averaged. Current literature suggests that the cut-off value that provides the best accuracy for the prediction of intracranial pressure >20 mm Hg is 5.7 to 6.0 mm, and increased intracranial pressure should be suspected with values above this threshold. The sensitivity and specificity in detecting increased intracranial pressure using a cut-off value of 5.7 to 6.0 mm are in the range of 87% to 95% and 79% to 100%, respectively.[34]

CAUTIONS WITH OCULAR US

- **If globe rupture is suspected, avoid any manipulation or pressure upon the globe or eyelid.**
- Limit the duration of ocular US examination, especially when using spectral and color Doppler, and **set the US unit for 'ocular imaging'**. The recommended exposure limits are half that of fetal imaging.
- Various artifacts may interfere with ocular ultrasound examination. Orbital emphysema can make it difficult to visualize contents of the orbit.
- Air bubbles within the vitreous, which may appear in the setting of trauma to the globe, may resemble an intraocular foreign body.

Acknowledgment: The authors gratefully acknowledge the contributions of John D. Mitchell, the author of this chapter in the previous edition.

REFERENCES

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

CHAPTER 242

Ear Disorders

Kathleen Hosmer

This chapter discusses common nontraumatic conditions affecting the external, middle, and inner ear. Selected traumatic conditions include auricular hematoma, burns, and frostbite. Lacerations to the ear are discussed in the chapter 40, "Face and Scalp Lacerations." Ear disorders in children are discussed in chapter 115, "Ear and Mastoid Disorders in Infants and Children."

ANATOMY

EXTERNAL EAR

The auricle, or pinna, is the visible external portion of the ear, whose trumpet shape enables it to collect air vibrations. It consists of a thin plate of elastic cartilage with a tightly adherent covering of skin. The external auditory canal is an S-shaped skin-lined tube that extends from the auricle to the tympanic membrane (TM). The outer one third of the external auditory canal is composed of an incomplete cartilaginous tube. Its thick skin supports hair follicles plus apocrine and sebaceous glands. The inner two thirds of the canal is composed of bone covered by a thin layer of tightly adherent skin, which is easily torn by minimal trauma.

The blood supply to the external ear is derived from the posterior auricular, superficial temporal, and deep auricular arteries. Venous drainage of the external ear is into the superficial temporal and posterior auricular veins, which then drain into the external jugular vein. The posterior auricular vein frequently connects to the sigmoid sinus, providing a route for extension of infected material into the intracranial cavity.

MIDDLE EAR

The middle ear is an air-containing cavity in the petrous temporal bone. It contains the auditory ossicles, which transmit vibrations of the TM to the perilymph of the internal ear. It communicates with the nasopharynx anteriorly via the eustachian tube and with the mastoid air spaces posteriorly via the aditus ad antrum (**Figure 242-1**).

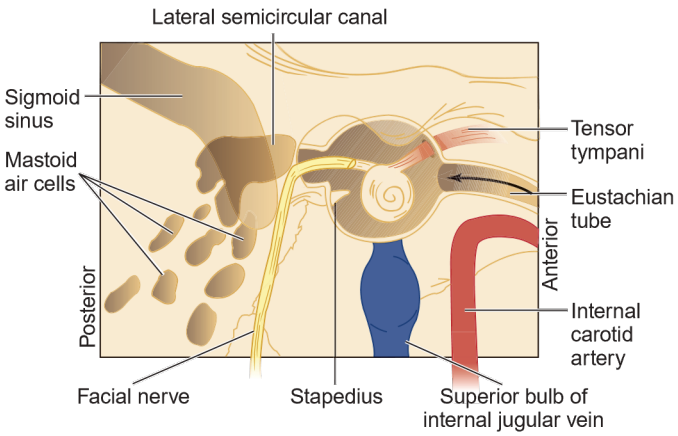

FIGURE 242-1. Sagittal section of the middle ear and related structures.

The TM is a thin, pearly gray, fibrous membrane that produces a cone-shaped light reflex anteroinferiorly when illuminated. Superiorly, the pars flaccida is the relatively slack portion of the membrane between the malleolar folds; the remainder of the membrane is tense and is called the pars tensa. The auditory ossicles are the malleus, incus, and stapes. Both the incus and the handle and lateral processes of the malleus are typically visible through the TM (**Figure 242-2**). Figure 242-1 shows the relationships of the facial nerve, sigmoid sinus, and internal carotid artery to the middle ear.

INNER EAR

The inner ear consists of the cochlea, which contains the auditory sensory receptors, and the vestibular labyrinth, which contains balance receptors. Cristae in the semicircular canals detect angular acceleration, and macules detect linear acceleration. Afferent nerves from the vestibular labyrinth connect to brainstem nuclei to maintain smooth movement of the eyes during head movement and to the cerebellum to control oculomotor and postural functions. Blood supply is from the vertebrobasilar system (**Figure 242-3**). The otolithic organs (utricle and saccule) lie in the vestibule. The internal auditory artery divides into the common cochlear artery and the anterior vestibular artery. The anterior vestibular artery provides the blood supply to the anterior and horizontal semicircular canals but not to the cochlea. Isolated occlusion of the anterior vestibular artery may therefore cause acute vestibular syndrome without hearing loss.

OTALGIA

Primary otalgia is caused by auricular and periauricular disease, whereas referred otalgia is caused by disease originating from remote structures.[1] Referred otalgia is common because the ear and several structures of the

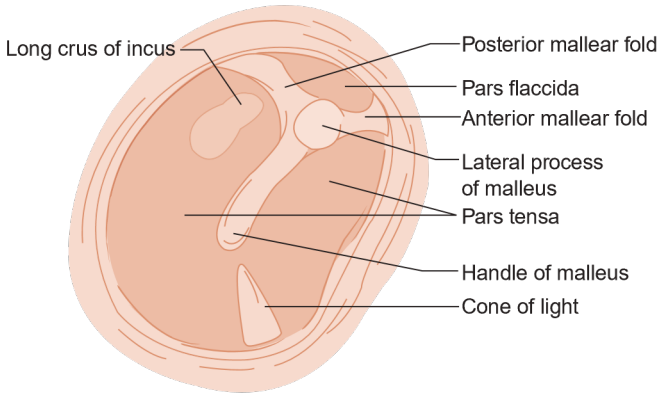

FIGURE 242-2. Right tympanic membrane as seen through the otoscope.

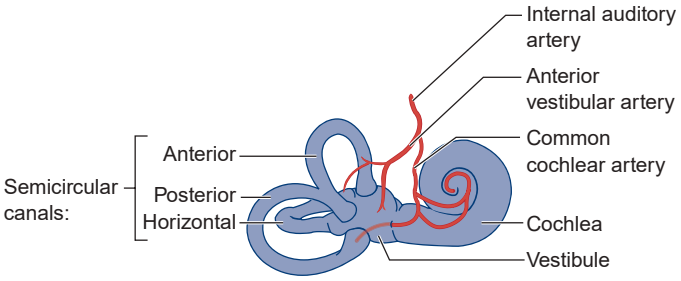

FIGURE 242-3. Schematic of the bony labyrinth containing the vestibular and auditory sensory organs.

head and neck share sensory innervation from the fifth, seventh, ninth, and tenth cranial nerves, as well as by the cervical plexus. **Table 242-1** lists common causes of primary and referred otalgia, including related neuralgias.[1]

TINNITUS

Tinnitus is the perception of sound without external stimulation. It may be constant, pulsatile, high- or low-pitched, hissing, clicking, or ringing. It is most prevalent between the ages of 40 and 70 years old.[2]

Tinnitus is divided into two types: objective and subjective. Objective tinnitus may be heard by the examiner. Subjective tinnitus is more common. Its exact origin is unknown, although it is believed to result from damage to cochlear hair cells. **Table 242-2** outlines causes of tinnitus.[2]

A number of drugs are associated with tinnitus, and some also cause hearing loss[2] (**Table 242-3**).

Accurate diagnosis usually requires referral to an otolaryngologist. Many medications have been suggested as therapies; antidepressants may help patients tolerate their tinnitus but do not change the course.

SUDDEN HEARING LOSS

Sudden hearing loss occurs over 3 days or less and is divided into sensorineural (cochlea, auditory nerve, or central auditory processing) and conductive (external ear, TM, and ossicles). Conductive hearing loss is more likely due to a reversible cause, such as otitis media, serous otitis, or a cerumen impaction. Indicators of poor prognosis include more severe hearing loss on presentation and the presence of vertigo. **Table 242-4**[3] lists causes of sensorineural hearing loss.

Viral infections, most typically mumps, have long been associated with sudden hearing loss.[4] Because of the terminal branches and interosseous location of the blood supply to the inner ear, the ear is uniquely vulnerable to a variety of vascular and hematologic diseases. Cogan's syndrome is an autoimmune disorder that presents with a bilateral hearing loss classically associated with tinnitus and vertigo.[5] Sudden hearing loss may also be caused by rupture of the TMs. Many common medications are implicated (Table 242-3).[3,5]

The evaluation begins with a complete history and physical examination. Differentiate conductive hearing loss from sensorineural hearing loss with a tuning fork. To perform the Weber test, place a vibrating tuning fork on the forehead and ask the patient where it is heard: it is normal to hear it equally in both ears; if the sound lateralizes to one ear, there is conductive hearing loss in that ear, or there is sensorineural loss in the opposite ear. To perform a Rinne test, place the vibrating tuning fork on skin over the mastoid bone of one ear, and then move the tuning fork to the entrance of the ear canal on the same side; the sound is normally heard better through air conduction at the entrance of the ear. If the sound is heard better over the mastoid bone, there is conductive hearing loss in that ear. Sudden conductive hearing loss may result from obstruction of the external auditory canal or from disturbances (or infection) of the TM or ossicles. Evaluate all current medications for possible ototoxic agents. A history of trauma or recollection of a "popping" noise preceding the hearing loss may indicate perforation of the

TABLE 242-1 Causes of Otalgia

Primary	Referred
Trauma	Temporomandibular dysfunction syndrome
Infection	Dental
Otitis externa	Abscessed teeth/dental carries
Otitis media	Malocclusion
Mastoiditis	Bruxism
Bullous myringitis	Eustachian tube dysfunction
Pinna cellulitis	Retro- and oropharyngeal
Cerumen impaction	Tonsillitis
Cholesteatoma	Abscess
Neoplasms	Neoplasm
Foreign bodies	Nasal cavity
	Sinusitis
Facial Neuralgias with Periauricular Pain	Deviated septum
Trigeminal neuralgia	Throat and neck
Ramsay Hunt syndrome	Foreign body
Great auricular neuralgia	Thyroid disease
	Neoplasm

TABLE 242-2 Common Causes of Tinnitus

Objective	Subjective
Vascular	Sensorineural hearing loss
Arteriovenous malformations	Hypertension
Arterial bruits	Conductive hearing loss
Mechanical	Head trauma
Enlarged eustachian tube	Medication side effects
Palatal myoclonus	Temporomandibular joint disorders
Stapedial muscle spasm	Anxiety, depression,
	Neurologic
	Acoustic neuroma
	Multiple sclerosis
	Benign intracranial hypertension
	Ménière's disease
	Cogan's syndrome

TABLE 242-3 Ototoxic Agents Causing Tinnitus and/or Hearing Loss

Loop diuretics	Chemotherapeutic agents
Ethacrynic acid	Cisplatin
Furosemide	Carboplatin
Bumetanide	Vinblastine
Salicylates and salicylate-containing compounds*	Vincristine
Nonsteroidal anti-inflammatory drugs*	Topical agents
Quinine	Solvents
Antibiotics	Propylene glycol
Aminoglycosides*	Antiseptics
Erythromycin	Ethanol
Vancomycin	Polymyxin B
	Neomycin

*Most commonly implicated agents.

TABLE 242-4 Causes of Sudden Sensorineural Hearing Loss

Idiopathic (71.0%)	**Trauma (4.2%)**
Infectious disease (12.8%)	Head injury
Unspecified respiratory infection (viral)	Acoustic trauma
Meningitis*	Barotrauma
Group A *Streptococcus*	**Vascular or hematologic (2.8%)**
Epstein-Barr virus	Cardiovascular disease
Toxoplasma gondii	Neurovascular disease
Syphilis	Hemorrhage (brain)
Herpes simplex virus	**Neoplastic (2.3%)**
Otologic disease (4.7%)	Vestibular schwannoma
Meniere's disease	Cerebellar angioma
Skull base or otologic surgery	**Other causes (2.2%)**
Autoimmune inner ear disease	Pregnancy related
Drug toxicity†	Nonotologic surgery

*Decreased incidence of hearing loss due to meningitis in the postimmunization era.
†See list of drugs in Table 3.

TM or ossicle dislocation. Coexistent tinnitus or vertigo may point to Ménière's disease. Also consider systemic illness.

The differential diagnosis includes both potentially reversible and potentially ominous causes (Table 242-4). If the physical examination does not identify the cause, consult otolaryngology. Treatment of idiopathic sudden sensorineural hearing loss is 60 milligrams of prednisone daily for 7 to 14 days or until follow-up, which is usually arranged within 2 weeks.[5-7]

ACUTE DIFFUSE OTITIS EXTERNA

Otitis externa includes infections and inflammation of the external auditory canal and auricle. It is divided into acute diffuse and malignant types. Acute diffuse disease is simply called *otitis externa* or *swimmer's ear.*

PATHOPHYSIOLOGY AND MICROBIOLOGY

Predisposing factors for the development of otitis externa are trauma to the skin of the external auditory canal and elevation of the local pH. Factors include frequent contact with water from swimming or bathing in hot tubs, pools, or freshwater lakes, and living in a humid environment. Trauma is most commonly due to scratching or overzealous disimpaction of cerumen. Cerumen is an acidic mixture of sebaceous and apocrine gland secretions and desquamated epithelial cells. It forms a physical barrier that protects the skin of the external auditory canal, whereas the acidic pH has antimicrobial properties.

The most common organisms are *Pseudomonas aeruginosa*, *Staphylococcus aureus*, Enterobacteriaceae, and *Proteus* species.[8] Otomycosis, or fungal otitis externa, is found in tropical climates and in the immunocompromised or after previous long-term therapy with antibiotics. Most cases are caused by *Aspergillus* or *Candida*.[9] Noninfectious causes include contact dermatitis from topical medications or resins in hearing aids, seborrhea, and psoriasis.

CLINICAL FEATURES

Acute diffuse otitis externa is characterized by pruritus, pain, and tenderness of the external ear. Physical signs include erythema and edema of the external auditory canal, which may spread to the tragus and auricle. Other signs are clear or purulent otorrhea and crusting of the external canal. As the disease progresses, the pain may become intolerable and occur with mastication or any movement of the periauricular skin. Increasing edema may eventually narrow the canal lumen and can lead to hearing impairment. In severe cases, infection may spread to the periauricular soft tissues and lymph nodes, and there may be lateral protrusion of the auricle secondary to inflammation.

TABLE 242-5 Topical Agents for Acute Diffuse Otitis Externa

Constituents	Comments
Ofloxacin 0.3%	Safe with perforations; one or two times per day
Ciprofloxacin 0.3%/dexamethasone 0.1%	Safe with perforations; two times per day
Acetic acid 2% solution	pH 4.5–6.0 (not safe with perforation)
Acetic acid 1%, hydrocortisone 2%	pH 3.0 (not safe with perforation)
Neomycin/polymyxin B/hydrocortisone	Ototoxic; higher risk of contact hypersensitivity; avoid in chronic otitis externa

TREATMENT

The treatment consists of analgesia, cleansing of the external auditory canal, acidifying agents, topical antimicrobials with or without steroids, and, in certain cases, adding an ear wick.[10,11] Cleansing may be done with gentle irrigation using hydrogen peroxide or saline, or gentle suction under direct visualization.[10] For the immunocompromised, atraumatic cleaning with aural suctioning under microscopic guidance can be done by an otolaryngologist.[10] Nonototoxic ototopical antibiotics are first-line therapy, particularly when the integrity of the TM is unknown or in the presence of a known TM perforation or tympanostomy tubes. Recommended topical agents are listed in **Table 242-5**.[11] Although there are few established cases of ototoxicity, there is a theoretical risk of both auditory and vestibular toxicity with the use of aminoglycosides, polymyxin, and acetic acid preparations.[10]

If cost is a factor and there are no known contraindications, acetic acid drops may be used because they are less expensive than the quinolones. In this case, a suspension should be used and not a solution; theoretically, a suspension has less chance of middle-ear penetration and resultant ototoxicity.

Instill the medication into the cleansed ear with the ear facing up, with this position held for 3 minutes. If edema of the external canal obstructs the lumen, insert a commercial wick to enhance delivery of topical drops throughout the full length of the canal.

For cases unresponsive to initial treatment, obtain bacterial and fungal cultures. Oral antibiotics are not routinely recommended[10] and should be reserved for febrile patients and those with periauricular extension (consider malignant otitis externa, discussed below). Instruct patients with otitis externa to avoid predisposing factors to eliminate recurrences. Strategies include ear plugs while swimming or bathing (cotton wool impregnated with petroleum jelly or commercial ear plugs), brief use of a hair dryer to remove water from the ear canal, and avoiding cotton-tipped applicators or other devices to remove cerumen.

In countries where otomycosis is more common, topical agents, such as clotrimazole 1% solution, are used.[12] Topical clotrimazole is available in the United States but not made specifically for otomycosis, and safety with ruptured TM has not been established. Fluconazole can be started (200 milligrams for one dose, then 100 milligrams daily for 3 to 5 more days);[13] however, the patient will need follow-up to determine the need for extended therapy. Patient growing *Aspergillus* in culture should be treated with voriconazole, 4 milligrams/kg PO two times daily.[9,13]

Instruct patients to follow up with their primary physician or an otolaryngologist in 1 week to be reevaluated; they should return to the ED for sudden worsening with fever or marked swelling.

MALIGNANT OTITIS EXTERNA

Malignant otitis externa is a potentially life-threatening infection of the external auditory canal involving the pinna and soft tissues with variable extension to the skull base, called *skull-base osteomyelitis.*

PATHOPHYSIOLOGY AND MICROBIOLOGY

Malignant otitis externa begins as a simple otitis externa that then spreads to the deeper tissues of the external auditory canal and infects cartilage, periosteum, soft tissue, and bone, with the normal anatomy of

the ear serving as the conduit for the spread of infection. Previously, >90% of cases were caused by *P. aeruginosa*,[13] but methicillin-resistant *Staphylococcus aureus* now accounts for 15% of cases.[14] Fungal disease occurs in diabetics and patients with immunocompromise.[15] Acquired immunodeficiency syndrome patients tend to be younger, have etiologic organisms other than *Pseudomonas*, and have a worse prognosis than patients without acquired immunodeficiency syndrome. The cerumen of diabetic patients has a higher pH than that of normal controls and represents an additional breakdown in local defense mechanisms. Small blood vessel disease of diabetics may lead to cartilaginous degeneration, further promoting the spread of infection.

CLINICAL FEATURES AND DIAGNOSIS

An individual with persistent otitis externa despite 2 to 3 weeks of topical antimicrobial therapy should be suspected of having malignant otitis externa. The typical presentation is severe otalgia (90%) and edema of the external auditory canal with otorrhea (70%).[16] Granulation tissue may be evident on the floor of the external auditory canal.

Examine both ears, inspecting both pinnas from the anterior, lateral, and posterior aspects. The infected ear will be erythematous, edematous, and more prominent than the unaffected ear. Assess nearby structures. Parotitis may be present, and trismus indicates involvement of the masseter muscle or temporomandibular joint. Cranial nerve involvement is a serious sign. The seventh cranial nerve is usually the first nerve affected by cranial extension, and the presence of dysfunction of the 9th, 10th, or 11th cranial nerve implies even more extensive disease. Lateral or sigmoid sinus thrombosis and meningitis are more serious possible complications.

Blood counts are typically normal. Erythrocyte sedimentation rate and C-reactive protein are frequently elevated but not essential for diagnosis[16] Clinical diagnosis and staging are confirmed by contrasted CT of the head or MRI.

TREATMENT

Institute antibiotics in the ED using imipenem in children; in adults, use an aminoglycoside and antipseudomonal penicillin or a cephalosporin or quinolone.[13] If fungal disease has grown from a prior culture, start voriconazole, 6 milligrams/kg IV every 12 hours. Selected cases of early infection may be managed as outpatients with oral quinolones. Mild cases are likely to completely resolve with a single course of antibiotic therapy, whereas more advanced stages may require IV antibiotics and possibly surgical debridement.

OTITIS MEDIA

Otitis media (OM) is primarily a disease of infancy and childhood (see chapter 118 for management of OM in children). While the management of OM in children and adults is similar, there are important differences in adults, highlighted below, especially for the management of OM with effusion.

PATHOPHYSIOLOGY AND MICROBIOLOGY

Viral upper respiratory tract infections precede or coincide with 70% of acute OM cases.[17] The most common associated viral pathogens are respiratory syncytial virus, adenovirus, and cytomegalovirus.[18] The most common bacterial pathogens recovered in acute OM are *Streptococcus pneumoniae* (43% to 49%), nontypable *Haemophilus influenzae* (29% to 70%), and *Moraxella catarrhalis* (15% to 28%).[17,19] The *H. influenzae* type b vaccine has no effect on nontypable *H. influenzae*. Most adults have never received this vaccine and thus remain unprotected from all *Haemophilus* flu strains. The predominant organisms involved in chronic OM are *S. aureus* (35%), *P. aeruginosa* (22%), *Aspergillus* (13%), and less commonly, anaerobic bacteria.[20] OM with effusion is differentiated from acute OM. In adults, OM with effusion is frequently associated with significant pathology: acute or chronic sinusitis in 66%, smoking-induced nasopharyngeal lymphoid hyperplasia and adult-onset adenoidal hypertrophy in 19% of cases, and head and neck tumors (mainly nasopharyngeal carcinomas) in 4.8%.[21] Adult patient with OM with effusion may also have symptoms of gastroesophageal reflux.[22]

CLINICAL FEATURES AND DIAGNOSIS

The typical ED presentation is a prodrome of an upper respiratory tract infection followed by sudden increase in otalgia, with or without fever. Otorrhea and hearing loss are variably present, while tinnitus, vertigo, and nystagmus are uncommon but possible findings. Diagnosis is clinical. The TM may be retracted or bulging. It may be red in color, indicating inflammation, or it may be yellow or white, as a result of middle-ear fluid. Pneumatic otoscopy almost uniformly demonstrates impaired mobility. Pain and new otorrhea (in the absence of external otitis) helps to confirm the diagnosis. **Always assess facial nerve function because of the nerve's proximity to the middle ear.** Guidelines are available for the diagnosis and management of OM in children,[23] but none have been published for adults. In adults, OM with effusion presents with ear discomfort or fullness or may present with decreased hearing without discomfort. OM with effusion is diagnosed based on physical exam findings of middle-ear effusion with little inflammatory changes, including pneumatic otoscopy showing an immobile TM. In adults, there will typically be other physical exam findings such as sinusitis or enlarged adenoids behind the uvula. Coexistent symptoms of reflux should be elicited.

TREATMENT

There are no treatment guidelines specifically for adults. The "wait-and-see" method recommended in children[24] has not been evaluated in adults. The preferred adult initial treatment is amoxicillin.[11] The dose in adults (weighing >40 kg) is 875 or 1000 milligrams every 12 hours, or 500 milligrams every 8 hours, for 7 to 10 days. Alternative agents include amoxicillin-clavulanate, cefdinir, or cefpodoxime. For OM unresponsive to initial therapy after 72 hours, consider changing to amoxicillin-clavulanate, levofloxacin, or moxifloxacin.

Provide pain control with acetaminophen or ibuprofen or with narcotics for severe pain. Topical agents such as antipyrine/benzocaine otic may also be given.[25] OM with effusion requires treatment with the same antimicrobials, but for 3 weeks, and prednisone may be added at follow-up. Patients with OM with effusion and coexisting symptoms of reflux should be treated with appropriate antireflux medications (see chapter 74, "Esophageal Emergencies").

Adults with OM should receive follow-up to assess treatment efficacy and to ensure that there is no anatomic obstruction to the eustachian tube, as, for example, from occult neoplasm. Any patient who presents with complications of OM or who appears septic should have urgent consultation for diagnostic and therapeutic tympanocentesis and admission for IV antibiotics.

COMPLICATIONS OF OTITIS MEDIA

Complications of OM are intratemporal and intracranial. Perforation of the TM is a common intratemporal complication and most often occurs in the pars tensa from the increased pressure of middle-ear secretions, with resultant otorrhea. Healing usually occurs in 1 week, although a chronic perforation may result. A temporary conductive hearing loss may occur from fluid in the middle ear. Hearing loss should resolve as the fluid is resorbed. Acute serous labyrinthitis may occur when bacterial toxins enter the inner ear through the round window. **Facial nerve paralysis** is an uncommon complication but requires emergent otolaryngology consultation.

Acute Mastoiditis and Cholesteatoma **Acute mastoiditis** results from spread of infection from the middle ear to the mastoid air cells by the aditus ad antrum. When this opening becomes blocked, the mastoid cavity becomes a closed space, and the mastoid air cells become inflamed and fill with fluid. The most common pathogens are *S. pneumoniae* (38%), *Streptococcus pyogenes* (11%), and *P. aeruginosa* (11%).[26] In addition to otalgia, fever, and otorrhea (especially in patient with *Pseudomonas*), patients with mastoiditis will have postauricular erythema, swelling, and tenderness, with protrusion of the auricle and obliteration of the postauricular crease. Diagnosis is suspected based

on the history and physical examination and confirmed on IV contrast CT scan. Mastoiditis requires admission for IV antibiotics, tympanocentesis, and myringotomy. For first episode, treat with ceftriaxone, 1 gram IV every 24 hours, or levofloxacin, 750 milligrams every 24 hours.[13] For recurrent episodes, treat with vancomycin, 1000 milligrams IV, and piperacillin-tazobactam, 3.375 gram initial dose IV, or imipenem. Incision and drainage of subperiosteal abscess or mastoidectomy may ultimately be required.

Aural **cholesteatomas** are collections of epidermis and exfoliated keratin within the middle ear or mastoid. As the cholesteatoma expands, it may erode the ossicular chain, bony labyrinth, or facial nerve canal. Cholesteatomas are often infected, and their intracranial extensions may be life threatening. Treatment requires otolaryngolic evaluation.

Intracranial Complications Intracranial complications of OM are more likely with chronic than with acute OM and are, in general, decreasing with the widespread use of antibiotics in the treatment of OM. However, suppurative intracranial extension is a severe complication, and suggestive signs and symptoms should be investigated appropriately. Meningitis and brain abscess are the most common intracranial complication of OM with an incidence of 0.42 per 100,000 per year.[27] The most prevalent causative organisms are *S. pneumoniae* (33%) and *Neisseria meningitidis* (23%).[27] Extradural abscess and subdural empyema are also potential complications.

Lateral Sinus Thrombosis Lateral sinus thrombosis is another ominous complication of acute OM. It arises from extension of infection and inflammation in the mastoid, with eventual inflammation of the adjacent lateral or sigmoid sinus. Reactive thrombophlebitis with mural clot formation, intraluminal empyema, or perforation of the venous wall may occur.

Headache is the most common symptom, with papilledema, sixth-nerve palsy, and vertigo being less frequently present. Angiography with venous phase and MRI are more sensitive than CT in diagnosing lateral sinus thrombosis. The employed antibiotic regimen should cover *Staphylococcus*, *Streptococcus*, and upper respiratory anaerobes, and have good penetration of the blood–brain barrier. A combination of IV penicillin or nafcillin, ceftriaxone, and metronidazole is one initial empiric regimen.[28]

BULLOUS MYRINGITIS

Bullous myringitis is a painful condition of the ear characterized by bulla formation on the TM and deep external auditory canal. The blisters are believed to occur between the highly innervated outer epithelium and the inner fibrous layer of the TM, explaining the severe otalgia. The blisters may be blood filled, serous, or serosanguineous. Reactive middle-ear effusions may accumulate. Otorrhea as a result of ruptured bullae is short lived. A reversible hearing loss is commonly associated with the condition and may be conductive, sensorineural, or mixed. This disorder is not caused by *Mycoplasma pneumoniae,* as commonly believed, but is a severe manifestation of the typical organisms that cause OM.[29] Treatment is as above for acute OM.

EAR HEMATOMA

An auricular hematoma can develop from almost any type of trauma to the ear. As a result of the lack of subcutaneous fat on the anterior surface of the auricle, blunt force applied to this area tends to shear the perichondrium from the underlying cartilage and tear the adjoining blood vessels. The cartilage depends on the perichondrial blood vessels for viability. Any interruption of the nourishing blood supply can result in necrosis. In addition, a subperichondrial collection can lead to stimulation of the overlying perichondrium, which can result in an asymmetric formation of new cartilage growth and deformity of the appearance of the external ear anatomy. The resultant deformed auricle has been referred to as "*cauliflower ear*," which is commonly seen in boxers or wrestlers secondary to repeated head/ear trauma. The auricular hematoma itself is a painful swelling that obscures the normal contour of the ear. The hematoma may accumulate immediately or several hours following an injury. Aspiration alone does not completely evacuate the clot and therefore leads to deformity and increased morbidity. The goal of treatment is to remove the fluid collection and maintain pressure in the area for several days to prevent reaccumulation of fluid.

ASPIRATION OF AND DRESSING FOR HEMATOMA OF THE EAR

Using sterile technique after local anesthesia, make a semicircular incision through the skin, and be careful not to incise the underlying perichondrium. The incision should be the minimal necessary to drain the underlying hematoma and positioned in an area with the least chance of cosmetic deformity. This is usually accomplished by incising the skin inside the inner curvature of the helix or anthelix. The hematoma can then be removed by gentle suction or curettage. Suture the incision after hematoma removal.

There are a few ways to prevent the hematoma from recurring. Place a dental roll or a firm sterile pledget coated with antibiotic ointment over the resutured site with through-and-through sutures connected to a similar bolster on the opposite side of the ear. Apply a light nonpressure dressing and reevaluate the ear within 24 hours to assure there has been no reaccumulation of the hematoma. A pressure dressing can also be employed if you do not want to suture a dental roll though the cartilage. Simply pack the helix with petroleum jelly–impregnated gauze and then place regular gauze both in front of and behind the ear. Lastly circle the head with a compressive wrap (**Figure 242-4**). Prophylactic antibiotics can be reserved for immunocompromised patients and should cover *P. aeruginosa* and *S. aureus*, the two likely participants in posttraumatic chondritis.

EAR FOREIGN BODIES

Cerumen loops/scoops, a right-angle hook, and alligator forceps are the instruments of choice for foreign body removal (**Figure 242-5**). Live objects should be drowned with a 2% lidocaine solution or viscous lidocaine, which immediately paralyzes the offending insect and provides modest topical anesthesia. The liquid can then be suctioned out with butterfly tubing and the insect removed with gentle suction or forceps under direct visualization. Remove all insect debris from the canal.

Irrigation with room-temperature water is adequate for small particles such as hard sand or cerumen and can mobilize distally positioned objects. Irrigation should not be used unless the TM is completely visualized and free of perforation. Organic matter that can expand when moistened is also a relative contraindication to irrigation.

Inspect the ear canal after removal of the foreign body to exclude injury to the canal skin, TM, and ossicles caused by the foreign body or its extraction. Small abrasions heal spontaneously. Topical antibiotics should be considered in cases where there was more serious cutaneous damage or where the foreign body consisted of organic material or generated a local inflammatory reaction (Table 242-5).

CERUMEN IMPACTION

Symptoms of cerumen impaction are decreased hearing, a sensation of pressure or fullness in the ear, dizziness, tinnitus, or otalgia. These symptoms are often precipitated by the use of cotton-tipped applicators. Most of the time, cerumen loops/scoops can be used to remove impacted cerumen (Figure 242-5). In particularly difficult cases or when the canal is completely occluded, softening of the material can be accomplished using half-strength hydrogen peroxide, sodium bicarbonate, mineral oil, or an over-the-counter preparation such as Debrox® (carbamide peroxide otic). Left in place for 30 minutes, such preparations soften the cerumen and facilitate its removal. If irrigation is the treatment of choice, it can usually be accomplished with an ear syringe, a flexible 18-gauge IV catheter, or a syringe attached to the tubing of a butterfly infusion catheter. Pretreatment with triethanolamine polypeptide oleate (Cereuminex®) improves success rates.[30] Use body-temperature irrigant to minimize the development of vertigo. Insert the catheter into the cartilaginous canal (external third) and gently irrigate along the superior portion of the external auditory canal. Using this technique, the pressure of the stream is directed toward the wall of the canal and not the TM.

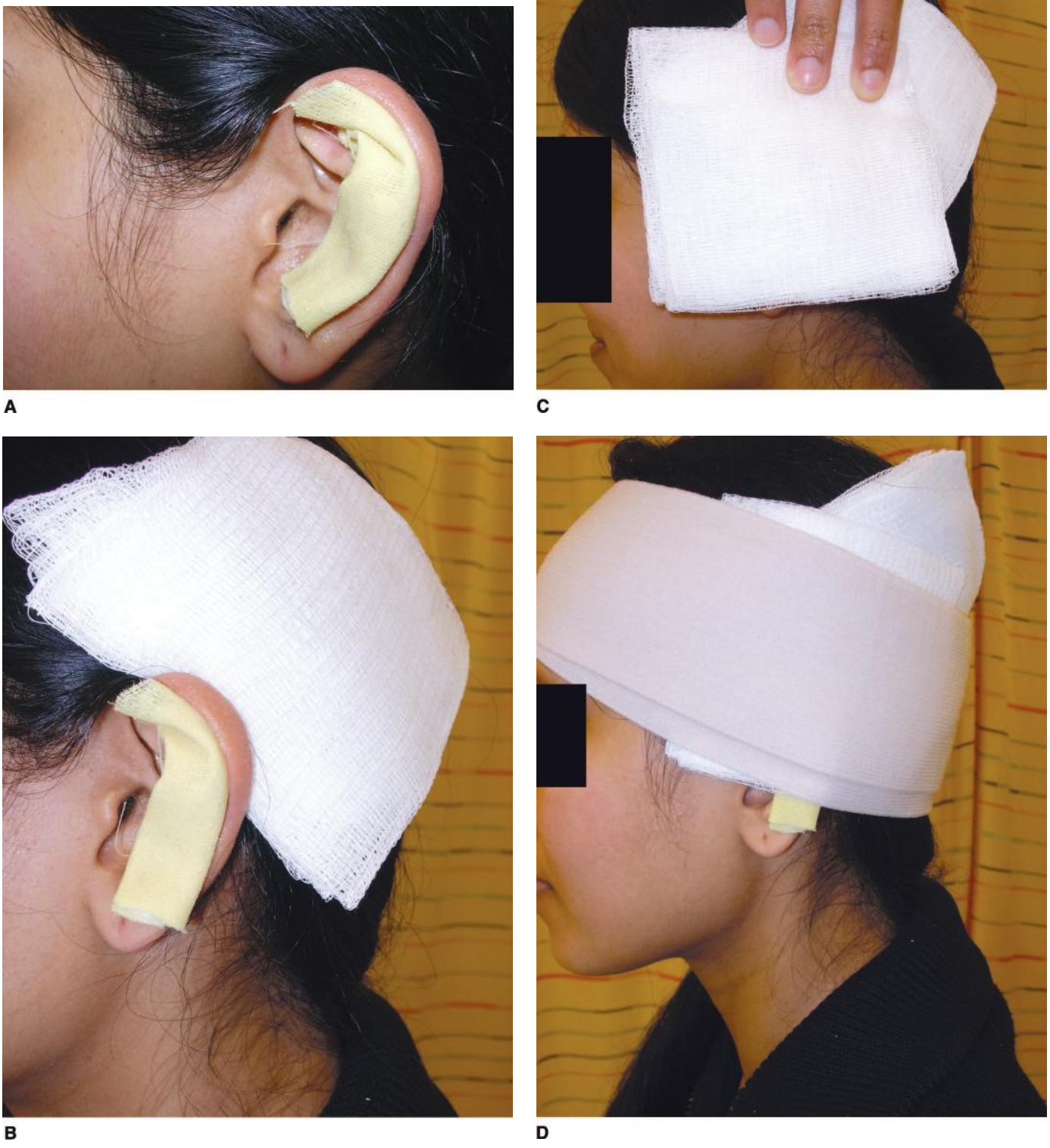

FIGURE 242-4. Stepwise progression to build an auricular bandage to help compress a drained hematoma in the auricle of the ear. Use a petroleum jelly–impregnated gauze inside the helix of the ear (**A**) and then add dry gauze to both the back (**B**) and front (**C**) of the ear. **D.** Wrap it lightly to add slight compression to hold the perichondrium to the auricular cartilage.

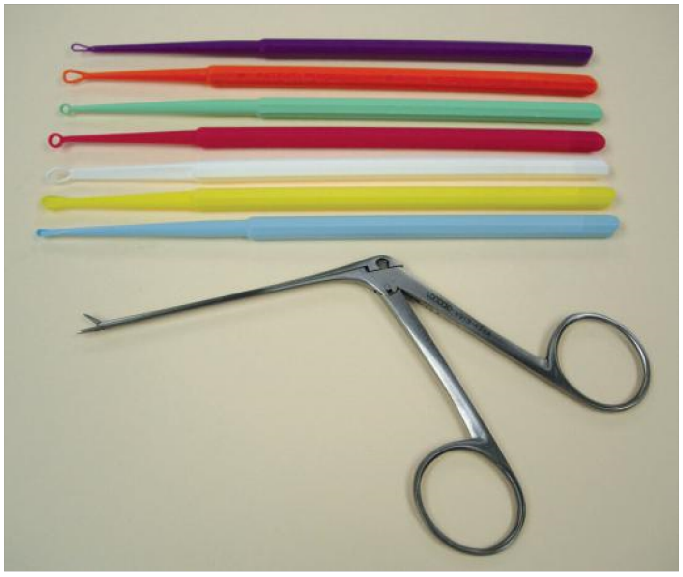

FIGURE 242-5. Different-shaped ear scoops and loops are useful to remove cerumen from an impacted ear. Miniature alligator forceps can be used to extricate foreign bodies from the external ear canal.

Irrigation of the canal when the middle ear is not infected often causes a temporary redness of the TM.

The most common iatrogenic injury associated with syringing of the ear is traumatic TM perforation. Predisposing factors for perforation include previous ear surgery, a previous or current history of OM, and severe otitis externa. When in doubt, it is safer to defer irrigation to an otolaryngologist. When determining if a perforation has occurred, it is important to rely on symptoms (sudden hearing loss, severe otalgia, or vertigo) rather than signs, because visualization of the TM may be impaired by the irrigating fluid and debris. In case of suspected perforation, reassurance, analgesia, and otolaryngology referral in 1 to 2 weeks are indicated. Prophylactic antibiotics are not necessary. If injury to ossicles is suspected, emergency ear, nose, and throat consultation is warranted.

TYMPANIC MEMBRANE PERFORATION

TM perforations can occur secondary to middle-ear infections or as a result of barotrauma, blunt/penetrating/acoustic trauma, or, on rare occasions, lightning strikes. Perforation is also discussed in the chapter 7, "Bomb, Blast, and Crush Injuries." When perforation is secondary to blunt or noise trauma, the perforation almost always occurs in the pars tensa, usually anteriorly or inferiorly. The pars tensa, the largest area of the TM, is only a few cell layers thick and thus is easily torn.

Symptoms are acute onset of pain and hearing loss, with or without bloody otorrhea. There may also be associated vertigo or tinnitus, but this is usually transient unless there has been injury to the inner ear or rupture of the round or oval windows. The TM should be completely visualized and the canal must be cleared of blood and debris.

Most TM perforations heal spontaneously. Patients with perforations secondary to blunt or noise trauma that are isolated injuries can be safely discharged and referred to a specialist for further evaluation and a formal audiogram as soon after the injury as possible. Patients should be instructed not to allow water to enter the canal of the ear. Topical or systemic antibiotics are not needed unless foreign material is suspected of remaining in the canal or in the middle ear. Perforations in the posterosuperior quadrant or those secondary to penetrating trauma have a greater likelihood of ossicular chain damage and should be referred to an otolaryngologist within 24 hours.

REFERENCES

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

CHAPTER 243

Face and Jaw Emergencies

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FACIAL CELLULITIS, ERYSIPELAS, AND IMPETIGO

Cellulitis and erysipelas are discussed in detail in the chapter 147, "Soft Tissue Infections." Impetigo is discussed in the chapter 136, "Rashes in Infants and Children." The differential diagnosis of facial infections is provided in **Table 243-1**.[1]

Cellulitis is a superficial soft tissue infection that lacks anatomic constraints.[2-4] Facial cellulitis is caused most commonly by *Streptococcus pyogenes* (group A β-hemolytic) and *Staphylococcus aureus*,[4] with an increasing predominance of methicillin-resistant *S. aureus*.[5] Less commonly, cellulitis may represent extension from deep space infections (see "Masticator Space Infection" section below). In children, buccal cellulitis from *Haemophilus influenzae* is now very uncommon if children have received the *H. influenzae* type b vaccine.[6]

Bedside US can exclude or identify facial abscess (**Figure 243-1**). CT can identify deep-seated, extensive infections that involve the soft tissues of the neck or pharynx.

Treatment is provided in **Table 243-2**. Duration of therapy is not well studied, but recommendations range from 7 to 14 days.[3,5,7,8] Treatment failures range from 15% to 20% for β-lactams (cephalexin, dicloxacillin, and amoxicillin-clavulanate) as well as for the anti–methicillin-resistant *S. aureus* therapies[9] due to the failure to cover methicillin-resistant *S. aureus* and streptococcal species, respectively. Cephalexin appears to be the most cost-effective therapy based on a probability of 37% for infection with *S. aureus* and a 27% methicillin-resistant *S. aureus* prevalence.[10] In selected cases, traditional β-lactam therapy may be added to anti-methicillin-resistant *S. aureus* therapy, but this strategy increases cost and potential for adverse effects of the medication.

Erysipelas is most common in the lower extremities (66% to 76%)[11-13] but is classically described as a disease of the face (see Figure 152-3). The nasopharynx is typically the source of bacteria.[14] In the majority of cases, erysipelas is caused by *S. pyogenes*.[3,4] Bullous erysipelas is a more severe form of the disease, and half of the infections reported in a 2004 case series were due to methicillin-resistant *S. aureus*.[15] Penicillin is the antibiotic treatment of choice[3,13] but is chosen as empiric therapy in a minority of cases.[11,12,16,17] If the suspicion exists of a staphylococcal infection (i.e., bullae, trauma, or the presence of a foreign body), alternatives include dicloxacillin, amoxicillin-clavulanate, or a cephalosporin[3] (Table 243-2). One randomized, prospective trial compared the use of a macrolide, roxithromycin, with penicillin and found no difference in efficacy.[18]

Impetigo is a discrete, superficial bacterial epidermal infection, characterized by amber crusts (nonbullous) or by fluid-filled vesicles (bullous) (see Figures 141-14 and 141-15). It is most common in children. *S. aureus* alone or in combination with *S. pyogenes* (group A β-hemolytic) is the most common cause of nonbullous impetigo. Bullous impetigo is always caused by *S. aureus*.[3,19,20] Treatment should cover streptococcal and staphylococcal species. Topical therapy is sufficient for uncomplicated patients with only a few nonbullous lesions.[21] Mupirocin, retapamulin, or fusidic acid ointment is recommended; however, some resistance is developing.[3,20,22,23] Consider oral antibiotics for extensive lesions or lesions that do not respond to topical therapy alone. Erythromycin and cloxacillin are superior to penicillin, but there is no clear preference between macrolides, β-lactamase–resistant penicillins, and cephalosporins.[3,19] Preferred choices are penicillinase-resistant penicillins (cloxacillin, dicloxacillin, amoxicillin-clavulanic acid) or first-generation cephalosporins (cephalexin).[3] For methicillin-resistant *S. aureus*, treat with trimethoprim-sulfamethoxazole or clindamycin[3] (**Table 243-3**).

SALIVARY GLAND INFECTIONS

There are three groups of salivary glands: the parotid, submandibular, and sublingual. The facial nerve passes through the superficial portion of the parotid gland, and the parotid (Stensen's) duct opens into the