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Up to Date on Etiology and Epidemiology of Hearing Loss

Larissa Vilela Pereira and Fayez Bahmad Jr.

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Abstract

Deafness is one of the most common communication disorders in humans. Approximately one out of every thousand infants is born with a significant hearing deficit. The prevalence of hearing loss increases dramatically with age. By age 65 years, one out of three of us will suffer from hearing impairment sufficient to interfere with the understanding of speech. Hearing impairment is a very heterogeneous disorder with a wide range of causes. Worldwide, estimates from the World Health Organization are that hearing loss affects 538 million people. Hearing loss may be classified into three types: sensorineural, involving the inner ear, cochlea, or the auditory nerve; conductive, when the outer or middle ear structures fail to optimally capture, collect, or transmit sound to the cochlea; and mixed loss, which is a combination of conductive and sensorineural hearing loss. In this chapter, we propose to briefly define each cause of hearing loss as follows: (1) outer ear causes (congenital, infection, trauma, tumor, dermatologic, and cerumen), (2) middle ear causes (congenital, eustachian tube dysfunction, infection, tumors, otosclerosis, tympanic membrane perforation, middle ear barotrauma, and vascular), and (3) inner ear causes (congenital or hereditary, presbycusis, infection, Ménière disease, noise exposure, inner ear barotrauma, trauma, tumors, endocrine/systemic/metabolic, autoimmune hearing loss, Iatrogenic, ototoxic, and neurogenic).

Keywords: Etiology, hearing loss, conductive hearing loss, sensorineural hearing loss

1. Introduction

Deafness is one of the most common communication disorders in humans. Approximately one out of every thousand infants is born with a significant hearing deficit, and the prevalence of hearing loss increases dramatically with age. By age 65 years, one out of three of us will suffer from hearing impairment sufficient to interfere with the understanding of speech. Hearing impairment is a very heterogeneous disorder with a wide range of causes.[1]

Worldwide, estimates from the World Health Organization are that hearing loss affects 538 million people.[1]

Hearing loss may be classified into three types:

- Sensorineural: involving the inner ear, cochlea, or the auditory nerve

- Conductive: when the outer or middle ear structures fail to optimally capture, collect, or transmit sound to the cochlea

- Mixed loss: a combination of conductive and sensorineural hearing loss

2. Outer ear causes

The outer ear comprises the auricle and the external auditory canal (EAC), and all hearing loss related to the outer ear is by nature a conductive hearing loss.

2.1. Cerumen

Probably the most common cause of conductive hearing loss is the complete blockage of the EAC by a cerumen impaction. Some patients are not able to clear it on their own or use Q-tips that push the cerumen down the ear canal. These individuals may need periodic cleaning to enhance their auditory capabilities.

2.2. Infection

Infections may lead to blockage of the EAC due to the accumulation of debris, edema, or inflammation. Acute otitis externa usually develops as a result of local trauma coupled with contamination by bacteria (or occasionally fungi—otomycosis or viral—herpes zoster oticus) after swimming, showering, or exposure to hot and humid conditions. With complete obstruction, a conductive hearing loss results. Diabetes mellitus and other immunocompromised states can predispose to developing malignant otitis externa.[2]

2.3. Congenital

The auricle and the EAC are derived from different embryologic tissue, and each may develop without the full maturation of the other sites. The EAC develops from the 8th to the 28th week of gestation, and the auricle itself forms from remnants of the first and second branchial arch during the 12th and 20th weeks. Problems can occur anytime during this developmental phase, and it is possible to have a normal auricle but an atretic canal. The anatomical course of the facial nerve is frequently altered in malformations of the ear and temporal bone, but facial nerve function is rarely affected by the malformation. Conductive hearing losses that result from congenital malformations may range from mild to severe.

Malformation of the auricle is termed anotia when there is complete absence of an external ear and microtia when there is a vestige present. Anotia and microtia may cause mild to moderate conductive hearing loss.[2]

Congenital atresia of the EAC occurs in approximately 1 per 10,000 births and are usually associated with other craniofacial abnormalities such as Treacher–Collins syndrome, Pierre Robin syndrome, or Crouzon disease. The malformation occurs unilaterally 4 times more frequently than it does bilaterally (Figure 1).[3]

The severity of the atresia determines how well the child hears, and some patients with congenital atresia have associated inner-ear abnormalities, but these abnormalities typically do not cause sensorineural hearing loss. Atresia or significant stenosis of the EAC causes moderate to severe conductive hearing loss.[3]

Figure 1. Congenital atresia of the EAC

2.4. Tumors

Malignant cancer of the ear canal is rare, and the most common histology type is squamous cell carcinoma. Other tumors include basal cell carcinoma, adenoid cystic carcinoma, adeno-carcinoma, and melanoma. Initially, cancer of the EAC is usually misdiagnosed as otitis externa because most patients complain of otorrhea, aural fullness, pain, itching, and hearing loss. However, after multiple failed attempts at treatment with ototopical drops and antibiotics, a

biopsy of the EAC should be obtained. Treatment of these malignant tumors varies with the specific neoplasm.[4]

Benign bony growths may also occlude the EAC with a resulting conductive hearing loss. The two most common benign growths are exostosis and osteoma.

Exostosis are the most common solid tumor of the EAC. They are periosteal outgrowths that develop in the bony ear canal and occur mostly in men who have had repeated exposure to cold water. The lesions are often multiple, bilateral, and form in the suture lines of the EAC bones. Surgical removal is performed in cases of conductive hearing loss or recurrent otitis externa.[5]

Osteoma, in contrast, is a solitary bony growth that is most commonly attached to the tympanosquamous suture line. Similar to exostoses, osteomas are not treated until they become so large that they affect hearing by occlusion or repeated infections because debris cannot exit the EAC.[6]

Benign polyps may occur as a result of other otologic conditions, such as chronic ear infections or cholesteatoma. Occasionally, benign polyps can grow large enough to occlude the lumen of the external auditory canal.

2.5. Trauma

Penetrating trauma to the external auditory canal or meatus due to bullet, knife, or fracture may cause mild or profound conductive hearing loss, depending on the degree of EAC occlusion. Ototopical drops prevent otitis externa, and external auditory canal stenting is required initially to ensure that the EAC does not develop significant stenosis. Surgical intervention is reserved for cases of stenosis.

3. Middle ear causes

As with the outer ear, all hearing loss associated with the middle ear is conductive hearing loss.

3.1. Congenital malformation

Malformation of the ossicular chain can cause conductive hearing loss. The most common ossicular abnormality observed is a missing or malalignment of the crura of the stapes. However, it is usually an abnormal incus or malleoincudal joint that causes the conductive hearing loss. Computed tomography (CT) scan is virtually always needed in order to make this diagnosis, and in some cases, exploratory tympanostomy may be required.[2]

3.2. Eustachian tube dysfunction

The eustachian tube serves to provide normal middle ear pressure when opened and to protect the middle ear from reflux and nasopharyngeal bacteria when closed. Abnormal function

occurs commonly in the setting of an upper respiratory infection (viral or bacterial), and it can also occur with allergies or tumors. It results in negative middle ear pressure causing reduction of tympanic membrane (TM) excursion and conductive hearing loss.

3.3. Infection

Acute otitis media (AOM) is a common childhood disorder that also frequently occurs in adults. Approximately 80% to 90% of all children will have developed at least one episode of OM by the time they enter school, and AOM accounts for more than 25% of the prescriptions for oral antibiotics annually.[7]

It is normally associated with pain, fever, and ear fullness as well as decreased hearing. Conductive hearing loss occurs because fluid filling the middle ear space prevents the TM from vibrating adequately, thereby diminishing movement of the ossicular chain.[8]

The middle ear may still be filled with serous or thick, tenacious fluid after the acute infection has been successfully treated. This fluid resolves within four to six weeks in 70% of cases. By an additional 12 weeks, 85% to 90% of all resolve the condition on their own. However, in the 10% to 15% in whom the fluid does not clear, it needs to be removed and the middle ear aerated in order to promote resolution of any conductive hearing loss. The fluid is usually cleared by either myringotomy and pressure equalization tube placement, or myringotomy and aspiration.[9]

3.4. Tympanic membrane perforation

Conductive hearing loss due to TM perforation is common. Clearly, the size, location, and nature of perforation will affect the degree of hearing loss. Small perforations and those located in the anterior–inferior quadrant cause the least amount of conductive hearing loss; near total or posterior–superior quadrant perforations have a much higher chance of causing significant hearing loss.[10]

Tympanic membrane perforations can arise as a consequence of either infection or trauma. Most perforations heal spontaneously. Occasionally, surgical correction is required, and repair of the perforation often corrects the conductive hearing loss.[7]

3.5. Otosclerosis

Otosclerosis is a primary disease of the temporal bone, leading to stapes ankylosis. Hearing loss is the main symptom. Complaints of continuous tinnitus and, eventually, vertigo are also observed. Otosclerosis is considered an autosomal dominant disease with incomplete penetrance being identified in three related genes: OTSC 1, OTSC 2, and OTSC 3. Treatment includes surgery, medical treatment, and sound amplification therapy alone or in combination.

3.6. Cholesteatoma

Cholesteatoma is a growth of desquamated, stratified, squamous epithelium within the middle ear space. Such collections of desquamated skin cells will erode bone slowly through a

combination of pressure necrosis and enzymatic activity. Infection accelerates the process of bony destruction. The formation of a cholesteatoma typically occurs after a retraction pocket has formed in the posterior/superior quadrant, often the result of poor eustachian tube function. It may also occur after tympanic membrane trauma, such as a traumatic, inflammatory, or iatrogenic perforation, with implantation of squamous cells.[11]

Conductive hearing loss can occur as one or all ossicles become destroyed. Left untreated, cholesteatomas may erode the tegmen, the sigmoid sinus, or even the inner ear, resulting in labyrinthine fistula, which causes severe or profound sensorineural hearing loss and vertigo. Thus, untreated, they can cause lateral sinus thrombosis, sepsis, brain abscess, facial paralysis, and even death. Treatment is surgical, usually involving a mastoidectomy.[7]

3.7. Neoplasm

Malignant tumors such as Langerhans cell histiocytosis or squamous cell carcinoma may also occur in the middle ear and can cause conductive hearing loss. However, these entities are relatively rare when compared with cholesteatoma.

3.8. Middle ear barotrauma

Barotrauma occurs when a patient is exposed to a sudden, large change in ambient pressure, often during diving or flying. Middle ear pressure becomes more positive with respect to ambient pressure during ascent until the eustachian tube is forced open. On descent, ambient pressure exceeds middle ear pressure until swallowing opens the eustachian tube.

Pressure in the middle ear normally equilibrates with ambient pressure via the eustachian tube. However, if upon descent with flying or diving this equalization is prevented by mucosal edema secondary to an upper respiratory infection, pregnancy, or anatomic variations, the negative relative pressure in the middle ear can lead to its filling with serous fluid or blood or to inward rupture of the TM. Symptoms vary from a sensation of pressure to hearing loss and pain, which may suddenly be relieved with rupture of the TM.[12]

Overpressurization of the middle ear can occur during ascent with flying or diving, but TM rupture is rare.

3.9. Vascular

Glomus tumors are the most common benign neoplasm of the middle ear. They arise from paraganglionic tissue from the promontory of the middle ear or the adventitia of the dome of the jugular bulb and may rarely show malignant potential.[8]

As tympanic tumors grow, they tend to fill the middle ear, with resultant pulsatile tinnitus with or without conductive hearing loss. They also erode bone as they enlarge, especially inferiorly, causing damage to cranial nerves. In addition, tumors may impede upon the ossicular chain and TM, thereby decreasing motility of either or both structures.[2]

Treatment may include surgical resection, embolization, and radiation.

4. Inner ear causes

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

4.1. Congenital or hereditary

Congenital hearing loss will be defined as any hearing loss that occurs at or shortly after birth that may be due to either a hereditary or nonhereditary cause. Nonhereditary etiologies involve an insult to the developing cochlea, including viral infections such as cytomegalovirus, hepatitis, rubella, toxoplasmosis, HIV, and syphilis. Some teratogenic medications may also affect the developing ear of the fetus, including recreational drugs, alcohol, quinine, and retinoic acid.[2]

Sensorineural hearing loss can be inherited in an autosomal dominant or recessive pattern; 90% is autosomal recessive, so that the children often have normal hearing parents. Sensorineural hearing loss also may be part of a syndrome or occur as a spontaneous mutation. The hearing deficit may be present at birth, be progressive from birth, or present when the child is older, or even early adult life. The most common testable genetic defect is an abnormal connexin 26.[1]

Congenital malformations of the inner ear also occur, these include anything from complete atresia to a common cavity of the cochlea. The most common malformation is a Mondini, where the normal two-and-one-half turns of the cochlea are replaced by one to one-and-one-half turns.

Patients who have congenital anomalies of either the inner or the middle ear may also develop perilymphatic fistulas (PLFs). PLFs alone can cause progressive or fluctuating sensorineural hearing loss.

4.2. Presbycusis

Presbycusis, or age-related hearing loss, is a common cause of hearing loss worldwide. This disorder is a complex and multifactorial, characterized by symmetrical progressive loss of hearing over many years. It usually affects the high frequencies of hearing, although its presentation and clinical course can be variable. Presbycusis has a tremendous impact on the quality of life of millions of older individuals and is increasingly prevalent as the population ages.[8]

Common complaints associated with presbycusis include the inability to hear or understand speech in a crowded or noisy environment, difficulty understanding consonants, and the inability to hear high pitched voices or noises. Tinnitus is often present.[2]

The prevalence of hearing loss increases with age, with up to 80% of functionally significant hearing loss occurring in older adults.

The World Health Organization (WHO) estimates that in 2025, there will be 1.2 billion people over 60 years of age worldwide, with more than 500 million individuals who will suffer significant impairment from presbycusis.

Hearing aids are able to benefit most patients with presbycusis, and cochlear implantation may benefit patients of any age who are not helped by hearing aids.

4.3. Infection

The most common infection of the inner ear is viral cochleitis in adults and meningitis in young children. Meningitis can access the cochlea by way of CSF-perilymph fluid connection and cause a profound sensorineural hearing loss by destroying the inner ear hair cells. Viral cochleitis usually manifests as a sudden sensorineural hearing loss and vertigo.[2]

Other causes of sudden sensorineural hearing loss include acoustic neuroma, perilymphatic fistula, Ménière disease, vascular insufficiency, multiple sclerosis, and other central etiologies. Although the primary etiology of sudden sensorineural hearing loss is almost always viral or a vascular ischemic event, patients with this presentation need to undergo audiometric evaluation as well as a magnetic resonance imaging (MRI) with gadolinium.

4.4. Ménière`s disease

Ménière`s disease is characterized by (1) spontaneous episodes of vertigo lasting several minutes to hours, (2) low-pitched tinnitus occurring or worsening during a vertiginous attack, (3) fluctuating low-frequency sensorineural hearing loss, and (4) aural fullness in the affected ear.[8]

The onset of symptoms is typically between the third and sixth decades, with a slight female preponderance. Endolymphatic hydrops is the main histopathologic correlate. Over time and with repeated attacks, the hearing deficit can become permanent and may even eventually involve all frequencies.

4.5. Noise exposure

Everyday noise exposure, compounded over time, has an impact upon our ability to hear. Constant exposure to loud noises can cause high-frequency sensorineural hearing loss, beginning with selective loss in 4000 Hz. With continued exposure, the notch widens and affects all high frequencies. Eventually, hearing loss can be seen in middle and lower frequencies. A short blast of loud noise also can cause severe to profound sensorineural hearing loss, pain, or hyperacusis. This usually involves exposure to noise greater than 120 to 155 dB.[2]

The mechanism by which excessive noise induces hearing loss includes direct mechanical damage of cochlear structures and metabolic overload due to overstimulation. Some potential metabolic effects are excess nitric oxide release that can damage hair cells, generation of oxygen free radicals that become toxic to membranes, and low magnesium concentrations that weaken hair cells by reducing the concentration of intracellular calcium.

Thus, hearing protection in the form of muffs or plugs is highly recommended anytime a person is exposed to loud noise.

4.6. Inner ear barotrauma

Barotrauma occurs when a patient is exposed to a sudden, large change in ambient pressure, often during diving or flying.

Inner ear barotrauma is a fairly uncommon injury but should be excluded in all cases of middle ear barotrauma. It can occur following the development of a sudden pressure differential between the inner and middle ear, leading to rupture of the round or oval window. The main symptoms are tinnitus, vertigo, and hearing loss. The resulting labyrinthine fistula and leakage of perilymph can result in permanent inner ear damage. The primary treatment of this complication is complete bed rest with head elevation to avoid increases in cerebrospinal fluid pressure. Deteriorating inner ear function generally requires tympanotomy and patching of the round or oval window.[2]

4.7. Trauma

Blunt trauma can result in sensorineural loss due to concussive forces of the inner ear fluids, which may cause a shearing affect on the cochlear organ of Corti. Blunt trauma may also lead to longitudinal or transverse temporal bone fracture.

The longitudinal type is most common (80%). It is usually caused by a blow to the temporal parietal region. Hearing loss is typically conductive and associated with tympanic membrane (TM) perforations and blood in the middle ear space.

A transverse fracture occurs following a blow to the occipital or frontal region (Figure 2). Fractures of this type usually run through the inner ear. If hearing is preserved to some degree, the most common reason for a conductive hearing loss is an ossicular injury, typically due to separation of the incudal stapedial joint and/or incus dislocation.[2]

Penetrating trauma typically causes sensorineural or mixed hearing loss. These injuries are usually due to gunshot wounds that upon impact cause significant temporal bone fractures.

4.8. Tumors

Most tumors of the inner ear are benign, although malignant tumors such as squamous cell carcinoma, sarcomas, adenoid carcinoma, and metastasis rarely occur. Benign bony tumors, including fibrous dysplasia and Paget disease, are also rare.[2]

The most common tumor that causes sensorineural hearing loss is an acoustic neuroma. Eighty percent of tumors arising in the cerebellopontine angle are acoustic neuroma. This is a benign tumor that usually arises from the Schwann's cells of the vestibular portion of the eighth cranial nerve. The most common complaint is an asymmetric progressive sensorineural hearing loss, which typically begins in the high frequencies and progresses to involve lower frequencies. Other symptoms include unilateral tinnitus, disequilibrium, dizziness, or headaches.[8]
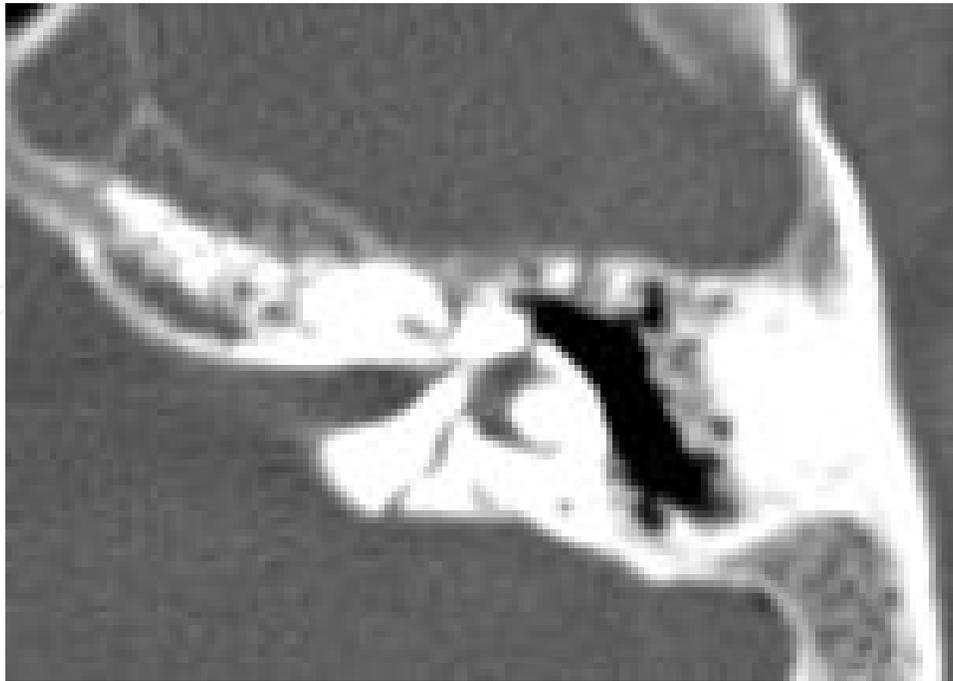
Figure 2. Transverse temporal bone fracture

4.9. Endocrine disorders

Various metabolic abnormalities have been known to either cause or be associated with sensorineural hearing loss. Thus, an evaluation of an unexplained sensorineural hearing loss should involve a complete laboratory evaluation to include the following: complete blood count with differential, blood sugar, thyroid function tests, and serologic test for syphilis.[2]

Diabetes has been associated with an approximately twofold increase in the prevalence of low- and midfrequency hearing impairment in adults; this might relate to the impact of diabetes on the vascular or neural components of the inner ear.[8]

Anemia or a white blood cell dyscrasia may lead to sensorineural hearing loss by an unknown mechanism that may involve decreased oxygenation, microblockage of vessels, or infection.

4.10. Autoimmune hearing loss

The autoimmune inner ear disease may be limited just to the ear, or it may be part of an overall systemic problem. Approximately one third of patients will have evidence of systemic autoimmune disorder such as Wegener granulomatosis, Cogan syndrome, rheumatoid arthritis, systemic lupus erythematosus, or polyarteritis nodosa.[2]

Autoimmune hearing loss is usually sensorineural, bilateral, and asymmetric, which is either fluctuating or progressive in nature.

The treatment choice for patients with autoimmune inner ear disease is high-dose glucocorticoids for up to 4 weeks. This often results in significant recovery of hearing.[2]

Cytotoxic medications such as cyclophosphamide, methotrexate, or azathioprine may be used if corticosteroids fail.

4.11. Ototoxicity

A great number of medications are known to cause damage to the ear. Anti-inflammatory, antibiotics, loop diuretics, antimalarials, chemotherapeutic agents, and ototopical medications may cause ototoxicity (Table 1).[8]

The hearing loss caused by antibiotic or chemotherapeutic agents usually begins at high frequencies, and with continued medication use, the hearing loss will become more pronounced and may even continue to worsen for a time after the drug is discontinued.

Several antibiotics cause ototoxicity. All oral aminoglycosides are ototoxic, and this effect is due to hair cell death from apoptosis. Different types of aminoglycosides show different patterns of ototoxicity. Streptomycin and gentamicin are primarily vestibulotoxic. Neomycin, amikacin, and tobramycin are primarily cochleotoxic.

Ototopical aminoglycoside drops have the potential to cause ototoxicity. However, it is believed that these medications do not have their normal ototoxic effect because the inflamed mucosa within the ear prevents significant drug penetration into the oval and round windows. Other oral antibiotics that can cause ototoxicity include erythromycin and tetracycline.

Medications		Effects
Antibiotics	**Aminoglycosides**	Vestibulotoxic
	Streptomycin	Vestibulotoxic
	Gentamicin	Cochleotoxic
	Neomycin	Cochleotoxic
	Amikacin	Vestibulotoxic and cochleotoxic
	Tobramycin	Cochleotoxic
	Macrolides	Cochleotoxic (synergism with aminoglycosides)
	Erythromycin	
	Glycopeptides	
	Vancomycin	
Anti-inflammatory	Aspirin (salicylates)	Cochleotoxic
Loop diuretics	Furosemide	Cochleotoxic (synergism with aminoglycosides)
Antimalarials	Quinine	Cochleotoxic
	Chloroquine	Cochleotoxic
Chemotherapeutic agents	Cisplatin	Cochleotoxic
Ototopical drops	Aminoglycoside drops (gentamicin and neomycin)	Potential ototoxicity in tympanic membrane perforation

Table 1. Medications related with ototoxicity

Many chemotherapeutic agents are known to cause hearing loss. These include cisplatin, 5-fluorouracil (5-FU), bleomycin, and nitrogen mustard. The worst ototoxicity occurs with cisplatin, which damage the outer hair cells of the basal turn of the cochlea, causing bilateral, symmetric, and high-frequency hearing loss.

High-dose aspirin (6–8 g/day) or other salicylates can cause a flat mild-to-moderate sensorineural hearing loss, but this is reversible with discontinuation of the drug.

Antimalarial medications such as quinine and chloroquine may also cause sensorineural hearing loss and tinnitus, but similar to salicylates, these effects are usually reversible. This is also true for high-dose nonsteroidal anti-inflammatory agents. Loop diuretics are an additional cause of temporary hearing loss and tinnitus.[2]

Heavy metals, including lead, mercury, cadmium, and arsenic, can all lead to hearing loss.

4.12. Neurogenic

Several neurologic disorders may cause sensorineural hearing loss: cerebrovascular accident or transient ischemic attack, Arnold–Chiari malformations (may stretch the auditory vestibular nerve, thereby causing hearing loss and/or vestibular complaints), and multiple sclerosis (can initially present as a sudden sensorineural hearing loss and/or vertigo). [2]

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