Ears are exquisitely sensitive organs. Treating pain is important in caring for patients with ear problems and will facilitate performance of the examination. Simple otitis externa can be treated with topical medications and débridement. Many cases of uncomplicated otitis media resolve spontaneously. Watchful waiting with use of a “rescue” antibiotic prescription has been shown to decrease unnecessary antibiotic use and improve patient and parent satisfaction. Subtle malalignments or malformations after repair of ear trauma can have profound cosmetic consequences. Pain from the teeth, pharynx, or temporomandibular joint or from cranial or cervical neuropathies can be referred to the ear. Hearing loss must be categorized as conductive or sensorineural. Conductive lesions can often be diagnosed clinically. Sensorineural hearing loss requires urgent referral to an otolaryngologist to improve the chance for recovery of hearing.

**Pathophysiology**

Anatomically, the ear is divided into three areas. The external ear extends outward from the tympanic membrane and serves to guide sound waves into the “business end” of the ear. Its external location places it at high risk for traumatic injuries, environmental exposure, obstruction, and infection. The middle ear extends inward from the tympanic membrane to the oval and round windows. It transfers mechanical energy from the outside world, through the ossicles in the middle ear, to the inner ear, where it is translated into signals that the brain interprets as sound. The enclosed nature of the middle ear predisposes it to accumulation of fluid, infection, and barotrauma. The inner ear is composed of the structures responsible for sound transduction and balance (organ of Corti, cochlea, vestibule [saccule and utricle], and semicircular canals). Dysfunction of this portion of the ear accounts for some visits to the emergency department (ED), but treatment options are limited and consist mostly of patient education, prognostication, limitation of further damage, and appropriate ear, nose, and throat (ENT) referral. The ear is surrounded by the middle cranial fossa superiorly, the mastoid air cells posteriorly, the cranial vault medially, and the temporomandibular joint and parotid glands anteriorly. Evaluation of these structures is a necessary part of the ear examination.

**Ear Pain**

Ear pain may be referred from or occur as a result of infections, trauma, or a foreign body affecting the ear.

**Infections**

Ear pain is commonly caused by infection. Any portion of the ear can become infected. The disease state is categorized by the portion of the ear that is primarily affected.

**Otitis Externa**

Infections of the outer ear canal most often begin when breakdown of the natural barriers allow infectious organisms to gain a foothold. This commonly occurs in the summer months, when warm weather and frequent water sports lead to excessive ear moisture, which washes out the cerumen and alkalizes the normally acidic environment—hence the term swimmer’s ear. Cotton swab trauma can be the inciting event, especially in diabetic patients.

The most common pathogens are *Pseudomonas aeruginosa* (frequently found in pools) and *Staphylococcus aureus*. Less common causes include chemical or contact dermatitis and fungal infections. The spectrum of disease ranges from mild (with minimal pain and inflammation of the canal) to severe (complete canal occlusion and exquisite pain). Further extension results in an invasive or systemic disease state called necrotizing external otitis (formerly malignant otitis externa; see later).

The diagnosis of external otitis is made from the history and findings of pain, pruritus, canal irritation, and edema on physical examination. Thick greenish discharge suggests *Pseudomonas*, whereas golden crusting implicates *S. aureus*. Other colored or black discharge may indicate fungal infections, of which *Candida* and *Aspergillus* are the most commonly isolated species. Small abscesses in the external ear canal can cause obstruction. These abscesses often require incision and drainage, as well as standard treatment of otitis externa.
Treatment

Treatment consists of débridement or aural toilet and antibiotics. Despite a relative lack of controlled studies, the American Academy of Otolaryngology–Head and Neck Surgery Foundation has released clinical practice guidelines based on evidence available as of 2005 (Fig. 27.1; also see the Patient Teaching Tips box).2 Briefly, these guidelines are as follows:

1. Distinguish acute external otitis from other causes of otalgia, otorrhea, and inflammation. Diagnostic criteria include rapid onset (2 to 3 days) and a duration of less than 3 weeks. Symptoms include otalgia, itching, or fullness. Signs include tenderness of the pinna or tragus or visual evidence of canal erythema, edema, or otorrhea.

2. Assess for factors that may complicate the disease or treatment (e.g., perforation of the tympanic membrane or eustachian tubes, immunocompromising states, previous radiotherapy). These factors raise the level of treatment needed and heighten suspicion for more invasive disease states such as necrotizing otitis externa (see later). These guidelines pertain to patients older than 2 years with normal states of health.

3. Pay attention to assessment and treatment of pain! Mild to moderate pain usually responds to acetaminophen or a nonsteroidal antiinflammatory drug alone or in combination with an opioid.

4. Topical preparations are first-line agents for the treatment of acute uncomplicated otitis externa. Reserve systemic therapy for immunocompromised patients or extension of disease beyond the ear canal. Topical therapy produces drug concentrations 100 to 1000 times that available with systemic administration and can thus overwhelm resistance mechanisms. No clear evidence points to the superiority of one particular treatment. Antiseptic and acidifying agents (e.g., aluminum acetate and boric acid) appear to work as well as antibiotic-containing solutions (e.g., solutions that contain cortisone and Neosporin or a fluoroquinolone). Corticosteroids in the drops decrease the duration of pain by approximately 1 day.3

5. Make sure that the patient can instill the drops correctly. Edema can prevent the drops from entering the canal. Debris or detritus should be removed or irrigated out. Placement of a compressed cellulose or ribbon gauze wick in the canal will enable the drops to penetrate, but placement can be painful. Within 1 to 2 days the canal edema should subside, and the wick falls out or can be removed (Fig. 27.2).

6. If you cannot be sure that the tympanic membrane is intact, use a nonototoxic, pH-balanced preparation such as ofloxacin and ciprofloxacin-dexamethasone.

7. Educate and reassess your patients. Pain should decrease significantly in 1 to 2 days and resolve by 4 to 7 days. Failure to improve may indicate more invasive disease (e.g., necrotizing otitis), inability of drops to reach the canal (wick needed), or noncompliance with therapy.

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**Fig. 27.1 Algorithm for the treatment of acute otitis externa. TM, Tympanic membrane. (Adapted from Rosenfeld RM, Brown L, Cannon CR, et al. Clinical practice guideline: acute otitis externa. Otolaryngol Head Neck Surg 2006;134;S4-23.)**

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**PATIENT TEACHING TIPS**

**Otitis Externa**

Most patients with otitis externa can be managed as outpatients. Follow-up in 1 to 2 days is indicated if a wick is placed for treatment, if oral antibiotic therapy is started, or if the pain does not begin to resolve in 24 to 36 hours. Patients should discontinue use of the drops after full resolution of their symptoms. Continued use of antibiotics (especially neomycin) can predispose to changes in the environment of the ear canal and foster fungal infections or sensitivity reactions.

Patients must avoid getting water in the ear during the healing process. Cotton balls soaked in petroleum jelly work well as earplugs. Any water that gets into the ear can be removed by gentle blow-drying.

Patients with evidence of significant immunocompromise or failure to improve in 1 to 2 days should be considered for admission to the hospital to be evaluated for more extensive disease.

Drying the ear after swimming or showering helps prevent otitis externa. Placing drops of acetic acid (vinegar) and rubbing alcohol in the ear two or three times a week during periods of heavy water exposure (summer vacation) helps dry the ear and restore the acidic environment that protects against otitis externa.
Acute otitis media is primarily a disease of children. In early childhood the eustachian tubes are not angled downward and do not drain well spontaneously. The relatively small tube size and higher frequency of URIs in children 6 to 24 months old lead to the highest incidence of otitis media in this age group. Another increase in the incidence of otitis media occurs at 5 to 6 years, which coincides with entrance into school and a higher frequency of URIs. The craniofacial abnormalities seen with some developmental disorders (e.g., Down syndrome) also predispose to the development of middle ear effusions.

Otitis media is one of the most common reasons for pediatric physician visits, with estimates that $5 billion is spent as direct or indirect costs annually. A significant proportion of cases are probably misdiagnosed, and guidelines have been issued to ensure proper diagnosis and thus curb wasting of resources.

Visualization of the tympanic membrane with identification of a middle ear effusion and inflammatory changes is necessary to establish the diagnosis with certainty. Effusions are signified on physical examination by bulging of the tympanic membrane, bubbles or fluid levels behind the membrane, loss of the light reflex (opacification or cloudiness of the membrane), and (most definitively) loss of tympanic membrane mobility on pneumatic insufflation.

Newer modalities, such as acoustic reflectometry and tympanometry, also demonstrate middle ear effusions but are not available in many EDs. Tympanic membrane injection (common in crying children) or the presence of fluid alone is not enough to make the diagnosis of AOM. Accompanying fever, pain, purulent drainage, or other systemic signs point to acute infection.

Chronic otitis media is defined as (1) the chronic presence of middle ear effusion in the absence of acute signs of infection or (2) chronic complications from otitis media, including persistent perforation of the tympanic membrane.

The role of infectious organisms in chronic otitis media is unclear. It was originally thought to be a noninfectious entity, but studies have shown the presence of bacteria (and bacterial DNA, mRNA, and proteins) in a biofilm model of chronic otitis media.
Treatment
American physicians have historically treated otitis media with antibiotics, whereas European physicians are typically less likely to do so. A 2005 study compared immediate antibiotic treatment with watchful waiting for nonsevere otitis media.\textsuperscript{10} In the watchful waiting group, 66% of children had complete resolution of symptoms with no antibiotic treatment, no adverse outcomes, cost savings, and similar patient satisfaction.

Treatment options and recommendations by the American Academy of Pediatrics for acute otitis media include the following:\textsuperscript{4}:

1. Pain management must be addressed in patients with AOM. Particular attention should be paid to pain management in the first 24 hours of any treatment regimen.
2. Observation without the use of antibacterial agents is an option for the first 2 to 3 days in selected children\textsuperscript{11} (Table 27.1). The child must be otherwise healthy and in a sound social environment with an adult capable of watching the child closely and returning to the physician if the condition deteriorates.
3. If antimicrobial treatment is chosen, the first-line agent should be amoxicillin, 80 to 90 mg/kg/day. With treatment failures or cases in which broader \( \beta \)-lactamase coverage is desired, amoxicillin, 90 mg/kg, with clavulanate, 6.4 mg/kg, in two divided doses can be used. Penicillin-allergic patients (non–type 1) can be treated with a third-generation cephalosporin (cefdinir, cefpodoxime, cefuroxime, or ceftriaxone). For patients with severe type 1 penicillin allergy, alternative treatments include azithromycin, clarithromycin, erythromycin-sulfisoxazole, and sulfamethoxazole-triamethoprim. Treatment is aimed at common pathogens, including \textit{S. pneumoniae}, nontypeable \textit{H. influenzae}, and \textit{M. catarrhalis}. \textit{Mycoplasma} species can also cause otitis media and are often responsible for blister formation on the tympanic membrane (bullous myringitis). Multiple virus species can cause otitis media and are obviously unaffected by antibiotics.
4. Failure of response in 2 to 3 days should prompt initiation of or a change in antibiotic treatment. If amoxicillin fails, alternatives include amoxicillin-clavulanate, cephalosporin (ceftriaxone), macrolides, and sulfa preparations.

Some authorities have suggested a compromise between meeting patients’ expectations and decreasing the inappropriate overuse of antibiotics.\textsuperscript{12,13} Patients can be given a “rescue” prescription, which they should have filled only if no improvement occurs in 2 to 3 days.

Chronic otitis media is not typically an emergency. The American Academy of Family Physicians, Pediatrics, and Otolaryngology–Head and Neck Surgeons have recently issued guidelines to direct the diagnosis and treatment of otitis media with effusion; they are summarized as follows:

1. Pneumatic otoscopy should be used to identify the presence of effusion.
2. The history and physical examination (to search for acute signs and symptoms of inflammation or infection) should be used to distinguish this disorder from AOM.
3. Otitis media with effusion should be managed with watchful waiting for 3 months in children who are not at risk for speech, language, or other learning disabilities.
4. Hearing tests (referral to an otolaryngologist) should be performed if the disease lasts longer than 3 months or earlier if any language, learning, or hearing problems are suspected.
5. Antihistamines and decongestants are ineffective and should not be used as treatment; antimicrobial agents and steroids do not have long-term efficacy and should not be used for routine management.

Middle ear effusions in adults should be able to be explained clinically (e.g., after URI) and should resolve within a few weeks. Any other circumstances require otolaryngologic evaluation.

### Table 27.1 Acute Otitis Media Treatment Guidelines

<table>
<thead>
<tr>
<th>AGE</th>
<th>CERTAIN DIAGNOSIS OF ACUTE OTITIS MEDIA</th>
<th>UNCERTAIN DIAGNOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;6 mo</td>
<td>Antibacterial therapy</td>
<td>Antibacterial therapy</td>
</tr>
<tr>
<td>6 mo to 2 yr</td>
<td>Antibacterial therapy</td>
<td>Severe illness: antibacterial therapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nonsevere illness: observation option*</td>
</tr>
<tr>
<td>≥2 yr</td>
<td>Severe illness: antibacterial therapy</td>
<td>Observation option*</td>
</tr>
<tr>
<td></td>
<td>Nonsevere illness: observation option*</td>
<td></td>
</tr>
</tbody>
</table>


*Observation: defer antibiotic treatment for 48 to 72 hours.
referral to evaluate for other nasopharyngeal disease, such as an obstructing tumor.

**Necrotizing (Malignant) Otitis Externa**

Necrotizing otitis externa (formerly known as malignant otitis externa) is aggressive extension of infection from the auditory canal to the skull base and other nearby bony structures. This complication occurs nearly exclusively in immunocompromised hosts, with elderly diabetic patients accounting for most of the affected population. It can be the initial complaint in patients with undiagnosed diabetes, and all patients with progressive ear infection need prompt evaluation for diabetes. The emergence of widespread human immunodeficiency virus infection now puts children at risk for a condition that was once almost exclusively an adult disease.  

Necrotizing otitis externa may be difficult to distinguish from simple otitis externa in the early stages, but exquisite otalgia and otorhea unresponsive to topical measures point to the former diagnosis. The pain often extends to the temporomandibular joint and gets worse with chewing. Granulation tissue is frequently seen at the inferior portion of the canal where the cartilage and bone meet, at the site of the fissures of Santorini. Inflammation of bony structures as a result of the osteomyelitis can cause nerve palsies (facial nerve most frequently involved). Progression of the infection inward can lead to catastrophic complications such as brain or epidural abscess, sinus thrombophlebitis, and meningitis. Evaluation with computed tomography (CT) or magnetic resonance imaging (MRI) can show the extent of the invasive process and may be helpful in evaluating for intracranial complications, but arranging for such an evaluation should not delay initiation of treatment.

More than 95% of cases of necrotizing otitis externa are caused by *P. aeruginosa*, and antibiotic therapy should be aimed at this organism. Since the introduction of semisynthetic penicillins, antipseudomonal cephalosporins, and antipseudomonal fluoroquinolones, mortality from this disorder has decreased from 50% to 10%. Empiric treatment with ciprofloxacin, 400 mg intravenously every 8 hours, is reasonable. Alternative treatments are an antipseudomonal penicillin (e.g., ticarcillin-clavulanate [Timentin], 3.1 g intravenously every 6 hours) and cephalosporins (e.g., ceftazidime, 1 to 2 g every 8 hours). Recently, resistance of *P. aeruginosa* to ciprofloxacin has been reported to be as high as 33%. Resistance is related to widespread use of quinolones for the treatment of URIs, topical preparations for otitis media and externa, and inadequate treatment courses in patients with malignant otitis externa.  

**Ramsay Hunt Syndrome**

The combination of ear pain, ipsilateral facial paralysis, and vesicular lesions characterize Ramsay Hunt syndrome, also known as herpes zoster oticus. This reactivation of latent varicella-zoster infection in the geniculate ganglion with spread to the eighth cranial nerve (and frequently cranial nerves V, IX, and X) results in both auditory and vestibular dysfunction.

Physical examination usually demonstrates vesicular lesions in the ear canal, but the variable course and innervation of the nervous structures may lead to involvement of the anterior aspect of the tongue, soft palate, pinna, and face. Because of the proximity of the ear to the eye, evaluation for ocular involvement is necessary. The disease tends to be self-limited and mortality is extremely rare, but deficits in nerve function and facial paralysis are common, and patients with such paralysis are much less likely to recover than those with Bell palsy.

Treatment is aimed at shortening the duration of the outbreak and controlling symptoms. Acyclovir and steroids are often used, but no clear prospective studies have been undertaken. In light of the known safety and effectiveness of anti–varicella-zoster or anti–herpes simplex drugs, acyclovir (800 mg five times per day) or famciclovir (500 mg three times per day) should be strongly considered, along with added prednisone. Aggressive analgesia is frequently needed for pain control. Vestibular symptoms can be treated with meclizine or diphenhydramine. Cranial nerve VII palsies can occur and lead to an inability to close the eye, which can cause drying and abrasions. Use of a moisturizer or lubricant ophthalmic ointment (Lacrilube) or other measures to moisten and protect the eye are often needed.

**Mastoiditis**

All cases of otitis media are accompanied by some subclinical fluid collection in the mastoid air cells, often seen on CT. Further blockage of the communicating spaces by mucosal edema and inflammation generates pus under pressure in the mastoid air spaces and results in what we know as clinically significant mastoiditis. Left untreated, the chronic inflammation results in abscess formation and resorption of trabecular bone.

This process can further extend outward or inward. Outward extension leads to subperiosteal abscess formation. This development is associated with the classic findings for mastoiditis: tenderness and erythema over the mastoid process, outward bulging of the pinna, loss of the postauricular crease, and fluctuance behind the ear. Inward extension leads to potentially catastrophic complications such as erosion into the cranial vault, meningitis, and brain abscess formation.

Mastoiditis is a clinical diagnosis, and physical findings should prompt CT evaluation to delineate the extent of the process. Treatment consists of supportive care and resuscitation, administration of antibiotics, and otolaryngologic or ENT consultation for surgical drainage. Antibiotic coverage should initially be broad spectrum and include coverage against common otitis media pathogens, anaerobes, and *Pseudomonas* and *Bacteroides* species.

**TRAUMA**

External ear trauma can be classified into contusions and ecchymoses; seromas and hematomas; and lacerations, tears, and avulsions. Fluid collections and anatomic disruptions require directed attention because of the propensity for necrosis or disfigurement if managed inappropriately.

**Blunt trauma** can cause blood to collect in the fascial plane between the cartilage and the perichondrium. The cartilage is an avascular structure that derives its nutritional support from the blood supply of the perichondrium, and separation of the two starves the cartilage. Furthermore, neo-cartilage formation in the fluid collection space leads to scarring and deformation (cauliflower ear). Fluid collections can
be drained by either needle aspiration or open evacuation. For cosmetic purposes, collections that form lateral (external) to the cartilage layer can be drained through a medial approach. A gentle compression dressing can then be applied by packing the ear canal with dry cotton and packing the rest of the auricle with a conforming material (gauze or foam). A gauze roll and an elastic bandage can then be carefully wrapped around the head to compress the entire bandage in place while avoiding overly tight placement, which could cause ear necrosis. Follow-up within 24 hours is needed to check for reaccumulation of fluid, which would need to be redrained.

Lacerations and avulsions need special repair techniques because of the cosmetic importance of ears. As with all facial wound repairs, minimal débridement (to minimize tissue loss) and alignment of visually eye-catching anatomic lines are key to aesthetic repair. Through-and-through lacerations of the pinna necessitate alignment and repair of the underlying cartilage. The use of deep sutures should be minimized (usually one or two), and small absorbable sutures should generally be used. The overlying skin can then be closed to realign the pinna rim first, followed by closure of the remainder of the defect. Similar technique should be used for earlobe clefts, which commonly occur after abrupt traction on earrings causes either a partial or complete tear of the earlobe. Compression packing should be applied to prevent reaccumulation of fluid. Complex disruptions with significant tissue loss can be managed conservatively, with referral for plastic or reconstructive repair at a later date to maximize the chance for a good cosmetic outcome.

**FOREIGN BODY**

Direct visualization of any foreign body in an ear is critical to identification of the object and aids in the choice of removal method. A small amount of lidocaine or mineral oil instilled into the ear anesthetizes or immobilizes most insects in the ear canal within about a minute.

Methods of foreign body removal are as follows:

- **Irrigation:** An intravenous catheter without a needle (18 to 20 gauge) can be used with a 10- to 20-mL syringe. Irrigating the superior portion of the canal seems to provide the best results. The force generated is well below that needed to perforate a normal tympanic membrane. Materials that swell when wet (vegetables, cellulose, wood) should not be removed by this method because of the risk for further swelling.
- **Forceps:** Small forceps (alligator forceps) can be used to grasp objects. Use of an ENT scope and speculum greatly facilitates the process (Fig. 27.3).
- **Cyanoacrylate:** A small amount of cyanoacrylate (e.g., Super Glue) can be applied to the blunt end of a cotton-tipped applicator and held against the impacted object for about 60 seconds to glue the foreign body to the applicator and allow gentle removal of it. This method should not be attempted in a moving, uncooperative patient.
- **Right-angle probe:** A small probe can sometimes be worked behind the object and used to pull it forward. This works best for loose or pliable objects.
Suction: A flanged end of thin plastic tubing (or a premade suction device) can sometimes be used to grasp smooth, regularly shaped objects (beads) or pieces of insects for removal.

If removal of a foreign body from the ear canal is difficult or impossible, in most cases the patient can be treated with pain and anxiety medications and followed up in an otolaryngologic clinic in 12 to 24 hours. Exceptions to this statement are lodged button batteries (risk for caustic damage from leakage) and signs of advanced infection (redness, fever, uncontrollable pain); such cases require otolaryngologic consultation in the ED.

Cerumen, or earwax, is a naturally occurring substance that cleans, protects, and lubricates the external auditory canal. Excessive accumulation of cerumen is one of the most common reasons that patients seek medical care for ear-related reasons. When associated with symptoms, it is recommended that clinicians use ceruminolytic agents (triethanolamine, docusate sodium, saline), irrigation, or manual removal to treat a patient with impacted cerumen.18

Sudden Hearing Loss

Anatomically and physiologically, the hearing process consists of two parts. Conduction refers to the mechanical transmission of sound waves from the external environment through the outer and middle ear to the round window. The sensorineural component refers to transduction of sound waves to electrical (neural) impulses and delivery of these impulses to the brain, where they can be interpreted as sound. Hearing can be impaired by dysfunction in either or both of these pathways. The first step in evaluating hearing complaints (and the primary guide to treatment) is to ascertain the location and extent of the hearing loss. The history and physical examination provide nearly all the information needed to guide ED treatment of hearing loss. The history must include details about the timing of hearing loss, laterality, previous episodes, associated symptoms (tinnitus, vertigo, or pain), preceding events (diving, plane rides, trauma), potential placement of a foreign body, environmental noise exposure, and potential ototoxic drugs.

Tuning fork tests provide the best clues to distinguish between conductive and sensorineural hearing loss. The key component of the test is to compare how well the ear hears conduction through bone versus conduction through air. A 512-Hz fork should be used. The Weber test compares the two ears with each other (Fig. 27.4). A vibrating fork is placed midline on the top of the head or between the front top teeth (some patients find this intolerable). The patient is asked which ear hears the vibrations better. Because outside sounds (from air conduction) suppress the perception of vibratory conduction, an ear with a conductive hearing defect will “hear” the fork vibrating through bone “louder” than the other ear will. So if the fork is heard louder in one ear, either that ear has a conductive deficit or the other ear has a neural deficit (Table 27.2).

Fig. 27.4 The Weber test compares hearing in the two ears with each other. A vibrating tuning fork is held midline against the patient’s forehead (A). The patient is asked whether one ear hears the fork more loudly. Unequal perception of sound indicates a conductive deficit in the loud ear or a neural deficit in the quiet ear. The Rinne test compares air and bone conduction in each ear independently. A vibrating tuning fork is held against the mastoid process (bone conduction; B) until the vibrations can no longer be heard. The still-vibrating tip is then moved near the canal opening to see whether the patient can still hear the vibration through air conduction (C). Longer or louder hearing through air conduction is normal. Longer or louder hearing through bone conduction indicates a conductive hearing deficit.
### Table 27.2 Interpretation of the Weber and Rinne Tests

<table>
<thead>
<tr>
<th>WEBER WITHOUT LATERALIZATION</th>
<th>WEBER LATERALIZES RIGHT</th>
<th>WEBER LATERALIZES LEFT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rinne both ears: AC &gt; BC</td>
<td>Normal</td>
<td>S/N loss in the left ear</td>
</tr>
<tr>
<td>Rinne left ear: BC &gt; AC</td>
<td>–</td>
<td>Combined loss: conduction and S/N loss in the left ear</td>
</tr>
<tr>
<td>Rinne right ear: BC &gt; AC</td>
<td>–</td>
<td>Conduction loss in the right ear</td>
</tr>
</tbody>
</table>

AC, Air conduction; BC, bone conduction; S/N, sensorineural.

### Table 27.3 Lesions That Cause Hearing Loss

<table>
<thead>
<tr>
<th>DESCRIPTION OF PATHOLOGY</th>
<th>ONSET/COURSE</th>
<th>ACTIONS OR TREATMENT</th>
<th>PROGNOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Conductive Lesion</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foreign body</td>
<td>Mass in external canal blocks sound conduction</td>
<td>Acute onset associated or not with pain, drainage, or odor</td>
<td>Removal. Evaluate for infection. Evaluate for TM perforation</td>
</tr>
<tr>
<td>Otitis externa</td>
<td>Edema and detritus obstruct external canal</td>
<td>Rapid onset. Pain, edema, swelling. Drainage, odor often present</td>
<td>Aural toilet to remove debris. Topical (± oral) antibiotics. Evaluate for necrotizing otitis</td>
</tr>
<tr>
<td>Exostosis</td>
<td>Bony growths obstruct canal. Often seen with prolonged exposure to cold water (divers)</td>
<td>Slow insidious onset. No pain or drainage unless causes complete obstruction</td>
<td>Evaluate for infection. Reassure patient. Refer to ENT</td>
</tr>
<tr>
<td>Tympanosclerosis</td>
<td>TM scarring from perforations or infections. Decreased mobility impairs sound conduction</td>
<td>Slow onset following perforations, trauma or infections</td>
<td>ENT referral. Reassurance</td>
</tr>
<tr>
<td>Perforated TM</td>
<td>Disruption of TM integrity results in impaired transmission of sound to ossicle</td>
<td>Acute onset. May follow direct trauma or sudden barotrauma. May have sudden relief from pain if caused by otitis media</td>
<td>Treat infectious causes. Counsel on importance of keeping water out of ear canal. ENT referral</td>
</tr>
<tr>
<td>Sterile effusion (barotrauma)</td>
<td>Fluid in middle ear dampens conduction through ossicles</td>
<td>Often following flight, diving, or URI. Bubbles can cause intermittent pain</td>
<td>Decongestants. Evaluate for infection. Follow-up</td>
</tr>
<tr>
<td>Acute otitis media</td>
<td>Pus (or fluid) in middle ear dampens conduction through ossicles</td>
<td>Acute to subacute onset, often following URI. Often associated with pain ± fever</td>
<td>Antibiotics (unless viral cause suspected), decongestants, pain control</td>
</tr>
<tr>
<td>Cholesteatoma</td>
<td>Trapped stratified squamous epithelial mass in middle ear. Interferes with ossicle conduction</td>
<td>Slow onset. Often history of previous perforations or chronic infections</td>
<td>ENT referral</td>
</tr>
<tr>
<td>Glomus tumor</td>
<td>Vascular tumor occupies middle ear space. Interferes with ossicle conduction</td>
<td>Slow onset. May be associated with rushing pulsatile sensation</td>
<td>ENT referral</td>
</tr>
<tr>
<td>Cancer</td>
<td>Squamous cell most common. Obstructs external canal</td>
<td>Slow onset. Often noticed first by others. Painless unless occlusion causes otitis externa</td>
<td>ENT referral. Evaluate for secondary infection</td>
</tr>
<tr>
<td>Lesions That Cause Hearing Loss—cont’d</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>DESCRIPTION OF PATHOLOGY</strong></td>
<td><strong>ONSET/COURSE</strong></td>
<td><strong>ACTIONS OR TREATMENT</strong></td>
<td><strong>PROGNOSIS</strong></td>
</tr>
<tr>
<td>Perilymph fistula (inner ear barotrauma)</td>
<td>Disruption of round or oval window allows leakage of perilymph into middle ear</td>
<td>Sudden onset of hearing loss often with tinnitus and vertigo. Frequently follows straining or abrupt change in pressure. Turning in direction of fistula exacerbates symptoms.</td>
<td>Complete bed rest. Elevate head of bed and avoid increases in CSF pressure. Severe symptoms or noncompliance may require hospitalization. ENT consultation for possible oval or round window patch</td>
</tr>
<tr>
<td>Viral cochleitis</td>
<td>Cochlear inflammation. Often following URI</td>
<td>Rapid onset. Often following URI</td>
<td>Steroids often used (no good data)</td>
</tr>
<tr>
<td>Presbycusis</td>
<td>Age-related hearing loss. May be related to previous chronic noise exposure</td>
<td>Slow onset. Usually symmetric. High frequencies most affected. Tinnitus may occur.</td>
<td>Hearing aid may help with both hearing loss and tinnitus</td>
</tr>
<tr>
<td>Acoustic neuroma</td>
<td>Benign schwannoma of 8th cranial nerve</td>
<td>Slow onset. Usually unilateral. May exhibit tinnitus, vertigo. May exhibit facial hyperesthesias or twitching</td>
<td>May require surgical excision if symptoms debilitating</td>
</tr>
<tr>
<td>Ototoxic agents</td>
<td>Direct toxicity to inner ear structures</td>
<td>Variable onset. High frequency most affected. Exposure to ototoxic drugs. May have associated tinnitus</td>
<td>Stop use of offending agent</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>Multiple demyelinating lesions interfere with nerve conduction</td>
<td>Often other associated neurologic findings. May wax and wane</td>
<td>Standard multiple sclerosis treatment (steroids, cytotoxic agents)</td>
</tr>
<tr>
<td>Stroke/CVA</td>
<td>Focal ischemic lesion of auditory nerve or auditory cortex</td>
<td>Sudden onset. Often associated with other neurologic deficits</td>
<td>Treat CVA risk factors (ASA, anticoagulants, glycemic control, BP control)</td>
</tr>
<tr>
<td>Meningitis</td>
<td>Infection enters inner ear through CNS-perilymph connection. Damages organ of Corti</td>
<td>Follows clinical picture of meningitis</td>
<td>Treat infection. Steroids may limit inflammation and damage</td>
</tr>
<tr>
<td>Meniere disease (endolymphatic hydrops)</td>
<td>Abnormal homeostasis of inner ear fluids (clinical diagnosis; definitive diagnosis made histologically)</td>
<td>Episodic spells of vertigo. Associated sensation of fullness, tinnitus, and SNHL or auditory distortion. Low-frequency ranges most affected</td>
<td>Reduce salt, caffeine, nicotine (vasoconstrictors) intake. Consider diuretics, antihistamines, anticholinergics. ENT referral</td>
</tr>
<tr>
<td>Chronic noise exposure</td>
<td>Direct mechanical damage to cochlear structures and hair cells</td>
<td>Slow onset. Usually high frequency most affected</td>
<td>Prevention measures (earplugs). Stop exposure</td>
</tr>
<tr>
<td>Skull trauma</td>
<td>Interruption of cranial nerve VIII, ossicle disruption, or shearing effects on organ of Corti</td>
<td>Sudden onset after trauma</td>
<td>ENT consultation for possible surgical repair</td>
</tr>
<tr>
<td>Autoimmune causes</td>
<td>Vascular or neuronal inflammatory changes</td>
<td>Bilateral asymmetric SNHL. May be fluctuating or progressive. Often other systemic autoimmune findings</td>
<td>Outpatient autoimmune evaluation. Steroids and cytotoxic agents may slow progression</td>
</tr>
</tbody>
</table>

ASA, Acetylsalicylic acid; BP, blood pressure; CNS, central nervous system; CSF, cerebrospinal fluid; CVA, cerebrovascular accident; ENT, ear, nose, and throat; SNHL, sensorineural hearing loss; TM, tympanic membrane; URI, upper respiratory infection.
Sensorineural hearing loss may stem from several causes, but there are few emergency treatment options. The patient can be counseled about the variable recovery rate, and some prognosis may be given on the basis of the suspected cause of the lesion. Viral causes and inflammatory or autoimmune causes may respond to steroid treatment started in the first few days. Steroids have been regarded as standard therapy for sensorineural hearing loss suspected to be of viral etiology, although no controlled trials have shown significant benefit. Steroids should be prescribed with caution, and care must be taken to rule out infections, which may worsen with steroid treatment. Steroids should be given only if prompt follow-up is ensured. Antiviral agents (acyclovir, famciclovir, valacyclovir) are also commonly prescribed because of the possible role of herpes simplex virus type 1 as an etiologic agent in sensorineural hearing loss. No clear evidence has, however, shown a better outcome with steroids plus antiviral agents than with steroids alone.

Patients with suspected perilymph fistulas need absolute bed rest with head elevation to avoid raising intracranial pressure (and increasing flow of cerebrospinal fluid through the fistula). Some patients may require admission and sedation for this goal to be achieved.

Other causes of sensorineural hearing loss are not likely to be identified in the ED. These cases need expedited follow-up with an otolaryngologist for MRI and audiometry. Many patients receive relief from reassurance that their hearing loss is not a life-threatening event, but the emergency practitioner should be cautious and not give an overly optimistic picture because hearing often does not return after this type of hearing loss.

REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES