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19 CHAPTER

Otorhinolaryngology

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Chapter contents

Emergencies

Key Points

- Drooling, posturing, and air hunger are classic signs of epiglottitis.
- A lateral neck radiograph showing a thumbprint sign can be diagnostic of epiglottitis.
- Although most peritonsillar abscesses should be drained, smaller and early abscesses may respond to medical therapy.
- Acute onset of wheezing in a child with no history of asthma should alert the clinician to a possible airway foreign body.
- A high index of suspicion is required for the timely diagnosis of retropharyngeal abscess.
- Adolescent boys with recurrent epistaxis may have a juvenile nasopharyngeal angiofibroma.

Epiglottitis

Epiglottitis (or "supraglottitis") is a condition that requires prompt attention by the physician. Epiglottitis results from bacterial (and rarely viral) infection of the supraglottic structures, that is, the epiglottis and arytenoid cartilages. A high level of suspicion is necessary to make a diagnosis and avoid significant morbidity. Rapid decompensation and complete loss of the airway are the sequelae of most concern. The physician should always be suspicious when a patient presents with fever, sore throat, and difficulty swallowing, and when the severity of oropharyngeal physical findings is not in proportion to the symptoms. Croup, tonsillitis, peritonsillar abscess, and other neck infection may be incorrectly diagnosed in these patients. Epiglottitis occurs mainly in children age 2 to 7 years, although infants, older children, and adults can be affected. Mortality rates of 6% to 7% have been reported in adults.

Signs and symptoms of epiglottitis include rapidly developing sore throat, high fever, restlessness, and lethargy. A "supraglottic," muffled voice is common. Many patients have difficulty with their saliva and drool. Classically, these patients are in a sitting position leaning forward, because this position tends to alleviate obstructive symptoms from the supraglottic swelling. They may show signs of "air hunger" or may have stridor.

Differential diagnosis includes tonsillitis, peritonsillar abscess, retropharyngeal abscess, airway foreign body, and

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Table 19-1 Distinguishing Features of Epiglottitis and Croup

Feature	Epiglottitis	Croup
Cause	Bacterial	Viral
Age	1 year to adult	1 to 5 years
Location of obstruction	Supraglottic	Subglottic
Onset	Sudden (hours)	Gradual (days)
Fever	High	Low grade
Dysphagia	Marked	None
Drooling	Present	Minimal
Posture	Sitting	Recumbent
Toxemia	Mild to severe	Mild
Cough	Usually none	Barking, brassy, spontaneous
Voice	Clear to muffled	Hoarse
Respiratory rate	Normal to rapid	Rapid
Larynx palpation	Tender	Not tender
Clinical course	Shorter	Longer

From Berry FA, Yemen TA. Pediatric airway in health and disease. Pediatr Clin North Am 1994;41:153.

croup. Physical examination with laryngoscopy is extremely useful in differentiating these diagnoses. Endoscopy should not be performed if there is concern of impending airway obstruction. Endoscopy will typically show erythema and edema of the epiglottis and arytenoid cartilages. Other findings include laryngeal tenderness on neck palpation, although palpation should be avoided when the diagnosis is being considered.

Any time the diagnosis of epiglottitis is in question, otorhinolaryngologic (ear-nose-throat, ENT) and infectious disease consultations are warranted. Placement of a tongue depressor has been known to precipitate acute airway obstruction and should be avoided entirely if epiglottitis is strongly suspected. Differentiation from croup can be difficult because there is considerable overlap of symptoms (Table 19-1) (Berry and Yemen, 1994). A lateral extended neck radiograph can help in the diagnosis. X-ray evidence includes the classic "thumbprint" sign. If epiglottitis is suspected or lateral neck radiography is confirmatory, the patient should be taken to the operating room (OR) for orotracheal intubation in the presence of an anesthesiologist and an otorhinolaryngologist. In any case of airway obstruction, cricothyrotomy or tracheotomy can be lifesaving, because orotracheal intubation can be difficult and sometimes impossible. Some patients, usually adults, may be treated expectantly with intravenous (IV) medications and intensive care unit (ICU) observation as long as personnel are available for control of the airway if necessary. If airway stability is questionable, observation is not recommended.

After control of the airway is achieved, cultures of the epiglottis should be obtained and directed antibiotics instituted. *Haemophilus influenzae* type b (Hib) is common and can be beta-lactamase producing. Other, less common organisms include beta-hemolytic streptococci, *Streptococcus pneumoniae,* and *Staphylococcus aureus.* Antibiotics should be administered parenterally; effective antibiotics include cefotaxime, ceftriaxone, ampicillin plus sulbactam, or ampicillin plus chloramphenicol. Steroids can be useful for edema and inflammation, but their effectiveness has not been proved in controlled studies..

The incidence of epiglottitis in children is decreasing since the introduction of the Hib vaccine in the late 1980s. However, the incidence has remained stable or slightly increased in adults.

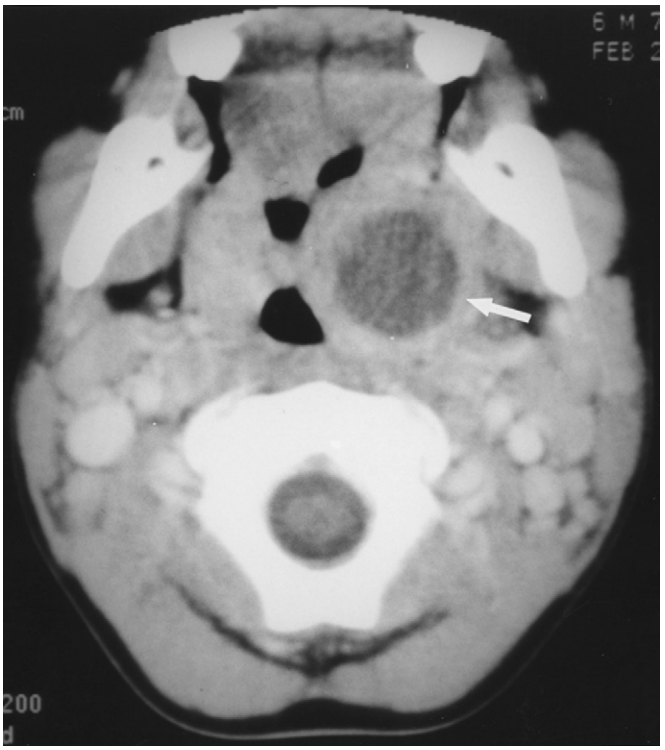


Figure 19-1 Large left peritonsillar abscess *(arrow)* that required surgical drainage.

Peritonsillar Abscess

A peritonsillar abscess is the accumulation of pus in the peritonsillar space that surrounds the tonsil. The same organisms responsible for common tonsillar infections— *Streptococcus* and *Staphylococcus* species and anaerobes—are also found in peritonsillar abscesses.

The typical signs and symptoms of peritonsillar abscess include fever, sore throat for 3 to 5 days, dysphagia, odynophagia, and a muffled, "hot potato" voice. Trismus is extremely common. Examination confirms asymmetric tonsils and peritonsillar edema and erythema. The soft palate and uvula are swollen and displaced away from the side of the abscess. It is often difficult to distinguish between abscess and peritonsillar cellulitis. If possible, it is helpful to palpate because fluctuance indicates a loculation of pus. Diagnosis is often made by clinical impression, but computed tomography (CT) can be confirmatory and useful when the diagnosis is uncertain (Fig. 19-1).

If untreated, a peritonsillar abscess may spontaneously drain, progress to involve the deep neck, or even lead to airway obstruction. The most important part of the treatment is drainage of

the abscess cavity by needle aspiration, incision and drainage, or tonsillectomy. Cultures of the aspirate can be obtained, and broad-spectrum antibiotics should be started. Appropriate antibiotics include ampicillin-sulbactam (Unasyn) or clindamycin (Cleocin). Many patients present with dehydration, and parenteral fluids should be given if necessary. Analgesics should be prescribed as needed. One or two doses of IV corticosteroids may be given to decrease inflammation and pain.

Children presenting with peritonsillar abscess should be admitted to the hospital. Treatment with IV hydration and parenteral antibiotics is appropriate initially. Patients with peritonsillar cellulitis/phlegmon or early abscess often demonstrate a rapid response to treatment, whereas those with a well-formed peritonsillar abscess do not improve. Drainage is necessary in nonresponders. Abscesses in cooperative adults can be drained under local anesthesia in the emergency department (ED) or office and treated in an outpatient setting. Children usually require general anesthesia for drainage, and a tonsillectomy may also be performed. An elective tonsillectomy is often recommended for any patient with a peritonsillar abscess to prevent recurrence, especially with a history of recurrent tonsillitis, although few, if any, controlled studies support this recommendation.

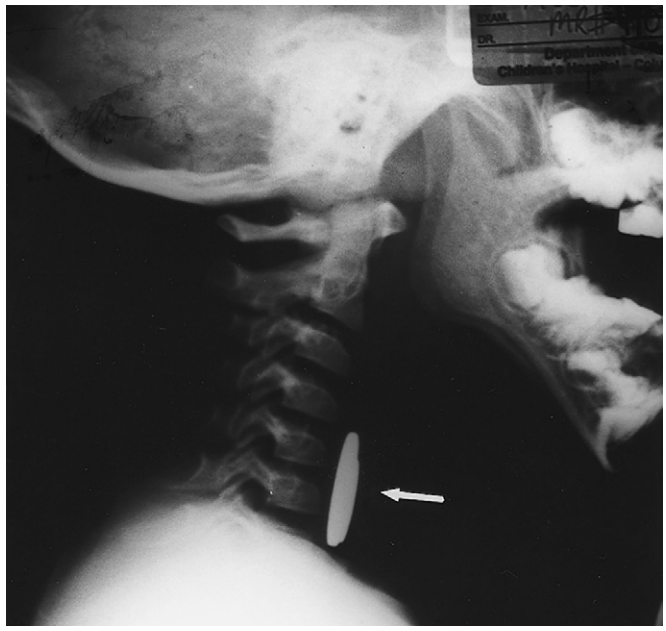


Figure 19-2 Lateral neck x-ray film of a child presenting with gagging and drooling, showing two coins *(arrow)* in the esophagus near the cricopharyngeal junction.

Sudden Sensorineural Hearing Loss

Although most types of hearing loss are nonurgent problems, sudden sensorineural hearing loss (SSNHL) deserves special note because it is considered "otologic emergency." Any patient complaining of sudden hearing loss requires prompt evaluation. An obvious cause such as cerumen impaction or middle ear fluid can be treated appropriately and routinely. If a cause is not identified, sudden sensorineural hearing loss should be suspected and prompt ENT referral arranged. SSNHL is thought to be secondary to vascular, thromboembolic, viral, or autoimmune causes. It may also be the result of ototoxicity. Without treatment, hearing returns in one third of patients, partial hearing returns in one third, and there is no improvement in the remaining third. Early intervention with oral corticosteroids (and possibly antivirals, directed at the herpesvirus [Awad et al., 2008]) appears to improve outcomes, although few controlled studies have been done. The Cochrane Collaboration believes that there is a lack of good-quality evidence to support the use of steroids and that more research is needed (Wei et al., 2006). Finally, for patients who do not respond to systemic steroids, controlled studies do support the use intratympanic infusion of corticosteroids, demonstrating improved outcomes (Xenellis et al., 2006). Intratympanic steroids are also appealing for patients with contraindications to oral steroids (e.g., "brittle diabetics")

Foreign Bodies

Swallowing or aspiration of objects is most common in children but also occurs in the adult population. These objects can become lodged anywhere in the upper aerodigestive tract.

Esophagus

The most common location for esophageal foreign bodies is at the level of the cricopharyngeal muscle. Other regions include the anatomic narrowings of the esophagus, such as the gastroesophageal junction, and the area of indentation of the esophagus by the left main stem bronchus and the arch of the aorta. Coins are by far the most common objects found in the esophagus in children. Chicken or fish bones are more common in adults.

The diagnosis of an esophageal foreign body is primarily based on the medical history and physical examination, with the aid of radiologic studies. Parents might witness ingestion of the foreign body and subsequent coughing, gagging, refusal to eat, or drooling. Often, however, the incident goes unwitnessed, and reliance on other diagnostic techniques is necessary.

Plain radiographs (including lateral films) are often diagnostic in pediatric patients because most esophageal foreign objects are radiopaque (Fig. 19-2). Other radiologic findings that can suggest a foreign body include increased soft tissue density in the prevertebral space, mediastinal widening, air-fluid levels in the esophagus, and paraesophageal air. Disk batteries *require a high index of suspicion* because they can cause significant tissue injury and lead to esophageal perforation *if they are not removed emergently*. They have a classic appearance when viewed laterally, approximating a dime resting on a nickel (similar to the appearance of Fig. 19-2).

If there is sufficient evidence of an esophageal foreign body, ENT consultation is indicated for rigid esophagoscopy and removal. When radiolucent objects have been ingested, contrast esophagography might be indicated, although esophagograms can give a false-negative result and can also complicate visualization during rigid esophagoscopy.

Airway

As with esophageal foreign bodies, airway foreign bodies are much more common in infants and young children. Many deaths from foreign-body aspiration occur in the home before medical intervention can be administered. The most

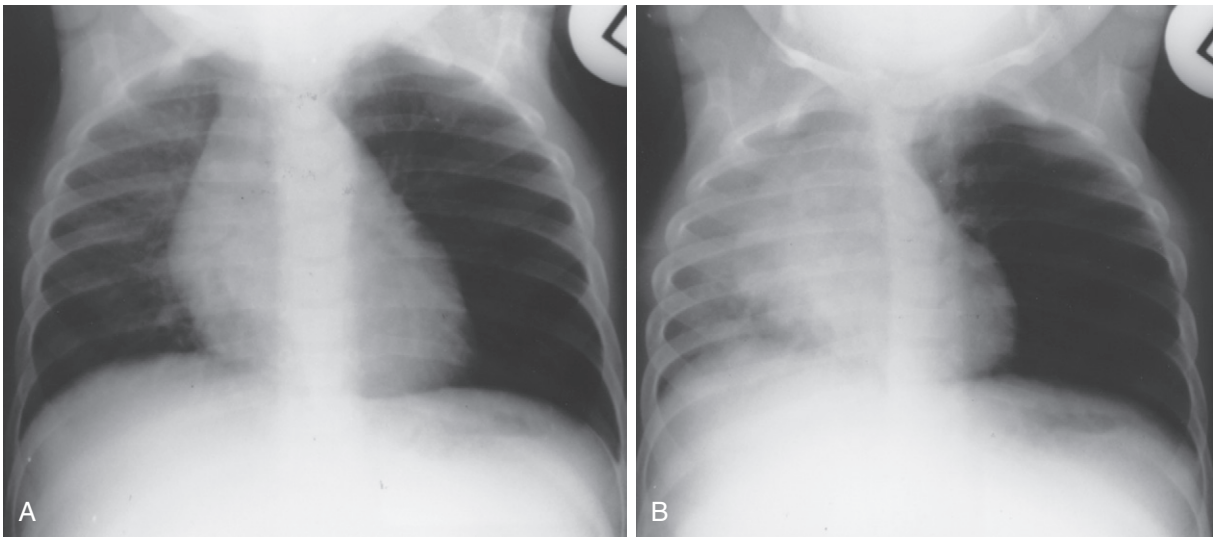


Figure 19-3 Inspiratory **(A)** and expiratory **(B)** radiographs of 8-year-old boy with left airway foreign body (peanut). No foreign object is visible on plain films, but the expiratory film shows overinflation of the left lung with mediastinal shift to the contralateral side.

frequently aspirated foreign bodies are foods, with nuts leading the list. Foreign bodies aspirated into the airway are usually found lodged in the bronchial tree but can also be found in the larynx or trachea. If the event is witnessed and results in complete airway obstruction, a Heimlich maneuver should be administered; however, the event is often not witnessed. Symptoms can include hoarseness, persistent cough, wheezing, or stridor if the foreign body is lodged in the trachea or larynx. Because the potential for morbidity and mortality is substantial, this condition requires urgent diagnosis and timely intervention to prevent catastrophe.

Any time a small child presents with wheezing or noisy breathing without a previous history of reactive airway disease, an airway foreign body should be included in the differential diagnosis. Typically, patients and parents recount a transient episode of coughing during eating that then subsided; the patient might even be symptom free for a time, then later have symptoms such as coughing or wheezing.

The most important diagnostic step for identifying a foreign body is a high index of suspicion. Careful auscultation of the lung fields is essential because subtle asymmetric differences may be found. Because most airway foreign bodies are radiolucent, chest radiographs can be normal, but abnormalities such as hyperinflation, atelectasis, or pneumonia can be present (Fig. 19-3). If plain radiographs are equivocal or normal and the patient is in stable condition, airway fluoroscopy can be helpful.

Consultation with a physician experienced in foreign body removal is required. Definitive treatment of airway foreign bodies is direct laryngoscopy and rigid bronchoscopy to indentify and remove the object.

Epistaxis

Although epistaxis is usually nothing more than a minor annoyance, some episodes are severe enough to require urgent medical attention and intervention. In rare cases, epistaxis can also be a life-threatening emergency.

Predisposing factors for epistaxis include trauma (facial trauma or self-inflicted digital trauma), dry weather, hypertension, bleeding dyscrasias (factor deficiencies, hereditary hemorrhagic telangiectasia, lymphoproliferative disorders), anticoagulation therapy (acetylsalicylic acid, heparin, warfarin), and intranasal tumors. Special consideration should be given to adolescent boys with recurrent epistaxis and nasal obstruction, because these symptoms might be the result of a juvenile nasopharyngeal angiofibroma. These are benign but locally aggressive tumors. All these potential risk factors should be considered because they must be addressed to treat the patient appropriately.

Epistaxis is classified according to its location. Bleeding from the anterior nasal cavity is most common and usually originates from a rich plexus of vessels at the anterior septum called *Kiesselbach's plexus* (Fig. 19-4). Bleeding from this location, although troublesome, is less likely to be severe and is usually easier to control than posterior epistaxis. Posterior epistaxis originates from the posterior two thirds of the nasal cavity and can be quite severe and much more difficult to control.

Initial management of epistaxis includes assessment and stabilization of vital signs. Rarely, severe bleeding can lead to airway compromise or hemodynamic compromise, or both, especially in patients with underlying cardiopulmonary dysfunction. The airway should be assessed and stabilized, urgently if necessary. Hypertension, if severe, should be controlled, with care taken to avoid subsequent *hypotension*. Hematologic studies, including complete blood count (CBC), prothrombin time, and partial thromboplastin time, should be ordered. Intravenous access should be established, allowing administration of fluids as well as IV medications, if necessary during treatment.

Treatment

Effective treatment requires adequate visualization and patient cooperation. The level of intervention by the primary care physician depends on level of experience, comfort level, and availability of appropriate supplies and equipment. ENT consultation should be considered when any of these prerequisites cannot be met. The patient should be reassured and

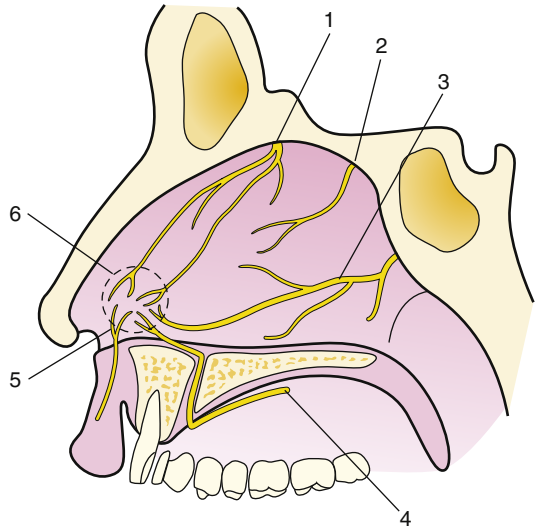


Figure 19-4 Kiesselbach's plexus. *1* and *2*, Anterior and posterior ethmoid arteries; *3*, septal branch of the sphenopalatine; *4*, greater palatine; *5*, branch from superior labial; *6*, Kiesselbach's plexus. *(From Colman BH, Hall IS (eds): Hall and Colman's Diseases of the Ear, Nose and Throat, 15th ed. Philadelphia, Churchill Livingstone, 2000, p 82.)*

given an explanation of the planned treatment. This results in better cooperation and decreases patient anxiety, improving treatment success. A bright headlight, nasal speculum, large nasal (Frazier tip) suction, and bayonet forceps are required. If the patient is monitored and stable, a small dose of IV narcotics titrated for analgesia and anxiolytic effect may be given. Extreme caution should be taken not to oversedate the patient. Also, instrumentation of the nose can lead to a significant vasovagal response that may be accentuated in a hypovolemic patient given narcotics. It is advised to err on the side of caution with regard to narcotic medication both during and after treatment.

All clots should be suctioned. They can be quite tenacious and require forceps for removal. The nasal cavity should next be topically anesthetized and decongested (a mixture of 4% lidocaine and phenylephrine works well). If a bleeding site is easily identified, it may be cauterized with a silver nitrate stick.

Anterior Packing

If suction, decongestion, and cautery do not stop the bleeding and the site is still thought to be anterior, an anterior nasal pack should be placed. This can be done with 0.5-inch petroleum gauze coated in antibiotic ointment. Alternatively, a variety of preformed packs are available. Merocel nasal packs or prepackaged inflatable packs are usually readily available and quite effective. Remembering that the nasal cavity extends *posteriorly* from the nostril and not *superiorly* facilitates placement. The pack should be coated in antibiotic ointment before placement.

Placement of the pack can be quite uncomfortable for the patient. Discomfort can be minimized by ensuring optimal decongestion and topical anesthesia of the nasal cavity. If using a Merocel pack, it is sometimes helpful to hydrate and expand the pack *before* placing it. This can be done with sterile saline or phenylephrine. Although the pack appears quite large after it is expanded, it decompresses readily and slides in easily once it is covered in antibiotic ointment. The entire length of the pack needs to be grasped with the bayonet forceps if this technique is employed. Preexpansion of the pack also minimizes further abrasions and bleeding that can occur with placement of the firm pack. This technique is especially advantageous if a septal deviation or spur exists on the bleeding side. Regardless of what type of pack is used, care must be taken not to distort or overstretch the nasal ala (nostril) once the pack is in place. This causes significant discomfort and can result in necrosis of the nostril.

After the pack is in place, the patient should be observed for further bleeding. If the patient remains stable, has no significant comorbid conditions, and has only a unilateral anterior pack, the patient may be discharged with mild narcotic pain medications and antibiotics for prophylaxis against toxic shock syndrome and sinusitis. The pack should be removed 2 to 5 days later, with instructions to use nasal saline and nasal ointment liberally for the next 2 weeks. Recurrent bleeding should prompt an ENT consultation.

Posterior Packing

If the bleeding is not controlled with an anterior pack, the origin of the bleeding may be posterior, requiring an *anterior-posterior pack.* An anterior-posterior pack is inserted similar to a nasogastric tube, then inflated with the *minimum amount* of saline to stop the bleeding. The recommended maximum amount of inflation should not be surpassed.

If a preformed anterior-posterior pack is not available or not effective, a traditional anterior-posterior pack may be placed. The posterior pack is necessary essentially to seal off the posterior nasal cavity and provide a buttress to prevent the anterior pack from slipping posteriorly. A Foley catheter and balloon is slid into the nasal cavity and serves as the posterior pack and buttress. Once the Foley catheter is in place, it is inflated with 5 to 10 mL of saline (or air) and pulled snugly to seal the posterior nasal choana. A large anterior pack is then placed, which should quickly control the bleeding.

Placement of a posterior pack requires experience, and even if it is successfully done, ENT consultation is indicated to assist with further bleeding, pack removal, and to monitor for pack complications. Anterior-posterior packs are associated with significant patient discomfort and potential complications during and after insertion. Local complications, including necrosis of the alae, septum, and palate, can occur, and close observation is required to prevent them. Hypoxemia can also result. Supplemental oxygen and monitoring of oxygen saturation is indicated. Patients with preexisting cardiopulmonary disease require closer observation, often in the ICU. The patient might require judicious doses of narcotics for pain and antibiotics for infection prophylaxis.

Other Techniques

Rarely, epistaxis cannot be controlled with packing, requiring further intervention. This can include intraoperative endoscopic cautery, endoscopic or open arterial ligation, or angiography with selective embolization of the offending vessel. There are advantages and disadvantages to each of these techniques. In general, the surgical techniques are usually favored over embolization if there are no significant contraindications to surgery. All the surgical techniques have very high success rates. Embolization is also effective when performed by an experienced invasive radiologist, but it does

carry the relatively low risk of inadvertent embolization of the internal carotid artery system and subsequent ischemic cerebral injury, which can be devastating.

Head and Neck Trauma and Respiratory Embarrassment

Key Points

- The ABCs of trauma include *a*irway, *b*reathing, *c*irculation, and *c*ervical spine clearance.
- Laryngeal and pharyngoesophageal injuries must be suspected in blunt or penetrating neck injuries.
- Intravenous access should be established and volume replacement initiated quickly.
- Isolated facial injuries can result in significant bleeding and can be associated with orbital or central nervous system injury.
- Overlooked facial fractures can result in long-term functional and cosmetic defects.

The ABCs of trauma should be remembered when treating a patient with cervicofacial trauma. This includes evaluation and treatment of *a*irway, *b*reathing, *c*irculation, and *c*ervical spine. The most pressing issue after significant face, head, or neck trauma is the potential for respiratory compromise, secondary to several causes. Altered mental status can lead to aspiration of blood or secretions with or without central hypoventilation. Comminuted facial fractures (midface or mandibular) can distort the oral and pharyngeal airway sufficiently to cause obstruction. An undetected expanding pharyngeal or neck hematoma can cause airway obstruction by extrinsic compression of the trachea or pharynx. Blunt or penetrating neck injuries can cause laryngeal fracture, bleeding, or hematoma, leading to critical airway obstruction.

Potential airway obstruction must be addressed quickly because complete obstruction can progress rapidly. The diagnosis is clinical because hypoxemia and carbon dioxide retention are late signs. Extensive facial edema or ecchymosis should arouse concern for facial fracture. A muffled voice can be the result of expanding hematoma. Laryngeal or tracheal injury should be suspected if the patient has a change in the voice, hemoptysis, subcutaneous emphysema, or stridor.

Stabilization of a compromised airway should be accomplished as soon as possible. Endotracheal intubation may be attempted, with plans for emergent cricothyrotomy as necessary. If time permits, the on-call anesthesiologist, trauma surgeon, or otorhinolaryngologist should be consulted to assist in airway management. Blind intubation (especially nasotracheal) or insertion of a laryngeal mask airway (LMA) is not recommended because this further compromises the already tenuous airway if intubation is unsuccessful. Although tracheotomy is the preferred procedure when endotracheal intubation is impossible or contraindicated, cricothyrotomy is also acceptable and can be lifesaving.

There is potential for significant blood loss after severe head and neck trauma. Intravenous access should be established and volume replacement initiated quickly. Bleeding from facial wounds can be controlled with direct pressure and suture ligation of arterial bleeding. Management of epistaxis is discussed earlier. Bleeding from the neck, or evidence of expanding hematoma, implies a major vessel injury and requires immediate operative exploration by a trauma surgeon, vascular surgeon, or otorhinolaryngologist.

Unrecognized pharyngeal and esophageal injury can result in life-threatening infection. These injuries might not be obvious on initial evaluation and require a very high index of suspicion. Contrast studies and endoscopy are usually required to confirm the diagnosis. Treatment can include repair of the injury or external drainage to allow healing.

Isolated facial injuries are rarely life threatening but still can result in significant bleeding and, rarely, airway compromise and permanent disability. Significant facial trauma should be evaluated in the ED. The potential for intracranial and cervical spine injuries should be considered when major facial injuries are present. Trauma of the periorbital region requires ophthalmologic evaluation. All lacerations should be inspected, cleaned, and sutured. Antibiotics should be used if contamination is likely.

Deeper injuries can result in facial nerve transection. If facial nerve weakness is detected, plastic surgery or ENT consultation is necessary for expedient nerve exploration and repair. The parotid salivary duct can also be injured and requires repair over a stent. Facial fractures should be evaluated with CT (both axial and coronal images). Possible mandibular fractures should be evaluated with plain x-ray films, including panographic (Panorex) films. Overlooked and untreated facial fractures can result in significant long-term functional and cosmetic deficits. Oral surgery consultation is sometimes required, especially with injury to the teeth or altered dental occlusion.

The Ear

Key Points

- In smokers with otalgia, the clinician should suspect laryngopharyngeal carcinoma.
- A white mass behind the tympanic membrane might be a cholesteatoma.
- Pneumatic otoscopy is most useful in confirming middle ear fluid.
- Tympanometry and acoustic reflex testing are complementary to audiometry in determining the location of hearing loss.
- A neoplastic process should be considered when a presumed infection does not respond to usual medical treatment.

Physical Examination

See the discussion online at www.expertconsult.com.

Otalgia

While the vast majority of patients with otalgia have an otologic cause, the clinician must recognize that otalgia may be *referred*. Sensory innervation of the ear includes cranial nerves V, VII, IX, and X, and therefore disorders of structures with similar innervation can cause otalgia. It is imperative that the physician not simply attribute otalgia to an ear infection unless the physical examination supports this diagnosis. Otalgia can result from dysfunction of the nose, sinuses, oral cavity, pharynx, larynx, dentition, temporomandibular joints, and salivary glands. These structures

Ear: Physical Examination

The typical symptoms associated with an otologic problem include hearing loss, ear pain (otalgia), tinnitus, vertigo, and drainage (otorrhea). The clinician should ascertain the timing, severity, duration, associated symptoms, and modifying factors.

Examination of the ear begins with inspection of the external ear, which includes the auricle and ear canal (Lucente and Har-El, 1999) **(eFig. 19-1).** In children, overly prominent ears, or *macrotia*, can result in significant teasing and may be surgically correctible with an otoplasty. The skin of the auricle should be carefully inspected for signs of infection, actinic changes, obvious lesions, or trauma. The ear canal is best inspected with an otoscope using the largest speculum that easily fits in the canal. The canal should have a small amount of brownish cerumen that need not be removed unless causing symptoms or obscuring the view of the entire tympanic membrane. The canal should be inspected for ulcerations, masses, erythema, drainage, or foreign bodies. Some patients have bilateral smooth, raised areas in the canal called *bony exostoses*. These are of no consequence unless they obstruct the ear canal. They are common in cold-water swimmers or surfers.

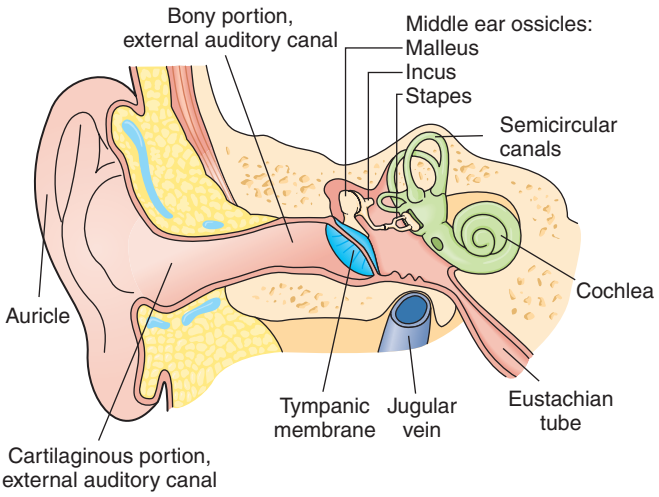
The tympanic membrane and middle ear can also be assessed with the otoscope **(eFig. 19-2).** The middle ear includes the tympanic membrane and the ossicles (malleus, incus, and stapes). The tympanic membrane should appear translucent, with fine vascularity along the part of the malleus that is attached to the membrane. A normal light reflex may be noted at the anterior-inferior portion of the tympanic membrane. The tympanic membrane should be inspected for dullness, erythema, retraction, bulging, perforation, or drainage. An attempt should also be made to look for any masses that might be visible behind the tympanic membrane. Whitish areas *on* the tympanic membrane (*tympanosclerosis* or *myringosclerosis*) are usually secondary to previous trauma or infection and are rarely of clinical significance. However, a white mass that is *behind the* tympanic membrane may be a cholesteatoma.

Clinicians should be comfortable with *pneumatic otoscopy*, which should be performed if middle ear effusion is suspected. A slightly larger otoscope tip or special soft tip is used so that the canal will be completely sealed. A small amount of positive and negative pressure is applied with either a small bulb attached to the otoscope handle or a small silicone elastic (Silastic) tube (a tourniquet tube) placed in the examiner's mouth. If the middle ear pressure is normal, the tympanic membrane moves equally back and forth. In cases of middle ear fluid or perforation, the tympanic membrane is sluggish or immobile.

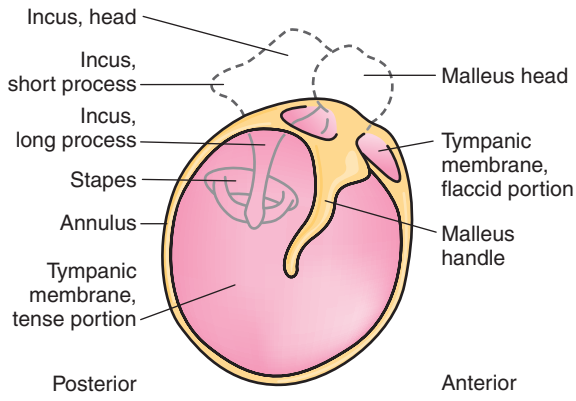
Although the same principles of an adult examination apply, otologic examination of young children and infants may be compromised by small ear canals and poor cooperation. Because crying may result in tympanic membrane erythema and obscure the diagnosis of acute otitis media, the otologic examination should be done first. Assistance from the parent or medical assistant is often required. Obstructing cerumen should be removed if the tympanic membrane is not well visualized. Pneumatic otoscopy may be necessary to confirm the presence of middle ear fluid.

A tuning fork examination using Weber's and Rinne's tests should be done if hearing loss is suspected. These tests help distinguish *conductive* (middle ear) and *sensorineural* (inner ear) loss. A 512-Hz tuning fork usually suffices, with additional tuning forks (256 and 1024 Hz) increasing sensitivity and specificity. *Weber's test* is performed by gently striking the tuning fork and placing it against the patient's forehead or upper incisors. If the patient hears the tone louder in one ear (referred to as *lateralizing* to that ear), there may be either a conductive hearing loss in the same ear or a sensorineural hearing loss in the contralateral ear. *Rinne's test* compares air conduction to bone conduction and complements Weber's test to help determine which ear is affected. Rinne's test is performed by gently striking the tuning fork and alternating placement against the mastoid tip (bone conduction) with placement directly in front of the ear (air conduction). With normal hearing or a sensorineural hearing loss, the patient will hear the tone louder in front of the ear (positive Rinne's test). In an ear with a conductive hearing loss, the patient will hear the tone louder over the mastoid tip (negative Rinne's test).

A *Dix-Hallpike test* is a specific examination technique to evaluate for benign paroxysmal positional vertigo (BPPV) **(eFig. 19-3).** The test is done by having the patient sit on an



eFigure 19-1 Coronal section of the ear. *(From Lucente FE, Har-El G. Essentials of Otolaryngology, p 2. Philadelphia, Lippincott–Williams & Wilkins, 1999.)*



eFigure 19-2 Tympanic membrane and middle ear ossicles. *(From Lucente FE, Har-El G. Essentials of Otolaryngology, p 3. Philadelphia, Lippincott–Williams & Wilkins, 1999.)*

examination table with legs outstretched. The patient's head is turned to one side and the patient is assisted back into a supine position, with the head still turned and allowed to gently extend past the edge of the table. The patient is asked to direct the eyes downward toward the floor and left open. The patient is asked to report any feeling of disequilibrium while the examiner inspects the eyes for any sign of nystagmus. After performing the test on one side, it is repeated on the other. A positive Hallpike test includes all the following: brief latency before subjective vertigo, rotatory nystagmus with the fast component directed toward the floor, spontaneous cessation of symptoms and nystagmus after several seconds (usually 15-30), and fatigability on repeat test. Some of these findings may be absent in BPPV. However, lack of spontaneous cessation or fatigability of vertigo or nystagmus is concerning for central nervous system (CNS) dysfunction.

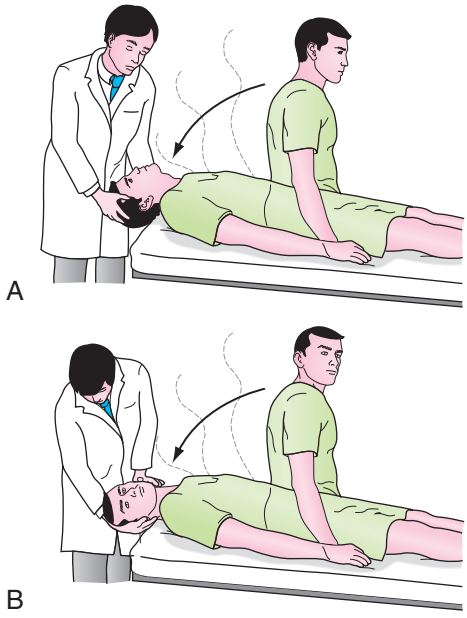
Although not technically part of the otologic examination, a complete neurologic examination should always be done, especially in a patient with a complaint of disequilibrium. This should include assessment of cranial nerve function, cerebellar testing, and Romberg's test. In addition, a cardiovascular assessment, including orthostatic vital signs, can also provide clues to a nonotologic cause of dizziness.

Audiometry and Tympanometry

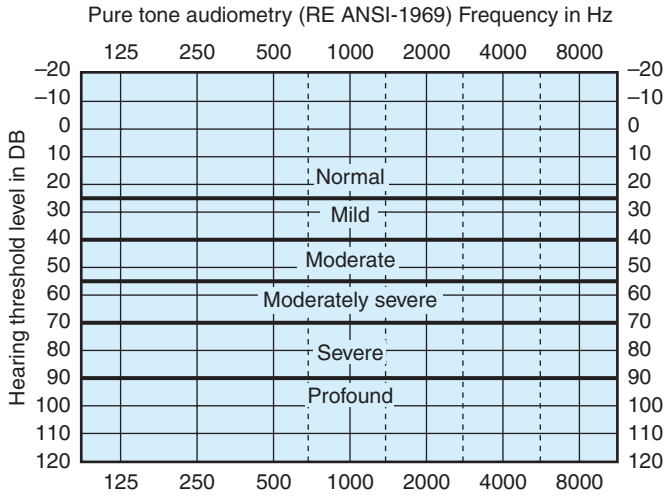
Audiometry and tympanometry are objective tests of the auditory system. They are essential and often complementary parts of a complete otologic evaluation. Audiometry consists of two parts: pure-tone and speech audiometry **(eFigs. 19-4 to 19-7)**

Pure-tone audiometry tests the patient's awareness of tones of various pitches that are presented to each ear individually. The measure of loudness is based on the decibel scale, which is logarithmic. Normal hearing is defined as hearing each tone (ranging from 250-4000 Hz) at levels at or below 20 dB. The tones are presented in each ear through air conduction via an earphone or through bone conduction via a bone-conduction vibrator placed at the mastoid tip. Presenting the tones in these two ways allows the conductive components of hearing (i.e., external and middle ear structures) and the sensorineural components (i.e., inner ear structures and cranial nerve VIII) to be assessed independently in each ear. The external ear canal should be cleared of cerumen before testing. Pure-tone audiometry allows an audiogram to be created, which graphically depicts both conductive and sensorineural hearing in both ears.

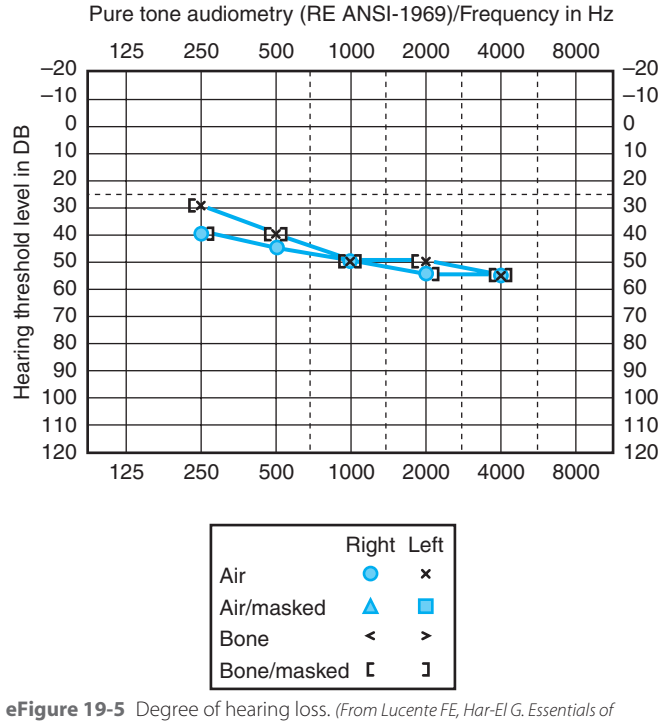
Speech audiometry is a test of inner ear function. It is performed by asking the patient to repeat single-syllable words (presented at an adequate volume to each ear individually).



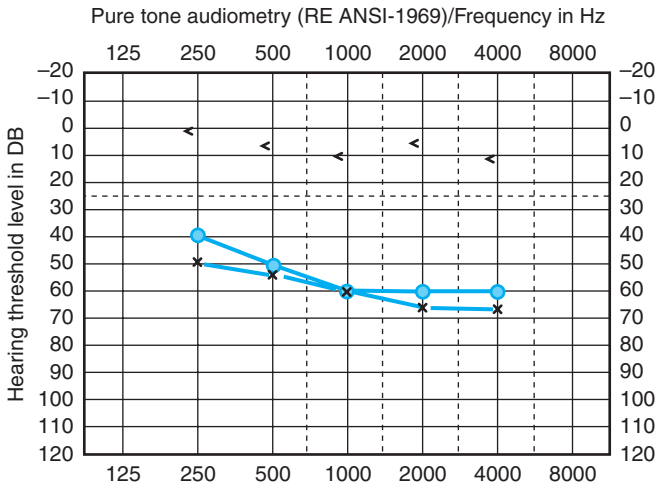
eFigure 19-3 Dix-Hallpike maneuver. *(From Andreoli TE. Cecil Essentials of Medicine, p 926. Philadelphia, Saunders, 1997.)*



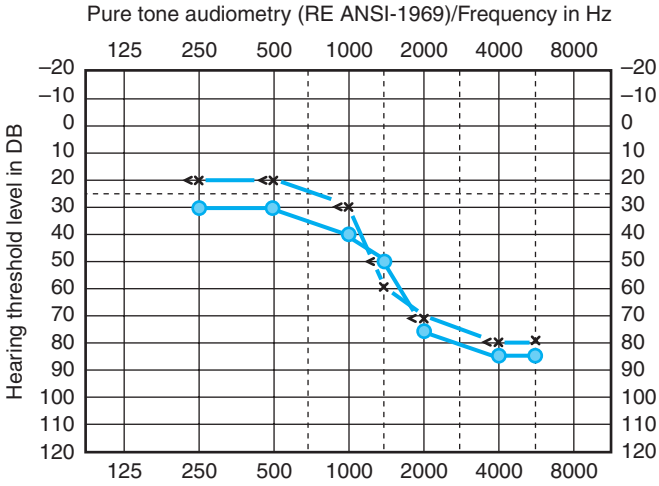
eFigure 19-4 Sensorineural hearing loss. *(From Lucente FE, Har-El G. Essentials of Otolaryngology. Philadelphia, Lippincott–Williams & Wilkins, 1999, p 73.)*



eFigure 19-5 Degree of hearing loss. *(From Lucente FE, Har-El G. Essentials of Otolaryngology, p 74. Philadelphia, Lippincott–Williams & Wilkins, 1999.)*



eFigure 19-6 Conductive hearing loss. *(From Lucente FE, Har-El G. Essentials of Otolaryngology, p 75. Philadelphia, Lippincott–Williams & Wilkins, 1999.)*



eFigure 19-7 High-frequency hearing loss. *(From Lucente FE, Har-El G. Essentials of Otolaryngology, p 76. Philadelphia, Lippincott–Williams & Wilkins, 1999.)*

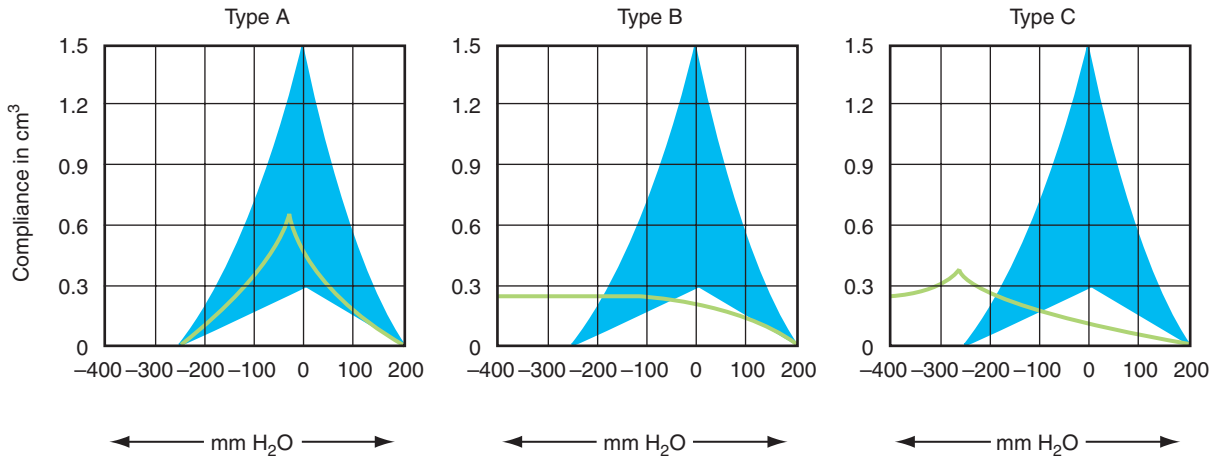
A score, called the *discrimination score,* is given to each ear. A discrimination score of less than 90% is considered abnormal. Asymmetry of the discrimination scores is suspicious of unilateral inner ear pathology (acoustic neuroma). Audiometry is an excellent, reliable test in cooperative patients. It has limitations for those who cannot (or will not) follow instructions (e.g., infants, young children, mentally impaired patients, malingerers). An abnormal audiogram should always be confirmed by tuning fork examination. An accurate audiogram clearly depicts hearing acuity and, most importantly, shows if any variation exists between the two ears. Special audiometric tests are done in cases of suspected malingering and are quite helpful in these cases.

Impedance audiometry or *tympanometry* measures resistance and compliance of the middle ear structures. It is also an indirect test of eustachian tube function. It is done by placing a pressure probe with an airtight seal in the meatus of the ear. The probe is capable of producing a small amount of positive and negative pressure while delivering a stimulus and measuring small changes in volume, thereby measuring compliance of the tympanic membrane **(eFig. 19-8).** The result produces a *tympanogram,* the configuration of which can be compared to standard patterns.

If eustachian tube and middle ear function are normal, the normal pressure and compliance yields a *type A* tympanogram. If there is negative middle ear pressure or middle ear fluid, abnormal tympanic membrane compliance and middle ear pressure yields an abnormal *type C* or *type B* (flat) tympanogram, respectively. A tympanic membrane perforation causes a type B (flat) tympanogram, but with a higher measured volume, because the middle ear is in communication with the external ear canal.

Special Tests

Auditory brainstem response (ABR) is a test of nerve conduction of the auditory nerve (cochlear division of cranial nerve VIII) in response to auditory stimuli. A normal ABR produces a characteristic waveform reflecting normal neural transmission from the eighth nerve to the brain. It can be done on conscious or unconscious patients. Although its evaluation



eFigure 19-8 Type A is a normal tympanogram. If there is negative middle ear pressure or middle ear fluid, tympanic membrane mobility will be impaired. Type B tympanogram indicates middle ear fluid, and type C indicates negative middle ear pressure. *(From Lucente FE, Har-El G. Essentials of Otolaryngology, p 77. Philadelphia, Lippincott–Williams & Wilkins, 1999.)*

is not as precise as with an audiogram, ABR is a completely objective test and does not require patient cooperation. It is most often used to test hearing in patients who cannot be tested with routine audiometry, including infants or young children who have failed infant hearing screening or OAE testing or those who are suspected of or at risk for hearing loss. Because it requires no patient feedback, ABR can be done on unconscious patients and possible malingerers. It may be ordered as a screening test for some patients with relatively low suspicion of an acoustic neuroma or for those not able to undergo MRI.

Otoacoustic emission (OAE) is also an objective test that measures the cochlea's response to noise. When presented with certain acoustic stimuli, the hair cells within the cochlea produce a predictable noise that can be measured. Assuming there is no conductive hearing loss, lack of production of stimulated OAEs indicates cochlear dysfunction and probable hearing loss. The test provides accurate information regarding hearing. OAE has been recommended to screen all infants for hearing loss and has been implemented in most U.S. states. Abnormal OAE testing in a neonate requires further evaluation, including repeat testing, possible ABR, and ENT evaluation.

Electronystagmography (ENG) is a test of the vestibular (balance) portion of the inner ear that relies on the neural relationships between the cranial nerve innervating the ears (cranial nerve VIII) and those innervating the eyes (cranial nerves III, IV, and VI). Placement of electrodes around the eyes allows detection of nystagmus (both spontaneous and elicited) that might not be visible to the observer. Abnormal nystagmus indicates CNS or peripheral (labyrinthine) dysfunction, or both. The test can also be performed using special goggles and video assessment referred to as *videonystagmography* (VNG).

must be thoroughly assessed, especially if the examination of the ear appears normal. This is especially true in smokers, whose initial symptom of laryngopharyngeal carcinoma may be otalgia. Otolaryngologic referral for laryngoscopy may be indicated with suspected referred otalgia. (See **eBox 19-1** and **eBox 19-2** online at www.expertconsult.com for differential diagnosis of otalgia.)

Tumors of the Ear

Tumors of the ear are rare. Classification is based on their location (external ear, middle ear, inner ear). A tumor of the external or middle ear is often easily diagnosed by visualizing a lesion on otoscopy. In some cases, symptoms may mimic infection (pain, otorrhea). Tumors of the inner ear can manifest with symptoms of hearing loss, tinnitus, disequilibrium, or facial weakness. (**eBox 19-3** online lists tumors of the ear by location.)

Vertigo

Key Points

- The most common cause of persistent vertigo is a peripheral vestibular disorder (38% to 56%).
- Central vestibular causes of vertigo represent less than 10% of all causes.
- Head movement always worsens the feeling of true vertigo.
- Benign paroxysmal positional vertigo is the most common cause of peripheral vestibular vertigo and is twice as common in women as men.
- Pneumatic otoscopy can cause nystagmus and vertigo in the presence of a perilymph fistula.
- Tinnitus is primarily caused by sensorineural hearing loss but occasionally can be the symptom of a vascular abnormality, hypermetabolic state, medication, or intracranial mass.

The sense of balance or equilibrium occurs when there is normal and harmonious function of several systems and organs in the body. These include the musculoskeletal system, the cardiovascular system, the central nervous system, the eyes, and the ears. Abnormal function of any of these can result in the sensation of dizziness or disequilibrium. The term *vertigo* is reserved to describe a perceived sensation of motion, usually spinning, of the person relative to the environment, or vice versa. Causes of disequilibrium can be categorized into one of three groups: *peripheral* (inner ear or labyrinthine), *central nervous system* (CNS), or *systemic* (e.g., cardiovascular, metabolic). Although not pathognomonic of a labyrinthine disorder, true vertigo most often indicates aberrant function of the inner ear.

Because patients use "dizzy" to describe many sensations, the actual sensation is best clarified by a detailed history (Box 19-1). The major studies on the causes of persistent dizziness, from Drachman and Hart (1972) to Davis (1994), all describe four diagnostic categories: lightheadedness, presyncope, disequilibrium, and vertigo. The investigators all conclude that the most common cause of persistent dizziness is a peripheral vestibular disorder (38%-56% of cases) followed closely by a psychogenic disorder (6%-33%). In about 25% of patients, the complaint is the result of the combined effects of multiple sensory deficits, medications, or orthostasis, leading to complaints of presyncope, lightheadedness, or disequilibrium. Finally, central vestibular etiologies are unusual and represent less than 10% of all causes.

Box 19-1 History for "Dizziness"

- Description of the sensation (including associated symptoms)
- Onset (acute, gradual)
- Duration (date sensation was first noted, length of time it lasts)
- Intensity (how troubling is it?)
- Exacerbations (activities, positions, circumstances that worsen the situation)
- Remissions (activities, positions, circumstances that make sensation better)
- Medications (prescription, herbal, over the counter)
- Other medical problems (e.g., diabetes, hypertension, heart disease)
- Psychosocial (any stressors?)

A thorough medical history allows the physician to distinguish between true vertigo (a sensation of spinning) and other sensations, such as presyncope, lightheadedness, and unsteadiness. The physical examination and laboratory evaluation are guided by the accuracy of the history. A sensation of vertigo originates from within the vestibular system but can be either *peripheral* (vestibular nerve and inner ear) or *central* (cerebellum, brainstem, thalamus, and cortex).

Questions regarding hearing and neurologic deficits can help elicit which part of the vestibular system is involved (Wiet et al., 1999) (see **eTable 19-1** online). Peripheral vertigo tends to be episodic, whereas central vertigo is constant. Neurologic symptoms or loss of consciousness do not occur with peripheral vertigo but are possible with central vertigo. Nystagmus, which is labeled by the direction of the fast component, can be present in both types of vertigo and can be horizontal or rotary; vertical nystagmus occurs only in central vertigo.

The physical examination should include assessment of orthostatic blood pressure changes, a complete ocular examination, tuning fork tests (Weber's and Rinne's), pneumatic otoscopy (elicits vertigo in patients with perilymphatic fistula), balance tests (Romberg's), gait (including tandem walking), and cranial nerve evaluation. The Dix-Hallpike maneuver (see **eFig. 19-3** online) is especially helpful in diagnosing benign paroxysmal positional vertigo (BPPV). Head movement always worsens the feeling of true vertigo. If it does not, the dizziness can be attributed to a cause other than vestibular dysfunction.

Laboratory testing can include an audiogram if no specific cause of vertigo can be found after the medical history and physical examination. Electronystagmography (ENG) is an objective study of the vestibular system and can help localize a vestibular lesion. Electrodes placed about the eye sense the movements of nystagmus as either spontaneous or initiated by maneuvers such as caloric testing, positioning, optokinetics, and pendulum tracing. A brain MRI scan is indicated in patients with unilateral otologic symptoms and in those unresponsive to treatment. Blood tests, when necessary, can include CBC, rapid plasma reagin (RPR), vitamin B_{12} level, folate level, drug screens, and heavy metal testing when indicated.

eBox 19-1 Differential Diagnosis of Otogenic Otalgia

External Ear

Diffuse external otitis
Furunculosis
Necrotizing (malignant) external otitis
Impacted cerumen
Perichondritis
Foreign bodies
Bullous myringitis
Herpes zoster oticus
Neoplasm
Otomycosis
Trauma
Keratosis obturans
Cholesteatoma of ear canal

Middle Ear or Mastoid

Acute otitis media
Acute mastoiditis
Barotrauma
Acute eustachian tube obstruction
Chronic otitis media
Subperiosteal abscess
Petrositis
Extradural abscess
Lateral sinus thrombosis
Neoplasm
Trauma
Postsurgical otalgia

From Lucente FE, Har-El G. Essentials of Otolaryngology, p 871. Philadelphia, Lippincott–Williams & Wilkins, 1999.

eBox 19-2 Possible Sources of Referred Otalgia

Through the Fifth Cranial Nerve

Nose and sinuses
Nasopharynx
Teeth
Temporomandibular joint
Salivary glands
Trigeminal neuralgia
Sphenopalatine neuralgia

Through the Seventh Cranial Nerve

Geniculate neuralgia

Through the Ninth and Tenth Cranial Nerves

Pharynx
Tonsils
Larynx
Tongue
Esophagus
Elongated styloid process

From Lucente FE, Har-El G. Essentials of Otolaryngology, p 96. Philadelphia, Lippincott–Williams & Wilkins, 1999.

eBox 19-3 Tumors of the Ear

External Ear

Malignant
Basal cell carcinoma
Squamous cell carcinoma sarcoma
Melanoma
Benign
Osteoma/exostosis
Sebaceous cyst
Keloid
Chondrodermatitis nodularis chronica helicis
Keratosis obturans—canal cholesteatoma
Adenoma

Middle Ear

Squamous cell carcinoma
Glomus tumors
Histiocytosis
Rhabdomyosarcoma
Hemangioma

Inner Ear

Acoustic neuroma
Facial neuroma

eTable 19-1 Comparative Features of Peripheral Vestibular Disorders

Feature	BPPV	Meniere's Disease	Vestibular Neuronitis	Perilymphatic Fistula
Hearing loss	No	Low frequency initially	Ultrahigh frequency (>8 kHz) or none	Variable
Tinnitus	No	Yes	Predominantly no	Variable
Type of vertigo	Positional	Spontaneous	Spontaneous	Variable
Duration of vertigo	1-2 min	20 min to 24 hr	24-48 hr	Variable
Physical examination	Positive Dix-Hallpike test	Nystagmus present in acute stage only	Nystagmus present in acute stage only	66% of patients have positive fistula test
Treatment	Repositioning maneuvers; surgery if dizziness persists	Diuretic as maintenance therapy; diazepam (Valium) for intractable vertigo	Diazepam during acute stage; vestibular exercises 72 hr after disease onset	Bed rest initially; surgery if dizziness persists

From Wiet RJ, Dinces EA, Rezall A, et al. Common ear, nose and throat problems. AAFP Home Study Self Assessment. Monograph 242, p 28, 1999.
BPPV, Benign paroxysmal positional vertigo.

Meniere's Disease

Meniere's disease is characterized by episodic severe vertigo lasting hours, with associated symptoms of unilateral roaring tinnitus, fluctuating low-frequency hearing loss, and aural fullness. Typical onset is in the fifth decade of life. The cause is uncertain but is speculated to result from allergic, infectious, or autoimmune injury. The histopathologic finding includes *endolymphatic hydrops*, which is thought to be caused by either overproduction or underresorption of endolymph in the inner ear.

Meniere's disease is a clinical diagnosis mostly based on history. Testing may be obtained to support the diagnosis and rule out other disorders. Audiometry often demonstrates a low-frequency sensorineural hearing loss. An FTA-ABS test may be obtained to rule out syphilis. ENG may demonstrate a unilateral peripheral vestibular weakness on caloric testing. When the diagnosis is uncertain, a brain MRI with contrast is obtained to evaluate for a retrocochlear lesion. The differential diagnosis of Meniere's disease includes acute labyrinthitis, neurosyphilis, labyrinthine fistula, autoimmune inner ear disease, vestibular neuronitis, and migraine-associated vertigo.

Although Meniere's disease has a highly variable clinical course, most patients have long symptom-free periods between clusters of episodes. The majority of patients have an excellent prognosis, with symptoms burning out over several years. However, some patients have a disabling course with frequent and severe attacks. On average, a moderate sensorineural hearing loss is the end result. The disease may become bilateral in about 45% of cases (wide variability exists).

Treatment of an acute episode involves vestibular suppressants and antiemetics. As with any vestibular disorder, vestibular suppressants should be limited for use during acute symptoms because of their addictive potential and impairment of central compensation. Maintenance therapy includes reduction of sodium intake to less than 1500 mg/day and a diuretic such as hydrochlorothiazide-triamterene (Dyazide). Patients are also instructed to minimize caffeine, alcohol, nicotine, and chocolate. Allergy treatment may be helpful in some patients. Most patients have adequate control of symptoms with this regimen.

Patients who fail conservative measures may be candidates for procedures and surgical treatment. Gentamicin, a vestibulotoxic aminoglycoside antibiotic, may be injected transtympanically into the middle ear to permeate into the inner ear. Control of vertigo may result in 90%, but with a risk of hearing loss. Endolymphatic sac decompression or shunting through a mastoidectomy appears to benefit most patients with minimal risk to hearing. Although a generally accepted procedure, adequate studies are lacking on its effectiveness. More invasive interventions, including vestibular nerve section and labyrinthectomy, are reserved for patients with severe disease who do not respond to other measures (Sajjadi and Paparella, 2008).

KEY TREATMENT

Treatment of acute episodes of Meniere's disease involves vestibular suppressants and antiemetics (SOR: A).
Maintenance therapy includes reduction of sodium intake and use of a diuretic (SOR: A) (Thirlwall and Kundu, 2006).

Vestibular Neuronitis

Acute vertigo associated with nausea and vomiting (but without neurologic or audiologic symptoms) that originates in the vestibular nerve is known as *vestibular neuronitis*. Vestibular neuronitis can occur spontaneously or can follow viral illness. Nystagmus is horizontal, with the fast component beating away from the affected side. The symptoms peak within 24 hours and usually last 3 to 4 days. Autopsy studies have shown cell degeneration of one or more vestibular nerve trunks, a finding similar to that seen in Bell's palsy, which affects the facial nerve. A short course (3-5 days) of vestibular suppressants (e.g., meclizine [Antivert], diazepam [Valium]) and antiemetics such as promethazine (Phenergan) can provide symptomatic relief in the acute setting.

Distinguishing between vestibular neuronitis and bacterial labyrinthitis or labyrinthic ischemia is important. The diagnosis of *bacterial labyrinthitis is* based on hearing loss and otitis media or meningitis, and *labyrinthic ischemia* can be distinguished by hearing loss plus associated neurologic symptoms with a history of vascular disease.

Benign Paroxysmal Positional Vertigo

The most common cause of peripheral vestibular vertigo in adults is benign paroxysmal positional vertigo. BPPV occurs in all age groups but more often between ages 50 and 70. The incidence of BPPV is 11 to 64 per 100,000 persons per year and is twice as common in women as men (Froehling et al., 1991). It is caused when otoconia particles from the utricle or saccule lodge in the posterior semicircular canal and is also referred to as *canalithiasis*. This causes the canal to be a gravity-sensing organ, and head movement results in displacement of the otoconia and a sensation of vertigo.

The Dix-Hallpike maneuver reproduces this vertigo in the patient, resulting in nystagmus (see **eFig. 19-3**). Characteristics of the nystagmus of BPPV include fatigability, a latency period of 1 to 5 seconds before nystagmus begins after the head is moved, short duration of nystagmus from 5 to 30 seconds, and reversal of the nystagmus components when the patient is returned to the sitting position. If these characteristics are not present and treatment is not successful, BPPV cannot be diagnosed. In such a case, a CNS lesion is possible. BPPV can be the residual effect of Meniere's disease, ear surgery, vestibular neuronitis, or ischemia of the inner ear. Head trauma, even when it is minor, can lead to BPPV. However, one third of cases are idiopathic.

Treatment of BPPV consists of performing repositioning maneuvers with the goal of returning the otoconia to the utricle or saccule (Yacovino, 2009). In addition, the patient may attempt to reposition the otoconia at home by sitting upright on the bed and rapidly lying supine with the affected ear facing downward. After 1 minute, the head should be repositioned with the opposite ear facing downward, and the patient should wait another minute. The patient should then return slowly to the upright seated position and repeat this exercise four more times. The entire process is completed twice daily until the symptoms have abated.

An expert panel convened by the American Academy of Otolaryngology–Head and Neck Surgery Foundation recommended against "routinely treating BPPV with vestibular suppressant medication such as antihistamines or benzodiazepines" (Bhattacharyya et al., 2008). Because of

the variability of symptoms, the clinician must judge each case independently. Resolution occurs in a few weeks or months, and the condition is benign, although it can recur.

KEY TREATMENT

Unless the diagnosis of BPPV is uncertain, radiographic imaging and vestibular testing are not recommended (SOR: B).
Patients with BPPV should be treated with an otoconia-repositioning maneuver (SOR: B) (Bhattacharyya et al., 2008).
Residual dizziness after successful repositioning procedures abates within 3 months without further treatment (SOR: B) (Seok, 2008).

Perilymph Fistula

Rapid changes in air pressure (barotrauma), otologic surgery, violent nose blowing or sneezing, head trauma, or chronic ear disease may cause leakage of perilymph fluid from the inner ear into the middle ear and result in episodes of vertigo. Associated signs and symptoms are variable but can include a sudden pop in the ear followed by hearing loss, vertigo, and sometimes tinnitus. Diagnosis can be determined by a fistula test, in which negative and positive pressures are applied to the tympanic membrane using pneumatic otoscopy, causing nystagmus and vertigo.

Labyrinthitis

As with vestibular neuronitis, labyrinthitis causes sudden and severe vertigo. In contrast to vestibular neuronitis, the patient also has tinnitus and hearing loss. The hearing loss is sensorineural, is often severe, and can be permanent. Labyrinthitis is caused by inflammation within the inner ear. The cause is most often a viral infection but can be bacterial. Bacterial labyrinthitis usually results from extension of a bacterial otitis media into the inner ear. A noninfectious *serous* labyrinthitis can also occur after an episode of acute otitis media. Other, less common causes include treponemal infections (syphilis) and rickettsial infection (Lyme disease).

Symptomatic treatment of labyrinthitis is similar to that for vestibular neuronitis. Antibiotics are recommended if a bacterial cause is suspected. As with acute otitis media, bacterial labyrinthitis can, in rare cases, lead to meningitis. Few other conditions cause the constellation of hearing loss, tinnitus, and vertigo, but cerebrovascular ischemia, meningitis, brain abscess, and encephalitis should all be considered. Although the vertigo should resolve over days to weeks, hearing loss and tinnitus can persist.

Drugs known to be ototoxic can cause acute onset of hearing loss and disequilibrium, although this is not true labyrinthitis. These drugs include salicylates, aminoglycosides, loop diuretics, and various chemotherapeutic agents. This cause should be considered in patients who complain of hearing loss or dizziness while taking these medications.

Tinnitus

Tinnitus is a term used to describe an internal noise perceived by the patient. It is usually, but not always, indicative of an otologic problem. Tinnitus is most often subjective, that is, heard only by the patient. However, it can be objective and heard by the patient and the examiner. In most cases, tinnitus is secondary to bilateral sensorineural hearing loss and requires no further evaluation. In rare cases, tinnitus can be a symptom of a vascular abnormality (aneurysm or arteriovenous malformation), hypermetabolic state, or intracranial mass that, if not evaluated, could result in delayed treatment. Middle ear and rarely external ear pathology can also cause tinnitus, as can numerous medications (Box 19-2). The patient's medications should be reviewed.

Evaluation of tinnitus begins with a complete medical history, including duration of symptoms, possible inciting event (e.g., acoustic trauma), and accompanying symptoms (e.g., vertigo, hearing loss, headache, vision changes). Specific questions regarding the tinnitus are critical: Is it unilateral or bilateral? What is the quality of the tinnitus (pitch, volume)? Does it sound like a heartbeat or rushing blood? Does it change? A complete ENT evaluation should be performed, and audiometry is mandatory.

In general, if the tinnitus is bilateral, not particularly intrusive, not pulsatile, and associated with symmetric hearing loss, it is likely secondary to the hearing loss itself. The hearing loss requires further evaluation with magnetic resonance imaging (MRI) with contrast if it is asymmetric.

In cases of *pulsatile* tinnitus with normal otoscopy, magnetic resonance angiography (MRA) is performed to evaluate for vascular abnormalities. If otoscopy identifies a retrotympanic mass, a temporal bone CT is obtained to evaluate for a vascular mass or abnormality. Blood tests can be performed to rule out anemia or hyperthyroidism, which can result in a hypermetabolic state and cause tinnitus secondary to increased blood flow near the cochlea. Auscultation of the neck, periauricular area, and chest may identify a bruit or murmur, indicating a need for a carotid duplex ultrasound

Box 19-2 Causes of Tinnitus

Subjective

Otologic: Presbycusis, noise-induced hearing loss, Meniere's disease, otosclerosis

Metabolic: Hyperthyroidism, hypothyroidism, hyperlipidemia, vitamin deficiency

Neurologic: Basilar skull fracture, whiplash injury, multiple sclerosis, meningitic effects

Pharmacologic: Aspirin, nonsteroidal anti-inflammatory drugs, aminoglycosides, tricyclic antidepressants, loop diuretics, heavy metals, oral contraceptives, caffeine, cocaine, marijuana

Dental: Temporomandibular joint syndrome

Psychologic: Depression, anxiety

Objective

Vascular abnormalities: Arteriovenous malformation, glomus tumors, stenotic carotid artery, vascular loops, persistent stapedial artery, dehiscent jugular bulb, hypertension

Tympanic muscle disorders: Palatomyoclonus, idiopathic stapedial muscle spasm

Patulous eustachian tube

Central nervous system anomalies: Congenital stenosis of the sylvian aqueduct, type 1 Arnold-Chiari malformation

From Lucente FE, Har-El G: Essentials of Otolaryngology. Philadelphia, Lippincott–Williams & Wilkins, 1999, p 110.

study or echocardiogram, respectively. Most cases of arterial pulsatile tinnitus are secondary to atherosclerotic carotid artery disease. Venous pulsatile tinnitus often improves with digital pressure over the internal jugular vein. Etiologies include idiopathic venous hum, a high-riding jugular bulb, or benign intracranial hypertension.

Effective treatment of tinnitus is difficult and usually requires various approaches. Finding and eliminating potential causes (especially pharmaceutical) is imperative. Patients should be counseled to avoid caffeine and nicotine. No single medicine has been proved effective in treating tinnitus. Antidepressants have shown promise, especially if depression coexists. Intravenous lidocaine eliminates tinnitus in some patients but is not practical and has obvious potential side effects. Various homeopathic treatments and nutritional supplements are effective in some cases, but most have not been evaluated in controlled studies. Hearing aids are beneficial in *masking* the tinnitus if hearing loss exists. Tinnitus maskers can be purchased that essentially drown out the tinnitus with various distracting noises. Biofeedback and a technique called *tinnitus retraining therapy* are helpful for some patients. These techniques can be learned through various publications or at a tinnitus treatment center. All patients with obtrusive tinnitus are encouraged to join the American Tinnitus Association, the largest tinnitus support group and an excellent source of reliable information.

Disorders of the External Ear

Otitis Externa

The most common cause of pain in the external ear is acute otitis externa. It affects 3% to 10% of the patient population. The pain is caused by inflammation and edema of the ear canal skin, which is normally adherent to the bone and cartilage of the auditory canal. The inflammatory reaction can be caused by bacteria, fungi, or contact dermatitis (see **eTable 19-2** online).

Cerumen protects the canal by forming an acidic coat that helps prevent infection. Factors that predispose to otitis externa include absence of cerumen, often from excessive cleaning by the patient; water, which macerates the skin of the auditory canal and raises the pH; and trauma to the skin of the auditory canal from foreign bodies or use of cotton swabs.

When a bacterial organism is suspected, treatment consists of cleaning the ear canal of any debris or drainage and then instilling antibiotic drops with or without steroids. Because the most common bacterial organisms in this infection are *Pseudomonas aeruginosa* and *Staphylococcus aureus,* drops containing ciprofloxacin or neomycin/polymyxin B are effective against these pathogens, combined with a steroid to decrease inflammation, pain, and pruritus (Ciprodex, Cortisporin, Coly-Mycin, Pediotic). A recent study found Ciprodex to be more effective against *P. aeruginosa* than neomycin/polymyxin B/hydrocortisone (Dohar et al., 2009).

The clinician must use judgment in assessing the severity of the infection and treat accordingly. If the infection spreads beyond the auditory canal, oral antimicrobials are indicated. If clinical improvement is not apparent after 48 hours, the patient needs to be reexamined for additional treatment or referral to an otorhinolaryngologist.

Fungal infections compose less than 10% of external otitis cases. The most common fungi are *Aspergillus niger* and *Candida* species and are more prevalent in tropical climates. Itching is a more common complaint than pain in fungal ear infections. Thorough cleaning of the ear canal is the primary duty of the physician in this infection. Drops that are effective include 2% acetic acid with or without a steroid. Clotrimazole drops or powder can also be used to treat fungal infections of the canal (van Bolen et al., 2003).

Approximately 90% of necrotizing (malignant) otitis externa is seen in immunocompromised patients such as diabetic patients, patients with acquired immunodeficiency syndrome (AIDS), and those receiving chemotherapy. Systemic antibiotics are mandatory in these cases. Antipseudomonal antimicrobials should be administered intravenously in the hospital setting, and surgical debridement is often necessary. Complications from necrotizing otitis externa include facial nerve palsy, mastoiditis, meningitis, and even death (Quick, 1999).

Other conditions that affect the external auditory canal include impacted cerumen, seborrheic dermatitis, psoriasis, contact dermatitis, and staphylococcal furunculosis. Symptoms and signs include pruritus, edema, scaling, crusting, oozing, and fissuring of the external auditory canal. Treatment of the underlying disease is the primary goal. Corticosteroid preparations are indicated for seborrheic dermatitis, psoriasis, and contact dermatitis. Oral antibiotics and sometimes incision and drainage are required for staphylococcal furunculosis.

KEY TREATMENT

Ciprofloxacin/dexamethasone 0.1% (Ciprodex Otic) applied ototopically in patients with acute otitis externa is more effective against *Pseudomonas aeruginosa* (most common isolate) than neomycin/polymyxin B/hydrocortisone (Cortisporin Otic) (SOR: A) (Dohar et al., 2009).

Using drops that have a steroid plus an antibiotic or acetic acid improves the cure rate compared to an antibiotic or acetic acid alone (SOR: A) (van Bolen et al., 2003).

Auricular Hematoma

Blunt auricular trauma, most commonly in wrestlers and boxers, may shear the perichondrium from the underlying cartilage, leading to a hematoma. The presence of a fluctuant swelling with loss of normal auricular landmarks helps to distinguish a hematoma from ecchymosis. If left untreated, an auricular hematoma may cause fibrosis and neocartilage formation, leading to a deformity of the auricle termed *cauliflower ear.* Therefore, treatment in a timely manner is recommended.

Although a Cochrane review could not define the best treatment for an acute auricular hematoma, a frequently successful treatment involves incision and drainage with dental rolls sutured to the anterior and posterior auricle (Fig. 19-5). Needle aspiration alone will often lead to recurrences. The bolster is usually left in place for 4 to 7 days, with the patient permitted to return to wrestling or boxing with headgear. Prophylactic antistaphylococcal antibiotics are given. For a long-standing hematoma or a cauliflower ear, debridement of fibrosis and cartilage is necessary (Jones and Mahendran, 2008).

eTable 19-2 Otorrhea from the External Ear Canal

Cause	Physiologic Process	Diagnostic Clue	Treatment
Cerumen	Accumulation of cerumen in external ear canal with possible infection	Impacted cerumen in external ear canal	Cerumen removal
Otitis externa	Inflammation of external ear canal, usually by *Pseudomonas aeruginosa*	Purulent drainage in external ear canal Edema or erythema of external ear canal Recent history of water in external ear canal History of diabetes mellitus History of canal trauma (with cotton swabs) Otalgia Polyp in external ear canal (see Box 19-5)	Otic antibiotic drops with or without oral antibiotics Aural cleaning Dry-ear precautions Consider ear wick if unable to visualize tympanic membrane
Foreign body	Foreign body impacted in external ear canal	Foreign body visualized in external ear canal History of inserting foreign body History of ear cleaning with cotton swabs Young person or person with mental retardation having had foreign body in nose	Removal of foreign body Otic antibiotic drops if evidence of infection or bleeding Referral to ENT if foreign body cannot be removed
Canal trauma	Trauma to skin of external ear canal caused by, e.g., cotton swab or bobby pin; possible head trauma	History of instrumentation such as ear cleaning Bloody otorrhea Laceration or bleeding visualized in external ear canal	Otic antibiotic drops Temporal bone CT if history of head trauma Complete audiologic evaluation Referral to ENT
Neoplasia	Neoplasms originating in or extending to external ear canal	Unilateral involvement Mass seen in external ear canal Polyp in external ear canal	Temporal bone CT Referral to ENT
Malignant otitis externa	Fulminant infection, almost always caused by *Pseudomonas* spp	Severe pain Patient has diabetes or is old Polyp in external ear canal	*Pseudomonas*-specific intravenous antibiotics Aural cleaning Temporal bone CT
Fungal otitis externa	Fungal infection	Visible hyphae Refractory to antibiotic therapy Patient has diabetes or is old or immunocompromised	Acetic acid otic drops Dry-ear precautions
Dermatosis	Allergic or irritant dermatosis	History of contact with known allergen or irritant Erythema and itching	Removal of causative agent Topical steroids Systemic steroids if severe Antibiotics if secondary infection is present
Psychogenic factors	Psychogenic condition	Continued reports of wet or crawling sensations in the external canal Irritated or possibly secondarily infected external canal Normal findings at evaluation for cause after patient has been treated for infection	Topical steroids Antibiotics if secondary infection is present Psychiatric referral

From Lucente FE, Har-El G. Essentials of Otolaryngology, p 99. Philadelphia, Lippincott–Williams & Wilkins, 1999.
CT, Computed tomography; *ENT,* otorhinolaryngologist (ears-nose-throat specialist).

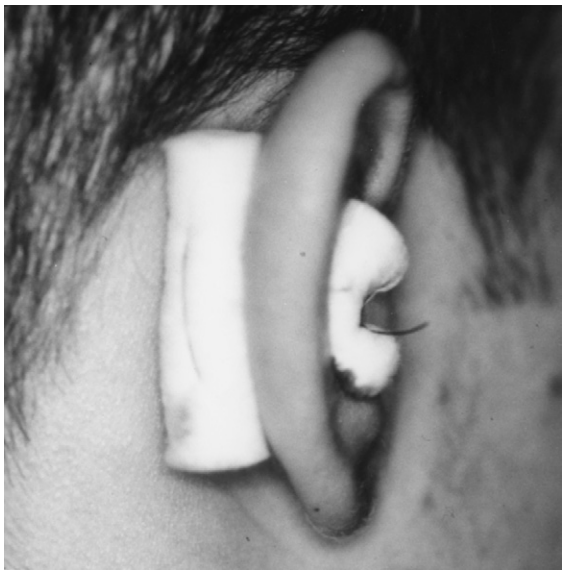


Figure 19-5 Photograph of 16-year-old boy who sustained auricular hematoma in wrestling match. He underwent incision and drainage with placement of dental rolls.

KEY TREATMENT

Successful treatment of auricular hematoma is incision and drainage with dental rolls sutured to the anterior and posterior auricle (SOR: B) (Jones and Mahendran, 2008).

External Auditory Canal Foreign Bodies

A common problem seen in family physicians' offices is a patient with an external auditory canal foreign body. A wide variety of objects can be found. In one study of 191 patients with aural foreign bodies, 27 different objects were discovered (Ansley and Cunningham, 1998). The most common were beads, plastic toys, pebbles, insects (especially cockroaches), popcorn kernels, earrings, paper, peas, cotton, pencil erasers, and seeds. When a patient presents with a chronic dry cough that has not responded to the usual measures, the physician should look for an aural foreign body (causing irritation of the ninth cranial nerve).

Removal of an external auditory canal foreign body is simplified if the object is in the lateral one third of the external auditory canal. Objects within the medial two-thirds pose a greater challenge. A variety of instruments can be used, depending on the object, including cerumen loops, alligator forceps, and otologic-tip suctions. Irrigation with body-temperature sterile water often dislodges the object. Hygroscopic objects such as vegetables, beans, and other food matter can swell and make the object even more impacted and should not be irrigated. Disk batteries should be removed immediately because of the possibility of liquefaction necrosis of the external auditory canal. Aural irrigation is contraindicated because wetting of the battery leads to leakage of electrolyte solution.

Smooth, round objects pose a difficult problem because, in trying to remove them, they are often pushed farther into the canal. Aural irrigation or even cyanoacrylate glue on the tip of a straightened paper clip is effective in removing objects that are difficult to retrieve. Methods to remove cockroaches or other insects include microscope immersion oil, mineral oil, or lidocaine. The effect of mineral oil or microscope immersion oil is to drown the insect, whereas lidocaine tends to make cockroaches crawl rapidly out of the canal (Bressler and Shelton, 1993). Otomicroscopy is often required for safe removal.

Depending on their age, fewer than 35% of patients should require anesthesia. The younger the patient, the more likely anesthesia will be required. Objects with sharp edges are best removed with an operating microscope with the patient under general anesthesia. Complications of foreign body removal include canal wall trauma and tympanic membrane perforation. Immobilization of the patient is the key to successful removal of aural foreign bodies, and at least two assistants are necessary.

Cerumen

Glandular secretions from the outer one third of the external auditory canal and desquamated epithelium combine to form cerumen. Cerumen is necessary to provide a hydrophobic and acidic environment to protect the underlying external ear canal epithelium and prevent infection. The external auditory canal is self-cleaning, with cerumen slowly pushed laterally to the external meatus.

Cerumen impaction is the symptomatic accumulation of cerumen in the external canal or an accumulation that prevents a needed assessment of the ear. Complete occlusion is not necessary. Symptoms may include hearing loss, tinnitus, pruritus, fullness, otalgia, cough, odor, and dizziness. Impaction often results from instrumentation with cotton-tipped applicators, which should be discouraged. Elderly patients with changes to external canal epithelium, patients with external canal abnormalities (e.g., osteomas, exostoses, stenosis), and users of hearing aids and earplugs are also at risk for impaction. Excessive cerumen production as a primary problem is relatively rare.

In most people, cleaning the external meatus with a finger in a washcloth while bathing is sufficient to maintain the ear canals. Treatment of cerumen impaction by the clinician may involve ceruminolytic agents, irrigation, or manual removal. Ceruminolytic agents include water-based, oil-based, and non-water-, non-oil-based solutions. A Cochrane review found that any type of ear drop (including water and saline) is more effective than no treatment, but study quality was lacking. Office irrigations may be performed using a large syringe with a large angiocatheter tip. The type of irrigant solution used is probably not critical, although a tepid or warm temperature is important to prevent the patient from becoming vertiginous from a labyrinthine caloric response. Instilling a ceruminolytic 15 minutes before irrigation may improve the success rate. Irrigations should not be performed in those with tympanic membrane perforations or previous ear surgery. Of note, irrigation with tap water has been implicated as a causative factor in malignant otitis externa. Therefore, instilling an acidifying ear drop after irrigation in diabetic patients is recommended. Manual removal requires knowledge of ear anatomy and special care to avoid trauma. A handheld otoscope with a curette and other instruments may be used. Otolaryngologists will often use binocular microscopy to aid with visualization. Those patients inquiring about ear

candling should be informed that it has not been shown to be effective and presents a risk of thermal injury to the ear (Burton and Doree, 2008).

Frostbite

The ears, nose, and cheeks, in that order, are most at risk for frostbite. Exposure to subfreezing temperature is the main risk factor, but wind chill also greatly affects heat loss from the skin by convection. Protective clothing greatly diminishes the risk.

There are three grades: grade I frostbite, in which the skin is erythematous and edematous; grade II frostbite, in which the skin blisters and forms bullae; and grade III frostbite, which results in local necrosis of the dermis over 1 to 2 weeks. To assess the severity of frostbite, the physician must examine the tissue from several hours up to 2 days after the typical skin blanching occurs.

Treatment consists of quickly warming the ear with gauze soaked in saline at 38° to 40° C (100.4°-104.0° F). Any blisters that form should be allowed to reabsorb spontaneously. Topical antibiotic ointment can be applied, and viability of the tissue should be assessed periodically.

Lacerations

When there is trauma to the ear requiring suture closure, careful realignment is mandatory to maintain the auricular contour. The extent of the injury should be evaluated thoroughly with the tissue anesthetized with 1% lidocaine (without epinephrine). This allows a careful evaluation of the wound as well as a meticulous suture closure. Lacerations involving only the skin can be closed with everting nonabsorbable sutures. An earlobe that is torn from earring trauma can be closed in layers using absorbable chromic gut suture to close the dermis and nonabsorbable 5-0 or 6-0 suture to close the skin. Lacerations that involve cartilage, perichondrium, and skin must also be closed in layers but might best be referred to a specialist in otorhinolaryngology or to a plastic surgeon.

Disorders of the Middle Ear

Key Points

- Tympanic blistering (bullous myringitis) is simply a variant of acute otitis media (AOM) and should be treated as such.
- The most common infection in children seen in a physician's office is AOM.
- Three criteria are necessary for the diagnosis of AOM: acute onset, middle ear effusion, and signs or symptoms of middle ear inflammation.
- Severe AOM is defined as moderate to severe otalgia and fever greater than 39° C (102.2° F).
- When the diagnosis of AOM is uncertain (<2 criteria), observation is allowed in nonsevere illness.
- Amoxicillin is the drug of choice in treating AOM (80-90 mg/kg/day in 2 divided doses).
- Influenza vaccine decreases the number of cases of AOM in children 6 to 24 months of age.
- Surgical treatment is necessary for all middle ear and mastoid cholesteatomas.
- Antihistamines, decongestants, antibiotics, and corticosteroids are not recommended for routine use in otitis media with effusion.
- All traumatic perforations of the tympanic membrane require audiologic evaluation to rule out sensorineural hearing loss.
- Sudden sensorineural hearing loss is an otologic emergency.
- Facial weakness in the setting of AOM can require myringotomy with or without tube insertion in addition to broad-spectrum antibiotics.

Bullous Myringitis

Bullous myringitis refers to painful inflammatory bullae on the tympanic membrane. The blebs appear hemorrhagic. It was formerly thought that bullous myringitis was caused by *Mycoplasma pneumoniae* infection. Roberts (1980), however, summarized six studies involving 858 patients with bullous myringitis, and *M. pneumoniae* was isolated from only one. The cause is usually viral but can be bacterial in some cases. Studies have confirmed that bacterial cultures from bullous fluid are similar to cultures from middle ear fluid taken from patients with acute otitis media. The main isolates are *Streptococcus pneumoniae, Haemophilus influenzae,* and beta-hemolytic streptococci. The tympanic blistering is probably a nonspecific reaction and simply a variant of acute otitis media that should be treated as such.

It is important to distinguish bullous myringitis from acute otitis externa, which requires topical treatment, and from herpes zoster oticus, which can lead to cranial neuropathy and requires antiviral treatment. Neither of these conditions is usually limited to only tympanic membrane involvement.

KEY TREATMENT

No evidence supports the role of *Mycoplasma pneumoniae* as the causative agent in bullous or hemorrhagic myringitis (SOR: A) (Kotikoskie et al., 2004).
Bullous myringitis should be treated with antimicrobials used to treat acute otitis media (SOR: A).

Otitis Media

Acute Otitis Media

The most common infection for which children are seen in a physician's office is acute otitis media (AOM). The annual cost of AOM in the United States is an estimated $5 billion (Bondy et al., 2000). By age 7 years, 93% of children have had at least one episode of AOM, and 75% have had recurrent infections. AOM can occur at any age, but the highest incidence is between 6 and 24 months in the United States.

The primary cause of bacterial colonization of the middle ear is eustachian tube dysfunction. Abnormal tubal compliance in addition to delayed innervation of the tensor veli palatini muscle leads to collapse of the eustachian tube. Aerobic and anaerobic organisms, as well as viruses, can contribute to middle ear infection (Heikkinen et al., 1999). The three most common bacteria involved in AOM are *S. pneumoniae* (25%-40% of cases), *H. influenzae* (10%-30%), and *Moraxella catarrhalis* (2%-15%) (Klein, 2004). Risk factors most often associated with AOM are child care outside

the home and parental smoking. Box 19-3 lists the common risk factors for AOM. A viral upper respiratory infection usually precedes an episode of AOM.

Three criteria are necessary to confirm the diagnosis of AOM: acute onset, presence of middle ear effusion, and signs or symptoms of middle ear inflammation (American Academy of Pediatrics [AAP], 2004; Level of evidence [Grade] B). Middle ear effusion can be diagnosed by direct visualization of air-fluid levels behind the tympanic membrane, a bulging drum, lack of movement on pneumatic otoscopy, or a flat tympanogram readout that indicates no tympanic membrane movement and therefore the presence of middle ear effusion. Redness of the tympanic membrane, pain, and fever are the most common signs and symptoms of middle ear inflammation (see **eBox 19-4** online). Erythema of the tympanic membrane without middle ear effusion is myringitis or tympanitis and is a separate diagnosis from AOM. Ear pain in the presence of a normal-appearing, flaccid tympanic membrane indicates causes other than AOM (Box 19-4).

The standard of care for the treatment of AOM in children older than 2 years is not to treat with antibiotics at the first visit, but to treat the pain and either observe the patient or prescribe an antimicrobial agent depending on certain criteria. The decision either to begin antibiotics or to observe the patient without them is based on the certainty of diagnosis, severity of symptoms, and age of the patient (AAP, 2004). (Table 19-2).

When all three criteria for the diagnosis of AOM are met (acute onset, middle ear effusion, and inflammation), the diagnosis is certain, and antibiotic therapy is indicated for any child 2 years old or younger (AAP, 2004; Grade A). For children older than 2 years, observation is an option if the illness is not severe and the parents can be relied on to report the patient's status and can obtain medication if necessary. Severe illness is defined as moderate to severe otalgia and fever higher than 39° C. (102.2° F.) When two or fewer diagnostic criteria are present, diagnosis is considered uncertain, and observation is allowed for children 6 months and older with nonsevere illness.

Box 19-3 Common Risk Factors for Acute Otitis Media (AOM)

- Male gender
- Bottle-feeding, especially in the supine position
- Exposure to upper respiratory tract infections (e.g., daycare setting, winter season)
- Genetic factors
- Ethnic factors (e.g., Inuit or Native American)
- Parental smoking
- Allergy
- Craniofacial abnormalities (e.g., cleft palate)
- Previous episode of AOM, particularly during the preceding 3 months)
- Use of a pacifier

From O'Handley JG. Controversies in the management of otitis media. Prim Care Rep 1999;5:43.

Box 19-4 Causes of Otalgia Other than Acute Otitis Media

- Abscessed teeth
- Cervical arthritis
- Dental malocclusion
- Nasopharyngeal carcinoma
- Sinus infection
- Sore throat
- Temporomandibular joint disorders

Table 19-2 Treatment of Acute Otitis Media

Features	Treatment
Low-Risk Patients	
Older than 6 years, no antimicrobial therapy within past 3 months, no otorrhea, not in daycare, and temperature <38° C (<100.5° F)	Amoxicillin: 40-50 mg/kg/day in divided doses for 5 days
High-Risk Patients	
Younger than 2 years, in daycare, treated with antimicrobials within past 3 months, otorrhea, or temperature >38° C (>100.5° F)	Amoxicillin: 80-90 mg/kg/day in divided doses for 10 days
Treatment Failure	
Signs and symptoms persisting after 3 days	Amoxicillin–clavulanic acid (Augmentin): 80-90 mg/kg/day for 10 days Cefuroxime axetil (Ceftin): 20-30 mg/kg/day bid for 10 days Ceftriaxone (Rocephin): 50 mg/kg intramuscularly for 1 dose
Penicillin-Allergic Patient	
Any	Cefuroxime axetil: <2 years, 125 mg bid; ≥2 years, 250 mg bid TMP-SMX (Bactrim, Septra): 8 mg/kg TMP, 40 mg/kg SMX, per 24 hours in 2 doses Cefprozil (Cefzil): 30 mg/kg/day in 2 doses Cefaclor (Ceclor): 40 mg/kg/day in 3 doses Cefixime (Suprax): 8 mg/kg/day as a single dose

TMP-SMX, Trimethoprim-sulfamethoxazole; *bid,* twice daily.

eBox 19-4 Common Signs and Symptoms of Inflammation and Fluid in the Middle Ear

Diminished hearing
Ear pulling
Fever
Irritability
Loss of appetite
Otalgia
Otorrhea
Tinnitus
Vertigo
Vomiting

From O'Handley JG. Acute otitis media. In Rakel RE (ed). Manual of Medical Practice, 2nd ed, pp 78-79. Philadelphia, Saunders, 1996.

Resistance of *Streptococcus pneumoniae* to penicillin is an increasing problem and ranges from 15% to 50% depending on the area. The mechanism of resistance is based on an alteration of penicillin-binding proteins rather than the production of beta-lactamase, as occurs with *H. influenzae* and *M. catarrhalis.* Resistance rates are higher in children than in adults, especially if the children are in daycare or have received antimicrobial therapy in the previous 3 months (Dowell and Schwartz, 1997).

The dose to treat AOM is 80 to 90 mg/kg/day in two divided doses (AAP, 2004). This allows the drug to overcome resistance in the causative organism (Dowell et al., 1999). For patients with a penicillin allergy, alternative medications include cefdinir, cefpodoxime, or cefuroxime. A meta-analysis found that first-generation cephalosporins have cross-allergy with penicillin, although the cross-allergy with second- and third-generation cephalosporins is negligible (Pichichero and Casey, 2007). Macrolides are not recommended for AOM in children because *H. influenzae* is the dominant organism causing AOM in this age group. Middle ear fluid becomes sterile 3 to 6 days after starting treatment (Carlin et al., 1991), so duration of therapy for uncomplicated AOM is 5 to 7 days, except for the child with an episode of AOM in the past 30 days, for whom a 10-day course of therapy is recommended (Pichichero and Brixner, 2006).

If the initial antibiotic fails to resolve symptoms in 72 hours (pain, fever, redness and bulging of the tympanic membrane, otorrhea), high-dose amoxicillin–clavulanic acid is recommended. Alternatives in penicillin-allergic patients include the antibiotics cited earlier. Patients who do not respond to amoxicillin–clavulanic acid therapy should be treated with intramuscular ceftriaxone for 3 days. This antibiotic in a single dose can also be used initially if the child is vomiting or unable to keep down oral medication. Doses of antimicrobials are given in Table 19-2.

Influenza vaccine has been shown to decrease the number of cases of AOM in immunized patients compared to controls and is recommended for all children age 6 to 24 months.

KEY TREATMENT

Antibiotics are most beneficial in children younger than 2 years with bilateral acute otitis media (AOM) and/or otorrhea (SOR: A).
For most other children with mild disease, close observation and follow-up is an option (SOR: A) (Vouloumanou et al., 2009).
Administration of the seven-valent pneumococcal vaccine (PCV7) in infancy reduces the risk for AOM by 6% to 7%. Administering PCV7 to older children with a history of AOM appears to have no benefit in preventing further episodes (SOR: A) (Jansen et al., 2009).
Second- and third-generation cephalosporins may be used to treat AOM in penicillin-allergic patients (SOR: A) (Pichichero and Casey, 2007).
Uncomplicated AOM may be treated for 5 to 7 days (Pichichero and Brixner, 2006).
SOR: A

Otitis Media with Effusion

Otitis media with effusion (OME) is defined as persistent middle ear fluid without pain, fever, or redness of the tympanic membrane. It is often the result of AOM but can occur de novo. About 90% of children have OME before they reach school age. About 80% to 90% of cases resolve within 3 months and 95% within 1 year. Table 19-3 provides the Agency for Health Care Policy and Research (AHCPR) guidelines for treatment of OME.

Tympanometry can be used to judge the presence of middle ear fluid. It is important to document the affected ear, the duration of the effusion, and the presence and severity of symptoms associated with OME. The latter include a feeling of fullness in the ear, popping, mild pain, hearing loss, balance problems, and delayed language development.

If OME persists for 3 months, a comprehensive hearing evaluation should be performed. A 40-decibel (dB) loss (or worse) in hearing bilaterally mandates referral for evaluation for polyethylene (PE) tube placement. Management of hearing loss between 6 and 39 dB depends on parent or caregiver preferences and can include strategies to improve the listening and learning environment or referral for tube placement. If the hearing loss is 5 dB or less, repeat testing in 3 months may be performed if the middle ear effusion continues at that time. Follow-up testing is recommended every 3 to 6 months until the effusion resolves, unless significant hearing loss occurs or there is evidence for structural abnormalities of the eardrum or middle ear. In these patients, PE tube placement is the preferred course.

When referring to a surgeon, the primary care physician must provide an adequate history of the duration of the middle ear effusion, developmental state of the child, and pertinent information such as a history of AOM. Physician and parental expectations for the referral should be clarified. Ultimately, the decision for PE tube placement should be based on a consensus among all parties involved. The possibility of repeat surgery after tube extrusion is 20% to 50%, and with reoperation, adenoidectomy is recommended in children with normal palates because it reduces the need for future surgery by 50%.

KEY TREATMENT

Otitis media with effusion is best diagnosed with pneumatic otoscopy.
In children with craniofacial abnormalities, visual impairment, autism, speech delay, or hearing impairment, appropriate management of OME is critical (AAFP et al., 2004).
Antihistamines and decongestants have not been shown to be effective and are not recommended for treatment of OME.
Antibiotics and corticosteroids are also not recommended for routine management of OME because they do not provide long-term efficacy.
SOR: A; B

Recurrent Otitis Media

Three episodes of AOM within 6 months with complete resolution between episodes or four episodes in 12 months defines recurrent otitis media. Although the evidence is conflicting, a double blind, placebo-controlled study comparing once or twice daily amoxicillin prophylaxis with placebo showed no benefit to using antibiotics. The authors recommended discouraging amoxicillin prophylaxis in children with recurrent otitis media not only because it is ineffective but also to prevent the acquisition of resistant pneumococci (Roark and Berman, 1997).

Tympanostomy tubes may be considered and may be beneficial in cases of recurrent AOM requiring multiple rounds of antibiotics within 6 to 12 months, especially if the episodes are severe.

Table 19-3 AHCPR Guidelines for Treatment of OME

Duration of OME	Treatment
6 weeks	Observation or antimicrobial therapy Hearing evaluation optional
3 months	Referral for hearing evaluation; with 20-dB hearing loss, patient should receive antimicrobial therapy or PE tube
4 to 6 months	Referral for PE tube if there is hearing loss

AHCPR, Agency for Health Care Policy and Research; *OME,* otitis media with effusion; *PE,* polyethylene.

Box 19-5 Complications of Otitis Media

- Acute mastoiditis
- Brain abscess
- Epidural abscess
- Facial nerve paralysis
- Labyrinthitis
- Meningitis
- Sigmoid sinus thrombophlebitis
- Subdural abscess
- Subperiosteal abscess
- Cholesteatoma

Chronic Suppurative Otitis Media

Chronic suppurative otitis media (CSOM) is the presence of persistent purulent otorrhea through a perforated tympanic membrane or tympanostomy tube. A persistent tympanic membrane perforation may result from acute otitis media, chronic eustachian tube dysfunction, or trauma. A cholesteatoma or rarely a tumor may also result in CSOM. Otorrhea may also be from chronic otitis externa, which may be difficult to distinguish from CSOM until treatment is initiated. Causes of otorrhea from the middle ear may not always be from middle ear bacterial infection (see **eTable 19-3**).

Associated symptoms often include hearing loss and tinnitus. Increasing pain, vertigo, or facial palsy imply a possible impending complication of CSOM (discussed later) and requires urgent otolaryngologist consultation. Binocular otomicroscopy allows better visualization and suctioning of purulent material compared to routine otoscopy. Imaging is reserved for medical treatment failures or if a complication is suspected. CT is helpful to evaluate for bony erosion. MRI is indicated with suspicion of CNS involvement.

Initial management should include culture and sensitivity of the discharge to allow appropriate antibiotic selection. If chronic otitis externa is suspected, the specimen should also be sent for fungal culture. Empiric antimicrobials should be started with coverage against the usual pathogens, which include *S. pneumoniae, H. influenzae, S. aureus, Pseudomonas* spp., and anaerobes. A Cochrane review demonstrated that ototopicals are superior to oral antibiotics. Quinolone ototopicals are safe for use in the middle ear. Topical aminoglycosides carry a risk of ototoxicity, but in some cases their use outweighs the risk. Systemic antibiotics may be required in severe cases or when copious drainage impairs administration of ototopicals. Aural toilet with an acetic acid solution (1 part distilled water, 1 part white vinegar) may be helpful to clear debris and provide antisepsis (Macfadyen et al., 2006a, 2006b).

If otorrhea resolves but a tympanic membrane perforation persists, the patient may be offered a tympanoplasty (tympanic membrane repair) to reduce the risk of recurrence and improve hearing. If medical therapy fails to control inflammation, a tympanomastoidectomy may be indicated to eradicate infection, aerate the middle ear and mastoid, and repair the tympanic membrane. *Chronic tympanostomy tube otorrhea* is treated the same as typical CSOM. In recalcitrant cases, however, tube removal or replacement may be indicated. Also, adenoidectomy may be considered because chronic adenoiditis may act as a nidus for infection.

Complications

Although rare since the advent of antibiotics, complications of otitis media must be recognized early to avoid significant potential morbidity and mortality (Box 19-5). Chronic or recurrent otitis media can result in scarring of the tympanic membrane *(myringosclerosis* or *tympanosclerosis)*, which alone is usually of no consequence. If scarring involves the ossicles, however, hearing loss can result. Tympanic membrane retraction or perforation can also occur.

Intratemporal Complications

Extension of the infection into the mastoid air cells can lead to *acute mastoiditis.* The signs and symptoms of acute mastoiditis are fever and postauricular tenderness, erythema, and edema. It is important to recognize that acute mastoiditis is a clinical and not a radiologic diagnosis. Therefore, inflammatory changes on a temporal bone CT must be correlated with examination findings to be called acute mastoiditis. Furthermore, acute mastoiditis must be distinguished from an auricular cellulitis secondary to acute otitis externa. *Facial paralysis* may result from AOM or CSOM because of inflammation along the facial nerve as it courses through the middle ear space. Treatment of acute mastoiditis and facial nerve paralysis includes IV antibiotics, insertion of a tympanostomy tube for drainage, and sometimes emergent mastoidectomy. Infection within the middle ear space can extend into the inner ear, leading to labyrinthitis. Symptoms may include vertigo, tinnitus, and sensorineural hearing loss. Expeditious initiation of broad-spectrum antibiotics and in some cases insertion of a tympanostomy tube may be necessary. *Petrositis* is a rare complication involving inflammation of the petrous apex mastoid air cells. *Gradenigo's syndrome,* which includes retro-orbital pain, otorrhea, and cranial nerve VI palsy, may result. Treatment includes IV antibiotics and surgical drainage.

Intracranial Complications

The most serious complications of otitis media involve CNS extension of the infection and include sigmoid sinus thrombosis, meningitis, and brain abscess. Warning signs of an impending CNS complication include increasing pain, headache, spiking fever, or altered mental status. Evaluation may include MRI and lumbar puncture. Suspicion of CNS complication often requires urgent neurosurgical, ENT,

eTable 19-3 Otorrhea from the Middle

Cause	Pathophysiologic Process	Diagnostic Clue	Treatment
Acute otitis media with perforation	Purulent effusion in middle ear space with subsequent perforation	Antecedent upper respiratory infection Otalgia preceding otorrhea Purulent drainage	Otic antibiotic drops with oral antibiotics Aural cleaning Dry-ear precautions
Chronic otitis media with perforation with or without cholesteatoma	Chronic inflammation of middle ear space and mastoid, usually caused by *Staphylococcus* and *Pseudomonas* organisms	Purulent drainage History of tympanic membrane perforation White or pearly appearing mass behind tympanic membrane Unresponsive to prior antibiotic therapy Polyp in external ear canal	Otic antibiotic drops Complete audiologic evaluation Temporal bone CT Culture of drainage if not responding to prior antibiotic therapy Referral to ENT
Trauma	Head trauma with temporal bone fracture and possible cerebrospinal fluid leakage	History of head trauma or ear trauma Bloody drainage Clear drainage that continues to accumulate Facial nerve weakness	Otic antibiotic drops Complete audiologic evaluation Temporal bone CT Dry-ear precautions Referral to ENT
Tuberculosis of middle ear	Hematogenous spread of *Mycobacterium tuberculosis* organisms to middle ear space	Positive result of PPD or anergy test History of pulmonary TB Thin, odorless drainage (chronic) No response to prior antibiotic therapy	Acid-fast bacillus stain and culture Antituberculosis medications if positive PPD test result or if high index of suspicion
Neoplasia	Neoplasm originating in or extending to middle ear space	Unilateral Pulsatile tinnitus Mass visualized Polyp in external ear canal	Complete audiologic evaluation Temporal bone CT Referral to ENT

From Lucente FE, Har-El G: Essentials of Otolaryngology, p 101. Philadelphia, Lippincott–Williams & Wilkins, 1999.
CT, Computed tomography; *ENT,* otorhinolaryngologist; *PPD,* purified protein derivative; *TB,* tuberculosis.

and infectious diseases consultations. High-dose IV antibiotics and sometimes urgent surgery (mastoidectomy or craniotomy) are required to prevent significant morbidity or mortality. Otitic meningitis is a major cause of morbidity in the pediatric population. Fortunately, vaccines against *H. influenzae* type B and *Pneumococcus* spp. (Prevnar) have decreased these occurrences.

A "cholesteatoma" is a destructive epithelial cyst in the middle ear that may extend to the mastoid air cells. The term is a misnomer because of the lack of cholesterol and presence of only squamous epithelium and keratin debris. The external ear canal and outer layer of the tympanic membrane are lined with squamous epithelium. Keratin debris is continuously sloughed as new epithelial cells mature. In a normal ear the debris slowly migrates to the external meatus, where it is washed away. In contrast to the external ear, the middle ear space is lined with respiratory epithelium, which produces no keratin debris. Cholesteatomas form when squamous epithelium is abnormally located within the middle ear space, allowing the keratin debris to accumulate.

Cholesteatomas often result in CSOM with findings of purulent otorrhea, polyps, and granulation. However, some cholesteatomas are dry, with the finding of a white mass visible behind the tympanic membrane or a white mass or crusting on the tympanic membrane itself. A cholesteatoma must be differentiated from *myringosclerosis,* which is usually flat, white scarring on the tympanic membrane. Enzymatic properties, inflammation, and pressure may lead to bone erosion and hearing loss. If left untreated, serious problems may result, including facial nerve paralysis, labyrinthine fistula, and intracranial complications (see Complications of otitis media).

Congenital cholesteatomas occur without a history of tympanic membrane perforation or retraction and are postulated to result from congenital rests of epithelium in the middle ear space. These may occur in children with no significant history of otitis media and are usually diagnosed as an incidental white mass behind the tympanic membrane.

Primary acquired cholesteatomas result from prolonged eustachian tube dysfunction. Negative middle ear pressure results in a *retraction pocket* of the tympanic membrane, usually at the region of the pars flaccida. Squamous epithelium may become trapped and accumulate in the retraction pocket, resulting in a cholesteatoma. Any retraction pocket of the tympanic membrane requires further evaluation to prevent progression to cholesteatoma.

Secondary acquired cholesteatomas occur as a result of a tympanic membrane perforation. In some cases, a perforation of the tympanic membrane may allow the outer squamous epithelium to migrate into the middle ear space, leading to cholesteatoma formation.

Although aggressive medical treatment may reduce inflammation, surgical treatment is necessary for almost all cholesteatomas. In less advanced cholesteatomas, the external auditory canal is spared in surgery, a "canal wall up" tympanomastoidectomy. Close follow-up is required because of the risk of recurrence from microscopic disease or persistent eustachian tube dysfunction. In more advanced disease, the posterior canal is removed, termed a "canal wall down" tympanomastoidectomy (modified radical and radical mastoidectomy). The canal wall–down procedures are more likely to result in a conductive hearing loss, but the mastoid cavity becomes accessible through a larger external canal, thereby exteriorizing the cholesteatoma. Typically, semiannual mastoid bowl debridement is necessary to remove squamous and ceruminous debris to prevent inflammation.

Traumatic Tympanic Membrane Perforations

Traumatic perforation of the tympanic membrane may result from barotrauma (water skiing/diving injuries, blast injuries, blows to side of head), ear canal instrumentation (cotton-tipped applicators, bobby pins, paper clips, cerumen curettes), or otitis media (see earlier discussion). The patient usually complains of acute pain that subsides quickly, associated with bloody otorrhea. Severe vertigo can occur but is transient in most cases. Persistent vertigo suggests inner ear involvement (perilymphatic fistula). Hearing loss and tinnitus are also common.

Findings may include fresh blood in the canal and around the perforation. Any medial canal clots or debris should not be removed or irrigated except under microscopy. Secondary bacterial infection may require treatment with ototopical antibiotics. Topical fluoroquinolones are safe for use in the middle ear. Audiologic evaluation is necessary to rule out sensorineural hearing loss. If a tuning fork examination indicates sensorineural hearing loss or is unreliable, the patient should be referred for complete audiologic and ENT evaluation.

In uncomplicated cases the perforation is expected to heal spontaneously over days to weeks. The patient should be instructed to keep the ear dry during this time. If the perforation has not healed after several weeks, a tympanoplasty to close the perforation and, if necessary, repair ossicles is indicated. Repair of the perforation may improve hearing, reduce infection, and prevent cholesteatoma formation.

Barotrauma and Barotitis

Changes in altitude while flying (or scuba diving) can lead to rapid changes in middle ear pressure, leading to accumulation of serous middle ear fluid or blood. Symptoms may include aural fullness, otalgia, and conductive hearing loss. In most cases the fluid is resorbed, although this may take several weeks. *Autoinflation* maneuvers (popping the ears) may hasten recovery. Oral and topical decongestants, nasal steroid sprays, or a short course of corticosteroids may be helpful. Antibiotics are indicated only if there are signs of infection. If fluid persists or is troublesome to the patient, a myringotomy allows the fluid to be drained. Tympanostomy tube insertion may be indicated for persistent middle ear fluid.

Rarely, a rapid change in middle ear pressure can lead to the creation of a *perilymphatic fistula* between the inner and middle ear. The patient complains of severe vertigo and hearing loss (sensorineural) (see Vertigo). Urgent ENT consultation is indicated.

Hearing Loss

Hearing loss results from an interruption in the transmission of sound or subsequent nerve impulses in one or more areas of the ear. Recognition and treatment of hearing loss are imperative; unrecognized or untreated hearing loss may

result in severe psychosocial ramifications in both adults and children. In the elderly population, hearing loss may lead to social withdrawal and depression. In the pediatric population, hearing loss may cause speech or cognitive delays. Hearing loss also has significant safety implications when it interferes with awareness of warning sounds (e.g., car horns, sirens, fire alarms). The four types of hearing loss follow:

1. *Conductive hearing loss* (CHL) occurs when there is a failure of normal propagation of acoustic energy through the conducting portions of the ear, which include the external auditory canal and the middle ear.
2. *Sensorineural hearing loss* (SNHL) occurs from dysfunction of the inner ear, which may be caused by a failure of the generation of nerve signals in the cochlea by the cochlear hair cells or propagation of electrical signals along the cochlear division of the eighth cranial nerve.
3. *Mixed hearing loss* (MHL) occurs when hearing loss results from both CHL and SNHL.
4. *Central hearing loss* can result from ischemic or traumatic brain injuries.

Hearing loss may be subclassified according to whether it is *acquired* or *congenital*. Hearing loss is further classified based on its *severity* (mild, moderate, severe, profound), *sidedness* (right, left, bilateral), *stability* (stable, progressive, fluctuating), and *cause*.

Evaluation includes noting the onset and duration of the hearing loss, any inciting events, the subjective severity of the hearing loss, and any psychosocial impact. Associated ear symptoms, medical history, and a history of ototoxic medication exposure are also important. Although history and examination provide clues to the etiology of the hearing loss, comprehensive audiometric evaluation is essential to making a diagnosis. Box 19-6 lists the most common types of CHL and SNHL.

Box 19-6 Common Types of Conductive and Sensorineural Hearing Loss

Conductive Hearing Loss

Cholesteatoma
Cerumen impaction
Foreign body in ear canal
Ossicular problems
Otitis media with effusion
Otosclerosis
Retracted tympanic membrane (eustachian tube dysfunction)
Tumor of the ear canal or middle ear
Tympanic membrane perforation
Tympanosclerosis

Sensorineural Hearing Loss

Acoustic neuroma
Diabetes
Hereditary (congenital) loss
Idiopathic loss
Meniere's disease
Multiple sclerosis
Noise-induced loss
Ototoxicity
Perilymphatic fistula
Presbycusis
Syphilis

Otosclerosis

Otosclerosis is caused by sclerotic fixation of the stapes and is the most common cause of CHL in the adult population with no previous history of trauma or infection. It is autosomal dominant in inheritance and more common in women than in men. Otosclerosis is usually bilateral and progressive. Treatment options include no treatment, amplification (hearing aid), fluoride treatment (stabilizes but does not improve hearing), and surgery (stapedectomy). Stapedectomy involves removing and replacing the stapes with a tiny prosthesis. This procedure has a success rate of greater than 95%. Risks of surgery, although rare, include worsened hearing, tympanic membrane perforation, changes in taste, and disequilibrium.

Sudden Sensorineural Hearing Loss

See Emergencies.

Presbycusis

Presbycusis is an all-inclusive term to describe the process of hearing loss related to aging. An estimated 30% to 35% of adults age 65 to 75 and 40% to 50% of adults older than 75 have hearing loss. Symptoms of presbycusis include gradually decreasing hearing acuity, especially for higher-pitch tones (women's and children's voices) and in certain situations (with background noise). Tinnitus is common.

The cause of presbycusis is likely multifactorial, but ultimately the loss of cochlear *hair cell* function is thought to be the cause in most cases. Hair cell damage or loss can result from chronic noise exposure, genetic predisposition, and ototoxic medications. The hearing loss may also be caused by neurovascular injury from chronic conditions such as hypertension or diabetes, which can affect the cochlea or cochlear nerve. Hormonal conditions such as hypothyroidism should be considered, as should unusual conditions such as tertiary syphilis. Central auditory problems might be the cause, from dementia, cerebrovascular disease, or cerebrovascular accident (CVA, stroke).

Although the term "presbycusis" implies sensorineural loss, conductive hearing loss should also be considered, including cerumen impaction, chronic OME, and otosclerosis (see Box 19-6).

Acoustic Neuroma

An acoustic neuroma (or more precisely, *vestibular schwannoma*) is a benign tumor that arises from the Schwann cells of cranial nerve VIII. Acoustic neuromas account for about 10% of all intracranial tumors. They are most commonly diagnosed in middle age. They are slightly more common in women than men. They are usually sporadic but may be associated with neurofibromatosis 1 or 2 (NF-1, NF-2). Most patients with NF-2 will develop bilateral acoustic neuromas. Acoustic neuromas in NF-1 are much less common.

The primary symptoms of vestibular schwannoma are asymmetric hearing loss (sensorineural) and tinnitus. The hearing loss is usually gradual in onset and progressive but can occur suddenly. Disequilibrium is not usually the chief complaint on presentation, but patients often admit to mild unsteadiness. Larger tumors can cause dysesthesia around the ear or facial weakness, or both. If the neuroma is diagnosed late, patients can manifest cerebellar symptoms and symptoms of mass effect and obstructing hydrocephalus.

After a complete neuro-otologic examination and audiologic evaluation, an MRI scan of the brain with fine cuts through the internal auditory canal with gadolinium contrast is necessary for diagnosis.

Treatment options include observation, surgery, or stereotactic radiotherapy. Most vestibular schwannomas require treatment to prevent cerebral complications from future growth. If the patient is aged or infirm, watchful waiting may be considered. Very small tumors may be observed because the rate of growth is often slow. Surgical treatment involves either a translabyrinthine resection if hearing is poor or a craniotomy for hearing preservation. Stereotactic radiotherapy has the obvious advantage of avoiding major surgery. Success is similar to surgery for smaller tumors. Shortcomings of this treatment include delayed facial paresis, tumor recurrence (which requires routine monitoring), and the potential for radiation-induced malignancies in the future.

Hearing Loss from Acoustic Energy

Excessive noise exposure is an important and usually preventable cause of hearing loss. Hearing loss can result from chronic or acute noise exposure, usually causing injury at the level of the cochlear hair cells. However, acute acoustic trauma can also cause injury to the tympanic membrane and middle ear structures.

Chronic noise exposure may be recreational or vocational. The Occupational Safety and Health Administration (OSHA) has established guidelines for safe limits for acute and chronic noise exposure to prevent occupational noise-induced hearing loss. Exposure to noise of 90 dB or less is permissible for up to 8 hours per day. As the noise intensity increases, the permissible duration of exposure decreases. OSHA outlines procedures for hearing protection and monitoring. These standards also can help provide guidelines to minimize excessive recreational noise exposure. Recreational activities known to cause excessive noise include hunting or target shooting with firearms, use of power tools or power lawn equipment, attendance at sporting venues, motor racing events, action movies, or concerts and listening to loud music on headphones. Hearing protection or avoidance is recommended for such activities.

Acute exposure to excessively loud noise can cause conductive or sensorineural hearing loss. CHL can result from a blast-type injury that leads to tympanic membrane perforation or ossicle injury. The conductive component of the hearing loss is usually reparable, but severe acoustic trauma can also cause sensorineural loss. SNHL from acute acoustic trauma is usually the result of temporary hair cell dysfunction or permanent injury, leading to transient or permanent *threshold shifts*, respectively. A concussive or blast injury (e.g., slap, airbag deployment to ear) can result in the formation of a labyrinthine fistula from the inner ear into the middle ear, which causes severe vertigo and SNHL. Most fistulas close spontaneously with bed rest, but some require middle ear exploration and repair. However, the sensorineural hearing loss is usually permanent.

Hearing Loss in the Pediatric Population

Congenital hearing loss is often hereditary but may be secondary to an intrauterine insult or infection. Of the hereditary variety, the majority are autosomal recessive and nonsyndromic. Risk factors for congenital hearing loss include family history of hearing loss, facial abnormalities, ICU admission, history of meningitis, syndromes known to be associated with hearing loss, low Apgar scores at birth, medications known to cause hearing loss (e.g., aminoglycosides), elevated bilirubin, some prenatal maternal infections, or suspicion of hearing loss.

Universal newborn hearing screening using otoacoustic emissions and auditory brainstem response allows early identification of impaired children. Intervention by age 6 months appears to improve language development. A temporal bone CT is often obtained to evaluate for inner ear malformations that would predispose the patient to further hearing loss with even mild head trauma. A genetics evaluation and counseling may be indicated. Mutations of the Connexin-26 gene, an autosomal recessive disorder, accounts for a significant percentage of nonsyndromic hereditary hearing loss. Hearing loss may coexist with other conditions (e.g., renal, ophthalmologic, thyroid, infectious, cardiac), so other testing may be indicated based on clinical suspicion.

In children with a significant hearing loss, hearing aids are recommended. A cochlear implant may be indicated for those with hearing so poor to be considered unaidable. A cochlear implant is a surgically implanted device that receives sound, converts it to electrical signals, and directly stimulates the cochlea. Results are excellent in properly selected patients.

An important and often unrecognized cause of hearing loss in children is chronic OME. Children with either unilateral or bilateral middle ear effusions refractory to medical treatment for more than 3 months should have audiometric testing. Myringotomy with tube insertion is indicated if bilateral CHL is found and should be considered in some cases of unilateral loss as well.

Treatment of Hearing Loss

Individual treatments for specific causes of hearing loss vary greatly. This section gives a brief overview of options available to improve hearing in patients with the most common causes of hearing loss.

Surgery is often performed for CHL. Myringotomy with or without tube insertion corrects hearing loss in cases of OME. The procedure is performed under brief general anesthesia in children or under local anesthesia for most adults. A tympanoplasty is performed for tympanic membrane perforations and to reconstruct ossicles with a prosthesis. A stapedectomy with placement of a prosthesis is often a successful option in patients with otosclerosis.

Cochlear implantation is indicated for profound SNHL in patients who do not benefit from conventional hearing aids. The procedure is indicated for adults and children as young as 12 months old.

Table 19-4 Causes of Facial Paralysis in Order of Occurrence

Cause	Percentage	Characteristics
Idiopathic (Bell's palsy)	60-85	Acute onset; viral prodrome (60% of cases)
Trauma	20-50	Acute-onset paralysis or paresis of previously functioning nerve
Herpes zoster	10-15	Ramsay Hunt syndrome with cranial nerve VII involvement, vestibular(vertigo), cochlear (hearing loss)
Tumor	10-15	Slow progression to complete paralysis
Birth	10-15	Part of congenital syndrome or birth trauma at delivery
Infection	4	Mastoiditis, otitis media, direct cranial nerve VII infection, Lyme disease
Brain lesion (central nervous system)	<10	Supranuclear or in brainstem

From Brody R, Har-El G. From Lucent FE, Har-El G. Essentials of Otolaryngology. Philadelphia, Lippincott Williams & Wilkins, 1999, p 131.

A variety of styles of hearing aids are used to rehabilitate hearing loss in patients with SNHL and CHL. The simplest (and least expensive) are larger, behind-the-ear aids with analog amplification of sound. The most complex (and most expensive) are completely-in-the-canal aids with programmable digital amplification. Several other types of aids fall between these two extremes. A certified audiologist, under the supervision of an otorhinolaryngologist, assists the patient with proper selection of an appropriate hearing aid.

Facial Nerve Paralysis

Facial paralysis occurs for various reasons. Possible causes are listed in Table 19-4. The eponym "Bell's palsy" is reserved for cases of idiopathic facial paralysis. It has been shown, however, that many if not most cases of idiopathic facial paralysis are actually caused by reactivation of latent herpesvirus living in the facial nerve or geniculate ganglion.

Although the most common cause of facial paralysis is indeed Bell's palsy, it is incumbent to rule out *other* potentially serious causes of facial paralysis before making this *diagnosis of exclusion*. Initially, a complete history and physical examination are required, including otologic and neurologic evaluation. The patient should be questioned regarding history of recurrent cold sores, which suggest herpetic involvement. Recent travel (especially camping) should be noted because Lyme disease is an often-overlooked cause of facial paralysis. Involvement of facial nerves is a concern in patients with a history of chronic otitis media or cholesteatoma. Other symptoms should be noted. Otalgia is common with Bell's palsy and does not always imply that the ear is involved. Of course, questions regarding risk factors for cerebrovascular disease or previous CVA should be obtained.

Evaluation of facial nerve function requires careful attention and comparison between the two sides of the face. The patient should be evaluated at rest and with voluntary movement. The patient should be asked to wrinkle the nose, raise the eyebrows, squeeze the eyes shut, and purse the lips to assess all branches of the facial nerve. The facial skin should be assessed, because a rash can indicate *herpes zoster oticus* (Ramsay Hunt syndrome). The eyes should be inspected to rule out exposure keratitis from lack of eye closure and dryness. If keratitis is suspected, ophthalmologic consultation should be obtained to prevent loss of vision. A complete neurologic examination must be done. If other neurologic deficits are found, neurology consultation is indicated. Lesions in the auditory canal should raise suspicion of herpes zoster oticus or malignancy of the external auditory canal with facial nerve involvement. Otitis externa and facial weakness can represent malignant otitis externa, especially if the patient is diabetic or immunocompromised. Signs of otitis media imply involvement of the facial nerve as it courses through the middle ear. The parotid gland and rest of the neck should be checked to rule out a parotid salivary gland mass that involves the facial nerve. Any of these associated ear findings should prompt ENT consultation because early intervention can improve outcome. Audiometry is recommended in the evaluation of facial paralysis because of the proximity of cranial nerves VII and VIII in the temporal bone.

In cases in which no identifiable cause is found, the diagnosis of Bell's palsy is made (though this diagnosis is not certain until a facial or acoustic neuroma has been ruled out either with return of facial function or with MRI scan). In the past, Bell's palsy was treated expectantly because the cause was not clear, making treatment difficult. Evidence now indicates that most Bell's palsy cases are secondary to a reactivation of the herpes simplex virus in the geniculate ganglion, causing neural edema and neurapraxia. On the basis of this research, treatment with antivirals and steroids are thought to improve outcomes. If no contraindications to steroids exist, prednisone (1 mg/kg, up to 60 mg, for approximately 7 days with a 7-day taper) is reasonable. In addition to steroids, an oral antiviral with activity against the herpesvirus is given (200 mg acyclovir [Zovirax] five times daily or valacyclovir [Valtrex] 500 mg twice daily for 7-10 days). Of the utmost importance is protection of the eye. Moisturizing eyedrops and nightly lubrication should be prescribed. Any signs of irritation should prompt an ophthalmology evaluation.

If the facial weakness is secondary to Ramsay Hunt syndrome, treatment is similar to that of Bell's palsy, but the clinical course and expected outcomes differ. This syndrome is caused by herpes zoster (rather than herpes simplex) involvement of the facial (geniculate), vestibulocochlear, and/or trigeminal ganglia. The infection causes pain and eventually vesicular eruptions around the auricle and external ear canal. Vesicles may appear only in the pharynx or hard palate in some cases. Facial weakness and at times dense paralysis are common. Hearing loss, tinnitus, and persistent vertigo also occur in 20% to 30% of patients (Adour, 1994). As with Bell's palsy, prompt initiation of oral steroids and acyclovir should begin when the diagnosis is suspected. This therapy can lessen vertigo and improve recovery of facial

nerve function, although outcomes are not as favorable as for Bell's palsy. Some patients struggle with persistent facial weakness, pain, and hearing loss (Robillard et al., 1988).

In cases of facial paralysis associated with otitis externa or otitis media, treatment differs. If malignant otitis externa is suspected, the patient requires hospital admission, control of diabetes, and infectious diseases and ENT consultation. IV antibiotics and sometimes surgical debridement are required. CNS complications are possible.

Facial weakness in the setting of AOM requires treatment with a broad-spectrum antibiotic covering the usual pathogens of otitis media. In addition, a myringotomy with or without tube insertion is thought to hasten resolution and improve outcomes by allowing decompression of the infection. This also allows a culture to be done and antibiotic sensitivities determined. If facial weakness occurs in the setting of chronic otitis media or known cholesteatoma, topical and systemic antistaphylococcal and antipseudomonal antibiotics should be started and an urgent ENT consultation obtained in hopes of preventing permanent facial paralysis and further complications.

Expected outcome for true Bell's palsy is full return of function in 80% of patients. The remaining 20% have variable recovery. If recovery is incomplete, MRI is indicated to rule out neoplastic process (most likely a facial neuroma) that could mimic Bell's palsy. Patients with diabetes mellitus have a higher incidence of Bell's palsy and often have a poorer outcome. Permanent facial weakness is also more common after herpes zoster oticus. Outcomes of facial paralysis secondary to the other causes discussed are variable and depend on the severity of the pathology, the patient's general state of health, and the response to treatment.

In patients who have poor return of facial function, rehabilitation is necessary. The most important goal is protection of the eye, followed by improved cosmesis. Ophthalmologic and ENT involvement is continued (Almeida et al., 2009).

The Nose and Paranasal Sinuses

Key Points

- A CT scan of the sinuses is not necessary to diagnose uncomplicated acute sinusitis. CT should be considered when the diagnosis is uncertain, treatment has failed, or a complication or neoplasia is suspected. MRI is useful in evaluation of sinus tumors.
- Adenoid hypertrophy is a common cause of nasal symptoms in children, but in an adult can indicate a lymphoproliferative disorder or HIV infection.
- A nasal foreign body should be suspected in a child who presents with recent unilateral nasal obstruction, rhinorrhea, and odor.
- Nasal polyps can be seen with asthma, cystic fibrosis, and rarely neoplasia.
- After allergen avoidance and antihistamines, topical corticosteroids are the mainstay of treatment for seasonal allergic rhinitis.
- Leukotriene receptor antagonists can reduce the symptoms of allergic rhinitis.
- Treatment to restore mucociliary function in sinusitis is as important as antimicrobial therapy.
- Unlike adults, children with sinusitis rarely complain of facial pain.
- Prompt diagnosis and treatment of a nasoseptal hematoma can prevent subsequent cartilage destruction.

History

Signs and symptoms of most nasal and sinus disorders include nasal congestion, rhinorrhea, bleeding, facial pressure, halitosis or pain, headache, cough, otalgia, facial or periorbital swelling, altered (diminished, absent, or distorted) sense of smell, or postnasal drainage. Initial evaluation of the patient with nasal complaints begins with a complete history, with specific questions directed at the timing and chronicity of the symptoms and modifying factors. The patient should be questioned specifically about previous nasal trauma.

Patients should be asked about prescription and over-the-counter nasal, sinus, and allergy medications. Many patients try OTC remedies before seeking medical advice. Excessive use of decongestant nasal sprays can exacerbate and even cause nasal obstruction secondary to *rhinitis medicamentosa* and rebound nasal congestion. The patient should also be questioned about previous nasal surgery. Rarely, the patient admits to unorthodox self-treatment that may be significant (e.g., peroxide irrigation, overzealous nasal cleaning). This can explain continued symptoms and may indicate an underlying problem. The underlying problem may be true nasal pathology or rarely may be obsessive-compulsive disorder manifesting as repeated nasal cleaning.

Information about past medical history and social history is also necessary. Knowledge of the patient's work environment may be relevant. Exposure to chemicals or fumes can cause nasal symptoms. Woodworkers are known to have a higher incidence of sinonasal carcinoma. A history of environmental allergies or immune dysfunction is relevant. Many medical conditions (e.g., asthma, autoimmune disorders) are associated with sinonasal dysfunction. Other conditions (e.g., hypertension) will limit the use of decongestants. History of migraine is noteworthy because migraine headaches can be confused with sinus pain. Some prescriptions can exacerbate or even cause nasal dysfunction, especially medications (antihistamines, diuretics, antidepressants) that can lead to excessive nasal dryness (sicca). Drug allergies should be noted. Previous nasal surgery, if done, may not have been successful or even led to increased problems. Cigarette smoking and excessive use of alcohol and caffeine have negative effects on mucociliary function that can lead to congestion. A history of nasal or facial trauma is important. Previous or current use of intranasal cocaine can lead to significant pathology and symptomatology.

Physical Examination

See the discussion online at www.expertconsult.com.

Radiography

Plain radiographs do not approach the sensitivity and specificity of CT but are useful in some cases. Plain films are often ordered in cases of facial trauma, especially isolated nasal trauma. The films can be complementary to the physical examination.

Nose and Paranasal Sinuses: Physical Examination

Examination of patients with nasal complaints should include a complete assessment of the face, oral cavity, oropharynx, nasopharynx, ears, lungs, nose, and sinuses. On initial inspection, it should be noted whether the patient prefers to breathe with the mouth open or closed. The face should be inspected for any visible asymmetry resulting from previous trauma or that can be associated with nasal and sinus tumors or polyps. The periorbital region should be inspected for swelling or erythema. The area over the maxillary and frontal sinuses should be palpated and percussed to elicit tenderness, and the external nasal skin should be inspected.

The oral cavity and oropharynx should be examined for evidence of mass effect, ulceration, or postnasal drainage. In children the size and appearance of the tonsils should be noted. The nasopharynx can be inspected indirectly with a small dental mirror to look for drainage or masses and to evaluate the adenoid size.

Examination of the ears might show coexisting otitis media with effusion that indicates sinus disease, adenoidal hypertrophy, or a nasopharyngeal mass. Auscultation of the lungs is performed because chronic sinusitis and pulmonary disease coexist in certain patient populations (COPD, asthma, cystic fibrosis, Wegener's granulomatosis, immotile cilia syndrome). The neck should be palpated, because sinus and nasopharyngeal neoplasms are often associated with cervical lymphadenopathy.

Intranasal examination is best done with a bright headlight and a nasal speculum. An otoscope with a large tip suffices and is often preferable in children. If a rigid or flexible endoscope is available, endoscopic examination of the nose provides optimum visualization of the entire nasal cavity but requires experience to perform properly. The appearance of the nasal mucosa should be inspected; healthy mucosa appears pink and moist. The quality of any rhinorrhea, if present, should be noted. The position of the nasal septum should be evaluated. Although most people have mild, asymptomatic septal deviation, moderate to severe septal deflections can cause nasal obstruction.

The inferior turbinates are easily seen low in the nasal cavity. They are sometimes mistaken for a mass or a polyp, especially if there is asymmetry between the right and left sides. The normal nasal cycle often causes the inferior turbinates to be of different size. Looking superiorly in the nasal cavity allows visualization of the middle turbinate. This is a critical structure because normal drainage of most mucus produced in the sinuses (~1 qt/day) converges on an area called the *ostiomeatal complex* or *ostiomeatal unit* (OMC or OMU), which empties into the *middle meatus*, the space under the middle turbinate. The superior turbinate is not visible without endoscopy. Inspection should be made for any abnormal lesions, ulcerations, recent bleeding, or (in children) foreign bodies. If significant rhinorrhea, nasal congestion, or septal deviation does not allow adequate inspection of the nasal cavity, ENT evaluation is necessary for a complete examination.

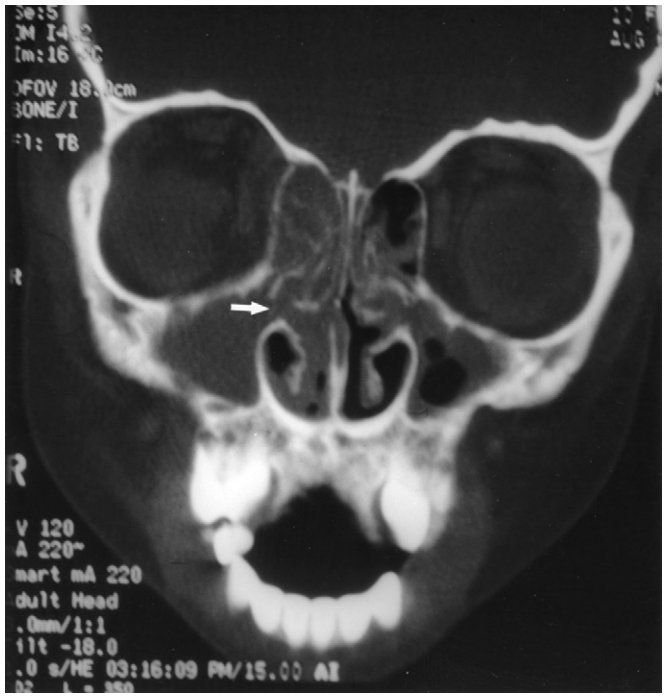


Figure 19-6 Coronal computed tomography scan showing complete opacification of the right maxillary and ethmoid sinuses with partial opacification of the left maxillary and ethmoid sinuses. Arrow indicates ostiomeatal complex.

Plain radiographs of the sinuses are still useful in certain circumstances. Plain sinus radiographs are reasonably accurate in assessing the maxillary and frontal sinuses in cases of *acute* sinusitis. Complete opacification or an air-fluid level in one of these sinuses usually indicates acute sinusitis. However, the relatively low sensitivity and specificity of plain films, especially in evaluating the ethmoid sinuses, have limited their usefulness.

Computed tomography has become an invaluable diagnostic tool for evaluating chronic nasal and sinus problems and has essentially supplanted the use of plain sinus films. CT of the sinuses allows unparalleled imaging of the complicated anatomy of the nose and paranasal sinuses. It has also increased understanding of the pathophysiology of sinusitis. CT scanning can show areas of mild mucosal thickening in the sinuses (indicating chronic sinusitis), complete opacification (seen in acute sinusitis, polyps, or sinus tumors), bone erosion, or abscess formation in adjacent critical structures such as the orbit or brain (Fig. 19-6). CT can show whether the *ostiomeatal complex* (the "bottleneck" of normal sinus drainage) is patent or obstructed and shows the myriad nasal and sinus normal variants, some of which predispose to sinonasal pathology.

Computed tomography of the sinuses should be ordered when the diagnosis of chronic sinusitis is suspected, medical treatment of sinusitis has failed and surgery is being contemplated, a complication of sinusitis is suspected, or a nasal or sinus mass is suspected. CT is not required as a confirmatory test in the treatment of uncomplicated acute sinusitis except in certain circumstances. The scan is helpful, however, in cases of *recurrent acute sinusitis* or when the diagnosis is not certain. Obtaining a scan during a patient's presumed infection allows the diagnosis to be confirmed or ruled out. Although some abnormalities require further treatment occasionally, the scan identifies abnormalities or variations of normal anatomy that require no intervention. Mucus retention cysts, for example, are seen in up to 20% of the population. Unless they are large or infection is suspected (the patient complains of pain in the vicinity of the cyst), no treatment is required.

Magnetic resonance imaging is not particularly helpful in evaluating sinusitis and has two main limitations in evaluating inflammatory sinus conditions. First, MRI often tends to be too sensitive, showing mucosal thickening that is clinically insignificant. Second, MRI fails to show bony anatomy, which is critical in diagnosis and surgical planning in chronic sinusitis. MRI is useful in evaluating suspected sinonasal tumors and fungal infections of the sinuses. The limitations of MRI and its relatively high cost compared with CT do not justify its routine use in evaluating chronic sinusitis. When incidental sinusitis is noted on an MR image and the degree of sinusitis is severe, is asymmetric, or the patient is symptomatic, treatment (and sometimes ENT referral) is indicated.

Clinical Problems

Complaints related to the nose and sinuses are among the most common seen in a family medicine practice. Acute rhinitis (the common cold), allergic rhinitis, and sinusitis compose the vast majority of these complaints and, taken together, result in an enormous socioeconomic impact in terms of missed workdays and schooldays and pharmaceutical costs. Nasal complaints are usually related to nasal congestion, rhinorrhea, bleeding, facial pressure or pain, headache, cough, otalgia, facial or periorbital swelling, altered (diminished, absent, or distorted) sense of smell, or postnasal drainage.

Epistaxis

See also earlier Emergencies section. (p. 300). Epistaxis can be caused by trauma, dry weather, hypertension, bleeding dyscrasias, anticoagulation therapy, and intranasal tumors. Adolescent boys with recurrent epistaxis and nasal obstruction might have juvenile nasopharyngeal angiofibroma. Epistaxis typically responds to conservative treatment, including nasal hydration with saline mist, nasal ointment, environmental humidification, avoidance of digital trauma, and control of hypertension if present. If bleeding continues to be a problem, the patient should be referred to an ENT consultant for a complete evaluation of the nasal cavities and possible cautery. Screening blood studies for coagulopathy may be necessary.

Nasal Obstruction

The sensation of unilateral or bilateral nasal obstruction is relatively common and can range from mildly annoying to extremely frustrating to the patient. Nasal obstruction may be associated with other symptoms such as rhinorrhea, lost or altered sense of smell, or facial discomfort. Nasal obstruction may result from pathology of the nasal cavity or nasopharynx. (**eTable 19-4** online summarizes the most

eTable 19-4 Differential Diagnosis of Nasal Stuffiness Based on Incidence, Age, and Gender

Incidence	Children (0-10 yr)	Adolescents (11-19 yr)	Adults (≥20 yr)
Common	Infection, viral Infection, bacterial Allergy Adenoid hypertrophy (young > old)	Infection, viral Infection, bacterial Allergy Nasoseptal deformities (M>F) Vasomotor rhinitis	Infection, viral Infection, bacterial Allergy Nasoseptal deformities (M>F) Rhinitis medicamentosa Environmental and occupational irritants (M>F)
Uncommon	Nasoseptal deformities Chronic sinusitis (bacterial) Septal hematoma Septal abscess Foreign body	Rhinitis medicamentosa Chronic sinusitis (bacterial) Septal hematoma Septal abscess Infectious mononucleosis Antrochoanal polyp (F>M)	Atrophic rhinitis Chronic sinusitis (bacterial) Metabolic or endocrine disorder Septal perforation Hypothyroidism Antrochoanal polyp (F>M) Diabetes mellitus Menses, pregnancy
Rare	Atrophic rhinitis Choanal atresia Cystic fibrosis Dysgammaglobulinemia Neoplasm	Atrophic rhinitis Angiofibroma (boys) Fibrous dysplasia (M>F) Rhinolith Tornwaldt's bursa	Malignant neoplasm Granuloma of pregnancy Midline lethal granulomatosis (Wegener's granulomatosis) Rhinolith Paget's disease

Modified from May M, West JW. The "stuffy" nose. Otolaryngol Clin North Am 1973;6:665.
M>F, Males (boys, men) affected more than females (girls, women); *F>M,* vice versa.

common causes, associated signs and symptoms, and treatment for nasal obstruction).

Physical Examination

See the discussion online at www.expertconsult.com.

Treatment

Successful treatment of nasal obstruction depends on making a correct diagnosis. Once the diagnosis has been established, a treatment plan should be developed. If the nasal obstruction is secondary to one of the various types of rhinitis, it is treated medically. This includes nasal steroids, antihistamines, leukotriene inhibitors, mucolytics, oral decongestants, topical decongestants, and nasal saline. These medications may be used alone or in various combinations. The choice of medications is determined by the severity of symptoms and the patient's medical history, response to treatment, and wishes. Oral steroids can be used in select severe cases but are associated with potential significant side effects. Nasal decongestant sprays are very effective for treating severe nasal congestion but should be used sparingly and never for longer than 3 days, to prevent rebound nasal obstruction (rhinitis medicamentosa). Allergy testing is done when allergies are suspected and the standard regimen is largely ineffective. Antibiotics are administered if a bacterial infection is suspected (acute rhinosinusitis).

Adenoid Hypertrophy

Adenoid hypertrophy is common in children. If identified in an adult, adenoid hypertrophy could indicate a lymphoproliferative disorder or HIV infection. The patient may present with nasal symptoms or symptoms of eustachian tube dysfunction. In the pediatric population, adenoid hypertrophy causes chronic or recurrent nasal obstruction, rhinorrhea, snoring, cough, or otitis media. The diagnosis is usually clinical but can be confirmed with lateral neck radiograph. If symptoms are severe or persistent, adenoidectomy is indicated; improvement is usually dramatic.

Foreign Body

A nasal foreign body should be suspected in a child with or without a history of previous nasal problems who presents with recent unilateral nasal obstruction, rhinorrhea, and odor. The nasal foreign body might not be visible secondary to the presence of mucosal edema, mucus, or pus.

If the foreign body is identified, removal may be attempted in a cooperative child. If removal is not possible or the diagnosis is uncertain, ENT consultation should be obtained. The ENT evaluation may be done in the office setting or in the operating room, depending again on patient cooperation and degree of suspicion. The nasal cavity is suctioned, decongested, and anesthetized with topical lidocaine. Endoscopy may be done. If the foreign body is seen, removal is undertaken.

If old enough, asking the child to blow the nose after decongestion might remove the foreign body or at least move it anteriorly. Removal can be difficult, and experience helps. Problems that can hinder removal include bleeding that obscures visibility. The foreign body can also be inadvertently pushed posteriorly. Softer foreign bodies, such as food matter and tissue paper, can disintegrate, requiring piecemeal removal.

A headlight and bivalve nasal speculum are recommended. Suction should be available. A small alligator or bayonet forceps is sometimes used, but may simply push the foreign body posteriorly. In many cases a useful instrument is a small, ball-tipped, right-angle probe, actually an otologic surgical instrument called an "attic hook." This can be gently passed posterior to the foreign body, turned 90 degrees, and then used to pull the foreign body anteriorly and out of the nose.

Once the foreign body is removed, the nasal cavity should be reinspected for retained, more distal foreign bodies. The other nasal cavity and ears should also be inspected because the child might be a "repeat offender." Antibiotics are recommended if there is evidence of obvious infection or complete removal is not certain and reexamination is planned.

Nasal Vestibulitis

A low-grade infection of the anterior nasal vestibule will cause chronic irritation, crusting, and sometimes bleeding. The examination typically shows mild erythema, cracking, and yellow crusting just inside the nostril, but it may be fairly normal. The etiology is usually *Staphylococcus aureus* but may be fungal. Herpetic infections are typically more severe and not as protracted. Treatment is with OTC topical antibiotic ointment (without neomycin because of patient sensitivity) and avoidance of irritating the area. If symptoms continue, methicillin-resistant *S. aureus* (MRSA) infection is possible and should respond to mupirocin ointment. Continued symptoms require ENT consultation.

Choanal Atresia

Choanal atresia is a common cause of nasal obstruction in children but can also be seen in adults. If bilateral, it manifests shortly after birth as an airway emergency, because neonates are "obligate nasal breathers" and cannot tolerate nasal obstruction. Typically they will oxygenate well while crying but will become cyanotic when crying stops and they cannot feed. This condition requires urgent ENT consultation. The airway is stabilized and the atresia repaired shortly thereafter. If unilateral, the atresia can go undiagnosed until later in childhood or even adulthood. The patient will report a lifelong history of nasal obstruction and rhinorrhea. Diagnosis is made with endoscopy and CT. Treatment is surgical.

Nasal Polyps

Nasal polyps are the result of nasal mucosal inflammation and edema. On examination, nasal polyps are usually silver-gray in color and may be translucent. If there is associated infection, polyps can appear erythematous or may be obscured by mucus. Polyps cause significant and sometimes complete nasal obstruction but are painless and insensate. Nasal polyps predispose the patient to sinusitis and often cause anosmia.

The exact cause of nasal polyps is unclear. Polyps are often associated with reactive airway disease and less often with environmental allergies. In children the presence of polyps should prompt testing for cystic fibrosis. Sinonasal tumor or fungal involvement should be considered, especially if the polyps are unilateral. If polyps are identified, further evaluation includes allergy and asthma testing and CT scan.

Nasal Obstruction

Physical examination should include a complete examination of the face, oral cavity, pharynx, and internal and external nose. The patients should be observed (while unaware) for signs of mouth breathing while at rest. This is especially important in children whose predominant symptom of nasal obstruction might be mouth breathing. Inspection should be made for stigmata of allergic rhinitis: circles under the eyes ("allergic shiners"), or a crease on the bridge of the nose secondary to the allergic salute. Children with long-standing nasal obstruction (often from adenoid hypertrophy) often have a classic appearance of open-mouth posture, periorbital edema, and narrow face: the *adenoid facies.* Previous nasal trauma may be evidenced by obvious external nasal deformity. The oral cavity and pharynx should be inspected for enlargement of the tonsils, which often coexists with adenoid hypertrophy. Thick drainage along the posterior oropharyngeal wall can indicate coexistent sinusitis. The palate should be examined for distortion or ulceration from a neoplasm arising in the sinuses or the nasal cavity. An attempt may be made to visualize the nasopharynx indirectly with a headlight and dental mirror.

Examination of the nasal cavity is done systematically. The nasal septum is inspected for evidence of significant septal deviation. The size of the inferior turbinates is assessed. The presence and quality of any rhinorrhea is noted. The presence or absence of polyps or other intranasal masses must be confirmed. Several diagnoses are listed in **eTable 19-4.**

Medical treatment is initially offered but is often inadequate. Initial treatment includes topical steroids, allergy treatment, and treatment of sinusitis. In many patients, endoscopic sinus surgery is an important adjunct to medical treatment and results in significant improvement in symptoms. Unfortunately, polyps often recur after surgery, requiring repeated removal.

Deviated Septum

Most patients have some degree of asymptomatic septal deviation, but in some patients it is severe enough to cause symptoms of obstruction. Septal deviation is usually the result of previous nasal trauma. The trauma might have seemed relatively minor at the time or might have resulted in a nasal fracture. Some deviated septums are congenital. Physical examination may clearly demonstrate the septum obstructing the nasal airway if anterior. If more posterior, nasal endoscopy or CT may be necessary to make the diagnosis. Any patient complaining of persistent nasal obstruction deserves further evaluation, especially if the cause is not immediately evident. Symptomatic septal deviation is readily treatable with outpatient surgery.

Septoplasty is done through an intranasal incision, allowing deviated portions of cartilage and bone to be replaced to the midline or removed, resulting in a symmetrically patent nasal airway. Septoplasty is often combined with a turbinate reduction procedure. The procedures are usually well tolerated. Postoperative pain, formerly a greater problem, usually resulted from the need for nasal packing and removal. Newer devices such as soft-silicone (Silastic) splints now cause much less postoperative discomfort than traditional packing.

In pediatric patients, septoplasty is not usually recommended because of concern about disrupting nasal and facial growth, although this risk appears to be low. For this reason, "limited" septoplasty may be considered in select patients.

Hypertrophied Turbinates

Inferior turbinate hypertrophy is relatively common in adults and children. This usually occurs with chronic inflammation, usually resulting from allergy or rhinosinusitis. Turbinate hypertrophy usually responds to medical treatment addressing the primary problem. If the turbinates remain significantly hypertrophied despite medical treatment, turbinate reduction is offered, using cautery, radiofrequency treatment, fracture, excision, laser treatment, or cryotherapy. Submucosal resection of a portion of the conchal bone and stromal tissue seems to provide the greatest success.

Rhinitis

Allergic Rhinitis

Seasonal allergic rhinitis affects 10% to 30% of adults and up to 40% of children in the United States. Although half the patients with allergic rhinitis have symptoms for only 4 months per year, 20% experience symptoms more than 9 months per year. Direct medical costs of treating this condition plus indirect costs of lost productivity and absences from work or school are estimated at up to $2.4 billion per year in the United States. Unfortunately, seasonal allergic rhinitis is not self-limiting and often coexists with more serious conditions such as asthma, sleep apnea, nasal polyps, sinusitis, and OME.

Understanding the immunologic mechanisms of seasonal allergic rhinitis directs the physician to the appropriate therapy. Allergens such as pollens are deposited on the nasal mucosa and processed by macrophages. The allergens are then brought to T lymphocytes and B cells, the latter producing immunoglobulin E (IgE), which in turn attaches to receptors on mast cells and basophils, causing the release of histamine and other inflammatory substances. The action of these substances on the nasal mucosal cells and nerve endings produces the localized symptoms of nasal discharge, nasal congestion, sneezing, and itching. This is considered the early or immediate phase of the allergic reaction. The delayed-phase response can occur 6 to 8 hours after the initial exposure, even when there are no allergens present. A continued influx of immune cells into the nasal mucosa causes a recurrence of symptoms.

The annual cycle of allergens causing allergic rhinitis begins in the early spring with the flowering of deciduous trees, followed by the release of pollen from grasses during the summer months and from weeds during the late summer months and early fall. Mold spores can be present throughout the year but increase during the warm months.

The diagnosis of seasonal allergic rhinitis is made primarily by history. Differentiation from the common cold is through examination of the nasal mucosa. A pale-pink to blue, boggy nasal mucosa usually indicates allergic rhinitis, whereas a red mucosa is more likely the result of a viral infection or nonallergic vasomotor rhinitis. A horizontal nasal crease on the nose of children and allergic "shiners" are other physical signs to look for in making the diagnosis. Seeing eosinophils under the microscope from a nasal smear using a Wright's stain also points to a diagnosis of allergic rhinitis. Skin testing for specific allergens may be done when the symptoms warrant. The term *perennial rhinitis* simply indicates that the symptoms are present throughout the year and are often caused by allergens (dog or cat dander, mold, dust mites).

The treatment is the same for perennial rhinitis and for seasonal allergic rhinitis. The link among seasonal allergic rhinitis, asthma, and sinusitis is based on the one-airway theory. Because the mucosa in each of those areas consists of basement membrane, a capillary system, mucous glands, goblet cells, and nervous innervation, each area reacts similarly to allergens and responds to like treatments.

After allergen avoidance, antihistamines are most often used to treat seasonal allergic rhinitis. They can also be beneficial for asthma, although they have not been traditionally used as part of asthma treatment. The second generation of nonsedating antihistamines has shown clear benefits in treating seasonal allergic rhinitis and asthma (Grant et al., 1995). (**eTable 19-5** online shows the onset of action, sedating properties, cardiac side effects, and dosage regimens of first- and second-generation antihistamines.) Cetirizine (Zyrtec) and loratadine (Claritin) can be taken with food, whereas fexofenadine (Allegra) is less well absorbed when taken with food. A topical antihistamine, azelastine (Astelin) has shown efficacy in treating allergic rhinitis.

Adding a decongestant such as pseudoephedrine or phenylephrine can help relieve nasal congestion through its vasoconstrictor properties. The patient should be cautioned about

eTable 19-5 Characteristics of Selected First- and Second-Generation Oral Antihistamines

Antihistamine	Onset of Action (hr)	Sedation	Possible Cardiac Side Effects	Formu-lation	Recommended Dosing Regimens
First-Generation Antihistamines					
Brompheniramine (Rondec, Dimetane, Bromphen)	1	Yes	No	Elixir	6-12 yr: 1 tsp q4-6h ≥ 12 yr: 2 tsp q4-6h
Chlorpheniramine maleate (Chlor-Trimeton)	1	Yes	No	Tablets	6-12 yr: 2 mg q4-6h ≥ 12 yr: 4 mg q4-6h
Diphenhydramine hydrochloride (Benadryl)	1	Yes	No	Tablets Liquid	6-12 yr: 12.5-25 mg q4-6h ≥ 12 yr: 25-50 mg q4-6h 6-12 yr: 12.5-25mg q4-6h ≥ 12 yr: 25-50 mg q4-6h
Second-Generation Antihistamines					
Cetirizine (Zyrtec)	1-2	Yes	No	Tablets Syrup	≥ 12 yr: 5-10 mg qd ≥6 yr: 5-10 mg qd
Fexofenadine (Allegra)	1-2	No	No	Capsules	≥12 yr: 60 mg bid
Loratadine (Claritin)	1-2	No	No	Tablets Syrup Reditabs	≥6 yr: 10 mg qd ≥6 yr: 10 mg qd ≥6 yr: 10 mg qd
Azelastine (Astelin)	0.5-1	Yes	No	Nasal spray	≥12 yr: 2 sprays each nostril bid

Modified from Corren J, Storms W, Young T. Contemporary issues in the management of allergic disorders: an update for the family physician. Clin Courier 1999;17:1.
q4-6h, Every 4 to 6 hours; *qd,* once daily; *bid,* twice daily.

Table 19-5 Doses for Nasal Corticosteroids

Nasal Spray	Age: Dosage
Mometasone furoate (Nasonex)	2-12 yr: 2 sprays each nostril qd (200 μg/day)
Fluticasone propionate (Flonase)	4-16 yr: 1-2 sprays each nostril qd (100-200 μg/day) ≥15 yr: 2 sprays each nostril qd (200 μg/day)
Budesonide (Rhinocort, Pulmicort)	≥6 yr: 2 sprays each nostril bid or 4 sprays each nostril qd (256 μg/day)
Beclomethasone diproprionate (Beconase, Vancenase, Beconase AQ)	6-12 yr: 1 spray each nostril bid (168 μg/day) ≥12 yr: 1 or 2 sprays each nostril bid (168 or 336 μg/day)
Flunisolide	6-14 yr: 1 spray each nostril tid or 2 sprays each nostril bid (1500-2000 μg/day) ≥14 yr: 2 sprays each nostril bid or tid (200-300 μg/day)
Triamcinolone acetonide (Nasacort AQ)	2-5 yr: 1 spray each nostril daily 6-12 yr: 2 sprays each nostril qd (200 μg/day) ≥12 yr: 2 sprays each nostril qd or bid (220-440 μg/day)

qd, Daily; *bid,* twice daily; *tid,* three times daily.

side effects such as nervousness, irritability, and insomnia when taking decongestants. Decongestants should be used carefully in patients with hypertension and with symptoms of prostatic hypertrophy.

Topical corticosteroids are effective in reducing the inflammation of the late-phase reaction (Weiner et al., 1998). The onset of action is within 24 to 72 hours, with full effects within 1 to 2 weeks. The initial dose may be two to three times daily for the nasal corticosteroids, followed by a reduction in the dose for maintenance (Jacobsen, 2001). The main side effects of corticosteroid nasal sprays are epistaxis (5% of patients) and nasal dryness (10%). These can be managed by using saline nose drops or a small amount of petroleum jelly before insufflation of the corticosteroid. No sign of nasal mucosal atrophy has been seen after as long as 1 year of therapy.

There is no evidence that nasal corticosteroids suppress adrenal function, but because of the concern about the effect of topical corticosteroids on the hypothalamic-pituitary-adrenal axis, several newer nasal corticosteroids have been developed to be less well absorbed. Mometasone (Nasonex) and fluticasone (Flonase) are less than 2% bioavailable, whereas budesonide (Rhinocort) and beclomethasone (Beconase, Vancenase) are 11% and 17% bioavailable, respectively. Age-appropriate dosages for the nasal corticosteroids are shown in Table 19-5.

Leukotriene receptor antagonists have been shown to inhibit the early phase of antigen response and to attenuate the late-phase inflammatory response. Several randomized, placebo-controlled trials have proved that antileukotrienes can reduce the symptoms of allergic rhinitis and improve the quality of life (Meltzer, 2002). In addition, they reduce nasal congestion and improve the sense of smell. Zafirlukast (Accolate) and montelukast (Singulair) are effective when patients either refuse or fail antihistamines and nasal sprays and can be used in combination with antihistamines. Zafirlukast can cause liver enzyme abnormalities and should not be prescribed for children younger than 12 years. Montelukast has no serious side effects and can be prescribed for children as young as 6 years.

Patients who respond poorly to pharmacotherapy, who develop adverse side effects, or who simply wish to use an alternative to pharmacotherapy may consider immunotherapy. They must realize the commitment to this therapy is 3 to 5 years, and that local reactions are seen in 15% of patients and systemic reactions in 0.5%. A cost comparison between immunotherapy and medication done under the auspices of the American Academy of Allergy, Asthma, and Immunology demonstrated a significant reduction in cost of single-injection immunotherapy over medication in allergic rhinitis (Huggins and Looney, 2004).

The family physician is crucial to the diagnosis and treatment and can educate patients about the importance of allergen avoidance, adherence to the treatment regimen, and potential side effects of the medications.

KEY TREATMENT

As immunotherapy for allergic rhinitis, montelukast shows efficacy similar to loratadine but less than intranasal fluticasone.
Montelukast used concomitantly with loratadine or cetirizine shows increased benefit compared with either drug alone (Nayak and Langdon, 2007).
Intranasal steroids continue to be the mainstay of treatment for allergic rhinitis. For steroid-intolerant patients, however, montelukast with loratadine or cetirizine can produce comparable results.
Intranasal antihistamine therapy is effective for both allergic rhinitis and vasomotor rhinitis (Kaliner, 2007).
SOR: A

Vasomotor Rhinitis and Idiopathic Rhinitis

The term "vasomotor rhinitis" is a misnomer because there is no inflammation. The primary symptoms are a feeling of nasal congestion and rhinorrhea. Box 19-7 lists some of the conditions that are included in the category of vasomotor rhinitis. Allergy skin tests in patients with vasomotor rhinorrhea are negative, with less than 25% eosinophils present on a nasal swab. These patients do not fully respond to topical or systemic corticosteroids. The condition suggests hyperactivity of parasympathetic tone, blockage of sympathetic tone with vasodilation of submucosal venous sinusoids, and excessive seromucous secretions from the mucous glands. A good analogy to vasomotor rhinorrhea is functional bowel disease. Treatment is with systemic decongestants and antihistamines or topical anticholinergic agents such as ipratropium bromide (Atrovent 0.06% nasal spray). Because of the persistence of symptoms, patients should be warned about excessive use of OTC nasal sprays, which can lead to rhinitis medicamentosa.

Rhinitis Medicamentosa

The prolonged use of topical decongestants for the nose can itself induce nasal stuffiness. The condition is caused by *rebound swelling* after dissipation of the decongestive effect

Box 19-7 Conditions Included under Vasomotor Rhinitis

Drug-induced rhinitis (reserpine, nonselective β-blockers, antidepressants, oral contraceptives)
Irritant rhinitis (smoke, gases)
Temperature- and humidity-induced rhinitis
Emotion- and stress-induced rhinitis
Hormonal rhinitis (pregnancy, premenstrual, hypothyroidism)
Idiopathic rhinitis

Modified from Mikaelian AJ. Vasomotor rhinitis. Ear Nose Throat J 1999;68:207.

of the nasal spray. Increasing the dose of the spray is the patient's response to the rebound swelling, and the vicious cycle is difficult to break without education and medical help.

To treat rhinitis medicamentosa, the patient must stop using the topical decongestant to allow recovery of the damaged nasal mucosa. To relieve the subsequent rebound mucosal swelling, topical and oral corticosteroids are recommended. The length of time needed to successfully treat rhinitis medicamentosa varies depending on the duration the patient has used nasal decongestants. It takes at least 2 weeks to reverse the edema and histamine sensitivity. Other forms of treatment include systemic antihistamines or decongestants, corticosteroid injection into the inferior turbinate, and nocturnal sedation. Surgery is helpful if nasal septal deviation is present. Graf and colleagues (1995) achieved a 100% success rate at the end of 6 weeks of nasal corticosteroid therapy and avoidance of nasal decongestants. It takes time and patience to educate the patient about rhinitis medicamentosa, and both are essential for the treatment to be successful.

Atrophic Rhinitis

Elderly patients are more prone to develop atrophic rhinitis, which leads to nasal congestion, crusting, and foul odor. Treatment consists of saline nose sprays and topical antibiotics. Atrophic rhinitis can also result from previous nasal surgery, use of cocaine, and autoimmune or systemic inflammatory disorders (e.g., lupus, Wegener's granulomatosis). If the cause is unclear, further workup is indicated.

Sinusitis

Symptoms of rhinitis and sinusitis are often very similar and even difficult to differentiate in many cases. Sinusitis implies inflammation of the mucosa of one or more of the paranasal sinuses. This usually coexists with rhinitis and is actually more accurately referred to as *rhinosinusitis.* Studies have shown that CT scans of patients with uncomplicated viral upper respiratory infections (URIs) have mucosal thickening and opacification of the sinuses. For this reason, most URIs are technically considered *viral rhinosinusitis.* In most cases, these changes resolve with time and symptomatic treatment. The terms *rhinosinusitis* or *sinusitis* are typically used when a bacterial infection of the sinuses is suspected. About 5% of viral URIs will progress to bacterial rhinosinusitis. An estimated 20 million cases of bacterial sinusitis occur in the United States annually, accounting for 9% and 21% of all pediatric and adult antibiotic annual prescriptions, respectively (Sinus Partnership, 2004).

Inflammatory conditions of the paranasal sinuses cause significant socioeconomic impact annually, secondary to considerable medical expense and missed workdays. Chronic sinusitis can be quite debilitating. Studies have shown that quality-of-life scores of patients with chronic sinusitis are often similar to those of other, more severe conditions (CHF, COPD). Chronic sinusitis can also exacerbate coexisting medical conditions, most notably reactive airway disease.

Sinusitis represents one of the most common disorders requiring antibiotic treatment in adults. The challenge to the clinician in evaluating the patient with possible sinusitis is to differentiate viral URI, allergic rhinitis, and even a migraine headache, which do not require antibiotics, from bacterial sinusitis, which does respond to antibiotic treatment. There still seems to be a public perception that antibiotics hasten recovery from the common cold. Some physicians prescribe antibiotics in these situations, not wanting to disappoint the patient and seeing no significant risk. In fact, evidence suggests that there is a greater likelihood of *harming rather than benefiting* the patient with inappropriate use of antibiotics (Scott and Orzano, 2001). The emergence of bacteria highly resistant to broad-spectrum antibiotics has forced the medical community to modify its behavior regarding the treatment of URIs. Antibiotics should not be prescribed unless a bacterial infection is certain or at least probable. The patient should be educated about the rationale for this and usually responds favorably.

The underlying cause of most cases of sinusitis is mucociliary dysfunction and sinus obstruction. The maxillary sinuses, anterior ethmoid sinuses, and frontal sinuses all drain through small ostia that converge into a small channel called the *ostiomeatal unit,* which then empties into the middle meatus, beneath the middle turbinate . Obstruction at the ostiomeatal unit leads to obstruction of these sinuses and secondary infection. The posterior ethmoid sinuses and sphenoid sinuses are usually affected later. Sinusitis most often follows a viral URI or an episode of allergic rhinitis. Mucosal edema, impaired local immunity, and ciliary dysfunction lead to impaired sinus drainage and mucus stasis, followed by bacterial infection. Less frequently, sinusitis can result from direct bacterial contamination from an infected tooth or trauma.

Sinusitis is classified into *acute* (symptoms up to 3 weeks), *subacute* (symptoms from 3 to 6 weeks), and *chronic* (symptoms longer than 6 weeks) cases. Cases of acute sinusitis that clear completely only to develop again quickly are referred to as *recurrent acute sinusitis.* Although the types of sinusitis share many characteristics, there are several critical differences in pathogenesis and treatment.

The most important risk factor for the development of sinusitis is rhinitis (e.g., viral, allergic). Other risk factors include anatomic abnormalities (abnormality within the sinuses, septal deviation, choanal atresia, foreign body, adenoid hypertrophy), nasal polyps (which can also occur secondary to chronic sinusitis), conditions of local or systemic immunodeficiency, cystic fibrosis, primary ciliary dysfunction (Kartagener's syndrome), secondary ciliary dysfunction (cigarette smoking, nasal decongestant abuse, cocaine abuse), gastroesophageal reflux disease (GERD), systemic inflammatory conditions (sarcoidosis, Wegener's granulomatosis), dental disease, and nasal or sinus tumors. Any of these conditions can mimic or cause rhinosinusitis. Further

workup or referral is indicated if a patient continues to struggle with nasal or sinus symptoms despite medical therapy.

The diagnosis of sinusitis is initially clinical. Imaging and cultures are not initially indicated (Reider, 2003). In 1996 the Task Force on Rhinosinusitis sponsored by the American Academy of Otolaryngology–Head and Neck Surgery developed diagnostic criteria for sinusitis. The signs and symptoms of sinusitis are divided into major and minor. *Major* signs and symptoms include facial pain and pressure, nasal congestion and obstruction, nasal discharge, discolored posterior discharge, anosmia or hyposmia, fever (acute only), and purulence on intranasal examination. *Minor* signs and symptoms include headache, otalgia or ear pressure, halitosis, dental pain, cough, fever (nonacute), and in children, fatigue and irritability. The diagnosis of sinusitis is *probable* if the patient has two or more major factors *or* one major and two or more minor factors. A *suggestive* history is indicated by the presence of one major factor or two minor factors.

Microbiology of sinusitis varies according to its chronicity. Acute sinusitis is most often *initially* viral. If symptoms persist, the likelihood of bacterial infection increases. Bacteria most often involved in acute sinusitis are *Pneumococcus* spp., *Haemophilus influenzae,* and *Moraxella catarrhalis,* with beta-lactamase production common in all these. Chronic sinusitis is caused by the same bacteria as in acute sinusitis, but anaerobic bacteria, *Pseudomonas* spp., and staphylococci become involved more often. The incidence of antibiotic-resistant bacteria, including MRSA and multidrug-resistant *Pneumococcus,* seems to be increasing. Polymicrobial infections are not uncommon.

Sinusitis can also be caused by fungi. *Invasive* fungal sinusitis (caused most often by *Aspergillus* or *Mucor* spp.) can be seen in patients with impaired immune function and poorly controlled diabetes. It is life threatening even with aggressive medical and surgical treatment. Much more common is a more indolent fungally mediated sinusitis. *Allergic fungal sinusitis* is seen in patients with normal immune function. This is often seen in association with nasal polyps and is thought to be the result of an aberrant immune response to the fungus rather than a true infection. Patients do not always have type I hypersensitivity to fungi. Secondary bacterial infection is often associated with this problem.

Rarer causes of sinusitis are secondary to mycobacterial or parasitic infection.

Complications of Sinusitis

Most cases of sinusitis would resolve with or without medical treatment. Sinusitis is usually treated, however, to avoid potential complications and hasten recovery. The proximity of the paranasal sinuses to the orbits and brain potentially allows infection to spread to these locations. Orbital and CNS involvement of sinusitis can lead to loss of vision and can be life threatening and therefore requires early recognition and treatment. Table 19-6 lists the potential complications of sinusitis and treatment recommendations. A high degree of clinical suspicion is required in cases of possible complicated sinusitis, especially in young children. Patients with a recent URI who present with periorbital erythema, vision change, increasing or severe headache, high fever, or altered mental status require *urgent evaluation* and treatment. Ophthalmologic, infectious diseases, and ENT consultations are obtained in cases of orbital complication. Periorbital and orbital cellulitis usually can be managed with intravenous antibiotics. The more severe orbital complications, however, usually require drainage procedures in combination with IV antibiotics. Surgical drainage also allows cultures to be obtained. Recovery from orbital complications is usually complete with prompt and aggressive treatment. Permanent vision impairment can occur even after appropriate treatment.

Table 19-6 Complications of Sinusitis

Complication	Physical Findings	Treatment
Periorbital cellulitis	Periorbital erythema, edema	Antibiotics: PO or IV
Orbital cellulitis	Erythema, edema, proptosis ± vision loss	IV antibiotics, close observation
Orbital abscess	Erythema, edema, proptosis ± vision loss	IV antibiotics + drainage, FESS
Cavernous sinus thrombosis	Erythema, edema, proptosis + vision loss	IV antibiotics + FESS
Meningitis	Headache, altered mental status, nuchal rigidity, fever	IV antibiotics ± FESS
Intracranial abscess	Headache, altered mental status, high fever	IV antibiotics + drainage, FESS
Mucocele or pyocele	Facial swelling ± fever ± pain	Drainage

FESS, Functional endoscopic sinus surgery.

The CNS complications require neurosurgical, ENT, and infectious diseases consultation. High-dose IV antibiotics are administered. Surgical drainage of the sinuses is sometimes recommended to treat the nidus of the infection and identify the exact pathogen. Recovery from CNS complications is more variable and depends on the patient's age and medical history, severity of the infection, and response to treatment.

Although not always complicated infections, sphenoid and frontal sinusitis deserve special mention. In some cases, drainage of the frontal sinuses is compromised. Chronic and recurrent frontal sinusitis can lead to both intracranial and ophthalmologic complications if untreated. Large *mucoceles* or *mucopyeloceles* can also form within the frontal sinus, causing disfigurement and diplopia. These conditions usually require surgical drainage. Similarly, sphenoid sinusitis can rarely be aggressive. The carotid artery and optic nerves traverse the lateral walls of the sphenoid sinuses. The sphenoid sinus occupies a space inferior and anterior to the cranial vault. Acute or long-standing sphenoid sinusitis can progress to CNS or eye complications, or both. If frontal or sphenoid sinus involvement is noted on CT scan, ENT evaluation is usually indicated.

Medical Treatment of Acute Sinusitis

Treatment of acute sinusitis is almost always medical. Medical treatment of sinusitis, in general, is intended to restore normal mucociliary function, eradicate infection, and improve patient symptoms. Treatment to restore mucociliary function is critical and is as important as antibiotic treatment.

Improved mucociliary function allows the patient's local immunity to function better and often leads to resolution of the infection.

The patient's medical history, including allergies, must be considered. Patients with poorly controlled hypertension or coronary artery disease may not tolerate decongestants. In acute cases of sinusitis, mucociliary function can be improved by a combination of medications, including oral or topical decongestants (topically for less than 3 days), mucolytics (guaifenesin), and nasal toilet (saline mist or irrigations). Nasal saline irrigations are available over the counter or can be homemade. Both 0.9% isotonic saline and hypertonic saline irrigations are extremely beneficial. Nasal steroids are *not indicated* for acute sinusitis but may decrease symptoms and hasten recovery in some patients. Antihistamines are usually not helpful, unless there is a strong allergic component, and can actually be counterproductive by increasing mucus viscosity and mucosal dryness. Oral steroids are usually not indicated in acute sinusitis, but they may be helpful in select patients.

This leaves the practitioner with the responsibility of using good clinical judgment to appropriately prescribe antibiotics to treat acute sinusitis. Antibiotics are empirically chosen based on the expected pathogens and local antibiotic-resistance patterns. The high incidence of beta-lactamase–producing strains of *H. influenzae* and *M. catarrhalis* and the penicillin-resistant pneumococci must be considered. More prudent use of antibiotics seems to have resulted in a plateau of the emergence of antibiotic resistance of these pathogens. MRSA seems to be more common, however, especially in chronic sinusitis.

According to Cochrane Collaboration recommendations for treatment of acute sinusitis, antibiotics provide a minor improvement in simple, acute (uncomplicated) sinus infections. However, 8 of 10 patients improve without antibiotics within 2 weeks. The small benefit gained may be overridden by the negative effects of antibiotics, both on the patient and on the population in general. For acute sinusitis confirmed by radiology or nasal endoscopy, current evidence is limited but supports the use of *intranasal steroids for acute sinusitis* as a monotherapy or as an adjuvant therapy to antibiotics. Clinicians should weigh the modest but clinically important benefits against possible minor adverse events when prescribing therapy.

Many antibiotics are indicated for the treatment of acute bacterial rhinosinusitis. In addition, there are antibiotics that do not have Food and Drug Administration (FDA) approval for treatment of sinusitis but are still appropriately used.

The Sinus and Allergy Health Partnership (2004) made the following comprehensive recommendations regarding the treatment of acute rhinosinusitis:

1. A bacterial infection should be suspected if symptoms of a viral URI do not improve after 10 days, or if symptoms worsen after 5 to 7 days.
2. Antibiotic resistance is common. Specifically, intermediate resistance of *Streptococcus pneumoniae* to penicillin (PCN) is 15%, and complete resistance is estimated at 25%. Resistance of *S. pneumoniae* and *Haemophilus influenzae* to trimethoprim-sulfamethoxazole (TMP-SMX) is common, as is resistance of *S. pneumoniae* macrolides. Beta-lactamase production of *H. influenzae* and *Moraxella catarrhalis* is 30% and 100%, respectively.
3. Selection of an antibiotic should be based on severity of symptoms, whether the patient has received an antibiotic in the last 4 to 6 weeks, and the response to current antibiotic therapy after 72 hours. Mild symptoms include rhinorrhea and fatigue. Moderate symptoms include congestion, low-grade fever, and facial pain.
4. The widespread use of fluoroquinolones for mild sinusitis may promote resistance to this class of antibiotics.
5. Antibiotic choices for adults with mild disease and no recent antibiotics include amoxicillin (1.75-4 g/day, with or without clavulanate), cefpodoxime proxetil, cefuroxime axetil, or cefdinir. TMP-SMX, doxycycline, azithromycin, erythromycin, and clarithromycin may be considered in PCN-allergic patients, but the failure rate may be as high as 20% to 25%. Failure of therapy should prompt reevaluation of the patient or a switch in therapy.
6. Antibiotic choices for adults with moderate disease or with mild disease who have received recent antibiotics include amoxicillin-clavulanate (4 g/day) or a respiratory fluoroquinolone (levofloxacin or moxifloxacin). Ceftriaxone (1-2 g parenterally for 5 days) or combination therapy for gram-positive and gram-negative bacteria may also be considered. Failure of therapy should prompt reevaluation of the patient, CT scan, endoscopy with culture, or a switch in therapy.
7. Antibiotic choices for children with mild disease and no recent antibiotics include amoxicillin (90 mg/kg/day, with or without clavulanate), cefpodoxime proxetil, cefuroxime axetil, or cefdinir. TMP-SMX, doxycycline, azithromycin, erythromycin, and clarithromycin may be considered in PCN-allergic patients (especially immediate type I hypersensitivity), but the failure rate may be as high as 20 to 25%. If the patient has a true type I hypersensitivity to beta-lactams, desensitization, sinus culture, CT scan, or other intervention may be necessary. Less severe reaction may allow use of another beta-lactam antibiotic. Failure of therapy should prompt reevaluation of the patient or a switch in therapy.
8. Antibiotic choices for children with moderate disease or with mild disease who recently received antibiotics include amoxicillin-clavulanate (90 mg/kg/day). Cefpodoxime proxetil, cefuroxime axetil, or cefdinir may be used if there is a nonsevere PCN allergy (rash). Cefdinir is preferred because of high patient acceptance. Ceftriaxone (50 mg/kg/day parenterally for 5 days) or combination therapy for gram-positive and gram-negative bacteria may also be considered. Failure of therapy should prompt reevaluation of the patient, CT scan, endoscopy with culture, or a switch in therapy.

As recurrence or severity of the infection increases, broader-spectrum antibiotics are indicated. Macrolides, fluoroquinolones, augmented penicillins, and cephalosporins are useful in these cases. Culture-directed antibiotic treatment may be indicated in more refractory cases. Obtaining a culture usually requires an ENT referral, because simply swabbing the nasal cavity is not reliable. Cultures can be obtained from an endoscopically guided middle meatus swab. Maxillary sinus aspiration can also be done but is more invasive and not much more accurate than a middle meatal culture. Adjunctive treatment to help improve mucociliary function becomes more important as recurrence increases.

KEY TREATMENT

The American Academy of Otolaryngology–Head and Neck Surgery produced a consensus statement of Clinical Practice Guidelines for Treatment of Presumed Sinusitis (Rosenfeld et al., 2007). The following summary guidelines are for acute viral sinusitis (VRS), presumed acute bacterial rhinosinusitis (ABRS), and chronic rhinosinusitis (CRS). The key symptoms of sinusitis were rhinorrhea, nasal obstruction, and facial pressure or pain.

STRONGLY RECOMMENDED TREATMENT

The quality of data supporting the benefits of treatment outweighing the potential harm is strong (Grade A, B):

1. Clinicians should attempt to differentiate between viral and bacterial sinusitis.
2. The level of pain should be assessed when treating ABRS.

RECOMMENDED TREATMENT

The benefits of treatment outweigh the risks, but the data are not as strong (Grade B, C):

1. Imaging studies are not recommended for cases of uncomplicated VRS.
2. If a decision is made to treat ARS, amoxicillin should be used as first-line therapy if no PCN allergy.
3. If the patient worsens or fails to improve in 7 days, antibiotics should be started or changed.
4. The clinician should attempt to differentiate CRS from recurrent ARS.
5. The clinician should assess the patient with CRS or recurrent ARS for conditions or anatomic abnormalities that would predispose the patient to these conditions.
6. The clinician should obtain a CT scan when evaluating a patient with recurrent ARS or CRS.
7. The patient should be educated on control measures for ARS and CRS.

OPTION

There is only weak evidence that the benefit of treatment outweighs the risk (Grade D):

1. Symptom relief should be offered when treating VRS.
2. Symptom relief should be offered when treating ARS.
3. Observation without use of antibiotics may be done in cases of uncomplicated ARS with temperature less than 101° F (T <38.3° C).
4. Diagnostic nasal endoscopy should be employed in the evaluation of recurrent ARS or CRS.
5. Testing should be done for allergy and immune system dysfunction in patients with recurrent ARS or CRS.

SOR: B

Medical Treatment of Chronic Sinusitis

Medical treatment of chronic sinusitis is based on the same principles as for acute sinusitis: improvement of mucociliary function and eradication of bacteria. If chronic sinusitis is suspected, CT scan of the paranasal sinus should be ordered to confirm the diagnosis before further treatment is initiated; treatment of chronic sinusitis requires more aggressive therapy, with potential side effects. This contrasts with acute sinusitis, for which CT is not necessary before treatment. In addition to confirming the diagnosis and severity of the infections, CT can identify abnormalities that can predict a poorer response to medical treatment. This includes a posterior septal deviation, polyps, allergic fungal sinusitis, and various sinus abnormalities. The scan may also arouse suspicion of sinonasal mass or tumor, which would require earlier ENT evaluation. For all these reasons, referral to an ENT should also be considered as risk of recurrence or chronicity increases.

Medical treatment of chronic sinusitis requires more diligence than for acute sinusitis. Mucociliary improvement is accomplished with the same medications as for acute sinusitis. It is noteworthy that there are *no* medications with indications for treatment of chronic sinusitis (and relatively few for acute sinusitis). Topical and systemic steroids can be used to improve drainage in cases of chronic sinusitis by decreasing mucosal inflammation, edema, and mucus production. Most topical nasal steroids must be used daily for several weeks to have significant benefit. Although oral steroids significantly improve symptoms, the effects may be short-lived, and the potential side effects must be considered. Patients with known allergies should be aggressively treated. It should be remembered, however, that antihistamines cause mucosal drying and mucus stasis. These may need to be stopped in patients with acute exacerbation of chronic sinusitis. Saline irrigations are extremely helpful. Allergy and asthma medications such as leukotriene inhibitors and IgE antagonists can also benefit coexisting sinusitis.

The efficacy of antibiotics in treating chronic sinusitis has not been validated in controlled studies in adults. Two consensus statements do report that antibiotic treatment is likely beneficial in adults but *not in children* (Duiker, 2004; Grade B). Because of the apparent benefit, antibiotics are typically used to treat chronic sinusitis in adults. An antibiotic with activity against staphylococci, as well as the more typical pathogens (with predicted antibiotic resistance), must be chosen. In cases of nasal polyps, an antibiotic with antipseudomonal activity may be necessary. Although no antibiotics have FDA indications for "chronic sinusitis," appropriate choices include macrolides, broader-spectrum cephalosporins, fluoroquinolones, and augmented penicillin. "Older" antibiotics, such as TMP-SMX, clindamycin, doxycycline, and linezolid, may be quite useful in documented staphylococcal infections. Also, the addition of rifampin may be useful in treating documented staphylococcal infections.

Once chronic rhinosinusitis is suspected, ENT consultation is indicated to assist in medical management and to offer surgical options if indicated. Also, simply obtaining a middle meatal culture is extremely helpful in choosing an appropriate antibiotic. Treating chronic sinusitis requires a longer course of antibiotic treatment, often 3 to 8 weeks.

According to Cochrane Collaboration recommendations for medical treatment of chronic sinusitis, *nasal saline irrigations* for the symptoms of chronic rhinosinusitis are well tolerated. Although minor side effects are common, the beneficial effect of saline appears to outweigh these drawbacks for the majority of patients. The use of topical saline could be included as a treatment adjunct for the symptoms of chronic rhinosinusitis.

Failure of Medical Treatment of Sinusitis

When medical treatment of recurrent acute sinusitis or chronic sinusitis is not successful, further evaluation and treatment are indicated. An ENT consultation should be obtained for patients unresponsive to medical treatment of chronic sinusitis. The risk factors listed previously should be considered and modified if possible. Allergy testing should be arranged. Immunologic testing may be indicated. Pulmo-

nary evaluation may be helpful if the patient has poorly controlled reactive airway disease.

If CT has not been done, a scan of the sinuses should be ordered to evaluate the anatomy of the sinuses. If CT has already been done, and symptoms have changed or significant time has elapsed since the first scan, a repeat scan may be helpful. Before repeating the scan, ENT consultation should be considered because the consultant might have a preference for type of scan ordered. A standard sinus CT scan often contains only coronal views; more detailed scans may be necessary for surgical planning.

The CT scan will show any evidence of chronic mucosal edema of the sinuses or even complete opacification. Partial or complete bilateral sinus opacification is typically seen in cases of long-standing chronic sinusitis or polyposis. Unilateral opacification or bony erosion is more worrisome for neoplastic disease or fungal sinusitis. The size of the turbinates and position of the nasal septum can also be assessed. In cases of medical failure or intolerance to continued aggressive medical treatment, surgery should be considered. *Endoscopic sinus surgery* (ESS), or *functional* ESS (FESS), has become the mainstay of surgical intervention for chronic sinusitis. Although earlier external surgical techniques are still occasionally indicated, FESS has the advantage of leaving no external scars and specifically addresses the critical area within the sinuses: the ostiomeatal unit. The procedure is typically done under general anesthesia but can be done under local anesthesia. Success rates of greater than 90% are expected and have been reported in many studies. Some patients experience relapse of their condition, especially if they had nasal polyps. Revision surgery is offered in these cases and is usually quite successful.

Successful sinus surgery often results in fewer and less severe infections and improved response to future medical treatment. The procedure is often done with septoplasty and turbinate surgery and is well tolerated. Advancements in minimally invasive surgical techniques have allowed preservation of more normal tissue and less surgical morbidity. Nasal packing is often not necessary. When used, resorbable packs are typically applied, obviating the need and discomfort of pack removal. Potential complications of ESS include bleeding, scarring leading to further sinus blockage, loss of smell, and rare orbital and CNS injury. For this reason, surgery is undertaken only after appropriate medical treatment and careful patient consideration.

According to Cochrane Collaboration recommendations for chronic rhinosinusitis, FESS as currently practiced is a safe procedure. The limited evidence suggests that FESS has not been demonstrated to confer additional benefit to that obtained by medical treatment (with or without sinus irrigation) in chronic rhinosinusitis. More randomized, controlled trials (RCTs) comparing FESS with medical and other treatments are required, with long-term follow up.

Advances in medical and surgical treatment deserve mention. Newer allergy and asthma medications (leukotriene inhibitors and IgE antagonists) show promise in the future treatment of chronic sinusitis. New antibiotics continue to be released to treat antibiotic-resistant bacteria. Compounding pharmacies have made available topical antibiotic and antifungal formulations that are quite effective in some patients. Topical treatment is appealing because it delivers extremely high concentrations of antibiotic to the target area, minimizing systemic side effects and, theoretically, development of resistance. Topical treatment seems more effective in patients with sinus surgery that created open sinuses, allowing antibiotic delivery. Research into the genetics of sinusitis continues and should lead to further advances in treatment.

Surgical advances include improved power surgical debriders that spare normal mucosa, have more efficient surgery, and reduce blood loss. Packing materials have improved and often are resorbable. Image-guided surgery has become more commonplace. Balloon techniques allow dilation of the sinus ostia without tissue removal. Although not a substitute for an experienced surgeon, these advances often allow more focused and safer surgery.

Pediatric Sinusitis

Although the sinuses are not completely developed until adolescence, children can still develop sinusitis, usually involving the ethmoid and maxillary sinuses. Young children can have 5 to 10 episodes of acute rhinitis (viral URI) in a year. The usual symptoms of a URI lasting longer than 2 weeks can indicate development of bacterial sinusitis. Other symptoms indicating sinusitis in children include nighttime cough and foul breath. Children rarely complain of facial pain, which is common in adults.

Diagnoses other than sinusitis should also be considered in children with prolonged URI-type symptoms. Previously undiagnosed choanal atresia or stenosis can be present. Unilateral or bilateral nasal foreign bodies can also cause these symptoms. Environmental allergies should be considered, as should immunodeficiency, GERD, and cystic fibrosis. Many children with asthma also have coexisting sinusitis, which can complicate asthma management. Recurrent sinusitis can cause or worsen asthma exacerbations.

Chronic adenoid hypertrophy or chronic adenoiditis can mimic sinusitis in children. Adenoid hypertrophy can also *cause* sinusitis secondary to nasal obstruction, mucus stasis, and subsequent infection. This can occur with or without the presence of tonsil hypertrophy. Adenoid hypertrophy can lead to facial changes caused by chronic mouth breathing. Children with long-standing nasal obstruction tend to have an elongated, narrow face with open-mouth breathing, the "adenoid facies." Differentiating chronic adenoiditis from sinusitis can be difficult because the symptoms may be identical and the disorders often coexist.

According to Cochrane Collaboration recommendations, limited evidence suggests that *intranasal corticosteroids* may significantly improve nasal obstruction symptoms in children with moderate to severe adenoidal hypertrophy. This improvement may be associated with a reduction of adenoid size. The long-term effect of intranasal corticosteroids in these patients remains to be defined.

Medical treatment of pediatric acute sinusitis is similar to that for adults: decongestants, nasal saline irrigations or mist, and antibiotics (Duiker, 2004). The pathogens are similar to those of adults: pneumococci, *H. influenzae,* and *M. catarrhalis.* Coexisting problems (e.g., allergies) should be controlled. Diligence on the part of the parents is required because children might not tolerate nasal saline sprays willingly. If compliance is ensured, medical treatment should result in improvement in the vast majority of patients.

Children who fail medical therapy should be referred to an otorhinolaryngologist. Often, adenoidectomy is recom-

mended. In properly selected patients, this procedure has a high success rate in greatly improving symptoms. For children unresponsive to medical treatment and adenoidectomy, a CT scan should be obtained. If significant sinusitis exists, the child likely has chronic sinusitis. Medical treatment should be reattempted because it might be more effective once the obstructing adenoids have been removed. If not previously done, the child should be screened for allergies, immune deficiency, and cystic fibrosis.

The efficacy of antibiotics in the treatment of pediatric chronic sinusitis has not been validated. Unlike adults with chronic sinusitis who likely benefit from longer courses of antibiotics, evidence-based research indicates that this is not the case for children (Duiker, 2004).

If symptoms persist, ESS is considered. The surgery targets the ostiomeatal unit in hope of improving sinus drainage and aeration. A ciliary biopsy may also be done to evaluate for ciliary dyskinesia. Cultures should also be obtained to tailor postoperative antibiotics. In properly selected patients, sinus surgery has an extremely high success rate. Although rare, significant risks, including bleeding and orbital and intracranial injury, must be considered. Surgery may be especially beneficial in children with coexisting pulmonary disease (asthma or cystic fibrosis).

Sinonasal Tumors

Intranasal and sinus tumors often manifest with symptoms identical to those of more benign sinonasal conditions. Nasal obstruction, facial pressure or pain, and bloody rhinorrhea are common symptoms of a neoplastic process within the nasal cavity or sinuses. Because these symptoms are also common with sinusitis, a high index of suspicion is required, and diagnosis is often delayed.

Tumors of the external nose are usually related to prolonged exposure to the sun. Basal cell and squamous cell carcinoma are most common (see Chapter 33). Intranasal tumors can be benign or malignant. The most common growth within the nasal cavity is the *benign squamous papilloma,* caused by the human papillomavirus (HPV). This typically appears as an exophytic lesion within the nose, often at a junction between squamous and respiratory epithelium, and causes irritation and bleeding. Treatment consists of simple excision, and recurrence is uncommon. Malignant degeneration is extremely unlikely.

A much more aggressive papillary lesion is an *inverted papilloma.* These tumors often manifest as unilateral polyps and can cause symptoms of nasal obstruction, bleeding, and sinusitis. These lesions require excision because they can be locally destructive, and malignant degeneration can occur. Endoscopic excision is usually possible, although external approaches are sometimes required.

The *juvenile nasopharyngeal angiofibroma* is a tumor that occurs exclusively in adolescent boys. The tumor is located within the nasopharynx but typically causes nasal symptoms. Patients present with nasal obstruction and recurrent epistaxis. Because nosebleeds and nasal obstruction are both common problems, a high index of suspicion is required to make a timely diagnosis. These tumors can be extremely aggressive, and surgical excision is required. Recurrences are possible, and radiation therapy is recommended for some patients.

Malignant tumors of the nose and sinuses include squamous cell carcinoma, adenocarcinoma, adenoid cystic carcinoma, hemangiopericytoma, osteosarcoma, and malignant melanoma. Again, early symptoms are similar to those of sinusitis, which often results in delayed diagnosis. The prognosis ranges from extremely good to extremely poor, depending on tumor type and stage. Orbital and intracranial extension often occurs. Both regional and distant metastases result in poorer prognosis. Aggressive treatment is required and involves combined treatment with surgery and radiation, sometimes with chemotherapy.

Nasal Polyps

Nasal polyps result from inflammation, with resultant exuberant swelling of the respiratory epithelium of the nasal and sinus mucosa. Typically, polyps cause symptoms of nasal obstruction and anosmia and can lead to sinusitis. Once formed, polyps have a grayish, translucent appearance. Nasal polyps are typically bilateral and are painless and insensate if touched. Typical nasal polyps are benign. Suspicion of neoplasia or fungal infection should be higher in cases of unilateral polyps. Their exact cause is not completely understood. Nasal polyps are often seen in atopic patients. Some patients are found to have nasal polyps, aspirin sensitivity, and asthma—the "asthma triad." Nasal polyps often coexist with chronic sinusitis. If seen in pediatric patients, nasal polyps should raise suspicion of cystic fibrosis.

Treatment of polyps consists of the usual medical treatment of sinusitis and allergies if present. *Oral steroids for nasal polyps* may reduce the need for surgery, but there are concerns about possible side effects with long-term oral steroid use. The side effects of short courses of oral steroids are less clearly defined. Duiker (2004) found only one trial that met inclusion criteria, involving 80 participants, about 75% of whom were randomized to receive oral prednisone. Quality-of-life and nasal symptom scores improved with a significant reduction in polyp size after 2 weeks of treatment versus no steroid treatment. However, the trial was small and of low methodologic quality. Oral steroids often result in temporary shrinkage of the polyps. Regrowth typically occurs shortly after the steroids are stopped. Topical nasal steroids are also sometimes effective in shrinking polyps or stabilizing their growth. Newer, more potent leukotriene inhibitors have shown promise in slowing or even reversing polyp growth in some patients, although these medications are not indicated for this use. If the polyps cause significant symptoms of nasal obstruction or sinusitis, ESS should be considered to remove the polyps and eradicate any chronic infection within the sinuses. Surgery also allows a biopsy to be done.

Although polyps often recur after ESS, the patient's subjective improvement is marked, and recurrence is often slow. In addition, the response to medical treatment is often greatly improved once the polyps have been removed. Revision surgery (and sometimes multiple surgeries over years) may be necessary.

Nasal Trauma

Traumatic injuries to the nose are extremely common and usually of little long-term significance. In some patients, however, trauma can result in significant cosmetic and

functional problems. In severe cases, nasal trauma can result in severe bleeding and cerebrospinal fluid (CSF) leakage and can even be life threatening.

Evaluation of a patient who has sustained nasal trauma requires a thorough history. The mechanism of injury must be understood. If the injury was recent, the patient must be examined for signs of cervical, mandibular, maxillary, orbital, or intracranial injury. Bleeding can be quite severe after isolated nasal trauma but usually stops spontaneously or with only digital pressure.

Initial evaluation includes assessment of the gross appearance of the face, with special attention to the possibility of other facial fractures (orbital, zygomatic, mandibular). Obvious deformity of the nose should be noted, although marked edema obscures this in some cases. Radiographs may be ordered, but their utility is variable because nondisplaced fractures usually require no treatment, and displaced nasal fractures are usually obvious on examination.

Intranasal examination is done to rule out the presence of a septal hematoma or CSF leakage. A septal hematoma results when bleeding occurs between the septal perichondrium and the underlying cartilage. The hematoma can be unilateral or bilateral. It results in a widened septum with nasal obstruction. Successful treatment requires prompt diagnosis followed by incision and drainage and packing to prevent reaccumulation. If untreated, and especially if bilateral, the hematoma leads to ischemic necrosis of the cartilage or can result in abscess formation. This can ultimately result in loss of enough septal cartilage to cause external nasal collapse, called saddle nose deformity. Because it is extremely difficult to repair, avoiding saddle nose deformity is paramount.

Severe bleeding after nasal trauma can result from a vascular injury of the ethmoidal, the sphenopalatine, or rarely the carotid arteries. ENT consultation should be obtained if severe bleeding persists. *CSF leakage* is diagnosed when clear drainage is seen dripping from one or both sides of the nose. Leakage can increase in a more dependent position. Nasal CSF leakage requires urgent ENT and neurosurgical consultation. It often resolves spontaneously but can lead to life-threatening problems such as pneumocephalus (air within the cranial vault), meningitis, and brain abscess.

Isolated nasal deformity after nasal trauma results from displacement of the nasal bones, the external nasal cartilages, or the septum. The nasal bones can often be repositioned with excellent results by performing a *closed reduction*. This is done under local or general anesthesia, usually after the initial edema has subsided and before the bones have set (7-10 days after injury). Sometimes an *open reduction*, which involves refracturing the nasal bones, is required. If the septum is greatly deviated, it can be repaired at the same time. If significant nasal deformity persists, a formal *rhinoplasty*, which more precisely addresses all aspects of the external nose, can be done later. In children with nasal fractures, closed reduction is usually recommended sooner than for adults because their fractures heal more quickly. Repair should be done within 7 days of the injury, if possible. Open reduction is generally not recommended in children because of concern for affecting future nasal growth. If necessary, rhinoplasty is delayed until nasal growth is complete, which is shortly after puberty.

KEY TREATMENT

Radiographic imaging is not recommended in patients who meet the clinical criteria for acute bacterial rhinosinusitis.
Diagnosis of ABRS can be made if signs or symptoms are present 10 days beyond the initial onset of upper respiratory symptoms, or if there is a worsening within 10 days after an initial improvement ("double sickening") (Rosenfeld et al., 2007).
Amoxicillin is the first line of treatment for most adults with ABRS (SOR: B).

Oral Cavity and Pharynx

Key Points

- The Infectious Diseases Society of America now recommends the rapid antigen test alone to confirm the presence of group A beta-hemolytic streptococcal (GABHS) pharyngitis in adults.
- Cephalosporins appear to be superior to penicillin in bacterial eradication and clinical cure in GABHS pharyngitis.
- Motor disorders of the esophagus are more likely to cause difficulty swallowing liquids, whereas mechanical obstruction produces dysphagia with both solids and liquids.
- From 23% to 60% of patients presenting with a globus sensation have gastroesophageal reflux disease.
- Snoring is the most common symptom of obstructive sleep apnea and is more common in men than women.

Physical Examination and Radiography

See the discussion online at www.expertconsult.com.

Acute Pharyngitis and Tonsillitis

Viral agents cause the majority of sore throats. Even when exudates are present, less than 15% of children and 10% of adults have documented group A beta-hemolytic streptococci (GABHS) as the cause. In children younger than 3 years, the predominance of a viral cause is even higher than in school-age children.

Pharyngitis caused by GABHS *(Streptococcus pyogenes)* has its peak incidence in late winter and early spring. The incubation phase is 2 to 5 days and leads to sudden onset of sore throat, painful swallowing, fever, and chills. Less frequent symptoms include headache, abdominal pain, and nausea. On physical examination, a purulent white exudate is often seen on the tonsils, and the anterior cervical nodes are tender and enlarged. There is sometimes a scarlet fever rash (a diffuse, erythematous, macular, rough rash that tends to coalesce) and soft palate petechiae. Scarlet fever is the result of exotoxin-producing strains of GABHS. Pastia's lines are caused by prominence of the rash in the flexor creases of the antecubital space or axilla. A strawberry tongue is another sign of GABHS.

A throat culture is the "gold standard" for diagnosing GABHS infection. A 5% sheep blood agar plate is used to plate the throat swab and can be read in 24 hours (sensitivity 96%). Serologic tests (antistreptolysin O [ASO] titer) are accurate but not practical to use in diagnosing an acute infection because of the time involved in obtaining results.

Oral Cavity and Pharynx

Physical Examination

A wide variety of disorders may be found in the oral cavity and pharynx. Information is obtained from a thorough history and systematic physical examination of this area.

Problems in the oral cavity are usually dental in nature, and all physicians should be able to describe abnormalities accurately in consultation with dental colleagues. Children have 20 deciduous teeth, and adults have 32 permanent teeth. The teeth are numbered beginning with the upper right third molar (tooth number 1). The upper left third molar is number 16. The left mandibular third molar is number 17, and the right mandibular third molar is number 32. Adults have four upper and four lower midline incisors and a canine tooth flanking the incisors (four total). The premolars are lateral to the canines (total four). If the "wisdom teeth" are present, adults have four upper molars per side and four lower molars per side (16 total). The appearance of the teeth should be noted and dental evaluation requested if necessary.

Particular attention is paid to mucosal appearance. All mucosa should be inspected. The color and contour should be pink, smooth, and moist. The presence of mucosal ulceration or mass is noteworthy. Mucosal abnormalities may be conditions that are congenital, infectious, autoimmune, traumatic, neoplastic, toxic, or irritative (cigarettes, oral tobacco). They may also be secondary to immune deficiency (HIV/AIDS), systemic inflammatory disorders, or nutritional deficiencies. Proper identification of these allows prompt diagnosis and treatment.

The tongue consists of papillae that cover most of the dorsal surface and include fungiform, filiform, and circumvallate papillae. The circumvallate papillae are a V-shaped group of papillae located between the anterior two thirds and the posterior one third of the tongue. These papillae may be mistaken for an abnormality by the patient or even the physician. The foramen cecum is a pit located midline at the junction between the anterior two thirds and the posterior one third of the tongue and represents the location of initial descent of the embryologic thyroid into the neck. The lingual tonsil lies at the base of the tongue and is a fairly common location for infection (lingual tonsillitis) as well as neoplasia (base of tongue cancer). The base of the tongue should be palpated if concern for neoplasia exists.

The two pairs of major salivary ducts should be inspected. Wharton's ducts drain saliva from the submandibular glands. They lie in the floor of mouth, just lateral to the lingual frenulum. Stenson's ducts supply saliva from the parotid glands. They open into the buccal mucosa, adjacent to the upper first molars. Each of these glands should be palpated. In cases of suspected sialoadenitis, the quality of the saliva should be inspected as the glands are massaged. If infection is present, purulence may be seen coming through the papilla of the duct of the affected gland.

The palatine tonsils should be inspected. They are part of the oropharynx. Their size, appearance, and symmetry should be noted. The size of the tonsils is graded on a scale of 1 to 4. A "1" tonsil is quite small, barely visible; "4" tonsils touch in the middle (kissing tonsils); "2" and "3" tonsils are mildly and moderately hypertrophic. Asymmetric tonsils require further investigation because this is a concern for malignancy, especially in adults.

Rapid antigen detection tests or optical immunoassays (OIAs) are the most popular tests for detection of GABHS. Although most manufacturers claim 95% to 97% true negatives (specificity), clinical trials suggest a lower figure of 90%. True positives (sensitivity) are claimed to be 90% to 95%, but again, clinical trials show closer to 60% to 80% sensitivity.

Scoring systems based on features of sore throat have been developed. The Centor criteria for screening consist of tonsillar exudates, tender anterior cervical lymphadenopathy, absence of cough, and history of fever (Centor et al., 1981). These findings in patients with sore throat can help determine the likelihood of GABHS. When three or four Centor criteria are met, the physician may use a rapid antigen detection test. However, Centor found that only 56% of adults with all four criteria had a positive throat culture, and only 30% to 34% of adults with three criteria had a positive culture. Therefore, 60% of adults with three or four Centor criteria are probably culture negative (Bisno, 2003). The negative predictive value of two or fewer Centor criteria is very high, and testing or treatment would not be indicated. The Infectious Disease Society of America (IDSA) now recommends the use of the rapid antigen test alone to confirm the presence of GABHS in adults (Bisno et al., 2002).

A study comparing five management strategies (no treatment or testing, empiric treatment with penicillin, throat culture, OIA with culture if results negative, OIA alone) found empiric therapy the least cost-effective and culture the most cost-effective strategy when GABHS prevalence was 10%. OIA alone was as effective as OIA followed by throat culture when OIA was negative (Neuner et al., 2003). Once a test result is positive, treatment with penicillin or a cephalosporin is recommended because there has been no incidence of in vitro resistance to these drugs.

Treatment for GABHS is penicillin VK, 250 mg two or three times daily in children and 500 mg three or four times daily in adolescents and adults for 10 days. Intramuscular penicillin G benzathine, 600,000 U for a child weighing less than 60 lb (27 kg) or 1.2 million U for patients weighing more than 60 lb, can be used if compliance is a problem or if the child cannot swallow or is vomiting. Mixing the benzathine penicillin with 300,000 U procaine penicillin alleviates some of the discomfort. Penicillin-allergic patients can use erythromycin, 40 mg/kg in two or four divided doses daily for 10 days. In recurrent disease, cephalexin, 12.5 mg/kg or 250 mg three or four times daily for 10 days, can be prescribed. Alternative antibiotics include cefpodoxime (Vantin), cefprozil (Cefzil), cefuroxime axetil (Ceftin), cefixime (Suprax), cefaclor (Ceclor), ceftibuten (Cedax), loracarbef (Lorabid), azithromycin (Zithromax), clarithromycin (Biaxin), and amoxicillin–clavulanic acid (Augmentin). Cephalosporins appear to be superior to penicillin in terms of bacterial eradication and clinical cure (Pichichero and Brixner, 2006).

After treatment, 15% of throat cultures remain positive for GABHS. This is considered a carrier state. One effective way to eliminate the carrier state is to use clindamycin (Cleocin), 20 mg/kg/day in three divided doses (maximum, 450 mg/day) for 10 days (Tanz et al., 1998). Contagiousness to others is inversely proportional to the length of time that GABHS is carried. Culturing contacts is indicated only when the contact is symptomatic. Pets have been considered a reservoir for GABHS, but some evidence casts doubt on this supposition (Wilson et al., 1995).

Although antimicrobial treatment is believed to decrease the suppurative (peritonsillar abscess) and immunologic (acute rheumatic fever, acute glomerulonephritis) sequelae of GABHS infection, much is not known. *Acute rheumatic fever* had become rare until 1986, when its incidence increased; any explanation is mere speculation. There is good evidence that antimicrobial treatment shortens the symptomatic period of GABHS and plays a part in preventing acute rheumatic fever. However, acute rheumatic fever can still occur in the presence of appropriately diagnosed and treated cases of GABHS pharyngitis and even in the absence of a symptomatic infection.

Only specific serotypes of GABHS (12, 49, 55, 57, Red Lake strain) cause *acute glomerulonephritis* (AGN) because of an antigen-antibody deposition on the kidney glomerular membrane. When a patient does have GABHS infection caused by one of these strains, only 15% develop AGN. Edema, hypertension, and rusty urine are the hallmarks of AGN and occur 10 days after the infection. Prompt treatment of GABHS pharyngitis with penicillin does not appear to prevent AGN. Treatment is symptomatic to control the blood pressure and edema.

The indications for tonsillectomy after GABHS infections are six episodes within 1 year or three to four episodes within each of 2 years (Pichichero, 2004). The severity of the infections and the total number of missed workdays or schooldays should also be taken into account when considering tonsillectomy.

Viral causes of pharyngitis include rhinovirus (20% of all pharyngitis) and coronavirus, adenovirus, and parainfluenza virus (5% of all pharyngitis) (Middleton, 1996). Coxsackievirus A can cause 1-mm to 2-mm red-ringed vesicles on the tonsils, uvula, and soft palate and is known as *herpangina*. Coxsackievirus A16 is the major cause of hand, foot, and mouth disease, a 1-week illness with 4-mm to 8-mm ulcers on the tongue and buccal mucosa and vesicles on the palms and soles. Incubation time for this illness is 4 to 6 days.

A type of pharyngitis similar to coxsackievirus A16 pharyngitis is caused by herpes simplex virus (HSV). Painful, shallow, red-bordered ulcers develop on the soft palate, gums, lips, or buccal mucosa and cause fever, pain, and lymphadenopathy. Acyclovir (Zovirax), 200 mg five times daily for 5 days; famciclovir (Famvir), 125 mg twice daily for 5 days; or valacyclovir (Valtrex), 500 mg twice daily for 5 days, can be used in severe cases of herpes stomatitis and lessen the duration of symptoms and viral shedding.

Epstein-Barr virus (EBV) as a cause of pharyngitis can mimic GABHS infection. It can also occur concurrently with GABHS infection. Studies have shown the two infections occurring together in 2% to 33% of cases. Prodromal symptoms to severe sore throat include malaise, anorexia, chills, and headache. Fatigue, lymphadenopathy, and hepatosplenomegaly can follow in 5 to 14 days. Pharyngitis with tonsillar hypertrophy and a membranous white tonsillar exudate lasts 5 to 10 days. Lymphadenopathy and hepatosplenomegaly can persist 3 to 6 weeks. Contact sports should be avoided for 6 weeks because of the possibility of splenic rupture. Complications that occur in less than 2% of patients with EBV infection include thrombocytopenia, hemolytic anemia, Guillain-Barré syndrome, Bell's palsy, transverse myelitis, and aseptic meningitis. Hepatitis can be seen in 20% to 50% of patients with EBV infection.

Diagnosis is made on clinical grounds supported by positive antibody tests. A CBC shows atypical lymphocytes, and

Box 19-8 Bacterial Causes of Pharyngitis Other than GABHS*

Group C β-hemolytic streptococci
Group G β-hemolytic streptococci
Anaerobes (*Peptostreptococcus*, *Fusobacterium*, and *Bacteroides* spp.)
Arcanobacterium haemolyticum
Chlamydia pneumoniae
Corynebacterium diphtheriae
Corynebacterium haemolyticum
Mycoplasma pneumoniae
Neisseria gonorrhoeae
Francisella tularensis (tularemia)
Yersinia enterocolitica
Haemophilus influenzae (epiglottitis)

*Group A β-hemolytic streptococci.

a monospot test is positive in 95% of cases of infectious mononucleosis (Middleton, 1996). Steroids (and rarely urgent tonsillectomy) are indicated when the tonsillar hypertrophy causes pharyngeal obstruction or when other life-threatening complications occur. Amoxicillin should be avoided because it often causes a rash in patients with infectious mononucleosis.

When the symptoms point to infectious mononucleosis but the monospot test or EBV titer result is negative, the patient may be infected with cytomegalovirus (CMV). The illness lasts 2 to 6 weeks and is characterized in older patients by higher fever and greater malaise but milder pharyngitis than with infectious mononucleosis. A CMV-specific immunoglobulin M (IgM) antibody test is the best means of diagnosing this infection. In immunocompromised patients, ganciclovir (Cytovene) or foscarnet (Foscavir) can control the infection. Bacterial causes of pharyngitis other than GABHS are shown in Box 19-8.

KEY TREATMENT

Although current treatment guidelines recommend oral penicillin V or intramuscular penicillin G benzathine as drugs of choice for GABHS, strong evidence supports cephalosporins as a first choice for this infection (SOR: A) (Casey and Pichichero, 2007).
In countries with low rates of rheumatic fever, 3 to 6 days of oral cephalosporins to treat GABHS has comparable efficacy to the usual 10-day oral penicillin regimen (SOR: A) (Altamimi et al., 2009).
Once-daily amoxicillin (750 mg to 1 g) for 10 days is as good as twice-daily or three-times-daily dosing (SOR: A)(Lennon et al., 2008).

Abnormalities of the Oral Region (eTable 19-6)

Swallowing Disorders

Patients who experience difficulty swallowing solids or liquids have *dysphagia*. About 7% of Americans experience dysphagia in their lifetime, and 30% to 40% of nursing home residents have swallowing disorders. Motor disorders of the esophagus are more likely to cause difficulty in swallowing liquid, whereas mechanical obstruction causes dysphagia with both solids and liquids.

Pharyngeal muscle weakness or CNS disease can lead to difficulty beginning the act of swallowing. The proximal one third of the esophagus is composed of striated or voluntary muscles, the middle one third is a combination of striated and smooth muscles, and the distal one third is entirely smooth muscle tissue. The two sphincters involved in swallowing are the upper and lower esophageal sphincters (UES and LES).

Food sticking in the throat is known as *oropharyngeal dysphagia*, and 80% of the cases are the result of neuromuscular disease (see **eBox 19-5** online for causes) (Trate et al., 1996). Inability to move solids from the esophagus to the stomach is referred to as *esophageal dysphagia*, which is more common than oropharyngeal dysphagia (see **eBox 19-6** online for causes). The history is extremely important in determining the cause of the dysphagia. If dysphagia is associated with chest pain, especially difficulty swallowing cold liquids, diffuse esophageal spasm is the most likely diagnosis.

A constant sensation of a lump in the throat is known as *globus hystericus* and is not necessarily associated with the act of swallowing. This diagnosis can be made only by ruling out anatomic or motor abnormalities of the pharynx, larynx, or esophagus. From 23% to 60% of patients presenting with a globus sensation have GERD as the origin (Ahuja et al., 1999). **eBox 19-7** online lists head and neck symptoms related to GERD versus gastroesophageal symptoms.

If the food bolus sticks in the distal esophagus, a stricture, malignancy, or ring is most likely the cause. Achalasia or degeneration of the nerve cell bodies of the myenteric plexus leads to esophageal dilation and food retention associated with increased tone of the LES.

Motor disorders of the esophagus cause progression of dysphagia over months to years. A carcinoma should be suspected when there is a rapid progression of dysphagia for solids in an older person with anorexia and weight loss; a history of smoking and alcohol use makes this diagnosis more likely. Medication-induced esophagitis is characterized by acute retrosternal pain exacerbated by swallowing. The most common medications associated with this syndrome are the tetracyclines (doxycycline, minocycline), potassium chloride pills, iron preparations, quinidine and its derivatives, aspirin, and nonsteroidal anti-inflammatory drugs (NSAIDs).

Another mechanical reason for dysphagia is an *esophageal web*, a thin diaphragm most often in the proximal esophagus. When associated with iron deficiency anemia, the condition is known as *Plummer-Vinson syndrome*. *Pulsion diverticula* are usually seen at the level of the cricopharyngeal muscle and occur predominantly in men older than 50 years. Regurgitation of food and liquid immediately after swallowing is the hallmark of this condition, with large diverticula that can cause almost complete obstruction. A *Zenker's diverticulum* originates from the posterior aspect of the esophagus and is bounded superiorly by the cricopharyngeal muscle and inferiorly by the inferior pharyngeal muscle. Treatment is usually surgical.

Dysphagia in the pediatric population is often secondary to tonsillar hypertrophy or esophageal foreign body. Diagnostic studies include a barium esophagram with views of the pharynx, esophagus, and stomach. To detect rings and early strictures, a barium-coated tablet or marshmallow can reveal the site of narrowing. When there is radiographic evidence of a lesion, esophagogastroscopy is indicated.

If no abnormality is found, esophageal manometry should be performed. The goal of esophageal manometry is to assess the characteristics of esophageal contractions and to define the LES and the UES and their response to swallowing. Only

eBox 19-5 Causes of Oropharyngeal Dysphagia

Neuromuscular Diseases

Central
Stroke
Amyotrophic lateral sclerosis
Brainstem tumors
Bulbar poliomyelitis
Multiple sclerosis
Parkinsonism
Peripheral
Diabetes, alcohol, or thyroid causing neuropathy
Motor end plate
Myasthenia gravis
Voluntary muscle
Dermatophytosis
Muscular dystrophy
Polymyositis

Local Structural Lesions

Cancer
Esophageal web
Thyromegaly
Zenker's diverticulum

From Trate DM, Parkinan HP, Fisher RS. Dysphagia: evaluation, diagnosis, and treatment. Prim Care 1996;23:417.

eBox 19-6 Causes of Esophageal Dysphagia

Mechanical Disorders

Intrinsic
Alkali-related scarring
Benign tumors
Carcinoma
Esophageal web
Schatzki's ring (distal esophagus)
Stricture secondary to chronic gastroesophageal reflux
Extrinsic
Mediastinal abnormalities (lymph node, thyroid)
Vascular compression (aortic aneurysm)

Motility Disorders

Achalasia
Amyloidosis
Anticholinergic medications
Diabetic neuropathy
Esophageal spasm
Presbyesophagus
Scleroderma

From Trate DM, Parkinan HP, Fisher RS. Dysphagia: evaluation, diagnosis, and treatment. Prim Care 1996;23:417.

eBox 19-7 GERD-Related Head and Neck Symptoms

Chronic cough
Food sticking in throat
Globus sensation
Halitosis
Hoarseness
Sore throat
Throat clearing
Water brash

GERD, Gastroesophageal reflux disease.

eTable 19-6 Some Disorders of the Oral Regions by Predominant Site of Involvement

Disorder	Description	Disorder	Description
Nonspecific Acute herpetic gingivostomatitis	Widespread ulcerating vesicular lesions	Verrucous carcinoma	Slow growing, exophytic, usually well differentiated, at site of snuff application, metastasis unusual, occurring late
Behcet's syndrome	Multiple oral ulcers similar to those of aphthous stomatitis	White sponge nevus	Thick, white folds over most of buccal mucosa except gingivae; benign
Cicatricial pemphigoid	Bullae that rupture quickly, leaving ulcers; ocular lesions that develop after oral lesions	**Palate** Infectious mononucleosis	Petechiae at junction of hard and soft palates
Condyloma acuminatum	Venereally transmitted wart forming cauliflower-like clumps	Kaposi's sarcoma	Painless, red to purple macules progressing to painful papules
Dyskeratosis	Occurs with erythroplakia, leukoplakia (with patch on mucous membrane that does not rub off), and mixed red-and-white lesions; precancerous	Necrotizing sialometaplasia	Large, rapidly developing ulcer, often painless; appears grossly malignant; heals spontaneously in 1-3 months
Erythema multiforme	Multiple bullae that rupture quickly, leaving hemorrhagic ulcers; includes Stevens-Johnson syndrome	Papillary inflammatory hyperplasia	Red spongy tissue, followed by fibrous tissue folds; benign
Hemangioma	Purple to dark-red lesions, similar to port-wine stain; benign	Pipe smoker's palate (nicotine stomatitis)	Red punctate areas at ducts of minor salivary glands, often with severe, usually benign, leukoplakia
Hereditary hemorrhagic telangiectasia	Localized dilated blood vessels	Secondary herpes simplex	Small papules quickly coalescing into series of ulcers

eTable 19-6 Some Disorders of the Oral Regions by Predominant Site of Involvement—cont'd

Disorder	Description	Disorder	Description
Lichen planus	Lacy pattern (Wickham-s striae) sometimes erosive; can become malignant	Torus palatinus	Overgrowth of bone in midline; benign
Lymphangioma	Localized swelling or discoloration; benign	Wegener's granulomatosis	Lethal midline granuloma with bone destruction, sequestration, and perforation
Mucocele (mucous retention cyst)	Soft nodule; if superficial, covered by thin epithelium; bluish	**Tongue and Floor of Mouth** Ankyloglossia	Tongue unable to protrude
Noma	Small vesicle or ulcer that rapidly enlarges and becomes necrotic	Benign lymphoepithelial cyst	Yellowish nodule on ventral part of tongue or anterior floor of mouth
Pemphigoid	Small, yellow or hemorrhagic, tense bullae; can last several days before rupture	Benign migratory glossitis (geographic tongue, erythema migrans)	Changing patterns of hyperkeratosis and erythema on dorsum and edges; desquamated filiform papillae in irregular circinate pattern, often with inflamed center and white or yellow border
Peutz-Jeghers syndrome	Brownish black melanin spots with gastrointestinal polyposis	Dermoid cyst	Swollen floor of mouth
Recurrent aphthous stomatitis (canker sores)	Small, painful ulcers or large, painful scarring ulcers	Enlargement of tongue	Localized or generalized, depending on how many teeth are missing; adjacent teeth may indent tongue
Syphilis	Chancre,* mucous patch, gumma	Fissured (scrotal) tongue	Deep furrows in lateral and dorsal areas
Lips Actinic atrophy	Thin, atrophic mucosa with erosive area; predisposes to neoplasia	Glossitis	Red, painful tongue; often secondary to another condition, allergic, or idiopathic
Angioedema	Acute swelling	Hairy tongue	Dark, elongated filiform papillae
Angular cheilitis (cheilosis)	Fissuring of corners of mouth, often with maceration	Linea alba	Thin white line on edge of tongue, usually bilateral
Cheilitis glandularis	Enlarged, nodular labial glands with inflamed, dilated secretory ducts; sometimes everted, hypertrophic lips	Lingual thyroid nodule	Smooth nodular mass of thyroid tissue follicles on far posterior dorsum of tongue, usually at midline
Cheilitis granulomatosa	Diffusely swollen lips, primarily lower lip	Ludwig's angina	Can compromise airway by forcing tongue superiorly and posteriorly
Cracking exfoliative cheilitis	Chronic desquamation of superficial mucosal cells	Median rhomboid glossitis	Red (usually) patch in midline of tongue, without papillae
Keratoacanthoma	Benign, locally destructive epithelial tumor resembling squamous cell carcinoma; regresses spontaneously in about 5 months	Neurilemoma	Persistent swelling, sometimes at site of previous trauma
Secondary herpes simplex (cold sores)	Short-lived vesicle followed by small, painful ulcer at vermilion border	Pernicious anemia	Smooth pale tongue, often with glossodynia or glossopyrosis
Verruca vulgaris (wart)	Pebbly surface	Ranula	Large mucocele penetrating mylohyoid muscle; can plunge deep into neck; swollen floor of mouth
Buccal Mucosa Aspirin burn	Painful white area; when wiped off, exposes inflamed area	Thyroglossal duct cyst	Midline swelling that moves upward when tongue protrudes
Fordyce's granules	Cream-colored macules, about 1 mm in diameter; benign	Tuberculosis	Ulcers on dorsum of tongue, cervical adenopathy
Hand-foot-and-mouth disease	Small, ulcerated vesicles	**Salivary Glands**	

Continued

eTable 19-6 Some Disorders of the Oral Regions by Predominant Site of Involvement—cont'd

Disorder	Description	Disorder	Description
Herpangina	Vesicles in posterior of mouth	Benign lymphoepithelial lesion (Mikulicz disease)	Unilateral or bilateral enlargement of salivary glands; often with dray mouth and eyes
Irritation fibroma	Smooth-surfaced, dome-shaped sessile	Sialadenitis	Swelling, often painful; benign
Koplik's spots	Tiny, grayish white macules with red margins near orifice of parotid duct	Sialolithiasis	Swelling (e.g., floor of mouth) that increases at mealtime or after eating a pickle
Linea alba	Thin white line, typically bilateral, on level of occlusal plane; benign	Sjögren's syndrome	See text section on salivary glands
Smokeless tobacco lesion	White or gray, corrugated; usually behind lower lip; tends toward malignancy	Xerostomia	Dry mouth

From Beers MH, Berkow R (eds). The Merck Manual of Diagnosis and Therapy, 17th ed, pp 752-754. White House Station, NJ, Merck Research Laboratories, 1999.
*Red papule developing into a painless ulcer with serosanguineous crust.

about 50% of patients show a definitive abnormality with this test. Provocative testing with manometry involves infusion of edrophonium, esophageal balloon dilation, or acid perfusion into the esophagus. Recording esophageal pH in an ambulatory setting can indicate if acid reflux episodes are present at the same time as the patient's symptoms.

The management of swallowing difficulties is directed toward the specific cause involved. **eTable 19-7** lists the treatments for the various causes of dysphagia.

Snoring and Obstructive Sleep Apnea

Snoring is extremely prevalent but is also the most common symptom of obstructive sleep apnea. OSA occurs when the upper airway collapses during sleep, leading to obstruction, hypoventilation, and hypoxemia. OSA has been associated with development or exacerbation of hypertension, coronary artery disease, pulmonary hypertension, poor concentration, impotence, obesity, depression, and increased risk of motor vehicle crashes. It is a potentially serious medical condition that can be overlooked if not specifically sought.

Men have OSA more than women, and children can also be affected. Adults with OSA are often overweight or stocky. They are told that they snore loudly, and episodes of respiratory obstruction or gasping might be witnessed by a family member. Symptoms of OSA include loud snoring, daytime fatigue, morning headache (secondary to hypoxemia), restless sleeping habits, and frequent catnaps (often unintentionally, sometimes while driving).

Physical examination should be performed with attention to the patient's body habitus. The oral cavity should be inspected for tonsillar hypertrophy and evidence of excess soft palate tissue. The size and position of the tongue should be noted. The nose should be inspected for nasal obstruction. The size and position of the mandible and its relationship to the neck should be evaluated.

Strong suspicion of sleep apnea should be confirmed with an overnight sleep study or polysomnogram. The sleep study measures intensity of snoring, presence of apnea or hypopnea (partial apnea), oxygen saturation, sleep efficiency, and cardiac rhythm. The data are compiled and analyzed by a sleep specialist. A numeric value of the total number of apneas and hypopneas is derived, called the apnea-hypopnea index or *respiratory distress index* (RDI). An RDI of less than 5 is considered normal. An RDI greater than 5 with or without oxygen desaturation indicates sleep apnea. Some patients are found to have normal RDI values but have snoring with disrupted sleep. This condition has been described as "upper airway resistance syndrome." Treatment is similar to that for OSA.

Treatment for OSA is either nonsurgical or surgical. Nonsurgical methods are always attempted first and include weight loss and assisted nighttime ventilation by continuous positive airway pressure. CPAP essentially splints the collapsed airway open with positive pressure, given as the patient initiates inhalation. It is generally well tolerated, safe, and extremely effective in compliant patients. Unfortunately, some patients either do not tolerate CPAP or choose not to use it. Surgery is a potential option in these patients.

Numerous surgical procedures exist for the treatment of OSA. The gold standard is tracheotomy, which bypasses the upper airway obstruction and is almost always successful in eliminating sleep apnea. Obviously, most patients do not see this as an appealing solution. For patients with severe OSA and morbid obesity, however, a tracheotomy can give extremely satisfying results. More popular procedures have been devised to try to eliminate (rather than bypass) the upper airway anatomic obstruction. Uvulopalatopharyngoplasty is used most often and entails removal of redundant oropharyngeal tissue, including the tonsils, the uvula, and a strip of soft palate. Its success depends on the severity of the OSA and the patient's anatomy. Other procedures use laser or radiofrequency tissue ablation to eliminate excess pharyngeal tissue. If nasal obstruction exists, it is corrected. In severe cases, procedures directed at the tongue base and facial skeleton have been effective in properly selected patients.

The Larynx

Key Points

- Most cases of acute stridor are secondary to inflammatory disorders such as croup, epiglottitis, and tracheitis.
- Neoplasia must be considered in adults with voice or swallowing complaints.
- Visualization of the laryngopharynx is required to evaluate any patient with persistent or severe stridor or with voice or throat symptoms.
- Parainfluenza virus is the most common cause of croup.
- Laryngomalacia is the most common cause of chronic stridor in neonates.
- Vocal cord nodules are always bilateral; polyps and cysts are usually unilateral.
- It is essential to elicit smoking and alcohol histories in patients with voice complaints because of the association with cancer.
- Although often idiopathic or iatrogenic, vocal cord paralysis requires thorough investigation.
- Vocal cord dysfunction is often misdiagnosed as asthma.

Initial Evaluation

Hoarseness, stridor, foreign body (globus) sensation, and dysphagia are all symptoms of laryngeal or hypopharyngeal pathology. When a patient presents with any of these persistent symptoms, a complete past medical and social history is essential. In addition, if hoarseness has persisted, laryngoscopy is indicated. Laryngoscopy can be performed indirectly using a mirror. If the hypopharynx and larynx are not adequately visualized with the mirror because of an excessive gag reflex, direct flexible fiberoptic laryngoscopy can be performed. A topical vasoconstrictor (e.g., 0.5% ephedrine) with a topical anesthetic (e.g., topical lidocaine) can be applied in the nose to improve patient comfort; however, this is not always necessary. The flexible fiberoptic nasopharyngoscope is then passed through the pharynx to visualize the larynx.

Radiography

Indications for imaging the larynx include evaluation of laryngeal tumors and traumatic laryngeal injuries. CT or MRI is performed to evaluate depth of invasion with tumors. CT is the preferred method for imaging malignant and nonma-

Radiography

Imaging of the oral cavity and oral pharynx can greatly aid in the examination of these areas in certain circumstances. Plain films or CT scanning of the mandible can aid in delineating fractures, infections, cysts, or tumors. A panoramic film is particularly useful in evaluating bony abnormalities of the mandible. CT and MRI scans are useful in evaluating tumors of the oral cavity and oropharynx. Not only do these scans help by differentiating solid, cystic, and vascular lesions, but they also can distinguish the depth of the lesion and its relationship to important neurovascular structures.

Imaging is also extremely useful for masses with extension into the submental and sublingual space. CT or MRI can help determine whether the lesion is inflammatory (Ludwig's angina), cystic (plunging ranula), vascular (hemangioma), or neoplastic (squamous cell carcinoma or salivary gland tumor). Enhanced CT scanning is particularly helpful in the diagnosis of peritonsillar abscess and in differentiating this condition from peritonsillar cellulitis. The scan sometimes shows poorly circumscribed "phlegmonous" changes that represent *early* abscess formation. This knowledge helps in formulating appropriate treatment for these patients.

eTable 19-7 Treatment of Dysphagia

Cause	Treatment
Carcinoma of distal third of esophagus with no metastases	Surgery
Carcinoma of middle third of esophagus	Surgery and radiation therapy and/or chemotherapy
Carcinoma of proximal third of esophagus	Radiation therapy
Benign strictures and rings less than 13 mm in diameter	Dilation with Hurst or Maloney mercury bougies
Neuromuscular disease, reflux esophagitis with inflammation, or scleroderma	Chronic antireflux medication, PPIs, or H_2 blockers and elevating head of bed
Achalasia	Botulinum toxin, pneumatic dilation of LES, sphincterotomy (Heller's procedure)
Diffuse esophageal spasm	Medications (anticholinergics, calcium channel blockers, nitrates, hydralazine)

PPI, Proton pump inhibitor; *LES*, lower esophageal sphincter.

lignant disease, although MRI has certain indications. Traumatic injuries are best evaluated by CT scan. The modalities to assess stridor are directed by the history and physical examination. Available imaging techniques include chest radiography (PA and lateral films), neck radiography (PA and lateral films), and fluoroscopy. Contrast esophagography may be necessary to evaluate for vascular anomalies that can cause airway and esophageal compression or to rule out tracheoesophageal fistulas.

Special Studies

Airway fluoroscopy is useful for evaluating pediatric stridor. Fluoroscopy provides a dynamic picture of the entire airway from the nasopharynx to the carina. This facilitates the identification of obstructive lesions or foreign bodies during both phases of respiration.

A more specialized examination called video laryngeal stroboscopy (VLS) is performed by a speech/language pathologist. This technique allows highly detailed views of the larynx and also allows evaluation of the gross movement of the vocal cords and their vibratory motion. VLS is an excellent modality to photo-document lesions of the laryngopharynx. VLS is also helpful to voice therapists in identifying and following patients with voice disorders, including hoarseness and paradoxical vocal cord dysfunction (see later discussion).

Finally, direct laryngoscopy using a rigid laryngoscope can be performed, usually in the OR. Indications include the need for further evaluation and biopsy of pathology of the larynx.

Laryngitis

Laryngitis is the most common cause of acute hoarseness. It is secondary to diffuse swelling of the larynx. Viral infections are the most common cause and are often associated with other upper respiratory tract symptoms. Treatment is conservative, and recommendations include relative voice rest and avoidance of inhalational substances such as cigarette smoke or other irritating substances. Humidification may be helpful. Symptoms from viral laryngitis usually improve within days; if hoarseness persists longer than the typical several days, the physician should consider other causes, such as bacterial infection with respiratory organisms (e.g., *M. catarrhalis, H. influenzae*). For this reason, a course of antibiotics directed at these pathogens may be helpful, especially if symptoms persist even after conservative treatment.

Other uncommon infectious causes of laryngitis include tuberculosis and syphilis. Fungal infections can also localize to the larynx. Of these, *Candida albicans* is the most common and is found in immunocompromised patients, patients using inhaled steroids, and those using long-term, broad-spectrum antibiotics. Characteristic findings on examination include a diffuse reddened mucosa covered by white patches. Topical treatment includes nystatin, miconazole, or clotrimazole; systemic therapy includes fluconazole or ketoconazole.

Stridor

Stridor is noisy breathing. Stridor is a symptom, not a definitive diagnosis. It results from turbulent airflow and results from some degree of airway obstruction, usually at the level of the laryngopharynx or trachea. Stridor can affect both adults and children, but children present special diagnostic and therapeutic challenges because the antecedent history may be limited, physical examination is more challenging, and their small airways are more susceptible to critical obstruction. Because of the small airway diameter in infants and children, even small and subtle abnormalities can cause stridor and obstruct the airway.

It is helpful to localize stridor in the respiratory cycle. In general, *inspiratory* stridor typically is caused by an obstruction at or above the level of the true vocal cords, whereas *expiratory* stridor is usually localized to the more distal tracheobronchial tree. Biphasic stridor is usually caused by an obstruction at the true vocal cords, typically at the immediate subglottic level.

The medical history provides valuable information in the evaluation of stridor. In the pediatric population, a history should include questions about cyanosis, feeding difficulty, failure to gain weight, and retractions. Time of onset of stridor, birth history, prematurity, and need for immediate intubation at birth may help to identify the cause of stridor. A past medical history of smoking or ethanol abuse in an adult patient should raise the suspicion of a neoplastic process. Factors that tend to exacerbate the stridor are noted. These include change of intensity of stridor when in different positions, when crying, and when feeding. Previous intubation or laryngotracheal trauma can lead to acquired subglottic or tracheal stenosis. The presence of fever and a history of acute onset may be significant for an infectious process, including epiglottitis, croup, or tracheitis. New-onset stridor or increased stridor may portend impending airway distress, which would require more immediate workup, close monitoring, and possibly the need to stabilize the airway.

The severity of the obstruction and the need for intervention are sometimes difficult to determine. A child with a partially obstructed airway can have stridor but may not appear to be in respiratory distress. Intervention might or might not be needed. Examination should include documentation of stridor in the phase of respiration and whether suprasternal or intercostal retractions are present. The patency of the nose, oral cavity, and oropharynx should be noted. Large tonsils and adenoids can contribute to obstruction of the airway. The neck should be palpated for any masses that can cause extrinsic compression. Significant obstruction can cause signs of tachycardia, tachypnea, confusion, restlessness, or obtundation. Visualization of the larynx is paramount. Flexible fiberoptic laryngoscopy is the most useful modality in the workup of stridor. This usually requires an ENT consultation. It is easily performed in infants and adults.

There are many causes of stridor in the adult and pediatric population. The differential diagnosis includes lesions affecting various parts of the airway (see **eBox 19-8** online). Pediatric patients are more likely to have congenital causes of stridor, whereas any age group may have stridor secondary to trauma; inflammation; neoplasia, polyps, or papillomas; and foreign bodies. Common congenital anomalies include laryngomalacia, true vocal cord paralysis, subglottic stenosis, laryngeal webs and clefts, subglottic hemangiomas, anomalous great vessels, and complete tracheal rings. Most cases of acute stridor are caused by inflammatory disorders, including croup, epiglottitis, and tracheitis. Chronic causes

eBox 19-8 Causes of Stridor

Nose

Nasal aperture stenosis
Choanal atresia (unilateral or bilateral)
Nasal polyposis
Neoplasia

Oral Cavity or Oropharynx

Ludwig's angina
Tonsil hypertrophy or acute tonsillitis
Peritonsillar abscess
Parapharyngeal abscess
Macroglossia
Retrognathia
Neoplasia

Larynx

Laryngomalacia
Bilateral vocal cord paralysis
Papilloma
Laryngotracheobronchitis (croup)
Epiglottitis
Papilloma
Neoplasia

Trachea

Neoplasia
Subglottic stenosis
Vascular anomalies
Hemangioma
Complete tracheal rings
Tracheoesophageal fistula
Tracheitis
Tracheomalacia
Enlarged thyroid
Foreign body
Neoplasia

of stridor are more likely to be congenital, neoplastic, or from airway stenosis.

The most common cause of acute stridor in the pediatric population is *croup* (laryngotracheobronchitis). The parainfluenza virus is the most common cause and affects primarily the subglottic area but can also affect other portions of the laryngotracheal complex. Stridor can be inspiratory or biphasic and is often associated with a barking cough. Radiographs usually show the typical subglottic narrowing caused by edema. The typical age group is 6 months to 2 years, but the condition can be seen in children up to 5 years. The infection and inflammation are usually self-limiting, and conservative management is recommended. Evidence supports the routine use of corticosteroids in most children with croup (Husby et al., 1993). Intervention at an earlier phase of the illness reduces the severity of symptoms and the rates of return to a health care practitioner for additional medical attention, ED visits, and hospital admissions. Many children respond to a single, oral dose of dexamethasone. For those who do not tolerate the oral preparation, nebulized budesonide or intramuscular dexamethasone are reasonable alternatives.

According to Cochrane Collaboration recommendations regarding glucocorticoids for croup, dexamethasone and budesonide are effective in relieving the symptoms of croup as early as 6 hours after treatment. Fewer return visits and (re)admissions are required, and hospital stay is shortened. Dexamethasone is also effective in patients with mild croup (SOR: A).

Severe cases of croup can manifest with significant respiratory distress or even obstruction. Therapy in these patients requires hospitalization (ICU in some cases); treatment includes corticosteroids, supplemental oxygen, fluids, humidification, nebulized racemic epinephrine aerosols, heliox, and occasionally intubation. Complications from croup include airway obstruction, pneumonia, pulmonary edema, and cardiac failure.

Laryngomalacia is the most common cause of chronic stridor in neonates and is characterized by high-pitched inspiratory stridor that can be intensified with agitation, feeding, and placement in the supine position. The disorder is caused by immaturity of the laryngeal cartilages and is typically seen on examination as an omega-shaped epiglottis and floppy aryepiglottic folds that partially obstruct the laryngeal inlet on inspiration. The key to the diagnosis lies in the history and typical findings on flexible fiberoptic laryngoscopy. Airway fluoroscopy provides a dynamic evaluation of the laryngotracheal complex and often shows a component of tracheomalacia as well (laryngotracheomalacia). Treatment is rarely needed, because the problem is self-limiting and resolves by 18 months of age. Gastroesophageal reflux is extremely common, because the increased pressure gradient needed for adequate ventilation causes an increase in acid reflux into the esophagus. This exacerbates the condition by causing a component of reflux laryngitis. Rarely, failure to thrive because of feeding problems, cor pulmonale, or persistent desaturations while asleep can indicate the need for surgical intervention to improve the airway. Even if uncomplicated laryngomalacia is suspected, ENT evaluation is still recommended to confirm the diagnosis.

True *vocal cord paralysis* is another common cause of congenital stridor and usually occurs at birth to 2 months of age. In newborns, the cause is usually injury to the vagus nerve sustained at birth, or it can be secondary to CNS abnormalities. Unilateral paralysis is fortunately more common than bilateral paralysis, which causes airway obstruction. The diagnosis is confirmed by flexible fiberoptic laryngoscopy. Workup also includes barium swallow, neck and chest radiography, and cardiology consultation to rule out a cardiothoracic cause of vagal paralysis. Treatment is rarely necessary because most patients improve or compensate adequately with the opposite vocal cord. Bilateral vocal cord paralysis manifests with a high-pitched stridor. MRI of the brain should be included in the workup to rule out hydrocephalus and Arnold-Chiari malformation. Emergent airway intervention by intubation or tracheotomy might be necessary.

Adults may also be affected by vocal cord paralysis, either unilateral or bilateral. Possible etiologies include neoplastic processes, traumatic injuries to the recurrent laryngeal nerve, or idiopathic causes. If the cause is known (e.g., occurring after cervical surgery resulting in recurrent laryngeal nerve injury), further workup may not be recommended. If the cause is not clear, imaging of the brain, neck, and chest is ordered to rule out a compressive lesion affecting the recurrent laryngeal nerve.

Recurrent respiratory papillomatosis occurs in all ages but is more common in children. It is the most common benign tumor of the airway and is usually found on the true vocal cords and supraglottic and subglottic areas. The causative agent is human papillomavirus. Symptoms usually begin with hoarseness or aphonia and progress to stridor and dyspnea at a later stage of disease. Treatment is by endoscopic removal using the carbon dioxide laser or pulsed dye laser (PDL), and recurrence is common. It can progress to complete airway obstruction and eventually death. Repeated procedures are required. Interferon has been used for severe cases. The antiviral drug cidofovir has demonstrated efficacy against recurrent respiratory papillomatosis and is considered a promising new adjuvant treatment for this disease. The HPV vaccination program in the female adolescent population may lead to a decreased incidence of pediatric laryngeal papillomas by reducing the vertical transmission of the virus from mother to baby.

Subglottic tracheal stenosis is caused by cicatricial scarring of the subglottic trachea and can be congenital or acquired. This area is often affected because the cricoid cartilage is the only complete ring in the trachea and is the narrowest segment in the airway. Acquired subglottic stenosis is most frequently a result of long-term intubation, with traumatic injury to the subglottic segment causing pressure necrosis and subsequent scarring. It may be idiopathic. Subglottic stenosis can occur in adults or children. Patients with severely stenotic segments causing significant respiratory symptoms usually require surgical intervention that includes splitting the subglottis with short-term stents, use of cartilage grafts, or placement of long-term stents.

Although many of the etiologies previously listed may also cause stridor in the adult population, most often an adult who presents with stridor will have this symptom secondary to subglottic tracheal stenosis, inflammation or edema of the larynx, neoplasia, or vocal cord paralysis. The workup is tailored to the history and endoscopic findings. In general, treatment of stridor depends on the etiology. The critical issue is stabilizing the airway in hopes of preventing catastrophe. Some conditions may respond to medical treatment, but any patient with stridor should be treated as having an impending

airway obstruction until proved otherwise. ICU observation may be required, with personnel available to stabilize the airway by intubation or tracheotomy. ENT consultation is recommended for further evaluation and more definitive treatment.

Laryngeal Trauma

Injury to the airway is an important cause of death in patients with head and neck trauma. Laryngeal trauma must be recognized early to avoid catastrophic sequelae. Securing the airway is the most important initial step in the management of these injuries to preserve life. The most preventable factor in morbidity and mortality is likely a delay in diagnosis. Less severe laryngeal injuries may initially go undiagnosed, whereas major injuries can lead to early mortality.

Blunt trauma is a common cause of death in motor vehicle crashes. The mechanism of blunt laryngeal trauma is typically caused by a hyperextension of the neck (i.e., against the dashboard) with compression and fixation of the larynx against the cervical spine, which leads to fracture or comminution of cartilage with associated soft tissue injury. Laryngotracheal disruption can occur from "clothesline" injury, which can occur with motorcycle and snowmobile accidents.

Penetrating trauma is becoming more common with an increase in civilian violence. Knife and gunshot wounds are the most common cause of death in homicide cases. Other traumatized structures in the neck can include the great vessels, the esophagus, and the cervical spine.

Signs of laryngotracheal trauma include tenderness over the larynx, anterior neck contusion, subcutaneous emphysema, palpable fractures or crepitus, loss of thyroid prominence, tracheal deviation, and hemoptysis. Symptoms include hoarseness, shortness of breath, inability to tolerate the supine position, and dysphagia.

Examination should include flexible fiberoptic laryngoscopy in every patient, if possible, to evaluate the anatomy and function of the larynx. Diagnostic imaging includes cervical spine films, chest films, and CT scan. Unless physical examination and flexible fiberoptic laryngoscopy are normal, CT should be done in most cases. The decision to take a patient to the OR is based on history, physical examination, flexible fiberoptic laryngoscopy, and CT scanning.

As with any trauma patient, management of the airway is of primary importance. Some controversy still exists on the optimal management of the airway. Most authors recommend awake/local tracheotomy as the safest and least traumatic method of securing the airway in an adult patient with laryngeal trauma. Some reports recount the disastrous outcome of a lost airway after attempted oral or nasal intubation in patients with laryngeal trauma. However, many still advocate intubation as the initial method of securing the airway. Emergency cricothyroidotomy can be performed if time does not permit a formal tracheotomy.

Hoarseness

All patients who present with hoarseness should be questioned about the history, the duration, and the progression of symptoms. Hoarseness may be categorized as chronic or acute. Acute hoarseness is rarely secondary to a malignant process. Acute hoarseness usually results from vocal abuse, laryngitis, or smoking. Malignancy should be considered in patients with chronic hoarseness, but the differential also includes GERD, polyps, nodules, neurologic disorders, papillomas, and functional voice disorders (see **eTable 19-8** online).

Other symptoms can coexist with hoarseness. Cough can be secondary to irritation of the vocal cords from acute or chronic inflammation but can also indicate cancer of the larynx or lung. Dysphagia or odynophagia can be present from disorders of the pharynx and esophagus. Hemoptysis with hoarseness should be considered secondary to a malignancy until proved otherwise. A history of smoking and vocal abuse is an important consideration. Clear visualization of the larynx by indirect or direct laryngoscopy is absolutely necessary for all patients who present with hoarseness that does not resolve on its own or with medical therapy. This can require referral to an otorhinolaryngologist unless the family physician has training and experience in the procedures.

An unusual cause of voice problems is *spasmodic dysphonia* (or laryngeal dystonia). The exact etiology is unknown but is thought to be a CNS condition classified under "focal dystonias." It typically causes a harsh staccato voice but may cause breathiness. Spasmodic dystonia responds poorly to voice therapy but has been shown to respond very well to botulinum toxin injections into the larynx, done endoscopically or externally. The treatment weakens the muscles and lessens the symptoms for several months. Repeat injections are usually done, and response to treatment can decrease over time.

Vocal Cord Paralysis

Vocal cord paralysis can manifest itself as hoarseness. However, many patients are able to maintain a relatively normal voice because of compensation from the opposite vocal cord. Patients can present with shortness of breath while conversing, cough when swallowing, aspiration, or recurrent pneumonia. They may complain of the inability to hold a breath while exerting against a closed glottis (Valsalva maneuver). Visualization of the larynx by indirect or flexible fiberoptic nasolaryngoscopy usually reveals an immobile, sluggish vocal cord in a paramedian position.

Paralysis can result from peripheral (recurrent laryngeal or vagus) nerve involvement or a CNS disorder (e.g., CVA). Approximately 90% of vocal cord paralyses result from dysfunction of the peripheral nerve. Most causes of paralysis are found after a careful history and physical examination (see **eBox 19-9** online). Because of the course of the recurrent laryngeal nerve around the arch of the aorta on the left and around the subclavian artery on the right, a chest x-ray film is initially necessary to rule out compression from an intrathoracic process invading or compressing the nerve. CT or MRI may be useful for imaging the brain or the course of the recurrent laryngeal nerves. VLS is very useful and often yields further diagnostic and prognostic information.

Surgical trauma remains the most common cause of unilateral vocal cord paralysis. This is common after thyroidectomy, carotid artery surgery, or transcervical spine procedures. Neoplastic processes, including thyroid, lung, and esophageal cancers, must always be ruled out as a cause of either compression or invasion. Skull base tumors and mediastinal lesions are less common causes of paralysis. Careful palpation of the neck to rule out masses and evaluation of other cranial nerves help identify these problems.

eBox 19-9 Causes of Vocal Cord Paralysis

Neoplasm

Causing Nerve Compression

Bronchial tumors
Esophageal tumors
Jugular foramen tumors (glomus jugulare)
Laryngeal carcinoma
Neck tumors
Thyroid tumors

Causing Direct Paralysis

Invasive thyroid cancer

Trauma

Iatrogenic

Carotid artery surgery
Cervical spine surgery (anterior approach)
Esophageal surgery
Heart surgery (ligation of patent ductus arteriosus)
Thyroid surgery

Blunt

Birth trauma
Clothesline injury
Fighting
Motor vehicle crash

Penetrating

Gunshot wound
Knife wound

Neurologic

In Children

Arnold-Chiari malformation (bilateral paralysis)
Hydrocephalus
Low tentorium cerebelli
Meningomyelocele

In Adults

Amyotrophic lateral sclerosis
Encephalitis
Multiple sclerosis
Poliomyelitis
Pseudobulbar palsy
Wallenberg's syndrome

Other

Idiopathic causes

eTable 19-8 Common Causes of Hoarseness

Condition	Cause
Acute	
Acute laryngitis	Viral or bacterial
Vocal cord hemorrhage	Vocal abuse or misuse
Postnasal drip	Sinusitis or allergies
Gastroesophageal reflux	Gastric acid
Neck trauma	Blunt or penetrating trauma
Intubation granuloma	Short or long periods of intubation
Psychiatric problems	Conversion disorder, hysterical aphonia
Chronic	
Chronic laryngitis	Viral, bacterial, or smoking
Malignancy	Squamous cell carcinoma, thyroid cancer
Vocal cord nodules	Vocal cord abuse or misuse
Vocal cord polyps	Vocal cord abuse or misuse, smoking, allergies
Papillomas	Recurrent respiratory papillomatosis
Vocal cord paralysis	Cancer, trauma, stroke, degenerative disease

KEY TREATMENT

The American Academy of Otolaryngology–Head and Neck Surgery produced a consensus statement of Clinical Practice Guidelines for Hoarseness (Dysphonia).

STRONGLY RECOMMENDED TREATMENT

The quality of data supporting the benefits of treatment outweighing the potential harm is strong (Grade A, B):

1. Clinicians should not routinely prescribe oral antibiotics to treat hoarseness.
2. Voice therapy should be employed for patients with hoarseness that affects voice-related quality of life.

RECOMMENDED TREATMENT

The benefits of treatment outweigh the risks, but the data are not as strong (Grade B, C):

1. Hoarseness should be diagnosed in a patient with an altered quality of voice.
2. Patients with hoarseness should be assessed with history or physical exam with attention to previous factors or treatments that may have affected the recurrent laryngeal nerve or larynx. This may include neck or chest surgery or radiation therapy, endotracheal intubation, tobacco use, or occupational vocal overuse.
3. The clinician may perform laryngoscopy (or refer) if hoarseness persists after 3 months, or sooner if suspicion of serious illness is high.
4. CT or MRI should not be performed until the larynx has been visualized (these tests may not be necessary).
5. Antireflux medications should not be prescribed in the absence of other signs or symptoms of reflux.
6. Clinicians should not routinely prescribe oral corticosteroids to treat hoarseness.
7. Laryngoscopy should be performed before recommending voice therapy.
8. Clinicians should advocate for surgery as an option in cases of suspected malignancy, soft tissue lesion, or glottic insufficiency.
9. Patients should be referred for possible treatment with botulinum toxin in cases of spasmodic dysphonia.

OPTION

There is only weak evidence that the benefit of treatment outweighs the risk (Grade D):

1. The clinician may perform laryngoscopy (or refer) at any time after diagnosis of hoarseness.
2. Antireflux medications may be prescribed in patients with hoarseness if there are signs of chronic laryngitis with laryngoscopy.
3. Patients with hoarseness should be educated on control and prevention methods.

SOR: A; B.

Bilateral vocal cord paralysis typically manifests with significant respiratory distress caused by obstruction of the glottis from bilateral medialization of vocal cords. Many of these patients need emergent establishment of the airway by intubation or tracheotomy. Causes of bilateral vocal cord paralysis include thyroid or cervical spine surgery or CNS disorders. Hydrocephalus or an Arnold-Chiari malformation can cause bilateral paralysis via brainstem herniation with stretching of the vagus nerves. Treatment in these circumstances is aimed at stabilizing the airway and treating the underlying problem, with the paralyzed cord usually returning to normal function after a few months.

There are numerous surgical techniques to improve vocalization. Endoscopic injection of autologous, allogenic, or alloplastic substances can provide temporary and even permanent improvement by medializing the weak vocal cord so that the mobile vocal cord can make contact. Open surgical approaches can also be performed for permanent unilateral paralysis, with excellent results. Medialization of the vocal cord with the use of alloplastic materials is now common. Surgical options to correct permanent bilateral vocal cord paralysis include removal of a portion of the arytenoids or vocal cords to open the airway; permanent tracheotomy is a last resort.

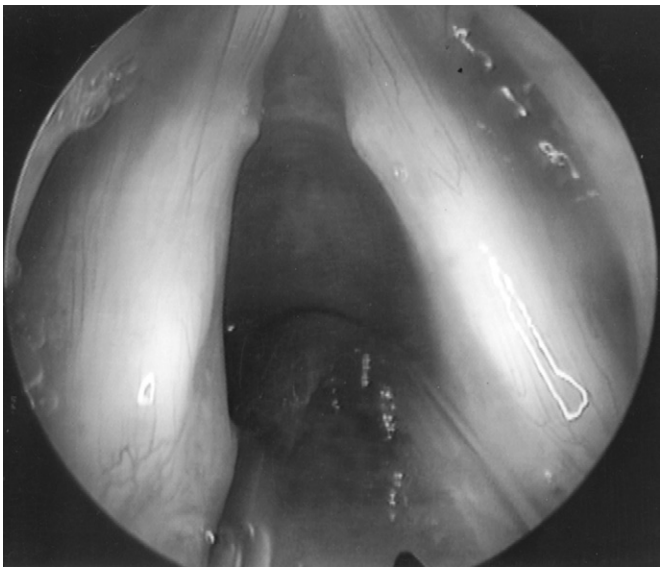


Figure 19-7 Intraoperative photograph of true vocal cords with nodules in 25-year-old teacher.

Vocal Cord Nodules

Vocal cord nodules result from long-term vocal overuse or abuse. Nodules are typically seen as symmetric raised areas at the anterior aspect of each vocal cord. These occur more often in women, boys, lecturers, coaches, and professional singers. The most common symptom is hoarseness and a persistent raspy voice. Smoking, allergies, and GERD tend to aggravate the condition and can prevent healing.

Nodules are always bilateral and classically occur at the junction of the anterior one third and posterior two thirds of the vocal cords (Fig. 19-7). Nodules must be distinguished from polyps, which are smooth and often unilateral, and granulomas, which tend to be located more posteriorly on the vocal cord. Treatment is initially conservative. The patient is referred for voice therapy, which consists of counseling, vocal reeducation, relative voice rest, and psychotherapeutic rehabilitation. Most patients respond after several sessions of voice therapy, but satisfactory improvement can take up to 12 to 18 months. Measures to control acid reflux and avoidance of irritating substances such as cigarette smoke are also routinely recommended. Surgery is reserved for rare patients who do not respond to voice therapy and consists of endoscopic removal. Postoperative voice therapy and acid reflux control are always indicated after surgical removal of nodules.

Reflux Laryngitis

Reflux laryngitis, also known as *laryngopharyngeal reflux* (LPR), is a relatively common condition. Many patients do not have the classic symptoms of GERD, including heartburn, and

the correct diagnosis is often initially overlooked. Constant throat clearing may be the only presenting symptom. Other manifestations include a feeling of a lump in the throat with a choking sensation *(globus pharyngeus)*, odynophagia, dysphagia, chronic cough, and hoarseness. The patient may also complain of postnasal drainage. Spicy foods, fats, caffeine, chocolate, beer, milk, and orange juice are known to exacerbate the condition by lowering LES pressure. Several medications increase reflux of acid into the esophagus, such as beta blockers, calcium channel blockers, diazepam, and progesterone. Obesity and sleep apnea also predispose the patient to GERD and reflux laryngitis.

A careful history and examination allow the diagnosis to be made. Findings on indirect or flexible fiberoptic laryngoscopy are nonspecific and include edema, erythema, and redundancy of the mucosa around the arytenoids and postcricoid area. Occasionally, small granulomas are present posteriorly near the vocal processes of the arytenoids. Studies that help confirm the diagnosis of GERD include barium swallow, pH probe, esophageal manometry, and esophagoscopy. Gastroenterology consultation may be necessary.

Often the diagnosis of reflux laryngitis cannot be confirmed or excluded with a trial of empiric therapy. The treatment for reflux laryngitis consists of diet and lifestyle modifications and acid-reducing medications. Diet modification includes avoidance of foods and substances known to increase acid reflux. Lifestyle modifications include avoidance of eating near bedtime, elevation of the head of the bed 6 to 10 inches (15-25 cm), weight loss, and avoidance of tight-fitting clothing. Medical therapy usually begins with a trial of H_2 blockers or preferably daily dosing of proton pump inhibitors (PPIs). Refractory or severe cases usually respond well to twice-daily PPI therapy. Although most patients respond to treatment, resolution of symptoms can take several months after initiating proper treatment. It is critical that visualization of the larynx be obtained early if the patient's symptoms persist, to rule out more serious pathology such as neoplasia.

Cancer of the Larynx

Squamous cell carcinoma is by far the most common malignancy of the larynx. Laryngeal cancer accounts for 1% to 4% of all malignancies detected every year. Peak age group is between 60 and 65 years old, and it is more prevalent in men than in women (8:1), although the incidence in women is rising. Malignancies of the larynx are most common in smokers and alcohol abusers. When both these factors are present, the risk of developing cancer becomes 50% greater than the additive risk of each. Only 2% to 5% of laryngeal cancer patients have no history of smoking. Thus, it is extremely important to elicit smoking and alcohol histories in patients with head and neck complaints.

Hoarseness can be a very early symptom, making most cancers of the larynx curable because they are detected early. For this reason, hoarseness should never be simply attributed to "laryngitis" without proper evaluation. Other symptoms, including sore throat and referred otalgia, can exist without hoarseness. These patients are often treated incorrectly with antibiotics for an extended period and referral is delayed, thereby increasing morbidity and mortality.

Detection of cancer requires visualization of the larynx. Indirect or direct laryngoscopy usually shows a discrete, well-circumscribed exophytic lesion in the endolarynx, most frequently on one of the true vocal cords. A critical concern is potential airway obstruction in bulky tumors; emergent airway intervention is fairly common. The neck should be palpated to evaluate for cervical lymphadenopathy. Metastasis to the lungs or brain is also possible.

Several modalities of treatment exist for laryngeal cancer. Carcinoma in situ and leukoplakia can be treated with laser vaporization. Invasive carcinoma is usually treated with radiation or surgery or both, depending on the stage of disease. Numerous partial-laryngectomy surgical techniques are available that have high cure rates and preserve the voice. Total laryngectomy with a permanent tracheostomy is reserved for late-stage and recurrent tumors. Speech restoration is still possible after total laryngectomy. In some institutions, cancers of the larynx are treated initially with radiation, with significant rates of cure and voice preservation. Radiation therapy and chemoradiation can also be used to preserve the larynx, avoiding total laryngectomy. Studies to determine the ideal chemo/radiation treatment are ongoing. Patients unresponsive to radiation or chemotherapy are treated surgically, usually with total laryngectomy. Neck dissection is required in patients with cervical metastases.

The Neck

Physical Examination

See the discussion online at www.expertconsult.com.

Neck Masses

Proper evaluation of a neck mass includes obtaining a complete history and performing a thorough examination of the neck and all mucosal surfaces. Imaging options include CT with contrast, MRI with contrast, and ultrasound, which may assist in the diagnosis and help define the extent of a lesion. Classification includes congenital, inflammatory, and neoplastic disorders. In general, pediatric neck masses are usually inflammatory, followed by congenital lesions. Neoplasia occurs less often in pediatric patients. In adults, neoplasia is by far the most common entity, with inflammatory causes less common and congenital masses rare.

Congenital Neck Masses

Congenital neck masses are common in children but can occur in any age group. These are the most common cause of noninflammatory neck masses in the pediatric population. Classifying these into midline or lateral location helps in the workup because congenital neck masses occur in consistent anatomic sites (see **eBox 19-10** online).

Midline Neck Masses

Thyroglossal duct cysts are the most common anterior midline neck masses in children. They are remnants of the descending tract of the thyroid gland from the foramen cecum at the base of the tongue to its normal position in the lower neck.

Neck: Physical Examination

Examination of the neck is an important part of the routine ENT examination. Of utmost importance is identifying abnormal masses. Several normal anatomic structures are present and can be confused for masses in some cases. These structures include low-lying submandibular glands, calcified carotid arteries, and transverse cervical processes. Masses in the neck can be secondary to enlargement of lymph nodes, thyroid masses, salivary gland masses, or a variety of other abnormalities. Any questionable neck mass should be referred to a head and neck surgeon if resolution does not occur promptly after appropriate therapy.

eBox 19-10 Differential Diagnosis of Neck Mass

Congenital

Midline

Dermoid cyst
Thyroglossal duct cyst
Ranula (plunging)

Lateral

Branchial cleft cyst
Lymphangioma (cystic hygroma)

Inflammatory

Acquired immunodeficiency syndrome
Atypical mycobacteria
Cat-scratch disease
Cervical adenitis (viral, bacterial)
Granulomatous (sarcoidosis)
Histoplasmosis
Infectious mononucleosis
Toxoplasmosis
Tuberculosis

Neoplastic

Benign

Epidermoid inclusion cyst
Fibroma
Lipoma
Neurofibroma
Paraganglioma
Schwannoma (neurilemoma)
Sebaceous cyst

Malignant

Lymphoma
Metastatic squamous cell carcinoma
Neurofibrosarcoma
Rhabdomyosarcoma
Salivary carcinoma
Thyroid carcinoma

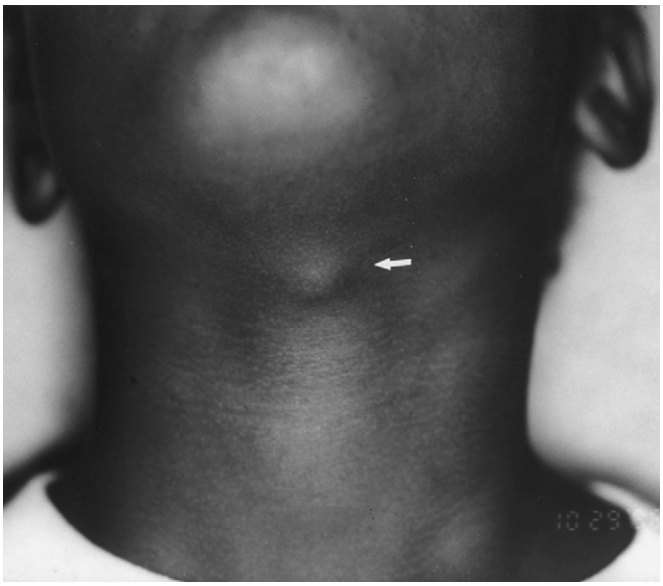


Figure 19-8 Photograph of 9-year-old child with midline neck mass *(arrow)* near the hyoid bone. Swallowing produced movement in the mass. Surgical excision with pathologic evaluation demonstrated a thyroglossal duct cyst.

Cysts or fistulas can occur anywhere along this tract and can intermittently become infected. Physical examination usually reveals a 2-cm to 4-cm mass in the anterior neck that moves with swallowing and elevates with tongue protrusion (Fig. 19-8). Treatment is surgical excision of the cyst and the tract, including the midhyoid bone, to the base of the tongue (Sistrunk procedure). There have been reports of neoplastic transformation of these cysts. Recurrence is possible.

Dermoid cysts typically develop along midline embryonic fusion planes and are composed of ectodermal and mesodermal embryonic remnants. Their usual location in the neck is in the submental area. They are also found frequently along the dorsum of the nose. Dermoid cysts tend not to move with swallowing or elevate with tongue protrusion, unlike thyroglossal duct cysts. Treatment is surgical excision.

Ranulas are cystic lesions that are usually present in the floor of the mouth. They can become "plunging" and extend through muscle planes into the upper midneck area. Plunging ranulas are thought to occur from mucus extravasation from a blocked salivary duct. Physical examination of a plunging ranula reveals a cystic mass in the submental area with or without a cystic mass in the floor of the mouth.

Lateral Neck Masses

Branchial cleft cysts are common congenital abnormalities found in the lateral neck area and are caused by failure of obliteration of the embryonic branchial clefts during development. Abnormalities can also manifest as sinus tracts or fistulas in the skin. These can become intermittently infected, especially after URIs.

Second branchial cleft abnormalities are by far the most common. These usually manifest as masses anterior to the sternocleidomastoid muscle with or without a fistulous opening. The sinus tract passes between the external and internal carotid arteries and ends in the tonsillar fossa. Treatment is complete surgical excision of the cyst and sinus tract after control of infection with appropriate antibiotics. First branchial cleft cysts are less common and manifest as a duplication abnormality of the external auditory canal (type I) or as an infected mass near the angle of the mandible with a sinus tract passing superiorly through the parotid salivary gland (type II).

Lymphangiomas (cystic hygromas) are congenital lymphatic masses often found in the neck. They usually manifest during the first year of life and often enlarge after a URI. They can also increase in size after hemorrhage into the cystic cavities. Lymphangiomas form from a failure of complete development and subsequent obstruction of the lymphatic system. They are most frequently found as an asymptomatic mass in the posterior triangle of the neck but can also be found anteriorly, causing airway or swallowing problems. Physical examination reveals a nontender, fluctuant, soft, spongy mass without discrete margins. Surgery is the mainstay treatment for lymphangiomas, although other modalities of interest include intralesional injection sclerotherapy and systemic interferon.

Hemangiomas are the most common congenital malformations. Most are cutaneous, but they can also be found in deep tissues. The most common deep location in the head and neck is the masseter muscle. Hemangiomas are characterized by appearance at or after birth, followed by a rapid proliferative phase at 6 to 18 months of age. The lesion then reaches a plateau phase, followed by a slow, involutional phase over 6 to 8 years. Even large, uncomplicated lesions left untreated usually undergo almost complete resolution. Conservative management is almost always recommended.

Certain locations, including the nasal tip, lips, and eyelids, can cause severe functional or cosmetic deformities, and referral for removal may be appropriate. Massive lesions can cause high-output heart failure or a consumptive coagulopathy (Kasabach-Merritt syndrome), whereas others can become ulcerated, infected, or hemorrhagic. Other forms of treatment for complicated lesions include interferon alfa-2b and corticosteroids. Cutaneous lesions may be treated with laser therapy.

Neoplastic Neck Masses

Benign lesions in the head and neck include *lipomas* and *fibromas* and require no treatment unless they cause significant functional or cosmetic deformity. Sebaceous cysts and epidermal inclusion cysts are also common and usually need to be excised because of the high incidence of recurrent infection.

All neck masses in adults should be presumed to be malignant until proved otherwise. The most common malignant neck mass in adults is metastatic *squamous cell carcinoma*. Primary tumors are usually found in the upper aerodigestive tract or skin. Smoking and alcohol abuse are etiologic factors. Careful examination of all mucosal surfaces of the head and neck is crucial to identify the primary tumor site. Endoscopy with biopsies is the first step in the diagnosis; surgical treatment of the primary site involves neck dissection, radiation, or both.

Lymphomas are one of the most common malignancies of the neck in children but can occur at any age. Patients can present with lymphadenopathy associated with constitutional symptoms (night sweats, fever, weight loss), hilar adenopathy, or hepatosplenomegaly. Diagnosis is confirmed

by excisional biopsy. Most head and neck lymphomas are treated with a combination of radiation and chemotherapy.

Rhabdomyosarcoma is a common childhood neoplasm with peak incidence at age 5 years. Early symptoms include a painless, enlarging mass or symptoms related to obstruction by tumor. Approximately 35% of rhabdomyosarcomas manifest in the head, neck, and orbit. Embryonal rhabdomyosarcoma and botryoid sarcoma (a variety of embryonal rhabdomyosarcoma) are most common in the head and neck, accounting for 75% of cases. Other forms include alveolar and pleomorphic. Diagnosis is made by excisional biopsy, and treatment is multimodal, including surgery, radiation, and chemotherapy.

Carotid body tumors are paragangliomas that arise from the adventitia of the common carotid bifurcation. They are thought to arise from derivatives of neural crest cells and are members of the diffuse neuroendocrine system. They can be associated with other tumors of similar origin, including medullary thyroid carcinoma, parathyroid adenoma, and pheochromocytoma, in up to 7% of cases. Familial incidence is 8% to 10%, and inheritance is autosomal dominant. A high incidence of bilateral carotid body tumors occurs in familial paraganglioma syndromes, and 1% to 3% of these tumors actively secrete substances such as catecholamines or serotonin. Symptoms of catecholamine secretion include headache, perspiration, palpitations, pallor, and nausea. Screening for blood and urinary catecholamines should be performed to rule out a secreting tumor or pheochromocytoma. Symptoms of serotonin secretion are carcinoid syndrome, including diarrhea, flushing, severe headaches, and hypertension.

Other tumors of the head and neck arise from neurogenic tissue. These include schwannomas, neurofibromas, neurofibrosarcomas, and neuroblastomas. These tumors can manifest with associated cranial nerve deficits. Treatment is surgical excision.

Inflammatory Neck Masses

Reactive lymph nodes from a viral infection should be treated expectantly, whereas bacterial infections such as streptococcal tonsillitis or pharyngitis should be treated with appropriate antibiotics. In some cases, inflamed lymph nodes can suppurate and become abscessed, requiring incision and drainage. Masses thought to be reactive lymph nodes not responding to conservative management (antibiotics) often need referral for further evaluation to obtain a definitive diagnosis.

Cervical lymphadenitis can be caused by atypical mycobacteria, which may appear as a subcutaneous abscess with erythematous overlying skin (Fig. 19-9). Treatment includes excisional biopsy and appropriate antibiotics according to culture sensitivities. Incisional drainage is contraindicated and can cause chronic fistulization.

Cat-scratch disease is another pediatric infection that can manifest with lymphadenopathy. Most patients recount exposure to a cat, and many have a cutaneous lesion representing an inoculation site. The diagnosis is made by serologic testing for *Bartonella henselae*. The disease is self-limited.

Tuberculous cervical lymphadenitis (scrofula), caused by *Mycobacterium tuberculosis*, can manifest with bilateral lower lymph node enlargement. It is usually associated with pulmonary involvement, and treatment is with a multidrug regimen. Nodes not responding to treatment should be excised.

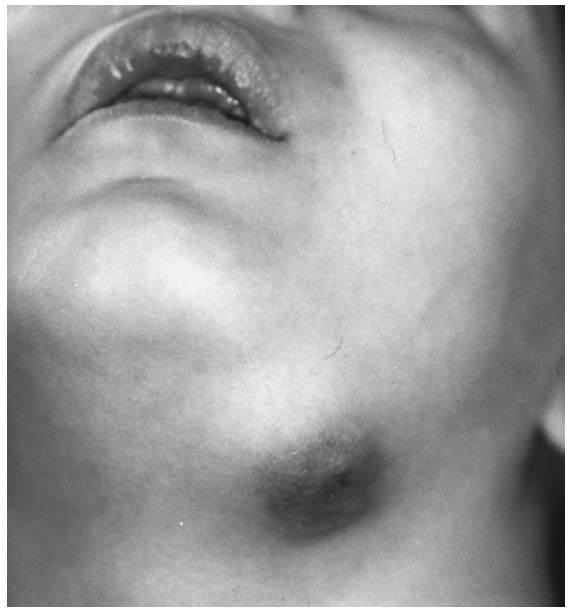


Figure 19-9 Photograph showing cervical adenitis with overlying skin erythema in a young child. Cultures revealed atypical mycobacteria.

Patients infected with *human immunodeficiency virus* (HIV) can present with asymptomatic lymph node enlargement. Persistent lymphadenopathy in AIDS patients is common, and most are followed if the nodes remain stable. Because of the higher incidence of non-Hodgkin's lymphoma and Kaposi's sarcoma in these patients, any suspicious neck masses with other constitutional symptoms should be referred for biopsy and tissue diagnosis. Fine-needle aspiration biopsy is appropriate, with indeterminate cytology requiring open excisional biopsy. Other possible causes of neck lymphadenopathy in immunocompromised patients include histoplasmosis, tuberculosis, atypical mycobacterial infections, and toxoplasmosis.

Sarcoidosis is a granulomatous disease that causes cervical lymphadenopathy and may be the presenting sign in 10% to 15% of cases. This disorder typically affects the African American population. Other findings include fever, sinusitis, parotid swelling, and hilar adenopathy on chest x-ray films. Diagnosis is classically made by tissue biopsy showing noncaseating granulomas. High angiotensin-converting enzyme level is common but not diagnostic. Other studies include cytoplasmic antineutrophil cytoplasmic antibody (c-ANCA) to rule out other granulomatous diseases, purified protein derivative and acid-fast bacillus stains to rule out tuberculosis, and Venereal Disease Research Laboratory (VRDL) and RPR tests to exclude syphilis.

Disorders of the Salivary Glands

The salivary glands consist of the paired parotid, submandibular, and sublingual glands and the minor salivary glands. Disorders of the salivary glands can be categorized into inflammatory, metabolic, and neoplastic problems (see **eBox 19-11** online). The history and physical examination facilitate differentiation among these categories.

eBox 19-11 Salivary Gland Enlargement

Inflammatory

Acute or chronic sialoadenitis
Cat-scratch disease
Cytomegalovirus
First branchial arch cysts or sinuses
Human immunodeficiency virus (lymphoepithelial cyst)
Mumps
Sialolithiasis
Tuberculosis

Metabolic

Medications
Sarcoidosis
Sjögren's syndrome

Neoplastic

Benign

Hemangioma
Oncocytoma
Pleomorphic adenoma (mixed tumor)
Warthin's tumor

Malignant

Adenoid cystic carcinoma
Lymphoma
Metastasis
Mucoepidermoid carcinoma
Squamous cell carcinoma

Inflammatory Disorders

Acute sialoadenitis is a common cause of painful enlargement of the parotid and submandibular glands. The organism is usually *S. aureus,* but it can also be caused by *S. pneumoniae* and other bacteria. The infection is secondary to salivary stasis caused by decreased production (dehydration, poor oral hygiene) or intrinsic or extrinsic obstruction (stones, strictures, masses). Patients present with exquisite tenderness over the gland, fever, and sometimes skin erythema. Purulence can be expressed from the duct with manual massage of the gland. Treatment includes antistaphylococcal antibiotics, adequate hydration, massage of the gland, sialagogues, and warm compresses. Abscess can occur and requires incision and drainage.

Mumps is a relatively common cause of painful unilateral or bilateral parotid gland enlargement in children. It is caused by a paramyxovirus, and diagnosis is confirmed by elevation of antibodies to the S and V virus antigens or by isolation of the virus in the urine. The incubation period is 2 to 3 weeks, and infection lasts 7 to 10 days. Treatment is conservative, with close follow-up to observe for possible complications such as pancreatitis, meningitis, orchitis, and hearing loss.

Sialolithiasis is a cause of intermittent salivary gland enlargement and is usually associated with eating. Most calculi occur in the submandibular duct, and most are radiopaque. Calculi arising in the parotid duct are less common and tend to be radiolucent. Symptoms include recurrent, unilateral, tender salivary enlargement that subsides within 24 to 48 hours. Physical examination usually reveals a palpable stone in the duct of the gland. Sialography is successfully diagnostic if a calculus is not palpable. CT scan and ultrasound are also effective in identifying calculi. Treatment depends on the location of the obstruction. Calculi near the terminal orifice are easily removed transorally. Symptomatic calculi located near the hilum of the gland usually require excision of the gland.

Patients with HIV infection can present with enlargement of salivary glands. Glands can become infiltrated with benign lymphoid tissue or *lymphoepithelial cysts.* The cystic lesions often occur in the tail of the parotid gland, and aspiration can provide temporary relief of symptoms. These patients are treated conservatively because cysts tend to recur after excision or aspiration procedures. Differential diagnosis of salivary gland enlargement in HIV-infected patients includes non-Hodgkin's lymphoma and Kaposi's sarcoma. Fine-needle aspiration or excisional biopsy of suspicious masses is performed for diagnosis of malignancy.

Included in the differential diagnosis of inflammatory lesions of the major salivary glands are tuberculosis, cat-scratch disease, CMV infection, and first branchial arch cysts and sinuses.

Autoimmune Disorders

Sjögren's syndrome is characterized by xerostomia (dry mouth) with or without parotid gland enlargement. Chronic inflammatory cells and lymphocytes infiltrate the glands, leading to fibrosis and atrophy of the parenchyma. Xerophthalmia suggests involvement of the lacrimal gland and causes a gritty or painful sensation of the eye. Primary Sjögren's syndrome involves the exocrine glands, only without association with other connective tissue disorders. Secondary Sjögren's syndrome is often associated with rheumatoid arthritis and other autoimmune disorders.

The SS-A and SS-B autoantibodies are positive in most cases of primary Sjögren's syndrome. Other useful laboratory tests include rheumatoid factor, antinuclear antibody, thyroid globulin antibody, and thyroid antimicrosomal antibody titers. Lip biopsy can confirm the diagnosis of Sjögren's syndrome, and referral to an immunologist is appropriate. Treatment includes proper oral hygiene, mouth rinses, and medications that stimulate salivary flow. Steroids are useful for severe cases.

Other causes of xerostomia include surgery and irradiation of the head and neck. Several common classes of medications can cause mouth dryness as a side effect. These include antihistamines, analgesics, anticonvulsants, antidepressants, and antihypertensives. Systemic disorders that can be associated with xerostomia include dehydration, diabetes, anemia, and overall debilitation.

Neoplastic Disorders

Most salivary gland neoplasms arise in the parotid gland, and most are benign. Approximately 80% of parotid gland tumors are benign, with *pleomorphic adenoma* (mixed tumor) being the most common in adults. *Hemangiomas* are the most common benign tumor in children, but malignancy in children is more likely than in adults when a solid mass is found in the salivary gland. Other benign tumors include Warthin's tumors (papillary cystadenoma lymphomatosum) and oncocytomas. The most common malignancy in adults and children is *mucoepidermoid carcinoma.* Other malignancies include adenoid cystic carcinoma, malignant mixed tumor, and squamous cell carcinoma. Treatment of salivary gland neoplasms is surgical excision.

Thyroid Masses

Careful examination for thyroid abnormalities should be performed on each patient in routine office visits. *Thyroid nodules* occur in about 5% to 10% of the population; of these, approximately 10% are malignant. Differential diagnosis of thyroid masses is presented in Box 19-9. Risk factors for malignancy include exposure to radiation and family history of medullary carcinoma. Factors that increase the possibility of carcinoma include hoarseness; age younger than 20 years or older than 45 years; male gender; presence of a firm hard nodule; and vocal cord paralysis.

The most common disorder in patients who present with a thyroid nodule is *nontoxic multinodular goiter.* The presence of multiple nodules is helpful in diagnosis, but only one dominant nodule may be apparent on physical examination. Nontoxic multinodular goiter may be endemic in iodine-deficient areas or sporadic. Nodularity is thought to be secondary to repeated episodes of deficiency of thyroid hormone, causing increased levels of thyroid-stimulating hormone (TSH), which results in hyperplasia of the gland. Symptomatic compression on the trachea or esophagus can occur. Surgery is reserved for functional problems caused by compression or to rule out malignancy. Chest radiography and CT are helpful in evaluating substernal extension of goiter.

Graves' disease is an autoimmune disease that causes diffuse thyroid enlargement, hyperthyroidism, infiltrative ophthalmopathy, and myxedema. It is the most common cause

Box 19-9 Thyroid Masses

Degenerative

Graves' disease
Nontoxic multinodular goiter

Thyroiditis

Acute thyroiditis
Chronic lymphocytic throiditis (Hashimoto's)
Fibrous thyroiditis (Riedel's)
Subacute thyroiditis (granulomatous, lymphocytic)

Neoplastic

Benign

Follicular adenoma

Malignant

Anaplastic carcinoma
Follicular carcinoma
Lymphoma
Medullary carcinoma
Papillary carcinoma

of hyperthyroidism and is more common in women age 20 to 50. Treatment modalities include antithyroid medications, radioactive iodine ablation, or surgical excision when medical treatment fails.

*Thyroiditis c*an cause nodular enlargement of the thyroid gland. Subacute thyroiditis may be a cause of intermittent hyperthyroidism from the release of stored thyroid hormone. Chronic lymphocytic (Hashimoto's) thyroiditis can cause diffuse nodular enlargement of the thyroid. Measurement of antithyroid microsomal antibodies is helpful but not specific for this disorder. Fibrous (Riedel's) thyroiditis is a rare cause of thyroid enlargement, and distinction from neoplasia can be difficult.

Neoplasms of the thyroid are classified as malignant or benign tumors. Follicular adenoma is the most common benign tumor. Malignancies include papillary (65%), follicular (20%), medullary (5%), and anaplastic (10%) carcinomas. Medullary carcinoma is associated with an elevated calcitonin level, with pheochromocytoma and parathyroid hyperplasia in multiple endocrine neoplasia (MEN) IIA, and with pheochromocytoma, mucosal neuromas, ganglioneuromatosis, and marfanoid body habitus in MEN IIB. Measurement of urinary catecholamines, vanillylmandelic acid, and metanephrine levels is necessary to aid in the diagnosis of pheochromocytoma, which must be treated before surgery for medullary thyroid carcinoma. Hypercalcemia is diagnosed by measuring serum calcium levels.

Initial evaluation of a suspected or palpable nodule includes a thyroid ultrasound and TSH level. If the TSH level is low, a radioiodine scan is obtained. If the nodule corresponds to an area of increased uptake, a biopsy is not necessary because almost all "hot" nodules are benign. Ultrasound will define the number and size of nodules and identify characteristics suggesting malignancy.

Fine-needle aspiration biopsy (FNAB) is indicated for nodules larger than 1 cm or with suspicious ultrasound or examination features. Ultrasound guidance may be used to improve the diagnostic yield for difficult-to-palpate nodules (see **eFig. 19-9**). FNAB is highly accurate. Results are reported as benign, indeterminate, malignant, or nondiagnostic. Benign nodules should be followed with serial examination and ultrasound. Repeat FNAB is indicated with a significant size change. An indeterminate diagnosis may represent a follicular lesion. Follicular neoplasms are not distinguishable as benign or malignant because fine-needle aspiration fails to identify the critical factor of vascular or capsular invasion. Therefore, a thyroid lobectomy may be necessary for diagnosis. Alternatively, a radioiodine scan may be obtained and surgery performed if the nodule is "cold." Nondiagnostic aspirates should undergo repeat FNAB. A malignant diagnosis often requires a total thyroidectomy with central compartment lymphadenectomy. Postoperative radioactive iodine is often given. Risks of thyroid surgery include recurrent laryngeal injury and hypoparathyroidism.

Prognosis for papillary thyroid carcinoma is excellent, especially in younger patients. Prognosis is good for medullary and follicular carcinoma and universally poor for anaplastic carcinoma (Cooper et al., 2006).

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The complete reference list is available online at www.expertconsult.com.

Web Resources

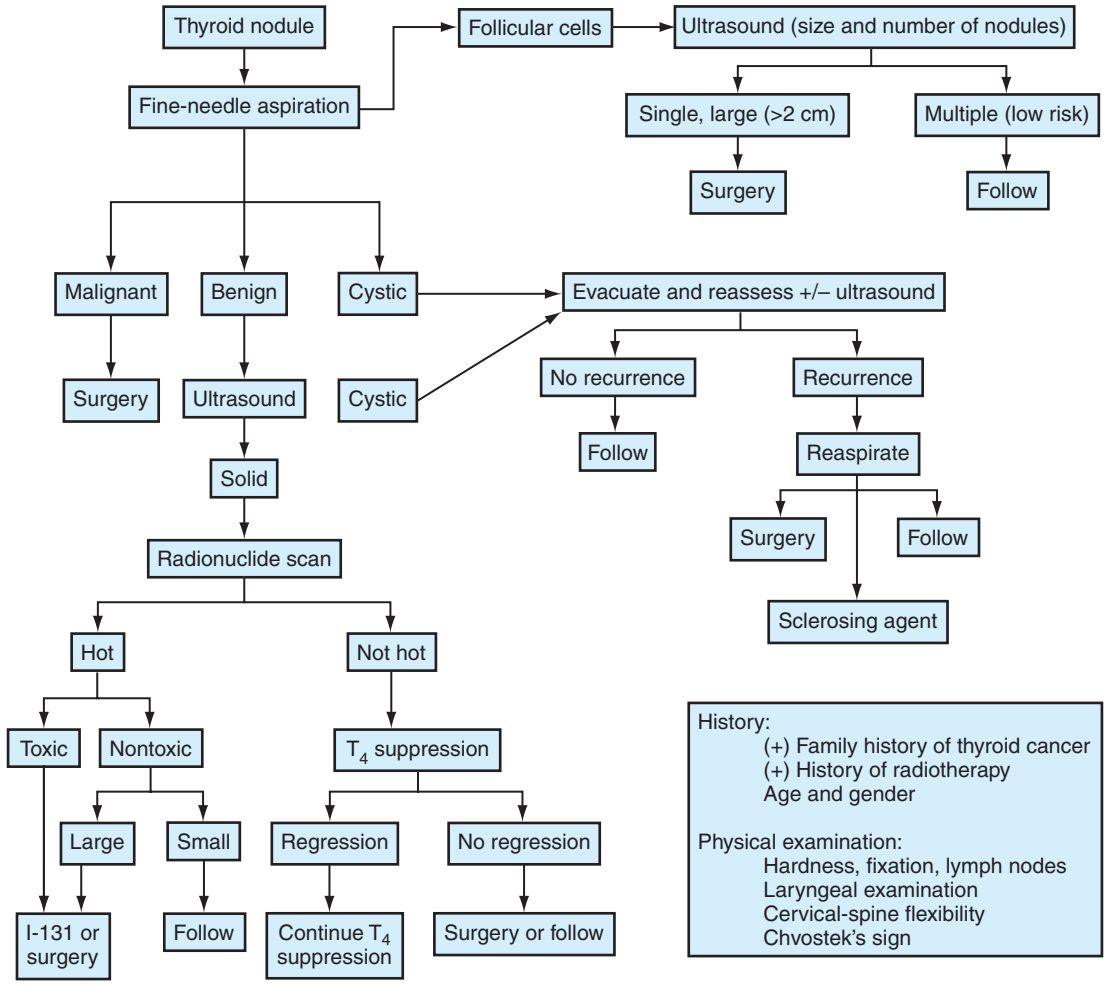
www.entnet.org
The American Academy of Otolaryngology–Head and Neck Surgery
Contains resources for physicians seeking information on ENT topics, as well as a section on patient education.

www.UTMB.edu/oto/
Dr. Quinn's Online Textbook of Otolaryngology, The Texas Nasal and Sinus Center, The Centers for Cancers of the Head and Neck, The Center for Audiology and Speech Pathology
Contains up to date information on all aspects of otolaryngology.

www.nidcd.nih.gov
The National Institute on Deafness and other Communication Disorders
Contains information about hearing, balance, smell, taste, voice, speech, and language

www.medlineplus.com
A service from the National Library of Medicine. Contains the most accurate database of the scientific medical literature plus a guide to over 9,000 prescription and OTC medications.

www.cochrane.org
The Cochrane Collaboration. Contains reviews of the latest literature in the field of otolaryngology.



eFigure 19-9 Algorithm for the diagnosis and treatment of thyroid nodules. *(From Gates GA. Current Therapy in Otolaryngology–Head and Neck Surgery, St Louis, Mosby–Year Book, 1998).*

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