HEARING LOSS

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In a broad sense, there are three types of hearing loss. The first, *sensorineural*, involves the inner ear or cochlea, the auditory nerve, or a central neural lesion. The second, *conductive hearing loss*, is caused by the inhibition of sound from gaining access to the inner ear. This inhibition can be caused by something as simple as an ear canal filled with cerumen, fluid in the middle ear, or something complicated such as ossicular chain fixation. The third type of hearing loss is a *mixed loss*, that is, a combination of sensorineural and conductive. This article outlines the causes for these types of hearing loss, how they relate to the pediatric and adult populations, and treatment options.

HEARING EVALUATION

Simple tests can be performed in the office by the physician to assess a patient's hearing status. A history of the hearing loss and familial tendency to such loss and a comprehensive physical examination, including gross evaluation of hearing with the use of a 256- and 512-Hz tuning fork, are mandatory. The two most useful tests are the Rinne and the Weber. In an ear with normal hearing, air conduction (sound waves traveling to the tympanic membrane) is always louder than bone conduction (sound transmitted via the vibration of the skull). In the Rinne test, the patient compares the loudness of the tuning fork when it is placed on the mastoid bone behind the ear (bone conduction) versus when the tuning fork is held in front of the ear (air conduction). A positive or normal Rinne test result has an air conduction that is louder than bone conduction. A negative Rinne test result (bone conduction louder than air conduction) indicates a conductive hearing loss. The Weber test consists of placing the tuning fork on the bridge of the forehead, nose, or teeth and asking the patient if the sound is louder in one ear or the other. In a person with normal hearing, the sound is heard equally in each ear. If the sound is heard louder in one ear,

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comparison with the Rinne test is useful in helping to determine a diagnosis. If bone conduction was louder than air conduction in one ear (Rinne test), the louder hearing ear, via the Weber test, is the ear with the conductive hearing loss.

Pneumoscopy is always performed to evaluate the mobility of the tympanic membrane. Positive pressure forcing air into the external auditory canal (EAC) displaces the tympanic membrane medially. When the pressure is released, negative pressure pulls the tympanic membrane out. A nonmobile tympanic membrane may be due to fluid in the middle ear cavity, a mass, or a stiff tympanic membrane. A tympanic membrane that moves only with negative pressure looks retracted. Hypermobile tympanic membranes, which are difficult to see with a hand-held otoscope, may indicate ossicular chain disruption or a poorly healed tympanic membrane perforation.

Formal audiologic assessment is performed by an audiologist in a soundproof environment. This evaluation provides detailed information regarding a patient's hearing ability. The formal audiogram with a tympanogram and site of lesion testing normally provides adequate information.

OUTER EAR

All hearing loss related to the outer ear is by nature a conductive hearing loss.

Congenital

Microtia, the absence or malformation of the pinnae, may cause a mild-to-moderate conductive hearing loss, but atresia or stenosis of the EAC produces maximal (60 dB) conductive hearing loss. Unilateral atresia or stenosis of the EAC is more common than bilateral. Atresia malformations of the EAC occur in approximately 1:10,000 births and can be associated with other craniofacial abnormalities, such as Treacher Collins syndrome, Pierre Robin syndrome, or Crouzon's disease.[15] The severity of the atresia determines how well the child hears and whether surgical intervention may be of benefit.

Treatment for congenital atresia of the EAC depends on several factors. In children with bilateral congenital atresias, bone conduction hearing aids are placed as soon as possible. In patients with unilateral atresia, no action is taken until the child reaches the age of 5 or 6 as long as normal hearing exists in the normal ear.[8] At the age of 5 or 6, a computed tomography (CT) scan of the temporal bones determines if any abnormalities exist in the middle or inner ear. Surgical treatment options are then considered.[16]

Infection

Conductive hearing loss resulting from infections occurs because of blockage of the EAC. Such may be due to the edema or inflammation of the canal or debris within the canal. Otitis externa, more commonly known as *swimmer's ear*, causes intense ear pain with significant edema of the EAC. Often the canal is filled with purulent, squamous debris. Otitis externa is usually due to a combination of local trauma coupled with water after swimming, showering, or exposure to a hot, humid environment. These conditions allow the proliferation of bacteria, most notably *Pseudomonas aeruginosa*, or occasionally fungi.

The hallmark of treatment for otitis externa is proper cleaning of the EAC and the administration of topical steroid and antimicrobial drops. Often the EAC is so edematous that drops do not traverse the EAC easily. In these instances, a cotton or Merocel wick is used not only to stent open the ear canal, but also to provide a means for the drops to penetrate down the entire EAC. These wicks should be removed after 24 to 48 hours and a new one applied if necessary. Acetic acid with hydrocortisone is an excellent initial topical otic drop to use for otitis externa because the acidity decreases the pH of the EAC, thus eliminating most bacteria as well as fungi. Systemic or oral antibiotic treatment is usually unnecessary for the treatment of otitis externa, but often it is difficult to rule out an underlying otitis media, and antibiotics are used in such circumstances.

Trauma

Penetrating trauma to the EAC or meatus resulting from a bullet, knife, or fracture may cause a mild or profound conductive hearing loss depending on the degree of EAC occlusion. Supportive care with EAC stenting is the initial treatment. Surgical intervention is reserved for when and if healing does not occur properly.

Tumors

Certain tumors of the EAC may cause a conductive hearing loss because of occlusion of the EAC. The most common EAC tumor is squamous cell carcinoma.[17] Cancer of the EAC is usually misdiagnosed as an otitis externa. A biopsy specimen, usually obtained after multiple failed attempts at otitis treatment, provides the diagnosis. Treatment of these malignant disorders is surgical extirpation with possible adjuvant irradiation.

Benign growths of the EAC may also cause occlusion, with a resultant conductive hearing loss. These benign growths can be either exostoses or osteomas. Exostoses are multiple benign bony growths of the EAC that usually occur in individuals who have had repeated exposure to cold water (e.g., swimmers in northern climates). When the exostoses become so large that they occlude the canal, surgical intervention clears such. Osteomas, solitary bony growths usually at the tympanomastoid suture line, are treated the same as exostosis.[9]

Endocrine

Although infectious in nature, necrotizing or malignant otitis externa may also be a cause of conductive hearing loss. This entity is seen only in patients with insulin-dependent diabetes or other immunocompromised state. The ear becomes swollen, erythematous, and painful to the touch. Usually osteomyelitis ensues; biopsy specimens demonstrate chronic and acute inflammation. Treatment consists of intravenous antibiotics for *Pseudomonas* and débridement. Hyperbaric oxygen treatment may help in the treatment by increasing oxygenated blood flow to the site of infection.

MIDDLE EAR

The middle ear consists of the tympanic membrane, the ossicular chain, and the middle ear space. Just as in the outer ear, all hearing loss associated with middle ear diseases consists of conductive hearing loss.

Congenital

Atresia, malformation or bony fixation of the ossicular chain, causes an apparent conductive hearing loss. Abnormal development of the ossicular chain is much more difficult to observe on otoscopic examination and is usually best seen with CT scan. Treatment consists of correcting the abnormality and restoring mobile ossicular continuity, or a hearing aid may provide significant benefit.

Infection

Otitis media is by far one of the commonest diagnoses made in the pediatric population and is also common in adults.[6] The annual cost of treatments for otitis media nationwide is greater than $5 billion. Reports demonstrate that 80% to 90% of all children have developed at least one episode of otitis media by the time they enter school.[7] Besides the obvious symptoms, such as pain, fever, and ear fullness, a decrease in hearing is usually reported. Hearing loss occurs because fluid fills the middle ear space, preventing the tympanic membrane from vibrating adequately.

Once the acute infection has subsided, the middle ear may still be filled with serous or thick fluid. Studies have shown that this fluid generally resolves in 70% of cases within 4 to 6 weeks.[18] In 8 to 12 weeks, 85% to 90% of all children or adults have reabsorbed the middle ear fluid.[5] In those patients in whom the fluid does not clear on its own, myringotomy and tube placement is recommended because it removes the fluid and allows the middle ear to aerate, heal, and resolve any conductive hearing loss. Most adults do not tolerate fluid in the middle ear space for longer than 2 to 4 weeks. Thus, myringotomy and aspiration is a common office procedure in the adult population.

Tumors

Although malignant tumors, such as squamous cell carcinoma or Letterer-Siwe disease, may cause conductive hearing loss, these entities are relatively rare when compared to benign growths, such as cholesteatoma and otosclerosis.

Cholesteatoma

By definition, cholesteatoma is a growth of desquamated stratified squamous epithelium. As keratin accumulates from the epithelial lining of the sac, it gradually becomes bigger with erosion of the ossicular chain or mastoid. Formation of cholesteatoma usually occurs after a retraction pocket has formed in the posterior-superior quadrant or after tympanic membrane trauma. If left untreated, cholesteatomas may eventually erode the bony covering of the tegmen into the middle fossa, the sigmoid sinus, or even the inner ear. The complications of cholesteatoma include lateral sinus thrombosis, sepsis, brain abscess, sensori-

neural hearing loss, vertigo, dysequilibrium, facial nerve paralysis, and death.[4] Therefore referral for management of cholesteatomas is mandatory. The treatment of cholesteatoma is surgical, usually involving a tympanomastoidectomy.

Otosclerosis

Otosclerosis is a bony overgrowth involving the footplate of the stapes. As the stapes becomes fixated, conductive hearing loss becomes significant. Treatment for otosclerosis and the accompanying hearing loss is either hearing amplification or stapedectomy. In stapedectomy, a small hole is placed in the footplate of the stapes, and the superstructure of the stapes is removed. A piston prosthesis is then placed from the incus into the stapedotomy hole, which, in turn, restores ossicular function.

Tympanic Membrane Perforation

Conductive hearing loss resulting from a tympanic membrane perforation is common. The degree of conductive hearing loss depends on the size and location of the perforation. Perforations that are almost near-total or located in the posterior-superior quadrant have a much higher chance of causing significant conductive hearing loss than small perforations in the anterior-inferior quadrant. Perforations may be caused by events such as a blast injury, barotrauma, foreign body trauma, temporal bone fractures, ear infections, or self-inflicted trauma from a Q-tip or other object or may persist after myringotomy tubes extrude. A patient with a perforation needs an audiogram to document the level of hearing. The ear should also be evaluated to ensure that skin is not trapped on the undersurface of the tympanic membrane, which could then lead to cholesteatoma formation. Surgical correction of the perforation is usually accomplished with a temporalis muscle fascia graft.

Temporal bone trauma from fractures or other blunt injury may cause a tympanic membrane perforation but, more importantly, may also cause ossicular discontinuity. When the ossicles are no longer attached to each other, a conductive hearing loss up to 60 dB can be produced. The diagnosis is often made with CT scans or the high compliance of tympanic membrane with pneumoscopy. Surgical management is indicated in these cases to restore ossicular continuity.

Vascular

Glomus tympanicum or jugulare tumors may cause conductive hearing loss by impeding ossicular or tympanic membrane motion. These benign paragangliomas arise within the ear, either from the adventitia of the dome of the jugular bulb or from the promontory of the middle ear. As these tumors enlarge, they erode bone and may eventually cause damage to cranial nerves 7 through 12 in addition to the conductive hearing loss. Identification on pneumoscopy is important. Often with pneumoscopy, the red mass behind the tympanic membrane blanches. Treatment is complete removal.[1] Radiation therapy may slow the growth of these tumors but does not cure them and is usually reserved for patients who cannot tolerate significant surgery.

INNER EAR

Disorders of the inner ear normally cause a sensorineural hearing loss. The cause may be associated with the cochlea, eighth cranial nerve, internal auditory canal, or brain.

Congenital and Hereditary

Congenital hearing loss can simply be thought of as a hearing loss that occurs at birth or shortly thereafter, which may be due to either a hereditary or a nonhereditary cause. Nonhereditary causes usually involve an insult to the developing cochlea, such as exposure to viral infections, including cytomegalovirus, hepatitis, rubella, toxoplasmosis, human immunodeficiency virus (HIV), and syphilis. Certain medications may also have a teratogenic effect on the developing ear, including recreational drugs as well as alcohol, quinine, and retinoic acid.

Hereditary sensorineural hearing loss may have an autosomal recessive or dominant pattern. Sensorineural hearing loss may be part of a syndrome or occur as a spontaneous mutation. Sensorineural hearing loss may be present at birth; may be progressive from birth; or present when the child is older, including teens or even early adult life. Studies have demonstrated that 90% of all hereditary sensorineural hearing loss is via an autosomal recessive pattern, thus explaining why these children often have normal-hearing parents.[11, 20]

Congenital malformations of the inner ear range from complete atresia to a common cavity of the cochlea. The most common malformation is a Mondini malformation, in which the normal 2.5 turns of the cochlea are replaced by 1 to 1.5 turns. Hereditary hearing loss may fluctuate, progress, or remain stable.

Patients with congenital anomalies of either the inner ear or the middle ear have a higher association with perilymphatic fistulas, which may cause progressive or fluctuating sensorineural hearing loss.[23, 24] In these patients, surgical exploration with repair of perilymphatic fistulas may be indicated.

Presbycusis

Presbycusis is the hearing loss everyone experiences as they age. Multiple factors, including genetics, medications, and a lifetime of noise exposure, influence the degree and rate of hearing loss. The hearing loss seems to become more significant in the sixth decade. Presbycusis is almost always symmetric and usually starts in the high-frequency range (Fig. 1). Frequent associated complaints with presbycusis are the inability to hear or understand speech in a crowded or noisy environment as well as having difficulty with high-pitched voices and noises. Occasionally, tinnitus is also present. Hearing aids benefit most patients. At times, hearing amplification is not tolerated by patients because of increased static, noise, discomfort, inability to understand speech, or cosmetic concerns. Patients with a unilateral or asymmetric sensorineural hearing loss do not have typical presbycusis and require further otologic evaluation.

Infection

The most common infection in adults is a viral cochleitis, whereas in young children it is meningitis.[2] Meningitis can access the cochlea by way of the

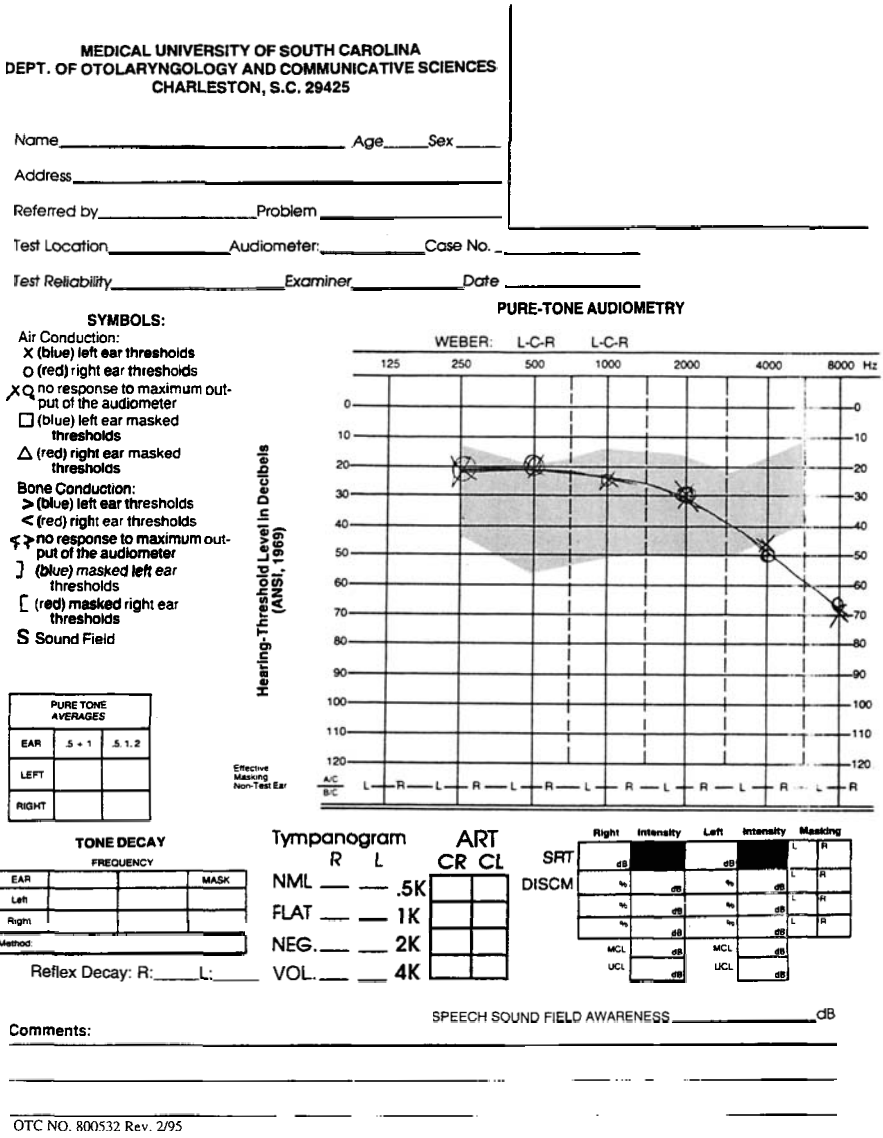

Figure 1. Audiogram depicting a usual high frequency hearing loss pattern commonly associated with presbycusis.

cerebrospinal fluid–perilymph fluid connection and cause a profound sensorineural hearing loss by destroying the inner hair cells. Many who have suffered profound sensorineural hearing loss resulting from infections or other causes may still be able to obtain hearing with the use of cochlear implants. A cochlear implant consists of an electrode array that is inserted into the cochlea. The

patient wears a processor similar to a beeper, which is connected to the implanted device underlying the skin via a magnet.

Viral cochleitis usually manifests itself as a sudden sensorineural hearing loss. Any patient who presents with complaints of sudden hearing loss, or even if it is not so sudden, should have audiometric evaluation. Although the primary cause of sudden sensorineural hearing loss is usually either viral in nature or a vascular ischemic event, there are other causes, such as acoustic neuroma, perilymph fistulas, Ménière's disease, vascular insufficiency, multiple sclerosis, and other central nervous system conditions. Therefore patients who present with sudden sensorineural hearing loss also should have a magnetic resonance (MR) imaging scan with gadolinium.[25] When the sudden sensorineural hearing loss is due to a virus or vascular cause, 70% to 90% of all patients experience anywhere from some to full recovery of hearing, which may take up to 3 months. It is thought that herpesvirus may be involved in sudden sensorineural hearing loss, just as it is thought to be a causative factor in Bell's palsy. Current treatment for sudden sensorineural hearing loss of unknown origin is a 10-day course of high-dose steroids (prednisone 60 to 80 mg every morning) and possibly a 7- to 10-day course of valacyclovir (Valtrex) (1 g three times a day) or famciclovir (Famvir). Prognosis for the sudden sensorineural hearing loss is usually good if it is primarily a high-frequency hearing loss pattern or a low-frequency hearing loss pattern, but those that are flat have the worst prognosis. Other treatment options for sudden sensorineural hearing loss are available, including carbogen, histamine, intravenous diatrizoate (Hypaque), and dextran.[12]

Ménière's Disease

Ménière's disease is described as episodic with vertiginous spells (lasting hours) associated with hearing loss, aural fullness, and tinnitus. Occasionally, only the auditory system may be affected. The patient then experiences episodic hearing loss that usually recovers within a 12- to 24-hour period. At times, the patient may also complain of associated ear fullness, tinnitus, or both. The hearing loss is almost always low frequency. Over time the hearing loss becomes permanent and may eventually involve all frequencies. The hearing loss spells may occur daily, weekly, or monthly. Standard medical therapy of low-salt diet, no caffeine or alcohol, and diuretics does not seem to prevent hearing loss, even though it helps the vertigo. Surgery has little benefit either. Chronic otitis media may also produce sensorineural hearing loss as a result of the effects of chronic toxin exposure, which eventually can permeate through the round window membrane and into the inner ear system.

Noise Exposure

To some degree, everyday noise exposure compounded over time is thought to have an impact on the ability to hear and, ultimately, the degree of presbycusis. Constant exposure to loud noises can clearly cause a high-frequency sensorineural hearing loss. Figure 2 represents a typical audiogram of noise-induced sensorineural hearing loss with the worst hearing level at 4000 Hz, then improving at 8000 Hz. To protect workers from noise exposure, the federal Occupational Safety and Health Administration (OSHA) has set standards. Employees exposed to levels greater than 85 dB over an 8-hour workday must be enrolled in a hearing conservation program and be provided hearing protection. Furthermore,

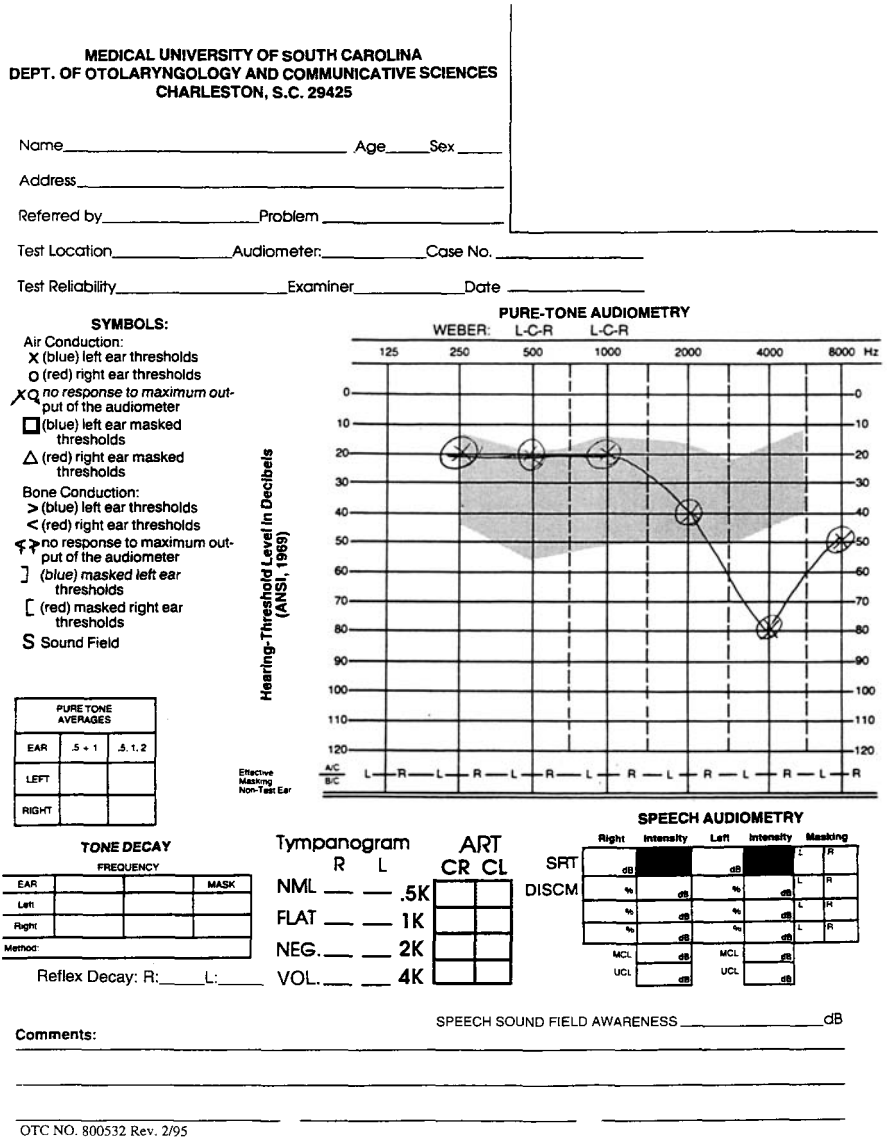

Figure 2. Audiogram depicting a noise exposed sensorineural hearing loss.

OSHA standards limit employees' exposure to noise such that if the time-weighted average is 90 dB, they may be exposed for 8 hours; 95 dB, for 4 hours; 100 dB, for 2 hours; and so on.[21] For personal reference, a blank shot from a 22-caliber pistol makes a noise of approximately 90 dB. Sudden exposure to noise, greater than 115 to 120 dB, may also cause a permanent hearing loss or result in hyperacusis (pain associated with loud noise).

Barotrauma

Barotrauma is caused by sudden, large changes in air pressure, such as those associated with diving or flying. During ascent with flying, the middle ear pressure increases until the eustachian tube is forced open. On descent, negative middle ear pressure develops until swallowing opens the eustachian tube. In diving, positive middle ear pressure is experienced on descent, whereas negative middle ear pressure is experienced with ascent. Failure to equilibrate the pressure may cause tympanic membrane perforations, rupture of cochlear blood vessels, perilymphatic fistulas, or middle ear accumulation of fluid or blood. Treatment is usually bed rest with the possible use of a burst (7 days) of high-dose steroids. If hearing continues to fluctuate, an exploration or repair of perilymphatic fistula is indicated because implosion may cause a leakage of fluid out of the round or oval window.

Trauma

Penetrating trauma usually causes a sensorineural hearing loss or mixed loss but more often than not causes a dead or anacoustic ear with total hearing loss. These injuries are usually due to gunshot wounds, which on impact cause significant temporal bone fractures.

Blunt trauma may cause a sensorineural hearing loss as a result of concussive forces of the inner ear fluids (shearing effect on the cochlea or organ of Corti) or a temporal bone fracture. Two types of temporal bone fractures exist: longitudinal and transverse. Longitudinal is most common (80%), usually caused by a blow to the temporal parietal region.[6] Hearing loss is usually conductive with an associated tympanic membrane perforation and blood in the middle ear space. Transverse fractures occur approximately 20% of the time and are due to blows to the occipital or frontal regions. These blows cause a fracture through the inner ear with a resultant *dead* ear. The commonest ossicular injuries associated with these fractures are a separation of the incudal stapedial joint and incus dislocation.

Iatrogenic trauma is usually caused by a Q-tip being placed too far into the ear, which can then not only cause a tympanic membrane perforation, but also it may cause subluxation of the stapes, or it may push the stapes into the vestibule. These maneuvers could cause a significant sensorineural hearing loss and quite possibly a vestibulopathy.

Tumors

Most tumors of the inner ear are benign. Malignant tumors, such as squamous cell carcinoma, sarcomas, or adenoid carcinoma, may occur but are rare. Benign bony tumors, such as fibrous dysplasia or Paget's disease, are also rare. The most common tumor causing sensorineural hearing loss is an acoustic neuroma. Acoustic neuromas are benign and usually originate from the vestibular portion of the eighth cranial nerve.

The most common complaint (90%) in acoustic neuroma is an asymptomatic or unilateral sensorineural hearing loss.[13] Other symptoms include unilateral tinnitus, dysequilibrium, dizziness, or headaches. Later findings could include facial hypesthesia or facial musculature twitching. Confirmation of the diagnosis is made by MR imaging with gadolinium. Treatment of acoustic neuroma de-

pends on the size of the tumor, the patient's medical condition, the age of the patient, and whether a tumor exists on the contralateral side (neurofibromatosis II) and the degree of dysfunction that the tumor is causing. Acoustic neuromas are slow growing, usually 1 to 2 mm per year, and thus small tumors without any symptoms can sometimes be observed and followed with repeated scans in the elderly or medically debilitated.[3] In most circumstances with small tumors, hearing-preservation surgical approaches are used, whereas for large ones, hearing is seldom preserved. Retaining facial nerve function is one of the primary goals of acoustic neuroma surgery as well as removal of the tumor. Other benign tumors that may cause a sensorineural hearing loss and occur in the cerebellar pontine angle include meningiomas, lipomas, and lymphoproliferative disease.

Endocrine and Systemic

Various metabolic abnormalities may be associated with sensorineural hearing loss. Blood chemistry levels are evaluated, especially for glucose, to ensure that the patient does not have diabetes, which, with its resultant small vessel disease, may cause ischemia to the cochlea. Complete blood count with differential is obtained to eliminate anemia or a white blood cell dyscrasia disorder. Hyperthyroidism and hypothyroidism are causes of sensorineural hearing loss, and thus thyroid function tests are obtained in addition to physical examination. Any patient with an unexplained hearing loss also receives a test for syphilis, fluorescent treponemal antibody absorption test (FTA/ABS), or microhemagglutination test with *Treponema pallidum* (MHA-TP). In children, urinalysis rules out protein spillage in the urine.

Autoimmune hearing loss is a relatively recent entity, dating from 1979, described by McCabe.[19] Autoimmune hearing loss usually consists of a bilateral asymmetric sensorineural hearing loss that may fluctuate or be progressive in nature. The condition may be limited just to the ear or may be part of an overall systemic problem, such as Wegener's granulomatosis, Cogan's syndrome, rheumatoid arthritis, systemic lupus erythematosus, Paget's disease, polyarteritis nodosa, and relapsing polychondritis. Laboratory work consists of obtaining an erythrocyte sedimentation rate, antinuclear antibody levels, and rheumatoid factor, and, at times, consideration is given to C-ANCA levels, C1q-binding, CH-50, Raji cell assays, and cryoglobulin counts. Blood may be sent to special laboratories to test for cochlear autoantibodies.

The first line of treatment in patients with autoimmune hearing loss is high-dose steroids, usually 60 to 80 mg prednisone every morning for 2 to 3 weeks. Once there has been a recovery of the hearing, the steroids are tapered gradually until hearing loss again occurs, and a long-term level of steroids required is established. In patients who fail steroid therapy, cytotoxic medications, such as cyclophosphamide, methotrexate, or azathioprine (Imuran), may be considered.[14]

Iatrogenic

Injuries that are iatrogenic may be surgically related, as in tympanomastoidectomy or stapes surgery, or may be medication related. Many drugs are ototoxic, with aminoglycosides the most well known. Although all aminoglycosides are ototoxic, gentamicin and streptomycin are more vestibulotoxic, whereas tobramycin and amikacin are more cochleotoxic. Whenever aminoglycosides are administered, hearing levels should be monitored. Dosages may be altered or a

different antibiotic substituted if hearing levels diminish. Other antibiotics that can also cause ototoxicity include erythromycin, vancomycin, and tetracycline, particularly in renally impaired patients.

As would be expected, chemotherapeutic agents may cause hearing loss. The most common chemotherapeutic agents include 5-fluorouracil, bleomycin, and nitrogen mustard, but the worst offender is cisplatin. The hearing loss exhibited with antibiotic or chemotherapeutic agents usually begins at high frequencies. With continued usage, the hearing loss can become more pronounced, even after the drug is discontinued. These hearing losses are irreversible.

Aspirin or salicylates can also cause hearing loss. The hearing loss and tinnitus are reversible with the discontinuation of the drug. The hearing loss is believed to be due to an enzymatic inhibition, and because of this, high doses (average of 6 to 8 g/d) are required to exhibit ototoxicity. Antimalarial medications, such as quinine and chloroquinine, may also cause sensorineural hearing loss and tinnitus, but this again is usually reversible. Loop diuretics and other medications (such as β-blockers) may cause a temporary sensorineural hearing loss and tinnitus.

Ototopical Medications

Several ototopical drops have the potential to cause ototoxicity. Cortisporin has neomycin, tobramycin, and gentamicin. There are few case reports that demonstrate a sensorineural hearing loss with the use of ototopical medications, and even in these, it is not clear that drops were the reason for the sensorineural hearing loss. It is believed that the inflamed mucosa prevents significant drug penetration into the oval or round window.

Neurogenic

Although cerebrovascular accidents or transient ischemic attacks may cause sensorineural hearing loss, there are other neurologic sequelae. Arnold-Chiari malformations may produce auditory vestibular complaints because of stretching of the eighth nerve complex, and, at times, this may be the only manifestation of Arnold-Chiari syndrome.[22] Multiple sclerosis can initially present as a sudden sensorineural hearing loss or vertigo in up to 10% to 15% of cases.[10] Diagnosis is usually confirmed with an MR imaging scan or analysis of cerebrospinal fluid.

CONCLUSION

There are many causes for both conductive hearing loss and sensorineural hearing loss. A complete history and physical examination should point to the problem. Whenever a hearing loss is suspected, an audiogram is essential. Anytime questions arise that concern the auditory or vestibular system, referral to an otolaryngologist is appropriate.

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