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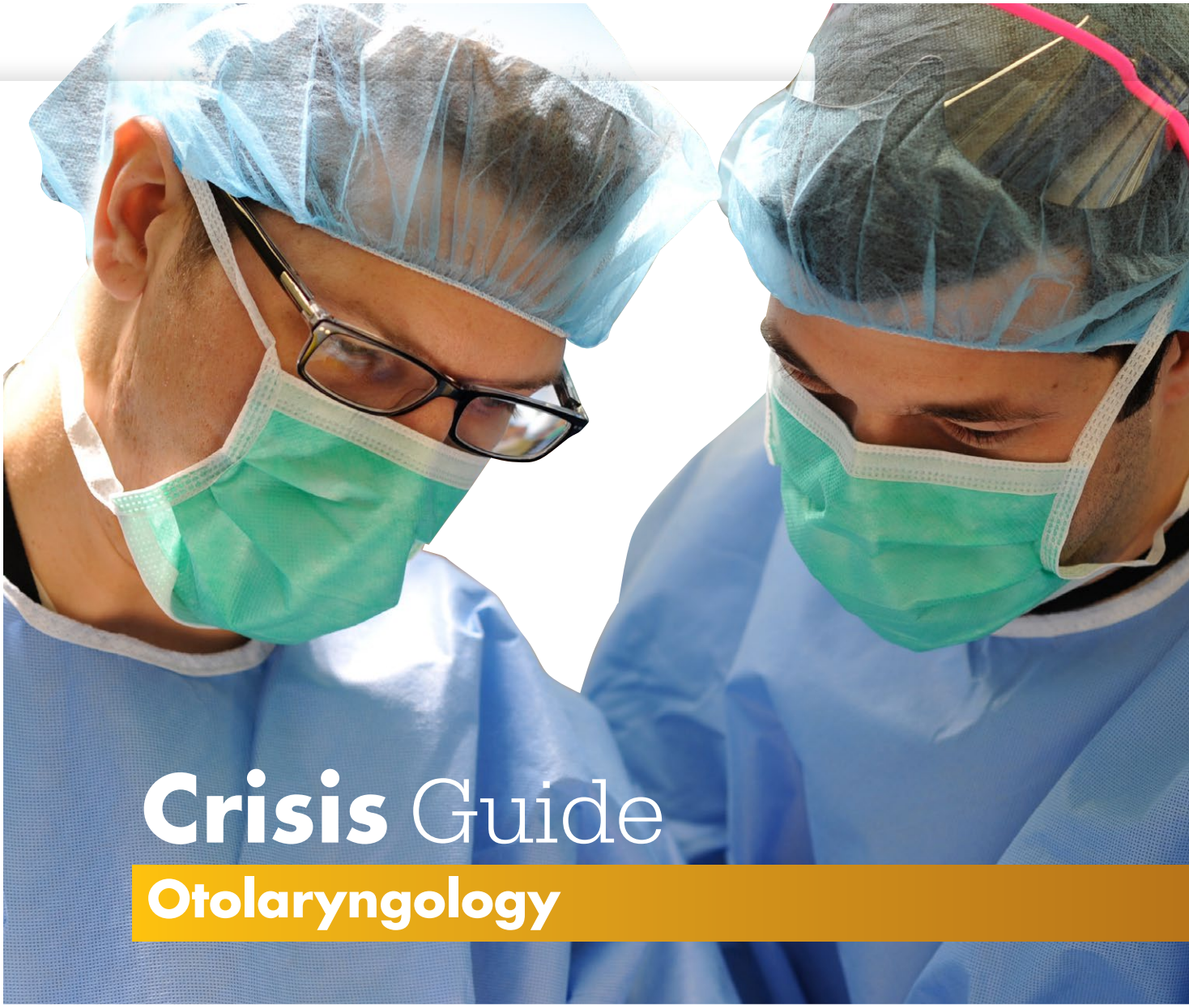

Crisis Guide

Otolaryngology

Editors:

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About
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This book is a collection of straighforward management strategies for common ENT urgencies. It was written and edited by the residents of the Emory University Department of Otolarygology – Head & Neck Surgery. This collection was originally created as a pocket reference for acute treatment of common ENT emergencies; it is not meant to be an all-inclusive text. The opinions and recommendations offered here are loosely collected from our training experience as well as the prevailing opnion in recent literature and major otolarygology texts.

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Introduction

Otolaryngology is an exciting field that usually allows for the practice of surgery and medicine in a controlled environment. However, like any medical field there are an abundanceof emergencies that require focused attention and rapid decision-making. In a crisis, the effective practitioner should be able to assimilate all available data and make clear decisions aimed at stabilizing an emergency.

Mission

Together with our customers
we are driven
to make healthcare better.

Common ENT Infections

Acute Otitis Media and Mastoiditis

Oswaldo Henriquez, M.D.

Clinical presentation of acute otitis media includes otalgia, fever, hearing loss, ear pulling and irritability, and upper respiratory symptoms. Upon otoscopic examination there can be appearance of thickening and erythema of the tympanic membrane, perforation with suppurattive otorrhea in the auditory canal, decreased visibility of middle ear landmarks, decreased tympanic mobility on pneumatic otoscopy, and presence of middle ear effusion. In most cases, clinical diagnosis should be sufficient alone. The three most commonly involved bacteria are Streptococcus pneumonia, Haemophilus influenzae, and Moraxella catarrhalis. In many cases Staphylococcus aureus and Pseudomonas aeurginosa may be identified.

Primary treatment of acute otitis media includes oral antibiotics to cover the above common pathogens as well as analgesia. Usually primary treatment is with amoxicillin or bactrim, or with amoxicillin + clavulante for persistent or recurrent infections. General recommendations are for a 10-14 day course of PO therapy for uncomplicated AOM. Recent large studies in the primary care literature advocate a “watch and wait” approach to treatment where antibiotics may be withheld for up to 3 days to allow for spontaneous resolution.

Complications of AOM are classified as intratemporal or intracranial. Intratemporal complications include coalescent mastoiditis, subperiosteal abscess, facial nerve paralysis, labyrinthine fistula, or abscess at the temporal root, petrous apex, or mastoid tip. Intracranial complications include meningitis, sigmoid thrombophlebitis, subdural empyema, epidural abscess, parenchymal abscess, and otic hydrocephalus.

Masoitditis is basically the spread of otitis media infectious process to the mastoid air cells. On physical exam there is presence of bulging erythematous tympanic membrance with erythema, tenderness, and edema over the mastoid area, and the classic protruding auricle. The work up includes a high-resolution CT scan of the temporal bone. On CT there should be presence of middle ear effusion with coalescent mastoid air cells or attenuation of the bony mastoid cortex. It is important to note that due to normal communication of the middle ear and mastoid cavity, any OM can demonstrate a fluid filled mastoid on imaging. MRI or contrast-enhanced CT can be helpful if there is concern for intracranial complications.

Treatment of mastoiditis can be medical or surgical depending on the severity of the infection. Usually these patients are admitted for antibiotic therapy, most commonly empiric vancomycin and ceftriaxone. Routine tympanostomy will aid in treatment and in diagnosis. Mastoidectomy is usually considered for failure of medical management or significant intracranial or intratemporal complications, based on clinical and radiologic findings. Often collaboration with neurosurgery or infectious disease specialties are necessary.

Necrotizing Otitis Externa

Ajani Nugent, M.D.

Necrotizing Otitis Externa (NOE, aka malignant otitis externa) is best viewed as being an osteomyelitis of the temporal bone and skull base. The populations most at risk are those with compromised immunity and diabetes.

Infections leading to NOE are most commonly caused by Pseudomanonas aeruginosa. It usually colonizes the EAC after exposure to water or minor trauma. Fungal NOE, especially Aspergillus fumingatus, can be seen in the HIV+ population.

Granulation tissue is usually found along the floor of the isthmus (bonycartilagenous junction). Spread to the skull base can occur via the fissures of Santorini and the tympanomastoid suture; the stylomastoid foramen and the jugular foramen are potentially affected.

Patients with NOE often present complaining of severe throbbing otalgia that is present for several weeks and is refractory to analgesics. There is commonly a prior history of benign otitis externa. Patients may also complain of headache, TMJ pain, trismus, and conductive hearing loss.

Visualization of the EAC and TM are necessary for diagnosis. Usually the EAC will require debridement. Granulation on the floor of the EAC at the bony-cartilaginous junction is nearly pathognomonic for NOE. Examination of cranial nerve function is also requisite for assessment of any intratemporal infection.

Workup should include WBC and ESR in addition to imaging of the temporal bone. Traditionally, Tc-99m nuclear scanning of the temporal bone is used establishing the diagnosis of osteomyelitis and serial Ga-67 imaging used to follow for resolution. However, in the acute setting, high-res CT scanning with contrast is often the most accessible and useful. Cultures taken from the EAC may be useful but should not delay treatment. Serum glucose and Hb1AC as well as an immunocompetency workup should be considered.

Topical fluoroquinolones and oral ciprofloxin are the most commonly prescribed antibiotic regimen. However the use of ototopicals in NOE is debated. Additionally frequent aural toilet (usually with microscopy) is required. Surgery is generally not required acutely, but should be considered for fluid collections or significant skull base necrosis.

The suggested treatment regimen involves Ciprofloxin 750mg BID for a course of 6-8 weeks. With intravenous administration reserved only for rare cases of recalcitrant disease, often due to resistant Psuedomaonas. Particular emphasis should also be placed on controlling hyperglycemia or any underlying immunosupression as well.

Complications of Acute Sinusitis

Praveen Duggal, M.D.

Sinusitis is the infection and inflammation of the paranasal sinus cavities. The usual process of events stems from an upper respiratory infection that causes increased mucosal secretions, edema, and blockage of sinus outflow tracts.

Common symptoms of acute sinusitis include: purulent nasal discharge, facial pain and pressure, headache, fever, cough, hyposmia, and otalgia. Causative organisms include Streptococcus pneumonia, Haemophilus influenza, and Moraxella catarrhalis. Most often bacterial sinusitis can be treated with antibiotics (eg. Fluorquinolones, amoxicillin+clavulonic acid, cephalosporins, or sulfa derivatives) for 10-14 days.

Complications of acute sinusitis can be classified as orbital, intracranial, or both.

Most infections can begin with facial erythema, periorbital edema, and tenderness. Later worrisome symptoms include proptosis, chemosis, vision change, ophthalmoplegia, and bilateral involvement.

Evaluation of orbital involvement includes a good history, evaluation of extraoccular movements, visual acuity, and imaging with CT or MRI. Imaging is indicated with orbital symptoms, failure of antibiotics, or evaluation of suspected subperiosteal abscess. MRI is more helpful if concern for intracranial complications.

Intracranial complications include meningitis, epidural abscess, subbural abscess, intracerebral abscess, frontal osteomyelitis (Pott's Puffy Tumor), and cavernous (or other venous) sinus thrombosis. Symptoms including altered mental status, cranial nerve palsies, nuchal rigidity, hearing loss, vomiting, and lethary should all be assessed. Medical management with IV antibiotics along with surgical intervention should be considered.

Orbital Complications of Sinusitis

	Common Findings	Treatment
Preseptal cellulitis	Swollen eyelids; extraocular muscles (EOM) intact; normal vision	Medical therapy (rarely, drainage of secondary abscess)
Orbital cellulitis	More diffuse orbital edema; ± impaired EOM; usually normal vision	Medical therapy ± sinus drainagea
Subperiosteal abscess	Proptosis; impaired EOM	Medical therapy; ± sinus drainage, ± abscess drainage
Orbital abscess	Severe exophthalmos, chemosis, complete ophthalmoplegia and visual impairment common	Medical therapy; sinus drainage, often; abscess drainage, usually
Cavernous sinus thrombophlebitis	Bilateral orbital pain, chemosis, proptosis, and ophthalmoplegia	Medical therapy; sinus drainage, often; ± anticoagulation

*Adapted from Chandler JR. The pathogenesis of orbital complications in acute sinusitis. Laryngoscope. 1970; 80:1414-1428

Invasive Fungal Sinusitis

Anita Sethna, M.D.

Acute invasive fungal sinusitis constitutes a surgical emergency due to its ability to progress rapidly. Different from allergic fungal sinusitis, this entity can carry with it a mortality rate of 50-80%. The fungi involved invade deep into the sinuses, nasal mucosa and soft tissues surrounding the sinuses such as the brain and orbits.

Fungal organisms are not uncommon in the nasal cavity. The reason people suffer from IFS is most commonly due to a compromised immune system. This can be from neutropenia secondary to hematologic malignancy, chemotherapy, or even diabetics with poor glucose control. The fungi that affect those with IFS are common to the environment: Rhizopus, Mucor, and Aspergillus.

Symptoms of this condition can be similar to bacterial sinusitis, such as fever or facial pain. In particular, while examining a patient suspected of IFS, make sure to assess cranial nerve function including gross vision, sensation over the face and extra-ocular movements. Inspect the hard palate and oral cavity in general.

On examination of the nose, look for necrotic, black tissue, mold spores, or any areas that appear paler than the surrounding tissues. The first areas to be involved are commonly the middle turbinates and septum. Classic teaching states the disease should be unilateral, but this is not always the case.

Radiologic imaging, CT of the sinuses without contrast, is helpful in visualizing areas beyond traditional bedside endoscopy, and can give hints as to where the disease may be originating. For example, changes in the retro-antral tissues showing obliteration or increased soft tissue replacing fat planes compared to the normal side, thickening of the nasal septal tissue, and a later finding of bony destruction.

Workup Essentials:

1. Check labs for neutropenia/BG levels (ANC<250=neutropenia, but don't exclude functional neutropenia).

2. Look at films for retro-antral fat thickening, bony destruction, unilateral disease, nasal mucosa abnormalities, soft tissue inflammation.

3. Scope patient: a straight scope distorts less—try to avoid decongestant because this may make tissues look pale falsely.

4. Do not random biopsy—but take a biopsy of anything suspicious for ischemia, necrotic black tissue or moldy appearing tissue. Call in pathology for frozen section.

5. Place specimen on a piece of gauze and into a sterile specimen cup (without formalin); place a few drops of saline to keep tissue moist and take to pathologist.

Acute Tonsillitis and Peritonsillar Abscess (PTA)

Elina Kari, M.D.

Patients in any age group can present with tonsillitis, where as the peritonsillar abscess is usually seen in patients 20-40 years old. The patient will usually complain of severe sore throat, odynophagia, dysphagia, drooling, malaise, referred otalgia, and fever. On physical exam there will be erythema and edema of anterior pillar, trismus, drooling, "hot potato voice", fluctuance, inferior displacement of tonsil, and deviation of uvula/soft palate to contralateral side. Tonsillitis will have inflammation of the actual tonsil with purulence but no edema of the anterior pillar.

Causative agents include anaerobic and aerobic organisms. Streptococcus species (especially Group A □-hemolytic Strep, Streptococcus pyogenes) are most common. Anaerobic species: Prevotella species and Peptostreptococcus species can also be seen.

The differential diagnosis includes peritonsillar cellulitis, infectious mononucleosis, retropharyngeal or parapharyngeal abscess, retromolar abscess, and neoplasia. Complications include: airway obstruction, ruptured abscess and aspiration, parapharyngeal abscess, glottic edema, poststreptococcal sequelae, and possible sepsis.

A CT scan is NOT needed for diagnosis of PTA in an adult who can open their mouth. CT scans will show a fluid collection medial to superior pharyngeal constrictor muscles. Consider if diagnosis of PTA is equivocal. The otolaryngologist needs the following equipment for successful treatment of a PTA: tongue blade, head light, yanker suction, 10 cc syringe, 1% lidocaine with epinephrine, hurricane spray, 18 gauge needle, #15 blade, hemostat, and culture swabs.

To treat, always remember your airway first. If there is obvious fluctuance, attempt drainage with #18 gauge needle aspiration. Spray Hurricane spray and infiltrate locally with 1% lidocaine + epinephrine. Aim for the superior pole of the tonsil or supratonsillar fossa. If you are not able to aspirate with needle, you may consider I+D with #15 blade followed with hemostat dissection to break up loculi. Take care to incise over an area that you have previously needled to ensure you are not near the carotid artery.

Patients should receive IV antibiotics and be discharged on a 7-10 day course of oral antibiotics. Tonsillitis patients may be discharged on clindamycin or augmentin. For a PTA the same antibiotics can be used. Steroids may be administered IV and a short course of steroids has been proven to help with trismus and pain upon discharge. Patients can be managed on an outpatient basis if patient does NOT show signs of toxicity, sepsis, airway compromise, or has significant co morbidities and is able to stay wellhydrated. Close follow-up within a few days is advised for patients with a true peritonsillar abscess. Patients with recurrent episodes are candidates for tonsillectomy performed 4-6 weeks after resolution of PTA and is usually recommended with a patient that has had 2 or more episodes of PTA. A Quincy tonsillectomy is a tonsillectomy performed during acute episode and is indicated for severe airway obstruction or failed incision and drainage.

Epiglottitis & Supraglottitis

Jodi D. Zuckerman, M.D.

Epiglottitis is an inflammation of the epiglottis, whereas supraglottitis is swelling of the surrounding structures including the aryepiglottic folds and arytenoid soft tissue. Epiglottitis is often seen in the pediatric patient, whereas supraglottitis is more commonly seen in the adult.

Causative organisms include: Haemophilus influenza, Klebsiella pneumonia, Candida albicans, Staphylococcus aureus, Neisseria meningitides, and varicella zoster. Caustic burns and trauma to the larynx can also trigger acute inflammation in the supraglottic structures.

Patients will present with drooling, dysphagia, respiratory distress, and possible stridor.

This is considered a surgical emergency and again the otolaryngologist must ensure a secure airway before further workup is pursued. Patients stable enough for laboratory and imaging may demonstrate a peripheral bandemia and “thumbprint sign” on lateral neck X-ray.

The key to management of this disorder is quick recognition (based on presenting signs and symptoms) and securing the airway early. Many of these patients can undergo awake fiberoptic intubation with the otolaryngologist on standby for an emergency tracheostomy. Classically however, great caution is required during any stimulation of the airway as this may trigger an acute decompensation.

After the airway is secured broad spectrum antibiotics are usually begun.

Retropharyngeal and Parapharyngeal Infections

Frederick Durden, M.D.

Retropharyngeal and parapharyngeal space abscesses can be common consults for the ENT physician. The parapharyngeal space is bounded by the parotid gland and mandible laterally, pharynx and pterygoid muscles medially, superiorly by the skull base and inferiorly by the hyoid bone. Spaces are then separated by the pre- and post- styloid compartments. The prestyloid space contains the maxillary artery, inferior alveolar, lingual and auriculotemporal nerves. The poststyloid space contains cranial nerve IX, X, XI, sympathetic chain, internal jugular vein and carotid artery.

Infections of the parotid and odontogenic sources can spread to this space. Any virus or upper respiratory infection that has significant lymphadenopathy can futher develop into a parapharyngeal space infection. Complex peritonsillar abscesses can also further develop into parapharyngeal space infections.

The retropharyngeal space is bounded by the pharyngeal constrictors anteriorly, alar fascia posteriorly, skull base superiorly, and mediastinum inferiorly. It contains lymphatic vessels draining from infections in the nose, sinus, nasopharynx, and oropharynx.

The organisms responsible for this infection in adults include: Streptococcus viridans, S. aureus, Klebsiella pneumoniae,, Bacteroides species, Staphylococcus epidermidis, Anaerobic Streptococci, E. coli, Prevotela species, and Mycobacterium tuberculosis. In children, common pathogens include: S. aureus, Haemophilus, B-hemolytic streptococcus, Bacteroides species, Peptostreptococcus species, Fusobacterium species, Prevotella species, and coagulase-negative Staphlococcus. There has recently been an emergence of Methicillin Resistant Staph Aureus and when this is the causative agent first line treatment usually includes clindamycin or sulfamethoxazole/trimethoprim pending sensativities.

Patients will present with fever, odynophagia, dysphagia, neck pain, poor oral intake, lethargy and irritability. Large or advanced abscesses may impinge on the airway causing respiratory distress and stridor. The physician should examine the oropharynx and look for oral pharyngeal bulge, or evidence of a source of infection such as a peritonsillar abscess or odontogenic abscess. Palpation of the neck may reveal cervical lymphadenopathy. A flexibile laryngoscopy is helpful to determine effect on the airway and if emergent management is warranted.

Treatment usually consists of airway stabilization, re-hydration, and broad spectrum antibiotics covering gram positive and anaerobic organisms. Mycobacterium tuberculosis can also be the causative agent for these abscesses and the patient should be placed on TB precautions and treated appropriately.

If patients are not responsive to antibiotic treatment or there is a surgically drainable abscess then incision and drainage may be necessary. Parapharyngeal abscesses may be drained through the mouth with direct laryngoscopy. Evaluation of the great vessel position is imperative prior to this approach. Most retropharyngeal and parapharyngeal abscesses will be readily palpable in the neck and a simple incision and drainage with drain placement is appropriate treatment.

Ludwig's Angina

Eric Berg M.D.

Ludwig's angina typically presents with rapid onset swelling of the floor of mouth with elevation of the oral tongue, drooling, and possible airway obstruction. It is the result of a rapidly spreading bilateral bacterial cellulitis of the sublingual, submental, and submandibular spaces. This infection may spread through the buccopharyngeal gap (space created by the styloglossus muscle as it passes between the middle and superior pharyngeal constrictors) causing adjacent retropharyngeal and eventual mediastinal infection.

Risk factors include poor dentition, tooth extraction, floor of mouth trauma, peritonsillar abscess, and intravenous drug abuse. In fact, ninety percent of cases are odontogenic in origin with seventy-five percent originating from the second and third manibular molars. Mortality rates approach 10% and are primarily due to airway compromise.

Evaluation of suspected Ludwig's angina begins with a thorough physical exam. Initial attention should be directed towards respiratory status and any need for airway intervention. The exam should then be focused on the oral cavity, oropharynx, and neck. Visualization of the oral cavity allows for assessment of oral airway patency. Teeth should be inspected for signs of infection. Most importantly, the floor of mouth should be palpated. In Ludwig's angina, the floor of mouth takes on a firm, woody consistency and pushes the tongue caudally. The displacement can extend to the base of tongue and lead to further airway compromise. Fiberoptic laryngoscopy should be utilized to better assess the patency of the oropharynx and hypopharynx.

Radiographic evaluation consists primarily of computed tomorgraphy of the neck with contrast. Findings range from diffuse inflammation and phlegmon to frank abscess of the sublingual, submental, and submandibular spaces. Plain radiographs can be used to assess degree of soft tissue swelling and caliber of the airway.

First and foremost, management should focus on the airway. Due to its rapid progression, a once stable airway may quickly become compromised, and one may consider securing the airway prior to this event. Frequent airway reassessment should be performed to avoid sudden loss of the airway. Oxygen should be provided as necessary. Should significant distress exist, the airway should be first secured with an oral or nasal airway, intubation with or without fiberoptic guidance, or tracheostomy if necessary. Heliox may also be considered. During airway evaluation, broad-spectrum antibiotics should be administered to cover oral flora. Incision and drainage and tooth extraction if an infectious focus is apparent may also be necessary.

Deep Neck Space Infections

Elina Kari, M.D.

Patients with upper respiratory infection, trauma, IV drug abuse, tooth infections, oral infections, and immunosuppresion are all at risk for deep neck space infections. Symptoms include sore throat, hoarseness, odynophagia, dysphagia, neck pain, stiff neck, fevers, and chills.

Signs include, neck asymmetry, mass, edema, erythema, crepitation, tenderness to palpation, trismus, fever, cranial nerve deficits, tachypnea, stridor, "hot potato" voice, tenderness over SCM, nuchal rigidity, contralateral torticollis, ipsilateral Horner's, vocal cord paralysis. Exam should always include flexible fiberoptic laryngoscopy in any question of possible airway compromise.

Complications of a deep neck space infection include upper airway obstruction, descending mediastinitis, pleural empyema, necrotizing fasciitis of the head and neck, pericarditis, jugular vein thrombosis (Lemierre's syndrome), septic shock, carotid pseudoaneurysm or rupture.

The work up usually includes obtaining a CT scan with IV contrast to define the presence of an abscess with ring enhancing elements. MRI may be used for soft tissue and nerve involvement. Laboratory work up includes: CBC with differential, blood chemistries, blood cultures, coagulation and clotting profiles, and abscess cultures.
To treat the abscess first and foremost, remember the airway. If the patient has signs of respiratory distress, manage their airway first. Some patients with severe deep neck infections may require an awake tracheostomy. Volume and metabolic resuscitation, often requires ICU admission. Management of deep neck infection varies depending on its location, extent, and severity. Initial management with IV antibiotics is appropriate for small fluid collections in patients with no airway compromise.

IV antibiotics should include coverage of gram positive bacteria, anaerobes, and possible gram negatives. MRSA infection should be suspected and treatment should include coverage against such resistant organisms. Surgery is reserved for patients who present with significant disease, airway compromise, necrotizing fascitis, or for those who do not improve after 48h of IV antibiotics. Keys to surgical treatment are wide exposure, multiple approaches and, depending on the location, blunt dissection for drainage of the abscess cavity. Necrotizing fascitis requires frequent debridement.

Relevant anatomy

- Visceral Space: (infrahyoid neck) space defined by the visceral layer of middle layer of deep cervical fascia. Space includes thyroid gland, trachea, esophagus.
- "Danger" Space: Between alar and prevertebral fascia. Extends from skull base to diaphragm. Contains sympathetic trunk and loose areolar tissue and allows rapid spread of infection.
- Prevertebral Space: Deep to prevertebral fascia, contains spinal cord and extends from skull base to coccyx.
- Carotid sheath space: formed by other fascial layers, extends from skull base to thoracic inlet. Contains carotid artery, jugular vein, vagus nerve.

Nasal Septal Hematoma

Avani Ingley, M.D.

Septal hematoma often occurs as a result of nasal trauma. Bleeding occurs due to bleeding, often bilateral, within the subperichondiral plane of the septum. Elevation of the mucosa off the cartilaginous septum leads to a loss of septal vascular supply. If the hematoma is not drained, fibrosis of the cartilage may occur, followed by necrosis. Without treatment, in 3-4 days septal perforation will occur. The resultant loss of septal support will lead to collapse of the nose and a saddle nose deformity. A septal abscess may also develop if the collect clot becomes infected.

Treatment of a septal hematoma or abscess involves urgent drainage, which can be performed in the clinic or emergency department. An incision should be made in the caudal septal mucosa and the hematoma evacuated. Consider placing a small nasal tampon or splint to prevent reaccumulation of blood. The patient should be seen in followup to observe healing and to remove any packing; antibiotics are a must when packing or splints are placed.

Bilateral septal hematoma.
Notice bulging septum, often causing nasal airway obstruction. Dashed line marks incision for drainage on the left.

Auricular Hematoma

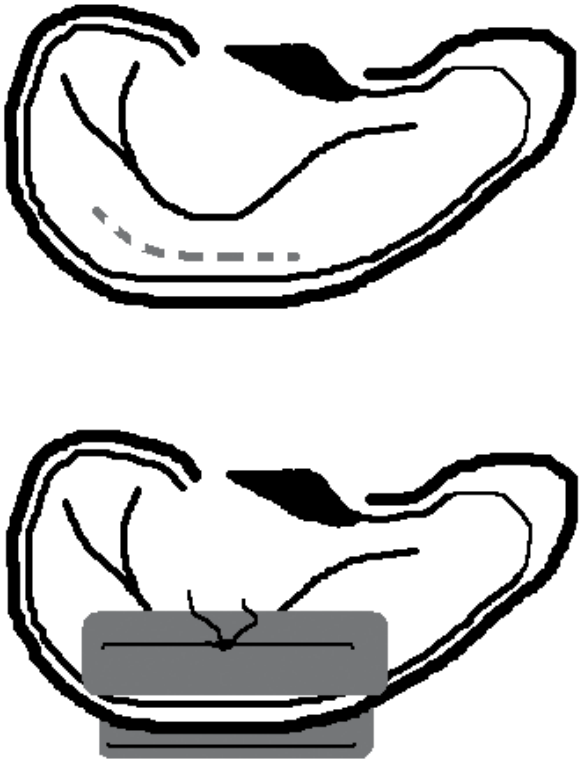
Adrienne Laury, M.D.

Auricular hematoma is a relatively common complication of direct trauma to the ear or side of the head. It is most commonly seen in wrestlers and is diagnosed by history and the loss of typical auricular landmarks on physical exam. The hematoma forms following a direct trauma as a result of the tearing of perichondrial blood vessles. These sheared vessels then result in a blood extravasation which separates the perichondrium from the anterior auricular cartilage. If not repaired, multiple complications can arise including infection, cartilaginous necrosis, and the most infamous – auricular chondropathy, aka “cauliflower ear”.

Drainage of an auricular hematoma is fairly straightforward and should be done in a timely fashion in order to prevent complications. The only contraindications to drainage include: hematoma presence >7 days or recurrent/chronic hematomas. Begin drainage by placing the patient in a lateral decubitus position on unaffected side. Cleanse area with antiseptic and anesthetize ear with lidocaine without epinephrine. Next, make a small, superficial incision approximately 5-10mm on the anterior portion of the ear along a natural skin fold such as the helical rim. Separate the skinand perichondrium from the hematoma and cartilage and compress or suction out the blood. Irrigate with saline. If possible, reapproximate the skin and perichondrium with absorbable sutures.

A compression dressing must be placed over the ear; this is essential to prevent reaccumulation of the hematoma. A simple dressing of bulky vaseline gauze on both sides of the helix, secured in place with a matressed heavy monofilament suture is most common and simplest. A circumfrential dressign may also be applied.

Oral antibiotics to cover for skin flora may be considered while the dressing is in place. Reevaluate and remove the compression dressing in 2-5 days. It is also important to remind the patient to avoid aspirin or NSAIDs as they may promote continued bleeding and reaccumulation of the hematoma.



I&D of Auricular Hematoma (top image, dashed line indicates possible incision site) followed by placement of compression dressing (bottom image, gray rectaongles represent gauze rolls on either side of auricule secured by heavy suture.

Mandibular Fractures

Eric Berg, M.D.

Fractures of the mandible are the second most common facial fracture behind only nasal fractures. Because of the mobility of the mandible and intimate involvement with the oral cavity, they have a relatively high complication and infection rate. The most common points of fracture are near the third molar, the mental foramen, and at the condylar neck. In addition, bilateral articulation at the temperomandibular joint makes bilateral fractures common. Fractures are described as favorable versus unfavorable based upon the muscular forces which serve to either reduce or displace fracture fragments.

Evaluation of a suspected mandibular fracture consists initially of a thorough examination of the oral cavity and surrounding soft tissue. Note should be made of signs of external trauma. Patients should be assessed for trismus and malocclusion. Anesthesia or parasthesias may be present if the fracture involves the inferior alveolar nerve. Examination of the oral mucosa may demonstrate gingival lacerations or a floor of mouth hematoma.

Radiographic evaluation consists primarily of computed tomography. It is more sensitive in the diagnosis of mandible fractures than panoramic views however it may not show bony detail of alveolar ridge and tooth fractures as well.

Most favorable fractures can be managed by closed reduction with maxillary mandibular fixation alone. Four to six weeks is a sufficient duration for symphysis, angle, and body fractures, however condylar fractures may benefit from a shorter duration to preserve post-fixation range of motion.

In spite of the simpler surgical approach of closed reduction, maxillary mandibular fixation is very uncomfortable for patients, requiring a liquid diet which may result in significant weight loss. Consideration of open reduction and internal fixation with plates alone may be appropriate for symphyseal, parasymphyseal, angle, and body fractures. Ramus and condyle fractures are often treated with closed reduction alone, as internal fixation is very difficult to achieve and often requires an open or endoscopic approach. Special consideration must be made in edentulous patients as arch bars are not an option. Further, children are more often treated with closed reduction so as to avoid growth disturbance.

Complications of fracture and management include infection, malocclusion, malunion, nonunion, TMJ ankylosis, TMJ dysfunction, trismus, tooth loss, facial or inferior alveolar nerve damage, and poor cosmesis. To minimize infectious risk, all open fractures should be treated with perioperative antibiotics.
Surgical management need not be emergent provided the patient is able to tolerate an oral diet and is often completed in an outpatient setting.

Nevertheless, emergent management must be considered in cases of airway obstruction, displacement of the condyle into the middle cranial fossa, or hemorrhage.

Orbital Floor Fractures

Casey Mathison, M.D.

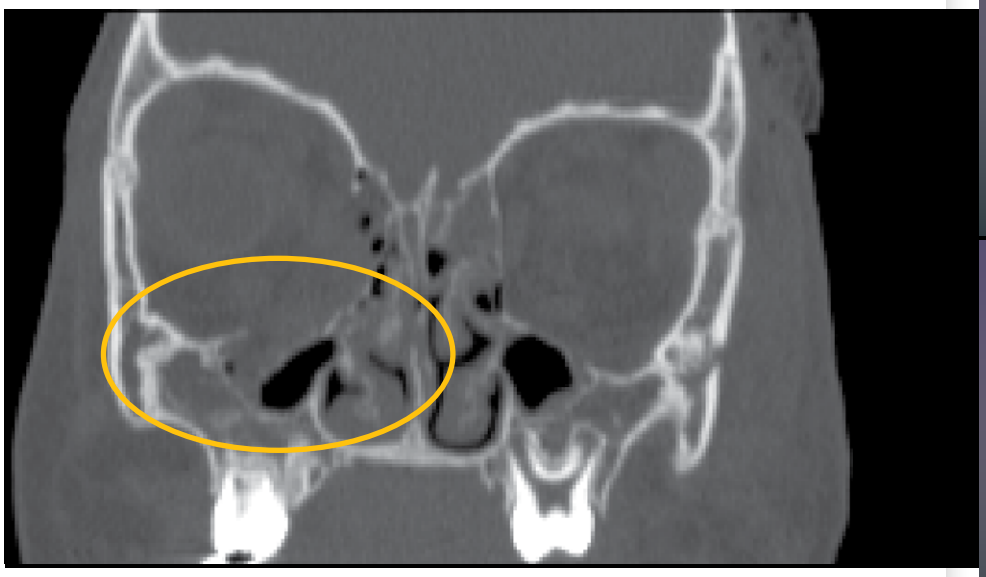
Orbital floor fractures are a common result of facial trauma. They are usually the result of blunt trauma to the face, such as an interpersonal altercation or a motor vehicle collision. Presenting signs and symptoms can vary, and timing of surgical repair (if needed at all) depends on the physical exam and radiographical findings.

Patients usually report being struck by a fist or a ball, or being in a car accident. Typical complaints include facial swelling or pain, blurry vision, diplopia (especially on up/down gaze), or numbness to the ipsilateral cheek.

The eye exam is the most important--you want to make sure that the globe has not been injured. If any abnormalities are found, ophthalmology should be consulted. The patient should be assessed for stable vitals signs, extraocular movements (may need to do forced duction), gross visual acuity, papillary reactivity, enophthalmos or exophthalmos, hypoesthesia to V2 distribution (cheek, teeth), gross facial asymmetry and depressions, and subcutaneous emphysema. You will almost always see facial swelling and ecchymosis.

The work up includes a CT scan of the face with coronal reformats. Entrapment of soft tissue, inferior rectus muscle, and fat should be evaluated on CT scan. Other facial fractures (ZMC, mandible, frontal/maxillary sinus, temporal bone) should be ruled out as well. Lastly, retro-orbital hematomas should be followed with serial eye exams.

Management of these fractures is almost always as an outpatient. Timing of operative repair depends on finding physical exam and CT findings. Indications for immediate surgical repair include: entrapment of extraocular muscles and non resolving oculocardiac reflex (bradycardia, heart block, nausea, vomiting) caused by entrapped tissue. Repair can wait 7 to 14 days when there are no immediate indications for repair and the swelling and ecchymosis can resolve. If after swelling subsides repair may still be pursued for cosmetic deformity and symptomatic diploplia. Patients can be observed if there are no visual disturbances and less than 50% floor involvement. Greater than 50% of the orbital floor involved with fracture has a high risk for late enophthalmos if left untreated.



Frontal Bone Fractures

Lindsey Arviso, M.D.

The frontal bone borders several important structures, most importantly, the brain. The supraorbital rim and roof of the frontal bone are the superior boundary of the orbit with the supraorbital and supratrochlear nerves nearby. The frontal bone gives contour to the forehead and encloses the frontal sinuses completely. Fracture of the anterior wall of the sinus, therefore, may result in deficits in sinus function or cosmesis. Posterior sinus wall fractures are more dangerous and may extend into skull base fractures requiring neurosurgical intervention.

When consulted for a frontal bone fracture, evaluate the forehead for motor and sensory function. The exit and path of the supraorbital and supratrochlear nerves could be involved in the defect. Visable or palpable defects or step-offs should be noted. Lacerations over non-operative fractures may be closed under sterile conditions in the emergency room.

A high-resolution axial CT should be performed to evaluate bony defects. Anterior wall fractures may be present that were not found on physical exam. Posterior table fractures with any degree of displacement are significant. Fractures may be described as linear horizontal, linear vertical, comminuted, and with or without nasal, ethmoid, and lacrimal bone (NOE) involvement.

The bones that provide protection to the orbital contents and anterior fossa are the thicker bones of the supraorbital rims and frontal bones surrounding the frontal sinus. In all, for repair of the frontal sinus, alignment of the bones is all that is really necessary for good healing and outcomes. The most important topics in regards to frontal fractures are the need for exploration or obliteration.

The anterior table is repaired for aesthetic purposes and to correct sinus outflow tract disruption. Obstruction of the nasofrontal ducts will lead to chronic sinus

infections. Depressed anterior wall fractures may also lead to sinus complications and should be explored to evaluate mucosal damage, however repair may be delayed for a period of conservative observation.

Posterior wall fractures are more serious in nature. A nondisplaced posterior table fracture can be observed if the anterior table does not require repair. With a displaced fracture of the posterior wall, one must consider the involvement of the dura and the brain. Dura and brain contents may have herniated into the frontal sinus if the defect is large enough and on the reverse, a frontal sinus mucocele could encroach and compress the brain. Obliteration eliminates the frontal sinus. As long as there is no ductal involvement, trephination or endoscopy are appropriate to exclude herniation of brain into the sinus. If a displaced posterior wall fracture is present with no soft tissue defect, sinus obliteration is performed at some centers.

Outpatient follow up with interval imaging to observe if sinus disease follows may also be appropriate. Delayed obliteration can be performed if chronic sinus disease then develops. Comminuted posterior frontal sinus fractures with intact anterior walls should be obliterated by cranialization to remove the posterior wall of the sinus and mucosa. This procedure prevents herniation or mucocele, however, the necessity of this is debated.

CSF leaks from defects in the skull base may occur from traumatic frontal fractures. These must be identified if possible due to the risk of meningitis. Skull base defects may be repaired at the time of fracture repair. Skull base disruption should be addressed early due to its many risks of complications. A bifrontal craniotomy or transglabellar subcranial approach with collaboration from neurosurgery allows direct visualization of the anterior fossa for repair.

Temporal Bone Fractures

Grace Leu, M.D. and Aaron Rogers, M.D.

Lateral skull base fractures are common with serious closed head injury. These uncommonly present a true correctible emergency but nonetheless the input of the otolaryngologist is important.

Clinical findings may include Battle sign, hemotympanum, TM perforation, hearing loss, or facial paralysis. CSF otorrhea is also fairly common.

Classically fractures are described as either longitudinal or transverse, relative to the axis of the petrous ridge; fractures are commonly a combination of the two types or even comminuted.

Longitudinal fractures are more likely to cause tympanic membrane perforation and conductive hearing loss. These are the most common type of fracture.

Transverse fractures are more likely to injure the facial nerve or cochlea. These are much less common than longitudinal fractures.
The vast majority of temporal bone traumatic CSF leaks will resolve spontaneously with bed rest and head elevation. Many clinicians will empirically place the patient on prophylactic antibiotics for several days as well.

Treatment of the traumatized facial nerve is a controversial issue. Operative decompression of a paralyzed nerve may be considered, especially in cases of late-onset paralysis or obvious physical disruption on imaging. Serial seventh nerve conduction studies are classically described but often may not alter patient management. Systemic steroid is often advocated.

Concerns of hearing loss are usually dealt with in the proceeding weeks after injury. Typically, patients are assessed with an audiogram after any hemotympanum has resolved (and after more acute issues are controlled). Surgical exploration with tympanoplasty or ossicular chain reconstruction may be warranted for persistent conductive hearing loss.

Traumatic TM perforations are usually treated with ototopical antibiotics to prevent infection (and promote spontaneous closure).

For significant ear canal lacerations an expanding sponge wick (Merocel or "Pope" pack) may be placed for several days to prevent scar contracture.

Injury to the carotid canal (and artery) must be ruled out with angiography if suspected on initial imaging.

Blunt Laryngeal Trauma

Aaron Rogers, M.D.

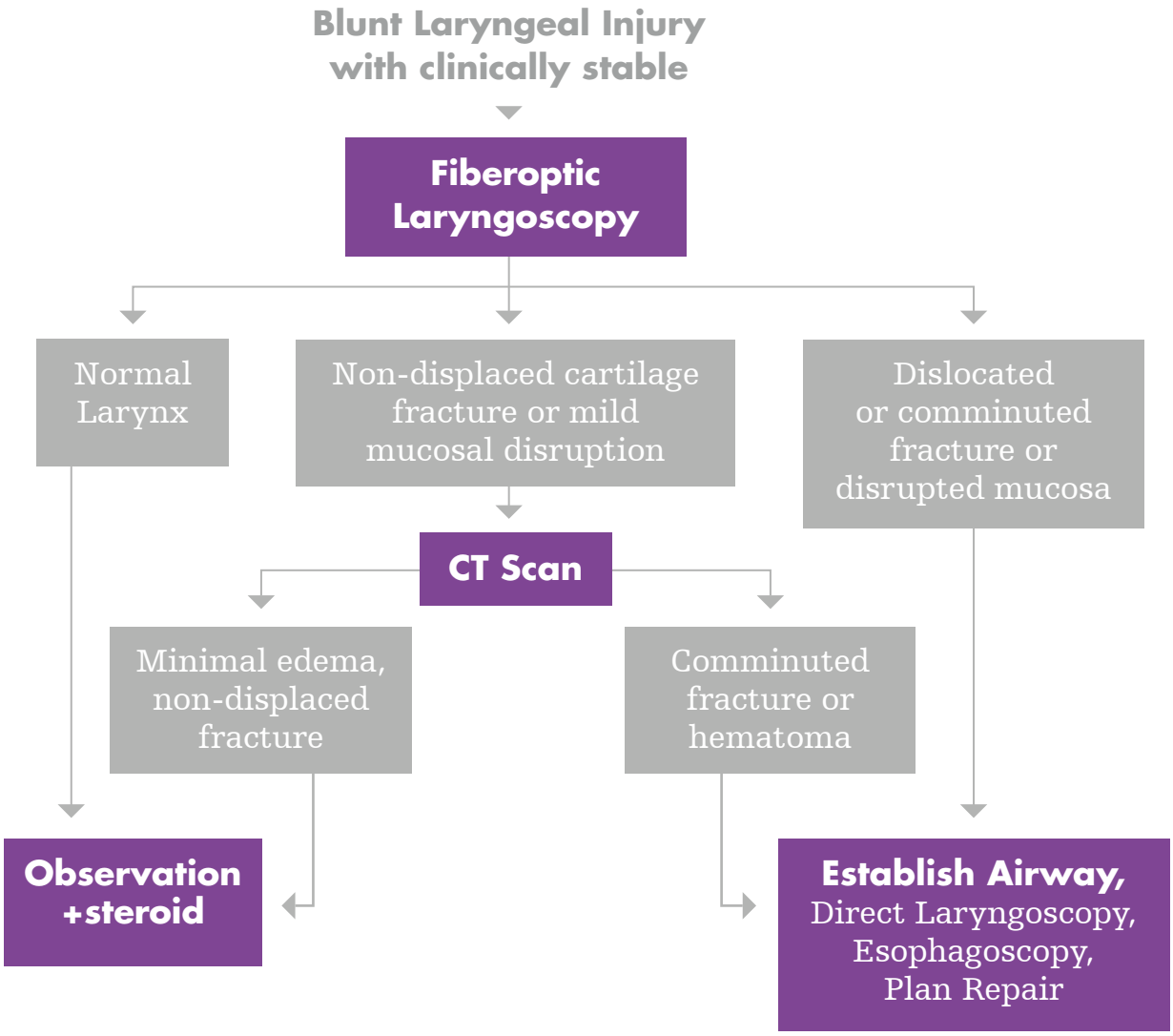
Trauma to the anterior neck can pose significant and rapid respiratory compromise. For this reason, the otolaryngologist is often consulted to evaluate the stability and patency of the upper airway. Intervention may include an urgent oral intubation or trachestomy, in addition to possible open repair of the cartilaginous larynx and mucosa.

The biggest challenge in early management is appropriately stratifying the patients who need a secured airway from those who are safe for observation alone. These are most often patients who have moderate blunt trauma to the Zone II neck with only minimal voice or respiratory disturbance.

Blunt laryngeal injuries can result in fracture of the laryngeal cartilages with or without dislocation, submucosal or external laryngeal hematoma, mucosal shearing leading to neck emphysema, upper airway bleeding, or even frank laryngo-tracheal separation.

The physical exam should start with an assessment of respiratory distress and voice disturbance. Palpation of the neck for crepitus (subcutaneous emphysema) or gross cartilaginous disruption is a must. Some authors argue to be wary of bedside canalization of open wounds as this could worsen bleeding and pose risk for laryngeal hematoma. Plain-film X-rays may reveal subcutaneous or mediastinal emphysema. If CT scanning has been done it is often very helpful, but probably should not be done until the airway has been evaluated. In general, fiberoptic laryngoscopy should be your first diagnostic tool (i.e. take it with you to the ED).

Smaller soft tissue injuries and nondisplaced fractures with a stable intact endolarynx can be managed with conservative measures. Patients should be admitted for 24 hr observation with prophylactic antibiotics and systemic corticosteroids. Larger injuries, disruption of the laryngeal framework, or dislocated arytenoids may require repair with rigid or wire fixation in an acute setting once an airway is established. Small injuries may be approached endoscopically.



Penetrating Neck Injury

Shatul Parikh, M.D and Jodi Zuckerman, M.D.

To determine the appropriate management of penetrating trauma to the neck, it is very important to consider the anatomic location of the injury. Initial management should consist of basic airway, breathing, and circulation steps of trauma assessment. The airway may be secured with intubation or a surgical airway. Bleeding should be controlled with pressure and fluid resuscitation. Cervical spine injury should be assumed and always use C-spine precautions. Obtaining a brief and accurate history of the mechanism of trauma can be helpful to provide clues as to where the injury may occur.

In the exam, one must look for signs of neurologic trauma and vascular injury. Shock, expanding hematoma, evolving stroke, hemoptysis, hematemesis, profuse bleeding, unequal pulses, and bruits can all be signs of vascular injury. Subcutaneous emphysema, stridor, hoarseness, or respiratory distress indicate tracheal or laryngeal injury. Neck pain, odynophagia, and fever can be signs of early injury to the esophagus. All penetrating injuries should be managed with physical exam, airway stabilization, and management of circulation. Obviously great vessel injury usually mandates early operative exploration.

Zone 1 of the neck: Superiorly bounded by cricoid and inferiorly by sternum and clavicle. Within this zone lies the subclavian artery and vein, dome of the pleura, esophagus, great vessels of the neck, recurrent nerve and trachea. In this zone there may be concomitant thoracic injuries including pneumothorax, hemopneumothorax, and tension pneumothorax.

In the patient with suspected Zone 1 injury, angiography is indicated to evaluate the vasculature. Early neck exploration may be considered. Evaluation with esophagoscopy, contrast swallow exam, and bronchoscopy may also be considered depending on the extent of injury.

Zone 2 of the neck: Superiorly bounded by the angle and inferiorly by the cricoid. Within this zone lies the larynx, pharynx, base of tongue, carotid artery, jugular vein, phrenic nerve, vagus nerve, and hypoglossal nerves.

In this zone the symptomatic patient should be taken to the OR for neck exploration. In an asymptomatic patient with penetration of the platysma, due to the relative ease of exposure and the risk to the carotid artery and jugular vein, neck exploration is usually advocated prior to other diagnostic entities.

Zone 3 of the neck: Everything between the angle of the mandible to skull base. Contains the internal and external carotid arteries, vertebral artery, and cranial nerves I-VIII.

Angiography is initially performed to assess for great vessel injury. If exploration is indicated the assistance of a neurosurgeon or vascular surgeon may be helpful. One may also consider percutaneous intralumenal procedures to control hemorrhage or psuedoaneurysm in this risky area. Threading a balloon catheter through the high neck wound to inflate and control hemorrhage is also reported.

Operative complications

Post -Tonsillectomy Hemorrhage

Clyde Mathison, M.D.

Bleeding after tonsillectomy usually occurs either during the first 24 hours (primary) or greater than 24 hours after surgery (delayed). Delayed hemorrhage is the most common. Primary post-tonsillectomy hemorrhages are due to surgical technique, whereas delayed hemorrhages relate to postoperative factors. Generally, a delayed hemorrhage is attributed to the sloughing of the primary eschar and usually occurs between postoperative day 5 and day 10.

The blood supply to the tonsil is highly variable and complex but is most commonly supplied by the ascending pharyngeal and ascending palatine branches of the external carotid artery.

When called to the Emergency Department to evaluate a post-tonsillectomy bleed, it is best to have several pieces of equipment on hand to order assess and control the bleeding. A head light, tongue depressors, and suction are used to facilitate better visualization. The proper protective equipment, especially eyewear, should always be worn. Most authors recommend removing any clot that is present in order to find where in the tonsillar fossa the bleeding is originating. More often than not, the bleeding is from either the superior or inferior poles.

Several methods have been described in order to stop the bleeding. Having the patient gargle cold water or suck on an ice cube is a simple method that occasionally works, and this can be done while you are gathering supplies. The most tried-and-true methods include cauterization of the fossa using silver nitrate and holding compression with a gauze or tonsil-sponge soaked in oxymetazoline, neosynephrine, or epinephrine. Infiltrating the bleeding site with epinephrine (1:1000) has also been successfully employed. All of these procedures require complete patient cooperation, which can be very difficult in children. If these bedside methods fail, then the patient should be taken to the operating room for an exploration with cauterization and/or suture ligation of the offending vessel.

It is the general rule to admit all of these patients for observation to see if the bleeding resumes. Labs, including a hemoglobin and hematocrit, should be drawn in order to assess the amount of blood lost. Many patients are nauseated from the ingested blood and should be placed on appropriate medication.

Carotid Blowout

Ajani Nugent, M.D.

The carotid artery is the main arterial supply of the head and neck. It is a paired vessel originating on the aortic arch on the left, and the brachiocephalic artery on the right. Both sides divide into the external and internal carotid arteries at the bifurcation at the superior border of the thyroid cartilage (approximately C4 level of the spine). The ICA continues on to the skull base without any branches to the neck. The ECA branches in the neck to provide flow to the superior thyroid artery, ascending pharyngeal artery, lingual artery, facial artery, occipital artery, posterior auricular artery, maxillary artery, and ends as the superficial temporal artery.

The carotid artery is very susceptible to damage from radiation. Carotid blowout is a term that refers to spontaneous rupture of the carotid arteries in patients most commonly preceded by neck irradiation and surgery. There are three subcategories of the carotid blowout syndrome. In a threatened carotid blowout, evidence by exam or radiograph demonstrating spontaneous hemorrhage is inevitable if corrective measures are not taken. In these cases, there has yet to be any evidence of bleeding. An impending blowout has already demonstrated self-controlled brisk bleeding known as sentinel bleeding from a compromised artery. An acute blowout is bleeding that is refractory to pressure and packing.

Arterial weakening secondary to manipulation during surgery, radical resection, wound infection, pharyngocutaneous fistula, recurrent tumor, and disruption to the vaso vasorum from radiation treatment are all contributors to this devastating complication.

Treatment options are limited. Arterial ligation may control hemorrhage but also increase risk of cerebrovascular accident. When being called to treat a potential carotid blowout, one should begin with basic ABC management. Ensure a proper airway, breathing, and circulation with resuscitation techniques. Manual compression of the neck or intraoral packing should be performed to increase transmural pressure and decrease bleeding surface area. Interventional radiology should be notified for the possibility of endovascular management with embolization. If the source of the bleeding cannot be identified, an angiogram may be additionally helpful and set the stage for future embolization. A balloon occlusion may be performed to test collateral circulation to the brain if the bleeding vessel was emobolized and occluded. If collaterals are poor, a stent may be placed. Surgical management is usually last resort and consists of arterial ligation and would debridement.

Tracheostomy Bleeding

Iman Naseri, M.D.

Bleeding from a tracheostomy site may occur within the first 24 to 48 hrs from the time of the surgery. The causes may stem from multiple factors, and typically include: requirement for anticoagulation, intrinsic or extrinsic coagulation deficiencies, exposed vessels or bleeding tissues from the procedure, and improper/excessive postoperative manipulation of the tracheostomy tube. Several specific operative techniques combined with various perioperative precautions can reduce such risks of postoperative bleeding.

General rules that apply to closed surgical wounds do not pertain to a tracheostomy wound. For example, we cannot place a drain or a wrap-around pressure dressing for a tracheostomy wound. Nevertheless, a variety of materials may be used to prevent potential bleeding foci by way of tamponade. These materials include a 'drain' sponge or a Kerlix-type gauze. Others include Surgicel or Vaseline strips. All such materials must be carefully packed inside the wound in the peri-stomal area using a curved clamp. They should not be placed outside the wound on the skin regardless of how much pressure it applies. This method would fail to prevent bleeding as it does not contact the fresh surgical wound. It is important to secure the end of any strip-type packing with tape to the surrounding skin to ensure that the packing is seen and properly removed during the postoperative period.

Whether the tracheostomy procedure is performed in the traditional open versus percutaneously, several principles must be practiced. If moderate to extensive bleeding is encountered during the tracheostomy, packing of the wound should be performed regardless of the patient's presumed bleeding tendency postoperatively. Careful attention must be given to prevent packing any material inside or close to the lumen of the trachea. This may potentially lead to aspiration of the packing material into the distal airway. Encountering anterior jugular vein(s) or thyroid tissue (usually the isthmus) may potentially increase postoperative risks of bleeding. Careful identification and control of bleeding from such vessels is crucial, in addition to proper cauteriziation of any exposed thyroid tissue. Proper tightening of the tracheostomy tube holder is important in helping with tamponade of potential bleeding foci especially when preventive packing is used. A general rule should be to tighten the strap around the neck enough so that only two fingers can be placed between the neck and the strap.

Most importantly, vigorous arterial bleeding must be considered a surgical emergency until proven otherwise. Hemorrhage from the innominate artery, termed a trachea-innominate fistula, has a mortality rate well over 50%. About half of such hemorrhages have sentinal bleeds. Management of these may include an angiogram or operative exploration for diagnosis and operation with likely sternotomy and innominate artery ligation for treatment. Such dire cases are fortunately rare but prompt action may be your only chance to save the patient.

Flap Compromise

Craig R. Villari, M.D.

The development of free-tissue transfer offers the ENT surgeon a valuable tool in reconstructive surgery. In comparison to a pedicled flap, the free flap technique requires microvascular anastomosis of the vessels from the donor site to the recipient site. There are multiple ways to check for flap compromise, but the underlying premise of each technique is the same - flap checks are aimed at verifying that both arterial and venous flow are present in the anastomosis. The bulk of flap failures will occur in the first 72 hours of the post-operative period. Therefore, diligent assessment of flap viability during that time frame is key to operative success.

When assessing a flap, the first thing a physician should do is to check the color and quality of the flap. An edematous or boggy flap can indicate venous obstruction; conversely, a pale flap can signal arterial compromise. Capillary refill should also be maintained and evaluated with a free flap. There are two ancillary techniques that can be used to augment the physical exam. The first requires the physician to stick the flap with a small-gauge needle and to examine the color of the resulting bleed. This technique is an indirect measure of vascular flow; any colored blood other than dark black blood can indicate good vascular patency.

Obviously, this is a subjective assessment so some surgeons prefer the use of Doppler technology to assess flow. Doppler signals can be obtained with implanted leads wrapped delicately around the anastomosed vessels or they can be obtained with hand-held probes placed on the surface of the flap. Both techniques convert the Doppler reading into auditory signals that can then be interpreted to evaluate patency of both arterial and venous systems. Arterial signals are periodic repetitive 'wooshes' that coincide with the patient's pulse; venous signals are a more constant background noise comparable to static coming from a television. If either, or both, of these signals cannot be found, the physician must suspect vascular compromise.

Once vascular compromise has been established, time is of the essence. The evidence for the short-term benefits of aspirin or clot-dissolving agents like streptokinase is limited; surgical exploration of the anastomosis remains the first-line therapy for flap compromise.

Cervical Hematoma

Candice Colby, M.D.

Post-operative neck hematoma is an airway emergency that can occur following any surgical procedure where the neck spaces have been violated. Bleeding can occur following thyroid surgery, neck dissection, or any other operation that violates the neck spaces. The risk is greatest when both sides of the neck have been operated on, as this will cause the trachea to move in a posterior fashion and collapse against the spine. Most postoperative bleeding occurs within a few hours of the operation but sometimes can occur much later. Commonly the bleed is from a small vessel within or just under the skin flap. Occasionally cervical hematoma is seen spontaneously in the absence of surgery, usually due to either a great vessel pseudoaneurysm or hemorrhagic thyroid or parathyroid adenoma.

Patients will present with neck swelling or tightness, or with progressive difficulty breathing. If neck hematoma is suspected, the patient should be closely monitored for expansion of hematoma and progression of symptoms. Inspect for tracheal deviation and degree of respiratory distress. Examine the neck for tense swelling, and whether it is pulsatile or if bruit is present - suggesting a larger vessel bleed that should be handled in a controlled setting to ensure patient safety. If present, inspect for surgical drain output (consistency and amount), bleeding from incision site, or ecchymotic change of the neck.

The determination to re-explore the hematoma is a clinical one, though threshold for doing so should be low. Airway compromise can occur quickly and any sign of such compromise should prompt quick action. Be prepared to explore and decompress the wound locally, in the absence of any anesthesia, if necessary to control the airway.

Whether performed in a dire emergency or in a more controlled fashion, usually blunt exploration, suction, and profuse irrigation of the surgical bed will identify the bleeding culprit. Sometimes this is a large identifiable vessel, other times no discrete vessel can be identified. In the latter case the vessel may have finally thrombosed, retracted into a muscle, or perhaps the the bleeding was due to a systemic coagulopathy allowing diffuse hemorrhage from arterioles and venules.

After the wound is irrigated and bleeding controlled, one may consider applying topical thrombotic agents such as exogenous thombin. Also, consider that significant supraglottic edema may not be much improved by drainage of the hematoma, so due caution should be taken upon extubation.

Preoperative studies may help to predict those at highest risk of bleeding, but do little in the way of prevention. Similarly placement of a surgical drain seems to do little to protect the patient from this complication.

Pharyngocutaneous Fistula

Katherine Hayes, M.D.

Pharyngocutaneous fistulas can develop following pharyngeal surgery (i.e. laryngectomy) as an inappropriate communication between pharyngeal mucosa and the skin of the neck. Several factors predispose a patient undergoing pharyngeal/laryngeal surgery to fistula formation. Pre-operative radiation damages tissues at the cellular level via oxidative stress and fibrosis, thus impeding proper wound healing after surgery. In addition, patients with malnutrition or systemic diseases such as COPD, diabetes, or CHF also suffer from poor wound healing and have increased risk of fistula formation post-operatively. Optimization of nutritional status and management of underlying chronic disease is critical. From the surgical standpoint, all efforts should be made to achieve impeccable, tight closure as the most effective method of preventing fistula formation.

There are a few signs which should trigger suspicion for fistula formation including neck edema, erythema, tenderness at suture sites, fever, and visible drainage. Many advocate a small local wound exploration as part of the workup of post-operative fever, serving to both identify a leak early and provide a clear drainage pathway medial to the great vessels. Fistulae most often develop within the first 7 days, but can be seen up to 6 weeks post-op. Usually clinical diagnosis will suffice, but barium swallow or methylene blue testing (visualize dye leaking from wound) can clinch the diagnosis. If a drain is in place or a sample of fluid can be obtained, elevated amylase confirms the presence of saliva in the fistula tract.

Conservative treatment is first line and includes initiation of tube feeding with NPO status, addition of clindamycin (or other broad spectrum antibiotic) if fever or wound erythema is apparent, and packing of the wound with gauze 2-3 times per day to facilitate wound healing and prevent further damage to surrounding skin and soft tissue. Placement of a temporary drain may be helpful to prevent seepage into a nearby stoma and avoid aspiration. Additionally, salivary diversion tubes may be placed in the pharynx in an attempt to reduce exposure of the wound to saliva.

One should be concerned if a lateral fistula has developed due to the possibility of soft carotid artery adventitial breakdown. Due to the risk of carotid blowout in this situation, further imaging with CT and closure with a local myocutaneous or pectoralis major flap is likely the best option. If conservative treatment for 2-3 weeks shows no signs of fistula closure, surgical closure becomes necessary. Primary closure is feasible in patients with adequate non-necrotic mucosa which can be approximated with minimal tension (this is uncommon). Other patients require secondary closure with regional muscle or fasciocutaneous flaps.

Hypocalcemia

G. Aaron Rogers, M.D.

Hypoparathyroidism after thryoidectomy is the most common cause of acute hypocalcemia seen by the otolaryngologist. The etiology is usually inadvertent removal or devascularization of the parathyroid glands following total, or completion, thyroidectomy. Theoretically, any neck surgery can have this complication.

Calcium circulates in blood as a cation, largely sequestered by albumin and other proteins. Free ionic calcium is the "active" form, however is difficult and expensive to measure directly. Total serum calcium is measured most commonly, and includes calcium bound to albumin. A simple formula is used to correct calcium levels in states of hypoalbuminemia:

Corrected Ca = Measured Ca + 0.8 x (4 - Albumin)

Additionally, states of hypomagnesemia will cause renal sacrifice of calcium to spare magnesium, making hypocalcemia even more difficult to treat.

Signs and symptoms of hypocalcemia in this setting are usually isolated to muscular complaints, tetany, and cardiac arrthymias. Generalized seizures and laryngospasm are also possible. Subacute mild hypocalcemia may also present with psychosis.

Chvostek's sign is a facial muscle spasm elicited by firmly tapping the parotid gland (proximal facial nerve). Trousseau's sign is carpopedal spasm seen after momentary ischemia to the forearm (as with a blood pressure cuff). EKG changes include a lengthening of the QT interval (and corrected QT interval), and a lengthening of the ST interval. T-wave changes may be seen as well. Heart block and ischemia may result.

There are various algorhythms for managing postoperative calcium replacement. After total thyroidectomy we routinely check serum calcium, albumin, and magnesium every 8-12 hours for 24 hours.

Calcium replacement of the asymptomatic patient usually begins when the corrected calcium is less than 8.4mg/dL. We typically replace with oral Calcium Carbonate + Vitamin D (OsCal D(r)) 500-1000mg TID. For calcium levels below 7mg/dL, replacement with IV calcium gluconcate 1-2g is appropriate.

For symptomatic patients (especially with EKG changes) IV calcium replacement is preferred. Adults who require multiple IV boluses may benefit from a calcium gluconate maintenance "drip" of 100mg/hr for 24 hours. Keep in mind that these patients will still benefit from the relative speed of preparation and administration of oral calcium; some physicians keep Tums® at the bedside.

When replacing with oral calcium remember that an acidic gastric environment is required for absorption, so tablets should be given with meals. Absorption will be unpredictable if the patient is on antacid medications.

When treating any patient for hypocalcemia, remember that the most common serious complication is heart block and arrhythmia. Some patients may require multiple EKG's or constant cardiac monitoring.

Medicine or endocrinology consults also may be helpful.

Cerebrospinal Fluid Leaks

Eric Berg, M.D.

Cerebrospinal fluid leaks may be caused by iatrongenic injury, trauma, or spontaneous occurance. Such a leak may be diagnosed immediately or may have a more delayed presentation. Leaks may also be seen with congenital anomalies, skull base neoplasms, or erosive processes. If no identifiable cause is found, the leak may be termed idiopathic.

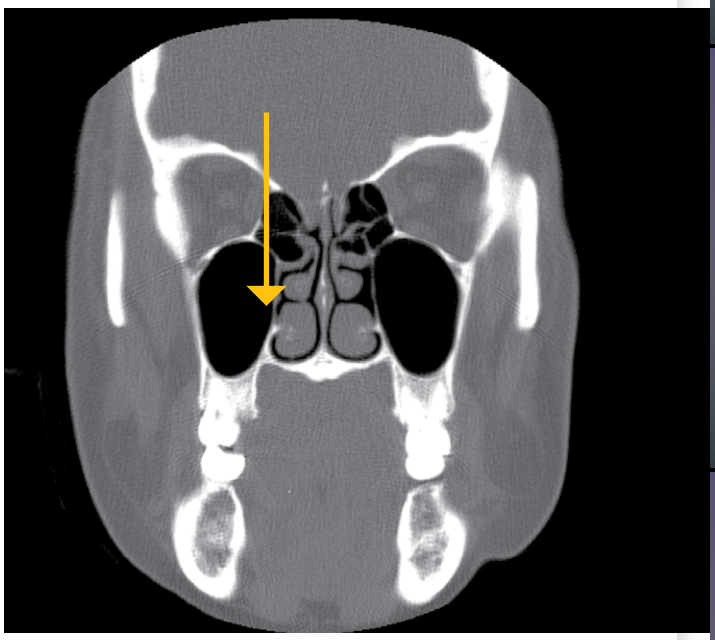
Patients typically present with complaints of clear, salty, watery nasal drainage from the nose or ears made worse when the head is lowered or with valsalva or strong coughing (all of which increase ICP). Defects involving the cribriform plate may present with disturbance of smell. It should be noted that CSF otorrhea may present as rhinorrhea via collection in the middle ear and drainage via the eustachian system.

Evaluation is basically a two step process; first one must confirm that the fluid is in fact CSF, then one must localize the defect(s). Fluid should be collected for analysis. The “halo sign” is non-specific and refers to fluid separation on gauze with an external fluid ring and a central spot of blood. A glucose level greater than 30 mg/cc suggests CSF but has a high false-positive rate and may be falsely-negative in the presence of active bacterial meningitis. The most specific test is an assay for □2-transferrin.

Once CSF has been confirmed, evaluation is generally followed by a high-resolution CT scan for bony evaluation. MRI to evaluate soft tissue is complementary. Interthecal radioactive dyes or fluorescein may be employed using cotton pledgets as a more sensitive but non-localizing test. Endoscopic evaluation may directly visualize a leak or even prolapsing cranial contents.

In the case of accidental trauma, management is initially conservative with bed rest, elevation of the head of bed, avoidance of strain, stool softeners, and osmotic and diuretic agents. Prophylactic antibiotics are controversial but empiric therapy is imperative if meningitis is suspected. If no improvement is seen after 2-3 days of conservative management, lumbar drain placement for CSF diversion or definitive surgical management should be considered. If a leak is a recognized surgical complication, repair should be attempted immediately.

Nontraumatic leaks and leaks of an extended duration are more likely to require surgical repair. Modern operative approaches are primarily endoscopic. Basic principles consist of selection and possible harvest of graft material, graft application, and graft stabilization. Post-repair management includes observation in an ICU with lumbar drain management for ~5 days in addition to the conservative measures outlined above. Strenuous activity should be avoided for approximately 6 weeks.



Inappropriate or delayed management of cerebrospinal fluid leaks may have devastating complications. Skull base defects leave patients vulnerable to meningitis, cerebral abscess, pneumocephalus, intracranial hemorrhage, and secondary cerebral compression. Prompt evaluation and management is essential.

Orbital Hematoma

Kara Prickett, M.D.

Orbital (or retrobulbar) hematoma is a rare but serious complication of trauma to the orbit or surgical intervention in or around the orbit. Due to the relative inelasticity of the periorbita and its firm fixation to the orbital rim, collection of blood within the orbital space can result in a rapid rise in pressure and development of orbital compartment syndrome. As pressures rise, they may reach levels sufficient to cause external compression of retinal veins and arteries. Resultant retinal ischemia can lead to permanent blindness in as little as 60-90 minutes.

The diagnosis of orbital hematoma is primarily clinical, and index of suspicion should rise if there is a plausible mechanism in the history. Almost all cases are preceded by surgery or trauma; symptoms may progress rapidly, or over several days, depending on the source of the bleed. Signs and symptoms suggestive of orbital hematoma include proptosis, chemosis, eye pain, progressive decrease in visual acuity, and opthalmoplegia. Fundoscopic examination may show papilledema and a pale optic disc, but is not always feasible in the emergency setting. Documentation of increased intraocular pressure is important to establish the diagnosis, with most reported cases having IOP > 40 mmHg (normal IOP 10-20 mmHg). Lab or imaging studies may delay sight-saving treatment and are not usually indicated.

The differential diagnosis includes retrobulbar edema (the most likely cause of proptosis in trauma patients), sub-periosteal hematoma, orbital emphysema, anterior ischemic optic neuropathy, central retinal artery occlusion, and traumatic injury to the globe.

Management should begin with standard ATLS protocol to assess for life-threatening injuries in any trauma patient. Urgent ophthalmologic consultation is always indicated. In rapidly progressive cases, immediate initiation of pharmacotherapy with osmotic agents and/or carbonic anhydrase inhibitors and orbital decompression via lateral canthotomy should be undertaken. Reassessment of intraocular pressure and visual acuity after orbital decompression is important; if acuity does not improve and pressures do not fall significantly, other diagnoses such as orbital emphysema or sub-periosteal hematoma should be considered. The patient should be admitted for serial examinations and monitoring. Cosmetic repair of the lateral canthus may be undertaken prior to discharge or at a later date with good result.

Performing a lateral canthotomy

Lateral canthotomy can be safely performed with the contents of a basic instrument tray. Lidocaine 1-2% with or without epinephrine should be infiltrated into the area of the lateral canthus. A small clamp can then be placed with one tine inside the lid and one tine outside the lid and directed toward the lateral orbital wall where the attachment of the lateral canthal tendon should be palpable. The clamp is tightened for 60 seconds to provide a guide for incision and to prevent undue bleeding. Incision of the skin and removal of the tendon from its insertion on the orbital rim may be done with a scalpel or (more safely) with small, sharp scissors. The plane of orbital septum and canthal tendon MUST be violated to provide adequate decompression.

Miscellany

Epistaxis

Lindsey Arviso, M.D.

Nosebleed is one of the most common conditions an otolaryngologist will be asked to evaluate. Usually from the anterior septum, 90% are located at Kiesselbach's plexus in Little's area, a confluence of vessels at the anterior septum. Injury or anything that produces hyperemia in the nasal mucosa predisposes to epistaxis, including: trauma, digital manipulation, vigorous blowing, sinusitis, allergy, neoplasm, hypertension, renal disease, hepatic failure, hemophilia, acquired thrombocytopenia, hereditary hemorrhagic telangiectasia, blood thinners, NSAIDs, thioridazine, dipyridamole, anticholinergics, nasal sprays, decongestants, and cocaine.

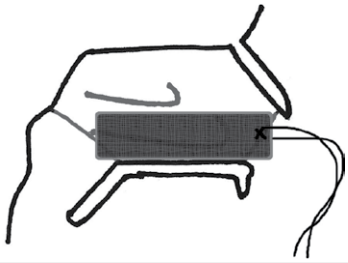
A significant nosebleed may require urgent attention and management. Similar to a trauma situation, large-bore peripheral IV access is a must. Face mask, gown, and gloves for the provider are pertinent. Take a history regarding inciting factors, therapies already attempted, and approximate blood loss.

First line treatment of epistaxis should include a trial of direct pressure applied for 15 minutes. Topical neosynephrine, epinephrine, or oxymetazoline applied liberally to the nasal mucosa are also very useful. Any contributing medical factors should be optimized if possible.

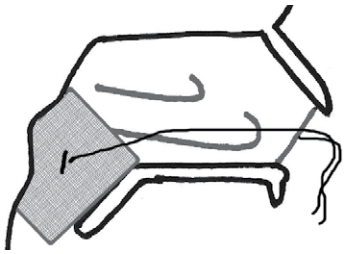
Cautery with silver nitrate may be attempted for small anterior bleeding vessels. Packing with an expanding nasal tampon or inflatable compression balloon pack is usually definitive but painful. Moderate bleeding from the posterior nasopharynx is often controlled by longer (10cm) nasal tampons or a double-balloon style pack (Epistat).

Tamponade of the nasopharynx can also be achieved via a Foley catheter balloon or posterior gauze packing. Failure of the above interventions may require percutaneous intralumenal embolization Epistaxis or surgical ligation.

Anterior Packing with Nasal Tampon



Posterior Packing with Gauze Roll



Posterior Packing with Foley Catheter

Angioedema

Jodi Zuckerman, M.D.

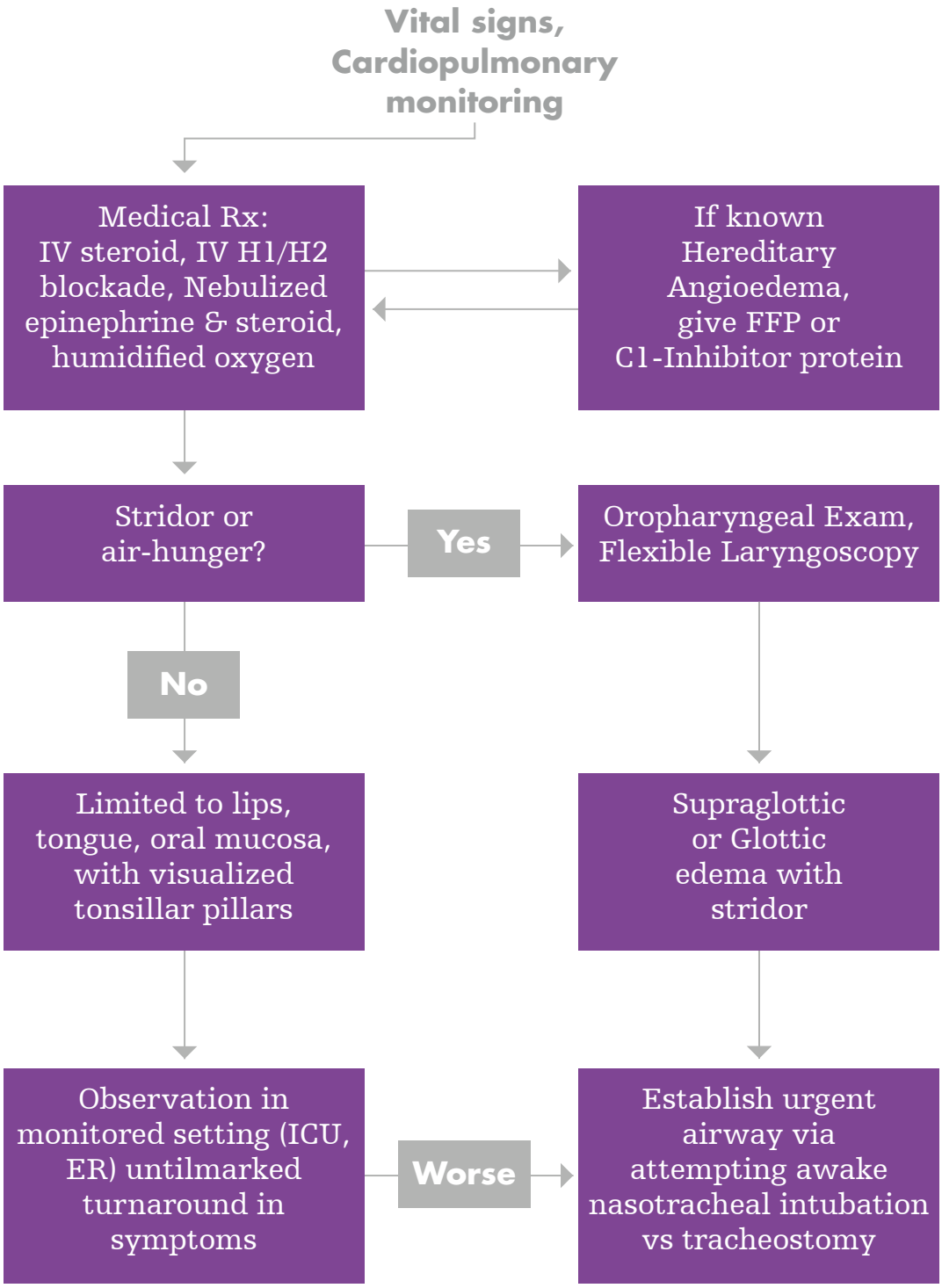
Angioedema is caused by post capillary venule inflammation that results in leakage and edema in the submucosal and subcutaneous layers. This is triggered by inflammatory mediators such as histamine, serotonin, and kinins. This can occur in the face, lips, floor of mouth, tongue, pharynx, and larynx.

The causes of angioedema are numerous and can be classified as IgE mediated, complement mediated, hereditary, idiopathic, and drug reaction (most commonly ACE inhibitors).

The on-call otolaryngologist needs the following items to be truly equipped in the work up of the patient with angioedema: tongue blade, light source, topical vasoconstricting and anesthetic agents, flexible laryngoscope, intubation supplies including (endotracheal tube and anesthetic agents), and lastly a tracheostomy tray.

The work up should begin with obtaining a history to provide details to the allergen or trigger. A physical exam should detail the oropharynx specifically determining tongue swelling and visualization of the posterior tonsillar fossa. Flexible laryngoscopy can then be performed to determine if swelling extends to the pharynx and larynx. Imaging is not indicated in this setting.

Definitive treatment is of course medical in nature, however stabilization of the airway may be required while awaiting resolution of symptoms. The decision on whether to establish an airway should be based on the overall clinical presentation, not a single exam finding. Most important is the patient's progression of symptoms over time.


Vital signs, Cardiopulmonary monitoring
Medical Rx: IV steroid, IV H1/H2 blockade, Nebulized epinephrine & steroid, humidified oxygen
If known Hereditary Angioedema, give FFP or C1-Inhibitor protein
Stridor or air-hunger?
Yes
Oropharyngeal Exam, Flexible Laryngoscopy
No
Limited to lips, tongue, oral mucosa, with visualized tonsillar pillars
Supraglottic or Glottic edema with stridor
Observation in monitored setting (ICU, ER) untilmarked turnaround in symptoms
Worse
Establish urgent airway via attempting awake nasotracheal intubation vs tracheostomy

Inhalation Injuries

Elizabeth Hoddeson, M.D.

Inhalation injuries can result from inhalation of noxious substances or from a fire, particularly in an enclosed space, with injury resulting from inhalation of smoke, hot dry gas, or steam. With hot dry gas, injuries are often limited to the oropharyngeal and upper airway due to the dissipation of heat in the nasopharyngeal and oropharyngeal tissues and glottic adduction reflex to heat to protect the lower airways. The heat capacity of steam is much greater than that of dry air, and the upper airways are often overwhelmed by the insult, resulting in a higher frequency of injury to the lower airway tissues. The mucosa of the respiratory tract is highly specialized in order to properly serve its important functions of humidification and prevention of small substrates from reaching and occluding the very small spaces of the lower airways. The lungs are highly susceptible to injury and cell death from exposure. Airways are left vulnerable to accumulation of mucus and substrates in lower airways that may form obstructive plugs and serve as a medium for bacterial growth.

Upon examination of a patient with suspicion for inhalation injury, signs that prompt concern include facial burns, singed facial and nasal hair, carbonaceous sputum, wheezing, stridor, cough, oropharyngeal edema, tachypnea, respiratory distress, copious sputum production, and thick secretions. Symptoms often evolve and can be delayed in situations of inhalation injury up to 24 – 36 hours. The best tests to detect inhalation injuries include direct fiberoptic laryngoscopy and bronchoscopy.

Conservative management of suspected inhalation injury may be initiated as the patient is closely monitored in order to detect possibly evolving symptoms; this includes cool humidified oxygen and systemic rehydration. Use of steroids in this setting is not supported in the literature. Definitive management of inhalation injury is securing the airway through early endotracheal intubation prior to development of an emergent airway situation. This allows for suctioning of lower airways and prevention of upper airway occlusion as edema progresses. Tracheotomy may be required either emergently, in situations of significant injury to the larynx in order to avoid further trauma by forced orotracheal intubation, or established as a secondary measure if ventilation dependency ensues or for very thick secretions difficult to manage through an endotracheal tube. Tracheotomy should be weighed carefully in the setting of significant burn to the neck, which will adversely affect wound healing and serve as a nidus for infection.

Pneumonia and tracheobronchitis are common complication in patients with inhalation injury due to decreased ciliary function for mucus and soot clearance and increased permeability of alveolar membranes resulting in lower airway flooding.

Facial Nerve Paralysis

Craig R. Villari, M.D.

The course of the facial nerve can be grossly characterized into three subdivisions: intra-cranial, intra-temporal, and extra-cranial. Paralysis can result from several mechanisms including congenital, traumatic, infectious, neoplastic, iatrogenic, and idiopathic processes. As with building a differential diagnosis for other conditions, the provider should obtain information about the onset, characteristic, contributing factors, and associated symptoms involved in the paralysis. The House-Brackmann grading system (below) is useful in communicating the degree of paralysis to other providers.

House-Brackmann Class Key Characteristics

Grade I - Full function
Grade II - Slight weakness
Grade III - Slight, but appreciable weakness; can still close eye
Grade IV - Incomplete eye closure but symmetrical facial muscular tone at rest
Grade V - Obvious disfigurement but retains some muscular function
Grade VI - No function

The facial nerve is susceptible to severe trauma resulting in transverse fractures of the temporal bone. Fortunately, for facial nerve function, those fractures are less common than longitudinal fractures. Treatment options for temporal bone fractures affecting the facial nerve include steroids and surgical decompression.

The more distal branches of the facial nerve are also vulnerable even with fairly superficial lacerations. After copious irrigation to cleanse the wound, early surgical neurroraphy using either primary or cable graft technique should be considered.

There are many causes of infectious facial nerve paralysis, but the most well studied are Ramsay Hunt Syndrome and Lyme disease. Ramsay Hunt syndrome results from the reactivation of latent herpes simplex virus. It is almost exclusively unilateral in presentation and is easily diagnosed when telltale painful vesicles are also present. Treatment requires a 7-10 day course of acyclovir and steroids. Ophthalmology should be consulted if eye involvement exists.

Lyme disease is caused by a spirochete transmitted in the bite of the Ixodes tick. The disease can present with unilateral or bilateral paralysis and is most-easily diagnosed when the classic 'bull's eye' rash is present. Treatment is focused on appropriate antibiotic treatment, usually doxycycline or cefuroxime.

Bell's palsy is the most common cause of facial paralysis. It is an idiopathic disorder of acute onset that is almost always unilateral. A stroke workup is necessary if there is sparing of forehead movement. Though the exact etiology of the disorder is unclear, it is usually treated with a 10 day course of steroids; of note, treatment does not alter disease prognosis if initiated 3-4 days after onset. Surgical decompression of the facial nerve may be considered when refractory to medical treatment or recurrent in nature.

It is important to remember several principles when treating a patient with facial nerve paralysis. Patients are sometimes seen by a neurologist before coming to an otolaryngologist's attention, but a full neurologic exam should be performed to rule-out other neurologic deficits. Additionally, one cannot underemphasize the importance of ophthalmologic care if there is any inability to protect the eye; depending on the degree of paralysis, simple eye lubrication or tarsorrhaphy may be indicated.

Sudden Sensorineural Hearing Loss

Praveen Duggal, M.D.

Sudden Sensorineural Hearing Loss (SSHL) is defined as a sensorineural hearing loss of greater than 30dB over three contiguous pure-tone frequencies occurring within a 3-day period. Sudden loss can occur from many causes such as infection, autoimmune disease (Sarcoid/Wegeners granulomatosis /Cogan syndrome), vascular compromise, retrocochlear pathology/tumor, trauma to the temporal bone, pharmacologic toxicity, or idiopathic. Most SSHL are idiopathic and include both abrupt and rapidly progressive losses without an explained cause or underlying pathology. Incidence of 5-20 per 100,000 persons a year. Average age ranges from 40-50's with equal sex distribution. SSHL is most likely a symptom that may have several different known and/or unknown etiologies. Four different pathways have been discussed as possible idiopathic SSHL causes including

- Viral infection
- Vascular compromise
- Intracochlear membrane rupture (double membrane break theory)
- Autoimmune mediated

One fourth of all SSHL patients report a viral infection within 1 month of their loss. SSHL has been shown to possibly be linked with damage by active or latent viruses to cause loss of hair cells or other inner ear structures. Inner ear vascular compromise from occlusion or obstruction may lead to reduction in cochlear blood supply. Rupture of any of the membranes separating the perilymph and endolymph may lead to a change in the membrane potentials needed for hearing. Autoimmune causes can be associated with other immune mediated disorders but remains unclear in SSHL.

Evaluation should include time onset and course of SSHL, symptoms associated with loss, and what occurred before/during/and after loss. Medications should be reviewed for any ototoxic side effects. A complete head and neck physical exam should be performed with thorough evaluation of the ears, as well as a thorough neurologic/cranial nerve evaluation. The Hum, Weber, and Rinne tests may be of use to determine side of loss and SSHL vs conductive hearing loss.

Labs should include an ANA, RF, and ESR to rule out autoimmune causes. A WBC as well as FTA-Abs can be ordered to rule out infection and possible syphilis.

Audiometry is a mandatory test that needs to assess baseline hearing and/or loss. ABR and OAE can be ordered to assess auditory nerve and cochlear function. An MRI with gadolinium is excellent for evaluating retrocochlear masses. Most agree this need not be done in an inpatient setting.

Treatment for SSHL should consist of treating the underlying etiology. Current empiric standard of care for idiopathic SSHL is a tapered course of oral corticosteroids. Prednisone 60mg over 4 days followed by a taper of 10mg every 2 days over 10-14 days is adequate. Greatest recovery is noted when steroids are initiated within the first 1-2 weeks of symptom onset. Little benefit noted after 4 weeks. Antiviral agents have mixed results, and if used should be given in combination with steroids. Intratympanic steroid injection has been effective in patients with severe/profound SSHL that have failed standard treatment. Carbogen is an inhalational vasodilator that can improve cochlear blood flow and help with sudden deafness, though it is rarely used.

Foreign Bodies

Avani Ingley, M.D.

Esophageal Foreign Bodies

Foreign body ingestion is a common problem in both adults and children. In adults, the most likely objects are fish bones, dentures, and meat. Children are most likely to swallow coins. Ninety-five percent of esophageal foreign bodies become lodged at the cricopharyngeus; other common locations are the gastroesophageal junction, and the indentations of the aortic arch and left mainstem bronchus.

Patients typically present with dysphagia, drooling, weight loss, chest pain, or fever. The first step in evaluation is a chest X-ray. It is key to get a lateral x-ray in children who have swallowed coins to confirm the number of coins in the esophagus. If X-rays do not reveal a foreign body and the patient has a convincing history and physical exam, then exam under anesthesia with either flexible or rigid esophagoscopy should be pursued. Barium swallow should be avoided as it may obscure the endoscopic view. Patients who are drooling or in any respiratory distress should be taken to the operating room urgently. Asymptomatic patients should be taken to the operating room within 24 hours. Patients with disc batteries should have them removed urgently.

Complications include esophageal perforation, aspiration, mediastinitis, pneumothorax, and pneumomediastinum.

Airway Foreign Body

Airway foreign bodies present a diagnostic and therapeutic challenge to the otolaryngologist. Typically, the patients present to the emergency department or primary care physician. Physicians should have a high index of suspicious for aspiration, especially in children because foreign body aspiration can mimic many other conditions. The most commonly aspirated objects are peanuts, balloons, hot dogs, or small toys. History from witnesses can be a key element in diagnosis.

Patients can present with choking, stridor, chest pain, hoarseness, or wheezing. A chest x-ray with inspiratory and expiratory phases should be performed to evaluate for radiopaque items and for air trapping. Objects can become lodged anywhere along the airway, but the most common are at the level of the larynx, in the trachea, or the right mainstem bronchus.

Management involves laryngoscopy and rigid bronchoscopy. Light sedation with short-acting muscle relaxants and jet ventilation should be used. Intraoperative with or without post-operative steroid should be given to reduce inflammation. If removal is not accomplished with rigid bronchoscopy, then a thoracotomy may be necessary.
ALWAYS LOOK FOR A SECOND OBJECT

Complications can include pneumonia, pneumothorax, pneumonitis, parenchymal loss, or chronic lung infections.

Nasal Foreign Body

Nasal foreign bodies should be removed with little delay as there is a potential for aspiration or airway obstruction. Instruments necessary include nasal speculum, nasal suction, small ear instruments with curvature such as (mastoid pick and wax curette), and foley catheters. With assistance from ancillary staff, the patient can be papoosed in place. After anesthetizing, the nose instruments can be used to grab the foreign object and remove it.

Ear Foreign Body

Most critical in this assessment is to determine what exactly the foreign body is. If it is a food particle or bean it may expand with irrigation and become extremely painful for the patient. If a report of a battery or a caustic substance is lodged in the ear, this must be removed immediately to avoid corrosive damage to surrounding structures. Otherwise, it is common practice to remove these foreign bodies under microscopy in clinic as an outpatient.

Tracheostomy Care

Anita Sethna, M.D.

Triaging tracheostomy problems is one of the most common issues that may arise on call. Common complications after tracheostomy and initial management strategies are listed below.

Bleeding: a separate chapter addresses tracheostomy bleeding. Consider packing with hemostatic gauze if not severe.

Pneumothorax: Occurs in up to 4% postoperatively. A chest xray is diagnostic. A small pneumothorax can sometimes be managed with high-flow oxygen (and avoiding positive pressure ventilation). Otherwise, a chest tube will be required.

Post-obstructive Pulmonary Edema: Due to acute relief of airway obstruction, the alveoli can become flooded with interstitial fluid (and even luminal fluid). CXR and clinical suspicion is diagnostic. Diuresis or positive pressure ventilation may be required.

Tracheitis: Some degree of tracheitis will always be present in trach patients. Minimizing irritation is possible by tracheal humidification, even by "aspirating" and coughing up sterile saline (often in 1-3mL bullets). Also, ensuring a firm trach holding strap will help prevent the tracheostomy tube from irritating the tracheal walls.

Mucous Plugging: If this is suspected, remove the inner canula, and replace or clean it. If the plug is distal in the airway then aggressive suctioning or even bronchoscopy may be necessary. Regular irrigation and humidification will help minimize this.

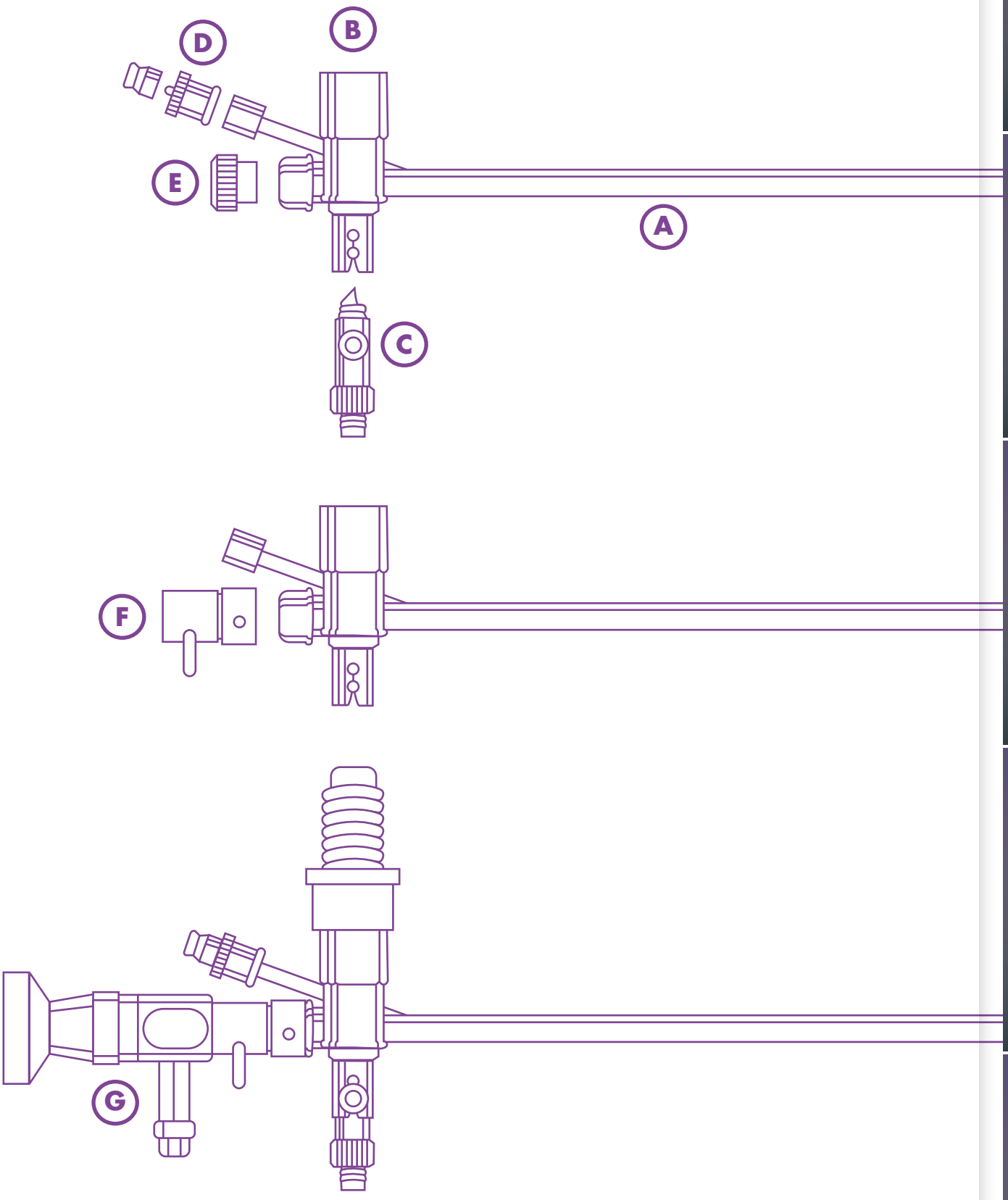
Tracheostomy Tubes: A size 6 Shiley tube will be large enough for most patients. Unless a patient requires positive pressure ventilation, a cuffless tube is usually best. Some may advocate a cuffed tracheostomy tube for aspirating or bleeding patients as well.

Ventilating Bronchoscope

Legend: Ventilating Bronchoscope Setup

A. Rigid Bronchoscope, bevel up with light port inferior, ventilator circuit port superior, visualization port left and accessory suction/irrigation port.
B. Ventilator circuit and port.
C. Prism adapter for light cord. Needed for visualization when not using a telescope, and still needed for creating a seal when illuminated telescope is used.
D. Accessory port cover and plug. Needed for seal.
E. Eyepiece or lens. Needed for seal when not using a telescope. Connects directly to bronch scope without using the bridge.
F. Bridge. Held by friction onto bronch scope and has locking mechanism to attach to telescope.
G. Telescope, locked to bridge with bridge pushed firmly onto bronch scope.

The bottom image demonstrates the assembled bronchoscope / telescope most commonly used. A camera and light cord are yet to be attached to the telescope. The bronchoscope is carefully slid through the larynx after direct laryngoscopy using a Parsons or similar open-ported rigid laryngoscope.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