dehicience of the medial borders of the platysma.
- Check for ptotic submandibular glands.

Surgical Technique

- Mark patient awake and sitting or standing. Mark the anterior borders of the SCM, the inferior border of the mandible, and the thyroid notch inferiorly. This outline is for the subcutaneous dissection.
- The access is a 3–4 cm incision that is 2–3 mm posterior to the submental crease. If the incision is placed in the submental crease, the crease will deepen.
- Local anesthetic with epinephrine is infiltrated into the region of the subcutaneous dissection. Tumescent anesthesia can also be employed. A 22-gauge spinal needle is commonly used to inject the region. Allow 5–7 minutes for vasoconstriction.
- The incision is made with a #15 scalpel blade through skin into subcutaneous tissue. Sharp dissection is performed around the entire incision for about 1 cm in the subcutaneous plane.
- The subcutaneous dissection is completed with facelift scissors leaving 3–5 mm of fat attached to the dermis so as not to skeletonize the neck.
- Once the subcutaneous dissection is completed and hemostasis is achieved, open liposuction can be easily performed under direct visualization using a 1–2 mm liposuction cannula with the tip open toward the platysma. Do not cross the inferior border of the mandible as this risks damaging the marginal mandibular nerve. Lipectomy allows for more fat removal than liposuction. If closed liposuction is preferred, it is done prior to subcutaneous dissection.
- The amount of central laxity of the platysma or fascia to be excised is determined by picking up the tissue with forceps.
- The edges of the platysma are sutured with a running locking 2-0 or 3-0 long lasting absorbable or permanent sutures. The platysma corset suturing begins superior and continues inferiorly, then is reversed to end superiorly. Some surgeons choose to perform mastoid-mastoid suturing techniques (e.g., Giampapa suture) to reposition the cervicomental angle.
- The skin incision is closed. A cervical dressing is applied.

Post-op Complications of Neck Liposuction and Submentoplasty

- *Over Resection of Fat* – avoided by using micro liposuction cannulas 1–2 mm. Treated with fat injections that can be harvested from thighs or abdominal fat.
- *Exposure of Platysmal Bands Without Platysmaplasty* – Botox injections may be used as palliative treatment. Platysmaplasty is a more enduring treatment.
- *Cobra Neck Deformity* – if overaggressive, lipectomy subplatysmal or uneven fat removal laterally. If separation of the platysma muscle midline, may plicate platysma muscle.
- *Submandibular Gland Ptosis* – descent with age or prominent gland. Treatment includes suture suspension (limited success). Superficial transection of the gland is more commonly performed.
- *Sialocele* – can occur from the parotid or submandibular gland. Parotid damage more common during facelift procedure and submandibular damage more likely after gland resection for neck recontouring. Treatment is serial aspirations with fluid tested for high levels of amylase (usually greater than 10000 u/L). Treatment includes antisialogogues or botulinum toxin A into the gland.

Tumescent Anesthesia

- Tumescent anesthesia is a technique of infiltrating large volumes of subcutaneous fluids

in order to produce anesthesia, tissue distention, and hydrodissection.
- The fluid typically contains lidocaine, saline, and epinephrine.
- Benefits include hydrodissection, minimized blood loss, decreased anesthesia requirements, decreased postoperative pain, bacteriostatic effects, and increased tissue firmness facilitating ease and quantity of fat removal.
- Tumescent fluid can be injected into the face and neck via manual pressure utilizing a syringe and a 22-G spinal needle. An infusion pump may also be used for larger volume injection.
- Tumescent anesthesia was first introduced by dermatologist, Dr. Jeffrey Klein for liposuction. He recommended a lidocaine dose between 35 and 45 mg/kg. The American Academy of Dermatology recommends a maximum dose of 55 mg/kg in patients weighing 43.6–81.8 kg.
- Lidocaine's onset of anesthesia is 15 minutes and maximum concentration occurs at 11–15 hours after injection.
- 20% of infiltrated lidocaine is removed during liposuction.
- The toxic level of plain lidocaine (without epinephrine) is 4.5 mg/kg when utilized for local infiltration. Lidocaine with epinephrine should be limited to 7 mg/kg for local infiltration.
- Tumescent anesthesia allows higher maximum doses of lidocaine than that of local infiltration. As a result of the large volume of saline and dilute epinephrine (vasoconstrictor), the volume is pushed interstitially. This slows systemic absorption of lidocaine and thus reduces peak serum lidocaine concentrations. This, in-turn, reduces the risk of systemic lidocaine toxicity.
- Levels only reached 0.8–2.7 mcg/mL postoperatively when using 35 mg/kg. Cytochrome P450 (hepatic CYP3A4) metabolizes lidocaine. 10% is excreted unchanged in the urine.
- Medications such as benzodiazepines, TCAs, SSRIs, antifungals, CCBs, and cimetidine are metabolized or inhibit CYP3A4. Concomitant use of these medications can lead to toxic levels of lidocaine.
- Lidocaine absorption is different in the face vs. the body. Absorption is faster in the face.
- Another concern of tumescent fluid is when large volumes are injected. This can result in volume overload and pulmonary edema. It is important to keep IV fluids to a minimum.

Klein's Formula

- Normal saline 1000 mL
- 1% Lidocaine 50 mL (500 mg)
- 1:1000 Epi 1 mL (1 mg)
- +/− 8.4% $NaHCO_3$ 10 mL (10 mEq)

- Final concentration: [0.05% lidocaine with 1:1,000,000 epi].
- Since smaller amounts of fluid are injected for anesthesia of the head and neck, a more concentrated solution may be used.

Lidocaine Toxicity

- Mild symptoms of toxicity are lightheadedness, headaches, visual disturbances, confusion, metallic taste, circumoral numbness, hypotension, sleepiness, and nausea/vomiting.
- As symptoms progress, muscle twitching, tinnitus, seizures, and eventually unconsciousness occur.
- In severe cases, bradycardia, significant hypotension, arrhythmias, asystole, and cardiac arrest can occur.
- Cardiac toxicity occurs at levels of 5–10 mcg/mL. Respiratory depression and cardiovascular collapse occur above 10 mcg/mL.
- Treatment includes airway maintenance, oxygen administration, intravenous fluids, benzodiazepines to control seizures, vasopressors, and a 20% intralipid infusion.
- The initial bolus of intralipid is 1.5 mL/kg followed by an infusion of 0.25 mL/kg/min with a maximal dose of 8 mL/kg.
- An example of a 20% intralipid is LipidRescue™. If the patient is unresponsive to initial interventions, immediately start CPR

and administer 20% intralipid 1.5 mL/kg over 1 minute followed immediately by an infusion rate of 0.25 mL/kg/min. Continue chest compressions to circulate the lipid. Repeat bolus every 3–5 minutes up to 3 ml/kg total dose until circulation is restored. If the BP declines, continue the infusion at 0.5 mL/kg/min until hemodynamic stability is restored.

Blepharoplasty

Eye Lid Anatomy

- Thinnest skin is on the eyelids without any subcutaneous fat.
- Orbicularis oculi: Innervated by CN VII. Palpebral (further subdivided into pretarsal and preseptal) and orbital segments. It functions as the eyelid protractor.
- Orbital septum: Thin fibrous tissue arising from arcus marginalis from orbital rims. Fuses with levator aponeurosis 2–5 mm above the tarsus (depending on race). Separates preseptal tissue from orbit.
- Orbital fat: Two fat pads on upper eyelid and three fat pads lower lid. Levator aponeurosis immediately beneath fat pads.
- Levator muscle: Innervated by CN III. Originates above annulus of Zinn near orbital apex. 40 mm in length. Transitions from muscle to aponeurosis over Whitnall's ligament (transverse support ligament). Inserts onto tarsus and pretarsal skin creating supratarsal lid crease.
- Mueller's muscle: Sympathetic innervation. Originates undersurface of levator muscle and inserts at superior border of tarsus.
- Tarsus (varies in ethnic groups): Upper – 8–10 mm in height; lower – 4–6 mm in height. Composed of dense connective tissue that maintains structural stability.

Pathophysiology

- Aging and actinic changes lead to degeneration of elastin and collagen resulting in lax skin.
- Weakening of orbital septum results in fat prolapse (steatoblepharon).
- Stretching or weakening of levator muscle can result in involutional ptosis (droopy eyelid).

Evaluation

- Understand patients concerns and expectations. Onset and duration of symptoms. Coexisting double vision.
- Identify risk factors: sun exposure, aging, smoking, inflammatory disorders, family history.
- Medications: blood pressure control, use of antiplatelet agents (stop 7 days prior to surgery if possible), and herbal supplements (fish oil, ginkgo biloba, garlic, vitamin E, etc.).
- Identify preexisting ocular conditions such as dry eyes (aggressive blepharoplasty may lead to chronic keratoconjunctivitis) and previous or upcoming ocular surgeries.
- History of prior cosmetic procedures including fillers or neurotoxins.

Eye Evaluation

- Baseline visual acuity exam and ocular motility exam.
- Brow: examine contour, symmetry, lateral eyelid fold; creases above brow may indicate ptotic brow (to help elevate lid out of field of vision). Female brow above orbital rim, male brow at rim. For female, the brow position should be 1–2 mm medial, 5–6 mm middle, 8–10 mm arch, 10–15 mm tail above orbital rim. For male, it should be 1–2 mm above the orbital rim for all segments [4, 5].
- Ocular motility and alignment: check for Bell's phenomenon (upward rotation of the eye with ipsilateral orbicularis contraction) and strabismus.
- Tear function tests such as Schrimer test. As patient looks up, place Schrimer strip in the temporal portion of the lower fornix. Normal

wetting of the strip should advance to 10–15 mm, <10 mm is abnormal. If abnormal, blepharoplasty not be advised; lagophthalmos may not be tolerated by patients with a history of dry eyes.
- Visual field testing – with and without lid elevation.
- Slit lamp examination.
- Photo documentation to allow preoperative and intraoperative evaluation. Photos may also be used to demonstrate postoperative changes to the patient.

Upper Lid Blepharoplasty

Preoperative Evaluation

- Eyelid exam for upper lid, rule out blepharoptosis, and brow ptosis.
- Upper lid height: determine MRD1 (margin reflex distance – from central corneal reflex to eyelid margin). MRD1 normally 4–4.5 mm, lower number usually means eyelid ptosis
- Lid function is tested for ptosis. Levator excursion test investigates distance from extreme upward to downward gaze with brow immobilized, normal 13–16 mm. Orbicularis strength (forced resistance to closure, subjective).
- Fat prolapse: note the amount of prolapse. Normally not much fat in the temporal fat that may indicate lacrimal gland prolapse.
- Examine eyelid crease, normal 9–11 mm superior to the eyelid margin. Ask patient look down as the eyelid fold is elevated. If less than 9 mm, consider reconstruction of eyelid crease and fat excision. If more, consider disinsertion of the levator aponeurosis.

Indications for Upper Lid Blepharoplasty

- Redundant or lax eyelid skin (dermatochalasis) with or without fat herniation (steatoblepharon) that results in functional visual obstruction or cosmetic concerns.

Surgical Technique Upper Lid Blepharoplasty

- In sitting position, mark natural eyelid crease (Fig. 6.9) for inferior edge of resection (if no crease: female: 8–10 mm, men 6–8 mm from the edge of the eyelid).
- Identify superior edge with pinch testing to determine the amount of skin resection with slight eversion of eyelashes. Must be below eyelid-brow junction. Safe rule: leave ~20 mm between margin and eyelid-brow junction.
- Anesthesia: sedation, oral, intravenous, or general depending on patient's comfort.
- Place topical anesthetic in eye prior to placement of corneal protectors to prevent ocular injury.
- Local anesthetic with epinephrine for hemostasis (inject subcutaneous for blepharoplasty).
- Skin incision: #15 blade, electro-cautery, Ellman RF, or laser. Gentle traction on skin allows precision and visualization.
- Layered dissection: skin only first followed by small layer of orbicularis excision centrally (important not to excise too much laterally or medially to preserve eyelid closure).

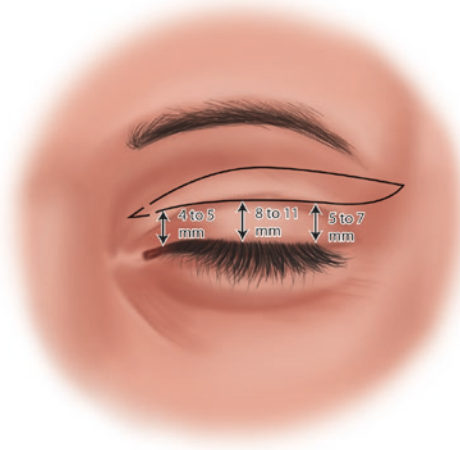

Fig. 6.9 Marking schematic for upper blepharoplasty. (Reprinted with permission from Holds JB, Ch. 10 Blepharoplasty pp. 75–82 in Manual of Oculoplastic Surgery, 4th ed. Mark R. Levine, et al. Springer, 2010)

- Create an incision through the septum to allow orbital fat prolapse.
- Medial fat pad is paler with a thicker capsule. Adequate hemostasis is crucial. Trim redundant fat anterior to the level of orbital rim. Do not force or pull fat. Central fat pad is usually preserved. Aggressive fat excision can result in hollow appearance.
- If lacrimal gland prolapse is noted, it can be suspended inside the orbital rim with mattress sutures.
- Skin closure: adequate hemostasis should be confirmed before closure. Skin only, dissolvable, or non-dissolvable suture can be used. Do not suture septum.

Note: If combining blepharoplasty with brow ptosis repair: perform brow ptosis repair prior to blepharoplasty incisions to prevent over-resection.

Postoperative Care

- Topical antibiotic ointment (ophthalmic) over sutures and eye.
- Prophylactic antibiotics (e.g., Keflex 500 mg q 6 hrs × 7 days).
- Cold compresses for the first 2 days.
- Follow-up within 1 week for suture removal.

Lower Lid Blepharoplasty

Preoperative Evaluation

Assess lower eyelid position, canthal position, tone, laxity, dermatochalasis, and proptosis.

- Lid function: orbicularis strength and eyelid position. Look for entropion or ectropion.
- Measure margin reflex distance (MRD2) – distance from light reflex to lower lid margin. If greater than 5.5 mm, it can be a sign of lower eyelid retraction.
- Lateral canthal position is 2–3 mm above medial canthal position.
- Test for tone (snap-back test): gently pull eyelid inferiorly and release; normal tone will result in return to baseline position immediately without blinking.
- Test for laxity (globe distraction test): Gently pull eyelid inferiorly. If able to pull a lower eyelid greater than 8 mm, than it is excessively laxed. If abnormal tone or laxity, patient will benefit from lid-tightening procedures to avoid malposition.
- Lower lid dermatochalasis can be tested by skin pinch test. May need skin excision or laser resurfacing for skin tightening.
- Fat prolapse: note the amount of prolapse, three fat pads in lower lid. Upward gaze can help in identification of prominent fat pads.
- Rule out thyroid eye disease, high myopia (long eyes), and bony asymmetry as causes of globe proptosis.

Surgical Technique

- Anesthesia: sedation, oral, intravenous, or general depending on patient's comfort.
- Place topical anesthetic in eye prior to placement of corneal protectors to prevent ocular injury.
- Local anesthetic with epinephrine is injected through inferior fornix. Infraorbital nerve block.

1. *Transconjunctival Approach (Most Preferred)*
   - Does not address anterior lamella skin excess but can be combined with skin excision.
   - Transconjunctival incision performed using (laser, Ellman RF, or monopolar cautery) 4–5 mm below tarsus along entire lid length. Incision is through the conjunctiva.
   - Blunt dissection through the capsulopalpebral fascia to expose the orbital septum.
   - Once the septum is exposed, gentle pressure on the globe will assist in locating lower fat pads.

- A small incision is made through the septum over each fat pad. The entire septum may be exposed, but this is often unnecessary in most cases.
- Without excessive tugging or manipulation, the exposed fat can be removed and sculpted.
- Always check for adequate hemostasis and avoid injury to inferior oblique muscle (between medial and central fads).
- Lids massaged superiorly after ideal contour is achieved.
- Skin-only closure, conjunctival suture is optional.

2. *Transcutaneous Approach*
   - Subciliary skin incision with #15C blade. Stay 2–3 mm inferior to lashes to avoid injury to hair follicles and this allows good cosmesis.
   - Carefully dissect skin flap 4–5 mm inferiorly. Avoid injury to pretarsal orbicularis.
   - Transect through preseptal orbicularis and septum to expose fat pads.
   - Sculpt medial, central, and lateral fat pads. Hypertrophic orbicularis oculi muscles may be judiciously removed if needed.
   - Advance skin superiorly and drape over the incision. Remove excess skin (very conservative).
   - The orbicularis oculi generally does not require resuspension. The skin may be closed with either resorbable or nonresorbable sutures.

Complications

- *Retrobulbar Hematoma/Hemorrhage:* may be caused by a bleeding vessel from the fat pad that retracts posteriorly or from bleeding edges of orbicularis muscle. Eye pain with progressive proptosis, ophthalmoplegia, and visual disturbance. Treatment is to remove sutures and possible lateral canthotomy with inferior canthotomy. Medical management includes hypotensive/osmotic agents (acetazolamide 500 mg IV or mannitol 1.5 g/kg), topical beta-blockers, oxygen therapy, and high-dose steroids (e.g., dexamethasone 3–4 mg/kg).
- *Lagophthalmos:* treatment initially should include lubricant eye drops with taping closure of eyelid at night. After 2 weeks, may initiate lid massage and stretch. If no resolution after 3 months, consider full thickness skin grafting from preauricular region or contralateral eyelid.
- *Excessive Skin Remnant or Fat:* allow 6 weeks for edema to resolve and removal of remaining skin/fat.
- *Lower Lid Hollowing:* treatment with autologous fat injection or dermal fillers such as hyaluronic acid.
- *Lacrimal Gland Prolapse:* reposition with a 5-0 non-absorbable suture passed through gland capsule to the periosteum of the anterior tip of the lacrimal gland fossa.
- *Suture Granuloma:* focal inflammation around the suture. Most resolve overtime, if persistent; injection of steroids, topical steroid application, or excision.

Brow Lift

- The eyebrow helps frame the eye and contributes to the perception of the eyelids.
- Eyebrow ptosis, especially laterally, contributes to the appearance of excess skin (Connell's sign) and cannot be optimally treated with blepharoplasty alone.
- The female eyebrow is ideally positioned in a vertical line with the medial canthus and nasal ala and horizontally aligns with the tail of the brow (Fig. 6.10). The female brow is ideally several millimeters above the orbital rim.
- The male eyebrow sits lower than the female eyebrow. The male brow sits along the level of the orbital rim.
- The eyebrow arches superolaterally with the maximal arch at or just lateral to the lateral limbus, before terminating in an imaginary line drawn between the nasal ala and lateral canthus.
- Eyebrow ptosis usually starts laterally and progresses medially with age. The frontalis

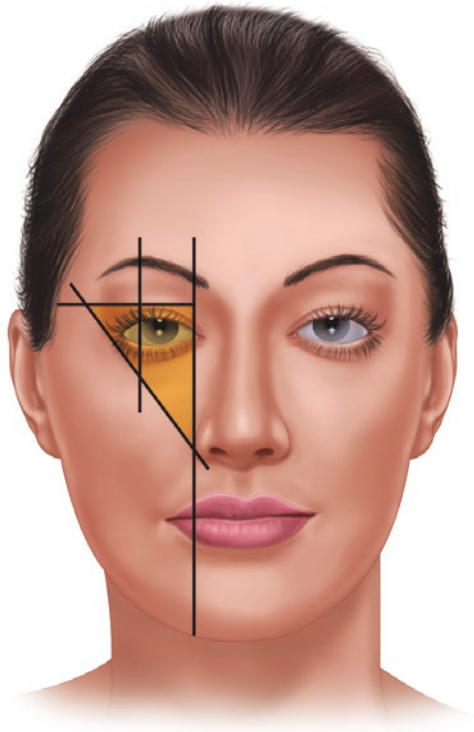

Fig. 6.10 Eyebrow position. (Reprinted with permission from Zimbler M. Cummings Otolaryngology 6th Edition. Elsevier Books; 2015)

has diminished activity lateral to the temporal fusion plane, which accelerates the effect of gravitational forces laterally. Compensatory frontalis elevation raises the eyebrow, but produces rhytids that become more visible as the skin and subcutaneous tissue thins. Lateral hooding will become more pronounced when the frontalis is relaxed. Chronic glabella contraction can lead to an angry appearance and depress the medial eyebrow.

Preoperative Evaluation

- Complete medical history including prior facial procedures, eyebrow tattoos, ophthalmic surgeries, facial nerve palsy, facial trauma, fluctuating symptoms, and cardiovascular risk factors.
- Prior LASIK surgery or symptomatic dry eye may necessitate a more conservative approach.
- Review of all current medications, including supplements, anticoagulants, or antiplatelet agents must be done. Patients on anticoagulants, or antiplatelet agents, have increased risk of bleeding even if the medicines are stopped preoperatively. A direct brow lift may be the best option for these patients, as hemostasis can be obtained under direct visualization, and less risk of postoperative hematoma.
- Eyebrow position and contour are judged with the frontalis relaxed.
  - With the patient's eyes closed, a hand can be placed on the forehead to stabilize the frontalis; the true brow position can then be appreciated when the patient opens their eyes.
  - Some patients remove brow hairs and they may have eyebrow tattoos, this must be recognized preoperatively.
  - Patients must be counseled on preoperative asymmetry.
- The facial nerve and eyebrow function are assessed to ensure full eyelid closure.
- The evaluation of the eyelids with the brow held to expected postoperative position to examine eyelid margin height (MRD) and residual dermatochalasis will accurately assess the need for adjuvant eyelid surgery.
- Assessment of hairline, forehead, and glabellar rhytids for incision planning:
  - Deep mid-forehead and suprabrow rhytids can be used to camouflage incisions.
  - Mild glabellar rhytids can be treated with neuromodulators. This can be combined with treatment of the lateral brow depressors in mild cases of brow ptosis. In more severe cases, forehead lifting with the release of the corrugators is recommended.
  - Corrugator myectomy can also be performed through an upper eyelid blepharoplasty incision if a brow lift is otherwise not indicated.
- The hairline and forehead length are important for procedure selection. Patients with a receding hairline, male-pattern baldness, or females who wear their hair back are poor candidates for a pretrichial incision. Pretrichial

forehead lifts shorten the forehead and coronal lifts will lengthen the forehead.
- All patients must have preoperative photos. A frontal view and oblique view to demonstrate secondary upper eyelid hooding are recommended. Any preoperative asymmetry must be captured by the photographs and documented and discussed with the patient.

Surgical Technique

- See Fig. 6.11 for incision markings for the various brow lift techniques.

1. *Indirect Brow Lift*
   - Performed through an open blepharoplasty incision, the periosteum is exposed at the superior orbital rim.
   - The retaining ligaments are released, and the brow fat pad sutured to the periosteum with 4-0 or 5-0 absorbable suture above the orbital rim.

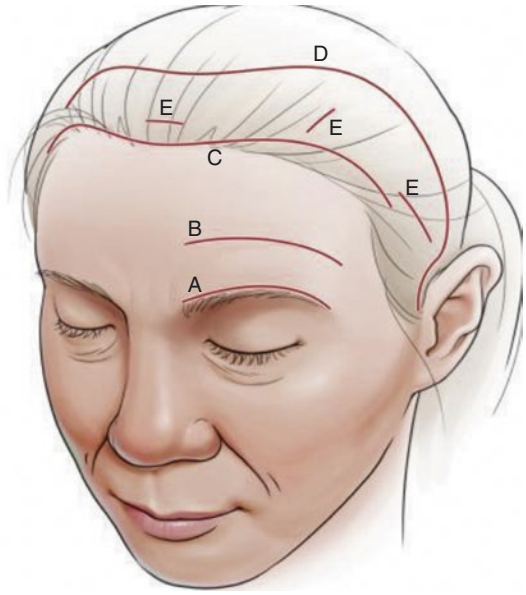

Fig. 6.11 Incision placement for different brow lift procedures. (A) Direct brow lift, (B) midforehead brow lift, (C) hairline incision brow lift, (D) coronal incision brow lift, (E) endoscopic brow lift. (Reprinted with permission from Benjamin W. Light B and Leong P. Brow Lift. Operative Otolaryngology. Elsevier; 2018)

   - Can be combined with a corrugator myotomy medially.
     - The supratrochlear and supraorbital neurovascular bundles can be injured medially.
     - Incomplete release or inadvertent transposition of corrugators can lead to abnormal contour and movement of the medial brow.
2. *Direct Brow Lift*
   - Greatest elevation per amount of tissue excised, also offers excellent control of brow contour. The most technically simple and shortest operation.
   - Traditionally used in males with heavy brows, patients with receding hairlines, or patients that do not want a more involved procedure.
   - Good for temporal brow ptosis, carrying the incision medially will result in a displeasing scar.
   - Can be combined with a mid-forehead lift to treat medial brow ptosis.
   - The redundant skin to be removed is marked preoperatively following the contour of the margins of the upper eyebrow.
   - The incisions are beveled in the direction of the eyebrow hairs to avoid damage to the follicles, with excision of underlying skin and subcutaneous tissue.
   - The frontalis is not touched.
   - Deep closure is important and done with buried 4-0 or 5-0 polyglactin sutures.
   - Skin closure is accomplished with a running 5-0 nylon or polypropylene.
   - Danger zones: the supratrochlear and supraorbital neurovascular bundles are located 1.7 cm and 2.7 cm, respectively, from a midline mark and courses along the anterior aspect of the frontalis. Damage will lead to scalp numbness.
3. *Pretrichial and Trichophytic Brow Forehead Lift*
   - Raises the eyebrows and lowers the hairline.
   - Effective in patients with a high or long forehead. Hairline and manner of wearing hair must be assessed preoperatively.

- The incision is placed slightly posterior to (trichophytic), or just anterior (pretrichial) to the hairline. This incision is generally confined medial to the conjoint tendons of the forehead.
- If lateral elevation is required, independent temporal incisions may be required.
- Traditionally, the incision is bevelled (10–15°) perpendicular to the hair follicles for preservation and regrowth through the incision. Identical bevel of the anterior and posterior incisions is vital for proper closure.
- Centrally, the dissection is in the loose areolar plane between the galea and periosteum (some surgeons advocate using the subperiosteal plane). Many also perform a trichophytic brow lift in the lipocutaneous plane in order to break up adhesions between the skin and subcutaneous tissue, thus eliminating static rhytids.
- Dissection is carried inferiorly to the level of the superior orbital rim.
- Corrugator release can be performed if indicated.
- Temporally the dissection plane is on the superficial aspect of the deep temporal fascia.
  - If unsure of the depth, a small incision to visualize the temporalis muscle can be performed.
  - The facial nerve is within the temporoparietal fascia and can be damaged if the dissection is too superficial.
  - The central and temporal planes are connected by the release of the conjoint tendon (fusion of periosteum, temporoparietal fascia, and deep temporal fascia).
- Wound closure is performed after an appropriate ellipse of skin is excised.
- Deep inciscions is close with 3-0 or 4-0 polyglactin.
- Superficial closure is performed with 4-0 or 5-0 polypropylene.

4. *Coronal Brow and Forehead Lift*
   - Infrequently used presently.
   - Lengthens forehead while raising brows.
   - Amount of skin to be removed can be done after flap or estimated in initial markings (best to err on excess than over-resection. If over-resection occurs, may need to mobilize the posterior flap).
   - An ear-to-ear incision is placed anterior to the coronal suture, 6 cm posterior to anterior hairline. Lateral extent is 1–2 cm above the ear insertion. Incisions should be bevelled with course of hair follicles.
   - The dissection is performed in the loose areolar plane between the galea and periosteum in the central region and just superficial to the temporalis fascia laterally.
   - Subgaleal dissection is performed until slightly above the supraorbital rim to allow access to glabellar musculature. Corrugator, frontalis, and procerus myotomies are performed as required.
   - Appropriate amount of tissue skin is excised and the skin is reapproximated. 1.5–2 cm of excision is required to gain 1 cm in brow elevation.
   - Reapproximate galea with 2.0 polyglactin followed by skin closure with staples or 3-0 polypropylene.
5. *Endoscopic Forehead Lift*
   - Raises the eyebrows and lengthens the forehead through small incisions (usually five incisions) within the hairline (Fig. 6.12).
   - Minimizes scarring and alopecia but not a good option for patients with high hairlines or males with severe brow ptosis.
   - Preoperative chemodenervation of the glabella can help with wound healing, particularly in patients with hypertrophic corrugators.
   - Facial nerve and sentinel vein – the facial nerve courses superiorly and medially from 0.5 cm below the tragus to 1.5 cm above the lateral eyebrow in the temporoparietal fascia just deep to the SMAS. The sentinel vein is located 3 cm lateral to the lateral canthus in a line drawn from the nasal ala. This marks the inferior termination of dissection unless a mid-face lift is concurrently performed.

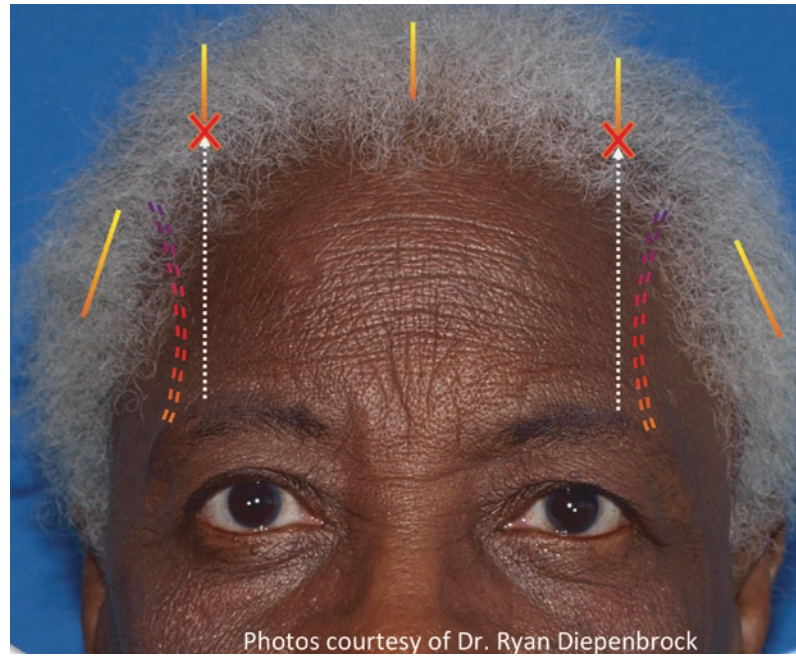

Fig. 6.12 Endoscopic brow lift incisions. (Image courtesy of Dr. Ryan Diepenbrock)

Procedure:

- Anesthesia can be local or general.
- Full strength local anesthesia with epinephrine is used along the orbital rims, incisions, and supratrochlear and supraorbital neurovascular bundles.
- Tumescent is used to infiltrate the remainder of the forehead.
- All incisions are placed approximately 2 cm within the hairline and made with a #15 blade.
- The central incision and paracentral incisions are vertical, approximately 1.5 cm in length and subperiosteal in depth.
- The central incision is directly above the center of the glabella. The radial incisions are approximately 3.0 cm lateral to the central incision at the desired level of greatest brow height.
- The temporal incisions are made in the center of a perpendicular line drawn from the nasal ala to the lateral canthus.

Dissection

1. Central
   - A blunt elevator is used for subperiosteal dissection, connecting the central incisions, posteriorly and inferiorly.
   - Can be done without the endoscope until 2 cm above the superior orbital rim (all other dissections are done under direct visualization).
   - Adequate posterior dissection is key for mobility and elevation (10 cm posteriorly up until lambdoid suture).
   - Initial dissection stops at the temporal line of fusion laterally.
2. Temporal
   - A #15 blade is used to incise the skin through the dermis, then meticulous dissection is performed to expose the "glistening" white deep temporal fascia.
   - Blunt dissection along the deep temporal fascia is performed anteriorly and posteriorly.
   - The conjoint tendon is released connecting the two planes of dissection. Dissection is carried from temporal to medial scalp rather than vice versa.
   - Dissection is then carried anteriorly toward the orbital rim. The periosteal attachments must be released for adequate mobilization and lift.
3. Superior Orbital Rim and Glabella
   - After elevation of the periosteum from the superior orbital rim, the periosteum is cut for a full release.

- This can be done delicately with a curved elevator as well as bluntly with scissors.
- Opening the periosteum will expose the glabella and supraorbital and supratrochlear bundles.
- Endo graspers are used to strip the fibers of the corrugators and a strip of procerus if indicated.
- Excision of the depressor supercili is not recommended as the periosteal spreading results in significant weakening.

4. Fixation
   - Endotines, lactosorb screws/periosteal sutures, and titanium percutaneous screws can be used.
   - Fixating at the posterior aspect of the paracentral incisions provides adequate elevation.
5. Closure
   - Laterally, the superficial temporal fascia is advanced temporally, secured to the deep temporal fascia, and closed with 3-0 polyglactin.
   - Deep closure is with 4-0 absorbable sutures.
   - The scalp incisions can be closed with interrupted non-absorbable/absorbable suture, or staples.

Postoperative Care

- All patients are advised to use topical antibiotic ointment, avoid showering for 24 hours postoperatively, use ice, and avoid physical activity in the perioperative period.
- Compression wraps can be placed over pretrichial, trichophytic, coronal, and endoscopic lift incisions at the time of surgery.
  - This decreases the risk of hematoma and promotes adherence of the periosteum.
  - Wraps are removed 1–2 days postoperatively.

Complications and Management

- *Alopecia*: most common in coronal incisions. Limited use of electrocautery including judicious use of bipolar cautery prevents unnecessary collateral damage to hair follicles. The skin and subcutaneous tissue should be incised at a 10–15 degree bevel to prevent transection and disruption of the hair follicles. Additionally, limiting suture passes through hair-bearing areas minimize follicle damage. Hair loss is usually temporary. Consider hair transplantation for cases where regrowth does not occur.
- *Bleeding:* significant bleeding is usually caused by the superficial temporal, supraorbital, supratrochlear, or zygomaticotemporal arteries. Opening of the incisions and identification of bleeding source with drain placement is necessary in some cases. Some surgeons routinely place drains postoperatively.
- *Facial Nerve Damage:* avoided by maintaining proper dissection planes. Will result in orbicularis and brow weakness. Damage to frontal branch may impair orbicularis oculi and require globe protection such as eye patches, temporary tarsorrhaphy, or gold weight implantation to upper eyelid.
- *Numbness and Paresthesias:* expected transiently. Damage to the supratrochlear or supraorbital bundles will result in permanent dysfunction.
- *Scarring and Depressed Incisions:* closure under tension increases risk. A proper multilayered closure minimizes risk. Laser resurfacing can be used for direct brow lift incisions once the wound has matured.
- *Asymmetry:* meticulous preoperative evaluation reduces risk. Can result from failure of fixation after an endoscopic lift. Revision with refixation is recommended in the early postoperative period in these cases.
- "*Surprised Look" with Brow Widening*: occurs with overzealous resection of the corrugators. Consider Botox in selected regions.
- *Lagophthalmos:* mild lagophthalmos is common in the early perioperative period and managed with topical lubrication. More common when combined with upper eyelid blepharoplasty, or if the patient has had prior surgery. Fixation devices can be explanted or revised if necessary.

Pearls

1. Meticulous preoperative planning to evaluate function, asymmetry, and the need for adjunct procedures are key for success.
2. Evaluation of the forehead length, hairline, the manner hair is worn, and rhytids to camouflage scars ensure proper procedure selection.
3. Visualization is the key to ensure proper dissection planes and avoid damage to neurovascular bundles.
4. Limited resection of the corrugator and procerus muscles minimizes postoperative contour abnormalities and brow widening.
5. Matching the bevel on both sides of the incision and meticulous wound closure minimize scarring.
6. Limitation of electrocautery, use of blade for incisions, and minimizing suture passes reduce the risk of alopecia.

Otoplasty

- Excessive otic projection or prominauris has two main root causes.
  - Lack of a well-defined antihelical fold; corrected with Mustardé sutures.
  - Excessive conchal bowl depth; corrected with Davis technique.
  - Combination of the two.
- Ideal candidates are preschool or kindergarten children (5–6 years old).
- Roughly 85% of ear growth is completed by 3 years old.
- Ear is fully developed by age 7–8. Cartilage is more pliable and easier to mold and manage.
- Studies have demonstrated an improved quality of life, increased self-esteem, and decreased psychosocial anxiety when performed at a younger age.
- Blood supply of the external ear consists of two branches of the external carotid artery: the posterior auricular artery and vein and the superficial temporal artery and vein.

Preoperative Evaluation

When the patient presents for an otoplasty consultation, it is first imperative to ask his or her chief complaint. In the pediatric population, excessive otic projection is often accompanied by ridicule from peers or siblings, thus pediatric patients are often eager and willing to undergo the procedure. Adults often state a long history of teasing dating back to their childhood.

During the history and physical, the surgeon should specifically assess the external and internal ear for the following:

- Symmetry
- Dystopia of the ears
- Anatomy of the ears
- Root cause of excessive projection (lack of antihelical fold/conchal bowl hypertrophy/combination)
- Flexibility and resiliency of the cartilage
- Signs of skin lesions or history of otitis media or externa
- Hearing loss
- Integrity and condition of the tympanic membrane and external auditory canal

In addition to a thorough history and physical exam, preoperative photos and measurements are imperative. The following is a list of recommended photos:

- A frontal view
- Right and left ¾ view
- Right and left profile views
- Submental vertex
- Posterior view
- Isolated ear photos with a ruler:
  - Conchal bowl depth
  - Measurement of the superior, middle, and inferior mastoid to helix distance

Physical Findings

- First the surgeon must access the degree of protrusion. This is accomplished by

determining the auriculocephalic angle and the scapha conchal angle.
- A normal auriculocephalic angle is roughly 25–35° (Fig. 6.13). An abnormal or protrusive angle is generally greater than 45°.
- The scapha conchal angle is normally 75–100° (Fig. 6.13).
  - Lack of an antihelical fold, which causes failure of the ear to fold normally will typically present with a scapha conchal angle of >100°.
- The depth of the conchal bowl is measured from the cavum concha to the anterior segment of the anti-helix. A normal depth should be roughly 8 mm.

Technique for Otoplasty

Preoperative Markings

- First, the proposed antihelix is drawn.
- Next, the proposed markings for the placement for the Mustardé sutures are drawn (Fig. 6.14).
  - Trapezoidal in shape with the anterior arm being slightly shorter than the posterior arm.
  - Markings should be placed roughly 7–8 mm on either side of the proposed crest of the newly formed antihelical fold.
- Using calipers or a ruler, the conchal bowl is measured to a depth of 8 mm from the transition line of the antihelix. This ensures a proper height of residual bowl remains after excision (generally, in a kidney bean fashion).
- Posterior auricular incision is marked. This is generally fusiform in fashion extending superiorly to inferiorly to gain surgical access to the entire ear. Incision is made 2–3 mm lateral to the sulcus.
  - This will ease dissection to the helix of the ear as well as promote good tissue approxi-

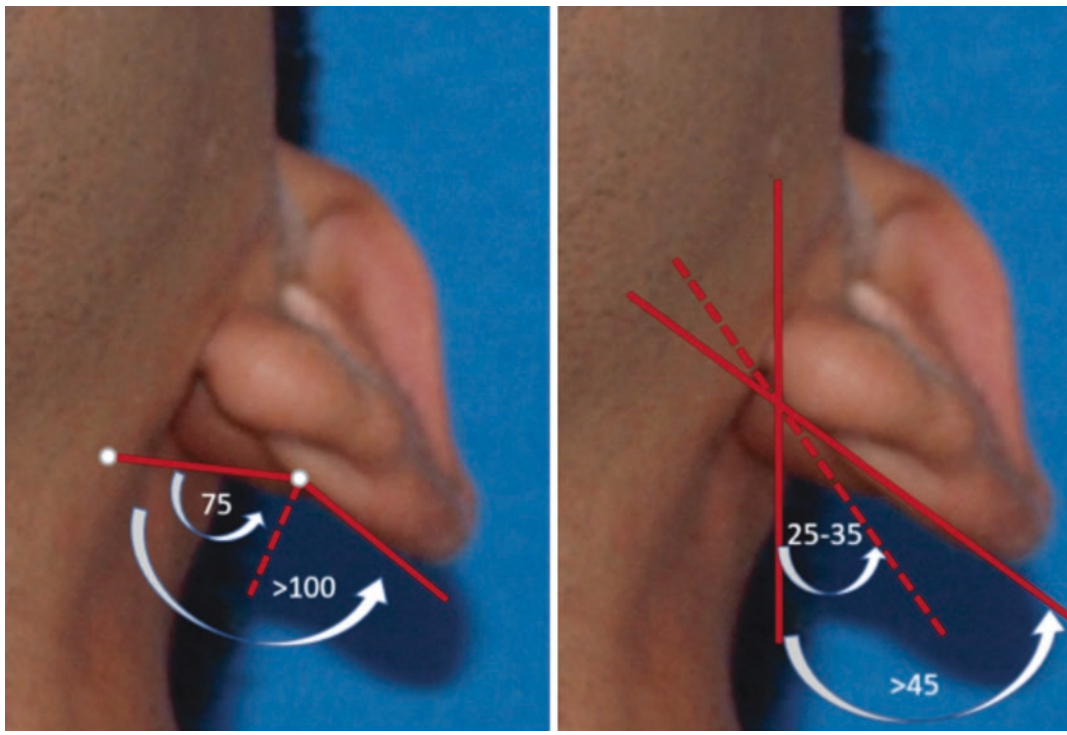

Fig. 6.13 A normal auriculocephalic angle is 25–35°. Greater than 45° is considered excessive. Scapha conchal angle is normally 75–100°. Greater than 100° is usually attributed to a lack of an antihelical fold. (Images courtesy of Dr. Ryan Diepenbrock)

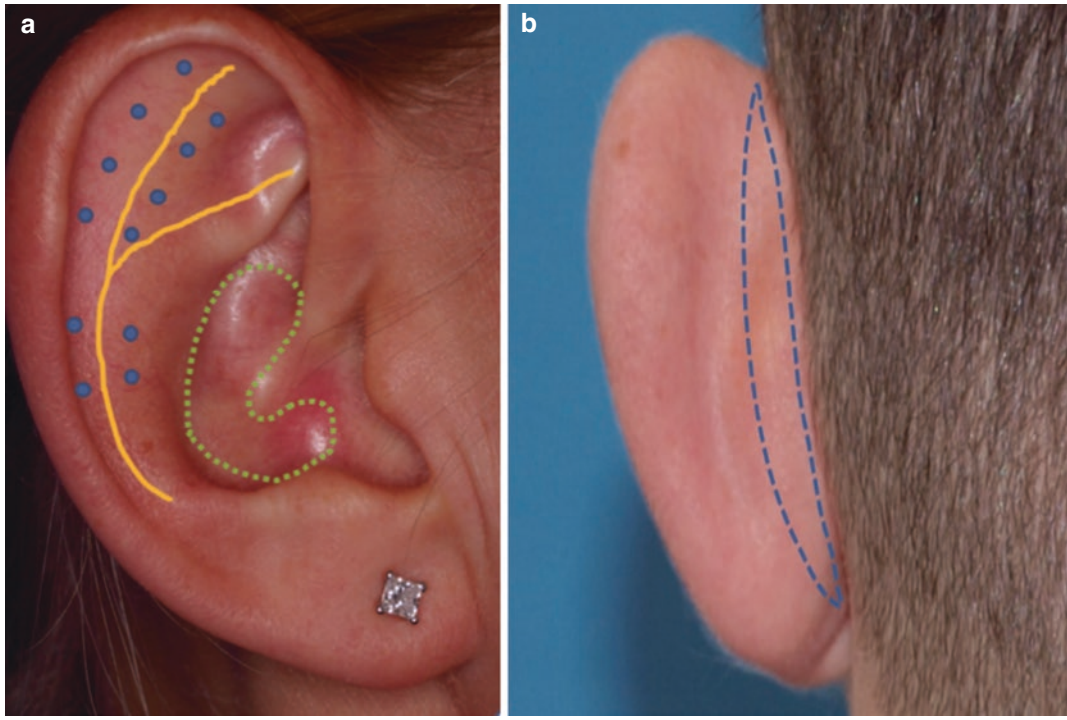

Fig. 6.14 (**a**) Proposed markings of the Mustardé sutures placed to form the new antihelical fold, and the planned cartilage excision (Davis technique) for conchal bowl resection. (**b**) Planned fusiform excision of skin in the posterior sulcus. The preoperative markings are crucial because they will be used during the surgery to facilitate tattooing of the cartilage for accurate suture placement and bowl reduction. (Images courtesy of Dr. Ryan Diepenbrock)

mation during closure; 5–10 mm wide depending on the amount of setback needed.
  - Only remove enough skin to facilitate extension of the incision laterally to ensure access to preoperative markings.

Intraoperative Markings

Prior to local anesthetic infiltration and nerve blocks, the surgeon may decide to use methylene blue to tattoo the cartilage.

- A 30-gauge needle (1/2″–3/4″) is passed from the external markings through the skin, cartilage, and posterior body of the ear.
- The needle is inserted into the tip of a 1 ml syringe filled with methylene blue and withdrawn.
- Each of the trapezoidal dots is inked.
- Outline of the kidney bean shaped marking is completed in identical fashion. The final markings should appear as in Fig. 6.14.

Surgical Procedure

- The fusiform marking is excised to expose the posterior ear cartilage.
- A subperichondrial dissection is completed until all the markings for the Mustardé sutures and conchal bowl excision are visualized. Usually the subperichondrial dissection is carried nearly the full distance to the helical cartilage. The methylene blue tattoos are visualized and Mustardé sutures commence.
- Mustardé sutures are utilized to create a crease in the cartilage in order to form a new antihelical fold.

- Using 4-0 Mersilene® sutures, the needle is passed from posterior to anterior through the cartilage; being cognizant not to buttonhole the skin. The knot is tied in a vertical vector to promote the curvature along the long axis of the newly formed antihelical fold. Usually two to three horizontal mattress sutures are placed.
- Once the antihelical fold is created, attention is turned to reducing the conchal bowl. The tissue posterior to the bowl must be excised to create what will essentially become the new floor of the conchal bowl. The tissue posterior to the bowl is removed to expose the mastoid fascia. The excised tissue will include subcutaneous tissue, fat, and a portion of the posterior auricular muscle. The mastoid fascia must be thoroughly exposed, smoothed, and free from remnants of superficial tissue.
- A full thickness incision through the cartilage of the conchal bowl is made. The cartilage is then dissected from the overlying perichondrium and removed.
- After verification of final measurements, mattress sutures (Furnas sutures) are placed with 4-0 Mersilene® at the superior, middle, and inferior aspects. The sutures are placed through the remaining cartilage and secured to the mastoid fascia. Next, the sutures are individually tied and placed under appropriate tension using a ruler or caliper to verify final position of the ear.
- The incision is then closed with a running 5-0 fast gut suture. Bolsters may be placed using dental cotton rolls or resorbable sutures through the bowl to the fascia to close dead space. Xeroform gauze is placed in the newly formed bowl as well in the sulcus. A dressing consisting of fluffs, Kerlix™, and Coban™ is then applied.

Postoperative Course

- The patient is discharged with a 5–7 day course of oral antibiotics, a non-steroidal anti-inflammatory medication, a small dose of narcotic analgesics, 3 days of postoperative steroids, and antibiotic ointment.
- The patient is seen the next day for follow-up. He or she is instructed to bring an athletic headband to the follow-up appointment. At the follow-up, the head wrap is removed and the surgical sites are inspected for hematomas and tissue integrity.
- Patients are instructed to wear the athletic headband for 24 hours per day for the first 2 weeks. At that point, they are to wear the headband in the evening and while sleeping for an additional 2 weeks.
- The patient is instructed to clean the incisions with 50:50 hydrogen peroxide/H_2O and liberally apply antibiotic topical ointment 4–5 times per day for at least 1 week.

Postoperative Complications

- *Infection* – perioperative antibiotics such as cefazolin to cover Staphylococcus aureus. Some prefer preoperative ciprofloxacin to cover Pseudomonas aeruginosa. Cultures for causative bacteria as well as sensitivities for post-op infection.
- *Hematoma* – bolster dressing, Xeroform™ gauze (3% bismuth tribromophenate in petrolatum blend with fine mesh gauze), headwrap. Follow-up in 1 day. Drain as needed, open incision, and localize the bleeding vessel or consider returning to the operating room. Requires early intervention and follow-up to prevent perichondritis.
- *Cauliflower Ear* – caused by untreated hematoma or fluid collection under the perichondrium (prevention is key).
- *Wound Dehiscence* – keep the wound clean with 50:50 hydrogen peroxide and water cleanses. Cover the wound with Xeroform® dressing/wet-to-dry packing. Consider reapproximation for large, non-healing wounds.
- *Tissue Sloughing or Necrosis* – topical vasodilators such as nitropaste or hyperbaric oxygen.
- *Perforation of External Auditory canal (EAC)* – primary closure with gut suture and otic antibiotic drops.

- *EAC Stenosis* – ensure bowl is set back, difficult to correct. Avoid problem.
- *Telephone Ear Deformity* – over-tightening or over-resection of middle-third antihelical region. Treatment of overcorrection with a Mustarde procedure can be corrected by removing the offending suture, scoring the cartilage and suspending appropriately.
- *Reverse Telephone Ear Deformity* – opposite of the above with over-tightening or over-resection of the superior and inferior antihelical regions. Treatment of overcorrection with a Mustarde procedure can be corrected by removing the offending suture, scoring the cartilage and suspending appropriately.

Chemical Skin Resurfacing

Skin Preparation Prior to Peels

- Tretinoin (Retin-A) 0.05% or 0.1% twice daily
  - Metabolite of vitamin A.
  - 2–4 weeks prior to treatment.
  - Thins and compacts stratum corneum (Fig. 6.15) by decreasing cohesiveness allowing easier penetration of peeling agents.
  - Induces type I procollagen.
  - Reduces melanin content.
  - Stimulates angiogenesis and epithelial cell mitotic activity leading to rapid wound healing.
  - Normalizes keratinization allowing for chemical peel agent to penetrate deeper and more evenly.
  - Not to be used within 1 year of laser resurfacing due to scarring.
  - Don't forget, retinoic acid (Isotretinoin, Accutane®) is contraindicated 1 year prior to treatment.
- Glycolic Acid 5–10%
  - 4–6 weeks preoperatively.
  - Reduces thickness of stratum corneum allowing increased chemical peel penetration.
- Herpetic Prophylaxis

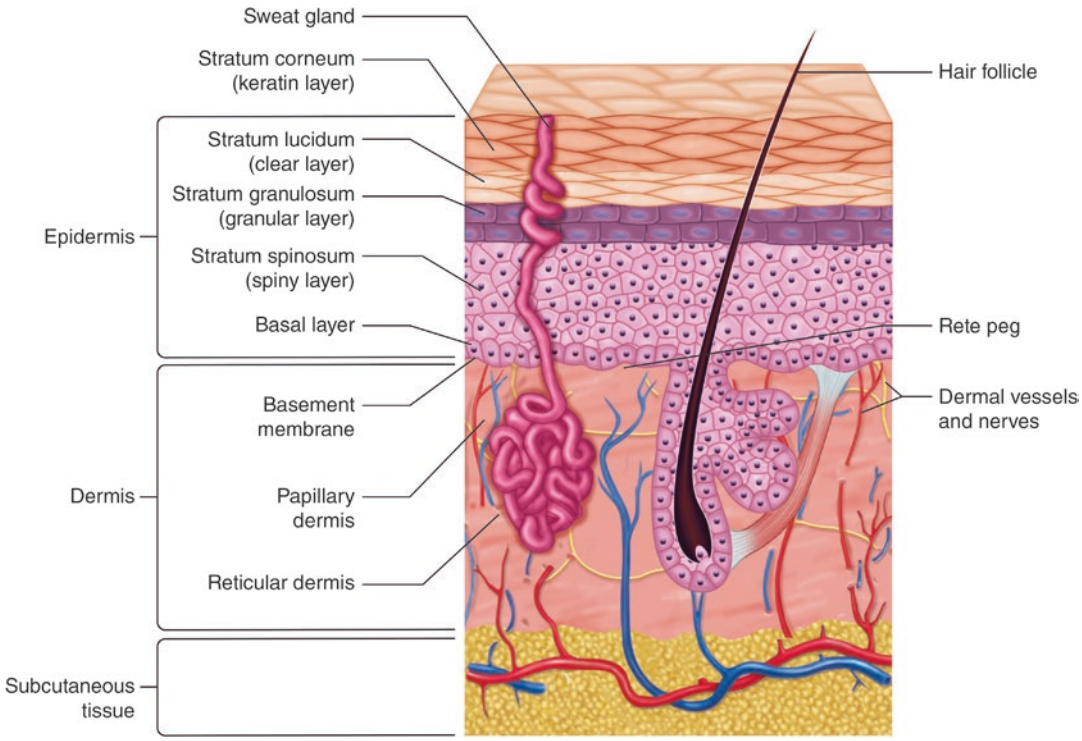

Fig. 6.15 Layers of epidermis. (Reprinted with permission from Young A. Rehabilitation of burn injuries. Phys Med Rehabil Clin 2002;13(1):86)

  - For medium-depth chemical peels or laser resurfacing.
  - Acyclovir 400 mg three times daily, valacyclovir 500 mg twice daily, or famciclovir 250 mg twice daily for 3 days prior to the procedure and continued for 10 days postoperatively.
- Hydroquinone 4%
  - Inhibits tyrosinase enzyme preventing melanocytic production of melanin.
  - Reduces risk of post-inflammatory hyperpigmentation.
  - Used twice a day.
- Sunscreen
  - Must be used daily to lessen the development of hyperpigmentation.
  - Allows skin to rest before peel/resurface.
  - Needs to be started 3 months prior.

Patient Selection

- Indications are for patients with extensive rhytids (will only treat passive), seborrheic or actinic keratosis, acne vulgaris, melasma, and post-inflammatory hyperpigmentation.
- Fitzpatrick skin types 1 and 2 are best candidates, 3–6 have a higher risk of post-inflammatory hyperpigmentation.
- Be cautious when treating neck skin due to reduced or limited adnexal dermal structures which may result in hypertrophic scar. This is due to reduced epiboly as pilosebaceous units provide progenitors of new epithelium. Superficial peel can be used more safely on neck skin.
- History of herpes labialis is a relative contraindication due to increased risk of scarring.
- Patients on hormone replacement therapy (birth control or menopause treatments) are at a higher risk of drug-induced increased melanocytic activity and pigmentary changes.
- Caution with patients with inflammatory skin conditions such as psoriasis or vitiligo, which can exacerbate and spread to face.
- Avoid patients with use of isotretinoin within past year.

Chemical Peeling

- Chemical peeling is a controlled exfoliation process.
- Classified by depth of burn (Fig. 6.16).
- Light peels are for fine lines and wrinkles.
- Medium and deep peels help improve scarring, texture, and blemishes.
- Work by causing keratolysis and keratocoagulation.

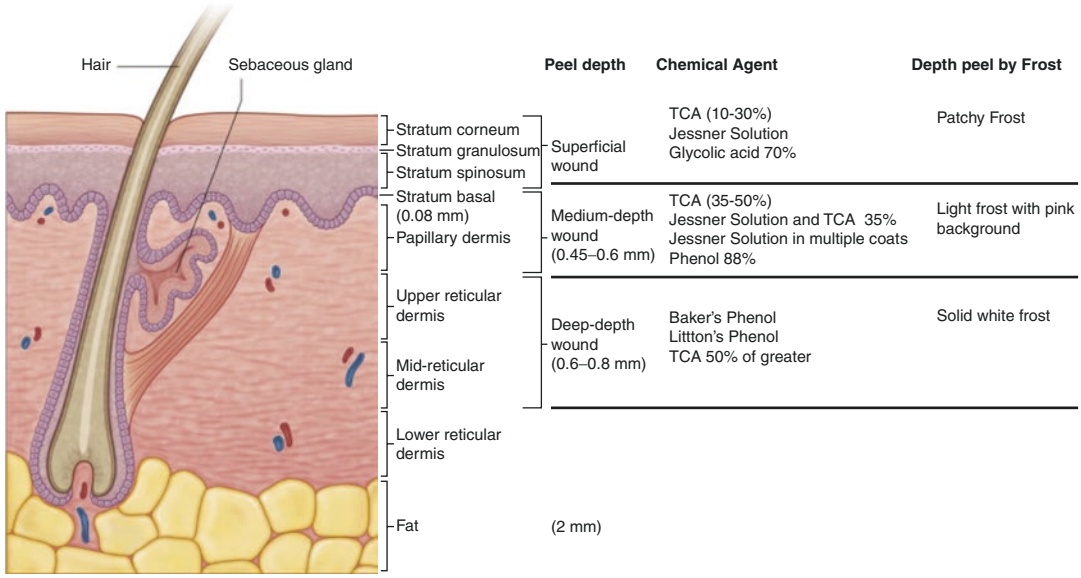

Fig. 6.16 Chemical peel wound depth and chemical agent. (Reprinted with permission from Landau M and Ghannam S. Surgery of the Skin 3rd Edition. Elsevier Books; 2015)

- Lighter peels usually work by keratolysis to interrupt adhesions for exfoliation but no effect on deeper wrinkles (e.g., glycolic acid, lactic acid, and salicylic acid).
- Deeper peels denature and coagulate proteins (e.g., TCA and phenols).
- White frosting is due to precipitation of salts.

Technique

- Mark the mandibular border prior to laying patient supine to aid transition line into neck.
- Anesthetize with local blocks and/or sedation if medium to deep peel planned. Handheld fan for cool air could be used for superficial peels.
- Prepare skin to remove oils, dirt, and impurities with agents such as acetone or alcohol on gauze pads.
- Peel of choice applied with cotton tip applicator with care around eyes. Deep line and scar treatment aided with swab stick to work in.
- Monitor for frost level for desired penetration. Neutralizing agent should be available (water for dilution is an option as well) for spills.
- Once depth of desired peel achieved, wash off with water/neutralizing agent and cleanse skin.
- Petroleum jelly is applied to face.

Postoperative Care:

- Facial rinses with tepid water or 0.25% acetic acid-soaked sponges to loosen desquamated skin cells and loosen coagulum 4 × daily for 10 days followed by application of petroleum jelly.
- Return to maintenance program including cleanser, sunscreen, and topical steroid.

Complications

- *Hyperpigmentation* – hydroquinone 4% and tretinoin treatments. Seen usually 30 days later.
- *Hypopigmentation* – can blend in with CO_2 laser or treat with topical oxsoralen cream 1% weekly. Usually occurs later on, about 6–12 months after treatment.
- *Infection* – herpes simplex should be treated with double the acyclovir/valacyclovir prophylactic regimen if provided or treated with traditional doses. *Candida albicans*, examine with KOH to confirm. Treat with topical antifungals and discontinuation of petroleum jelly dressing. Bacterial infection can be cultured and treated with parenteral or topical antibiotics.
- *Milia* – clogged hair follicles may form cyst-like structures. Normally resolve with improved skin hygiene. May be treated with needle evacuation or topical tretinoin.
- *Scarring* – treated with injection of corticosteroids. 585-nm pulsed dye laser can also be used to treat the area. Application of occlusive silicone strips may help as well.
- *Persistent Erythema* – erythema lasting over 6 months, most redness should resolve by month 3 after treatment. Firstly, patient must stop all skin care as the increased sensitivity of the skin may be the cause. Question patient about skin exposure to sun and use of sunblock. Examine with potassium hydroxide test for subclinical fungal infection, if positive treat with antifungal. If all negative, treat with topical steroid cream, expect to resolve in 12 weeks.

Laser Resurfacing

Patient Selection

- Similar to chemical peel.

Laser Ablation and Pre-treatments

- Appropriate skin care is essential for facial rejuvenation.
- Cleansing to remove oil, dirt, bacteria, and dead skin. Mild detergents such as Cetaphil® or Neutrogena® are recommended.

- Moisturizer to decrease epidermal water loss.
- Daily sun protection prevents premature aging and decreases postoperative inflammatory hyperpigmentation.
- Prescription of topical retinoids improves appearance of photodamaged skin and increases collagen production resulting in smooth tone and texture.
- Use of retinoids prior to lasers or chemical peels also aids in re-epithelialization postoperatively.

Ablative Skin Rejuvenation

- Light amplification by stimulated emission of radiation (LASER) has become a popular modality for skin resurfacing.
- Fractionated CO_2 laser resurfacing is safe and effective for photoaging and treatment of wrinkles. It vaporizes small areas (MAC – micro ablative columns) of skin in a grid-like pattern with undamaged skin in between to allow faster healing.
- Energy: amount of energy delivered to each MAC per pulse (increasing energy increases depth and coagulation).
- Spot size: diameter of each MAC (small spot size with high energy increases depth of penetration).
- Fluence: energy per MAC divided by spot size area (allows for comparison of different lasers) in J/cm^2.
- Pulse duration: amount of time to deliver each pulse.
- Power: energy per MAC divided by pulse duration (higher power results in deeper penetration).
- Density: number of MACs per unit area of grid (higher density decreases distance between MACs – increased erythema and edema). Lower density is recommended in areas of hair follicles and thin skin such as eyelids.
- Differences between lasers depend on the media in which the excited photon energy is released.
- CO_2 laser: infrared energy at wavelength of 10600 nm that is specific for water. Ideal for skin as it consists of 70% water. Higher risk of lateral thermal damage as it is also absorbed by proteins and fats. Minimal fluence necessary is 4–5 J/cm^2.
- Er:YAG laser (erbium:yttrium-aluminum-garnet): infrared radiation at wavelength of 2940 nm. More specific for water than CO_2 laser. Lower risk of adjacent thermal damage but higher risk of bleeding. Better for elevated lesions or scars. Minimal fluence necessary is 0.5–1.7 J/cm^2.
- Hybrid lasers: combination of CO_2 and Er:YAG.
- Anesthesia is administered with regional blocks with 1–2% lidocaine with epinephrine or subcutaneous infiltration of dilute 0.3% lidocaine with epinephrine. Topical proparacaine to eyes before placing metallic eye shields.
- Post-laser care involves dilute vinegar soaks (antibacterial and antifungal). Aquaphor or Vaseline® to act as a barrier and moisturizer. Cool compresses. Avoid cosmetics until the skin surface is healed.

Botulinum Toxin

- Botulinum toxin A, trademarked under the name Botox®, inhibits the release of the neurotransmitter acetylcholine at the neuromuscular junction of nerve terminals, causing a temporary paralysis of the injected muscle. It targets SNAP-25, a presynaptic membrane protein containing acetylcholine vesicles.
- Other available forms are abobotulinumtoxin A (Dysport®), prabotulinumtoxinA-xvfs (Jeuveau®) and incobotulinumtoxin A (Xeomin®).
- All are derived from the gram-positive spore-forming bacteria *Clostridium botulinum* type A.
- Botox® cosmetic is approved by FDA for the temporary improvement of glabellar rhytids caused by the procerus and/or corrugator muscles and crow's feet caused by lateral orbicularis oculi in adult patients 18–65 years of age.

In 2017, it was approved for the temporary improvement in the appearance of moderate to severe forehead lines associated with frontalis muscle activity in adults. This approval makes the brand the first and the only neurotoxin indicated for three facial treatment areas – forehead lines, crow's feet lines, and glabellar lines.

- It must be recognized that the use of Botox® for the eyelids, nasal, cervical, and perioral areas for cosmetic results is considered off-label.
- The paralytic effect of neurotoxins ranges from 3 to 6 months; however, the FDA approval is for 3–4 months duration depending on the brand. Recovery is based on new axonal sprouting and new SNAP-25 production.
- Peak effect is 14 days, will start seeing results in 48 hrs.
- The lethal dose in 50% of humans (LD_{50}) for Botox® is 2500–3000 Units, or approximately 40 U/kg.

Evaluation of the Neurotoxin Patient

HPI

- What experience does the patient have with neurotoxin? Previous treatment, with whom, and a record of injection points with dosing.
- What expectations does he or she have with neurotoxin treatment? It will treat dynamic wrinkles only, not static or deep wrinkles.
- Medical history
  - Blood thinning medications or herbs as this can lead to excessive bruising.
  - Medications that interfere with neuromuscular transmission such as curare-type medications, aminoglycosides, muscle relaxants, or anticholinergics may potentiate Botox® effects.
  - There is a small risk of developing clinical antigenicity to albumin contained within each vial, and thus rendering the Botox® product ineffective. Directly inquire about any known allergy to albumin.

Table 6.7 Neuromuscular conditions contraindicated for Botox treatment

Neuromuscular disorders
Myasthenia gravis
Amyotrophic lateral sclerosis (ALS)
Multiple sclerosis (MS)
Eaton-Lambert syndrome

  - A history of specific neuromuscular conditions is pertinent to ask the potential neurotoxin patient, as the product must be used with caution in these patients as it can exacerbate preexisting conditions (Table 6.7).

- Maxillofacial exam
  - Rule out skin infections.
  - Evaluate for heavy use of foundation to hide wrinkles and hairstyle.
  - Evaluate for thickened scars as this may be a relative contraindication for injection site due to lack of dissipation.
  - Ptosis should be evaluated as weakened frontalis muscle can lead to exaggerated eyelid droop.
  - It is also important to have photo documentation.

Important Facial Musculature in Botulinum Toxin Treatment (Fig. 6.17)

- Muscles commonly treated and their clinical effects are presented in Table 6.8.

Reconstitution of Botox® (Allergan)

- Store refrigerated (2–8 °C), can store up to 24 months, once opened manufacturer recommends use within 4 hours.
- Reconstitute with sterile, preservative-free saline. Stir, do not mix vigorously.
  - 1.25 mL saline per 50 U vial = 4 U/0.1 mL
  - 2.5 mL saline per 100 U vial = 4 U/0.1 mL

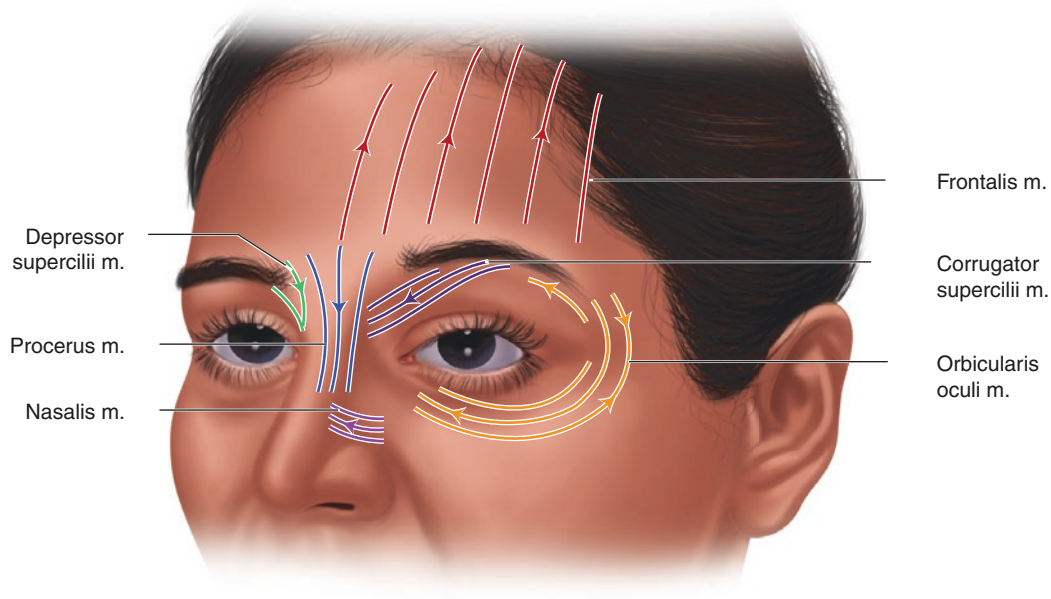

Fig. 6.17 Facial muscles of expression and their action. Arrows indicate the direction of contraction. The mimetic lines are created perpendicular to the pull. (Reprinted with Permission from Qaqish C. Atlas of the Oral and Maxillofacial Surgery Clinics. Elsevier. 2016)

Table 6.8 Mimetic muscles of the face

Muscle	Action	Clinical effect
Orbicularis oculi	Closure of eyelids and lateral eyebrow depression	Crow's feet
Corrugator supercilii	Depresses and adducts medial half of eyebrow	Creates vertical furrows above the nose "11's"
Procerus	Depresses medial eyebrow	Creates horizontal furrows above the nose; bunny lines
Depressor supercilii	Depresses head of eyebrow	Depresses head of eyebrow
Frontalis	Furrows forehead, elevates eyebrows	Creates forehead horizontal furrows
Orbicularis oris	Purses and puckers lips	Vertical lip lines
Depressors of mouth: depressor labii inferioris depressor anguli oris	Depresses lateral angle of mouth	Marionette lines
Platysma	Depresses lower lip and jaw	Platysmal banding

Treatment of Botox®

- Obtain informed consent for treatment.
- Determine your treatment plan prior to injecting, ask patient to activate muscles of planned injection to identify areas of maximum muscular contraction.
- Ensure makeup is removed, apply ice to the area for 20 sec, and pretreat skin with alcohol.
- Place patient in upright position.
- May enter skin and muscle either in an oblique or perpendicular injection.
- Inject directly into muscle except for crow's feet, which are injected subdermally.
- Peri-orbital injections: injection sites placed at least 5–10 mm from orbital rim and 5 mm medial and lateral to supraorbital nerve.
- Avoid forehead injections lateral to the lateral canthus to prevent inhibition of temporalis function.
- Inject at least 2 cm above eyebrow to prevent ptotic brow.
- Change the needle as needed to diminish pain from dulling; injection slowing can also diminish pain.

Table 6.9 Suggested Botox injections

Forehead (frontalis)	2–4 U per injection site
Glabella (procerus and corrugator)	5–7 U per injection site
Crow's feet (lateral orbicularis)	3–5 U per injection site

- Apply pressure for 1–2 min.
- Instruct patients not to disturb the treated area for 4 hours to prevent spread to unwanted regions.

Suggested Botox® (Allergan) treatment dosages (Table 6.9)

Post-op Instructions/Expectations

- Expect skin wheals posttreatment; usually resolve within 20–30 minutes.
- Ice the areas after treatment.
- Remain upright for 4 hours and do not apply makeup, scrub, or wash face for 4 hours (this prevents diffusion of the product).
- Exercise may be resumed the next day.
- A noticeable effect may be seen in 3–5 days with peak effect in 30 days.

Complications

- *Post-op Bleeding* – apply pressure; do not rub as this may spread the toxin to undesired areas.
- *Hematoma* – usually self-resolving.
- *Chemodenervation of Unwanted Muscle* – involving the levator palpebrae superioris causing temporary blepharoptosis (this can be temporarily reversed by treatment with alpha-adrenergic agonist drops (e.g., apraclonidine 0.5%)); ptosis usually does not last as long as the intended treatment (on average lasts about 3 weeks). Chemodenervation of zygomaticus muscle can create lip asymmetry.
- *Inability to Close Eye* – paralysis of orbicularis oculi. Tape eye shut and placement of eye lubricant advised until paralysis resolves.
- *Diplopia* – undesired entry of botulinum around extraocular muscles may lead to a binocular diplopia. This should be a transient problem. Refer to an ophthalmologist for evaluation (possible prism lenses can be prescribed until effect has diminished).
- *Xerophthalmia* – botulinum affecting lacrimal gland will block acetylcholine release leading to diminished tear production. Repeated treatment may cause a chronic dry eye syndrome. Supportive care with lubricating eye drops; should reverse in 3–6 months.
- *Short Duration of Desired Effect* – some patients reported to develop antibodies if given injections within 1-month time period. Best to allow 3 months between injections and less than 400 units. May use botulinum toxin type B as alternative.

Injectable Fillers

- The most commonly used fillers used in the USA today are composed of hyaluronic acid (HA). It is composed of a linear polysaccharide of repeating disaccharide units of glucuronic acid and N-acetylglucosamine.
- HA's mechanism of action is its attraction and its ability to bind over 1000× its weight in water, thus ballooning and increasing volume in atrophied areas.
- The effect of HA fillers can be seen immediately and can last on average for 6–12 months. Longevity due to process of isovolumetric degradation, the degraded filler products draw water as the filler degrades.
- HA fillers are FDA approved for injection into the mid-to-deep dermis (superficial) for correction of moderate to severe facial wrinkles and folds such as nasolabial folds and marionette lines.
- Juvedérm Voluma XC® is approved for deep injection into the cheek to correct age-related volume loss.
- HA fillers can be reversed with the injection of hyaluronidase.

- Calcium hydroxyapatite fillers consists of hydroxyapatite spheres in a soluble gel medium, and because of this composition, it maintains a longer duration of action, averaging 9–18 months; they are useful for treatment of heavier folds (nasolabial), e.g., Radiesse®.
- Poly-L-lactic acid fillers initiate an inflammatory reaction that induces the production of collagen in the injected area; their effect is not immediate. Useful for larger zone contouring in the deep dermal or subcutaneous plane and are FDA approved for the treatment of HIV-associated lipoatrophy (Sculptra®, FDA approved in 2004).

Evaluation of the Filler Patient

- Similar to the neurotoxin patient; see previous section.
- Non-animal stabilized hyaluronic acid products should not be used by people with previous allergies to hyaluronic acid products.

Common Treatment Areas with Dermal Fillers

- Used to treat moderate to severe wrinkles or tissue deficiencies.
- Nasolabial folds (laugh lines, "parentheses," lines between the nose and the corner of the mouth).
- Marionette lines (vertical lines that laterally circumscribe the chin).
- Tear troughs (shallow area underneath eyes; depression of the medial lower eyelid just lateral to the anterior lacrimal crest and limited in its inferior aspect by the inferior orbital rim).
- Nasojugal fold (a shallow groove in the skin that extends downward and lateral from the medial canthus).
- Lips.
- Chin (deepened nasolabial folds, deficient genial projection).
- Cheek volume (malar volume).
- Glabellar region (may be used to augment neurotoxin treatment in this area).
- Pre-jowl area (hollowing of the area on either side of the chin at the jawline).

Treatment Technique of Fillers

- Premark the intended injection sites with surgical pen.
- Choose an anesthetic (topical, local blocks, or none).
- Position the patient in upright position. May allow patients to use a mirror to evaluate effect and allow the patient to take part in the procedure.
- Inject into the subdermal plane in the predetermined marked areas.
- Always aspirate prior to injection to ensure appropriate placement and avoid intravascular deposition.
- Massage the injected areas manually and instruct the patient to do so.
- Apply ice to treatment areas.

Complications

- *Tyndall Effect* – superficial injection of HA, treatment is with 15–50 IU of hyaluronidase.
- *Tissue Necrosis due to Vascular Compromise* – apply 2% nitroglycerin paste immediately, then q5m × 2 hr. Prescribe ASA 325 mg sublingual immediately, then take 1 tab PO daily. Prednisone 24–40 mg for 3–5 days. If hyaluronic acid is used, reversal with hyaluronidase should also be used; should not be used in patients with a history of allergy to bee stings. Warm compresses to the area. Applying a ½ inch strip of 2% nitroglycerin paste to the affected area will also stimulate vasodilation. Consider Hyperbaric oxygen therapy (HBO) if massive necrosis. Keep wounds covered with topical antibiotics.
- *Volume Irregularity* – treat with massage to redistribute filler or add additional filler to repair under filled area. Consider hyaluronidase if hyaluronic acid used. Cross-linked

collagen can be softened with steroid or hyaluronidase injection.
- *Blindness* – highest risk in glabella region. Immediate injection of hyaluronidase into the treated area followed by immediate referral to an ophthalmologist.
- *Herpes Simplex Infection* – reactivation of infection, especially in lip area. Patient with HSV infection should be given prophylactic antiviral therapy (e.g., valacyclovir 500 mg BID) 2 days before treatment.
- *Foreign Body Granuloma* – a chronic inflammatory reaction. Treatment is with hyaluronidase or intralesional corticosteroids.

Filler Pearls

- Observe the needle as it enters the skin; when the bevel can no longer be seen, you have reached the intradermal plane.
- There are two main injection techniques: serial puncture and linear threading.
- Serial puncture: multiple small amounts of filler are sequentially injected into the subdermal plane immediately anterior to the previous injection (i.e., multiple entry points).
- Linear threading: continuous deposition of filler through one subcutaneous entry point.

Rhinoplasty Case Example

A 27-year old male, ASA 1, no medications, No known drug allergies, presents for consultation with a chief complaint:

"My nose is too big (Fig. 6.18)."

- *Describe your preoperative evaluation.*
  - First complete a thorough medical history to include any history of nasal trauma, nasal functional deficiencies, history of prior surgery, seasonal allergies, chronic or acute sinus disease. Psychiatric history including screening for SIMON (single, immature, male, overly expectant, narcissistic).

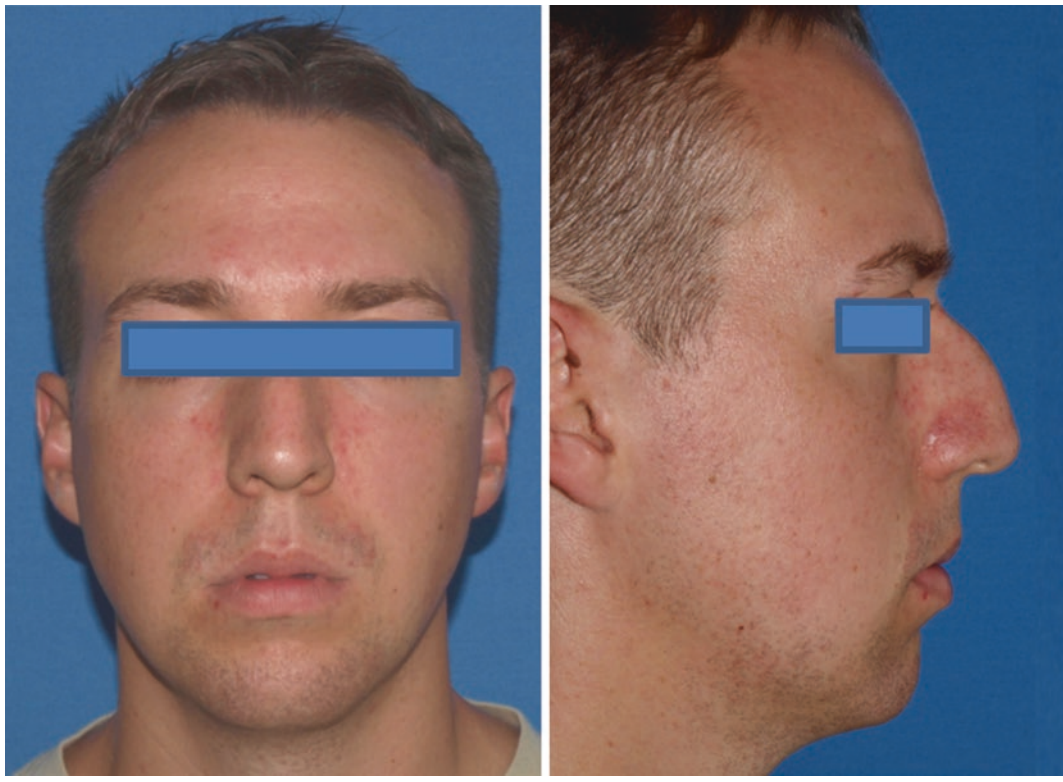

Fig. 6.18 Rhinoplasty case figures. (Images courtesy of Ryan Diepenbrock)

  - Pre-operative photos to include frontal, right and left ¾, right and left profile, and submental vertex. An anterior rhinoscopy should be completed to evaluate the nasal septum and turbinates. A CT scan may be beneficial. Perform a Cottle's test.

- *Describe a Cottle's test.*
  - I would apply upward and lateral traction to the skin of the cheek and ask the patient to sniff. If the sensation of blockage improved, there is likely a nasal obstruction.

- *What comprises the internal nasal valve?*
  - It is made up of the septum medially, the caudal end of the upper lateral cartilage laterally, and the anterior end of the inferior turbinate inferolaterally.

- *What comprises the external nasal valve?*
- It comprises the lower lateral cartilage, nasal septum, and the nasal floor.

- *Describe what you see in the above photos.*
  - The patient has a smooth radix curve with the nasal tip coincident with the midsagittal plane. The alar base is of appropriate width for the patient's race. The nasal dorsum is wide with a slightly asymmetrical gull wing appearance. On the profile view, the patient has a good radix take-off and projection with a significant dorsal hump. The nasal tip is well projected and slightly under-rotated.

- *What procedures could you offer to address his chief complaint?*
- An open rhinoplasty to include dorsal hump reduction, lateral osteotomies, minor tip work, and a columellar strut.

- *After completing the hump reduction, what post-op condition are you most concerned about?*
- An open roof deformity.

- *Describe an open roof deformity.*
  - An open roof deformity is when the dorsum of the nose is over-reduced and the nasal bone, and upper lateral cartilage is visible through the skin. If the internal nasal valve will not be too restricted, osteotomies can be made at the lateral aspect of the nasal bones. The mobile nasal bones then are mobilized medially to close the open-roof. If the internal nasal valve will be restricted, spreader grafts should be placed prior. They should be sutured to the dorsal septum to lateralize the upper lateral cartilages.

- *What is external nasal valve collapse? And what is normally done to fix this issue?*
- It is the collapse of the ring of cartilage around the nostril. This is usually corrected with alar batten grafts.

- *What do you see in* (Fig. 6.19)*?*
  - *Saddle nose deformity*
- *What is a saddle nose deformity?*

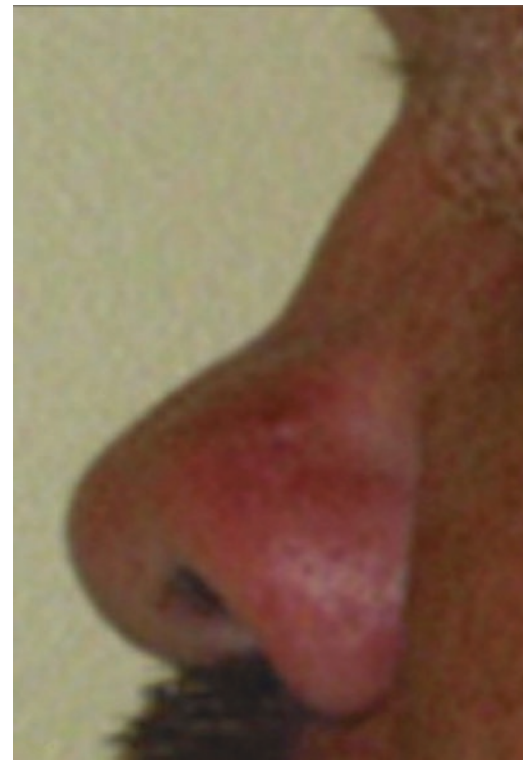

Fig. 6.19 Rhinoplasty complication. (Reprinted with permission from Brenner K and Calvert J. Masters Techniques in Rhinoplasty. Elsevier Books; 2011)

- A loss of nasal dorsal height due to deficiency or collapse of the bony and/or cartilaginous nasal dorsum.

- *How is a saddle nose deformity treated?*
- With dorsal onlay graft.

- *What kind of graft do you use?*
  - I would harvest cartilage from the septum (other sources include rib, ear, and calvarial bone, and synthetics like Medpor® or Gortex® are reserved for cases where there is insufficient autogenous source).

Rhytidectomy Case Example

A 68-year-old female presents to your office with a chief complaint of "I don't like the way I look anymore" (Fig. 6.20).

- *How would you proceed?*
- Try to narrow down the chief complaint to specific problems. Ask the patient what they don't like when they look in a close-up mirror or photo, specifically what bothers them.

- *She states "her tired eyes, her jowls, and her neck." What else would you like to know?*
- Review medical conditions, medications, and allergies. Assess for smoking history and uncontrolled diabetes.

- *She has a past history of smoking. She is on a statin for high cholesterol and takes an H_2 block for acid reflux. What do you do next?*
- Perform a full facial aesthetic evaluation. Evaluate the amount and degree of skin redundancy. Ask the patient if she has previously had facial cosmetic surgery. Take standardized pictures.

- *Describe what you see in the pictures* (Fig. 6.20)*?*
- This is an older Caucasian female.
- She is a Fitzgerald class III and Glogau scale III. Her forehead has deep static rhytids. Her eyebrows appear in good position and form.

Fig. 6.20 Rhytidectomy case figures. (Images courtesy of Anthony Alessi)

She has dermatochalasis of her upper lids. Crow's feet present bilaterally. She has lower eyelid festooning. She has deepening of the nasojugal groove (tear drop deformity). She has descent of the malar fat pads contributing to jowling. She has deepened nasolabial folds. Her nasal dorsum appears midline. Her nasal tip has good definition. Her alar bases are coincident with inner canthi. Her columellar show is appropriate. She has perioral rhytids, marionette lines, and platysmal banding. She lacks definition of the inferior border of the mandible.

- *What procedures do you perform?*
- I would recommend a rhytidectomy with a cervicoplasty procedure to treat her midface, lower face, and neck. To address her eyes, I would recommend upper and lower blepharoplasties.

- *How would you perform a rhytidectomy*?
- I would begin by marking the patient in the preoperative setting to include the planned incision design for the face-lift.
- After the patient is prepped and draped, I would anesthetize the planned incisions with 2% lidocaine with 1:100,000 epinephrine. I would then administer tumescent anesthesia for the planned areas of dissection
- Next, the temporal, endaural incisions would be made with a #15 blade through the skin to the lipocutaneous layer. After elevation of the skin flap, the SMAS is identified *(managed according to the surgeon's preference. If you are not an experienced facelift surgeon, it is recommended that you simply perform a SMAS plication with 3-0 polypropylene sutures or other comparable permanent sutures.)* The SMAS should be pulled in a posterior/superior vector.
- Next, hemostasis is obtained, excess skin is trimmed, and key sutures are placed. The deep sutures are placed to suspend the deep tissue to alleviate tension on the skin, especially below the ear. The skin is then closed.
- A drain may be placed and the head is wrapped.
- The patient is followed up the next day to evaluate for hematomas.
- Sutures are removed at 6–7 days.

- *You have a follow-up on a rhytidectomy on post-op day one. You remove all of the facelift/necklift dressings and you see this. What do you see* (Fig. 6.21)*?*
- Hematoma

- *What do you do?*
- Perform needle decompression with an 18-G needle and syringe. Once the hematoma is evacuated, apply a pressure dressing.

- *When do you see her back?*
- The next day.

- *The next day you find that the hematoma is back. What do you do now?*

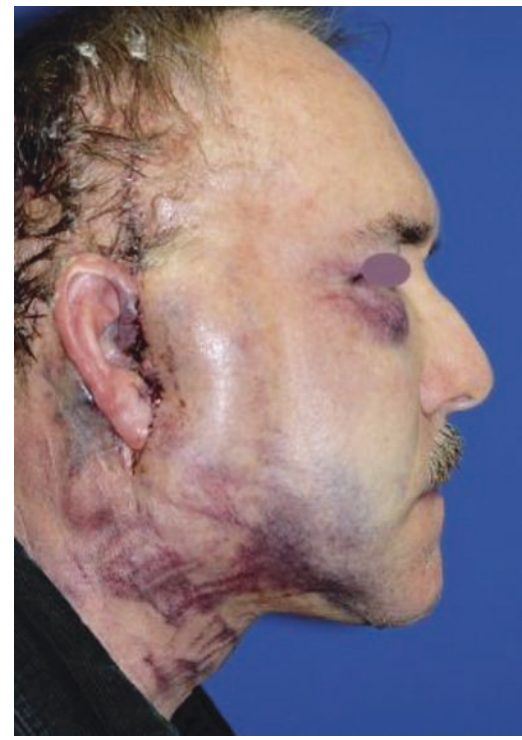

Fig. 6.21 Facelift complication. (Reprinted with permission from. Cleven R. Avoiding Patient Dissatisfaction and Complications in Facelift Surgery. Facial Plastic Clinics of North America. Vol 17 issue 4; 2009)

- Take the patient back to surgery and remove the blood clot. Identify the bleeder and coagulate it.

- *On postoperative day #5, the patient returns for suture removal. You notice on the right side that the skin below the sideburn and above the anterior superior helix looks very dark. The area is the size of a quarter. What do you think is going on and what do you do?*
- The edge of the skin flap is undergoing necrosis due to a lack of blood supply. Apply an antibiotic ointment TID until the area granulates in. You may apply a vasodilator medication such as nitro-paste. You may also consider HBO therapy. After complete healing, assess if further treatment is necessary.

Blepharoplasty Case Example

A 64-year-old female presents with a chief complaint of "My eyelids look heavy and I can't place eye-liner anymore" (Fig. 6.22).

- *How would you evaluate this patient?*
- Review the patient's medical conditions, especially by questioning about any endocrine disease history such as diabetes or thyroid disease. I would ask if there is any prior history of eyelid or globe surgery or history of periorbital trauma. Question if there are recent vision changes or a history of xerophthalmia.

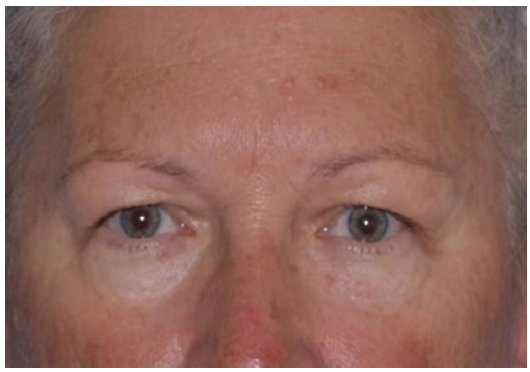

Fig. 6.22 Blepharoplasty case example. (Image courtesy of Dr. Ryan Diepenbrock)

- *What would be included in your physical exam?*
- Position of the lid crease and any asymmetries, brow position, upper lid ptosis, scleral show of the inferior limbus, integrity of the skin, and lower lid supporting structures. Look for pseudoherniation of the fat in the upper and lower lids as well as orbicularis oculi hypertrophy.
- Perform an eye exam in the standard fashion evaluating for pupil reactivity, size, equality, shape, and accommodation. Evaluate the visual fields and assess visual acuity. (Some may endorse a referral to an ophthalmologist for "clearance" and Schirmer's test.)

- *How would you assess the integrity of the lower and upper lids?*
- Distraction test, pull test, snap test, pinch test, and checking for Bell's phenomenon.

- *What photos would you take?*
- Frontal, right and left ¾ view, right and left profile view, periorbital views with eyes open, eyes closed, upward gaze, and inferior gaze.

- *Where is the normal upper lid crease position for a female?*
- 8–10 mm from edge of the eyelid.

- *What is MRD 1?*
- Margin reflex distance. The measurement from the light reflex of the pupil to the lower margin of the eyelid (ciliary line). Normal is 4–5 mm. If less than 3 mm, you must evaluate for lid ptosis.

- *The patient is a good candidate for upper eyelid blepharoplasty. Describe the procedure.*
- The patient consents and is medically cleared for surgery and anesthesia. The procedure may be accomplished with local anesthetic alone, with sedation, or under general anesthesia. The patient is marked in the pre-op region. The markings include the upper lid tarsal crease and the superior portion of the skin to be excised. The marking should leave at a minimum of 20 mm of residual skin from the

ciliary line to the transition of the brow and lid skin.
- The region is anesthetized with local anesthetic with epinephrine.
- Next the skin is excised, followed by muscle.
- With gentle pressure on the globe, prolapsed fat may be contoured or removed via opening the septum and removing fat.
- After judicious hemostasis, the incision is closed with suturing of skin only with 6-0 polypropylene.

- *After the procedure, the patient is found to have 2 mm lagophthalmos in the PACU. Are you concerned?*
- At this point no, instruct the patient to clean the incision and keep it moist. Apply ice for the first 24 hours followed by warm moist heat after 24 hours.

- *The patient returns at 6 days for suture removal and now has 3 mm of lagophthalmos and cannot close with lid without significant effort. What will you do?*
- Remove the sutures. Instruct the patient to begin gentle massage and stretching of the upper lid. Prescribe moisturizing eye drops and follow-up in 1 week.

- *After 2 months of following the patient, there is no improvement in lagophthalmos* (Fig. 6.23). *The patient now complains of xerophthalmia and is quite frustrated with the inability to close the eyelids completely. What is your next course of action?*
- Consider interpositional full-thickness skin grafts or placement of gold or platinum weights. Depending on the level of comfort in managing this complication, you may consider referral to oculoplastic surgeon.

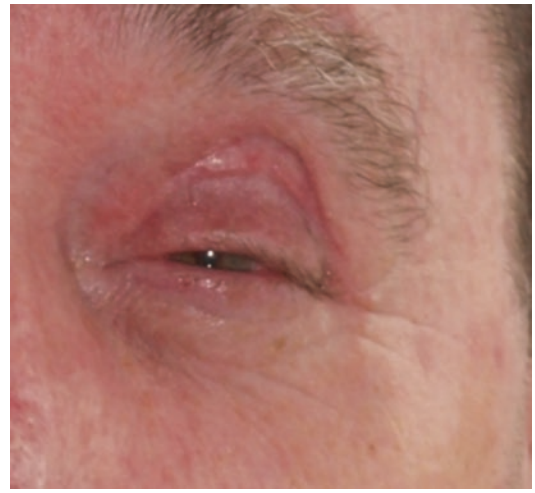

Fig. 6.23 3 mm of lagophthalmos 2 months status post upper lid blepharoplasties secondary to untreated wound dehiscence along the incision line. (Image courtesy of Dr. Ryan Diepenbrock)

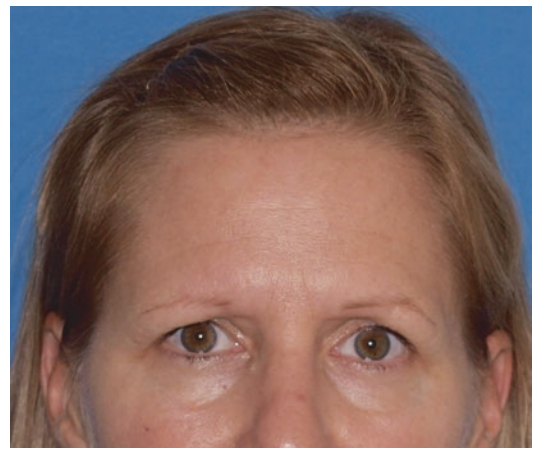

Fig. 6.24 Browlift case example. (Images courtesy of Dr. Ryan Diepenbrock)

Browlift Case Example

A 48-year-old presents for aesthetic consultation (Fig. 6.24). *Her principal complaint is puffiness of the right upper eyelid and drooping of her eyebrows. She has no past medical or surgical history, no history of trauma, takes no medications or supplements, and has no allergies.*

- *How would you determine if a blepharoplasty or a brow lift is indicated?*
- With the eyes closed and frontalis relaxed, a hand is used to stabilize the frontalis with the eyebrow in appropriate position. If dermatochalasis is no longer apparent, a brow lift without concurrent blepharoplasty is recommended.

- *Which are your options for surgical correction of brow ptosis?*
- Direct brow lift, pretrichial brow lift, trichophytic brow lift, coronal brow lift, or endoscopic brow lift.

- *What are some disadvantages of the direct brow lifting technique in this patient?*
- Medial brow ptosis cannot be corrected by direct brow lifting alone, thin eyebrows limit the ability to camouflage the incision.

- *What are the major disadvantages of coronal forehead lifting?*
- Alopecia and forehead lengthening.

- *To what depth is the dissection performed during trichophytic brow lifts?*
- Centrally, the dissection can be performed in the subperiosteal, pre-periosteal, or subcutaneous planes. Temporally, the dissection is performed to the level of deep temporal fascia.

- *What is the course of temporal branch of the facial nerve?*
- The facial nerve courses superiorly and medially from 0.5 cm below the tragus to 1.5 cm above the lateral eyebrow in the temporoparietal fascia just deep to the SMAS.

- *What other neurovascular bundles must be considered? Where are they located?*
- The supratrochlear and supraorbital neurovascular bundles are located 1.7 cm and 2.7 cm from a midline mark and courses along the anterior aspect of the frontalis.

- *After undergoing a forehead lift, the patient has mild lagophthalmos (1.5 mm) the first postoperative day. How would you manage this? What concurrent procedure increases the risk of postoperative lagophthalmos?*
- Topical ophthalmic lubricating drops and lubricating ointment at night is sufficient for mild dryness and lagophthalmos. Concurrent blepharoplasty or ptosis repair increases postoperative lagophthalmos.

- *While discussing the risks, benefits, and alternatives of browlifting, the patient asks if both sides will look exactly the same. How would you address this?*
- Postoperative asymmetry is a risk of brow/forehead lifting, especially if preoperative asymmetry exists. This should be discussed with the patient and documented before informed consent is obtained. The patient's preoperative exam and photographs should be reviewed with the patient to demonstrate the existing asymmetries.

- *After undergoing a trichophytic forehead lift, the patient develops severe forehead pain, swelling, and profuse bleeding from the incisions the night of surgery. What is the most likely diagnosis and appropriate course of action?*
- Hematoma formation and hemorrhage from the supraorbital or supratrochlear artery. Opening the surgical incision and obtaining hemostasis are indicated.

- *Your patient states that she is very difficult to "numb" and requests full-strength local anesthetic for the entire forehead instead of tumescent dilute anesthesia. What is the major risk of this and symptoms?*
- Lidocaine toxicity. Major symptoms are myocardial depression, peripheral vasodilation, bradycardia, hypotension, drowsiness, tremors, paresthesias, and convulsions.

- *Why is the forehead length important in evaluation for endoscopic forehead lifting?*
- An endoscopic forehead lift raises the forehead. Patients with long foreheads are not good *candidates for this procedure.*

- *What is the average thickness of the bony skull in the parasagittal region?*
- 7.5 mm (varies 4–11 mm). This is a safe region to place a bone screw, as it is free of venous lakes. The midline region has a thickness of 7 mm but has multiple midline lakes.

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Maxillofacial Trauma

7

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Basic Trauma Principles

Hypovolemia (Table 7.1)

- Can be caused by bleeding or massive urinary or gastrointestinal fluid losses.
- One or two large bore (≥16 gauge) peripheral IVs should be inserted.
- Focused abdominal sonography for trauma (FAST) in a hypotensive patient to rule out intra-abdominal hemorrhage or cardiac tamponade.

Glasgow Coma Scale (Table 7.2)

- Objective measure of patient's neurological status and used serially to track clinical progress.
- Can be applied to patients 5 years of age and above.
- Use best response (left vs. right differential).
- Score 8 or less, early airway protection encouraged due to concern of respiratory arrest or hypoxia leading to secondary brain injury.
- Score minimum is 3.
- Requires serial exams and a CT scan of the head.

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Table 7.1 Classes of Hemorrhagic Shock. Based on a 70 kg Person

Classes of Hemorrhagic Shock				
	1	2	3	4
Blood loss (ml)	<750	750–1500	1500–2000	>2000
Pulse rate (BPM)	<100	100–120	120–140	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Respiratory rate (per minute)	12–20	20–30	30–40	>40
Urine output ml/hr	>30	20–30	5–15	Scarce
Mental status	Slightly anxious	Mildly anxious	Confused	Lethargic

Table 7.2 Glasgow Coma Scale. Mnemonic to remember points allotted for each GCS component "4 Eyes, Jackson 5 (voice) and V6 (motor)"

Eye opening response	Verbal response	Motor response	Points
No response	No response	No response	1
To pain only	Incomprehensible	Extension in response (decerebrate posturing)	2
To verbal stimuli, command, speech	Inappropriate words	Flexion in response to pain (decorticate posturing)	3
Spontaneous–opening with blinking	Confused conversation, but able to answer	Withdraws in response to pain	4
	Oriented	Purposeful movement to pain	5
		Obeys commands for movement	6

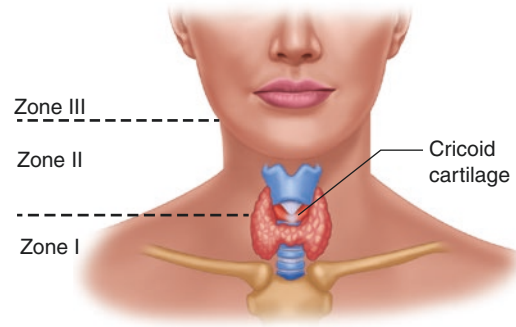

Fig. 7.1 Zones of the neck. (Reprinted with Permission from Georgopoulos C. Chap. 86 – Neck Trauma. Sixth Edition. Emergency Medicine Secrets. Elsevier Inc.; 2016)

Head Injury Classification

- Severe Head Injury/Coma – GCS score of 8 or less.
- Moderate Head Injury – GCS score of 9–12.
- Mild Head Injury – GCS score of 13–15.
- Denotation of T after the score is applied to an intubated patient.

Zones of the Neck for Penetrating Trauma (Fig. 7.1)

- Zone 1 – thoracic inlet to cricoid cartilage.
- Zone 2 – cricoid cartilage to angle of the mandible.
- Zone 3 – angle of the mandible to the base of the skull.
- Zones 1 and 3 are usually worked up using conventional or, more commonly now, CT angiography due to difficulty in access.
- Due to the multiple organ systems represented in the neck, multiple systems can be impacted by a single penetrating insult. Insults may result in cervical spine, carotid, esophageal, and/or laryngeal injuries.
- Patients who are unstable or present with hard signs such as bruits, thrills, large/pulsatile hematomas require immediate exploration.

Principles of Fixation

Rigid Fixation:
- Fixation that prevents interfragmentary movement when a load is applied.

Semi-Rigid Fixation:
- A form of fixation that is not of sufficient strength to prevent interfragmentary movement during loading but is adequate to allow union of bone.

Load Bearing:
- Hardware of sufficient strength that is able to bear the entire load.
- Plates and screws to immobilize the fractured segments.
- Use of thicker, rigid plates with bicortical screws to immobilize the fracture segments or lag screws.
- Requires at least three screws on each segment when plates are used.

Load Sharing:
- Form of hardware that is unable to bear all functional load across fracture.
- Open reduction, internal fixation without relying solely on the plates and screws to immobilize the fracture segments.
- Uses miniplates and monocortical screws along the lines of osteosynthesis as described by Champy.

Ideal Line of Osteosynthesis of the Mandible:
- Described by Maxime Champy in 1976 [1].
- A line around the mandible where plating the tension and compression forces are balanced, thus offering the best biomechanical advantage for positioning of plates and screws.

Plates and Screws for Internal Fixation

- Non-locking plates/screws:
  - Plates must be adapted intimately to the bone.
  - Compression of the plate onto the bone may cause bone resorption under the plate.
- Locking plates/screws:
  - Screws lock into the plate while it is being tightened.
  - Does not require a perfect adaptation of the plate to the bone.
  - The plate bears the load of mechanical forces.
- Plate and screw dimensions:
  - Most plates are composed of titanium.
  - Plating systems are named by the outer diameter of the screws.
- Thread shape:
  - Self-drilling – insertion without pre-drilling or tapping.
  - Self-tapping – requires pre-drilling of a pilot hole, the insertion of the screw will create its own thread in the bone.

Bone Healing

- Bone healing is altered by types of fixation and mobility of the fracture site in relation to function.
- Primary bone healing:
  - No fracture callus forms.
  - Heals by a process of:
    (a) Haversian remodeling directly across the fracture site if no gap exists (contact healing)
    (b) Deposition of lamellar bone if small gaps exist (gap healing).
  - Requires absolute rigid fixation with minimal gaps.
- Secondary bone healing:
  - Bony callus forms across fracture site to aid in stability and immobilization.
  - Occurs when there is mobility around the fracture site.
  - Secondary bone healing involves the formation of a subperiosteal hematoma, granulation tissue, and then a thin layer of bone forms by membranous ossification. Hyaline cartilage is deposited, replaced by woven bone and remodels into mature lamellar bone.

Approach to the Facial Trauma Patient

History of Present Illness

- Mechanism of injury and loss of consciousness are important points to note. It is difficult to assess a history from an unstable trauma patient and, therefore, witnesses should be questioned.
- Confirm ATLS/PALS has been performed. Threshold for intubation should be low. Ensure appropriate consultations have been made, e.g., neurosurgery, ophthalmology, orthopedic surgery, and pediatric surgery, etc., if necessary.
- Ensure C-spine has been evaluated and appropriate precautions taken. It's desirable to have the head and spine cleared prior to surgical intervention if possible.

Physical Exam

- After confirming the patient is stable and the airway secure, begin the head and neck trauma exam. This should be systematic and concise.
- Evaluate general demeanor and responsiveness. Patient cooperation may be extremely difficult with pediatric or inebriated patients.
- Evaluate neurological status to determine the level of consciousness (Glasgow scale).
- Evaluate for facial asymmetry, lacerations (rule out Stenson duct or facial nerve injury), edema and ecchymosis.
- Examine the cranial nerves II–XII and note any paresthesia (V1, V2, V3), or facial nerve deficits.
- If the orbit is involved, evaluate extra ocular movements, pupillary reaction, direct and consensual visual reflexes, monocular (indicative of retinal detachment) or binocular diplopia (can be secondary to edema or entrapment and restriction of gaze). Tonometry should be used for evaluating intraocular pressure. A fundoscopic exam is indicated for for evaluating the retina and optic nerve, and hyphema. A slit lamp exam is useful in evaluating the eyelids, lacrimal system, cornea and to rule out the presence of foreign bodies. Exam should assess for the presence of proptosis, dystopia (disturbance in globe position in the vertical and horizontal planes) or enophthalmos. Look for periorbital ecchymosis (Racoon eyes). Evaluate for telecanthus. Consider ophthalmology exam/clearance.
- Evaluate the ears for the presence of ecchymosis behind the ears (Battle's sign) or otorrhea, which may be indicative of a base of skull fracture (if positive neurosurgical consultation is required). Rinne and Weber exam to screen for hearing. Otoscopic exam to evaluate tympanic membrane and EAC (if injury apparent ENT should be consulted).
- Evaluate for exit wounds if a projectile was involved.
- Evaluate the midface for loss of projection, edema, ecchymosis, and step deformities.
- Evaluate the nose for asymmetries, blood, rhinorrhea, and septal hematoma.
- Evaluate jaws for range deviations on opening (this may indicate a condylar or subcondylar fracture), arch step deformities, lingual ecchymosis (highly indicative of a mandible fracture), hematomas, and intraoral lacerations.
- Evaluate the state of the dentition (dental fractures, missing teeth, changes in occlusion). For a pediatric patient correlate dental development with chronological age.
- Evaluate for a chin laceration, preauricular edema, or ecchymosis as these can be suggestive of a condylar fracture.
- The floor of mouth swelling or the possibility of airway compromise should be noted.

Imaging

- High-resolution maxillofacial computed tomography (CT) is the gold standard for evaluating facial trauma. Consider obtaining 3D reconstruction for surgical treatment planning and using it as an aid to discuss treatment with the family of the patient.
- If trauma is isolated to the mandible or dentition an orthopantomogram can be used.

- Plain film X-rays have limited value in the maxillofacial trauma patient.
- If teeth are missing or unaccounted for secondary to the trauma then an abdominal X-ray (KUB) and chest X-ray must be performed.

Mandibular Trauma

Nomenclature

- Fracture types:
  - Simple/closed – not opened to the external environment.
  - Compound/opened – fracture extends into an external environment.
  - Comminuted – splintered or crushed.
  - Greenstick – only one cortex fractured.
  - Pathologic – pre-existing disease of bone leads to fracture.
- Muscle Action Classification:
  - Vertically favorable vs. non-favorable, based on resistance to medial pull.
  - Horizontally favorable vs. non-favorable, based on resistance to upward movement.

Physical Exam

- Tenderness over the region of suspected fractures.
- Malocclusion:
  - Anterior open bite – bilateral condylar or angle fractures.
  - Unilateral open bite – ipsilateral angle, condylar and parasymphyseal fractures.
  - Posterior crossbite – symphyseal and condylar fractures with splaying of the posterior segments.
  - Prognathic bite – TMJ effusions.
  - Retrognathic bite – condylar or angle fractures.
- Loss of form – bony contour change, soft tissue depressions, deformities.
- Loss of function – can be from guarding, pain, or trismus.
  - Deviation on opening toward the side of the condylar fracture.
  - Inability to open due to impingement of coronoid or ramus on the zygomatic arch.
  - Premature contacts from alveolar, angle, ramus, or symphysis fractures.
- Edema.
- Abrasions/lacerations – the potential for compound fractures.
- Ecchymosis – especially floor of the mouth, symphyseal, or body fracture.
- Crepitus with manipulation.
- Altered sensation/paresthesia.
- Loss of teeth – require chest X-ray to rule out aspiration if not accounted.

Radiographic Evaluation

Panoramic Radiograph:
- Most informative radiographic tool.
- Shows entire mandible and direction of fracture (horizontal favorable, unfavorable).
- Disadvantages:
  - Patient must sit up-right/cooperative/non-sedated or intubated.
  - Difficult to determine buccal/lingual bone and medial condylar displacement.
  - Some details are lost/blurred in the symphysis, TMJ and dentoalveolar regions.

Mandible Series:
- Towne's view, anteroposterior and both oblique views.

Computed Tomography (CT):
- Excellent for showing intracapsular condyle fractures.
- Can get axial, sagittal, and coronal views; 3-D reconstructions.
- Disadvantage:
  - Expensive.
  - A larger dose of radiation exposure compared to plain film.
  - Difficult to evaluate the direction of fracture from individual slices (reformatting to 3-D overcomes this).

Treatment of Mandible Fractures

Closed Reduction (Table 7.3):
- Contraindications:
  - Medical conditions that should avoid intermaxillary fixation.
  - Alcoholics.
  - Seizure disorders.
  - Mental retardation.
  - Nutritional concerns.
  - Respiratory diseases (COPD).
  - Unfavorable fractures.

Techniques for Closed Reduction:
- Erich arch bars.
- Ivy loops.
- Essig Wire.
- Intermaxillary fixation screws.
- Splints.
- Bridal wires.

Length of Intermaxillary Fixation:
- Based on multiple factors:
  - Type and pattern of fracture.
  - Age of patient.
  - Involvement of intracapsular fractures.
- Average adult – 3-4 weeks.

Table 7.3 Advantages and disadvantages of closed reduction

Advantages	Disadvantages
Low cost	No absolute stability (secondary bone healing)
Short procedure time	Oral hygiene difficulty
Can be done in a clinical setting with local anesthesia or sedation	Possible TMJ sequelae
Easy procedure	Muscular atrophy/ stiffness
No foreign body in patients	Myofibrosis
	Possible effect on TMJ cartilage
	Decreased range of motion
	High degree of compliance required
	Weakness of muscles due to disuse
	Osteoporotic changes due to disuse

- Children 15 years or younger – 2-3 weeks.
- Elderly patients – 6-8 weeks.
- Condylar fractures – 2-4 weeks.

Open Reduction

- Implies accessing the fracture through skin or mucosa to aid in visualization and reduction of the fracture.
- Indications:
  - Unfavorable/unstable mandibular fractures.
  - Patients with multiple facial fractures that require a stable mandible for basing reconstruction.
  - Fractures of an edentulous mandible fracture with severe displacement.
  - Edentulous maxillary arch with opposing mandible fracture.
  - Delayed treatment with interposition of soft tissue that prevents closed reduction techniques to re-approximate the fragments.
  - Medically compromised patients.
  - Gastrointestinal diseases.
  - Seizure disorders.
  - Compromised pulmonary health.
  - Mental retardation.
  - Nutritional disturbances.
  - Substance abuse patients.

Contraindications for Open Reduction
- If a simpler method of repair is available, maybe better to proceed with those options.
- Severely comminuted fractures.
- Patients with healing problems (radiation, chronic steroid use, transplant patients).
- Mandible fractures that are grossly infected.

Edentulous Mandible Fractures

- Biomechanics differ for edentulous fractures compared dentate mandible fractures:
  - Decreased bone height leads to a decreased buttressing effect (alters plate selection).

  - Significant bony resorption in the body region.
  - Significant effect of muscular pull, especially the digastric muscles.
  - Incidence of fractures highest in the body.
  - Atrophy creates saddle defects in the body.
- Biological differences:
  - Decreased inferior alveolar artery (centrifugal) blood flow.
  - Dependent on periosteal (centripetal) blood flow.
  - Medical conditions that delay healing.
  - Decreased ability to heal with age.
- Closed Reduction.
  - Use of circummandibular wires fixated to the piriform rims and circumzygomatic wires with patient's denture or Gunning style splints.
  - Requires IMF – usually longer periods due to age.
- Open Reduction Techniques:
  - Treat mandible >20 mm as dentate mandible.
  - Requires load bearing type plates.
  - Due to poor healing quality of bone and reduced osteoprogenitor cells, bone grafts are commonly incorporated to transplant osteocompetent cells and augment mandible.
  - Reduction may be aided by the adaptation of miniplates at the inferior border.

Condylar Process Fractures

- Usually unilateral, and from indirect trauma from the opposite side of insult.
- Will have ipsilateral premature closure and midline pull on side of the fracture.

Classifications:

- Wassmund Scheme [2]:
  - I – minimal displacement of the head (10–45°).
  - II – fracture with tearing of medial joint capsule (45–90°), bone still contacting.
  - III – bone fragments not contacting, condylar head outside of capsule medially and anteriorly displaced.
  - IV – head is anterior to the articular eminence.
  - V – vertical or oblique fractures through the condylar head.
- Condylar fractures (AO classification – see Fig. 7.2):
- Goals of condylar fracture repair:
  - Pain-free mouth opening with an opening of 40 mm or greater.
  - Good mandibular motion of jaw on all excursions.
  - Restoration of pre-injury occlusion.
  - Stable TMJs.
  - Good facial and jaw symmetry.

Treatment Options for Condylar Fractures

- Non-surgical – diet, observation, and physical therapy.
- Closed Reduction:
  - Treated with a short course of IMF with post-operative physical therapy.
- Open Reduction [3]:
  - Zide's Absolute Indications:
    (1) Middle cranial fossa involvement with disability.
    (2) Inability to achieve occlusion with closed reduction.
    (3) Invasion of joint space by a foreign body.
    (4) Lateral capsule violation and displacement.
  - Zide's Relative Indications:
    (1) Bilateral condylar fractures where the vertical facial height needs to be restored.
    (2) Associated injuries that dictate early or immediate function.
    (3) Medical conditions that indicate open procedures.
    (4) Delayed treatment with misalignment of segments.

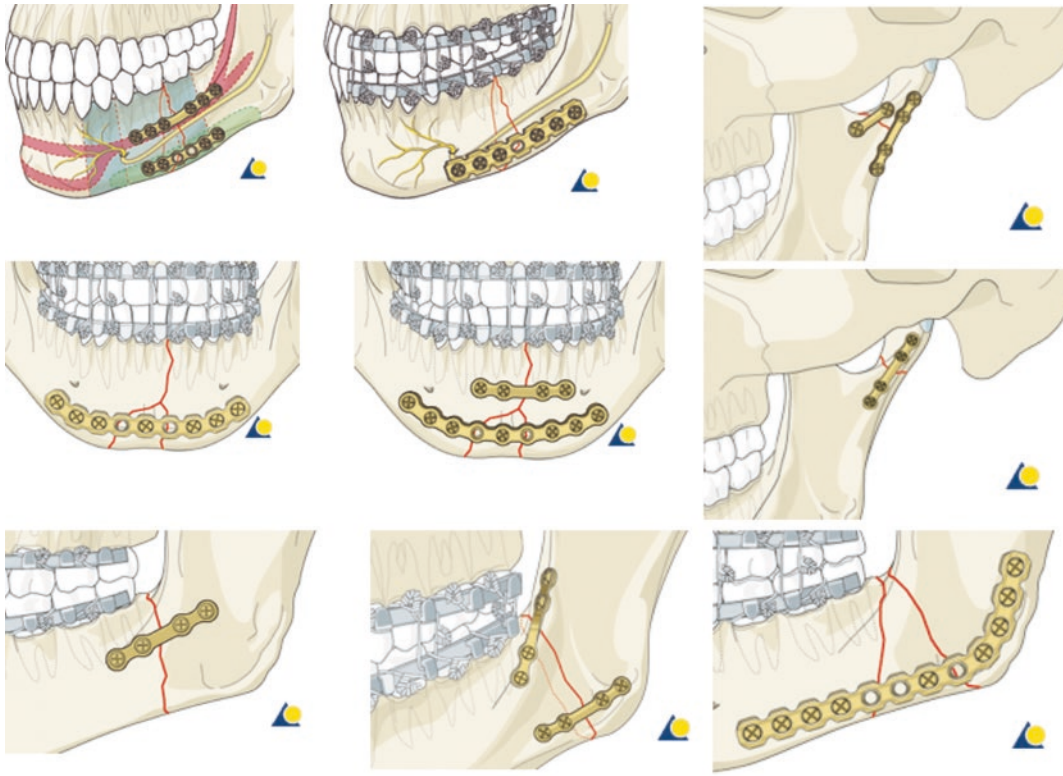

Fig. 7.2 AO Classification of Condyle Fractures. The condylar neck region can be divided into high and low halves by equally dividing the distance between the sigmoid notch line and the lateral pole line. (Copyrighted by AO Foundation, Switzerland)

- Closed reduction techniques rarely produce pain, limit function, or produce growth disturbances.
- Open reductions techniques show an early return to normal function, but are technique sensitive, time extensive, and can lead to facial nerve dysfunction depending upon surgical approach.

Teeth in the Line of Fracture

Generally accepted tooth to be removed if:

- Gross mobility.
- Periapical pathology.
- Preventing reduction.
- Roots with a fracture.
- Exposed root.
- Delay in repair from time of fracture.
- Recurrent infection at the fracture site despite antibiotic therapy.

Complications

Malunion – Fracture that has healed in non-anatomic alignment. More common when complex fractured with multiple segments. May also be due to occlusion that is forced into position, loose IMF, inadequate reduction, or poor adaptation of a fixation plate. If there are minor dental discrepancies, orthodontic or occlusal adjustments can be used as treatment. Early recognition may allow for breaking down hardware and proper alignment. Late identification can mean osteotomies at the fracture sites or orthognathic

concepts with surgical stents to reestablish occlusal and facial qualities.

Non-union – Arrested healing after the appropriate time has passed (mobility after 4 weeks without treatment and 8 weeks with surgical management). Can be multifactorial but includes mobility at the fracture site, poor reduction, infection, substance abuse, delay in treatment, or tooth in line of fracture. Diagnosis is characterized by persistent pain, mottled bone at the fracture site, mobility of mandible, and sign of hardware loosening/failure. Treatment occurs after infections are controlled and oral/cutaneous communications have closed. Establish occlusion and a rigid fixation plate is placed. Screw placement is recommended to be 1 cm from borders of segments as some bone is thought to be non-vital. The bone edges are smoothed, and commonly a cancellous bone graft is used to reconstruct the continuity defect if needed.

Osteomyelitis – Complaint of continued pain, paresthesia, feeling of the mobility of plate. Diagnosis can be made with labeled white blood cell scans (indium-111), bone scans (technetium 99), MRI, or biopsy of bone. Treatment can involve removal of hardware with closed reduction, resection/debridement/cortication of bone, placement of rigid fixation, IV antibiotics, and consideration of hyperbaric oxygen therapy.

LeFort Fractures

- Transfacial fracture of the midface, involving the maxillary bone and surrounding structures in either a horizontal, pyramidal, or transverse direction. It involves the pterygomaxillary suture and the nasal septum.
- Classified according to the experiments by anatomist Rene LeFort.

Classifications (Fig. 7.3)

LeFort I (Horizontal Fracture) – extends above the apices of the maxillary dentition across the nasal septum and maxillary sinuses. Posteriorly it extends through the pyramidal process of the palatine bone and the pterygoid processes of the sphenoid bone. It also may involve the fracture of the palate.

LeFort II (Pyramidal Fracture) – extends from the nasofrontal region down through the medial orbital wall, crossing the inferior orbital rim and zygomatic buttresses. Posteriorly similar to a LeFort I fracture.

LeFort III (Complete Craniofacial Disjunction) – fracture lines extend through the nasofrontal junctions, zygomaticofrontal articulations, zygomaticomaxillary suture, temporozygomatic suture, pterygomaxillary junction, medial and lateral orbital walls, and superior articulation of the nasal septum.

Rarely is there a pure category of fracture; usually there's a mixed combination.

Physical Exam

- Generally, look for increased facial height (equine facies), loss of anterior projection (dishpan facies). Look for edema, lacerations, contusions, hematoma formation, and ecchymosis.
- Ocular – pupils, extraocular muscle function, visual acuity, ocular pressure, subconjunctival hemorrhage, infraorbital nerve sensation, and intercanthal distance.
- Battle Sign – ecchymosis in the mastoid region, suggestive of a base of skull fracture. Can be seen in midfacial fractures due to high-energy injuries.
- Palpate – look for tenderness, crepitus, bony step deformities, mobility of segments at zygomaticofrontal and nasofrontal sutures, or maxillary mobility.
- Nasal – rhinorrhea, septal hematoma, epistaxis, mobility of nasofrontal suture.
- Intraoral exam – evaluate dentition, relative class III malocclusion, vestibular ecchymosis, tooth loss/fracture, and occlusion and anterior open bite.

Radiographic Evaluation

Maxillofacial CT with axial, coronal, and sagittal cuts. There are four key areas to evaluate.

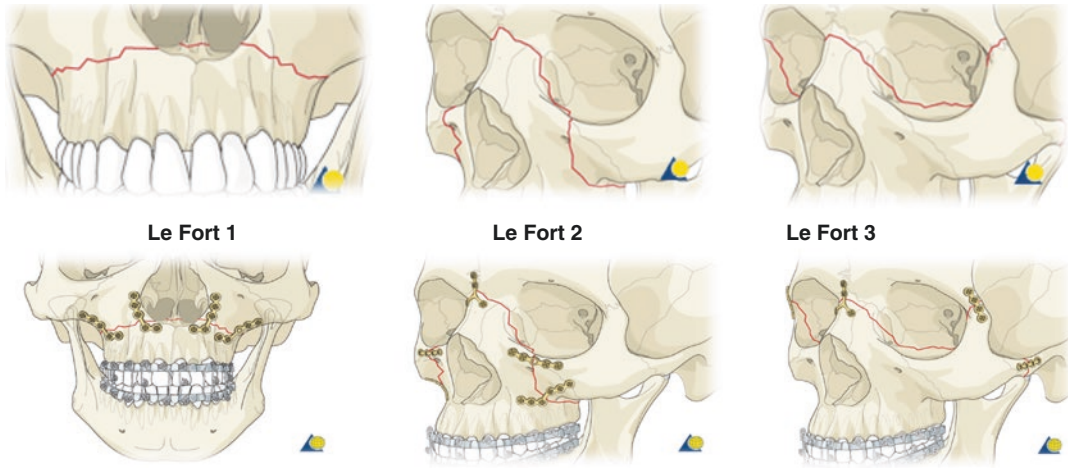

Fig. 7.3 LeFort fracture patterns and recommended plating schemes. (Copyrighted by AO Foundation, Switzerland)

- Pterygoid plates, strong indication of LeFort fracture.
- Lateral margin of nasal fossa, suggestive of LeFort I fracture.
- Inferior orbital rim, suggestive of LeFort II.
- Zygomatic arch, suggestive of LeFort III.

Principles of LeFort Fracture Management

- Nondisplaced fractures without clinical compromise can be managed by a soft diet with observation or 4–6 weeks of IMF.
- Edentulous patients may be treated with open treatment or observation.
- Treat as soon as possible. The longer open or compound fractures are untreated, the greater incidence of infection and malunion.
- Fixate fractures to allow immobilization and optimal healing.
- Use buttresses for fixation.
- Restore preoperative occlusion.
- Ensure to treat nasal complex and orbital fractures as indicated.

Approaches to LeFort I

- Access is via the transoral approach.
- Place into IMF (consider using disimpaction techniques if necessary to aid in the alignment of fractures and restoration of occlusion).
- Reduce fractures and plate stabilization at piriform rims and zygomaticomaxillary buttresses.
- Check occlusion.

Approaches to LeFort II

- Access is via transoral and/or periorbital/coronal approach.
- Place into IMF.
- Reduce fractures and plate stabilization at the piriform rim, zygomaticomaxillary buttresses, orbital rim, and management of NOE component if necessary.
- The orbital floor should be treated last after ensuring that the zygoma and the maxilla are in the proper position to prevent increased orbital volume.
- Check occlusion.
- Nasal repairs as needed.

Approaches to LeFort III

- Access is usually via a combination of the transoral, lower lid, coronal approaches.
- Good mobilization.
- Place into IMF.
- Reduce fractures ensuring restoration of adequate facial height and width.
- Fixate starting at the frontozygomatic suture, nasal region, zygomaticomaxillary but-

tresses, piriform rims, and zygomatic arch as needed.
- Check occlusion.
- Reduce/reconstruct orbit.
- Nasal reduction as needed.

Description of the Maxillary Vestibular Approach

- Length of the incision depends on the area of interest or extent of injury. Can be unilateral or bilateral.
- Incision is placed ~3–5 mm superior to the mucogingival junction making sure to leave adequate unattached gingiva for closure. (Usually extends posteriorly to first molar.)
- Incise through mucosa, submucosa, and periosteum down to the bone using electrocautery Bovie or #15 blade.
- Subperiosteal dissection superiorly and anteriorly to piriform buttress and posterior to zygomaticomaxillary buttress.
- May encounter infraorbital canal and neurovascular bundle if dissection extends enough superiorly.
- If necessary, may need to dissect nasal mucosa from the lateral wall, floor, or septum using a Freer elevator.

Complications

- *Malocclusion Noted After IMF Is Released* – Occlusion may spring open after the IMF is released. Remove fixation and then place patient back in IMF and make sure occlusion is stable and condyles are in the fossa before fixating fractures again; remove from IMF and recheck occlusion.
- *Malocclusion Noted 1 Week Postoperatively* – Most likely loss of fixation. Obtain new imaging to confirm and if fixation failed then return to OR to correct. If malocclusion is minor and fixation appears intact on imaging, then allow full healing of fractures and refer for orthodontic correction of the minor malocclusion.
- *Bleeding During Mobilization of the Maxilla* – If from the pterygoid muscles at posterior maxilla, then treat with surgicel or fibrin sealants. If from the pterygoid plexus, then treat with local anesthesia and packing. If from the terminal branches of the maxillary artery (descending palatine, PSA, or sphenopalatine arteries) treat with vessel clips and/or anterior and posterior nasal packs. If local hemostatic measures are used and bleeding is still not controlled, then consider embolization with interventional radiology. Late bleeding may occur due to pseudoaneurysm formation.
- *Malunion* – May require osteotomies or onlay grafting to address the resulting anatomical anomaly.
- *Nonunion* – Continual mobility noted after 8 weeks after the fixation has been placed. May require removal of faulty fixation, bone grafting, and placement of rigid internal fixation.

Zygomaticomaxillary Complex (ZMC) Fractures

- The zygoma has four articulations (frontozygomatic, zygomaticomaxillary, zygomaticosphenoid, and zygomaticotemporal). These sutures represent common areas of fracture points.
- It should be noted that the weakest portion of the zygomatic arch is not the zygomaticotemporal suture, but a point approximately 1.5 cm posterior to this suture.

Classification of ZMC Fractures

- The most commonly quoted is Knight and North, based on the direction of displacement on a Water's view radiograph.
  - Group 1 – nondisplaced.
  - Group 2 – arch fractures.
  - Group 3 – unrotated.

  - Group 4 – medially rotated.
  - Group 5 – lateral rotation outward.
  - Group 6 – complex fractures.
- Zingg Classification based on review of CT scans.
  - Type A fractures are incomplete zygomaticomaxillary complex and broken into three subcategories:
    - A1 – Isolated arch fracture.
    - A2 – Isolated lateral wall.
    - A3 – Isolated inferior orbital rim.
  - Type B – monofragment with all four buttresses.
  - Type C – comminution of zygomatic bone.

Physical Exam

- ZMC fractures are also orbital fractures. An ocular exam is imperative including visual acuity, assessment of extraocular muscles in the six cardinal fields of gaze, integrity of rim, ecchymosis, hyphema, shape of pupil (traumatic mydriasis or iridodialysis), reactivity of pupil, size of pupil, subconjunctival ecchymosis, periorbital edema/ecchymosis, and chemosis and position of the globe. Deepening of the supratarsal crease is one of the earliest signs of enophthalmos.
- Flattening of the malar eminence. Decreased projection is best assessed from a bird's eye view.
- Depression in the preauricular region denoting flattening of the zygomatic arch.
- Antimongoloid slanting (due to disruption of the frontozygomatic suture and inferior displacement of Whitnall's tubercle).
- Neurological disturbances over the distribution of the infraorbital nerve.
- Step deformities denoting discontinuity of the orbital rim, zygomaticomaxillary buttress, and frontozygomatic region.
- Ecchymosis in the maxillary vestibule (Guerin's sign).
- Trismus if coronoid is impinged and possibly spasm of masseter.
- Pupillary level – fracture of orbital floor allows for displacement of suspensory ligaments and Tenon's capsule causing hypoglobus of the affected side.

Radiographic Evaluation

- CT is the gold standard for evaluation of ZMC fractures. It allows for the visualization of all buttresses and to assess the degree of displacement and/or comminution. It also allows for assessing the orbital floor, muscle entrapment, and the integrity of the globe.
- CT scans obviate the need for plain radiographs.
- Plain radiographs that were used in the past for assessing ZMC fractures were the Caldwell and Submentovertex views (submentovertex views are still used intraoperatively to assess adequate reduction of the zygomatic arch).

Management of Zygomatic Fractures

Zygomatic Arch Fractures

- Isolated zygomatic arch fractures can be approached via a Keen or Gillies approach.
- Some surgeons wire/suture a finger splint or Fox shield over the arch to maintain reduction while healing.
- Closed reduction has also been described using a towel clip to aid reduction.
- ORIF of zygomatic arch fractures. Not performed for isolated arch fractures. Usually stabilized with miniplates as part of a high-impact ZMC fracture or a panfacial fracture.

ZMC Fractures

- It is important to employ a systematic sequence when treating ZMC fractures. Multiple approaches are necessary to expose

the frontozygomatic, zygomaticomaxillary, orbital rim, and orbital floor regions. Minimum two points of fixation are required.
- Recommended sequencing of fixation:
  - Fixate the frontozygomatic region first to restore facial height of the complex.
  - Fixate the zygomaticomaxillary buttress region to restore facial projection and to ensure that the medially rotated body is back in its normal anatomical position.
  - Fixate the orbital rim to define orbital volume and facial volume.
  - The orbital floor should be managed last as it is critical that the aforementioned sites are placed back into alignment to prevent enophthalmos and facial widening.
- Alignment of the sphenozygomatic suture is a good indicator of the three-dimensional position of the zygoma.

Complications

Malunion/Asymmetry – May result in facial widening and/or malar flattening. Can be managed by osteotomies (difficult due to lack of bony landmarks), onlay grafts, alloplastic implants, or a combination of the aforementioned. Consider navigational instrumentation when using custom implants.

Enophthalmos – Due to increase in orbital volume or atrophy of fat. Posttraumatic enophthalmos is difficult to manage. Requires placement of space-occupying material such as bone or prosthetic material behind the globe to displace it anterior. Consider custom implant.

Blindness – Rare but devastating either by direct trauma to globe or retrobulbar hematoma. In retrobulbar hematoma, the patient will have pain, proptosis, elevated intraocular pressure, and decrease in visual perception (first decrease in red-green color perception followed by decreased visual acuity).

Retrobulbar Hemorrhage – Managed by a lateral canthotomy or by reopening the surgical wound used for periorbital access to allow for decompression.

Vertical Dystopia – Reconstitution of orbital floor height with autogenous bone, alloplastic implant, or custom plate.

Orbital Fractures

Anatomy

- Quadrilateral/pyramidal bony cavity with base facing anteriorly. The widest dimension is 1 cm posterior to the orbital rim.
- Volume: 30 ml, 4 cm horizontal dimension, 3.5 cm vertical on average [4].
- Medial walls parallel to each other; lateral walls at 90 degrees to each other. Lateral wall to medial wall 45 degrees. (Fig. 7.4).

Bones of the Orbits

- Orbital roof (two bones): Frontal and lesser wings of sphenoid.
- Lateral wall (two bones): Greater wing of sphenoid and zygomatic bone.
- Orbital floor (three bones): Maxillary bone, zygomatic bone, and palatine bone.
- Medial wall (four bones): Frontal process of maxillary, ethmoid (lamina papyracea), lacrimal and sphenoid bones.

Anatomic Landmarks

- Inferior orbital fissure gives rise to the infraorbital groove at about 2.5–3.0 cm posterior to the orbital rim; exits the infraorbital foramen about 5 mm below the infraorbital rim.
- Superior orbital fissure: CN III, IV, VI, sensory nerve V1, sympathetic fibers, superior ophthalmic vein, recurrent and middle meningeal artery.
  - Separates the greater and lesser wings of the sphenoid.
  - Delineates between orbital roof and lateral orbital wall.

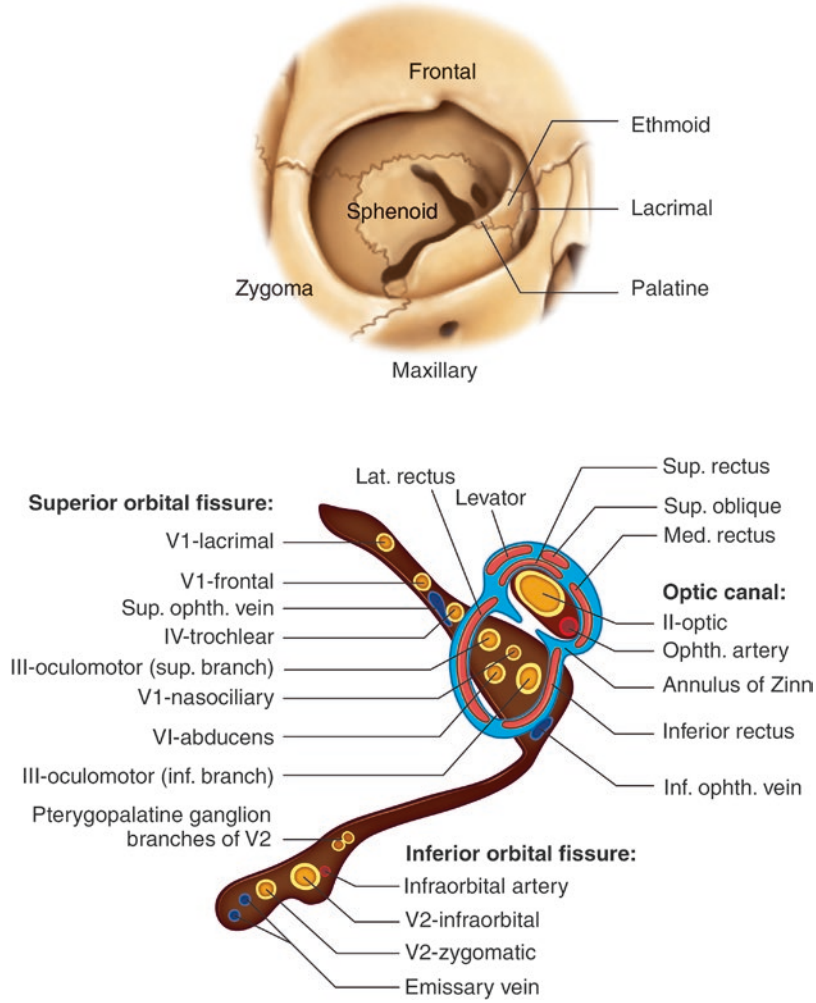

Fig. 7.4 Anatomy of the orbit and its contents. (Reprinted with permission from Bevans and Moe [4])

- Inferior orbital fissure: sensory nerve V2, parasympathetic branch of pterygopalatine ganglion and inferior ophthalmic vein.
- Optic canal: optic nerve, ophthalmic artery, and sympathetic fibers.
- Whitnall's tubercle: located 10 mm below the FZ suture and 3–4 mm inside the lateral orbital rim. Attachments (1) lateral horn of levator aponeurosis, (2) lateral canthal tendon of the eyelids, (3) Lockwood's ligament, (4) check ligaments. All four of these comprise the lateral retinaculum.
- Annulus of Zinn: a tendinous ring of fibrous tissue at the apex of the orbit surrounding the optic nerve that is the origin of the rectus muscles of the eye.
- Safe Dissection: All measurements are from an intact anterior lacrimal crest. Anterior ethmoidal foramen 24 mm, posterior ethmoidal foramen 36 mm, optic foramen 42 mm [4].

Eye Lid Anatomy

- Layers of the eye (skin, subcutaneous tissue, orbicularis oculi, septum, tarsal plate, conjunctiva) (See Fig. 7.5).
- Orbital septum: dense connective tissue arising from the orbital periosteum; forms the anterior boundary of the orbit. 1–2 mm below the infraorbital rim, it fuses with an area of thickened periosteum known as the *arcus marginalis.*

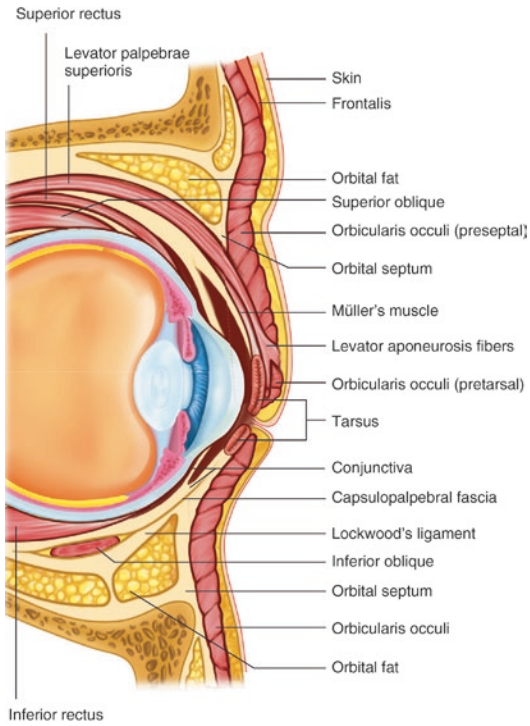

Fig. 7.5 Sagittal view of the orbital soft tissues. (Reprinted with permission from Bevans and Moe [4])

- Tarsus: dense fibroconnective tissue; approximately 1–1.5 mm thick, approximately 25 mm in horizontal length.
  - Upper eyelid: 10–12 mm in height.
  - Lower eyelid: 3–5 mm in height.
  - Contains meibomian glands which form lipid layer of tears.
- Orbicularis oculi (CN VII).
  - Palpebral (pretarsal, preseptal): reflex eyelid closure.
  - Orbital (covers orbital rims): forceful voluntary eyelid closure.
- Levator palpebrae superioris (CN III): main retractor of upper eyelid.
- Muller's (superior tarsal) muscle (sympathetic): responsible for the tone of the upper eyelid, ~2 mm of lift; minor retractor of the upper eyelid.
- Whitnall's ligament.
  - Supports the superior anterior eyelid; provides vertical support for the orbit.
  - Inserts superomedially on the frontal bone behind the trochlea; inserts superolaterally near the frontozygomatic suture.
  - Key landmark that marks the transition from levator muscle to aponeurosis (at the junction of the levator muscle and aponeurosis).
- Capsulopalpebral fascia: main retractor of the lower eyelid; terminal extension of the inferior rectus muscle.
- Inferior tarsal muscle (sympathetic): minor retractor of the lower eyelid.
- Lockwood's ligament.
  - Lower lid counterpart to Whitnall's ligament (formed by conjoined fascia of inferior rectus and inferior oblique muscles).
  - Inserts on the medial and lateral canthal ligaments as well as the bony orbital rim.
- Medial canthal ligament (tripartite attachment).
  - Anteriorly inserts onto maxillary bone, posteriorly onto posterior lacrimal crest, superiorly onto orbital process of frontal bone.
- Lateral canthal ligament.
  - Inserts onto Whitnall's tubercle (zygomatic bone) 2–3 mm inside the lateral orbital rim, 1 cm inferior to the frontozygomatic suture.
- Nasolacrimal duct opens into the inferior meatus of the nasal cavity 10 mm behind the nasal aperture; reflux of tears is prevented by Hasner's valve.

Terminology

- Anisocoria – different sizes of the pupils.
- Diplopia – double vision. May be monocular (concern for retinal attachment or lens dislocation) or binocular (unequal movement of eyes usually due to edema or possible entrapment).
- Enophthalmos – inward positioning of the globe, usually due to increased volume of orbit.
- Exophthalmos – outward positioning of the globe, usually due to edema but concern for hematoma or in-fracture versus out-fracture of orbit walls.
- Hyperglobus – superior positioning of the globe.
- Hypoglobus – inferior positioning of the globe.

- Hypertropia – misalignment of eyes, a form of vertical strabismus.
- Proptosis – synonymous with exophthalmos.
- Ptosis – drooping of the eyelid.

Evaluation

- History, mechanism of injury: High velocity/low velocity; in high-velocity injuries increased concern for penetrating globe injuries, concomitant injuries such as head, spine, or NOE, etc.

Physical Evaluation

- Inspection – look for edema, ecchymosis, subconjunctival hemorrhage, and proptosis.
- Evaluate for exophthalmos; Hertel (based off zygomaticofrontal suture) or Naugle exophthalmometers (based off of the frontal bone, more useful when lateral orbital rim involved).
- Palpation – used to assess the presence of bony discontinuity/steps indicative of fracture.
- Visual acuity (Snellen chart), visual fields (Goldman chart, confrontation), pupils (afferent pupillary defect), EOM (entrapment, forced duction test to r/o incarceration of orbital contents, diplopia in forward gaze is of most concern), slit lamp (Wood's lamp (cobalt blue) for corneal injury), fundoscopic (vitreous or retinal hemorrhage), tonometry pen (pressures normal 10–20 mmHg).
- Confrontation exam: Testing the visual fields consists of confrontation field testing in which each eye is tested separately to assess the extent of the peripheral field. To perform the test, the individual occludes one eye while fixated on the examiner's eye with the non-occluded eye. The patient is then asked to count the number of fingers that are briefly flashed in each of the four quadrants.
- Sensory changes in infraorbital distribution, document sensory testing level.
- Intraocular injuries – shape of lens, traumatic iritis, hyphema, angle recession, commotio retinae/Berlin's edema, vitreous hemorrhage, retinal tears/holes/detachments.
- Muscle entrapment. Ductions can illicit nausea, vomiting, and bradycardia due to oculocardiac reflex. In children, be cautious of a "white eyed" blowout fracture.

Radiological Examination

- A helical CT w/o contrast scanning with 1–3 mm cuts in axial and coronal planes with soft tissue and bone windows.
- Useful to identify craniofacial deformities, globe and optic nerve integrity, orbital foreign bodies (metallic), and diagnosing a retrobulbar hemorrhage.
- MRI – Poor bony details but can aid in globe trauma or foreign body detection.

Classification

- Linear: fractures maintain periosteal attachment and do not result in a defect with orbital content herniation.
- Blow-out: most common; limited to one wall and typically 2 cm or less in diameter. Infraorbital rim remains intact.
- Complex: extensive fracture involving 2 or more walls.

Management

- Consider ophthalmology evaluation to document globe or vision concerns.
- Most fractures may be observed for 2 weeks for resolution of motility disturbance resulting from edema, hemorrhage, or rectus muscle contusion.
- Indications for fracture repair:
  - Large orbital fractures >50% orbital floor; enophthalmos >2 mm, diplopia in primary gaze.
  - Asymptomatic patients without the aforementioned signs and symptoms – observation.
  - Muscle incarceration is a true emergency as entrapped tissue will become ischemic.

Signs of oculocardiac reflex require emergent surgical intervention.
- Consider antibiotics (with sinus coverage), sinus precautions, nasal decongestants, ice packs, and head of bed elevation.
- If status of one eye is blinded, be more guarded to operate.

Surgical Approaches

1. Transconjunctival:
   - Lower rate of eyelid malposition.
   - Direct access to the orbital floor can be extended with a lateral canthotomy and transcaruncular incision to get wide access to lateral and medial walls.

Technique:
- A corneal shield with ophthalmic-grade bacitracin/ocular lubricant is placed on the globe.
- Local with vasoconstrictor is injected under the conjunctiva to aid in hemostasis as well as around the lateral canthus if lateral canthotomy is planned.
- Using a 15 blade, a sharp incision is made through the lateral canthus. The tip of an iris scissor is placed inside the palpebral fissure, extending laterally to the depth of the underlying lateral orbital rim (lateral extension should not exceed 7 mm, ensuring a safe distance from the temporal branch of the facial nerve). The scissors are used to cut horizontally through the lateral palpebral fissure (incising through skin, orbicularis muscle, orbital septum, lateral canthal tendon, and conjunctiva).
- Using the lateral orbital rim as a stop, inferior cantholysis is performed by turning the orientation of the scissors vertically to incise the inferior canthal ligament.
- The conjunctiva is approached using blunt-tipped pointed scissors to dissect through the small incision through the conjunctiva, made during the lateral canthotomy. The conjunctiva is bluntly undermined over the orbital septum and extended as far medially (3 mm away from the caruncle).
- Scissors are then used to incise the conjunctiva below the curvature of the tarsal plate. (2 5–0 nylon traction sutures may be used through the cut edge of the bulbar conjunctiva to assist in retraction and to hold the corneal shield in place.)
- The inferior bony orbit is palpated. With retraction of the orbital contents and the lower lid, the dissection continues to the orbital rim, taking care to stay lateral to the lacrimal sac.
- Periosteal elevators are used to strip the periosteum over the orbital rim, anterior surface of the maxilla, zygoma, and orbital floor.
- A broad malleable retractor is placed to protect the orbit and to confine any herniating periorbital fat.
- After exploring the orbit, releasing entrapped tissue, and identifying the bony landings, the orbital floor is reconstructed to support the globe contents.
- Forced duction test is used to ensure uninhibited mobility of globe.
- A 4–0 Vicryl® is used to reattach the lower limb of the lateral canthal tendon.
- Subcutaneous sutures and 6–0 skin suture are placed along the horizontal lateral canthotomy.
- Some surgeons reapproximate the bulbar conjunctiva with 6–0 fast gut suture in a single or running fashion suture.

2. Subciliary:
   - Good access to floor.
   - Higher incidence of scarring, ectropion/entropion.
   - The skin incision is made 2 mm below the gray line. There are three approaches for dissection down to the orbital rim with the subciliary approach.
     - 1. Skin.
     - 2. Deep to the orbicularis oculi muscle (skin-muscle flap).
     - 3. Step dissection.
   - It is more common to utilize a skin-muscle flap, as it is less likely to lead to a lid malposition after healing. Violating the orbital septum integrity leads to a higher risk of vertical lid shortening.

Technique:

- A corneal shield with ophthalmic-grade bacitracin/ocular lubricant is placed on the globe.
- The skin incision is made 2 mm below the gray line.
- The path of dissection deep to the orbicularis oculi muscle includes the pretarsal orbicularis muscle in the elevated skin muscle flap if the skin incision is placed across the tarsus.
- With the skin muscle flap and the step technique, maintaining the integrity of the orbital septum is paramount. The incision through the periosteum for entry into the floor of the orbit is made beneath the infraorbital rim (3 mm below).
- A subperiosteal dissection is accomplished posteriorly exposing the orbital walls.
- Periosteal elevators are used to expose the orbital floor, release entrapped tissue, and identify stable margins.
- A broad malleable retractor is placed to protect the orbit and to confine any herniating periorbital fat.
- After exploring the orbit, releasing entrapped tissue, and identifying the bony landings, the orbital floor is reconstructed to support the globe and its contents.
- Closure is usually performed in two layers. The periosteum is reapproximated with a resorbable suture. The skin is then closed with a 6–0 non-resorbable or fast-resorbing suture.

Complications

Orbital Implant Infection – implant removal, culture and start antibiotics.

Implant Migration or Extrusion Early – repositioning with additional fixation.

Implant Migration or Extrusion Late – requires implant removal.

Ectropion – due to shortening of the anterior lamellae. May require tarsal strip.

Entropion – due to shortening of the posterior lamellae. May require Quickert-Rathbun suturing technique (passing a gut suture through the inferior fornix anteriorly toward the lashes). Severe cases may require grafting with oral mucosa.

Persistent Enophthalmos – due to improper position of implant, fat atrophy, and tissue loss.

Sympathetic Ophthalmia – injury-induced autoantibodies to uveal tissue; ~80% occur within 3 months.

- Treatment options include:
  - Enucleation: entire removal of globe without rupture.
  - Evisceration: leave the sclera +/− cornea.
  - Exenteration: entire contents of the orbit.

Retrobulbar Hemorrhage – 1% incidence, signs/symptoms: pain, proptosis, decreased visual acuity, increased intraocular pressure, and ophthalmoplegia.

- Manage medically with IV infusion 20% mannitol 2 g/kg to shrink the vitreous humor, acetazolamide (Diamox ®) 500 mg bid, or steroids.
- Manage surgically with lateral canthotomy with cantholysis.

Nasolacrimal Duct Injury

- Jones I test: few drops of fluorescence dye or propofol in the lower conjunctival sac, observe for fluorescein/propofol in the nose. If not identified, perform Jones II test.
- Jones II test: irrigate the punctum and inject fluorescein into the (SAC) puncta/canaliculi.
  - If fluorescein is seen, then the blockage is after the lacrimal sac; if not, then the blockage is near the punctum or canaliculus.
- If a laceration is present and visible, early repair is advocated, but reasonable to wait 3–6 months if no laceration is present.
- Primary repair: dilate with bowman probe, place stent (Crawford tube, Jackson tube) through the puncta and nasolacrimal duct opening in the nose, suture both ends with 8–0 PDS sutures, and leave the stent for 3 months.
- Secondary repair: dacryocystorhinostomy; the goal is to create a bony window between the lacrimal sac and nose.

Corneal Abrasions – Symptoms: pain, tearing, photophobia, foreign body sensation, treatment with topical antibiotics.

Hyphema – Bleeding from torn vessels at the root of the iris in the anterior chamber; signs/symptoms: positional blurred vision, photophobia.

- Grade 1: ¼ of anterior chamber; Grade 2: ½; Grade 3: ¾; Grade 4: complete coverage of the anterior chamber, aka blackball or 8-ball hyphema.
- Manage with atropine 1% ophthalmic drops bid/qid (dilates pupil and immobilizes the iris to prevent further bleeding), timolol ophthalmic drops bid (beta-blocker to decrease intraocular pressure, acetazolamide 500 mg PO bid (carbonic anhydrase inhibitor, IOP > 35 mm Hg), steroids, bed rest with HOB elevated.
- Complications: 2.5% to 38% re-bleeding most common 2–5 days post-injury; glaucoma after one year; corneal blood staining in 5% [5].

Afferent Pupillary Defect (Marcus Gunn pupil) – Swinging-flashlight test: light in the affected eye produces mild to no consensual light pupillary reflex; then swinging light to normal eye produces equal constriction; then swinging light to affected eye produces dilation during direct light stimulation.

Traumatic Optic Neuropathy – Decreased vision in the affected eye; ipsilateral afferent pupillary defect.

- Thought to be due to vascular insufficiency; goal is to reduce microvascular spasm and soft tissue edema.
- Treatment with large dose steroids (methylprednisolone 30 mg/kg IV loading dose, then 5.4 mg/kg/hr. IV infusion for 23 hours) within 8 hrs.

Traumatic Mydriasis – Pupillary dilation due to interruption of the parasympathetic innervation.

- Results in anisocoria; treatment with 2% prilocaine; may resolve over several days or weeks.

Traumatic Iritis – Inflammation of the anterior chamber of the eye, onset within 3 days of trauma.

- Symptoms: dull pain, tearing, and photophobia.
- Treatment with cycloplegic agents: scopolamine 0.25 or cyclopentolate 2%.

Horner's Syndrome – injury to sympathetic nerves supplying the globe.

- Triad of signs: (1) miosis (unopposed parasympathetic), (2) eyelid ptosis (decreased Muller's muscle tone), (3) anhidrosis (sweat glands).
- Diagnosis: 4% cocaine drops to the affected eye fails to dilate compared to the unaffected pupil.

Superior Orbital Fissure Syndrome – Ophthalmoplegia (CN III, IV, VI), lid ptosis (CN III), mydriasis and loss of direct pupillary light reflex (CN III parasympathetic fibers). Treatment is dependent on etiology. Retrobulbar hematoma (see above). If superior orbital fissure narrowed in the setting of fractures, then surgical intervention for the displaced fracture segments is required. IR may be indicated in the setting of a carotid-cavernous fistula. Ophthalmology and neurosurgery should be consulted as an intracranial/transethmoidal approach may be indicated should decompression in the posterior orbit be necessary.

Orbital Apex Syndrome – Superior orbital fissure syndrome + injury to CN II (loss of vision and direct and consensual pupillary light reflex). Treatment is dependent on etiology. Retrobulbar hematoma (see above). IR may be indicated in the setting of a carotid-cavernous fistula. If superior orbital fissure narrowed in the setting of fractures, then surgical intervention for the displaced fracture segments is required. Ophthalmology and neurosurgery should be consulted as an intracranial/transethmoidal approach may be indicated should decompression in the posterior orbit be necessary.

Naso-Orbito-Ethmoid (NOE) Fractures

NOE anatomy – the NOE complex consists of the nasal bones, frontal processes of the maxilla, nasal process of the frontal bone, and the medial orbital wall (comprised of the lacrimal bone and ethmoid bones).

Manson and Markowitz Classification

Based on the condition of the central fragment (frontal process of the maxilla) and the medial canthal tendon.

- Type I – no comminution of the central fragment and the tendon is intact.
- Type II – comminution of the central fragment and the tendon is intact.
- Type III – severe comminution of the central fragment and the tendon is avulsed.

Imaging

- CT scan is the most valuable tool for assessment with axial and coronal view 1.5 mm slices. Axial cuts can aid in the diagnosis of the status of the frontal sinus and the medial canthal tendon.

Physical Evaluation

- Depressed nasal dorsum (saddle nose deformity).
- Crepitus.
- Telecanthus – intercanthal distance should be coincident with the width of the alar bases. Clinician would perform the bow-string test to confirm disruption of the medial canthal tendon. Intercanthal distance varies between ages, races, and gender. Normal range for a white adult is 28–35 mm. Intercanthal distances greater than 35 mm are suggestive of medial canthus involvement and 40 mm or more are diagnostic for traumatic telecanthus.
- Almond-shaped eyes – due to the detachment of medial canthus.
- Periorbital edema and ecchymosis.
- Anosmia – damage to cribriform plate leading to damage of olfactory nerves.
- Paresthesia/anesthesia along distribution of the infraorbital nerve.
- Epiphora – occurs due to obstruction within the nasolacrimal apparatus. Jones I and Jones II tests. Early onset may be due to swelling.
- Enophthalmos (remember the medial wall being involved can also lead to increased orbital volume).
- Epistaxis.
- Rhinorrhea – CSF leak noted as thin blood-tinged discharge from nose. Test for beta-2 transferrin. May also send sample for glucose and chloride level. Chloride is usually greater and glucose is less than serum. Halo test involves placing a drop of the bloody rhinorrhea on filter paper and seeing a center of blood and a straw-colored halo.

Treatment:

- As the name implies, nasal and orbital treatment is required in these fractures.
- Type I and type II fractures are treated by securing the main fragment(s) in an anatomically reduced position.
- Type III fractures require a canthopexy with a posterior superior vector.
- Approaches usually include overlying laceration, orbital approaches, and coronal approaches.

Nasal Dorsal Augmentation/ Reconstruction

- Often the dorsal nasal bones need reconstruction due to weak support. This is seen as a saddle nose deformity with or without flattening of the nasal dorsum.
- Treatment is commonly done with the outer cortex of the calvarium as it is relatively flat

and easily recontoured. It is stabilized by a small bone plate.
- Extension of bone graft should reach region of lower lateral cartilages for nasal tip support.

Management of the Avulsed Medial Canthal Tendon via Canthopexy

- Transnasal wiring technique. The vector of fixation is posterior and superior to the lacrimal fossa.
- Suturing the tendon to a miniplate in the NOE region. A non-resorbable suture is used and the vector of fixation is posterior and superior to the lacrimal fossa.
- Mitek anchoring procedure – use of suture anchoring device for management of medial canthal tendon. The vector of fixation is posterior and superior.

Complications

Dacryocystitis – infection of lacrimal sac due to obstruction. Treatment with antibiotics such as penicillin-based drugs.

Epiphora – first attempt lower lid massage; if no improvement, dacryocystorhinostomy should be considered. In this procedure, an incision is placed 6 mm from the medial canthal angle and dissection is carried to the lacrimal sac. An H incision is made in the nasal soft tissue and lacrimal sac. The posterior flaps are sutured together. The puncta are intubated with a Crawford tube and passed through the openings of the nose. The ends of the Crawford tube are tied and the anterior flaps of the nasal mucosa and lacrimal sac are sutured together. The orbicularis muscle and skin are closed. The stent is left in place for 3–6 months.

Nasal Fractures

- Nasal bones are the most commonly fractured facial bones in adults.
- Paired nasal bones are attached superiorly to frontal bone and laterally to the frontal processes of the maxilla.
- The inferior attachment of the nasal bones is to the upper lateral cartilage.
- Nasal septum rests on the nasal crest of maxilla.
- Nasal septum is thick posteriorly along the bony junction with vomer and ethmoid bones.
- Kiesslebach's plexus, located along the anterior aspect of the septum, is the confluence of anterior ethmoidal artery, posterior ethmoidal artery, nasopalatine artery, and septal branch of the superior labial artery; the plexus is the most common site of epistaxis.

Physical Evaluation

- Swelling.
- Bruising around mid-face.
- Epistaxis (anterior and posterior nasal bleeding).
- Deviation of nasal complex.
- Mobility of nasal complex upon digital manipulation.
- Difficulty in breathing (congestion).
- Numbness of midface.
- Peri-nasal lacerations.
- Loss of nasal projection (especially true with naso-orbital-ethmoid fractures).
- Anosmia.
- Trauma to the nose can also create trauma to the nasal septum; septal hematoma can occur with nasal trauma and must be evaluated and treated immediately; the nasal septum is the major source of support for the nasal complex.
- It is always advisable to inquire about previous nasal fractures or the appearance of the nasal complex prior to injury (i.e., looking at the driver's license photograph of the patient).

Radiographic Evaluation

- Computed tomography without contrast is the imaging modality of choice for initial evaluation; plain X-rays are acceptable but only in isolated cases and clearly do not offer as much information as a CT scan.

Treatment

- Fractures of the nasal bone can be treated with closed or open reduction under general anesthesia.
- If treated within the first 10–14 days following injury, most nasal fractures can be appropriately and predictably treated with closed reduction.
- Closed reduction involves administration of a vasoconstrictor into the nasal cavity and then digitally, or through the aid of appropriate instrumentation (e.g., Boise elevator), reducing the nasal bones in their appropriate premorbid state.
- If a septal hematoma is present, it must be drained immediately at the time of the initial evaluation; otherwise, collection of blood between the septal cartilage and the mucoperichondrium will eliminate the only source of blood supply to the cartilage, resulting in cartilage necrosis and future saddle nose deformity.
- Oftentimes, the nasal septum is also displaced and requires appropriate reduction back on the crest of the maxilla.
- Open reduction of nasal fractures may be required when the injury is older than 10–14 days and the nasal bones can no longer be manipulated easily.
- Open reduction requires incision within the nose in order to perform osteotomies (endonasal lateral osteotomy) or accessing the fracture sites through existing lacerations in order to reduce fractures.
- Internal fixation of nasal bones with titanium plates and screws is seldom performed in isolated cases of nasal fracture.
- Placement of nasal packing or intranasal splints is advisable to maintain the reduction and to aid in hemostasis.
- External splints along the dorsum are also useful in order to maintain the external architecture of the nose during the healing period.
- If internal packing is used, systemic antibiotics should be administered for a few days.

Post-operative Management

- External and internal packings are typically removed within the first 7 days following repair.
- Systemic decongestant and nasal saline rinses can be used in the post-operative phase.
- Topical nasal decongestants should not be used for longer than 48 hours; chronic use of nasal decongestants interfere with normal nasal mucosal thickness and increase the need for frequent usage (rhinitis medicamentosa, aka rebound nasal congestion).
- Persistent nasal edema and swelling should be expected; typically, majority of the swelling is resolved in the first few weeks after surgery.
- Normal nasal function and airflow will be impaired for the first few weeks; patients need to be reassured of this possibility.
- Persistent nasal airflow obstruction and/or nasal complex deviation beyond 2–3 months will require a post-traumatic rhinoplasty.
- Full thickness laceration, especially along the nasal tip, can compromise the vascularity of the nasal tip; therefore, open structure rhinoplasty should be delayed for 12 months to avoid tip necrosis.

Complications

Bleeding – treat with anterior and posterior nasal packs. If local hemostatic measures are used and bleeding is still not controlled, then consider embolization with interventional radiology.

Post-Traumatic Nasal Deformity – wait until 1 year after the initial surgery. Consider septorhinoplasty after 1 year.

Frontal Sinus Fracture

- Requires between 800 and 1600 lbs. of force for fracture. (Much higher than mandible, NOE, or zygoma.) Should look for other injuries.

- The mucosal fluid exits the frontal sinus through the ostium located on the posteromedial portion of the sinus floor.
- 15% of the population has a true nasofrontal duct facilitating frontal sinus drainage into the middle meatus of the nose. The remaining population drains via the hiatus semilunaris to the nasal frontal tract.
- No universally accepted classification system of frontal sinus fractures exists. However, Gonty's classification is often used, and describes the location, extent of bony injury, and associated fractures.

Gonty's Classification

- Type 1 – isolated anterior table.
- Type 2 – anterior and posterior table fractures.
- Type 3 – posterior table fracture.
- Type 4 – comminuted fracture.

Radiography

- CT scan modality of choice: axial (anterior and posterior table evaluation) and coronal (frontal recess evaluation) slices are important.
- Thin cut (1 mm), high-resolution facial CT scan with reconstructions in the axial, coronal, and sagittal planes are standard for evaluation of frontal sinus fractures.
- Head CT scan is important for the evaluation of pneumocephalus, extradural hematoma, and subarachnoid hemorrhage, which are commonly associated with frontal sinus fracture and necessitate neurosurgical consult.
- Cervical spine evaluation and potential CT or MRI imaging is important as a cervical spinal fracture or ligamentous injury should be ruled out prior to definitive management of the fracture to prevent spinal cord injury during patient positioning and/or intubation.

Physical Evaluation

- Evaluate for laceration over frontal bone (possible direct access for repair).
- Glabellar swelling.
- Depression of frontal bone (not always visible due to swelling/hematoma).
- Supraorbital numbness.
- Eyelid ecchymosis.
- Air emphysema/crepitus.
- Rhinorrhea – present as a clear or strawberry-colored fluid; however visual inspection is often limited due to the presence of blood and nasal secretions.
  - A halo test, where the fluid is dropped onto a tissue paper can reveal the presence of CSF by formation of a clear ring around the blood.
  - Samples of CSF will show high chloride, low potassium, and low glucose concentration (>30 mg/dl) compared to normal serum.
  - Intrathecal fluorescein dye injection with imaging.
  - The definitive test for CSF rhinorrhea is the beta-2 transferrin assay. Collect nasal secretions in sterile tube and send to the lab. Can be held at room temperature for 1 week without compromise of sample. The assay is based on a western blot, which takes 4 days to process. Requires 5 cc of fluid collection for accurate diagnosis.
  - β-trace protein (βTP) may also be used as a diagnostic marker, but not reliable in patients with renal deficiency or bacterial meningitis.

CSF Leak

- Management of CSF leak should be done in collaboration with a neurosurgical consult. A CSF leak typically results from a posterior table fracture with an associated dural tear. The presence of a CSF leak can be managed conservatively with observation (the use of prophylactic antibiotics is controversial).
- If the CSF leak does not resolve with observation within 7 days, neurosurgical management may include placement of a lumbar drain to decrease the intracranial pressure or direct repair of the dural tear (if the drain is not successful).

Anterior Table Fractures

- Treatment of anterior table fractures is dictated by the aesthetic deformity secondary to the fracture. In the case of nondisplaced and minimally displaced fractures, the patient can be treated with observation alone. Decongestants may be indicated to aid in sinus system pressure relief. Consider 6 week reimaging to ensure fluid levels are dissipated and frontal duct system is intact.
- Typically, displacement of the anterior table is defined as 1–2 mm, or greater than the width of the anterior table; however, there is no accepted standard.
- After addressing the timing of surgical intervention, the approach must be planned.
  - In the case of an open frontal sinus fracture, the existing soft tissue laceration(s) can typically be used to expose the fracture and extended as needed to provide adequate visualization.
  - Options for exposure of closed frontal sinus fractures include the coronal and supraorbital approaches.
  - Additional options for exposure that are used by some authors include the frontalis rhytid approach.
  - Endoscopically assisted procedures have also been described with anterior wall fractures without duct involvement.
- Resorbable plates/screws may be a good alternative, as these bones are not load bearing.

Nasofrontal Duct Involvement/ Management/Obliteration (Ablation)

- Commonly patency is tested intraoperatively by injection of dye (fluorescein, methylene blue, propofol) into the duct/tract with a large bore catheter and observation for passage into the nasal sill.
- If outflow is compromised or uncertain, a sinus obliteration procedure is recommended. The key steps include complete exposure of the sinus, obliteration of the entire mucosal surface, and addressing the frontal sinus drainage tract.
- Complete removal of the mucosa is important in order to prevent mucocele formation. Depending on the size of the sinus and fracture, osteotomies should be considered to increase access and visualization. The borders of the cavity can be visualized with illumination with a fiberoptic cord or discovered using long pickups to identify borders. Care should be taken to remove mucosa invaginated into the foramina of Breschet. Goal is to ensure a "safe sinus."
- The lining of the sinus floor, containing the mucosa of the nasofrontal ducts, is then elevated, inverted, and placed back into the infundibulum to obstruct the outflow tract. The sinus floor is then typically packed with local tissue to ensure separation of the inverted mucosa from the sinus. Typically, a small piece of temporalis fascia or muscle is used, but a thin piece of calvarium can also be harvested and trimmed for this purpose. Synthetic fibrin sealants are an alternate option for occluding the nasofrontal duct.
- The optimal method for obliteration of the remaining free space is controversial. The most common materials used are abdominal fat or iliac crest bone. Additional autologous tissues that are commonly used include fascia, muscle, and pericranium.
- The use of synthetic materials such as hydroxyapatite, methyl methacrylate, bioglass, gelfoam, etc. is less common but has also been described [6, 7]. These synthetics are not recommended due to the risk of infection from poor vascularity.
- Another commonly used and accepted technique for sinus obliteration is spontaneous osteogenesis, which occurs when the sinus cavity is left empty.

Posterior Table Fractures

- These fractures rarely occur in isolation and are typically associated with an anterior table fracture. Additionally, the sinus floor and frontal sinus drainage tract may be involved.

- Indications for non-operative management of a posterior wall fracture include non-displaced fractures without a cerebrospinal fluid (CSF) leak.
  - A non-displaced fracture with a small CSF leak may be observed for up to 7 days for resolution of the CSF leak.
  - Conservative treatment includes bed rest, stool softeners, elevation of the head of bed between 35–45 degrees, and sinus precautions. CSF leaks greater than 72 hours may require a lumbar subarachnoid drain. Those leaks lasting greater than 7 days require surgical intervention.
- A posterior table fracture is generally considered displaced if the bone has moved a distance equal to or greater than one table width.
- Options for operative treatment of a posterior table fracture include:
  - Frontal sinus obliteration with or without cranialization.
  - Frontal sinus obliteration alone can be considered in cases with a displaced posterior table fracture that involves less than 25% of the posterior table, has minimal to no comminution, and does not have an associated CSF leak [7].

Cranialization

- In the case of large or highly comminuted displaced posterior fracture fragments, a cranialization procedure should be performed.
- It involves frontal craniotomy, repair of dura, debridement of the damaged brain segment, repair of dural lacerations, removal of the posterior wall, removal of the mucosal lining of the sinus, and plugging the nasofrontal ducts; a pericranial flap is used to separate the sinus from the splanchnocranium [8].
- The brain is allowed to fill into the extradural space and the anterior table is reconstructed.

Approaches to Frontal Sinus Fractures

- If there is a division of the sinus by a septum, no treatment of the unharmed portion is necessary.
- In the case of open fractures, the most common approach is an extension of the laceration to obtain adequate exposure.
- The most common approach used for closed frontal sinus fracture is the coronal (bitemporal) approach.
- Additional options include the supraorbital, frontalis rhytid approach, the endoscopic approach, and combined open and endoscopic techniques.
- The coronal approach provides the best visualization and is ideal for bilateral frontal sinus fractures that necessitate a wide exposure.
  - This approach has an acceptable cosmetic result and the bulk of the incision is well hidden within the patient's hair.
  - Another advantage of this approach is that it facilitates harvesting of cranial bone graft if needed.

Coronal Approach Technique

- To aid in hemostasis and dissection, local anesthetic or tumescent may be used to insufflate the planned dissection.
- Consideration for hairline and lack thereof: The incision normally curves anterior at the vertex 5 cm behind the hairline. In the bald patient, consider a more posterior incision. Access is created the more inferior the incision extends, if the arches require exposure (incision may extend to the inferior portion of the ear lobule).
- The traditional initial incision is extended sharply through skin, subcutaneous tissue, and galea between the temporal lines exposing the loose areolar plane. Blunt dissection is used to elevate in all directions, but primarily anterior.
- Extension below the temporal line can be carefully completed using the subgaleal plane as a guide to bluntly dissect alongside the anterior helix. A sharp incision is made down to the instrument.
- Following the incision, additional hemostasis can be obtained with Raney clips.
- Continued exposure should be performed in the areolar tissues of the subgaleal plane. This can be developed easily with blunt dissection.

Lateral tension of the flap is due to the remaining attachments to the temporalis fascia and should be relieved to allow for anterior displacement of the flap.

- Approximately 2–3 cm superior to the supraorbital rims, the pericranium can be incised and the dissection can proceed in a subpericranial plane to obtain exposure.
- A periosteal elevator can be used at this point to continue the dissection and care should be taken to preserve the integrity of the pericranium for use as a vascularized flap if desired.
- If additional exposure is needed, the superficial temporalis fascia can be excised at the root of the zygomatic arch meeting the horizontal incision above the orbital rims at a 45-degree angle. The temporal branch of the facial nerve should be safely located on the undersurface of the temporoparietal fascia.
- The orbital foramen/notch may be osteotomized to allow release of the neurovascular bundles and further retraction.
- Access to subcondylar region can be reached through a coronal flap by detachment of the masseter muscle or osteotomizing the arch with attachment of the masseter.
- Closure should be performed in a layered fashion to minimize drooping. The temporalis fascia is often over suspended to minimize drooping and protect the facial nerve. Use of a flat suction drain is based on surgeon preference. If a running suture was placed for hemostasis at the beginning of the procedure, this should be removed.

Follow Up

Weekly follow up for 1 month. Every 3 months for the first year and every year up to year 5. CT scans are recommended at years 1, 2, and 5 or if symptomatic.

Complications

- *Meningitis* – Inflammation of the arachnoid membrane and the pia mater extending throughout the subarachnoid space, brain, spinal cord, and ventricles. Acute fever, headache, stiff neck, and confusion are common to meningitis. Kernig sign (inability to flex the leg with thigh at a right angle to the trunk) or Brudzinski sign (flexion of hips and knees when neck is flexed) may be present 30% of the time. Diagnosis made with CT scan of the head to rule out a mass or lesion; blood cultures and CSF examination for protein, glucose, cell count; and gram stain. Antibiotics are empirically used until cultures available with neurosurgical consultation.
- *Mucocele and Mucopyocele* – Mucoceles form from retained sinus mucosa with compromised sinus ventilation leading to mucous-filled lesions. A mucopyocele forms when the mucoceles are infected. Treatment involves obliteration of the sinus.
- *Intracranial Abscess* – Patients will normally have a subacute onset of illness and not appear toxic. Common signs are mental status changes, focal neurologic deficits, fever, nausea/vomiting, and seizures. Treatment involves neurosurgical consultation, parenteral antibiotics (e.g. third generation cephalosporins) with possible craniotomy for aspiration/drain placement.
- *Cavernous Sinus Thrombosis* – Clinical signs include headaches, ptosis, ophthalmoplegia, paresthesia of ophthalmic and maxillary branch of CNV, papilledema, and periorbital edema. Imaging best visualized with MRI with gadolinium but contrast enhanced head CT is also valuable. Treatment includes, broad spectrum antibiotics, anticoagulation with heparin, and sinus drainage. High dose steroids are controversial but may reduce cranial nerve dysfunction.
- *Contour Deformity* – Allow for swelling to resolve completely. May correct with bone grafting, bone cement, or custom alloplastic implants.

Panfacial Fractures

- Fractures involving the lower, middle, and upper portions of the face.

- About 20% associated with spine fractures (spine films should be taken).
- The facial buttresses act as pillars of strength and are useful in reconstruction for outcome and because they typically have thicker bone compared to the interposed areas.
- The vertical buttresses run in a cranial to caudal direction and are important for maintaining facial height. From anterior to posterior, they include the nasomaxillary, zygomaticomaxillary, pterygomaxillary, and posterior mandibular buttresses. The pterygomaxillary buttress is the only one that is not typically surgically reconstructed because it is inaccessible.
- The horizontal buttresses run in an anterior to posterior direction and are important to maintain facial projection. From inferior to superior, these include the mandibular, maxillary, zygomatic, and frontal buttresses.

Imaging

- Maxillofacial CT with axial, coronal, sagittal, and 3D reconstruction.
- Ensure cervical spine films are taken and read prior to treating the patient.
- Head CT without contrast to rule out intracranial involvement. Pneumocephalus, extradural hematoma, and subarachnoid hemorrhage commonly seen with panfacial trauma.
- Chest films to r/o aspiration of teeth or other materials. The right mainstem bronchus is more often obstructed because it is wider and more vertical than the left.

Patient Evaluation/Early Management

- ATLS protocol starting with a primary survey and evaluation of life-threatening problems.
- Airway protection, perfusion, and hemodynamic stability are confirmed or established.
- Medical history should be obtained when possible, either from the patient or family and friends.
- As previously described for the subunit, a full head and neck exam should be completed.
- Patient should be inspected for rhinorrhea and otorrhea. Cerebrospinal fluid (CSF) rhinorrhea and/or otorrhea will present as a clear or strawberry-colored fluid.
- Oral examination to identify integrity of occlusion, which can be useful as a stable base for reconstruction.

Treatment

- Extensive access to the entire facial skeleton is typically required in these fractures; therefore, a combination of surgical approaches may be needed. Patients may need tracheostomy for long-term airway control. Submental intubation may be considered if long-term airway control is not of concern.
- The timing of repair is dependent on associated injuries and can proceed once other life-threatening injuries have been addressed and stabilized. Waiting for swelling to subside is helpful to aid in appreciation of bony reduction; however, waiting too long may lead to callus formation and malunion. Consider steroid administration to expedite edema resolution.
- There is no "one size fits all" approach to developing a plan for surgical reconstruction of panfacial fractures. The choice of approach is dependent on the fracture characteristics. It is generally advisable to work from known to unknown (meaning that reconstruction should begin with the less comminuted fractures where anatomic reduction can be more easily assessed and then proceed to the more comminuted regions where bridging constructs and bone grafting may be required).
- There are two general sequences described that allow for a systematic approach to these injuries, and they include the bottom up and inside out approach, and the top down and outside in approach. Once you choose a method, you should not deviate.
- Best to visualize all fractures before you begin fixation.

Bottom Up and Inside Out

Use mandible as the foundation for reconstruction.

Typical Surgical Sequence:

- Maxillomandibular fixation – consider prefabricated splint, made from stone models, for comminuted dentate segment fractures.
- Mandibular bony/ramus/angle/symphysis fractures. Make sure to keep pressure at the angle of mandible to prevent splaying and increasing of lower facial width. Treatment of symphysis fractures may require over bending the plate to prevent splaying of the lingual cortex (always treat the dentate segment first).
- Condylar fractures if indicated to restore vertical height. If one condyle is intact, it may only require elastic training after period of maxillomandibular fixation.
- Treat the zygomaticomaxillary complex next. Fixate the frontozygomatic region first to restore facial height of the complex. Fixate the zygomaticomaxillary buttress region to restore facial projection and to ensure that the medially rotated body is back in its normal anatomical position. Fixate the orbital rim to define orbital volume and facial volume. The orbital floor should be managed last as it is critical that the aforementioned sites are placed back into alignment to prevent enophthalmos and facial widening.
- Naso-orbitoethmoid complex.
- Frontal sinus.
- Implants/augmentation – such as dorsal struts.
- Soft tissue support/repair.

Top-Down Approach

In the top down and outside approach, some authors feel there is no need to address a fractured condyle. Some authors choose this approach after restoring the occlusion.

- Frontal sinus/supraorbital rims sinus fracture.
- Zygomaticomaxillary complex.
- Naso-orbitoethmoid complex.
- Maxillary/palatal/LeFort.
- Maxillomandibular fixation.
- Subcondylar fractures.
- Mandibular bony/ramus/angle/symphysis fractures. Make sure to keep pressure at angle of mandible to prevent splaying and increasing of lower facial width. Treatment of symphysis fractures may require over bending the plate to prevent splaying of the lingual cortex.
- Implants/augmentation – such as dorsal struts.
- Soft tissue support/repair.
- In either approach, it is important to use the dental arches, mandible, sphenozygomatic suture, and intercanthal region as key landmarks to obtain an anatomic reduction.
- In the case of highly comminuted fractures or regions with extensive bone loss, primary bone grafting is recommended. The most common areas that necessitate bone graft include the frontal sinus, medial wall and floor of the orbit, the nasal dorsum, and zygomaticomaxillary buttress.
- Calvarial bone graft is typically used as it is exposed through the coronal approach and may have a lower incidence of resorption.
- The ribs and pelvis are also sites that can be used to obtain structural bone graft. All structural bone graft should be included in the fixation construct to minimize motion and increase the chance of graft incorporation.

Soft Tissue Injuries

Foreign Body

- The common foreign body is glass and easily detected in plain films if superficial.
- Negative CT scan does not rule out foreign body.
- The radiodensity of wood, plastic, and vegetative materials is very low and often missed.
- Wood will show on MRI.
- Ultrasound aids in detection of most foreign bodies in soft tissue.

Tetanus

- Tetanus is a neuromuscular disease caused by *Clostridium tetani,* a spore forming, gram-

positive, strictly anaerobic bacillus found in soil, intestines, and feces.
- Spores germinate to produce exotoxin tetanospasmin, a potent neurotoxin that is carried to the nerve terminals blocking spinal cord inhibitory neurons, which causes trismus, spasm of facial expression muscle (Risus sardonicus), and spasms of voluntary skeletal muscles [9].
- The CDC recommends tetanus part of immunization programs for children and is included in the DTaP (diphtheria, tetanus, and pertussis) vaccination.
- Tetanus prophylaxis should be evaluated for contaminated wounds.
- Tetanus toxoid should be administered if the patient has not been administered vaccination over 10 years, failed to complete a primary tetanus vaccination of at least three doses, or has an unclean wound and has not received tetanus vaccination in over 5 years (booster dose of 0.5 ml intramuscular).
- If no history of immunization or uncertain/incomplete, passive immunity with human tetanus immune globulin should be administered (250 U intramuscular single dose).

Soft Tissue Management

- Due to the rich vascularity of the face, facial wounds that are clean can be closed within 48 hrs. This differs from non-head wounds that should be closed no more than 19 hours after insult.
- Inoculation of infectious organisms must exceed 10 organisms/gram tissue for gram-positive and gram-negative bacteria. The number of bacteria present is of more concern than the species.
- Staphylococcus and Streptococcus are the species most involved in the contamination of facial skin.
- Tissue crushing injuries are 100 x more susceptible to infection.
- Wounds must be debrided of all foreign materials.
- Pulsatile (high pressure) irrigation requires pressure of 7 lb. Psi to remove adherent bacteria with a balanced salt solution or a scrub brush.
- Povidone-iodine and hydrogen peroxide are toxic to fibroblasts and must be diluted to a point where bactericidal effectiveness is compromised.
- Limit debridement of tissues (much will survive due to vascularity).
- Wounds should be closed in a tension-free manner, taking into account the phases of wound healing and Langer's lines of skin tension.
- Wounds maximally ever gain 80% of the strength of intact skin.
- Sutures recommended to be removed in the face in 7–10 days, and in thin tissue areas such as eyelids in 3–5 days (tensile strength regained is only 5–10% at this time point).
- Causes of marks include (1) epithelialization due to extended stay of sutures, (2) tissue necrosis secondary to tension across suture line, and (3) reactive suture type.
- Subcuticular sutures can remain up to 4 weeks.

Bites/Rabies

- Dog and feline bites – *Pasteurella multocida, Streptococci,* and *Staphylococcus aureus.* Domestic dogs on average have a biting force of 320 lbs. of pressure [10].
- Human Bites – *Eikenella corrodens, Staphylococcus Aureus, Haemophilus influenzae,* and *Corynebacterium*. Additionally, consideration should be given to Hepatitis B and C, herpes simplex virus, syphilis, tuberculosis, and HIV, which can be transmissible through human bites [10]. Human biting force on average is 120 lbs. of pressure.
- Unlike bites to regions other than head and neck, non-infected wounds should be closed primarily due to the abundance of vascularity. Early closure leads to the best aesthetic outcome.

- Wounds with exposed cartilage are most likely to become infected.
- Treat with high-pressure irrigation with normal saline.
- Antibiotic coverage of choice is amoxicillin and clavulanic acid (Augmentin®). If penicillin allergic, then consider doxycycline and metronidazole.
- Rabies is an important consideration following an animal bite. It is a viral infection of single stranded RNA virus (rhabdovirus family).
- Bats, raccoons, skunks, and foxes are the major reservoirs in the United States due to vaccination programs of domesticated animals.
- Bites from wild animals or domesticated animals (allowed to roam in rural or semi-rural areas) should be presumed to inoculate with the rabies virus.
- Domesticated animals should be observed for 10 days for changes in behavior. In the event of erratic behavior, the animal should be euthanized and brain examined for evidence of rabies.
- Human rabies virus infections are divided into two forms: (1) encephalitic ("furious") and (2) paralytic ("dumb") [11, 12]. The encephalitic form occurs with the hydrophobia, delirium, and agitation. The paralytic form shows symptoms ascending paralysis, hypophonia, polyneuropathy, and symmetrical quadriparesis (paralysis is usually more severe in the bitten limb).
- Spread of the virus is via peripheral nerves to the central nervous system.
- Post exposure prophylaxis includes involved passive immunity by giving 20 IU/kg human rabies immunoglobulin directly around the wound and any remaining volume intramuscularly [11]. 1 ml of human diploid cell vaccine or purified chick embryo cell vaccine should be given intramuscularly on days 0, 3, 7, 14, and 28.

Parotid Injury

- Buccal branch of the facial nerve often runs together with the parotid duct. Can estimate the course of the duct by drawing a line between the tragus and midportion of the upper lip.

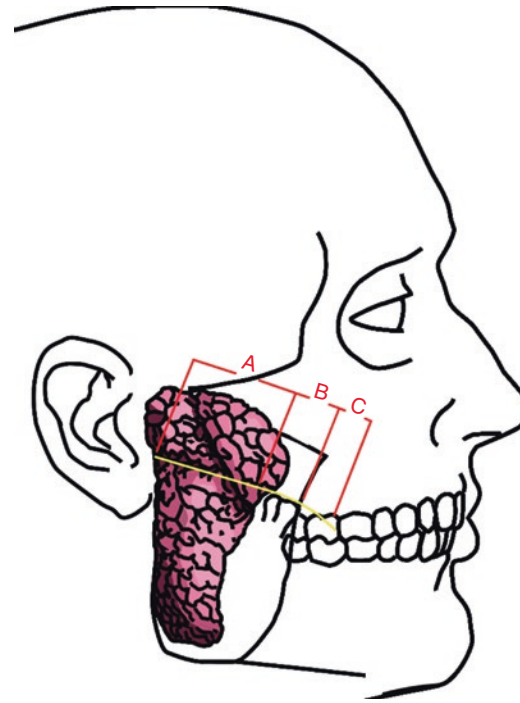

Fig. 7.6 Anatomic classification of parotid duct injuries. (Image courtesy of Erik Steenberg)

- Often transection of the buccal branch of the facial nerve is accompanied by duct injury.
- Van Sickels divided Stensen duct in three distinct sites (Fig. 7.6) [13]. Site A corresponds to the most proximal part of the duct, intraglandular. Site B corresponds to the part of the duct that is located superficial to the masseter muscle. Site C corresponds to the part of the duct located anterior to the masseter muscle and subsequently enters the buccinators. The duct terminates intraorally, adjacent to second maxillary molar. If injury is at sites B or C, attempt to identify stumps for repair. For site A injuries, treatment is only closure of parotid capsule; these injuries have lower complication rates.
- Lacerations require routine repair of the soft tissue injury. Consider drain placement. Intermittent aspiration may be required and compression dressing must be done.
- Sialocele.
  - Formed by leak of saliva into glandular or periglandular tissue.

- Check to ensure saliva via amylase levels >10,000 u/l is confirmatory.
- Treatments include: pressure dressing and multiple aspirations with or without antisialogogues (propantheline 15 mg PO QID half hour prior to meals), octreotide, parasympathetic denervation (tympanic neurectomy), secondary duct repair, intraoral fistula creation (dochoplasty), low radiation (1800 rad/treatment for more than 6 weeks total of 30 Gy) and for nonresponders Botox (10–20 units of botox-A), superficial or total parotidectomy.
- Anticholinergic pharmacotherapy in the form of propantheline, scopolamine, or glycopyrrolate may be used to reduce saliva production and the risk of recurrence.

Table 7.4 House-Brackmann scale for facial nerve injury

House-Brackmann Grading scale		
Grade	Description	Characteristics
I	Normal	Normal facial function in all areas
II	Mild dysfunction	Synkinesis – Hypokinetic/ uncoordinated facial movement with symmetry at rest
III	Moderate dysfunction	Noticeable weakness or synkinesis; symmetry at rest; complete eye closure with maximal effort
IV	Moderately severe dysfunction	Obvious weakness/ disfigurement; symmetry at rest; incomplete eye closure with maximum effort
V	Severe dysfunction	Only perceptible movement, asymmetry at rest
VI	Total paralysis	No movement

Stensen Duct Repair

Repair should be done preferably in the first 24 hrs.

- Anesthesiologist to avoid sympatholytics or only short acting agents.
- Use ketamine to encourage salivary flow.
- Identify distal end with 20–22-gauge silastic tube via the opening of the duct, which can be identified with a lacrimal probe.
- Identify proximal end of duct, may be eased by parotid massage to encourage salivary flow.
- Repair duct with 6–0 nylon.
- Stent to be kept in place 5 days up to 3 weeks and given sialogogues (lemon drops) to prevent scarring.

Facial Nerve Transection

- The House-Brackmann scale is used to test facial nerve function (Table 7.4).
- Facial nerve repair should be attempted posterior to a line drawn perpendicular to the lateral canthus (Fig. 7.7).
- Repair within 72 hours, prior to Wallerian degeneration and loss of ability to identify nerve with stimulator.
- Use nerve stimulator to identify distal end; proximal end identification may be aided by retrograde dissection; repair the epineurium with 9–0 nylon on a GS-8 needle. Use three sutures to obtain anastomosis. Fibrin glue and collagen tubes have been used to aid anastomosis.

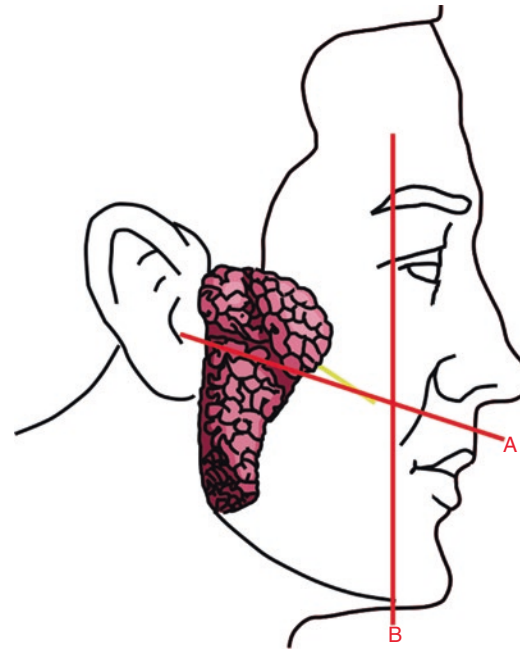

Fig. 7.7 A line joining the midline of the upper lip with the tragus of the ear approximates the course of the parotid duct. Injury along this line behind a line from the lateral canthus to the mental foramen has great chance of injuring the parotid duct or gland and the facial nerve. (Figure courtesy of Erik Steenberg)

Laceration of Submandibular Gland

- Repair of duct normally unnecessary as will form a fistula in the floor of mouth. If submandibular fistula forms, serial aspirations with pressure bandage will normally resolve this. If not, the submandibular gland should be removed.
- Sialodochoplasty done for pathological resections that include the floor of the mouth.

Lacrimal Apparatus

- Laceration through medial canthal region and NOE injuries should raise suspicion for nasolacrimal injury.
- Consider ophthalmology consult to rule out globe injury.
- Epiphora may be present.
- Anesthetize the medial canthus, dilate punctum with dilator, and pass silicone intubation stent (Crawford tube); pigtail probe is passed through the intact punctum and canaliculus to identify the transected portion.
- Cannulate upper and lower punctum and thread stent into nose (below inferior turbinate), cut the steel rods, and tie a knot. Allow 3 months to heal, remove tube through the segment visible in the corner of the eye.
- Alternatively, may use a stainless steel rod threaded with black silk or monocanalicular monoka® stent.
- Chronic lacrimal duct obstruction can be managed with a dacryocystorhinostomy.

Scars

- Wounds kept moist heal faster than those exposed to air:
  - Keratinocytes migrate sooner.
  - Prevents hypoxia which drives angiogenesis and retention of growth factors.
  - Affords protection against exogenous organisms.
  - Retains water and proteolytic enzymes, which debride the wound.
- Silicone sheeting has been shown to aid in keeping moisture in and keeping out bacteria. The sheets are permeable, which allow some oxygen to enter the wound. In addition, they control tension on the wound to prevent stretching and irritation. They have also been shown to suppress fibroblast activity and decrease capillary activity and collagen deposition leading to decreased dermal thickness (decreases scarring). They are to be used after epithelialization has occurred.
- Hypertrophic scars develop within the borders of the wound. Keloids are scars that extend outside of the wound borders. Treatment includes intralesional steroids that can be started at 1 month post-op (e.g. Triamcinolone 40 mg/ml, 0.2 ml given every 3 weeks for 3 months). Aggressive injections can lead to significant atrophy.
- Silicone sheeting, flashlamp-pumped pulsed-dye laser 585 nm or 1064 nd:YAG non-ablative laser, dermabrasion at speed of 35,000 rpm with diamond fraise burrs of medium course can also be used.
- Radiotherapy, 15–20 Gy over 6 sessions, should be considered for refractory cases.
- Scar can take up to 1 year to mature as collagen remodels. It is imperative to allow adequate time to healing. A delay of at least 6 months is recommended.

Ear

- Fluoroquinolones are prudent for injuries that involve the cartilage to cover *Pseudomonas aeruginosa*. However, it is toxic to developing cartilage and should not be given to patients under 18 years of age. If perichondritis develops, assume it is from this pathogen.
- The ear is extremely vascular and only requires small pedicle for revascularization.
- Elastic cartilage found in the ear, which is relatively avascular, is not commonly sutured as this may devitalize the area. If suture is required, then a fine chromic suture is recommended.
- If there is partial avulsion, classically the Mladick technique (retroauricular pocket) is performed: de-epithelize amputated auricle, perform anatomic cartilage reattachment, and bury into retroauricular pocket. The second stage (2 weeks later) is cartilage elevation and split thickness skin graft.
- The Baudet technique for ear repair [14]: amputated auricle's posterior surface is de-

epithelized, cartilage fenestrated, retroauricular pocket raised, and anterior pinna skin sutures placed. Second stage: ear elevation and split thickness skin graft.
- Temporoparietal fascia flap may be used to cover denuded cartilage and a split thickness skin graft.
- Auricular hematoma (usually from blunt trauma) can lead to cartilage destruction and replacement with fibrous tissue. Early treatment with needle evacuation (incision and drainage for late treatment). A bolster dressing should be left in place for 7 days.

Nasal Hemorrhage
- Exam should be performed after achieving profound anesthesia and vasoconstriction for visualization. Anesthesia and vasoconstriction for exam can be achieved with 4–10% cocaine topical solution (max 1 mg/kg for infant or 2–3 mg/kg for adult). Another option is utilizing local anesthesia with oxymetazoline-soaked neuro sponges.
- Septal hematomas are drained with small mucosal incision or needle drainage. Nasal pack or septal stent secured with a multiple pass 4-O suture technique is utilized to prevent recurrence.
- Nasal bleeds are more commonly anterior (from Kiesselbach area aka Little's area), where posterior bleeding is commonly from the sphenopalatine artery and the posterior pharyngeal artery (Woodruff's plexus) (Fig. 7.8).
- Control of an anterior bleeding may include [15]:
  - Finger compression for 10 minutes.
  - Topical vasoconstrictors.
  - Silver nitrate application.
  - Nasal sponge cut to 4–6 cm (from 10 cm sponge) coated in petroleum jelly removed in 48 hrs.
  - 1/2 inch petroleum jelly soaked gauze removed after 48 hrs.
  - Balloon tamponade coated with Sodium Carboxymethylcellulose, e.g., Rapid Rhino © (to aid in gel coat and to encourage platelet aggregation) soaked 30 seconds in sterile water (not normal saline as it may interfere with hydrocolloid fabric). Fill the bladder of the rhino with air from a syringe and monitor tactile feedback of pilot cuff (cuff becomes rounded and feels firm). Remove in 24–72 hours.
- Control of a posterior bleed is most commonly managed with the use of a 14 French Foley catheter with a 30 ml balloon. Fill it

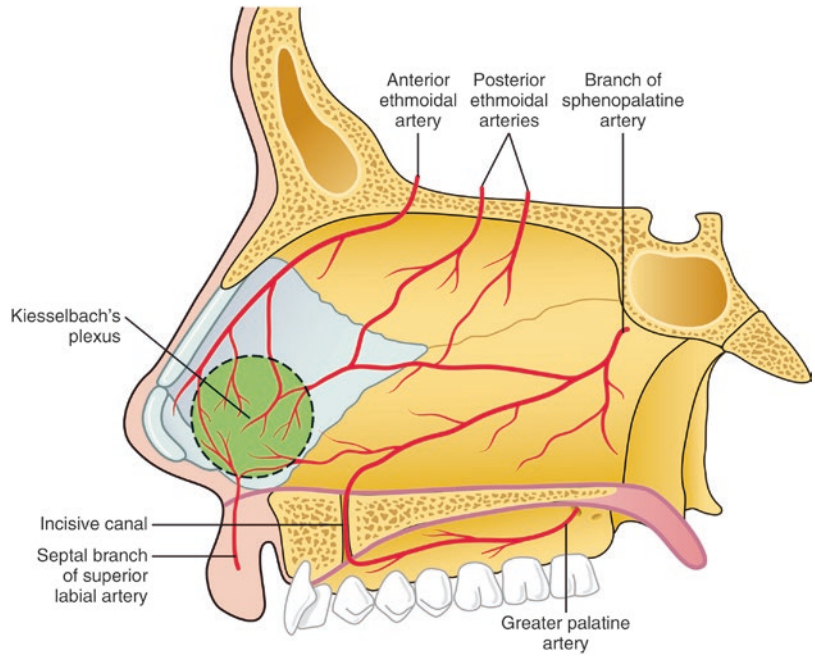

Fig. 7.8 Arterial supply to nasal septum. (Reprinted with permission from Waldman S. Chap. 147 – Neuradenolysis of the Pituitary: Needle-Through-Needle Technique. Fourth Edition. Atlas of Interventional Pain Management. Elsevier Inc.; 2015)

with 10–15 ml saline. Retract the Foley so the balloon is wedged and add 3–5 ml of more saline. Remove in 3 days. Of note, some doctors use air versus saline due to aspiration risk if the catheter were to rupture. On the contrary, leakage of air renders the catheter ineffective.
- Nasopulmonary reflex – mediated by trigeminal and vagal nerves. Can be seen in patients with COPD, advanced pulmonary or cardiac conditions. Nasal packing can cause a 15 mm Hg drop in arterial oxygen pressure [15].
- If packing measures fails, then consider endoscopic sphenopalatine artery ligation (EPSAL) for a posterior bleed or anterior ethmoid artery ligation for anterior bleed. Extensive bleeding may require ligation of both regions.

Lip Laceration
- Defects of up to 25% of the width of the upper lip can be closed primarily, and 30% of the lower lip.
- Misalignment of 1 mm of the vermillion border can be detected by the human eye.
- Intermediate defects up to 2/3 of the upper or lower lip can be reconstructed with either the Abbe flap or the Estlander flap.
- Larger defects may require Karapandzic flap, Gilles flap, or a Webster-Bernard flap.

Pediatric Maxillofacial Trauma

Nasal Fractures

- Common fractures in the pediatric population.
- Edema frequently masks the fracture.
- Diagnosis is usually made by clinical exam but a plain film or CT scan can be used.
- Clinical exam must include ruling out other associated fractures and ruling out the presence of a septal hematoma. If a septal hematoma is noted, it must be drained as in the adult patient.
- Treatment is usually observation or closed reduction. Rarely is open reduction necessary.
- Growth disturbances can occur and be associated with premature ossification of the septovomerine or nasoethmoid suture. This may lead to a restriction in midface growth.

Naso-Orbital-Ethmoid Fractures

Rare injuries but have significant effects on growth when displacement occurs and is not repaired. Premature ossification or obliteration of the frontoethmoidal, frontolacrimal, frontomaxillary, nasomaxillary, or ethmoidomaxillary may result in midface hypoplasia in the vertical and anterior/posterior planes.

- If the fractures are nondisplaced, then observation is acceptable.
- If displaced, precise reduction and fixation must be performed (consider resorbable plating systems).
- If there is telecanthus, then the canthal ligament must be resecured. If the location of the disruption is unclear, then it must be secured in a more superior and posterior position. Ideal treatment is within the first 4 days of injury.

Orbital Fractures

- The pattern of orbital fractures may be influenced by the changing craniofacial ratio of the growing child.
- Signs of globe injury such as asymmetric pupils, hyphema, torn bulbar conjunctiva, and corneal damage warrant a prompt evaluation by an ophthalmologist.

Fronto-Orbital Injuries (Orbital Roof Fractures)
- Occur primarily in children <7 years of age.
- Secondary to a proportionally larger cranium and a lack of rudimentary sinuses present.
- Orbital roof fractures have a greater likelihood of associated neurocranial injuries.
- Non-displaced orbital roof fractures may be observed.
- Neurosurgical consultation is advised as these fractures may extend into the frontal bone.

- If the bones are displaced, extraocular muscle movements are affected, or intracranial injury is confirmed, then an open approach is indicated via a coronal incision.
- Consider using a resorbable plating system.

Lower Orbital Fractures (Orbital Floor and/or Medial Wall)

- Occur primarily in children >7 years of age.
- Increased vulnerability of the face due to growth and pneumatization of the paranasal sinuses.
- Clinical signs include ecchymosis, diplopia, restricted upward gaze (entrapment), enophthalmos, hypoglobus, loss of globe support, and loss of orbital volume.
- Trapdoor fractures are linear, medially hinged, minimally displaced, and run along the infraorbital nerve canal.
- Assess for the presence of "white eyed" orbital floor fractures (no subconjunctival hemorrhage or overt signs of orbital trauma is seen) that can result in muscle necrosis of the inferior rectus muscle due to entrapment. The oculocardiac reflex may be seen in this patient population (intractable nausea and vomiting, bradycardia, and occasionally syncope.)
- Early intervention is crucial (2 days maximum).
- The consequence of late detection and treatment is muscle ischemia and permanent gaze restriction.
- Resorbable mesh or Gelfilm ® are adequate materials for the reconstruction of most orbital floor/wall fractures.
- If there is a large defect, a calvarial bone graft may be necessary.

Zygomatic Complex Fractures

- Clinical findings similar to that of the adult patient; periorbital ecchymosis and edema, bony step offs, paresthesia associated with the infraorbital nerve (V2) on the side affected, and subconjunctival hemorrhage.
- Ophthalmology consult required if orbital components involved.
- Minimal or non-displaced fractures can be observed.
- Displaced fractures require an open approach and one-point fixation is usually adequate for the non-comminuted pediatric zygomatic complex fracture.
- Access can be achieved with the transconjunctival, buccal, upper bleph, or lateral brow approaches.
- Comminuted fractures may require a coronal approach.
- Maxillary tooth buds are still present in children under 6 years of age and must be taken into account when fixating a fracture from the maxillary vestibule approach.

Maxillary Fractures (LeFort)

- Rare fractures in children. Prevalence increases after age 12 when sinuses have pneumatized.
- Open reduction and internal fixation is the preferred technique for management.

Mandible Fractures

- Mandible fractures account for 5–50% of all pediatric facial fractures.
- Children's mandible fractures have a high tendency to have greenstick fractures secondary to the fibroelastic properties of the bone during its development. Displaced and comminuted fractures are rare.
- Attention to the age and state of development of the dentition is necessary. At 6 months of age the first deciduous incisors erupt. By age 2.5 most children have a full complement of deciduous teeth. Root resorption of the primary teeth occurs between the ages of 5–9, and between 9–12 years of age mixed dentition is present.
- The high vascular supply to the periosteum by the inferior alveolar nerve and high osteogenic potential is the reason for early fracture healing (2–3 weeks) in the pediatric mandible fracture patient.
- Treatment of mandible fractures is dictated by fracture location, patient age, stage in development, severity of injury, and the presence of displacement or comminution.

  - Infants less than 1 year of age should be observed. Diet modification is not required.
  - For mandible fractures in young children that are not displaced and occlusion is stable, conservative management, close observation, and soft/liquid diet is appropriate treatment.
  - Displaced fractures need to be stabilized and immobilized.
  - Open reduction and internal fixation (ORIF) should be used only for fractures significantly displaced or comminuted.
- Techniques for stabilization and immobilization in pediatric patients include maxillomandibular fixation, fabrication of a lingual or occlusal splint, open reduction internal fixation, or a combination of these techniques.
- If maxillomandibular fixation (IMF) is used, closure for 2–3 weeks is adequate for children less than 12 years of age. Less time, 1–2 weeks, is appropriate, if the fracture involves the condylar process.
- In a patient without adequate dentition to secure arch bar placement skeletal fixation with wires at the circum-mandibular, circum-piriform, circum-orbital, and circum-zygomatic regions can be used to attain maxillomandibular fixation.
- Acrylic splints can be fabricated to provide stability to the mandible. They can be used when no deciduous or permanent teeth are present or fabricated on the occlusal or lingual aspects of those teeth that are present. They require taking impressions, pouring diagnostic models, cutting the models at the regions of fracture and restoring the segments to proper occlusion with wax. An acrylic splint is then fabricated off of these models and secured to the patient's mandible with wires (either circumdentally or circum-mandibular).
- A Risdon cable can also be used as a substitute to bulky arch bars that will not conform to the small deciduous teeth. The technique which entails twisting a long 24-gauge wire together and is secured to the posterior molars with additional 24-gauge wires was described by Risdon.
- Titanium plates and screws may be used although may require a secondary surgery for removal in patients 2–3 months after the initial placement. This is secondary to concerns regarding migration of the titanium plates and inhibition or alteration of growth. This is controversial, as some surgeons believe a secondary surgery may further lead to disturbances in growth. The author of this section (J.P.) typically removes titanium plates and screws in patients less than 13 years of age 2 months after the initial placement.

Symphyseal and Parasymphyseal Fractures

- Can be treated with IMF or ORIF.

The canine region is important to evaluate as the permanent canine may be at different stages of development and susceptible to injury at multiple regions including the inferior border if the tooth bud is still present. Placement of screws in this region should be avoided if ORIF is used.

Angle, Body, and Ramus Fractures

- Less susceptible regions for fractures and growth disturbances.
- Can be treated conservatively with IMF, traction, or with ORIF.
- If a greenstick fracture is present, observation is a reasonable choice of management.
- Circum-mandibular wires are useful at times to hold and provide stability to a distracted mandibular body fracture.

Condylar Fractures

- Laceration or ecchymosis of the chin should give rise for suspicion for a condylar fracture.
- Fractures of the condyle are one of the most frequent types of mandible fractures. These are also the most commonly missed and undiagnosed fractures.
- Generally categorized as extracapsular (low or subcondylar fractures extending toward the ramus) or intracapsular (fracture of the condylar head or high condylar neck above the sigmoid notch).
- With bilateral condyle fractures, clinical presentation is loss of projection and an anterior open bite.

- Based on the development of the mandible, children less than age 6 are more likely to have intracapsular fractures and those older than 6 years of age are more likely to have condylar neck or extracapsular fractures. The marrow within the condylar unit has an abundance of osteogenic progenitors, which could lead to an exuberant osteoblastic response, and hence ankylosis.

Management of Condylar Fractures

- Those condylar fractures with no evidence of malocclusion with a reproducible bite can be treated with observation and soft diet. Strong consideration should be made for intracapsular fractures due to the high risk of ankylosis.
- If the fracture is immobilized in IMF, 7–14 days is generally adequate. Elastics guidance can be used to promote the function of the joint after this time frame. This is usually followed by physical therapy to regain maximal opening and to help shape the remodeling of the new condyle. This also helps to reduce the possibility of ankylosis.
- Indications for ORIF of condylar fractures are few and include:
  - Condylar head fractures avulsed from the capsule and fossa.
  - Condylar fractures that have been displaced intracranially.
  - Bilateral condyle fractures with comminuted midfacial fractures.
  - Unacceptable occlusion after a closed technique trial has failed.

Metallic Plates and Screws

Advantages

- Superior mechanical handling.
- Greater resistance against torsional forces and allowance of compression.
- Titanium is biocompatible, hypo-allergenic, and inert.

Disadvantages

- Passive migration of metal plates in children.
- Distortion of future MRI or CT scans.
- Possible need for secondary surgery for subsequent removal.
- Growth disturbance.
- Thermal sensitivity.

Biodegradable Plates and Screws

Advantages

- Does not require additional surgery for removal.
- Blunt and non-penetrating biodegradable screw tip avoids potential odontogenic injury.
- Decreased potential obstruction to tooth eruption.

Disadvantages

- Time-consuming and technique-sensitive process of plate adaptation.
- Complex bending of the plates requires a heat source to allow the polymer chains to bend and not fracture. This may present a problem when fixating regions that require more complex shaping.
- Higher rate of visible or palpable hardware postoperatively due to greater thickness than metal.
- Foreign body reaction or sterile abscess may occur during the biodegradation and absorption process.
- Polyglycolic acid (PGA) and pure poly-L-lactic acid (PLLA) have caused adverse reactions during degradation.
- Unable to be re-sterilized.
- Limited shelf life.

Examples of Resorbable Plates Currently on the Market

- Inion CPS® system, Tampere, Finland.
- Zimmer-Biomet Lactosorb® (Lorenz Plating System).
- DePuy Synthes Rapidsorb® Rapid Resorbable Fixation System.

- Stryker Delta System®.

Management of Dentoalveolar Trauma

- Clinical Manifestations – malocclusion, mobile teeth and/or alveolar bone, gingival soft tissue lacerations, fractured teeth, missing teeth, pain with chewing or biting, and dentures not seating properly.
- Physical Exam.
  - Evaluate for any lacerations or soft tissue wounds intraorally.
  - Evaluate for any bony steps or malocclusions.
  - Evaluate dentition for fractures, mobility, or displacement of teeth.
  - Percussion and pulp test teeth.
- Radiographic Imaging – Panorex and/or CT scan.
  - Evaluate for presence of root fracture.
  - Determine degree of extrusion or intrusion.
  - Pathology.
  - Evaluate root development and pulp chamber/root canal.
  - Evaluate for jaw fractures.
  - Evaluate for tooth fragments or foreign bodies.

Classification

- Crown Crack – crack or incomplete fracture of enamel without loss of tooth structure.
- Ellis Fracture Classification.
  - Class I – Confined to the enamel.
  - Class II – Enamel and dentin involved.
  - Class III – Enamel, dentin, and exposed pulp involved.
  - Class IV – Root fracture.
- Tooth Displacement.
  - Intrusion or Extrusion.
  - Labial, Lingual, or Lateral displacement.
- Avulsion.
  - Complete displacement of tooth from its socket.
- Alveolar Process Fracture.
  - Fracture of alveolar bone with or without tooth involvement.

Treatment

- Most crown cracks and/or fractures that only involve enamel/dentin can be treated with dental restorations.
- If the pulp is exposed, then pulp capping or pulpotomy may be indicated.
- Root and/or crown fractures that extend past the gingival crevice usually require removal. Orthodontic extrusion or crown lengthening may be an option depending on apical extent of injury.
- No acute treatment is recommended for sensitivity. Although, removing occlusal contacts may help relieve pain.
- Intrusion.
  - Orthodontic assisted eruption is favored: must be done slowly over 3-4 weeks and once in position must be stabilized for 2-3 months.
  - Endodontic treatment is based on follow-up findings.
  - If a deciduous tooth is intruded, it may be extracted if it is impeding eruption of permanent tooth.
- Extrusion.
  - Usually can be repositioned and splinted for 1–3 weeks.
  - Endodontic treatment is usually needed, and patient should be evaluated at follow-ups.
- Displacement
  - Reposition tooth and alveolus and splint.
  - Repair any gingival lacerations.
  - Follow up to determine the state of pulp and periodontal damage to determine further treatment.
- Avulsion.
  - Rinse tooth immediately with patient's saliva or saline and replant immediately.
  - Try to limit contact with root surface.
  - If patient cannot replace tooth, then it should be placed in storage medium (HANKS Balanced Salt Solution or milk). Do not scrape walls of socket or root sur-

face, as this will destroy viable periodontal tissue.
  - Semi rigid splint for 7–10 days.
  - Strict follow up to evaluate for root resorption and ankylosis as well as need for endodontic treatment.
- Alveolar Fractures.
  - Place segment into proper position.
  - Stabilize for 4 weeks using arch bars or acrylic/composite splint.
  - Teeth in segment may need endodontic treatment.

Stabilization Periods (Table 7.5)

Table 7.5 Dentoalveolar stabilization periods

Dentoalveolar injury	Duration of stabilization
Mobile tooth	7–10 days
Tooth displacement	2–3 weeks
Replanted tooth (mature)	7–10 days
Replanted tooth (immature)	3–4 weeks

Case Example

You are paged by the ED about a 51-year-old male that was involved in an MVC with rollover. The patient was a restrained driver with airbag deployment and prolonged extrication. There was loss of consciousness and the patient was intoxicated with ethanol. ATLS has been completed and the patient is stable per vital signs. Secondary survey reveals that the patient's injuries were isolated to the maxillofacial region. You see the patient at the bedside the next morning. Ophthalmology has already evaluated the patient and has ruled out globe injury (Fig. 7.9).

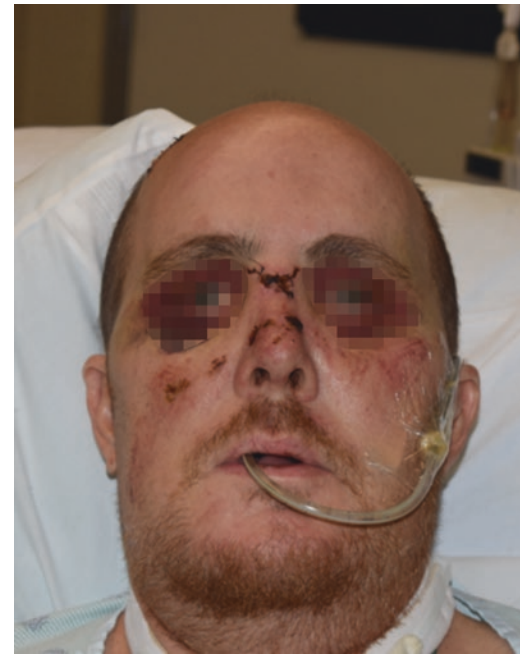

Fig. 7.9 Frontal photograph of panfacial patient. (Images are courtesy of Drs. Aaron Figueroa and Damian Findlay)

- *How do you want to proceed?*
  Get a thorough history from family members, trauma team members, chart review, and witnesses if available. Next, conduct a thorough head and neck physical exam.
  Exam reveals the following:
  - *Middle-aged male with male pattern baldness. Bilateral periorbital edema, temporal subconjunctival injection, and ecchymosis. Pupils are reactive to light. Frontal edema with appreciable depression. Steps noted at the left frontozygomatic articulation and left orbital rim.*
  - *Mobility of the nasal bones; there are tagging sutures in the nasal dorsum region.*
  - *Intraoral exam reveals left upper vestibular ecchymosis with mobility of the maxilla. Dentition is intact. A left posterior open bite and mobility at the left mandibular angle are appreciated. The fracture involves the mesial periodontal space of tooth #17. Orogastric tube placed due to excessive emesis due to oropharyngeal bleeding.*

- *What do you want to do next?*
  Get a maxillofacial CT with 3D reconstruction.

- *What do you see on the provided radiography* (Fig. 7.10)*?*
  1. Left unfavorable mandibular angle fracture.

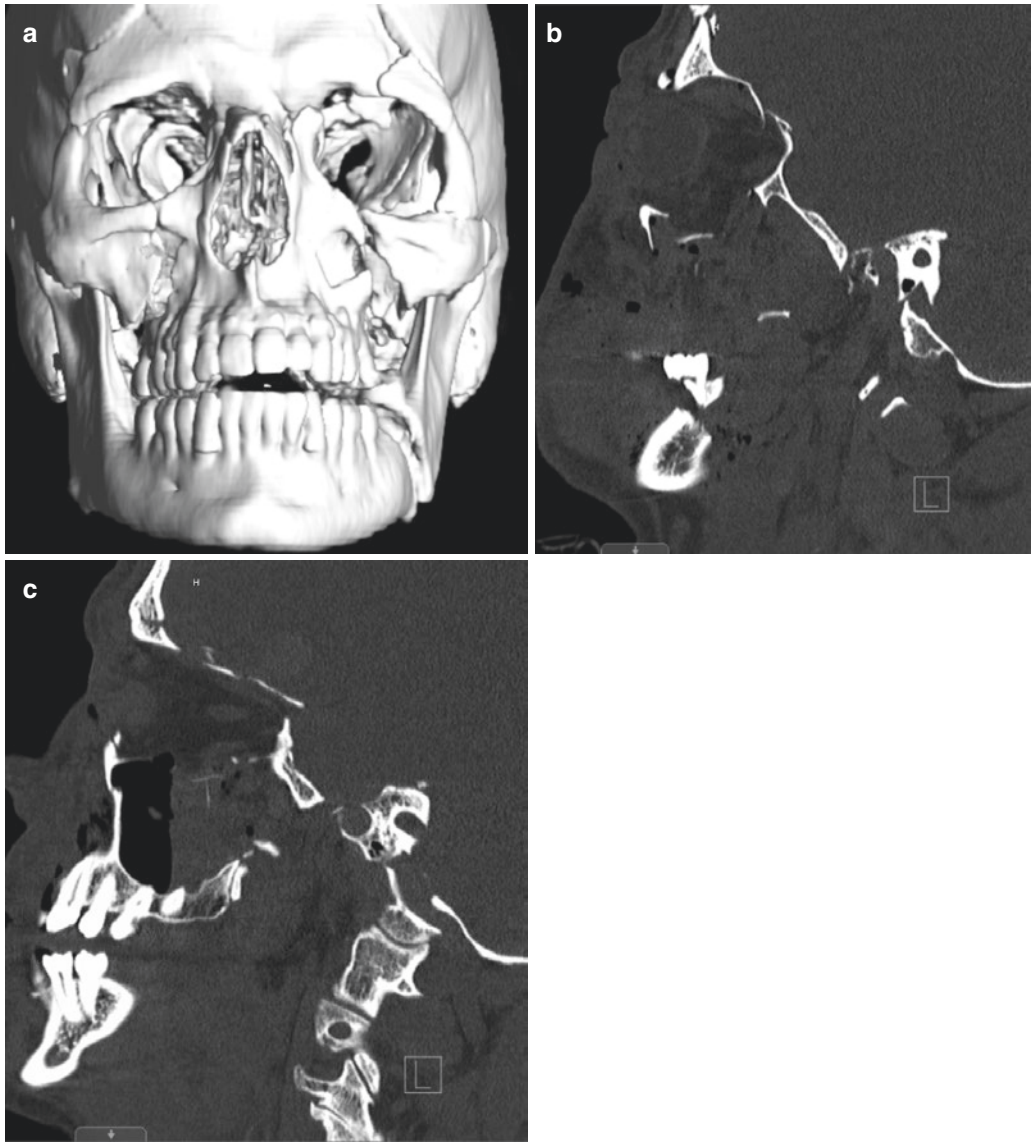

Fig. 7.10 Clinical case example of panfacial fracture from blunt trauma. (**a**) 3-D reconstruction of panfacial fracture. (**b**) CT scan without contrast sagittal view at the level of left orbit. (**c**) CT scan without contrast sagittal view at the level of right orbit. (Images are courtesy of Drs. Aaron Figueroa and Damian Findlay)

2. Left displaced ZMC fracture with involvement of the buttress and rim.
3. Right minimally displaced ZMC fracture with buttress and rim involvement.
4. Nasal bone fracture.
5. Depressed frontal bone region.
6. Bilateral Orbital floor fractures.

- *What's your diagnosis?*
  1. Depressed frontal bone fracture.
  2. Bilateral LeFort II fractures.
  3. Left displaced open mandibular angle fracture.
  4. Nasal bone fracture.

- *Can you describe the LeFort fracture patterns?*
  LeFort I (Horizontal Fracture) – extends above the apices of the maxillary dentition across the nasal septum and maxillary sinuses. Posteriorly it extends through the pyramidal process of the palatine bone and the pterygoid

processes of the sphenoid bone. It also may involve the fracture of the palate.
LeFort II (Pyramidal Fracture) – extends from the nasofrontal region down through the medial orbital wall, crossing the inferior orbital rim and zygomatic buttresses. Posteriorly similar to a LeFort I fracture.
LeFort III (Complete Craniofacial Disjunction) – fracture lines extend through the nasofrontal junctions, zygomaticofrontal articulations, zygomaticomaxillary suture, temporozygomatic suture, pterygomaxillary junction, medial and lateral orbital walls, and superior articulation of the nasal septum.

- *How would you approach this fracture?*
  My approach would be a bottom-up approach. I would first establish occlusion to allow for stability and provide a stable base for horizontal and vertical relationships to build on.
  - *Apply Erich arch bars and place the patient in maxillomandibular fixation.*
  - *Access fractures sites* via *Risdon approach, coronal, bilateral subconjunctival, and maxillary vestibular approaches.*
  - *I would begin fixation at the angle of the mandible using a reconstruction plate at the inferior border. Next I would fixate zygomaticomaxillary buttress followed by the infraorbital rims with miniplates. The frontal bone would then be reduced and secured with multiple low profile miniplates. The orbital floor fractures would be reconstructed with Medpor ® orbital reconstruction implants. The nasal bones with be reduced and stabilized with internal and external splinting.*

  **Author's Note*. It is easy to go down the rabbit hole of overwhelming detail. Allow the examiner to guide you down this hole if they wish to. A brief description normally suffices.

- *What comprises the lateral retinaculum?*
  Lateral horn of the levator aponeurosis, lateral check ligament, Lockwood's inferior suspensory ligament, lateral canthus.

- *How would you manage the tooth in the line of fracture if a vertical root fracture appreciated?*
  I would remove this non-restorable tooth as it poses a risk for infection.

- *Where is the nasofrontal ostium located in relation to the frontal sinus?*
  Its location is in a medial posterior position. The majority of the time, it is not a true duct but a recess.

- *How can patency of the duct be assessed?*
  With the frontal duct exposed, injection of sterile solutions such as propofol or methylene blue can be done into the ostium and evaluated for its passage into the nares.

- *If the duct is found to be non-patent, what is your treatment?*
  My choice would be for plugging of the duct/recess and obliteration of the sinus. This would involve carefully removing the remaining sinus mucosa. At the level of the duct, bone is used to plug the duct and covered with a pericranial flap.

- *What other materials may be used to obliterate the sinus?*
  Muscle such as a temporalis flap, fat, autogenous bone or alloplastic materials such as glass ionomer cement or hydroxyapatite.

- *What is Guerin's sign?*
  Ecchymosis in the maxillary vestibule denoting a zygoma fracture.

- *What is the earliest sign of enophthalmos in the acute traumatic setting?*
  Deepening of the supratarsal crease.

- *You are seeing your patient post-op day #2 from ORIF. The patient is tolerating a diet. He complains of intense ocular pain and a decrease in visual acuity. Exam reveals a proptotic and tense globe. What do you think is going on and how do you manage it?*

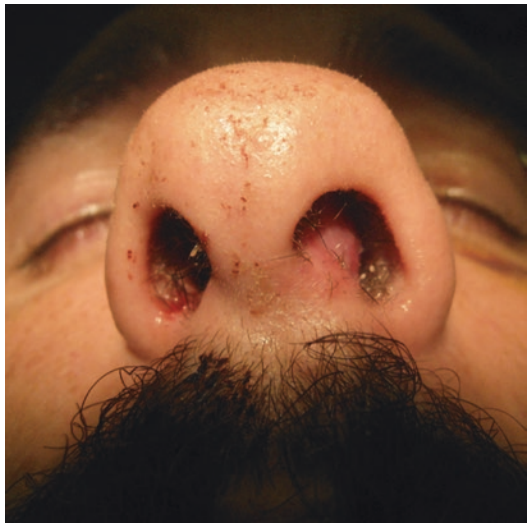

Fig. 7.11 Trauma complication. (Courtesy of Dr. Tirbod Fattahi)

A retrobulbar bleed. Perform a lateral canthotomy or remove the transconjunctival sutures to decompress the globe. Get an ophthalmology consult.

- *What is the condition seen below and how do you manage it* (Fig. 7.11)*?*
  Nasal septal hematoma. Managed by inserting a 27-gauge needle and aspirating the blood. A nasal packing should be placed for several days to prevent recollection of blood.

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Part III

Oral Pathology, Maxillofacial Reconstruction, and Cleft Lip/Palate

Oral Pathology

8

Naomi Ramer, Molly Cohen, Trina Sengupta, Chandni Desai, John W. Hellstein, Beomjune B. Kim, Ryan J. Smart, Waleed Zaid, and Prashanth Konatham-Haribabu

Odontogenic Cysts

- Cysts derived from the tissues involved in odontogenesis.

Dentigerous Cyst

- Represents 1/3 of all odontogenic cysts.
- Associated with the crown of an impacted or unerupted permanent tooth and rarely involves an unerupted deciduous tooth.
- Caused by an accumulation of fluid between the reduced enamel epithelium and the crown of the tooth.
- Has three variants:
  1. Central – cyst surrounds the entire crown of the tooth (attached to the tooth at the CEJ). The most common variant.
  2. Lateral – cyst grows lateral along the lateral aspect of the tooth.
  3. Circumferential – cyst surrounds the tooth and gives the appearance that the tooth is within the cystic cavity.
- Teeth most commonly involved (in descending order):
  - Mandibular third molar.
  - Maxillary cuspid.
  - Maxillary third molars.
  - Mandibular second premolars.
- Can cause bony expansion, root displacement, and root resorption.

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Radiographic Appearance

- Well-delineated unilocular or multilocular radiolucency.
- Root resorption may be evident.
- Impacted tooth may be displaced a long distance from its original site.

Treatment

- Size and location of the cyst will dictate treatment.
- Small lesions – enucleation.
- Larger lesions – marsupialization (followed by enucleation after decompression).
- Recurrence is uncommon.

Periapical Cyst (Radicular Cyst)

- All ages, and all teeth, can be affected.
- Most common cyst of the jaw bones.
- Occurs most commonly in the maxilla.
- Forms as a sequela of chronic inflammation in a preexisting periapical granuloma.
- Cyst is usually located at the apex of a nonvital tooth.

Radiographic Appearance

- Well-circumscribed unilocular radiolucency around the apex.
- May cause root resorption or tooth displacement.

Treatment

- Endodontic therapy.
- Extraction of tooth with apical curettage/enucleation.
- Endodontic therapy with apicoectomy.

Residual Cyst

- All ages.
- Cyst that has been left in the jaw bone after the associated tooth has been extracted.
- More common in maxilla.

Radiographic Appearance

- Well-defined radiolucency.

Treatment

- Simple excision.

Lateral Periodontal Cyst

- Also has a polycystic variant known as the botryoid odontogenic cyst (appears in a grape-like cluster).
- Most common in second–fourth decades.
- Arises in the periodontal ligament along the lateral aspect of the root of a tooth.
- Teeth are vital.
- Intrabony counterpart of the gingival cyst of adults.
- More common in males.
- Mandible: Canine-premolar region.
- Maxilla: Lateral incisor-canine region.
- Asymptomatic.

Radiographic Appearance

- Well-defined (unilocular or multilocular) radiolucency along the lateral surface of tooth root.
- Multilocular appearance seen in botryoid variant.

Treatment

- Enucleation

Glandular Odontogenic Cyst

- Large variation in the age of presentation, second to eighth decades of life (average fifth decade).
- Rare cyst that can show clinically aggressive behavior (expansion, pain, and paresthesia).
- Has a strong predilection for the anterior mandible and usually crosses the midline.
- Can have histological features of a low-grade mucoepidermoid carcinoma.

Radiographic Appearance

- Unilocular or multilocular well-defined radiolucency surrounded by a sclerotic border.
- Larger cysts may cause bony expansion and cortical disruption.

Treatment

- Curettage.
- Some surgeons advocate for marginal or en bloc resection due to high recurrence (30%) [1].

Odontogenic Keratocyst (OKC)

- Most commonly in second–third decade.
- Associated with the PTCH tumor suppressor gene.
- Derived from the rests of dental lamina (rests of Serres).
- Slight male predilection.
- More common in the mandible – third molar/ramus area.
- Maxilla: posterior region is the most common area for occurrence.
- Aggressive growth potential with higher tendency to recur (most recurrences are within the first 5 years after original treatment).
- Most are asymptomatic.
- Commonly displace the inferior alveolar nerve (IAN).
- Will resorb roots of teeth.
- Histologically will see a cyst lined by a thin layer of parakeratotic stratified squamous epithelium, which is usually 6–8 layers thick (rete ridge formation is inconspicuous).
- May be associated with nevoid basal cell carcinoma syndrome, also known as Gorlin syndrome. Features include:
  - Multiple basal cell carcinomas.
  - Multiple OKCs.
  - Palmer and plantar pits.
  - Calcified falx cerebri.
  - Rib anomalies (bifid, missing, or partially developed).
  - Spina bifida.
  - Hypertelorism.
  - Enlarged head circumference due to frontal bossing.
  - Cleft lip and palate.

Radiographic Appearance

- Unilocular or multilocular radiolucency.
- Displacement or resorption of teeth may be noted.

Treatment

- Treatment depends on size, extent, and location of lesion.
- Enucleation and curettage with peripheral Ostectomy. Large cysts may be decompressed prior to treatment.
- Cryotherapy with liquid nitrogen offers penetration up to 1.5 mm into the bone. It is difficult to work with as the peripheral tissues are at risk of injury and need to be protected. Additionally, if the inferior alveolar nerve is exposed, there is a high rate of paresthesia with reasonable recovery. Fractures of the mandible are also a known risk.
- Chemical cauterization with Carnoy's solution (mixture of ethanol, chloroform, glacial acetic acid, and ferric chloride). It is known that chloroform is a carcinogen, thus its usage has been banned in North America, rendering the use of Carnoy solution as banned. There is a modified form without chloroform, but the recurrence rate is considerably higher as chloroform is essential for its successful use.
- Some authors advocate for en bloc resection with 1 cm margins due to the high recurrence rate and the aggressive nature of the lesion.

Calcifying Odontogenic Cyst (Gorlin Cyst)

- Age: second and third decade.
- Occurrence: less than 1% of odontogenic cysts [1].
- Calcifying odontogenic cysts (COCs) behave in a benign fashion.
- Most cases are within bone, 5–17% are found extraosseous [1].
- Maxilla and mandible have equal distribution as it relates to occurrence.

Radiographic Appearance

- Unilocular or Multilocular with radiopaque structures that represent calcification.
- Displacement or resorption of adjacent teeth may be seen.
- May be associated with an unerupted tooth.

Table 8.1 Origin of odontogenic tumors

Odontogenic epithelium	Odontogenic mesenchyme	Both
Ameloblastoma	Central odontogenic fibroma	Ameloblastic fibroma
Calcifying odontogenic cyst (Gorlin Cyst)	Odontogenic myxoma	Ameloblastic fibro-odontoma
Calcifying epithelial odontogenic tumor	Cementifying fibroma	Ameloblastic fibrosarcoma
Adenomatoid odontogenic tumor	Cementoblastoma	Odontoma
Squamous odontogenic tumor		
Clear cell odontogenic tumor		

Treatment

- Conservative surgical removal.
- Low recurrence rate.

Odontogenic Tumors

- Neoplasms derived from the tissues that are involved in odontogenesis (Table 8.1).

Tumors Consisting of Odontogenic Epithelium

Ameloblastoma

- Most common epithelial odontogenic tumor.
- Age: 30–70 years.
- Most common in the mandible with a predilection for molar/ramus area.
- Causes jaw expansion.
- High rate of recurrence.

Radiographic Appearance

- Unilocular or multilocular radiolucency.
- Referred to as a "soap bubble" appearance when the loculations are large.
- Referred to as a "honeycomb" appearance when the loculations are small.
- Well-defined borders.
- Root resorption of adjacent teeth may occur.
- May simulate a dentigerous cyst when associated with an impacted tooth.
- There are many histological subtypes (See Table 8.2).

Unicystic Ameloblastoma

- 13–21% of all cases of ameloblastoma [2].
- Mimics a dentigerous cyst.
- Most common in mandibular third molar region.
- Younger age group than multicystic ameloblastomas.
- Slow growing, paresthesia uncommon.
- There are three histological variants (Table 8.3).

Table 8.2 Ameloblastoma histological subtypes

Follicular pattern	Most common. Islands of odontogenic epithelium A single layer of columnar ameloblast-like cells surrounds the central core. Central core resembles stellate reticulum. Ameloblast-like cells nuclei exhibit reversed polarity from basement membrane and subnuclear vacuoles.
Plexiform pattern	Interconnecting elongated islands of Od. Epithelium.
Acanthomatous pattern	Squamous metaplasia in the center of the islands.
Granular cell type	Eosinophilic granular cells.
Desmoplastic type	Well-collagenized stroma. May mimic a fibro-osseous lesion radiographically. More common anterior mandible/maxilla.

Table 8.3 Unicystic ameloblastoma subtypes

Unicystic	Epithelium fulfills criteria of ameloblastoma.
Intraluminal type	Nodules of ameloblastic cyst lining project into the lumen.
Mural type	Tumor islands present in the cystic wall.

Radiographic Appearance

- Unilocular radiolucency.

Peripheral Ameloblastoma

- Arises from rests of dental lamina, or from the basal cell layer of the surface epithelium.
- Exophytic mass of the tooth-bearing area.
- Normally does not invade the underlying bone.
- Less aggressive than the intraosseous counterpart.

Malignant Ameloblastoma

- Malignant (metastasizing) ameloblastoma: Metastasizes, but metastatic deposits are histologically benign.
- Overall rate <1% of ameloblastomas [1].
- Most metastases are to the lungs followed by the cervical lymph nodes.
- Ameloblastic carcinoma: histologically malignant with hyperchromatism, increased nuclear-to-cytoplasmic ratio, and presence of high mitoses [1].

Treatment of Conventional or Unicystic Ameloblastoma with Extraluminal Invasion

- Enucleation and Curettage – this procedure is not considered curative and should be used for palliative purposes such as when there is a major anesthetic or surgical risk for the patient. The particulation of an ameloblastoma with enucleation and peripheral ostectomy is known to liberate tumor cells within the soft tissue and cause debilitating recurrences. This type of treatment has recurrence rates approaching 70–85% (recurrence usually occurs within 5 years) [1].
- Marginal or block resection – curative form of treatment with resection using 1.0–1.5 cm bony margins and one uninvolved anatomical margin [3, 4]. Maxillary lesions may be more aggressive since they are not contained by cortical bone.

Treatment for Other Types of Ameloblastoma

- Unicystic ameloblastoma with intraluminal confinement – enucleation with long term follow up.
- Peripheral ameloblastoma – treatment with local surgical excision with 2–3 mm margins. Recurrence to bone treated with further local excision.
- Malignant ameloblastoma – En bloc resection of the primary tumor with wedge resection of the lung (71% cases travel to lung) and possibly chemotherapy [5]. Requires a multidisciplinary approach.
- Ameloblastic carcinoma – resection with 2–3 cm bony lesions with neck dissection. Consideration for chemotherapy with platinum-based agents and radiotherapy.

Calcifying Epithelial Odontogenic Tumor (Pindborg Tumor)

- Uncommon odontogenic tumor of the bone.
- Commonly located in the mandibular premolar region.
- Presents as a slow painless expansion.
- Age: fourth–sixth decade.
- No sex predilection.

Radiographic Features

- Unilocular or multilocular radiolucency (Fig. 8.1).

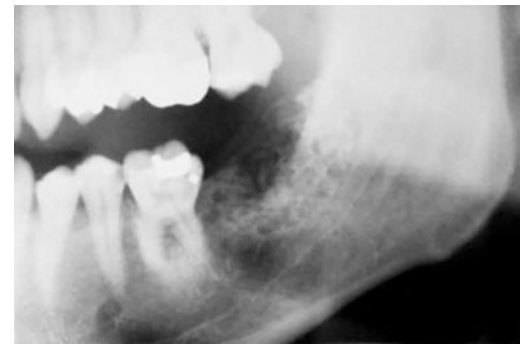

Fig. 8.1 Calcifying epithelial odontogenic tumor, aka Pindborg tumor. Can be unilocular or multilocular radiolucent lesion. (Reprinted with permission from Neville et al. [1])

- Usually well-delineated lesion, but up to 20% have ill-defined borders [1].
- May be associated with an impacted tooth.
- CT scan may show an expanding mass.
- Calcified structures (Liesegang ring calcifications) can be seen within the lesion. Amyloid-like material is found within the stroma.

Treatment

- Conservative local resection with peripheral ostectomy has a recurrence rate around 15% [1, 6].
- Some surgeons advocate for resection with 1–1.5 cm margins and an uninvolved anatomical barrier.

Adenomatoid Odontogenic Tumor/ Cyst

- 2/3rds seen in teenage females.
- Location: Anterior maxilla more commonly than anterior mandible.
- 2/3rds seen in association with impacted canines.
- Slow growing, usually asymptomatic.

Radiographic Findings

- Unilocular, well circumscribed.
- Snowflake-like calcifications.

Treatment/Behavior

- Conservative enucleation, removed easily from its bed due to a thick fibrous capsule.

Tumors Consisting of Odontogenic Mesenchyme

Odontogenic Myxoma

- Arises from odontogenic ectomesenchyme.
- No sex predilection.
- Occurs more commonly in the mandible than maxilla.
- Young adults, 25–30 years of age [1].
- Can cause expansion of the affected area and displace/resorb teeth.
- Histologically will see a myxoid stroma containing spindle- and stellate-shaped cells.

Radiographic Appearance

- Multilocular radiolucency is more common but soap bubble and honeycomb patterns do occur.

Treatment

- Curettage – used for palliation and small lesions.
- Resection – curative form of treatment with resection using 1.0–1.5 cm bony margins and one uninvolved anatomical margin [7].

Cemento-Ossifying Fibroma (Cementifying or Ossifying Fibroma)

- A distinct form of ossifying fibroma, which is confined to tooth-bearing area of jaws.
- Third–fifth decade with a female predilection.
- Occurs more commonly in the mandible.
- Slow growing, painless, and may cause large expansion.

Radiographic Appearance

- Well circumscribed, round radiolucent or radiopaque lesion.
- Displacement of teeth.

Treatment

- Conservative enucleation for a small, well-demarcated lesion, as those lesions are usually encapsulated.
- Large lesions require resection with 5 mm borders. No need to remove involved soft tissue as tumor is encapsulated.

Cementoblastoma (True Cementoma)

- Solitary lesion found in continuity with a tooth root.
- Teeth are normally vital.
- Site: mandibular premolar or molar.
- Age: most patients are less than 30 years old.
- May have expansion and discomfort.

Radiographic Appearance
- Well-defined dense radiopaque mass in continuity with the tooth root with a radiolucent halo around the lesion.
- Periodontal ligament space surrounds the mass to distinguish from hypercementosis.

Treatment
- Excision, often with loss of involved tooth (low recurrence rate).
- May also consider endodontic treatment with root resection.

Tumors Consisting of Odontogenic Epithelium and Odontogenic Mesenchyme

Ameloblastic Fibroma

- First and second decade.
- Slightly more common in males.
- Posterior mandible more commonly involved.
- Bony expansion.

Radiographic Appearance
- Unilocular or multilocular radiolucency with well-defined borders.
- Commonly associated with unerupted tooth.
- Cortical expansion common.

Treatment
- Conservative surgical excision is recommended initially, with close follow up.
- More aggressive excision for recurrent lesions.
- Ameloblastic fibrosarcoma may develop in the setting of recurrent ameloblastic fibroma.

Ameloblastic Fibrosarcoma

- Third decade of life.
- Predilection for the mandible.
- Rapid clinical growth, often with pain and swelling.
- Malignant counterpart of ameloblastic fibroma.
- May arise de novo or from pre-existing ameloblastic fibroma.

Radiographic Findings
- Poorly defined destructive radiolucency.
- Unilocular or Multilocular.
- Expansile.

Treatment
- Radical surgical excision with 1–1.5 cm margins and uninvolved margin.
- Lesions do not usually metastasize, but patients may die from uncontrolled local disease [8].

Ameloblastic Fibro-Odontoma (Ameloblastic Dentinoma)

- Younger population than ameloblastic fibroma (average age of 9) [1].
- Mandible is affected more commonly than maxilla
- No longer considered a separate entity by the WHO but a developmental stage of odontoma.

Radiographic Appearance
- Well-defined unilocular or multilocular lesion.
- Contains calcifications and often an unerupted tooth.

Treatment
- Enucleation and curettage.

Odontoma (Compound and Complex)

- The most common odontogenic tumor.
- Compound odontomas are more common in anterior maxilla. Complex odontoma are found in the posterior region of either jaw.

Radiographic Appearance
- Compound odontoma: appears as a small, tooth-like structure often surrounded by a radiolucent halo.
- Complex odontoma: radiopaque mass also surrounded by a radiolucent rim.
- Odontomas may be found associated with a tooth that has failed to erupt.

Treatment

- Excision.

Fibro-Osseous Lesions

- Fibro-osseous lesions – disease processes characterized by normal bone being replaced by fibrous tissue containing a mineralized product.

Fibrous Dysplasia

- Characterized by normal bone being replaced by fibrous tissue.
- Associated with a GNAS gene mutation/deletion. The timing of the mutation during embryogenesis determines the extent of involvement.
- Can involve one bone if mutation is late (monostotic) or multiple bones (polyostotic) if mutation is early on.
- Monostotic disease:
  - Most common form of the disease.
  - Equal male and female predilection.
  - Usually diagnosed in the second decade of life.
  - Can involve the craniofacial bones, ribs, femur, and tibia.
  - Maxilla is affected more than the mandible.
  - Manifests as a slow, painful growth of the jaw. Adjacent teeth may be displaced by the expansile lesion.
  - Maxillary lesions can cause obliteration of the antrum if extensive enough and cause a functional issue. Maxillary lesions can extend to the zygoma, sphenoid, ethmoid, and frontal bone (craniofacial fibrous dysplasia).
  - Lesions tend to stabilize after skeletal maturation and even regress in a few cases.
- Polyostotic disease is usually associated with syndromes that have cutaneous and endocrine abnormalities.
  - Jaffe-Lichtenstein syndrome – polyostotic fibrous dysplasia with café au lait pigmentation.
  - McCune-Albright syndrome – polyostotic fibrous dysplasia with café au lait pigmentation and endocrinopathies (precocious puberty, hyperparathyroidism, hyperthyroidism, and hypercortisolism).
  - Mazabraud syndrome – polyostotic fibrous dysplasia with intramuscular myxomas.

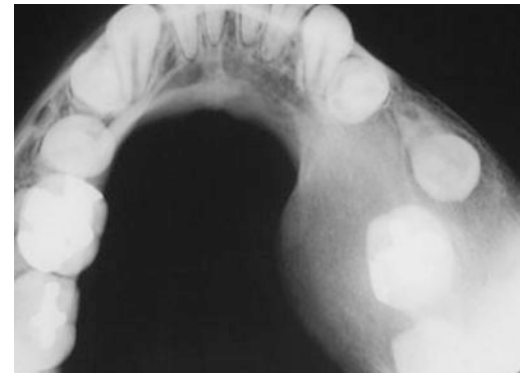

Fig. 8.2 Occlusal radiograph demonstrating ground glass appearance and expansion of the mandible seen in fibrous dysplasia. (Reprinted with permission from Neville et al. [1])

Radiographic Appearance

- Lesion appears with "ground glass" opacity and poorly defined margins. Periapical radiographs often demonstrate an ill-defined lamina dura (Fig. 8.2)

Treatment

- Conservative management is the treatment of choice.
- Patients with minimal cosmetic and functional issues may not require surgical treatment.
- Surgical contouring, shaving, and debulking may be performed for the purpose of severe cosmetic deformity and functional problems. Combined orthodontic treatment and orthognathic surgery may be needed should a malocclusion ensue
- High concern for blood loss after surgery, consider blood banking prior to surgery.
- Complete surgical removal should be considered for aggressive lesions or lesions refractory to repeated debulking.

- Radiation is contraindicated due to the risk of post irradiation sarcoma development.
- Bisphosphonates may help to relieve bone pain (must consider the risk of MRONJ should subsequent surgery be needed).

Cemento-Osseous Dysplasia (COD)

- Common in Black females, fourth–fifth decade of life.
- Teeth are vital.
- Radiographic and clinical findings can lead to a presumptive diagnosis (black female with multiquadrant or lower anterior teeth involvement).
- Pathological entity in which bone is replaced by a cementum-like material.
- Three variants:
  1. Focal COD:
     - Involves a single site.
     - Shows a female predilection and occurs from the third to sixth decade of life.
     - Mostly involves the posterior mandible in region of first or second molar.
     - Radiographic appearance ranges from radiolucent, to mixed, to completely radiopaque.
  2. Periapical COD:
     - Predilection for the anterior mandible periapical region.
     - Predilection for female patients (mostly in females of African descent).
     - Tend to be asymptomatic.
     - Early lesions tend to be radiolucent and mimic periapical granulomas or cysts.
     - Lesions tend to mature and become a mixed radiopacity and ultimately become radiodense with a thin radiolucent rim.
  3. Florid COD (Fig. 8.3):
     - Shows multifocal involvement (can involve all four quadrants).
     - Predilection for African American females.
     - Lesions are often asymptomatic and found on routine radiographic screening.
     - May develop swelling if simple bone cysts occur in long-standing lesions.

Diagnosis

- Teeth are vital.
- Radiographic and clinical findings can lead to a presumptive diagnosis (Black female with multi-quadrant or lower anterior teeth involvement).

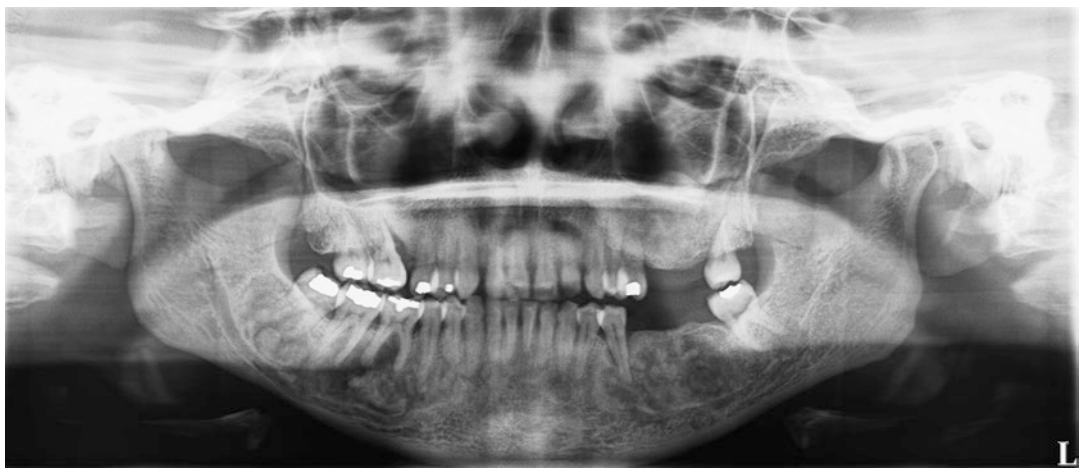

Fig. 8.3 Patient with multiquadrant cemento-osseous dysplasia (florid osseous dysplasia) . (Courtesy of Dr. Damian Findlay)

Treatment

- Lesions are not neoplastic and, therefore, surgical removal is usually not necessary
- Sclerotic lesions are hypovascular, which makes them prone to necrosis/infection from minor traumatic insults (e.g., extractions/implant placement).
- Patients with COD should have regular dental prophylaxis and follow up to prevent dental disease leading to tooth loss (periodontitis/caries).
- Patients with evidence of osteomyelitis should undergo debridement with saucerization.

Central Giant Cell (CGC) Tumor

- Intraosseous lesion that may exhibit nonaggressive (most cases) or aggressive behavior.
- Nonaggressive lesions grow slowly, expand bone, are painless, and do not resorb roots or cortical bone.
- Aggressive lesions will be symptomatic, occur in younger patients with rapid growth.
- Diagnosis for the aggressive subtype requires possessing one major or three minor criteria:
  - Major: (1) Greater than 5 cm (2) Recurrence after treatment.
  - Minor: (1) Rapid growth (2) Clinical displacement or loosening of tooth (3) Cortical thinning or perforation (4) Radiographic evidence of tooth resorption or displacement.
- Unknown origin – widely assumed to be trauma induced (but little supporting evidence).
- Female predilection (2×).
- The majority of cases are seen before the third decade of life, with a peak between the ages of 5 and 15.
- Most cases involve the mandible (usually anterior mandible).
- Nonaggressive lesions are usually discovered on routine radiographic examination or as a result of painless expansion.
- Aggressive lesions tend to be aggressive resulting in pain, expansion, paresthesia, root resorption, and tooth displacement.
- Histologically similar to brown tumors of hyperparathyroidism. Parathyroid hormone assay for primary hyperparathyroidism levels (with hypercalcemia) or for secondary hyperparathyroidism (with hypocalcemia) not routinely required as earlier concerning signs should be evident. Alkaline phosphatase elevation not necessarily needs to be assayed as it is normally elevated in growing children. CGCs demonstrate multinucleated giant cells within a spindle mononuclear cell stroma histologically.

Radiographic Features

- Well-delineated unilocular or multilocular radiolucency (Fig. 8.4).
- Displace teeth.
- Resorb interradicular bone.

Treatment

- Enucleation and curettage with peripheral ostectomy, recurrence rate 20% (more frequently in large lesions).
- En bloc resection with 1 cm margins for recurrent lesions.
- Conservative treatment for large lesions:
  - Intralesional corticosteroid injections – 1:1 mixture of local anesthetic and triamcinolone 10 mg/mL weekly for 6 weeks. 2 cc

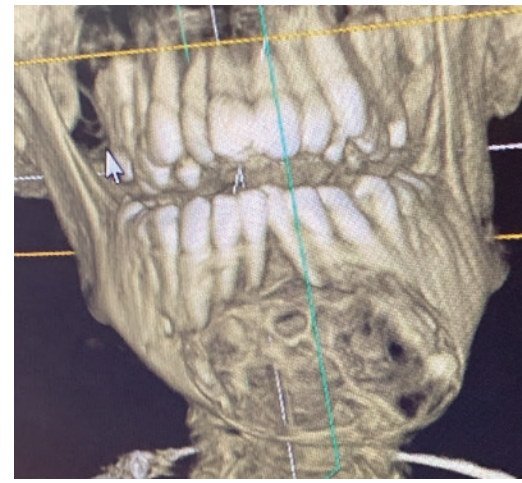

Fig. 8.4 Central giant cell tumor. Multilocular radiolucency of anterior mandible . (Image courtesy of Dr. Robert Reti)

for every 1 cc of lesion visible on an orthopantogram; has a high recurrence rate.
  - Submucosal or nasal (less absorption predictability) calcitonin: 100 units calcitonin/day for 6 months. If nausea occurs, may reduce to 50 units/day. No radiographic changes are seen for approximately 9 months and requires 18–21 months for treatment. May also be used to debulk tumor for smaller area of resection if complete resolution is not achieved.
  - Subcutaneous Interferon alpha-2a. Side effects of fever, fatigue, and flu symptoms. Usually used in conjunction with curettage and postoperative treatment for 6 months to prevent recurrence.
  - Imatinib – tyrosine kinase inhibitor.
  - Bisphosphonates.
  - Newer treatment protocols with Rank-L inhibitor Denosumab® is showing to be effective as part of care.

Peripheral Giant Cell Granuloma

- Soft tissue equivalent of central giant cell granuloma.
- Caused by trauma or irritation.
- Common tumor of the oral cavity. Part of 3Ps (1) Pyogenic Granuloma (2) Peripheral Ossifying Fibroma (3) Peripheral Giant Cell Granuloma.
- Normally smaller than 2 cm with a sessile or pedunculated base.
- Blue/purple in color.
- Female predilection.
- Common age 30–60.
- Treatment is excision down to the bone.

Medication-Related Osteonecrosis of the Jaw (MRONJ)

- The medications implicated in MRONJ are antiresorptive medications and antiangiogenic medications.
- Bisphosphonates and RANK ligand inhibitors are antiresorptive medications. These medications work by diminishing or altering osteoclasts, the cells responsible for bone resorption. RANK ligand inhibitors prevent osteoclast differentiation while bisphosphonates inhibit osteoclast function and increase osteoclast apoptosis. These medications tend to concentrate in areas of bone repair and remodeling possibly explaining their affinity for the jaws and the selectivity of osteonecrosis in this area.
- Oral bisphosphonates are most commonly used for the treatment of osteoporosis and osteopenia. They are less commonly used for Paget's disease of bone and osteogenesis imperfecta.
- Intravenous bisphosphonates for the treatment of osteoporosis are also available as once yearly zoledronic acid and four times yearly ibandronate infusions. Intravenous bisphosphonates are also used for the management of bone metastases of solid tumors, especially breast, prostate, and lung cancers as well as the lytic lesions of multiple myeloma. The RANK ligand inhibitor denosumab is also used to treat osteoporosis and manage bone metastasis (See Tables 8.4 and 8.5 for a list of medications associated with MRONJ).
- Antiangiogenic medications prevent the formation of new blood vessels and are used as a cancer treatment. They include the classes of drugs known as tyrosine kinase inhibitors and monoclonal antibodies inhibiting vascular

Table 8.4 Bisphosphonates

Drug	Indication	Route
Alendronate (Fosamax®)	Osteoporosis	Oral
Risendronate (Actonel®)	Osteoporosis	Oral
Ibandronate (Boniva®)	Osteoporosis	Oral or IV
Zolendronate (Reclast®)	Osteoporosis	IV
Zolendronate (Zometa®)	Bone metastases	IV
Pamidronate (Aredia®)	Bone metastases	IV

Table 8.5 RANK ligand inhibitors

Drug	Indication	Route
Denosumab (Prolia®)	Osteoporosis	Subcutaneous
Denosumab (Xgeva®)	Bone metastases	Subcutaneous

endothelial growth factor. There is currently little research on the association between osteonecrosis and these medications, but reports indicate that a low risk exists.

- The 2014 AAOMS position paper on MRONJ requires three characteristics to be present to distinguish MRONJ from other conditions [9]:
  1. Current or previous treatment with antiresorptive or antiangiogenic agents.
  2. Exposed bone present for more than 8 weeks that can be probed through an intraoral or extraoral fistula in the maxilla or mandible.
  3. No history of radiation therapy or obvious metastatic disease to the jaws.

Radiographic Appearance

- Radiographic changes are often seen before clinical evidence of necrosis and include increased radiopacity in the areas of increased bone remodeling such as tooth-bearing alveolar ridges.
- Periosteal hyperplasia is occasionally seen.
- In severe cases, a moth-eaten radiolucency with central sequestra is seen.

MRONJ Staging System [10]

- Stage 0 – Non-exposed bone variant:
  - Patients present with no necrotic bone but have other non-specific symptoms such as bone pain and unexplainable tooth pain.
  - Patients may have loose teeth with no evidence of periodontal disease and fistulae unassociated with pulpal necrosis.
  - Radiographic bone changes (usually areas of increased radiopacity) that cannot be attributed to another source may be seen.
- Stage 1 – Exposed and necrotic bone or fistulae that probe to bone in asymptomatic patients with no evidence of infection.
- Stage 2 – Exposed and necrotic bone or fistulae that probe to bone in symptomatic patients with evidence of infection.
- Stage 3 – Exposed and necrotic bone or fistulae that probe to bone in symptomatic patients with evidence of infection and one or more of the following:
  - Exposed necrotic bone extending beyond the alveolus.
  - Pathologic fracture.
  - Extra-oral fistula.
  - Oral antral/oral nasal communication.
  - Osteolysis extending to the inferior border of the mandible or sinus floor.

Preventative Management

- Patients should be evaluated for both acute and potential sources of oral infection prior to beginning bisphosphonate therapy. Patients with dentures should be evaluated for areas of trauma, as well. Sources of infection should be eliminated and bisphosphonate treatment initiation should be delayed until there is adequate osseous healing (takes about 1 month if conditions permit). Excellent oral hygiene should be stressed.
- For patients taking bisphosphonates for osteoporosis for 4 years or more or for those who are taking bisphosphonates along with systemic corticosteroids or antiangiogenic agents, a drug holiday should be considered beginning 2 months prior to dentoalveolar surgery and continuing until osseous healing is complete. A drug holiday is not necessary for osteoporosis patients on bisphosphonates for less than 4 years.

Treatment [10]

- For patients with MRONJ, regardless of stage, all mobile bony sequestrum should be gently removed to allow soft tissue healing.
- Stage 0 disease, treatment is pain management, antibiotics if necessary, and close monitoring.
- Stage 1: use oral antimicrobial rinses.
- Stage 2: disease is treated with antimicrobial rinses and systemic antibiotics (penicillin being the drug of choice). For penicillin-allergic patients, fluoroquinolones, metronidazole, clindamycin, doxycycline, or erythromycin may be used. Microbial suscep-

tibility testing should be performed, and antibiotics should be adjusted accordingly. In cases refractory to antibiotic therapy, dead bone should be removed surgically.
- Stage 3: patients will require antimicrobial rinse, antibiotics, and debridement with possible resection and antibiotic therapy. Histologic diagnosis is required for all bone removed in any of the above cases to rule out metastatic disease.

Osteoradionecrosis of the Jaws

- Osteoradionecrosis (ORN) of the jaws is a complication of radiation therapy for head and neck cancer.
- It occurs when irradiated bone becomes exposed through a wound in the overlying skin or mucosa and persists without healing for three or more months.
- In order to diagnose ORN, it must be proven that this necrotic bone is not related to tumor recurrence, tumor necrosis, or metastatic disease. The clinical differential diagnosis for ORN includes medication-related osteonecrosis of the jaw, metastatic disease, primary bone malignancy, and fibro-osseous lesions.
- The risk of developing ORN following radiation ranges from 5% to 15% with most cases arising in the mandible between 4 months and 3 years after completion of radiation therapy [11, 12]. This condition affects older males as would be expected, as this is the group most likely to have head and neck cancer.

Risk Factors for ORN

- The most important factor associated with bone necrosis is radiation dose. The majority of cases of ORN are reported in patients who received a radiation dose of greater than 60 Gy. Those who received brachytherapy are also at an increased risk.
- Another factor associated with a risk of developing ORN is a primary tumor located in the tongue, floor of mouth, alveolar ridge, retromolar triangle or tonsil.
- If the cancer is Stage III or IV or a prior surgery was done for the primary tumor that included a mandibulectomy or ostectomy, then the patient is also at higher risk.
- Periodontal disease, poor oral hygiene, dental extractions after radiation, alcohol use, tobacco use, and poor nutrition can all contribute to the development of ORN.

Clinical Findings in ORN

- Most cases of ORN arise following trauma such as tooth extraction, but some are spontaneous.
- ORN typically begins as a focal area of mucosal breakdown with exposure of the underlying bone. With progression, patients experience neuropathic pain, trismus, and chronic drainage compounded by the xerostomia, dysgeusia, dysphagia, and decreased tongue mobility that commonly follow radiation to the head and neck.
- If untreated, ORN will eventually lead to pathologic fracture.

Radiographic Appearance

- A panoramic film is the initial study of choice for ORN and will show poorly demarcated sclerotic radiolucent areas intermixed with lytic areas. Pathologic fracture and/or sequestra may be seen.
- CT scan can be used to more accurately delineate the lesion and to see interruptions in cortical margins. CT could show hypodense/hyperdense changes in the bony architecture.

Pathophysiology

- In 1983, Marx suggested a theory for the pathogenesis of ORN called the 3H theory.
- He proposed that radiation causes hypoxic-hypocellular-hypovascular tissue and that

microorganisms play a contaminant but not causative role. This theory helps to explain why the mandible is affected far (up to 24 times) more than other bones of the head and neck. The mandible is most likely to be in the line of radiation to the head and neck and it is less vascularized than other bones. A more recent theory suggests that because osteoclasts suffer radiation damage early, suppression of bone turnover may be the initial event in ORN.

Preventative Measures

- Prevention of ORN is the most crucial aspect of management of these patients.
- The main goal of prevention is to avoid the need for tooth extraction during or following radiation.
- Prior to beginning radiotherapy, a patient's dental and oral health should be evaluated. The aim of a pre-radiotherapy dental evaluation is to reduce the risk that the patient will need an extraction following radiation. If teeth in the area to receive radiation are extensively decayed or infected, partially impacted, and/or have pocket depths more than 5 mm, they should be extracted.
- Following extractions, 2–3 weeks of healing is advised before the initiation of radiation treatments. If necessary, extractions can be done within 4 months following completion of radiation therapy as tissue repair and healing are still taking place. Within 5–6 months, the progressive fibrosis and loss of vascularity that lead to ORN have begun.
- An important aspect of the pre-radiotherapy dental evaluation is patient education on the risk of ORN and the importance of excellent oral hygiene. Patients should be given a high-fluoride toothpaste and daily fluoride treatment, either brush-on or in a custom tray, to prevent caries caused by radiation-induced xerostomia. Edentulous patients should be monitored for breakdown of tissue below dentures.
- Advances in radiation therapy may also help to reduce the risk of ORN. Intensity-modulated radiation therapy (IMRT) and three-dimensional conformal radiation therapy allow for higher doses of radiation with less damage to surrounding tissues. Less damage to normal tissue in the mandible will reduce the risk of ORN.

Hyperbaric Oxygenation

- HBO therapy is an extension of Marx's theory that ORN is caused by hypoxia, hypocellularity, and hypovascularization.
- HBO therapy increases tissue oxygenation by stimulating angiogenesis. It also stimulates fibroblast proliferation and collagen formation, which leads to healing. Additionally, the increased oxygen tension is bactericidal and bacteriostatic.
- Optic neuritis and existing neoplasia are the only contraindications to HBO.

Marx HBO Protocol for Treatment and Prevention

Treatment of Osteoradionecrosis

- Patients receive 30 HBO dives where a dive is defined as 100% oxygen breathed at 2.4 atmospheres for 90 minutes. After 30 dives, the patient is re-evaluated. If there is improvement such as decrease in the amount of exposed bone, granulation tissue formation, or re-mucosalization, the patient completes a full course of up to 60 total dives for full mucosal cover.
- If there is no improvement after the initial 30 dives, the patient is advanced to stage II.
- Stage II treatment is trans-oral debridement or sequestrectomy with primary mucosal repair. If healing progresses, the patient completes up to 60 dives. If there is recurrent bone exposure, the patient is advanced to stage III.
- In stage III, after a minimum of 30 dives, the patient undergoes a resection to bleeding bone with primary wound closure and external fixation. The patient then dives until there is

complete mucosal closure or a total of 60 accumulated dives. Ten weeks later, the patient progresses to stage III-R and is given 20 more dives. Reconstruction is completed, followed by ten more dives with jaw fixation for 8 weeks. Patients who present with a pathologic fracture, oro-cutaneous fistula, or osteolysis of the inferior border of the mandible begin treatment as stage III.

Prevention of Osteoradionecrosis

- Patients that have received previous tumoricidal head and neck radiation should receive prophylactic HBO therapy prior to oral surgical procedures.
- Patients will require 20 dives of HBO followed by 10 dives post operatively.

Other Treatment Modalities

- Frequent visits to the oral surgeon for removal of any sequestrae from the necrotic lesions.
- Ultrasound has been shown to increase angiogenesis and stimulate collagen and bone production.
- Consider pentoxifylline (Trental®) and tocopherol (Vitamin E) for prevention and treatment of ORN.
- The pentoxifylline-tocopherol-clodronate (Pentoclo) drug combination has also been used with some success to treat refractory ORN or as a prophylactic regimen when extractions are unavoidable.

Cutaneous Melanoma

- Melanoma is a malignant neoplasm of melanocytic origin.
- Melanocytes are pigmented dendritic cells located in the base of the epidermis, the eye, and in the epithelia of the nasal cavity, oropharynx, anus, vagina, and urinary tract.
- Melanoma can arise de novo or from a melanocytic nevus (benign counterpart).
- Ultraviolet radiation exposure is the most important risk factor for cutaneous malignant melanoma (CMM). Other risk factors include fair complexion, light hair or eyes, a family history of melanoma, and a personal history of dysplastic or excessive nevi.
- CMM is most commonly diagnosed in white adults with a median age of 61, but it can occur over a broad age range.
- In patients younger than 40, there is a female predilection likely related to tanning bed use, while in older patients there is a male predilection.
- Clinically, CMM can resemble a melanocytic nevus. The "ABCDE" mnemonic (See below) can be a helpful tool when deciding if a pigmented lesion is benign or malignant.
  - A = Asymmetry.
  - B = Border irregularity, scalloping, or poor definition.
  - C = Color variegation.
  - D = Diameter. Melanomas are usually greater than 6 mm in size.
  - E = Evolving. Describes a mole that is changing in size, shape, or color.

4 Major Types of Melanoma

1. Superficial Spreading
   - Superficial spreading melanoma is the most common form of melanoma and is most likely to be found on the trunk and extremities. Clinically, it demonstrates a combination of colors from tan and brown to pink and blue. Its outline is sharply marginated and will have one or more peninsula-like protrusions, and its surface may have a papule or nodule that extends above the surface.
2. Lentigo Maligna
   - Lentigo maligna melanoma is clinically large and mostly flat with rare papules that may represent foci of invasion. It has variegated tan, black, and brown flecks of color, and the outline is irregular. They occur more commonly on the face and neck.

3. Nodular
   - Nodular malignant melanoma has a uniform brown-black or blue-black color and may present as a smooth-surfaced nodule, elevated plaque with irregular outlines, or polypoid ulcerated tumor. It is more common on the trunk and extremities.
4. Acral Lentiginous Melanoma
   - Acral lentiginous melanoma is a rare type of melanoma that arises on the palms, soles, mucous membranes, and nail beds in patients of African or Asian descent. It is the most common form of oral melanoma. The prognosis tends to be worse than the other subtypes usually due to delays in diagnosis.

Prognosis

- The prognosis of melanoma, regardless of the subtype, is most closely linked to primary tumor thickness.
- Breslow depth is a measurement in millimeters of the tumor cell depth from the granular layer to the base of the melanoma (See Table 8.6). Surgical margins area is based on Breslow depth. For melanomas with a Breslow depth greater than 1–4 mm or with ulceration, a sentinel lymph node biopsy is recommended. If positive, a lymph node dissection is indicated. For depths greater than 4 mm, lymph node dissection should be considered.
- Clark's staging measures the depth based on skin anatomy, but is no longer used in melanoma staging.
- TNM system takes into account tumor thickness, ulceration, regional metastases, distant metastases, and serum lactate dehydrogenase (Table 8.7).
- The vast majority of cutaneous melanomas are treated with surgery alone where a 1 cm margin is adequate for tumors with a thickness of 1 mm or less.
- 2 cm margins are recommended for deep tumors.
- Lymph node dissection is performed on patients with clinically evident regional

Table 8.6 Breslow depths and recommended margins

Melanoma Breslow depth	Margin of resection
Melanoma-in-situ	0.5
≤1 mm	1
1.01–2.0 mm	1–2
>2 mm	2

Table 8.7 Melanoma TNM classification of cutaneous disease

T classification	**Thickness (mm)**	**Ulceration status/mitoses**
TX	Primary tumor cannot be assessed (for example, curettage or severely regressed)	Not applicable
T0	No evidence of primary tumor	Not applicable
Tis	Melanoma in situ	Not applicable
T1	Melanomas 1.0 mm or less in thickness	(a) Without ulceration and <0.8 /mm (b) With ulceration <0.8 and with or without ulceration 0.8–1 mm
T2	Melanomas >1.0–2.0 mm	(a) Without ulceration (b) With ulceration
T3	Melanomas >2.0–4.0 mm	(a) Without ulceration (b) With ulceration
T4	Melanomas more than 4.0 mm	(a) Without ulceration (b) With ulceration
N classification	**No. of metastatic nodes**	**Nodal metastatic mass**
NX	Patients in whom the regional nodes cannot be assessed (for example, previously removed for another reason)	
N0	No regional metastases detected	

Table 8.7 (continued)

N1	One node (one tumor involved node or in transit, satellite, and/or microsatellite metastasis with one tumor involved node.)	(a) One clinically occult node (i.e., detected by SLN biopsy). (b) One clinically detected (c) No regional lymph node disease
N2	Two–three nodes (two–three tumor involved nodes or in transit, satellite, and/or microsatellite metastasis with one tumor involved node.)	(a) Two or three clinically occult nodes (i.e., detected by SLN biopsy). (b) Two or three, at least one clinically detected. (c) One clinically occult or clinically detected.
N3	Four or more tumor-involved nodes or in transit, satellite, and/or microsatellite metastases with two or more tumor involved, or any number of matted nodes without or with in transit, satellite, and/or microsatellite metastases.	(a). Four or more clinically occult nodes (i.e., detected by SLN biopsy). (b). Four or more nodes, at least one clinically detected or presence of any number of matted nodes. (c). Two or more clinically occult or clinically detected and/or presence of any matted nodes.
M classification	**Site**	**Serum lactate dehydrogenase**
M0	No detectable evidence of distant metastases	NA
M1a	Metastases to skin, muscle, or non-regional lymph nodes	Normal
M1a(0)	Metastases to skin, muscle, or non-regional lymph nodes	Normal
M1a(1)	Metastases to skin, muscle, or non-regional lymph nodes	LDH elevated
M1b	Metastases to lung, with or without M1a sites of disease	Normal
M1b(0)	Metastases to lung, with or without M1a sites of disease	Normal
M1b1	Metastases to lung, with or without M1a sites of disease	LDH elevated
M1c	Metastases to non CNS visceral sites with or without M1a or M1b sites of disease	Normal
sM1c(0)	Metastases to non CNS visceral sites with or without M1a or M1b sites of disease	Normal
M1c(1)	Metastases to non CNS visceral sites with or without M1a or M1b sites of disease	LDH elevated

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metastasis, and radiation therapy is used for tumors with a high suspicion of recurrence. Due to a recent discovery that up to 50% of cutaneous melanomas have BRAF mutations, *BRAF* inhibitor treatments, namely vemurafenib and ipilimumab, are approved for the treatment of metastatic melanoma.

Mucosal Melanoma

- Head and neck primary mucosal melanoma (PMM) is a rare and aggressive malignancy, 1% of all melanomas and 10% of all head and neck melanomas [14].
- It is important to mention that, due to their rarity, PMMs are poorly understood. If PMM is suspected, it is mandatory to rule out metastatic disease.
- Head and neck PMMs arise mainly in the oral cavity, nasal cavity, and paranasal sinuses but can also occur in the pharynx and larynx.
- These tumors develop primarily in the fifth to eighth decade of life.
- Unlike their cutaneous counterpart, PMMs have no association with UV exposure, but inhaled and ingested carcinogens such as smoking and formaldehyde may play a role in pathogenesis. Many report an equal sex distri-

bution, but some series show a slight male predilection.
- Sinonasal PMM presents as many other sinonasal diseases with nasal obstruction, facial pain, persistent rhinorrhea, or epistaxis.
- CT and MRI are used to define the extent of the tumor.
- PMM can have a variety of radiographic appearances from an enhancing, expansile, benign-appearing mass to a destructive mass. PET/CT is used for staging.
- A chest X-ray is also taken to rule out metastatic disease.
- PMMs of the larynx and pharynx are very rare, and the most common presenting symptom is hoarseness or symptoms similar to sinonasal tumors.
- Oral mucosal melanoma (OMM) usually presents as widespread or multiple pigmented lesions with ill-defined borders; although, depigmented/amelanotic lesions are seen in up to 10% of cases.
- Depigmented lesions will appear as macules or lobulated, exophytic masses with normal mucosal color or vascular appearance. Necrosis, ulceration, and tissue invasion are also commonly noted, though patients are usually asymptomatic.
- Acral lentiginous melanoma is the most common form of OMM.
- The most common locations for OMM are the maxillary alveolar ridge and hard palate.
- The bone adjacent to the involved mucosa may demonstrate a "moth-eaten", irregular, and destructive resorption pattern on radiography.
- The "ABCDE" checklist described in the cutaneous melanoma section may be useful in distinguishing these lesions from racial pigmentation, amalgam tattoo, Peutz-Jeghers syndrome, and Addison disease.
- The risk of cervical lymph node involvement is higher in OMM than other mucosal melanomas, and 25% of patients with OMM present with lymph node metastasis.
- Commonly accompanied by satellite lesions that are in adjacent tissues but without direction connection.

Table 8.8 Mucosal melanoma of the head and neck TNM classification

Primary tumor (T)	
T3	Mucosal disease. Tumor is limited to mucosa and immediate underlying soft tissue, regardless of thickness of greatest dimension: for example, polypoid nasal disease, pigmented or non-pigmented lesion of the oral cavity, pharynx or larynx.
T4	Moderately advanced or very advanced.
T4a	Moderately advanced disease. Tumor invading deep soft tissue, cartilage, bone, or overlying skin
T4b	Very advanced disease. Tumor involving brain, dura, skull base, lower cranial nerves (IX,X,XI,XII), masticator space, carotid artery, prevertebral space, or mediastinal structures
Regional lymph nodes (N)	
NX	Regional nodes cannot be assessed
N0	No regional lymph node metastases detected
N1	Regional lymph node metastases present
Distant metastases (M)	
M0	No distant metastases
M1	Distant metastases

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- Currently, the 8th edition AJCC TNM classification is the most widely accepted staging system for mucosal melanomas (Table 8.8). This classification uses anatomic extent of the tumor, regional lymph node metastases, and distant metastases to stage PMM of the head and neck. Because of their aggressive behavior, head and neck PMMs are classified as a minimum of stage III. It should be noted that Tx-T2 are not applicable.

Treatment

- Complete surgical resection with wide dissection of 3 cm with PMM [15].
- Lymph node dissection is performed on patients with evident regional metastasis. In the absence of clinically evident nodes, sentinel lymph node biopsy is useful to identify patients who would benefit from elective neck dissection.
- Radiation therapy is mainly used when the patient is a poor surgical candidate or margins are inadequate. Indications for radiotherapy

are ill-defined, but it is currently used only in advanced or recurrent disease.
- Chemotherapy is offered mainly as palliative care. Appears to have no increase in 5-year survival [14].

Basal Cell Carcinoma (BCC)

- The most common cancer that rarely causes death or results in metastasis. BCC can be locally aggressive or destructive causing significant morbidity.
- Commonly seen in males >40 years of age.
- Approximately 80% of BCCs occur on the head and neck with most cases on the face. Oral BCC has been reported rarely, but most cases are likely misdiagnosed odontogenic or salivary gland tumors.
- Risk factors:
  - UV radiation is the largest risk factor, in particular, recreational exposure to the sun during childhood and adolescence.
  - Fair complexion, red or blonde hair, and light eye color.
  - Immunosuppression.
  - Radiotherapy treatment.
  - Exposure to arsenic or tar.
- Molecular pathogenesis
  - Results from inappropriate activation of the sonic hedgehog signaling pathway. Sonic hedgehog proteins bind to and inactivate the PTCH1 tumor suppressor gene on chromosome 9q22. Germline mutations of PTCH1 are found in patients with nevoid basal cell carcinoma (Gorlin) syndrome. Gorlin syndrome is an autosomal dominant disorder, but around 40% of cases represent a new mutation.

BCC Has Three Main Subtypes

1. Nodular
   - The most common subtype with lesions appearing as shiny or pearlescent papules or nodules with telangiectasias. There may be crusting over a central depression or ulceration with a rolled border, sometimes referred to as a rodent ulcer. The differential diagnosis for a non-ulcerated nodular BCC includes molluscum contagiosum, sebaceous hyperplasia, amelanotic melanoma, and intradermal nevus. Ulcerated lesions are clinically similar to squamous cell carcinoma and keratoacanthoma.
2. Superficial
   - The second most common subtype. These lesions are well-circumscribed, scaly, pink-red macules or plaques with a crust or thin rolled border. They are found most commonly on the trunk and extremities and clinically may resemble psoriasis or early amelanotic melanoma. Least aggressive form of BCC.
3. Morpheaform
   - The rarest type representing 5–10% of all BCCs. They present as indurated plaques that are pink-to-white and shiny with ill-defined borders. They may be difficult to identify as they clinically resemble scars, and they tend to behave more aggressively than the other subtypes.

Staging

Basal cell carcinoma is staged using the AJCC cancer staging for cutaneous squamous cell carcinoma and other cutaneous carcinomas (Table 8.9). High-risk features are a tumor thicker than 2 millimeters, a tumor that has invaded down into the lower dermis or subcutis, a tumor that demonstrates perineural invasion, a primary tumor of the ear or hair-bearing lip, and a microscopically poorly differentiated or undifferentiated tumor.

Treatment [1, 15]

- The goal of treatment is complete removal of the tumor.

Table 8.9 AJCC 8th edition TNM classification of cutaneous squamous cell carcinoma and other cutaneous carcinomas

Primary tumor (T)	
TX	Primary tumor cannot be assessed
Tis	Carcinoma in situ
T1	Tumor 2 cm or less in greatest dimension
T2	Tumor ≥2 cm and <4 cm in greatest dimension.
T3	Tumor ≥4 cm in greatest dimension and/or perineal invasion and/or deep invasion and/or minor bone erosion.
T4a	Tumor with gross cortical bone/marrow invasion
T4b	Tumor with skull base invasion and/or skull base foramen involvement.
Regional lymph nodes (N)	
NX	Regional nodes cannot be assessed
N0	No regional lymph node metastases
N1	Metastasis in a single ipsilateral lymph node, ≤3 cm in greatest dimension and ENE (−).
N2a	Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension and ENE (−)
N2b	N2b Metastasis in multiple ipsilateral lymph node, none >6 cm in greatest dimension and ENE (−)
N2c	N2c Metastasis in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension and ENE (−)
N3a	N3a Metastasis in a lymph node >6 cm in greatest dimension and ENE (−)
N3b	N3b Metastasis in a single ipsilateral node >3 cm in greatest dimension and ENE (+); or multiple ipsilateral, contralateral, or bilateral nodes, any with ENE (+); or a single contralateral node ≤3 cm and ENE (+)
Distant metastases (M)	
Mx	Distant metastasis cannot be assessed
M0	No distant metastases
M1	Distant metastases

ENE extranodal extension
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- Recurrence and metastases are rare, so local control is most important.
- Tumors at low risk for recurrence are treated with electrodessication and curettage or surgical excision.
- High-risk features for BCC include:
  - Lesions of 6 mm or more in the "mask" area of the face (skin of the central face, eyelids, eyebrows, periorbital, nose, lips, chin, mandible, auricular region, and temple).
  - Lesions more than 1 cm in head and neck sites other than the "mask".
  - Lesions with poorly defined clinical borders.
  - Lesions with perineural invasion.
  - Recurrent lesions.
  - Lesions in immunosuppressed patients.
  - Lesions that have an aggressive histological growth pattern.
  - Lesions in a site of prior radiotherapy.
- Lesions with a high risk for recurrence or that are in sites where tissue conservation is crucial are often treated with Mohs surgery.
- Radiation may be used for patients unable to have surgery.

Squamous Cell Carcinoma

Relevant Anatomy

- The oral cavity is located in the most rostral portion of the gastrointestinal tract and extends from the vermillion border of the lip anteriorly to the hard and soft palate junction/circumvallate papillae posteriorly.
- The subsites of oral cavity include:
  1. Anterior two-thirds of the tongue
  2. Floor of mouth
  3. Buccal mucosa
  4. Retromolar trigone
  5. Hard palate
  6. Lips
  7. Maxillary gingiva
  8. Mandibular gingiva
- Primary lymphatic drainage of the oral cavity is to the upper cervical lymph nodes including level 1a (submental nodes), level 1b (submandibular nodes), level 2 (upper jugular nodes), and level 3 (middle jugular nodes). Up to 15% of tongue carcinomas can have "skip metastasis" to level 4 (lower jugular nodes) without involving levels 1, 2, and 3 [16]. This predictable pattern of lymphatic drainage has led to a type of selective lymphadenectomy called "supraomohyoid neck dissection", which includes the removal of lymph node levels 1–3. Some surgeons routinely include level 4

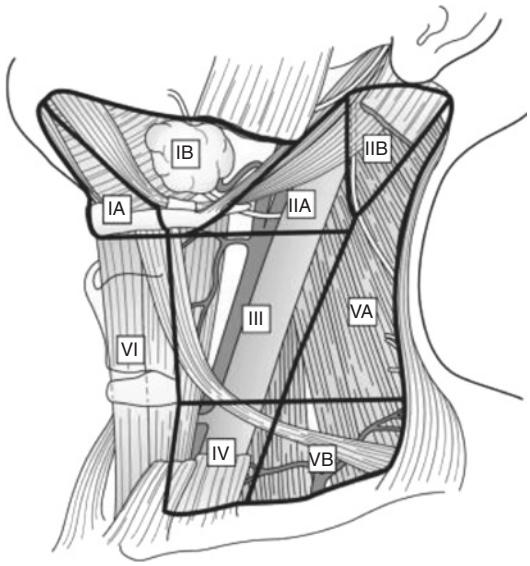

Fig. 8.5 Levels and sublevels of the neck. (Reprinted with permission from Cheng and Schmit [21])

as well for tongue carcinomas due to the possibility of skip metastasis (See Fig. 8.5 for levels of neck).

- The oropharynx is bounded anteriorly by the oral cavity, superiorly by the nasopharynx, and inferiorly by the hypopharynx.
- The subsites of oropharynx include:
  1. Soft palate.
  2. Tonsils and tonsillar pillars (palatoglossus and palatopharyngeus muscles).
  3. Base of tongue (behind the circumvallate papillae).
  4. Posterior pharyngeal wall.
- The oropharynx is primarily drained by the jugular lymph node chain in levels 2, 3, and 4 in addition to retropharyngeal and parapharyngeal nodes. The soft palate, posterior pharyngeal wall, and base of tongue subsites are known to have rich bilateral lymphatic drainage.
- The understanding of cervical nodal groups is essential in the management of head and neck cancer patients. It defines the extent of cervical lymphadenectomy (neck dissection) or radiation field, facilitates communication among clinicians, and has prognostic implications (the involvement of levels 4–5 results in a worse outcome than levels 1–2).
- In general, the first echelon nodes for the oral cavity SCC are levels 1, 2, and 3, meaning that these levels are the first lymph node groups that cancer cells travel through before reaching lower lymphatic chains in levels 4 and 5 with the exception of some tongue SCC.
- Level 1a (submental lymph node group) is a triangular region bounded superiorly by the mandible, bilaterally by the anterior belly of the digastric muscle, and inferiorly by the hyoid bone. The mylohyoid muscle forms the floor of level 1a.
- Level 1b (submandibular lymph node group) is a triangular region bounded superiorly by the mandible, anteriorly by the anterior belly of the digastric muscle, posteriorly by the stylohyoid/posterior belly of the digastric muscle, and inferiorly by the hyoid bone. The mylohyoid muscle and hyoglossus muscle form the floor of level 1b.
- Level 2 includes the upper jugular lymphatic chain and is bound superiorly by the skull base, anteriorly by the sternohyoid muscle, posteriorly by the posterior border of the sternocleidomastoid muscle (SCM), and inferiorly by the carotid bifurcation (radiographically this level coincides approximately with the lower border of the hyoid bone).
- Level 2 is divided into level 2a and level 2b. Level 2a includes the nodes anterior to the spinal accessory nerve (CN 11) and level 2b (submuscular recess above the splenius capitis and levator scapulae fascia) includes the nodes posterior to CN11.
- Of note, there is a controversy regarding the removal of level 2b for clinically node-negative neck in oral cavity SCC due to a small percentage of level 2b involvement (6%) without the involvement of other levels and dreaded complication of shoulder dysfunction and pain syndrome from dissection of CN 11. Most surgeons, however, include level 2b in neck dissection.
- Level 3 includes the middle jugular lymphatic chain extending from the carotid bifurcation

above to the omohyoid muscle below (radiographically, this level coincides approximately with the lower border of the cricoid cartilage). It is bounded anteriorly by the sternohyoid muscle and posteriorly by the posterior border of SCM.
- Level 4 includes the lower jugular lymphatic chain extending from the omohyoid muscle above to the clavicle below. Again, it is bounded anteriorly by the sternohyoid muscle and posteriorly by the posterior border of SCM.
- Level 5 is the posterior triangle of the neck bound by the posterior border of SCM, the anterior border of the trapezius muscle, and the clavicle. Level 5 is divided by the horizontal line at the level of the lower border of the cricoid cartilage into level 5a around CN 11 (accessory chain) and level 5b (transverse cervical and supraclavicular chain). Of note, level 5 is rarely involved in oral cavity SCC and is not included in neck dissection for clinically node-negative neck.
- Level 6 is the central compartment nodal group bound bilaterally by the carotid artery, superiorly by the hyoid bone, and inferiorly by the sternal notch. Level 6 includes the pretracheal, paratracheal, recurrent laryngeal, and precricoid (delphian) nodes. Level 6 drains the thyroid gland, glottic and subglottic larynx, cervical esophagus, and apex of pyriform sinuses and is not included in neck dissection for oral cavity SCC.

Background of SCC

- The most common type of malignancy of the oral cavity is squamous cell carcinoma (SCC). SCC arises from the lining cells of the oral cavity, which are frequently exposed to carcinogens such as tobacco, alcohol, and betel nut. This explains why more than 90% of oral cavity cancers are SCC. Other malignant tumors of the oral cavity include minor salivary gland cancers, sarcomas, mucosal melanomas, and lymphoma.
- In the United States, the lateral border of the tongue is the most common subsite of oral cancer followed by the floor of the mouth. Buccal mucosa and retromolar trigone subsites are next in frequency.
- Carcinogens cause cancer by DNA mutations in the regions of proto-oncogenes and/or tumor suppressor genes. Cumulative genetic mutations create cancer cells, which by definition have an uncontrollable growth potential, and the ability to invade surrounding tissue and metastasize to distant sites.
- Major risk factors for oral cavity SCC include tobacco (initiator) and alcohol (promoter). As a risk factor for developing SCC, tobacco increases the risk 5–9 fold, and alcohol increases the risk 3–9 fold. Combined use of tobacco and alcohol has a synergistic effect rather than an additive effect, estimated as high as a 100-fold risk increase [17, 18].
- In Southeast Asia and India, the use of betel nut products is implicated in the high prevalence of oral submucous fibrosis and oral cavity SCC. Excessive sun exposure can lead to SCC of the lips (lower lip>>upper lip by 9:1).
- Some studies have implicated poor oral hygiene; chronic mechanical trauma such as from ill-fitting dentures; vitamin A, C, or E deficiency; and a number of infectious agents including Candida species and syphilis as risk factors for oral cancer.
- Pathologic or pharmacologic immunosuppression such as in patients with Acquired Immune Deficiency Syndrome (AIDS) or history of organ transplantation increases the risk of cancer.
- Moreover, there are various genetic conditions that can cause oral SCC including Li-Fraumeni syndrome, Fanconi anemia, dyskeratosis congenita, etc.
- Of note, oropharyngeal cancer is a completely different entity from oral cavity cancer in terms of anatomical location and biological/clinical behavior.
- Human papillomavirus (HPV) causes more than 80% of oropharyngeal SCC, as opposed to 3–5% in oral cavity cancers. This accounts

for a current rise in the number of oropharyngeal cancer in young male patients.
- HPV positive tumors generally have improved response to radiation and improved survival when compared with HPV negative tumors. Thus, the status of HPV carries both therapeutic and prognostic implications in the management of these tumors, meaning less dose of radiation may be used in these patients with comparable cure rates and less radiation toxicity. However, currently there is no consensus on deintensification of treatment regimens in these patients and further research is needed on how HPV status can be translated into clinical practice.

Clinical Presentation

- Oral SCC patients can be asymptomatic and diagnosed with SCC as an incidental finding on dental/oral examination especially in the early stages (stages 1 and 2). Other symptoms include pain, numbness, swelling or lump inside the mouth or in the neck, bleeding, ill-fitting dentures, dysphagia, odynophagia, dysphonia, loose teeth, change in voice (either from the direct invasion of the laryngeal structure or from the recurrent laryngeal nerve involvement), trismus (from invasion into the masticator space) and ear pain (from invasion of the lingual nerve (CN5) in the oral cavity or the glossopharyngeal (CN9)/vagus nerve (CN10) in the oropharynx).
- In very advanced stages, patients can present with cachexia (wasting syndrome accompanied by severe weight loss, muscle atrophy, loss of appetite, and fatigue).
- Oral SCC can present with an exophytic mass or an endophytic ulceration. In addition, oral SCC can present with a non-ulcerated surface as a white patch (leukoplakia), a red patch (erythroplakia), or a mixed white-red patch (erythroleukoplakia). In advanced stages (stages 3 and 4), patients typically present with palpable cervical lymph nodes.
- The most common sites of distant metastasis include the lungs, bone, and liver.
- Patients may develop oral SCC from premalignant lesions such as leukoplakia, erythroplakia, or oral lichen planus.
  - Leukoplakia is the most common premalignant lesion, defined as "a white patch or plaque that cannot be characterized clinically or pathologically as any other disease," and must be removed or followed closely based on pathologic findings. Any dysplastic findings warrant complete removal of the lesion.
  - Proliferative verrucous leukoplakia (PVL) is a variant of leukoplakia with multifocal corrugated/papillomatous lesions of the oral cavity with a high recurrence rate. It is generally not associated with smoking habits. Most PVL will go on to develop oral SCC or verrucous carcinoma.
  - Erythroplakia is defined as "a red patch or plaque that cannot be characterized clinically or pathologically as any other disease," and must be excised completely with adequate margins due to much higher malignancy potential than leukoplakia (The redness of the lesion is due to the atrophic epithelium lacking keratin and thereby allowing the underlying vasculature to show through and produce a red appearance [1]). Almost all erythroplakia lesions demonstrate dysplasia, carcinoma-in-situ, or invasive SCC.
  - Oral lichen planus is a mucocutaneous disease characterized by T-cell mediated degeneration of the basal cell layer with low malignant potential. Erosive and atrophic subtypes are considered premalignant and warrant long-term follow-ups and biopsy with any change of symptoms or signs.
  - Oral submucous fibrosis is a premalignant condition with progressive fibrosis of the submucosal (lamina propria) layer and atrophy of the overlying epithelium of the oral cavity resulting in severe trismus, associated with areca and betel quid chewing commonly in Southeast Asia and India. Periodic follow-ups are necessary.

Oral SCC Work-up

- Chief complaint in patient's own words, e.g., "I have a white spot on my tongue."
- History of present illness (HPI) /ROS
  - Problem-focused and detailed exploration into patient's chief complaint
  - Location, quality, onset, duration, severity, exacerbating or alleviating factors, and associated symptoms described above in the clinical presentation section.
  - Depression among these patients is quite common and should be dealt with seriously. Referral to a psychologist or psychiatrist may be necessary.
- Past Medical History/Past Surgical History
  - History of previous head and neck cancer and radiation therapy/surgical therapy should be obtained. These patients are prone to recurrent cancer or second primary cancer due to "field cancerization" of the upper and lower aerodigestive tract (carcinogens result in cumulative DNA mutations in the entire lining of this tract). This information can alter the treatment decision.
  - Significant medical comorbidities (especially cardiopulmonary diseases) are common in this population due to the age and smoking/alcohol habits and can affect the choice of treatment modality. Non-surgical candidates can be treated with radiation therapy with or without chemotherapy.
  - Perioperative medical and nutritional optimization is usually necessary.
  - Functional status of the patient is an important piece of information that determines the eligibility for chemotherapy. Various scoring systems such as Karnofsky, ECOG/WHO/Zubrod, and Lansky scoring systems are generally used.
  - If vascularized microvascular flap reconstruction is contemplated, then history of trauma or surgery to the donor site should be questioned.
- Medications/Allergy
- Social history pertinent to oral SCC patients includes smoking pack-year history, tobacco chewing/dipping, alcohol/drug use, and betel nut chewing (patients from Southeast Asia or India). Family support is another important factor in the care of oral SCC patients.
- Family history of hereditary conditions such as Fanconi anemia or dyskeratosis congenital that can lead to oral SCC development should be sought.
- Physical Examination
  - Vital signs and body weight measurement (5% weight loss over the prior 1 month or 10% weight loss over 6 months should prompt consideration for prophylactic feeding tube placement.)
  - A focused head and neck examination is mandatory for these patients in addition to generalized physical examination.
  - Any signs of respiratory distress from compromised airway such as rapid shallow breathing, sniffing position or drooling should be noted.
  - Approximately 30% of patients present with regional node involvement at presentation. Visual inspection and palpation of the neck is performed in a systematic fashion to examine the cervical lymph nodes. Painless, hard, matted, fixed, and/or greater than 1–1.5 cm lymph nodes should be considered cancerous until proven otherwise.
  - Intraoral examination should include visual inspection and palpation of the lesion or mass and describe the color, texture, consistency, fixation, size, endophytic vs. exophytic nature, and tenderness. Also, the anatomic extent of the lesion should be described.
  - The cranial nerves V, VII, X, XI, and XII should be examined.
  - In-office flexible fiberoptic nasopharyngoscopy may be performed for assessment of extent of disease and identification of second primary. Also,

panendoscopy (laryngoscopy, bronchoscopy, and esophagoscopy) is usually performed at the time of surgery for the same purpose.
  - Complete dental evaluation is mandatory. Any non-restorable teeth should be extracted at least 2 weeks before the commencement of radiation therapy. Active dental caries and periodontal disease should be treated aggressively to eliminate the source of infection and to decrease the risk of osteoradionecrosis (ORN).
- Incisional biopsy is mandatory to establish a definitive diagnosis. Any suspicious lesion persisting for more than 2 weeks after elimination of potential source of trauma should be biopsied in order to rule out malignancy. Early detection of oral cancer is crucial in improving the prognosis.
- Radiographic images (See Table 8.10).

Imaging Studies

Labs

- CBC
- Chem-10
- PTT/INR
- Liver function test (LFT) is ordered in addition to routine preoperative blood work as an initial screening test to rule out liver metastasis. Upon abnormal findings, CT of the abdomen is ordered for confirmation of distant metastasis.

Oral SCC Staging/TNM Classification

- Currently, oral cavity SCC is staged based on the tumor, node, metastasis (TNM) staging system by the American Joint Committee for Cancer (AJCC).

Table 8.10 Radiographic imaging in workup for SCC

Dental radiography	Radiographs are obtained to evaluate dentition and bony involvement by SCC. These are two-dimensional films and can show bony erosion/invasion only of the alveolar process but not of the buccal or lingual cortices. Approximately 50% of demineralization is required for radiolucency to be discernible on the X-ray and, thus, the sensitivity of these studies is limited.
CT	Most commonly used imaging study for this purpose due to its better ability to show central necrosis, extracapsular spread, and bony detail (evaluate osseous invasion). Lymph nodes that are greater than 1.5 cm at levels I and II, >1 cm for level III and above are considered metastatic. Lymph nodes that have lost the typical oval shape or have become rounded with a necrotic center are more suspicious for harboring metastatic disease.
MRI	Better for perineural invasion. Concern for decreased nodal metastasis. Over estimation of bony invasion due to inflammatory response. MRI with contrast in T1 weighted image best imaging for tongue invasion.
PET-CT	Primary staging modality for head and neck cancer is controversial. Post-treatment evaluation after radiation therapy or chemoradiation therapy (recommended 3 months after completion of treatment) and for evaluation of unknown primary tumor. Does not differentiate increased metabolic activity due to inflammation or infection versus malignancy. Deoxy-glucose (FDG) is radiolabeled glucose molecules used as a radiotracer in PET scans. Any tissue with high metabolic rate has high standard uptake value (SUV) of FDG and lights up on PET scans. Thus, inflammation or physiologic uptake of muscle lowers the specificity of the study. Very high uptake of fluorodeoxyglucose (FDG), termed "FDG-avid", is suggestive of metastasis. Lymph nodes with an SUV greater than 2.5 are considered pathological.
CXR	Considered acceptable initial screening evaluation for distant metastasis or second primary cancer. With any abnormal findings on CXR, CT of the chest should be obtained. Lungs, Liver and bones are the most common distant metastatic sites for SCC.

- Nomenclature:
  - aTNM: Autopsy classification, performed post mortem and no evidence of cancer prior to death.
  - cTNM: A small case c is used as a prefix for clinical staging given by clinical examination of a patient. (For example, cN1 means a single suspicious ipsilateral node 3 cm or less in greatest dimension found on physical examination or imaging studies).
  - pTNM: A small case p is used as a prefix for pathologic staging given by histologic examination of the surgical specimen (For example, pN1 means a single positive ipsilateral node 3 cm or less in greatest dimension based on pathologic examination of cervical lymph nodes by a pathologist).
  - rTNM: Similarly, a prefix r is used for stage of a recurrent tumor.
  - 'm' suffix: More than one primary at a single site (pT(m)NM).
  - 'y' prefix: Classification performed during or after initial multimodality therapy (ycTNM or ypTNM).
- *T-stage* is determined by the surface dimension of the primary tumor and/or by the extent of invasion into the surrounding tissue. Depth of invasion (DOI) is measured by distance in millimeters from the "horizon" of the basement membrane (Table 8.11).
- N-stage is determined by the size, number, and laterality of the involved cervical lymph nodes.
- Of note, the nodal status is the single-most important prognostic factor for head and neck cancer. Stage I/II survival rate >80% over a 5 year rate. Having a single positive lymph node will reduce the prognosis by 50%.
- ENE – extranodal extension. Extension of metastatic carcinoma, within a lymph node, through the capsule, and into the surrounding connective tissue. This designation is given regardless of associated stromal reaction [13] (Table 8.12).
- M-stage is determined by the presence or absence of distant metastasis. Distant metastasis suggests an incurable disease and the patients are treated palliatively (Table 8.13).

Table 8.11 Tumor size classification according to the AJCC 8th edition for oral squamous cell carcinoma

T category	T criteria
Tx	Primary tumor cannot be assessed
Tis	Carcinoma in situ
T1	Tumor ≤2 cm with DOI ≤5 mm
T2	Tumor ≤2 cm with DOI >5 mm and ≤10 mm or tumor >2 cm and ≤4 cm with DOI ≤10 mm
T3	Tumor >4 cm or any tumor with a DOI > 10 mm but ≤20 mm
T4a	Moderately advanced local disease tumor invades adjacent structures only (e.g., through cortical bone of the mandible or maxilla or involves the maxillary sinus or skin of the face) or extensive with bilateral tongue involvement or DOI >20 mm Note: Superficial erosion of bone/tooth socket (alone) by a gingival primary is not sufficient to classify a tumor as T4.
T4b	Very advanced local disease. Tumor invades masticator space, pterygoid plates, or skull base and/or encases the internal carotid artery.

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Table 8.12 Regional lymph node invasion for TNM classification of oral squamous cell carcinoma

M category	M criteria
M0	No distant metastasis
M1	Distant metastasis.

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Table 8.13 Definition of distant metastasis of TNM classification of oral squamous cell carcinoma

N category	N criteria
Nx	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single ipsilateral lymph node, 3 cm or smaller in greatest dimension ENE(−)
N2	Metastasis in a single ipsilateral node larger than 3 cm but not larger than 6 cm in greatest dimension and ENE(−); *or* metastases in multiple ipsilateral lymph nodes, none larger than 6 cm in greatest dimension and ENE(−); *or* in bilateral or contralateral lymph nodes, none larger than 6 cm in greatest dimension, and ENE(−)

Table 8.13 (continued)

N category	N criteria
N2a	Metastasis in a single ipsilateral node larger than 3 cm but not larger than 6 cm in greatest dimension, and ENE(−)
N2b	Metastases in multiple ipsilateral nodes, none larger than 6 cm in greatest dimension, and ENE(−)
N2c	Metastases in bilateral or contralateral lymph nodes, none larger than 6 cm in greatest dimension, and ENE(−)
N3	Metastasis in a lymph node larger than 6 cm in greatest dimension and ENE(−); *or* metastasis in any node(s) and clinically overt ENE(+)
N3a	Metastasis in a lymph node larger than 6 cm in greatest dimension and ENE(−)
N3b	Metastasis in any node(s) and clinically overt ENE(+)

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- Anatomic Stage/Prognostic Groups:
  - Stage 0: Tis N0 M0
  - Stage 1: T1 N0 M0
  - Stage 2: T2 N0 M0
  - Stage 3: T3 N0 M0, T1,2,3 N1 M0
  - Stage 4a: T4a N0,1 M0, T1,2,3,4a N2 M0
  - Stage 4b: T4b Any N M0
  - Stage 4c: Any T Any N M1
- In general, early stage 1 or 2 disease defines a relatively small primary tumor (T1 or T2) without any nodal involvement or distant metastasis (N0M0).
- Advanced stage 3 or 4 cancers include larger primary tumors (T3 or T4) with metastases to regional nodes or distant sites.

Management of SCC

- Early stage oral SCC (stages 1 and 2) is usually treated with either surgery or radiation therapy alone. The survival rates from each treatment modality are comparable.
- However, surgery is generally preferred to radiation therapy for several reasons. Surgery provides a surgical specimen that pathologists can evaluate and provide meaningful information such as the depth of invasion, histological grade, and adverse pathologic features of perineural invasion, lymphovascular invasion, extracapsular spread or multiple node involvement. This information demonstrates the degree of aggressiveness of the tumor and can be used to help predict the disease course and to guide further management with adjuvant radiation ± chemotherapy.
- In contrast, since radiation therapy does not provide a specimen, much useful information is lost. Secondly, radiation therapy is associated with not only tumor eradication but also normal tissue injury and scarring. As a result, surgery becomes much more difficult in cases of recurrent or persistent cancer. Lastly, the oral cavity is particularly at risk for post-radiation complications such as xerostomia and osteoradionecrosis. Thus, unless the patient's medical comorbidities preclude surgery, surgery remains the preferred treatment modality for early stage oral cancer.
- Surgery involves resection of primary tumor including a margin of normal tissue. The generally accepted pathologically negative margin is >5 mm, but due to tissue shrinkage, intraoperative clinical circumferential margins measured and excised by the surgeon are 1–1.5 cm.
- Due to anatomical constraints, this margin may not be achievable (for example, tumor approaching the mandibular bone or the internal jugular vein (IJV)). In these cases, the next uninvolved anatomic barrier (the mandibular bone or IJV) should be removed. For example, marginal mandibulectomy is performed for a tumor abutting the mandible. If cancer invades into the marrow space of the mandible, segmental mandibulectomy should be performed with at least 1 cm normal bony margins.
- Advanced stage oral cancer (Stages 3 and 4): Often, surgery is the initial treatment modality followed by adjuvant radiation therapy ± chemotherapy.
- Upon initial evaluation and diagnosis of oral SCC, the patient is presented at a multidisciplinary tumor board where surgical oncologists, radiation oncologists, medical oncologists, pathologists, social workers,

radiologists, and a dietician discuss the optimal treatment strategy for each individual patient.

- Indications for adjuvant radiation (the National Comprehensive Cancer Network (NCCN) guidelines) include T3/T4 tumors, positive or close (<5 mm) resection margins, 2 or more cervical lymph nodes containing metastatic cancer, perineural or lymphovascular invasion, extracapsular spread, and T3/T4 tumors. Adjuvant radiation should be given within 6 weeks after surgery based on the studies that showed poorer outcome with delayed treatment.
- Potential complications of radiation therapy include radiation mucositis, dermatitis, trismus, dysphagia, xerostomia, osteoradionecrosis, and dental caries. With intensity-modulated radiotherapy (IMRT), radiation dose to the critical organs such as salivary glands, pharyngeal constrictor muscles, and mandible is reduced with the achievement of similar rates of tumor control, thus, lowering the incidence of radiation toxicities.
- Indications for adjuvant chemotherapy include positive resection margins and extracapsular spread based on the US intergroup trial (RTOG 9511) and the European trial (EORTC 22931) that showed improved survival outcome and locoregional control with adjuvant chemoradiation therapy with Cisplatin for these two adverse features [19]. Chemotherapy for head and neck cancer is used to sensitize cancer cells to the effects of radiation.
- Cisplatin is the most commonly used and favored chemotherapeutic agent for head and neck cancers, given weekly or every 3 weeks. Side effects of cisplatin include ototoxicity, nephrotoxicity, peripheral neuropathy, and gastrointestinal toxicity. Carboplatin may be used for patients with renal insufficiency.
- Cetuximab (monoclonal antibody targeting the endothelial growth factor receptor (EGFR)) is an FDA-approved alternative used for patients that cannot tolerate Cisplatin therapy.
- Radiation with or without chemotherapy can be chosen as primary treatment modality if the tumor involves vital anatomic structures or if the patient is not medically fit to undergo surgery.
- Upon completion of treatment, the primary oncologic surgeon periodically follows up patients. Because over 80% of cancer recurrences are detected within the first 24 months following initial treatment, patients are generally seen every 1–3 months for the first 2 years, though institutional variation does exist. After 2 years, patients are seen every 4–6 months.
- Tumor surveillance also includes periodic LFT and imaging studies such as CXR, CT scans, and PET scans to help detect locoregional recurrence, distant metastasis, or second primary cancer.

Management of the Neck

- Most oral SCC patients benefit from some form of cervical lymphadenectomy (neck dissection).
- Patients with clinically negative neck (N0 neck) with >20% chance of having occult regional metastasis (clinically and radiographically unidentifiable cancerous node due to small quantity of cancer cells present in the node) receive selective neck dissection (SND) [20]. In SND, only the nodal groups determined to be at highest risk for metastasis (first echelon nodes) are removed. If <20%, the morbidity from neck dissection such as shoulder dysfunction is considered to exceed the benefit gained from it.
- For oral tongue SCC with depth of invasion >4 mm is believed to have >20% chance of occult regional metastasis and warrants selective neck dissection for N0 neck. If the lesion approaches within 1 cm of the midline, bilateral neck dissection is warranted [21].
- For oral cavity SCC, SND involves the removal of lymph node levels 1–3 and is called "supraomohyoid neck dissection (SOHND)." As described before, level 4 is often included for tongue carcinomas due to skip metastasis.

- N0 oropharyngeal SCC requires removal of levels 2–4 (lateral neck dissection), if surgery is selected as primary treatment. SND can be therapeutic for a limited neck disease [22]. Locoregional control rates for SND are comparable to MRND for N0. SND is also useful for staging of the neck in the clinically N0 neck and guides further adjuvant therapy (see indications of adjuvant therapy below).
- Patients with nodal positive neck (N+ neck) generally receive comprehensive neck dissection with removal of level 1–5 lymph nodes [23].
- Comprehensive neck dissection is classified into radical neck dissection (RND) and modified radical neck dissection (MRND) and extended neck dissection (END) [24].
- MRND is further classified into three types depending on which non-lymphatic anatomic structures are preserved.
  1. RND: Radical en bloc lymphadenectomy including levels 1–5 along with SCM, IJV, and CN 11. All other types of neck dissection are derived from RND. RND is currently reserved for extensive cervical involvement (N3) or matted lymph nodes with gross extracapsular spread and invasion into the SCM, IJV, or SCM.
  2. MRND: Same as RND, except preservation of one or more non-lymphatic structures (SCM, IJV, and/or CN 11) not invaded by cancer. The rationale behind MRND is to reduce shoulder dysfunction, improve cosmesis, and reduce the likelihood of bilateral IJV resection in case of contralateral neck involvement (10% mortality with bilateral resection of IJV).
     - Medina classification:
       - MRND Type 1: CN 11 preserved.
       - MRND Type 2: CN 11, IJV preserved
       - MRND Type 3: CN 11, IJV, and SCM preserved.
  3. Extended neck dissection: RND or MRND in addition to removal of traditionally preserved anatomic structures such as the carotid artery, hypoglossal nerve, or level 6 or 7 nodes. END is reserved for cancer spread into these extended locations.
- Sentinel lymph node biopsy (SLNB) for oral SCC is not widely accepted. SLNB involves removal and intraoperative histologic evaluation of the first echelon lymph node. The procedure involves injection of radioactive tracer and blue-dye (isosulfan blue or toluidine blue) around the lesion and identification of the sentinel node with preoperative lymphoscintigraphy and intraoperative gamma probe. If metastatic cancer cells are found within the node, completion neck dissection is performed. If negative for metastasis, the patient can be spared possible complications from invasive neck dissection procedure. The sensitivity and specificity of SLNB are found to be >90% especially with serial sectioning at 150-micron sections of the specimen.

Surgical Technique for a Neck Dissection

- There have been several types of incisions described for various access, but the most common is a standard utility incision, which allows excellent exposure of levels 1 through 4 and can be well camouflaged within a neck crease (Fig. 8.6).
- When performing a neck dissection with the standard utility incision, the patient is placed in a supine position on the OR table with a shoulder roll to allow slight extension of the neck.
- The incision is marked from the mastoid process in a gentle curve over the mid-portion of the neck up to the contralateral submental region.
- This is then infiltrated with a local anesthetic with epinephrine for hemostasis.
- Incision is made with the scalpel through skin and subcutaneous tissues.
- Bovie electrocautery is used to dissect through the subcutaneous tissues exposing the platysma.
- Platysma is then sharply divided with care taken to preserve a clean edge of it for closure.

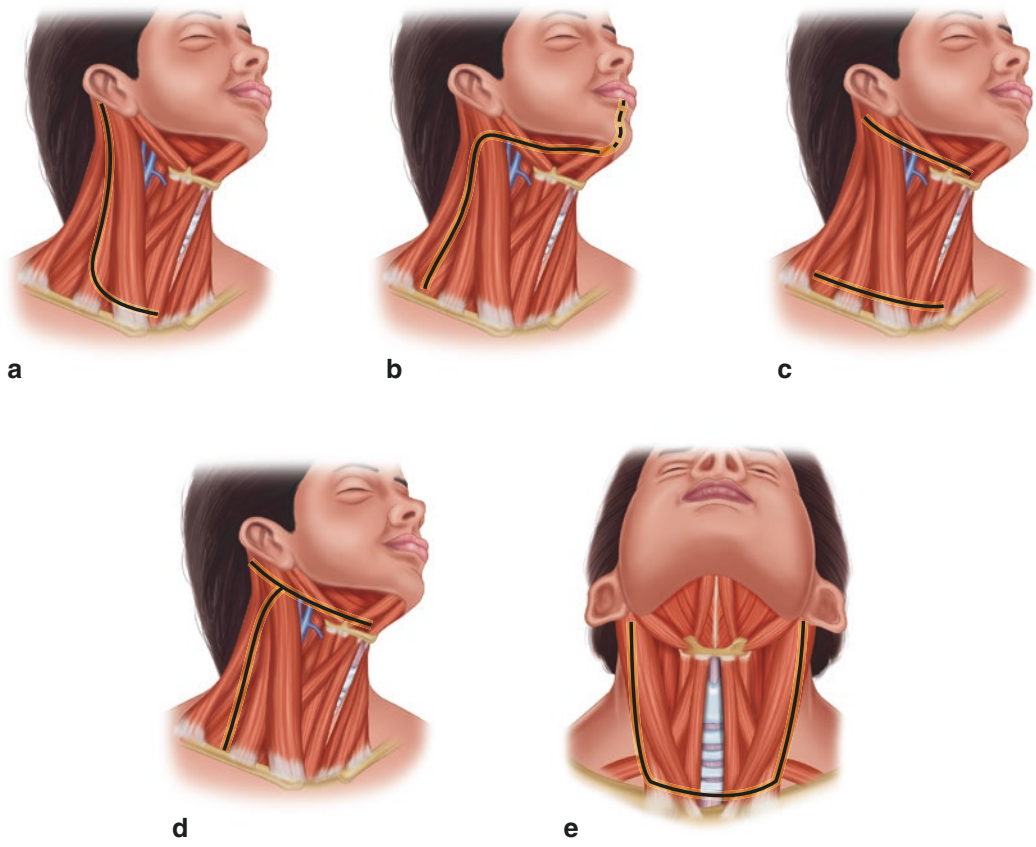

Fig. 8.6 Incisions for radical and modified radical neck dissections. A, Utility Incision. B, Boomerang. C, McFee. D, Modified Schobinger. E, Apron or bilateral hockey stick. (Reprinted with Permission from Robbins et al. [31])

- Subplatysmal flaps are elevated superiorly until the inferior border of the mandible and inferiorly until the clavicle. The external jugular vein and greater auricular nerve are found coursing transversely over the sternocleidomastoid from inferior to superior, and during elevation of the platysmal flap, the structure should be preserved. The first cranial nerve that is often encountered is the marginal mandibular branch of the facial nerve.
- Often, there are aggregates of lymph nodes in level IB that make protecting the nerve with a Hayes Martin maneuver not feasible. Therefore, the nerve should be identified with loupes and/or a nerve stimulator. Once it is identified, it can be retracted superiorly and protected during the remainder of the neck dissection.
- The fascia over the sternocleidomastoid muscle is divided.
- Working from a lateral to medial direction, this fascia is then elevated until the muscle can be retracted laterally exposing levels 2, 3, and 4.
- The spinal accessory nerve is then identified and dissected from the surrounding fascia to the level of the posterior belly of digastric.
- With this completed, the nerve can be retracted and the lymph node tissues from levels 2a and 2b can be dissected off the underlying fascia over the splenius capitis muscle. The dissection is then carried inferiorly staying medial to the transverse cervical nerves. This allows

adequate dissection of level III lymph nodes off of the internal jugular vein without violation of deeper structures such as the phrenic nerve or thoracic duct.
- The lymph nodes are then dissected off of the internal jugular vein, carotid artery and retracted medially. Care is taken to avoid injury to the vagus nerve. With that complete, the lymph nodes can be dissected from levels 1A and 1B.
- Starting with the contralateral anterior belly of digastric, level IA lymph nodes are sharply dissected from the mylohyoid muscle and digastric muscles. Care should be taken in this area as bleeding is often encountered from the submental branch of the facial artery.
- As the dissection progresses into level 1B, the posterior edge of the mylohyoid muscle is retracted anteriorly and the salivary gland is retracted out of the submandibular fossa. The lingual nerve is identified along with the submandibular ganglion and submandibular duct.
- The duct can be ligated and divided and the gland dissected from the fascial attachments in this area. Deep to the digastric muscle is the hypoglossal nerve and ranine veins. Care should be taken not to injure the hypoglossal nerve or damage these veins as this can result in troublesome bleeding.
- Once that is complete, the lymph node specimen can be elevated from the remaining fascial attachments and oriented for pathological evaluation. The neck is usually closed with 1–2 large suction drains.
- The platysma is closed and then the skin is closed in separate layers.

Complications of Neck Dissection

Neurological Dysfunction

- Spinal Accessory Nerve: Leads to "Shoulder Syndrome" due to loss of function of trapezius muscle. Symptoms include (1) pain, (2) limitation in arm abduction at shoulder, (3) winging of scapula, and (4) loss of contour of the shoulder due to muscle atrophy. Intentional on radical neck dissection and not a complication. Treatment entails physical therapy.
- Trunk of facial nerve: Temporary palsy reported as high as 58% in parotidectomy [25]. Managed best at time of transection with primary repair. Delayed treatment can involve cross face facial nerve, hypoglossal facial nerve crossover, or microneurovascular free tissue transfer. Static and dynamic slings from temporalis fascia or fascia lata can help with cosmesis but offer little function.
- Hypoglossal Nerve: Leads to difficulty with swallowing and speech. Tongue will deviate toward the affected side.
- Phrenic Nerve: Will cause ipsilateral elevation of diaphragm and is seen most often on postoperative chest radiograph. Signs include mediastinal shift, paradoxical movement of the lung, cardiac irritations (palpitations, tachycardia, or extrasystoles), and dyspnea [26]. Gastrointestinal symptoms include nausea, vomiting, and abdominal pain due to displacement of abdominal content.
- Vagus Nerve: Loss of sensation to tonsillar region, posterior 1/3 tongue, and pharynx. Can see diminished movement of soft palate and deviation of uvulae. Patients have difficulty swallowing and hence aspiration risk.

Thoracic Duct Injury: Primarily in left neck dissection. Can lead to large loss of plasma, triglycerides, fatty acids, electrolyte disturbances, reduction of immunocompetence due to depletion of circulating lymphocytes and lead to hypoalbuminemia [26–28]. Intraoperatively, after neck dissection, a request to anesthesia to observe for chyle egress (milky/creamy fluid) is important. If identified, over suturing the duct followed by a repeat valsalva maneuver to ensure resolution is performed. Lab testing of the fluid for a triacylglycerol level greater than 110 mg/dL (1.24 mmol/L) or the presence of chylomicrons is considered pathognomonic of chyle leak [29].

If identified post operatively, conservative management is done with pressure dressings, suction drains or negative wound pressure ther-

apy, bed rest, head of bed at 30 degrees or more, diet modification with a fat-free diet or medium-chain fatty acid diet, and use of broad-spectrum antibiotics. If no resolution within 14 days, surgical exploration is indicated [27]. Surgical intervention is recommended sooner for high output chyle leaks (>500 cc/day) for 7 days. At the time of surgery when the damaged duct is identified, it can be ligated, covered with a muscle flap or treated with a sclerosing agent [27].

Pharmacological management with Octreotide 100 μg subcutaneous every 8–12 hours to 200 μg subcutaneous every 8 hours is considered controversial. Side effects include nausea, vomiting, diarrhea, elevated liver enzymes, bradycardia, and interference with enteral nutrition uptake [27, 29].

Carotid Artery Blowout (See Fig. 8.7)**:** Risk is 3–4% of all patients who undergo radical neck dissection [30]. Prior radiotherapy increases risk 7-fold [26, 30]. Usually preceded by heralds or sentinel bleeds and may stop with minor packing or will cease spontaneously. If suspected, management initially includes securing a definitive airway, fluid resuscitation with large bore needles and pressure. Patients at risk who have heralds bleed should undergo angiography to identify bleeding sites, vessel aneurysm, or tumor blush [30]. If artery is exposed, immediate coverage with vascularized tissue is necessary [26].

In case of frank blow out, vessel ligation is initiated. This is done through an incision through the anterior border of the sternocleidomastoid. The sternocleidomastoid is retracted posteriorly exposing the carotid sheath that is then entered. The internal jugular vein is then retracted laterally, and after mobilization of the vagus nerve the common carotid is ligated [30].

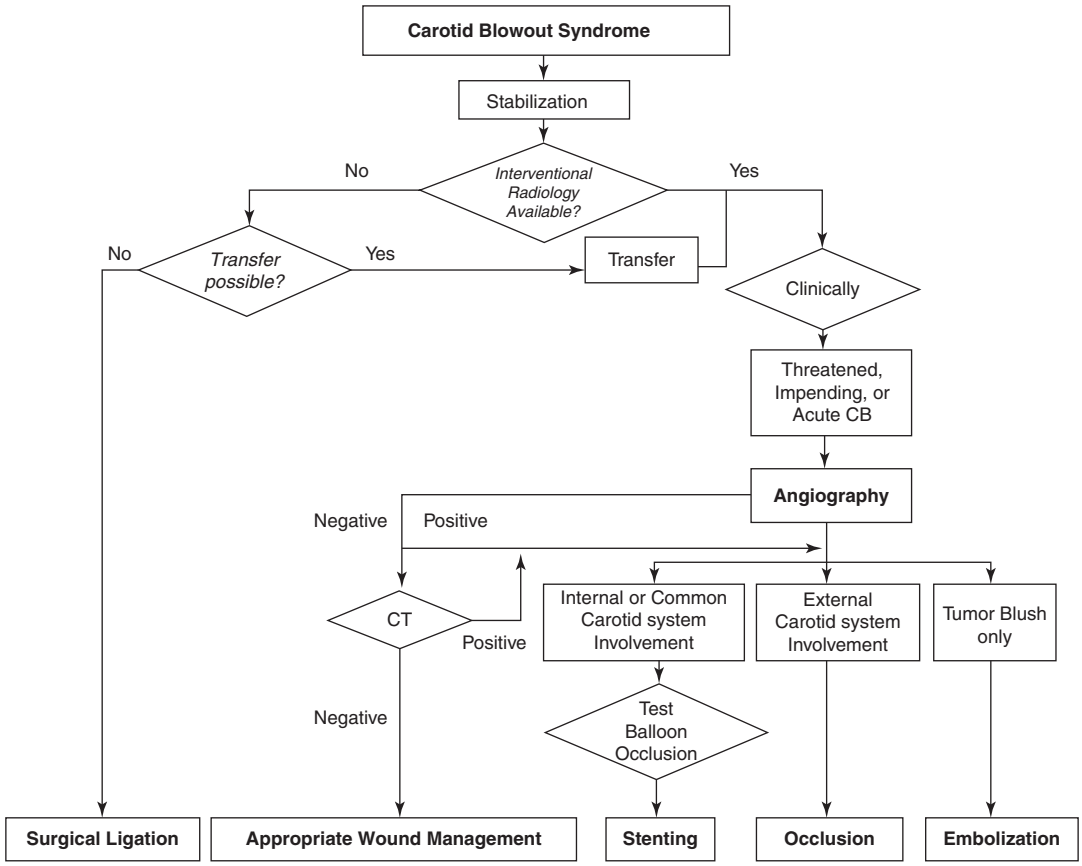

Fig. 8.7 Management of carotid artery blowout. (Reprinted with permission by Pool and Goyal [32])

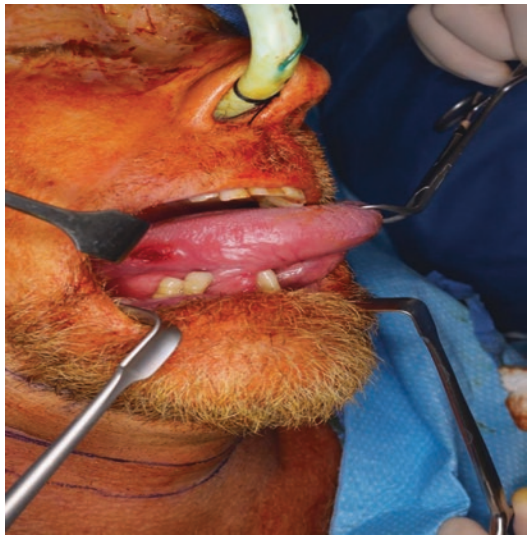

Fig. 8.8 Bleeding ulcer on the right posterolateral surface of the tongue. (Courtesy of Dr. Beomjune Kim)

Case Examples

Case 1

A 65-year-old male patient presents with a chief complaint of "a lesion on the right tongue" (See Fig. 8.8*). He first noticed this lesion 2 months ago but did not think much of it, because it was not painful. However, he developed intermittent bleeding from the lesion. Denies chest pain and shortness of breath.*

- *Past medical history – hypertension, arthritis.*
- *Past surgical history – adenoidectomy and tonsillectomy, arthroscopy of the right knee.*
- *Medications – Norvasc, multi-vitamins.*
- *Social history – 40 pack-year history of cigarette smoking, drinks 3–5 cans of beer daily.*
- *Family history – His father died of a heart attack at the age of 72, his mother is alive and healthy.*

- *What would you like to do next?*
  I would perform a complete physical exam.

  Physical exam reveals a well-developed and well-nourished gentleman with no palpable lymph node in the neck. Intraorally, 1.5 cm ulceration with rolled borders is found on the right posterolateral surface of the tongue. The area is friable and easily bleeds. The tongue has full range of motion. His oral hygiene and dentition are in fair condition without gross caries or active periodontal disease. No other suspicious lesions or masses are found on exam. He has no cranial nerve deficits.

- *What would you like to do next?*
  Based on the history and physical exam findings, the lesion is very suspicious for oral SCC, especially with his long-standing history of smoking and alcohol. I would perform a biopsy.

- *Would you perform an excisional or incisional biopsy and why?*
  Incisional biopsy must be performed. Only a small portion of the lesion should be biopsied. If cancer is high on your differential diagnosis, excisional biopsy is contraindicated because it interferes with the determination of surgical margins at the time of definitive surgery.

- *The histopathologist's diagnosis returns as moderately differentiated invasive SCC with 4.5 mm depth of invasion. What would you do next?*
  As there is a risk for a synchronous tumor, a nasopharyngoscopy examination would be necessary to evaluate the subepiglottic and supraepiglottic regions, posterior oropharynx, and nasopharynx.

- *Where is the most common metastasis for oral SCC?*
  The lungs.

- *Your examination does not identify a synchronous tumor. What is next?*
  I would order some imaging and laboratory studies.

- *Which studies would you want and why?*
  An orthopantogram to aid in the evaluation of the remaining dentition and invasion into the

adjacent bone. To evaluate cervical lymph nodes, CT neck with IV contrast is obtained. A chest X-ray to evaluate for pulmonary metastasis and pulmonary disease. Liver function tests to rule our hepatic metastasis. A complete metabolic panel, complete blood count and coagulation profile would also be requested as it is not uncommon for metabolic, electrolyte, and nutritional deficits to be encountered in cancer patients. Also an electrocardiogram would be ordered due to his age, cardiac disease, and long smoking history.

- *His panorex does not show any signs of periapical pathology, dental caries, or chronic periodontitis. Chest X-ray returned. CXR is clear without any nodules and LFT is within normal range. CT scan did not show any suspicious node or bony invasion. Remaining blood work and EKG are all within normal limits. How would you stage this patient's cancer?*
  The AJCC staging system is used to determine the TNM stage of this patient.
  This patient has superficial tongue carcinoma with a surface dimension of 1.5 cm, which belongs to clinical T stage 1 (cT1). CT of the neck with IV does not show any suspicious node (cN0). CXR and LFT results are within normal limits (cM0).
  This patient has clinical stage 1 oral tongue squamous cell carcinoma (cT1 cN0 cM0).

- *How would you treat this patient?*
  Smoking cessation is crucial to minimize perioperative pulmonary and wound complications and to prevent recurrent cancer after treatment. I would present this case to my tumor board.
  Early stage oral SCC can be treated with either surgery or radiation alone. I would review with the patient the pros and cons of each approach; however, I would highly recommend surgery as the primary treatment. This patient has no serious medical comorbidities that would preclude surgery. However, the cons and pros of each option should be discussed with the patient.
  I would perform a partial glossectomy with at least 1 cm tumor-free surgical margins. Intraoperative frozen section analysis of margins would be performed for the preliminary assessment of margins. The ipsilateral neck has to be treated based on the depth of invasion >4 mm, which suggests >20% chance of occult regional metastasis. For oral tongue SCC, a supraomohyoid neck dissection is indicated.

- *Which group of lymph nodes would you include?*
  I would remove levels 1–4 due to concern for skip metastasis. It is easily accessible and there is a high chance of skip metastasis in tongue cancers in the N0 neck.

- *What is a supraomohyoid neck dissection?*
  Supraomohyoid neck dissection removes lymph node levels I-III while preserving the spinal accessory nerve, internal jugular vein, and sternocleidomastoid muscle.

- *Would you reconstruct the tongue and if so how?*
  If more than 1/3 of tongue volume is resected, regional or distant flap reconstruction is usually performed. Radial forearm free flap is an excellent choice for this defect due to its thin and pliable skin paddle. Larger defects are usually reconstructed with bulkier flaps like anterolateral thigh (ALT) free flap or rectus myocutaneous flap. These patients benefit greatly from speech and swallow evaluation and therapy after oral cancer resection.

- *The final pathology shows:*
  - *Tumor dimension: 1.3 cm × 1.2 cm × 0.7 cm.*
  - *Margins: all negative with closest margin 9 mm from the posterior margin of the specimen .*
  - *0/33 positive lymph nodes.*
  - *Lymphovascular invasion: not found.*
  - *Perineural invasion: not found.*

  - *Extracapsular spread: N/A.*
  - *Grade: Moderately differentiated squamous cell carcinoma.*
  - *Pathologic Stage 1 (pT1 pN0 pMX).*

- *Does this patient need any adjuvant therapy?*
  No adverse features (such as T3/T4 primary tumor, positive margins, extracapsular spread, perineural/lymphovascular invasion, two or more metastatic lymph nodes) are found. Adjuvant therapy is not recommended.

- *What is the next step?*
  In order for early detection of recurrence or second primary cancer, the patient needs to be seen every 1–3 months for the first 2 years and then 4–6 months for the following 3 years. After patients are disease-free for 5 years, they are considered cured. However, they should still be seen annually after 5 years because these patients are at life-long risk of developing another cancer.

Case 2

*A 55-year-old Caucasian male patient is referred by his general dentist after a biopsy-proven SCC of right floor of the mouth (*Fig. 8.9*). He has difficulty eating and has lost 3 pounds over the last 2 months. He denies any swallowing, chest pain, or breathing difficulty. He also complains of right ear pain.*

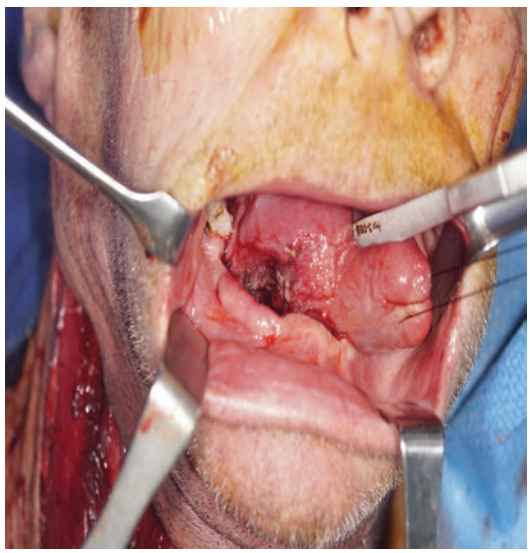

Fig. 8.9 Photo demonstrating floor of the mouth lesion. (Courtesy Dr. Beomjune Kim)

- *Past Medical History – hypertension and hypercholesterolemia.*
- *Medications – Lisinopril, Atorvastatin*
- *Allergy – NKDA*
- *Social History – 30 year-pack history of cigarette smoking, heavy alcohol (6 pack of beer a day). He works at an auto body shop.*
- *Family History – Father died of colon cancer at the age of 68. Mother survived breast cancer and is otherwise healthy.*
- *ROS –Weight loss and otalgia per HPI.*

- *How would you work this patient up?*
  With his diagnosis of oral SCC, he needs to be staged for further management. Again, start with thorough H and P and a review of systems.

- *What would you like to do next?*
  A complete physical examination including a nasopharyngoscopy.

  Exam reveals a slender gentleman in no acute distress. The neck is soft without palpable lymph nodes. Intraoral exam reveals a painful 3 cm endophytic tumor in the right floor of mouth with rolled borders extending to the right ventral tongue. The tumor is not fixed to the anterior mandible. No lingual nerve, mental nerve, or hypoglossal nerve deficits are noted. Nasopharyngoscopy does not show any abnormal tissue.

- *Would you like any imaging?*
  I would order several imaging studies including CT of the neck with IV contrast CXR, and PET-CT.

- *CXR is negative for any suspicious findings.*

- *What do you see on the CT (See* Fig. 8.10*)?*
  CT of the neck with IV contrast shows an enhancing mass in the right floor of mouth and tongue without any evidence of bony erosion.

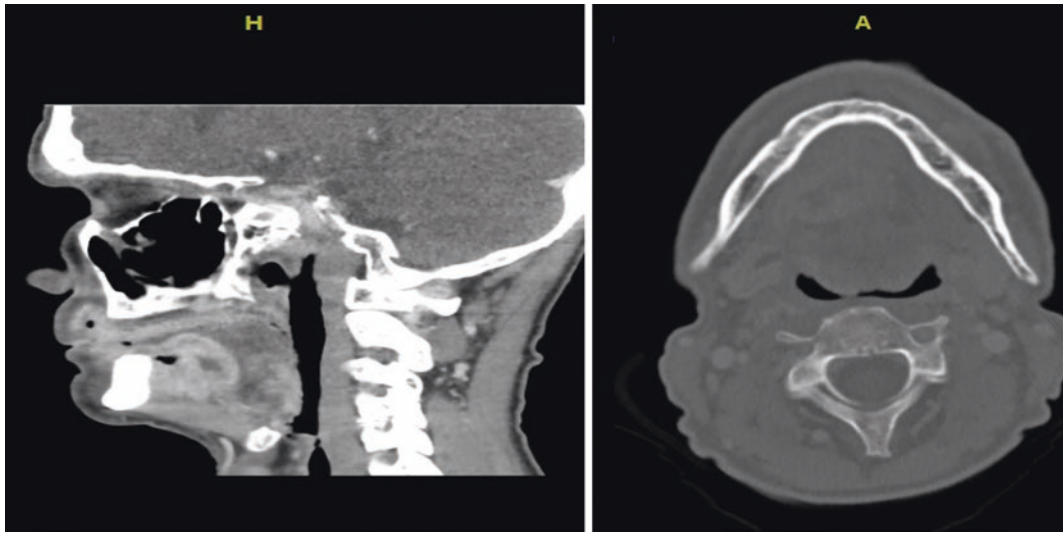

Fig. 8.10 Sagittal and axial slices. (Courtesy Dr. Beomjune Kim)

CT of the neck in axial view does not show any sign of mandibular cortical erosion.

- *PET-CT also shows a large mass in the floor of mouth and tongue with SUV of 6.5 as well as a 1 cm right level 2a node with SUV of 2.5. PET-CT does not show any suspicious distant metastasis. How does a PET-CT detect cancer cells?*
  It evaluates the uptake of the cells using the marker 18-fluorodeoxyglucose.

- *Any other laboratory tests you would like to order?*
  I would request to do complete blood count, basic metabolic panel, liver function tests, and prealbumin blood tests as well as an EKG.

- *Significant laboratory findings include prealbumin of 13, hematocrit of 34, and slightly elevated liver function tests and coagulation panels attributable to his heavy alcohol consumption.*

- *What would be your clinical staging?*
  Based on these work-ups, he has cT2 cN1 cM0 (Stage 3) squamous cell carcinoma of the floor of mouth.
  Tumor size up to 4 cm in greatest dimension without invasion into the mandible or extrinsic muscles of tongue belongs to T2. A single ipsilateral lymph node up to 3 cm belongs to N1. No distant metastasis belongs to M0.

- *How would you manage his cancer?*
  Based on his advanced clinical stage 3 oral SCC, he will most likely benefit from combined modality therapy with surgery and adjuvant radiation with or without chemotherapy. Thus, he should be presented at a tumor board.
  A dental exam should be completed and nonrestorable teeth should be extracted.
  Nutritional counseling and optimization are essential to improve his surgical and disease outcome.
  Smoking cessation should be discussed. Both psychological and pharmacological intervention may be necessary. Composite marginal mandibulectomy is chosen based on clinical and radiographic finding that the mandibular bone is not directly invaded. The presence of metastatic node warrants comprehensive neck dissection with removal of levels 1–5. In this case, I would offer a modified radical neck dissection type III due to morbidity.

- *What is a composite mandibulectomy?*
  If any sign of bony involvement is found intraoperatively, composite segmental mandibu-

lectomy should be performed. Composite mandibulectomy involves removal of soft tissue with mandibular bone.

- *What is a marginal mandibulectomy?*
  "Marginal mandibulectomy" means removal of mandibular bone with preservation of the mandibular continuity as opposed to "segmental mandibulectomy" which involves removal of an entire segment of mandible with disruption of mandibular continuity.

- *Final Pathology:*
  - *Greatest tumor dimension of 3.5 cm*
  - *Moderately differentiated SCC*
  - *Posterior inferior soft tissue positive margin*
  - *(+) lymphovascular invasion*
  - *(+) perineural invasion*
  - *4/34 positive metastatic nodes without extracapsular spread*

- *What is the TNM stage?*
  pT2 pN2b pMX

- *What adjuvant therapy would he need based on the final path report?*
  He has many adverse features including positive margins, multiple positive lymph nodes, perineural invasion, and lymphovascular invasion. Therefore, he would benefit from adjuvant radiation therapy. Moreover, due to the positive margin status, he would benefit from the addition of cisplatin (typical dose: 100 mg/m^2 every 3 weeks for 3 doses) to adjuvant radiation therapy.

- *When would you take post-radiation PET-CT?*
  Usually, post-radiation treatment PET-CT is performed at a minimum of 12 weeks after treatment to reduce the false positive rate from post-treatment inflammation.

- *Describe what you see in this image (*Fig. 8.11*)?*
  Axial CT of the neck with IV contrast shows a right metastatic 29 mm level 1 lymph node with central necrosis.

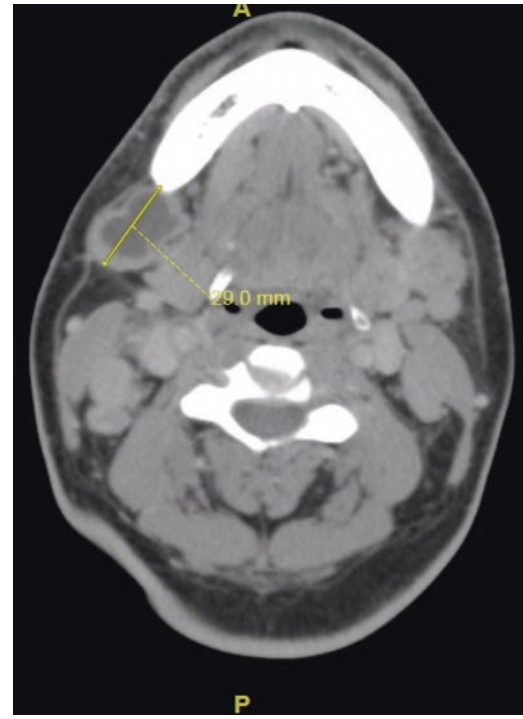

Fig. 8.11 Axial CT slice demonstrating enlarged lymph node. (Courtesy Dr. Beomjune Kim)

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Reconstruction

9

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Cancellous Cellular Marrow Grafts and Corticocancellous Block Grafting

Terms:

- Osteoconduction – graft material acts as a scaffold for vascular tissue and mesenchymal cells.
- Osteoinduction – stimulation of osteoprogenitor cells to differentiate into new bone forming cells (osteoblasts).
- Osteogenesis – transfer of vital osteoblasts to contribute to the growth of new bone.
- Allograft – derived from the same species. Can provide osteoconduction and osteoinduction.
- Autograft – graft obtained from the same individual. Provides osteoconduction, osteoinduction, and osteogenesis (See Table 9.1).
- Xenograft – graft from a species that is non-human. Provides osteoconduction.
- Alloplastic graft – graft from synthetic materials.
- Creeping substitution – process by which osteoclastic activity creates new vascular channels, with osteoblastic bone formation,

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Table 9.1 Autogenous bone graft sites

Harvest site	Bone available
Calvarium	No cancellous bone. Use as cortical onlay or bone mill for particulate graft.
Anterior ilium	Up to 50 mL, corticocancellous block also available.
Posterior ilium	Up to 120 mL, corticocancellous block also available.
Tibia	Up to 25 mL cancellous bone available. 1 × 2 cm cortical block.
Symphysis	Corticocancellous block up to 2 × 6 cm, 5 mL if milled for a particulate graft.
Maxillary tuberosity	Very small amount of cancellous bone for socket grafting or peri-implant or periodontal defects.
Ramus	Corticocancellous block of 2 × 5 cm.
Rib	Non-cancellous. Use as a costochondral graft or strut.

resulting in new haversian systems and osteogenesis from the graft.

Tibial Bone Graft

- Ease of access makes this a suitable procedure to be done in the outpatient surgery center or office.
- Contraindications to harvesting bone from the proximal tibia include patients with history of surgery in the area or implanted hardware, acute infection of the soft tissues over the surgical site.
- Relative contraindications would include patients with a history of metabolic bone disease.
- Up to approximately 25 mL of cancellous bone (Table 9.1) can predictably be harvested from the proximal tibia, which makes this donor site good for many maxillofacial indications including alveolar cleft grafting, sinus elevation, and socket grafting.

Anatomy and Surgical Technique

There are two approaches that have been described for harvesting bone from the proximal tibia.

1. Medial approach.
2. Lateral approach that centers over the lateral tibial plateau, also known as Gerdy's tubercle; most common approach. In this review, we will discuss the lateral approach.
   - To minimize difficulty with dissection and postoperative pain, it is important to avoid the tibialis anterior muscle, which attaches inferiorly to the harvest site when the incision is placed over Gerdy's tubercle.
   - The attachments to this tubercle include the fascia lata above and the anterior tibialis muscle below. Together, these form the iliotibial tract that stabilizes the knee and hip during gait. Therefore, minimal stripping of these structures during dissection will minimize pain during ambulation post-operatively.
   - There are no major blood vessels or nerves located between the skin surface and periosteum of the bone over Gerdy's tubercle; although, the lateral genicular artery does travel transversely above the tubercle and the anterior tibial artery travels along the anterior tibial surface below this tubercle.
   - The common peroneal nerve courses inferior to the tubercle and should not be found within the surgical site in a proper dissection.

Surgical Technique (Lateral Approach)

- The patient is positioned with the knee partially flexed and medially rotated and prepared in a sterile fashion.
- The skin and subcutaneous tissue should be infiltrated with epinephrine containing local anesthetic for bleeding and pain control.
- 2 cm incision is marked directly over the palpable ridge of Gerdy's tubercle. This incision is parallel to the tibial plateau (articulating surface) and oblique to the long axis of the tibia. An incision is made through the skin and subcutaneous tissue down to the periosteum.
- Once the periosteum is incised, a small portion of the anterior tibialis muscle inferiorly

and the fascia lata above will be stripped to allow access to the cortex.
- A fissure bur, under copious irrigation, is used to make a 1.5–2.0 cm circular corticotomy. This can be removed with an osteotome and mallet or a periosteal elevator.
- Curette is inserted, and in a rotational manner, the cancellous bone is harvested. The curette should be inserted transversely across the tibia in a downward direction. The risk of perforating the subchondral bone at the superior edge of the tibia and violating the knee joint is small; but proper care during curetting to avoid the area will minimize the chance of that complication.
- Once the graft harvest is complete, the graft is placed into a 10 mL syringe and compacted. Tibial cancellous bone has a higher composition of fat compared to iliac cancellous grafts. It is important to extrude the extra fat cells and concentrate the progenitor cells.
- This is stored on ice until ready for graft inset and the leg is closed. Bovine microfibrillar collagen (Avitene®) or absorbable gelatin sponge (Gelfoam®) can be placed into the harvest site for hemostasis.
- The wound is then closed in layers and a dry sterile dressing is applied.

Complications and Management

- Tibial bone graft complications include infection, gait disturbance, osteomyelitis, hematoma, seroma, fracture, and violation of the joint space.
- *Ecchymosis and Swelling of the Lower Leg and Ankle* – more of a pseudo complication, can be decreased by keeping the limb elevated. Although normal weight bearing is permitted postoperatively, strenuous activities should be avoided, as this will increase pain and swelling. Resolves spontaneously.
- *Violation of the Joint Space or Fractures of the Tibia* – treated with non-weight bearing therapy, splinting, and orthopedic surgery consultation.
- *Osteomyelitis* – MRI to evaluate for depth of invasion and true osteomyelitis. Consult orthopedic surgery. Infectious disease and wound therapy consult for possible hyperbaric oxygen and appropriate long-term antibiotic therapy.

Calvarial Bone Graft

- Shown to have rapid revascularization and limited resorption that allows for resistance to remodeling and soft tissue displacement.
- Limited morbidity and donor site deformity as well as donor site proximity to the recipient site makes calvarial bone useful in both craniofacial and dentoalveolar reconstruction.
- The graft is mostly cortical, which makes it a durable graft for not only ridge augmentation but also orbital and craniofacial reconstruction.

Anatomic Considerations

- Although there are several regions of the skull available for harvest, the most common is the parietal area. The skull is thickest in this area (average 6.3 mm) [1] and does not overlay dural sinuses or arteries.
- There are no major nerves in the area. In most patients, the parietal scalp is fully hair bearing, which makes for acceptable scar camouflage.
- Superior sagittal sinus runs 5 mm parasagittal to the midline. To avoid injury to the superior sagittal sinus, harvest should be 2 cm away from midline and 2 cm away from thin squamous portion of temporal bone inferiorly.

Surgical Technique

- The parietal bone can be accessed by either a coronal incision with wide elevation of the scalp (which is convenient when upper facial skeletal exposure is needed in the case of trauma) or through a linear incision directly over the donor site.
- After instilling the area with local anesthetic (with epinephrine), a scalpel is used to make an incision through the SCALP (skin, subcu-

taneous tissue, aponeurosis, loose areolar connective tissue, pericranium). The periosteum can be incised separately, although this is not necessary.

- The loose connective tissue plane easily dissects (either blunt finger dissection or sharp dissection with the back of a scalpel blade) from the pericranium. This allows for separate incision through the pericranium. Separate elevation of the pericranium may lead to less blood loss, as this is less disruptive to cranial perforating vessels.
- Raney clips (or a running suture) should be applied to the scalp edge instead of extensive electrocautery for hemostasis. Extensive electrocautery can damage the skin and hair follicles and increase the risk of alopecia over the incision.
- Once the incision is made, a periosteal elevator can be used to elevate the scalp in a subpericranial plane until adequate bone is exposed for harvest.
- A fissure bur under copious irrigation is used to outline the desired graft through the outer table. The bur can be used to bevel the outer margin of the corticotomy to facilitate placement of a curved osteotome to complete the outer table harvest.
- With a curved osteotome, gentle malleting is done radially around the graft to separate the graft from the donor bed.
- Once the graft harvest is complete, the graft is set aside in saline, and bleeding at the donor site is controlled. This is most commonly accomplished with bone wax. The resulting skull defect may be filled with hydroxyapatite cement or a titanium mesh to avoid a post-harvest deformity, although this is not always required.
- The wound is then closed in layers. Care should be taken to approximate the galea (aponeurosis) as this will decrease scar width.

Complications

Complications associated with harvesting calvarial bone include the possibility of infection, alopecia, intracranial passage of instrumentation, dural tear, epidural hematoma, subgaleal hematoma, contour deformity, and scarring.

Perforation of Inner Cortex/Dural Tear Craniotomy is frequently required to extend the visual field to identify underlying dural or parenchymal injury. If injury to cortex is identified neurosurgical consultation is required. Most tears can be treated with direct repair with a non-resorbable suture such as Nurolon™ (Ethicon). Larger defects may require grafting. Post-operative non-contrast head computed tomogram (CT) should be sought to rule out intraparenchymal hemorrhage.

Anterior Iliac Crest

- Max of 50 cc of uncompressed cancellous bone (up to 5 cm defect) – remember 1 cm defect requires about 10 cc of bone graft.
- Harvest site is located between Anterior Superior Iliac Spine (ASIS) and tubercle of ilium (which is 6 cm posterior to ASIS).
- ASIS – attachment for external oblique muscles (medially), tensor fascia lata (TFL) laterally. Dissection laterally should be minimized as to prevent postoperative gait disturbance and pain. Inferior to anterior iliac crest are the gluteus medius and minimus muscles, which attach to the lateral cortex.
- Iliacus muscle attaches to the medial surface of iliac crest (reflected during medial dissection)
- Sensory cutaneous nerves (there are no motor nerves overlying the anterior ilium):
  - The most commonly encountered nerve is the lateral cutaneous branch of the iliohypogastric nerve (L1, L2) – this nerve courses over tubercle of the ilium. Damage to this nerve causes sensory disturbance over the lateral anterior third of the ilium.
  - Lateral cutaneous branch of subcostal nerve (T12, L1) courses over ASIS passing just inferior to iliohypogastric nerve.
  - Lateral femoral cutaneous nerve (L2–3) – most inferior of all three nerves of interest,

courses medially between psoas major and iliacus, deep to the inguinal ligament, and perforates TFL to innervate lateral skin of thigh. In 2.5% of people, this nerve courses within 1 cm of ASIS placing it at risk during inferior dissection. Damage to this nerve can result in meralgia paresthetica (dysesthesia and anesthesia of lateral thigh) [2].

- Perforators from deep circumflex iliac artery and vein originating from the external iliac system – located on the medial aspect of the ilium are the predominant vascular supply. The most common source of bleeding is from the superior gluteal artery (internal iliac system).
- Left hip is most often chosen for donor site to prevent interference with driving.

Surgical Technique

- Patient is placed in a supine position and a soft roll such as a saline bag or sandbag wrapped in a towel is placed to elevate the hip.
- Retract skin medially with the surgeon's nondominant hand, so that the resulting scar will end up lateral to the iliac crest and less likely to be irritated by clothing. The resulting scar is also more cosmetic.
- Mark the incision site (4–6 cm in length and placed 1–2 cm anterior to tubercle of the ilium and 1 cm posterior to ASIS). Infiltrate the planned area of dissection with local anesthetic with epinephrine.
- The incision is oblique along the anterior iliac crest – this will avoid the iliohypogastric and subcostal nerves which are superior and the lateral femoral cutaneous nerve which is inferior-medial.
- Layers of the incision – skin, subcutaneous tissue, Scarpa's fascia, and muscular aponeurosis.
- Plane of dissection – between TFL (laterally) and external oblique and transverse abdominus muscles (medially) which is in an avascular plane. The dissection in this plane will lead straight to the iliac crest periosteum (sharply transected). The iliacus muscle is sharply dissected from the medial surface.
  - *NOTE: This approach constitutes the medial approach.*

    Disadvantages of medial approach: higher incidence of meralgia paresthetica (lateral femoral cutaneous nerve) and postop ileus.
  - *The lateral approach requires dissecting away the tissues lateral to the crest, which are the TFL and gluteus medius muscles. There is less intra-abdominal injury since the dissection is lateral, but higher postop pain and gait disturbance (TFL).*
- Depth of harvest: 5 cm – depth at which cortical plates fuse
- Techniques for graft harvesting:
  - Clamshell: mid-crestal osteotomy, fold medial and lateral cortices over to expose underlying bone marrow (good technique for cancellous-only harvest) – if you need a larger quantity of bone, full-thickness corticocancellous block can be harvested (maximum 4–6 cm) – limited anteriorly by ASIS and posteriorly by tubercle of the ilium – if you want to decrease risk of fracture of ASIS, leave 3 cm of intact bone posterior to it.
  - Trapdoor: either medial or lateral cortex with attached musculature is pedicled like a hinge to gain access to marrow.
  - Tschopp: oblique osteotomy of iliac crest, pedicled onto external oblique muscle
  - Tessier: medial and lateral oblique osteotomies, both aspects pedicled to gain access
  - Trephine technique: incision only 2 cm in length, no medial or lateral stripping, trephine used to perforate crest, angulated 30° to vertical
- Bone wax and/or microfibrillar bovine collagen can be used to aid in hemostasis. A low suction drain may also be placed to prevent postoperative hematoma.
- Closure – need to reapproximate periosteum over crest followed by a layered closure.

Complications

- *Hematoma* – if non-expanding, pressure packing may be applied. If expanding, require surgical exploration.
- *Massive Hemorrhage* – superior gluteal artery is the usual culprit. This is caused by harvesting proximal to and/or retracting too aggressively near the greater sciatic notch. Treatment includes exploration, ligation, embolization by IR; do not try to blindly clip it or place hemostat, it has the risk of damage to the sciatic nerve or superior gluteal nerve.
- *Seroma* – needle aspiration (if large) vs. pressure dressing (if small).
- *Nerve Injury* – can be from direct injury to nerve, fibrosis, entrapment during closure, hematoma, or seroma causing external nerve compression.
  - Lateral femoral cutaneous (meralgia paresthetica).
  - Iliohypogastric (loss of sensation to lateral gluteal and suprapubic regions).
  - Subcostal nerve (loss of sensation to the lateral hip).
- *Infection of Donor Site* – removal of sutures to allow drainage. Take culture, local wound care, and antibiotics to treat infection.
- *Gait Disturbance* – stripping of TFL, gluteus medius (abductors). Normally self-limiting. Physical therapy to aid in reestablishing gait and possible walking aid in the interim.
- *Bony Fracture (ASIS, Tubercle of Ilium)* – caused by harvest too close to ASIS, due to action of Sartorius and TFL muscles (stay at least 2 cm away from ASIS to avoid this complication). Management is usually nonsurgical, including pain management and bedrest (if greenstick fracture). Large fractures or significant displacement may require fixation, and an orthopedic consult is warranted.
- *Intra-abdominal Injury* – this is a surgical emergency and requires an exploratory laparotomy.
- *Adynamic Ileus* – cessation of mechanical peristalsis of the bowel. This is managed by bowel rest, electrolyte correction, nasogastric suction (for decompression), and minimal use of narcotics. Should this therapy fail, promotility agents such as metoclopramide (Reglan) may restore bowel function. Most commonly this is a time-limited issue. If not self-resolving, further physical examination and imaging to rule out bowel injury is critical.
- *Sacroiliac Instability* – pain in lower back or pubis (posterior destabilization of SI joint). Fusion of SI joint may be needed in future if persistent pain.
- *Abdominal Wall Hernia* – risk factors: >4 cm block harvest, female gender, or obesity. Will require general surgery consultation.

Posterior Iliac Crest

- Up to 100 cc of uncompressed bone available (up to 10 cm defect).
- Superior cluneal nerve (L1–3) – pierces lumbodorsal fascia, travels superior to posterior iliac crest, and provides sensation to posteriormedial buttocks.
- Middle cluneal nerve (S1–3) – traverses through the sacral foramina and innervates the medial buttocks.
- Sciatic nerve (L4–5, S1–3) is 6–8 cm below the level of posterior iliac crest – should not be encountered.
- Blood supply – perforators from the subgluteal artery.
- Additional surgical time to reposition patient.
- Concern for endotracheal tube displacement risk during patient maneuvering.
- Unable to perform simultaneous procedures.

Surgical Technique

- Patient in prone position – 210-degree reverse hip flexion.
- 6–10 cm curvilinear incision is drawn following the course of posterior iliac crest. The superior and inferior boundaries of the field

are defined by superior and middle cluneal nerves. The incision should end inferiorly approximately 3 cm lateral to gluteal crease centered over the insertion of gluteus maximus muscle (bony protuberance in triangular fossa). The area of dissection is infiltrated with local anesthetic with epinephrine.
- The incision layers – skin, subcutaneous tissue, lumbodorsal fascia (separates abdominal and gluteal musculature), and periosteum over the posterior iliac crest.
- The gluteus maximus muscle is stripped from the tubercle using a blade or electrocautery against the cortical bone. Additional exposure may be gained by reflection of the gluteus medius with a Keyes periosteal elevator.
- 5 cm × 5 cm posterior iliac crest osteotomy of lateral cortical plate to access cancellous bone. Limit harvest at least 4 cm from PSIS to avoid violation of the sacroiliac joint.
- Bone wax and/or microfibrillar bovine collagen can be used to aid in hemostasis.
- Closure in layers; reapproximate periosteum and lumbodorsal fascia.
- A drain should be placed to prevent postoperative hematoma.

Complications

- *Arterial Injury* – superior gluteal artery, postop gluteal compartment syndrome (treated with ligation; if continued bleeding, may need exploratory laparotomy via retroperitoneal approach, or embolization by IR).
- *Ureteral Injury* – postop hematuria, abdominal distention, ileus – from excessive electrocautery usage near the greater sciatic notch (usually while you are trying to control bleeding from superior gluteal vessels). Urology consult is indicated. Treatment may include placement of a ureteral stent or surgical repair.
- *Nerve Injury* – cluneal nerves, posterior pelvic pain radiating to buttocks.
- *Gait Disturbance* – weak abductors (mainly gluteus medius) – from excessive stripping.

Microvascular Free Tissue Transfer

- The term free flap (also known as autologous tissue transfer and microvascular free tissue transfer) is used to describe the transplantation of tissue with its own blood supply from one site of the body to another. The circulation in the transferred tissue is reestablished by anastomosis of the transferred arteries and/or veins to recipient vessels in the host bed (Table 9.2).
- Free flaps may be comprised of skin, muscle, nerve, bone (or any combination of these).

Anterolateral Thigh Flap (ALT)

- The ALT is a fasciocutaneous perforator flap based on the descending branch of the lateral circumflex femoral artery. It runs in the intermuscular septum between the rectus femoris (RFM) and vastus lateralis muscle.
- Long vascular pedicle length from 8–16 cm if measured the entire length of the flap.
- Flap is fairly thick due to the presence of copious subcutaneous tissue. Some authors have described "defatting" the flap at the time of harvest to thin the flap; however, this carries the risk of compromising venous circulation within the flap.
- The flap can be raised with only fascia and thin subcutaneous fat without the skin. This will result in a much thinner fascial flap.
- Popular due to the versatility of the flap in design.
- Donor site can often be closed primarily leading to minimal donor site morbidity.

Surgical Technique

- A line is drawn from the anterior-superior iliac crest to the lateral aspect of the patella. This line roughly corresponds to the intermuscular septum between the rectus femoris and vastus lateralis muscles. At the midpoint of this line, a 5 cm circle is scribed (centered on the line). Within this circle (the lateral two

Table 9.2 Free Flap Indications and Vascularity

Free flap	Components	Pedicle length/caliber	Indications
Radial Forearm Free Flap (RFFF)	Skin and fascia. Tendon (palmaris longus) if suspension is needed, bone (radius) if small bone is required.	Long pedicle if taken at take-off from brachial artery. Large caliber 2–4 cm with two venae comitantes or cephalic vein for drainage.	Thin flap is great for intraoral soft tissue defects or tongue reconstruction or lip reconstruction if tendon is included.
Anterolateral Thigh (ALT)	Skin, muscle (vastus lateralis), fascia, large flap up to 10 × 25 cm can be harvested	5–7 cm length, 1.5–3 mm diameter vessel, descending branch of the lateral femoral circumflex artery.	Large facial or intraoral defects, scalp defects, orbitocraniofacial resections, gunshot wounds. May be too thick in obese patients.
Deep Circumflex Iliac Artery (DCIA)	Vascularized iliac crest bone, iliacus muscle, with or without skin.	4–8 cm length, 1.5–3 mm diameter vessels, deep circumflex iliac artery and venae comitantes.	Maxillary or mandibular reconstruction, may require vein grafts if inadequate pedicle length.
Scapula	Skin and bone (lateral border of scapula).	Up to 7 cm length, 2–4 mm vessel diameter, subscapular artery	Mandibular ramus reconstruction, maxillary reconstruction.
Free Fibula Flap (FFF)	Bone, muscle (flexor hallucis longus cuff or adjacent soleus muscle), and skin.	Pedicle length depends on the length of bone needed but can be 5+ cm, 2–4 mm diameter vessel, peroneal artery, and venae comitantes.	Maxillary or mandibular reconstruction, can use closing osteotomies to establish arch form.

quadrants most commonly) is where perforating vessels can be most readily identified. Once this is accomplished, the flap is designed centered over the perforator.

- Flap elevation begins by making a skin incision along the medial margin of the flap. This is carried through the skin, subcutaneous tissue, and fascia over the rectus femoris muscle.
- Once in the subfascial plane, gentle blunt dissection can be carried laterally until the perforating vessels are identified. (Most commonly the perforating vessels are muscular and pierce the vastus lateralis muscle. About 9% of the time, the perforators are septal. The presence of septal perforators does simplify flap harvest but when muscular perforators are encountered, a small cuff of vastus lateralis can be safely taken to protect the perforator.)
- Once the perforator is identified, the intermuscular septum between the vastus lateralis and rectus femoris muscles is dissected and the flap pedicle can be identified. (Motor nerve branches to the vastus lateralis muscle are commonly seen running with the artery and can be separated and preserved.)
- The pedicle is traced back to the takeoff from the lateral circumflex femoral artery. The flap is then incised around the lateral aspect.
- The fasciocutaneous portion of the flap is completely dissected. (Perforators are dissected from the muscle if needed.)
- Once the tumor is ablated and the size of the defect is defined, back cuts are made on flap to the desired size and modified as required.
- Vessels are clamped proximally and ligated, and the flap is delivered from donor site.
- Donor sites up to 8–10 cm wide can usually be closed primarily; if the flap is larger than this, skin graft closure can also be used but is less cosmetically pleasing.

Complications

Specific to the ALT flap paresthesia or anesthesia over the lateral thigh, seroma formation, wound infection. Paresthesia over the lateral thigh is not usually very bothersome. Seromas may require drainage and infections should be treated with antibiotics to cover for skin flora.

Herniation of Muscle There are reports of vastus lateral and rectus femoris muscle herniation. Requires exploration and repair. Repairs include direct closure or coverage with a split thickness skin graft. Larger hernias may be treated with a polypropylene mesh [3, 4].

Rectus Femoris Muscle Necrosis [4] Uncommon complication from ligation of the lateral circumflex artery proximal to take-off of the descending branch during harvest. Authors recommend when a larger pedicle is needed to place a vessel loop at the site of planned vessel harvest prior to definitive ligation to evaluate blood flow to the rectus femoris muscle.

Compartment Syndrome [4] Increase in pressure within a closed fascial space resulting in a decrease in capillary flow. If the deep fascia was used for closure, treatment describes release of the deep fascia. If deep fascia closure was not performed and compartment syndrome was discovered, debridement with VAC therapy has been described.

Radial Forearm Free Flap

- The radial forearm free flap is a fasciocutaneous flap based off the radial artery.
- Venous outflow is provided by either the venae comitantes or the cephalic vein.
- Used mostly for floor of mouth, tongue, lip, and buccal mucosa reconstruction. Defects that require more bulky tissue such as subtotal glossectomy and large skull base tumors are better suited with a bulkier flap such an ALT [5, 6].
- This flap is also being described to include a portion of the radial bone, the palmaris longus tendon, or brachioradialis muscle.
- Because the skin of the volar forearm is quite thin and pliable, this flap has become a workhorse flap in oral cavity, laryngeal and pharyngeal reconstruction. It is also useful for resurfacing defects where a thinner flap is desirable.
- Long vascular pedicle with large caliber vessels allows for easy anastomosis.
- There are usually no long-term sequelae of the donor site with hand and wrist mobility and finger strength being preserved in usual flap harvests.
- Major complaint is unaesthetic donor site due to scarring or poor color match when skin grafts are used for closure.

Preoperative Considerations

- Note any recent intra-arterial or intravenous lines placed as they may compromise flap vascularity. “No stick” order should be placed for patients presenting for reconstruction.
- Best to harvest from non-dominant hand in the event that there is a donor site complication that would compromise the hand or reduce hand strength or mobility.
- A preoperative Allen test is useful for determining whether the patient has acceptable ulnar collateralization. Many patients undergoing oral cavity reconstruction have poor peripheral circulation and an Allen test can be difficult to interpret. Therefore, the use of a pulse oximeter on the thumb when doing the test can improve sensitivity of the exam. Adequate compression of the ulnar and radial arteries should result in complete cessation of the arterial waveform on the pulse oximeter. Release of the ulnar artery should result in return of pulsatile flow but with an attenuated waveform. Then, release of the radial artery should result in restoration of the complete amplitude of the waveform. (In the traditional description, reperfusion of the fingers or nail beds should be seen in 15–20 seconds). In this scenario, adequate ulnar collateral flow is evident, and the patient should tolerate radial artery harvest without a risk for devascularization.
- Further vascular imaging studies are generally not necessary. In the event that the patient has a concerning Allen test, the patient can still undergo radial forearm free flap harvest; how-

ever, the radial artery should be reconstructed immediately with vein grafting. While the cephalic vein is commonly used for venous outflow of the flap, it can be used for reconstruction of the radial artery while the venae comitantes are used for the venous circulation of the flap.

Radial Forearm Harvest Technique

- The radial artery is palpated and marked as is the cephalic vein. The appropriately sized flap is then drawn over the radial and volar surface of the forearm centered over the radial artery. The distal aspect of the flap margin is marked approximately 1 cm from the distal wrist crease.
- A tourniquet is utilized and inflated to 250 mmHg for exsanguination.
- Flap elevation begins at the distal aspect of the segment. A#15 blade is used to incise the skin, subcutaneous tissue, and fascia along the distal margin of the flap. Curved hemostats are then used to dissect the cephalic vein as well as the radial artery and radial artery venae comitantes. They are then ligated and transected.
- Next, starting on the medial and distal corner, the flap is elevated in a subfascial plane. Care must be taken during this portion of the flap elevation to avoid dissecting the fascia from the radial artery. When elevating the flap over the flexor carpi ulnaris, palmaris longus, and flexor carpi radialis tendons, the paratenon over those tendons should be preserved to aid in skin graft take.
- Flap elevation is continued to the radial distal edge and, while lifting the flap, the two superficial dorsal branches of the radial nerve can be identified and left in place during flap elevation. This minimizes paresthesia over the dorsum of the hand along the thumb and index finger. Once the flap is elevated to the proximal margin, the proximal aspect of the flap is incised with care not to transect the cephalic vein.
- Releasing incision is then opened to the antecubital fossa. Vessel loops are placed around the cephalic vein distally and this vein is followed proximally and dissected out of the subcutaneous tissues with care being taken to ligate and divide any branches.
- The brachioradialis muscle is retracted laterally and the flap is elevated to place gentle tension on the radial artery. Vessel loops were placed around the radial artery and it is traced proximally as branches are ligated and divided.
- The cephalic vein and radial artery are traced to the antecubital fossa both in order to provide adequate pedicle length and also to improve vessel caliber.
- The tourniquet is deflated, and the flap is reperfused for 20 minutes.
- The flap can then be harvested by ligating and dividing the radial artery and two venae comitantes as well as the cephalic vein.
- The flap is passed into the oral cavity for inset and anastomosis.
- Closure of the radial forearm donor site is most commonly accomplished by primary closure of the releasing incision and then skin graft application over the flap donor defect. Some surgeons prefer a full-thickness skin graft harvested from the medial surface of the upper arm as this area is already prepped into the surgical field and a full-thickness skin graft gives thicker coverage for the flexor tendons. Others will utilize a split thickness skin graft harvested from a distant site such as the thigh.
- If a skin graft is used, it should be perforated to allow seepage of fluid. A bolster is kept in place for 5–7 days and the splint is kept in place for 4 weeks.

Complications

Delayed Wound Healing Failure of the skin graft resulting in tendon exposure, infection, and decreased mobility of the wrist or fingers due to scarring. Treatment options depend on the extent of necrotic tissue. The tissue may be debrided and a second skin graft attempted. The defect may be allowed to heal by secondary intention, by covering with moist gauze until healed [7, 8].

Hand Ischemia Can normally be avoided if Allen test is properly conducted, or can use color flow doppler if Allen test is not conclusive [8–11]. Can occur if the radial artery is the major blood supply to the hand, damage to the ulnar artery, or insufficient collateral blood flow between arterial systems. Treatment is an interpositional vein graft to the divided stump from either the saphenous or cephalic vein.

Decreased Pincer Grasp and Hand Strength Can result from harvesting a portion of the radial bone.

Infection Antibiotics with activity against skin flora are indicated if signs of infection are present. Serial debridement of necrotic material and irrigation of the wound is recommended. Cultures should be taken and considered for infectious disease consultation.

Nerve Injury Complaint of dysesthesia in the distribution of radial nerve, but this becomes less noticeable overtime [8, 10].

Fibula Free Flap

- Excellent option for reconstruction of any mandible defects (most commonly from neoplasm, osteonecrosis, and trauma).
- Ideal for composite osseous defects that require reconstruction of adjacent oral lining or external skin.
- The fibula is a long, thin bone that articulates with the lateral condyle of the tibia proximally and with the connective tissue of the ankle mortise distally. It is a non-weight bearing bone with a relatively thick cortex circumferentially.
- Bone height varies from 9 to 15 mm with a total length of approximately 35 cm, typically up to 25 cm can be harvested.
- The peroneal artery (PA) and its venous comitantes provide vascular supply to the fibular free flap. The external diameter of the peroneal artery is 1.5–2.5 mm and the pedicle length is between 2 and 6 cm [12].

Fascial Compartments

- The lower leg is separated into compartments dictated by the tibia and fibula bones as well as fascial planes (Fig. 9.1).
- The tibia and fibula with their interosseous septum separate the anterior and posterior compartments. The anterior lower leg is further subdivided into anterior and lateral compartments by the anterior intermuscular septum.

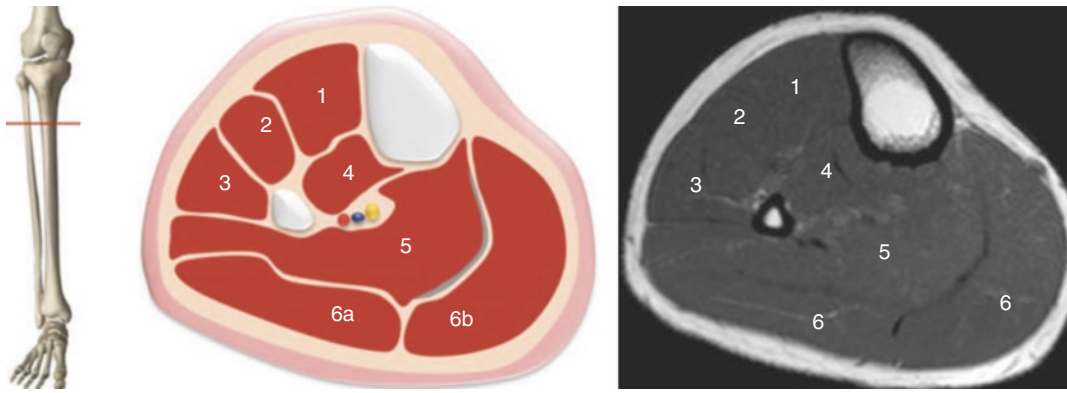

Fig. 9.1 1 Tibialis anterior; 2 extensor digitorum and hallucis; 3 peroneus longus; 4 tibialis posterior; 5 soleus; 6 gastrocnemius lateralis (6a) and medialis (6b). (Reprinted with permission from Silvestri et al. [13])

- The posterior lower leg is subdivided into deep and superficial compartments by the transverse intermuscular septum.
- The lateral and posterior compartments are separated by the posterior intermuscular septum which importantly carries the skin perforator vessels essential to skin paddle harvest.
- Knowledge of these fascial compartments and their contents is essential when harvesting a fibular flap.

Preoperative Workup

The integrity of the lower limb and foot vasculature is essential before planning for a fibular free flap. Work up should be focused on identifying abnormal vascular patterns to the foot as well as identifying compromised vessel quality. Imaging is recommended to confirm three vessel run-off.

History

- Inquire about cardiovascular disease risk factors (coronary artery disease, peripheral vascular disease, smoking, etc.).
- Specifically check for history of claudication, dependent edema, venous thrombosis, varicose veins, prior lower limb surgery, and prior lower limb trauma.

Physical Exam

- Inspect for signs of peripheral vascular disease including edema, induration, varicosities, shiny/hairless skin, and cold feet.
- Palpate dorsalis pedis and posterior tibial pulses.
  - "Modified" Allen test: Apply pressure to dorsalis pedis artery while palpating for posterior tibial pulse and vice versa to eliminate retrograde flow.
- ABI (Ankle-Brachial Index): Objective measure to detect arterial insufficiency although its use is primarily historical in this setting due to improved imaging modalities.

Imaging

1. Magnetic Resonance Angiography.
2. Computed Tomographic Angiography (Fig. 9.2).
3. Conventional Angiogram.
4. Color-Flow Doppler Imaging (may also play a role in localizing perforators).

Guidance Point PVD doesn't necessarily preclude use of FFF; however, there is a higher risk of thrombotic events and flap complications. Other osteocutaneous flaps should be considered in these patients, such as the scapula, since the vascular supply is more proximal and less affected by PVD.

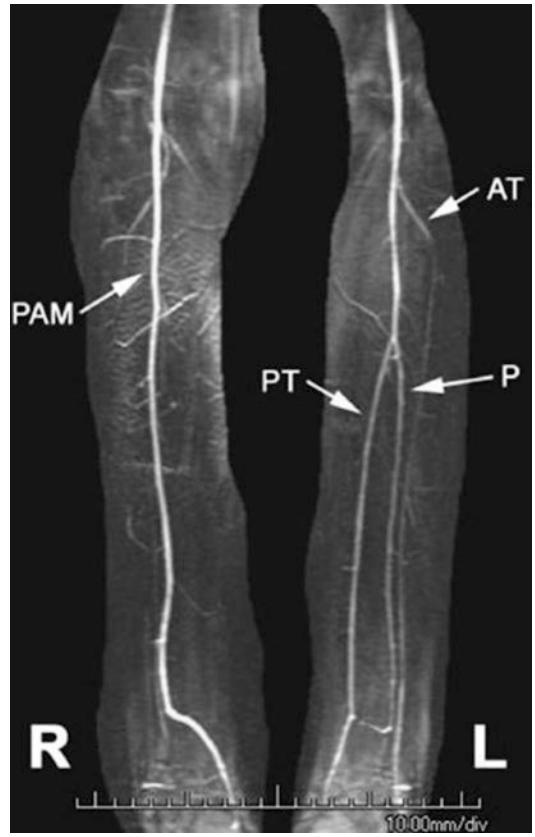

Fig. 9.2 Peronia Arteria Magna of right lower leg on angiography. Lower left leg demonstrating all three vessel branches of the popliteal artery (1) anterior tibial (AT) (2) posterior tibial (PT) and (3) peroneal artery. (Reprinted with permission from Betz and Betz [14])

Variation in Arterial Supply

- Surgically significant anomalies occur in 10% of population, 5.2% of any given limb.
- Variations are clinically asymptomatic but impact surgical candidacy.
- Infra-popliteal Arterial Branching Classification (Kim-Lippert's Classification System) [15]:
  - I: Normal level of branching
  - II: High-Division of PA (at or right above the knee)
  - III: (10.37% Overall) Hypoplastic/aplastic branching with altered distal supply (Table 9.3).
  - IV: PA vessel caliber variation
    - IV-A: Hypoplastic (not recommended, but may not fully preclude FFF harvest given anastomoses of vessels as small as 1 mm possible)
    - IV-B: Aplastic (absolute contraindication)

Fibula Free Flap Technique

- Exsanguinate leg with compressive bandage. Apply thigh tourniquet inflated to 250–350 mmHg.
- Plan a long anterior curvilinear incision to transverse intermuscular septum, along anticipated skin paddle. Making a lengthy anterior incision along the fibula allows for ease of access as well as preparation for variations in skin perforator anatomy.

Table 9.3 Type III subtypes of lower leg branching patterns

Type III (10%)			
Subgroup	IIIA	IIIB	IIIC
Notes	2-vessel runoff Deficient vessel: PT (63%)	2-vessel runoff Deficient vessel: AT (29%)	Single-vessel runoff (Peronia Arteria Magna). See Fig. 9.2. Deficient vessel: Both tibial arteries (8%)
Implication	Possible but unwise	Possible but unwise	Absolute contraindication

- Incise skin and soft tissue to the fascia overlying the peroneus muscles.
- Identify intermuscular septum and locate perforators to the skin.
  - Dominant perforators are typically located in the middle and distal thirds of the fibula.
- Dissect along the length of the anterior aspect of fibula via elevation of the peroneus longus, peroneus brevis (which are retracted anteriorly), and extensor hallucis longus.
- Continuing dissection along the bone to the medial aspect reveals the interosseous membrane.
- Proximal and distal fibula bone cuts are made (at least 6–8 cm preserved on both ends to maintain ankle and knee stability and provide access for adequate vascular pedicle length).
- Making posterior skin cuts down to the subfascial plane at this point helps in fully mobilizing the fibula.
  - Visual identification and protection of skin perforator(s) are crucial to avoid compromise of the skin paddle.
- The fibula is retracted laterally exposing the interosseous membrane. Transection of interosseous membrane to separate the fibula from tibia exposes the peroneal pedicle below the membrane.
- Distal peroneal artery and veins are identified after the distraction of fibula. Ligate and transect distal pedicle.
- Dissect the pedicle from distal to proximal while separating the chevron shaped tibialis posterior muscle.
  - It is prudent to preserve a cuff of muscle along the fibula (as well along with the periosteum) as to not injure the multiple musculoperiosteal nutrient branches to the fibula bone.
- It is important to be aware that the peroneal vessels will begin to course medially in an oblique fashion as they near the junction of the posterior tibial artery.
- Transect the flexor hallucis longus muscle (leaving a cuff attached to flap) and ligate the pedicle proximally.

Table 9.4 Traditional flap inset and orientation (other options are feasible depending on the soft tissue constraints)

	Right fibula		Left fibula	
	Anterior exiting vessels	Posterior exiting vessels	Anterior exiting vessels	Posterior exiting vessels
Right mandible defect	Skin paddle positioned over neomandible for mucosal defect	Skin paddle inferior to neomandible for external defect or transposed for mucosal defect	Skin paddle inferior to neomandible for external defect or transposed for mucosal defect	Skin paddle positioned over neomandible for mucosal defect
Left mandible defect	Skin paddle inferior to neomandible for external defect or transposed for mucosal defect	Skin paddle positioned over neomandible for mucosal defect	Skin paddle positioned over neomandible for mucosal defect	Skin paddle inferior to neomandible for external defect or transposed for mucosal defect

  - It is typically most useful to ligate the peroneal pedicle near its take off to maximize pedicle length as well as vessel diameter; however, a small stump of the pedicle should be left in situ to avoid damage to the remaining tibioperoneal trunk.
- The flap is now harvested in preparation for contouring, insertion, and anastomosis (Table 9.4).
- Fibula donor site is closed by first reapproximating the lateral compartment muscles to the soleus. The remainder of the wound is closed in a layered fashion in a tension free manner. Skin grafting may be required.

Use of Microsurgery in Head and Neck Reconstruction

Recipient Vessels

- Commonly, the vascular system proximal to the site of reconstruction or within the reach of the vascular pedicle of the chosen flap is utilized.
- Occasionally due to prior operation, radiation therapy, or poor vessel quality, more distant vessels must be utilized.
- Commonly, the superficial temporal artery and vein are used for upper face and scalp reconstruction.
- The facial artery and vein or external jugular vein are convenient vessels for oral cavity and lower facial reconstruction.
- The anterior jugular veins should be used with caution when the patient has undergone tracheotomy placement as these vessels may be compromised inferiorly.
- The internal jugular vein and its branches routinely provide improved caliber veins with higher vessel pressures.
- Commonly vein branches sized at least 3 mm in diameter can be found along the internal jugular vein.
- The lingual and superior thyroid artery can be used when the facial artery is not acceptable and the transverse cervical artery at the base of the neck can be utilized when no other superior options are available.
- In cases where a neck exploration is required, careful handling of the tissue is imperative to avoid injury to the recipient vessels. The vessels should be gently tied with silk ties or vascular clips and dissected sharply.
- Electrocautery or high-energy cautery including harmonic scalpels should not be used directly on the recipient vessels as this increases the risk for intimal damage and later thrombosis.
- Excessive handling of the vessels during dissection can lead to shearing, tearing of branches and uncontrolled bleeding, spasm, and delamination of the intima of the vessel.
- Patients with atherosclerosis or prior radiation may have friable vessels with sclerosis of the adventitia. These vessels are more prone to dissection and thrombosis. Any defect noted within the lumen of the vessel should be

trimmed back until the vessel demonstrates a smooth, uniform intima.

Anastomosis

- The most common type of anastomosis is the end-to-end anastomosis of the donor and recipient vessel.
- End-to-side anastomoses are also relatively common.
- Most of the flaps used within the head and neck have a vessel caliber similar to those in the neck.
- Techniques to overcome vessel discrepancy include [16]:
  1. Trimming the smaller vessel back (2:1 discrepancy).
  2. Dilating the smaller vessel gently (2:1 discrepancy).
  3. Spatulation – the end of the smaller vessel is incised longitudinally. The apex of the incision is sutured first to the recipient vessel and the anastomosis is completed allowing accommodation for the vessel size (between 2:1 and 3:1 discrepancy).
  4. Beveling the edge of the smaller vessel, no more than 30° (between 2:1 and 3:1 discrepancy).
  5. End to side anastomosis (>3:1 discrepancy).
- Vessels are typically sewn with 9-0 or 10-0 nylon suture on a tapered-point needle.
- While hand sewn venous anastomoses are still common, the use of vein couplers has simplified vein anastomosis. Several coupler diameters are available and chosen based on the best fit to both the donor and recipient veins. The couplers may also be equipped with a Doppler sensor to facilitate flap monitoring post-operatively.
- Couplers are best used for vessels more equal in size, but some surgeons have used it up to a 3:1 discrepancy.
- They have been shown to result in completion of anastomosis 4–5 times faster than hand sewing.

Flap Vitality

- The most common cause of vascular failure of a flap is venous congestion vs. arterial thrombus (4:1).
- Most congestion occurs within 48 hours of surgery.
- After 12 hours of ischemia, free flap salvage is not possible. It is common to have hourly checks for the first 24 hours and 2-hour checks for the next 48 hours.
- The rate of flap necrosis is around 4% [17].
- Hemostasis is imperative since hematomas can result in external compression of the vessels resulting in occlusion.
- It is important to minimize vasoactive medications that can result in vasospasm within the flap.
- Since veins are low-pressure vessels, they are susceptible to torsion and external compression by hematomas.
- Redundant vessels may appear to lay flat when the head is turned and the neck extended during the operation. But, when the neck is in a neutral position or flexed, the vessels may become tangled and occluded.
- Delayed arterial thrombosis is less common. Several pharmacological agents have been described to reduce thrombotic complications, but no regimen has been established as superior:
  - Aspirin blocks thromboxane A2 production that has vasoconstrictor activity and aids in platelet binding. This is usually started immediately after surgery and the length of treatment postoperatively varies widely but commonly lasts between 30 and 90 days.
  - Heparin binds to antithrombin III which causes an increased activity, preventing the activation of factor IIa, XIIa, IXa, and Xa [5]. Heparin is not typically utilized due to the risk for bleeding and hematoma formation which could have a devastating effect at the site of the micro-surgical anastomosis. Heparin irrigation is commonly used as a vessel irrigant given its highly negatively charged state and affinity for the vessel wall and its ensuing antithrombotic effects.

  - Dextran has been described in vascular and microvascular surgery to improve vascular patency. Low molecular weight dextran has both an electrochemical and rheological effect on the vessel wall and red blood cells respectively. There is concern for antigenicity and a test dose is usually given before an infusion is started. Non-cardiogenic pulmonary edema, respiratory distress syndrome, renal damage, and cardiac overload have been associated with dextran infusion [5, 18].

Flap Monitoring

- If there is evidence of impaired venous drainage of a flap or decreased arterial inflow, the most common problem is structural in nature. The pedicle may be compressed or twisted. The perforating vessels may be twisted or stretched too tightly. The vein may be kinked or compressed by a hematoma. In any one of those instances, the patient should be taken back to the operating room for exploration of the flap as soon as compromise is identified.
- The earliest signs of flap vascular congestion may be increased turgor or a faint bruise within the flap. This can progress to diffuse and dark ecchymosis as the problem advances. Often, the Doppler signal will remain normal or near normal until late stages of congestion, and therefore, should not be solely relied upon for flap monitoring.
- Flap color and character of bleeding on pin prick are perhaps the most important tools in the diagnosis of vascular compromise in the postoperative period.
- Methods of flap evaluation include:
  - Clinical Evaluation: Pinprick, surface temperature (difference of 3 °C associated with arterial insufficiency and 1–2 °C with venous insufficiency) [18], capillary refill, turgor of tissue, serial photography (taken time of surgery when well perfused and during flap checks to evaluate changes).
  - Doppler: internal, external, and laser.
  - Pulse Oximetry: Commonly quoted, best to monitor digit.

Pinprick Test

- Medium gauge needle used to pierce flap.
- Arterial occlusion will have minimal to no bleeding. The turgor of the tissue is decreased due to the lack of inflow of blood.
- Venous occlusion will cause a rapid bleed of dark blood. The turgor of the tissue is increased due to inability to clear venous blood.

Tracheostomy

- The term tracheostomy refers to the surgical creation of an artificial opening into the trachea.
- The indications for tracheostomy include the need to bypass the upper airway and in patients who require prolonged intubation, such as those with encephalopathy due to trauma or cerebrovascular disease. A tracheostomy both protects the laryngeal tissues and trachea from prolonged intubation, facilitates pulmonary physiotherapy and suctioning. It also allows for weaning of sedation while maintaining a secured airway. Additionally, it can be used for severe sleep apnea.
- The tube should correspond to the size of the patient's trachea (in general, a size 8 tube will work for most men and a smaller tube such as a size 6 will work for most women).
- In obese patients, extra-long (XLT) tracheostomy tubes with proximal or distal extensions can be used to reduce the risk of tracheostomy tube displacement.
- The cuff on the tube should be inflated to check for leaks and then lubricated with water-soluble lubricant to facilitate passage into the trachea.

Technique for Tracheostomy (Fig. 9.3)

- Extend the patient's neck to facilitate distraction of trachea out of thoracic cavity which also brings the anterior surface of the trachea closer to the skin. This may be contraindicated

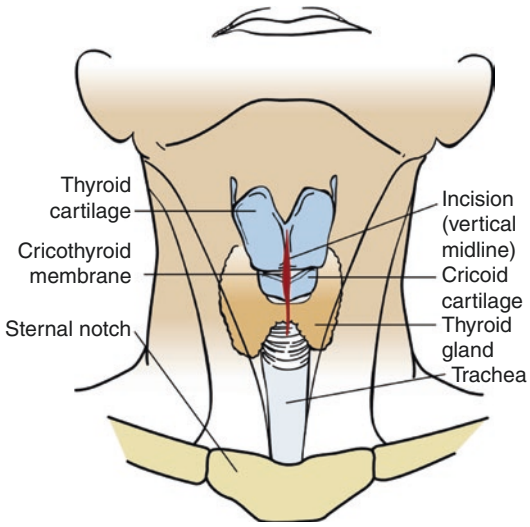

Fig. 9.3 Tracheostomy anatomy. (Reprinted with permission from Roden [54])

in certain cervical spine conditions such as injury or inflammatory spinal disorders or in cases of atlanto-occipital instability.

- Identify the thyroid notch, cricoid cartilage, bilateral sternocleidomastoid muscles (SCM), and sternal notch and mark.
- Approximately half-way between the sternal notch and cricoid cartilage, a transverse incision is marked. This can extend from the anterior edge of SCM to the contralateral anterior edge of SCM.
- The incision is then infiltrated with a local anesthetic containing epinephrine for hemostasis.
- The skin is then incised into the subcutaneous tissues. Careful blunt dissection is carried out in the subcutaneous tissues. The superficial layer of the deep cervical fascia should be divided vertically taking care not to damage the anterior jugular veins and branches. They can be retracted laterally or ligated and divided.
- The strap muscles are next encountered. The median raphe between the infrahyoid strap muscles are divided and retractors are placed to laterally retract the sternohyoid and sternothyroid muscles.
- The thyroid isthmus commonly lies within the superior aspect of this dissection and can either be retracted cephalad or ligated and divided. This can be accomplished by dissecting around the isthmus circumferentially and then applying silk suture ligatures or with electrocautery.
- After this is complete, the trachea is plainly visualized. The pretracheal fascia is divided.
- Next, the anesthesia team is advised to deflate the endotracheal tube cuff to avoid rupture of the cuff and a cricoid hook is placed from inferior to the cricoid cartilage. Gentle traction is then applied cephalad.
- 3-0 silk stay sutures can be placed prior to the incision; these facilitate retraction of the cut margins of the trachea and, therefore, placement of the tracheal tube.
- Using an 11 blade, a transverse incision is made through the membranous portion of the trachea between the second and third tracheal rings (above this the tube may erode through the cricoid cartilage leading to subglottic stenosis, below this there is risk to the mediastinal structures such as the innominate artery) [19]. A heavy tissue scissor is then used to transect one to two rings inferior to the transverse incision.
- Several incisions have been described, but a "T" incision is the simplest. A Bjork flap, which is an inferiorly based anterior tracheal wall flap, can be used when creating a tracheal stoma for long-term tracheotomy.
- The anesthesia team is asked to slowly withdraw the endotracheal tube. Once the tube clears the tracheotomy site, a tracheal dilator can be inserted to gently enlarge the opening.
- A lubricated tracheostomy tube is then inserted into the lumen of the trachea (lubrication reduces the likelihood of damage to the balloon, which can be easily damaged on insertion). This is held in place while the stylet is removed, and the inner cannula is inserted and ventilator circuit is attached.
- The presence of end-tidal CO_2, chest rise with ventilation, and bilateral breath sounds confirm placement.
- The tracheal tube flange can then be secured to the skin and tracheotomy ties passed around the neck for added security.

Complications

- *Bleeding* – usually due to untreated injury to the anterior jugular vein or inadequate control of the highly vascular thyroid. It commonly occurs during incision into the trachea (the critical portion of the procedure when the airway is not protected) and is usually caused by small vessels along the lateral trachea. Extensive electrocautery should be avoided here as the recurrent laryngeal nerve travels in the area of the tracheoesophageal groove. Use of a bipolar cautery can be useful for controlling bleeding in this location. Some surgeons advocate the application of Surgicel® once the tracheal tube is in place. This may help with minor nuisance bleeding post-operatively, but caution should be exercised during tracheal tube change as these friable pieces of Surgicel® can become dislodged into the trachea and cause airway obstruction.
- *Wound Infections* – are possible as these wounds are contaminated with airway flora. Local wound care with dry sterile dressings or wet to dry dressings and antibiotic coverage usually suffice.
- *Pneumothorax and Subcutaneous Emphysema* – can occur due to wide dissection done during tracheostomy, tracheostomy tube displacement, and early ventilation prior to tube security. Must identify source of air (rule out hole in trachea – posterior wall tear commonly reported) or displacement of tracheostomy tube. Repair of the hole in the trachea and bypassing the hole with the tube are possible options. Pneumothorax, if small, can be treated with watchful waiting and serial chest X-rays. If the pneumothorax is large, emergent needle decompression with chest tube placement is indicated. Subcutaneous emphysema, once source controlled, will gradually resolve. Subcutaneous fenestrated catheters can be placed for severe cases. Consider warm heat and antibiotic coverage.
- *Tracheal Tube Displacement* – the most devastating complication. This can occur during initial placement or postoperatively and should be recognized promptly. Intraoperatively, a lack of breath sounds, lack of chest rise, or lack of end tidal CO_2 necessitates prompt re-evaluation of tracheal tube placement. Post operatively respiratory distress, inability to pass a suction cannula, or poor oxygenation requires rapid evaluation of the position of the tracheal tube in a controlled environment. A bronchoscope can be useful for visualization, but the surgeon should not waste time with these maneuvers if the patient is in respiratory distress. In the case of a dislodged tracheal tube, an attempt at re-securing the airway via translaryngeal intubation should be undertaken. If a displaced tracheal tube is attempted to be replaced prior to maturation of tracheostomy tract (rule of thumb prior to 7 days), it may create a false passage into the neck. Once the airway is secured, re-exploration of the tracheotomy in a controlled environment can be accomplished and the tracheal tube replaced.
- *Tracheal Stenosis* – is linked to prolonged endotracheal intubation and wide dissection of the trachea during tracheotomy. Surgical options include tracheoplasty or laser excision of scar tissue.
- *Tracheo-Innominate Fistulas* – can result in life-threatening bleeding. It results from the tracheal tube eroding through the anterior wall of the trachea and the posterior wall of the innominate artery as it crosses the trachea. A "herald" or "sentinel" bleed can precede a massive exsanguinating hemorrhage; but this is not always the case. Over inflation of the tracheotomy cuff may aid in stopping the bleeding. If a patient is bleeding and the innominate artery is the suspected source, the Utley maneuver is indicated (maneuver by which a gloved finger can be inserted into an enlarged tracheotomy incision and used to tamponade the innominate artery against the sternum or proximal clavicle until a thoracotomy can be performed).
- *Tracheocutaneous Fistula* – formation of a fistula from the skin into the trachea. Commonly seen in patients who have been cannulated for about 1 year [20]. Treatment is to remove the scar tissue. The strap muscles are then dissected and repositioned over the trachea.

Closure of skin and subcutaneous tissue performed after.

- *Tracheoesophageal Fistula* – formation of a fistula between the posterior wall of the trachea and the anterior wall of the esophagus. These commonly require an interpositional muscle flap for repair of the esophagus with prolonged bypass of the esophagus with a nasogastric tube.

Muscular Flaps

- Local Flaps – flaps created with tissue adjacent to the defect.
- Regional Flaps – located at a distance from the donor site with its own bloody supply.

Pectoralis Major Myocutaneous Flap

- Can be used to reconstruct soft tissue defects of oral, oropharyngeal, skull base, esophageal, partial tracheal, and pharyngeal defects. Also used to cover exposed major vessels and cutaneous defects of the neck and gain additional soft tissue neck bulk after radical neck dissection.
- Pectoralis major provides adduction and internal rotation of arm. Expected to have some functional postoperative decrease in arm and shoulder function.
- Arc of rotation limits ease of use and normally best for defects anterior to the retromolar region of the jaw and inferior to the ear lobe [21].
- Good alternative for those who are not free flap candidates or for flap salvage.
- Can be harvested with rib to reconstruct segmental defect.
- One stage surgery, with possibility of two teams.
- Distinct color mismatch to the face and hair growth (in male patients) in areas that are normally bare.
- Creates a bulge over the clavicle and neck that will atrophy over time due to denervation.
- Arterial Supply: Pectoral branch of thoracoacromial artery, lateral thoracic artery, superior thoracic artery, and intercostal artery (Fig. 9.4).
- Motor innervation to the muscle from the medial and lateral pectoral nerves (branches of the brachial plexus).
- Venous drainage via venae comitantes of the accompanying arteries that drain into the axillary vein.
- Sensory innervation via the anterior cutaneous branches of the intercostal nerves.
- A 6 × 6 cm flap can be harvested in men without need to skin grafting. This can be doubled in females [21].
- In patients with Poland syndrome, the flap cannot be utilized on the ipsilateral side of the defect. Poland syndrome is a congenital anomaly characterized by unilateral hypoplasia of pectoralis major and minor with associated unilateral brachysyndactyly.
- In patients with breast implants, discussion of removal of implant should be forewarned.

Technique

- First measure the defect of the recipient site.
- A string/suture is placed at the clavicle and swiveled to the margin of the defect point to aid in marking the most inferior portion of the skin paddle.
- The dimensions of the paddle are transferred to the donor site medial and inferior to the nipple. In females, the inframammary crease corresponds to the inferior edge of the skin paddle. (A medial or lateral incision is now decided. A lateral incision is more cosmetic and allows for a wider arc of rotation but closure in females may be more difficult due to need for retraction of breast tissue.)
- Incision is carried through the skin and subcutaneous tissue on the lateral aspect of the incision.
- Dissection is carried down to the pectoralis fascia.

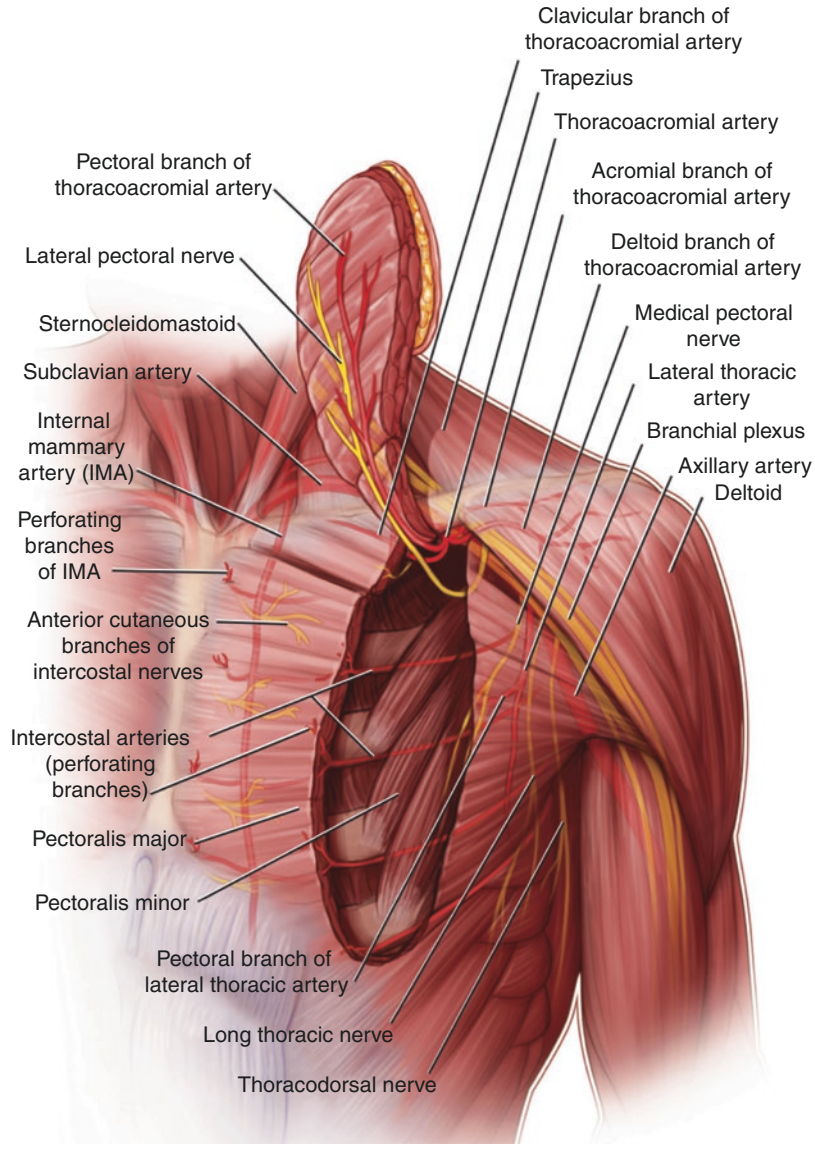

Fig. 9.4 Pectoralis flap anatomy. (Reprinted with permission from Wei and Wai [22])

- The curvilinear extension of the flap is now dissected laterally toward the free margin of the pectoralis major muscle and the insertion of the muscle superiorly and medially toward the sternal and clavicular origins of the muscle.
- The remainder of the skin paddle incision is now completed, and the skin paddle is sutured down to the fascia.
- The flap is now elevated between the pectoralis major and minor muscles. The inferior attachment of the pectoralis muscle to the ribs must be released.
- Once the flap is released, the pectoral nerves are released to aid in arc of rotation and to induce atrophy of the muscle to improve esthetics by reducing bulk.
- Lateral attachment to the humeral insertion is released via blunt dissection.
- A tunnel is now created into the head and neck. A subplatysmal plane of dissection is carried over the clavicle. Most circumstances neck exposure is part of operative field. If sternocleidomastoid still present, the plane is superficial to this muscle. Tunnel should be of

adequate size and typically should accommodate the width of four fingers.
- The flap is now inset.
- Chest is closed in layers.
- A skin graft may be required depending on defect size.

Complications
- More common complications include infection, hematoma/seroma, and wound dehiscence.
- *Flap Necrosis* – must ensure no twisting of flap. Adequate tunnel width to the neck is necessary to avoid pressure on the flap. Avoid tight bandages. Treatment with hyperbaric oxygen has not shown to provide much improvement. For partial flap necrosis conservative treatment with debridement of necrotic tissue with wet to dry dressings. Consider hyperbaric oxygen for partial necrosis. For complete necrosis, flap should be brought down and a new flap should be performed for reconstruction.
- *Osteochondritis* – more commonly seen in patients that have donor site repaired with skin graft [23]. The cause is thought to be due to rib exposure leading to infection and loss of blood supply to the periosteum (due to disruption of thoracoacromial artery). Treatment involves IV antibiotics and debridement/resection of osteocartilaginous segment.
- *Hematoma/Seroma* – large undermining for flap development can lead to large dead space [23]. Drain placement and achieving hemostasis is paramount. Compression bandaging leads to concern for flap compromise. If seroma forms, serial aspirations may be preferred. If minor hematoma, aspiration with mild pressure bandaging is indicated. Major hematomas require identification and control of vessel.
- *Pneumothorax* – if air leak is noted on Valsalva maneuver, a suction catheter should be placed into breach. A purse string suture is then placed, and under suction the catheter is removed, and the purse string is tightened. Small tears will normally resolve on their own. Serial chest X-rays are taken to observe progression. A large pneumothorax or instability of patient will require a chest tube.
- *Shoulder Dysfunction* – shoulder weakness with difficulty in adduction and rotation of arm is commonly seen. Preservation of clavicular portion may reduce functional disturbance. Physical therapy may aid in regaining partial function. If neck dissection done concomitantly, this may be a complication of this procedure.

Temporoparietal Fascia Flap

- Supplied by superficial temporal artery and vein. Vein runs on the surface of fascia and anterior to the artery. Vein runs superficial to the fascia while arteries covered by fascial fibers. Superficial temporal artery bifurcates 2–4 cm above the zygomatic arch.
- Innervation is from the auriculotemporal nerve.
- Temporal branch of the facial nerve runs deep to the temporal fascia. It can be mapped by a line drawn starting from the tragus to a line 3 cm above and 2 cm lateral to the supraorbital rim surgically. The frontal branch is noted to be 1.5 cm lateral and superior to the eye brow [24].
- Very thin flap, 2–4 mm [25], difficulty in dissecting the superficial portion.
- Up to 12 × 14 cm size, making it difficult to use for large defects [25, 26].
- Thin pliable tissue with good extension.
- Great for orbital, auricular, and maxillary reconstruction.
- Flap can be harvested with cutaneous tissue (skin/forehead) or cranial bone.

Technique
- The superficial temporal artery can be identified and mapped using doppler if desired.
- The incision is marked through a preauricular crease in front of the tragus and extends superficially into a hemi-coronal incision (the incision should be parallel to the direction of the hair follicles).
- Superficial dissection through the subcutaneous fat adhered to the fasciae. Anterior exten-

sion is done to a safe length to the expected course of the frontal branch of the facial nerve. Superiorly the extension is carried to the vertex of the scalp.
- Check the arc of rotation with a suture or lap sponge from the preauricular area.
- Release the fascia with the desired pedicle from the underlying temporalis muscle. The galea is separated from the temporoparietal fascia along the margins unto the subgaleal supraperiosteal layer.
- The release is completed in the subgaleal areolar tissue (Merkel's space) down to the zygomatic arch [25].
- A subcutaneous tunnel should be formed to allow extension of flap to defect without putting pressure on the flap.

Complications
- *Transient or Permanent Alopecia* – avoided by deepening the dissection and not dissecting hair follicles.
- *Frontal Branch of Facial Nerve Damage* – Nerve innervates the frontalis muscle and runs deep and transversely along the temporoparietal fascia. Avoid taking flap anterior to the temporal hairline.
- *Temporal Hollowing* – may volumize bone with hydroxyapatite, fat grafting, or custom implants.
- *Flap Failure* – uncommon, risk if pedicle compromised during harvest or kinking of vessel on rotation. Normally, does not leave a defect due to thinness of flap. May treat area of involvement conservatively with debridement if easily accessible. Alternative flap may be required to treat defect.

Other Regional Flaps

Paramedian Flap/Median Forehead Flap

- Color of forehead makes a great match for face and nose.
- Can incorporate supratrochlear artery as part of flap. Artery is approximately 2 cm from the mid glabella [27].
- Supratrochlear artery emerges from supratrochlear foramen, travels superficial to the corrugator muscles and deep to the orbicularis oculi. It ascends 2 cm before piercing the frontalis muscle [28].
- Median forehead flap designed to capture both supratrochlear arteries while the paramedian flap is aligned vertically over the supratrochlear notch to capture a single side of the artery [28].
- The flap raised in supraperiosteal plane. The flap will contain skin, subcutaneous tissue, and frontalis muscle. If nasal reconstruction is planned, the area of nose should be thinned of most of the subcutaneous tissue.
- The pedicle width should be 1.5 cm [28].
- The forehead is closed primarily after careful undermining. Any areas that are not primarily closed on the forehead will heal by secondary intention.
- The division of the pedicle is most reliably done at 3 weeks [27]. Flap viability can be tested by tying a rubber band to the flap and evaluating color/perfusion.
- Must rule out prior history of cutaneous malignancies within the expected skin paddle. Also ensure no scars are within the potential region of the pedicle.

Melolabial Flap/Nasolabial Flap

- A cutaneous flap harvested from the skin lateral to the melolabial crease. Can be harvested with a superior or inferior base, depending on the defect site.
- Superior flaps commonly used to reconstruct nasal, palatal, or oral sulcular defects.
- Inferior flaps are mostly used to address lip, floor of the mouth, and buccal mucosa defects [29, 30].
- It is supplied by the branches of the facial artery and is drained via the facial angular vein [30, 31]. The facial artery runs deep to the mimetic muscles [30, 32].
- Advantage: good color match especially of the skin of the lips and caudal lateral nose [31]. The scar can be positioned parallel or in the melolabial fold that can provide cosmesis.

- The incision is placed 1–2 mm lateral to the melolabial fold to prevent a flattened appearance.
- Most commonly used for cutaneous reconstruction. The flap is rotated into place (a tunnel flap for single stage has also been described) and 3 weeks later the pedicle is divided.
- Intraoral defects require transbuccal tunneling. It is imperative to deepithelialize the tunneled portion of the flap.
- Possible ectropion and scleral show are dependent on the extent of the flap. Scarring may cause facial asymmetry. There is limitation in the arc of rotation.

Facial Artery Musculomucosal Flap (FAMM Flap)

- Based on the facial and angular arteries.
- Can be based inferiorly (anterograde flow) – best for floor of mouth, tongue, gingival, alveolar, and lower lip reconstruction.
- Can be based superiorly (retrograde flow) – best for palate, skull base, conjunctiva, intranasal lining of nose, nasal septum, and upper alveolar defects [30, 33].
- The flap can be up to 2 cm wide but must take into consideration the position of Stensen's duct.
- The mucosal surface can be utilized for moderate tongue defects with the advantage of no visible scars.
- There is limitation in the arc of rotation, a second stage surgery required for division after 3 weeks and may limit mouth opening [33, 34].

Tongue Flap

- Can be a random or axial flap.
- The axial flap is based off the dorsal-lingual branch of the lingual artery.
- Random flaps are most commonly used and include the dorsal tongue and lateral tongue flap.
- Dorsal tongue flap can be based anteriorly or posteriorly, fed by dorsal lingual artery [35]. Posterior based flap is best soft palate, posterior buccal mucosa, and retromolar region. Anterior based flap is best for anterior floor of mouth, lips, and hard palate.
- The random tongue flap can be as thin as 3 mm [36]. Flaps can be as thick as 10 mm and up to 2/3rd of the dorsum of the tongue can be raised up to circumvallate papillae [35, 36].
- Design should be 20% bigger than defect.
- The pedicle can be divided in 2–3 weeks and debulking (if required) is done at no earlier than 3 months [36].
- A double-door tongue flap has been described for large buccal mucosa defects. An incision is used to split the lateral tongue horizontally. The superior and inferior flaps are undermined and raised to cover the mucosal defects. Three weeks later the flaps are divided [37].
- The sliding posterior tongue flap is an axial flap based on the dorsolingual branch of the lingual artery. It is used to treat lateral tongue defects up to 6 cm by advancing the posterior tongue after releasing from the hyoid bone and the vertical septum [36].
- Patients with psychiatric disorders or cognitive delay may not be good candidates due to the need for tongue immobility.

Submental Artery Island Flap

- Based off the submental artery, a branch of the facial artery, and the submental vein.
- Useful for reconstructing facial skin, oropharynx, esophageal, nasopharyngeal, floor of mouth, retromolar, soft palate, and tongue defects and maxilla [38, 39].
- Advantages: Good color and texture match for cutaneous defects, rich vascularity allowing a flap as large as 15 × 6 cm [40]. The flap is thin and pliable with low morbidity and good cosmesis.
- Disadvantages: Risk of transfer metastatic lymph nodes at level I [41] and in males the beard hair may be a nuisance in non-hair bearing areas.

Buccal Fat Pad Flap

- Buccal fat pad is composed of three lobes (anterior, intermediate, and posterior). The posterior lobe has four extensions from the posterior portion: buccal, pterygoid, pterygopalatine, and temporal. The main body lies on the anterior border of the masseter [42].
- Fat contains some stem cells.
- Grafts normally epithelialize in 4 weeks with squamous epithelium.
- Utilized for small and proximal defects.
- Most commonly used for oral antral fistulae but uses include repair of oncological defects, cleft palate, drug induced osteonecrosis, and osteoradionecrosis.
- Blood supply from the buccal and deep temporal branch of the maxillary artery and the superficial temporal artery from the facial transverse branch [43].

Lip Reconstruction

- Lower lip is supplied by the branches of the facial artery: inferior labial artery, horizontal labial artery, and vertical labial artery. Sensation is from the mental branch of the inferior alveolar nerve. Motor innervation provided by marginal mandibular branch of the facial nerve [44].
- Upper lip is supplied by the superior labial artery of the facial artery. Sensation is from the infraorbital branch of the maxillary division of the trigeminal nerve. Motor innervation from the zygomatic branch of the facial nerve.
- Reconstruction of lip is commonly described as a simple algorithm based on the size of the defect.
- Lip excisions are commonly combined with vermilionectomies (also known as a lip shave).
- Lip shave procedures are indicated for cases of actinic cheilitis, leukoplakia, or carcinoma in situ [45].
- Irregularity of the vermilion, as little as 1 mm, may be noticed by an observer speaking at a distance [45].
- Lip-switch flaps result in a smaller oral opening.
- Loss of 50% of one lip only results in a 25% decrease in the total oral circumference.
- Almost complete regain in the sensation of lip with return of pain, touch, and temperature (cold then hot) over first year [46].

Upper Lip

Defects 1/4 of the Upper Lip

- V, W, shield, and pentagonal incisions are commonly used for primary closure of defects [45, 47].
- A wedge excision will cause some asymmetry of the upper lip. Cosmesis of upper lip may be compromised due to three esthetic subunits of upper lip (philtrum and two lateral segments) [45]. Consider Abbe flap or lip switch for cosmesis.
- T- Excision (Fig. 9.5): Bilateral advancement flap for upper lip. Most commonly used for centrally located lesions. A rectangular excision is made surrounding the lesion. The horizontal portion of the flap is created in the nasal sulcus with Burow's triangles relieved in the

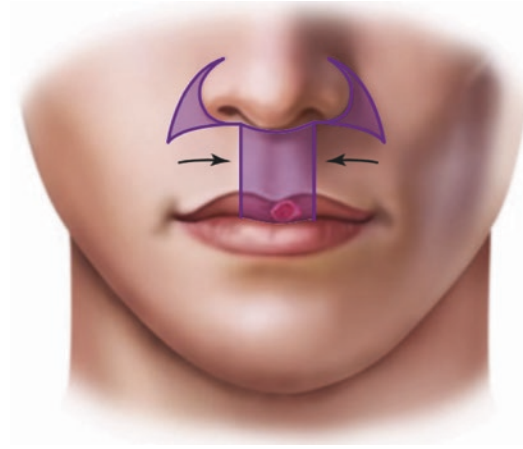

Fig. 9.5 T-excision. The lesion is excised in a full-thickness fashion leaving a defect in the central upper lip. Partial-thickness perialar excisions of skin and subcutaneous tissue can be used to gain further advancement of the wound edges. (Reprinted with permission from Urbanek and Bailey [45])

crease of the nasal alar folds. The edges are advanced medially. The philtrum and cupid's bow are lost in this procedure.

Lower Lip

Defects 1/3 of Lower Lip

- V, W, and shield excision more commonly used. Wedge resection is more acceptable in the lower lip.
- Lateral (rectangular) advancement flap (Fig. 9.6): Used more commonly for centrally located lesions. A rectangular incision is made, and a full thickness incision is made along the labiomental crease. Burow's triangles are excised to facilitate closure. The edges are advanced medially for closure [45, 46].

Defects 1/3 to 2/3 of Upper Lip

- Abbe Flap (Fig. 9.7): Cross lip transfer of full thickness lip tissue based on the inferior labial artery. Triangular wedge from lower lip is designed to reconstruct a wedge excision of the upper lip region. The flap is raised, transposed 180°, and inset. The donor site is closed directly. Division occurs 21 days later. The width of the wedge is about ½ designed slightly smaller, but height should match [46–48].
- Primary closure if less than 50% of lip, but unaesthetic.
- Karapandzic Flap (Fig. 9.8): A rotation neurovascular advancement flap, good for 2/3 or subtotal reconstruction of the upper or lower lip. The lesion is resected in a rectangular full thickness fashion. Semicircular partial thickness incisions in skin and mucosa are performed from the edge of the skin defect toward the nasolabial and labiomental folds bilaterally. The vasculature and the nerves are identified and released to allow stretch without damage. The facial nerve branches, sensory branches of CN V2 and V3, and superior and inferior labial arteries are preserved as incisions are not of full thickness in the area of the flaps [41, 45–49]. This does lead to microsomia as no additional tissue is recruited.
- McGregor Flap: Modification of Gilles (see below). Flap is rotated around the commissure and transfers tissue from the melolabial region, which prevents microstomia. Does not restore natural looking vermillion [49].

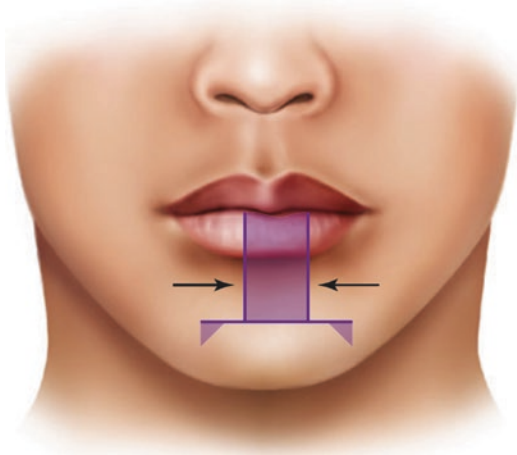

Fig. 9.6 Lateral advancement flap. The lesion is excised in a full-thickness fashion and the wound edges are advanced medially. Burow's triangles may be incorporated into the resection to prevent bunching of the tissue during wound advancement and closure. (Reprinted with permission from Urbanek and Bailey [45])

Defects 1/3 to 2/3 of Lower Lip

- Stein Flap (Fig. 9.9): Double Abbe flap with preservation of the philtrum but harvesting on either side of the philtrum [45]. Leads to greater denervation of the reconstructed lip and not particularly esthetic, less favored.
- Reverse Abbe Flap: Abbe flap based on the superior labial artery.
- Johanson Stair-Step Flap (Fig. 9.10): Good for defects up to 2/3 of lower lip. Can be raised unilateral or bilateral. Full thickness rectangular resection. A partial thickness stair step incision is made inferiorly and laterally with 8 mm × 10 mm blocks respectively. Burow's triangle is excised in the bottom of the staircase. The skin flaps laterally are undermined and brought medially.

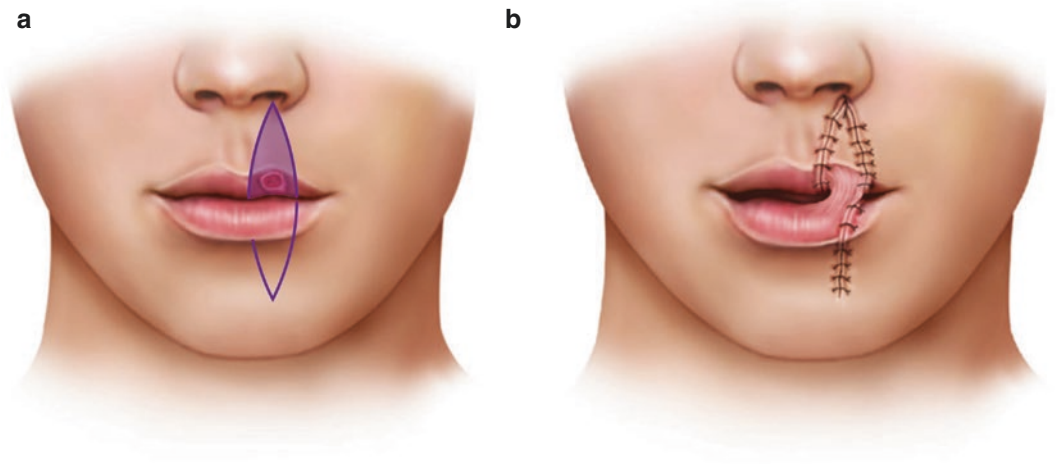

Fig. 9.7 Abbe flap. (**a**) Triangular excision of the upper and lower lip incision is made in a full-thickness fashion with extension up to the vermillion to preserve the vascular pedicle. Which is typically based medially. (**b**) The lower lip flap is then rotated superiorly and inset into the upper lip defect. The lower lip defect is closed primarily. After 21 days of healing, the vermillion tissue pedicle is divided and inset. (Reprinted with permission from Urbanek and Bailey [45])

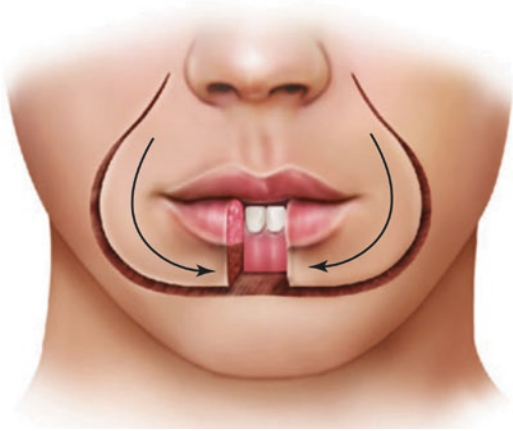

Fig. 9.8 Karapandzic flap. Incisions are carried along the labiomental crease and into the nasolabial creases. The incisions are made through the skin and subcutaneous tissue. Dividing muscle and mucosa as needed to gain mobility. The flaps are advanced medially and the defect is closed in a layered fashion. (Reprinted with permission from Urbanek and Bailey [45])

- Karapandzic Flap: See above.
- Gillies Fan Flap (rotation-advancement nasolabial flap) (Fig. 9.11): Transfers of tissue around the commissure toward defect. Based on the superior labial artery. Moves cheek and lip tissue. A rectangular resection of the defect. A curvilinear full thickness incision is extended laterally and superiorly running into the labiomental crease and the nasolabial fold. A full thickness flap is then rotated medially recruiting lip and cheek tissue. Will result in blunting of the commissure.
- Modified Webster-Bernard Flap (Fig. 9.12): Good for near total or total defects of the lower lip. Utilizes bilateral cheek advancement flaps. Places Burow's triangles in relaxed skin tension lines in the mesiolabial fold. Scar leads to "jump man" stick figure.

Defects of the Commissure

- Estlander Flap: Lip-switch flap for reconstruction of pericommissural defects of upper or lower lip. Single stage flap with no need for sectioning of the pedicel. Apex of the flap is made into the nasolabial or labiomental crease. The flap is transposed 180° from the upper lip to the lower lip or vice versa. This procedure results in a smaller oral stoma and indistinct commissure, which may require further revision.

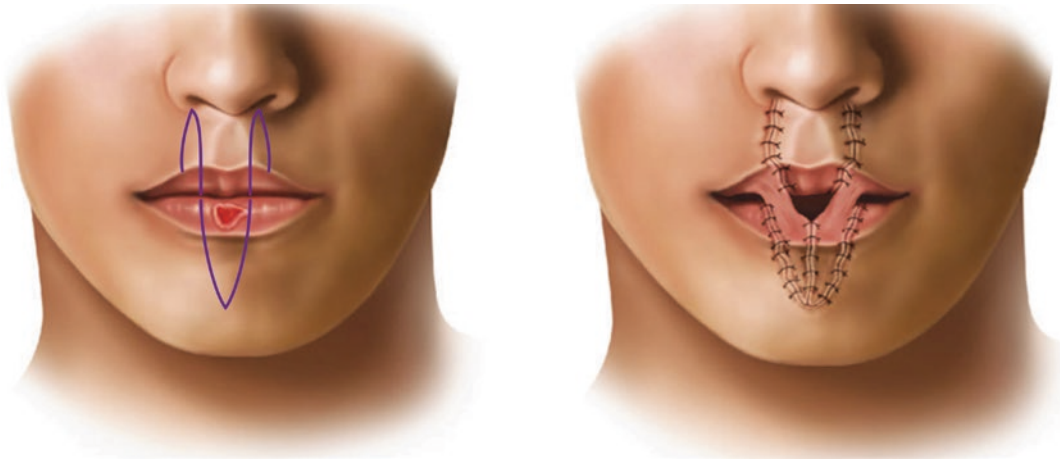

Fig. 9.9 Stein flap. Triangular flaps from the central upper lip is used to reconstruct a defect of the central lower lip. Like the Abbe flap, once sufficient collateral circulation has formed, the vermillion pedicles are divided and inset. (Reprinted with permission from Urbanek and Bailey [45])

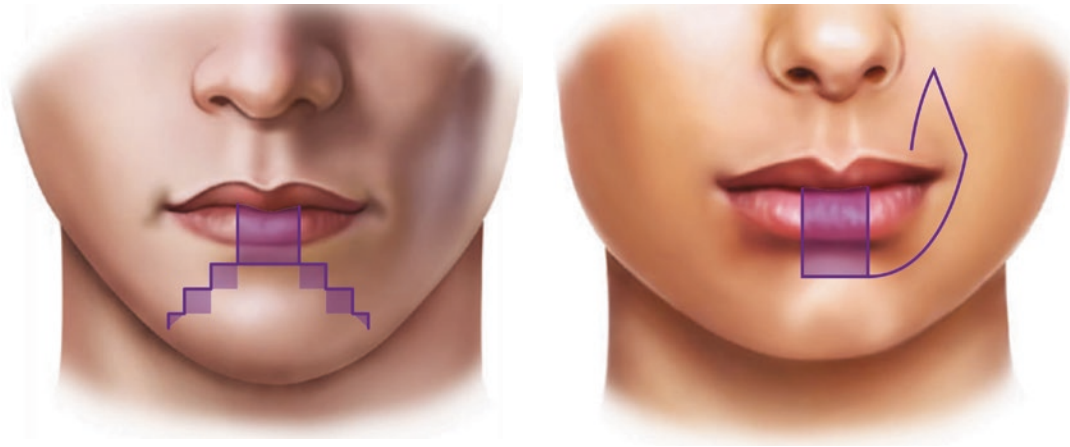

Fig. 9.10 Johanson flap. Full thickness rectangular incision of the lip defect. Partial thickness staircase incision design with undermining laterally. Burow's triangles are made at the bottom of the staircase to aid in medial movement of the flap. (Reprinted with permission from Urbanek and Bailey [45])

Fig. 9.11 Gillies fan flap. The flap is designed to transfer tissue from around the commissure. A full-thickness flap is then raised and rotated medially to bring lip and cheek tissue to the defect. (Reprinted with permission from Urbanek and Bailey [45])

Complete Lip Reconstruction, Upper and Lower

- Radial Forearm Free Flap: Thin, hair free, pliable skin flap with ease of harvest. Color match is acceptable. Thin tissue and lack of lip definition are esthetic drawbacks. Loss of vermillion but this can be recreated with medical tattooing [47, 50].
- Anterior Lateral Thigh Flap: Thicker flap with ability to debulk as needed. Can retain sensation with lateral femoral cutaneous nerve harvested. Does contain hair bearing skin and increased variability in dissection, making it less desirable to use [50].

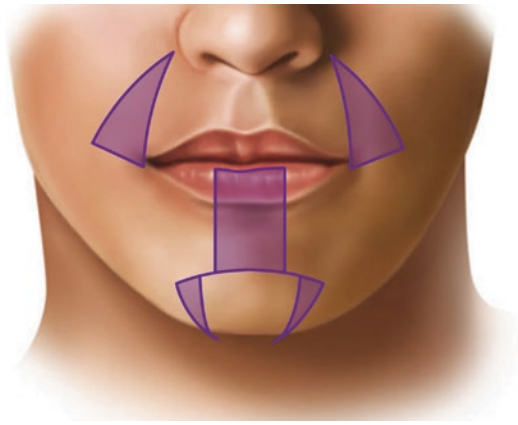

Fig. 9.12 Modified Bernard Webster Flap. Involves a full thickness excision of the pathological up to the labiomental crease. Four Burow's triangles of partial thickness are made at the melolabial crease and the labiomental creases to allow medial advancement of the tissue. (Reprinted with permission from Urbanek and Bailey [45])

Maxillary Reconstruction

- Reconstruction of the maxilla poses a unique challenge to maxillofacial surgeons as there are a wide variety of defects resulting from tumor ablation, trauma, or congenital deformities. Loss of midfacial structures are psychosocially and functionally impairing to patients, particularly when teeth, orbital contents, and facial pillars are involved.
- Several classification systems exist for describing maxillary defects and reconstruction, but perhaps the most widely used system by OMS is the Brown Classification, first described in 2000 then updated in 2010 [51, 52]. This is particularly helpful as it combines both horizontal and vertical defects of the midface with integration of dentoalveolar and functional deficiencies.

Brown Classification (Fig. 9.13)

- Vertical defect classification
  - Class I: Infrastructure defect of the midface, or maxillary alveolus.
  - Class II: Class I plus suprastructure and result in oral/antral communications.
  - Class III: Class II plus defect of the inferior orbital rim and floor
  - Class IV: Class III plus exenteration of orbital contents.
  - Class V: Orbitomaxillary defects.
  - Class VI: Nasomaxillary defects.
- Horizontal defect classification
  - Class A: central palatal defect
  - Class B: is ½ or less of the unilateral palate and alveolus
  - Class C: anterior maxillary defect
  - Class D: greater than ½ palatal and alveolar defect.
- It is important to recognize that achieving successful maxillary reconstruction requires separate goals across two distinct platforms. Oftentimes, achieving facial harmony is not congruent with establishing ideal occlusal arrangements and vice versa.

Goals of Maxillary Reconstruction

Facial form

- Restoring vertical and horizontal buttresses.
- Restoring soft tissue contours.
- Restoring smile.

Function

- Establishing a partition between the neck and aerodigestive tract.
- Establishing a partition between the sinonasal cavities and the oral cavity to allow for speech.
- Maintain oral competence.
  - Lip form for seal.
  - Labial vestibule for saliva.
  - Speech and swallowing.
  - Mastication.
- Using prosthetics as an adjunct for establishing the vertical appropriate positioning of the jaws is also appropriate.

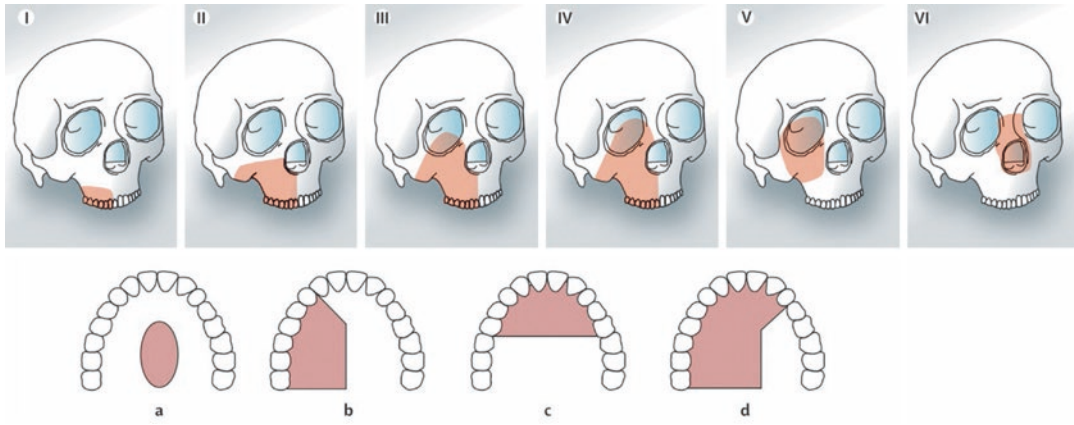

Fig. 9.13 Brown classification of maxillary defects. (Reprinted with permission from Brown and Shaw [52])

Defect Specific Reconstructive Algorithm

Brown Class I (Loss of Alveolus)

Goals: Optimize Alveolar Support

- Options:
  - Tooth supported prosthetics
  - Implant supported prosthetics
  - Local flaps (buccal sliding, palatal rotation)
  - Regional flaps (tongue flap, temporalis flap)
  - Free flaps (radial forearm flap, anterolateral thigh flap)
- This is generally an uncommon defect as it is a pure dentoalveolar or infrastructure defect. By definition, only soft tissue and alveolus below the level of the sinus are missing and several options exist for functional reconstruction. The soft tissue can be reconstructed with local/regional flaps, skin or mucosal grafts, or a small soft tissue free flap. The majority or all of the bony and dentate defects can be replaced prosthetically as the facial pillars are spared.

Defects

- IA defects – the primary goal is to seal the central oronasal communication and this can be done with vascularized soft tissue:
  - Palatal rotation flap
  - Tongue flap
  - Temporalis flap
- IB or IC defects
  - Fixed partial denture (FPD) on existing teeth or implants.
  - Removable partial denture (RPD)
- ID defects
  - FPD/hybrid prosthesis/overdenture (OD) supported with implants after sinus floor augmentation.
  - FPD/hybrid/OD using "All-on-4" configuration.
  - FPD/hybrid/OD using anterior implants and zygomatic implants.
  - Choosing between FPD/hybrid/OD

    Transition line vs. smile line – if smile line shows transition of teeth to gingiva, consider hybrid with pink porcelain.

Brown Class II (Loss of Alveolus and Malar Support)

- Goals: alveolar support, malar projection, nasal support, and seal oral cavity from the nasal and sinus cavities.
- Options:
  - Soft tissue Free flap
  - Osteocutaneous Free flap
  - Regional flap (Temporalis)
  - Zygomatic implant supported prosthesis
  - Obturator prosthesis

- Class II defects create an issue of both the available alveolar bone and also now sinus closure and the vertical and horizontal 3-dimensional buttressing that is critical for malar projection and facial support.
- There are also now diverging pathways for dealing with these issues. The first decision tree is whether the patient needs/wants teeth. If it is a smaller posterior defect, a soft tissue coverage plan may be sufficient. If teeth are needed or desired, then we must decide upon a prosthetic or reconstructed route.

Soft Tissue Reconstruction of Brown Class II Defects

- A soft tissue free flap provides immediate reconstruction and is straightforward. A single operation can close the antral/nasal communication and restore speech and swallowing. Additionally, it does not limit future prosthetic options as zygomatic implants can still be utilized for prosthetic support. The soft tissue option, however, is only useful in class IIB defects as there is no bony lip support for the anterior maxilla. Class IIC/D defects reconstructed this way will have a significant upper lip deformity resulting in poor esthetics and lip incompetence.

Osteocutaneous Free Flap Reconstruction of Brown Class II Defects

- In most cases where osteocutaneous free flaps are chosen for maxillary reconstruction, the fibula proves to be the most useful. The bony segments can be osteotomized to re-create the alveolus/dental arches and can accept dental implants at the time of surgery. The flap has a long pedicle length to tunnel into the neck for anastomosis and can be harvested with a skin paddle to reconstruct the mucosal defects.
- When designing the flap, it is tempting to place the bony segments at the vertical level of the adjacent alveolus; however, it is almost impossible to close the soft tissue over this as the fibula skin paddle is thicker than the adjacent mucosa. It is critical to under correct the alveolus height by a few millimeters to allow for passive soft tissue closure to prevent dehiscence or fistula formation from the nose/sinus.
- Positioning of the bone flap in relation to yaw, pitch, and roll can be difficult. Particularly with multiple segment reconstruction, as the flap typically fills the surgical field during inset and obscures the surgeon's frame of reference. The problem can be mitigated via wide exposure (Weber-Fergusson) and intraoperative CT scan or intraoperative navigation. This is especially important if implants will be placed in the fibula to ensure they are restorable.

Obturator Reconstruction

- Least surgically complex options such as bone, teeth, and soft tissue are replaced with one prosthetic. This may or may not be supported with dental implants.
- Functionally, it can seal the sinus, provide soft tissue facial support, is easy to hygienically maintain, and restore speech and mastication.
- The disadvantage, however, is that it requires a highly specialized prosthodontist or maxillofacial prosthodontist and can be expensive. In some cases, it cannot provide a complete seal and requires special care on the part of the patient to maintain cleanliness.
- One major benefit is that the obturator can be removed, so direct visualization of the tumor bed can be accomplished for tumor surveillance.

Brown Class III (Loss of Maxillary Alveolus, Malar Support, and Orbital Support)

- Goals: Alveolar support, malar projection, orbital projection, nasal support, seal oral cavity from the nasal and sinus cavities.
- Options:
  - Osteocutaneous free flap (fibula, scapula, DCIA).

  - Zygomatic implant supported prosthetic.
- The primary challenge in Brown Class III defects lies in reconstructing malar projection of infraorbital rim/anterior maxillary wall and alveolus. Though this can be accomplished with prosthetic and zygomatic implants, the author (A.P) favors osteocutaneous free flaps for several reasons: immediate hard and soft tissue reconstruction, possibility for implant supported dental reconstruction, and the ability to withstand radiotherapy (most Brown Class III defects are oncologic defects from tumors invading the bone).
- The double barrel configuration highlighted in the previous section can be utilized here, with one segment reconstructing the orbital rim and another for the alveolus. If needed, orbital floor mesh can be placed and fixated to the fibular segment at the rim.
- For cases where there is a through and through or complex soft tissue defect as well, the subscapular system is utilized for free flap reconstruction. The versatility and chimeric nature of the scapula osteocutaneous flap make it indispensable for complex composite maxillofacial reconstruction as multiple segments of bone and multiple soft tissue paddles can be harvested and oriented three-dimensionally.

Brown Class IV (Loss of Maxillary Alveolus, Malar Support, Orbital Support, and Orbital Contents)

- *Goals*: Alveolar support, malar projection, orbital projection, nasal support, seal oral cavity from the nasal and sinus cavities, seal intracranial cavity from nasal and sinus cavities, and obturate dead space.
- *Options*:
  - Osteocutaneous free flap (fibula, scapula, DCIA).
  - Double free flap (osteocutaneous fibula + soft tissue flap).
- Structurally, the class IV defect is similar to the class III defect, with the addition of an orbital exenteration defect. Oftentimes, these are resultant from ablative surgery of maxillary tumors invading the orbit and skull base.
- Most principles of reconstruction of class III defects apply here; however, one must take into consideration the possibility of an intracranial communication, either via the orbital apex or formal anterior skull base resection. If this is the case, as it often is, vascularized soft tissue is all that is needed to seal the dura/brain from the paranasal sinuses. The scapula "mega-flap," incorporating the scapular and parascapular skin paddles, scapula bone, and latissimus dorsi muscle, is of greatest utility in these cases.
- Alternatively, a double free flap may be used, such as a fibula osteocutaneous flap to restore facial and alveolar bone as well as the oral mucosal defect, and an anterolateral thigh flap to obturate the orbit and seal of the nasal cavity and sinus from the intracranial contents.

Flap Design and Considerations

1. Fibula
   - For maxillary reconstruction, using the contralateral fibula will orient the skin paddle toward the oral cavity and pedicle coming off the posterior of the flap. This allows for the most direct tunneling of the vessels into the neck while placing skin on the oral side.
   - In many cases (Brown II and above), there will be a resultant lateral nasal mucosal defect. The skin paddle can be partially folded to also reconstruct this wall with a small bridge of deepithelialized skin in between the mouth and nose.
2. Scapula
   - This is less critical as the orientation of the bone, skin, and vessels is much more flexible. Ease of harvest takes priority as the flap is usually raised with the patient in lateral decubitus.
   - If the scapula tip is being used based on the thoracodorsal vessels, be sure to flip the bone 180° so the vessels can come off posteriorly into the neck.
3. DCIA
   - Some surgeons prefer the ipsilateral iliac crest for maxillary reconstruction with vessels oriented posteriorly. This allows for the

thick crest to be positioned toward the oral cavity while the thinner cut surface can be contoured to the orbital rim and piriform aperture. In most cases, the internal oblique muscle only is harvested to reconstruct the soft tissues as the skin paddle is thick and fixed to the bone. The muscle is thin and flexible and is wrapped over the crest into the oral cavity to close the surgical fistula.

Case Example #1

Reconstruction Case

56-year-old male with history of tonsil squamous cell carcinoma treated with chemoradiation 5 years prior presents with nonhealing left oromandibular wound and exposed bone. Associated symptoms included trismus, foul smelling odor, and severe pain. Computed tomographic imaging showed a left mandible fracture with surrounding osteosclerosis. The diagnosis of osteoradionecrosis was made. The decision was made to take patient for composite mandible resection with immediate fibular reconstruction (See Fig. 9.14).

- *PMH: Tonsil cancer, GERD*
- *Medications: Omeprazole*
- *Allergies: NKDA*
- *PSH: None*
- *Social: Social alcohol. History of 1 PPD smoker. Works as a mechanic.*

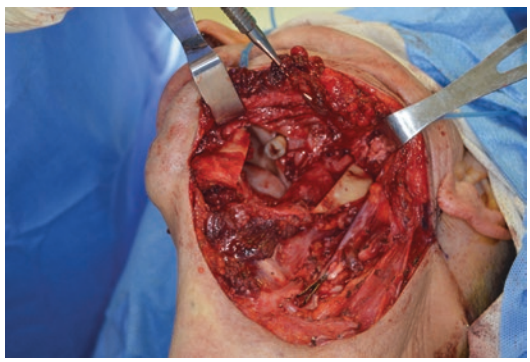

Fig. 9.14 Left mandible oromandibular defect after surgical resection. (Courtesy of Dr. Brett Miles)

- *Why is a fibula free flap a viable option for reconstructing the defect (Please comment on the defect pictured below)?*

  The composite mandibulectomy site displays a combined osseous defect with an adjacent mucosal and cutaneous defect. The fibular free flap would be ideal as the defect requires reconstruction of adjacent oral mucosa, skin of the face, and bone. The fibula can also be easily contoured with osteotomies for reconstruction of the neomandible. The bone stock that is available allows dental implant reconstruction. The fibula is a good osteocutaneous reconstructive choice for this patient due to his young age and lack of risk factors for peripheral vascular disease. Additionally, the lengthy pedicle is useful in radiated fields with low recipient vessel quality. A non-vascularized graft would not be appropriate due to the lack of vascularity in the tissue bed and exposure to the oral cavity and the neck greatly reducing its success.

- *How much bone can be harvested from the fibula?*

  Bone height varies from 9 to 15 mm with a total length of approximately 35 cm, typically up to 25 cm can be harvested.

- *Why would plate-only reconstruction not be a poor option for this patient?*

  Due to the fact that the region has poor vascularity, plate-only osseous reconstruction would have an unacceptably high wound complication rate.

- *What preoperative imaging would you request prior to fibular reconstruction?*

  A CT-angiogram of the lower extremities is indicated for preoperative confirmation of three-vessel flow for potential surgical candidates.

- *What other studies could you request?*

  Magnetic resonance angiography, ankle-brachial index screening, and doppler studies are alternatives.

- *What is a normal three vessel runoff?*

  A normal three vessel run of would show the popliteal artery branches into the anterior tibial artery, posterior tibial artery, and the peroneal artery. Commonly the peroneal artery branches from the posterior tibial artery (called the tibioperoneal take-off)

- *This patient underwent CT-angiogram of the lower extremities that demonstrated a normal 3-vessel runoff pattern. What modalities are available for postoperative flap monitoring?*
  - Temperature, color, turgor assessed on exam.
  - External Doppler, implantable Doppler, laser Doppler monitoring devices available.

- *What are the advantages and disadvantages of using a fibular free flap?*

  Advantages
  - Thick cortical bone that allows the fibula to tolerate mastication forces.
  - Large length of potential harvest allows for large segments of mandible to be reconstructed.
  - Large length of vascular pedicle allows the use of distant neck vessels.
  - Two teams may work simultaneously.
  - Skin can simultaneously be harvested with bone graft.
  - Higher success rate of vs. non-vascularized grafts
  - Does not rely on adjacent vascular bed.

  Disadvantages
  - Increased operative time
  - Increased cost
  - ICU admission
  - Height of fibula leads to difficulty in implant reconstruction.

- *What is a double barrel fibular flap?*

  It involves the removal of a 1 cm segment of the fibular bony segment and the bone flap is folded upon itself to increase the height of reconstruction.

- *The reconstruction was completed, and the donor site was closed* via *primary closure. The patient returned 3 weeks later showing evidence of wound dehiscence and muscle necrosis. What are the possible mechanisms for this presentation? How do you proceed?*
  - Compartment syndrome: Compartment syndrome is difficult to detect in early stages. Necrosis of underlying muscles may develop despite initial healing at the incision site and intact dorsalis pedis/posterior tibial pulses. Harvest inherently causes tissue damage and muscle ischemia. The resultant edema may cause intracompartmental pressure to exceed perfusion pressure of anterior and posterior tibial artery.
  - Iatrogenic trauma: Damage to the posterior tibial artery can occur during the procedure.
  - Congenital vascular anomaly: this should have been excluded in preoperative examination
  - Excessive wound tension (most common)

- *What should be done in the setting of wounds that may have excessive tension when closing?*

  A skin graft should be used whenever primary closure may produce excess tension. Another option includes the use of a tissue expander to expand the dorsal skin of the calf.

- *Patient presents with an equinovarus deformity as well as loss of sensation on the anterior and lateral calf and dorsum of the foot. What is the mechanism of injury and how could it have been prevented?* (Fig. 9.15)

  Injury to the common peroneal nerve can occur due to iatrogenic dissection or excess traction. The nerve wraps posterolaterally around the neck of the fibula, and beneath peroneus longus muscle, where it then splits into deep and superficial branches. It is most vulnerable when the proximal fibula and head are harvested. The best way to avoid this com-

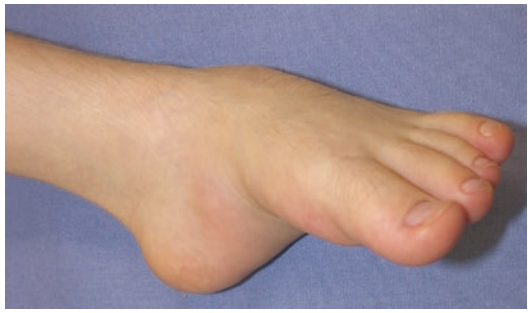

Fig. 9.15 Equinovarus deformity. (Reprinted with permission from Coughlin et al. [53])

plication includes identifying the nerve early in dissection as well as leaving 6–7 cm segment of bone attached to the knee.

- *A fibula was used to reconstruct a defect after resection of an ameloblastoma. Initial surgical margins appeared clear but final pathology review indicates one of the bony margins is positive. How do you manage this?*

  The pathology needs to be re-resected. After about 6 weeks, the skin paddle should have allowed adequate healing to the oral mucosa to seal the wound. The wound can be opened carefully through the neck incision (being careful not to injure the pedicle) and second non-vascularized bone graft can be applied to the new gap after re-resection of the positive margin or the proximal segment of the mandible can be rotated counterclockwise to establish bony continuity depending on the patient's anatomy, occlusion, and size of the re-resection.

- *What if the pathology being resected was a squamous cell carcinoma and the final path showed a positive bony margin?*

  Current National Comprehensive Cancer Network (NCCN) guidelines recommend re-excision when feasible. However, treatment should not delay adjuvant therapy. In cases where a positive margin is present, adjuvant chemotherapy and radiation therapy are recommended.

Case Example #2

Maxillary Reconstruction Case

48-year-old male with biopsy proven low grade adenocarcinoma of the right maxilla causing expansion of the hard and soft palate and crossing the midline (Fig. 9.16). *Mass had been increasing in size over the past 2 years. Associated symptoms included right maxillary jaw pain that radiates to the right ear causing 8/10 pain. Computed tomographic imaging showed a 6 × 6 × 5 cm mass centered in right maxillary sinus causing destruction of the lateral and medial walls of the right maxillary sinus and hard palate, extending to the right nasal cavity and abutting the right orbital floor* (Fig. 9.17). *The decision was made to take the patient for composite right maxillary resection with immediate fibular reconstruction.*

- *PMH: Gastroesophageal reflux disease.*
- *PSH: Right inguinal hernia repair at 4-years-old.*
- *Medications: None.*
- *Allergies: NKDA.*
- *Social: Denies tobacco and alcohol use.*

- *What are the surgical margins required for this entity?*

  The surgical margins require 1.5 cm.

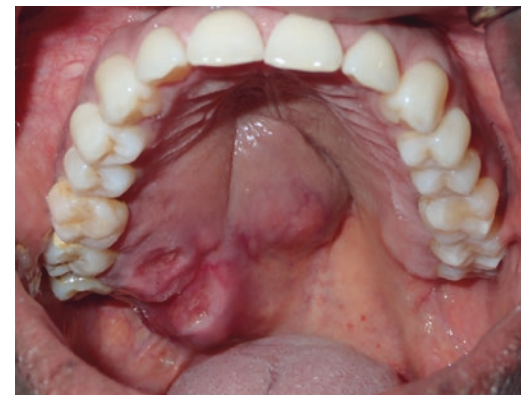

Fig. 9.16 Maxillary reconstruction case image. (Courtesy of Dr. Fayette Williams)

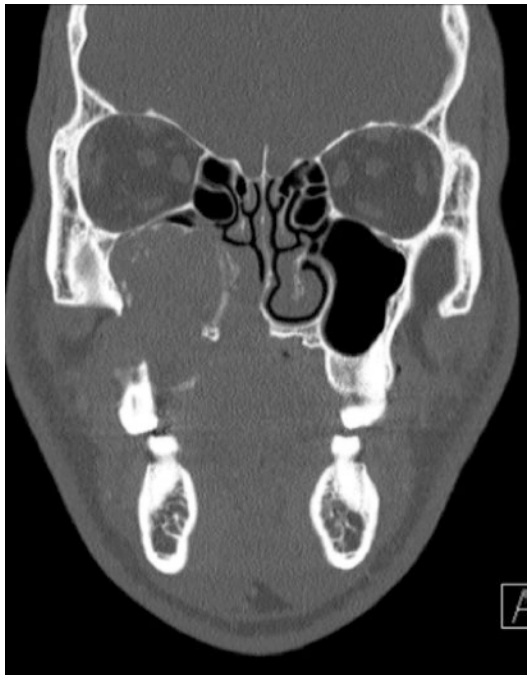

Fig. 9.17 Bone window CT, coronal cut for case #2. (Courtesy of Dr. Fayette Williams)

Fig. 9.18 Defect after resection of lesion of the right maxillae. (Courtesy of Dr. Fayette Williams)

- *Why is a fibula free flap a viable option for reconstructing the defect* (Fig. 9.18)*?*

  The maxillary resection created an osseous and mucosal defect. According to the Brown and Shaw Classification, this is a Class III D defect, which involves the loss of orbital support in addition to loss of cheek and dental arch support. The fibula free flap offers bony support to the orbit and facial skin, as well as use of skin paddle for oral mucosa that was included as a part of resection. Additionally, a fibula free flap offers reliable dental reconstruction with endosseous dental implant placement, immediately or delayed. Careful selection of osteotomy sites allows the fibula to be contoured and used for both orbital support and dental arch support, which extends across the midline. Furthermore, the length of pedicle is adequate to perform microvascular anastomosis without the need for vein grafting, as the pedicle must be tunneled to reach the neck.

- *What is the blood supply for the fibula free flap?*

  Blood Supply: Peroneal artery and venae comitantes.

- *What preoperative imaging would you request prior to fibular reconstruction?*

  A CT angiogram of the lower extremities to confirm three-vessel flow. The three vessels are anterior tibial, posterior tibial, and peroneal arteries. May use conventional angiography, MRA, or a doppler study.

- *What are the contraindications for fibula free tissue transfer?*

  Peronea arteria magna (where one dominant peroneal artery perfuses the whole foot), two vessel runoff, and open wound of the leg.

- *What are additional options available for maxillary reconstruction?*

Obturator

- Advantages: Offers immediate reconstruction, including dental reconstruction, of defect and provides tissue support to the face. Allows for easy surgical site surveillance.
- Disadvantages: Requires additional procedures to modify obturator as tissue heals and contracts. Not a viable option for a long-term orbital support. Poor fitting obturators are difficult to care for. Poor dexterity or trismus may render the obturator useless.

Lateral Scapula Free Flap

- Advantages: Osteocutaneous potential for both hard and soft tissue defects. Harvest site morbidity is low, scar is hidden. May use for maxillary or mandibular reconstruction, including orbital support. Potential for chimeric flap design to include the latissimus dorsi, cutaneous branch, and skin paddle.
- Disadvantages: Bone stock not as good as fibula, making dental implants more difficult. Possible shoulder dysfunction. Single team approach and thus longer surgery.
- Blood supply: Circumflex scapular artery which divides into periosteal, transverse, and descending cutaneous arteries and their respective venae comitantes, most frequently from the subscapular system.

Deep Circumflex Iliac Artery Free Flap

- Advantages: Provides bony structure that closely resembles mandible and may also be used for maxillary defects. Provides good bulk of bone facilitating dental implant placement. Can be used for both hard and soft tissue reconstruction with skin paddle and/or muscular component (for oral mucosal reconstruction).
- Disadvantages: Vascular pedicle is short and most likely requires vein graft for maxillary reconstruction. Skin paddle can be very bulky making reconstruction of skin defects difficult. Donor site morbidity and the likely need for mesh support and concerns for hernia formation.
- Blood Supply: Deep circumflex iliac artery and venae comitantes.

Radial Forearm Free Flap

- Advantages: Reliable flap that can be used for soft tissue reconstruction. Not as bulky as an ALT flap and is ideal for tongue and floor of mouth reconstruction. May also be used for palatal defects including the hard and soft palate. Long pedicle length (10–12 cm). Low morbidity. Also offers osteocutaneous reconstruction utilizing the radial bone allowing for reconstruction of small bone defects of the maxilla or mandible or to provide orbital support. Two team approach.
- Disadvantages: Some patients do not have adequate palmar arch from the ulnar artery, thus blood supply to hand is compromised if the radial artery is harvested (Allen's test required). Not enough tissue available for large bulky defects. If radial bone is also harvested, the bone stock is not ideal for dental implants. Postoperative sensory changes at the dorsal aspect of thumb and index finger. Large scar on forearm.
- Blood Supply: Radial artery, cephalic vein, and venae comitantes.

Anterolateral Thigh Free Flap

- Advantages: Provides more tissue bulk for large oral defects. Ideal for large skin defects and offers pliable tissue, which can be contoured to existing anatomy. Low morbidity and allows primary closure without skin grafting. Two team approach.
- Disadvantages: Variable perforator blood supply. Can be too bulky for some reconstructions. Relatively short pedicle length.
- Blood Supply: Descending branch of the lateral circumflex femoral artery and its venae comitantes.

Temporalis/Temporoparietal Fascia Flaps

- Advantages: Versatile flap that can be used for maxillary/mandibular soft tissue reconstruction. Can provide coverage of defects and watertight seal. Reliable vascularity. May be used for facial reanimation.
- Disadvantages: Temporal hollowing, and some may require osteotomy of the arch to gain enough length.
- Blood Supply: Anterior deep temporal artery, posterior deep temporal artery, middle temporal artery.

- *What should be done in the setting of persistent oronasal fistula?*

  Depending on the size of the defect and reliability of the palatine arteries after resection, palatoplasty is an option. With larger defects, or previous failure of palatoplasty, a radial forearm free flap or other soft tissue flap may be used.

- *What is an option for dental reconstruction if using a soft tissue only free tissue transfer?*

  Zygomatic implants.

- *What is the difference between hypoglobus and orbital dystopia?*

  Hypoglobus is the inferior displacement of the globe commonly due to lack of bony orbital support as seen in cases of trauma of midface reconstruction. Orbital dystopia is the displacement of the entire orbit as commonly seen among the pediatric craniofacial patient population. May be evident as hypertelorism (horizontal) or vertical discrepancy when comparing the symmetry of the orbits.

- *Describe the difference between hyper- and hyponasality.*

  Hypernasality: Airflow escapes into the nasal cavity. Often a result of velopharyngeal dysfunction resulting from palatal clefting but may be acquired as such from Le Fort advancement, or maxillectomy. Numerous genetic syndromes are associated with hypernasality including DiGeorge syndrome, Treacher Collins syndrome, Prader-Willi, etc.

  Hyponasality: Air passage into the nasal cavity is restricted as seen with enlarged tonsils, inflammation and swelling from any number of etiologies (i.e., common cold), deviated septum, tumors, etc.

- *What are some ways to monitor a free tissue transfer?*

  Pen Doppler, internal Doppler, external color Doppler, pin prick to visualize bleeding, temperature, turgor assessment.

- *What is the most common reason for flap failure?*

  Vascular thrombosis is the primary cause of free flap failure. Venous thrombosis is more common than arterial thrombosis. When venous thrombosis occurs, the flap can appear boggy and have a bluish/purple hue with brisk capillary refill. The majority of flap failures take place within the first 48 hours of the postoperative period.

- *What is Ohngren's line and how does it relate to overall prognosis in regard to maxillary/midface tumors?*

  A plane extending from medial canthus to the angle of the mandible in the sagittal plane delineating suprastructure and infrastructure of the midface/maxilla. Tumors that are above this line, superior and posterior within the suprastructure, tend to have a worse prognosis when compared to infrastructure tumors (anterior maxilla).

- *What are some of the different approaches to the maxilla?*

  Weber Ferguson incision, split mandibulotomy, and midface degloving.

- *What are ways to decrease bleeding when performing maxillary surgeries?*

  Meticulous surgical technique, reverse Trendelenburg positioning, use of electrocautery, permissive hypotension, use of injectable vasoconstrictors, and vasoconstrictor or thrombin impregnated packing gauze.

- *What happens if performing an osteotomy and massive bleeding is encountered?*

  Pack the site, complete the osteotomy, then try to localize and control the source of bleeding. If unable to control the bleed, the site should be aggressively packed with an urgent consultation to interventional radiology for arterial embolization.

- *What is the most common source of bleeding when performing maxillary surgery?*

  The descending palatine artery which branches from the internal maxillary artery. The ascending palatine branch from the facial artery and the ascending pharyngeal artery supply blood flow to the down-fractured maxilla.

- *What are examples of regional flaps that may be utilized in the case of head/neck free flap failure?*

  Pectoralis major myofascial or myocutaneous flap, supraclavicular artery island flap, temporalis flap.

- *If there are signs of flap failure (i.e., loss of Doppler signal, negative pin prick, dusky and congested skin paddle, wound margin dehiscence) what should be the appropriate management?*

Emergent exploration in the operating room under general anesthesia. Assess for potential hematoma or seroma formation causing compression which results in compromised flap perfusion. Open up previous neck incision and assess the geometry of the pedicle and identify any kinking of the vessels. If geometry appears appropriate, the next step is to identify venous and arterial thrombosis. Venous thrombosis is more common due to lower blood flow and lower pressures when compared to arterial flow. If thrombus is identified, a thrombectomy should be performed. The use of antithrombotic agents such as heparinized saline or Fogarty catheter are used locally during and after thrombectomy. Thrombolytic therapy (streptokinase, tissue-plasminogen activator) may also be used to treat thrombosis or used in addition to surgical exploration/thrombectomy. Thrombolytic therapies are infused directly into the resected end of anastomosis or into a side branch to prevent systemic side effects.

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Cleft Lip and Palate

10

Pat Ricalde, Imran Ahson, and David E. Urbanek

Epidemiology/Embryology

- Cleft lip with or without cleft palate occurs in 1/940 live births [1].
- Cleft palate without cleft lip occurs in 1 in 1574 live births [1].
- Native Americans have the highest incidence at 1/500 live births [2].
- African Americans have the lowest incidence at 1/2500 live births [2].
- Cleft lip occurs more often in males (on the left side), and cleft palate occurs more in females. This is more likely to occur on the left side as there is a delay in the rotation of the palatal process of the maxillae on the left side.
- The incidence of isolated cleft palate is higher in females (3:2); 50% of isolated cleft palates are associated with a sequence or syndrome (e.g., Pierre Robin, Stickler, van der Woude, 22q deletion anomalies). Cleft lip with or without a cleft palate occurs with a 15% syndromic association [3].
- Formation of the lip, nose, and palate involves controlled proliferation, adhesion, apoptosis, and fusion of prominences. Failure of this process results in cleft formation.
  - 6 weeks gestation: Median nasal process fuses with maxillary process to form upper lip, philtrum of the lip, base of nose, and primary palate. Failure of fusion results in a clefting of the lip and/or alveolus.
  - 8–12 weeks gestation: Palatine shelves of maxillary processes merge in the midline to fuse with nasal septum/vomer to form secondary palate. This fuses from anterior to posterior. The degree of clefting is dependent on timing of disruption.
- The primary palate denotes the anatomy that is anterior to incisive foramen including the incisors, alveolus, and nasal spine. The secondary palate denotes all structures posterior to the incisive foramen including the soft/hard palates and the uvula.
- Complete cleft lips extend into the nares and the alveolus.
- Complete cleft palate involves the soft and hard palate and associated musculature.
- A submucosal palatal cleft is an incomplete cleft resulting from failure of the submucosal levator muscle to fuse completely in the midline (patients may clinically have a bifid uvula, zona pellucida, or absent posterior nasal spine).

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- Factors associated with cleft include family history, maternal smoking and alcohol use, maternal zinc deficiency, advanced parental age, folate deficiency in the periconception period, exposure to alcohol, and certain medications (e.g., retinoids, corticosteroids, anticonvulsants including phenytoin and valproic acid).

Classification Systems

- The Veau system has been proposed for classification of palatal cleft. Group 1 includes soft palate only. Group 2 includes cleft of the soft and hard palate. Group 3 represents complete unilateral cleft lip and palate. Group 4 includes complete bilateral cleft lip and palate (Fig. 10.1).
- The striped Y of Kernahan and Stark classification is a symbolic representation of the untreated cleft lip and palatal deformity with the incisive foramen as the dividing point. Right and left are distinguished for clefts of the lip, alveolus, and premaxilla.
- Boxes 1 and 4 reflect clefts of the right and left lips.
- Boxes 2 and 5 represent clefts of the right and left alveolus.

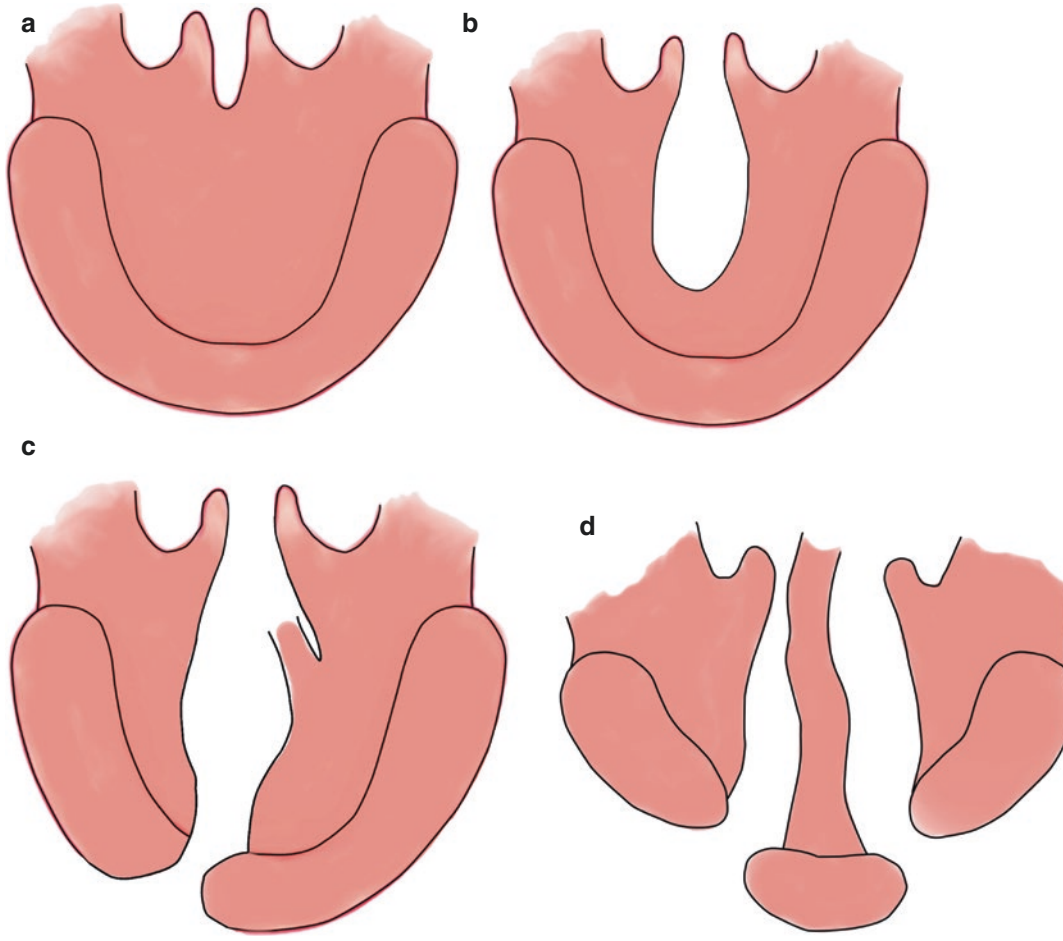

Fig. 10.1 Veau classification system. (**a**) Veau I cleft of the soft palate. (**b**) Veau II cleft of soft and hard palates. (**c**) Veau III unilateral cleft. (**d**) Veau IV bilateral cleft. (Modified from Butler CE. Head and Neck Reconstruction. Saunders; 2009.)

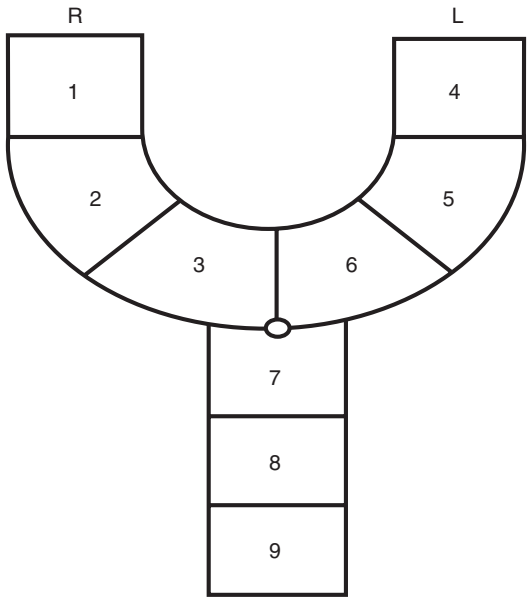

Fig. 10.2 Striped Y of Kernahan and Stark classification of clefts

- Boxes 3 and 6 represent clefts of the right and left premaxilla. The circle at the center reflects the incisive foramen.
- Box 7 reflects an isolated hard palate cleft.
- Box 8 represents a soft palate cleft.
- Box 9 represents a submucous cleft. It does not describe the degree of cleft (e.g., complete or incomplete cleft lip) or its functional impact (e.g., presence of velopharyngeal insufficiency) (Fig. 10.2).

Cleft Management

- A team approach at specialized cleft centers is the standard of care for patients with clefts to provide coordinated and continuous care through the child's growth. Members include surgeons (oral and maxillofacial, ENT, or plastic), pediatric dentists, orthodontists, speech pathologists, geneticists, audiologists, social workers, psychologists, and pediatricians.
- Ultrasound can identify clefting as early as 13–14 weeks of gestation. The sensitivity in detecting CL ± CP prior to 18–20 weeks is much lower [4]. This allows for anticipatory guidance and referral to a cleft team for interdisciplinary management.
- At birth, appropriate diagnostic testing includes echocardiogram and chromosomal array for isolated cleft palate patients in light of associated syndromes.

Sequence of Management

- At birth, a lactation consultation and/or speech therapy consultation is indicated. Bottles are available with customized nipples (e.g., pigeon bottle, Haberman with a one-way valve and reservoir spaces, Dr. Brown's cleft palate bottle) to reduce the child's work in feeding. Additionally, a Mead Johnson bottle is compressible to aid in forward flow of fluids.
- Try feeding promptly after birth. Keep child upright with frequent breaks. Feeds over 30–35 minutes will often fatigue the child and expend more calories than consumed.
- Preoperative orthopedics – During the first few weeks of life, lip and/or nasal taping or naso-alveolar molding appliances may be utilized to prepare the labial-naso-alveolar complex for surgery with a regularly adjusted appliance that helps guide growth and improve tension-free lip closure. The goal is to approximate the segments to within 5 mm. The need for frequent adjustments increases the risks of irritation and ulceration which are drawbacks of this therapy. Latham proposed appliances with screw activation and pin retention for better control of molding. Risk of growth restriction and the need for anesthesia are detractors from this therapy, as well as the baby having to undergo additional procedures.
- Lip adhesion procedure is a separate soft tissue closure used as part of a staged unilateral or bilateral cleft repair with the goal of narrowing the defect at the time of definitive closure. Normally carried out at 3–4 months of age. The major disadvantages is the need for an additional surgery with potential increase risk of scarring and questionable impact on the definitive repair.

- Primary lip repair (cheilorrhaphy) is usually performed around 10 weeks of age to reduce risks of anesthesia (rule of 10's). The timing of primary lip repair weighs the benefits of functional improvement (reestablishing muscular anatomy and function) with the risks of early surgery from anesthesia and growth limitation of surgical scarring.
- "Rule of 10's": 10 weeks old, 10 pounds in weight, and 10 mg/dL of hemoglobin.
- Audiology screening/ENT evaluation. Should be conducted before 6 months. Children with cleft palate have difficulty controlling middle ear pressure due to eustachian tube dysfunction as a result of abnormal insertions of the levator veli palatini and tensor veli palatini. With impaired ability to equalize middle ear pressure, infants with clefts usually have fluid in the middle ear space which can result in chronic otitis media, and conductive hearing loss. If left unmanaged, this can lead to permanent hearing loss. Treatment consists of myringotomy tubes or fluid evacuation.
- Primary cleft palate repair (palatoplasty in one or multiple stages) is generally performed between 9 and 18 months and timed with speech development to avoid compensatory misarticulations. Staged-soft palate includes soft palate closure at 6–10 months followed by hard palate at 1–3 years. Early palate repair (before 9 months) increases the risk of maxillary growth restriction.
- Correction of velopharyngeal insufficiency (including pharyngeal flap, sphincter pharyngoplasty, or mucosal augmentation procedures such as filler or fat grafting) is completed at 3–5 years following nasoendoscopy demonstrating poor adaptation of the soft palate to the posterior pharyngeal wall.
- Nasolabial revision is delayed until completion of nasal growth with anticipation that a definitive rhinoplasty may still be required. It is preferable to delay at least until the time of alveolar bone grafting so that concomitant treatment can be performed.
- Alveolar grafting is generally completed at 6–9 years, based on development of the dentition. Grafting is timed with formation of canine root (1/2 to 2/3 formation) to allow emergence into the cleft site. Some surgeons also time grafting to coincide with the development of the central or lateral incisor.
- Early orthodontics may be needed in the mixed dentition stage with the goal of maintaining transverse dimension, encouraging maxillary growth, and/or preparing for alveolar bone grafting. A second phase of orthodontics is often required with the permanent dentition.
- Orthognathic surgery (if required) is planned at 14 to 16 years of age in females, 16 to 18 years in males to address the maxillary hypoplasia, mandibular skeletal disharmony, and sleep-disordered breathing that is common in cleft patients.
- Rhinoplasty is often delayed at least 6 to 12 months following maxillary surgery due to changes in septum and tip support with movement of the anterior nasal spine.
- Cleft scar revision can be completed any time after 5 years of age but is ideally performed at the time of alveolar bone grafting.

Surgical Management of the Cleft Lip

Primary Lip Repair Techniques

Millard Rotational-Advancement Flap

(See Fig. 10.3)

- Millard proposed the rotation advancement flap. The goal of the Millard repair is the development of a three-layered closure following excision of hypoplastic tissue at the cleft margins. Orbicularis oris muscle continuity is reestablished and incision lines are designed to fall within the natural philtral ridges of the lip to promote symmetry. The repair also allows for columellar lengthening. Bilateral repair is complicated by lack of quality tissue, a short columella, and upward rotation of the premaxillary segment. The goal of the bilateral repair is reconstruction of the nasal floor, orbicularis continuity, and the maxillary vestibule.

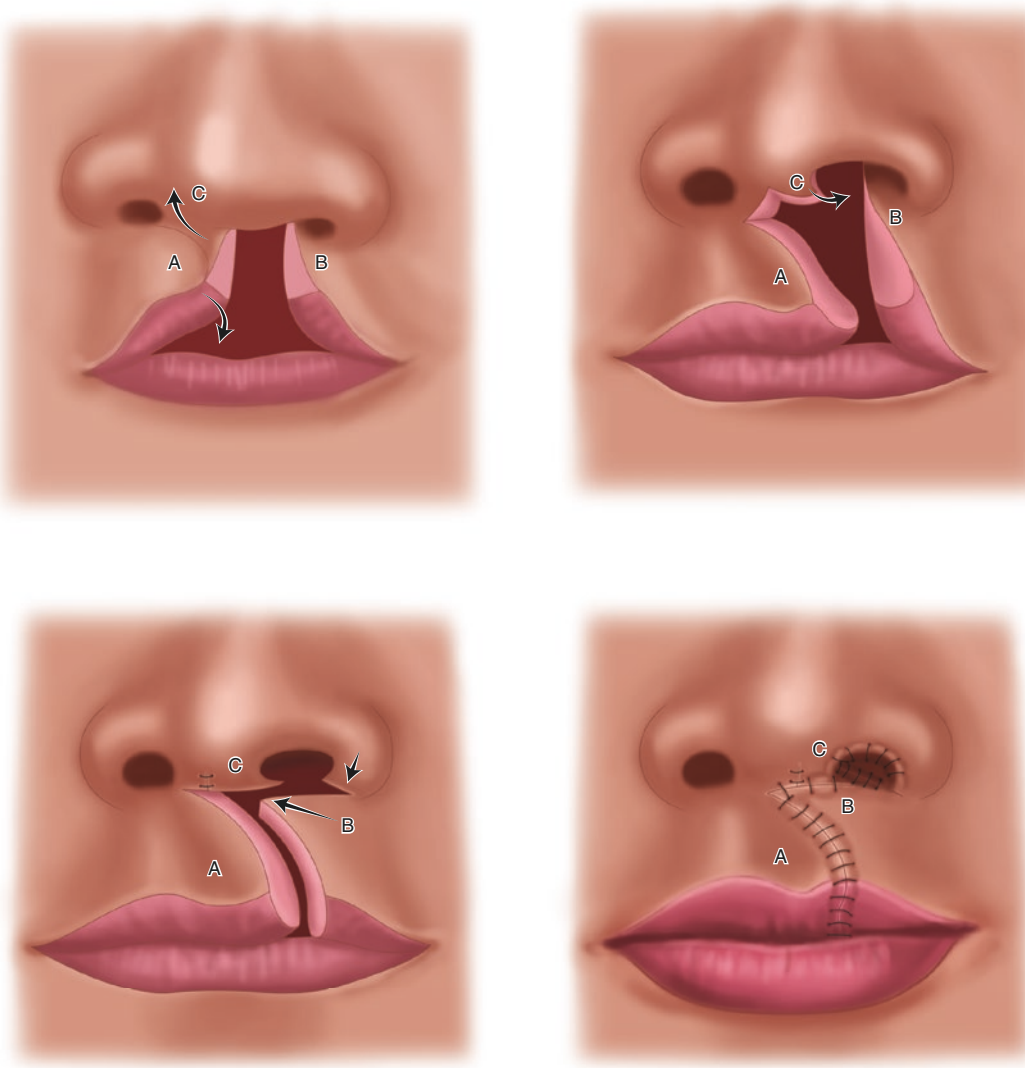

Fig. 10.3 Millard rotational-advancement flap

- The reason the technique is called a "rotational-advancement" flap is because the non-cleft side is cut in such a way that the tissue *rotates* (A) to create a longer vertical width and the cleft side *advances* (B) horizontally. This allows the scar to be hidden in a normal philtral line. This adds tissue to the deficient non-cleft side of the lip and the cleft side of the nasal columella. The premise for this and all techniques include a three-layered closure (skin, orbicularis oris, and mucosa); the excision of hypoplastic tissue from the cleft margins; and re-approximation of anatomic structures.

Delaire Technique (See Fig. 10.4)

- This technique is often used for bilateral cleft lips. Similar to the Millard technique, in that the cleft side advances while the non-cleft side rotates.
- In the case of a bilateral cleft, the technique is slightly modified. Both lateral sides are treated the same way, in that the design is the same and the muscles are dissected. The only

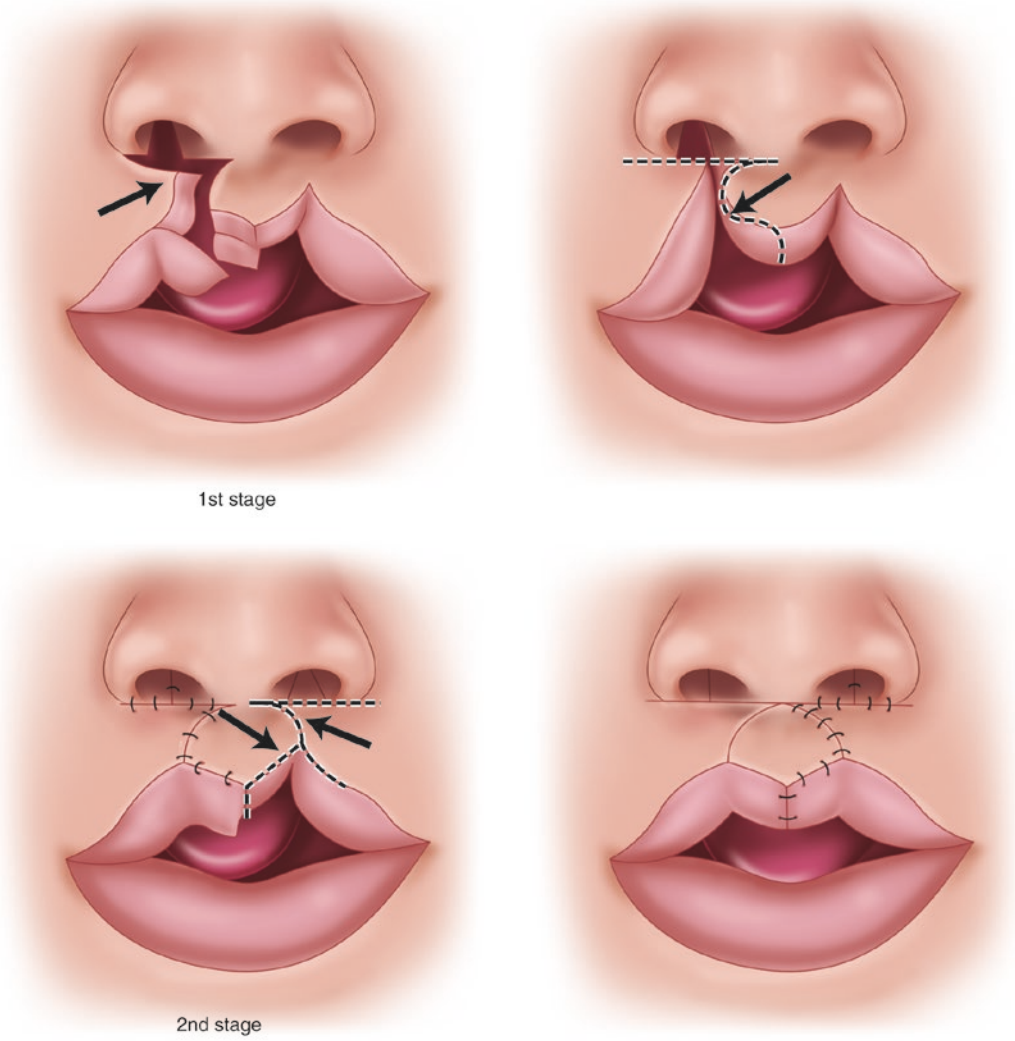

Fig. 10.4 The Delaire technique

difference is the prolabium, which is often proclined anteriorly. The prolabium is dissected with the following flap design seen in Fig. 10.4.

- The difference lies in the treatment of the nose, as the nasal tissue is gently dissected to create a laxity that allows for reapproximation in a symmetrical position. With the incisions open after the initial dissection of the Millard technique, a pair of tenotomy scissors are used to dissect over the lower lateral cartilages (still beneath the skin, cartilages not exposed) to free this tissue from its abnormal insertions. After the nasal tissue is dissected, the muscle and skin are reapproximated with additional sutures placed in the area of the nasal floor to create symmetry between the nostrils.
- A nasal bolster is often placed to maintain the nasal shape postoperatively.

Tennison-Randall (See Fig. 10.5)

- A Z-plasty technique that some feel is best used in wider clefts or if more vertical repositioning of Cupid's bow is needed. Proponents of this technique argue that the triangular

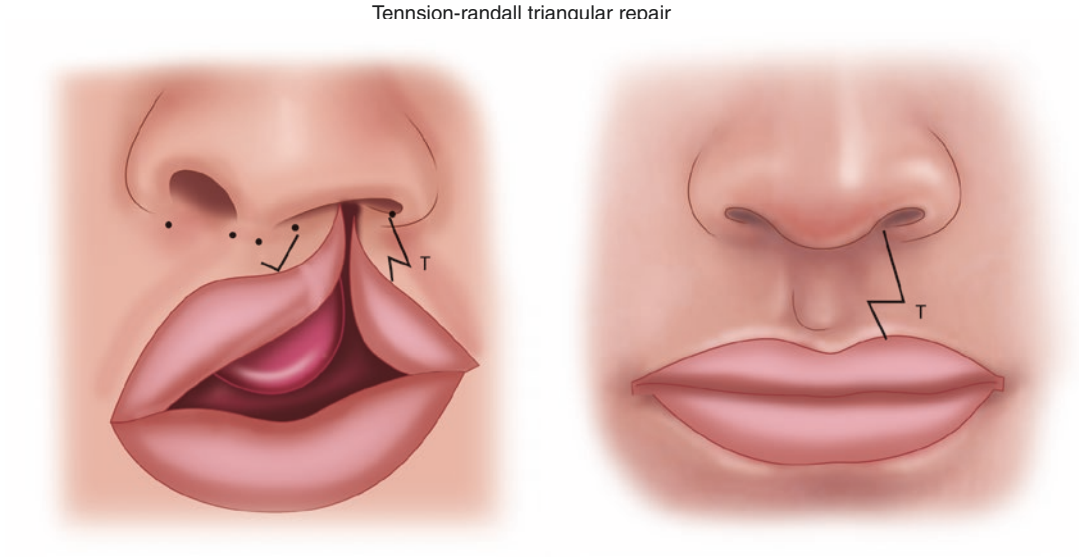

Fig. 10.5 Tennison-Randall technique

design avoids wound contracture and lip length shortening seen with the rotation and advancement flap operation. A major disadvantage of this technique is the scarring that crosses the philtral column which may contribute to the asymmetry.

Cleft Lip Repair Complications

Vermillion Deformities Vermillion deformities of the lip may occur secondary due to inadequate approximation of the marginal portion of the deep orbicularis or of the medial and lateral white roll at the primary repair, or excessive resection of the vermillion. Vermillion deficiencies are more common in bilateral clefts due to a dearth of tissue. May require a revision surgery.

Whistle Lip Deformity Inadequate release and advancement of mucosa and the vermillion can result in an indentation at the junction of the vermilion. The result is inadequate bulk of the lip with excessive show of the central incisors when the lips are in repose. Treatment includes nonkeratinized epithelium mucosal-submucosal flap procedures or fat grafting. Severe deformities may require an Abbe flap.

Hypertrophic Scar Can be managed by watchful waiting, scar revision, or steroid injection.

Nasal Asymmetry Can be managed at the time of nasolabial revisions or patient may require definitive rhinoplasty for management of asymmetry after orthognathic surgery.

Surgical Management of the Cleft Palate

Muscles of the Palate (See Fig. 10.6)

Muscles, innervation. and actions include the following:

- Tensor veli palatini – trigeminal nerve – tenses and depresses soft palate.
- Levator veli palatini – pharyngeal nerve – elevates the palate.
- Musculus uvulae – pharyngeal nerve – draws uvula upward and forward.
- Palatoglossal – pharyngeal nerve – draws palate down and narrows pharynx.
- Palatopharyngeal – pharyngeal nerve – draws palate down and narrows pharynx.

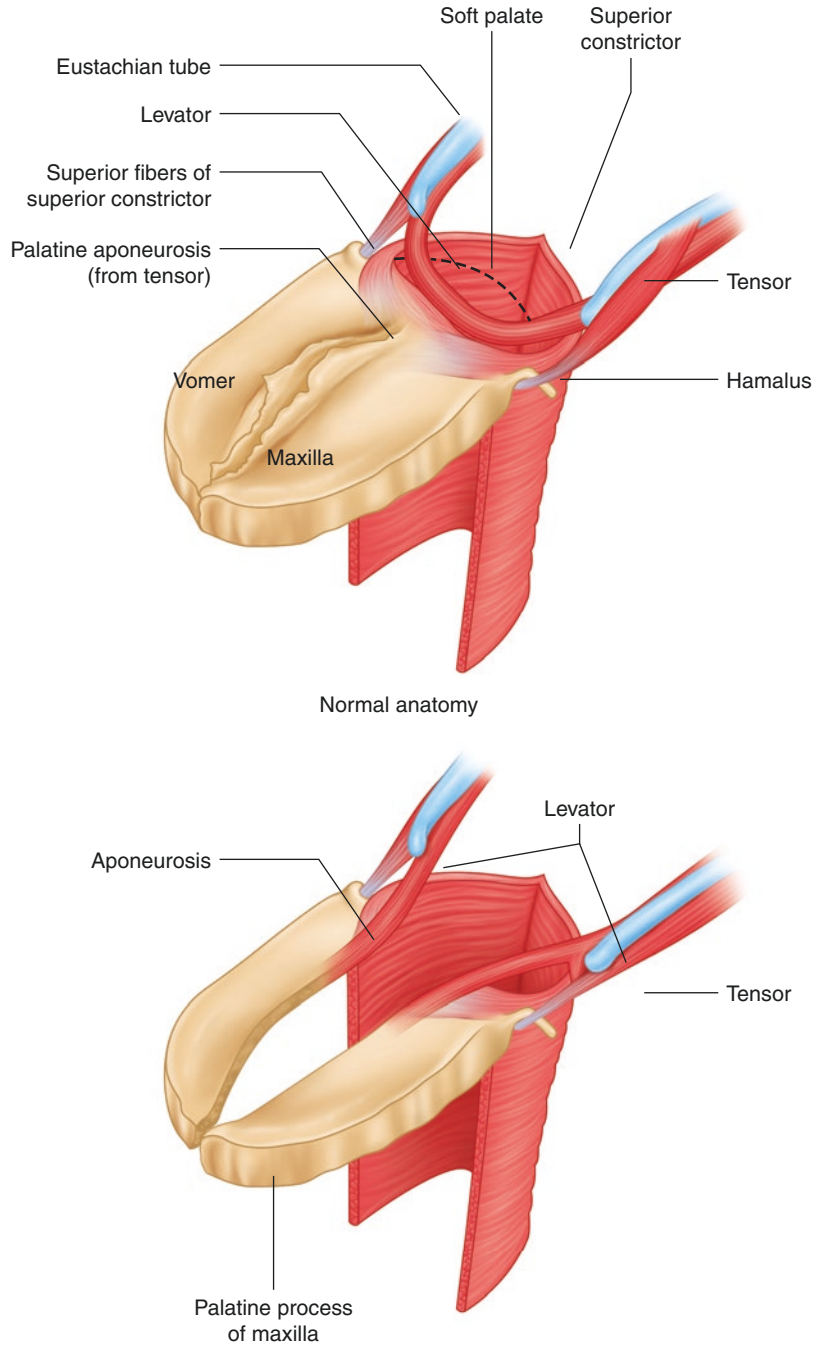

Fig. 10.6 Muscles of the palate (normal and cleft)

Palatal Repair Techniques

- Cleft palate repair has two goals, namely, the watertight closure of the oronasal communication and the anatomic repair of the musculature within the soft palate, which is critical for normal creation of speech.
- In the cleft patient, the tensor and levator veli palatini, as well as the palatoglossal and palatopharyngeal muscles, abnormally insert into the posterior hard palate in an AP orientation and have to be reoriented into their normal transverse alignment.

Modified von Langenbeck Procedure (See Fig. 10.7)

- In this procedure, two full thickness flaps are created and mobilized with layered dissection of the soft palatal tissues. The anterior and posterior portion remain connected to periosteum to

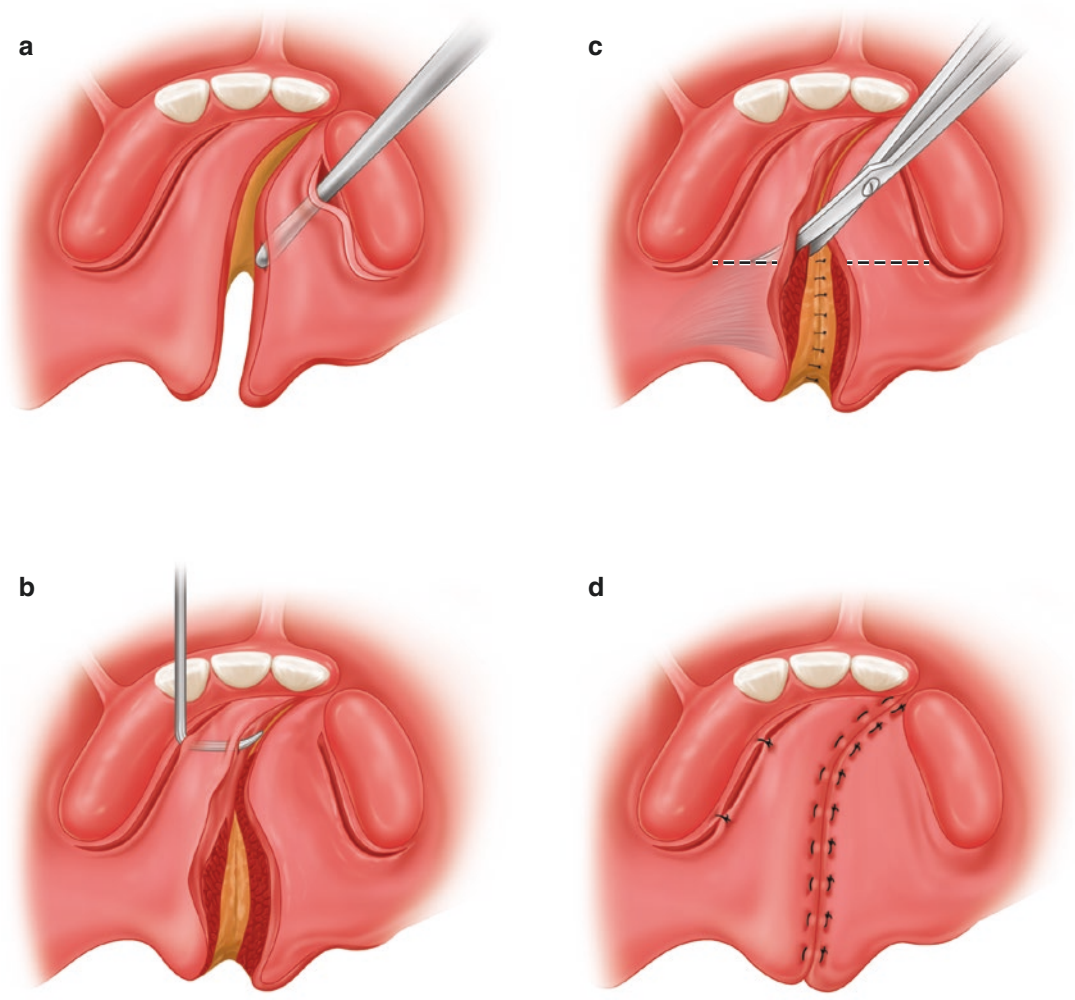

Fig. 10.7 Modified von Langenbeck procedure

increase circulation. Having an anterior attachment can decrease the visualization for nasal mucosa closure, and there is usually an area laterally that is allowed to granulate in. This healing through secondary intention can increase pain and create scar formation that restricts growth. For these reasons, it has fallen out of favor.

Bardach Two-Flap Technique (See Fig. 10.8)

- The most commonly used procedure, two flaps based off of the greater palatine artery are raised, dissecting either two layers (hard palate – nasal and oral mucosa) or three layers (soft palate – nasal mucosa, muscle, and oral mucosa). Dissecting the muscle off the posterior edge of the soft palate allows closure to reapproximate a more physiologic orientation.

Furlow Z-Plasty Technique (See Fig. 10.9)

- The Furlow Z-plasty was created to allow for lengthening of the hard palate by reorienting the muscles in a more physiologic reapproxi-

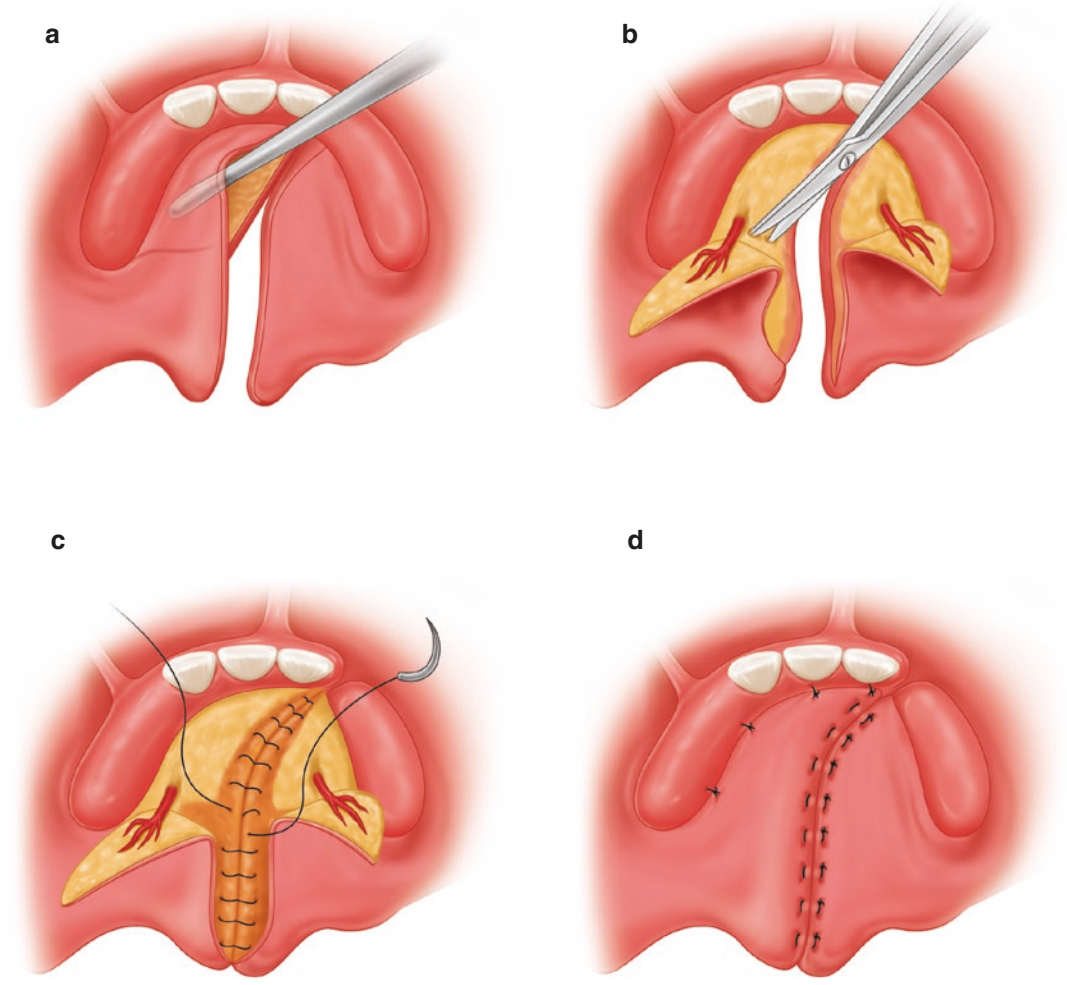

Fig. 10.8 Bardach two-flap technique

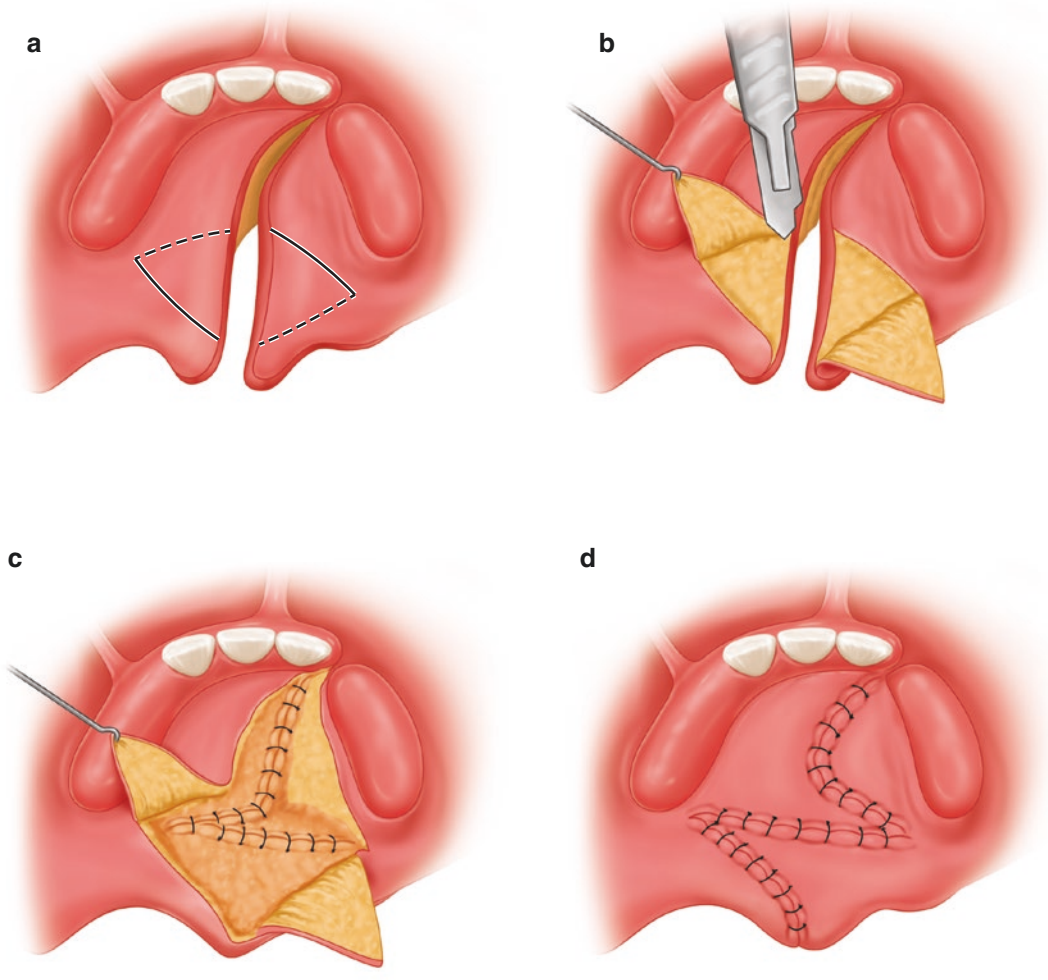

Fig. 10.9 Furlow Z-plasty technique

mation. One side is myomucosal and the other side is mucosal. This method is more technically difficult and has a higher rate of oronasal fistula formation.

V-Y Pushback Technique (See Fig. 10.10)

- Sometimes described as a modification of the von Langenbeck technique with anterior pedicle release, this technique involves the release of the muscles of the soft palate from the posterior edge of the hard palate and from the periosteum on the nasal side, allowing the creation of a more physiologic muscle sling and soft palate. There is an anterior two-layer closure and posterior three-layer closure, but still has an area that heals by secondary intention, which leads to severe growth restriction.
- A major advantage is maximization of palatal length to decrease postoperative VPI.

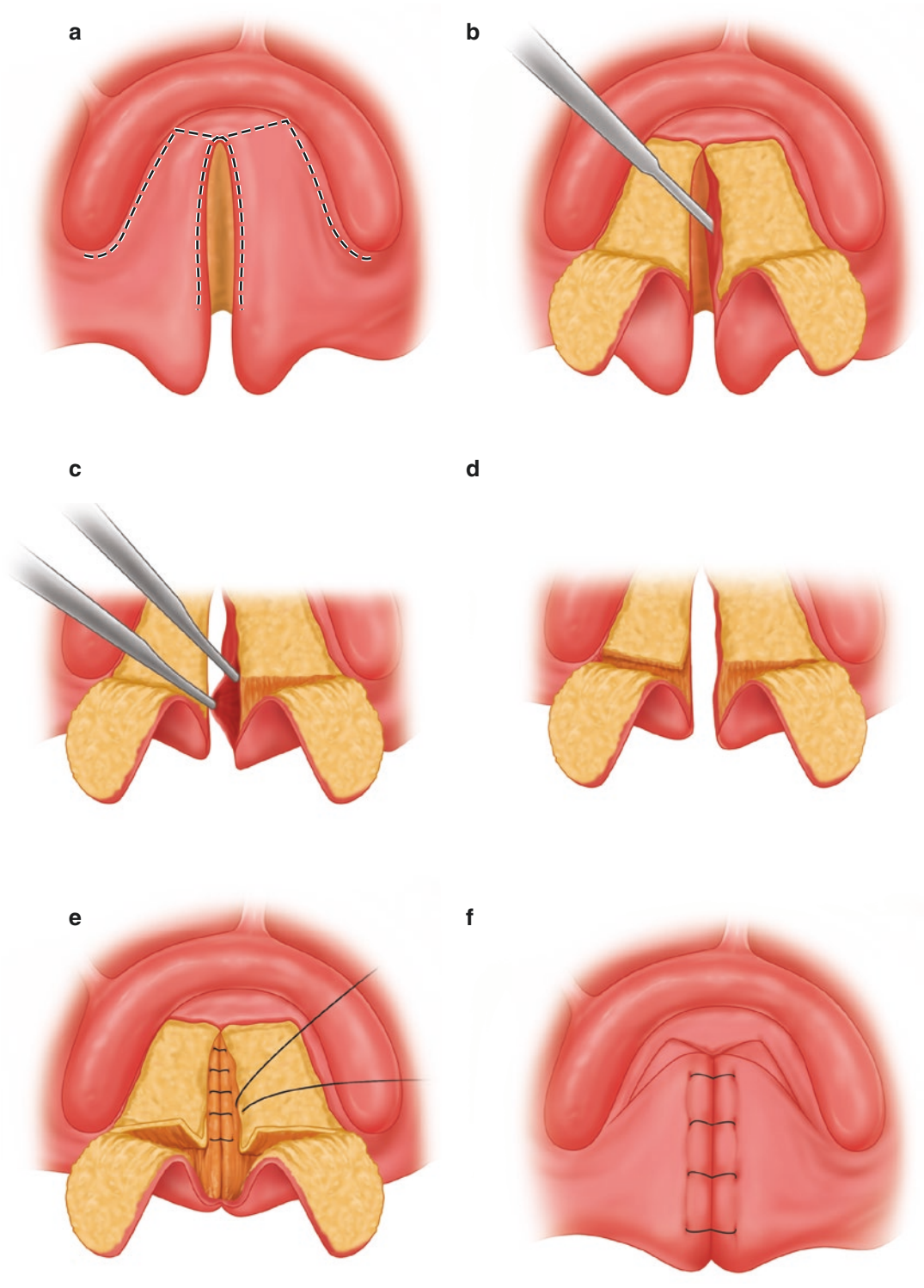

Fig. 10.10 V-Y pushback technique

Complications of Cleft Palate Repair

Oronasal Fistulae The most common sites for development of fistulae are at the junction of the hard/soft palate and the incisive foramen. Smaller fistulae may be addressed with regional palatal flap repair. Larger fistulae can be treated with robust regional palatal flaps, tongue flaps, facial artery myomucosal flaps (FAMM flaps), and temporalis flaps. Surgeons may consider waiting for further growth prior to repair to avoid maxillary growth restrictions secondary to surgery if speech or function not affected**.** Depending on age of patient, repair of fistulae may also be combined with the alveolar bone graft, or with orthognathic surgery so as to minimize the number of surgical interventions.

Velopharyngeal Incompetence VPI manifests as hypernasality and nasal emission. Diagnosis can be confirmed with nasoendoscopy with or without videofluoroscopy. The surgical management of VPI includes pharyngeal flap, pharyngoplasty, posterior pharyngeal wall augmentation, and palatal revision.

Velopharyngeal Insufficiency (VPI)

Velopharyngeal Insufficiency (VPI)

- Also called velopharyngeal incompetence, it is the incomplete closure of the velopharyngeal sphincter, and thus incomplete separation of the oral and nasal cavities during speech.
- It is the term of choice when diagnostic studies have clearly determined that a true physical limitation is present.
- The term velopharyngeal dysfunction (VPD), however, is used when there is some sort of malfunction, but the cause remains unclear.
- It is estimated that about 1/4 of patients will demonstrate some sort of VPD after cleft palate repair.
- Signs and symptoms of VPI:
  - **Hypernasal speech** – is the production of inordinate nasal resonance during the production of vowels produced by the inappropriate coupling of the nasal and oral cavities.
  - **Nasal emission** – nasal air escape during production of consonants requiring high oral pressure. Nasal emission may be audible or not.
  - **Nasal substitution** – incomplete closure of the velopharyngeal port causes sound to be produced as a nasal consonant rather than an oral consonant. For example, "B" becomes "M" and "D" becomes "N."
  - **Compensatory misarticulations.**
    - With VPI, the child cannot build up oral pressure to produce fricatives (e.g., S) or plosives (e.g., P). Instead, the child will attempt to create those pressures below the level of the velopharyngeal port. Such misarticulations may include glottal stops, pharyngeal stops, and pharyngeal fricatives.

Work Up and Diagnosis

Speech and Language Pathologist (SLP)

- An essential role of the SLP is to determine if the VPI is the result of true physical limitations, and therefore requires surgical management, or more related to learning or habituation of patterns, which would require speech therapy. In cases of severe and consistent VPI, it is easy to make the diagnosis and, therefore, treatment recommendations. However, in borderline cases, it is difficult to make this determination, and surgery may not be the proper treatment. In complex cases such as these, a period of speech therapy will provide additional and often necessary diagnostic information.
- Timing of evaluation – Evaluation for VPI is dependent on the development of speech in the child. It is impossible to accurately judge velopharyngeal function in infants and toddlers (up to 3 years of age). However, during the preschool years (3 to 5 years of age), as the child's sound repertoire, articulation, and

expressive language expand, velopharyngeal function can be assessed more accurately. A level of cooperation during the evaluation is also required on the part of the child.

Clinical Evaluation of VPI

- Observe speech for hypernasality, nasal substitutions, and compensatory articulation errors. These signs should serve only as a "red flag" for the surgeon. Formal speech testing and interpretation should be left to the SLP.

Tests for Nasal Emission

- Mirror test: a mirror is held under the patient's nares during production of pressure-sensitive sounds to observe for fogging.
- See Scape test: See Scape is a device that resembles an incentive spirometer. A flexible tube is placed into the patient's nare, and as the child produces pressure-sensitive sounds, a styrofoam stopper inside a connected vertical tube rises as a result of nasal emission.
- Nasal palpation: vibration from hypernasality can be palpated by placing one's fingers on the nasal sidewalls.
- Auscultation with a stethoscope (remove the drum and use the tube), flexible plastic listening tube, or straw.
- Nasopharyngoscopy:
  - Involves using a flexible fiberoptic endoscope to visualize velopharyngeal function. The nasal surface of the velum, and lateral and posterior pharyngeal walls can be visualized without interfering with speech production.
  - Is the primary instrumental assessment tool of most cleft palate-craniofacial teams because it does not expose the patient to radiation.
  - Patient cooperation is essential for a successful evaluation.
  - Is the most appropriate instrumental measure for diagnosing an occult submucous cleft of the palate, and for assessing velopharyngeal closure after a pharyngeal flap has been performed.
- Video Fluoroscopy:
  - Uses fluoroscopy to visualize the anteroposterior movement of the velum during a variety of speech tasks. Barium is instilled through the nose and provides visualization of the margins of the velopharyngeal structures.
  - Is performed by a radiologist.
  - Involves exposing the child to radiation, and because of this is a less frequently used test for VPI.

Treatment of VPI

Nonsurgical Management

- Prosthetics such as a speech bulb or palatal lift (if surgery is not an option).
- Speech therapy – for cases where velopharyngeal mislearning (faulty articulation) is the cause of hypernasality or nasal emission.

Surgical Management

- Timing of surgery – surgical management of VPI ideally occurs around age 5, however can be done at a later age as well.
- Indications of surgery – patients who have consistent VPI as determined by a SLP and experienced surgeon.

Contraindications of surgery:

- Patients who decline surgery by choice.
- Patient has known or suspected risk for airway obstruction.
- Patient has intermittent or inconsistent closure that responds well to speech therapy.
- Patient has incomplete diagnostic results.
- Patients who have an aberrant and medial position of the internal carotid artery, such as in those with velocardiofacial syndrome. Velocardiofacial syndrome (VCFS) – anomalous internal carotid arteries have been shown to be a frequent feature in VCFS and pose a potential risk of iatrogenic injury and hemorrhage during surgery. Preoperative cervical vascular imaging studies are recommended to define the course of these vessels.

Surgical Correction of VPI

- The patterns of velopharyngeal closure can be thought of as consisting of two main categories of movement: anteroposterior movement in which the velum contacts Passavant's ridge and lateral pharyngeal wall movement. Varying degrees of velum and pharyngeal wall motion contribute to many sphincteric closure patterns.
- Passavant's ridge – a bulge on the posterior pharynx above the arch of the atlas (C1), produced by forceful contraction of the superior pharyngeal constrictor. This ridge also may be associated with velopharyngeal incompetence as a compensatory mechanism to assist with velopharyngeal closure.

Pharyngeal Flap Surgery

- If the patient has undergone palatal repair and has been diagnosed with velopharyngeal incompetence, he/she may undergo pharyngeal flap surgery in order to correct hypernasality and nasal air escape.
- The goal of surgery is to develop a functional seal between the nasal cavity and the oral cavity. This is accomplished by taking tissue from the posterior pharynx and attaching it to the soft palate, effectively decreasing the opening between the nasal cavity and the oral cavity (also called the velopharyngeal port).
- There are multiple different methods, but the most commonly used techniques are either an inferiorly based or superiorly based pharyngeal flap. Below are descriptions of two superiorly based repairs.

Superiorly Based Pharyngeal Flap
(See Fig. 10.11)

- Indicated when there is adequate lateral pharyngeal wall movement. Tissue is taken from the posterior pharyngeal wall and attached to the soft palate, creating a midline subtotal obstruction of the oral and nasal cavities with two small lateral openings that ideally remain open during respiration and nasal consonant production and close for consonants.
- Involves elevating a myomucosal flap from the posterior pharyngeal wall and insetting it into the velum such that it converts the single velopharyngeal aperture into two lateral ports separated by the pharyngeal flap.
- It is the most commonly performed surgical technique used to treat VPI.
- For VCFS, preoperative cervical vascular imaging (CT/MRI angiogram) is obtained to rule out medial internal carotid arteries (ICA). Treatment options if aberrant vessel location identified:
- "Cautious" surgery.
- Alternate procedure such as a Furlow palatoplasty.
- No surgery and re-evaluate over time to see if things change with growth.
- Posterior pharyngeal veins are the most common site for bleeding and are visible on the prevertebral fascia.
- ICAs are located laterally and lateral dissection is avoided.

Sphincter Pharyngoplasty (Orticochea Dynamic Pharyngoplasty)

- Indicated for those with markedly impaired or absent lateral pharyngeal wall motion (when there is adequate posterior movement of the velum).
- Creates a smaller midline pharyngeal port.
- Scheduled for a minimum of 3 months after a tonsillectomy and adenoidectomy. These must be done carefully to preserve the posterior tonsillar pillars.
- Testing of VPI is repeated 6–8 weeks after T&A.

Alveolar Clefts

Alveolar clefts (Fig. 10.12) involve a separation of the bony alveolar ridge and can range from a simple notching in the buccal alveolus to a wide gap creating an oronasal fistula and disturbances in the eruption of teeth. The alveolar cleft is managed with bone grafting and rearrangement of the soft tissues.

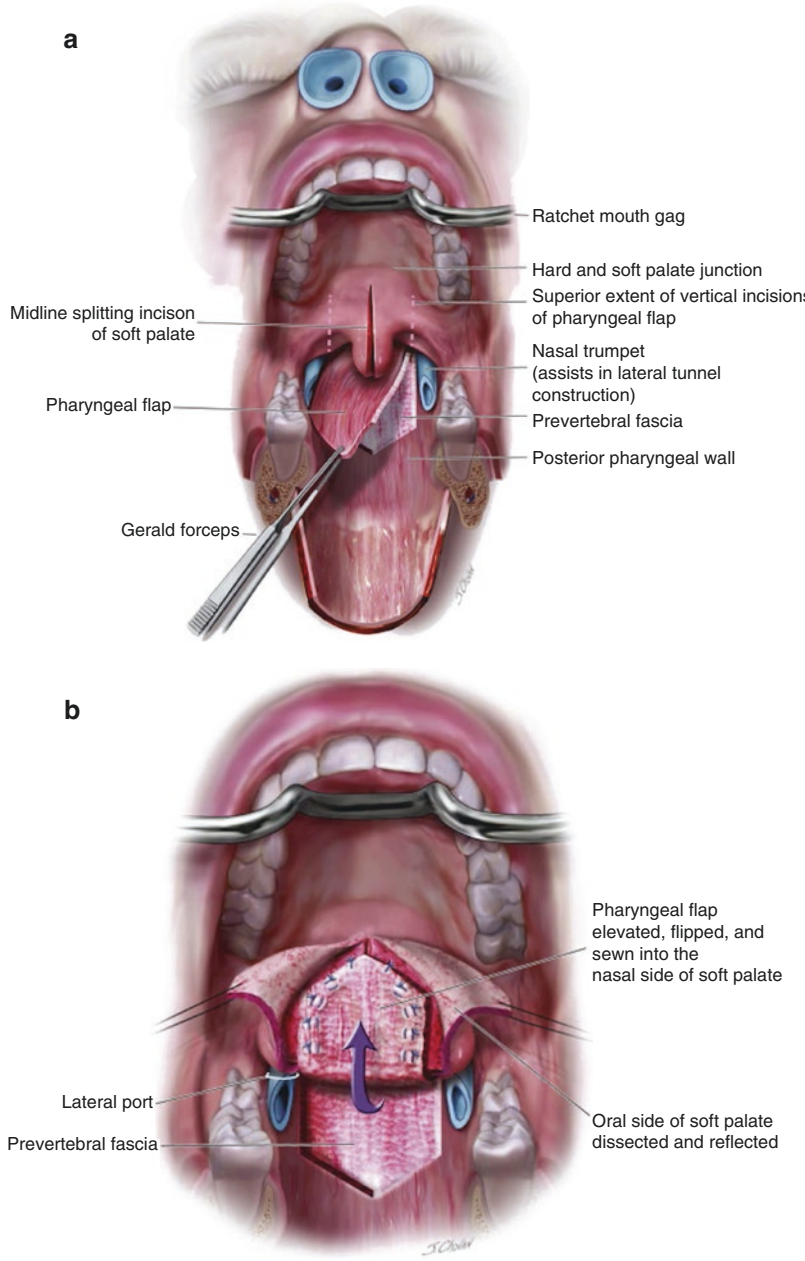

Fig. 10.11 Superiorly based pharyngeal flap. (Reproduced with permission from Posnick, JC (2000) The staging of cleft lip and palate reconstruction: infancy through adolescence. In: *Craniofacial and Maxillofacial Surgery in Children and Young Adults*, edited by JC Posnick. Philadelphia: WB Saunders)

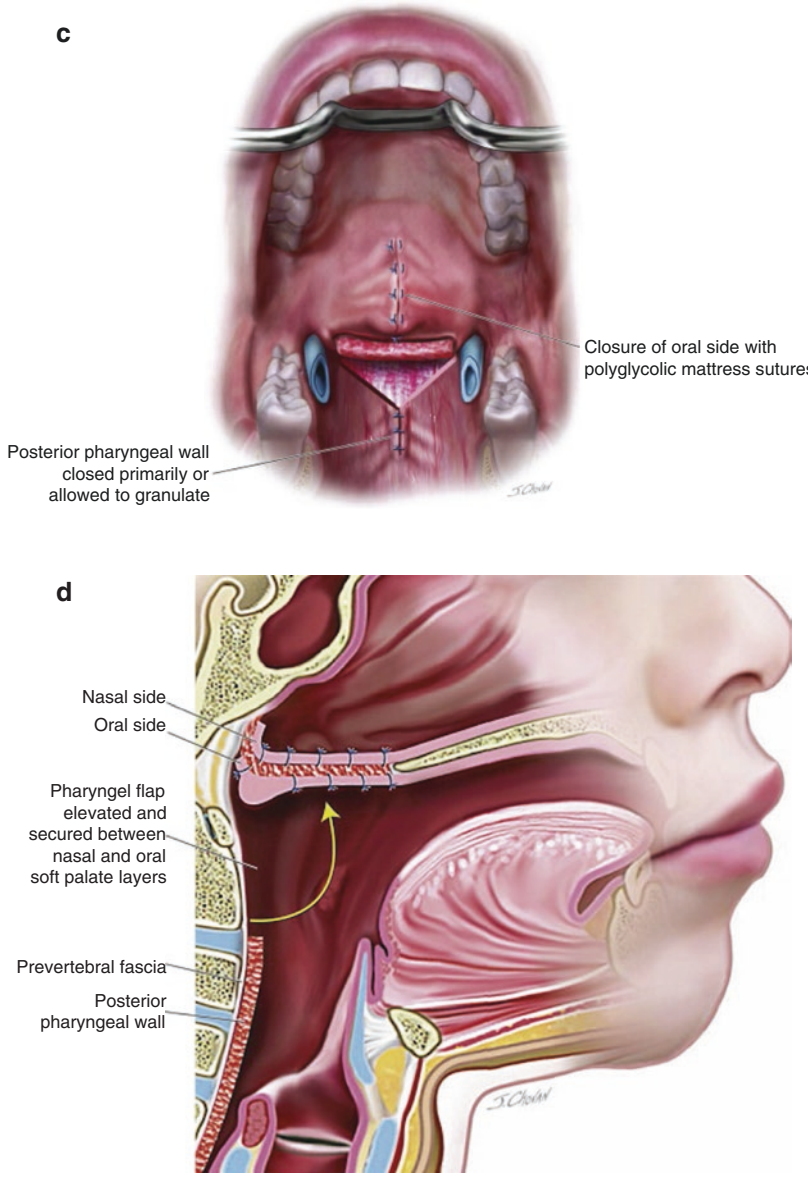

Fig. 10.11 (continued)

Presentation of a Cleft Patient

- The anatomy of a patient with an alveolar cleft can look very different from patient to patient, and therefore, an understanding of the different presentations and anatomical differences is important to treatment plan the patient appropriately.
- In unilateral clefts, the segment of the maxilla that is on the side of the cleft is known as the "lesser" segment (the "greater" segment being the non-cleft side).
- The cleft itself can range from a small notch to a wide gap with large oronasal fistula.
- Common presentations include medial collapse due to a lack of transverse stability, resulting in crossbite.

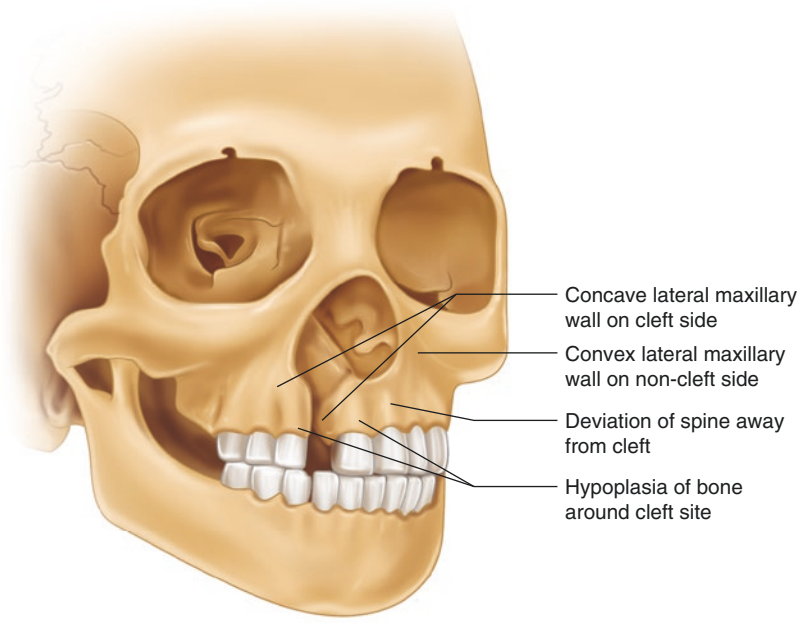

Fig. 10.12 Alveolar Cleft

- Teeth along the cleft side can be missing, hypoplastic, malrotated, or supernumerary. The periodontal support on the cleft side of the adjacent incisor and canine tend to be deficient.
- In bilateral clefts, the presentation can be varied as well. The premaxilla is commonly rotated anteriorly due to the unrestricted growth of the vomeropremaxillary suture. It also may be slightly mobile due to the flexibility of the pediatric bone. Occasionally the premaxilla may be missing due to the presence of a true midline cleft, or due to iatrogenic injury to the blood supply. If present, care must be taken at all times to preserve its vascularity due to a lack of collateral circulation from lateral anastomoses.
- Knowledge of the three-dimensional structure of the cleft will be important in helping you visualize the technique, as well as describing the information on the boards (Fig. 10.13).

Goals of Therapy

- Allow eruption of dentition.
- Provide support to the adjacent periodontium.
- Stabilization of maxillary segments.
- Closure of oronasal fistulae.
- Improve speech and language development.
- Provide appropriate tissue for dental health.
- Provide adequate bone for future dental implant therapy.
- Reconstruct nasal floor and lift the alar base.
- Allow for greater lip support.
- Cosmesis.
- Greater self-esteem.

Nomenclature

The nomenclature for alveolar bone grafting is based on the time of bone grafting in comparison to the lip repair and dentition. This is divided into:

- Primary (at or before the time of the cleft lip or palate repair).
- Secondary (later in life). Among secondary bone grafting, the timing can further be broken down into:
  - Early: 3–5 years.
  - Early mixed dentition: 6–8 years.
  - Late mixed dentition: 9–12 years.
  - Late: 13 years and above.

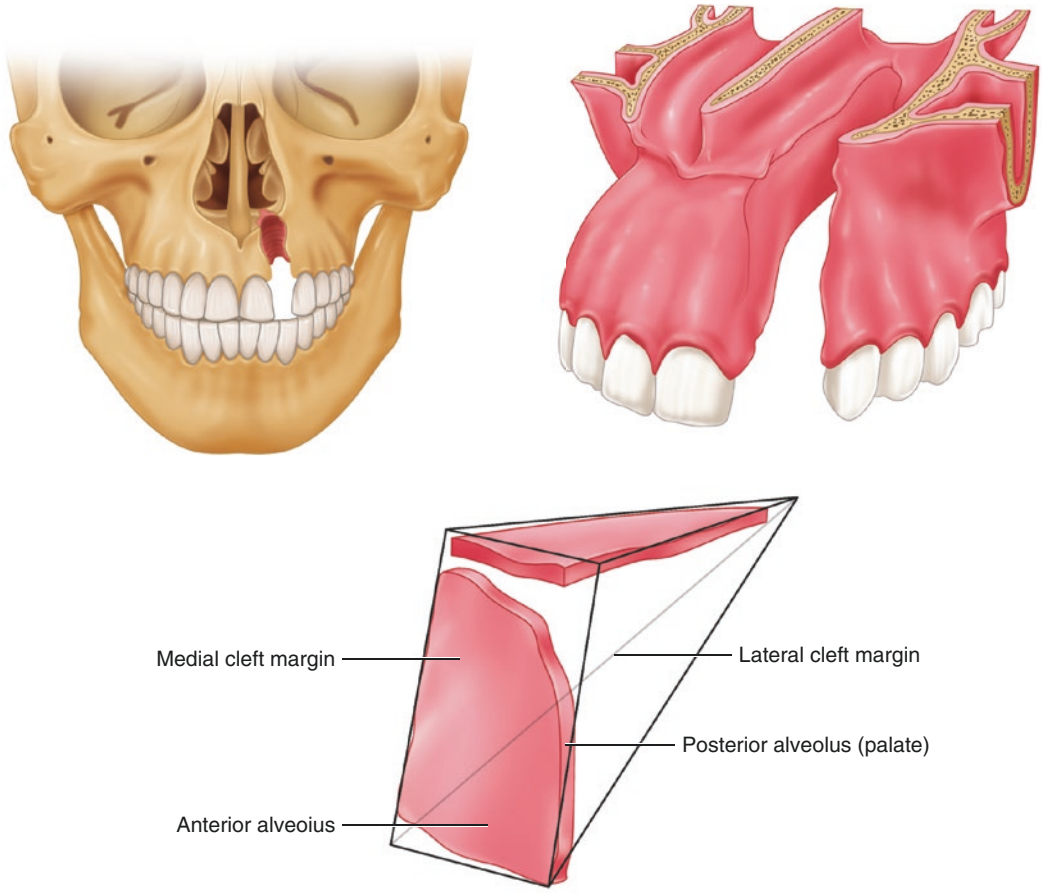

Fig. 10.13 3D structure of alveolar cleft

Gingivoperiosteoplasty Can be done with naso-alveolar molding techniques or at the time of primary lip repair. Gingivoperiosteoplasty approximates the alveolar cleft soft tissue resulting in a gingivoperiosteal tunnel. It is hoped that this will create bone healing so as to avoid the need for bone grafting. The procedure is not appropriate for wide clefts as the segments need to be within 2 mm of each other. The downside to this procedure is the potential for early scarring and restriction of maxillary growth, injury to the cleft and adjacent teeth blood supply, and increased costs/psychological burden to the child and family.

Primary Grafting The rationale behind grafting at the time of the initial lip repair is to prevent maxillary arch collapse and allow the teeth to migrate into the alveolar process. In bilateral cases, it can also stabilize the premaxilla at an earlier age. Unfortunately, these procedures have resulted in severe growth restrictions and have largely been abandoned.

Secondary Grafting

Early If done too early, it may lead to maxillary growth restriction.

Early Mixed Dentition Most commonly alveolar bone grafting is performed when the canine root is ½ to 2/3rds formed. There are many other factors to consider, and grafting based on development of central and lateral incisors is gaining favor among some cleft centers, but outcome

studies still lacking. Advantages include improved clinical crown height and periodontal support for the adjacent central incisor.

Late Mixed Dentition Grafting after the eruption of the canine has been shown to have a lower success rate due to the gradual loss of bone along the distal surface of the central incisor and the distal surface of the canine root. Once the cementum is exposed, the bone graft is unable to adhere to and make up for these losses. In addition, the positive effects of the tooth erupting into the bone graft does not occur leading to a decreased volume of remaining bone after grafting has been completed.

Late The late bone grafting requires extensive orthodontic forces to separate the collapsed arches, as well as having the same issues as grafting in the late mixed dentition. The success rate decreases as age increases. At this point, rather than attempting a procedure with a decreased success rate, it may be prudent to use a removable partial denture as an obturator. Another option is modified orthognathic surgery, during which time the arches are placed in the appropriate alignment, and the lesser segments are advanced to decrease the cleft dental gaps. With the intimate contact of the arches, the bone graft success is much higher.

Surgical Technique for Secondary Bone Grafting (Fig. 10.14)

- Although the technique may vary in timing, the main objective of the surgery is to fill in the pyramidal bone defect caused by the cleft (see below).
- The anterior flaps are raised first, to gain access to the underlying nasal mucosa.
- Next, the nasal floor must be recreated, forming the superior portion of the pyramid. The posterior portion of the pyramid is recreated by reapproximating the palatal tissue.
- Finally, bone graft is packed into the pocket of tissue that has now been created, and the anterior flaps are closed over the bone graft in a watertight seal.

Design of the Flaps

Split Thickness Component As the incision is carried superiorly, dissection remains anterior to the cleft to expose the lateral aspect of the anterior nasal spine and the lower pyriform rim. At the junction of the nasal cavity and vestibule, the flap should have a split thickness component (at the most anterior margin).

Full Thickness Component The anterior flaps abutting the alveolar bone are full thickness mucoperiosteal flaps, with their blood supply from the periosteum and lateral circulation. The design can range from a sulcular incision carried laterally or sparing the gingival sulcus. To get the tissue appropriately mobile, some surgeons use a back cut at the area of the first molar. The nasal flaps are created from the tissue adhering to the lateral and medial walls of the bony pyramid, extending posteriorly. These are raised in a full thickness fashion, except for the anterior component which is split thickness. The palatal flaps are full thickness mucoperiosteal flaps as well, with gingival sulcular incisions extending as far distal as necessary to ensure appropriate mobilization.

Bilateral Alveolar Clefts The main difference in the flap design of bilateral clefts is taking care to minimize dissection that would compromise the blood flow to the premaxilla (Fig. 10.15). The remaining blood flow to the premaxilla is from the midline branches of the posterior septal artery and to a lesser extent the lateral nasal and terminal branches of the anterior ethmoidal artery. These vessels pass from a superior to inferior direction, so care must be taken to have minimal dissection superiorly and focus on freeing tissue from the lateral aspect of the premaxilla as shown below.

Donor Site

- The gold standard for alveolar cleft repair is bone from the iliac crest that is harvested as a cancellous graft. The particulate structure of the graft allows for easy packing into the cleft site and conformity to the shape of the arch.

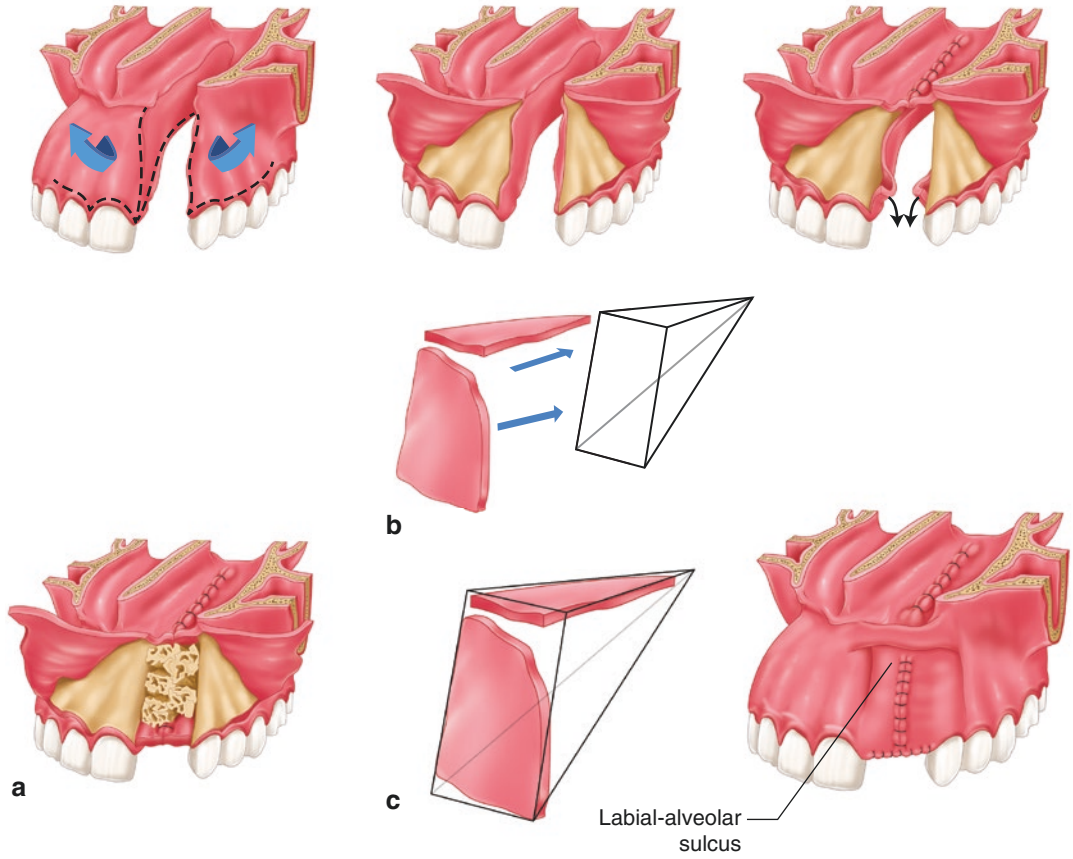

Fig. 10.14 Secondary bone grafting technique. (*Note: Some surgeons prefer to raise the palatal component second and retract the tissue to gain better visualization of the cleft to help in closing the nasal floor)

- Harvest from children is similar except the crest is cartilaginous. Cartilage cannot be harvested as it represents an ossification center. The cartilage is reflected to one side and bone below is harvested to prevent a contour deformity.

Other Sources of Bone Grafting for Alveolar Clefts

- Rib:

  Pros: Sole source of bone in primary grafting, large amount of bone.

  Cons: Morbidity (pain, scar, pneumothorax).

- Calvarial bone:

  Pros: Membranous bone, same field, hidden scar.

  Cons: Little cancellous, patient perception.

- Tibial bone grafting:

  Pros: Cancellous bone.

  Cons: Donor morbidity, moderate amount of bone.

- Mandible:

  Pros: Membranous bone, same field, hidden scar.

  Cons: Little bone, risk to dentition, mental nerve.

- Bone morphogenic protein:

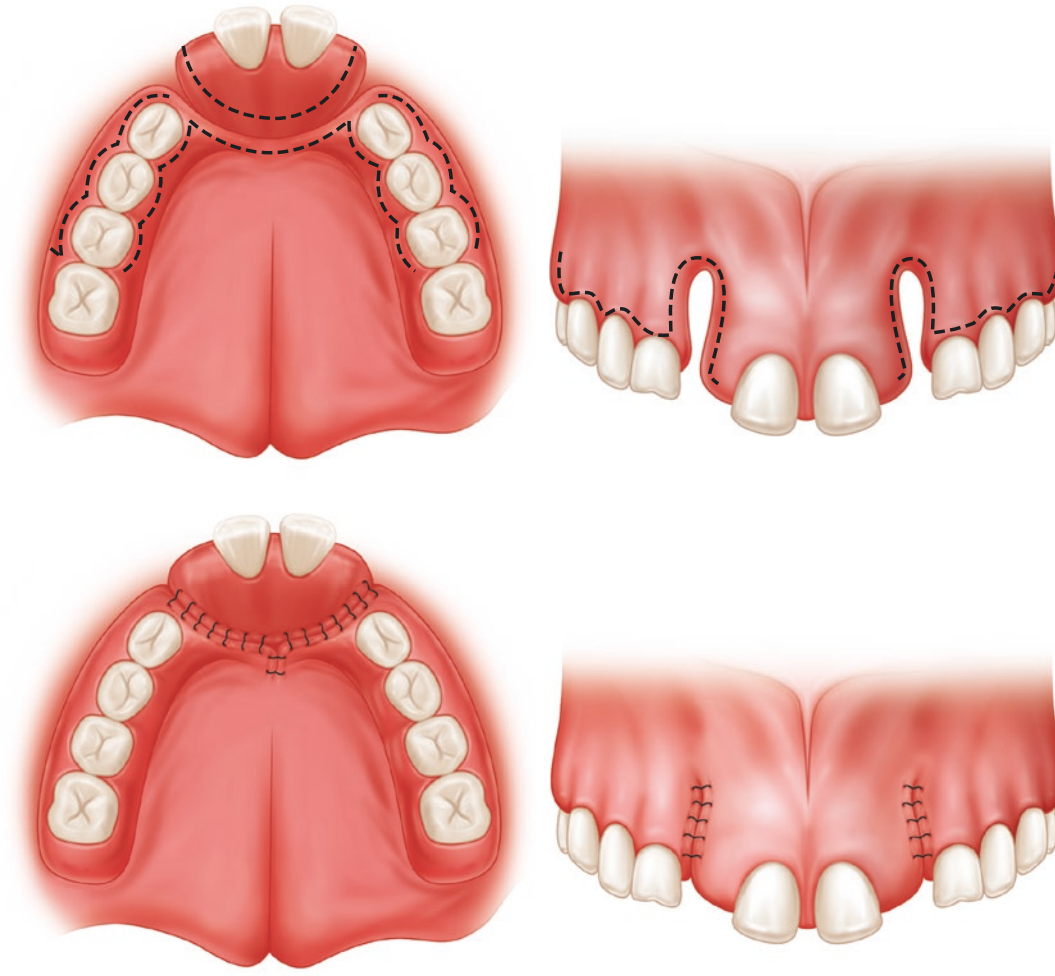

Fig. 10.15 Secondary bone grafting of a bilateral alveolar cleft

Pros: Reduce OR time, can be mixed with autologous or allograft, reduced risk of harvest site.

Cons: Higher expense, risk of ectopic bone formation, severe swelling/edema, and increased medico-legal risk. BMP is poorly studied in children and is contraindicated by the FDA for use in children.

Perioperative Dental Support:

- Highest success rates occur in a multidisciplinary setting. Consultation with other specialists and regular follow-up ensures that the patient is treated at the appropriate age and the appropriate steps are completed preoperatively to maximize success. Specifically, consultation with orthodontists and pediatric dentists preoperatively is mandatory for the following reasons:
- Orthodontic alignment of the arch form – Presurgical and postsurgical orthodontics are associated with superior secondary alveolar bone grafting outcomes. Presurgically, the teeth are repositioned into a more ordered alignment to facilitate surgery. Postsurgically, the functional stress on the grafted bone promotes alveolar remodeling.

- Approximately 4 to 6 months of orthodontic treatment should be anticipated in preparation for alveolar bone grafting.
- Pediatric dentists – Provide routine dental care. Pedodontists can also take occlusal radiographs which allow for preoperative visualization of the cleft site and allows for postoperative comparison.

Complications of Alveolar Grafting

Graft Exposure One of the most common complications is exposed bone. When the graft is exposed, the treatment is conservative therapy with antibiotics, chlorhexidine, removal of sequestrate, and optimizing hygiene. If secondary surgery is needed, a minimum of 3 months should be allowed between surgeries to allow for scar maturation.

Oronasal Fistulae The most common sites for development of fistulae are at the junction of the hard/soft palate and the incisive foramen. Smaller fistulae may be addressed with regional palatal flap repair. Larger fistulae can be treated also with regional palatal flaps, or with tongue flaps, facial artery myomucosal flaps (FAMM flaps), and temporalis flaps. May consider waiting for further growth prior to repair to avoid maxillary growth restrictions secondary to surgery if speech or function not affected.

Cleft Cases

Case 1

You have been consulted for a healthy 3-month-old male baby that presents to the craniofacial clinic with the following appearance (See Fig. 10.16):

Fig. 10.16 Cleft case image

- *What is your diagnosis?*

  This is a patient with unilateral complete cleft lip with associated complete cleft palate.

- *How would you treat this patient?*

  Once it has been determined that the patient has been deemed fit for surgery and has been evaluated by all members of the craniofacial team, I would plan for the primary lip repair. I would treat this patient with a primary lip repair using a modified Delaire technique (Use whatever technique you feel most comfortable with here. If you feel more comfortable using the description of the Millard technique, use that).

  After evaluation by anesthesia, I would recommend an oral RAE tube taped to the lower lip to confirm no tension on the upper lip. I would prep and drape the patient in a sterile fashion and make a mark with a surgical marker the edges of my flap design as well as making a mark with a syringe dipped in methylene blue. I would then inject the patient with lidocaine 1% with 1:100,000 epinephrine. Once 5 minutes have elapsed, I would I would then make an incision along these margins, removing the hypoplastic tissue at the cleft margins. Once the hypoplastic tissue is removed, I would dissect the orbicularis oris muscle from each side. Consistent with the Delaire technique, I would dissect bluntly the lateral crura and extend my dissection superiorly and laterally on the cleft side to achieve tension-free closure and to allow the deformed nose to be placed in a more symmetric position. I would first reapproximate the nasal floor by reapproximating the muscles on the lateral margins to the midline. This would reorient the perinasal musculature downward. Next, I would place on stitch in the fibrofatty tissue of the lateral nasal ala and subcutaneous tissue of the midline, and then tie to reapproximate the nostril opening on opposite side. I would then reapproximate the muscle layer with 4-0 polyglactin suture. This would reconstruct the orbicularis oris. Now that the muscle has been reapproximated, I would place one stitch to reapproximate the white line of the lip and then I would reapproximate the underlying mucosa. Lastly, I would reapproximate the skin flaps. After all suturing is complete, I would place a layer of topical skin adhesive in overtop the sutures.

- *Would you treat the palate?*

  No. I would wait until 12 months to treat the palate, as the normal time for treatment is 9–18 months. If completed during this time frame, the incidence of maxillary restriction is at its lowest point without affecting speech development (approx. 18 months).

- *Are cleft palates more common on the right or left side?*

  They are more common on the left.

- *Is there a proposed theoretical explanation?*

  In the embryological rodent model, it has been observed that the right palatal shelf reaches the horizontal position faster than the left during palatal shelf fusion. This delay is believed to occur also in humans which contributes to a higher incidence of left-sided palatal clefts.

- *The same patient returns to you at age 1 year old for palatal repair. How would you treat this patient?*

  I would close the palate at this point, using a Bardach two-flap technique. I would make incisions on either side of the cleft and dissect full thickness anteriorly until reaching the levator palatini muscles posteriorly. After this, my dissection would be split thickness and dissect the mucosa off of the muscle layer. I would then dissect the nasal floor and the muscle layer independently. Finally, I would reapproximate the tissue in layers, first with the nasal floor, then the muscle (reorienting them along the midline, which would reconstruct the palatoglossus, palatopharyngeus, levator veli palatini, and tensor veli palatini) and then the overlying mucosal layer.

- *During the palatal closure, you accidentally sever the greater palatine neurovascular bundle. What do you do?*

  Continue with the surgery. Although the known vascular supply has been severed, there

is still enough circulation from the palatal soft tissue pedicle. In essence, this flap changes from and "axial" pattern to a "random" pattern.

- *During the palatal closure, you have a tear in the mucosal component of your soft tissue flap. What do you do?*

  You can either excise the tear (usually done if a midline tear); or repair it; or place a layer of collagen membrane in the exposed area (the collagen membrane will only be sufficient, if there is an underlying muscle or nasal mucosal layer); or bring a buccal fat pad flap over as an additional tissue layer.

- *The patient follows up in 2 weeks and there is a fistula at the incision line. What do you do?*

  The size of the fistula would dictate treatment. Regardless of size, wait 6 months so that everything has scarred appropriately. Some fistulae resolve spontaneously. If the fistula is small and at midline, you can do a local closure or a redo of the palatoplasty. If it is large, you may need to do a revision palatoplasty. Other options include a buccal mucosal flap or a tongue flap.

- *The fistula resolves, but at age 3 the patient follows up and has been having speech problems, specifically a large amount of hypernasality. Why is this patient having these issues? How would you diagnose the patient?*

  The patient most likely has velopharyngeal insufficiency. This is an inability of the palate to create a separation between the nasal airway and the oral airway. The methods of detecting this include direct mechanism such as nasopharyngoscopy, multi-view video fluoroscopy, or indirect methods such as voice and resonance assessment by a speech pathologist. Considering the patient's age, a speech pathology evaluation is usually used due to less burden on the patient.

- *How would you treat the velopharyngeal insufficiency?*

  Pharyngeal flap surgery is indicated. Some surgeons will wait until age 5, but with a large degree of hypernasality verified by speech pathology, it is unlikely to resolve. If the patient has normal lateral movement of his pharynx, but difficulty in the midline, I would use a superiorly based pharyngeal flap (first dissecting to the prevertebral fascia, then splitting the uvula and insetting the flap into the mucosal pocket).

- *Preoperative work up reveals a diagnosis of Velocardiofacial syndrome. Are there any concerns with this type of patient?*

  Yes. Many of these patients have medially displaced carotid arteries. Dissection in the posterior pharynx could lead to vascular injury. This would require an MRA before surgery.

- *At what age would you treat the alveolar cleft and why?*

  Alveolar grafting is generally completed at 6–9 years of age based on development of the dentition. It has been described to graft once the formation of canine root is (1/2 to 2/3), but also important to carefully assess the development of the central and lateral incisor (if present).

- *Your orthodontist does not have a significant amount of experience in craniofacial patients and asks what you would like with presurgical orthodontics. What do you tell them?*
  - Expand collapsed segments.
  - Avoid displacement of the teeth into the cleft.
  - Remove traumatic crossbites with the mandible.
  - Position premaxilla for symmetry.

- *Where would you harvest the bone graft?*

  I would harvest a particulate bone graft from the iliac crest.

- *The patient's mother is extremely concerned about harvesting from the hip. What other sites could you harvest from?*

  Mandible, calvarium, tibia, or possibly rib.

- *What is BMP? Are there any side effects associated with using it?*

BMP is a protein complex belonging to a family of growth factors that promote osteoinduction. It is stored in the bone matrix and is available commercially for use in bone grafting. It can stimulate bone formation without the presence of bone graft and is usually placed in a carrier such as a collagen sponge.

Disadvantages of its use include a high cost and postoperative swelling at the graft site. Other disadvantages are ectopic bone formation and the theoretical risk of teratogenicity and carcinogenicity. In addition, it is FDA contraindicated for use in children. Considering that long-term studies have not been completed in pediatric patients and that the outcomes are equivalent to autogenous bone grafting in pediatric populations, BMP should be used only as a last resort.

- *How could you design/close your flaps?*

  Mucogingival flap designs include (lateral sliding flap was detailed above) the following:
  - Buccal Finger flap
    - Good blood supply.
    - Adequate soft tissue for closure.
    - Shortens the vestibule.
    - Provides nonkeratinized tissue in the area of the erupting canine.
  - Oblique Sliding Flap
    - Adjacent attached mucosa from lesser and greater segments brought to cleft site.
    - Adequate attached gingiva for tension-free closure of wide alveolar clefts.
    - Minor decrease in vestibular depth.
  - Lateral Sliding flap
    - Excellent blood supply.
    - Brings attached gingiva to area of cleft.
    - Tension-free closure.
    - Shortening of vestibule (although less).
    - Relies on secondary healing of mucosa on denuded area posterior to cleft.

  I would close the oronasal fistula first. This would create a floor to pack the bone graft. I would then close the palatal component creating a pocket to place the bone graft. I would then place the bone graft and then close the buccal flaps over top making sure to create a watertight closure. In addition, I would place a premade splint in the maxilla to stabilize the premaxilla during the bone grafting period. This splint also aids in patient compliance with the no chew diet.

- *What if you can't close the buccal flaps?*

  You can create a back cut at the posterior edge or make cuts in the periosteum to give additional laxity.

- *Why shouldn't you elevate medially in the premaxilla region to gain tissue laxity?*

  Elevation medially towards the premaxilla may compromise blood supply.

- *During the transfer of the bone graft to the mouth, the assistant drops the bone graft on the ground. What do you do?*
  - If you have prepped and consented for the other hip, you can discard the bone graft and harvest from the opposite hip.
  - If you would prefer to save the bone graft, you can wash the bone for 3 minutes with normal saline, place the bone in 10% betadine or 0.04% chlorhexidine for 20 minutes and then wash again for 3 minutes with normal saline.
  - You could harvest at another location after receiving parental consent.
  - You can close the wounds and return another time after the marrow regenerates.

- *The patient returns to the clinic and particles of bone are seen protruding from the bone graft site. The parents report that they have been spitting these particles out for the past few days. What do you do?*

  Place the patient on regimen of peridex and perform gentle superficial debridement with follow-up until the area is fully mucosalized. Although a portion of the graft has been lost, there may be some underlying osseoinduction. Take postoperative radiographs in 3 months and if there is no continuity, you can attempt the procedure again. The other option is orthognathic surgery when growth is complete.

- *The patient has a remaining oronasal fistula. When would you operate?*

  Any surgery should be delayed for 3 months to allow full integration of the graft. If the patient still has an oronasal fistula, a secondary procedure using local tissue rearrangement can be completed if necessary or can be delayed until orthognathic surgery.

Case 2

- *A 4-month-old male with an isolated cleft lip is brought by his mom to your office for consultation. Describe what you see (*Fig. 10.17*).*

  Complete right unilateral cleft lip with asymmetry of alar base of the nose.

- *Is cleft lip more common on the right or left? Is it more common in males or females?*

  Isolated cleft lips are more common on the left and is most common in males.

- *How would you proceed?*
  - Review medical history and pertinent perinatal history.
  - Review any other abnormalities/syndromic features. Ask if any other specialists are involved in his care. Ask about history of clefting in the family and issues with feeding.
  - Perform physical exam.
  - Explain to the mother the protracted course of treatment and the need for multidisciplinary care.

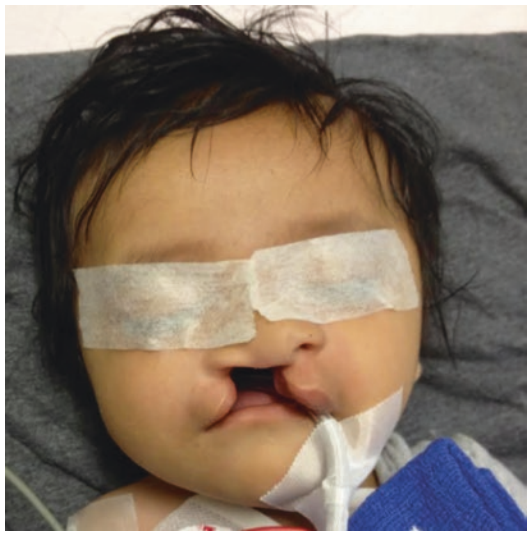

Fig. 10.17 Complete right unilateral cleft lip with asymmetry of alar base of the nose. (Image Courtesy of Dr. Damian Findlay)

- *At what week of gestation, could a cleft be identified?*

  Ultrasound can identify clefting as early as 13–14 weeks of gestation. The sensitivity in detecting CL ± CP prior to 18–20 weeks is much lower.

- *What are associated risks of cleft formation?*

  Chemical exposure (e.g., alcohol or cigarette smoke), radiation, viral infection, maternal hypoxia, teratogenic drugs (e.g., valproic acid and benzodiazepines), nutritional deficiencies, and genetics may play a role.

- *What can a mother do to reduce her chance of her having a child with a cleft?*

  Avoid exposure to known teratogens and take folic acid during pregnancy.

- *What is the difference between a complete and incomplete cleft of the lip?*

  An incomplete cleft only involves the part of the lip and/or alveolus. A complete cleft lip extends into the nares and the entire lip and associated musculature.

- *From an embryological standpoint how does of cleft lip form?*

  Lack of fusion of the median nasal processes and lateral maxillary processes.

- *What is the sequence of management you would expect for a patient with CL?*
  - Primary lip repair (cheilorhinoplasty), timing based on the rule of 10's.
  - Nasolabial revision is delayed until ages 3–5 years.
  - Alveolar grafting is generally completed at 6–9 years based on development of the dentition.

- Early orthodontics begins in the mixed dentition stage with the goal of improving arch form and width, allowing more space for eruption of teeth, and opening fistulae for surgical access.
- Orthognathic surgery if required is planned for 14 to 16 years of age in females, 16 to 18 years in males to address maxillary hypoplasia common in cleft patients.
- Rhinoplasty is often delayed at least 6 to 12 months following maxillary surgery due to changes in septum and tip support with movement of the anterior nasal spine.
- Cleft Scar Revision: Any time after 5 years of age, but preferably at the time of alveolar bone grafting.

- *What is the goal of cleft lip repair?*

  A three-layer closure of skin, muscle, and mucosa that approximates normal tissue and excises hypoplastic tissue at the cleft margins.

- *What are some surgical maneuvers to correct a cleft lip deformity?*
  - Tennison-Randal – a Z-plasty technique.
  - Delaire – rotation-advancement technique.
  - Millard – rotation-advancement technique.

- *Patient presents to your office and is unhappy with his cleft lip repair. What do you see?*

  This is a whistle lip deformity. It is caused either by scar retraction or inadequate reapproximation of the margin portion of the orbicularis oris and skin. This is usually treated with a V-Y plasty and ensuring correct reapproximation of the underlying muscle (Fig. 10.18).

Fig. 10.18 Whistle lip deformity

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Part IV

Anesthesia and Medicine

Outpatient Anesthesia

11

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It should be no surprise that it is expected that you will perform safe outpatient anesthesia in your office. Per the AAOMS parameters of care, all patients whom are an ASA class II and above should be considered for consultation with a physician for medical clarification of the patient's medical condition and clearance to assist in making appropriate decisions for the setting and depth of anesthesia.

Accepted Definitions of Sedation and Anesthesia (AAOMS Parameters of Care 2017) [1] (Table 11.1)

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

Moderate Sedation/Analgesia (Conscious Sedation) is a drug-induced depression of consciousness during which patients respond purposefully[1] to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.

Deep Sedation/Analgesia is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully[1] following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.

General Anesthesia is a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to maintain ventilation 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-

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

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Table 11.1 Continuum of depth of sedation: definition of general anesthesia and levels of sedation/analgesia

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

Modified from Gross et al. [2]

induced depression of neuromuscular function. Cardiovascular function may be impaired. Because sedation is a continuum, it is not always possible to predict how an individual patient will respond. Hence, practitioners intending to produce a given level of sedation should be able to rescue[2] patients whose level of sedation becomes deeper than initially intended. Individuals administering moderate sedation/ analgesia should be able to rescue[2] patients who enter a state of deep sedation/analgesia, whereas those administering deep sedation/analgesia should be able to rescue[2] patients who enter a state of general anesthesia.

Airway Assessment

The goal of the evaluation is to predict the difficulty of mask ventilation and endotracheal intubation, should either be required during surgery. Critical components of the airway assessment include the patient's general appearance, dental exam, Mallampati score, maximum incisal opening, thyromental distance, mandibular protrusion, and BMI.

- *Dental Exam* – look for mobile/carious teeth, edentulism, crowns, dentures, orthodontic appliances, and large tongue/tonsils.
- *Mallampati Score* (See Fig. 11.1) – Original classification scheme was first described in 1985 as a clinical sign to predict difficult intubation and included 3 classes. In the modified Mallampati (by Samsoon and Young) a 4th class is included. Score of 3 or 4 indicates potential difficult intubation. Assessment of patient seated upright with head in neutral position, mouth open, tongue protruded without phonation.
  - Class I: soft palate, uvula, tonsillar pillars, and fauces are visible.
  - Class II: superior 2/3 of uvula and soft palate are visible.
  - Class III: <1/3rd of uvula and soft palate are visible.
  - Class IV: soft palate not visible.
- *Maximum Incisal Opening* (MIO) – normal is >40 mm, <35 mm should be considered limited. Be cautious of patients with a history of temporomandibular joint disorders or a history of head and neck radiation. The ability to perform specific airway maneuvers depends in part on the degree that a patient is able to maximally open. At a MIO of 20 mm, it is possible to insert an oral or nasal airway, GlideScope ™, and perform a fiberoptic intubation. As the MIO increases to 30 mm, insertion of a laryngeal mask airway (LMA) becomes possible. At a MIO of 40 mm, direct laryngoscopy (DL) intubation may be performed.

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

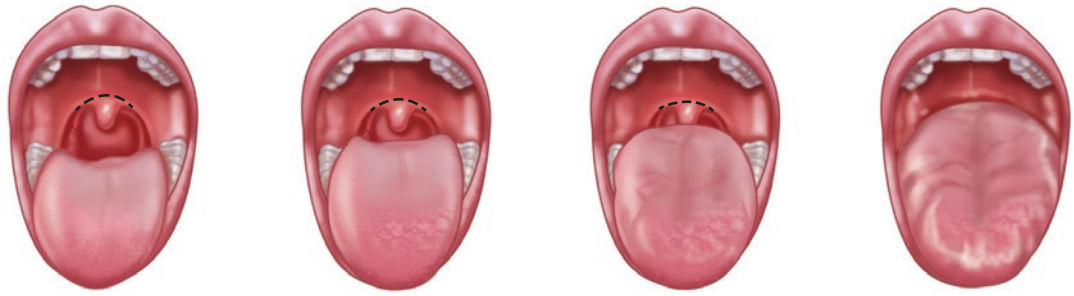

Fig. 11.1 Mallampati classification. (Reprinted with permission from Strauss and Noordhoek [11])

- *Thyromental Distance* – the distance between the top of the thyroid cartilage and the menton of the mandible. It is an indicator of the ability to displace the tongue during direct laryngoscopy. A distance of <6.5 cm (three finger breadths) may indicate difficulty with intubation.
- *Mandibular Protrusion* – evaluate patient's ability to protrude the lower jaw so that the mandibular incisors are in front of the maxillary incisors. This maneuver correlates with the clinician's ability to sublux the mandible during laryngoscopy.
- *Upper Lip Bite Test* (ULBT) – assesses a patient's ability to reach and cover their upper lip with their mandibular incisors (similar to mandibular protrusion test).
  - Grade 1: fully covers the upper lip with lower incisors.
  - Grade 2: partially covers the upper lip with lower incisors.
  - Grade 3: cannot reach the upper lip with lower incisors.
- *Body Mass Index (BMI) and Obesity* – patients often have increased parapharyngeal fat which can cause difficulty with airway maneuvers and these patients are more prone to desaturation during sedation. On the opposite spectrum, be extremely cautious of those with a BMI of <18.5, as they are at a higher rate of mortality with anesthetic challenges. Patients with a BMI of <18.5 are more prone to hypokalemia, dehydration, delayed gastric emptying, decreased GFR, and have a predisposition to aspiration.
  - BMI Scale (m/kg^2):
    Normal: 18.5–24.9
    Overweight: 25–29.9
    Obese: 30–39.9
    Morbidly obese: 40–49.9
    Super obese: >50
- *Neck Circumference* – if greater than 43 cm (17 inches), associated with difficulty for intubation, more predictive than BMI.

Laboratory Tests and Other Studies

- Testing should be based on the history and physical of the patient and the nature of the surgical procedure.
- An electrocardiogram (EKG) should be obtained in all adults over the age of 65, and any patient with a history of hypertension (HTN), cardiac disease, substance abuse, or eating disorder.
- A fasting fingerstick glucose (FSG) should be obtained on all diabetics both prior to surgery and postoperatively prior to discharge.
- Other lab tests that may be indicated depending on the patient's reported history include Hematocrit (Hct), chest X-ray (CXR), urine pregnancy test (b-hCG), BMP, LFTs, coagulation studies, or echocardiogram.

Evaluation of Functional Status

- During the preoperative evaluation, it is important to ask patients about their exercise

capacity, as this is a significant determinant of perioperative risk. In general, patients with good exercise tolerance are at lower risk for cardiopulmonary complications during anesthesia. One commonly used measure of exercise tolerance is metabolic equivalent tasks (METs).

- MET is a physiological measure that expresses the energy cost of performing various physical activities. It is expressed as the ratio of a patient's metabolic rate during a specific physical activity over the reference metabolic rate, which is the resting or basal oxygen consumption of a 40-year-old, 70-kg man (3.5 mL O_2/kg/min or MET 1) [3].
- Perioperative risk is elevated in patients with <4 METs. Activities of <4 METs include watching TV, shopping, golfing with a cart, and walking slowly (2–3 mph).
- Activities associated with >4 METs include climbing a flight of stairs, bicycling, walking >4 mph, and performing housework.

American Society of Anesthesiology (ASA) Classification

American Society of Anesthesiology Clinical Information [Internet]. Schaumburg, IL: American Society of Anesthesiologists; 2017. ASA physical status classification system; [approved 2014 Oct 15; Available from: https://www.ncbi.nlm.nih.gov/books/NBK441940/]

- ASA Class I: A healthy patient (healthy, nonsmoking, no or minimal alcohol use).
- ASA Class II: *mild* systemic disease (WITHOUT substantive functional limitations).
  - For example, current smoker, social alcohol drinker, pregnancy, obesity (BMI 30–40), well-controlled DM/HTN, mild lung disease.
- ASA Class III: *severe* systemic disease that *limits activity but is not incapacitating* (substantial functional limitations).
  - For example, poorly controlled DM/HTN, COPD, morbid obesity (BMI ≥40), active hepatitis, alcohol dependence or abuse, implanted pacemaker, moderate reduction of ejection fraction, ESRD undergoing regularly scheduled dialysis, history (>3 months) of MI, CVA, TIA, or CAD/stents.
- ASA Class IV: *severe* systemic disease that is a *constant threat to life*.
  - For example, recent (< 3 months) MI, CVA, TIA, or CAD/stents, ongoing cardiac ischemia or severe valve dysfunction, severe reduction of ejection fraction, sepsis, DIC, ARD, or ESRD not undergoing regularly scheduled dialysis.
- ASA Class V: moribund patient who is not expected to survive without operation.
- ASA Class VI: declared brain-dead patient whose organs are being removed for donor purposes.
- The addition of "E" denotes emergency surgery – added when a delay in treatment of the patient would lead to a significant increase in the threat to life or body part.

NPO Guidelines

In 2017, the American Society of Anesthesiology published a set of practice guidelines regarding preoperative fasting prior to anesthesia. These guidelines were meant to reduce the occurrence and severity of complications related to perioperative pulmonary aspiration of gastric contents [4].

The ASA's preoperative fasting guidelines for elective procedures requiring general anesthesia, regional anesthesia, or sedation/analgesia are summarized as follows:

- Clear liquids – at least 2 hours.
  - Examples of clear liquids include water, fruit juice without pulp, carbonated beverages, clear tea, plain gelatin, and black coffee. These liquids should *NOT* include alcohol. The volume of liquid ingested is less important than the type of liquid ingested.
- Breast milk – at least 4 hours.
- Infant formula – at least 6 hours.

- Non-human milk – at least 6 hours.
- Solids, light meals, non-clear liquids – at least 6 hours.
- Heavy meal or fried/fatty foods – at least 8 hours.

Note: The routine use of preoperative gastrointestinal stimulants (e.g., metoclopramide), medications that block gastric acid secretion (e.g., H_2 antagonists, PPIs), antacids (e.g., sodium citrate, magnesium trisilicate), antiemetics (e.g., droperidol, ondansetron), to decrease the risk of pulmonary aspiration in patients who have no apparent increased risk for pulmonary aspiration is *NOT* recommended. The use of anticholinergics (e.g., atropine, glycopyrrolate) is *NOT* recommended to reduce risk of pulmonary aspiration.

Airway Management

Oxygen Delivery Methods

Mouth to Mouth

- Exhaled air contains 17% oxygen.

Nasal Cannula

- Each increase liter per minute is approximately 4% above room air.
- $FiO_2 = 20 + [4 \text{ L/min} \times \text{L/min}]$.
- Flow greater than 4 L/min, although delivering 36%, may be uncomfortable.

Simple Facemask

- Flow 8–12 L/min.
- FiO_2: 35–65%.
- Increase approximately 4% per liter flow.

Non-Rebreather

- Flow rate should be min 6–15 L/min.
- FiO_2: 60–100%.
- Delivered oxygen ~60%, each L/min increase will raise FiO_2 ~5%.

Mask Ventilation

- Can be used for spontaneously breathing patients or apneic patients via positive pressure ventilation.
- Uses:
  - Primary means of ventilation for a short procedure.
  - Bridge to establishing a more definitive airway.
  - Pre-oxygenation prior to GA induction.
  - Valuable rescue technique when intubation proves difficult.
- Caution if full stomach, severe facial trauma or C-spine fracture.
- Difficulty occurs when ventilating via the facemask is not possible because of an inadequate mask seal (e.g., beard hair), excessive gas leak, and/or excessive resistance to the ingress or egress of gas. This may be helped by oro/naso-pharyngeal airways.
- Risk factors for bag mask ventilation difficulty include age greater than 55 years, BMI > 26 kg/m^2, edentulism, presence of a beard, and history of snoring [5].

Oropharyngeal Airways

- Follows the curvature of the tongue, pulling the tongue and epiglottis away from the posterior pharyngeal wall.
- Not appropriate for use in conscious patients (can cause coughing, retching, or laryngospasm due to irritation at base of tongue). Best used in the deeply anesthetized patient.
- Sized by measuring from the corner of a patient's mouth to the angle of the jaw.

Nasopharyngeal Airways

- Less stimulating than oropharyngeal airways, more appropriate for lightly anesthetized patients.
- Lubricate prior to insertion and insert with the bevel facing the nasal septum.

Laryngeal Mask Airway (LMA)

- Supraglottic airway that is blindly inserted into the pharynx.
- Provides a patent conduit for ventilation, oxygenation, and delivery of anesthetic gases without tracheal intubation.
- Less invasive than intubation but provides a more definitive airway than a facemask.
- Can be used for either spontaneous ventilation or PPV.
- Allows for delivery of O_2 and inhaled anesthetics during spontaneous ventilation or via PPV at pressures up to 20 cmH_2O.
- Flexible LMA Tube – allows the tube to be moved out of the surgical field without displacement of the cuff, or loss of seal for the anesthesiologist.

Endotracheal Intubation

- Gold standard for airway management.
- Establishes a definitive airway.
- Maximal protection against aspiration of gastric contents.
- Allows for delivery of O_2, inhalational anesthetics and allows for PPV with higher airway pressures than with a facemask or supraglottic airway (LMA).

Emergency Percutaneous Airways

Cricothyrotomy

- Invasive technique that provides access to the airway in situations when either noninvasive maneuvers have failed or when it is clinically indicated as a primary plan to secure the airway.
- Not considered a permanent airway, and after placement plans should be made for either the removal or conversion to a formal tracheostomy.
- Technique:
  - Step 1: Extend the head and neck and identify and immobilize the cricothyroid membrane. (Make an initial vertical incision if identification is not possible.)
  - Step 2: Make a horizontal stab incision through the skin and cricothyroid membrane and keep the blade in place.
  - Step 3: Use a tracheal hook to apply caudal and outward traction on the cricoid cartilage; remove the scalpel.
  - Step 4: Insert the ETT tube (6.0 cuffed mm) or tracheostomy tube (No.4 cuffed tracheostomy tube) and inflate the cuff.
  - Step 5: Ventilate with a low-pressure source.
  - Step 6: Confirm pulmonary ventilation.
- Note: In children <6 years of age, cricothyrotomy is contraindicated because the cricoid cartilage is the narrowest portion of the airway and the isthmus of the thyroid gland typically reaches the level of the cricothyroid membrane. Needle cricothyrotomy with transtracheal jet ventilation is indicated in this pediatric population.
- Conversion to tracheostomy is normally recommended within 72 hours to prevent subglottic stenosis [6].

Transtracheal Needle Ventilation

- Allows for 30 minutes to 2 hours of ventilation.
- Must be avoided in those with tracheal trauma.
- Technique:
  - Step 1: Extend the head and neck and identify and immobilize the cricothyroid membrane. Normally it is 2 cm in width and 2–3 cm below the laryngeal prominence [7].
  - Step 2: Puncture cricothyroid membrane at a 90° angle with saline-filled syringe with 14 gauge catheter needle for adults or 18 gauge for pediatric and draw back until air enters the syringe. This indicates entry to airway.
  - Step 3: Advance catheter off caudally at a 30–45° angle.
  - Step 4: Attach syringe to 100% wall oxygen at 50 psi for adults, 10–25 psi for children 8 years or older, or 5–10 psi for children 5–8 years of age [7]. A bag-valve mask using the connector from a 7 ET tube inserted into the back of a plunger less 3 mL Luer-lock syringe can also be used.

Pediatric Airway and Anatomy Considerations

Pediatric Airway Is Smaller

- In the pediatric airway, there is greater risk of airway obstruction from small foreign bodies.
- Minimal swelling of the small pediatric airway will result in a relatively greater reduction in airway diameter than would occur in the larger airways of the adult.
- The internal diameter of the appropriate endotracheal tube for a child will roughly equal the size of that child's little finger, but this estimation may be difficult and unreliable.
- For children 1–10 years of age, an estimate based on the child's age is available using the following equation.
- Uncuffed endotracheal tube size = (age in years/4) + 4.
- Intubation is preferred method of airway security.
- Cricothyroidotomy is contraindicated in children less than 12 years of age.
- Emergent airway is a tracheostomy.

Large Tongue Relative to a Small Mouth

- This increases the risk of the tongue obstructing the airway than in the adult. This makes it essential that there is correct positioning of the head and jaw when opening the airway.

Infants Have a Larger Occiput

- The large occiput of the infant flexes the head forward when he/she is placed prone on a flat surface. This is important in airway-opening maneuvers and cervical spine immobilization.
- Care must be taken not to hyper-extend the neck, as this may result in airway obstruction or spinal cord damage in the event of a cervical spine fracture.

Infants Are Obligatory Nose Breathers

- In the first 4–6 months of age, infants breathe exclusively through the nose and will experience respiratory distress if the nose is blocked. Care must be taken to ensure that the nares are patent in cases of trauma involving the infant patient.

Trachea/Presence of Tonsils and Adenoids

- The cartilaginous nature of the pediatric airway renders it soft which makes it more collapsible than the adult airway.
- The tonsils in toddlers and young children may be enlarged, contributing to airway obstruction and making nasal passage of an endotracheal tube difficult.

Larynx Is Higher and More Anterior

- The larynx sits at the level of the 2nd–3rd cervical vertebrae in the young child, compared with the 6th–7th cervical vertebrae in the adult.

Shape of the Epiglottis

- The epiglottis of the young child is floppy and projects posteriorly. This makes the technique of tracheal intubation more difficult.

Cricoid Ring Is the Narrowest Point in the Airway

- The cuff of the endotracheal tube sits at the level of the cricoid ring, which then takes up valuable airway diameter. In addition, the cricoid region is lined with pseudostratified, ciliated epithelium bound to areolar tissue, which is susceptible to edema. For these reasons, an uncuffed endotracheal tube is used for pediatric intubation.

The Length of the Trachea Is Smaller

- The pediatric trachea is comparatively shorter than that of the adult, and the risk of dislodgement of the endotracheal tube is greater.

Cervical Spine

- Larger head, in particular in the occipital region. This has been previously discussed with regard to the airway. However, it is also very important in cervical spine alignment. The ligaments of the pediatric vertebral column are relatively lax, compared to those in the adult spinal column. This increases the likelihood that movement of vertebrae may occur, resulting in injury to the normal spinal cord.
- The fulcrum is at C1–2 not C6–7. Therefore, cervical spine injuries in children under the

age of 8 most commonly occur in the first three vertebrae, whereas in the adult, injuries tend to be lower in the vertebral column.

Difficult Airway Algorithm [8]

The figure below (see Fig. 11.2) summarizes the steps for the management of the difficult airway.

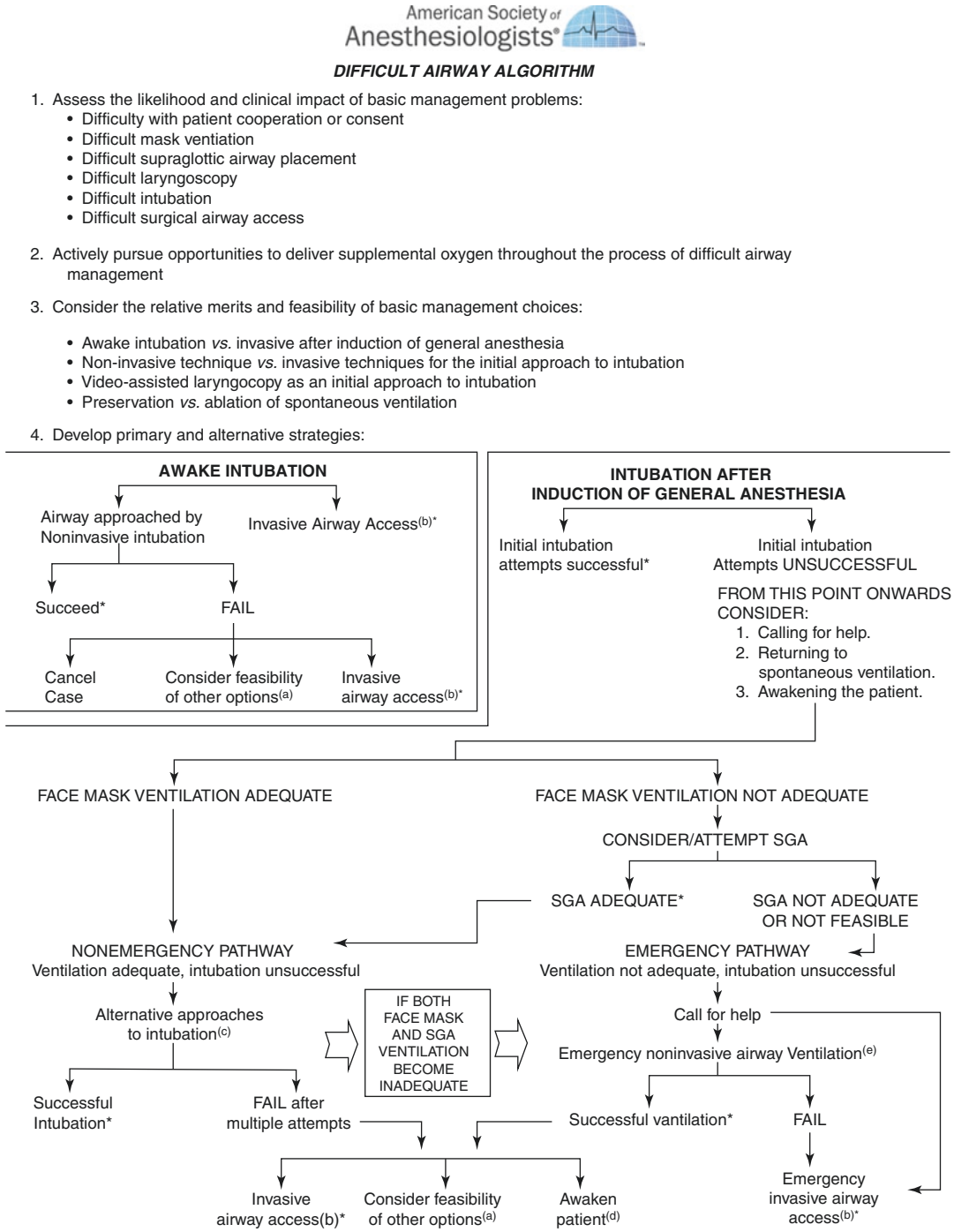

Fig. 11.2 Difficult airway management algorithm. (Reprinted with permission from Apfelbaum et al. [8])

AAOMS Standards for Basic Anesthetic Monitoring During Deep Sedation and General Anesthesia [1]

- During all anesthetics, the patient's oxygenation, ventilation, circulation should be continually evaluated.
- Temperature measurement instruments should be available.

Oxygenation

- Must use a quantitative method of assessment such as pulse oximetry – the variable pitch pulse tone and low threshold alarm must be audible to the anesthesia provider.
- *Pulse Oximetry*
  - Based on the red and infrared light absorption characteristics of oxygenated and deoxygenated hemoglobin.
  - Photodetector only amplifies light of an alternating intensity (pulsating artery).
  - Oxygenated Hgb absorbs more infrared light and allows more red light to pass through.
  - Deoxygenated (reduced) Hgb absorbs more red light and allows more infrared light to pass through.
  - Two wavelengths emitted: (1) 660 nm (deoxygenated Hgb) and (2) 940 nm (oxygenated Hgb).
  - After the transmitted red and infrared signals pass through the measuring site and are received at the photodetector, the oxygenated vs. non-oxygenated Hgb to the total amount of Hgb is expressed as a fraction of the hemoglobin pool in the oxygenated form.
  - False high readings occur with carbon monoxide as COHgb (elevated in smokers).
  - Methemoglobinemia will overestimate the SaO_2, and readings will not fall below 85%.
  - SpO_2 reported as accurate with a systolic BP > 80 mmHg.
  - Oximetry is in the presence of anemia accurate to 2–3 g/dL of hemoglobin.

Ventilation

- Continual monitoring for the presence of expired carbon dioxide (end tidal CO_2 Fig. 11.3).
- Continual observation of chest rise and auscultation of breath sounds (consider precordial stethoscope).

Circulation

- The electrocardiogram (ECG) shall be continuously displayed from the beginning of anesthesia until preparing to leave the anesthetizing location. Leads II and V5 are more sensitive to ischemia.
- Arterial blood pressure and heart rate must be determined and evaluated at least every 5 minutes.
- If providing general anesthesia, must continually evaluate circulatory function by at least one of the following: palpation of a pulse, auscultation of heart sounds, monitoring of a tracing of intra-arterial pressure, ultrasound peripheral pulse monitoring, or pulse plethysmography or oximetry.

Temperature

- Must be monitored when clinically significant changes in body temperature are intended, anticipated, or suspected.

Recovery and Discharge Assessment

Modified Aldrete Score

Assigns a score of 0–2 to the following categories: activity, breathing, circulation, consciousness, and oxygen saturation (Table 11.2) [10].

A score of 9 out of 10 is required for discharge from the facility/PACU.

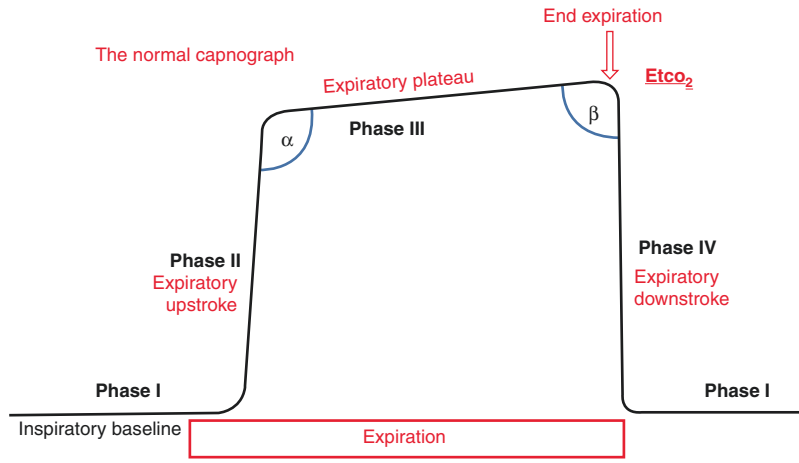

Fig. 11.3 The capnographic of the normal waveform. Evaluated with the change in concentration of CO_2 (mmHg) gas over a function of time (normally seconds). Phase 1 – exhalation of CO_2 free gas from dead space (inspiratory baseline). Phase 2 – rapid rise in CO_2; combination of dead space and alveolar gas. Slow uptake in this phase may represent an upper airway obstruction, obstruction of the endotracheal tube, or bronchospasm. A shark fin pattern, without an expiratory plateau, may be suggestive of obstructive lung disease such as asthma of COPD. Phase 3 – exhalation of mostly alveolar gas (expiratory/alveolar plateau). Phase 4 – inhalation with return to baseline CO_2 concentration levels (expiratory downstroke). (Reprinted with permission from Manifold et al. [9])

Table 11.2 Modified Aldrete score

Variable evaluated	Score
Activity	
Able to move 4 extremities on command	2
Able to move 2 extremities on command	1
Able to move NO extremities on command	0
Breathing	
Able to breathe deeply and cough freely	2
Dyspnea	1
Apnea	0
Circulation	
Systemic blood pressure ≤20% of preanesthetic level	2
Systemic blood pressure 20–30% of preanesthetic level	1
Systemic blood pressure ≥50% of preanesthetic level	0
Consciousness	
Fully awake	2
Arousable	1
Not responsive	0
Oxygen saturation (pulse oximetry)	
>92% while breathing room air	2
Needs supplemental O_2 to maintain saturation >90%	1
<90% with supplemental O_2	0

Pharmacological Principles

Pharmacokinetics

- Describes the absorption, distribution, metabolism, and excretion of drugs.
- Describes how the body affects the drug.

Pharmacodynamics

- Describes therapeutic and toxic organ system effects of drugs.
- Describes how the drug affects the body.
- Knowledge of a drug's pharmacokinetics and pharmacodynamics defines the relationship between the dose of drug administered and the resulting pharmacological effect as depicted by the dose-response curve.

Theories of Anesthetic Action

Past understanding of anesthetic actions attempted to identify a unitary hypothesis of

anesthetic effects. This hypothesis proposes that all inhalation agents share a common mechanism of action at the molecular level. This was previously supported by the observation that the anesthetic potency of inhalation agents correlates directly with their lipid solubility (Meyer-Overton rule). There is an ongoing debate as to the mechanism of anesthetic action. Anesthetic interactions at specific protein ion channels, as well as more nonspecific membrane effects, may combine to produce the anesthetized state.

General anesthesia is an altered physiological state characterized by reversible loss of consciousness, analgesia, amnesia, and some degree of muscle relaxation. The multitude of substances capable of producing general anesthesia is remarkable: inert elements (xenon), simple inorganic compounds (nitrous oxide), halogenated hydrocarbons (halothane), ethers (isoflurane, sevoflurane, desflurane – see Table 11.3), and complex organic structures (propofol). A unifying theory explaining anesthetic action would have to accommodate this diversity of structure. In fact, the various agents probably produce anesthesia by differing sets of molecular mechanisms. Inhalational agents interact with numerous ion channels present in the CNS and peripheral nervous system. Nitrous oxide and xenon are believed to inhibit N-methyl-D-aspartate (NMDA) receptors. NMDA receptors are excitatory receptors in the brain. Other inhalational agents may interact at other receptors (e.g., gamma-aminobutyric acid [GABA]-activated chloride channel conductance) leading to anesthetic effects.

Table 11.3 Different MAC values for commonly used inhalation anesthetics

MAC in adults 30–55 years		
Anesthetic	In O_2	In 60–70% N_2O
Desflurane	6.00	2.83
Halothane	0.75	0.29
Isoflurane	1.15	0.50
Sevoflurane	2.3	1.3
Xenon	71	
Nitrous oxide	104	—

MAC (Minimal Alveolar Concentration)

- Minimum alveolar concentration or partial pressure at one ATM which will prevent "gross purposeful movement" in response to a surgical stimulus in 50% of patients (essentially ED_{50}).
- Clinically, 1.25–1.3 times MAC is required to include 90+% of patients.
- Higher MAC, the less potency of the inhalational anesthetic (Table 11.3).
- Choice of inhalation agent is commonly determined by its physiological effects on the body (Table 11.4).

This Level of Partial Pressure Gradient Depends Upon

- F_i Inspired concentration.
- V_m Minute volume.
- Transfer to the alveoli.
- Transfer from the alveoli.
- CO Cardiac output.
- AV Partial pressure gradients.
- Transfer to the brain.

Other Factors that Influence the Uptake or Elimination of Inhalational Agents

- Concentration Effect – when an inhalational agent is administered in a low concentration, for example 1% volume, uptake of half the volume results in a 0.5% volume concentration. If, however, 80% volume concentration is administered, uptake of half the volume

Table 11.4 Cardiovascular effects of inhalational anesthetics compared

	Cardiac output	Systemic vascular resistance	Mean arterial pressure	Heart rate
Halothane	↓	↔	↓	↓↓
Enflurane	↓↓	↓	↓↓	↑
Isoflurane	↓	↓	↓	↑
Desflurane	↔	↓	↓	↑
Sevoflurane	↔	↓	↓	↔
N_2O	↓	↑	↔	↑
Xenon	↔	↔	↔	↓

results in a 66–70% volume concentration as the alveolar volume contracts.
- Second Gas Effect – when two inhalational agents are administered (one in a large concentration and another in a small concentration), the first agent may increase the concentration of the second agent. This follows the same concept as the concentration effect.
- Diffusion Hypoxia – the rapid wash out of oxygen by an inhalational agent, which results in a hypoxic alveolar concentration of oxygen.
- Overpressure Technique – in order to rapidly establish the desired partial pressure of an inhalational agent, the circuit is primed at the highest concentration and the agent is delivered at high concentration via facemask until the rapid induction is achieved. This technique, in conjunction with pre-priming of the circuit with the maximum deliverable concentration of agent will allow "Vital Capacity Induction."

Wash Out or Elimination

In general, wash out is the inverse of wash in. Factors affecting wash out include agent solubility, duration of anesthetic, and minimally, metabolism.

Inhalation Considerations

1. Soda Lime Reactions:

- Carbon dioxide absorption is important to allow rebreathing of volatile agents possible. Usually NaOH or KOH mixed with hardeners such a silica and kieselguhr. Contain an indicator that changes color when exhausted.
- CO is produced from extremely dry soda lime.
- Sevoflurane is unstable in soda lime and produces a Compound A (Compound A is nephrotoxic).
- Amsorb is used instead of soda lime, which eliminates these reactions.

2. Halothane Hepatitis

- About 20% of halothane is metabolized to trifluoroacetic acid, chloride, and bromide.
- Trifluoroacetic acid combines with liver proteins forming a hapten that activates an antibody reaction on repeated exposure leading to immune-mediated liver destruction.

Intravenous Agents

Propofol (Diprivan®)

- Sedative-hypnotic.
- Propofol: 2,6-diisopropylphenol 1% injectable emulsion that is an intravenous sedative-hypnotic agent used for induction and maintenance of anesthesia or sedation.
- Mechanism of action is via potentiation of GABA receptor, causing depression of the reticular activating system.
- It contains soybean oil, glycerol, egg lecithin, and depending upon the manufacturer it may contain EDTA, sodium metabisulfite or benzyl alcohol to retard microbial growth.
- It has a fast onset of action, usually within 40 seconds (one arm-brain circulation) due to high lipid solubility.
- Short duration of action is due to rapid clearance. Propofol is primarily metabolized in the liver and then excreted by the kidneys. The metabolites of propofol are thought to be inactive.
- Extra-hepatic metabolism of propofol occurs in the kidneys. A minor amount of propofol is excreted unchanged in the feces and lungs.
- Propofol has a half-life of 4–7 hours and a clearance of 20–30 (mL/kg/min).
- Central Nervous System (CNS) Effects:
  - The hypnotic action of propofol is mostly mediated by binding to the GABA receptor sites.
  - Propofol has poor analgesic properties.
  - It reduces intracranial pressure (ICP) and cerebral blood flow (CBF).
  - It has anticonvulsant and antiemetic effects.
- Cardiovascular System Effects:
  - A cardiovascular depressant with direct myocardial depression (negative inotropism), decrease in systemic vascular resistance and hypotension. Inhibits normal

baroreflex response to hypotension with only a small increase in heart rate.
  - Bradycardia and asystole have occurred in healthy adults after propofol induction.
- Respiratory System Effects:
  - Profound respiratory depressant.
  - Causes apnea following induction dose.
  - Inhibits hypoxic respiratory drive and depresses normal response to hypercarbia.
  - Reduction in tidal volume and respiratory rate causing reduction in minute ventilation.
  - Reduction in upper airway reflexes and protects against bronchospasm via bronchodilation.
  - Lower incidence of wheezing on induction.
- Side Effects:
  - Pain on injection is common. This can be reduced by prior injection of lidocaine or mixing propofol with lidocaine.
  - Muscle twitching, spontaneous movement, or hiccupping can occur with propofol induction.
- Caution needed for the following patients:
  - Elderly – consider 20% lower doses, administer bolus more slowly and/or in smaller increments.
  - Impaired cardiac function – use lower doses or select other agents.
  - Reports of avoidance with patients with peanut, soy, and egg allergy. A review by the American Academy of Allergy, Asthma and Immunology reports its safe use in these patients without special precautions. The majority of reports on allergic reaction to propofol have involved patients without allergy to peanuts, soy, or egg.
- Clinical Uses:
  - *Induction of General Anesthesia*
    Adults: 1–2.5 mg/kg
    Children: 2.5–3.5 mg/kg
  - *Maintenance of GA*
    Infusion of 100–200 mcg/kg/min
  - *Sedation*
    Infusion of 25–100 mcg/kg/min
    Intermittent bolus (adults): 20–50 mg
  - *Antiemetic*
    10–20 mg bolus

Ketamine (Ketalar®)

- Partially water-soluble and highly lipid-soluble derivative of phencyclidine. It provides dissociative anesthesia, which causes disassociation between thalamus and limbic system. It also renders the patient in a cataleptic state where the patient appears conscious but is unable to process or respond to a stimulus.
- N-methyl-D-aspartate (NMDA) receptor antagonist.
- Potent analgesic.
- High lipid solubility and low protein binding ensure rapid onset of action.
- Metabolized in the liver to several metabolites, some of which retain activity (norketamine).
- Inactive metabolites excreted by the kidneys.
- Central Nervous System Effects:
  - Increases CBF and ICP.
  - Anticonvulsant effect.
  - Induces analgesia, amnesia, and hypnotic properties.
  - Unpleasant psychomimetic effects.
  - Not recommended for patients with intracranial pathology.
  - Emergence Phenomenon: vivid, disturbing dreams, hallucinations, out-of-body experience, and delirium. These reactions are limited by pretreatment with benzodiazepines.
- Cardiovascular System:
  - Significant increase in systemic blood pressure, heart rate, and cardiac output.
  - These indirect effects result from centrally mediated sympathetic stimulation.
- Respiratory System:
  - Ventilatory drive is minimally affected with induction dose.
  - Transient hypoventilation and rarely apnea can follow a rapid intravenous bolus.
  - Upper airway reflexes remain largely intact.
  - It is a potent bronchodilator.
- Side Effects:
  - Psychomimetic effects.
  - Increase in salivation; attenuated by premedication with an anticholinergic such as glycopyrrolate.

  - Can increase the risk of laryngospasm, especially in children.
- Clinical Uses:
  - *Premedication for special needs patients and uncooperative pediatric patients.*
  - *Induction of GA.*
    IV: 1–2 mg/kg.
    IM: 3–5 mg/kg.
  - *Sedation.*
    IV: 0.2–0.5 mg/kg, intermittent boluses.
    PO: 1mg/kg.

Midazolam (Versed®)

- 1,4-Benzodiazepine compound.
- Sedative-hypnotic.
- Mechanism of action is via GABA potentiation.
- Water soluble, converts to lipid-soluble compound by exposure to pH of blood.
- Administered IV, IM, IN.
- Intranasal, buccal, and sublingual administration of midazolam is effective.
- Highly lipid soluble ensures rapid onset.
- Highly protein bound.
- Metabolized by liver with metabolites mainly excreted in urine.
- Fast onset.
- Excellent anterograde amnesia.
- Central Nervous System Effects:
  - Activation of GABA receptor complex.
  - Minimal effect outside CNS.
  - Anterograde amnesia and anxiolysis.
  - Anticonvulsant.
  - Mild muscle relaxant mediated at spinal cord level.
  - Reduce CBF and ICP.
- Cardiovascular System Effects:
  - Minimal effects: Slight decrease in arterial blood pressure, cardiac output, and peripheral vascular resistance.
- Respiratory System Effects:
  - Minimal depression of ventilation.
  - Decreased ventilatory response to CO_2, which is usually not significant. It is more severe when administered in conjunction with opioids.
- Side Effects:
  - Allergic reactions extremely rare to nonexistent.
  - Hiccups (singultus) upon administration.
- Clinical Uses:
  - *Premedication and sedation.*
    IV: 1–2 mg/kg.
    PO: 0.5–1 mg/kg.
    IM: 0.05–0.1 mg/kg.
    IN: 0.5 mg/kg.
  - *Induction of GA.*
    IV: 0.1–0.4 mg/kg.
  - Suppression of seizure activity.
  - Sedative effects are increased with elderly patients, decreased doses are recommended.
- *Reversal by Flumazenil (Romazicon®)*
  - Flumazenil is a benzodiazepine receptor ligand with high affinity. It interacts with the receptor in a concentration-dependent manner. It is a competitive antagonist at the benzodiazepine receptor site. It does not displace the agonist, but rather occupies the receptor when an agonist dissociates from the receptor.
  - Initial dose: 0.2 mg IV one time over 15 seconds.
  - Repeated doses: 0.2 mg may be given every minute until the desired level of consciousness is achieved.
  - Maximum total dose 1 mg.
  - Most patients respond to 0.6–1 mg.

Fentanyl (Sublimaze®)

- Narcotic agonist-analgesics of opiate receptors (primarily μ) that inhibit ascending pain pathways.
- Fast onset and short duration of action.
- Primarily metabolized by the liver (CYP3A4).
- End products mostly eliminated by the kidneys.
- Central Nervous System Effects:
  - Analgesia and sedation without loss of consciousness.
  - Tolerance and dependence with repeated opioid administration.
  - Higher incidence of nausea vomiting due to stimulation of chemoreceptor trigger zone in the medulla oblongata.

- Cardiovascular System Effects:
  - In general, does not seriously impair cardiovascular function.
  - Causes bradycardia.
  - Arterial blood pressure often falls from bradycardia, vasodilation, and decreased sympathetic reflexes.
- Respiratory System Effects:
  - Depress ventilation, especially respiratory rate.
  - Apneic threshold is elevated and hypoxic drive is decreased.
  - Can induce chest wall and laryngeal muscle rigidity.
- Side Effects:
  - Nausea and vomiting due to stimulation of chemoreceptor trigger zone in the medulla.
  - Constipation.
  - Tolerance and physical dependence with repeated administration.
  - Pruritus.
  - Hypothermia.
  - Shivering.
- Clinical Use:
  - 2 μg/Kg IV.
  - *Opioid Reversal by Naloxone* (Narcan®).

    Naloxone is a competitive opioid receptor antagonist.

    Its affinity for opioid μ receptors appears to be much greater than for opioid κ or δ receptors. Naloxone has no significant agonist activity.

    Initial adult dose: 0.4 mg to 2 mg IV.

    If the desired response is not obtained, doses should be repeated at 2–3 minute intervals, generally up to a total dose of 10 mg.

    Initial pediatric dose: 0.01 mg/kg IV; if desired response is not obtained, may give 0.1 mg/kg IV.

Neuromuscular Blocker Agents

Succinylcholine (Anectine®)

- A depolarizing noncompetitive agent that works at the cholinergic receptor, which has a rapid onset (30–60 seconds) and short duration (2–3 minutes).
- It is associated with muscular pain and anaphylactic reactions.
- It is known as a trigger agent for malignant hyperthermia.
- Repeated doses are associated with bradycardia and possible asystole. Pretreatment with atropine lessens this complication.
- Rarely (1 in 4000 patients), a deficiency of the enzyme pseudocholinesterase occurs, which would prolong its metabolism and thus the duration of action substantially.
- *Clinical Use*:
  - Facilitation of endotracheal intubation and rescue from laryngospasm. Consider pretreatment with atropine.
  - Intubation dose: 0.3–1.1 mg/kg IV single bolus.
  - Rescue from laryngospasm dose: use 20% of intubating dose, 20 mg intravenously or consider intubating dose if rapid sequence intubation may be planned.

Serious Pediatric Risks

- There have been rare reports of acute rhabdomyolysis with hyperkalemia followed by ventricular dysrhythmias, cardiac arrest, and death after the administration of succinylcholine to apparently healthy children who were subsequently found to have undiagnosed skeletal muscle myopathy, most frequently Duchenne's muscular dystrophy.
- Rescue from laryngospasm and facilitation of emergent intubation is still performed primarily with succinylcholine, but alternatively, increasing the depth of anesthesia with propofol or a volatile anesthetic agent (Sevoflurane) may be considered. Rocuronium may also be considered as an alternative to succinylcholine.

Rocuronium (Zemuron®)

- Non-depolarizing muscle relaxant that works at the cholinergic receptor.
- Has a much faster onset of action than other non-depolarizing agents, especially when

used in higher doses, it is almost comparable with succinylcholine.

- *Clinical Use:*
  - Intubation dose: 0.45–0.6 mg/kg IV bolus.
  - Rapid sequence intubation: 0.6–1.2 mg/kg IV bolus.
- *Reversal Agent*
  - Sugammadex, a cyclodextrin, is the first selective relaxant-binding agent; it exerts its reversal effect by forming tight complexes in a 1:1 ratio with steroidal non-depolarizing agents (e.g., rocuronium).

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Liver Disease

12

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Liver Function

- The liver plays a major role in maintaining homeostatic, immunological, and synthetic processes. Hence, liver dysfunction and liver failure have significant consequences on overall health.

Hepatitides

- There are five forms of viral hepatitis (A, B, C, D, and E).
- The leading causes of chronic viral hepatitis are chronic hepatitis B and C.
- The pathophysiology of all viral hepatitis is an inflammatory-mediated process with active hepatocellular damage and necrosis with a lobular inflammatory response.
- Viral hepatitis is considered acute when inflammation lasts 6 months or less.
- With chronic viral hepatitis, the longstanding repetition of the inflammation and healing process leads to liver fibrosis.

Hepatitis A

- RNA virus with an average incubation period of 35–70 days.
- Typically, it is a self-limiting process leading to acute hepatitis.
- Clinical illness manifests as malaise, myalgias, arthralgia, anorexia, nausea, vomiting, diarrhea, low-grade fevers, and/or jaundice.
- Clinical features usually subside over 2–3 weeks.
- There is no chronic form of hepatitis A.
- Mode of transmission: fecal-oral, sewage-contaminated shellfish.
- Prevention: pooled gamma globulin, hepatitis A vaccine.
- Complications: Hepatitis A can lead to fulminant liver failure. It has <0.2% mortality rate [1].

Treatment: Hepatitis A can be prevented with a vaccine. There is no specific treatment for hepatitis A (treatment is just supportive care). Physical exertion, alcohol, and hepatotoxic agents should be avoided.

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Hepatitis B

- DNA virus with an average incubation period of 60–110 days that has an insidious onset leading to acute hepatitis.
- Chronic hepatitis develops in 1–5% of immunocompetent adults, a substantial proportion of immunocompromised adults and as high as 90% of infected children [2].
- After an infection with hepatitis B there are four phases: (1) immune tolerant, (2) immune clearance, (3) inactive HBsAg carrier state, and (4) reactivated chronic hepatitis B.
- Mode of transmission: vertical transmission (mother to fetus), percutaneous, and sexual.
- Risk factors: IV drug use, unprotected sex with multiple sexual partners, incarceration, needle sticks, tattoos, body piercings, hemodialysis, and blood transfusions.
- Prevention: hepatitis B immunoglobulin, hepatitis B vaccine.
- The clinical features of acute hepatitis B are similar to that of hepatitis A.
- Complications: The clinical course associated with acute hepatitis B ranges from asymptomatic to fulminant disease and death in a few days. If aminotransferase levels remain elevated for more than 6 months, the patient is considered to have chronic hepatitis B.
- 20% of chronic hepatitis B cases progress to cirrhosis [2].
- Hepatocellular carcinoma develops in cirrhotic patients at a rate of 2–4% per year [2].

Treatment: Hepatitis B vaccines are available. Treatment of acute hepatitis B is the same as hepatitis A, supportive care centered. Chronic hepatitis B patients with active viral replication may be treated with nucleoside or nucleotide analogs, with the goal of lowering HBV DNA levels and possible seroconversion.

Hepatitis C

- RNA virus with an average incubation period is 35–70 days.
- The clinical illness of acute hepatitis C is often mild and usually asymptomatic. Majority of cases progress to chronic liver disease.
- Mode of transmission: percutaneous.
- Risk factors: IV drug use, incarceration, needle stick, tattoos, body piercing, hemodialysis, and blood transfusion.
- Prevention after exposure: interferon plus ribavirin.
- Complications**:** Chronic hepatitis C is clinically indistinguishable from other causes of chronic hepatitis. Cirrhosis occurs in 20% of affected patients after 20 years [3]. Patients are also at risk for developing hepatocellular carcinoma. Hepatitis C has been shown to be associated with mixed cryoglobulinemia and membranoproliferative glomerulonephritis. HCV infection increases a patient's risk of developing non-Hodgkin lymphoma.

Treatment: Unlike hepatitis A and B there is no vaccine for hepatitis C. Both antiviral medication and interferon plays a role in the treatment of hepatitis C. Use of peginterferon in patients with acute hepatitis C decreases the risk of developing chronic hepatitis C. Ribavirin can be added to the course of peginterferon if HCV RNA does not clear with peginterferon alone. Direct-acting and host-targeting antiviral agents are changing the treatment of chronic hepatitis C. Many patients are now able to clear their chronic hepatitis C infections after a multidrug regimen (e.g., Harvoni ® and Mavyret®). Patient response rates to these treatment regimens depend on the specific genotype of HCV they are infected with. Patients may also be treated with liver transplantation.

Alcoholic Liver Disease (ALD)

- Excessive alcohol intake leading to a fatty liver, followed by hepatitis and cirrhosis.
- The consumption of 50 g daily (5 standard drinks) of alcohol for 10 years or more puts individuals at risk for ALD.

- The clinical course of ALD includes a spectrum of disease: alcoholic steatosis, alcoholic steatohepatitis, cirrhosis, and hepatocellular carcinoma.
- The pathophysiology of alcoholic liver disease starts with acute or chronic inflammation leading to parenchymal necrosis of the liver. The liver inflammation and hepatocyte degeneration at the level of alcoholic steatosis is reversible with cessation of alcohol use. The dreaded complication of alcoholic liver disease is progression to cirrhosis.

Treatment: Alcohol cessation and nutritional support are the first step in the treatment of ALD. Folic acid, thiamine, and zinc are particularly important to administer when deficiencies are noted. Daily administration of methylprednisolone or equivalent corticosteroid for 1 month may reduce short-term mortality in patients with alcoholic hepatitis and encephalopathy.

Nonalcoholic Fatty Liver Disease (NAFLD)

- The aggregation of hepatic fat that is not due to alcohol use is known as nonalcoholic fatty liver disease.
- NAFLD is one of the most common liver diseases in the United States and worldwide.
- The predisposing factors for NAFLD are metabolic syndrome, diabetes mellitus type 2, total parenteral nutrition, history of gastric bypass surgery, and some medications.
- The pathophysiology of this disease is not fully understood, and the treatment involves managing the primary medical problem.

Autoimmune Hepatitis

Autoimmune hepatitis (AIH) is among the less common cause of chronic liver disease. The pathophysiology of AIH is characterized by destruction of hepatocytes by T-cell and autoantibody-mediated molecules. Primary biliary cirrhosis and primary sclerosing cholangitis are among these autoimmune diseases.

Primary Biliary Cirrhosis

- Primary biliary cirrhosis (PBC) manifests due to chronic autoimmune destruction of *small* intrahepatic bile ducts and cholestasis.
- Patients with PBC are at risk for hepatocellular carcinoma.
- PBC onset is usually insidious.
- Most commonly occurs in women aged 40–60 years.
- PBC has both genetic and environmental risk factors, and often it is associated with other autoimmune diseases such as Sjogren syndrome, autoimmune thyroid disease, Raynaud syndrome, scleroderma, hypothyroidism, and celiac disease.
- The clinical manifestations of PBC include xanthomas, pruritus, hyperpigmented skin, jaundice, and fatigue.
- Laboratory findings include elevated alkaline phosphatase/aminotransferases. Patients are positive for antimitochondrial antibodies serologically.

Treatment: Ursodeoxycholic acid is the only FDA-approved treatment for PBC. It has been shown to slow progression, stabilize histology, improve long-term survival, and reduce the chance of developing esophageal varices. Per clinical trial studies, cortisone has no positive impact in PBC treatment. Liver transplantation increases the chance of survival, but disease can recur in the graft.

Cirrhosis

Even though there are many etiologies of liver disease, the underlying pathophysiology is hepatic inflammation and fibrosis leading to liver cirrhosis and ultimately liver failure. This process

leaves patients with an inability to synthesize coagulation factors and metabolize toxic chemicals.

- Cirrhosis is the progression of chronic liver disease to fibrosis and scarring of hepatocyte architecture. The liver might have areas of regenerative nodules surrounded by dense fibrotic tissue. Fibrosis deposition increases resistance of intrahepatic blood flow, which is the cause of portal hypertension.
- The clinical sequelae of portal hypertension include gastroesophageal varices with hemorrhage, ascites, and hypersplenism.
- A history of esophageal varices can complicate intubation. An esophagogastroduodenoscopy may be useful prior to surgery.
- The third spacing of fluid into the peritoneal cavity is due to hypoalbuminemia, salt and water retention, and excessive lymphatic fluid formation.
- Infection of ascites fluid, known as spontaneous bacterial peritonitis, is a dreaded complication in surgical inpatients with cirrhosis as it has a high mortality rate. Timely administration of antibiotic therapy is critical for survival.
- Other important sequelae of advanced cirrhosis are hepatorenal syndrome (due to dehydration and poor renal blood flow), hepatic encephalopathy, hepatopulmonary syndrome (due to intrapulmonary shunting leading to a V/Q mismatch), portopulmonary hypertension, malnutrition, coagulopathy, and hematologic abnormalities.
- Abnormalities in the coagulation system is an expected complication in the cirrhotic patient. There is not only a decreased biosynthesis of clotting factors, but also diminished clearance of anticoagulants (protein C and antithrombin).
- Patients with portal hypertension may have thrombocytopenia due to sequestration of platelets as a result of hypersplenism.
- Patients are more prone to infections due to destruction of the hepatic reticuloendothelial system (also known as the mononuclear phagocytic system).

Management of Cirrhosis

- Abstinence from alcohol.
- Patients are placed on a diet rich in calories and protein.
- HAV, HBV, pneumococcal, and yearly influenza vaccines are indicated due to deficiencies in immunosurveillance.
- Varices are managed endoscopically with banding, sclerotherapy, and balloon tamponade.
- Ascites and edema are treated with diuretics (spironolactone in combination with furosemide) and when the ascites is severe enough, refractory to diuretics, or causing respiratory compromise, large volume paracentesis is indicated.
- A transjugular intrahepatic portosystemic shunt (TIPS) is an effective treatment for both severe refractory ascites and variceal bleeding refractory to standard therapy.
- Spontaneous bacterial peritonitis is treated with antibiotics and supplemental IV albumin.
- Hepatorenal syndrome is treated with diuretic withdrawal, infusion of albumin, and a regimen of vasoconstrictors; in some situations modified dialysis methods are indicated.
- Hepatic encephalopathy is treated with lactulose to decrease systemic ammonia levels.
- Liver transplantation is indicated in selected cases of irreversible, progressive chronic liver disease.

Patient Management

- Perioperative mortality of patients with chronic liver disease depends on the progression of the disease. For the most part, a patient with liver disease is safe for surgery until their disease has advanced to cirrhosis. Even then, a patient with compensated cirrhosis can be a good surgical candidate. It is important to discuss the patient's current medical status with their hepatologist.
- Two scoring systems used to assess the severity and survival probability of chronic liver

disease are the Child-Pugh score and Model for End-Stage Liver Disease (MELD) equation.
  - MELD predicts the 90-day mortality without liver transplantation. It is based on sodium, INR, bilirubin, and creatinine. Its values range from 6 to 40, with a score of 40 predicting a 90% mortality in 3 months.
  - Child-Turcotte-Pugh (CTP) score predicts survival rate up to 2 years. It is based on bilirubin, albumin, PT (or INR), ascites, and hepatic encephalopathy. It places patients into three categories: A, B, and C based on points with increasing severity. The projected 2-year survival for category A is 85%; for category B, 57%; and for category C, 35%. Patients with CTP class A and B can proceed with surgery after medical optimization of encephalopathy and coagulopathy. CTP class C and acute hepatic dysfunction have a perioperative mortality rate of more than 80%, and all elective surgery should be deferred. Per American Society of Anesthesiology, patients who have alcohol dependence/abuse disorder or active hepatitis have an ASA III classification.
- The history and physical exam should be used to identify potential liver disease risk factors such as intravenous (IV) drug use, excessive alcohol use, and hepatotoxic medications.
- Physical exam signs of liver disease include spider nevi, jaundice, splenomegaly, ascites, hepatomegaly, and caput medusa (engorged paraumbilical veins).
- Evaluate for cirrhotic cardiomyopathy. ECG will show increase in Q-T interval. Preoperative stress test and echocardiogram should be considered in coordination with cardiology consultation.
- Assess baseline oxygen saturation, as patients may have respiratory compromise secondary to hepatopulmonary syndrome (triad of liver disease, increased alveolar arterial gradient, and intrapulmonary shunting) or portopulmonary hypertension. The severity of either does not correlate with the severity of liver disease. Higher risk for perioperative mortality. Evaluate for platypnea-orthodeoxia syndrome [4].
- CBC to assess for anemia, leukopenia, and thrombocytopenia. Thrombocytopenia is primarily treated with platelet transfusions. A bone marrow stimulant drug named Eltrombopag can reduce the need for platelet transfusion.
- Liver function tests – liver synthetic function is measured via levels of albumin, PT/INR, and biliary system dysfunction via levels of hepatic transaminases and bilirubin.
- Coagulopathy commonly due to declining synthetic function of the liver. Malabsorption and malnutrition may exacerbate the situation. Patients may require perioperative fresh frozen plasma (every 3–5 hours due to short half-life) or recombinant activated factor VIIa (expensive alternative). Consider vitamin K 10 mg IM prior to surgery [4].
- Intranasal desmopressin has been shown to be as effective as blood transfusion in achieving hemostasis in cirrhotic patients with moderate coagulopathy undergoing dental extraction [5].
- Thrombocytopenia is a common concern due to sequestration from portal hypertension and splenomegaly. Consider transfusion if platelet levels drop below 50,000.
- CBC, coagulation studies, and a complete metabolic panel are required to calculate MELD and CTP scores.
- Nutritional status in patients with cirrhosis is normally poor. Serum albumin, prealbumin, and triglycerides should be considered part of work up if there are signs of cachexia. Nutrition consult may be considered.
- Sensitivity to fasting with hypoglycemia is possible due to impaired gluconeogenesis.
- The intraoperative management of patients with chronic liver disease aims toward preserving hepatic function. Patients with viral hepatitis might need a lower dose of medication due to increased central nervous system sensitivity. On the other hand, alcoholic patients exhibit cross-tolerance to volatile and

intravenous anesthetics. Patients with cirrhosis have an unpredictable response to different anesthetic agents.

- Rapid-sequence induction should be used in patients with large ascites. The presence of ascites increases gastric volumes and leads to delayed gastric emptying. These two issues, along with upper gastrointestinal variceal bleeding, are considered a significant aspiration risk.
- The termination of induction agent action is based on redistribution and not metabolism or excretion. Therefore, standard induction dosages and agents such as propofol, etomidate, ketamine, and midazolam can be used as long as cardiac output and blood pressure are maintained.
- The neuromuscular blockers that are cleared independent of hepatic metabolism are cisatracurium, atracurium, and succinylcholine. These agents are a safe option as they preserve hepatic blood flow and oxygen delivery. Other non-depolarizing agents require larger doses due to increased volume of distribution [4, 6].
- Titrate parenteral drugs to effect. Altered pharmacokinetics resulting from reduced albumin and plasma proteins lead to an increase in the amount of unbound drug in circulation.
- Amide local anesthetics are degraded for the most part in the liver. Consider reduction in the amount of amide anesthetic. Bupivacaine, the most likely culprit in amide anesthetic-induced liver damage, causes damage via an idiosyncratic hypersensitivity reaction.
- Benzodiazepines that undergo glucuronidation (e.g., oxazepam, temazepam, and lorazepam) are unaffected by liver disease. Drugs such as diazepam and midazolam undergo hydroxylation [6]. Repeat dosing of benzodiazepines such as midazolam should be titrated to effect as their half-life, and elimination is prolonged in patients with advanced liver disease.
- Isoflurane and sevoflurane are the safe volatile agents for patients with significant liver disease as they have been found not to reduce hepatic blood flow. Nitrous oxide does not cause liver injury but might decrease the hepatic blood flow. Halothane should be avoided as it is believed to cause immune mediated liver damage [6].
- Opioid half-lives are prolonged and lower dosages should be used. Fentanyl and remifentanil clearance appears unaffected [4].
- Propofol retains a short half-life even in patients with decompensated cirrhosis [6].
- During the induction and maintenance of anesthesia, the blood pressure of a patient with advanced liver disease should be kept at baseline. A predominant feature of a cirrhotic patient's cardiovascular system is a generalized hyperdynamic state. This consists of increased heart rate, cardiac output, and plasma volume, with reduced systemic vascular resistance and blood pressure. Given the common hyperdynamic circulation in this patient population, vasopressors are often needed to avoid hypotension. Hypotension should be avoided in order to preserve hepatic arterial perfusion [4].
- Fluid replacement in patients with chronic liver disease is challenging. These patients are on a sodium-restricted diet preoperatively. Overall fluid administration should be limited to 5 ml/kg/h versus the usual 10 ml/kg/h in order to prevent volume overload. Consider pulmonary artery catheterization to monitor volume status and cardiac performance [4].
- Perioperative red blood cell transfusion may lead to hypocalcemia and nitrogen overload. Colloid intravenous fluids (albumin) are preferable in order to avoid sodium overload and fluid shifts.

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Cardiovascular Disease

13

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Hypertension

- Persistently elevated arterial blood pressure of 130/80 or mm or higher Hg in adults.
- Diagnosed by 2 elevated readings of at least 130/80 mmHg on 2 or more visits.
- American Heart Association (AHA) and American College of Cardiology (ACC) update to the JNC 7 Classification [1]:
  - Normotension <120/80 mmHg
  - Elevated 120–129/<80 mmHg
  - Stage I 130–139/80–89 mmHg
  - Stage II >140/90 mmHg

Essential Hypertension (HTN)

- Hypertension with no identifiable cause.
- 90% of patients diagnosed with HTN have essential HTN [2].
- Associated with the following defects:
  - Decreased vascular response to vasodilators such as prostaglandins and nitric oxide.
  - Renal defect which leads to retention of sodium chloride.
  - Increase in sympathetic tone.
  - Neural reflex defects.
  - Increased angiotensin II and renin secretion.
- Risk factors associated with hypertension include the following:
  - Obesity and sedentary lifestyles
  - Diabetes
  - Alcohol
  - Aging
  - Smoking
  - OSA
  - Family history
  - Ethnicity (African American)
  - Sex (Males have higher rates of HTN)

Secondary Hypertension

- Hypertension with an identifiable cause. Examples include the following:
  - Pheochromocytoma
  - Renal artery stenosis
  - Polycythemia vera
  - Cushing syndrome
  - Coarctation of the aorta
  - Hyperaldosteronism
  - Pregnancy
  - Renal parenchymal disease

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- Sequelae of untreated/poorly controlled hypertension (result of end organ damage):
  - Left ventricular hypertrophy
  - Ischemic heart disease
  - CHF
  - Renal insufficiency
  - Retinopathy and vision loss
  - Cerebrovascular accident
  - Peripheral vascular disease

Treatment of Hypertension

- Secondary hypertension – treat underlying cause.
- Lifestyle modification – weight loss, smoking cessation, decreased in sodium intake, exercise, and reducing alcohol consumption.
- Pharmacologic therapy:
  - Calcium channel blockers – decrease the influx of calcium ions resulting in vasodilation and a reduction in blood pressure (e.g., amlodipine, felodipine, diltiazem, verapamil).
  - Ace inhibitors – block the conversion of angiotensin I to angiotensin II (AII). AII is responsible for vasoconstriction and liberating aldosterone. Inhibiting vasoconstriction and decreasing the effects of aldosterone results in a reduction in blood pressure (e.g., lisinopril, fosinopril, enalapril, captopril, ramipril).
  - Angiotensin II (AII) receptor blockers (ARBs) – block the effects of AII through antagonism of AII receptors leading to decreasing vasoconstriction and aldosterone secretion. (e.g., losartan, valsartan, olmesartan, telmisartan).
  - β-blockers – block β-adrenergic receptors resulting in a decrease in myocardial contractility, decrease in renin production, and relaxation of smooth muscles (e.g.,metoprolol, atenolol, esmolol, carvedilol, labetalol).
  - Thiazide diuretics – block the reabsorption of NaCl in the distal convoluted tubule of the nephron leading to a contracted intravascular volume (e.g., hydrochlorothiazide, chlorthalidone).
  - Vasodilators – work by decreasing vascular smooth muscle tone (e.g., hydralazine, sodium nitroprusside).
  - Alpha-2 adrenergic agonist – works on central adrenergic receptors leading to decreased norepinephrine release (e.g., clonidine).
  - Direct renin inhibitor – prevents renal release of renin with a subsequent decrease in AII production (e.g., aliskiren).

Patient Management

- Obtain recent labs/studies to assess end organ damage (BUN/Creatinine, EKG, CBC).
- Defer elective surgery if preoperative blood pressure is not controlled. Refer immediately to physician for hypertensive urgency (BP >180/120 with no signs/symptoms of end organ dysfunction). Patients are treated with oral antihypertensives with gradual reduction of BP over the course of a few days.
- Metabolism of amide anesthetics can be reduced in patients taking beta-blockers [3].
- Monitor blood pressure closely and be prepared to treat intraoperative hypertension and hypotension:
  - Ephedrine and phenylephrine are commonly used to treat hypotension. Be cautious of the reflex bradycardia with phenylephrine usage.
  - Patients taking antihypertensives should continue their medications. Know side-effect profiles of the antihypertensives that the patient is taking.
  - A basic metabolic panel should be taken on patients taking ARBs and ACE inhibitors to rule out hypokalemia.
  - Patients taking ARBs and ACE inhibitors are more prone to anesthesia-induced hypotension.
  - Monitor EKG intraoperatively to rule out myocardial ischemia.
  - Use local anesthesia with a vasoconstrictor judiciously based on recommendation of 0.4 mg of epinephrine according to the AHA recommendations.

- Anxiolysis to reduce stress-induced hypertension.
- Monitor for signs/symptoms of orthostatic hypotension due to antihypertensive medications.
- Avoid medications that increase sympathetic tone (e.g., ketamine).
- Contact EMS for evidence of a hypertensive crisis (BP 180/120 with signs/symptoms of myocardial ischemia, bradycardia, hypertensive encephalopathy, dyspnea, chest pain, confusion, nausea/vomiting, headache, seizures, and pulmonary edema). Hypertensive crisis- BP is gradually reduced to not lead hypotension and subsequent myocardial ischemia and cerebrovascular ischemia.

Atherosclerosis and Ischemic Heart Disease

Atherosclerosis Hardening of the arteries due to lipid accumulation within the arterial wall.

Risk Factors

- Genetics – familial hyperlipidemia due to a mutated low density lipoprotein (LDL) receptor.
- Dyslipidemia – Having a total cholesterol of 240 mg/dl increases the risk of a coronary event [2]. Elevated LDL levels correlate with an increased incidence of atherosclerosis and coronary artery disease (CAD). Elevated high density lipoprotein (HDL) levels correlated with being protective against atherosclerosis and CAD.
- Tobacco – enhances oxidation of LDL, causes endothelial dysfunction, and causes increased platelet adhesiveness.
- HTN – damages the endothelium which leads to increased permeability to lipoproteins.
- DM – nonenzymatic glycosylation of LDLs increases the antigenicity of LDLs.
- Metabolic syndrome – cluster of HTN, hyperlipidemia, insulin resistance, and abdominal obesity.
- Lack of physical activity.
- Estrogen status – Physiologic estrogen levels raise HDL and lower LDL. Menopausal women are at an increased risk for ischemic heart disease (IHD).

Pathophysiology of Atherosclerosis

- Damage to the endothelium occurs.
- Lipoproteins then traverse the intima and leukocytes are recruited via chemotaxis. Macrophages imbibe LDL to form foam cells.
- Smooth muscle cells of the media layer secrete an extracellular matrix that traps the lipoprotein and gives bulk to the lesion.
- This matrix gives rise to the fibrous cap. As the lesion increases in size, the fibrous cap thins. Rupturing of the fibrous cap, leading to exposure of the thrombotic lipid core, which is now exposed to the blood. This may lead to an acute coronary syndrome (ACS).

Complications of Atherosclerosis

- Embolization of an atherosclerotic plaque to distant sites and can cause infarction of the affected organ (e.g., CVA).
- Weakening of vessel walls that may lead to aneurysm formation.
- Peripheral artery disease.
- Renal artery stenosis.
- Myocardial infarction.

Ischemic Heart Disease (IHD)

- Disease process secondary to stenotic coronary arteries that leads to ischemic sequelae from a myocardial oxygen supply and demand imbalance.
- Myocardial oxygen is dependent on oxygen supply and coronary blood flow. Myocardial oxygen demand is determined by wall stress, heart rate and contractility.

Consequences of IHD

Stable Angina Transient chest discomfort due to a fixed atheromatous plaque secondary to a myocardial oxygen supply and demand imbalance.

- Symptoms include dyspnea on exertion, retrosternal chest pain that may radiate to the arm or jaw. Patients will often describe the symptoms as a pressure or an elephant sitting on their chest. Patients may place a clenched fist over the sternum (Levine's sign). Symptoms normally appear when a vessel is at least 70% stenotic. The symptoms normally cease after 5–10 minutes with rest.
- Diagnostic workup – EKG may show ST depression or T wave inversion. Stress testing is done (bike, treadmill, or pharmacologic) to assess cardiac reserve. Pharmacologic testing may be carried out with dipyridamole thallium in persons unable to exercise. Echocardiogram is used to assess wall function, ejection fraction, and valvular function. Coronary angiography is used to assess stenotic coronary arteries (gold standard).

Acute Coronary Syndrome Disease processes along a continuum secondary to a ruptured atherosclerotic plaque with subsequent formation of a thrombus within the coronary vessel.

- *Unstable angina* – occurs secondary to a coronary thrombus that is partially occlusive. Patients have chest pain that is not relieved by rest. Can see signs of ischemic changes on an EKG with negative cardiac enzymes.
- *Non-ST segment elevation MI* – due to partially occlusive thrombus that results in a subendocardial infarction. Patients present with chest pain, nausea, dyspnea, and diaphoresis. EKG shows ST depression or T wave inversion. Will see elevated serum biomarkers such as troponins and CK-MB. (CK-MB is used to assess for early reinfarction due to its shorter half-life in comparison to troponins.)
- *ST segment elevation MI* – due to an occlusive thrombus that results in a transmural infarct. Will see ST segment elevations and serum biomarkers. Symptoms similar to NSTEMI.

Complications of MI (STEMI or NSTEMI) – may lead to fatal arrhythmias, conductions blocks, cardiogenic shock, ventricular wall rupture, and heart failure.

Treatment of IHD

- Nitrates – cause venodilation, which decreases preload (determinant of wall stress) and dilates coronary arteries.
- Beta-blockers and calcium channel blockers – decrease oxygen demand by decreasing heart rate and contractility.
- Ranolazine – inhibits sodium channels in myocardial cells, which leads to less intracellular calcium and decreased contractility.
- Percutaneous coronary intervention – balloon-tipped catheter is placed in a peripheral artery and maneuvered into the stenotic coronary vessel. A bare metal or drug eluting stent is then deployed to increase the patency of the coronary vessel. Patients are placed on antiplatelet drugs to decrease coronary thrombosis, as the stents are thrombogenic (drug-eluting stents are more thrombotic than bare metal stents). Drug-eluting stents decrease the rate of epithelialization.
- Coronary artery bypass grafting (CABG) – grafting done to bypass obstructive coronary vessels. Preferred for multivessel disease.

Treatment of MI

- Morphine – used for analgesia and anxiolysis.
- Oxygen – increases oxygen supply to the myocardium.
- Nitrates – improve coronary flow.
- Aspirin – decreases platelet aggregation.

- Beta-blockers – decrease myocardial oxygen demand.
- Transfer to hospital (remember time is myocardium) for PCI with stent deployment or fibrinolytic therapy if the hospital does not have interventional cardiology capabilities.

Treatment Used to Prevent Recurrent Episodes

- Beta-blockers – decrease myocardial oxygen demand and contractility via antagonism of beta-adrenergic receptor. Beta-blockers also increase the amount of time spent in diastole, which is the phase when coronary perfusion occurs (e.g., metoprolol, carvedilol, labetalol).
- Calcium channel blockers – decrease myocardial oxygen demand and contractility via antagonism of calcium channels. Calcium channel blockers increase the amount of time spent in diastole, which is the phase when coronary perfusion occurs (e.g., amlodipine, nifedipine, verapamil, diltiazem).
- Nitrates – cause venodilation, which decreases preload (determinant of wall stress) and dilates coronary arteries (e.g., isosorbide mononitrate, isosorbide dinitrate).
- ADP receptor inhibitors – decrease ADP activation of platelet aggregation to prevent coronary thrombosis. Examples are clopidogrel, prasugrel, and ticagrelor (reversible ADP inhibitor).
- ACE inhibitors – decreases the angiotensin II vasoconstriction, which decreases the afterload. ACE inhibitors also decrease aberrant cardiac remodeling (e.g., lisinopril, enalapril, quinapril, captopril).
- Statins – HMG Coa reductase inhibitors that decrease circulating LDL levels decreasing the rate atheroma formation (e.g., simvastatin, atorvastatin).

Patient Management

- Assess functional capacity. Metabolic equivalents (METS) are physiologic measures of the amount of energy expended during activities compared to energy expenditure at rest. 1 MET is based on the basal oxygen consumption of a 40-year-old, 70-kg male. This helps with risk stratification of functional capacity.
  - >10 METS excellent functional capacity (jumping rope or strenuous sports).
  - 7–10 METS good functional capacity (jogging or calisthenics).
  - 4–6 METS moderate functional capacity (power walking, sexual activity, leisure biking).
  - <4 METS poor functional capacity (watching television, writing). Patients with less than 4 metabolic equivalents should undergo noninvasive cardiac testing. Consider treating patients with less than 4 metabolic equivalents in the hospital setting.
- Non-cardiac surgery can be carried out 6 weeks after an episode of ACS.
- Anxiolysis to prevent increasing myocardial oxygen consumption.
- Supplemental oxygen.
- Ensure profound analgesia to prevent sympathetic stimulation.
- Provide adequate fluid infusion to prevent hypotension. Must use judiciously in patients with concomitant CHF.
- Avoid sympathomimetic agents as they increase heart rate and blood pressure (e.g., ketamine).
- Be judicious with usage of drugs that can depress myocardial contractility and decrease blood pressure (e.g., propofol).
- Consult the patient's physician to help with risk stratification.
- Use adjunctive hemostatic agents in patients on antiplatelet therapy.
- Monitor EKG for occult signs of cardiac ischemia.

- Maintain heart rate and BP within 20% of preoperative values.
- Consider treating high-risk patients in a hospital setting.

Congestive Heart Failure (CHF)

- Condition characterized by the inability of the heart to pump enough blood to meet the metabolic demands of the body.
- Compensated heart failure is due to compensatory mechanisms such as an increase in sympathetic tone that decreases pulmonary congestion and fluid retention.
- Decompensated heart failure is due to acute or gradual onset of signs and symptoms of pulmonary and systemic congestion.
- Systolic failure – due to impaired contractility of the heart or high afterload (chronic volume overload from mitral and aortic regurgitation, dilated cardiomyopathies, HTN, aortic stenosis). Ejection fraction <40%.
- Diastolic failure – due to impaired diastolic relaxation or ventricular failing of the heart (caused by left ventricular hypertrophy, restrictive cardiomyopathy, myocardial fibrosis, myocardial infarction). Can have preservation of the ejection fraction (>50%).

Risk Factors
- Ischemic heart disease.
- Hypertension.
- Myocardial infarction.
- Valvular diseases.
- Congenital heart disease.
- Cardiomyopathies.

Left-Sided Heart Failure Signs and Symptoms
- Dyspnea.
- Paroxysmal nocturnal dyspnea.
- Orthopnea.
- Clinically can appreciate a third heart sound (S3) and pulmonary rales. S4 sound with diastolic failure.

Right-Sided Heart Failure Signs and Symptoms
- Most common cause is left-sided heart failure.
- Abdominal discomfort.
- Anorexia.
- Hepatomegaly.
- Peripheral edema.
- Jugular venous distension.

New York Heart Association Classification of CHF

- CLASS I – Heart disease with no symptoms or limitation of physical activity.
- CLASS II – No symptoms at rest and slight limitation with ordinary activity.
- CLASS III – Marked limitation of activity with minimal exertion.
- CLASS IV – Symptoms at rest. Severe limitation of activity.

CHF Workup

- EKG – look for signs of ischemia, infarctions, and dysrhythmias.
- Echocardiography – assess ejection fraction and wall dyskinesia.
- Chest radiograph – assess for cardiomegaly and pulmonary edema.
- Clinical exam – auscultation for signs of volume overload (rales and S3) and S4 heart sound. Assess for pedal edema, JVD, and hepatomegaly.
- Brain natriuretic peptide (BNP) – used to diagnose and monitor CHF progression. BNP >35 pg/ml for diagnosis of non-acute onset CHF and >100 pg/ml for acute onset CHF [2].
- Basic metabolic panel – to assess electrolyte status prior to starting diuretic therapy and to monitor electrolytes for patients taking diuretics.
- Liver function tests to evaluate hepatic function and congestion.
- Fasting lipid to assess IHD and metabolic syndrome.

- Fasting glucose levels to assess for DM, as this is a risk factor and common comorbidity for CHF.
- CBC and TSH to exclude anemia and thyroid disease, respectively, as etiologies.

Treatment of CHF

- Diuretics – used to treat systemic and pulmonary congestion (e.g., Lasix. Consider spironolactone to prevent potassium wasting).
- Beta-blockers – decrease myocardial oxygen consumption.
- Digoxin – increases cardiac contractility. Be familiar with the signs and symptoms of digitalis toxicity which include xanthopsia (vision deficiency leading to predominance of yellow vision) nausea, vomiting, confusion, paresthesias, ventricular tachycardia, premature ventricular contractions, heart block, bigeminy, trigeminy. Narrow therapeutic range. 0.5–0.9 ng/ml [4].
- Ace inhibitors – decrease afterload and prevent aberrant cardiac remodeling.
- Nitrates – venodilators that decrease preload.
- Ventricular assist devices – used to assist the diseased ventricles to maintain cardiac output.

Patient Management

- Patient positioning is paramount as a recumbent position could lead to dyspneic episodes.
- Supplemental oxygen.
- Assess chemistry studies to look for electrolyte imbalances (e.g., hypokalemia).
- Avoid NSAID use as renal perfusion is based on prostaglandin vasodilation in patients with CHF. NSAID use may put patients at risk for renal failure.
- Avoid drugs that depress myocardial activity or increase myocardial oxygen consumption.
- Use intravenous fluids judicially to prevent volume overload.
- Consider treatment in a hospital setting for those with decompensated heart failure.

Valvular Heart Disease

General Terms

- Stenosis – Reduced blood flow through a valve secondary to pathologic stiffening or decreased opening of valve.
- Regurgitation – When blood flows back through a valve, in the reverse direction than normal, while the valve is closed.

Mitral Valve Disorders

Mitral Stenosis

- Decrease in the size of the mitral valve orifice resulting in decreased blood flow across the valve during diastole and increased atrial pressures and volume.
- The most common cause of mitral stenosis is rheumatic heart disease.
- Rheumatic heart disease leads to diffuse thickening of the mitral leaflets, calcification of leaflets and annulus, and possibly commissural fusion.
- It can also occur secondary to congenital disease with chordal fusion or papillary muscle malposition.
- With the congenital presentation, the papillary muscles may be abnormally close together and may merge into a single papillary muscle.
- Other causes include prior mitral valve repair procedures, carcinoid syndrome, left atrial myxoma, fenfluramine use, systemic lupus erythematosus (SLE), and rheumatoid arthritis (RA).
- Clinical signs and symptoms:
  - Dyspnea on exertion.
  - Chest pain.
  - Hemoptysis.
  - Low-pitched early diastolic murmur best heard over the apex.
  - Hoarseness due to left atrial enlargement and impinging on the recurrent laryngeal nerve (Ortner syndrome).
  - Atrial fibrillation.
  - Atrial thrombus formation and embolization.

Workup

- EKG to assess for left atrial enlargement, atrial fibrillation and left ventricular hypertrophy.
- Chest radiograph to assess for pulmonary edema, cardiomegaly, and possible calcifications of valves.
- Echocardiogram to assess valvular function and wall function.

Treatment

- Symptoms of mild to moderate disease are treated with diuretics to decrease left atrial pressure.
- Control of heart rate is imperative because tachycardia impairs left ventricular filling and increases left atrial pressure. Heart rate control can be achieved with beta-blockers, calcium channel blockers, or digoxin.
- Other treatment options include percutaneous balloon valvuloplasty with Wilkins score <8 and valve replacement for symptomatic patients with a mitral valve area of 1.5 cm^2 or less.
  - Wilkins score ranges from 0 to 16 based on mobility, subvalvular thickening, thickening and calcification of the mitral valve on echocardiography [5].
- When there is concomitant atrial fibrillation, anticoagulation is required due to significant increase in risk of embolic stroke.

Mitral Regurgitation

- An increase in the size of the mitral valve orifice, resulting in back flow of blood across the mitral valve during systole. This causes an increase in left atrial pressure and a decrease in left ventricular stroke volume and cardiac output.
- The fraction of left ventricular stroke volume which regurgitates is dependent on the size of the mitral orifice, the heart rate, and pressure gradient across the valve.
- Conditions such as ischemic heart disease, rheumatic heart disease, endocarditis, mitral valve prolapse, hypertrophic cardiomyopathy, congenital heart disease, among others, can result in papillary muscle dysfunction, mitral annular dilation, or rupture of chordae tendineae leading to dilation of the mitral valve orifice.
- Mitral regurgitation causes a decrease in forward left ventricular stroke volume and cardiac output. Left atrial volume overload results from regurgitated blood entering the left atrium during systole. This leads to pulmonary congestion.
- When mitral regurgitation develops gradually, patients are often asymptomatic for years. Volume overload changes the left ventricle into a larger and more compliant chamber capable of delivering larger stroke volumes, which leads to ventricular hypertrophy and increased compliance of the left atrium.
- With time, volume overload results in decreased myocardial contractility and a drop in the ejection fraction occurs.
- Atrial fibrillation may result secondary to the left atrial and ventricular enlargement. With acute mitral regurgitation, there is no time for left atrial or ventricular compensation, so pulmonary edema and/or cardiogenic shock develops.
- Clinical signs and symptoms:
  - Dyspnea on exertion.
  - Fatigue.
  - Orthopnea.
  - Cough.
  - Heart palpitations.
  - S_3 heart sound if CHF ensues.
  - Holosystolic blowing murmur at the cardiac apex radiating to the axilla.
  - Atrial fibrillation.

Workup

- EKG to assess for enlarged P waves denoting left atrial enlargement, atrial fibrillation, and left ventricular hypertrophy.
- Chest radiograph to assess for pulmonary edema, cardiomegaly, and possible calcifications of valves.

- Echocardiogram to assess valvular function and wall function.

Treatment

- Symptomatic patients with chronic mitral regurgitation have been shown to have a decrease in regurgitation, an improvement in symptoms, and improved exercise tolerance with use of ACE inhibitors, beta-blockers, and biventricular pacing.
- Vasodilators (ACE inhibitors and ARBs) are useful in the medical management of acute mitral regurgitation due to decreasing afterload (which reduces LVH).
- Mitral valve surgery is indicated in symptomatic patients (even if the ejection fraction is preserved), patients with new onset atrial fibrillation and severe regurgitation. Mitral valve repair is preferred over valve replacement.
- Valvular tissue prosthetic valve patients require anticoagulation for 3 months after surgery.
- Mechanical valve therapy requires lifelong anticoagulation. Patients anticoagulated with warfarin are maintained at a target INR of 2.5–3.5.

Mitral Valve Prolapse (MVP)

- Prolapse of one or both mitral leaflets into the left atrium during systole, this may occur with or without regurgitation.
- Results from floppy or myxomatous mitral valve.
- Most common form of valvular heart disease.
- Risk factors include Marfan's syndrome, Ehlers-Danlos syndrome, rheumatic heart disease, myocarditis, osteogenesis imperfecta, thyrotoxicosis, and systemic lupus erythematosus.
- With the anatomic variant, there is redundant and thickened leaflets. This typically occurs with connective tissue disorders or in elderly men.
- With the functional variant, there is mild bowing and normal appearing leaflets. These patients have a decreased risk of complication compared to the anatomic variant.
- MVP can result in complications such as cerebral embolic events, infective endocarditis, dysrhythmias (supraventricular and ventricular), and sudden death from rupture of a chordae tendineae.
- A hyperadrenergic syndrome associated with MVP has been described. This may be responsible for some of the non-cardiac symptoms, such as anxiety, palpitations, dyspnea, fatigue, atypical chest pain, and orthostatic hypotension. Clinical signs and symptoms:
  - Dyspnea on exertion.
  - Fatigue.
  - Anxiety.
  - Orthostatic hypotension.
  - Midsystolic click appreciated upon auscultation.
  - Cardiac dysrhythmias.

Workup

- Cardiac MRI.
- Echocardiogram to assess valvular function and wall function.

Treatment

- Most patients with MVP with or without regurgitation can be treated conservatively.
- Beta-blockers and SSRIs have been used to treat the associated hyperadrenergic syndrome. Beta-blockers are also effective for the management of associated dysrhythmias.
- Mitral valve repair is favored over replacement for patients with concomitant severe regurgitant and left ventricular disease.

Aortic Valve Disorders

Aortic Stenosis

- Decrease in the aortic valve area resulting in obstruction of blood flow into the aorta and increased left ventricular pressures.
- Congenital unicuspid or bicuspid aortic valve, hypertension, hypercholesterolemia, smoking, rheumatic heart disease, endocarditis, and certain genetic markers (Notch 1) are risk factors.

- In order to maintain stroke volume, there is an increase in left ventricular pressure. The stenotic valve produces an increase in cardiac afterload. This results in an increase in myocardial work and concentric left ventricular hypertrophy, leading to an increase in myocardial oxygen demand.
- Patients can often experience angina pectoris as a result, despite the absence of coronary artery disease.
- Patients become symptomatic when the valve area is 1 cm^2 or less.
- Eventually compensatory mechanisms are overcome and a decrease in the ejection fraction arises. Systolic and/or diastolic left ventricular dysfunction can occur and lead to CHF with associated exertional dyspnea.
- Syncope is a late and common sequela.
- A degree of aortic regurgitation is almost always associated with aortic stenosis.
- Clinical signs and symptoms:
  - Dyspnea on exertion.
  - Fatigue.
  - Syncope.
  - Angina.
  - Sudden death.
  - Signs of heart failure with severe LV disease.
  - Crescendo-decrescendo systolic murmur.

Workup

- EKG to assess for atrial fibrillation and left ventricular hypertrophy.
- Chest radiograph to assess for pulmonary edema and cardiomegaly.
- Echocardiogram to assess valvular function and wall function.

Treatment

- In asymptomatic patients with aortic stenosis, medical management consisting of blood pressure and cholesterol control can be used.
- Valve replacement can be delayed until symptoms arise.
- Although asymptomatic, severe aortic stenosis should be surgically managed. Aortic valve replacement is the mainstay of treatment.
- Valve replacement can be performed as a surgical (open) aortic valve replacement (SAVR) or a transcatheter aortic valve replacement (TAVR).
- TAVR is recommended for high-risk surgical patients.
- In younger patients with congenital valvular disorders, there is still a role for percutaneous balloon valvuloplasty.
- Bioprosthetic valve recipients require anticoagulation for the first 3–6 months.
- Mechanical valve recipients require lifelong anticoagulation. Patients receiving warfarin have a target INR of 2.5–3.5. Patients also require lifelong antiplatelet therapy with aspirin.

Aortic Regurgitation

- Disease of the aortic leaflets or of the aortic root, resulting in backflow of blood across the aortic valve into the left ventricle during diastole.
- Leaflet abnormalities can result from infective endocarditis, rheumatic fever, bicuspid aortic valve, and use of anorexigenic drugs.
- Abnormalities of the aortic root can result from idiopathic aortic root dilation, hypertension-induced aortic annular ectasia, aortic dissection, osteogenesis imperfecta, syphilitic aortitis, Marfan's, Ehlers-Danlos, RA, ankylosing spondylitis, and psoriatic arthritis.
- Regurgitation results in both pressure and volume overload of the left ventricle and a decrease in cardiac output resulting in left ventricular hypertrophy.
- Patients can experience angina pectoris, which may occur in the absence of coronary artery disease.
- Left ventricular failure can also occur, resulting in pulmonary edema and volume overload.
- Clinical signs and symptoms:
  - Dyspnea on exertion.
  - Fatigue.
  - Angina.

  - Signs of heart failure with severe LV disease.
  - Decrescendo high-pitched diastolic murmur along the left sternal border in the second right intercostal space.

Workup
- EKG to assess for left ventricular disease.
- Chest radiograph to assess for pulmonary edema and cardiomegaly.
- Echocardiogram to assess valvular function and wall function.

Treatment
- Afterload reduction with ace inhibitors and calcium channel blockers.
- Loop diuretics in patients with pulmonary congestion.
- Surgical replacement of the diseased aortic valve is recommended before the onset of permanent left ventricular dysfunction, even if patients are asymptomatic. Also recommended in patients with acute aortic regurgitation due to trauma, endocarditis, or dissection.

Tricuspid Valve Disorders

Tricuspid Regurgitation

- Tricuspid annular dilation resulting in backflow of blood across the tricuspid valve into the right atrium during systole.
- Right ventricular enlargement, pulmonary hypertension, infective endocarditis (intravenous drug users are at risk for tricuspid valve endocarditis), carcinoid syndrome, rheumatic heart disease, tricuspid prolapse, and Ebstein's anomaly are associated with tricuspid regurgitation.
- Often associated with mitral or aortic valve disease.
- Usually functional, caused by tricuspid annular dilatation secondary to right ventricular enlargement.
- Mild regurgitation can be a normal finding at any age.
- Clinical signs and symptoms:
  - Dyspnea on exertion.
  - Fatigue.
  - Peripheral edema.
  - Hepatomegaly.

Workup
- EKG to assess for wall stress, right axis deviation, right atrial enlargement, and atrial fibrillation.
- Chest radiograph to assess for pulmonary edema and cardiomegaly.
- Echocardiogram to assess valvular function and wall function.

Treatment
- Mild disease is usually treated with diuretics.
- Definitive treatment usually requires elimination of the underlying cause. Treatment also includes improving lung function, treating left heart failure symptoms, and reducing pulmonary HTN.
- Surgical management of isolated tricuspid disease is rarely performed.

Tricuspid Stenosis

- Decrease in the size of the tricuspid valve orifice resulting in decreased blood flow across the valve and increased right atrial pressure.
- Rare in the adult population. Rheumatic heart disease with coexisting tricuspid regurgitation and often mitral or aortic valve disease. Carcinoid syndrome and endomyocardial fibrosis are also causes.
- Clinical signs and symptoms:
  - Dyspnea on exertion.
  - Fatigue.
  - Peripheral edema.
  - Hepatomegaly.

Workup
- EKG to assess for wall stress, right axis deviation, right atrial enlargement, and atrial fibrillation.
- Chest radiograph to assess for pulmonary edema and cardiomegaly.

- Echocardiogram to assess valvular function and wall function.

Treatment
- Initial management is directed toward relieving fluid congestion with diuretics.
- When tricuspid valve replacement is indicated, bioprosthetic valves are always preferred over mechanical.

Pulmonic Valve Disorders

Pulmonic Regurgitation

- Annular dilation of the pulmonic valve resulting in backflow of blood across the valve into the right ventricle during diastole.
- High pressure causes: pulmonary hypertension.
- Low blood pressure causes: connective tissue disorders, congenital, post repair of Tetralogy of Fallot, carcinoid syndrome, infective endocarditis, rheumatic heart disease.
- Rarely symptomatic, right heart volume overload develops.
- Clinical signs and symptoms:
  - Dyspnea on exertion.
  - Fatigue.
  - Peripheral edema.
  - Hepatomegaly.

Workup
- EKG to assess for wall stress, right axis deviation, right atrial enlargement, and atrial fibrillation.
- Chest radiograph to assess for pulmonary edema and cardiomegaly.
- Echocardiogram to assess valvular function and wall function.

Treatment
- Rarely needs specific treatment, other than treatment of primary cause.
- Bioprosthetic valve replacement sometimes indicated.

Pulmonic Stenosis

- Decrease in the pulmonic valve area resulting in obstruction of blood flow into the pulmonary arteries and increased right ventricular pressures.
- Usually congenital, acquired causes include rheumatic heart disease, infective endocarditis, or previous surgeries.
- Clinical signs and symptoms:
  - Dyspnea on exertion.
  - Angina.
  - Peripheral edema.
  - Hepatomegaly.
  - Ejection click with a split S_2.

Workup
- EKG to assess for wall stress, right axis deviation, right atrial enlargement, and right ventricular hypertrophy
- Chest radiograph to assess for pulmonary edema and cardiomegaly.
- Echocardiogram to assess valvular function and wall function.

Treatment
- Surgical balloon valvotomy.

Patient Management

When performing office-based procedures or deciding if a patient is a surgical candidate in the OR, it is imperative to have a firm understanding of their underlying pathology and its severity.

- Consultation with the patient's cardiologist or PCP is often indicated.
- Patients with valvular disease, whether treated or not, are often on anticoagulants or have indications for endocarditis prophylaxis. These patients are often fragile with decreased cardiac reserve. Small changes in heart rate, secondary to pain or local anesthesia, can result in large changes in cardiac output.
- Measures to avoid anxiety and prolonged tachycardia should be considered.

Anesthetic Considerations

Mitral Stenosis

- Avoid excessive perioperative fluid administration.
- Avoid Trendelenburg's position. Manage tachycardia aggressively.
- Avoid ketamine because of its effects on the heart rate.
- Control blood pressure to decrease afterload.

Mitral Regurgitation

- Maintain a normal to slightly elevated heart rate.
- Bradycardia may result in left ventricular volume overload. Increases in systemic vascular resistance should also be avoided.

Mitral Valve Prolapse

- If there is associated regurgitation, the patient should be treated the same as a patient with regular MR.

Aortic Stenosis

- Prevention of hypotension and preservation of cardiac output. Normal sinus rhythm must be maintained. Agents like ketamine (which increase HR) and propofol (which decreases SVR) should be avoided.

Aortic Regurgitation

- Heart rate should be kept above 80 bpm.
- Bradycardia results in an increase in the duration of diastole, resulting in more time for regurgitation of blood.
- Abrupt increases in systemic vascular resistance can also precipitate left ventricular failure.

Tricuspid Regurgitation

- Nitrous oxide can be a weak pulmonary artery vasoconstrictor and can increase the degree of regurgitation.

Orthotopic Heart Transplantation

Clinical Highlights

- Most recipients are >60 years of age.
- Projected half-life for cohort of recipients is 11 years [6].
- Primary indication for transplantation is non-ischemic cardiomyopathy.
- Other indications:
  - Ischemic heart disease.
  - Adult congenital heart disease.
  - Retransplantation.
  - Valvular heart disease.
- Cardiac allograft vasculopathy (immune mediated process) accounts for 33% of all deaths in patients surviving after 5 years. Other causes include malignancy and infection (secondary to immunosuppression) [7].
- Patients are on lifelong immunosuppression (induction dose followed by maintenance therapy).
- Immunosuppressive drugs have their own specific side effects which have clinical implications.
- Maintenance therapy usually involves 2 drugs with or without a corticosteroid (prednisone).
- Tacrolimus and mycophenolate mofetil are the most common immunosuppressants used.
  - Tacrolimus – nephrotoxic and neurotoxic. Also shown to cause hypertension, hyperlipidemia, and hyperglycemia.
  - Mycophenolate mofetil – causes leukopenia and a variety of gastrointestinal symptoms.
- Rapamycin – known to cause myelosuppression and hyperlipidemia.
- Cyclosporine – known to cause gingival hyperplasia, gastric atony, hyperkalemia, and hypomagnesemia. Cyclosporine has also been found to increase the duration of action of vecuronium and pancuronium. Also known to enhance the effects of fentanyl and pentobarbital.

- Azathioprine – most significant side effects are myelosuppression and hepatotoxicity.
- Steroids can cause adrenal suppression, hypokalemia, hypocalcemia, poor wound healing, osteoporosis, fluid retention, steroid myopathy.

Physiological Changes Associated with a Transplanted Heart

- The most profound difference between the native heart and the transplanted heart is denervation.
- Efferent denervation ablates the resting parasympathetic tone responsible for maintaining baseline heart rate.
- Transplanted patients generally have an increased baseline heart rate of 90 to 100 beats per minute [8]. Loss of direct sympathetic innervation means that the cardiac response to physiologic stressors (exercise, hypovolemia, vasodilatation, pain, light anesthesia) is mediated by circulating catecholamines and, as a result, tends to occur much less quickly.
- Predominantly parasympathetic responses (visceral traction, abdominal insufflation, oculocardiac reflex, vasovagal bradycardia, hypertension-induced bradycardia, cardiac response to carotid massage, Valsalva maneuvers) are absent.

Denervation Pharmacologic Implications

- The denervated heart no longer responds normally to indirect-acting medications (medications that mediate effects via the autonomic nervous system).
- Administration of drugs such as neostigmine, physostigmine, pyridostigmine, edrophonium, glycopyrrolate, atropine, digoxin, and nifedipine no longer produce their anticipated heart rate effects.
- Indirect-acting drugs such as ephedrine have a decreased effect.
- Direct-acting drugs such as glucagon, norepinephrine, epinephrine, isoproterenol, dopamine, and beta-blockers are effective choices for managing hemodynamics in these patients.
- Reflex responses such as the bradycardia expected after administration of phenylephrine may not be present.

Other Physiologic Changes in the Transplanted Heart

- A mild decrease in ventricular function.
- Mild to moderate diastolic dysfunction which renders the patient dependent on preload for maintenance of cardiac output, and an increase in resting coronary blood flow as a result of loss of adrenergic tone.
- Early post-transplant patients can have significant ECG abnormalities. If a portion of the patient's native right atrium was retained during surgery, the ECG may demonstrate 2 P waves: 1 from the native atria.
- Ectopic ventricular beats are common in the first several weeks after transplant (usually diminishes).
- Supraventricular dysrhythmias (atrial premature beats, atrial fibrillation, and atrial flutter) are common after transplantation, but are also associated with episodes of acute rejection. New onset supraventricular dysrhythmias should heighten suspicion for rejection.
- Can also see atrioventricular bradyarrhythmias requiring pacemaker insertion.
- By 10 years after transplantation, 99% of recipients have hypertension, 14% have severe renal insufficiency (creatinine >2.5 in 8%, chronic dialysis in 5%, and renal transplant in 1%), 93% have hyperlipidemia, and 37% have diabetes [9].

Patient Management

- Physical examination
  - The preoperative physical examination should focus on looking for evidence of volume overload suggesting ventricular failure (increased jugular venous pressure, S3 gallop, peripheral edema, hepatomegaly). The preoperative evaluation should also include a chest radiograph to look for pneumonia or other signs of cardiopulmonary disease.

- Exercise tolerance and functional status are excellent preoperative screening tools.
- Echocardiography needed to assess left ventricular function.
- An ECG is needed to assess for dysrhythmias and rule out acute ischemia (useful in looking for evidence of old infarcts (Q waves) as the transplanted heart patient may not have angina symptoms)
- Laboratory evaluation should include a complete blood count to rule out anemia, myelosuppression, leukocytosis, and thrombocytopenia.
- Blood urea nitrogen and creatinine can help to evaluate renal function in the presence of probable hypertension.
- Preoperative antibiotics due to immunosuppression.
- Stress-dose steroids should be considered in patients receiving corticosteroids as part of their immunosuppressive regimen.
- Cyclosporine can enhance the effects of pentobarbital and fentanyl as well as prolong the action of vecuronium and pancuronium. Cyclosporine also causes gastric atony which is a risk for emesis and aspiration.
- Patients further removed from surgery have a higher risk of cardiac allograft vasculopathy, malignancy, diabetes, and renal insufficiency.
- There should be strong consideration to treating these patients in the operating room setting due to the precarious nature of these patients.

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14 Respiratory Diseases

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Lung Volumes and Capacities (Fig. 14.1)

- Total lung capacity (TLC) is the total volume of air that can be contained in the lung.
- Functional residual capacity (FRC) is the volume of air in the lungs after normal exhalation.
- Expiratory reserve volume (ERV) is the volume of air that can still be expired after normal exhalation.
- Inspirational reserve capacity (IRC) is the maximum volume of air that can be inspired at the end of normal inspiration.
- Forced vital capacity (FVC) is the maximum volume of air that can be exhaled.
- Residual volume (RV) is the volume of air remaining in the lungs at the end of forced exhalation.
- Inspiratory capacity (IC) is the maximum volume of air that can be inspired.
- Tidal volume (VT) is the volume of air during normal inspiration.

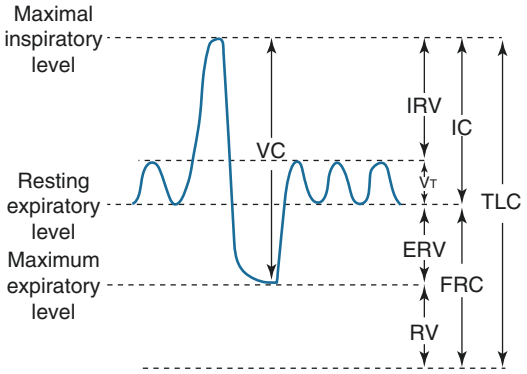

Fig. 14.1 Lung volumes. (Reprinted with permission from Mayer A and Maier L. Murray and Nadel's Textbook of Respiratory Medicine. Elsevier Health. 2016)

- Vital capacity (VC) is the total volume of air that can be expired after maximum inhalation.
- Forced expiratory volume (FEV1) is air that can be expired in 1 second.
- FEV1/FVC ratio represents the proportion of vital capacity that is expired in the first second of forced expiration. Normal value is 80%.
- FRC serves as a reservoir of oxygen. FRC increases with obstructive lung disease and decreases with lung restrictive disease. FRC is also low in the pediatric population.

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Categories of Lung Disease (Table 14.1)

Asthma

Asthma**:** Chronic obstructive disease with bronchiolar inflammation and hyperresponsiveness that displays reversible, variable, and recurrent airway obstruction.

- Diagnosis: Asthma is a clinical diagnosis, which can be confirmed with a positive response to bronchodilator during a pulmonary function test.

Asthma Signs and Symptoms

- Wheezing
- Dyspnea
- Chest tightness
- Cough both productive and nonproductive
- Tachypnea
- Atopy – greatest risk factor for the development of asthma

Table 14.1 Categories of pulmonary disease with examples

Obstructive	Restrictive parenchymal	Restrictive extraparenchymal
Asthma	Pulmonary fibrosis	Myasthenia gravis
COPD	Sarcoidosis	Diaphragm paralysis
Cystic fibrosis	Radiation-induced interstitial lung disease	Muscular dystrophy
		Obesity
		Kyphoscoliosis

Classification and Treatment of Asthma (Table 14.2)

Status Asthmaticus

- Bronschospasmic episode that does not respond to treatment and is considered life threatening.

Treatment

- Supplemental oxygen to maintain an SaO2 of >90%.
- β_2-agonists by metered dose inhaler every 15–20 minutes.
- Intravenous corticosteroids.
- IV magnesium sulfate.
- Epinephrine 0.3 mg 1:1000 administered subcutaneously.
- Tracheal intubation for a PaCO2 > 50 mm Hg.

Table 14.2 Classification of asthma and treatment regimen

Asthma classification	Symptom frequency	Night-time symptom	FEV_1	β adrenergics	Steroids	Leukotriene inhibitor	Cromolyn
Mild intermittent	<2 per week	<2 per month	>80%	Short acting <2 days per week	No	Alternative	No
Mild persistent	>2 per week	>2 per month	>80%	Short acting >2 days per week, but not daily and not more than 1 time on any day	Inhaled low-dose	Yes	Alternative
Moderate persistent	Daily	>1 night/ week	60–80%	Short acting daily, can add long acting	Inhaled med-dose	Yes	PRN
Severe persistent	Continuous	Frequent	<60%	Short >2 × /day and can add long acting	Inhaled and high-dose systemic	Yes	PRN

Patient Management

- Assess the severity of asthma at the preoperative visit. Investigate frequency, triggers, hospitalizations/ER visits, and need for intubation. Patient with a history of recent hospitalizations, a history of poorly controlled disease (e.g., moderate or severe persistent), or history of hospitalizations that require intubation should be treated in a hospital setting.
- For elective cases, optimization of disease should be achieved and postponement of elective surgery until resolution of symptoms for at least 6 weeks [1].
- Assess oxygen saturation levels on room air (<95% is concerning).
- Auscultation of chest to rule active disease processes such as wheezing. A silent chest does not preclude the possibility of bronchospasm.
- Prophylactic preanesthetic use of β_2-agonist inhaler to reduce chances of bronchospasm is recommended. Very light sedation or deep general anesthesia is desired to avoid stage II, which increases odds of laryngospasm/bronchospasm.
- Use of anesthetic agents that result in bronchodilation is preferred, such as propofol, ketamine, sevoflurane, and isoflurane. Desflurane is very pungent and should be avoided.
- Liberal use of intravenous hydration is recommended to make airway secretions less viscous.
- Medications that result in histamine release should be avoided such as meperidine and morphine. Also, opioids should be used judiciously to decrease the risk of chest wall rigidity.
- Use of adenosine and other nonspecific β-blockers (e.g., Labetalol) should be avoided since they will result in bronchoconstriction.
- Be cautious using NSAIDs and cyclooxygenase inhibitors as this can induce an asthma attack and rash. Some patients may have Samter's triad (nasal polyps, ASA sensitivity, and asthma) and are also sensitive to the aforementioned drugs.
- Be aware of signs and symptoms of bronchospasm and status asthmaticus. Contact EMS for suspected status asthmaticus.

Cystic Fibrosis (CF)

Cystic Fibrosis: Autosomal recessive disease resulting in altered chloride and water transport (CFTR gene) across epithelial cells; this prevents sodium reabsorption by epithelial sodium channels. Can impact the respiratory, gastrointestinal, and reproductive systems.

- Diagnosis: Sweat chloride concentration exceeding 60 mEq/L in addition to one or more of the following: chronic airway disease, exocrine pancreatic insufficiency, or cystic fibrosis in a first-degree relative. At birth, failure of passing meconium is highly suggestive of CF.
- Patients have a high incidence of respiratory infections that manifest as productive cough, pansinusitis, and dyspnea.
- Patients may also exhibit signs of malabsorption including diarrhea, constipation, and greasy smelly stools.
- Salty crystal accumulation on skin and salty tasting skin.
- Heat and exercise intolerance.
- Infertility due to azoospermia and atresia of the vas deferens may also be present.
- Signs of fat-soluble deficiency:
  - Vitamin E – peripheral neuropathy and hemolytic anemia.
  - Vitamin K – decreased efficacy of vitamin k–dependent factors and increased bleeding susceptibility.
  - Vitamin D – risk for reduced bone density.
  - Vitamin A – impaired vision in low light setting and dry skin.
- Decreased FEV_1 and FEV_1: FVC ratio.

Treatment

- Currently no cure for CF exists.
- The main goals are proactive treatment of airway infection; many are on at least one antibiotic prophylactically to suppress infection, usually azithromycin.
- Acute pulmonary functions are treated empirically. Sputum cultures should be taken prior to the initiation of therapy.
- Chest therapy and use of dornase alfa to loosen secretions are commonly utilized.
- Involvement of pancreas leading to diabetes is common. Thickened bile secretions can lead to biliary obstruction and eventually cirrhosis. Lack of digestive enzymes may lead to malabsorption and difficulty in absorbing fat-soluble vitamins A, D, E, and K. Patients are treated with pancreatic enzyme replacement.
- Failure of these major organs might require pancreatic, lung, or liver transplant.

Patient Management

- Management of these patients is best carried out with the aid of pulmonologist or cystic fibrosis specialist.
- Optimization of pulmonary status in terms of management of secretions and infections is the key.
- Active infections must be ruled out prior to surgery.
- Consider chest physiotherapy prior to surgery.
- Electrolytes, LFTs, and chest radiographs are useful prior to anesthesia along with continuation of medications.
- Risk of developing a pneumothorax is risk high (nitrous oxide should be avoided).
- Procedure should be carried out later in the day to allow for clearance of secretions that have accumulated overnight.
- Ketamine is relatively contraindicated due to increased secretions.
- Prophylactic β agonist with metered dose inhaler is recommended.
- Irritant vapors such as isoflurane such and desflurane are less useful than sevoflurane [2].
- Short-acting anesthetic agents such as propofol and sevoflurane may be the most appropriate choice.
- Consider arterial line for frequent blood gas monitoring [2].
- Avoid nasal intubation if possible due to higher incidence of polyposis [2].

Chronic Obstructive Pulmonary Disease (COPD)

- COPD is an irreversible disease that causes airway obstruction by either chronic bronchitis and/or emphysema (Table 14.3).
- Risk factors include smoking (most common), respiratory infection, occupational exposure to environmental substances, and alpha-1 antitrypsin deficiency.
- Signs and symptoms include wheezing, chronic cough, productive cough, hyperinflation of chest, weight loss, fatigue, and dyspnea on exertion.
- Advanced symptoms include pursed lips, cachexia, and pulmonary hypertension leading to neck vein distention and peripheral edema.

Table 14.3 Classification of COPD

Stage	Diagnostic value	Treatment
I (Mild)	FEV_1/FVC < 70% FEV_1 > 80%	Short-acting bronchodilator (albuterol)
II (Moderate)	FEV_1/FVC < 70% FEV_1 50–79%	Long-acting bronchodilator (e.g., Salmeterol, Formoterol, Bambuterol) and anticholinergics (e.g., Atrovent, Spiriva, Combivent)
III (Severe)	FEV_1/FVC < 70% FEV_1 30–49%	Inhaled steroid
IV (Very severe)	FEV_1/FVC < 70% FEV_1 < 30%	Oxygen, pulmonary rehabilitation

- Chronic bronchitis ("blue bloaters") results in chronic hypersecretion of mucus in the bronchi resulting in increased resistance to airflow and irreversible airway obstruction.
- Emphysema ("pink puffers") is enlargement of the airway due to destruction of the airway walls distal to bronchioles. This leads to loss of elasticity/recoil. Symptoms include dyspnea, cough, sputum production, and decreased exercise tolerance. They typically have a barrel chest due to hypertrophy of the accessory muscles used during respiration.
- COPD patients typically exhibit hypercarbia and hypoxemia. Advanced disease can lead to pulmonary hypertension and cor pulmonale.

Diagnosis

- Pulmonary function tests including spirometry are used to confirm suspected COPD.
- Bronchitis diagnosis is based on a history of a productive cough that has been present for at least 2 consecutive years.
- Blood labs may show hypercarbia, polycythemia, decreased serum alpha$_1$-antitrypsin levels, and leukopenia.
- Chest radiographs are used to look for evidence of lung nodules, bullae, hyperinflated lungs, masses, or fibrotic changes.
- Pulse oximetry at rest, during exertion, and during sleep is useful to evaluate for hypoxemia and the need for supplemental oxygen.

Patient Management

- COPD patients should be optimized prior to surgery (consult with a pulmonologist and treatment of active infections).
- Smoking cessation for 6 weeks is recommended as oxygen carrying capacity and mucociliary transport improve and has been shown to reduce postoperative pulmonary complications [3, 4].
- EKG to rule out right-sided heart disease and ischemic heart disease [5].
- Chest X-ray warranted if concern for respiratory infection or occult malignancy.
- Presence of extensive bullae translates to a higher risk pneumothorax [5].
- Chest physiotherapy prior to surgery to loosen secretions and dislodge mucus plugs. Signs of active infection such a pyrexia, purulent sputum, or worsening cough may warrant delaying surgery until improvement.
- Nitrous oxide has the potential to expand and possible rupture pulmonary bullae.
- Consider β-2 agonist and an antimuscarinic (e.g., robinul) to increase airway patency.
- Prolonged recovery should be anticipated due to air trapping when using inhalational agents.
- Avoid over oxygenation as this will cause a ventilation-perfusion mismatch due to inhibition of autoregulatory mechanism known as hypoxic pulmonary vasoconstriction. This leads to more blood flow to poorly ventilated areas. Maintain oxygen saturation between 88% and 92% [6].
- On ventilator, watch out for breath trapping (intrinsic positive PEEP) may lead to right heart strain. Consider increasing inspiratory to expiratory ratio to allow more exhalation time closer to 1:3 to 1:5 [5].
- Chronic hypercapnia leads to a decrease in ventilatory drive in response to elevated CO_2 levels. Chemoreceptors in the medulla oblongata reset and initiate ventilation at a higher concentration of CO_2. Respiration drive is mostly dependent on anoxic stimulation of peripheral chemoreceptors (another reason to not excessively oxygenate the patient).
- Patients have limited ability to tolerate hypoventilation because they have a diminished response to hypercarbia. Opioids diminish the respiratory drive and predispose the patient to apnea (use opioids judiciously).
- Local anesthesia is preferred.

Pulmonary Embolism

Pulmonary embolism – acute, partial, or complete obstruction in the pulmonary arterial vasculature leading to a ventilation perfusion mismatch.

Sources of emboli include the following:

- Thrombus from a vein of the lower extremity.
- Mural thrombus that develops from atrial fibrillation.
- Fat embolism from long bones.

Signs and symptoms:

- Pleuritic chest pain
- Dyspnea
- Tachypnea
- Hemoptysis
- Coughing
- Jugular venous distension
- Cyanosis
- Rales and rhonchi
- Diminished breath sounds

Risk factors include (Virchow's triad – stasis, damage to the endothelium, and a hypercoagulable state) the following [7]:

- Immobility (stasis – e.g., lack of ambulation after surgery)
- Malignancy
- Disability or obesity
- Oral contraceptives
- Pregnancy
- Factor V Leiden, antiphospholipid syndrome, and protein C and S deficiency
- Deep vein thrombosis (DVT)
- Recent long bone fracture
- Hormone replacement therapy

Diagnosis:

- Signs and symptoms of a PE.
- D-dimer assay – highly sensitive. If negative then a PE is unlikely.
- Chest radiograph may show cardiomegaly, atelectasis, pulmonary effusions, and a raised diaphragm.
- CT angiography.
- Venous ultrasonography to rule out DVT.
- Arterial blood gas – can see hypoxemia, respiratory alkalosis, and hypocapnia [7].
- Wells criteria >4 probable PE (Table 14.4).

Treatment

- Options include anticoagulation to prevent progression of the thrombus and to decrease additional thrombotic episodes. Consideration is given for fibrinolytics and embolectomy for massive pulmonary emboli.
- Initial treatment is with unfractionated heparin (to a goal PTT of 60–85), low molecular weight heparin with bridging to warfarin for a goal INR of 2–3.
- If anticoagulation is contraindicated, then an inferior vena cava filter is indicated.
- 3 months of oral anticoagulation is recommended for most patients (pulmonary embolism provoked by surgery, unprovoked first PE with no history of DVT, and PE provoked by nonsurgical transient risk factor). Lifelong anticoagulation is required for patients with an unprovoked PE, recurrent DVTs, massive PEs, and malignancies associated with thrombosis.
- Common oral anticoagulants used are warfarin (Coumadin®), dabigatran (Pradaxa®), rivaroxaban (Xarelto®), apixaban (Eliquis®), and edoxaban (Savaysa®).

Patient Management

- Consult with physician managing the patient's anticoagulation to determine whether or not a drug holiday from the anticoagulant is possible.
- Obtain preoperative INR if warafin is utilized.
- Trend INR and PTT for inpatients that are being bridged to Coumadin®. Generally an of INR < 1.5 is suitable to more invasive procedures and patient is to continue bridging pro-

Table 14.4 Wells criteria to determine likelihood of PE [8]

Criteria	Points
Clinical signs/symptoms of DVT	3.0
High suspicion of PE as the diagnosis	3.0
Heart rate > 100 BPM	1.5
Surgery within 1 month or immobility >3 days	1.5
History of PE or DVT	1.5
Malignancy	1.0
Hemoptysis	1.0

tocol until goal INR achieved with first dose evening or morning after procedure.
- Use judicious hemostatic measures in the setting of anticoagulation.
- Avoid nasal intubation in the setting of general anesthesia procedures.
- Employ DVT prophylaxis (e.g., sequential compression device and postoperative heparin).
- Encourage early ambulation after surgery.
- Avoid NSAID use in the setting of anticoagulation to decrease the chance of perioperative bleeding.

COVID 19

Background

- Coronavirus disease 2019 is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).
- It is a single-stranded, positive sense RNA virus [9].
- SARS-CoV-2 is a betacoronanvirus, one of the four genera of coronaviruses. Betacoronavirus are of great clinical concern in humans as they produce the lineage of viruses that cause the common cold.
- The betacoronavirus also circulates in animals, but rarely do the animal viruses infect people.
- Previous outbreaks of coronaviruses included SARS-CoV-2 and Middle East respiratory syndrome (MERS-CoV), which are zoonotic and have their origins in bats. There is evidence that SARS-CoV-2 may be originated in horseshoe bats due to similarities in genomic architecture [10].
- Coronaviruses are human viruses that attack the human respiratory system. SARS-CoV-2 is the seventh member of the coronaviruses that infect the human respiratory system.
- The first clusters of the novel coronavirus, SARS-CoV-2, appeared in Wuhan, China in 2019 with patients presenting with pneumonia of unknown etiology [11].
- Transmission from person to person occurs through direct contact and droplets spread through coughing, sneezing, and talking. There is a possible role for fomites in that contaminated surfaces can harbor viral particles.
- Symptomatic and asymptomatic transmission can occur with a high rate of infectivity.
- Increased susceptibility to severe disease in patients with multiple comorbidities (HTN, DM, COPD) and elderly patients.
- Men have a higher susceptibility to severe disease compared to women.
- African Americans and Latinos are at a higher risk of morbidity in comparison to their White counterparts.

Pathogenesis

- Peptomers on the surface of the virus are the glycoproteins responsible for entry into host cells.
- SARS-COV-2 gains entrance through points such as the nasal, ocular, and oral cavity mucosae by binding to the human angiotensin-converting enzyme II (ACE2) receptors in these tissues via the peptomers on the viral surface.
- Alveolar epithelial cells have an abundance of cells with ACE2 in the pulmonary parenchyma which is why fulminant lung disease may occur. Binding activates immune cells inducing secretion of inflammatory mediators into the pulmonary vessels.
- Associated systemic cytokine storm can lead to widespread thrombosis and multiple organ involvement and has a serious impact on morbidity and mortality.
- Complications include acute respiratory disease, acute kidney injury, myocardial injury, thrombotic events, Kawasaki disease, multiorgan failure, and Guillain-Barre syndrome.

Signs and Symptoms

- Symptoms appear after an incubation period of 1–14 days with 5–6 days being the average [10, 11].
- The most common symptoms at onset include: fever, cough, sore throat, myalgia, fatigue, dyspnea, headache, loss of taste (dysgeusia), and smell (anosmia) [12].
- About 25% of patients have diarrhea [9].
- Rhinorrhea and sore throat surprisingly do not commonly occur [9].
- Most cases are mild and self-limiting.
- Severe cases progress to severe pneumonia, acute respiratory syndrome (ARDS), multiorgan dysfunction syndrome, thrombotic manifestations, cardiac arrest, shock, and death secondary to cytokine storm.

Diagnosis

- On physical exam, patients may appear tachypneic with labored breathing. They typically have fever exceeding 39 °C/102.2 °F (extreme age or immunodeficient patient may not develop fever).
- Hypotension may lead to tachycardia and cool extremities.
- Ophthalmic signs include conjunctival secretions, injection, and chemosis.
- Dermatological signs include erythematous rashes and petechiae.
- Polymerase chain reaction tests are standard for diagnosis. Samples include nasal and oral swabs to test for virus RNA. Bronchial fluid can also be sampled for viral RNA.
- CBC with differential.
  - WBC: Low to normal with higher than normal neutrophils.
- Elevated CRP, ESR, and D-dimer.
- CXR may show infiltrates and evidence of pneumonia.
- Computed tomography (CT) scan of the chest may reveal ground glass opacities, perihilar lymphadenopathy, and consolidations.

Head and Neck Manifestations

- Maculo-papular, acral, urticariform, vesicular, and vascular obstruction lesions are the most common cutaneous manifestations of COVID-19 [13].
- Common oral manifestations specifically include: xerostomia, vesiculobullous lesions, aphthous-like lesions, dysgeusia, facial pain [14].
- Salivary glands may be a reservoir for the virus.
- Cause-effect relationship has not been solved at present; however, research is being conducted on whether or not oral lesions are related to immunosuppression rather than the virus itself.
- Temporal and oral pigmented lesions are associated with hydroxychloroquine use.

Treatment

- Mostly supportive (antipyretics, analgesics, fluid resuscitation, and non-ventilator oxygen therapy). Unstable patients may require mechanical ventilation or extracorporeal membrane oxygenation (ECMO).
- Quarantining of patients with symptoms and a proven diagnosis at home. Hospitalized patients with a confirmed diagnosis and patients under investigation (PUI) should be kept on an isolated COVID-19 unit.
- Broad spectrum antiviral drugs, such as HIV-protease inhibitors and nucleoside analogues, offer some viral attenuation.
- The antiviral remdesivir and dexamethasone, at the time of this writing, have shown some success in the treatment of COVID-19.

Management of Patients in the Setting of COVID-19

- SARS-CoV-2 is sensitive to ultraviolet light and heat, 75% ethanol, ether, chloroform,

chlorine disinfectants, and other disinfectants [15].
- 10% betadine found to be viricidal to particles (need at least 15 seconds of contact).
- Judicious use of PPE, face masks, and medical grade N95 masks.
- Oral and maxillofacial surgeons and staff are considered to have a considerable risk of transmission due to aerosol producing procedures. There are high viral loads in the sinonasal and oropharyngeal regions; therefore, preoperative baseline testing for risk stratification is paramount. Nasoendotracheal intubation also increases the risk of transmission.
- Phone screenings prior to appointments should be considered.
- Frequent handwashing, social distancing in waiting areas, and disinfection of high contact areas are mainstays to controlling the spread of the virus [14].
- Consider using negative pressure rooms in the operating room setting for patients with unknown COVID-19 status.
- Influenza vaccination should be considered in the midst of the COVID-19 pandemic.
- COVID-19 is an ever-evolving pandemic; the prudent surgeon should continue to monitor and follow changing scientific literature and federal, state, and local public health guidelines as well.

Allergic Rhinitis

Background

- Allergic rhinitis is an IgE-mediated inflammatory process of the nasopharynx, oropharynx associated with allergen exposure.
  - Overall prevalence of about 25–40%, which peaks in childhood and adolescence [16, 17].
  - Continuous exposure due to home or work contamination may cause some symptom variability in perennial allergic rhinitis (sinus mucosal thickening and postnasal discharge).
  - Perennial allergic rhinitis is usually developed in adult life and may be caused by cockroach allergens, fungal spores, or latex.
  - Generally, occurring in atopic patients [16, 18].
    - 40% of allergic rhinitis patients also display symptoms of asthma.
    - 70% of asthmatics experience rhinitis.
  - Associated with asthma, rhinosinusitis, otitis media, sleep disorders, and there is a strong association with dental malocclusion due to chronic mouth breathing [18].
- Nonallergic rhinitis with eosinophilia syndrome (NARES) occurs in middle decades of life and is associated with anosmia, chronic sinusitis, and aspirin intolerance.
  - Resembles allergic rhinitis but occurs with nonspecific stimuli (e.g., chemical odors and position changes).
- Vasomotor rhinitis is a form of noninflammatory rhinitis with unknown etiology.
  - Often triggered by nonspecific stimuli such as smoke or cold air.
- Rhinitis medicamentosa is caused by rebound vasodilation after use of nasal decongestants are used for an extended period of time.

Pathophysiology

- Nasal mucosa traps particles which are then moved by ciliary action to the pharynx leading to lysozyme release of protein allergens.
- Common inhaled allergens originate from pollen, mold, dust mites, animal hair, dander, and insects.
- Initial interaction occurs between allergen and intraepithelial mast cells, which are sensitized to IgE. Mast cells then degranulate lib-

erating histamine, prostaglandins, and leukotrienes.
- With hyperemic and edematous mucosae during symptomatic seasons, adverse reactivity is enhanced.
- Biopsy specimens of nasal mucosa during seasonal rhinitis show submucosal edema with mostly eosinophilic infiltrates.

Signs/Symptoms

- Cardinal symptoms include clear and watery rhinorrhea, sneezing, pruritus of nose, palate, throat, or ears [18].
- Ophthalmic symptoms include itching, conjunctival injection, and epiphora [18].
- Obstruction of the sinus ostia and eustachian tubes.
- Increase in nasal polyps (mucosal protrusions with eosinophilic fluid).
- Chronic signs and symptoms of allergic rhinitis, especially in children, include:
  - Allergic shiners: dark circles beneath the eyes that form from chronic venous congestion.
  - Dennie's sign: prominent folds originating near the medial canthus and traversing the lower lid.
  - Transverse nasal crease along the lower third of the nose from nasal itching.
  - Mouth breathing and adenoid-type facies.

Diagnosis

- In seasonal allergic rhinitis, diagnosis mainly depends on accurate history of symptoms concurrent with pollination of offending plants.
- Recurrent viral or bacterial infection and tonsillar and adenoidal hypertrophy must be ruled out.
- Demonstration of immunologic specificity for IgE is critical to etiologic diagnosis, particularly in severe cases.
  - Skin-prick test of allergens in question is a quick and reliable method to identifying allergen sensitized mast cells.
  - An intradermal test may follow if the skin test is inconclusive.
- Nasal secretions will be rich in eosinophils and useful for ruling out nonallergic rhinitis when diagnosis is not clear.

Treatment

- Prevent exposure to known offending allergens.
- Treatment of symptoms is the most commonly used treatment.
- Oral antihistamines in the H_1 class are effective for nasal itching and lacrimation. First-generation drugs are more lipophilic and cross the blood-brain barrier and tend to produce sedation (e.g., diphenhydramine). Second-generation drugs are less lipophilic and hence less sedating and have decreased psychomotor impairment (e.g., loratadine and fexofenadine).
- Nasal H_1 antihistamines are also available and have been shown as efficacious as oral form (e.g., azelastine). Significantly reduces congestion and itching compared to oral form but may cause alteration in taste and smell [18].
- Decongestants α-adrenergic agents can be used topically for nasal congestion (e.g., oxymetazoline or pseudoephedrine).
  - Limited use due to rebound rhinitis (in use longer than 5 days) and systemic hypertension are side effects reducing the duration of their efficacy. Its use should be limited for acutely severe rhinitis.
  - Pseudoephedrine should be avoided in patients with narrow angle glaucoma, urinary retention, pregnancy, and severe hypertension.
- Intranasal glucocorticoids are the most effective in relief of established rhinitis (e.g., fluticasone, beclomethasone, and budesonide). Effects occur 7–8 h after administration [18]. Side effects include:
  - Epistaxis range from 4% to 8% in first 2 weeks [18].
  - Local irritation occurs in approximately 10% of patients [18].

  - Candida overgrowth is a rare side effect, whereas nasal irritation is a common side effect of this medication.
- Immunotherapy or hyposensitization requires gradually increasing concentrations of offending allergens.
  - This lasts 3–5 years with weekly or monthly injections being the most effective.
  - Should be avoided in patients with unstable asthma and significant cardiovascular disease.
- Systemic monoclonal antibody treatment blocks mast cell and basophil sensitization (e.g., montelukast, zafirlukast, and zileuton).

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Renal Disease

15

Simon Bangiyev and Jaclyn A. Tomsic

- The kidney has metabolic and synthetic functions [1, 2]. The metabolic functions take place via a balance of filtration, reabsorption, and secretion.
- The functions of the kidney include the following:
  - Elimination of metabolic waste and nonessential materials.
  - Fluid balance, electrolyte balance, and composition.
  - Maintenance of acid/base levels.
  - Secretion of renin by the juxtaglomerular cells.
  - Secretion of erythropoietin, conversion of vitamin D, calcium and phosphorus homeostasis.
  - Regulation of blood pressure.
- Renal function is measured by the glomerular filtration rate (GFR).
  - GFR is estimated using the Cockroft-Gault formula = [(140−age) × (lean body weight in kg) × (0.85 if female)/(serum creatinine mg/dL) × 72].
  - GFR is expressed per 1.73 m^2 surface area; affected by age, sex, and body size.
  - Average GFR for adult male = 130 mL/min [3].
  - Average GFR for adult female = 120 mL/min [3].
  - Chronic kidney disease occurs when GFR is reduced by at least 50 mL/min or when it is lower than 60 mL/min/1.73 m^2 [3].
  - GFR is most commonly measured using the body's clearance of the creatinine; creatinine is a by-product of muscle metabolism; it is almost exclusively filtered through the glomeruli of the kidney, therefore, making it a good clinical indicator of renal function.

Stages of Renal Dysfunction and Failure

Acute Renal Failure (ARF)/Acute Kidney Injury (AKI)

- The loss of renal function over hours to days that results in disturbances in fluid, electrolyte, and acid-base homeostasis. Diagnosis is based upon a serum creatinine increase by more than 0.5 mg/dL or a serum creatinine concentration rise of more than 25% in a patient with chronic kidney disease and a reduction of GFR by 50% [4].
- This is most commonly measured using creatinine levels as it approximates GFR closely.

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- Blood urea nitrogen (BUN) levels are also helpful, but less reliable due to other distracting factors (e.g., GI bleeding, increased protein intake, low urine output/dehydration, use of catabolic drugs such as steroids and tetracycline) as they can cause false elevation [3]. Also, up to 50% of BUN can be reabsorbed, while a negligible amount of creatinine is secreted.
- The most accurate estimate of renal function is a 24-hour urine collection, which is used to compare differences in plasma to urine creatinine and nitrogen levels.
- ARF is classified into three categories (Table 15.1) [3, 4]:
  - Prerenal azotemia – conditions that cause a fall in GFR because of reduced glomerular perfusion pressure (BUN:CR > 20:1 and a FENA < 1%).
  - Intrinsic renal failure – direct damage to the structures of the kidney.
  - Post renal failure – obstruction from either upper or lower urinary tract.

Table 15.1 Causes of AKI

Pre-renal (60%)	Renal (intrinsic) (30%)	Post-renal (obstructive) (10%)
Hypovolemia – decreased renal perfusion: hemorrhage, diarrhea, diuretics Hypotension/ reduce blood flow: cardiac failure, sepsis, dehydration Drugs: ACE inhibitors, NSAIDs (altering PG and ATII levels that maintain renal perfusion) Intraoperative: stimulation of renin-angiotensin-aldosterone axis causes vasoconstriction Postoperative: intravascular volume depletion (redistribution of ECF, CHF, MI, vascular obstruction)	Acute tubular necrosis (ATN) – Injury that directly damages the tubular epithelial cells Common causes are toxic, septic and ischemic Toxins: ethylene glycol, contrast dye, myoglobinuria, NSAIDS, aminoglycosides and amphotericin B Ischemia: embolism, dissection, cardiovascular surgery, severe blood loss, and severe hypotension Sepsis Acute interstitial nephritis (AIN) – edema and inflammation of the renal interstitium Drugs implicated in AIN: penicillin, diuretics, cimetidine, NSAIDs	Renal vein occlusion Urinary tract obstruction Anticholinergic-associated bladder dysfunction from anesthetic agents or antihistamines

Evaluation for Acute Renal Failure

- Detailed history and physical including pertinent labs (CBC with differential, CMP, coagulation profile, urinalysis, urine electrolytes).
- FE_{Na} = fractional excretion of sodium in urine; measures the differences between sodium and creatinine in the plasma (P) and urine (U); (U Na/P Na)/(U Cr/P Cr) × 100%.
- Clinical signs include the following:
  - Patient may or may not be oliguric (<400 mL/d) or anuric (<50 mL/d).
  - Volume depletion: orthostatic hypotension, tachycardia, dry mucous membranes.
  - Dermatologic signs: rash, purpura, livedo reticularis (mottled reticular lace-like purplish skin discoloration caused by capillary obstruction), gangrene, digital cyanosis (AIN or renal artery occlusion) [3].
  - Cardiovascular signs: third heart sound, jugular venous distension, and peripheral and pulmonary edema (cardiac failure).
  - Upper quadrant tenderness (ureteral obstruction or renal infarction).
  - Laboratory findings of acute renal failure (Table 15.2).
- Urinalysis findings in ARF:
  - Brown granular casts and epithelial cells represent ischemia or nephrotoxic ARF.
  - Heme in the absence of red blood cells represents rhabdomyolysis.
  - Eosinophils associated with fever, rash, peripheral eosinophilia represent AIN.
  - Red cells casts, protein, red blood cells represent glomerulonephritis.

Table 15.2 Laboratory findings in acute renal failure

Pre-renal	Renal	Post-renal
Urinalysis normal BUN:CR ratio 20:1 (characteristic) Urinary Na < 20 mEq/L Urine osmolality >500 mOsm/kg FENa < 1%	Urinalysis abnormal and BUN:Cr ratio normal Urinary Na > 40 mEq/L Urine osmolality <350 mOsm/kg	Normal urinalysis BUN:Cr ratio elevated FENa > 1%

Management of ARF

- Preoperative hypotension and volume depletion can lead to perioperative renal ischemia and, therefore, the aforementioned must be addressed prior to surgery.
- Identify potential risk factors: volume depletion, hypotension, sepsis, nephrotoxic exposure, preexisting chronic kidney disease.
- Elective surgery should be postponed until abnormalities have improved.
- Discontinue or use NSAIDs cautiously. NSAIDs inhibit the synthesis of prostaglandins, which are vital in the maintenance of renal blood flow and GFR. This puts the kidney at risk for perioperative failure in the susceptible patient.
- Consider short-term discontinuation of ACE inhibitors and angiotensin receptor blockers (ARBs). These inhibit actions on the efferent arterioles and worsen ARF [3].
- Avoid radiocontrast (RC) dye in patients with elevated BUN/Cr or chronic renal insufficiency. RC dye can cause direct toxic effects on the kidney or alter the production of nitrous oxide leading to acute failure [3]. Consider pre-treatment with N-acetylcysteine or sodium bicarbonate, which are protective agents; always minimize contrast loads and administer post-procedure hydration.
- Treat underlying cause postoperative ARF [3]:
  - Identify and eliminate causative agents.
  - Aggressive hydration.
  - Eliminate all nephrotoxins.
  - If obstructive, relieve obstruction.
  - Dialysis is the last resort if there is fluid overload, significant electrolyte abnormalities, and acid–base imbalances that are not relieved by the preceding measures.

Chronic Renal Disease (CRD)/ Chronic Kidney Disease (CKD)

- CRD is permanent renal insufficiency that develops over months to years caused by the structural intrinsic damage of the glomerulus or tubulointerstitial system resulting in damaging and irreversible complications. CRD occurs when the GFR is reduced to 50 mL/min. CRD eventually leads to ESRD at which point dialysis or transplant is mandatory to prevent death (Table 15.3).
- Chronic renal failure: pathophysiologic process with several known causes that leads to a decrease in nephrons and function. Detected by combination of imaging, blood and urine analysis, and GFR <60 mL/min for 3 or more months [5, 6].
- End stage renal disease (ESRD): the irreversible loss of kidney function such that the patient is permanently dependent on renal replacement therapy (dialysis or transplantation); GFR <15 mL/min [5].

Comorbidities and Sequelae in Patients with CRD

- *Cardiovascular*:
  - Causes up to 50% of mortality in ESRD patients [3].
  - Dyslipidemia is a common finding. CAD is found in about 40% of ESRD patients.
  - 40% of all patients on end stage dialysis have CHF [3].

Table 15.3 Stage of chronic renal failure [5–7]

Stage	Description	GFR (mL/min/1.73 m^2)
1	Slight kidney damage with normal or increased filtration	>90
2	Mild decrease in kidney function	60–89
3	Moderate decrease in kidney function	30–59
4	Severe decrease in kidney function	15–29
5	Kidney failure/ESRD	<15

  - Left ventricular hypertrophy is found in about 75% of all ESRD patients.
  - Patients develop hypertension due to inability to regulate salt/water balance and chronically elevated levels of ATII.
- *Anemia*:
  - ESRD patients develop anemia of chronic disease due to altered and decreased production of erythropoietin that increases the production of red blood cells in the tissues in response to decreased oxygen levels.
  - In general, the hemoglobin level in ESRD patients should be maintained between 11 and 12 g/dL.
  - Transfusing ESRD patients can be complicated by the development of blood antibodies; this can decrease an ESRD patient's chance at a donor kidney. However, if transfusion is indicated, typically when a patient is symptomatic or hemoglobin falls below 7 g/dL, it should be performed [3].
- *Platelet Dysfunction*:
  - ESRD patients suffer from a qualitative platelet defect and are at increased risk of intraoperative and postoperative bleeding from uremic platelet dysfunction [3, 7].
- *Gastrointestinal*:
  - Patients tend to have nausea and vomiting and are predisposed to developing an ileus.
  - Increased risk of aspiration.
- *Glycemic Control*:
  - Many patients with CKD have DM, which may have been the causative factor leading to CKD.
  - Glucagon, growth hormone, cortisol, epinephrine, and norepinephrine can be released due to surgical stress and anesthesia, worsening insulin resistance and deficiency in diabetics. This can lead to hyperglycemia and ketogenesis.
  - Diabetics are also at increased risk of becoming hypoglycemic postoperatively.
- *Risk of Infection*:
  - Patients with CKD have impaired phagocytosis, neutrophil chemotaxis, and malnutrition.
- *Secondary Hyperparathyroidism*:
  - Occurs as the kidneys lose the ability to convert vitamin D with a concomitant decrease in intestinal absorption.
  - Patients will have a total body decrease in calcium and be hypocalcemic. This leads to secretion of parathyroid hormone, which acts to increase serum calcium by bony resorption and an increase in intestinal absorption. This leads to renal osteodystrophy, which can be seen radiographically.
- *Electrolyte/Acid –Base Disturbances*:
  - Patients may be hyperkalemic due to the inability of the kidney to secrete potassium. A concomitant metabolic acidosis may occur due to hyperkalemia and inability to secrete hydrogen ions.
- *Oral Manifestations*:
  - Halitosis
  - Stomatitis
  - Gingival bleeding and petechiae
  - Osteolytic changes in the jaws, loss of lamina dura around teeth in more severe cases
  - Accelerated accumulation of calculus
- Uremia
  - Syndrome characterized by anorexia, altered mental status, vomiting, anemia, fatigue, and coagulopathy that mirror the kidneys' inability to perform its excretory, secretory, and synthetic functions. BUN concentration correlates with symptoms and reflects the patient's response to therapy.

Drug Considerations in Patients with CKD (Table 15.4)

- Patients with CRD and ESRD have abnormalities in drug metabolism leading to decreased clearance and prolonged effect.

Dialysis

- Dialysis is the process of removing excess fluid, solutes, and nitrogenous wastes. It is also known as renal replacement therapy.

Table 15.4 Drug safety in patients with CRD

Drugs to avoid	Drugs safe to use in ESRD patients
Aminoglycosides Radiographic contrast media NSAIDs Benzodiazepines (may be considered in reduced doses) Meperidine Morphine (with caution) Most inhaled anesthetics (transient but reversible depression) Barbiturates (may be used in reduced doses) Succinylcholine (causes hyperkalemia and its active metabolite is renally excreted) Non-depolarizing muscle relaxants	First-generation cephalosporins (empiric prophylaxis) Fentanyl Halothane, desflurane, nitrous oxide Propofol (hepatic clearance) Atracurium (action not prolonged; muscle relaxant of choice)

- There are two primary types of dialysis: hemodialysis and peritoneal dialysis.

Hemodialysis:
- Filtering of blood by diffusion across a semipermeable membrane to remove toxins while adding required substances [8].
- The blood is heparinized and passed through a dialyzer.
- Requires patients to obtain indwelling arteriovenous access; this can be achieved through ports into central veins (typically used temporarily while awaiting fistula maturation, 3–6 months) and surgically created fistulas. Patients must visit a dialysis center 3 days/week to receive treatment.

- Complications: shunt infection, shunt thrombosis and failure, hypotension, chronic blood loss [8].

Peritoneal Dialysis
- The instillation of dialysate solution into the peritoneal cavity, allowing toxins to passively diffuse into solution for removal [8].
- This treatment may be done at home, allowing patients to travel and is well tolerated.
- It must be done up to five time per day lending to longer treatment times and clearance of toxins is sometimes inadequate.
- Complications: catheter tunnel infections, peritonitis.

Dialysis Indications (AEIOU)
- *A*cidosis
- *E*lectrolyte disturbances (e.g., hyperkalemia)
- *I*ntoxications (e.g., methylene glycol, lithium)
- *O*verload (volume)
- *U*remia

Patient Management of Renal Disease

- Patients with CKD are tenuous surgical and anesthesia candidates because they often have other comorbidities including myocardial dysfunction, coronary artery disease, and peripheral vascular disease.
- Work up for comorbidities: EKG, echocardiogram, hemoglobin A1C.
- Optimize medically by controlling comorbidities (e.g., DM, HTN, CHF).
- They are less capable in handling changes in fluids, electrolytes (e.g., sodium, potassium, and phosphorus), acid loads, and the metabolism or excretion of medications.
- They must also be considered immunocompromised and, therefore, are at an increased risk for infections.
- Antibiotic prophylaxis is indicated for the first 6 months after fistula placement as transient bacteremia can lead to infection.
- Clinically these patients are at an increased bleeding risk due to platelet dysfunction. Consider preoperative pharmacological hemostatic agents such as DDAVP (use with caution to prevent volume overload), cryoprecipitate, and conjugated estrogens to prevent bleeding (DDAVP – administered IV or intranasally at 0.3 μg/kg 1 hour before surgery/cryoprecipitate – 10 U over 30 minutes IV 1 hour before surgery/conjugated estrogens – 0.6 mg/kg/d IV or 2.5–25 mg PO 5 days before surgery).
- Patients should undergo dialysis the day before surgery to minimize uremic complications and decreased risk of bleeding in the set-

ting of being heparinized. The hemodialysis process also destroys platelets.
- Utilize good surgical technique by achieving primary closure and utilize agents such as Gelfoam, Surgicel®, Floseal®, and topical thrombin if necessary.
- Correction or normalization of abnormalities preoperatively (electrolyte and acid–base disturbances).
- Avoidance of nephrotoxic agents and tight glycemic control can significantly decrease the risk of perioperative complications and postoperative infection [3].
- Preoperative labs: CBC (rule out anemia), coagulation studies, CMP (assess creatinine, calcium, magnesium, and potassium levels), urinalysis, and bleeding time.
- Erythropoietin can be administered preoperatively in the setting of anemia. The hematocrit can be raised up to 30% by administration of erythropoietin.
- Discontinue diuretics to avoid intraoperative hypotension and volume depletion.
- Optimize blood pressure control and consider holding ARBs and ACE inhibitors to reduce the risk of hypotension.
- Avoid using arm with AV shunt for monitoring blood pressure and venipuncture.
- Consider anti-emetic and pro-motility medications preoperatively due to the risk of aspiration.
- Adjust dosages of drugs excreted by the kidney accordingly.
- General anesthesia with volatile inhalational anesthetics reversibly depresses renal function and is short lived once discontinued. Sevoflurane has a metabolite known as compound A, which is nephrotoxic. Halothane, enflurane, and isoflurane are generally safe in ESRD.
- Depolarizing agents such as succinylcholine can increase potassium. Potassium must be closely monitored. As long as the potassium is not acutely elevated, succinylcholine can safely be used in ESRD patients.
- All non-depolarizing agents undergo some degree of renal excretion with prolonged duration of action and, therefore, should be dose adjusted. Atracurium and cisatracurium undergo spontaneous degradation via Hofmann degradation and ester hydrolysis and are generally safe.
- Propofol is mainly metabolized by the liver (none of the metabolites have been shown to be active). The presence of renal failure and uremia does not have an effect on the pharmacology of propofol, and there is no significant decrease in the clearance in ESRD patients. Furthermore, no significant dosing adjustments are necessary in ESRD patients. However, propofol can decrease myocardial function and causes hypotension that can worsen renal failure.
- Fentanyl is very lipid soluble and rapidly redistributed to inactive tissues. It is slowly released into the plasma and then cleared. In moderate doses, it is safe for use in renal failure patients.
- Benzodiazepines undergo hepatic metabolism and can be used with caution. Be mindful of the by-products of conjugation; diazepam forms two active metabolites: desmethyldiazepam and oxazepam, which can cause prolonged sedation.
- Avoid nephrotoxic drugs, e.g., NSAIDs, diphenhydramine, chlordiazepoxide, cimetidine, aminoglycosides, IV contrast dye, cephalosporins, erythromycin, tetracycline, acyclovir [9].
- Lidocaine (amide anesthetics), codeine, clindamycin, metronidazole are safe to use.
- Avoid meperidine as the normeperidine metabolite can induce seizures.
- Consider discontinuing all antiplatelet agents (aspirin, dipyridamole) 72 hours prior to surgery.
- Must be judicious when administering fluids, as there is a narrow margin of safety as it relates to insufficient and excessive fluid administration.

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Hematologic Disease

16

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Anemia

- Anemia is defined as a decrease in red blood cells (RBC), leading to less oxygen carrying capacity and delivery to end organs.
- Normal hemoglobin in adult males is 14–18 g/dL and in adult females 12–16 g/dL.
- Normal Hct in adult males is 40–52% and in adult females is 35–47%.
- Anemia may be caused by disorders of bone marrow production, red cell maturation, increased destruction, and iron deficiency. Anemia is subclassified into microcytic, normocytic, and macrocytic (Table 16.1). Clinical symptoms of anemia are fatigue and decreased exercise tolerance.

Iron and lab studies are used to determine the type and etiology of anemia. The most common studies used are as follows:

- Ferritin: marker of iron stores (also an acute phase reactant).

Table 16.1 Type of types of anemia and examples [1]

Microcytic	Normocytic	Macrocytic
Iron deficiency Thalassemia Sideroblastic anemia Lead poisoning	Acute blood loss Hemolysis Anemia of chronic disease Anemia of renal failure Myelodysplastic syndromes Aplastic anemia Hemolytic anemias	Folate deficiency Vitamin B_{12} deficiency Drug toxicity Alcoholism/ chronic liver disease

- TIBC (total iron binding capacity): indirect measure of transferrin saturation levels.
- Transferrin: protein which transports iron within the blood.
- MCV (mean corpuscular volume): average RBC volume; this value categorizes anemia:
  - Microcytic: MCV ≤ 80 fL
  - Normocytic: MCV = 80–100 fL
  - Macrocytic: MCV ≥ 100 fL
- Reticulocyte: immature erythrocyte (new RBC that is usually 1–1.5 days old).
- Reticulocyte count: fraction of RBCs consisting of reticulocytes that indirectly indicates the bone marrow activity of the erythrocyte line; expressed as a percentage; normal value 1%.

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Microcytic Anemias

Iron Deficiency Anemia

- Etiology is the decreased synthesis of heme. Iron deficiency anemia is the most common cause of anemia in the United States of all ages and both genders.
- Causes of iron deficiency anemia:
  - Blood loss (most common) – PUD, gastritis/NSAIDs, polyps/colorectal cancer, menorrhagia.
  - Increased utilization – pregnancy, infants/children.
  - Decreased intake – infants/children, elderly.
  - Decreased absorption – celiac sprue.

Treatment

- Iron supplementation
- Transfusion in specific cases for severe anemia
- Treat the underlying disease process

Thalassemia Anemias

A group of autosomal recessive anemic disorders due to the abnormal production of globin chains.

Alpha-thalassemia

- Decrease in synthesis of the alpha-globin chain of hemoglobin (Hgb).
- Common in Southeast Asia and Black Americans.
- Four genes control the production of the alpha chain of Hgb; combinations of deletions of 1, 2, 3, or all 4 of these chains cause different types and degrees of alpha-thalassemia.
- One or two deletions cause mild anemia with little symptoms and no need for treatment.
- Three gene deletions lead to HbH or four beta-chain disease, which causes a severe hemolytic anemia due to macrophage destruction of RBCs due to the excess beta chains.
- Four gene deletions are called Hb Bart and is incompatible with life.

Beta-thalassemia

- Decrease in the synthesis of the beta-globin chain of Hgb.
- Common in Black Americans, Greeks, and Italians.
- There are mild and severe forms:
  - Beta-thalassemia minor: mild microcytic anemia due to DNA splicing defect; decrease in HbA, increase in RBC count, HBA2, and ferritin production; no treatment.
  - Beta-thalassemia major: severe hemolytic anemia due to a nonsense mutation with formation of a stop codon; no production of HbA, increased production of HbA2 and HbF; these patients have lifelong danger of iron overload and therefore lifelong need for transfusion.

Macrocytic Anemia

- Increase in RBC size, commonly caused by either Vitamin B_{12} or folate deficiency.
- Most common cause of Vitamin B_{12} deficiency is pernicious anemia (decrease in intrinsic factor due to autoimmune destruction of gastric parietal cells).
- Most common cause of folate deficiency is alcoholism.
- Vitamin B_{12} is found in meat, eggs, and dairy products.
- Folate is found in green vegetables and animal proteins.
- Characteristic difference between Vitamin B_{12} and folate deficiency: neurologic disease (seen in Vit B_{12} deficiency but not folate deficiency).
- Lab findings in both: decreased Vitamin B12 or folate, increased homocysteine, pancytopenia and hypersegmented PMNs (PMN with more than five lobes).
- Other less common causes of macrocytic anemia: chemotherapy, hemolysis, liver disease, myelodysplastic syndromes, hypothyroidism

Normocytic Anemia

There are a number of conditions under this category that are beyond the scope of this, text but the following will be touched upon briefly.

Anemia of Chronic Disease (ACD)

- Most common anemia in hospitalized patients.
- Common causes: chronic inflammation (rheumatoid arthritis, tuberculosis), alcoholism, malignancy.
- Caused by decreased synthesis of heme.
- Lab findings: decreased iron, TIBC and iron saturation; increased ferritin
- ACD is microcytic 10–30% of the time.

Acute Blood Loss or Hemorrhage

- This may occur due to external causes (trauma, peptic ulcers) or internal causes (ruptured abdominal aortic aneurysm) and results in signs of volume depletion such as hypotension and tachycardia.

Aplastic Anemia

- Aplastic anemia is a bone marrow disorder.
- There are many causes of aplastic anemia and morbidity is high as only 10% of cases make a full recovery.
- In general, there is a destruction of multipotent myeloid stem cells with inadequate production of differentiated cell lines.
- The majority of cases are idiopathic; the most common known causes are drugs, commonly chemotherapy drugs (alkylating agents, antimetabolites).
- Other causes include chemical toxins (insecticides, benzene, parathion), viral infection (EBV, CMV, parvovirus B19, non-A and non-B hepatitis), and whole body radiation therapy.
- Lab findings: pancytopenia, low reticulocyte count (hallmark), hypocellular bone marrow.
- Clinical findings: fatigue, malaise, pallor, mucosal bleeding, petechia, infection.
- Treatment: withdrawal of offending agent, RBC and platelet transfusion, bone marrow transplant.

Hemolytic Anemias [2]

In general, hemolytic anemias are characterized by hemolysis of erythrocytes. These processes exhibit an increase in serum bilirubin leading to jaundice and an increase in reticulocytes as the bone marrow compensates for the loss of red blood cells.

Autoimmune Hemolytic Anemia (AIHA)

- Extravascular hemolysis due to the excessive destruction of RBCs by the liver and the spleen by autoantibodies against foreign RBC antigens.
- Warm type (70%) associated with IgG antibodies.
- Cold type (30%) associated with IgM antibodies.
- SLE (systemic lupus erythematosus) is the most common cause of AIHA.
- The direct antihuman globulin test (DAT or Coombs test) is the most important marker for AIHA.

Hereditary Spherocytosis

- An autosomal dominant RBC membrane defect.
- Intrinsic membrane defect of ankyrin or spectrin that causes extravascular hemolysis due to loss of RBC membrane and formation of spherocytes (small, round RBCs with no central pallor).
- Lab findings: increase in MCHC, RDW, RBC osmotic fragility (diagnostic for hereditary spherocytosis); normocytic anemia with spherocytes.
- Clinical findings: jaundice (due to increased unconjugated bilirubin), increased incidence of gallstones (due to increased conjugated bilirubin), splenomegaly.
- Treatment: splenectomy (Howell-Jolly bodies present after splenectomy).

Sickle Cell Anemia (SCA)

- Autosomal recessive disorder – intrinsic defect causing a hemoglobinopathy that causes extravascular hemolysis of sickled RBCs.
- Most common hemoglobinopathy in patients of African descent.
- SCA is a missense point mutation where valine is substituted for glutamic acid at the sixth position of the beta-globin chain of Hb; this mutation causes sickling of RBCs.

Table 16.2 Clinical findings and complications of sickle cell anemia

Clinical findings and complications in SCA (HbSS)
Aplastic crisis (due to parvovirus B19 infection)
Dactylitis (swelling of hands and feet in infants)
Acute chest syndrome (vaso-occlusion of pulmonary capillaries; high morbidity)
Aseptic necrosis of femoral head
Autosplenectomy (due to accumulation of sickled cells)
Increased risk of infection from encapsulated organisms (*Streptococcus pneumonia, Neisseria meningitidis, Haemophilus influenzae type b*) and *Salmonella paratyphi* osteomyelitis
Pain crises (due to vaso-occlusion), e.g., priapism
Splenic sequestration crisis

- Key pathologic processes in SCA: severe hemolytic anemia and vaso-occlusive crises (Table 16.2). Management is based on symptoms. Fluid hydration, pain management (PO or IV), oxygen therapy ($O_2 < 92\%$), and steroids.
- Treatment: hydroxyurea (increases HbF which prevents sickling) and bone marrow transplant.

Leukemia

- Leukemia is a disease of the mesenchymal cells of the blood in which there is an abnormal proliferation or an increased lifespan of myeloid or lymphoid cells.
- In the simplest form, all blood cells originate from a pluripotent stem cell, which can then differentiate into a myeloid (bone marrow) precursor or lymphoid (lymphatic tissue) precursor. Therefore, leukemias are classified as either myeloid or lymphoid and then subclassified as acute or chronic depending on its characteristics.
- Myeloid cells form – erythrocytes, polymorphonuclear lymphocytes (PMNs), macrophages/monocytes, platelets, eosinophils, and basophils.
- Lymphoid cells form – plasma cells (B cells), T cells, or natural killer (NK) cells.
- Pathogenesis of leukemias – block in stem cell differentiation leading to proliferation of neoplastic leukemic cells.
- General characteristics of all leukemias:
  - Increased number of circulating leukocytes in the blood.
  - Infiltration of the bone marrow by leukemic cells (both mature and immature);
  - Most common sites of leukemic infiltration are the spleen, liver, lungs, skin, lymph nodes.
  - Bone marrow failure: anemia (decreased RBCs), granulocytopenia of mature WBCs (leading to infections), and thrombocytopenia (decreased platelets) leading to hemorrhage.

Myeloid Leukemias [3, 4, 5]

- In myeloid leukemias, there is a somatic gene mutation resulting in the loss of control at one level of production which then leads to increased, decreased, or normal production of the cells of that line.
- The production of the other cells in that cell line is then indirectly disturbed. This disturbance, which can be quantitative or qualitative, causes crowding or suppression within the bone marrow.
- Therefore a common characteristic of myeloid leukemias is their infiltration of the bone marrow with a decreased number of normal myeloid cells of poor function.

Acute Myelogenous Leukemia (AML) [6, 3]

- This type of leukemia produces an increase in the number of immature myeloid cells (20%) in the bone marrow leading to bone marrow failure.
- Hematopoietic insufficiencies in the form of granulocytopenia, thrombocytopenia, and anemia are found.
- The incidence of AML is 2.3/100,000 people and increases with age and most commonly occurs between the ages of 15 and 59.
- AML accounts for upwards of 80% of acute leukemia in adults. It is more common in men than women. Common risk factors for AML are listed in Table 16.3.

Table 16.3 Common risk factors for the development of AML

Hereditary	Environmental	Drugs
Fanconi's anemia Down syndrome Klinefelter's syndrome Patau's syndrome Neurofibromatosis	Benzene Petroleum Radiation	Alkylating agents: Busulfan Cisplatin Carboplatin Chlorambucil Cyclophosphamide

- Fatigue is the most common presenting symptom. Other symptoms include dyspnea on exertion, dizziness and in the older patient angina, weight loss, anorexia, fever, and infection susceptibility.
- Gingivitis/swollen gums (leukemic infiltration) are symptoms of the neutropenia common in AML.
- Coagulation irregularities (bleeding and easy bruising) caused by thrombocytopenia.
- Physical examination yields splenomegaly (due to infiltration with leukemic cells), fever, infection and lymphadenopathy, and evidence of coagulopathy (ecchymosis, telangiectasia, petechiae, purpura, and bleeding gums).
- Work up includes blood tests (CBC, coagulation studies, blood smear, BMP, blood culture), bone marrow aspiration, and appropriate imaging.
- CBC – reveals anemia and thrombocytopenia; patients with AML may have high, normal, or low WBC counts.
- Coagulation studies – AML patients most commonly develop DIC (disseminated intravascular coagulation).
- Blood smear – increased number of circulating myeloblasts and Auer rods (splinter or rod-shaped granules within the cytosol of leukemic myeloblasts, most often found in the AML M3 subtype). Schistocytes or "helmet cells" (fragmented irregularly shaped RBCs with pointed ends common in DIC).

Treatment

- The most common treatment strategy for AML is the combination of an anthracycline drug, often idarubicin or daunorubicin, and the antimetabolite, cytosine arabinoside (Ara-C).

Chronic Myelogenous Leukemia (CML) [6, 3, 4]

- CML is a malignant myeloproliferative disorder associated with the Philadelphia chromosome characterized by the increase of myeloid cells in the peripheral blood and bone marrow.
- Philadelphia chromosome is the result of a translocation of the *BCR* gene from chromosome 22 with the *ABL* gene from chromosome 9. It is not hereditary but results from spontaneous somatic mutation.
- CML occurs rarely in children and is more common in older adults >60 years of age and in men. It accounts for approximately 25% of all leukemias.
- Risk factors include exposure to ionizing radiation and benzene.
- It has a very indolent course. It is often discovered incidentally on blood analysis.
- Patients may present with signs of anemia, fatigue, weight loss, and anorexia. Oral manifestations of CLL include petechiae, mucosal bleeding, acute periodontitis, and candidiasis.
- CBC shows WBC count between 50,000 and 200,000 cells/μL with myeloid cells in all stages of development on smear.
- Normocytic or macrocytic anemia is seen.
- Thrombocytosis or thrombocytopenia may occur.
- Bone marrow aspirate shows myeloblasts <10% and hypercellularity.

Treatment

- Tyrosine kinase inhibitors including imatinib (Gleevec®), dasatinib, nilotinib, and bosutinib are commonly used as first line of treatment. They are normally given orally and length of treatment is indefinite as long as disease remains controlled.
- Interferon-α is commonly used a second line of treatment or those whom are intolerant to tyrosine kinase inhibitors.
- Chemotherapy with stem cell transplant.

Lymphoid Leukemias [6, 3, 5]

- Lymphoid leukemias occur as a result of a single progenitor cell mutation.

- These mutated clones begin in the lymph nodes in lymphoid leukemias and spread throughout the lymphoid system and to the liver, spleen, and meninges.

Acute Lymphocytic Leukemia (ALL)

- Malignancy characterized by proliferation of immature lymphoid cells in peripheral blood, bone marrow, and other organs.
- ALL can be a lymphocytic B-cell, T-cell, or NK-cell neoplasm with 85% being B cell. NK cell is the least common. T-cell ALL is more common in adolescents and NK-cell ALL more common in adults.
- ALL in general is characterized by an infiltration of lymphoblasts or a clonal lymphoid stem cell disease.
- ALL is the most common childhood leukemia, accounting for 2/3rd of childhood leukemia and only about 20% of adult leukemia.
- Patients often present with signs of infection, fatigue, pallor, and spontaneous bleeding (often orally).
- Lymphadenopathy is common as is extranodal involvement of the CNS, testicles, liver, and spleen.
- Candidiasis, mucosal inflammation, neutropenic ulceration of the oral mucosa, chloroma, and herpetic infections that involve both keratinized and non-keratinized mucosa may also be seen.
- An entity known as *numb chin syndrome (NCS)*, a condition characterized by a sensory neuropathy and numbness involving the distribution of the mental nerve, may also be seen as a lone presenting symptom [7].
- CBC may show leukopenia, leukocytosis, or a normal count therefore bone marrow biopsy is the gold standard for diagnosis as well as classification.
- Bone marrow shows 20% or more bone marrow lymphoblasts confirming diagnosis of acute lymphoblastic leukemia.
- A normocytic anemia with thrombocytopenia is often seen.
- Work up requires a CT of neck, chest, abdomen, and pelvis. CT/MRI head for those with neurological symptoms.

Treatment

- ALL is the leukemia that is considered the most responsive to therapy, and at least 2/3rd of patients can be considered cured.
- Childhood cure rates have been cited as high as 85–90% while adult rates 60–80%.
- Common chemotherapeutic regimens include tyrosine kinase inhibitors for Philadelphia chromosome-positive disease in addition to vincristine, daunorubicin, prednisone, and cyclophosphamide (these agents are used without a tyrosine kinase inhibitor for Philadelphia chromosome-negative disease).

Chronic Lymphocytic Leukemia [3, 8, 5]

- Lymphoproliferative disorder with the proliferation and accumulation of mature-appearing neoplastic B-cells that cannot differentiate into plasma cells.
- CLL is the most common overall leukemia, most commonly in adults over the age of 60.
- Clinical presentation is indistinct, and up to 70% of patients are asymptomatic at diagnosis.
- It most often appears as generalized lymphadenopathy in patients over 60 years of age. Fatigue, weakness, frequent infections, and bleeding tendencies are also common.
- Patients may present with the classic "B symptoms" of fever (>38 °C), night sweats, and weight loss (>10% over a 6-month period or less).
- Laboratory findings include a peripheral WBC count of 15,000–200,000/μL comprised mostly of immature WBCs, rendering the patient neutropenic due to the lack of immunocompetent cells.
- Characteristic of CLL are "smudge cells" on the peripheral blood smear, which are fragile leukemic cells that become damaged during slide preparation and therefore appear smudged.
- Normocytic anemia and thrombocytopenia are also common.
- Patients with CLL also have an increased incidence of autoimmune diseases including autoimmune hemolytic anemia and autoimmune thrombocytopenia (ITP).

Table 16.4 Rai staging system for CLL

Stage	Findings	Median survival
0	Lymphocytosis alone	>15 years
1	Lymphocytosis with lymphadenopathy	9 years
2	Lymphocytosis with spleen or liver involvement	5 years
3	Lymphocytosis with anemia	2 years
4	Lymphocytosis with anemia and thrombocytopenia	2 years

- Hypogammaglobulinemia is frequently present in CLL patients, making them more susceptible to bacterial and viral infections.

Treatment

- Both treatment and prognosis are guided by staging. There is no standard curative treatment, and treatment is with goal of prolongation of life and symptom relief.
- The most frequently used CLL staging system is the Rai system. The Rai system is based on lymphocytosis +/− other cell count abnormalities and organ involvements (Table 16.4).

Asymptomatic patients may simply be observed.

- Symptomatic patients with lymphadenopathy or splenomegaly with B symptoms may be treated with oral alkylating agents (cyclophosphamide, chlorambucil, nitrosoureas, dacarbazine) with or without prednisone.

Bleeding Diatheses

Hemophilia

X-linked recessive bleeding disorder (two types A and B).

- Hemophilia A [9, 10]
  - Congenital deficiency in clotting factor VIII. Results from Factor 8 gene mutations on the X-chromosome.
  - X-linked recessive inheritance pattern.
  - Males are affected more than females.
- Hemophilia B [9, 10]
  - Congenital deficiency in clotting factor IX. Results from Factor 9 gene mutations on the X-chromosome.
  - X-linked recessive inheritance pattern.
  - Males are affected more than females.

- Diagnosis
  - With a known family history, often times with genetic testing a diagnosis can be made in utero or in the neonatal period.
  - Patients with mild disease may not be diagnosed until a major trauma or surgery in adolescence or as a young adult.
  - Partial thromboplastin time is prolonged. Prothrombin time is normal. Platelet count is normal.
  - Hemophilia A – factor VIII activity level below 40% of normal [10].
  - Hemophilia B – factor IX activity level below 40% of normal [10].
- Symptoms – Clinical features of hemophilia A and B are very similar. Classification is based on the severity of the disease:
  - Severe disease – Factor activity levels are below 1% of normal. Spontaneous bleeding multiple times per year.
  - Moderate disease – Factor activity levels are between 1% and 5% of normal. Infrequent spontaneous bleeding but prolonged bleeding as a result of trauma or surgical procedures occurs.
  - Mild disease – Factor levels are greater than 5%, but below normal. Prolonged bleeding with trauma or surgical procedures.
  - Hemarthrosis of multiple joints can occur and with repeat episodes can result in severe joint damage.
  - Intracranial hemorrhage in newborns.
  - Muscle bleeding, gastrointestinal bleeding, hematuria, easy bruising.
  - Patients can develop compartment syndrome from the development of large hematomas.

Treatment

- Non-pharmacologic: patient education, avoidance of contact sports.
- Patients should avoid NSAIDs and ASA.
- Regular orthopedic evaluation with joint involvement.
- Guided by type of bleeding or surgical intervention.
- Recombinant factor concentrates of factor VIII or IX. Goal is to raise the levels 80–100% prior to surgery and maintain 50% 1–2 weeks postoperatively.
- Factors should be administered 20 minutes postoperatively due to short half-lives.
- Hemostatic adjuncts to factor concentrate administration:
  - Anti-fibrinolytic agents such as tranexamic acid.
  - Desmopressin (DDAVP).
    Administered intravenously, subcutaneous or intranasal.
    Can be used for mild to moderate hemophilia A as it increases Factor VIII:C and vWF levels.
    Avoid excess water intake to prevent hyponatremia.

Von Willebrand Disease (vWD)

- Congenital platelet bleeding disorder due to a deficiency or dysfunction of von Willebrand factor (vWF).
- vWF is responsible for mediating platelet adhesion, adhesion to the endothelium, and prevention of degradation of Factor VIII.
- Most commonly inherited bleeding disorder (not sex linked). Autosomal dominant inheritance pattern is the most common.
- May be acquired by hematoproliferative disorders or in autoimmune disorders such as systemic lupus erythematosus or hypothyroidism.
- Divided into three types:
  - Type 1 – Partial quantitative deficiency (most common type).
  - Type 2 – Qualitative deficiency and is further divided into different subtypes (2A, 2B, 2N, 2M).
  - Type 3 – Total or near total quantitative deficiency (autosomal recessive).
- Symptoms – mucous membrane type bleeding:
  - Gingiva.
  - Menorrhagia.
  - Epistaxis.
  - Gastrointestinal.
- Type III can result in more severe symptoms including joint bleeding and mimics hemophilia.
- In many cases, a patient may not have symptoms until major trauma or surgical intervention results in excessive bleeding.
- Diagnosis
  - CBC, PT – normal. PTT normal unless Factor 8 is affected.
  - No single laboratory test available, recommendation for three assays for initial screening:
  - Ristocetin cofactor assay – measures ability of patient plasma to agglutinate platelets by addition of an antibiotic Ristocetin (off market due to known to cause thrombocytopenia and platelet agglutination).
  - vWF antigen (see decrease).
  - Factor VIII coagulant activity (see decrease).
- Multimer analysis aid in identifying subtype, only done once abnormality detected.

Treatment [11, 12]

- Type 1
  - Desmopressin (DDAVP)
    Causes release of stored vWF from endothelial cells.
    Intravenous 0.3 μg/kg over 20 minutes.
    Intranasal 150 μg spray to each nostril.
    Monitor free water intake as there is a risk of hyponatremia and seizures.
- Type 2 and 3
  - DDAVP in some type 2 variants can cause an increase in abnormal vWF and is often avoided.
  - Cryoprecipitate can be used to treat all types of vWD. Contains Factors 8, 9, vWF, fibrinogen, and fibronectin. Its use is more

often reserved to treat qualitative forms. There is additional risk of transfection.
  - Factor VIII concentrate is rich in vWF.
  - Humate-P (vWF/Factor VIII) concentrate.
  - Amicar – Antifibrinolytic agent given orally or IV.

Thrombophilic Disorders

Antithrombin III Deficiency
- Inherited deficiency of antithrombin III leading to increased risk for recurrent thrombosis (e.g., DVT and PE).
- Concern for heparin resistance.
- Antithrombin III is an inhibitor of thrombin and activated coagulation factors leading to increased protease activity and fibrin formation.
- Type I – Quantitative deficiency and Type II – Qualitative deficiency.

Treatment
- Patients may be treated with heparin and bridged to warfarin therapy if history of thrombosis.
- May require FFP, cryoprecipitate, or AT III concentrate perioperative.

Protein C Deficiency
- Qualitative or quantitative deficiency in protein C, which inhibits blood coagulation by inactivation of factors 5 and 13 causing a prothrombotic state.
- Protein C is a vitamin K-dependent coagulation factor synthesized in the liver.
- Autosomal dominant inheritance pattern.
- Can be hereditary or acquired, e.g., liver damage, DIC, or vitamin K deficiency
- Hypercoagulable state results from qualitative or quantitative deficiency in plasma glycoprotein C required for inactivation of clotting factors Va and VIIIa leading to hypercoagulability.
- Type I – Quantitative deficiency and Type II – Qualitative deficiency.

Treatment
- Patients may be on warfarin if history of thrombosis.
- Acute thrombosis may require heparanation and/or transfusion of FFP to increase protein C levels.

Factor V Leiden
- Hereditary gene mutation resulting in resistance to activated protein C.
- Resistance to inactivation of factor V by activated protein C secondary to a F5 gene mutation.
- Autosomal inheritance pattern.
- Heterozygosity results in 5–10 fold increased risk for thrombosis.
- Homozygosity results in 50–100 fold increased risk for thrombosis.
- Symptoms are associated with recurrent venous thromboembolism (VTE) [13, 14].
  - Deep vein thrombosis of the lower extremities.
  - Pulmonary embolism.
  - Superficial thrombophlebitis.
  - Arterial thrombosis is not generally associated with hypercoagulable states, but arterial embolism can occur from venous thromboembolism crossing a patent foramen ovale.
- Infants may present with thrombotic purpura or purpura fulminans and disseminated intravascular coagulation.
- Initial VTE usually occurs in early adulthood.

Treatment
- Early ambulation after surgery.
- Mechanical methods of anticoagulation.
  - Compression stockings.
  - Intermittent pneumatic compression devices.
  - Venous foot pumps.
- Subcutaneous heparin injection for DVT prophylaxis may be warranted based on risk of VTE in each patient based on surgical procedure and the expected postoperative course.

- Long-term anticoagulation with warfarin is sometimes indicated.
  - Patients should be anticoagulated with heparin until vitamin K-dependent clotting factors are sufficiently inhibited otherwise there is an increased risk of thrombosis and skin necrosis with use of warfarin.

Lymphoma

Lymphoma – malignancy of lymphocytes that usually arises in lymph nodes.

There are two main types (Hodgkin and non-Hodgkin lymphoma).

Hodgkin Lymphoma (HL)
- Male predilection (between age 15 and 35).
- Presents with painless lymphadenopathy (usually supraclavicular)/75% in the neck [15].
- Presents with B symptoms – night sweats, fevers, and weight loss.
- Spleen involved 20% of the time.
- Can also see extranodal involvement – bone marrow, lungs, liver, bone.
- Characterized by the presence of Reed-Sternberg malignant cells that are seen on histopathology (binucleated cells with owl eye nuclei).
- Work Up
  - Biopsy with histopathology.
  - Imaging – CT and PET scan for staging.
  - Clinical exam.
  - CBC and CMP – look for anemia, leukocytosis, thrombocytosis, hypoalbuminemia.

Treatment
- External beam radiation (20 Gy).
- Chemotherapy (adriamycin, bleomycin, vinblastine, dacarbazine).

Non-Hodgkin Lymphoma (NHL)
- Malignant lymphoproliferative lesions that are not Hodgkin lymphoma.
- 85% are of B-cell origin [15].
- Present with B symptoms and painless lymphadenopathy.
- Can also see extranodal involvement – bone marrow, lungs, liver, bone.
- Characterized as low, intermediate or high grade.
- Cells express CD20 marker.
- Viruses may play a role – EBV (Burkitt's lymphoma), HHV, HTLV, HIV, HEP B.
- Work Up
  - Biopsy with histopathology.
  - Imaging – CT and PET scan for staging.
  - Clinical exam.
  - CBC and CMP – look for anemia, leukocytosis, thrombocytosis, hypoalbuminemia.
  - HIV, HEP B serological assays.

Treatment
- External beam radiation.
- Rituximab (anti-CD20 monoclonal antibody).
- CHOP chemotherapy (cyclophosphamide, doxorubicin, vincristine, prednisone).
- Stem cell transplant.

Multiple Myeloma

- Malignancy characterized by the neoplastic transformation of plasma cells producing a monoclonal immunoglobulin.
- Proliferation occurs in the bone marrow and results in osteolytic lesions, hypercalcemia, osteopenia, and pathologic fractures.
- Disease of older adults (mean age 66 years old).
- Occurs in people of African descent 2–3 times more than in whites [15].
- Associated signs/symptoms:
  - Bone pain secondary to osteolysis.
  - Anemia (usually normochromic and normocytic).
  - Increase in urine monoclonal protein.
  - Increase in serum total protein.
  - Renal failure due to light chain nephropathy.
  - Hypercalcemia due to osteolysis.
  - Susceptibility to infection due to incompetent plasma cells.
- Work Up
  - Clinical exam.
  - Protein electrophoresis to detect monoclonal protein.
  - Serum and urine M protein.

  - Peripheral smear shows Rouleaux formation (stacked RBCs resembling coins), leukopenia, and thrombocytopenia.
  - Bone marrow biopsy to assess for myeloma cells.
  - CBC.
  - CT and PET scan for survey of bones.

Treatment

- External beam radiation for focal bony processes not responsive to chemotherapy.
- VRD chemotherapy (velcade, revlimid, decadron).
- Stem cell transplant.
- Anti-resorptive agents for lytic lesions – bisphosphonates and denosumab (risk of MRONJ).

Patient Management

- Preoperative CBC, coagulation studies, and tests specific for the disorders.
- Consult with hematologist.
- Type and Screen/Type and Cross in case of need for blood product replacement.
- Transfuse preoperatively if necessary. Remember one unit of PRBC increases hemoglobin by 1 g/dL and hematocrit by 3%. One unit of platelets increases the platelet count by 5000–10,000/microliters (50,000 is the minimum count accepted for most surgery).
- Maximize hydration to prevent crises in sickle cell anemia and to decrease hemolysis.
- Perioperative and postoperative antibiotics for patients with leukemia.
- In lymphoma/leukemia, blood products should be given with caution as concern for graft versus host disease.
- In lymphoma/leukemia, dexamethasone may precipitate tumor lysis syndrome.
- Consider risk of MRONJ in multiple myeloma patients that have taken bisphosphonates or other anti-resorptive drugs that are implicated in MRONJ.
- Preoxygenation is paramount as volatile anesthetics and hypothermia can shift the oxyhemoglobin curve to the left, which decreases oxygen tissue delivery.
- Patients with vWD may require DDAVP or Humate P (factor VIII and vWF concentrate) preoperatively to prevent bleeding.
- Patients with hemophilia may require factor VIII or IX replacement prior to surgery. Also, can use FFP for emergent cases or as an adjunct.
- Due to bleeding concerns consider avoiding administering an inferior alveolar nerve block and avoid floor of mouth injections to avoid hematoma formation around airway structures. Postoperative observation should be extended in addition to monitor bleeding and airway compromise. Adjunctive hemostatic agents should be employed.
- DVT/PE prophylaxis with anticoagulants (heparin/lovenox) and sequential compression devices is indicated for patients with prothrombotic disorders.
- Patients with hemophilia are low risk for a thromboembolic event. However, in the setting of patients who are undergoing surgeries that will limit their mobility are considered higher risk due to infusion of procoagulants.

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Connective Tissue Disorders

17

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Systemic Lupus Erythematosus (SLE)

- A multisystem chronic inflammatory disorder characterized by autoantibody production responsible for immune-mediated tissue damage.
- SLE is mostly seen in women of child-bearing age with the African American, Asian, and Hispanic demographic having the higher prevalence. The clinical variance in presentation and expression of the disease differs dramatically between patients. Most patients with SLE will experience remissions and exacerbations.
- The most common symptoms are constitutional symptoms (fever, fatigue, weight gain), arthralgias, pancytopenia, photosensitive rash (e.g., malar rash/butterfly rash), and serositis.
- Arthritis and arthralgias occur in 90% of SLE patients during the course of their disease. It may vary as mild joint pain to deformities. It usually presents as a symmetric inflammatory arthritis of the knees, wrist, or small joints of the hand.
- Jaccoud-like arthropathy initially resembles the ones observed in rheumatoid arthritis but, classically, it is "reversible." It is characterized by subluxation of metacarpophalangeal joints, "swan-neck" and Boutonniere deformities. Hyperextension at the interphalangeal joint of the thumb may also be present. The absence of erosions on radiography distinguishes it from those of RA. Patients do not tend to have prolonged morning stiffness.
- It should be noted that procainamide (antiarrhythmic) is known to cause a drug-induced SLE that will resolve weeks after discontinuation.

Multisystem Manifestations of SLE

- Renal – Renal involvement is found in the majority of SLE patients with about half of these having a significant nephritis. The most common nephropathy is immune complex-mediated glomerulonephritis, which may result in hypoalbuminemia. Oliguric renal failure may result as GFR is reduced.
- Pulmonary – Pleuritis is the most common manifestation of SLE and occurs in up to 30% of patients sometime in the course of their disease [1]. Pleural effusions, pulmonary arterial hypertension, and chronic interstitial lung

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disease, although less common, may be seen. Pulmonary function tests will show a restrictive lung pattern. A chest X-ray for evaluation of pulmonary involvement is warranted in this patient population.

- Cardiac:
  - Pericarditis occurs in 50% of patients with SLE, although usually small and asymptomatic, found mostly at time of autopsy or coincidental finding on imaging [2]. It may manifest itself as substernal chest pain with relief of symptoms while bending forward and exacerbated on inspiration.
  - Libman-Sacks endocarditis (aka verrucous endocarditis) has been recognized as a characteristic valvular abnormality in SLE. The pea size granular lesions normally involve the posterior leaflet of the mitral valve, although all four valves may be involved. The lesion may spread to the endocardium causing regurgitation. Advanced atherosclerosis is prevalent in SLE patients.
  - Patients are more prone to cardiac arrhythmias.
  - Hydroxychloroquine is known to cause a prolonged QT interval.
- Maxillofacial – The cutaneous and mucous membranes are commonly affected. The oral cavity may have lichenoid lesions, nondescript erythematous patches, or granulomatous lesions. These lesions commonly are found on the buccal mucosa or the hard palate. Involvement of the lower lip may show lupus cheilitis, which involves the vermillion of the lower lip. The characteristic butterfly facial rash with nasolabial fold sparing is the classic rash of SLE (seen in up to 60% of patients) [1].
- Hematology – All three blood cell lines can be affected. Anemia of chronic disease is commonly seen, and it is a normochromic, normocytic anemia with low serum iron and lower transferrin. Hemolytic and aplastic anemia may be present. Leukopenia and thrombocytopenia are seen in about half of SLE patients. Patients with SLE are at a higher risk of thromboembolism due to the presence of antiphospholipid antibodies/syndrome.
- Neuropsychiatric – Patients have neuropsychiatric manifestations as a result of inflammation. Manifestations include an acute confusional state, seizures, mood disorders, psychosis, aseptic meningitis, optic neuritis, and central and peripheral neuropathies.

Diagnosis

- Diagnosis is based on a constellation of symptoms and not serological testing alone. It should be noted that more than 50% of SLE patients will not have met four criteria at one time, but will have during the course of their disease. ANA is the most common specific antibody found in SLE, but anti-DSDNA and anti-Sm are more specific. The American College of Rheumatology requires four or more of the criteria met and exclusion of other reasonable diagnosis [3].
- DOPAMINE RASH is a mnemonic for SLE.
  - *D*iscoid rash
  - *O*ral ulcers
  - *P*hotosensitivity
  - *A*rthritis > 2 joints
  - *M*alar rash
  - *I*mmunologic criteria: anti-Sm Ab, anti-DSDNA
  - *N*eurologic symptoms: seizures, psychosis
  - *E*SR elevated
  - *R*enal disease
  - *A*NA positive
  - *S*erositis: pleurisy, pericarditis
  - *H*ematologic disorders: hemolytic anemia, leukopenia, thrombocytopenia

Treatment

- NSAIDs can often control arthritis and serositis. Patients are to avoid sun exposure due to photosensitivity.
- Corticosteroids should be limited to treat acute or subacute symptomatology but are effective at managing thrombocytopenia and hemolytic anemia as well as suppressing glomerulonephritis and cardiovascular abnormal-

ities. Topical steroids can be used to treat cutaneous and mucosal lesions. Hypoadrenalism and emotional disturbances can be major side effects. Heavy use of corticosteroids in treating SLE patients may lead to early-onset osteoporosis.

- Antimalarial drugs such as hydroxychloroquine (Plaquenil®) are used long term to control disease flare-ups. Long-term use, greater than 400 mg daily, can lead to eye toxicity and should be monitored every 6–12 months.
- Severe disease symptoms require immunosuppressant agents such as alkylating agents (e.g., cyclophosphamide), purine synthesis inhibitors (e.g., azathioprine), and disease-modifying agents such as methotrexate (folate agonist). Consider biologic agents (e.g., rituximab, belimumab) for refractory cases.

Patient Management

- Consult with patient's rheumatologist.
- Elective surgery should be deferred until flare-ups have resolved.
- Preoperative laboratories: CBC, aPTT, and BMP.
  - Rule out anemia, thrombocytopenia, coagulopathies, renal compromise, and electrolyte disturbances.
- EKG to rule out ischemic heart disease and rhythm disturbances.
- Auscultation to assess for cardiac murmurs and pulmonary involvement.
- Consider supplemental antibiotics in the setting of immunosuppression [3].
- Consider steroid supplementation for adrenal suppression.
- Avoid nephrotoxic drugs. Also, maintain urine output by avoiding hypotension and hypoperfusion.
- Airway considerations include mild laryngeal inflammation, vocal cord paralysis, subglottic stenosis, and laryngeal edema with acute obstruction may be seen in SLE which may make intubation and extubation difficult [3].
- Patients may be anticoagulated due to hypercoagulable state and should be managed appropriately.

Rheumatoid Arthritis (RA)

- A chronic systemic inflammatory disease of unknown etiology characterized by inflammatory polyarthritis (synovitis) with progressive destruction of joint, bone, and articular cartilage.
- RA affects females more than males (3:1).
- It usually affects patients in the fifth to sixth decade of life.
- It is a symmetrical polyarthropathy and the joints involved tend to be warm to the touch. Morning stiffness and swelling of the joints involved over 1 hour are hallmark signs of RA.
- Classic features are ulnar deviation and subluxation of the metacarpophalangeal joints, the swan-neck deformity (hyperextension of the proximal interphalangeal joint), and Boutonnière (hyperflexion proximal interphalangeal joint) deformities (See Fig. 17.1). This differs from osteoarthritis, which involves weight-bearing joints. Other major joints normally involved are the wrists, knees, elbows, ankles, and hips.
- Rheumatoid nodules are present in 1/5th of patients with RA. They are non-painful, firm, and normally present on the extensor surface of the forearms, but can be anywhere.
- The cervical spine is commonly involved, most often a C1–C2 (atlantoaxial) subluxation. When separation is severe, the odontoid process can exert pressure on the spinal cord and impair blood flow through the vertebral arteries. The deformity can be easily observed on a lateral plain film of the cervical spine of which up to of 3 mm or more gap can be seen between the anterior margin of the odontoid process from the posterior margin of the anterior arch of the atlas. Limitation of neck extension should be practiced with SLE patients to demonstrate

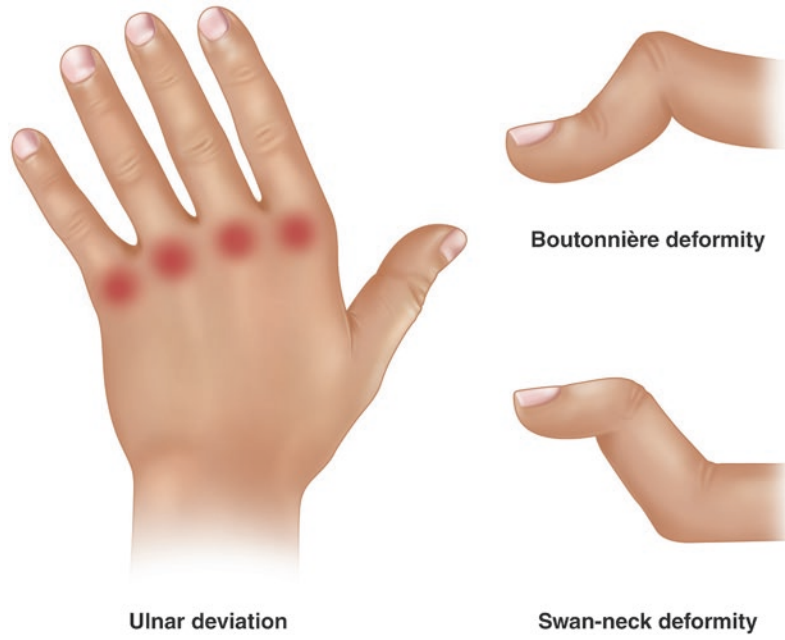

Fig. 17.1 Hand deformities seen in RA. (Modified from Rahman and Giles [17])

range of motion to avoid damage to the spinal cord.

Multisystem Manifestations of RA

- Cardiovascular – In the cardiovascular system, pericarditis, myocarditis, CAD, aortitis with aortic dilatation leading to a possible aortic regurgitation and advanced atherosclerosis may be present.
- Ocular – Eye pain and redness due to keratitis, scleritis, and episcleritis; foreign body and dryness sensation with keratoconjunctivitis and sicca. If severe enough, visual acuity can be compromised.
- Neurological – Neuropathy secondary to deposition of complexes into the vaso nervosum.
- Pulmonary – In the pulmonary system, pleural effusions and rheumatoid nodules may develop. If the costochondral joints are involved, this may create a restrictive lung disease pattern due to restricted wall movement. Preoperative pulmonary function studies may be indicated for those with advanced disease.
- Hematological – Anemia of chronic disease may be seen. Felty's syndrome (RA with splenomegaly and leukopenia) may occur. Small vessel vasculitis may manifest as digital infarcts and represent a poorly controlled state of the disease.

Diagnosis

- Not based solely on lab results. Common lab findings include rheumatoid factor titers increased, elevated erythrocyte sedimentation rate, and elevated C-reactive protein.
- Diagnostic Criteria for Rheumatoid Arthritis (symptoms must be >6 weeks & 4/7 criteria must be met):
  - Morning stiffness (≥1 hour).
  - Swelling of 3+ joints.
  - Swelling of hand joints.
  - Symmetrical swelling.
  - Subcutaneous nodules.
  - Serum rheumatoid factor.
  - Radiographic evidence of erosive arthritis.

Treatment

- Treatment of the disease may include anti-inflammatory drugs like corticosteroids (e.g.,

prednisone) to decrease swelling and stiffness. Long-term use can lead to osteoporosis, poor wound healing, adrenal suppression, and hyperglycemia.
- Disease-modifying antirheumatic drugs (DMARDs) are used to slow and halt progression of the disease. They include methotrexate, hydroxychloroquine (Plaquenil®), antimalarials, minocycline, and tofacitinib (Xelijanz®).
- The preferred medication methotrexate acts by inhibiting metabolism of folic acid via dihydrofolate reductase. Close monitoring is required to monitor for liver dysfunction and bone marrow suppression.
- Hydroxychloroquine requires yearly eye exams to rule out macular toxicity.
- Monoclonal antibody biologics – entanercept (Enbrel®), infliximab (Remicade®), and Adalimumab (Humira®) work by inhibiting TNF-α.

Patient Management

- Preoperative laboratories: CBC and BMP.
  - Rule out anemia, thrombocytopenia, renal compromise, and electrolyte disturbances.
- EKG to rule out ischemic heart disease.
- Cardiopulmonary exam to assess for cardiac murmurs and pulmonary involvement.
- Consider PFTs if there is presence of restrictive lung disease.
- Consider supplemental antibiotics in the setting of immunosuppression.
- Consider steroid supplementation for adrenal suppression.
- Avoid nephrotoxic drugs.
- Intubation considerations:
  - Hoarseness or stridor in this patient group may be a sign of cricoarytenoid joint involvement, which limits glottic opening making intubation difficult.
  - Additionally, TMJ involvement with limitation of opening should be identified before any anesthesia delivered.
  - Flexion deformity of the cervical spine may make intubation difficult due to difficulty with extension.
  - Atlantoaxial subluxation should be confirmed preoperatively with a radiograph. When present, care must be taken to when extending the neck during laryngoscopy to prevent spinal cord damage.

Osteoarthritis (OA)

- A painful degenerative process involving progressive deterioration of articular cartilage and remodeling of subchondral bone that is not primarily inflammatory.
- OA effects females to male 2:1 and occurs predominately later in life at ages 50 and above [4].
- Risk factors are age, female gender (especially post-menopausal), obesity, smoking, trauma from physical activity, occupation of physical nature, and muscle weakness.
- The hallmark of osteoarthritis is damage to articular cartilage. Cartilage normally distributes loads across joint surfaces to aid in smooth movement.
- Changes in the cartilage progresses through roughness, fibrillation, fissuring, and eventual erosion of the cartilage. The erosive process activates chondrocytes to release proteolytic enzymes causing further degeneration. The unprotected bone underlying the articular cartilage (subchondral bone) results in thinning (attrition) leading to subchondral cysts early on and later to subchondral sclerosis as bone formation is outpaced by bone resorption.
- The large weight-bearing joints (knees and hips) are the most commonly affected.
- Symptoms are prominently aching and pain that is worse with function. Morning pain is normally brief (less than 30 minutes). Joint instability, locking, sensation of grinding, and limited range of motion are common findings.
- Small joints of the hands are the most commonly affected. Osteophytes of the distal interphalangeal joints (Heberden nodes) or the proximal interphalangeal joints (Bouchard's nodes) can lead to visible and palpable nodes.

- Common radiographic findings are joint space narrowing, subchondral sclerosis, and osteophytes.

Treatment

- Treatment is mostly symptomatic.
- Ambulatory aids such as canes/walkers or motorized carts can allow patients to retain independence if the lower extremities are involved.
- Physical therapy and exercise programs can aid in range of motion and decrease pain.
- Most commonly used analgesics are NSAIDs or acetaminophen.
- Nutritional supplementation such as glucosamine and chondroitin are unproven and neither has been shown to reduce pain.
- Neuroreactive agents such as SNRIs have shown positive results.
- Intra-articular injections with glucocorticoids, platelet-rich plasma, or hyaluronic acid derivatives may be used if conservative treatment modalities are ineffective.
- Surgery is indicated for patients that do not respond to conservative therapy and the disease impacts their quality of life. Surgical procedures include arthroscopy, arthroplasty, and joint replacement may be indicated for severe disease.

Patient Management

- NSAIDs are commonly prescribed. May have higher risk of gastric ulcers and platelet suppression.
- Total joint replacement is common in this population. Consult with orthopedic surgeon for recommendation of prophylaxis.
- TMJ may be involved making airway management difficult.

Obesity

- An abnormally high amount of adipose tissue compared with lean muscle mass with an excess body weight >20% over the predicted ideal body weight. A common measurement used to categorize (Table 17.1) weight is body mass index (BMI). BMI is calculated by:

Table 17.1 Body mass index

Category	BMI (kg/m^2)	Obesity class
Underweight	<18.5	
Normal weight	18.5–24.9	
Overweight	25.0–29.9	
Obesity	30.0–34.9	Obesity class I
	35.0–39.9	Obesity class II
Extreme obesity	≥40	Obesity class III

$$\text{Body mass index}\left(\text{BMI}\right) = \text{body weight}\left(\text{kg}\right) / \text{height}\left(\text{m}^2\right)$$

- Obese patients have a higher incidence of cardiovascular disease, respiratory disease, and diabetes. They have depressed immune function, which leads to high perioperative infection rates and cancers. Thromboembolic events are twice that of non-obese patients.
- Cardiovascular implications:
  - Obesity-induced cardiomyopathy is due to volume overload and vascular stiffness, which causes progressive heart failure.
  - Hypertension is seen six times more often than in lean patients (uncontrolled hypertension can lead to heart failure and pulmonary hypertension).
  - Higher risk of hyperlipidemia in obese patients.
- Pulmonary implications:
  - An extrinsic restrictive pattern is seen which causes a decreased functional residual capacity. If severe enough, this can lead to right to left pulmonary shunting due to increased closing volumes.
  - Rapid oxygen desaturation can follow induction.
  - There is also increased oxygen consumption due to metabolic activity of excess adipose.
- Obstructive sleep apnea is common with features making masking the patient difficult. A long-term consequence of OSA is a condition called obesity hypoventilation syndrome

(Pickwickian syndrome), which is characterized by episodic central apnea events due to desensitization of hypercarbia. Even light sedation can cause airway/respiratory collapse.
- Gastrointestinal/hepatobiliary implications:
  - Due to higher residual gastric volumes and lower pH, they are at higher risk of aspiration events.
  - Non-alcoholic fatty liver disease.
  - Cholelithiasis.
  - Higher rate of colon and hepatobiliary cancers.
- Musculoskeletal implications – Osteoarthritis due to overloading/degeneration of weight bearing joints.
- Endocrine implications – Patients are at a higher risk of diabetes mellitus.

Treatment

- Lifestyle modifications including diet and exercise are the first line of treatment.
- Adjuvant pharmacotherapy may be considered for patients with a BMI greater than 30 kg/m^2 or a BMI of 27–30 kg/m^2 with concomitant weight-related complications.
- Pharmacological therapy may include serotonin, norepinephrine, and dopamine reuptake inhibitors. These drugs are known to increase blood pressure, heart rate, and pulmonary hypertension. Clinicians must be cautious of serotonin syndrome.
- Fenfluramine/phentermine aka "fen-phen" has been off the market since 2009. Patients with a history of fen-phen use should be worked up by a cardiologist to rule out valvulopathy.
- Surgical treatment of obesity is recommended with a BMI of 40 kg/m^2 or patients with a BMI of 35 kg/m^2 whom have significant comorbidities.
- Surgical interventions include the following:
  - Restrictive therapies such as gastric banding or sleeve gastrectomy, with a goal of gastric pouch of 10–12 mm in diameter.
  - Malabsorptive treatment such as bilipancreatic diversion or duodenal switch which creates a gastric pouch of 200–250 cc in volume and bypassing a portion of the small intestine creating a gastroileostomy.
  - The roux-en-Y-gastric bypass is both a malabsorptive and restrictive treatment. A gastric pouch is connected to a roux limb (intestinal segments accepting food) to the jejunum.
  - It should be noted patients whom have had these procedures are advised to avoid NSAIDs.

Patient Management

- Post-malabsorptive or combined treatments, deficiencies of Vit B12, iron, and vitamin K are common which could lead to anemia and coagulopathy.
- Patients may also develop dumping syndrome and postoperative diet should be monitored, especially for sugars.
- Protein-calorie malnutrition can lead to hypoalbuminemia, which may result in marked fluid shift such as into lungs and soft tissues. Close fluid monitoring or replacement is paramount.
- Anesthetic drug administration should be closely evaluated, as it is difficult to predict, due to large volume of distribution and increased total blood volume. Drugs should be injected based on the lean body weight (ideal body weight + 20%) versus the total body weight.
- Many anesthetic drugs such as propofol, benzodiazepines, and narcotics tend to be lipophilic and may have a reduced length of effect.
- Remifentanil acts similarly in obese and non-obese patients.
- Venipuncture tends to be difficult on these patients, and the patient should be asked for previous blood draw sites.
- Blood pressure cuffs should be appropriately sized with the cuff bladder 75% of the arm circumference.
- Pre-oxygenation with 100% oxygen for 3–5 minutes prior to anesthesia induction is key in these patients as they have minimal reserve.

- Use of nitrous oxide is discouraged especially in setting of pulmonary hypertension.
- Laryngoscopy may be difficult to navigate due to excessive adipose of the oral and parapharyngeal tissues.
- If planning for intubation, the head-elevated laryngoscopy position (HELP) or an inflatable rapid airway management positioner can aid in aligning airway.
- It must be kept in mind that obesity places a patient as ASA class III, and the patient may not be an appropriate candidate for office-based anesthesia.

Muscular Dystrophy

- Group of inheritable myopathies causing progressive death of muscles fibers.
- Commonly due to breakdown of dystrophin-glycoprotein complex [5].
- Symmetric wasting of muscles without skeletal muscle denervation.

Duchenne Muscular Dystrophy (Pseudohypertrophic Muscular Dystrophy)

- Most common neuromuscular disease.
- X-linked recessive pattern inheritance, incidence 1 in 3600 boys [6].
- Mutation in the dystrophin gene leads to a loss of the mechanical link between the sarcomere and sarcolemma leading to progressive necrosis of the muscle fibers.
- Presents in early life with delayed and impaired motor skill development, learning disabilities, and attention deficits [3, 4].
- Progressive weakening leads to these patients being wheelchair bound early in life around age 12 [6].
- Commonly the life span of these patients is 20 years of age with causes of death most commonly due to respiratory failure, heart failure, arrhythmias, or pneumonia.
- Clinical Features:
  - Calf muscle pseudohypertrophy and thigh muscle wasting. Calf false enlargement due to irregular muscle formation with replacement with scar tissue, fat, and connective tissue.
  - Tongue commonly also affected with pseudohypertrophy, which can add to respiratory difficulty.
  - Increased susceptibility to long bone fractures.
  - Kyphoscoliosis of the spine leads to reduction in respiratory function.
  - Joint contractures are seen in the ankles, knees, hips, and elbows.
  - Weakness of pharyngeal musculature leads to aspiration, nasal regurgitation, and speech with nasal quality [6].
  - Sleep-disordered breathing common.
  - Cardiac:
    Mitral regurgitation due to papillary muscle dysfunction [5].
    Tachycardia with tall R waves.
    Prone to arrhythmia development (secondary to cardiac muscle fibrosis).

Laboratory Findings:

- Serum CK levels greatly elevated (commonly 100× normal).
- EMG will demonstrate myopathic features.
- Muscle biopsy is diagnostic with atrophy and hypertrophic muscles strands with inflammatory infiltrates. Increased connective tissue between muscle strands.
- Immunostaining will show absence of dystrophin.
- DNA testing is confirmatory with analysis of dystrophin gene.

Becker Muscular Dystrophy

- Milder form of Duchenne dystrophy.
- X-linked recessive disease affecting the dystrophin gene [7].
- More rare with incidence of 1:18,000 male births [6].

- Presents later in life (>5 years of age).
- Learning disabilities less common.
- Features symmetrical proximal weakness with calf pseudohypertrophy [8].
- CK elevation is not as pronounced as in Duchenne dystrophy.
- Steroids not commonly used.
- Lifespan of these patients are into their 40s [7].
- Heart failure and arrhythmias occur later in life.

Myotonic Muscular Dystrophy

- Second most inherited muscle disease, 1:8000 [6].
- Striated and smooth muscles are affected.
- Autosomal dominant inheritance include two types:
  - Type 1 – affects mostly facial, oropharyngeal, forearm, and foot dorsiflexors.
  - Type 2 – peripheral weakness mainly.
- Commonly see muscle pain and weakness.
- Clinical Features:
  - Frontal balding.
  - Ptosis.
  - Temporal and masseter wasting.
  - Speech is hypernasal and slurred.
  - Intellectual impairment.
  - Myotonia – slow relaxation after muscle activation involuntary or not.
  - Peristalsis is slow and patients often have dysphagia. Constipation is common and encopresis may occur due to anal sphincter weakness.
  - Cardiac dysrhythmias common and progressive. Heart blocks are commonly seen. Can lead to sudden death in patients.
  - Endocrine dysfunction common: hypothyroidism, adrenocortical insufficiency, diabetes mellitus, and testicular atrophy (leading to testosterone deficiencies and male infertility).
  - Subcapsular lens cataracts.
  - Central sleep apnea.

Patient Management

- Patients with Duchenne muscular dystrophy are often managed with high-dose steroids. Steroids are given daily to reduce muscle necrosis and prolong ambulation. This can lead to osteoporosis and weight gain. Long-term steroid use may also require supplementation due to adrenal insufficiency prior to surgical procedures.
- Patients may be placed on ACE inhibitors and β-blocker to reduce myocardial fibrosis and reduce oxygenic demands on heart, respectively.
- Digoxin may be prescribed in cases of cardiac failure, concern for digitalis toxicity, and hypokalemia.
  - Digoxin toxicity early signs are nausea, vomiting, and visual disturbances (yellowing or blurred vision). EKG will show classic "reverse check" ST segments. Treatment with digoxin-specific antibody.
- Preoperative EKG and chest X-ray to evaluate for heart failure and conduction abnormalities.
- Cardiac pacemaker may be present in patients with conduction abnormalities. Electrocautery may interfere with device and requires deactivation prior to surgery. Placement of prophylactic defibrillator/pacer pads.
- Cardiomyopathy expected (mild symptoms might be exaggerated by volatile anesthetics depressant effects). Desfluroane is the agent of choice [9]. Consider total IV anesthesia (TIVA) to reduce risk.
- Succinylcholine is contraindicated due to risk of rhabdomyolysis and hyperkalemia. Non-depolarizing agent should be chosen such as Rocuronium.
- High risk for malignant hyperthermia, most commonly from halothane inhalation and succinylcholine administration.
- Delayed gastric emptying in these patients. Consider longer NPO protocol. Consider H_2-agonist and metoclopramide prior to induction [9].
- Concern for pulmonary aspiration susceptibility due to pharyngeal muscle weakness.

- TMJ has higher prevalence of subluxating on intubation.
- Decreased pulmonary reserve and respiratory muscle weakness. Patients may require prolonged need for mechanical ventilation. Additionally, pulmonary insufficiency may occur 36 hours postoperatively [5]. May require ICU postoperative monitoring.
- Sleep-disordered breathing common, postoperative CPAP/BiPAP should be available.
- Limit/avoid narcotics to reduce anti-respiratory effect for pain management.

Myasthenia Gravis

- Autoimmune disorder affecting postsynaptic neuromuscular transmission of the nicotinic ACH receptor due to the production of autoantibodies. It is characterized by weakness and fatigue of the skeletal muscles with improvement with rest.
- Female more affected than males 3:2.
- Clinical Features:
  - Fatigue of the voluntary muscles, most commonly eyelids (ptosis) and extra-ocular muscles. Will see furrowed brow to compensate for eye weakness.
  - 85% of patients will go on to develop generalized weakness [10].
  - Thymoma present in 10–15% of patients and 2/3rd will have thymic hyperplasia [7, 8].
  - Speech is slowed, nasal, and slurred (dysarthria).
  - Jaw claudication – progressive difficulty with chewing.
  - Face appears expressionless with a straightened smile.
  - Respiratory weakness that may lead to failure.
  - Bulbar involvement manifested as chewing and swallowing difficulty.
  - Peek sign – patient closes eyes and after a short time scleral show begins due to orbicularis oculi weakness.
  - Myasthenic crisis – acute exacerbation of symptoms with respiratory compromise.

Diagnosis

- Anti-acetylcholine receptor antibody test is the confirmatory diagnostic test of choice.
- Edrophonium chloride (Tensilon) test. Patient is injected with anticholinesterase with improved strength. Often diagnostic in patients with ptosis or ophthalmoparesis.

Treatment

- Pyridostigmine (anticholinesterase) first line of treatment.
- Immunosuppressants such as prednisone, cyclosporine, fujimycin (Tacrolimus®), and IV immunoglobulin may be provided to attenuate immune destruction of acetylcholine receptors.
- Plasmapheresis indicated in those without improvement or refractory to other treatments.

Patient Management

- Consider reduced or avoidance of sedatives or opioids in those with reduced respiratory reserve. Short-acting opioids such as remifentanil are preferred.
- Warn patient of possible need for prolonged ventilatory support.
- Avoid neuromuscular blocking drugs if possible, especially long acting, due to variable response and treatment with anticholinesterases. Usually resistant to succinylcholine and sensitive to non-depolarizing muscle relaxants.

- Rapid sequence intubation, succinylcholine should be adjusted to 1.5–2 mg/kg [7, 8].
- Nitrous oxide is safe [11].
- Avoid aminoglycosides (e.g., gentamycin) as they are known to impair neuromuscular transmission. Erythromycin, azithromycin, fluoroquinolones, and tetracycline have reports of increased myasthenic weakness [11].
- Inhalational agents such as sevoflurane, isoflurane, and halothane are known to reduce neuromuscular transmission and may be exacerbated in the myasthenic patient.
- Reports of adverse effects with ketamine and etomidate [10].
- Propofol is safe in these patients.
- Corticosteroids cause increased need for succinylcholine but increased sensitivity to non-depolarizing agents [10].
- Controversial on reversal of neuromuscular blockage due to concern postoperative weakness due to cholinergic crisis or inadequate muscular transmission [10].
- Cholinergic crisis, excess of acetylcholine. Normally due to administration of excess anticholinesterase drugs resulting in involuntary twitches and fasciculation and muscle weakness. Differentiate from myasthenic crisis by administration of the edrophonium test. In myasthenic crisis, pupils will be dilated as well.

Ehlers-Danlos Syndrome (EDS)

Ehlers-Danlos syndrome – a group of inheritable diseases that result from a defect in synthesis or structure of fibrillar collagen leading to changes in skin, tendons, blood vessels, and viscera.

- Multiple types with the transmission being autosomal dominant for most forms.
- 80% of cases fall under the classical and hypermobile (most frequent) types [12]. There are six major subtypes based on the Villefranche nosology.
- Vascular EDS subtype is life threatening due to spontaneous rupture of small and large arteries.

Classical Features:
Seen in both classical and hypermobile subtypes [12]:

- Joint hyper mobility in both large and small joints with common subluxation. May result in double-jointed fingers.
- Skin hyperelasticity/hyperextensibility.
- Gorlin sign – ability to touch tongue to tip of nose, 5× more common in EDS patients.
- Abnormal scarring.
- Extremely soft skin.
- Easily bruised.
- Hernias are common.

Patient Management

- Skin tape placement should be minimized and removed carefully due to skin fragility.
- Individuals can be resistant to local anesthetic, most commonly seen in hypermobility subtype.
- Be cognizant of TMJ subluxation during intubation.
- Higher risk of occipitoatlantoaxial instability in this population.
- Reduce airway pressures due to pneumothorax risk.
- Postural orthostatic tachycardia syndrome prevalent in hypermobile variant, ensure adequate hydration.
- Consider avoiding NSAIDs for pain control due to bruising risk.
- Cardiac evaluation to monitor risk of aortic root dilatation and mitral valve prolapse propensity, usually non-significant.
- Commonly have dental issues with poor periodontal health due to fragility of periodontium, especially in vascular subtype.
- Subtypes with vascular fragility should have type and cross match.

- 26% of patients have platelet aggregation defects. Consider discussion with hematologist for plan of action (commonly desmopressin) [13]. Additionally, use of prophylactic tranexamic acid should be considered.

Marfan's Syndrome

Marfan's syndrome – an autosomal dominant connective tissue disorder affecting cardiac, skeletal, and ocular tissue.

- Mutation in extracellular matrix protein fibrillin-1, leading to structural deformities in connective tissue.
- Autosomal dominant inheritance.
- Diagnosis requires effort between PCP, cardiologists, ophthalmologist, geneticist, and orthopedist based on the Ghent criteria.

Clinical Features (Table 17.2) [14, 15]:

Therapies

- β-blocker – reduced HR <100 BPM has been shown to be protective for aortic root growth. Non-selective β- and α-blockers help reduce HR and BP reducing hemodynamic stress.
- Angiotensin II receptor blockers may also be used for protection against aortic root dilatation from hemodynamic stressors.
- ACE inhibitors reduce volume overload secondary to valvular dysfunction.
- Lifestyle changes – avoid stimulants (e.g., caffeine), breathing against resistance (e.g., wind instruments), and contact sports.

Patient Management

- AHA does not recommend antibiotic prophylaxis.
- Cardiology consult to evaluate for valvular function and aortic root dilatation size. Need to be aware of signs for congestive heart fail-

Table 17.2 Clinical features of Marfan's disease

Skeletal	Cardiac	Ocular	Facial	Other
Dolichostenomelia – overgrowth of long bones. (Patients tend to be tall and thin) Pectus carinatum (outward displacement of ribs) or pectus excavatum (inward displacement ribs) Thoracolumbar scoliosis Protusio acetabuli – inward bulge of acetabulum of the hip Long fingers compared to palm (arachnodactyly) Joint hypermobility. ***Steinberg sign (Thumb sign)*** *– Distal phalanx and thumbnail extend beyond the ulnar side of the hand when fingers are folded over the top of the thumb* ***Walker-Murdoch sign (Wrist sign)****: thumb and fifth finger nail beds overlap when encircling the wrist*	Atrioventricular valve thickening leading to valvular prolapse Aortic dissection/ aneurysm due to weakened arterial media Mitral valve prolapse	Ectopia lentis Severe myopia Hypoplastic iris Predisposition to retinal detachment and early-onset glaucoma Antimongoloid slants	Dolicocephaly Enophthalmos Retrognathia Micrognathia Malar hypoplasia High-arched palate	Striae atrophicae Decrease lung sounds due to apical blebs Risk of sleep apnea Dural ectasia Scoliosis leads to reduce total lung capacity which results in a restrictive lung disease pattern Spontaneous pneumothoraces may occur secondary to bleb rupture Hernias due to defects in the abdominal wall

ure. Monitor EKG. Consider echocardiogram for patients with known aortic involvement.
- Control of blood pressure to reduce shear forces on vessels. Perioperative β-blocker should be continued.
- Replenish fluids to maintain blood volume and forward flow.
- Pulmonary function should be performed to evaluate for restrictive lung disease (higher risk in patient with scoliosis). Chest X-ray should be taken for evaluation of pulmonary blebs and pneumothoraces.
- Ensure patient positioning does not allow for laxed joints to become traumatized. Ensure that TMJ dislocation does not occur during laryngoscopy.
- Airway may be difficult due to propensity for high palatal arch, TMJ subluxation, retrognathia, tracheomalacia, and malar hypoplasia [16].
- Ventilatory pressures should be reduced to prevent barotrauma.
- Consider pulmonary function tests in patients with severe thoracic skeletal abnormalities.

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Endocrine Disease

18

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Thyroid Disease

Thyroid Physiology

- Thyroid gland produces, stores, and releases thyroid hormones, thyroxine (T3) and triiodothyronine (T4), whose main purpose is to regulate the body's physiological functions.
- The thyroid gland is constructed of thyroid follicles, each filled with the protein thyroglobulin.
- The thyroid hormones are attached to thyroglobulin protein and stored as colloid in the gland, and T3 and T4 are stored in a ratio of 15:1, respectively.
- The release of thyroid hormones is regulated by thyroid stimulating hormone (TSH), an anterior pituitary hormone, which is produced in the hypothalamus.
- Release of the thyroid hormones is achieved through proteolysis from thyroglobulin and eventual diffusion into the circulation.
- Once released from the gland, majority of T3 and T4 are bound to various carrier proteins such as thyroid-binding globulin (TBG).
- T3 is mostly produced by conversion from T4 in liver and kidney; 80% of T3 is produced in this manner by deiodination of T4 by the converting enzyme deiodinase (I, II, or III), whereas only 20% is produced directly by secretion from thyroid gland.
- T3 is much more potent than T4, due to its higher receptor affinity at various tissue receptors, and thus mediates most of the body's systemic effects. However, only the unbound form of T3 is active [1–3].

Thyroid Testing

- The standard testing for thyroid function includes the measurement of T3, T4, and TSH levels.
- The T4 assay, also known as thyroxine test, is elevated in 90% of patients with hyperthyroidism, and depressed in 85% of patients with hypothyroidism.
- The T3 test checks for levels of triiodothyronine and an abnormally high level usually indicates Graves' disease.
- T3 is used predominantly to detect hyperthyroidism if T4 is measured to be normal, as T3 may be the only hormone in excess. T3 however can be low due to various factors that impair the peripheral conversion of T4 to T3 such as seen in euthyroid sick syndrome (ESS).

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- TSH test is the gold standard for the diagnosis and treatment of thyroid dysfunction and is most useful in detecting hypothyroidism. The normal TSH level is between 0.4 and 4.0 mIU/L. Often in primary hypothyroidism, levels are greater than 20 mIU/L. During the treatment of hypothyroidism, the goal of thyroid replacement therapy is to normalize TSH levels [1].

Hyperthyroidism

- Hyperthyroidism is a condition in which the thyroid gland is overactive and exposes the body tissues to excessive amounts of thyroid hormone. The most common etiologies are multinodular diffuse goiter and Graves' disease.
- Manifestation of hyperthyroidism includes warm skin, sweating, increased ventricular contractility, tachycardia, elevated systolic blood pressure, weight loss despite hyperphagia, diarrhea, palpitations, emotional lability, skeletal muscle weakness, restlessness, and heat intolerance. Graves' disease patients might also exhibit exophthalmos from increased volume of retro-orbital fat [1, 2].

Graves' Disease

- Autoimmune condition, which results in hyperstimulation of TSH receptors from autoantibodies produced by the immune system.
- It is the most common cause of hyperthyroidism.
- The defining features of the disease, in addition to the usual manifestation of hyperthyroidism, include goiters, exophthalmos and pretibial myxedema. These last two signs are truly "diagnostic" of Graves' disease.
- Treatment of Graves' disease includes antithyroid drugs, which reduce the production of thyroid hormone. Other treatment modalities include radioiodine therapy and thyroidectomy.

Toxic Multinodular Goiter

- Also known as Plummer's disease, is a condition where the thyroid contains autonomously functioning thyroid nodules, with resulting hyperthyroidism.
- It is another common cause of thyrotoxicosis; however, symptoms associated with the disease are considered less severe than those with Graves' disease.
- It occurs most often in older adults, with risk especially high in females over 55 years old.
- It has typical symptoms of hyperthyroidism, but the exophthalmos and pretibial myxedema seen in Graves' do not occur.
- The disease is usually treated with beta-blockers such as propranolol, radioiodine therapy, and thyroidectomy.

Subacute Thyroiditis

- Also called granulomatous thyroiditis or De Quervain thyroiditis, is a less common variant of thyroiditis, which can cause hyperthyroidism in its initial stage, followed by a transient period of hypothyroidism.
- Etiology is thought to be from viral infections, especially ones affecting the ear, sinus, or throat. The viral infection induces tissue damage of the thyroid gland to release thyroid hormone into the circulation.
- The disease usually affects women aged 30–50 years.
- Since thyroiditis is a form of inflammation, the thyroid often presents with pain and discomfort in the thyroid, however the presentation can be insidious. Other symptoms include myalgia, fever, fatigue, malaise, palpitations, sweating, and heat intolerance.
- Laboratory work up includes TSH, T3, T4, and ESR. Patients will usually have suppressed TSH, elevated T4 and T3, and high ESR.
- During the initial phase of illness, T4 and T3 are elevated in almost all patients. In contrast to Graves, total T3:T4 ratio usually is less than

20, and radioiodine uptake in the acute stage of the disease is low [1, 4–6].

Treatment of Hyperthyroidism

- Therapy for most forms of hyperthyroidism include antithyroid drugs which include propylthiouracil and methimazole. These medications prevent production of T4 and conversion of T3 from T4.
- Beta-adrenergic antagonist can also be used to control symptoms of sympathetic activation such as palpitations, trembling, and anxiety until thyroid hormones level normalizes. Beta-adrenergic antagonists often obtain immediate temporary relief but do not treat hyperthyroidism or any of its long-term effects (e.g., propranolol).
- Radioactive iodine-131 is used as an ablative treatment and is given orally.
- Total thyroidectomy is rarely indicated except for thyroid adenomas or malignancy. It is uncommonly used for treatment of hyperthyroidism since most common forms of hyperthyroidism are very effectively treated with radioactive iodine [2].

Thyroid Storm

- An acute exacerbation of hyperthyroidism that is life threatening, and occurs most commonly in undiagnosed or undertreated hyperthyroid patient.
- Common triggers are stress of surgery or non-thyroid illness. During thyroid storm, patient will be induced into a hypermetabolic state caused by excessive release of thyroid hormones.
- Symptoms: dysrhythmias, myocardial ischemia, congestive heart failure, hyperthermia, shaking, change in consciousness, nausea, vomiting, diarrhea, and tachycardia. Heart failure and pulmonary edema can rapidly occur and cause death. An early sign is often very elevated systolic pressure and low diastolic pressure.
- Treatment: Less severe forms can be managed with supportive measures such as cooling blankets, IV fluids, electrolyte correction, EKG monitoring, oxygen supplementation, and medicine to manage agitation. Severe forms may require further medication with sodium iodide (blocks the release of stored thyroid hormone), propylthiouracil, hydrocortisone (prevents conversion of T4 to T3), and propranolol [1, 2, 4].

Patient Management

- Hyperthyroidism, if unmanaged, has a high risk for cardiac dysrhythmias or heart failure. Thus one of the primary goals in the management of patients with hyperthyroidism is to attempt to have the patient reach a euthyroid state with normalized thyroid levels prior to elective surgery. Preoperative assessment should aim for goals of resting heart rate of less than 85 bpm and normal thyroid function tests.
- In the case of emergency surgeries, beta-sympathetic antagonists can be used to attenuate sympathetic symptoms acutely and should be used in all hyperthyroid patients unless contraindicated. β-Adrenergic blockade such as propranolol given over 12–24 hours decreases sympathetic manifestations such as tachycardia, anxiety, heat intolerance, and tremor.
- Glucocorticoids should be administered to decrease hormone release and reduce the peripheral conversion of T4 to T3.
- Titrate doses on non-depolarizing muscle relaxers as there is a percentage of these patients that also have myasthenia gravis as a comorbidity.
- Avoid sympathomimetic agents such as ketamine, epinephrine, atropine, and ephedrine.
- Ocular protection with lubricants and tape in cases of ophthalmopathy.
- Monitor EKG for dysrhythmias.
- Postoperative period: Beta-sympathetic antagonist should be continued until the antithyroid drugs have taken effect. In the morning of surgery, all antithyroid medications should be

continued. Antithyroid drugs such as propylthiouracil and methimazole prevent the thyroid from producing excess amounts of the hormone. However it takes at least 8 weeks for the effect of medication to take place, and thus should be started several months prior to elective surgery.

Hypothyroidism

- Hypothyroidism (also known as myxedema) is a common condition where the thyroid gland has decreased production of thyroid hormone resulting in inadequate circulating levels of T4 or T3, or both. Disease development can be insidious and patients often have no or only mild symptoms, making the clinical diagnosis difficult.
- Signs and Symptoms: The disease can manifest with generalized reduction in metabolic activity, cardiac and respiratory depression, fatigue, slow mental functioning, hyponatremia, constipation, cold intolerance, slow movements, depression, constipation, thinning of hair, hair loss, weight gain, thickened tongue, thyroid nodule, periorbital edema, and bradycardia [1, 2, 4, 7, 8].
- Primary failure of the thyroid gland is responsible for 95% of cases of hypothyroidism, and is due to decreased production of thyroid hormone, despite adequate TSH production. Etiologies of primary hypothyroidism include thyroiditis (Hashimoto's disease), medications (iodine, propylthiouracil, methimazole), iodine deficiency, irradiation to neck, hereditary defects in biosynthesis, previous treatment with radioactive iodine, and previous thyroidectomy.
- Secondary hypothyroidism is responsible for the remaining incidence and is caused by either hypothalamic or pituitary disease.

Hashimoto's Thyroiditis

- The most common cause of hypothyroidism in countries with populations that have normal amount of dietary iodine.
- The disease is caused by an autoimmune process, which gradually leads to destruction of the thyroid gland. The thyroid gland exhibits atrophy of thyroid follicles and depletion of thyroid epithelial cells due to large infiltrates of lymphatic germinal centers. This leads to a decrease in thyroid hormone production despite normal levels of TSH.
- It is most commonly seen in women between 45 and 65 years old.
- Patients often present with painless, nontender, diffuse enlargement of the thyroid gland and feeling of discomfort and tightness in the throat.
- Lab values usually show low T4 and T3, high TSH, and presence of antibodies against thyroid peroxidase (TPO). A normal level of TSH reliably excludes hypothyroidism [1, 2, 4, 7, 8].
- Treatment: Treatment of symptomatic hypothyroidism is with hormone replacement therapy such as levothyroxine [1].

Myxedema

- Is a state of decompensated hypothyroidism characterized by hypoglycemia, hypercapnia, hypoventilation, hypotension, susceptibility to cardiac dysrhythmias, hypothermia, stupor or coma, delirium and hyponatremia.
- It usually occurs in elderly and is considered a medical emergency and requires immediate management due to its high mortality rate.
- Only emergent lifesaving surgeries should be done in the setting of myxedema coma.
- It is commonly treated with IV doses of T4 or T3, glucocorticoid therapy, airway management, and supportive measures.
- T3 has a much more rapid onset and should be used first. Patients should be treated in the intensive care unit with continuous cardiac monitoring.

Patient Management

- In patients with uncontrolled hypothyroidism, the concern is heart failure, mental confusion,

delayed wound healing, and hypotension. These patients will often exhibit bradycardia, decreased cardiac output, and decreased peripheral resistance since thyroid hormone is essential for myocardial contractility and catecholamine function. Heart failure only rarely occurs in the absence of coexisting heart disease.
- Similar to hyperthyroidism, it is imperative that patient be restored to a euthyroid state prior to oral surgery procedures.
- Treatment of symptomatic hypothyroidism is with hormone replacement therapy. For mild to moderate hypothyroidism, elective surgery under general anesthesia can be done with little risk for serious complications.
- Consider increasing NPO time due to a decrease in gastric emptying.
- Patients are more prone to hypotension due lack of myocardial contractility response and response to peripheral catecholamines.
- Patients are more prone to upper airway obstruction due to numerous factors: enlarged tongues, goiters, increased parapharyngeal swelling, and fat.
- Patients display increased sensitivity to anesthetic drugs.
- Vigilant monitoring intraoperatively, however, should be done to detect early signs of congestive heart failure, hypotension, and hypothermia.

Diabetes Mellitus (DM)

Diabetes Mellitus Type 1 and Type 2

- A metabolic disorder, which results in a defect in insulin secretion, action, or both, resulting in hyperglycemia.
- Type 1 diabetes mellitus, also known as insulin-dependent diabetes mellitus, is due to impaired production of insulin and occurs early in life. Type 1 diabetes, also referred to as juvenile onset diabetes, usually manifests during youth. The disease occurs due to loss of the insulin-producing beta cells of the islet of Langerhans in the pancreas. This occurs due to an autoimmune process and results in low level of circulating insulin.
- Type 2 diabetes mellitus, formally called non-insulin-dependent diabetes mellitus, occurs due to an altered number and affinity of peripheral insulin receptors and generally occurs later in life. Type 2 diabetes may also have features of reduced insulin secretion. It is the most common type of diabetes mellitus. Etiology is primarily due to lifestyle factors and genetics.
- The hormone insulin is a main regulator of glucose levels in the blood.
- Beta cells of islet of Langerhans in the pancreas produce insulin, which is released in a rapid surge during the first 10–30 minutes after a meal.
- The functions of insulin are to increase glycogen synthesis, decrease gluconeogenesis, increase potassium uptake, and increase lipid synthesis. However, insulin's most important function is to elicit a drastic increase in uptake of glucose by cells, especially in skeletal muscle cells.
- Hyperglycemia in diabetic patients is thus a manifestation of inability for proper uptake of glucose into liver, muscle, and adipose cells.

Diagnostic Criteria for DM

- Fasting glucose of >126 mg/dl on 2 or more occasions.
- Glucose tolerance test (readings greater than 200 mg/dl 2 hours after a 75 gram glucose load).
- HgB A1C of 6.5 or greater.
- Non-fasting plasma glucose ≥200 mg/dl and symptoms of DM.

DM Sequelae

- Peripheral neuropathy.
- These patients usually develop coronary disease at a young age, and are at risk for silent (painless) ischemic episodes due to myocardial neuropathy.

- Diabetic nephropathy – prolonged intraglomerular pressure can lead to renal insufficiency.
- Diabetic retinopathy.
- Increased risk of ischemic heart disease due to glycosylation of LDLs.
- Diabetic cheiroarthropathy, which is often seen in stiff joint syndrome, is limited joint mobility in the hands. This is due to waxy skin-thickening over the dorsum of the hand with restricted mobility of the fingers. It is diagnostic with a positive "prayer sign" which is an inability to fully oppose the palmar surfaces of the digits since patients cannot fully extend their metacarpophalangeal joints (MCPs), proximal interphalangeal joints (PIPs), and the fifth distal interphalangeal joint (DIP).

Class of Drugs to Treat Diabetes

- Insulin – promotes the uptake of glucose into muscle, adipose, and liver tissue.
  - Fast acting (lispro, aspart).
  - Short acting (regular).
  - Intermediate acting (NPH, lente).
  - Prolonged acting (Glargine, levemir).
  - Combined insulin treatment – Novolog 70/30 (70% aspart protamine and 30% aspart) and Humolog 75/25 (75% lispro protamine and 25% lispro).
- Oral hypoglycemics
  - Biguanides – decrease hepatic gluconeogenesis and decrease intestinal glucose absorption (e.g., Metformin). Risk of lactic acidosis in the setting of renal insufficiency with usage.
  - Sulfonylureas – stimulates beta cells to produce insulin (e.g., Glipizide, Glyburide). Risk of hypoglycemia with usage.
  - Thiazolidinediones – work intracellularly to promote insulin sensitivity in adipose, hepatic and muscle tissue. Also decrease triglycerides and increase HDL (e.g., Rosiglitazone, Pioglitazone). Weight gain is a known side effect.
  - Meglitinides – bind ATP-dependent potassium channel to increase beta cell secretion of insulin (e.g., Repaglinide).
  - GLP1 agonist – synthetic peptide of glucagon-like peptide that stimulates insulin secretion and decreases glucagon secretion (e.g., Exantide aka Byetta®).
  - DPP4 inhibitor – inhibits DDP4 which prevents inactivation of GLP1 and GIP leading to increased insulin secretion (e.g., Sitagliptin aka Januvia®).

Diabetic Ketoacidosis (DKA)

- DKA is a metabolic condition that occurs secondary to an insulin shortage that results in hyperglycemia, ketonemia, and an anion gap metabolic acidosis.
- Signs/symptoms – vomiting, confusion, polydipsia, polyuria, dehydration, abdominal pain, Kussmaul breathing, hyperglycemia, hyperkalemia, ketotic "fruity" breath, dry mucous membranes, and hypotension.
- Triggers – CVA, MI, infection (UTI, gastroenteritis, pneumonia), stress, cocaine usage, and inadequate insulin administration.
- Treatment:
  - Fluid rehydration.
  - Insulin.
  - Management of hyperkalemia (remember total body potassium may be low with GI losses).
  - Bicarbonate for treatment of the metabolic acidosis.
  - Identification and treatment of underlying cause.

Patient Management

- Glycemic goals: optimal glycemic goals are postprandial glucose of 120–200 mg/dl range 1–2 hours after a meal.
- Peripheral neuropathy, which increases risk for intraoperative hypotension and cardiorespiratory arrest. These patients usually develop coronary disease at a young age, and are at risk for silent (painless) ischemic episodes

due to myocardial neuropathy. Monitor EKG for signs of ischemia.

- Infections are common among diabetic population, and patients are susceptible to infections due to impaired chemotaxis and phagocytosis by monocytes and neutrophils. The use of prophylactic and postoperative antibiotics is of importance when performing oral surgery procedures to prevent the development of serious infections in poorly controlled diabetics with a fasting glucose of >250 mg/dl [9].
- Stiff joint syndrome is common in type 1 diabetics and can be a significant risk during airway management due to limited mobility of atlanto-occipital joint motion.
- Thyroid disease should be ruled out as type 1 DM has an association with other autoimmune diseases such as Graves' disease and Hashimoto thyroiditis.
- Due to the risk of perioperative and postoperative complications, a thorough history and physical, a recent ECG, recent labs including glucose, potassium, creatinine, and urinalysis are essential.
- Preoperatively, the physician should be cognizant of signs of hypoglycemia such as tachycardia and diaphoresis.
- Preoperative and postoperative finger stick to assess glucose levels.
- Obtain a recent HbA1c. Gives a true clinical picture of glycemic control over a 3-month period.
- Use corticosteroids judiciously. Corticosteroids may be of benefit as it relates to treatment of edema, airway management in the setting of infection, and management of postoperative nausea/vomiting. However, there is also an increased risk of immunosuppression and worsening hyperglycemia. The benefits of usage must outweigh the risks.
- Diabetic patients needs to be properly hydrated prior to surgery if NPO. Patient should receive 5–10 g/hr (100–200 mL of 5% dextrose solution hourly) if NPO.
- Maintenance of hydration during surgery – avoid lactated ringers due to the conversion of lactate to glucose. Blood glucose of <100 mg/dl, consider D5W with 0.45% NS and consider NS for patients with a blood glucose >130 mg/dl.
- Insulin considerations – Type 1 diabetics require insulin, otherwise long-standing type 1 diabetics will undergo rapid development of diabetic ketoacidosis. Blood glucose level can be managed with a wide dose range of both short-acting and long-acting insulin. A short-acting insulin sliding scale should be added in addition to the basal insulin for patients admitted to the hospital. The goal preoperatively is to avoid hypoglycemia and severe hyperglycemia.
- The basal insulin should be decreased by half prior to surgery, which allows prevention of ketoacidosis development and concomitantly prevention of hypoglycemia. Short- and rapid-acting insulin should be held. Surgery should be scheduled early in the morning to prevent hypoglycemia after prolonged periods of NPO.
- Due to diabetic autonomic neuropathy, gastric emptying is often delayed, and patients may be at increased risk of aspiration during sedation procedures. Consider longer NPO periods.
- Oral hypoglycemic considerations – Glucose levels should be measured before and after surgery. In general, on the day of surgery, oral regimen such as sulfonylureas and meglitinides should be discontinued. Short-acting insulin can be administered on a sliding scale as a substitute to maintain glycemic control. Metformin is discontinued preoperatively due to its association with lactic acidosis in the setting of hypotension, poor perfusion, or hypoxia. Discontinuation of sulfonylureas is due to its association with hypoglycemia and increased risk of perioperative myocardial ischemia and infarction. Thiazolidinediones, however, can be continued due to low risk of hypoglycemia.

Adrenal Gland Physiology

- The adrenal gland is composed of the outer cortex and inner medulla.
- The adrenal cortex synthesizes three groups of hormones: glucocorticoids (cortisol), mineralocorticoids (aldosterone and 11-deoxycorticosterone), and androgens (dehydroepiandrosterone).
- The two most important hormones are the adrenal cortex hormones cortisol and aldosterone, while the adrenal androgens are not as physiologically significant.
- The adrenal medulla produces norepinephrine and epinephrine.
- Corticotropin releasing factor (CRF) from the hypothalamus controls the pituitary hormone adrenocorticotropic hormone (ACTH), which in turn manages the secretion of cortisol.

Cushing's Syndrome

- Cushing's syndrome is a disease of excessive free plasma glucocorticoids.
- It occurs due to chronic use of glucocorticoid products such as prednisone, dexamethasone, and prednisolone or from increased endogenous production. Iatrogenic means from chronic steroid use is the most common cause, while endogenous Cushing's syndrome is a rare disease.
- Various diseases such as pulmonary, rheumatologic, neurologic, and renal diseases as well as post-transplant patients often necessitate chronic use of high doses exogenous steroids. These patients are thus at risk for Cushing's syndrome.
- The other causes of Cushing's syndrome are either ACTH independent (primary adrenal) or ACTH dependent (pituitary or ectopic). Cushing's disease, an ACTH-dependent etiology, is due to a pituitary tumor, which produces excess amount of ACTH. This surge in ACTH subsequently causes increased production of cortisol from the adrenal glands. Ectopic tumors, also ACTH-dependent tumors, are usually due to neoplasms outside of the adrenal gland. Common ectopic cancers accountable are medullary thyroid cancer, lung cancers, pheochromocytomas, and pancreatic islet tumors.
- Signs and Symptoms: Cushing's syndrome can manifest with buffalo hump, weight gain, truncal obesity, plethora, striae, cognitive dysfunction, depression, proximal muscle weakness, osteopenia, hyperglycemia, and hypertension.

Diagnosis:

- Laboratory tests are focused on locating an etiology for the many causes of Cushing's syndrome. These tests include blood cortisol levels, 24-hour urine free cortisol levels, dexamethasone suppression test, and ACTH level.
- 24-hour urine free cortisol level is usually the initial test, and a concomitant ACTH level will help identify the etiology of Cushing's syndrome.
- The dexamethasone suppression test is the best test to locate etiology for Cushing's syndrome. Dexamethasone, an exogenous steroid, would normally suppress ACTH production and thus cortisol secretion in a normal person. The test is usually conducted at night, and either a low or high dose dexamethasone is given, and a blood cortisol level is drawn 9 hours later. A low dose of dexamethasone, through negative feedback, decreases cortisol production (cortisol levels lower than 3–5 ug/dl) in normal patients with no pathology in the adrenal pituitary axis. A high dose of dexamethasone causes decreased cortisol levels in an ACTH-producing pituitary tumor, but cortisol levels are not suppressed on adrenal adenoma or ectopic ACTH-producing tumors.
- Treatment: Cushing's syndrome depends on the etiology of the disease. For exogenous iatrogenic Cushing's syndrome, gradual cessation of causative drug usually cures the disease. Pituitary and adrenal tumors are treated with surgical excision, and ectopic ACTH-producing tumors are treated with modalities geared to the specific tumor.

Adrenal Insufficiency

- Primary adrenal insufficiency is a rare entity, and results in a decrease in production of both glucocorticoids and mineralocorticoid secretion. It is caused by the anatomic destruction of the gland from various causes, such as tuberculosis or fungal infection. Autoimmune adrenal destruction is responsible for 80% of the cases (e.g., Addison's disease) [10].
- Secondary adrenal insufficiency is due to decrease or loss of glucocorticoid secretion only. Etiology is due to suppression of the hypothalamic-pituitary axis by exogenous steroids or endogenous steroids (e.g., tumor). Administration of high doses of glucocorticoids is the most common cause of secondary adrenal insufficiency.
- Treatment: Replace glucocorticoids (e.g., hydrocortisone or cortisone acetate divided throughout day) and mineralocorticoids (e.g., fludrocortisone once daily) in physiological manner.

Addison's Disease

- Disorder of insufficient adrenocortical synthesis and secretion of glucocorticoids and mineralocorticoids.
- Characterized by weakness, anorexia, arthralgia, abdominal pain, hyperpigmentation, anorexia, hypotension, and disruption of electrolytes (hyponatremia and hyperkalemia) [10].
- Diagnosis: When low morning cortisol levels are detected, a corticotropin stimulation test may be performed. If morning levels are less than 3 mcg/dL, no further testing is required.

Patient Management

- Patients with adrenal disease are at an increased risk for infection and delayed wound healing. Use of prophylactic and postoperative antibiotics are recommended to minimize risk of serious infection [10].
- In the setting of patients taking exogenous steroids, the hypothalamic-pituitary axis is normally not suppressed with low-dose steroid use (prednisone of 5 mg or less), alternate day dosing, and if used less than 3 weeks consecutively [11]. In these circumstances, no supplemental steroids (stress dose) are needed prior to procedures. However, if stress dose of steroids is not given in the setting of chronic high-dose steroid use, adrenal crisis can be precipitated during major surgery. This is due to adrenal insufficiency resulting from the inability of the body to mount an adequate cortisol response to stress.
- Cushing's Syndrome:
  - Obesity makes patients higher risk sleep apnea and gastroesophageal reflux.
  - Glucose intolerance common, invoke sliding scale for glycemic control.
  - Ensure proper patient positioning due to fragile skin and higher risk for osteoporosis.
- Addison's Disease:
  - Check serum potassium preoperatively and regularly.
  - Supraphysiological administration generally overstated and patient with primary adrenal insufficiency generally require 100 mg of IV hydrocortisone.
- Adrenal crisis is a life-threatening physiological state, brought about by major physical stress.
  - It will manifest as severe circulatory collapse and hypotension, not responsive to vasopressors. Patient will also have a fever, lethargy, severe flank or abdominal pain, tachycardia, delirium, and eventually coma.
  - Secondary adrenal insufficiency patients caused by exogenous steroids, autoimmune or inflammatory conditions have 1–2% risk of adrenal crisis [11] and do not require as high doses seen in adrenal insufficiency (Table 18.1). The dosing is dependent on the degree of surgical stress.

Table 18.1 Perioperative glucocorticoid regimen for secondary adrenal insufficiency

Glucocorticoid regimens for patients with secondary renal insufficiency		
Degree of surgical stress	Intraoperative steroids	Taper
Minor – e.g., dentoalveolar	Usual dose	None
Moderate – e.g., orthognathic	Hydrocortisone 50 mg or equivalent preoperatively	Continue 25 mg of hydrocortisone every 8 hours for 24 hours, then resume regular dosage
Major, e.g., free flap	Hydrocortisone 100 mg or equivalent preoperatively	Hydrocortisone 50 mg every 8 hours for 24 hours, then taper by half every day to usual dosage

Modified from Yeh et al. [11]

Parathyroid Glands

- The parathyroid gland produces parathyroid gland hormone (PTH) in response to circulating calcium levels in the bloodstream. As calcium levels decrease PTH increases and conversely as calcium levels increase PTH release decreases. PTH therefore plays a significant role in calcium homeostasis.
- In order to achieve this, PTH works on the GI tract, the renal system, and bony structures. For example, if the parathyroid gland were to respond to a low serum calcium level it would increase the production of PTH thus causing a sequence of events leading to sequestration of calcium. PTH will cause the GI system to uptake as much calcium as possible, the renal system will be signaled to retain calcium by decrease in its excretion, and finally the bone will ultimately undergo osteoclastic activity to release calcium into the circulation. Thus if the parathyroid gland recognized a state of hypocalcemia, it would increase the amount of PTH production causing the opposite reactions at the end organs therefore decreasing the amount of circulating calcium.

Hyperparathyroidism

- Primary causes – an adenoma or enlargement of one of the four parathyroid glands situated behind the thyroid over produces PTH.
- Secondary causes – hypocalcemia due to other systemic effects leading to the release of excess PTH.
- Patients may have maxillofacial manifestations such as the following:
  - Browns tumor which is a giant cell lesion.
  - Loose teeth, altered eruption, root malformation.
  - Sialolithiasis.

Hypoparathyroidism

- Causes – surgically removed parathyroid glands, irradiated parathyroid glands, autoimmune process, hereditary, hyper-/hypo magnesium.
- DiGeorge's syndrome: associated with hypoplasia or aplasia of the parathyroid glands.
- Dentition: enamel hypoplasia, malformed roots, missing teeth.
- Neurologic: paresthesia of lips and/or tongue.
- Muscular: facial muscle spasms, muscles of mastication spasms.

Hypercalcemia

- Causes:
  - Hyperparathyroidism (most common).
  - Medications.
  - Vitamin supplements.
  - Cancer.
  - Disease entities, e.g., tuberculosis, pneumonia, sarcoidosis.
- Consequences:
  - CNS: lethargy, fatigue, confusion, seizure, coma.
  - Renal: increased urination, thirst, nephrolithiasis, renal insufficiency.
  - Musculoskeletal: decrease strength, osteo-

porosis (calcium sequestered from bone).
  - Gastrointestinal: nausea, vomiting, abdominal pain.
  - Cardiac: arrhythmia (short QT).

Hypocalcemia

- Causes:
  - Hypoparathyroidism (most common).
  - Gastrointestinal malabsorption.
  - Hypophosphatemia.
  - Magnesium levels.
  - Vitamin D depletion.
  - Medications.
- Consequences:
  - Neuromuscular: paresthesia, muscle spasm.
  - CNS: seizures, dementia.
  - Dermatologic: course skin, brittle nails, hair loss.
  - Skeletal: osteoporosis (not enough calcium to be sequestered by the bone).
  - Cardiac: arrhythmia (long QT).

Hyperparathyroidism or Hypoparathyroidism

- Decrease in bone calcium:
  - Poor calcium uptake or calcium sequestration from the bone during childhood (e.g., Albright's syndrome).

Treatment

- Treat the underlying cause of disturbance for calcium or PTH levels.

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Neurological Disorders

19

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Amyotrophic Lateral Sclerosis (ALS)

- ALS is a progressive neurodegenerative disease of the upper and lower motor neurons due to degeneration of lateral corticospinal tracts, which will eventually lead to muscle failure.
- Commonly affects men between the ages of 40 and 60.
- Also known as Lou Gehrig's disease.
- Presents with progressive muscle weakness, hyperreflexia (due to upper motor neuron involvement), muscle fasciculations (due to lower motor neuron involvement), and atrophy.
- Maxillofacial implications – dysphagia due to pharyngeal muscle weakness leading to an aspiration risk (bulbar nuclei involvement), tongue weakness with fasciculations, and sialorrhea due to inability to handle secretions.
- Death is usually due to respiratory complications (aspiration, inadequate ventilation due to diaphragm, and accessory muscle weakness). Patients also have an inadequate cough reflex.

Treatment

- No curative treatment.
- The drug riluzole, a glutamate blocker, may provide neuroprotection and extend survival.
- Supportive treatments include antibiotic prophylaxis for aspiration, gastrostomy, tracheostomy, and mechanical ventilation.

Patient Management

- Consult the patient's physician to ascertain baseline respiratory status.
- Consider treatment in the operating room for airway management. Increased risk of aspiration due to bulbar involvement, respiratory weakness, and sialorrhea. Consider anticholinergic preoperatively.
- Succinylcholine may cause hyperkalemia secondary to denervation and immobilization.

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- General anesthetics may lead to prolonged respiratory depression.
- Nondepolarizing blockade may lead to prolonged neuromuscular blockade.
- Patients prone to autonomic dysfunction, which manifests as tachycardia and orthostatic hypotension.
- Avoid regional anesthesia as it may exacerbate disease.

Alzheimer's Disease

- Alzheimer's disease (AD) is a neurodegenerative disease characterized by the progressive loss of cortical neurons and the formation of amyloid plaques and intraneuronal neurofibrillary tangles.
- The progressive loss of neurons eventually leads to a relative deficiency in cortical acetylcholine transmission (resulting from loss of neurons in the nucleus basalis).
- AD is the most common neurodegenerative disease and the most common cause of dementia in persons older than 65 years of age.
- Patients with AD show a slow, progressive decline in intellectual function. Hallmarks of the disease include impaired memory, judgment, and decision-making, and emotional lability. Extrapyramidal signs, apraxias, and aphasias are present late in the course of the disease.
- Classification – AD is categorized into two forms (with continued further research, it is very likely that AD will continue to be subcategorized into more precise etiological categories):
  1. Familial form (early-onset AD)
     - Rare.
     - Early onset, usually before age 60.
     - Autosomal dominant pattern of inheritance.
     - Is almost universal in older patients with Down syndrome (trisomy 21).
  2. Sporadic form
     - More common.
     - Typically occurs after 60 years of age.

Workup and Diagnosis

- The clinical diagnosis of AD is by exclusion.
- Definitive diagnosis is usually made on postmortem examination via autopsy (presence of accumulation of amyloid plaques and neurofibrillary tangles).
- MRI imaging is preferred in diagnosis of AD. Will demonstrate marked cortical atrophy with ventricular enlargement.

Treatment

- There is no cure for AD and treatment focuses on control of symptoms. Even with treatment, prognosis is poor.
- The pharmacological agents for AD include cholinesterase inhibitors (tacrine, donepezil, rivastigmine, and galantamine), and the glutamate antagonist, memantine.
- Antipsychotics, antidepressants, and anxiolytics may be useful for patients with behavioral disturbances.

Patient Management

- Patients with AD are often confused and can be uncooperative. While some patients may tolerate a procedure with local anesthesia, many may need some sort of procedural sedation.
- Inquire who makes health care decisions for the patient if the patient does not possess capacity (power of attorney/health care advocate).
- As a general rule, anesthetic dosages should be decreased due to increased sensitivity and decreased pharmacokinetics resulting in a slower distribution and reduction in clearance [1].
- As a general rule, doses of sedative-hypnotics should be reduced by 30% [1].
- Preferred medications include short-acting sedative-hypnotics, anesthetic agents, and narcotics since they allow a more rapid recovery.

- Patients are at a higher risk of delirium and postoperative cognitive dysfunction after an anesthetic challenge [1].
- Centrally acting anticholinergics, such as atropine and scopolamine, may contribute to postoperative cognitive dysfunction and should be avoided if possible.
- Glycopyrrolate, which does not cross the blood–brain barrier, is the preferred agent when an anticholinergic is needed.
- Have the patient continue their AD medications preoperatively.

Parkinson's Disease

- Parkinson's disease (PD) is a neurodegenerative disease characterized by the classic triad of bradykinesia, rigidity, and resting (pill-rolling) tremor.
- It is caused by the progressive loss of dopaminergic neurons in the pars compacta of the substantia nigra in the basal ganglia.
- The depletion of dopamine results in the diminished inhibition of neurons in the extrapyramidal motor system and unopposed stimulation by acetylcholine, thereby producing the tremor of Parkinsonism.
- PD typically affects individuals older than 60 years of age.
- Other signs include postural instability, bradyphrenia (slowness of thought processes), akinesia (lack of spontaneous movement), and problems with walking.
- Patients note unilateral symptoms initially, which subsequently spread to involve both sides.
- Tremors – are rhythmic, alternating flexion and extension of the thumbs and other digits. They are typically described as "pill-rolling tremors." Tremors are more prominent at rest and tend to disappear during voluntary movement.
- Rigidity – first presents in the proximal muscles of the neck but may progress to absence of head rotation when turning the body and loss of arm swings when walking.
- Akinesia – facial immobility presenting as infrequent blinking and paucity of facial expression.

Workup and Diagnosis

- The diagnosis of PD is made according to the clinical signs of tremor, bradykinesia, muscular rigidity, and postural instability.
- Neuroimaging of no value.
- Dopaminergic challenge (administration of levodopa) provides clinical support for diagnosis if improvement demonstrated.

Treatment

- The goal is to increase the concentration of dopamine in the basal ganglia.
- Carbidopa-levodopa (Sinemet®) is the mainstay of treatment of PD. Levodopa is a dopamine precursor that is combined with the decarboxylase inhibitor, carbidopa, which prevents peripheral conversion of levodopa to dopamine, thereby optimizing the amount of levodopa that can enter the CNS. Carbidopa-levodopa can also be combined with the catechol-O-methyltransferase (COMT), entacapone (the three-drug combination marketed as Stalevo®), which blocks the peripheral breakdown of levodopa and, therefore, maximizes the amount of levodopa that can cross the blood-brain barrier.
- Selegiline and rasagiline are type B monoamine oxidase (MAO-B) inhibitors that help control PD symptoms by inhibiting the catabolism of dopamine in the CNS. They are used in patients with early PD and delay the need for levodopa treatment. Anticholinergics and antihistamines are also used to antagonize the effects of acetylcholine.
- Surgical treatment – patients who fail medical treatment are candidates for surgical intervention (such as thalamotomy, pallidotomy, or implantation of a deep brain stimulator).

Patient Management

- Have the patient continue their medications preoperatively. The half-life of levodopa is short and abrupt withdrawal can cause worsening of muscle rigidity and interfere with ventilation.
- Nausea/vomiting are side effects of levodopa and may be treated with ondansetron, dexamethasone, and transdermal scopolamine.
- Avoid phenothiazines (promethazine), butyrophenones (droperidol), and metoclopramide because their antidopaminergic activity can exacerbate symptoms.
- Diphenhydramine may be used for patients with tremor.
- Propofol is relatively safe and may have some antiparkinsonian effects via GABAergic and glutamate transmission [2].
- Opioids should be used judiciously as patients with PD have a higher incidence of chest wall rigidity [2]. If a patient is treated with monoamine oxidase inhibitors (MAOI), reduction in opioid dosage is advised due to decrease in liver metabolism of narcotics.
- Be mindful of serotonin syndrome risk on patients taking MAOI (hypertension, tachycardia, hyperthermia, diaphoresis, confusion and agitation).
- The chronic administration of levodopa may result in depletion of norepinephrine stores in the autonomic nervous system, which may sensitize adrenergic receptors to epinephrine administered with local anesthetic cartridges, and result in elevated blood pressure. Ketamine may be relatively contraindicated because of this and should be used judiciously.
- Many patients with PD have baseline autonomic dysfunction which can manifest as orthostatic hypotension, labile blood pressures, and sialorrhea [2].
- Monitor arterial blood pressure carefully. Hypotension should be treated with fluids and small doses of phenylephrine (a direct-acting vasopressor), rather than ephedrine.
- Inhalational anesthetics decreases dopaminergic transmission and may exacerbate symptoms [2].

Multiple Sclerosis

- Multiple sclerosis (MS), an inflammatory demyelinating disease of the central nervous system (CNS), is marked by discrete episodes of neurological dysfunction, termed relapses, followed by periods of remission.
- Such a course is termed relapsing-remitting and occurs in about 82% of patients. The remaining patients (18%) have a primary progressive course without discrete relapses. Many patients with relapsing-remitting MS eventually advance to a steady irreversible progression termed secondary progressive MS [1].
- The cause remains unknown; however, evidence suggests that MS is an autoimmune disease triggered by unknown environmental factors.
- Affects women more frequently than males (2:1).
- Onset commonly occurs between the ages of 20 and 40 years [1].
- Pathogenesis – demyelination preferentially occurs in the periventricular areas of the brain, due to autoimmune processes involving T and B autoreactive cells.
- Symptoms – based on multifocal involvement (reflects sites of demyelination). May manifest as visual disturbances, gait disturbances, ascending paresis, limb paresthesias. MS may present as bilateral trigeminal neuralgia.

Workup and Diagnosis

- MRI with gadolinium is the best study to demonstrate demyelination in the CNS due to its ability to delineate between normal myelin and MS lesions.
- CSF analysis will typically show increased intrathecal synthesis of immunoglobulin G (IgG). Isoelectric focusing gel of CSF will show oligoclonal banding of IgG.
- Evoked potential testing measures electrical activity in parts of the CNS caused by light, sound and touch. A patient with MS will have reduced neurotransmision from stimuli. Visual evoked potential is the most commonly used as it is the most sensitive of the evoked potentials studies to diagnose MS. Brain stem and somatosensory evoked potentials can also be utilized.

Treatment

- Treatment of MS consists of management of acute relapses aimed at reduction of the severity and frequency of these relapses, and slowing the progression of the disease.
- Treatment of major relapses is with IV methylprednisolone (500–1000 mg daily for 3–5 days), and sometimes followed by a taper of oral prednisone.
- Other therapies include treatment with interferon-B, glatiramer acetate, natalizumab, mitoxantrone, and plasmapheresis.
- There is no cure for MS.

Patient Management

- Surgical stresses are associated with exacerbation of symptoms [3]. Provide adequate anxiolysis and pain management to avoid exacerbations.
- Elective surgery should be avoided during periods of relapse, regardless of the anesthetic technique used.
- The informed consent discussion should include the possibility of worsening MS symptoms.
- Avoid increases in body temperature, which will lead to an exacerbation of symptoms.
- Consider stress-dosing the patient who is on long-term steroids.
- Patients taking interferon should have a CBC to exclude anemia, neutropenia, and thrombocytopenia.
- There are no recognized interactions with general anesthetics in patients with MS.
- Patients with advanced disease may have a labile cardiovascular system due to autonomic dysfunction. Be prepared to treat labile blood pressures and heart rates.
- A patient's ability to cough, exhale deeply, or clear secretions may be impaired and put them at risk for aspiration.
- Avoid succinylcholine in patients with paralysis or paresis because of the potential for hyperkalemia.

Seizure Disorders

Seizures and Epilepsy

- Seizure is caused by a sudden onset of abnormal, highly synchronous discharges of neurons.
- They can present with a wide variety of signs and symptoms that vary according to the location in the brain that is affected. These may include involuntary movements, sensations, and impaired consciousness.
- Seizures are divided into phases:
  - Ictus – the seizure itself.
  - Ictal phase – period of time during the seizure.
  - Postictal phase – the time after the seizure.
  - Interictal phase – the time in between seizures.
- The term "symptomatic seizure" refers to a seizure that occurs secondary to a medical or neurologic illness in which the brain function is temporarily deranged. Such secondary causes include fever in children, metabolic derangements (e.g., hypoglycemia or hyponatremia), intoxication, acute head injury, and hypoxic-ischemic conditions.
- In an individual, seizures are typically stereotyped; however, a person can have more than one type of seizure and a particular seizure type can have varying intensities
- 10% of people have a seizure during their lifetime. There is a bimodal distribution of incidence and prevalence with epilepsy being more common in childhood and old age [2].
- Epilepsy is a chronic disorder defined as recurrent seizures resulting from congenital or acquired factors in the cerebral cortex (e.g., cerebral scar, malformation, tumor, abnormal gene expression).
  - The term *seizure disorder* is synonymous with epilepsy.
  - Two separate unprovoked seizures are required to make the diagnosis of epilepsy.

Classification of Seizures

- Seizures are classified into two broad types depending on the area of the brain affected (partial vs. generalized). They are also classified according to the level of one's consciousness during the seizure episode (simple vs. complex).
- Focus of seizure activity
  - Partial seizure – originate from a single focus of cortical neurons.
  - Generalized seizure – involves diffuse activation of neurons in both cerebral hemispheres.
- Level of consciousness
  - Simple seizure – no loss of consciousness.
  - Complex seizure – altered level of consciousness.

Types of Seizures

- Simple partial seizures (SPS)
  - A focal-onset seizure without impairment of consciousness.
  - Is commonly called an aura and can serve as a warning that a more intense seizure is imminent. Auras are commonly seen in patients with partial epilepsy.
- Secondary generalized tonic-clonic seizure
  - A focal-onset seizure that spreads throughout the brain to create a secondary generalized seizure. These seizures are also known as grand mal seizures.
- Primary generalized seizures – begin diffusely and involve both cerebral hemispheres simultaneously from the outset.
  - Absence seizures (petit mal)
    - Occur mainly in children.
    - Demonstrate sudden, momentary lapses in awareness with staring. May have rhythmic blinking or automatisms (nonpurposeful, repetitive behaviors). Behavior and awareness return to normal immediately after the seizure ends.
  - Myoclonic seizure
    - Manifests as rapid, recurrent, brief muscle jerks that occur bilaterally or unilaterally without loss of consciousness.
  - Primary generalized tonic-clonic seizure
    - Begins with a few myoclonic jerks or abruptly with a tonic phase lasting 20–60 seconds, followed by up to 1 minute of a clonic phase, followed by a postictal state.

Types of Epilepsy

- Idiopathic partial epilepsy
- Symptomatic partial epilepsy
  - Most common type of epilepsy, and in all cases has an underlying focal abnormality in the cerebral cortex (e.g., scar, malformation, tumor, or abnormal gene expression)
- Post-traumatic epilepsy
- Idiopathic generalized epilepsy
- Symptomatic secondary generalized epilepsy
  - Occurs in people with multifocal or diffuse brain dysfunction from early in life. These patients will have tonic and atonic "drop seizures," in which the patient will fall, with no protective reflexes if they are upright during the seizure. As such, they should wear a helmet to protect against head injury.

Workup

- MRI – the preferred method for studying brain structure in patients with epilepsy.
- Standard EEG (electroencephalography) – the most important diagnostic test for seizures and epilepsy. Used to identify locations of seizure foci and characterize their electrical properties.
- Electrocorticography – involves the placement of electrodes directly onto the cerebral surface to aid in foci identification during surgical resection.

Treatment

- Treatment begins with single drug therapy, where the dosage is adjusted to achieve control. Additional drugs are added when monotherapy fails.

- When one antiepileptic drug is substituted for another, the original agent must not be discontinued until the new one is added, otherwise status epilepticus may result.
- Antiepileptic drugs (AEDs)
  - In all idiopathic generalized epilepsies (IGEs), valproate and lamotrigine are the first-line agents and are efficacious in controlling the majority of patients.
  - Carbamazepine, oxcarbazepine, topiramate, levetiracetam, lamotrigine, and zonisamide are the current first-line agents for partial seizures.
  - Phenytoin is the most commonly used AED in partial epilepsy in developed countries.
  - Phenobarbital is the most widely used AED in the world because of its low cost.

    These medications (minus gabapentin) undergo hepatic metabolism, and some (carbamazepine, phenytoin, and barbiturates) lead to enzyme induction and alter their own pharmacokinetics.

    All AEDs depress cerebral function and can lead to sedation, cognitive dysfunction, and incoordination.

Surgical Treatment of Seizures

- Indicated for patients who do not respond to or cannot tolerate the side effects of pharmacologic therapy.
- Partial seizures may respond to resection of a pathologic region.
- Vagal nerve stimulator (VNS) implantation (typically left side to avoid cardiac complications due to right vagus nerve supply to SA node).
  - More conservative treatment than other surgical procedures for seizure.
  - Swiping a magnet over the device can give extra stimulation and sometimes abort a seizure.
  - Once implanted, a patient cannot have an MRI of the neck. MRIs of the head with standard strengths may be permitted. Consult with radiology for recommendations.

Status Epilepticus (SE)

- A continuous episode of prolonged seizure activity or two or more occurring in sequence without recovery of consciousness between them.
- It is a medical emergency as it is life threatening.
- The most common cause of SE is abrupt withdrawal of antiepileptic drugs (non-compliance) in a person with epilepsy. The longer the SE lasts, the more difficult it is to terminate and the more likely it is to cause permanent brain damage.
- Treatment of status epilepticus [3, 4]
  - Activate EMS/911 if no self-resolution in 5 minutes.
  - Airway Breathing Circulations (ABCs), vitals, ECG. Patient may require airway protection with endotracheal intubation.
  - Supplemental oxygen.
  - Establish venous access – consider administering benzodiazepines. Consider drawing labs for electrolytes, toxicology, anticonvulsant drug levels, and hematology.

    Midazolam: 10 mg for >40 kg, 5 mg for 13–40 kg.

    Lorazepam: 0.1 mg/kg/dose, max: 4 mg/dose.

    Diazepam 0.15–0.2 mg/kg/dose, max: 10 mg/dose.
  - Rule out hypoglycemia and treat as needed (50 mL of 50% dextrose).
  - Routine glucose administration is not indicated because hyperglycemia can exacerbate brain injury.
  - Arterial blood gas draws to monitor for metabolic acidosis and to assess ventilation.
  - Hyperthermia frequently occurs and requires active cooling.
  - If seizure continues, consider second-line therapy such as phenobarbital (10–15 mg/kg at 100 mg/min) or phenytoin (15–18 mg/kg T 50 mg/min) or anesthetic dose of propofol.

Patient Management

- Consult with neurologist.
- Identify seizure triggers, frequency, and specific type of seizures. Driver's licenses status can help qualify the severity and control of seizures. Patient may not be able to hold license unless well controlled.
- Utilize drugs that raise the seizure threshold (e.g., benzodiazepines and propofol) and avoid drugs that lower the seizure threshold (e.g., methohexital, flumazenil, ketamine, and meperidine).
- Ketamine is controversial as it is proconvulsant at low doses, but anticonvulsant at higher doses [4].
- Sedation produced by antiepileptic drugs may have an additive effect with other anesthetics.
- Etomidate should be avoided as it is reported to increase risk for postoperative seizures [4].
- Nitrous oxide is safe to use as is most inhalation agents with the exception of enflurane [4].
- Phenytoin and carbamazepine can shorten the duration of nondepolarizing muscle relaxants.
- Opioids although considered safe increase EEG activity and in fact are used during epilepsy surgery to aid in identifying offending foci. Alfentanil is contraindicated due to its particularly excessive impact of EEG activity [4].
- Tramadol should be avoided.
- Consider intraoperative electrocorticography to assess for characteristic interictal patterns.
- Under general anesthesia, seizure activity can still occur with or without movement. Utilize electrocorticography, blood pressure, and heart rate as surrogates to determine whether an occult seizure is occurring.

Spinal Cord Injury

Acute Spinal Cord Injuries

- Acute spinal cord injury occurs in 1.5–3% of all major trauma victims [5, 6].
- 4–5% of traumatic head injury have concurrent injury to the spine usually C1–C3 [6].

Clinical Manifestation

- Cardiovascular effects
  - Initial sympathetic stimulation with increase in BP.
  - Followed by neurogenic shock, which causes hemodynamic changes and lasts for 1–3 weeks and occurs with injuries above T4–6. Symptoms include the following:

    Hypotension caused by loss of sympathetic tone and decrease in systemic vascular resistance.

    Increase in venous capacitance and pooling

    Bradycardia and decreased ventricular function in lesions above T5 (sympathetic innervation to the heart = T1–T4)
  - Autonomic reflexia in chronic stage with lesions above T7.
  - Hypotension is more severe with cervical spine injuries than with thoracic and lumbar cord injuries.
- Respiratory effects
  - Paralysis of diaphragm and apnea for lesions above C4 (C3, 4, 5 keeps the diaphragm alive).
  - Variable loss of intercostal and abdominal functions in lesions below C5.
  - Decrease in functional residual capacity.
  - Decrease in forced vital capacity.
  - Decrease in maximum inspiratory and expiratory pressure.
  - Hypoventilation with hypercapnia and hypoxemia.
  - Neurogenic pulmonary edema may occur.
  - Pneumonia from atelectasis, impaired cough, aspiration of gastric contents.
  - Risk of pulmonary embolism.
- Other effects
  - Spinal shock refers to a loss of reflexes and is a common misnomer for neurogenic shock.
  - Flaccid paralysis and absence of tendon reflex in the initial stages.
  - Spastic paralysis and abnormal reflexes in chronic stage.

  - Chronic renal infection from instrumentation (indwelling catheters).
  - Up to 50% of patients with C spine injury also have traumatic brain injury.

Preoperative Evaluation

- C-spine radiographs obtained for patients who meet any of the five criteria:
  1. Midline tenderness
  2. Focal neurologic deficits
  3. Intoxication
  4. Abnormal sensorium
  5. Painful distracting injury
- Two-thirds of trauma patients have multiple injuries that interfere with proper C spine evaluation.
- Evaluation ideally CT or MRI.
- If patient is unstable, plain films with portable X-ray machines are obtained of AP and lateral views.
- Treatment of C spine fracture or dislocation is immediate immobilization to limit neck flexion and extension – requires hard neck collars (limit movement by 25%), immobilization by halo-thoracic devices are the most effective [6].
- During direct laryngoscopy with manual in-line stabilization (assistant's hand placed on each side of the face with fingertips resting on the mastoid process and applying downward pressure against the table to immobilize the head in a neutral position).
- For intubation, consider video laryngoscopy as it reduces pressure and force on C spine.
- Movement of unstable C spine can mechanically deform the spinal cord; elongation of the cord can compromise blood supply to the spinal cord by narrowing the longitudinal blood vessels.
- Spinal cord injury without radiographic abnormality is more common in children and is thought to be due to a temporary distortion/distraction of spinal cord without fracture of bony structure.

Chronic Spinal Cord Injuries

- Weeks after acute spinal cord injury, reflexes gradually return.
- Common sequelae of chronic spinal cord injury result from the initial traumatic neurological insult [6].
- Early (2 years after injury)
  - Frequent UTIs due to inability to empty the bladder completely which may result in calculus formation and ultimately renal failure.
  - Skeletal muscle spasticity due to overactivity of the sympathetic system occurs.
  - Chills and fevers due to altered thermoregulation.
  - Decubitus ulcer due to immobility.
  - Autonomic hyperreflexia due to neurogenic dysregulation, which can lead to severe hypertension when stimulated below the level of the lesion. It is a medical emergency and occurs in 50–75% of patients with a lesion above T6 [7].
- Late (30 years after injury)
  - Decubitus ulcer
  - Skeletal muscle or joint pain
  - GI dysfunction
  - Cardiovascular dysfunction due to inactivity, reduced muscle mass, and development of metabolic syndrome. 40% of patients die due to cardiovascular disease [7].
  - Chronic UTI and renal calculus formation.

Patient Management

- Two primary goals are to maintain adequate oxygenation/ventilation by securing airway and to maintain spinal cord perfusion pressure.
- Autonomic hyperreflexia is a risk for labile swings in heart rate and blood pressure.
- Awake fiberoptic laryngoscopy under topical anesthesia is an alternative if the patient is cooperative and blood and secretions do not obscure the airway. Coughing during fiberop-

tic intubation may result in C spine movement as well.
- IV access commonly difficult due to reduction in blood volume, pooling of blood in lower limbs and reduced cutaneous blood flow.
- 50% of chronic spinal cord injury patients commonly have anemia and therefore, maintaining oxygenation is paramount. The threshold for transfusing blood products should be low in cases with massive amounts of blood loss [7].
- During direct laryngoscopy, manual in-line stabilization (assistant's hand placed on each side of the face with fingertips resting on the mastoid process and applying downward pressure against the table to immobilize the head in a neutral position).
- For intubation, consider video laryngoscopy as it reduces pressure and force on C spine.
- Rapid sequence intubation (RSI) and non-RSI are both appropriate.
- Absence of compensatory sympathetic nervous system response in patients with C spine or high T spine cord injury make them vulnerable to decreases of BP following changes in body position, blood loss, positive pressure ventilation.
- Due to hemodynamic changes, crystalloid infusion and vasopressors may be necessary to maintain adequate perfusion pressure. CNS response to hypotension is often blunted.
- MAP should be kept between 85 and 90 mm Hg to maintain spinal cord perfusion [8, 9].
- Temperature should be monitored and patients may require warming via blankets and fluids due to poikilothermia.
- Patients are often anticoagulated for DVT prophylaxis due to immobility for 3 months after acute injury as the risk is as high as 85% without prophylaxis [7]. Prophylaxis is not routinely indicated 3 months after acute spinal injury. This is due to the counteraction of venous stasis by muscle spasms of the lower limbs, femoral artery atrophy, and venous stability.
- Volatile and IV anesthetics should be used judiciously based on the hemodynamic state of the patient.
- Muscle relaxants used based on operative site (preferred agents have sympathomimetic effects such as pancuronium).
- Denervated muscles are more susceptible to excessive potassium release upon administration of depolarizing blocking agents during the first 6 months; therefore, use of non-depolarizing agents are indicated. This can lead to fatal cardiac dysrhythmias. Succinylcholine does not provoke excessive release of potassium during the first few hours after spinal cord injury and its short onset and duration are beneficial.
- Due to intercostal muscle and diaphragmatic weakness, patients are in a hypoventilatory state after general anesthesia. Patients may require prolonged intubation.

Cerebrovascular Accident

- Cerebrovascular accident (CVA), also known as stroke, is a sudden onset of neurological deficits that occurs secondary to cerebral ischemia or cerebral hemorrhage.
- Transient ischemic attack (TIA) – sudden onset of focal neurological deficits that resolve quickly (usually within 24 hours).
- Reversible ischemic neurological deficit (RIND) – neurological deficit that lasts >24 hours but resolves in <3 weeks.
- Risk factors – hypertension, smoking, atrial fibrillation, diabetes, ischemic heart disease, peripheral vascular disease, increased homocysteine levels, excessive alcohol consumption.
- Patients with carotid bruits, known carotid artery stenosis, or vertebrobasilar insufficiency are prone to the development of ischemic stroke.
- Common symptoms – hemiplegia, aphasia, dysarthria, hemineglect, seizures, and gait disturbances. In hemorrhagic strokes, headache, loss of consciousness, nausea, and vomiting are common. Symptoms manifest clinically based on the anatomical area of neurological involvement (e.g., hemiplegia due to corticospinal tract involvement).

- Craniomaxillofacial manifestations – include facial droop, ptosis, ocular muscle weakness, weakened parapharyngeal muscles (weakened gag and swallowing reflex), anosmia, visual field deficits, and disturbances in tongue movement.
- Aphasia
  - Broca's aphasia – disturbance in a patient being able to articulate/express themselves.
  - Wernicke's aphasia – disturbance in comprehension resulting in patients speaking fluently without making sense.
- Two categories of stroke
  - Ischemic stroke – CVA that occurs secondary to cerebral ischemia due to the narrowing or occlusion of the cerebral arterial vasculature. This can occur secondary to a thrombus forming in an atherosclerotic carotid artery. It can also occur secondary to an embolus as in the case of a mural thrombus in the setting of atrial fibrillation.
  - Hemorrhagic stroke – CVA that occurs secondary to leakage or rupture of cerebral vessels. This can occur secondary to subarachnoid hemorrhage, arteriovenous malformations, and/or bleeding secondary to anticoagulation.

Treatment of Transient Ischemic Attack (TIA)

- Patients with TIAs typically have a normal neurological examination, but should be evaluated and treated urgently due to an increased risk of future stroke development.
- Workup for TIA
  - Neuroimaging – CT scan without contrast
  - Echocardiography (rule out potential cardiac source of emboli)
  - Vascular imaging – carotid Doppler, CTA, MRA
  - Magnetic resonance imaging
  - Coagulopathy workup for younger patients
  - CBC, coagulation studies, pregnancy test (females in child-bearing age group), cardiac enzymes, and CMP
- Symptomatic atheromatous lesions
  - 70–99% occlusion – benefit from carotid endarterectomy (CEA) [10, 11].
  - Complete occlusion – 100% stenotic lesion makes it impossible for any surgery.
  - Consider arterial stenting/angioplasty in symptomatic patients with <50% stenosis and in those in which CEA may be contraindicated [12].
- Anticoagulation (e.g., warfarin, apaxiban, rivoroxaban, dabigatran) in the setting of chronic atrial fibrillation.
- Antiplatelet drugs (e.g., aspirin, clopidogrel, aspirin/ dipyridamole) in the setting of peripheral vascular disease.
- Statins reduce risk of stroke even in the absence of hyperlipidemia.

Treatment of Ischemic Stroke

- Patients with acute ischemic stroke may be candidates for intravenous (IV) thrombolytics, which must be administered ideally within a 3-hour window of stroke onset to be effective. The window can be increased to 4.5 hours in select patients.
  - IV tissue plasminogen activator (tPA) in patients who meet criteria and in whom treatment can begin within 3–4.5 hours from initial symptom.
  - Conditions prohibiting tPA include presence of hemorrhage on a brain CT, uncontrollable hypertension, extreme hypoglycemia or hyperglycemia, concurrent use of warfarin, or increased bleeding risk from recent surgical or invasive surgical procedures.
- Maintain adequate oxygenation.
- Endovascular thrombectomy may be indicated in specific cases.
- Maintain adequate blood pressure. Rapid decrease of blood pressure can lead to decreased cerebral perfusion, which could

worsen cerebral ischemia. BP should not exceed 185/110 mm Hg, especially while using thrombolytic therapy [13].
- Glucose level should be monitored. Hyperglycemia and hypoglycemia should be avoided.
- Intubation should be considered for patients that cannot maintain a patent airway in the setting of decreased protective airway reflexes.
- Maintain normothermia with antipyretics as well as cooling blankets when needed as hyperthermia may worsen neurological injury.
- Initiate deep venous thrombosis/pulmonary embolism prophylaxis.
- Initiate antithrombotic therapy within 48 hours.
- Transfer the patient to stroke unit.

Treatment of Hemorrhagic Stroke

- Discontinue and reverse all anticoagulants and/or antiplatelet drugs if possible.
- Consider administration of fresh frozen plasma (FFP) or prothrombin complex concentrate (PCC) for patients that are coagulopathic.
- Neurosurgical consultation to assess whether evacuation or placement of an external ventricular drain (EVD) is beneficial in the setting of increased cerebral pressure (ICP).
- The target MAP from intracranial hemorrhage without increased ICP is 110 mm Hg. If there is an increase in ICP, the ICP should be monitored and titrated to maintain CPP between 60 and 80 mm Hg [5].
- If patient is intubated, mechanical hyperventilation helps reduce ICP.
- Elevated ICP can also be managed with mannitol or hypertonic saline; avoid hypotonic solution and hypervolemia.
- Maintain normothermia with antipyretics as well as cooling blankets when needed as hyperthermia can worsen neurological injury.
- Glucose level should be monitored. Hyperglycemia and hypoglycemia should be avoided.
- Initiate pneumatic compression devices for deep venous thrombosis/pulmonary embolism prophylaxis upon admission.
- Transfer the patient to stroke unit.
- The risk of a major cardiovascular event, including a new ischemic stroke, acute myocardial infarction, or cardiovascular mortality is most increased in the first 9 months following an ischemic stroke. After 9 months, the risk plateaus at a stable yet still elevated level [14].

Patient Management

- Identify high-risk patients (e.g., risk factors and recent CVA).
  - The patient should be considered ASA class II if patient had a CVA >6 months earlier and has no evidence of residual neurologic deficit.
  - The patient should be considered ASA class III if patient had a CVA >6 months earlier and has some evidence of residual neurologic deficit.
  - The patient should be considered ASA IV if patient had a CVA <6 months earlier or if substantial residual deficit remains [15].
- Elective treatment should be delayed if the risk is high and proper preoperative treatment has yet to be rendered to decrease risks (e.g., anticoagulation and carotid endarterectomy). The previous dogma was to delay elective procedures for 6 months. However, the latest evidence shows that patients may undergo elective treatments if they are stable and risk factors are controlled.
- Consult with PCP and neurologist if preoperative treatment has been rendered and stroke occurred >3 months earlier.
- Maintain adequate blood pressure. It is paramount to prevent rapid decreases in blood pressure as this may lead to cerebral ischemia.
- Judicious use of sympathomimetic agents (e.g., ketamine and local anesthesia with epinephrine) to prevent hypertension is paramount.

- Maintain adequate oxygenation.
- Monitor ECG.
- Discuss holiday from anticoagulants with physician in the setting for procedures where significant bleeding is expected.
- Certain stroke patients may be at risk of aspiration if the neurological lesion involved the bulbar nuclei with residual swallowing/gag deficits.
- Be cognizant of residual deficits and the potential implications (e.g., hemiplegia patients are at a higher risk of DVT due to difficulty with ambulation).

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Electrolyte and Acid-Base Disturbances

20

Simon Bangiyev, Dustin R. Altmann, Tarek Korban, and Damian Findlay

Definition/Pathophysiology

- Normal arterial blood pH is 7.35–7.45, and maintenance of this physiologic acid-base balance is critical to biochemical reactions in the body. In physiological solutions, an acid is a compound that contains hydrogen and reacts with water to produce hydrogen ions [H^+] and a base reacts with water to form hydroxide ions [OH^-] [1].
- Acidosis is defined as arterial blood pH <7.35 and is considered severe when pH <7.20. Severe acidosis produces the following [2]:
  - Hypotension – direct myocardium and smooth muscle depression, reducing cardiac contractility and peripheral vascular resistance.
  - Hypoxia of tissues (despite rightward shift in Hb-O_2 dissociation curve).
  - Ventricular fibrillation threshold is decreased.
  - Hyperkalemia – K^+ moves extracellularly in exchange for H^+ moving intracellularly.
  - CNS depression (confusion, loss of consciousness (LOC), seizures) – more pronounced in respiratory than metabolic acidosis.
- Alkalosis is defined as arterial blood pH >7.45 and is considered severe when pH >7.60. Physiologic effects of alkalosis include [2] the following:
  - Hypoxia of tissues – hemoglobin has increased affinity for O_2 giving up less O_2 to tissues (Hb-O_2 dissociation curve shifts to the left).
  - Hypokalemia – H^+ moves extracellularly shifting K^+ intracellularly.
  - Hypocalcemia – increased Ca^{2+} binding to plasma proteins, decreasing serum Ca^{2+} causing cardiovascular depression and neuromuscular irritability.

Compensatory mechanisms – protection of physiologic pH, which occurs in three phases (Table 20.1):

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Table 20.1 Acid-base disorders and compensatory responses

Disorder	Primary change	Compensatory response
Respiratory		
Acidosis	Increased $PaCO_2$	Increased HCO_3–
Alkalosis	Decreased $PaCO_2$	Decreased HCO_3–
Metabolic		
Acidosis	DecreasedHCO_3–	Decreased $PaCO_2$
Alkalosis	IncreasedHCO_3–	Increased $PaCO_2$

- Chemical Buffering [1–3]
  - Bicarbonate (H_2CO_3/HCO_3^-) – most important for extracellular fluid (ECF) buffering.
    $H_2O + CO_2 \leftarrow$Carbonic anhydrase$\rightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$
    Effective against metabolic, but *not* respiratory acid-base disorder.
  - Hemoglobin (HbH/Hb^-) – important buffer in blood.
  - Intracellular proteins (PrH/Pr^-) – intracellular buffering.
  - Phosphates ($H_2PO_4^-/HPO_4^{2-}$) and ammonia (NH_3/NH_4^+) – urinary buffers.
  - Bone buffering:
    Acidic conditions will demineralize bone causing the release of alkaline compounds ($CaCO_3$ and $CaHPO_4$).
    Alkaline conditions will increase deposition of carbonate in bone.
- Respiratory compensation – whenever possible.
  - Ventilatory changes are mediated by chemoreceptors in the brainstem.
    Minute ventilation increases with acidosis → "blowing off" CO_2 to increase pH.
    Minute ventilation decreases with alkalosis → retain CO_2 to decrease pH.
- Renal compensation – slower but more effective.
  - Kidneys regulate bicarbonate (HCO_3^-) reabsorption/excretion, form new HCO_3^-, eliminate titratable acids and ammonium ions.

Blood Gas Analysis

- Arterial or venous blood is collected in a heparin-coated syringe, air bubbles eliminated, placed on ice, and analyzed as soon as possible. Serial examinations are necessary.
- Arterial Blood Gas (ABG):
  - Most commonly utilized "gold standard."
  - Invasive – risk of nerve injury or hematoma.
- Venous Blood Gas [1]:
  - PO_2 represents tissue extraction, not pulmonary function.
  - PCO_2 usually 4–6 mm Hg higher than $PaCO_2$, except in case of severe shock, or $PaCO_2$ >45 mm Hg.
  - pH is usually 0.03–0.04 lower than arterial pH.
  - Bicarbonates, lactates, and base excess are similar to ABG.

Blood Gas Interpretation

- Correlate changes in pH with changes in CO_2 or HCO_3 (Tables 20.1 and 20.2)
  - Respiratory disorder → pH and CO_2 change in the opposite direction.
    Each 10 mm Hg change in CO_2 should change arterial pH by about 0.08 in the opposite direction.
  - Metabolic disorder → pH and CO_2 change in the same direction.
    Each 6 mEq change in HCO_3 also changes arterial pH by 0.1 in the same direction.

Table 20.2 Acid-base disorders' diagnosis

pH				
	→ Increased	$PaCO_2$	→ Increased	Metabolic alkalosis
			→ Decreased	Respiratory alkalosis
	→ Decreased	$PaCO_2$	→ Increased	Respiratory acidosis
			→ Decreased	Metabolic acidosis

- If the pH change is greater or less than predicted, a mixed acid-base disorder is present.
- In metabolic acidosis, calculate the plasma anion gap. In metabolic alkalosis, measure urinary chloride.

Metabolic Acidosis

- Defined as a primary *decrease in bicarbonate* [HCO_3^-]. It is further categorized as anion gap or non-ion gap acidosis.
- Anion gap in plasma is defined as the difference between the *measured* cations (Na^+) and *measured* anions (Cl^- and HCO_3^-). In actuality it represents the difference in *unmeasured* anions and cations. Because electroneutrality must be maintained, a true anion gap cannot exist.
  - Increased anion gap is the result of:
    Either increased unmeasured cations such as K^+, Ca^{2+}, Mg^{2+}
    Or decreased unmeasured anions such as plasma proteins (albumin), lactic acids, keto acids, phosphates, sulfates.
- Causes:
  - Anion gap "MUDPILERS" mnemonic – caused by either the ingestion of toxins or increased production of endogenous non-volatile acids.
    *M*ethanol ingestion
    *U*remia – renal failure causes the inability to excrete non-volatile acids
    *D*iabetic ketoacidosis, starvation ketoacidosis
    *P*araldehyde, paracetamol/acetaminophen ingestion
    *I*ron, isoniazid ingestion
    *L*actic acidosis
    *E*thanol, ethylene glycol ingestion
    *R*habdomyolysis
    *S*alicylate/aspirin ingestion
  - Non-ion gap (hyperchloremic) – primarily either GI or renal wasting of bicarbonate.
    GI losses of HCO_3^- – diarrhea, intestinal/pancreatic fistulas, and ileal obstruction.
    Renal losses of HCO_3^- – renal tubular acidosis, hypoaldosteronism.
    Dilutional – rapid and large volume bicarbonate-free fluid (0.9% NaCl).
- Respiratory compensation – *Kussmaul's breathing:* hyperventilation in response to acidemia.
  - Decreased blood pH stimulates respiratory centers to increase minute ventilation, which in turn lowers $PaCO_2$ by "blowing off" CO_2, shifting pH toward normal.
- Treatment – correction of underlying cause:
  - ESRD – hemodialysis.
  - Lactic acidosis – supplemental O_2, fluid resuscitation, circulatory support.
  - DKA – IV fluids, insulin.
  - Hemorrhage – RBC transfusion: Hb buffers both CO_2 (carbonic acid) and nonvolatile acids.
  - Sodium bicarbonate ($NaHCO_3$) – effective in non-gap metabolic acidosis because problem is bicarbonate loss. It is not effective in anion gap acidosis.

Metabolic Alkalosis

- Defined as a primary *increase in bicarbonate* [HCO_3^-]. Metabolic alkalosis can be subdivided into chloride-sensitive and chloride-resistant metabolic alkalosis.
  - Chloride-sensitive alkalosis is associated with *extracellular fluid depletion* and NaCl deficiency. Sodium (Na^+) and volume depletion cause bicarbonate (HCO_3^-) to be reabsorbed in the kidney because physiologic maintenance of ECF volume is given priority over acid-base balance. Diuretics are the most common cause of chloride-sensitive metabolic alkalosis.
  - Chloride-resistant alkalosis is associated with *extracellular fluid expansion* secondary to mineralocorticoid excess, causing increased

aldosterone-mediated Na^+ and ECF reabsorption in exchange for H^+ secretion.

- Causes:
  - Chloride-sensitive alkalosis – *ECF depletion* and NaCl deficiency.
    GI – vomiting, gastric drainage (continuous NG suctioning)
    Renal – diuretics, post-hypercapnic, low chloride intake
    Sweat – cystic fibrosis
  - Chloride-resistant alkalosis – *ECF expansion* and enhanced mineralocorticoid activity.
    Hyperaldosteronism, refeeding syndrome
  - Miscellaneous
    Citrate in blood products after massive transfusion, acetate-containing colloid solutions, alkaline administration with renal insufficiency, hypercalcemia (bone metastases), sodium penicillins, glucose feeding after starvation.
- Respiratory compensation – hypoventilation.
  - Increased blood pH depresses respiratory centers, retaining CO_2 and increasing pH.
  - Less predictable than the ventilatory response to metabolic acidosis because hypoxemia will activate oxygen chemoreceptors stimulating ventilation.
- Renal compensation
  - Bicarbonate is able to be rapidly excreted in large quantities by the kidneys.
- Treatment:
  - Correction of the underlying disorder.
  - Ventilatory control to decrease minute ventilation and correct respiratory component.
  - Chloride-sensitive treatment, in the setting of volume depletion:
    IV saline (NaCl) and potassium (KCl).
    H_2-blocker therapy for excessive gastric fluid loss.
  - Chloride-resistant treatment, in the setting of volume overload:
    Aldosterone antagonist (spironolactone).
  - Severe metabolic alkalosis, pH >7.6
    Hemodialysis.
    IV hydrochloric acid, ammonium chloride, or arginine hydrochloride.

Respiratory Acidosis

- Defined as a primary *increase in $PaCO_2$*. The increase in arterial CO_2 is due to either increased production or reduced ventilation. The increase in CO_2 drives the bicarbonate reaction to the right ($H_2O + CO_2 \leftrightarrow H_2CO_3 \leftrightarrow H^+ + HCO_3^-$) increasing H^+ and decreasing pH, but [HCO_3^-] is minimally affected.
- Causes:
  - Hypoventilation [1, 2]:
    CNS depression – anesthetics, OSA, cerebral trauma
    Neuromuscular disorders
    Chest wall restriction – obesity, kyphoscoliosis, burns
    Upper airway obstruction – laryngospasm, foreign body, tumor, OSA
    Lower airway obstruction – asthma, COPD, tumor
    Lung disease – aspiration, pneumonia, PE, fibrosis, pulmonary edema, ARDS
    Ventilator malfunction – rebreathing from exhausted soda lime, incompetent one-way valve
  - Increased CO_2 production:
    Malignant hypothermia
    Thyroid storm
    Sepsis
    Prolonged seizure activity
    Extensive burn injury
- Acute compensation is limited; chronic renal compensation is usually fully compensated.
- Confusion, headache, and fatigue are common. Severe respiratory acidosis leads to CNS depression termed CO_2 narcosis.
- Renal compensation:
  - Increased reabsorption of HCO_3^- (bicarbonate).
  - Increased excretion of titratable acids. Hydrogen phosphate (HPO_4^{2-}) is secreted and combines with H^+ to be eliminated in the urine as $H_2PO_4^-$ as part of the phosphate buffer.

  - Increased renal production of ammonia (NH_3). The NH_3 combines with H^+ to be eliminated in the urine as NH_4^+.
- Treatment:
  - Increasing alveolar ventilation will "blow off" excess CO_2, restoring balance between production and ventilation.
    Mechanical ventilation for severe acidosis (pH <7.2), CO_2 narcosis, and respiratory muscle fatigue.
  - Supplemental oxygen for concurrent hypoxemia.
  - Correct underlying respiratory cause:
    Bronchodilation, narcotic reversal, diuresis for lung compliance.
  - Reducing CO_2 production is only useful in specific cases. E.g., Dantrolene for MH or antithyroid medication for thyroid storm.
  - Patients with chronic respiratory acidosis may have a predominantly hypoxic respiratory drive, not a hypercarbic respiratory drive. The goal is to avoid over-ventilation and return $PaCO_2$ to baseline. Normalizing $PaCO_2$ to 40 mm Hg may produce relative hyperoxia and cause severe hypoventilation.

Respiratory Alkalosis

Defined as a primary *decrease in* $PaCO_2$. This is a result of hyperventilation relative to CO_2 production. It is considered severe when pH >7.60.

- Causes [1–3]:
  - Central stimulation – pain, anxiety, ischemia, stroke, tumor, infection, fever, drug-induced.
  - Peripheral stimulation – hypoxemia, altitude, asthma, pulmonary edema, CHF, PE, severe anemia.
  - Unknown mechanism – sepsis, metabolic encephalopathy.
  - Iatrogenic – ventilator induced.
- Renal compensation:
  - Bicarbonate can be rapidly excreted in large quantities by the kidneys.
- Treatment:
  - Correction of the underlying process.
  - In rare cases, severe respiratory alkalosis may require sedation.

Management of the Patient with Acid-Base Disorders

- Elective surgeries should be postponed until the underlying cause is corrected.
- Acidosis can potentiate the effects of most anesthetic agents depressing the CNS and cardiovascular systems.
- Succinylcholine should be avoided in hyperkalemia acidosis to avoid further increases in potassium.
- Acidosis lowers seizure threshold.
- Metabolic acidosis (lactic acidosis) contributes to coagulopathy leading to the potential for increased bleeding.
- Alkalosis in combination with hypokalemia can cause severe arrhythmias. It may potentiate nondepolarizing muscle relaxers, but may be related to concomitant hypokalemia. The consequences of severe alkalosis are more prominent with respiratory than metabolic alkalosis.
- Respiratory alkalosis prolongs opioid-induced respiratory depression.
- Respiratory alkalosis causes cerebral and coronary ischemia from arteriolar vasoconstriction.

Electrolyte Disturbances

Disorders of Sodium

Hyponatremia

- Sodium concentration less than 135 mEq/L.
- Acute hyponatremia is hyponatremia that has developed in less than 48 hours (chronic >48 hours).
- Hyponatremia can be classified into mild (130–135 mEq/L), moderate (120–130 mEq/L), or severe (<120 mEq/L).
- Normal blood sodium range is between 135 and 145 mEq/L.

Causes of Hyponatremia [4]

- Most cases are due to impairment in renal water excretion, most often due to an inability to suppress ADH secretion as in syndrome of inappropriate antidiuretic hormone (SIADH). SIADH is often associated with pulmonary pathology, malignancies, and CNS infections/tumors.
- Volume depletion seen in diarrhea and vomiting.
- Heart failure.
- Cirrhosis.
- Hypothyroidism.
- Pregnancy.
- Thiazide diuretics, selective serotonin reuptake inhibitor (SSRIs), MDMA.
- Polydipsia: a large intake of water – seen in psychiatric illnesses and in competitive athletes.
- Advanced renal failure.
- Mannitol exposure.
- Hyperglycemia that leads to pseudohyponatremia.
- Hyponatremia due to hyperlipidemia or hyperproteinemia.

Signs and Symptoms of Hyponatremia [5]

- Primarily neurologic symptoms – irritability, seizures, confusion, lethargy, which are secondary to neurologic dysfunction induced by cerebral edema – provoked by hypoosmolality – can lead to osmotic demyelination.
- Other symptoms include muscle cramps, nausea, and fatigue.
- Signs include decreased skin turgor, low jugular venous pressure, orthostatic hypotension, signs of heart/liver failure.

Treatment [5, 6]

- In cases of hypovolemic hyponatremia (most common), volume resuscitation with normal saline is indicated. Correction of volume is initiated prior to correction of osmolality.
- For asymptomatic patients, infuse a 50 ml bolus of 3% saline to prevent further decrease in sodium.
- In symptomatic patients, infuse a 100 ml bolus of 3% saline over 10 minutes. Boluses may be repeated.
- The maximum rate of correction should be 8 mEq/L in a 24-hour period to avoid osmotic demyelination syndrome (ODS), which can lead to seizures, confusion, paralysis, and cessation of central respiratory drive.
- Raising serum sodium by 4–6 mEq/L generally alleviates symptoms and prevents herniation.

Hypernatremia

- Sodium concentration more than 145 mEq/L.
- Normal blood sodium level is between 135 and 145 mEq/L.

Causes of Hypernatremia [4]

- Usually due to loss of water from vomiting, diarrhea, burns, sweating and diuretics causing hypovolemic hypernatremia. (hypovolemia = water and solute loss).
- Diabetes insipidus (kidney is unable to reabsorb water) and pronounced insensible losses (e.g., sweating and respiratory tract). Causes euvolemic hypernatremia. (dehydration = water loss).
- Hyperaldosteronism and Cushing's syndrome. Both cause hypervolemic hypernatremia due to salt and water retention.
- Renal failure that causes hypervolemic hypernatremia.

Signs and Symptoms of Hypernatremia [5]

- Lethargy, weakness, muscle weakness, tremors, irritability, and seizures if severe (osmolality causes water diffusion out of the brain).

Treatment [2, 3]

- In cases of hypovolemic hypernatremia, volume resuscitation with normal saline is indicated until the patient is euvolemic. The plasma osmolality is then corrected with 5% dextrose or 0.45% normal saline.
- In cases of hypervolemic hypernatremia (e.g., hyperaldosteronism), treat the underlying cause and diuresis with a loop diuretic. Dialysis may be indicated in the setting of renal failure.
- Patients with euvolemic hypernatremia will require 5% dextrose in water intravenously

(rate of 3–6 mL/kg/hour for acute and 1.35 mL/kg/ hour for chronic). Patients with diabetes insipidus will also require desmopressin therapy (1–2 micrograms).
- The goal of therapy is to lower the serum sodium by 1–2 mEq per hour. Normonatremia should be restored slowly to prevent cerebral edema.

Disorders of Potassium

Hypokalemia
- Potassium concentration less than 3.5 mEq/L (normal blood level is 3.5–5.5 mEq/L).

Causes of Hypokalemia [4]
- Uncontrolled diabetes leading to DKA
- Gastrointestinal or urinary losses – vomiting, excessive nasogastric suction, diarrhea, insulin administration, or diuretics (most common cause)
- Hypothermia
- Antipsychotics: risperidone, quetiapine
- Mineralocorticoids
- Respiratory or metabolic acidosis

Signs and Symptoms of Hypokalemia [5]
- Below 3.0 meq: GI complications including ileus, nausea, vomiting, and abdominal distension.
- Below 2.5 meq: muscle weakness which can lead to rhabdomyolysis and respiratory failure due to diaphragmatic weakness.
- Can be associated with magnesium depletion.
- EKG abnormalities: ST depression, decrease of the T wave, U wave increase, prolongation of the QT interval. Can lead to torsades de pointes (especially in the setting of magnesium deficiency) and ventricular tachycardia.

Treatment [5, 6]
- The goal is to prevent life-threatening complications, treat the underlying cause, and replete the potassium deficit.
- Treat magnesium deficit, as potassium treatment will be refractory in the setting of hypomagnesemia. Hypomagnesemia prevents potassium excretion into the ECF by impacting the Na/K/ATPase pump and ROMK potassium channels.
- Oral potassium raises the serum potassium by 1–1.5 mEq/L after an oral dose of 40–60 mEq.
- Intravenous potassium (potassium chloride) raises the serum potassium 0.25 mEq/L after administration of 20 mEq iv. Should be administered at a rate of 10 mEq/hour in peripheral veins to prevent venous irritation. Higher doses may be given via central line.
- Consider potassium-sparing diuretics when indicated.
- Monitor physiologic effects (e.g., muscle weakness and EKG changes).

Hyperkalemia
- Potassium concentration more than 5.5 mEq/L (normal blood level is 3.5–5.5 mEq/L).

Causes of Hyperkalemia [4]
- Either from increased ingestion of K (rare).
- Impaired K excretion (e.g. kidney disease).
- Metabolic acidosis – excess hydrogen ions in the cells leads to potassium movement into the extracellular fluid.
- Insulin deficiency – insulin promotes movement of K into cells. Total body may be low in the setting of vomiting seen in DKA (redistributive hyperkalemia due to movement of K from cells to ECF).
- Increased tissue catabolism (trauma, tumor lysis syndrome).
- Heparin administration.
- Beta-blockers.
- Red cell transfusion.
- Succinylcholine use for patients with extensive trauma, burns, chronic infection, or neuromuscular junction disorders.
- Digoxin poisoning.
- For impaired K secretion, the causes are reduced aldosterone secretion, aldosterone resistance, and reduced distal tubules sodium and water delivery.
- Drugs that interfere with the RAAS or use of potassium-sparing drugs.

- Pseudohyperkalemia may occur due to RBC lysis in lab collection tube.

Signs and Symptoms of Hyperkalemia [5]

- Malaise and gastrointestinal disturbances including ileus, nausea, vomiting.
- Cardiac sequelae and respiratory failure due to diaphragmatic weakness.
- EKG abnormalities: ST depression, peaked T wave, U wave increase, bradycardia, bundle branch blocks, prolongation of QRS complex and PR interval. Can lead to torsades de pointes (especially in the setting of magnesium deficiency) and ventricular tachycardia.

Treatment of Hyperkalemia [5, 6]

- The goal is to prevent life-threatening complications, treat the underlying cause and decrease the serum potassium.
- Administration of calcium gluconate (1000 mg over 2–3 minutes intravenously) to antagonize/stabilize the cardiac membrane.
- 10 U insulin administration with ampule of 50% dextrose (D50) to prevent hypoglycemia to shift K intracellular.
- Beta-2 adrenergic agonists to shift potassium intracellular. E.g., salbutamol 10–20 mg nebulized.
- 1–2 amp sodium bicarbonate (if metabolic acidosis) over 10 minutes to shift K intracellular.
- Dialysis for patients with renal impairment.
- Loop diuretics to excrete K via kidneys.
- Gastrointestinal cation exchangers (e.g., patiromer and sodium polystyrene sulfonate).
- Kayexalate.
- Monitor physiologic effects (e.g., muscle weakness and EKG changes).

Disorders of Calcium

- Normal total corrected calcium range is 8.5–10.2 mg/dl. Measurements should be based on the corrected calcium levels as low albumin states can result in lower serum calcium levels.
- Corrected calcium = measured calcium (mg/dl) + 0.8 [4−albumin (mg/dl)].

Hypocalcemia

- Calcium concentration less than 8.5 mg/dl of the total corrected calcium levels.

Cause of Hypocalcemia [4]

- Inadequate PTH secretion (e.g., hypoparathyroidism)
- Vitamin D deficiency or resistance
- Diuretics (loop)
- CKD
- Pancreatitis
- Tumor Lysis syndrome
- Post-op parathyroidectomy or thyroidectomy
- Rhabdomyolysis
- Abnormal magnesium metabolism (induces PTH resistance or deficiency)

Signs and Symptoms of Hypocalcemia [5]

- Perioral and acral paresthesias
- Carpopedal tetany
- Trousseau's sign – carpal spasm elicited by inflating a blood pressure cuff
- Chvostek's sign – facial muscle spasms elicited by tapping on the facial nerve in the preauricular region
- Exaggerated tendon reflexes
- Seizures
- EKG abnormalities: prolongation of the QT interval. Can lead to torsades de pointes (especially in the setting of magnesium deficiency) and ventricular tachycardia

Treatment [5, 6]

- The goal is to prevent life-threatening complications, treat the underlying cause, and replete the calcium deficit.
- Treat magnesium deficit, as calcium treatment will be refractory in the setting of hypomagnesemia.

- Measurements to assess and then treat the underlying cause: PTH, vitamin D, magnesium, creatinine, amylase
- Oral calcium for mild/chronic hypocalcemia – calcium carbonate or calcium citrate in an oral dose of 500 mg QID.
- Intravenous calcium (calcium gluconate) – 10% calcium gluconate in normal saline or 5% dextrose (1000 ml solution) is given at 50 ml/hour that is the equivalent of 50 mg per hour.
- Monitor physiologic effects (e.g., muscle weakness and EKG changes).

Hypercalcemia

- Calcium concentration greater than 10.2 mg/dl of the total corrected calcium levels.

Causes of Hypercalcemia [4]

- Most common causes are from primary hyperthyroidism (elevated PTH) and malignancies (breast cancer, PTH-related protein, bone breakdown, or bony metastasis).
- Impaired vitamin D metabolism (i.e., kidney disease).
- Sarcoidosis.
- Milk-alkali syndrome.
- Vitamin D intoxication.
- Thiazide diuretics.
- Lithium.
- Familial hypocalciuric hypercalcemia.
- Paget disease.

Signs and Symptoms of Hypercalcemia [5]

- Pneumonic (stones, bones, moans, and psychiatric overtones)
- Malaise and gastrointestinal disturbances including ileus, nausea, vomiting, constipation (moans)
- Hypotonia
- Depressed tendon reflexes
- Bone pain secondary to osteitis fibrosa (bones)
- Cognitive deficits, anxiety, depression (psychiatric overtones)
- EKG abnormalities: shortening of QT interval and ST segment
- Polydipsia and nephrolithiasis (stones) and renal colic

Treatment

- The goal is to prevent life-threatening complications, treat the underlying cause, and decrease the serum calcium.
- Measurements to assess and then treat the underlying cause: PTH, vitamin D, creatinine.
- Monitor physiologic effects (e.g., muscle weakness and EKG changes).
- Mild cases with no symptoms may just require treatment of the underlying cause and adequate oral hydration.
- Severe hypercalcemia requires:
  - Volume expansion with isotonic saline to cause a diuresis (initial rate of 200–300 mL/hour).
  - Loop diuretic in the setting of renal/heart failure (e.g., furosemide).
  - Salmon calcitonin (4 IU/kg).
  - Zoledronic acid 4 mg intravenously, pamidronate 60–90 mg intravenously over 2 hours, or denosumab 60 mg subcutaneously. Patients should be informed of the risk of medication related osteonecrosis of the jaw (MRONJ).
  - Dialysis.

Management of Patients with Electrolyte Disturbances

- When possible, electrolyte should be corrected preoperatively to decrease perioperative morbidity and mortality.
- In the case of emergency surgery, the following strategies must be employed/considered:
  - Continuous EKG monitoring to assess for unstable cardiac rhythms and be prepared to treat precarious rhythms.
  - Baseline labs and frequent measurements of intraoperative labs to assess correction.
  - Consider invasive hemodynamic monitoring to assess cardiac function and to guide fluid resuscitation. This is also helpful

should diuresis in the setting of hypernatremia and hypercalcemia be indicated. [2]
- Some electrolyte disturbances (e.g., hyponatremia and hypokalemia) have a hypovolemic component. Hypovolemia will be exacerbated by the induction of anesthesia. This should be managed judiciously with vasopressors, fluids, and inotropes [5].
- Repletion of magnesium as it can impact calcium and potassium repletion.
- Avoid rapid correction of sodium to prevent ODS [6].
- Avoid hyperventilation, which can exacerbate hypokalemia. Hypokalemia can prolong the effects of neuromuscular blocking drugs due to its effect on skeletal muscle [5].
- Avoid use of succinylcholine and lactated ringers as both can raise serum potassium, which can be detrimental in the setting of hyperkalemia [5].
- Respiratory and metabolic acidosis can also exacerbate hyperkalemia.
- Low calcium levels can precipitate laryngospasm.

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Psychiatric Disease

21

Marc Dentico-Olin and Damian Findlay

Mood Disorders

Major Depressive Disorder (MDD)

- Depressed mood that lasts at least 2 weeks with a constellation of associated symptoms.
- Signs and symptoms (SIGECAPS mnemonic for remembering associated symptoms):
  - *S*leep (increased sleep during the day or decreased sleep at night)
  - *I*nterest (loss of interest)
  - *G*uilt (feeling of worthlessness)
  - *E*nergy (lack of energy – fatigue)
  - *C*ognition/concentration (reduced cognition or difficulty in concentration)
  - *A*ppetite (increased or decreased appetite)
  - *P*sychomotor (anxiety or lethargy)
  - *S*uicide (ideation)

Treatment for MDD

- Psychotherapy – cognitive behavioral therapy.
- Electroconvulsive therapy.
- Antidepressants:
  - Selective serotonin reuptake inhibitors (SSRIs) – prevent reuptake of serotonin in the synaptic cleft that increases the amount of serotonin, which enhances neurotransmission, e.g., citalopram, escitalopram, fluoxetine, paroxetine, and sertraline.
  - Selective serotonin – norepinephrine reuptake inhibitors (SNRIs) – prevent reuptake of serotonin and norepinephrine in the synaptic cleft that increases the amount of serotonin and norepinephrine, which enhances neurotransmission, e.g., duloxetine, venlafaxine, desvenlafaxine, milnacipran and levomilnacipran.
  - Tricyclic antidepressants (TCAs) – prevent reuptake of serotonin and norepinephrine in the synaptic cleft that increases the amount of serotonin and norepinephrine, which enhances neurotransmission, e.g., clomipramine, imipramine, amitriptyline, nortriptyline, and protriptyline.
  - Monoamine oxidase inhibitors (MAOIs) – inhibit the activity of monoamine oxidase, thus preventing the breakdown of monoamine neurotransmitters such as serotonin, epinephrine, and norepinephrine, e.g., isocarboxazid, nialamide, phenelzine, tranylcypromine, bifemelane, moclobemide, pirlindole, rasagiline, and selegiline.

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Bipolar Disorder (aka Manic Depression)

- Mental disorder with periods of depression and mania (elevated mood).
- Signs and symptoms of mania:
  - Inflated self-esteem/grandiosity.
  - Insomnia.
  - Pressured speech.
  - Engaging in risky behavior despite consequences (e.g., exuberant shopping, gambling, and engaging in risky sexual behavior).
  - Racing thoughts.

Treatment for Bipolar Disorder

- Patients are usually on an antidepressant and a mood stabilizer.
- Mood stabilizers include lithium, anticonvulsants, and antipsychotics.
- Lithium – alkali metal used for manic management. Lithium may cause diabetes insipidus. Lithium toxicity can manifest as cardiac toxicity (AV blocks, sinus bradycardia, ventricular irritability), seizures, and hypotension. Narrow therapeutic index (0.6–1.2 mmol/L). Avoid steroids and ibuprofen in these patients as these may increase lithium bioavailability.
- *Anticonvulsant mood stabilizers*
  - Carbamazepine
  - Valproic acid
  - Lamotrigine
  - Topiramate
- *Antipsychotic mood stabilizers*
  - Olanzapine

Serotonin Syndrome

- An adverse drug reaction producing excess serotonergic effects of the central nervous system [1, 2].
- Serotonin syndrome typically occurs shortly after an increase in the dose of a serotonin agonist (a MOI or an SSRI inhibitor) or after the addition of a second serotonergic agent, such as tramadol or dextromethorphan [3, 4].
- Occurs rapidly, normally within 24 hours.
- It is typically characterized by a triad of changes in mental status, neuromuscular activity, and autonomic function.
- It is a clinical diagnosis, as there is no lab test that is confirmatory. Serotonin blood levels do not correlate with clinical symptoms [1].
- Important to rule out infections, cocaine or MDMA abuse, lithium or anticholinergic overdose.
- Symptoms include hypertension, diarrhea, tachycardia, hyperthermia, diaphoresis, ataxia, myoclonus, mydriasis, hallucinations, and confusion.
- Concern for rhabdomyolysis, ventricular arrhythmia, respiratory arrest, and coma.

Treatment of Serotonin Syndrome

- Lorazepam 1–2 mg iv push for agitation.
- Methysergide 2–6 mg to counteract serotonin.
- Labetalol or propranolol for HTN and tachycardia.
- Cyproheptadine 4 mg po to counteract serotonin.

Patient Management

- Avoid drugs that may trigger serotonin syndrome (fentanyl, tramadol, ondansetron).
- Look for signs of serotonin syndrome.
- EKG monitoring for lithium-induced dysrhythmias.
- Patients on MAOIs are at risk for orthostatic hypotension.
- Careful use of sympathomimetic agents – vasoconstrictors may have exaggerated effects, especially in patients on TCAs. Ephedrine should be avoided in patients with MAOIs. Ketamine use should be carefully considered due to sympathetic effects.
- CYP450 inhibitors such as macrolide antibiotics may lead to toxic levels of TCAs.

Eating Disorders

Psychiatric disorder characterized by abnormal eating habits with psychological implications and excessive concern about body weight.

Bulimia Nervosa

- Binge eating disorder followed by purging at least one time per week for 3 months. Patients have strong negative views on their personal shape and weight with a personal feeling of self-worthlessness. Commonly dentition will have worn enamel on the lingual surface of the teeth from excess vomiting.

Anorexia Nervosa

- Eating disorder characterized by purposeful restricted caloric intake leading to low body weight with poor body image and self-evaluation. BMI (less than 18.5 kg/m^2) in the tenth percentile is highly correlated with anorexia [4].

Signs/Symptoms of eating disorders:

- Low body mass index.
- Dehydration.
- Hypertrophic salivary glands.
- Hypokalemia, hypocalcemia, hyponatremia, and hypoproteinemia.
- Resting bradycardia.
- Amenorrhea and atrophy of breasts in females.
- Seizures.
- Delayed gastric emptying.
- Hypothermia.
- Anxiety.
- Dry skin (normally scaly, yellow, and erythematous patches) and hair loss (alopecia).
- Lethargy.
- Hypokalemic hypochloremic metabolic alkalosis may occur secondary to excessive vomiting. Acidosis may be seen in those patients abusing laxatives.
- Russell sign – callus on dorsal surface of index and long finger due to induced vomiting (seen in bulimia).

Treatment for Eating Disorders

- Treatment is primarily with psychotherapy and medical monitoring of the physiological sequelae.
- Underlying associated anxiety/depression may be treated with pharmacotherapy.
- Primary treatments of eating disorders with antidepressants have been shown effective in the treatment of bulimia, but have shown mixed results with anorexia.

Patient Management

- Fluid rehydration – patients are commonly dehydrated leading to hypotension.
- Amplified response to anesthetic medication – slowly titrate medications. A reduction in fat levels leads to an increase in bioavailability of lipophilic drugs, such as propofol and ketamine. Decrease in plasma proteins leads to an increase in bioavailability of other at home medications and those delivered during anesthesia that bind to plasma proteins such as benzodiazepines. Deeper than desired level of anesthesia may be reached unintentionally. Patients may require an extended time to emerge from anesthesia administration.
- Bradycardia – concern for blunting of autonomic response, which may further reduce heart rate; be cautious with narcotics.
- Preoperative laboratories/test [5]:
  - EKG – preoperative EKG should be monitored for dysrhythmias including PVCs, T wave inversions, and heart blocks.
  - CBC – review white blood cell, platelet, and preoperative hemoglobin. This may be severely reduced due to malnutrition. Consider preoperative antibiotics or blood products for emergency surgeries.

  - Chemistry 10 Panel – evaluate for hydration based on BUN/creatinine ratio elevation. Rule out unsafe levels of electrolytes.
  - NPO period – increase of NPO time due to delay in gastric emptying. Consider antiemetic to prevent vomiting due to delay in gastric emptying.
  - LFTs – hypoproteinemia and reduced liver function may make patients more susceptible to bleeding and may decrease wound healing.

Schizophrenia

- Psychiatric disorder characterized by chronic and recurrent psychosis in the form of positive and negative symptoms.
- Positive symptoms may include the following:
  - Hallucinations (auditory, visual, somatic, olfactory).
  - Patients also display delusions, which are fixed false beliefs.
  - Disorganized thoughts and speech (neologisms, tangential speech, word salad).
  - Bizarre behavior.
- Negative symptoms may include the following:
  - Apathy.
  - Flat affect.
  - Anhedonia – lost interest in activity.
  - Aloofness.
  - Cognitive impairment (executive functioning, memory impairment, social cognition impairment).

Treatment

Antipsychotics are the first-line treatment for schizophrenia. This group of medications has significant side effects.

- *First-generation antipsychotics* – mechanism is via dopamine receptor (D_2) blockade. These drugs have effects on other receptors, such as serotonin type 2 ($5\text{-}HT_2$), alpha-1, histaminic, and muscarinic receptors.
  - Haloperidol (Haldol)
  - Fluphenazine
  - Chlorpromazine
  - Perphenazine
- *Second-generation antipsychotics* – mechanism is via D_2 blockade. Reduced extrapyramidal side effect profile than the first generation. These drugs have side effects of hypotension, weight gain, and glucose intolerance.
  - Risperidone (Risperdal®)
  - Clozapine (Closaril®) – risk for agranulocytosis, requires serial CBCs.
  - Olanzapine (Zyprexa®)
  - Lurasidone (Latuda®)
  - Quetiapine (Seroquel®)
  - Ziprasidone (Geodon®)
- Both generations of each drug in this class has various effects on other receptors, such as serotonin type 2 ($5\text{-}HT_2$), alpha-1, histaminic, and muscarinic receptors which may lead to extrapyramidal side effects including the following:
  - Rigidity.
  - Bradykinesia.
  - Dystonias.
  - Tremor.
  - Akathisia.
  - Tardive dyskinesia, which is an involuntary movements of the perioral region extremities.
  - Prolonged QT interval which may degenerate into torsades de pointes and cardiac arrest.

Neuroleptic Malignant Syndrome

- Potentially fatal complication of dopamine agonist due to depletion of dopamine in the CNS. Manifests over 24–72 hours [4].
- Diagnosis of exclusion, no laboratory tests. The diagnosis is usually satisfied if a combination of rigidity, altered mental status, pyrexia, and elevation of creatine kinase levels.

- It is slower in onset than serotonin syndrome, with symptoms occurring in 1–3 days after neuroleptic treatment.
- Clinical signs include the following [3]:
  - Mental status changes (delirium).
  - Muscular rigidity (lead pipe rigidity which can lead to rhabdomyolysis). Rhabdomyolysis can lead to acute renal failure (see elevated CPK). Patients can also have trismus, dysphagia, and prominent sialorrhea.
  - Pyrexia (mean temperature is 103 °F).
  - Autonomic instability – tachycardia, tachypnea, labile blood pressure, dysrhythmias.
  - Electrolyte disturbances – hypocalcemia, hypomagnesemia, hyperkalemia.
  - Disseminated intravascular coagulation (DIC).
  - Respiratory failure from chest wall rigidity.

Treatment of NMS

- Withdrawal of the dopamine antagonist offending agent.
- Supportive therapy (fluids for dehydration and myoglobinuria).
- Replenish electrolytes.
- Antipyretics and cooling blankets for hyperthermia.
- Control blood pressure.
- May need mechanical ventilation if respiratory failure develops.
- Benzodiazepines for agitation and psychosis.
- Consider bromocriptine (2.5 mg 3 times a day) until symptoms resolve [3].

Patient Management

- EKG to assess QT interval – higher risk for ventricular dysrhythmia.
- Assess for substance abuse (50% of schizophrenia patients have comorbid substance abuse disorder).
- Tremor may make IV placement and retention difficult.
- Tardive dyskinesia may create difficulties with oral surgical procedures.
- Avoid ketamine as it may worsen hallucinations and decrease the seizure threshold.
- Antipsychotics may cause an ileus (risk factor for vomiting and aspiration).
- Obtain an appropriate level of sedation as surgical stress can worsen psychotic symptoms.
- Postural hypotension secondary to beta adrenergic blockade.
- Look for signs of neuroleptic malignant syndrome.

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Pregnancy

22

Zain Manji and Aaron D. Figueroa

Physiologic Changes During Pregnancy

Cardiovascular

- Decrease in systemic vascular resistance.
- Increase in cardiac output.
- Increase in heart rate.
- Increased cardiac workload can result in ventricular hypertrophy.
- Benign systolic ejection murmur is common due to increased HR and blood volume; resolves after delivery.
- Uterine compression of the inferior vena cava, leading to venous stasis and deep venous thrombosis.
- Decrease in oncotic pressure leads to pedal edema.

Hematological

- Increase in plasma exceeds that of erythrocytes, leading to physiologic anemia aka "hemodilution" [1, 2].
- Hypercoagulable state increases the risk for deep venous thrombosis and pulmonary embolism:
  - Reduction in protein S activity.
  - Activated protein C resistance.
  - Increase in coagulation factors except XI and XIII [2].
  - Pressure from gravid uterus causes endothelial damage.
  - Leukocytosis due to hormonal changes.
  - Suppression of the immune system. Decreased chemotaxis and cell-mediated immunity [2].

Respiratory

- There is relaxation of the rib cage allowing for a more horizontal position and upward displacement of the diaphragm.
- Pulmonary Function:
  - Tidal volume – Increases
  - Respiratory rate – Increases
  - Minute ventilation – Increases
  - Expiratory reserve volume – Decreases
  - Residual volume – Decreases
  - Functional residual capacity – Decreases
- Respiratory changes result in respiratory alkalosis, due to increase in minute ventilation.
- Increase in oxygen consumption.
- Rapid desaturation during periods of apnea.

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Genitourinary

- Increase in glomerular filtration rate [1].
- Increase in renal blood flow.
- Increase in creatinine clearance.
- Third trimester, bladder capacity reduction due to pressure from enlarged uterus. Hydroureter found in 90% of pregnancies [2]. See increased rate of UTI due to urinary stasis.

Gastrointestinal System

- Decrease in lower esophageal sphincter tone.
- Increase in gastric emptying time.
- Increase in gastric pressure, can cause pyrosis (gastroesophageal reflux) complaint in 70% of pregnancies [1, 2].
- Increase in bile secretion.
- Increase in gallstone formation.

Endocrine

- Insulin resistance.
  - Fasting glucose levels are lower due to glucose utilization by the fetus.
- Hypoglycemia can result from insulin resistance and glucose utilization by the fetus, especially in times of fasting.
- Estrogen increases thyroxine-binding protein, which increases total levels of T3 and T4.
  - Circulating free T3 and T4 remain unchanged.

Oral Manifestations in the Pregnant Patient

Due to physiological and hormonal changes common oral manifestations are seen:

- Chronic gingivitis.
- 1–5% patients develop pregnancy tumors (pyogenic granulomas) due to increased angiogenesis and local irritating factors such a plaque buildup. Normally self resolves [1, 3].
- Decrease in salivary pH can lead to decreased mucosal desquamation and dental decay.
- Melanosis of the skin and mucosa due to increase in estrogen and progesterone [1].

Diseases of Pregnancy

Preeclampsia

- Preeclampsia is a pregnancy-induced condition due to abnormal placental implantation. It results in hypertension that occurs after 20 weeks of gestation or postpartum, accompanied by either proteinuria or other maternal organ dysfunction.

Treatment

- Term
  - Delivery of the fetus.
- Pre-term
  - Mild preeclampsia.
    Conservative management – control of blood pressure, fluid management, and frequent observation/fetal monitoring.
    Bed rest.
    Delivery at 37 weeks.
  - Severe preeclampsia.
    Delivery of the fetus regardless and management of sequelae.
    Antihypertensive therapies are aimed at prevention of abruptio placentae and stroke; indications include chronic hypertension and severe hypertension during labor or delivery.
    Agents of choice: labetalol, hydralazine, and nifedipine.
    Seizures prophylaxis is managed by administration of magnesium sulfate.

Eclampsia

- Form of severe preeclampsia characterized by seizures or coma without any other brain pathology.
- Can be associated with respiratory failure, kidney failure, coagulopathy, stroke, and cardiac arrest.

Treatment

- Patient positioning into lateral decubitus position (reduce pressure vena cava), suctioning of secretions, and supplemental oxygen.
- Administration of magnesium sulfate for seizure management.
- Use antihypertensive medications to control blood pressure.
- Emergent delivery of the fetus irrespective of gestational age.
- Management as needed for other sequelae.

HELLP Syndrome

- Syndrome characterized by hemolysis, elevated liver transaminases, and low platelet counts.
- Occurs in conjunction with eclampsia or preeclampsia.
- Patients may develop a subcapsular hepatic hematoma, which can rupture and lead to severe intra-abdominal bleeding and disseminated intravascular coagulation.
- High maternal and perinatal morbidity/mortality rate.
- Immediate delivery for pregnancies regardless of gestational age if mother is unstable.
- If gestational age is less than 33 weeks and mother is stable, consider corticosteroids administration to allow for fetal maturation and improve platelet count. Delivery within 48 hours if maternal condition stabilized after steroid administration.

Gestational Hypertension

- Elevated blood pressure during pregnancy not associated with proteinuria or any preexisting chronic hypertension. SBP ≥140 mm Hg and/or DBP ≥90 mm Hg (average of at least 2 measurements taken at least 15 minutes apart) [4].
- May develop into preeclampsia if proteinuria develops.
- May develop into chronic hypertension if it remains 3 months after delivery.
- Treated with antihypertensive medication, concern for HELLP, preeclampsia, end organ damage, and risk of maternal stroke.
- Women with severe hypertension with SBP 160 or DBP 110 mm Hg in pregnancy require urgent antihypertensive therapy because it is considered an obstetrical emergency [4].

Gestational Diabetes

- Any degree of glucose intolerance first recognized during pregnancy, diagnosed on glucose tolerance test.
- Pregnancy results in maternal insulin resistance in an effort to shunt glucose to the developing fetus.
- Women who are unable to compensate for this insulin resistance with increased production develop gestational diabetes.
- Risk factors include increased age, obesity, family history, history of gestational diabetes.
- Treatment consists firstly of lifestyle changes and oral hypoglycemic and insulin only when necessary.

Medication and Pregnancy (Table 22.1)

- Category A – Controlled studies in humans have failed to demonstrate a risk to the fetus, and the possibility of fetal harm appears remote.
- Category B – Animal studies have not indicated fetal risk, and human studies have not been conducted, or animal studies have shown a risk, but controlled human studies have not.
- Category C – Animal studies have shown a risk, but controlled human studies have not been conducted, or studies are not available in humans or animals. Drugs should only be given if the potential benefit justifies the potential risk to fetus.
- Category D – Positive evidence of human fetal risk exists, but in certain situations, the drug may be used despite risk.

Table 22.1 Medications commonly used by the oral and maxillofacial surgeon and their use in pregnancy

Drug	FDA category	Use during pregnancy	During breastfeeding
Local anesthetics			
Lidocaine	B	Yes	Yes
Mepivacaine	C	Yes	Yes
Bupivacaine	B	Yes	Yes
Articaine	C	Yes	Yes
Prilocaine	B	Yes	Yes
Analgesia			
Aspirin	C/D	No, causes intrauterine growth restriction. Intracranial brain bleeds.	No
Acetaminophen	B	Yes	Yes
Ibuprofen	B/D	Avoid third trimester, may close PDA	Yes
Etodolac	B/D	Avoid third trimester	Yes
Ketorolac	B/D	Avoid third trimester	Yes
Naproxen	B/D	Avoid third trimester	Yes
Codeine	C	Associated with first trimester malformations. May use small doses for short duration	Yes
Oxycodone	B	Associated with first trimester malformations. May use small doses for short duration	Yes
Fentanyl	B	Yes	Yes
Antibiotics			
Penicillins	B	Yes	Yes
Amox + clavulonic acid	B	Yes	Yes
Azithromycin	B	Yes	Yes
Erythromycin	B	Yes	Yes
Clindamycin	B	Yes	Yes
Gentamicin	B	No, concern for ototoxicity in fetus	Use with caution, poorly absorbed orally Infants can develop diarrhea [5, 6]
Metronidazole	B	Use with caution, reduced form is teratogenic, but humans not capable of reducing	Use with caution, reduced form is teratogenic, but humans not capable of reducing
Cephalosporins	B	Yes	Yes
Tetracycline	D	No, cause tooth staining and decreased bone development	No
Nystatin	B	Yes	Yes
Fluconazole	C	Use with caution	No
Vancomycin	B	Yes [7]	PO, poorly absorbed, low risk. IV concern GI upset due to change in bacterial flora
Chlorhexidine rinse	B	Yes	Yes
Sedatives/ Hypnotics			
Benzodiazepine	D	No, risk for fetal craniofacial anomalies	No
Barbiturates	D	No, risk for fetal craniofacial anomalies	No
Propofol	B	Use with caution	Yes, minimal amount in milk
Nitrous oxide	Not assigned	Controversial	Controversial

Information gathered and modified from Turner and Aziz [1], Moore [8], and Cengiz [9]

- Category X – Evidence of fetal abnormalities and fetal risk exists based on human experience, and the risk outweighs any possible benefit of use during pregnancy.

Patient Management in Pregnancy

- Avoid elective care in the first trimester, urgent treatment only.
- Second trimester is the safest and most ideal time for routine treatment to be provided, though this should focus on the elimination of active disease that could cause a potential problem later in the pregnancy.
- Supine hypotension may develop later in pregnancy; if this occurs during treatment, roll the patient to the left side. This occurs secondary to compression of the inferior vena cava.
- Hypertension appreciated at baseline may be an early sign of preeclampsia.
- Patients are more prone to emesis due to relaxation of the lower esophageal sphincter and longer gastric emptying times.
- It is estimated that a radiation dose of >10 Gy (5 Gy first trimester) can cause congenital fetal anomalies [1]. The radiation exposure to the fetus is estimated to be 1/50,000 as to the mother's head. A standard full mouth series, orthopantogram, or cone beam radiation exposure to the fetus is estimated 1×10^{-5} to 1×10^{-2} Gy [1]. Radiographs can be obtained relatively safely, but should only be made if necessary to complete treatment. All appropriate protective equipment should be used including the lead apron and thyroid collar.
- Anesthesia and surgery can result in preterm labor, which is of greatest concern in the third trimester. All elective surgery should be delayed postpartum.
- Maintain oxygenation, blood pressure and avoid hyperventilation during anesthesia.
- Consider local anesthesia whenever possible [10]. Pregnant patients are more sensitive to local anesthesia and have reduce onset time for conduction block [2].
- Consultation with the patient's obstetrician should be considered to determine the need for intraoperative fetal monitoring.
- Perioperative antibiotics may be indicated in the setting of gestational diabetes and reduced immune capability. All infections should be treated aggressively with early incision and drainage [1, 2].
- N_2O is controversial, as evidence of use without scavenging unit in dental assistants has shown increase in spontaneous abortion. N_2O inactivates methionine synthetase.
- Ensure adequate hydration and electrolyte balances if history of nausea and vomiting.

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23 Human Immunodeficiency Virus and Acquired Immunodeficiency Syndrome

Zain Manji and Jerri D. Hines

Definition/Pathophysiology

- HIV is an RNA retrovirus that infects CD4 cells that causes varying levels of immunosuppression with the possible progression to AIDS.
- AIDS – the result of progressive HIV infection in which a person has a weakened immune system and meets specific diagnostic criteria (see below).
- HIV is an infectious disease transmissible by sexual contact, exposure to infected blood/organ donations, perinatally, or via IV drug abuse.
- For several years, HIV-infected patients can remain clinically asymptomatic; however, during this phase the CD4 count gradually decreases complicated with development of opportunistic infections, autoimmune conditions, and neoplasms.
- HIV infection can manifest itself along a continuum as it may progress from initial infection to latency to progression to AIDS.
- According to the Centers for Disease Control (CDC), a patient has AIDS in the presence of HIV in combination with:
  1. A $CD4^+$ T-cell count of 200 cells/μL or less.
  2. A $CD4^+$ T-cell percentage of total lymphocytes of 15% or less.
  3. One or more of AIDS-defining illnesses (Table 23.1).

Table 23.1 AIDS defining illnesses

Burkitt's lymphoma
Candidiasis of bronchi, esophagus, trachea, or lungs
Coccidioidomycosis
Cryptococcosis, extrapulmonary
Cryptosporidiosis, chronic intestinal for longer than 1 month
Cytomegalovirus (CMV)
Encephalopathy (HIV-related)
Herpes simplex
Histoplasmosis
Invasive cervical cancer
Kaposi's sarcoma (KS)
Lymphoma
Mycobacterium avium complex (MAC)
Mycobacterium tuberculosis
Pneumocystis jirovecii pneumonia
Progressive multifocal leukoencephalopathy
Salmonella septicemia
Toxoplasmosis of the brain
Wasting syndrome

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Diagnostic Testing [1]

1. *Screening enzyme immunoassay (EIA)* for IgM and IgG anti-HIV antibodies (usually detectable 3–5 weeks after infection), if positive then Western blot performed.
2. CBC with differential (HIV patients exhibit hematologic disorders).
3. Albumin and prealbumin (nutritional status).
4. CD4 (reflection of immune status).
5. Viral load (predictor of rate of progression to AIDS).

Signs and Symptoms:

- Acute seroconversion occurs 2–3 weeks after inoculation.
- Acute symptoms mimic flu-like symptoms – fever, fatigue, headache, night sweats, pharyngitis, myalgias, and arthralgias.
- Generalized lymphadenopathy is often seen until initiation of treatment with highly active, antiretroviral therapy (HAART).
- Weight loss, cachexia (temporal wasting), and failure to thrive often seen in AIDS.
- Cardiovascular Manifestations – can see left ventricular hypertrophy, diastolic heart failure, myocarditis, abdominal aortic aneurysms, and aortic dissections [2].
- Pulmonary Manifestations – patients are predisposed to pulmonary infections/malignancies including [2] the following:
  - *Pneumocystis jiroveci* pneumonia
  - Pulmonary TB
  - Nocardia
  - Bacterial lung abscesses
  - Pulmonary Kaposi's sarcoma
- Neurological Manifestations – peripheral neuropathy, cerebral toxoplasmosis, CNS lymphoma, progressive multifocal leukoencephalopathy (PML), meningitis (*Cryptococcus neoformans*, HIV, and TB).
- Endocrine Manifestations – progressive HIV infection can cause glucose intolerance and adrenal insufficiency.
- Hematological Manifestations – can see leukopenia, anemia, and thrombocytopenia. HIV patients are also predisposed to hypercoagulable states.
- Renal Manifestations – HIV-associated nephropathy can lead to ESRD. Protease inhibitors have been implicated in acute tubular necrosis and nephrolithiasis.
- Maxillofacial Manifestations – can see wasting in the temporal, nasolabial, and orbital regions due to lipodystrophy from HIV or from antiretroviral treatment. Seborrheic dermatitis of the scalp or molluscum contagiosum of the forehead may be seen. Parotid gland enlargement may also be seen due to lipodystrophy, lymphoma, or lymphoepithelial cysts.
- Oral Manifestations – high risk for HIV periodontitis/gingivitis. Xerostomia is common. Fungal infections such as candidiasis may manifest when CD4 count drops below 500 cells/μl. In the setting of immunosuppression, aphthous ulcers often manifest and there is also a predilection to ulcerations secondary to fungal infections such as cryptococcus and histoplasmosis. Neoplasms such as Kaposi's sarcoma (related to coinfection with HPV-8) and lymphoma may be seen. Oral papillomas secondary to HPV infection are also common.

Treatment

- Highly active, antiretroviral therapy (HAART) – a combination of multiple antiretroviral medicines used to inhibit HIV replication. Usually three drugs – two nucleoside reverse transcriptase inhibitors (NRTIs) + a protease inhibitor or non-nucleoside reverse transcriptase inhibitors (NNRTI):
  - Nucleoside reverse transcriptase inhibitors (NRTI): zidovudine (AZT), lamivudine (3TC), stavudine (D4T), tenofovir, Emtricitabine Side effects include nausea, headache, peripheral neuropathy, anemia, and thrombocytopenia.
  - Non-nucleoside reverse transcriptase inhibitors (NNRTI): nevirapine, efavirenz (EFV). Side effects are dizziness, rash, vivid dreams, and suicidal thoughts. Emtricitabine/tenofovir/efavirenz (Atripla®) is a combination drug used in the treatment of HIV. Emtricitabine/tenofovir (Truvada®) is a combination drug used in the treatment and

prevention of HIV. It is also used in the treatment of Hepatitis B (valuable in the setting of HIV/Hep B co-infection). Truvada® is also approved for Pre-exposure prophylaxis (PREP) to decrease the transmission of HIV in patients engaging in high risk behavior.
  - Protease inhibitors: ritonavir. – Side effects include GI disturbance, glucose metabolism, and paresthesias.
- Immunotherapy: interleukin II or interferon-alpha can increase CD4 levels.
- Trimethoprim/sulfamethoxazole (Bactrim®) – if CD4 count falls below 100 cells/μl to decrease the risk of *Pneumocystis jirovecii* pneumonia.
- Macrolide (azithromycin or clarithromycin) – empirical management if CD4 count falls below 50 cells/μL to decrease the risk of *Mycobacterium avium* complex (MAC) infection.

Patient Management

- A thorough pre-anesthetic evaluation should be supplemented with laboratory data. Assessment of perioperative risk should focus on the patient's immunological/clinical status.
- Consider testing for concurrent blood-borne infections such as hepatitis B or C as infection routes are similar (especially in intravenous drug users).
- Communication with the physician (PCP or infectious disease) prior to surgery is paramount in clinical decision-making.
- The laboratory workup should include CBC, CHEM 10, LFTs, glucose, coagulation studies, immunological status via CD4+ lymphocyte cell count, and viral load during the previous 3 months.
- Chest radiographs and EKG should also be performed in all patients for cardiopulmonary abnormalities and screening for opportunistic infections such as tuberculosis. Patients with a history or signs of pulmonary or cardiac dysfunction should undergo further evaluation with ABG and ECG.
- Blood transfusions should be used judiciously due to higher risk for transfusion-related immunomodulation (TRIM) and can result in increased viral loads [2].
- Hypercoagulability increases risk for thromboembolic complications warranting the need for early mobilization and/or prophylaxis.
- Non-neutropenic HIV and AIDS patients follow standard antibiotic prophylaxis protocol.
- Patients taking certain antiretroviral medications such as protease inhibitors and NNRTIs may have impairment of CYP450 hepatic enzymes prolonging the effects of benzodiazepines, opiates, and lidocaine [3]. Etomidate, atracurium, remifentanil, and desflurane are not dependent on CYP450 hepatic metabolism and, therefore, are preferable drugs.
- Calcium channel blockers may enhance hypotensive effects with patients using PI and NNRTIs.
- For emergent situations, the need for succinylcholine (hyperkalemia or hyperpyrexia) is considered a potential risk in the HIV patients with progressive neuropathy, myopathy, or muscle wasting.
- Propofol should be used judiciously as the interaction between propofol and NRTIs may both potentially promote mitochondrial toxicity and lactic acidosis.
- The presence of neurologic manifestations, such as overt dementia, may increase brain sensitivity to opioids or benzodiazepines. Autonomic instability may also be present depending on the extent of neurological involvement.
- Pulmonary complications can occur as a consequence of opportunistic infections leading to respiratory distress and hypoxemia.
- Stress response to surgery may be more profound after deep/general anesthesia, which in turn may lead to impaired immune function that can persist for multiple days.

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Substance Abuse

24

Marc Dentico-Olin and Peter K. Cudjoe

Alcohol Abuse

Alcohol use disorder is characterized by a problematic pattern of alcohol use leading to clinically significant impairment or distress, as manifested by multiple psychosocial, behavioral, or physiologic features.

- Physical exam – Look for evidence of liver disease and anemia. Pallor, jaundice, scleral icterus, telangiectasia, ascites, fetor hepaticus ("breath of the dead"), caput medusa, palmar erythema, peripheral edema, and splenomegaly.
- Hepatic function – Can see a spectrum of changes from reversible fatty changes to cirrhosis. Increased serum transaminases (AST, ALT) due to cell injury and death, hyperbilirubinemia, and decreased production of clotting factors. Hypoproteinemia due to albumin depletion. Immunosuppression due to depletion of the reticuloendothelial system.
- Hematology – Anemia (GI bleeds, bone marrow depression, or nutritional deficiencies), thrombocytopenia (bleeding), leukopenia, and abnormal leukocyte chemotaxis (both cause impaired wound healing and increased risk of infection).
- Neurologic function – CNS depressant and can cause respiratory depression and death. Effects potentiated by concomitant use of narcotics, benzodiazepines, and barbiturates. Chronic alcohol abuser may require more anesthetic agents to produce desired level of anesthesia (but with greater risk).
- Nutritional deficiencies – Chronic alcoholics are frequently malnourished. Ethanol interferes with absorption, metabolism, and storage of vitamins (specifically thiamine, folate, and vitamin B6). Thiamine deficiency may produce ophthalmoplegia, ataxia, confusion (Wernicke's encephalopathy puts patients at risk for Wernicke-Korsakoff syndrome). Thiamine deficiency may also cause weakness, fatigue, myalgias, and anorexia. Folate deficiency may contribute to megaloblastic anemia.
- Cardiac – May have an associated cardiomyopathy or low-output heart failure (leading cause of death).
- Endocrine – Hypogonadism, testicular failure, and gynecomastia.
- GI – Increased incidence of gastritis, pancreatitis, and liver failure.
- Respiratory – Acute alcohol ingestion can cause hyperventilation and an increase in dead space ventilation. Aspiration of gastric con-

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tents with chemical pneumonitis and pulmonary infections is an increased risk with chronic alcoholism.

Alcohol Withdrawal Syndrome

- Begins 6–24 hours after the last intake of alcohol.
- The signs and symptoms include tremors, agitation, nausea, sweating, vomiting, hallucinations, insomnia, tachycardia, hypertension, delirium, and seizures.
- The most dreaded complication is delirium tremens (hyperthermia, tremors, and seizures), which has a high mortality rate.
- Treatment includes benzodiazepines (such as diazepam or chlordiazepoxide), electrolyte/fluid deficit correction, and administration of a banana bag (thiamine, folic acid, magnesium, and a multivitamin).
  - The goal of thiamine therapy is to prevent Wernicke-Korsakoff syndrome (ataxia, anterograde amnesia, nystagmus, and peripheral polyneuropathy).
  - Beta-blockers are also used for the management of tachycardia.

Patient Management

- The *CAGE* questionnaire (>2 is concerning for alcohol having an effect on health and social well-being) [1].
  - Have you ever felt you should *C*ut down on your drinking?
  - Have you been *A*nnoyed by other people criticizing your drinking?
  - Have you ever felt *G*uilty about drinking?
  - Have you ever taken a drink in the morning to steady your nerves or ease a hangover (*E*ye-opener)?
- Labs-CBC, Basic Metabolic Panel, LFTs.
- Postpone surgery for acutely intoxicated patient.
- Preoperative labs: CBC, BMP, and LFTs.
- EKG – for patients with known alcohol-induced cardiomyopathy.
- Evidence of active hepatitis – postpone surgery and obtain a medical consult.
- Coagulopathy management:
  - Goal INR < 3 and platelets >50,000 (depending on surgery).
  - If emergency surgery is indicated, treatment in a hospital setting should be employed as vitamin K, clotting factors, fresh frozen plasma, or platelets may also be necessary to correct coagulopathies.
  - Utilize local hemostatic measures to aid in bleeding control.
- Replenish electrolytes as required.
- Monitor glucose levels closely.
- Delirium tremens prophylaxis with benzodiazepines.
- Antibiotic prophylaxis due to immunosuppression.
- Patients are often volume depleted secondary to dehydration and will require fluid resuscitation.
- Patients are more prone to hypotension as a result of hypoalbuminemia that causes third spacing.
- Patients are less sensitive to endogenous or parenteral catecholamines which can contribute to hemodynamic instability.
- During emergency anesthesia with presumed alcohol intoxication, rapid sequence induction is indicated due to possible aspiration of stomach contents. Alcohol, opioids, and trauma are high risk factors for aspiration.
- Esophageal varices are a concern for rupture in unsuccessful endotracheal intubation.
- Chronic alcohol use increases dose requirements for general anesthetic agents as the minimal alveolar concentration is increased.
- Intravenous drug doses that are increased (e.g., propofol, thiopental, and opioids) can exacerbate the risk of cardiovascular instability in patients who may be suffering from cardiomyopathy, heart failure, or dehydration.
- In acutely intoxicated, non-habituated patients, the minimal alveolar concentration (MAC) of inhalational agents is reduced. Acutely intoxicated patients are more sensitive to the effects of opioids, benzodiazepines, and barbiturates. Sensitivity to other drugs may occur due to competitive inhibition from elevated blood ethanol concentration. Volatile

agents compete with ethanol for binding on neuronal gamma-aminobutyric acid (GABA) and glycine receptors.
- The distribution and metabolism of anesthetic drugs are altered by hypoalbuminemia and hepatic impairment. Neuromuscular blocking agents that undergo hepatic metabolism may have a prolonged duration of action.
- Increased risk of postoperative infection due to immune deficiency resulting from alcohol adverse effects on hematologic system causing leukopenia and altered cytokine production.
- Acute confusion or delirium after an operation can be mitigated by attention to pain control measures, oxygenation, and correction of metabolic disturbances. Consider intravenous haloperidol (0.5–10 mg may be repeated after 20–30 minutes) for acute agitation. Other medications include chlorpromazine, risperidone, and olanzapine.

Cocaine

- An amphetamine that blocks the reuptake of norepinephrine, serotonin, and dopamine transporting mechanisms in the CNS leading to intense stimulation, euphoria, and pleasure. Cocaine has a negative impact on several different systems.
- Cardiovascular system – The majority of concern is the patient's cardiovascular system and changes in vasculature due to cocaine's effects and especially with the risk of developing cocaine-induced cardiomyopathy in chronic abusers. Acute cocaine use can cause coronary vasospasm, myocardial ischemia/infarction, and ventricular dysrhythmias. EKG findings may show prolonged QRS and QT intervals and premature ventricular contractions, which can degenerate into deadly ventricular dysrhythmias. The myocardium is also sensitized to endogenous catecholamines.
- Respiratory system – There is a risk of alveolar hemorrhaging and pulmonary edema from crack cocaine (smoked) use. Snorting cocaine can lead to mucosal ulcerations which can cause epistaxis and nasal septal destruction. Cocaine users also can have chronic coughs with an increased risk of bronchospasm and diffusion capacity abnormalities.
- Hematologic system – Cocaine activates platelets, increases platelet aggregation, and promotes thrombus formation. This adds further risk for cardiac and cerebrovascular events intra-operatively. Cocaine-induced thrombocytopenia can also occur which is similar to idiopathic thrombocytopenia purpura.
- Nervous system – Cocaine alters pain perception, leading to potentially lower pain thresholds. This creates challenges in analgesia administration, both intra-operatively and postoperatively. Presynaptic reuptake of norepinephrine, dopamine, serotonin, and tryptophan is prevented, leading to intense activation of the sympathetic nervous system. May see an acute elevation in blood pressure, tachycardia, and predisposition to ventricular tachyarrhythmias and seizures.
- Gastrointestinal system – Chronic cocaine use also causes delayed gastric emptying, which is a risk for aspiration. As a result, aspiration prevention is an important consideration preoperatively.

Patient Management

- Determine last use of cocaine and severity of use. No consensus on the timing of treatment. Anecdotally, 8 hours is sufficient for general anesthesia if the patient is stable. For office-based anesthesia, consider waiting 24 hours to 1 week or consider treatment in a hospital setting.
- Urine testing can detect cocaine and its metabolites for up to 6 days or even 10 days for chronic users.
- Avoid sympathomimetic agents (e.g., ketamine) during sedations, as the myocardium of patients using cocaine is more sensitive to catecholamines. Monitor use of local anesthetics containing epinephrine due to dysrhythmias.
- Be prepared to treat intraoperative myocardial ischemia (e.g., nitroglycerin, esmolol, mor-

phine, oxygen). There is evidence that alpha-adrenergic blockade is sufficient to treating coronary artery vasospasm. Beta-adrenergic blockade may accentuate cocaine-induced coronary artery vasospasm and can result in paradoxical rise in blood pressure.
- Be prepared to treat cocaine-induced seizures (e.g., benzodiazepines and oxygen).
- Consider treatment in a hospital setting for emergent procedures or patients with severe cocaine-induced cardiomyopathy.

Marijuana

- Marijuana is a recreational drug from the cannabis plant.
- Its desired effects are euphoria, increased awareness of sensation, and increased libido.
- The most active cannabinoid in marijuana is Δ9-trans-tetrahydrocannabinol (THC).
- Low dose use stimulates the sympathetic nervous system, which leads to hypertension and tachycardia. High dose use inhibits the sympathetic nervous system, which leads to hypotension and bradycardia.
- Chronic usage can result in compromised pulmonary function (depressed pulmonary clearance and tar deposits) and increased airway reactivity. These patients are more prone to bronchitis and pneumonia. This makes patients more susceptible to anesthesia-related respiratory complications (e.g., laryngospasm and bronchospasm).
- Chronic users exhibit tolerance and cross tolerance, which may lead to decreased levels of sedations/combativeness during intravenous sedations.

Patient Management

- Determine last use of marijuana. Cancel cases when intoxication is suspected. For office-based anesthesia, consider waiting 72 hours after consumption in elective cases [2].
- Consider a dose of steroids in patients with recent use, as there is a documented risk for uvular edema [2].
- Monitor EKG and blood pressure continuously throughout the procedure as studies have documented acute use to cause PVCs, atrial fibrillation (Afib), and AV block [3]. Drugs with known sympathomimetic action should be avoided in acute users.
- Auscultation preoperatively to rule out active pulmonary abnormalities, similar pulmonary complications as seen in tobacco use [2].
- Consider administering albuterol and glycopyrrolate as the tracheobronchial tree is more sensitive to stimuli.
- Patient will be more sensitive to inhaled anesthetics.
- Myocardium more sensitized due to increased levels of epinephrine, be judicious with sympathomimetics.
- THC depletes acetylcholine stores and exerts an anticholinergic effect and thus creates a potentiation of the non-depolarizing muscle relaxants.
- Platelets drop due to ADP release that causes platelet aggregation and increased bleeding risk [4].
- Patients may exhibit combative behavior during sedation.
- Patient may require higher doses of benzodiazepines, opioids, and propofol due to cross-tolerance.
- Chronic users require higher doses of narcotics to obtain analgesia [5].

Heroin/Opioid Abuse

Heroin (diacetylmorphine) – opioid drug that is synthesized from morphine, which is a naturally occurring substance extracted from the seed of the opium poppy plant. The desired effect is an intense transcendent state of euphoria. Heroin can be inhaled, smoked, or injected intravenously.

- Opioids – Narcotic drugs that are derived from the opium plant. These drugs bind to opioid receptors (mu, delta, and kappa) in the CNS and PNS resulting in the desired effect of anal-

gesia. Other common effects are euphoria, sedation, nausea, constipation, chest wall rigidity, and respiratory depression.
- Chronic usage may lead to abnormal pain sensitivity, including hyperalgesia and allodynia. Synthetic opioids include oxycodone, hydrocodone, codeine, meperidine, methadone, hydromorphone, and fentanyl.
- Opioid use disorder can involve misuse of prescribed opioid medications, use of diverted opioid medications, or use of illicitly obtained heroin. This disorder causes significant impairment or distress, as manifested by multiple psychosocial, behavioral, or physiologic features. This disorder has a high morbidity and mortality rate.
- Opioid intoxication/overdose manifestations include miosis, respiratory depression, motor slowness, euphoria, slurred speech, gastric atony, and pulmonary edema.
- General inspection – Poor hygiene and malnourishment. Inspect limbs for cutaneous scarring and hyperpigmentation due to repetitive IV injections (IV access may be difficult to obtain). Infected or inflamed skin excoriations (histamine release causes intense itching and scratching).
- Gastrointestinal – Hepatitis (B and C) is the most frequent complication of intravenous heroin abuse. Roughly 40% of IV heroin users have been exposed to some form of hepatitis. Patients with hepatitis B and C are at risk for developing fulminant liver failure and hepatocellular carcinoma. Look for signs of liver disease (jaundice, telangiectasia, ascites, scleral icterus, etc).
- Pulmonary – Higher risk for lung abscesses, bacterial pneumonia, respiratory depression, pneumonitis, and fibrosis.
- Cardiac – Higher risk of endocarditis. Any new murmur or unexplained fever in these individuals should be considered as endocarditis until proven otherwise.
- Immune system – higher risk infection due to propensity for malnourishment and HIV.
- Acute intoxication is treated with naloxone (opioid receptor antagonist) and supportive care in a hospital setting.

Opioid Withdrawal Syndrome

- Syndrome that manifests after abrupt withdrawal of opioids.
- Symptoms include tachycardia, hypertension, lacrimation, yawning, nausea, vomiting, diarrhea, muscle spasms, abdominal cramping, hyperthermia, diaphoresis, and mydriasis.
- Treatment can include the following:
  - Supportive for GI distress – fluid and electrolyte correction.
  - Methadone – mu opioid agonist. Used for the management of opioid dependence.
  - Buprenorphine – synthetic mixed mu agonist-antagonist. This drug is also used for opioid dependence. It can be used alone or in combination with naloxone.
  - Clonidine – alpha-2 adrenergic agonist used to attenuate the sympathetic manifestations of withdrawal (tachycardia, hypertension, mydriasis, diaphoresis).

Patient Management

- Obtain preoperative labs:
  - LFTs (prothrombin time, PTT, INR).
  - Hepatitis titers.
  - HIV titers.
  - CBC to rule out thrombocytopenia from splenomegaly.
- Any doubt of a patient's sobriety or apparent acute intoxication should result in a cancelled appointment. Order toxicology screening.
- Intravenous access may be difficult due to collapsed veins and fibrosis.
- Avoid preoperative administration use of mixed agonist/antagonist (buprenorphine) as it could precipitate withdrawal syndrome.
- Illicit narcotic use just prior to the surgery will have additive effects with other CNS-depressant drug administration (respiratory or cardiovascular failure may result in emergency resuscitation).
- Tolerance – patients usually require higher doses of an opioid to achieve the desired effect. Increased tolerance to the effects of

narcotics may result in overdosing and respiratory depression.
- Cross tolerance – many of these patients develop cross tolerance to other CNS depressants which could manifest itself as difficulty in achieving a profound level of anesthesia.
- Patients have propensity for hypotension.
- Be prepared to treat opioid overdose (naloxone, supportive care, and contact EMS).
- Preoperative antibiotics (prophylaxis for patients with a known history of endocarditis).
- Postoperatively, pain management can be difficult due to tolerance. Use acetaminophen-based products sparingly for patients with compromised liver function. Consider using NSAIDs instead of opioids. If opioids are indicated, the medication should be given sparingly and only for short periods of time. The amount and type must be tailored to suit the surgical procedure. Preoperatively discuss the pain management plan with patient, primary care physician, or pain manager/drug abuse manager.
- Patients may also have a prolonged QT interval which can degenerate into torsades de pointes.

Amphetamines/Amphetamine Analogs

Methamphetamines

- Methamphetamines are potent central nervous stimulators. 3,4-Methylenedioxymethamphetamine (MDMA AKA ecstasy) and methamphetamine hydrochloride (AKA crystal meth or ice) are commonly abused methamphetamines. Both are stimulants that increase neurotransmitters, especially at the 5-HT receptor, and slow the reuptake of the neurotransmitters dopamine, norepinephrine, and serotonin, in parts of the central nervous system. They have psychedelic effects that last for up to 6 hours and are typically taken by mouth. Ecstasy is commonly packaged in a pill form with fillers. "Molly" is pure molecular MDMA without the fillers.
- The desired effects are euphoria, aphrodisia, heightened sensation, and increased empathy.
- Physical manifestations include mydriasis, hyperthermia, tachycardia, hypertension, diaphoresis, anorexia, tremors, dehydration, bruxism, and insomnia.
- Cardiovascular effects – Autonomic hyperactivity is a major feature in patients presenting with MDMA toxicity and is dose-dependent.
- Serotonin syndrome – Condition in which central 5-HT receptor hyperstimulation results in classic findings of hyperthermia, mental status changes, autonomic instability, and altered muscle tone and/or rigidity.
- Hyponatremia – Various cases of seizure and death secondary to hyponatremia have been reported. The occurrence of hyponatremia after MDMA use is multifactorial, stemming from increased water intake, excessive sweating with physical exertion, and the release of vasopressin leading to syndrome of inappropriate antidiuretic hormone (SIADH).
- Neurologic effects – MDMA, like other amphetamines, can lead to a variety of potentially fatal neurologic outcomes, including subarachnoid hemorrhage cerebral infarction, or intracranial bleeds.
- Hepatotoxicity –Growing evidence suggests that MDMA may harm the liver. Hepatotoxicity ranges from asymptomatic liver injury with confirmation of elevation of liver function tests to fulminant acute hepatic failure.
- Dental caries – Long-term usage of methamphetamines can lead to dental caries secondary to a decrease in salivary production (Meth Mouth).

Bath Salts

Bath salts (MDPV, mephedrone, and methylone) – the principal active ingredients of bath salts products are synthetic cathinones. Cathinones are beta-ketone amphetamine analogs.

- Phenethylamines, including traditional amphetamines and the newer synthetic compounds, share multiple pharmacodynamic properties.

Stimulation of alpha and beta adrenergic receptors is primarily responsible for the acute sympathomimetic effects of amphetamines which include hyper-alertness, hypertension, tachycardia, mydriasis, and diaphoresis.

- Phenylethylamines also cause the release of neurotransmitters (dopamine, serotonin, and norepinephrine) and may also inhibit their reuptake. These neurotransmitters lead to the intense euphoria and hyper-alertness.
- Alpha-pyrrolidinopentiophenone (alpha-PVP) also known as FLAKKA (street name) – Alpha-PVP is chemically similar to other bath salts and takes the form of a white or pink, odorous crystal that can be consumed through many methods (e.g., eaten, snorted, injected, or vaporized in an e-cigarette). It is a potent stimulus for the release of dopamine in the brain, even more so than cocaine and methamphetamine.
- Cardiovascular abnormalities – tachycardia, hypertension, chest pain, dysrhythmias.
- Severe paranoia.
- Hallucinations, muscle spasms, insomnia, agitation, anxiety, delusions, seizures, and aggression. The stimulant effects of these drugs can last up to 4 hours.
- Major concern is excited delirium, which is considered a medical emergency due to the violent behavior and hallucinations often accompanied by seriously elevated body temperature. Excited delirium is a controversial proposed condition that manifests as a combination of delirium, psychomotor agitation, anxiety, hallucinations, speech disturbances, disorientation, violent and bizarre behavior, insensitivity to pain, elevated body temperature, hypertension, tachycardia, and superhuman strength.
- Cases of suicide by self-mutilation are not uncommon.
- Intense muscle spasms can precipitate rhabdomyolysis.

Patient Management

- Basic metabolic panel – assess sodium levels as the patient may require treatment of SIADH.
- Urine drug testing.
- Acute intoxication signs symptoms – case should be cancelled/postponed.
- EKG to monitor for signs of ischemic changes.
- Signs of acute intoxication – Anxiety, extreme agitation, panic reactions, and seizures will require benzodiazepines and perhaps restraints if the patient is showing loss of self-control.
- Clozapine results in a marked and immediate reversal of MDMA-induced hyperthermia, via inhibition of brain metabolic activation and blockade of skin vasoconstriction.
- Avoid sympathomimetic drugs such as ketamine.
- Beware of signs/symptoms of serotonin syndrome.
- Potential risk of dental trauma secondary to extensive decay during laryngoscopy, which is an ingestion/aspiration risk.
- The main acute danger of MDMA is its association with hyperpyrexia reaction and subsequent rhabdomyolysis.
- The hyperthermia associated with MDMA is very similar to severe heat stroke shown with neuroleptic malignant syndrome, serotonergic syndrome, and malignant hyperthermia. Carvedilol is effective in reducing MDMA-induced hyperthermia. Dantrolene to treat hyperthermia is evidenced throughout the literature.
- Be prepared to treat intraoperative myocardial ischemia (e.g., nitroglycerin, esmolol, morphine, oxygen). There is evidence that alpha-adrenergic blockade is sufficient to treating coronary artery vasospasm. Beta-adrenergic blockade may accentuate amphetamine-induced coronary artery vasospasm.
- Be prepared to treat induced seizures (e.g., benzodiazepines and oxygen).
- EMS should be contacted for signs and symptoms of excited delirium. In the hospital setting, benzodiazepines are used to treat agitation.
- MAC decreased with acute intoxication and increased with chronic use.

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Medical Emergencies

25

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Cardiopulmonary Resuscitation

- CAB: Compressions, Airway, and Breaths (Fig. 25.1).
- Compressions are the priority (advanced airways not a priority, as there is no gain in immediate rate of survival).
- Give rescue breaths every 6–8 seconds if advanced airway in place (better 24 hours survival if advanced airway in place).
- Compress at a rate of 100–120/min with a depth of:
  - At least 2 inches (5 cm) for adults.
  - At least one-third the depth of the chest, about 2 inches (5 cm), for children.
  - At least one-third the depth of the chest, about 1½ inches (4 cm), for infants.
- Cycles of 30 compressions: two rescue breaths, use AED as soon as it available.
- In pediatric rescue attempt, use 15:2 ratio if a second rescuer arrives.

Automated External Defibrillator (AED)

- Two dysrhythmias can be treated: pulseless ventricular tachycardia and ventricular fibrillation.
- AEDs use a biphasic algorithm of shocks (120–200 J).
- Two pad placements positions:
  - Anterolateral – below right collarbone.
  - Anteroposterior – left side chest between breastbone and nipple and left side back next to spine.
- Pediatric pads are for those 8 years of age and younger. You can use adult pads on children but not child pads for adults.
- Chest hair should be removed. It can be removed rapidly with additional pads.
- Victims submerged in water should be removed and the chest should be dried (okay if victim is in small puddles or snow).
- Do not place pads directly on implanted defibrillators, pacemakers, or transdermal patches as they may block the shock. Additionally, shocks may damage the implanted devices and reduce the chances that pacing spikes are misinterpreted by AED.

Drugs That Can Be Given via ETT

- Acronym to aid remembering is NAVEL. In general, 2–2.5 × IV dose.
- *N*aloxone
- *A*tropine – pediatric 0.04–0.06 mg/kg.

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https://doi.org/10.1007/978-3-030-48880-2_25

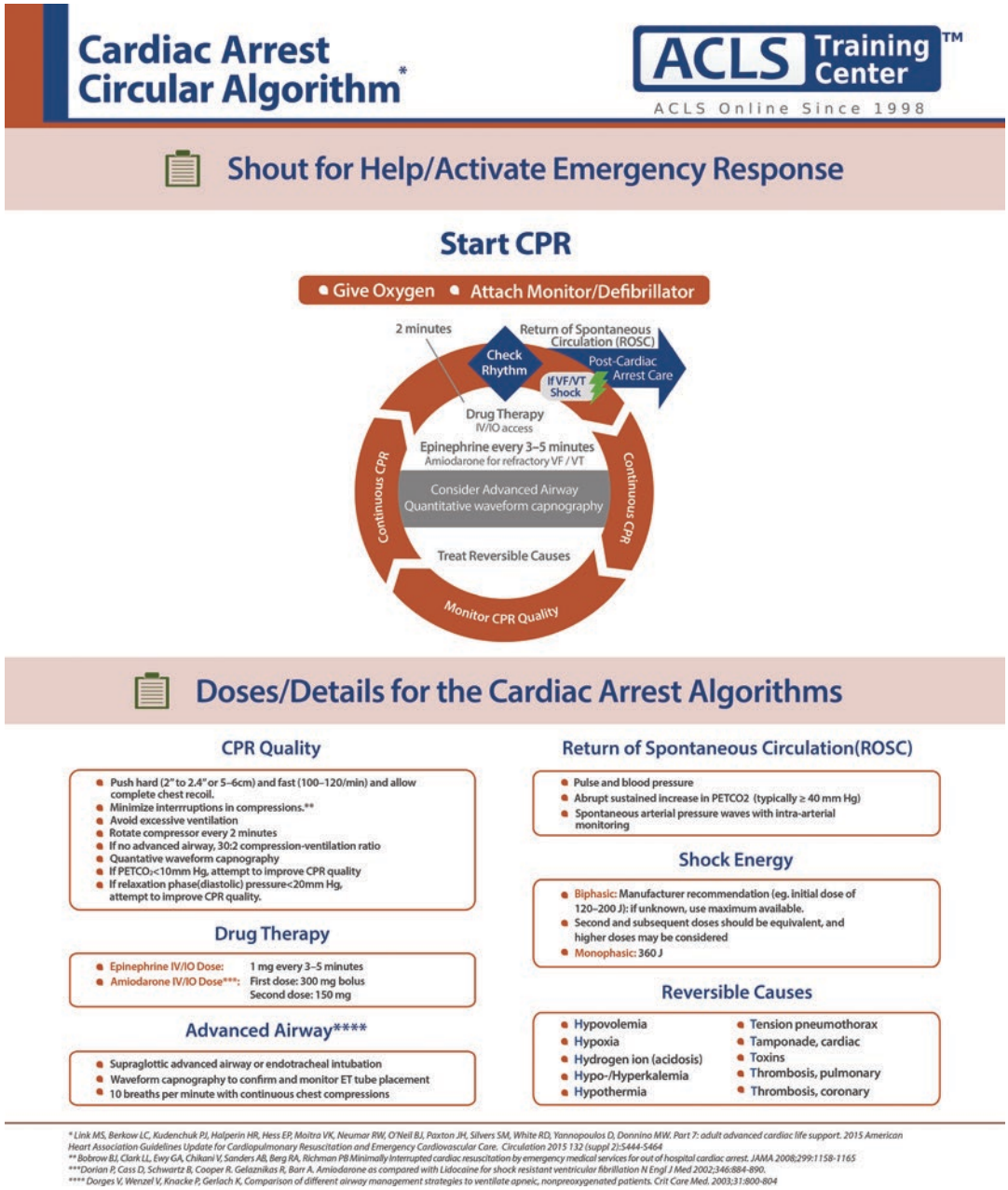


Fig. 25.1 Respiratory arrest algorithm. (Copyright 2016 ACLS Training Center, https://www.acls.net)

- *V*asopressin – data shows equally effective as IV. 40 units diluted in 10 cc saline.
- *E*pinephrine – pediatric dose should be 10 × IV.
- *L*idocaine

Hypertension (HTN)

- Perioperative HTN increases risk for ischemia, infarction, heart failure, and cerebrovascular accident.
- BP of less than 180/110 mmHg without end organ disease is not a risk factor for perioperative cardiac complications.
- Pulse pressure >80 mmHg is a more sensitive indicator for cardiovascular and cerebrovascular risk than SBP or DBP.
- Aggressive reduction in BP >20% may cause ischemic damage to brain, heart, and kidney.
- Hypertensive urgency – BP >180/120 mmHg without organ damage. Treated with oral/IV medications [1, 2].
- Hypertensive emergency – BP >180/120 mmHg with end organ damage (e.g., chest pain, changes in vision, confusion, nausea/vomiting), require hospitalization and rapid decrease in BP (MAP reduction 25% first hour and to target BP over the next 6 hours) to limit organ damage with IV medications [1, 2].
- Intraoperative high BP – stop stimulus and confirm BP. Clinician should ensure an adequate level of sedation, profound local anesthesia, and optimal oxygenation. Treat with antihypertensives.

Antihypertensives

Esmolol
- Ultra-short acting beta-1 selective blocker.
- Broken down via RBC esterase in the cytosol (not dependent on renal or hepatic clearance).
- 5–10 mg over 1 minute every 3 minutes with a maximum dosage of 300 mg.
- Onset within 1 minute with duration 10–20 minutes.
- Excellent agent for HTN with tachycardia.

Labetalol
- Non-selective beta-blocker and selective alpha-1 blocker.
- Good for HTN with tachycardia.
- 5–10 mg IV every 10 minutes with a maximum dosage of 300 mg.
- Onset within 5 minutes with a duration 3–6 hours.

Hydralazine
- Direct arterial vasodilator.
- Causes reflex sympathetic activation and tachycardia.
- Good treatment of choice for HTN with bradycardia (avoid in patients at risk for myocardial ischemia).
- 2.5–5 mg IV over 2 minutes (redose every 10 minutes) with a maximum dosage of 25 mg.
- Onset 5 minutes with duration of action of 2 hours.

Nitroglycerin
- Venodilator at low doses, which reduces preload and cardiac output.
- Arterial vessel dilator at high doses, which decreases afterload allowing blood entry to the aorta.
- 5–10 mcg/min with increase by 5–10 mcg/min every 5 minutes IV.
- Onset 2–5 minutes with a duration 10–20 minutes.
- Must ask if the patient has used erectile dysfunction medications within 48 hours (washout period) as it can lead to hypotension unresponsive to vasopressors.
- May use 0.4 mg sublingual every 5 minutes for a total of three doses.
- Good for HTN with bradycardia.
- Initial medication of choice for treatment of angina.
- Contraindicated for patients with hypotension (SBP <90 or ≥30 mmHg below baseline) and use of phosphodiesterase inhibitors.

Hypotension

- Generally said to be a BP <90/60 mmHg. No widely accepted definition intraoperative but widely held as a drop of systolic arterial blood pressure >25% from baseline [3].
- Leads to inadequate tissue perfusion to vital organs.
- Place the patient in supine position and elevate the legs.
- Administer supplement 100% oxygen.
- 250–500 cc NS or LR infusion (cautious use in patients with CHF or severe renal disease as to prevent volume overload).
- Check oxygenation and ventilation with auscultation of heart and lungs.
- Recheck blood pressure often.
- Attempt to determine the source (allergic reaction, hypovolemia, anesthesia depth, pulmonary embolism, pneumothorax, etc.).

Medications to Treat Hypotension

Atropine
- Cholinergic antagonist.
- Most useful in hypotension with bradycardia.
- 0.5 mg increments every 2–3 minutes to a max of 3 mg.
- 0.5 mg IM or sublingual every 5 minutes to max 3 mg.

Ephedrine
- Hypotension with normal heart rate.
- α and β agonist.
- 2.5–5 mg IV every 5–10 minutes to a 50 mg max dose.
- 25 mg IM or sublingual q 5–10 minutes to a 50 mg max dose.
- Onset 1 minute (peak 15 minutes) with a duration 1 hour.
- Available as 50 mg/mL (need to dilute 9 cc of sterile saline with 1 mL solution to obtain a concentration of 5 mg/mL).

Phenylephrine
- Selective α agonist.
- Used to treat hypotension with tachycardia or if an increase in heart rate should be avoided.
- Available as 10 mg/mL (need a 1% solution, dilute 1 mL in 9 mL of saline then take 1 cc and further dilute in 9 mL of saline allowing for a 100 mcg/mL).
- 100 mcg/mL q 5 minutes.
- Onset 2–3 minutes.

Tachycardia

- Tachycardia is a heart rate >100/min.
- Symptomatic tachycardia is tachycardia with symptoms: shortness of breath, angina, dyspnea on exertion, and mental status change or clinical signs such as pulmonary edema, rhonchi, hypotension, peripheral edema, jugular distension, ischemic EKG changes (Fig. 25.2).
- Heart rates >150/min not considered a normal physiological response to stress.
- Obtain ECG and ensure IV access.
- Rule out common causes of sinus tachycardia including pain, dehydration, and hypoxia.
- Supraventricular (SVT) origin – QRS complex is narrow with an origin at or above AV node. E.G. – Sinus tachycardia, A-Flutter, A-fib, paroxysmal supraventricular tachycardia (PVST).
- Ventricular origin – QRS wide with a ventricular origin. E.G – ventricular tachycardia.
- Always rule out the Hs and Ts (Table 25.1).

Bradycardia

- Normal HR for adults 60–100/min.
- Bradycardia – HR < 60/min (generally rate <50/min associated with symptoms).
- Bradycardic rhythm's examples include sinus bradycardia, first degree AV block, second degree type I (Wenckebach/Mobitz I), second degree type II (Mobitz II), and third degree AV block (Fig. 25.3).
- Symptomatic bradydysrhythmia's signs: hypotension, CHF, pulmonary congestion, and acute MI. Symptoms: angina, fatigue, altered mental status, light-headedness. (See Fig. 25.4 for treatment algorithm).

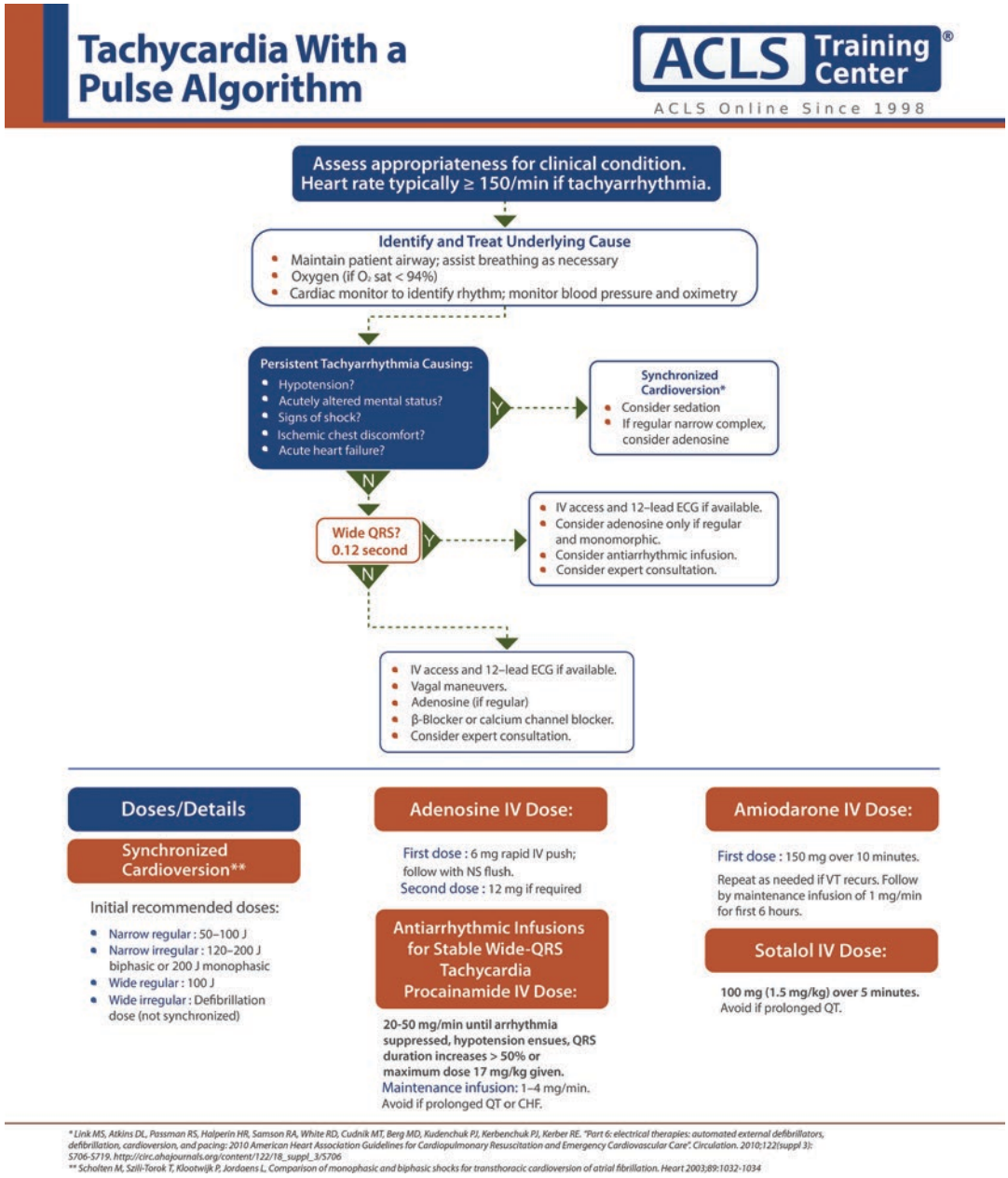


Fig. 25.2 Tachycardia with pulse algorithm. (Copyright 2016 ACLS Training Center, https://www.acls.net)

Table 25.1 The most common correctable causes of arrhythmias

Reversible causes	
Hs	Ts
*H*ypoxia	*T*ension pneumothorax
H^+ (acidosis)	*T*amponade (pericardial)
*H*yper/hypokalemia	*T*oxins (overdose, digoxin, Ca^{2+} blockers, beta-blockers)
*H*ypothermia	*T*hrombosis (P.E. or MI)
*H*ypovolemia	

Angina

- Chest pain due to inadequate blood flow to the myocardium as a result of demand–supply imbalance within the coronary arteries.
- Classic signs of angina: retrosternal pressure/discomfort, SOB, pain along ulnar surface of left arm.
- Chest pains that resemble angina: esophageal disorders, esophageal motility disorders, biliary colic, costochondritis, pericarditis, pulmonary embolism.
- Treatment pneumonic is ONAM (oxygen, nitroglycerin, aspirin, morphine). Aspirin may decrease absorption of nitroglycerin.

Classifications

- Chronic stable ischemic heart disease (stable angina) – follows a precipitating event and is relieved by rest or use of sublingual nitroglycerin.
- Unstable – occurs at rest and usually prolonged >20 minutes (considered an acute coronary syndrome).
- Prinzmetal – occurs at rest, cyclical, caused by coronary artery vasospasm.

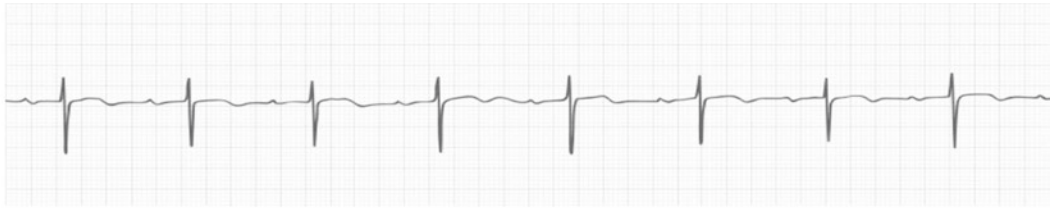
Management of a Patient with Angina (for treatments please see Fig. 25.5):

- Pulse oximetry, EKG, BP monitors.
- Oxygen delivery 4 L/min nasal cannula or 6 L/min nasal hood.
- Concerning EKG findings include:
  - ST elevation of two or more leads of 2 mm + in leads V2, V3 and 1 mm + in all other leads
  - Hyper acute T waves (earliest EKG finding)
  - ST segment depression (horizontal or down sloping)
  - Q waves or T wave inversions should alert for transfer to acute care setting.
- Ensure diastolic BP >90 mmHg and no erectile dysfunction agents use within 24 hours if sildenafil (Viagra®) or 48 hours tadalafil (Cialis®).
- Provide 0.4 mg SL tablet or spray q 5 minutes as needed as symptoms resolve (be aware of side effects such as hypotension or reflex tachycardia).
- EMS to be called if three doses of nitroglycerin over 15–20 minutes fails to relieve symptoms or if after one dose for unstable angina.
- Non-enteric coated ASA (4 tabs 81 mg or 1 tab 325) crushed or chewed (may give 300 mg rectal suppository if cannot be given by mouth).
- Morphine 2–4 mg IV initial; 2–8 mg IV subsequent if unresponsive to nitroglycerin. Reduces peripheral resistance (afterload) and venous capacitance (preload).
- ACLS protocols as needed; monitor vital signs closely.

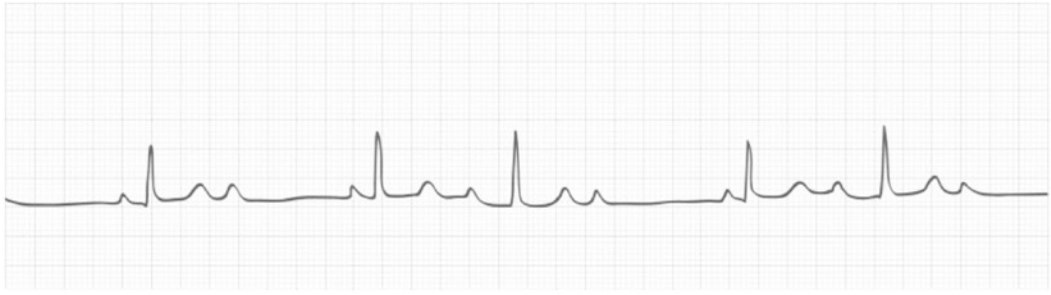
Acute Coronary Syndromes

- Unstable angina – angina not relieved by rest secondary to a partially occlusive coronary thrombus. Cardiac enzymes are usually negative.
- Acute Myocardial Infarction – myocardial necrosis that occurs secondary to an occlusive thrombus. In order for a patient to be diagnosed with a myocardial infarction, they must have at least two of the following three criteria, according to the World Health Organization:
  1. Angina consistent with ischemia.
  2. Elevation of cardiac markers in blood (Troponin-I, CK-MB, Myoglobin).
  3. Characteristic changes on electrocardiographic tracings taken serially.
     - Types of myocardial infarctions:
       - Non-ST Segment Elevated Myocardial Infarction (NSTEMI) – results in a subendocardial infarct.

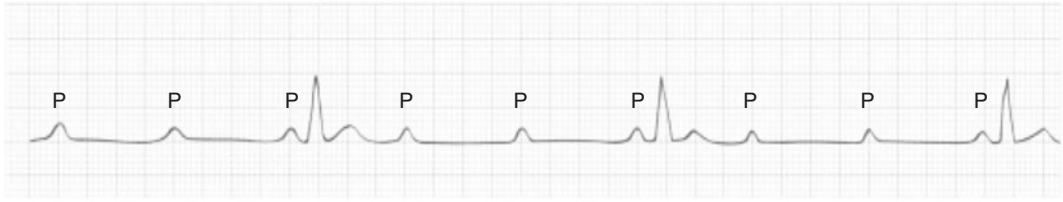
Atrioventricular Blocks



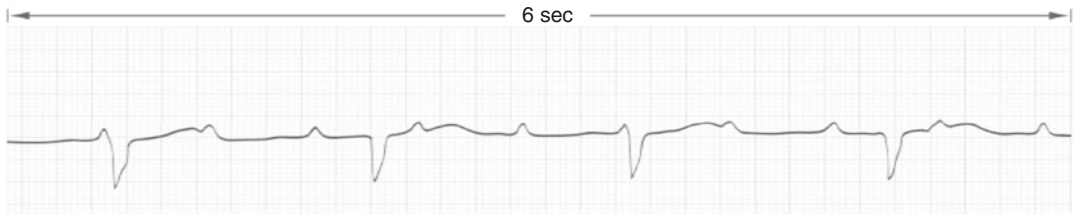
First-degree AV Block. PR interval (>200 msec)



Second Degree AV Block Type I – PR elongation until non-conducting atrial impulse (in this EKG between 2nd and 3rd beats).



Second Degree AV Block Type II – no PR widening, narrow QRS, conduction disturbances infranodal. In this example a 3:1 conduction block is shown.



Third Degree AV block aka complete hear block – absent conduction of all atrial impulses with slow escape rhythm. Pacemakers above the His bundle are associated with a narrow-Complex QRS at a HR of 45 to 60 /min, whereas pacemakers at or below the His bundle produce a wide-complex QRS at a HR of 30 to 45 /min.

Fig. 25.3 Bradyarrhythmias

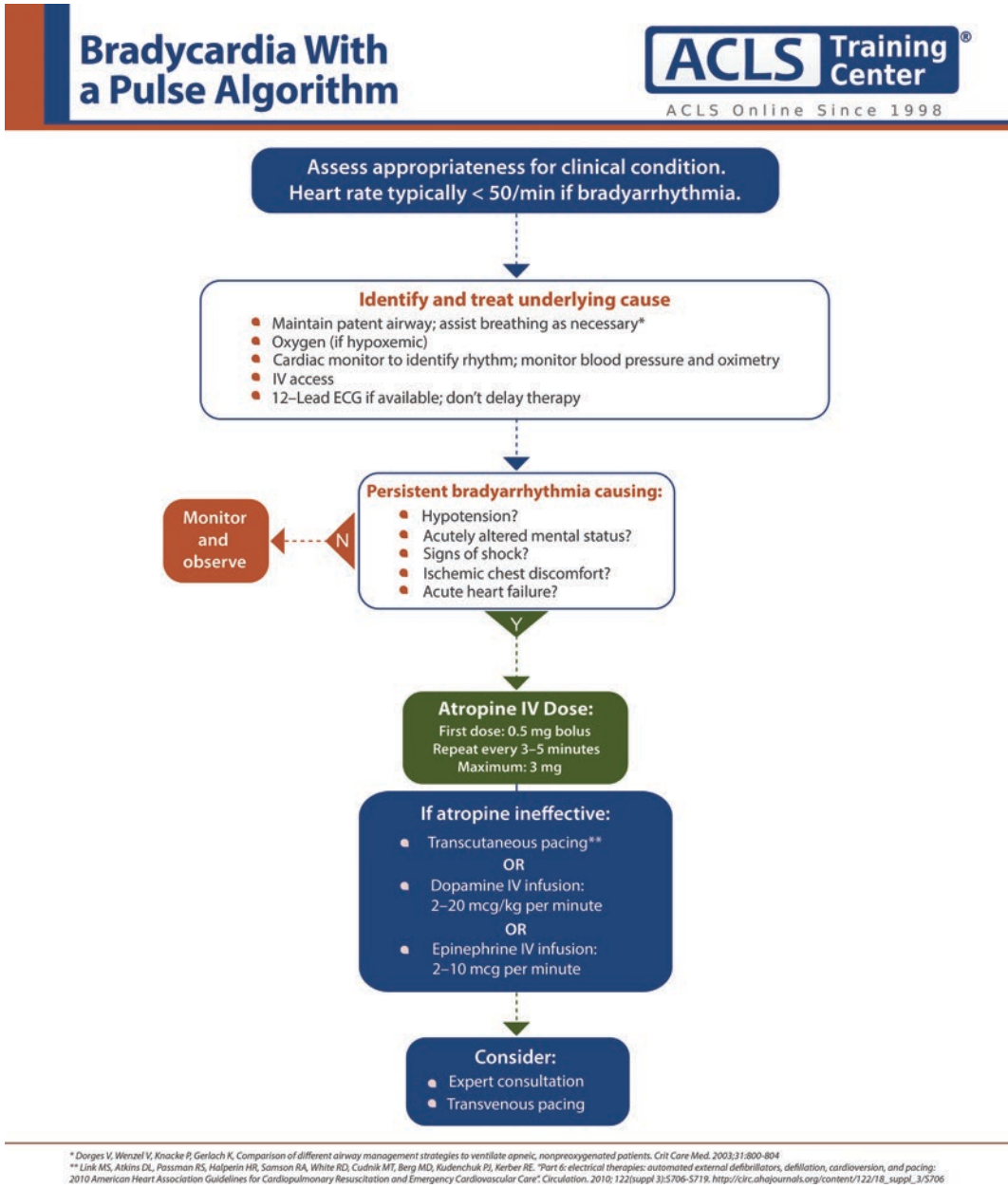


Fig. 25.4 Bradycardia with pulse algorithm. (Copyright 2016 ACLS Training Center, https://www.acls.net)

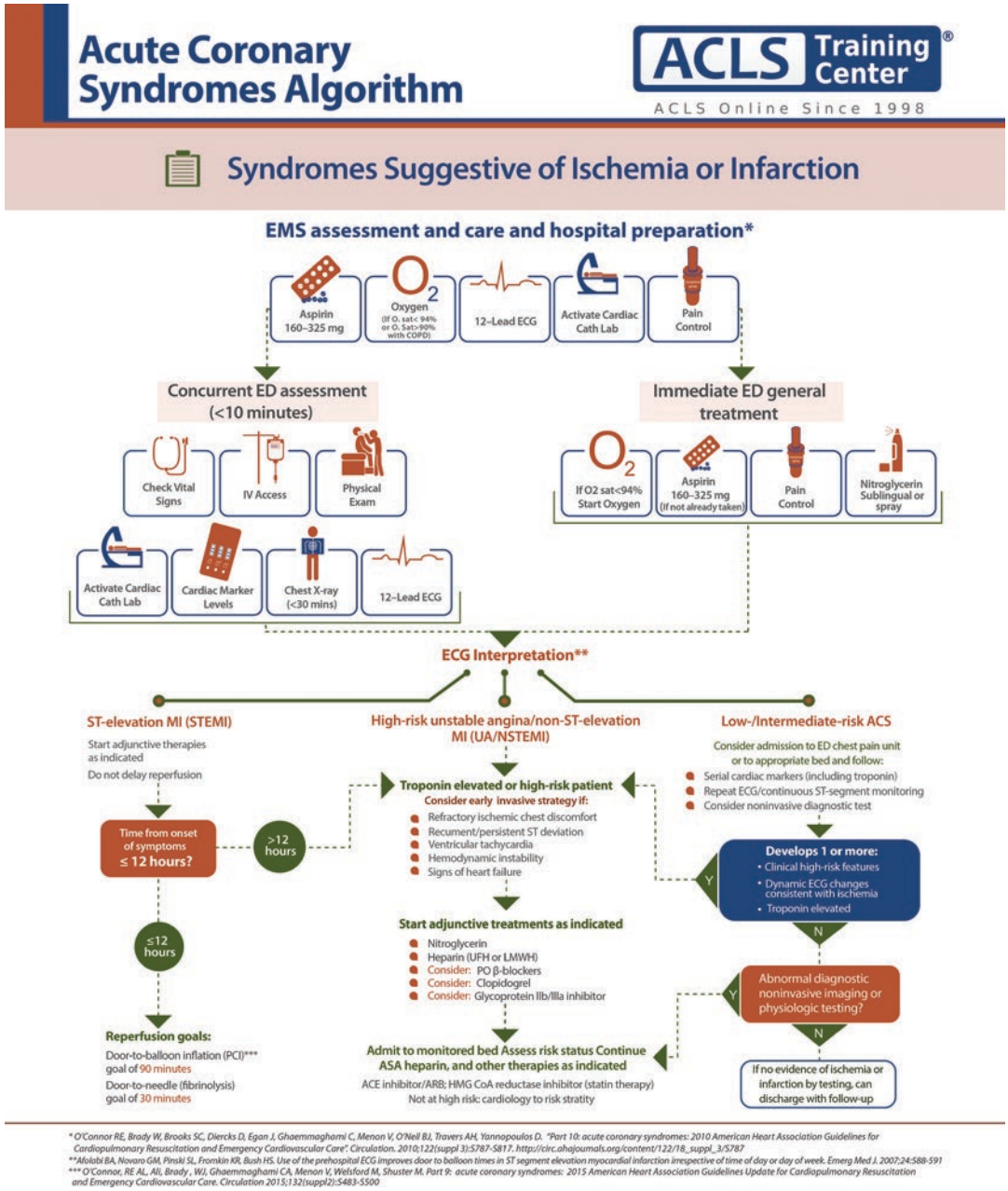


Fig. 25.5 Acute coronary syndromes algorithm. (Copyright 2016 ACLS Training Center, https://www.acls.net)

  - ST Segment Elevated Myocardial Infarction (STEMI) – results in a transmural infarct.

Treatment of ACS

- Unstable Angina – Treatment is similar to stable angina. Therapy with heparin may be indicated to prevent progression of the thrombus. Patients may be started on anti-platelet agents.
- Myocardial Infarction Treatment
  1. Fibrinolysis – option for patients with STEMI and symptoms within 12 hours and if percutaneous coronary intervention is not available within 90 minutes of arrival to ED. Fibrinolytic agents: (1) recombinant tissue plasminogen activator (rTPA), fibrin specific (2) streptokinase, activates plasminogen to plasmin. Ideal time of door to drug ≤30 minutes.
     - Contraindications: systolic BP ≥185 or diastolic ≥110 mmHg, right vs. left BP differential >15 mmHg, structural disease CNS, closed head/facial trauma w/I 3 months, stroke >3 hours or <3 months, recent trauma/surgery or GI/GU bleed, history of intracranial hemorrhage, blood thinners, clotting issues, pregnancy, serious systemic disease, blood glucose <50 or ≥400 mg/dL. High risk patients that have HR >100/min or BP <100 mmHg, pulmonary embolism, shock or required CPR should be given PCI treatment.
  2. Percutaneous coronary intervention (PCI) – a procedure to open narrow coronary arteries via catheterization of a peripheral artery leading to deployment of either a balloon and/or stent. It has proven to be superior to fibrinolytic therapy if greater than 3 hours from onset. Ideal door to balloon inflation time goal ≤90 minutes.

Cardiac Enzymes

- Troponins:
  - Cardiac-specific proteins not normally present in the serum of healthy individuals (troponin I and troponin T).
  - Presence of either troponin levels are equally diagnostic and levels rise 3 hours after onset, remain elevated for 7–14 days after. Hence, good to detect MI even up to 2 weeks after onset.
- Creatine Kinase-Myocardial Band (MB):
  - Found mainly in the heart muscle.
  - Best marker to test for early reinfarction.
  - Starts to rise 3 hours after onset, peaks at 24 hours, and normalizes within 48–72 hours.
  - Facilitation of MI diagnosis by calculating CK index. Index greater than 2.5 suggests cardiac muscle damage.

Premature Ventricular Complexes (PVC)

- Ectopic impulses originating in the ventricular myocardium.
- See widened QRS complex without a preceding P wave (Fig. 25.6).
- Volume of blood ejected during PVC is smaller than that ejected during a sinus beat.
- Common reasons for PVC development: hypoxemia, catecholamine excess (pain, anxiety, caffeine, cocaine, or amphetamines), MI, valvular heart disease, electrolyte imbalances, or digitalis toxicity.
- Lown's criteria is used to classify PVC according to frequency and morphology (Table 25.2) [4]. First three classes, if asymptomatic, do not require treatment.
- PVC should be treated if 6 or more PVCs/min, polymorphic, occur in runs of ≥3, or R-on-T phenomenon. Must treat even if asymptomatic, as there is an increased risk of developing life-threatening ventricular dysrhythmia (Lown's criteria 3–5).

Treatment:

1. Ensure defibrillator is available.
2. Rule out causative factors if setting allows (office vs. hospital setting).

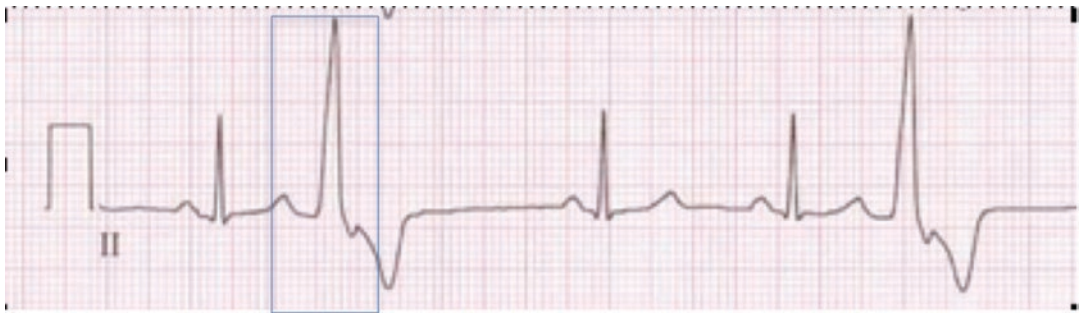


Fig. 25.6 Example of PVC

Table 25.2 Lown's criteria for PVC grading

Class	Description
1	<30/hour
2	≥30/hour
3	Multiform or multifocal
4A	2 consecutive
4B	≥3 consecutive runs of ventricular tachycardia
5	R on T phenomenon

3. Treat with β-blockers such as esmolol (500 mcg/kg IV) or Metoprolol (5–10 mg IV q15 mins). Antidysrhythmics are indicated if VT develops.

Ventricular Tachycardia (VT)

- A rapid heartbeat that originates in one of the ventricles of the heart that occurs when three or more consecutive ventricular beats occur at a heart rate of more than 120 (usually between 150 and 200).
- Designated as sustained if lasts for 30 seconds or more or non-sustained (self-terminating).
- QRS complexes >0.12 seconds.
- Designated as monomorphic ventricular tachycardia when QRS complex appears at a regular size and rate.
- Designated as polymorphic (e.g. Torsades de pointes) when multiple ectopic foci or there is presence of a re-entry circuit.
- May be hard to distinguish monomorphic VT from SVT.
  - SVT more likely if: (1) QRS morphology closely resembles ECG in sinus rhythm (2) dysrhythmia responds to vagal maneuvers.
  - VT more likely if (1) no relationship P wave/QRS complexes (2) QRS in chest leads all similar appearance with dominant positive or negative deflection.
- Treatment requires immediate CPR with early defibrillation (best within 3–5 minutes and should be used as early as possible if available). A series of chest compressions is not recommended before defibrillation. Drug therapy includes epinephrine, amiodarone, and vasopressin.
- Always attempt to rule out reversible causes (Hs and Ts).

Ventricular Fibrillation

- Uncoordinated fluttering of ventricles leading to cessation of cardiac output (incompatible with life).
- Treatment requires immediate CPR with early defibrillation (best within 3–5 minutes and should be used as early as possible if available). A series of chest compressions is not recommended before defibrillation. Drug therapy includes epinephrine, amiodarone, and vasopressin (Fig. 25.7).
- Always attempt to rule out reversible causes (Hs and Ts).

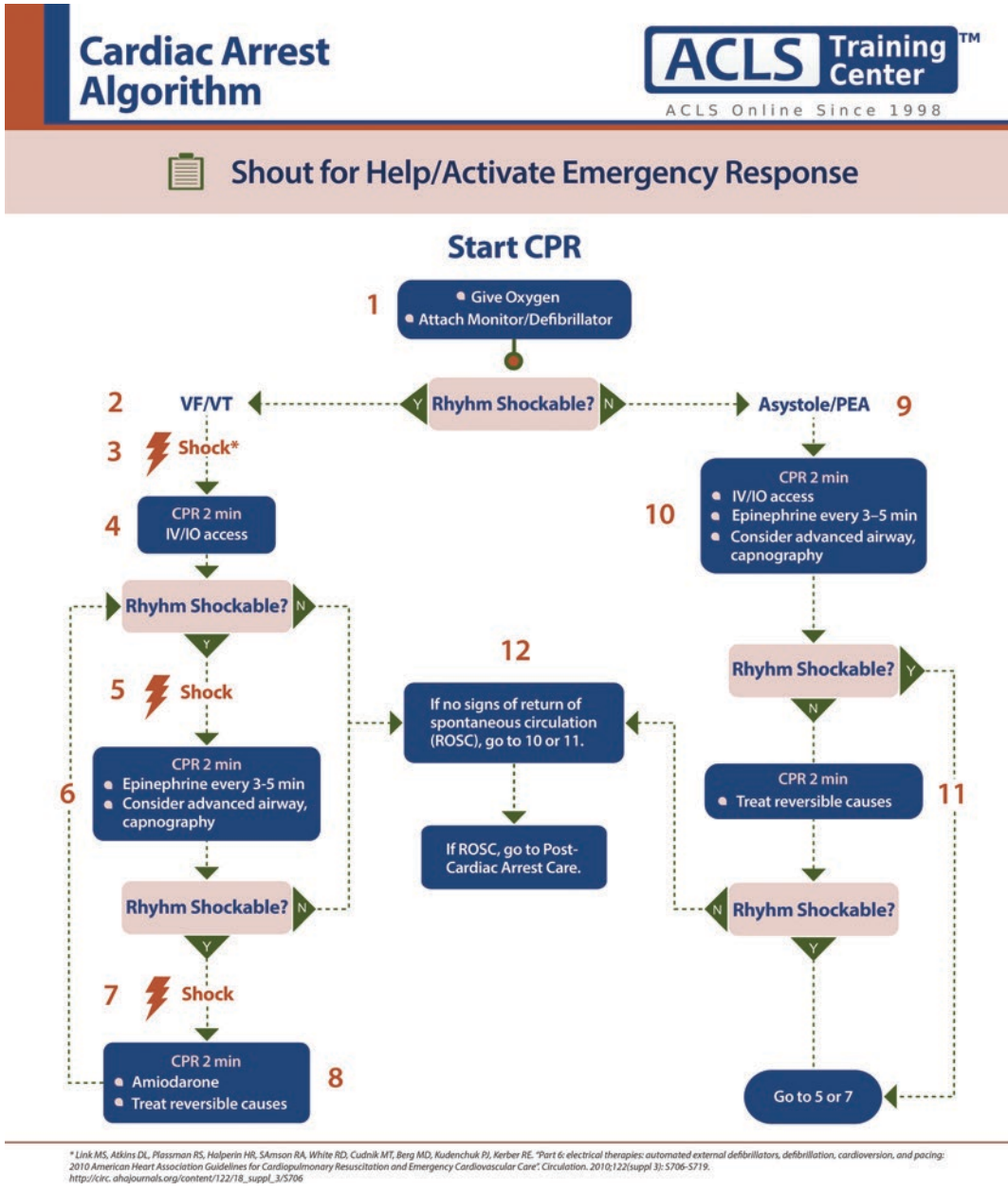


Fig. 25.7 VF/VT/asystole/PEA algorithm. (Copyright 2016 ACLS Training Center, https://www.acls.net)

Pulseless Electrical Activity

- Presence of organized cardiac electrical activity without sufficient mechanical contraction to produce a palpable pulse.
- Poor prognosis for survival and neurological outcome.
- Any rhythm or heart rate may be present without a pulse.
- No benefit from defibrillation (CPR is most effective with minimal interruptions).
- Normally caused by reversible condition and can be treated if identified (e.g., cardiac tamponade, hypovolemia, drug overdose).
- Treatment See Fig. 25.7.

Torsades de Pointes

- Atypical VT with a polymorphic QRS.
- Non-uniform delay of repolarizations producing early after depolarizations (exhibited as a long QT interval).
- Seen in electrolyte imbalances (e.g. hypokalemia, hypomagnesaemia), persistent bradycardia, and drugs that block cardiac potassium currents.
- Many medications can prolong the QT interval and predispose to Torsades de pointes (e.g., erythromycin, methadone, and phenothiazine).
- Normally symptomatic and can lead to ventricular fibrillation.
- Treatment is magnesium sulfate (loading dose 1–2 g IV diluted in D5W/NS which is given over 5–20 minutes).

Allergic Reactions

- Majority of anaphylaxis cases are type I (IgE mediated).
- Can also be non-immune (anaphylactoid reaction) as the mediators are released by direct interaction with offending drug.
- Class I reactions, B lymphocytes produce IgE that binds to mast cells and basophils that cause degranulation leading to release of histamines and other autacoids such as leukotrienes and prostaglandins.
- Severity ranges from simple contact dermatitis to Stevens-Johnson syndrome and toxic epidermal necrolysis.
- The most common are feelings of warmth and tingling. Pruritus with accompanying flushing and urticaria is also common.
- Angioedema – swelling of dermis and subcutaneous/submucosal tissues due to vascular leakage. Often involves swelling of the lips and eyelids.
- Can lead to airway compromise due to laryngeal swelling.
- Anaphylaxis – life-threatening condition with cardiovascular collapse, interstitial edema, and bronchospasm.

Treatment of Mild Allergy

- Diphenhydramine 50 mg IV/PO or 100 mg IM.
- Histamine may circulate for 3 days or more, so oral diphenhydramine should be given every 4 hours for the first 2 days. Warn the patient of sedative effects with both oral and written instructions.

Treatment of Severe Allergic Reaction/ Anaphylaxis

- Secure and maintain airway.
- Follow ACLS protocols.
- Fluids: 1 L bolus LR for adults or 20 mL/kg LR or NS in children.
- Epinephrine:
  - IM 1:1000 0.15 mg if 10–25 kg or 0.3 mg >25 kg in vastus lateralis or deltoid muscles. Repeat q 5–15 minutes based on response.
  - IV: 10 mcg to 1 mg bolus q 2 minutes in adults, or 1–10 mcg/kg bolus q 1–2 minutes

for children every 2–5 minutes based on response.
- β-2 agonist inhaler for bronchospasm such as an albuterol metered dose inhaler (90 mcg/activation). Usually 2–3 puffs and can use a chamber if patient cannot coordinate inhalation.
- Antihistamines:
  - Histamine 1 blocker – diphenhydramine 0.5–1 mg/kg IV for children or 50 mg for adults. Children 6–12 years old 25 mg IV.
  - Histamine 2 blocker – famotidine dosage for adults is 20 mg IV, or children 0.5 mg/kg IV.
- Steroids: Hydrocortisone 1–2.5 mg/kg IV or 100 mg IV over 1 minute, methylprednisolone 1 mg or dexamethasone 4–12 mg IV slowly over 1 minute.
- Patient to be admitted or transferred to an acute care facility for further monitoring.

Malignant Hyperthermia

- Hypermetabolic state that occurs on exposure to volatile anesthetics agents (except nitrous oxide) and succinylcholine.
- Due to a genetic mutation in the ryanodine receptor, found on the sarcoplasmic reticulum of skeletal muscle, which lowers the threshold of calcium release channel activation [5].
- Elevated calcium levels lead to sustained muscle contraction.
- Linked to Central Core Disease and King-Denborough Syndrome.
- Possible association with Duchenne Muscular Dystrophy, Becker Muscular Dystrophy, and exercise-induced rhabdomyolysis and/or history of dark-colored or brown-colored urine (myoglobinuria) post previous anesthesia [6].
- Early signs are sinus tachycardia, hypercarbia with no improvement of compensatory increase in minute ventilation, and masseter spasm.
- Later signs include dysrhythmias, peaked T waves on EKG due to hyperkalemia, increase in core temperature, dark blood in surgical site, and whole-body spasm.
- Testing includes caffeine-halothane contracture test and RYR1 gene testing.

Malignant Hyperthermia Treatment

- Stop procedure, discontinue volatile agents administration, and call for assistance (911 if office based).
- Call Malignant Hyperthermia Hotline early in progression if personnel available to assist. 1-800-MHHYPER (1-800-644-9737).
- "Tubes in all orifices."
- Hyperventilate with 100% oxygen.
- Dantrolene 2.5 mg/kg IV as bolus and q 5–10 minutes up to 10 mg/kg. 1 mg/kg IV every 6 hours for 72 hours for prevention. Preparation of Dantrolene – 20 mg dantrolene bottle mixed with 60 mL sterile water and 3 g of mannitol.
- IV fluids to maintain urine output to 2 mL/kg/h. Supplement with mannitol 0.25 g/kg IV or furosemide 1 mg/kg IV.
- Surface ice packs and intracavity lavage of the stomach and bladder with cold saline. (Goal to cool core to 38 °C).
- Correct metabolic acidosis with sodium bicarbonate 1–2 mEq/kg IV.
- Hyperkalemia treated with calcium chloride 5–10 mg/kg IV or regular insulin 0.15 U/kg in 1 mL/kg of 50% dextrose.
- Frequent blood gases every 15 minutes until abnormality is stopped.
- Treat cardiac dysrhythmias as needed.
- Observe in ICU and monitor for renal failure, recurrence, myoglobinuria, and disseminated intravascular coagulation.

Laryngospasm

- Spasm of the glottis muscles including false and true vocal cords.
- More common during light sedation than deeper planes.
- Classified as complete or incomplete.

- Classic sign is a high-pitched stridor or crowing for a partial laryngospasm, while silent during complete spasm.
- Other signs include tracheal tug, flared nostrils, or suprasternal retraction (paradoxical breathing).

Laryngospasm Treatment

- Stop surgery, suction airway, and pack off surgical site.
- Administer 100% oxygen with positive pressure ventilation via a full-face mask to mechanically break the spasm (consider deepening the level of sedation).
- May attempt to depress chest to force air to elicit a patent airway. Consider succinylcholine 10–20 mg (subparalyzing dose) IV if partial obstruction. Rocuronium 0.6–1.2 mg/kg IV if history or suspicion of malignant hyperthermia.
- 1–2 mg/kg (intubating dose) should be administered and ETT tube placed if complete spasm. Consider administration of atropine 0.02 mg/kg to prevent bradycardia.
- Concern for post obstructive pulmonary edema (can occur hours later). Consider admission to hospital for monitoring.

Bronchospasm

- Lower airway obstruction due to contraction and spasm of bronchial smooth muscle.
- Signs include wheezing, both inspiratory and expiratory, fixed thoracic cage in an inspiratory position, and cyanosis.

Treatment of Bronchospasm

- Stop surgery, suction airway, and pack off surgical site.
- Administer 100% oxygen via positive pressure ventilation.
- If the patient is responsive, administer 6–10 puffs via an MDI (β-2 agonist albuterol). If deeply sedated and airway patent attempt nebulized, albuterol or 4–8 puffs with space chamber attachment.
- Epinephrine:
  - IM 1:1000 0.15 mg if 10–25 kg or 0.3 mg >25 kg in vastus lateralis or deltoid muscles. Repeat q 5–15 minutes based on response.
  - IV: 10 mcg to 1 mg bolus q 2 minutes in adults, or 1–10 mcg/kg bolus q 1–2 minutes for children every 2–5 minutes based on response.
- Diphenhydramine 25–50 mg IV for histamine control.
- 1–2 mg/kg succinylcholine (intubating dose) should be administered and ETT tube placed. Consider administration of atropine 0.02 mg/kg to prevent bradycardia.

Emesis

- Oral expulsion of gastrointestinal contents.
- Risk for aspiration leading to chemical/bacterial pneumonitis.

Treatment Emesis

- Turn patient to right side and in a Trendelenburg position to spare left lung.
- Suction oral cavity.
- Administer 100% oxygen and auscultate both lungs for wheezing and rhonchi.
- If the patient cannot maintain oxygen saturation, then transfer to emergency department. Consider monitoring in hospital setting for serial chest X-rays and blood gas analysis as aspiration pneumonitis can present hours later.

Intra-arterial Injection

- Leave catheter in place.
- Administer 10 cc of 1% lidocaine or procaine as vasodilation may decrease arteriospasm.
- Consider transfer to hospital for vascular surgery consultation.

Hypoglycemia

- Signs and symptoms caused by elevated levels of glucagon and epinephrine (autonomic response) and insufficient glucose (neuroglycopenic symptoms).
- < 60 mg/dL: anxiety, irritability, nausea, vomiting, flushing, hunger, and sweating.
- <50 mg/dL: see neuroglycopenic symptoms such as inattention, headache, lethargy, blurry vision, agitation, confusion, and focal neurological defects.
- <30 mg/dL: seizures and coma may ensue.

Treatment of Hypoglycemia

- Glucose measurement.
- IV access.
- Supplemental oxygen.
- EKG and BP monitoring.
- If the patient is able to swallow, give 15 g of a simple carbohydrate (such as 6 oz of regular soda or 1 tablespoon honey). Glucose does not absorb through oral mucosa (needs to be swallowed).
- If the patient is unable to swallow, obtain IV access. Adults D50W, 1 amp. Unpredictable rise in glucose levels from 40 to 350 mg/dL. Children should be given D25W, 2–4 mL/kg. Due to hypertonicity of the solution, small veins may sclerose. Response to IV dextrose is within 5 minutes but peaks at 30 minutes.
- If no IV access, provide glucagon SC or IM 1 mg for adults or 0.5 mg for children. Glucagon causes the liver to convert glycogen to glucose via glycogenolysis. The response is within 10 minutes (peaks 30 minutes and the duration is 1–2 hours). Not effective in patients with depleted glycogen stores such as alcoholics, malnourished, or elderly patients.

Stroke AKA Cerebrovascular Accident (CVA)

- The National Institute of Neurological Disorders and Stroke defines stroke as an acute brain disease of vascular origin with neurological dysfunction that persists longer than 24 hours.
- Classified by its cause: (1) ischemic or (2) hemorrhagic.
- Majority of ischemic strokes are thrombotic, caused by atherosclerotic plaques while embolic origin is mostly from the atrium of the right heart as seen in atrial fibrillation or left ventricle from an MI.
- Hemorrhagic strokes are mostly intracerebral while the remaining are subarachnoid.
- Hallmark of ischemic or hemorrhagic stroke is contralateral deficits of motor and sensory nerves of the cerebral hemisphere involved.
- Pre-hospital assessment for stroke is known as the Cincinnati Prehospital Stroke Scale (Fig. 25.8).
- "Time is brain" – The National Institute of Neurological Disorders and Stroke set of goals for timing of treatment:
  - Immediate general assessment by a stroke team, emergency physician, or other expert within 10 minutes of patient arrival.
  - Order STAT non-contrast CT.
  - Neurologic assessment by stroke team and CT scan performed within 25 minutes of arrival.
  - Interpretations of CT scan within 45 minutes of ED arrival.
  - Initiation of fibrinolytic therapy, if appropriate, within 1 hour of hospital arrival and 3 hours from onset of symptoms. rTPA can be administered in "well screened" patients who are at low risk for bleeding for up to 4.5 hours (Fig. 25.9).
  - Door-to-admission time of 3 hours in all patients.

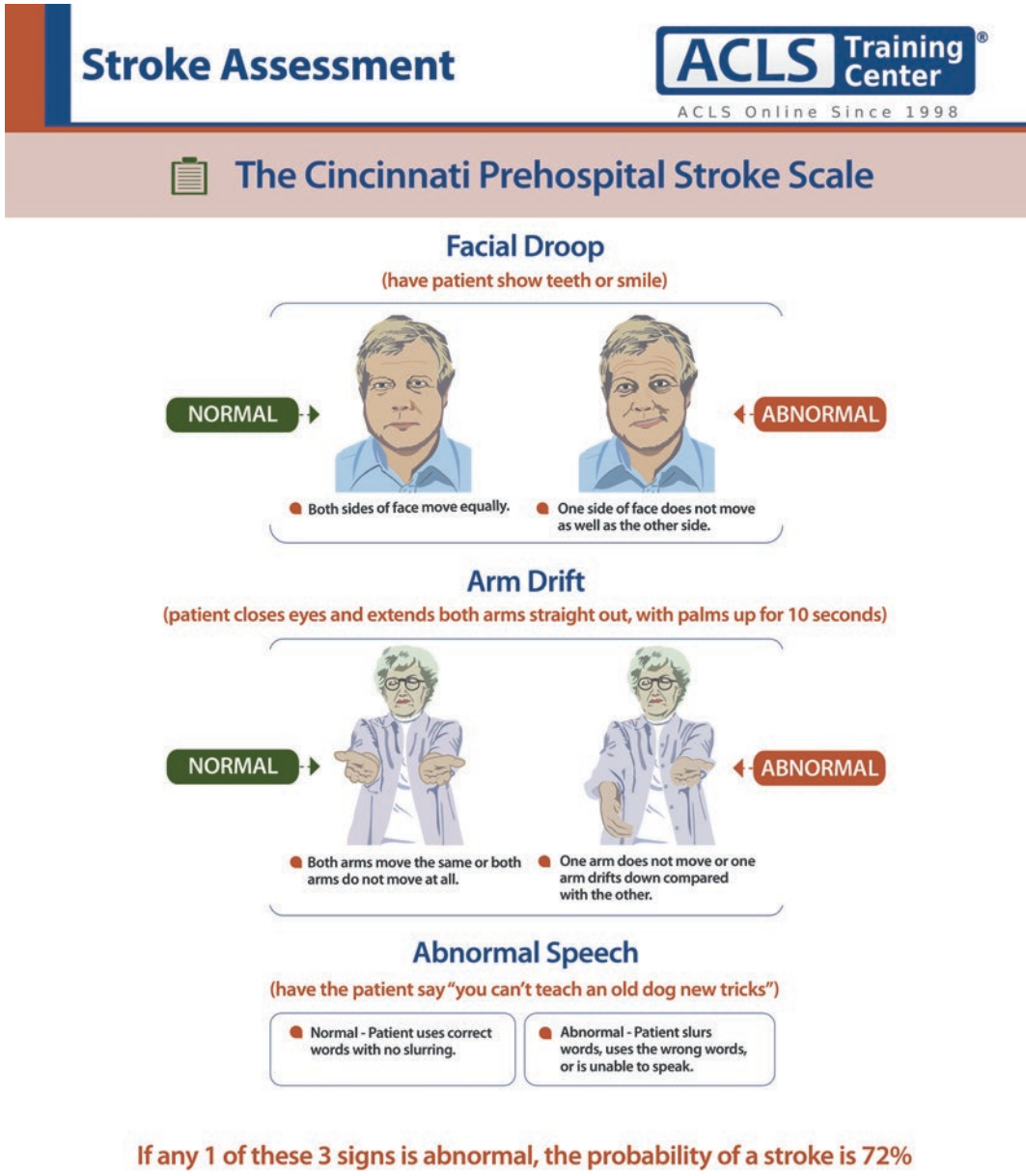


Fig. 25.8 Stroke assessment and treatment. (**a**) Cincinnati pre-hospital stroke assessment. (Copyright 2016 ACLS Training Center, https://www.acls.net)

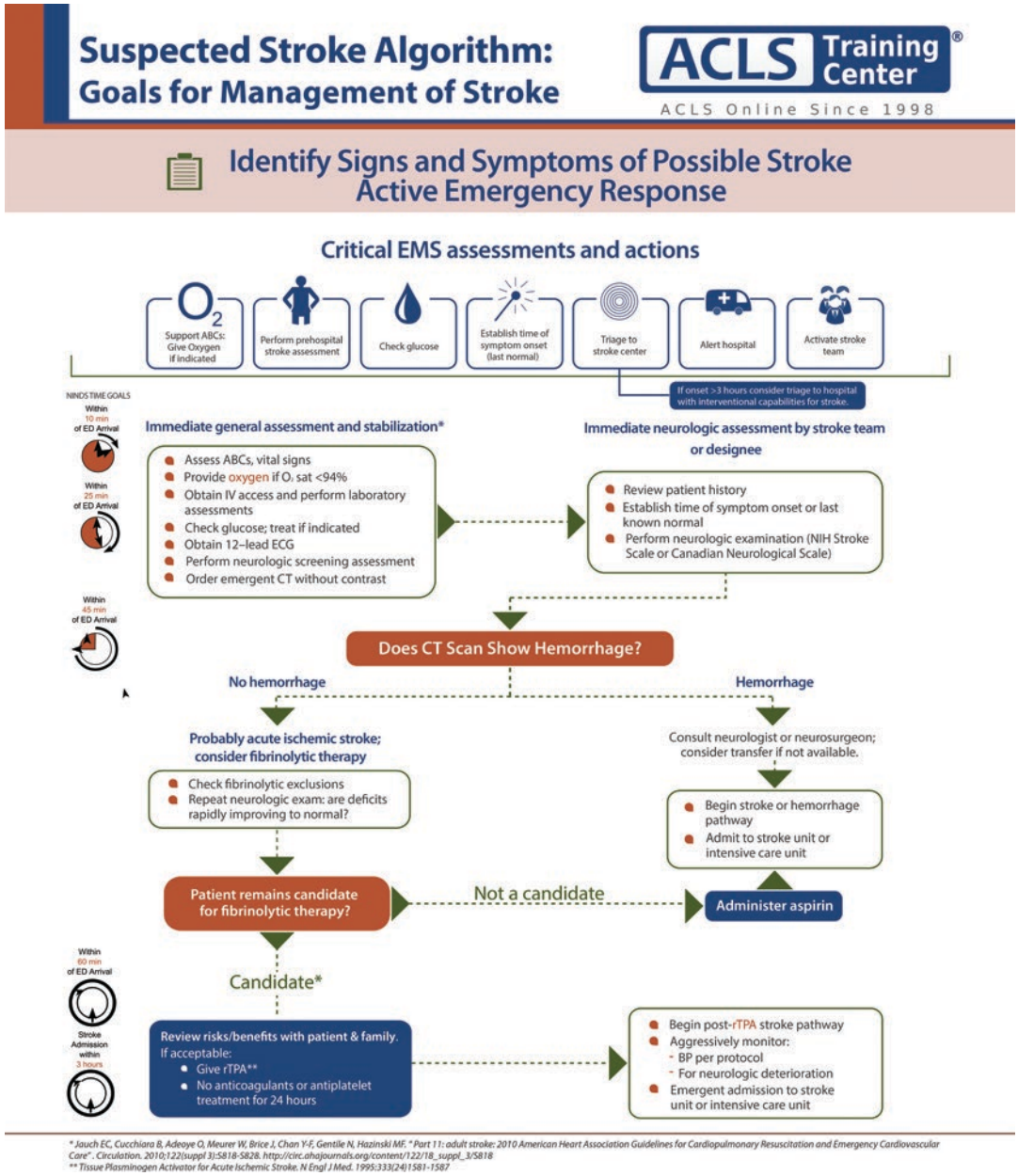


Fig. 25.9 Stroke treatment algorithm. (Copyright 2016 ACLS Training Center, https://www.acls.net)

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Medicine Case 1

26

Zain Manji, Damian Findlay, and Robert Reti

- *A 25-year-old female is referred to you for extraction of the third molars. The patient has the following medical history:*
  - PMHX – Asthma, Von Willebrand disease, ADHD
  - Meds – Advair, Ventolin, Singulair, Ritalin
  - Allergy – Seasonal
  - PSHx – Appendectomy
  - SocHx – Tobacco socially

- *What is asthma?*

  Asthma is a chronic reversible lower respiratory tract disease characterized by wheezing, dyspnea, coughing, and chest tightness.

- *How is asthma classified and what is the classification based on?*
  - Intermittent
  - Mild persistent
  - Moderate persistent
  - Severe persistent
- The asthma classification is based on the occurrence of symptomatology, interference with normal activity, and impact of obstruction (altered FEV_1).

- *What are some triggers for asthma attacks?*
  - Dust
  - Mold
  - URI
  - Pollen
  - Anxiety/stress
  - NSAIDs
  - Exercise

- *How do NSAIDs trigger asthma attacks?*

  Prostaglandins and leukotrienes are both liberated via the arachidonic acid pathway. NSAIDs are COX inhibitors, which shunt the pathway to lead toward leukotriene production. Leukotrienes are potent bronchoconstrictors. This is the rationale for leukotriene inhibitors in the management of asthma.

- *What are the mechanisms of action of Ventolin® and Advair®?*

  Ventolin (generic name albuterol) is a short acting Beta-2 selective agonist, which leads to smooth muscle relaxation and bronchodilation. Advair (generic for fluticasone and salmeterol) is a combination of a steroid and Beta-2 selective agonist. Fluticasone (steroid) works to decrease chronic inflammation. Salmeterol is a long acting Beta-2 selective

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agonist to dilate the bronchioles. Both are used for the chronic management of asthma.

- *What are some considerations for sedating asthmatics in the office setting?*
  - Consider administering albuterol preoperatively.
  - Preoperative auscultation to rule out active wheezing and rhonchi. Consider delaying surgery until resolved.
  - Consider using sedatives with bronchodilatory properties (ketamine and propofol).
  - Review medical history for recent attacks, hospitalizations, and upper respiratory tract infections.

- *What is ADHD?*

  Attention deficit hyperactivity disorder is a disease characterized by the inability to concentrate with bouts of excessive activity. The disease is believed to be related to decreased activity of dopamine and catecholamines in the prefrontal cortex, which governs attention, focus, executive planning, and organization.

- *What is the mechanism of action of Ritalin®?*

  Ritalin® (generic methylphenidate) prevents the reuptake of dopamine and catecholamines in the central nervous system.

- *What are some considerations for sedating patients with ADHD in the office setting?*
  - Patients on stimulants may be more combative (restless) during sedation procedures.
  - Stimulants increase sympathetic tone. Consider avoiding drugs that increase sympathetic tone (e.g., ketamine), which could act synergistically to increase the incidence of tachycardia, hypertension, and dysrhythmias.
  - Stimulants are known to be nauseating. Consider prophylactic antiemetic administration.

- *What is Von Willebrand Disease?*

  An autosomal dominant bleeding disorder that impacts coagulation by diminished platelet adhesion and/or factor 8 deficiency. Von Willebrand disease can be quantitative and/or qualitative.

- *What is Von Willebrand factor (VWF)?*

  A glycoprotein involved in platelet adhesion that also acts as a carrier for factor 8. VWF is made in the endothelium.

- *What lab tests are often abnormal in VWD?*
  - Low plasma VWF levels
  - Prolonged bleeding time
  - Abnormal ristocetin test
  - Elevated PTT

- *What are some considerations for treating patients with VWD in the office setting?*
  - Hematology consult to determine the subtype of VWD.
  - Laboratory tests (lists above).
  - DDAVP or Humate P (if low factor 8 levels) administration prior to surgery dependent on hematology recommendations.
  - Use of hemostatic agents intraoperatively such as gelfoam, surgicel, avitene, and lidocaine 2% with 1:50,000 epinephrine.
  - Suture extraction sites to aid in hemostasis.
  - Postoperatively consider tranexamic acid rinses.

- *Prior to inserting the intravenous catheter, the patient appears diaphoretic and begins breathing deeply and using accessory muscles with expiratory wheezing. What do you think is happening?*

  Patient appears to be having an acute asthma attack. I would maintain the patient on supplemental oxygen via nasal cannula. I would then administer albuterol via a metered dose inhaler (MDI). I would monitor O_2 saturation and for resolution of symptoms via inspection and auscultation.

- *After administering several rounds of the MDI, the patient continues to use accessory musculature. The oxygen saturation is 68%. What do you do?*

  I would contact EMS and administer intramuscular epinephrine.

- *How much epinephrine?*
  0.3 mg 1:1000

- *5 minutes after administration of intramuscular epinephrine, the patient does not appear to improve clinically as the saturation is 58% and the patient becomes cyanotic. Auscultation reveals diffuse wheezing throughout both lung fields. What do you do next?*

  I would administer an intubating dose of succinylcholine and place an appropriate-size endotracheal tube. I would administer albuterol via the endotracheal tube and ventilate the patient with 100% oxygen.

Medicine Case 2

27

Marc Dentico-Olin, Damian Findlay, and Robert Reti

- *A 60-year-old female presents to your office for consultation for extraction of her remaining mandibular teeth with delivery of an immediate complete denture. The patient is asymptomatic. The patient has the following medical history:*
  - PMHx – hypertension (HTN), hyperlipidemia (HL), Diabetes Mellitus (DM), coronary artery disease (CAD), myocardial infarction (MI), congestive heart failure (CHF) (on cardiac transplant list)
  - Meds – Metoprolol, Lisinopril, Lasix, Lipitor, Insulin, Brilinta, Digoxin
  - Allergies – NKDA
  - PSHx – Percutaneous coronary intervention (five stents), lipoma removal, left and right great toe amputation
  - SOChx – Former smoking 1 PPD for 10 years, social alcohol use, works as a cook

- *What is hypertension?*

  Persistent elevated arterial pressure of 130/80 mmHg. Will require two separate hypertensive readings (of at least 130/80 mmHg) separated by 2 minutes on two or more visits.

- *How is hypertension classified?*
  - Normotension <120/80 mmHg
  - Elevated 120–129/<80
  - Stage I 130–139/80–89
  - Stage II >140/90

- *How is end organ damage for hypertension assessed?*
  - Auscultation (assess for murmurs)
  - Ophthalmoscopy (assess for retinopathy, exudates, hemorrhages, papillary edema)
  - EKG (assess for ventricular hypertrophy and ischemia)
  - CBC (rule out polycythemia vera)
  - BMP (assess BUN/creatinine to rule out renal injury and potassium to assess for hyperaldosteronism and Cushing's disease).
  - Urinalysis to assess for proteinuria and GFR/creatinine clearance reduction
  - Sonography for arterial wall thickening
  - Ankle/brachial indices

- *What is the mechanism of action of lisinopril and metoprolol?*
  - Lisinopril is an ACE inhibitor, which prevents liberation of angiotensin 2, which is a potent vasoconstrictor.
  - Metoprolol is a beta blocker that decreases sympathetic tone to the myocardium, which decreases the heart rate and ultimately the blood pressure.

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- *Name some causes of secondary hypertension*
  - Hyperaldosteronism
  - Hyperthyroidism
  - Renal artery stenosis
  - Pheochromocytoma
  - Cushing's disease

- *What are some considerations for treating hypertensive patients in the OMS setting?*
  - Take preoperative BP readings.
  - Monitor BP throughout sedation procedures.
  - Monitor EKG for ischemic changes.
  - Be prepared for labile swings in blood pressure for patients taking ACE inhibitors, direct renin inhibitors, and ARBs.
  - Patients should take antihypertensives the morning of the procedure.
  - Avoid using medications that can elevate the blood pressure (e.g., ketamine).
  - Use local anesthesia with vasoconstrictor judiciously.

- *What is diabetes mellitus?*
  - A metabolic disease characterized by elevated blood sugar due to insufficient insulin production or insulin resistance.

- *How might untreated DM present?*
  - Polyuria
  - Polydipsia
  - Polyphagia
  - Signs of diabetes ketoacidosis

- *How is DM diagnosed?*
  - Fasting glucose of >126 mg/dl on two or more occasions.
  - Glucose tolerance test (readings greater than 200 mg/dl 2 hours after a 75 g glucose load).
  - HbA1c of 6.5 or greater.
  - Non-fasting plasma glucose ≥200 mg/dl and symptoms of DM.

- *Why are diabetics more susceptible to infections and poor wound healing?*

  Diabetics have decreased neutrophil chemotaxis and phagocytosis secondary to glycosylation of the neutrophils. They also have decreased humoral immunity and depression of the antioxidant system.

- *What are some precipitants of diabetic ketoacidosis?*
  - Poor glycemic control
  - Myocardial infarction
  - Infection (e.g., urinary tract infection)
  - Influenza
  - Stroke
  - Stress
  - Pancreatitis
  - Trauma
  - Alcohol and illegal substance abuse
  - Gastrointestinal disturbances (e.g., vomiting)

- *What are some long-term complications of DM?*
  - Nephropathy
  - Neuropathy
  - Retinopathy, cataracts, and glaucoma
  - Cardiovascular disease (increased risk of HTN, MI, peripheral vascular disease (PVD), and cerebral vascular accident (CVA))

- *Can you name a dreaded fungal infection with maxillofacial manifestations seen in poorly controlled diabetics?*

  Rhinocerebral mucormycosis.

- *What are some classes of drugs used to treat DM?*
  - Biguanides
  - Sulfonylureas
  - Insulin
  - Thiazolidinediones
  - Meglitinides
  - GLP1 agonists
  - DPP4 inhibitors

- *Why are diabetics typically on ACE inhibitors?*
  - ACE inhibitors are renoprotective in that they decrease intraglomerular pressure.

- *What is Kussmaul breathing?*

  Deep labored breathing often seen in diabetic ketoacidosis (DKA), which serves to blow

off hydrogen ions during exhalation (respiratory compensation for metabolic acidosis).

- *What are some considerations for managing hospitalized OMS patients with DM?*
  - Endocrinology/medicine consult to assist with management.
  - Diabetic diet.
  - Avoid corticosteroids if possible.
  - Serial glucose readings.
  - Serial CBCs in the infected patient to assess for resolution.
  - Comprehensive metabolic panel.
  - Urinalysis to assess for proteinuria and presence of ketones.
  - Prandial and postprandial insulin.
  - Sliding scale insulin. Patients may have increased blood glucose secondary to adrenal cortisol release. This is often due to pain and the traumatic insult of the surgery.
  - Tight glucose control in the critically ill patient (<110 mg/dl).
  - Patients may have atlanto-axial stiffness, which could make intubation difficult.
  - Consider discontinuing metformin for procedures where profound bleeding is expected (metformin can cause lactic acidosis).
  - Reduction of long-acting insulin prior to the surgery to prevent hypoglycemia in the setting of being NPO.

- *What are some considerations for treating diabetic patients in the OMS office setting?*
  - Early morning appointments.
  - Finger stick glucose levels prior and after procedure.
  - Have metabolic sugar sources in stock (orange juice, candy bars).
  - Consult with PCP to discuss altering insulin dosage prior to surgery.
  - Consider making the patient NPO for ~10 hours due to gastroparesis.
  - Consider giving a prophylactic antiemetic.
  - Avoid corticosteroids.
  - Ensure adequate hydration with intravenous fluids.
  - If glycemic control is not optimal, consider perioperative and postoperative antibiotics.
  - Imperative to assess EKG during sedation procedures to assess for signs of myocardial ischemia (diabetics may have silent ischemia due to autonomic neuropathy).
  - Be prepared to treat hypoglycemia (symptoms of hypoglycemia usually occur with a blood glucose of 70 mg/dl – confusion, fatigue, diaphoresis, nausea, seizures):

    Sugar source – candy, juice, glucose Tabs, D50, D5W, Glucagon

- *What preoperative blood glucose level warrants postponing surgery when treating the diabetic patient?*

  At a blood glucose value >250 mg/dl, an osmotic diuresis may occur which makes the patient susceptible to DKA and dehydration.

- *Is there a concern for using lactated ringers in diabetics?*

  Lactate can be converted to glucose, which increases the risk of hyperglycemia.

- *Why are diabetics at a higher risk of CAD/MI?*
  - Glycosylation of LDLs renders them more antigenic. A more profound inflammatory response renders the fibrous cap of atheromas more susceptible to rupture. Rupture of the fibrous cap exposes the lipid core, which leads to thrombosis.

- *Why are patients with CAD often on Beta Blockers?*
  - Beta Blockers decrease the heart rate, which decreases myocardial oxygen consumption.

- *What is the mechanism of action of Brilinta® (ticagrelor)?*
  - Brilinta® (generic name ticagrelor) is an ADP receptor antagonist that prevents platelet aggregation and coronary thrombosis.

- *Name other antiplatelet agents used to prevent coronary thrombosis.*
  - Aspirin
  - Clopidogrel

- Prasugrel
- Ticlopidine
- Abciximab

- *Why are patients with ischemic heart disease placed on ACE inhibitors?*

  ACE inhibitors decrease aberrant cardiac remodeling and decrease afterload.

- *What is congestive heart failure?*

  Failure of the heart to pump blood to meet the metabolic needs of the body.

- *What are the two types of CHF?*
  - Right sided heart failure – most common cause is left sided heart failure. Signs/symptoms include: JVD, hepatomegaly, pedal edema.
  - Left sided heart failure – signs/symptoms include: dyspnea, orthopnea, hemoptysis, paroxysmal nocturnal dyspnea.

- *How do you work up a patient with CHF prior to surgery?*
  - CBC – assess the oxygen carrying capacity of the patient.
  - BMP – electrolyte assessment.
  - EKG – look for signs of ischemia and ventricular hypertrophy.
  - Echocardiogram – assess wall movement, valvular function, and ejection fraction.
  - Cardiology consult to assist with management.
  - BNP levels.

- *You decide to treat the patient in the office with a mild sedation. Your preoperative vital signs are within normal limits and the EKG shows a normal sinus rhythm. You administer 5 mg of versed and 50 μg of fentanyl to achieve an adequate level of sedation. You achieve profound anesthesia with three carpules of lidocaine 2% with 1:100,000 epinephrine and one carpule of 0.5% Marcaine with 1:200,000 epinephrine. 3 minutes into surgery you notice that the patient has the following vital signs:*
  - *BP – 180/95*
  - *HR – 105*
  - *Saturation – 96%*
  - *EKG – NSR*

- *How do you want to proceed?*

  I would ensure that I have achieved profound anesthesia and that the resultant hypertension and tachycardia are not secondary to pain. I would also continue to monitor the EKG for ischemic changes.

- *You have determined that you have achieved profound anesthesia and the EKG appears normal. You take a new blood pressure 5 minutes later and the BP is 188/97 with a HR of 107. What do you do?*

  I would stop the procedure and administer 20 mg of labetalol.

- *What is the rationale for administering the labetalol?*

  Labetalol is an alpha- and beta-adrenergic agonist. This will result in a decrease in the heart rate and in peripheral vasodilation.

- *Why should you be cautious in using labetalol in this patient?*

  Labetalol can block the adrenergic response to hypoglycemia, which can make hypoglycemia difficult to identify.

- *Why is the management of tachycardia in this patient paramount?*

  Management is paramount because tachycardia increases the patient's myocardial oxygen demand. The patient has a history of ischemic heart disease and tachycardia puts them at risk for a perioperative ischemic event.

- *The blood pressure and tachycardia normalize and you complete your procedure. 20 minutes into postoperative monitoring, the patient complains of mild substernal chest pain and you notice the following EKG. What is concerning about the EKG? What do you do?* (Fig. 27.1)
  - This EKG shows ST depression which could be indicative of a myocardial infarc-

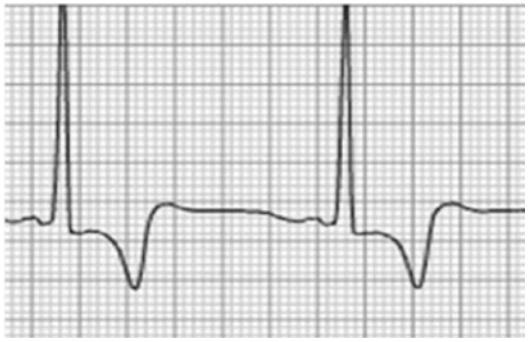

Fig. 27.1 ST depression in EKG. (Reprinted with permission from Cordeiro A, Moraes A, Cerutti V, et al. Clinical determinants and prognostic significance of the electrocardiographic stain pattern in chronic kidney disease patient. J Am Soc Hypertens. 2014;8(5):312–20)

tion or myocardial ischemia.
- I would contact EMS and administer 100% oxygen.
- I would administer 0.4 mg sublingual nitroglycerin (if SBP >90 mmHg), chewable aspirin (160–325 MG), and iv morphine (1–3 mg at 5-minute intervals).
- I would monitor vital signs for signs of decompensation (bradycardia, hypotension, ventricular dysrhythmias, and hypoxia).
- BLS as necessary.

- *If the patient did not complain of chest pain and the EKG appeared the same, would your management change?*

  No, the patient is at risk of having a silent myocardial infarction, as she is diabetic.

- *Patient was taken to the hospital and was treated successfully for an NSTEMI. What is the difference between an NSTEMI and STEMI anatomically?*

  The NSTEMI is an infarct that affects the subendocardial region whereas the STEMI is a transmural (full wall thickness) infarct.

- *The patient is informed, by her cardiothoracic surgeon, that they have found a suitable donor for her heart transplant. One year later, the patient is referred back to you for placement of implants in sites #22 and 27 to facilitate an implant overdenture. The patient is now 1 year s/p orthotopic heart transplant. Her new list of medications include tacrolimus, cyclosporine, mycophenolate mofetil. What are some new treatment considerations for this patient?*
  - Consultation with cardiologist.
  - Physical examination.

    The preoperative physical examination should focus on the patient's airway, heart, and lungs. Should look for evidence of volume overload suggesting ventricular failure (increased jugular venous pressure, S3 gallop, peripheral edema, hepatomegaly).

    The preoperative evaluation should also include a chest radiograph to look for pneumonia or other signs of cardiopulmonary disease.
  - Exercise tolerance and functional status are excellent preoperative screening tools.
  - Echocardiography needed to assess left ventricular function.
  - An ECG is needed to assess for dysrhythmias and rule out acute ischemia (useful in looking for evidence of old infarcts (Q waves) as the transplanted heart patient may not have angina symptoms).
  - Laboratory evaluation should include a complete blood count to rule out anemia, myelosuppression, leukocytosis, and thrombocytopenia. Blood urea nitrogen and creatinine can help evaluate renal function in the presence of probable hypertension.
  - Stress dose steroids should be considered in patients receiving corticosteroids as part of their immunosuppressive regimen.
  - Cyclosporine can enhance the effects of pentobarbital and fentanyl as well as prolong the action of vecuronium and pancuronium. Cyclosporine also causes gastric atony which is a risk for emesis and aspiration. Gingival hyperplasia may lead to fragility of gingivae which increases the risk of bleeding during airway manipulation.
  - Patients further removed from surgery have a higher risk of cardiac allograft vasculopathy, malignancy, diabetes, and renal insufficiency.

- Patient is at a higher risk of "silent ischemia" as the transplanted heart is denervated.
- Consider perioperative and postoperative antibiotics in the setting of anti-rejection medications that render the patient immunocompromised.
- Strong consideration should be done to treat these patients in the operating room setting due to the precarious nature of these patients.
- Catecholamine response differs in the transplanted heart requiring direct acting sympathomimetics. Dobutamine and isoprenaline are superior to dopamine as effective ionotropes. Atropine has no effect on the transplanted heart; therefore, in treating bradycardia and hypotension, isoprenaline and epinephrine should be readily available.

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