



Workplace Safety and Insurance
Appeals Tribunal

Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

Hearing Loss and Tinnitus

Discussion paper prepared for

The Workplace Safety and Insurance Appeals Tribunal

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This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

HEARING LOSS AND TINNITUS

Types of Hearing Loss

Hearing loss in any individual at any given time is a combination of the following factors:

- a. Congenital (what they were born with)
- b. Acquired (what they developed as the result of pathologic exposures or processes during their lifetime)

Conceptually three types of hearing loss exist:

1. Sensorineural
2. Conductive
3. Mixed (a combination of sensorineural and conductive hearing loss)

A sensorineural hearing loss exists with injury to the cochlea or cochlear nerve. This is the type of hearing loss that is found in routine unprotected daily exposure to loud noise potentially injurious to hearing in the occupational work force.

A conductive hearing loss occurs when there is some interference of sound transmission or vibration due to pathology involving the external and/or middle ears. This type of loss might be found in an individual, for example, with a large tympanic membrane (TM) perforation where mechanical vibrations along the ossicular chain are dampened.

A mixed hearing loss occurs when both a sensorineural and conductive hearing loss are present at the same time. For example an individual with a large TM perforation who received topical antibiotic ear drops for the treatment of a middle ear infection that caused inadvertent toxicity to the inner ear in addition (i.e. topical ototoxicity).

Common Causes for Hearing Loss

Conductive Hearing Loss	Sensorineural Hearing Loss
1. External otitis (acute and chronic)	1. Occupational or Noise Induced Hearing Loss (NIHL)
2. Wax	2. Presbycusis
3. Exostoses/osteomas	3. Meniere's Disease
4. Acute Otitis Media	4. Ototoxicity (Systemic and Topical)
5. Otitis Media with Effusion	5. Cochlear Otosclerosis
6. TM perforations	6. Trauma
7. Chronic Suppurative Otitis Media (CSOM) a. Safe or mucosal CSOM b. Cholesteatoma	7. Acoustic neuromas (vestibular schwannomas)
8. Otosclerosis	8. Sudden Sensorineural Loss

Common Causes for Sensorineural Hearing Loss

1. Noise Induced Hearing Loss

According to the 1990 Noise and Hearing Loss Consensus Conference, "Noise Induced Hearing Loss (NIHL) results from damage to the ear from sounds of sufficient intensity and duration that a temporary or permanent sensorineural hearing loss is produced. The hearing loss may range from mild to profound, may result in tinnitus (unwanted head noise) and is cumulative over a lifetime". Occupational NIHL and presbycusis (degenerative hearing from aging change) represent the two most common causes of sensorineural hearing loss in society today.

Two types of noise exposure are associated with NIHL: transient and continuous.

Impact (i.e. the collision of two solid objects as might occur in a forge plant) or impulse (i.e. the sudden noise of an explosion) noise are examples of transient noise where there is a rapid rise in sound pressure levels and a very quick decline over 0.2 sec. Constant or steady state noise by comparison remains relatively constant and lasts longer although fluctuations in sound intensity may occur. Although short lived, most impact/impulse noise typically has peak intensity levels much higher than found in steady state noise exposure. All things being equal most noise in industry however is a combination of primary continuous and superimposed impact type noise.

When susceptible, unprotected ears are exposed to loud noise potentially injurious to hearing, the inner ear seems to react in one of three ways: by adapting to the noise (i.e. the inner ear seems to "toughen" in some individuals), by developing a transient threshold shift (TTS), or a permanent threshold shift (PTS).

TTS refers to a transient sensorineural hearing loss lasting hours to a few days. Hearing thresholds are depressed until the metabolic activity in the cochlea recovers. For this reason workers ideally should be out of noise for at least 24 hours if not 48 hours prior to audiometric testing to avoid the effects of TTS on hearing.

PTS refers to a permanent loss of sensorineural hearing which is the direct result of irreparable injury to the organ of Corti. Noise induced deafness generally affects hearing between 3000-6000 Hz with maximal injury centering around 4000 Hz initially, an important point to remember.

a. The 4000 Hz Audiometric Dip

The 4000 Hz “notch” or “dip” in sensorineural hearing has been a classic finding in NIHL over the years. Some noise sources however such as gunfire may maximally affect hearing at 6000 Hz. Noise exposure from chipping machines and jackhammers characteristically damage the higher frequencies severely before affecting the lower frequencies.

Why the 4000 Hz frequency appears more affected than other frequencies continues to generate some controversy. There is good pathologic evidence however that demonstrates maximal cochlear hair cell loss in the tonal areas where the 4000 Hz hair cells normally reside in both animals and humans.

b. Individual Susceptibility to Noise

Individuals vary in their susceptibility to noise and the damage it may cause to the cochlea.

To date, susceptibility has not been shown to be dependent on gender (males vs. females), skin colour, any known diseases, mental attitude towards the noise, chemical exposure, pre-exposure hearing loss or smoking.

Of interest, one universal finding is that on average hearing threshold levels (HTLs) in the right ear are better than the left ear by about 1dB. This however is of no practical importance clinically.

c. Asymmetry of Sensorineural Hearing Loss from Noise

In general terms NIHL usually demonstrates a 4000 Hz dip. It should also be symmetric bilaterally.

Whether one ear is more resilient to noise in the same individual (the so-called “tough vs. tender” ear argument) while of academic curiosity is not based on any known pathologic basis to date. Nevertheless some individuals exposed to noise not infrequently demonstrate some asymmetry to their hearing loss. This is however usually related to the fact that one ear receives a greater exposure to the noise

than another. For example, truck drivers in North America not infrequently have a greater degree of hearing loss in their left ear (when the window is rolled down the left ear would be exposed to more sound from the engine). Those who fire guns often demonstrate a greater degree of hearing loss in the ear closest to the barrel (the left ear in a right-handed shooter) because that ear would be closest to the explosion and the other ear would be protected by a “head shadow”.

Please remember that when an asymmetric sensorineural hearing loss exists steps often need to be taken to exclude pathologic causes (i.e. tumours, other inner ear disorders, etc.)

d. Basic Facts Concerning Noise Induced Hearing Loss (NIHL)

The following statements tend to reflect what is agreed upon by the majority of scientists and physicians who deal with NIHL.

1. Noise exposure can produce a permanent hearing loss that may affect speech communication.
2. Noise induced hearing loss (NIHL) may produce a temporary threshold shift (TTS), permanent threshold shift (PTS) or a combination of both.
3. A PTS is caused by destruction of certain inner ear structures that cannot be replaced or repaired.
4. The amount of hearing loss produced from a given noise exposure varies from person to person.
5. NIHL initially affects higher frequency hearing (3-6 kHz range, for nearly all occupational exposures) than those frequencies essential for communication (i.e. 500, 1000 and 2000 Hz).
6. Four major factors determine the effects of exposure to noise overall:
 - Overall noise levels.
 - Spectral composition of noise.
 - Duration and distribution of exposure during a typical workday.
 - Cumulative noise exposure over days, weeks and years.
7. Exposure to noise:
 - a. Daily noise exposure (8hrs) > 90 dB for over 5 years (or equivalent) causes varying degrees of hearing loss in susceptible individuals.

- b. Amount of NIHL is related to the exposure level (i.e. the intensity of the sound) i.e. the 3 dB doubling rule (increase sound levels by 3 dB and you decrease by 1/2 the safe unprotected exposure time to noise).

i.e. 90 dB for 8 hours without wearing hearing protection 94 dB for 4 hours without wearing hearing protection 97 dB for 2 hours without wearing hearing protection, etc.
 - c. NIHL is a decelerating process; the largest changes occur in the early years with progressively smaller changes in the later years (so-called Corso's theorem). See section on Presbycusis.
 - d. NIHL first affects hearing in the 3-6 kHz range, for nearly all occupational exposures; the lower frequencies are less affected.
 - e. Once the exposure to noise is discontinued, there is no substantial further worsening of hearing as a result of noise unless other causes occur.
 - f. Previous NIHL does not make the ear more sensitive to future noise exposure.
 - g. Continuous noise exposure over the years is more damaging than interrupted exposure to noise which permits the ear to have a rest period.
8. Where the worker is exposed to sound level of 90dB or greater over an eight-hour work day, a number of controls may be implemented to minimize noise exposure, including
- engineering controls (required where feasible);
 - administrative controls (limiting employee exposure duration to the above mentioned time schedule); and
 - personal protective hearing devices (ear plugs, ear muffs, etc.).

2. Presbycusis

Progressive age related sensorineural hearing loss is often called presbycusis. In susceptible individuals the early effects of presbycusis are occasionally seen around age 40. Around age 55-60, an individual's hearing starts to worsen at a faster rate. For this reason a correction factor for presbycusis is applied in occupational hearing loss claims depending on the jurisdiction (in the Province of Ontario, for example, a correction factor of 0.5 dB/ year of age > 60 years) is typically applied for presbycusis.

The pathologic basis for presbycusis appears to be one of gradual devascularization of the cochlea and loss of functioning hair cells. Secondary to hair cell loss one can often see progressive neuronal dropout along the cochlear nerve. The majority of changes histopathologically are noted in the basal turn of the cochlea where the high frequency hair cells and their corresponding cochlear nerve neurons are found. Nevertheless the

changes seen in presbycusis are typically non-specific and can also be seen in a vast number of pathologies including the effects of noise upon the inner ear.

Clinically hearing loss from presbycusis appears to be an accelerating process unlike hearing loss in NIHL. In this regard the effects of aging in the absence of other factors cause a loss of hearing at all frequencies whose rate of growth becomes more rapid as age increased (especially after 60 years): an important point to remember in this context.

Unfortunately, there is no specific treatment available that will prevent age related hearing loss at present. To a large degree hearing loss with age is genetically primed; in other words the hearing your parents had as they aged is often passed on to you.

a. Controversies between presbycusis and NIHL

In the adjudication process of an occupational NIHL claim it is often difficult to separate the total amount of hearing loss from noise and age related change.

For example, not everyone as they age will experience age-related presbycotic change (changes from presbycusis are variable with some individuals experiencing greater degrees of age related change than others).

Moreover, exposure to high level noise early on may produce hearing loss more rapidly than aging such that the aging process has a negligible effect (i.e. the more that has been lost early on, the less there is to lose later on).

b. Dobie's and Corso's Theorems

The effects of noise exposure and aging on hearing when not combined are reasonably well-understood. When the two processes are combined the resultant pathology and its effects upon hearing are not as well understood.

Although it seems logical to “subtract” the age-related effects from the total hearing loss in order to quantitate the amount of hearing loss due to noise, this is really quite simplistic when one considers that aging effects and noise exposure effects can at times be practically indistinguishable for the most part audiometrically.

Because compensation claims have required some consideration of presbycusis and its role in the total hearing loss of an individual various correction factors have been applied.

Dobie's theorem states that the total hearing loss from noise and age are essentially additive (this is the theory put into practice when a standard correction factor for age after 60 years is applied in the Province of Ontario).

Corso's theorem on the other hand states that any correction for age should be based on a variable ratio (as individuals age the assumption is that the effects of presbycusis

variably accelerate by decade). This certainly generates a more complicated mathematical model but probably more closely approaches what is happening physiologically.

Nevertheless the quantification of hearing loss attributable to age when occupational NIHL is present is really quite a complex phenomenon.

3. Menière's Disease

This is an inner ear disorder characterized by episodes of vertigo (an illusion of movement) lasting minutes to hours, fluctuating hearing loss and tinnitus (unwanted head noise). Frequently there is a sense of pressure or fullness in the ear during attacks.

Usually one ear is involved initially although over time the other ear becomes affected in nearly 50% of cases. The hearing loss is typically a low-frequency sensorineural loss that fluctuates initially often reverting close to normal between attacks in the early stages. Over time the severity of the hearing loss progresses. Occasionally both a low-frequency and a high-frequency loss occurs but not usually the type of high frequency loss seen following noise exposure.

Pathologically there is distension of the inner ear membranes by excess fluid. It is not known if this results from excess production or inadequate drainage of fluid. When the distended membranes rupture the resulting admixture of inner (endolymph) and outer (perilymph) fluids causes electrolyte disturbances (i.e. the so-called Na⁺ - K⁺ intoxication theory) leading to dizziness. After its collapse the membrane heals and the cycle recommences. However the natural history is enigmatic with unpredictable periods of exacerbation and remission.

Treatment is medical in most cases involving a low salt diet and diuretics and vestibular sedatives. Currently there is research into the application of intermittent pressure / pulses to the inner ear via the eardrum (i.e. the Meniett Device). When vertigo is incapacitating the balance function of the inner ear may be attenuated or ablated by the trans-tympanic instillation of gentamicin with relative preservation of hearing. In the last resort the whole inner ear can be destroyed surgically by a procedure called a labyrinthectomy. Unfortunately hearing cannot be preserved in this procedure.

4. Cochlear Otosclerosis

Otosclerosis usually results in a conductive hearing loss from stapes footplate fixation due to new bone growth in this area. (See Appendix B, Middle Ear) Nevertheless the otosclerotic foci can involve any part of the hard bone (otic capsule) surrounding the

inner ear. When the foci primarily affect the cochlea the patient may present with a chronic progressive sensorineural hearing loss in one or both ears. If the footplate as well as the cochlea is involved then a mixed (conductive and sensorineural) loss might result.

The diagnosis of cochlear otosclerosis is usually made when a family history of otosclerosis exists and other rare causes for a chronic progressive loss can be excluded. The presence of a pink-flamingo hue to the middle ear on otoscopy (so-called Schwartze's sign), absent stapedial reflexes on audiometry and bone density changes involving the surrounding bone of the inner ear best appreciated on high-resolution CT scanning also helps in the diagnosis. As otosclerosis is a lifelong condition, the sensorineural hearing loss from cochlear otosclerosis is often superimposed on hearing loss from advancing age (i.e. presbycusis). The sensorineural hearing loss from otosclerosis progresses at an average rate of 5.5 dB/decade, higher than that seen in presbycusis.

Although no medical treatment can ever reverse the sensorineural hearing loss, treatment with oral sodium fluoride may minimize and possibly stabilize an individual's hearing.

5. Trauma

Physical injury to the ear is usually the result of blunt trauma in the circumstances of a significant head injury. Most patients suffering deafness by this mechanism will have had at least transient unconsciousness and have been admitted to hospital.

When physical trauma is severe enough to cause a temporal bone fracture (the temporal bone is the larger part of the skull bone that houses all the structures of the ear), two types of fractures occur; longitudinal and transverse.

In general terms longitudinal fractures are much more common and tend to result in a fracture line through the roof of the middle ear and ear canal. Bleeding from the ear is not unusual. The hearing loss noted is usually conductive and usually arises from discontinuity of the ossicular chain although any combination of conductive and sensorineural hearing loss can occur. The pathognomic sign of a longitudinal temporal bone fracture is the so-called "step deformity" in the deep ear canal. Exploration of the middle ear surgically with correction of the ossicular chain or insertion of a prosthesis is called an ossiculoplasty.

Transverse fractures of the temporal bone occur when the fracture lines run directly through the hard bone of the otic capsule. A fracture through this bone (which is the hardest bone in the body) implies the force of the injury was severe and often incompatible with survival. If the individual survives there is usually complete loss of hearing and vestibular function on the involved side. Facial paralysis from an injury to the facial nerve (a nerve that runs in close approximation to the inner ear) typically

occurs. On examination blood is usually seen in the middle ear behind the ear drum (a hemotympanum).

Trauma however can also be penetrating (i.e. a Q-tip through the ear drum), thermal (i.e. a welder's spark down the ear canal), electrical (accidental electrocution), explosive and even implosive (i.e. professional bell and scuba divers who try to "pop" their ears too vigorously). The resultant hearing loss depending on the mechanism can be conductive, sensorineural or a combination thereof.

When a column of air is forced down the ear canal in an explosive fashion (i.e. a slap to the ear, a bomb blast, etc.) the TM often ruptures in its central portion and the loss is usually conductive until the drum repairs itself. Persistence of a conductive loss after the TM has healed would suggest continued problems with the ossicular chain.

6. Ototoxicity

Ototoxicity is defined as the tendency of certain substances to cause functional impairment and cellular damage to the tissues of the inner ear, especially to the cochlea and the vestibular apparatus. Toxic substances can be delivered systemically either via the blood stream or topically through perforations/ventilation tubes in the ear drum.

Aminoglycoside antibiotics are powerful weapons in the treatment of certain bacterial infections. Unfortunately these antibiotics can cause varying degrees of cochlear, vestibular and renal toxicity. Careful and regular monitoring of auditory function (especially in the ultra high frequencies > 8000 Hz) and serum antibiotic levels may help the physician predict when ototoxic effects are occurring. The prolonged use of topical aminoglycoside antibiotics to treat middle ear pathology in the presence of a TM perforation is not without some risk which should always be kept in mind.

Antimalarial drugs such as quinine and chloroquin unfortunately have ototoxicity as a side effect if taken in excess. Reversibility however is relatively common once the medications have been discontinued. Platinum- based chemotherapeutic drugs (i.e. cisplatin) for the treatment of malignancy (cancer of the breast, lung, etc.) have been especially well documented to cause cochleotoxicity. Of interest the first course of cisplatin often demonstrates which patients are vulnerable to cochlear damage and what may happen in future if continued treatment courses are required.

7. Acoustic Neuroma (AN)

This is a pathologic misnomer since this tumour is strictly a schwannoma of the vestibular nerve. (See Appendix A, Medical Terminology) It arises in the internal auditory canal where nerves run between the inner ear and the brain. Pressure

on or devascularization of the acoustic nerve or inner ear causes hearing loss, usually a high-frequency loss with reduced speech discrimination. Usually the loss is progressive but occasionally may be sudden. ANs are overwhelmingly unilateral, but in association with a rare genetic disorder, neurofibromatosis type 2, they may be bilateral.

Although benign this tumour has serious health implications for the patient and is sought for by otolaryngologists when there is an undiagnosed asymmetry in the hearing. The Auditory Brainstem Response (ABR) is used as a screening test while a gadolinium enhanced Magnetic Resonance Imaging (MRI) study is the gold standard investigation.

8. Sudden Sensorineural Hearing Loss

This diagnosis is given to the individual who experiences a sudden hearing loss in one ear. The extent of the loss varies from a partial loss at one frequency right up to a profound loss at all frequencies with a profound discrimination loss. Patients can sometimes identify the moment of occurrence or wake with it or appreciate it only when they go to use the phone.

These sudden losses are considered by the profession to be most likely viral in origin. Many recover spontaneously and do so within six months. Severe losses and those associated with vertigo have a worse prognosis. Patients if seen early enough i.e. within 2 - 3 weeks are usually treated with a tapering schedule of Prednisone (a potent steroids). The addition of oral anti-viral therapy, intratympanic (IT) steroid injections and/or hyperbaric O₂ treatments also have their proponents.

Audiology

The formal recording of an individual's hearing forms the basis of the audiogram. For the purposes of compensation, the most reliable audiograms will likely be performed by an audiologist who typically has a masters' degree or doctorate in audiology.

Sound intensity is measured in what is called a decibel (dB). The audiometer used to test hearing has been especially calibrated so that each frequency of sound tested for hearing has been normalized where 0 dB represents the lowest intensity of sound that the majority of people with normal hearing can hear from large population studies. It is important to recognize that 0 dB does not mean an absence of sound. In fact some people with "superhuman" hearing can even hear sound intensities less than this relative value of sound pressure. Another concept to appreciate is that the dB scale is a logarithmic (an exponentially increasing) rather than arithmetic (linear increasing) scale which makes it difficult to apply a percentage hearing loss to any individual. For example the difference between 10 and 40 dB in sound pressure intensities is not a

4x's increase but actually an increase by a factor of 1,000 or 10^3 in sound pressure intensities!

The following audiometric tests form the foundation for most assessments of hearing. These include what is called conventional audiometry and impedance (immittance) testing with tympanometry:

Conventional Audiometry

a. Pure Tone Audiogram (PTA)

An individual's threshold hearing to pure tones at different frequencies (250 - 8000 Hz) is performed.

Air conduction (AC) thresholds are delivered via headphones and the individual is asked to respond to the sound of the lowest intensity (in dB) they hear at the frequency being tested. When a conductive hearing loss is suspected the ear canal and inner ear mechanisms for transmission of sound energy to the inner ear are bypassed by placing a bone vibrator over the mastoid which directly stimulates the inner ear. Bone conduction (BC) thresholds are obtained in this fashion. In complex situations (mixed hearing losses) it may also be necessary to "mask" the ear not being tested (to prevent crossover of sound to the other ear).

In a pure sensorineural hearing loss, AC thresholds should be the same as BC thresholds. In a pure conductive hearing loss BC thresholds will be better than AC thresholds. In a mixed hearing loss, elements of both sensorineural and conductive hearing loss are present. When AC thresholds are better than BC thresholds in a tested ear this usually implies an exaggerated hearing loss is present.

The pure tone audiogram forms the basis for assessing if an individual qualifies for Workplace Safety and Insurance Board (WSIB) benefits in the Province of Ontario. A weighted pure tone average from frequencies at 500, 1000, 2000 and 3000 Hz is required for this determination from AC thresholds if a pure sensorineural hearing loss is present or from BC thresholds if any conductive element to hearing loss is present. (See Appendix C, Question 1 for an explanation why the 4,000 Hz frequency is not used in occupational hearing loss claims).

b. Speech Reception Threshold (SRT)

Complex words with equal emphasis on both syllables (so called spondaic words such as "hotdog", "uptown", "baseball" etc.) are given an individual at the lowest intensity they can hear. As a general rule the SRT value should roughly equal the pure tone average in the speech frequencies at 500, 1000 and 2000 Hz. If there is a significant discrepancy this could imply an exaggerated hearing loss is present as well.

c. Speech Discrimination Scores (SDS)

A list of phonetically balanced single syllable words (these are words commonly found in the English language in everyday speech such as “fat”, “as”, “door” etc.) are presented to an individual at 40 dB above their speech reception threshold (SRT) in the ear being tested. Most individuals with normal sensorineural hearing should get over 80% of the words correct at this level. When speech discrimination scores are especially poor this implies that there may be a lesion involving the cochlear nerve (i.e. acoustic neuroma) and that further investigation may be necessary.

Impedance Testing with Tympanometry

In this test a probe is placed into the ear canal that both emits a sound and can vary pressure within the canal which causes the ear drum to move.

a. Middle ear pressure measurements (acoustic immittance)

This tells whether the pressure in the middle ear is within normal limits and provides some indirect measurement of Eustachian tube function. Pressures between -100 to +100 are considered to be normal. In general terms if the Eustachian tube is functioning normally pressure on both sides of the ear drum should be similar (i.e. a “0” reading).

b. Stapedial reflex testing

Stiffening of the ear drum from loud noise occurs when the stapedius muscle contracts in the middle ear. Most individuals with normal sensorineural hearing will exhibit a reflex at 70-80 dB above their hearing threshold level at the frequency being tested.

Absent stapedial reflexes are usually seen in middle ear pathology such as otitis media with effusion or otosclerosis. When reflexes occur at less than a difference than 70-80 dB above hearing threshold at the frequency tested this is indirect evidence of a phenomenon called recruitment which is usually seen in cochlear pathology (i.e. Meniere’s disease).

c. Tympanometry and Types of Curves

If one measures how the ear drum moves when pressure is changed in the external ear canal from negative to positive a series of curves can be attained. Clinical correlation has demonstrated that the following curves usually are seen in these pathologies (Figure 1):

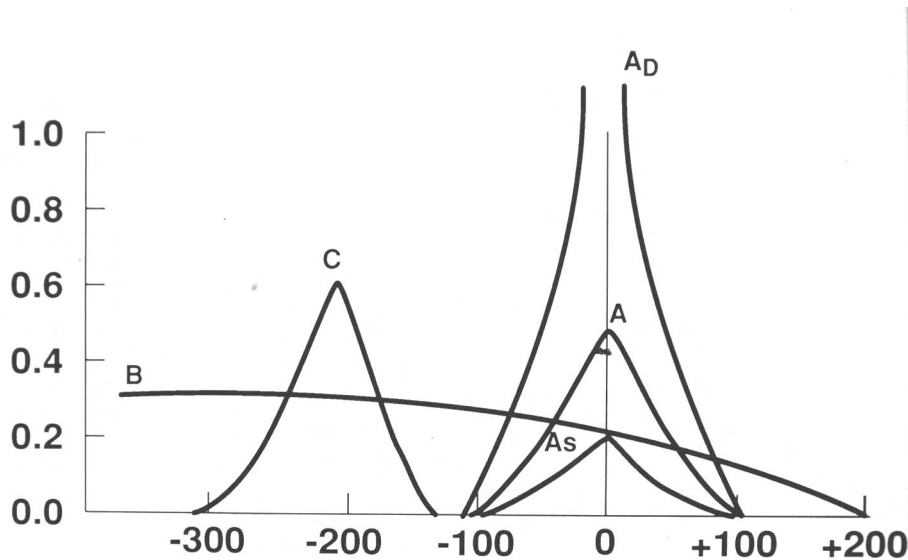


Figure 1 - Schematic representation of tympanometry curves

- A - Normal
- As- Otosclerosis or ossicular fixation
- AD- Ossicular discontinuity
- C- Eustachian tube dysfunction
- B- Middle ear atelectasis (the TM is rigidly fixed to the middle ear) or otitis media with effusion (glue ear)

If a TM perforation is present it is impossible to obtain a “seal” of the middle ear or a tympanogram.

Evoked Response Audiometry

The ability to measure minute electrical potentials following sound stimulation of the cochlea (i.e. evoked response) provides us with information concerning the cochlea (i.e. electrocochleography), the cochlear nerve and brainstem (i.e. auditory brainstem response or ABR) and higher cortical auditory pathways (i.e. threshold evoked potentials, cortical evoked response audiometry). An experienced tester, typically an audiologist, is required to perform these technically demanding tests.

The indications and relative importance of these tests are described below.

1. Electrocochleography (ECoG) - This test measures electrical activity in the cochlea during the first 2 msec of cochlear stimulation. ECoG’s chief value lies in its ability to demonstrate wave 1 of the auditory brainstem response (ABR) and whether waveform morphology is suggestive of changes thought to occur in endolymphatic hydrops, the pathophysiologic substrate of Menière’s disease.

2. Auditory Brainstem Response (ABR) - This term is synonymous with the term brainstem evoked potential (BEP) or the brainstem evoked response audiogram (BERA). This test measures electrical waveforms obtained in the first 10 msec from cochlear stimulation. Waveform morphology is thought to arise from the cochlear nerve and the various relay stations in the brainstem the electrical response has to travel through. Changes in waveform morphology and latency of the ABR can be quite helpful in the assessment of an individual with an asymmetric sensorineural hearing loss if an acoustic neuroma is suspected.
3. Threshold Evoked Potentials (TEP) - These electrical waveforms are usually identified between 50 - 200 msec following cochlear stimulation and are thought to represent cortical pathways of electrical activity. One advantage of TEP testing is that the electrical waves can provide us with information concerning the actual threshold at a certain frequency an individual hears and as such gives us some objective measurement of an individual's hearing that is not dependent on a voluntary response. The test is often indicated in an individual if there are concerns regarding an exaggerated hearing loss.

A Quick Primer for Understanding Audiograms

General Principles

- Air conduction (AC) determines the severity of hearing loss.
- Bone conduction (BC) determines the inner ear portion of hearing.
- AC and BC should be the same in a sensorineural hearing loss.
- If AC thresholds are greater than BC thresholds this usually implies there is a conductive component to hearing loss from pathology involving the external or middle ear.
- If BC thresholds are greater than AC thresholds this would typically suggest an exaggerated hearing loss or malingering.

Hearing Loss Classification Based Upon dB level

- 26-40 dB = mild
- 41-55 dB = moderate
- 56-70 dB = moderate-severe

- 71-90 dB = severe
- 91+ dB = profound

Audiogram Nomenclature*

	Right	Left
Air Conduction	O	X
Masked Air Conduction	Δ	Δ
Bone Conduction	<	>
Masked Bone Conduction	[]

* When reviewing an audiogram it is always important to carefully check each audiogram legend before interpretation as variations in the symbols used might exist in different testing facilities.

Common Audiometric Configurations in Certain Disease Pathologies

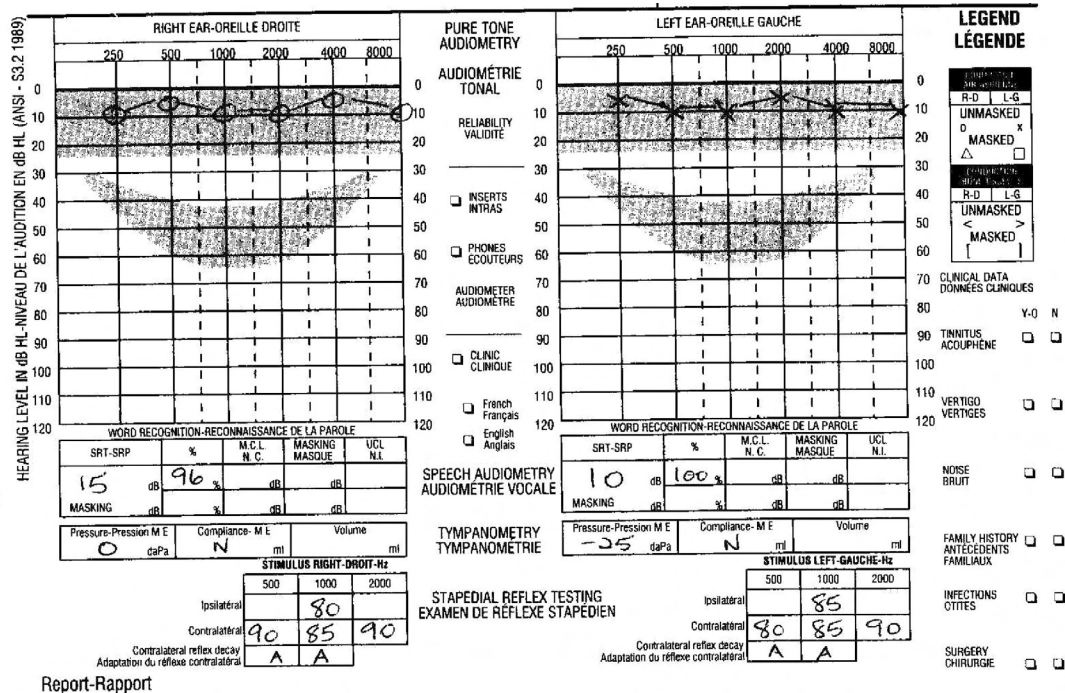


Figure 2 - Normal audiogram

1. Noise Induced Hearing Loss

In its classic presentation a notched sensorineural hearing loss is noted at 4000 Hz that should be relatively symmetric. Middle ear pressures should be normal, stapedia reflexes present and a normal tympanogram noted.

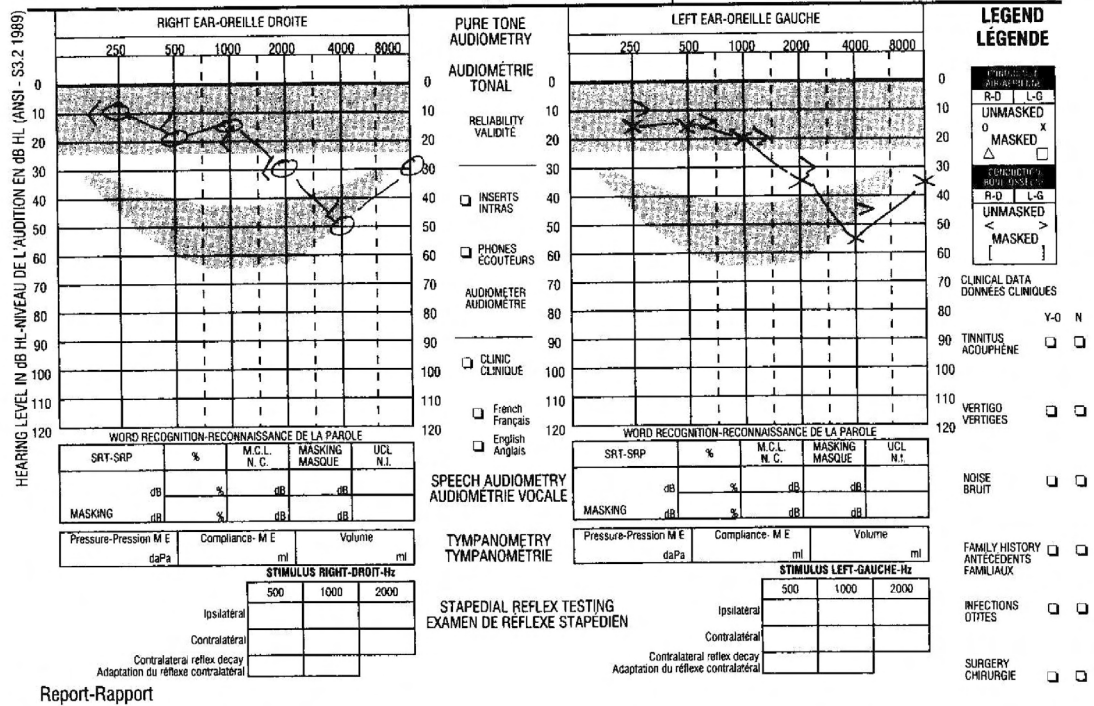


Figure 3 - Audiogram in NIHL case

2. Presbycusis

Age related hearing loss typically presents with a bilateral symmetrical high frequency sensorineural hearing loss.

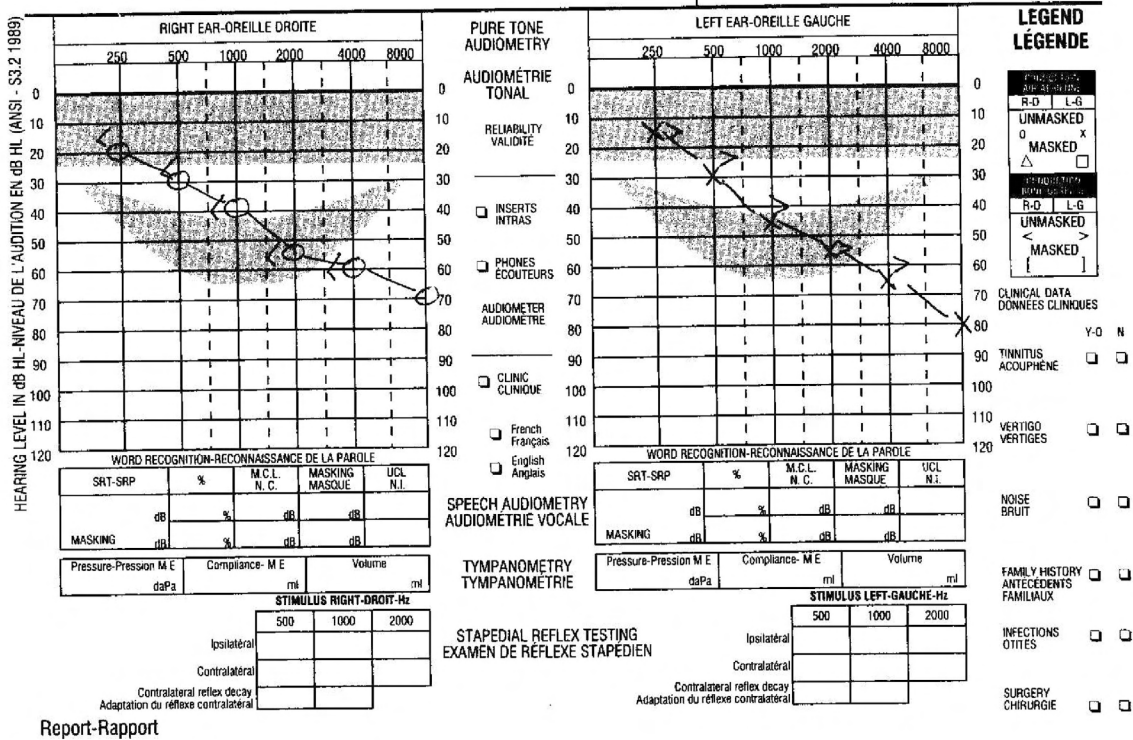


Figure 4 - Audiogram in presbycusis case

3. Menière's Disease

A low frequency sensorineural hearing loss is pathognomonic for this condition. Fluctuation in hearing is often noted on sequential audiometry.

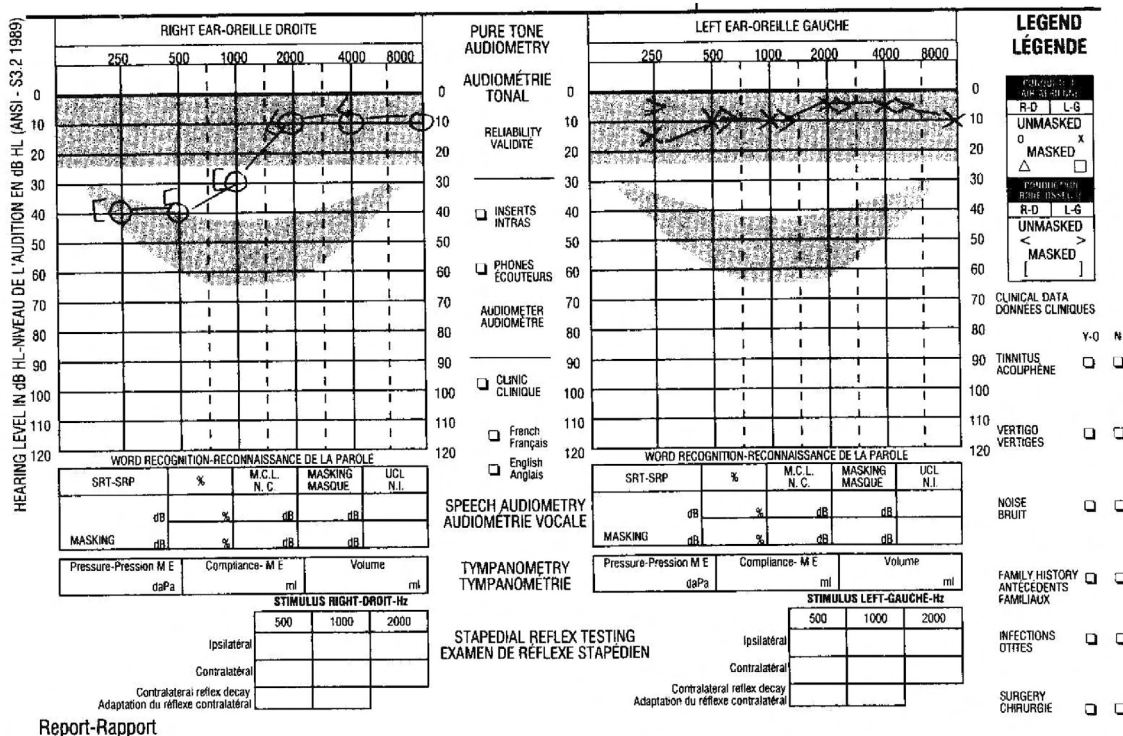


Figure 5 - Audiogram in Menière's Disease (Right side affected)

4. Congenital Hearing Loss

Individuals usually present with hearing loss early in life. A mid-frequency hearing loss is sometimes noted. This is often called a “cookie-bite” audiogram.

5. Exaggerated Hearing Loss

There are often numerous discrepancies noted such as the presence of acoustic reflexes at levels below volunteered pure tone thresholds, discrepancies between volunteered pure tone average and speech reception thresholds etc. Repeat testing and evoked response audiometry is often helpful.

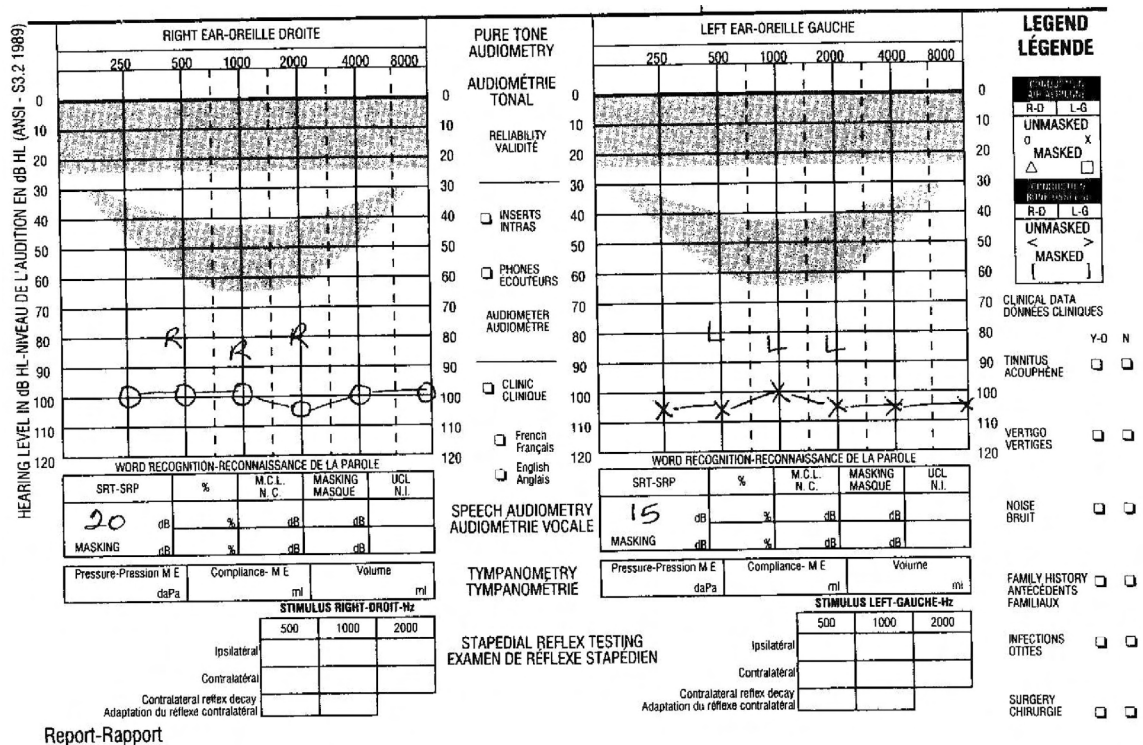


Figure 6- Audiogram in exaggerated hearing loss case

Tinnitus

Tinnitus (from the Latin, “to ring a bell”) by definition is unwanted head noise. It is a common problem that affects an estimated 20% of the population at any given time. Tinnitus can be either subjective or objective.

Subjective tinnitus can only be appreciated by the affected individual, is usually associated with a sensorineural hearing loss of some type and is the most common type noted. It is usually described as a constant sound (i.e. ring, buzz, hum, etc.) that is worse in the absence of competing background noise (i.e. at night).

Objective tinnitus, on the other hand, is quite rare but by definition is a noise that can be appreciated by an observer. Usually the tinnitus here is described as being pulsatile or clicking. Causes for objective tinnitus include the presence of vascular middle ear

tumours (i.e. glomus tumours), aneurysms near the inner ear / skull base or from the repetitive contractions of the middle ear muscles (so-called middle ear myoclonus).

Regardless of cause tinnitus can be quite disturbing for many individuals and can certainly affect their well-being.

Treatment for tinnitus includes the use of more pleasurable competing background noise (i.e. keeping the radio on at night, wearing a Sony Walkman, etc.), aids to improve hearing or masking devices that dampen the unwanted head noise. Tinnitus maskers are noise generators worn like a hearing aid that produces a sound of similar frequency to an individual's tinnitus. When tinnitus is severe enough to affect an individual's well-being a trial of pharmacological treatment is generally recommended. Certain anti-depressants (especially the tricyclic or SSRI classes) and anti-convulsants (medications used for seizure control) have been effective for some individuals. New therapies involving tinnitus retraining strategies are currently under investigation.

a. Tinnitus in NIHL Claims

In the context of a NIHL claim, tinnitus not infrequently is noted. Compensation is available for a non-economic loss (NEL). The problem with tinnitus however is that it is difficult to quantify objectivity and to fully appreciate how it affects an individual's well-being. In general terms when a claim for tinnitus exists the medical assessor ideally would like to have the following criteria (as suggested by the Veteran's Administration [VA] in the United States) present before a NEL award is made:

1. The claim for tinnitus should be unsolicited.
2. The tinnitus must accompany a compensable level of hearing loss (i.e. a tinnitus match audiometrically).
3. The tinnitus should be present for at least 2 years.
4. The individual affected has undergone treatments to try to alleviate their perceived unwanted head noise (i.e. medication trials, prosthetic devices, psychiatric intervention, etc.).
5. Evidence to support a personality change or a sleep disorder as a result of the tinnitus.
6. No history of substance abuse.
7. A history of tinnitus supported by statements from the family.

Hyperacusis

Hyperacusis is a curious phenomenon where an individual describes an uncomfortable, sometimes painful hypersensitivity to sounds that would be normally well tolerated in daily life. In some instances it can be quite disabling for the affected individual who tends to withdraw from any exposure to uncomfortable noise. Many individuals affected often have normal hearing. The term “phonophobia” has been used interchangeably but erroneously implies that it is more of a psychological disorder which is certainly not the case.

Why hyperacusis arises is poorly understood. It has been documented at times to follow an incident of acute acoustic trauma. Sometimes it arises without a cause being identified. Investigations such as a magnetic resonance imaging (MRI) are generally required to exclude the remote possibility of central nervous system pathology. Mean comfort levels (MCL's) and uncomfortable loudness levels (ULL's) during audiometry provide some information concerning the threshold intensity of sound an individual can tolerate.

Tinnitus and hyperacusis not infrequently co-exist in an affected individual.

Treatment usually requires the gradual introduction of increasing sound intensity that becomes better tolerated with time. Cognitive behavioural therapy (CBT) has also demonstrated promise in a recent randomized controlled study.

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Appendix A

Glossary

1. Definitions for Impairment/Handicap/Disability*

- **Permanent Impairment of Hearing** - When any anatomic or functional abnormality produces a permanent reduced hearing sensitivity.
- **Permanent Handicap for Hearing** - The disadvantage imposed by an impairment sufficient to affect the individual's efficiency in the activities of daily living (ADL).
- **Permanent Disability** - A person is permanently disabled or under permanent disability when the actual presumed ability to engage in gainful activity is reduced because of handicap and no appreciable improvement can be expected.

2. Medical Terminology

Acute Otitis Media (AOM) - Middle ear infection usually caused by pathogenic bacteria.

Acoustic Neuroma (Vestibular Schwannoma) - A benign brain tumour that arises on the vestibular nerve usually within the internal auditory canal (IAC). The tumor is derived from the schwann cells that produce the myelin covering for the nerve. A more pathologically correct term would be that of a vestibular schwannoma (VS). One general maxim in medicine is that "*an unexplained unilateral sensorineural hearing loss should be considered an acoustic neuroma until proven otherwise*".

Cholesteatoma - Invasion of the middle ear/mastoid by skin usually originating from TM retractions. The two major properties of cholesteatoma include chronic infection and bone erosion. Hearing loss is generally present. Because of the brain's proximity possible life-threatening complications can occur if left untreated (i.e. meningitis, brain abscess etc.).

* Adapted from the American Medical Association (AMA) *Guides to the Evaluation of Permanent Impairment-Third Edition 1990*.

Chronic Suppurative Otitis Media (CSOM) - A general term for any chronic persistent and recurrent bacterial infection of the middle ear/mastoid. It is typically painless until a complication arises. It is associated with hearing loss and an intermittent often malodorous discharge from an affected ear.

Meniere's Disease - A classic inner ear disorder associated fluctuant sensorineural hearing loss, tinnitus and episodic attacks of vertigo lasting minutes-hours. The pathology is thought to arise from excess fluid in the inner ear leading to membrane ruptures (so-called endolymphatic hydrops).

Otitis Media with Effusion - An encompassing term that describes the presence of fluid behind the TM. It can be watery (i.e. serous otitis media), glue-like (i.e. mucoid otitis media) or a combination of both.

Otosclerosis - Genetically inherited condition (approximately 1 in 20 people carry the otosclerosis gene) associated with the development of new, immature bone which primarily involves the stapes footplate leading to a progressive conductive hearing loss. When the otosclerotic foci affect the cochlea this can lead to progressive sensory neural hearing loss (so-called "cochlear otosclerosis") additionally.

Ototoxicity - Tendency of certain drugs/substances to cause functional and cellular damage to the inner ear, especially in the endorgans of hearing and balance. Certain antibiotics (especially the aminoglycoside class), antimalarials, chemotherapeutic agents and even excessive doses of ASA can be toxic to the inner ear.

Presbycusis - Hearing loss associated with age. Although changes may take place as early as age 40, hearing loss due to aging usually starts to accelerate around ages 55-60 yrs and continues with age.

Tinnitus - Unwanted head noise commonly described as a ringing, buzzing, humming noise. It is typically associated with some degree of sensorineural hearing loss.

Tympanosclerosis - Calcification of the middle layer of the eardrum that looks like a patch of white chalk. Infers that an individual has had previous ear infections. Usually of no clinical consequence to an individual's hearing unless the ossicular chain involved.

3. Surgical Terminology

Mastoidectomy - Procedure designed to exteriorize disease in the mastoid air cells and adjoining middle ear. Procedure usually performed for CSOM especially when due to cholesteatoma.

Ossiculoplasty - Procedure where the ossicular chain is repaired in order to try and improve hearing.

Stapedectomy/Stapedotomy - Surgical procedures for otosclerosis (an inherited disorder where the stapes footplate becomes fixed by new bone growth involving the otic capsule). In stapedectomy surgery the entire stapes is removed (including its footplate) and a prosthesis is inserted that is attached to the incus connecting to the inner ear. In stapedotomy a small hole is placed into the stapes footplate leaving the remaining portion of the footplate intact. The suprastructure is removed and a similar prosthesis is used for reconstruction.

Tympanoplasty - When used in its most simplistic context it means the repair of a previous TM perforation.

Ventilation (myringotomy) Tube - The placement of an open ended tube or “grommet” into the TM acts like an artificial Eustachian tube which helps ventilate the middle ear space. The incision to place the tube in TM is called a *myringotomy*. The procedure is usually done to resolve a conductive hearing loss from otitis media with effusion.

4. Audiological Terminology

Audiogram - This is the standard test to assess an individual’s hearing. It can be recorded on a graph or a digital format. The pure tone audiogram measures the individual’s hearing at certain frequencies at the minimal intensity of sound (in dB) necessary to hear.

Decibel (dB) - A decibel is an accepted measure of sound pressure level used to describe sound intensity. It is based on 1 Bel (B) being equal to an accepted sound pressure level of 0.0002 dynes/cm². Because of the large numbers involved in sound pressure measurement dB scales have been created for convenience (i.e., 100 Bel = 10² Bel = 0.02 dynes/cm² = $2_{(\log 10)}$ Bel or 20 dB; 10,000,000 Bel = 70dB). The greater the dB reading at any frequency, the worse an individual’s hearing is.

Evoked Response Audiometry - Measures electrical responses within the inner ear, cochlear nerve and central nervous system that are generated by loud repetitive clicks. Types of evoked response audiometry include:

- a. **Auditory Brainstem Response (ABR)** - Synonymous with the term BERA (brainstem evoked response audiometry) or auditory BEP’s (brainstem evoked potentials). This test measure electrical activity along the cochlear nerve and at the various relay stations in the brainstem between 1-10 msec of stimulation. This test is typically performed when an asymmetric sensorineural hearing loss is present when there is concern a retrocochlear lesion such as an acoustic neuroma or MS might be present.
- b. **Cortical Evoked Response Audiometry** - Synonymous with Threshold Evoked Potential (TEP) testing. This test looks at electrical waveforms in the cortical areas of the brain between 50-200 msec after cochlear stimulation. The presence of waveforms following stimulation with the lowest intensity sound an individual’s cochlea hears

provides us with a reasonable estimation of an individual's hearing (usually within 5-10 dB of the anticipated threshold hearing at the frequency tested).

This test is typically performed as one of the tests to confirm or exclude whether an exaggerated hearing loss (malingering) is present.

- c. **Electrocochleography** - An evoked response test that primarily looks at the electrical activity generated within the cochlea and the cochlear nerve before it reaches the brainstem. This test is primarily performed to identify the presence of endolymphatic hydrops (the pathophysiologic correlate of Meniere's disease).

Frequency- Number of times one complete sound waved occurs in 1 second. The audiogram records this as cycles/sec or Hertz (Hz). As a general rule the higher the frequency, the higher the pitch.

Otoacoustic Emissions (OAEs) - Evoked test that measures minute responses to sound thought to arise from the contractile elements of the outer hair cells. Depending on the frequency of the sound certain parts of the cochlea will emit an "echo" in response.

- a. Transient OAEs are created from broadband noise stimulating the outer hair cells. It provides reasonable "pass/fail" results that help determine whether an individual's sensorineural hearing is better than 30dB. As a result it has become an ideal neonatal screening technique for deafness.
- b. Distorsion product OAEs (DPOAEs) are currently being employed in more complete neonatal screening for hearing loss and have a potential role in screening for exaggerated hearing loss.

Impedance Testing - Part of the conventional audiometric test battery that helps measure middle ear pressures and whether ipsilateral and contralateral stapedial reflexes are present /absent.

Recruitment - Perceived abnormal progressive loudness of noise in an individual with a sensorineural hearing loss. Thought to be a hallmark of cochlear dysfunction. Not infrequently seen in Meniere's disease.

Speech Discrimination Score (SDS) - The percentage score an individual correctly identifies when presented with a list of phonetically balanced (PB) words usually tested at 40dB above their hearing threshold.

Speech Reception Threshold - The dB threshold level at which an individual is first able to hear speech and recognize spondaic words (i.e. words like hotdog, baseball, uptown, downtown, ice cream etc.)

Stapedial Reflex Testing - Measures reflex contraction of the stapedius muscle. This occurs to a loud noise typically 70-80 dB above an individual's hearing level at the frequency tested which causes stiffness of the ossicular chain and tympanic

membrane Absent stapedial reflexes are usually seen in otosclerosis due to stapes footplate fixation for example. Sometimes the responses can be very large if there is an ossicular chain discontinuity.

Tympanometry - Formal tracing of how the TM moves when pressure is altered in the external auditory canal. Helpful in addition to impedance testing in determining if middle ear pathology is present.

5. Specific Noise Induced Hearing Loss Terminology

Acoustic Trauma - Damage to the ear caused by a single exposure to an impulse noise of high density.

Continuous Noise - A noise that remains relatively constant in sound level for at least 0.2 seconds.

dBA - Measurement of a sound level using the A scale of a sound-level meter; with the A filter, low and very high frequencies are attenuated, thus giving greater weight to those frequencies most likely to be damaging to the ear.

dB HL - Hearing level referenced to a pressure of 20 uPa; this reference level corresponds to the amount of acoustic energy that is just audible for those frequencies to which humans are most sensitive.

Dosimeter - An instrument that integrates a function of constant varying sound pressure over a specified time period in such a manner that it directly indicates a noise dose and estimates the hazard of an entire exposure period.

Impact Noise - A type of transient noise produced by the collision of two masses (i.e. pile driver) and consisting of a succession of positive and negative pressure peaks of slowly declining amplitude.

Impulse Noise - A type of transient noise produced by the sudden expansion of gasses (i.e. gunfire, explosion etc.) and characterized by single positive pressure peak then a rapid return to normal atmospheric pressure.

Permanent Threshold Shift (PTS) - The permanent depression in hearing sensitivity resulting from exposure to noise at damaging intensities and durations.

Temporary Threshold Shift (TTS) - The temporary (usually several hours) depression in hearing sensitivity resulting from exposure to noise at damaging intensities and durations.

Time Weighted Average (TWA) - The sound level or noise equivalent that, if constant over an eight hour day would result in the same exposure as the noise level in question (assuming a 5 dB doubling rule). Different jurisdictions apply different rules depending on whether a 3 or 5 dB criteria is used.

- a. *3 dB rule* - If risk to hearing is simply related to the total energy in noise exposure then doubling of sound energy would result in a 3 dB increase in noise level. This rule would require that exposure time to noise be halved for each 3 dB intensity increase in order to maintain the same risk to hearing.
- b. *5 dB rule* - The statement that risk to hearing is related to the total energy in a noise exposure *plus the physiologic processes* in the auditory system that may limit the deleterious effects of continuous or subsequent overstimulation by the noise; this rule defines equal hearing risk to noise exposure for which exposure duration has been halved for each 5 dB increase in intensity.

Appendix B

Anatomy and Physiology of the Ear

The ear is a complex organ that has dual responsibility for hearing and balance (vestibular) function. Conceptually it has three distinct anatomical components which include the external, middle and inner ear.

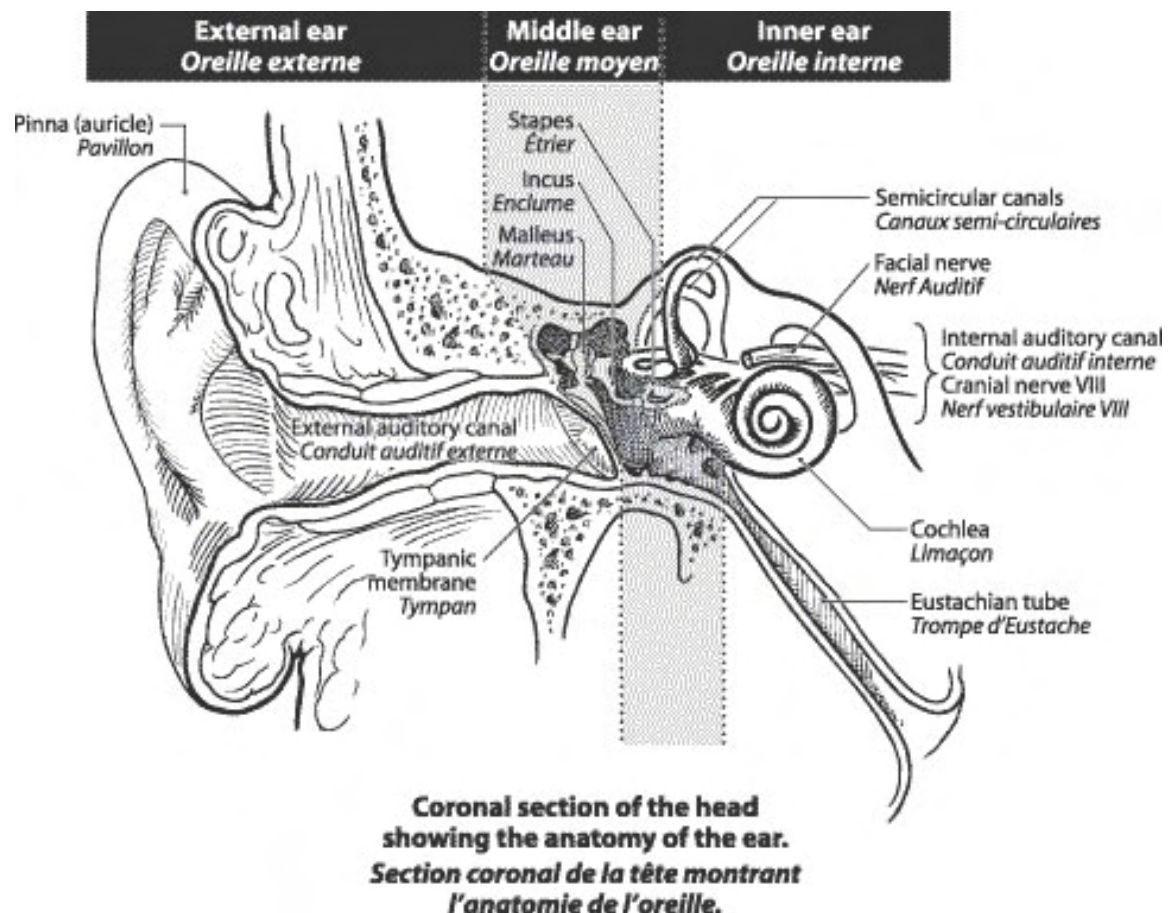


Figure B-1 - Anatomical drawing of the ear

a. The External Ear

The external ear consists of the auricle (pinna) and the ear canal. The auricle is a flattened, funnel shaped appendage that contains a cartilage framework covered by skin. In humans the auricle has rudimentary functions for gathering sounds and in sound localization which is much more important in animals. To some degree it helps to protect the ear canal from foreign bodies and insects.

The ear canal (external auditory canal) originates from the concha (so-called bowl) of the auricle and extends medially to the tympanic membrane. It is approximately 25-30mm in length and can be thought of as having a cylindrical shape. The first part of the ear canal is cartilaginous and is lined by thick, hair bearing skin that contains numerous sebaceous and ceruminous glands. The deeper part of the ear canal is bony, lacks skin appendages and is lined by thinner skin densely adherent to the periosteal lining of the bone.

The ear canal's primary function is to provide a conduit for airborne sound waves to reach the tympanic membrane. The anatomy favours the transmission of sound waves typically between 500-8,000 Hz which interestingly represents the common speech frequencies of most individuals. A secondary function is to protect the tympanic membrane from trauma. Somewhat unique is the ear canal's ability to self-cleanse. Continuous migration of epithelium from the deeper to the lateral portion of the ear canal tends to keep the ear clean. Wax and debris can collect in the ear canal when this migratory mechanism fails or is overloaded from infectious or inflammatory conditions.

b. The Middle Ear

The middle ear cleft is a more extensive designation of the middle ear that has three main components which include the middle ear cavity, mastoid air cell system and the Eustachian tube. Proper function of the Eustachian tube which connects the middle ear cavity to the back of the nose (nasopharynx) is important for the normal middle ear aeration and function. Malfunction of the Eustachian tube remains the leading cause for most middle ear disorders.

The mastoid air cell system consists of a series of small mucosal lined air cells interconnected with each other and the middle ear proper. Their actual role in humans remains unclear. The air cell system is typically underdeveloped or even absent (sclerotic) in individuals with a past history of recurrent ear disease. Their infection can sometimes lead to a mastoiditis with serious consequences.

Important structures of the middle ear include the tympanic membrane and the ossicular chain. The tympanic membrane or ear drum is an ovoid, pale gray, semi-transparent membrane positioned obliquely at the medial end of the ear canal. It is a three layered structure whose outer epithelial layer is contiguous with the skin of the external auditory canal, its middle layer is fibrous and its inner layer is composed of the

same mucosal lining found throughout the middle ear cleft. It is anatomically divided into 2 parts: the pars tensa and the pars flaccida (located in what is called the attic region of the tympanic membrane). The pars tensa or lower four-fifths of the ear drum is conically shaped and has a strong fibrous middle layer which provides strength. The pars flaccida or upper one-fifth is less distinct and has a poorly developed middle fibrous layer. This makes the pars flaccida especially susceptible to negative middle ear pressure and prone to retractions that can lead to the formation of retraction pockets. When retraction pockets fail to be self-cleansing they fill with debris and desquamated skin that can lead to secondary infection and bone erosion better known as cholesteatoma.

The ossicular chain consists of the three small interconnected mobile bones known as the malleus (hammer), incus (anvil) and stapes (stirrup). The tensor tympani and stapes muscles help stabilize the bones along with suspensory ligaments of connective tissue inside the middle ear. Clinically the malleus is attached by its handle to the tympanic membrane and is readily visible in most individuals on otoscopic examination. Medially the arch (crura) of the stapes attaches to the inner ear via its footplate (which is a thin, flat oval piece of bone approximately 7mm X 3mm in dimension). The blood supply to the incus can be tenuous compared to other structures of the middle ear. When pathology affects its blood supply it can lead to the erosion of its long process which is attached to the stapes. This in turn can result in an ossicular discontinuity.

c. The Inner Ear

The inner ear is the sensory end organ within the hardest bone of the body known as the petrous portion of the temporal bone. Covered by an otic capsule its membranous portion (or labyrinth) contains a delicate system of fluid-filled canals lined with sensory neuroepithelium necessary for hearing and balance perception. The separate but intimately related perilymphatic and endolymphatic compartments of the inner ear and their ionic constituents are efficiently controlled by the stria vascularis of the cochlea and the dark cells of the vestibular apparatus.

The cochlea specifically refers to the sensory portion of the inner ear responsible for hearing. It is a three chambered organ containing anatomical spaces known as the scala media, scala vestibuli and scala tympani. The sensory end organ for hearing is known as the organ of Corti. It contains a single row of inner hair cells (IHC's), three rows of outer hair cells (OHC's) and numerous supporting cells draped by the gelatinous tectorial (TM) membrane which lies on a basilar membrane. The basilar membrane (BM) separates the organ of Corti (OC) found in the scala media from the scala tympani. Another structure called Reissner's membrane (R) forms the roof of the scala media (M) which separates this from the scala vestibuli (V).

The scala media contains a potassium rich fluid known as endolymph. The scala vestibuli and scala tympani (T) by comparison contain fluid rich in sodium called perilymph. This ionic difference between these chambers forms the basis of the

endocochlear electrical potential which is necessary for the conversion of mechanical activity from fluid movement within the cochlea to electrical activity arising from the hair cells. Series of generation potentials culminate to form action potentials that transmit this electrical activity along the cochlear nerve fibres to the brainstem and onto the higher centers of auditory processing.

The vestibular portion of the inner ear consists of three fluid lined semicircular (superior, lateral and posterior) canals and the area called the vestibule which houses the otolithic macular endorgans. Within each semicircular canal is an area called the ampulla which contains hair cells sensitive to low frequency bidirectional fluid displacement that occurs with angular acceleration type movements of the head. The hair cells of the otolithic endorgans (utricle and saccule) are covered by otoliths (literally “little stones” or crystals of calcium carbonate). The hair cells of the vestibular macula respond primarily to gravity and linear accelerations. Abnormalities in vestibular end organ function often result in the patient experiencing the subjective complaint of vertigo.

Fig. B-2A Section through the Cochlea

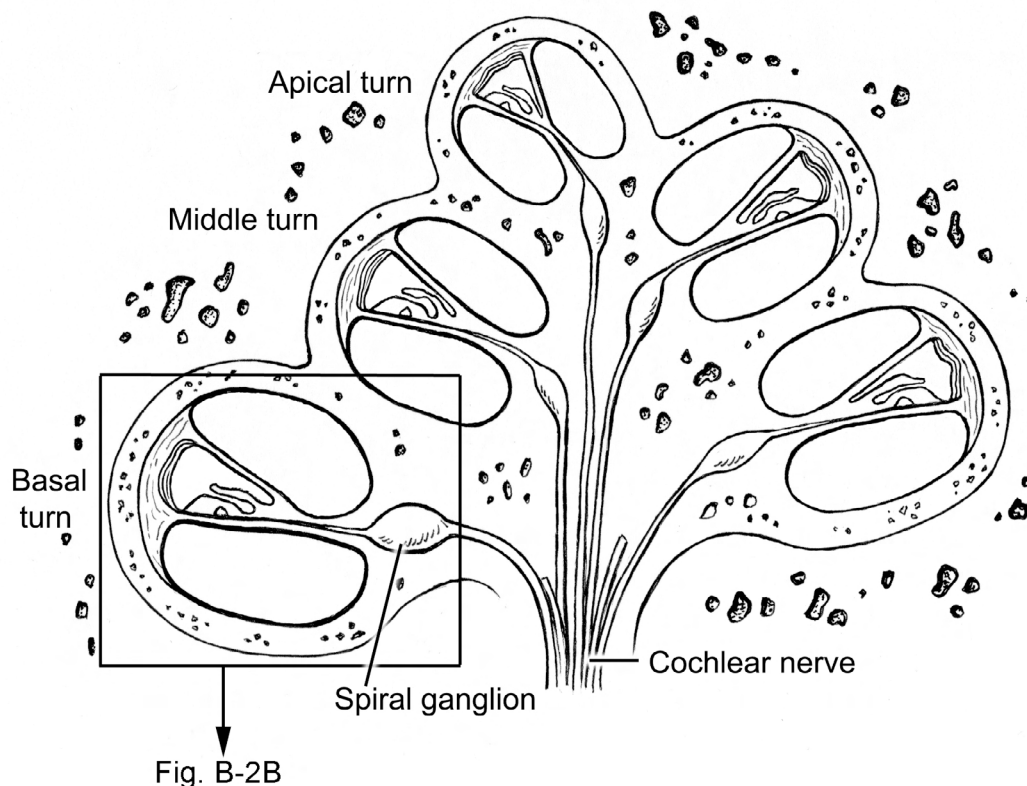


Figure B-2A - Section through the cochlea

Fig. B-2B Section through one turn of the Cochlea

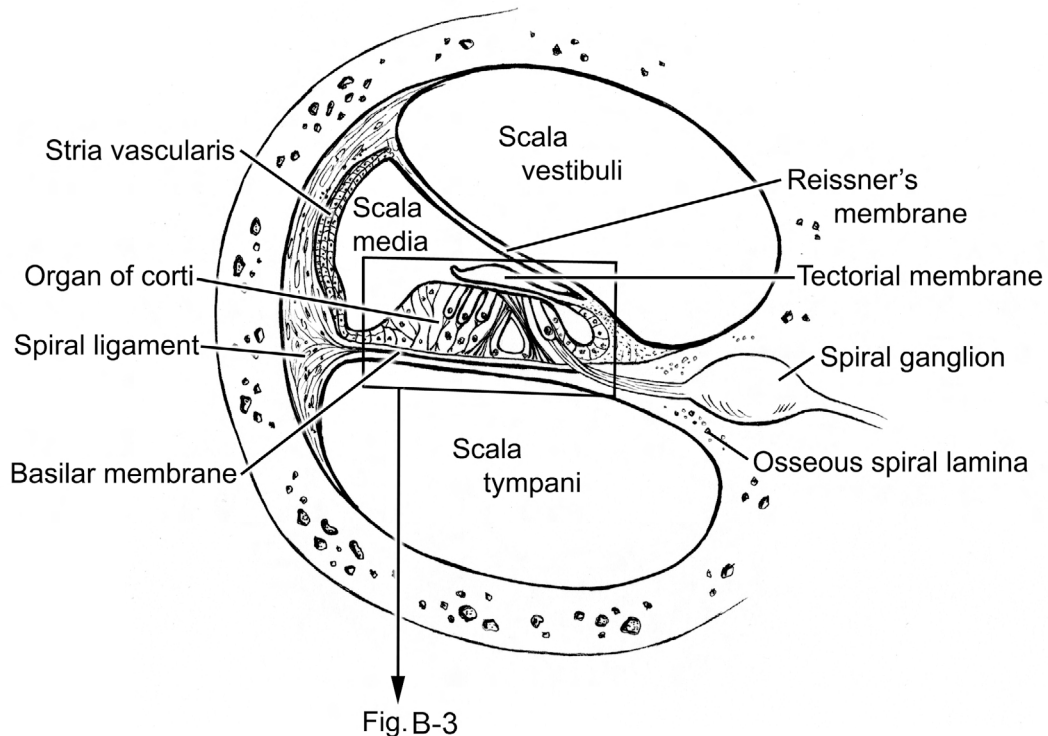


Figure B-2B - Section through one turn of the Cochlea

Physiology of Hearing

Sound vibrations are picked up by the pinna and transmitted down the external auditory canal where they strike the TM causing it to vibrate. The sound vibrations are then transmitted across the air-filled middle ear space by the 3 tiny linked bones of the ossicular chain: the malleus, incus and stapes . The mechanical vibrations are transmitted to the inner ear via the vibrations of the stapes footplate.

When the mechanical vibrations of the stapes footplate reach the inner ear they create traveling waves in the cochlea. The hair cells change these mechanical vibrations from the waves into electrochemical impulses that can be interpreted by the central nervous system (CNS). The tiny cilia (little hairs) on top of the hair cells (both inner and outer) are covered by a gelatinous membrane called the tectorial membrane. Fluid waves in the inner ear cause a deflection of both the tectorial and basilar membranes that surround the organ of Corti. The cilia move and generate a nerve impulse called a generation potential (GP). When enough generation potentials occur they result in what is called an action potential (AP). The transmission of electrical activity along the cochlear nerve will ultimately make its way through a series of nuclear relay stations

within the brainstem (this concept forms the basis of the electrophysiological test called the *auditory brainstem response* or *ABR*). Electrical signals are then forwarded to the auditory cortex in the temporal lobe for decoding. How we perceive what certain electrical signals represent in our auditory cortex forms the basis for the field of *psychoacoustics* (ie the perception of sound).

Although the vast majority of the cochlear nerve fibres are termed *afferent* (ie nerves that carry electrical activity from the inner ear to the brain), within the cochlear nerve itself we have a small number of nerve fibres designated as *efferent* (nerves that carry electrical activity from the brain to the inner ear). Most of the efferent fibres seem to land on the outer hair cells of the organ of Corti. This apparent internal “feedback loop” is thought to be responsible for “fine tuning” by inhibiting some unwanted electrical impulses and by changing the mechanical properties of the basilar and tectorial membranes. The presence of active non-muscular contractile elements within the outer hair cells and their effects on movement of the basilar membrane is used to explain the concept of *otoacoustic emission* (OAE) testing.

Of interest when the outer hair cells become injured or affected by pathology they also lose their ability to “fine tune” the electrical responses arising from the inner ear. The phenomenon of *recruitment* represents an abnormal sense of loudness. Distortion of certain sounds arises when enough hair cells are damaged such that the hearing threshold is reduced. When the sound gets loud enough the inability to “fine tune” sound becomes lost and more nearby hair cells are drawn into the firing needed to create an electrical signal; hence the distortion of a loud sound.

Although an individual may have apparently normal hearing it does not necessarily mean the cochlear nerve is undamaged. It is estimated that up to 75% of the auditory nerve supplying a certain section of the cochlea can be injured without causing an appreciable change in pure tone threshold hearing. This may be one reason why certain individuals with tumors arising on the nerves of balance and hearing better known as *acoustic neuromas* (vestibular schwannomas) often preserve their tonal perception of sound yet have problems with its discrimination (i.e. they know someone is talking on the telephone but can’t understand what is being said in the affected ear).

Fig. B-3 Schematic Diagram of the Organ of Corti

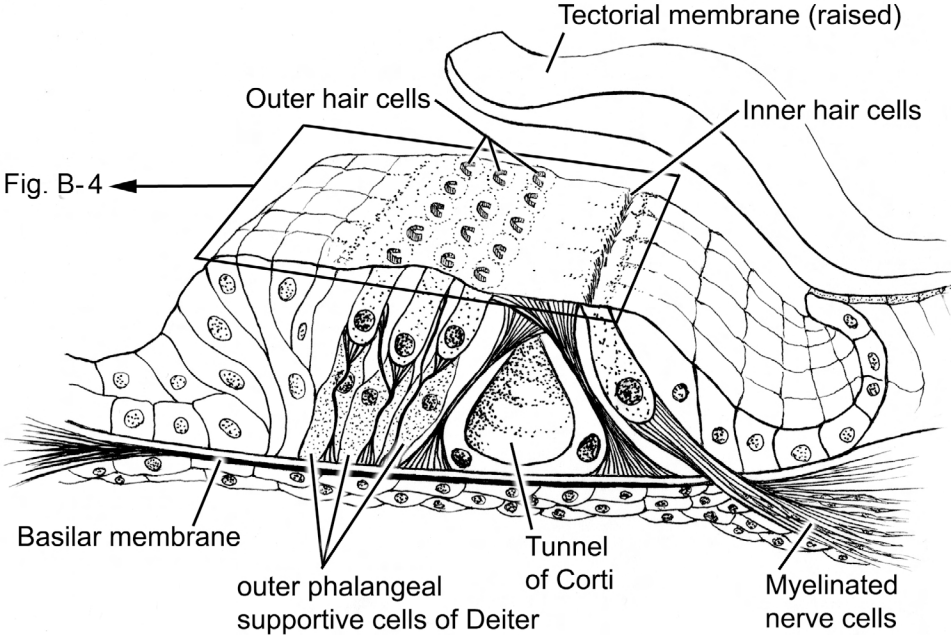


Figure B-3 - Schematic Diagram of the Organ of Corti

Fig. B-4 Electron Microscopy Schematic of Inner and Outer Hair Cells

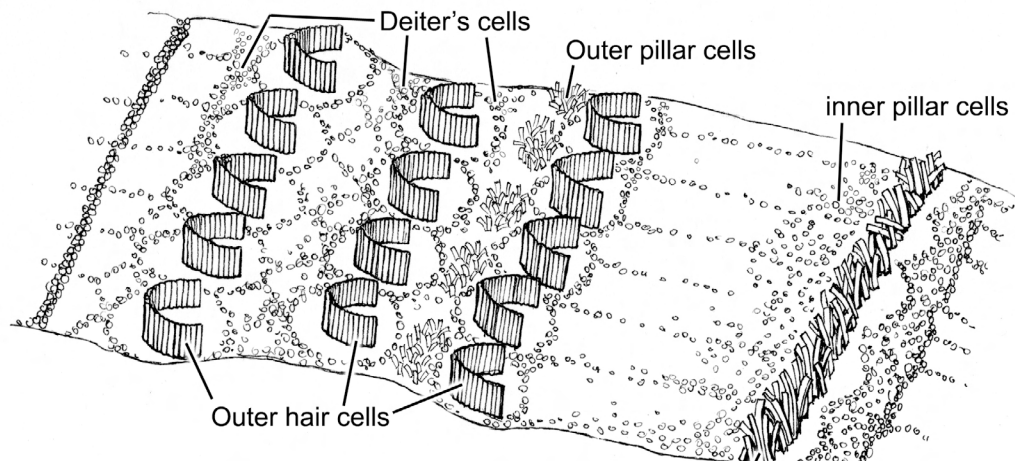


Figure B-4 - Electron Microscopy Demonstrating Inner and Outer Hair Cells

Some Physical Considerations of Sound

Sound is the propagation of pressure waves through a medium such as air and water for example. It can be a simple sound commonly known as a pure tone or it may be complex when we think of speech, music and noise. A cycle of a pure tone is represented by a sine wave appearance with an area of compression followed by rarefaction.

Pure tones have several important characteristics. Frequency represents the number of cycles per second or Hertz (Hz). Low sounds tend to have a long wavelength relative to higher pitched sounds which have shorter wavelengths by comparison. The physiologic correlate of frequency is pitch. In general terms the greater the frequency the higher the pitch of the sound and the greater the intensity the louder we hear it. The degree of intensity or loudness of a sound is measured in decibels (dB). In complex sounds the interaction of its pure tone components forms the basis of its complexity or its psychological counterpart known as timbre.

Appendix C

Questions and Controversies in NIHL (Occupational Hearing Loss)

1. Why are the frequencies of 0.5, 1, 2 and 3 kHz used for NIHL awards when hearing loss is often greater at 4, 6 and 8 kHz?

This is a very relevant question that is based on the definition of how hearing loss causes a handicap for the affected individual in their ability to identify spoken words or sentences under everyday conditions of normal living.

Although the higher frequencies are initially affected in NIHL an individual will really only begin to demonstrate a significant handicap to hearing to everyday speech in a quiet surrounding when the average of the hearing thresholds at 500, 1000, 2000 (\pm 3000) Hz is above 25 dB. The prediction of a handicap did not appear to be improved by the inclusion of other frequencies > 4000 Hz.

The inclusion of the 3000 Hz threshold value for NIHL awards is not always required depending on the jurisdiction. It should be appreciated that while a decrease in hearing sensitivity at 3000 Hz has little effect upon hearing speech in quiet, it produces a handicap in the presence of competing background noise (i.e. restaurants, family gatherings, church etc) or when speech is distorted. Most would not be expected to have problems with the telephone (most telephones rarely transmit sounds > 3000 Hz).

2. What do other jurisdictions consider to be disabling hearing loss (provincial, national and international)?

In the province of Ontario the Workplace Safety and Insurance Board (WSIB)'s Operational Policy Manual (OPM) Document No. 16-01-04, entitled "Noise Induced Hearing Loss, On/After January 2, 1990" states that workers with an occupational noise induced hearing loss sufficient to cause a hearing impairment may be entitled to benefits when there is a hearing loss of 22.5 dB in each ear when the four speech frequencies (500, 1000, 2000 and 3000 Hz) are averaged.

The WSIB accepts the following circumstances as persuasive evidence of work-related conditions in claims for sensorineural hearing loss:

- *Continuous exposure to 90dB (A) of noise for 8 hrs per day, for a minimum of 5 years, or the equivalent; and*
- *A pattern of hearing loss consistent with noise induced sensorineural hearing loss.*

A presbycusis (aging) correction factor of 0.5 dB is deducted from the measured hearing loss (averaged over the 500, 1000, 2000 and 3000 Hz frequencies) for every

year the worker is over the age of 60 at the time of the audiogram. The hearing loss that remains after the presbycusis adjustment is then used to determine entitlement to benefits. Entitlement to health care and rehabilitation benefits is available when the adjusted hearing loss is at least 22.5 dB in each ear.

It is important to also appreciate that decisions can also be made on an exceptional basis which the WSIB has recognized.

Exceptions

Since individual susceptibility to noise varies, if the evidence of noise exposure does not meet the above exposure criteria, claims will be adjudicated on the real merits and justice of the case, having regard to the nature of the occupation, the extent of the exposure, and any other factors peculiar to the individual case.

A list of Provincial and Territorial Comparisons for NIHL is provided below*

Province	Exposure Requirements	a. Entitlement Criteria b. Minimum hearing loss level for hearing aid benefits	Presbycusis correction factor (or other)	Tinnitus compensation a. In Claim b. In PI
British Columbia (BC)	85 dBA-8h/day over 2 years	a. Average at 500, 1000 and 2000 Hz b. Hearing loss > 30 dB/30dB bilaterally for hearing loss benefits	Use of <i>Robinson's Tables</i>	a. Yes b. NA
Alberta (AB)	85 dBA-8h/day over 2 years	a, b. Average at 500, 1000, 2000 and 3000 Hz >140dB to be considered for hearing aids and/or pension award	Use of <i>Robinson's Tables</i>	a. Yes b. NA
Saskatchewan (SK)	85 dBA-8h/day over 2 years	a. Average at 500, 1000, 2000 and 3000 Hz b. Hearing loss > 35 dB bilaterally	No deduction	a. Yes b. 2%
Manitoba (MB)	85 dBA-8h/day over 2 years	a. Average at 500, 1000, 2000 and 3000 Hz b. Hearing loss > 26.25 dB bilaterally for benefits	2 dB/year after age 60	a. Yes b. 2%
Ontario (ON)	90 dBA-8h/day over 5 years	a. Average at 500, 1000, 2000 and 3000 Hz b. Hearing loss > 22.5 dB for benefits	0.5 dB/year after age 60	a. Yes- medical opinion and tinnitus match required b. 2%

Province	Exposure Requirements	a. Entitlement Criteria b. Minimum hearing loss level for hearing aid benefits	Presbycusis correction factor (or other)	Tinnitus compensation a. In Claim b. In PI
Quebec (QB)	NA	a. NA b. Hearing loss > 30 dB for benefits	NA	a.b. NA
New Brunswick (NB)	NA	a. Average at 500, 1000, 2000 and 3000 Hz b. Hearing loss >25 dB for benefits	NA	a. Yes b. NA
Prince Edward Island (PEI)	85 dBA-8h/day over 2 years	a. Average at 500, 1000, 2000 and 3000 Hz b. Hearing loss >25 dB in one ear for benefits	0.5 dB/year after age 60	a. Yes b. 2%
Nova Scotia (NS)	NA	a.b. Average at 500, 1000, 2000 and 3000 Hz > 100 dB	2 dB/year after age 60	a. Yes b. NA
Newfoundland (NF)	85 dBA-8h/day over 2 years	a. NA b. Hearing loss >25 dB for benefits	0.5 dB/year after age 60	a. Yes b. NA
Yukon	85 dB-8h/day over 5 years (but must have worked for 2 years in the Yukon)	a. Average at 500, 1000, 2000 and 3000 Hz b. Hearing loss must be > 25 dB for benefits	None accounted for	a. Yes b. 2%
Northwest Territories and Nunavut	90 dBA-8h/day over 5 years	a.b. NA	NA	a.b. NA

NA-Not available, PI-permanent impairment

* as of 2006

The above list does demonstrate a lack of uniformity in NIHL claims and benefits within Canada. A comprehensive list of US comparisons for NIHL and benefits can be found in Table 35.1 pages 797-799 in the Chapter concerning State and Federal Formulae Differences in Occupational Hearing Loss, 3rd Edition. Sataloff and Sataloff. 2006

3. Does previous noise exposure make an ear more sensitive to future noise exposure?

Apparently it does not. It is generally thought that if an ear has suffered a permanent threshold shift from a noise induce etiology further noise exposure will cause less

damage than would occur in a normal ear to a similar exposure. This is based primarily on animal models which have demonstrated that at the frequency range of maximum damage the increase in a noise induced PTS from the second exposure was smaller for ears with greater pre-existing loss (the so-called, “you can’t further damage what has already been damaged” rule)

4. Does previous NIHL accelerate the onset of presbycusis?

This is question that continues to intrigue auditory research scientists. As previously noted the effects of noise exposure and aging on hearing when not combined are reasonably well understood. When the two processes are combined, the resultant pathology and its effects upon aging are not as well understood.

It is likely that the two effects are not additive (Corso’s theorem) but from a practical point of view this is how they are usually viewed with regards to compensation claims (Dobie’s theorem).

Some generally accepted principles (according to the American Academy of Otolaryngology (AAO)-Head and Neck Surgery 1994 Guidelines) with regards to age related change note that:

- a. At any given age for frequencies above 1000 Hz men will have more age related hearing loss than women.
- b. Age related hearing loss affects all frequencies although the higher frequencies are usually more affected.
- c. Age related hearing loss is an accelerating process where the rate of change increases with age.

5. Does permanent damage to the cochlear hair cells caused by noise exposure contribute to the eventual development of a hearing disability?

Yes it does. One however has to qualify this by saying that there is a significant amount of redundancy within the inner ear as it pertains to hearing. In other words many hair cells in a similar region of the cochlea will produce a certain frequency response to sound stimulation. Hair cell loss can continue until a certain critical point is breached with the individual unaware of a hearing deficit. Once the critical loss of hair cells occurs the individual will notice a hearing loss.

One can speculate that this might be one of the reasons an individual early on in their exposures may not be aware of a hearing loss, only to appreciate a hearing loss later in life when other factors such as presbycusis change occur.

6. At what age does presbycusis begin to make a material difference to hearing disability and at what age can its effects be seen on an audiogram?

When we are born we can hear frequencies up to 20,000 Hz. Over the years we hear less and less. Because we tend to make little use of frequencies > 8000 Hz we do not become aware of a hearing loss in general until the frequencies < 8000 Hz are affected.

Although we think of presbycusis as an age related event not all individuals will develop this condition. Moreover we really don't have a lot of good prospective longterm studies over 4-5 decades that can ultimately answer this question completely. Upon saying this however we can actually demonstrate that many individuals will start to show early changes in hearing as early as age 40 years (in subjects screened to rule out other ear disease and noise exposure).

With regards to the rate of hearing loss noted in presbycusis, recent evidence from longitudinal prospective studies from Denmark and the UK indicate that the actual rate of deterioration seems to be influenced by age; those over 55 years showed a higher rate of deterioration of up to 9 dB/decade against a deterioration of 3 dB/decade for those under 55 years.

Future genetic studies may provide us with further information concerning those at greater risk for progressive hearing loss from presbycusis in future.

7. Can moderate workplace noise exposure causing a TTS and repeated exposures later cause a PTS?

Yes. Repeated noise exposure that causes a temporal threshold shift (TTS) can ultimately lead to a permanent threshold shift (PTS) with repeated exposures. It is agreed that hearing loss and injury to the ear increases with the noise level, the duration of exposure, the number of exposures and the susceptibility of the individual.

A TTS is considered to represent a pathological metabolically induced fatigue of the hair cells or other structures within the Organ of Corti. Its development and recovery are proportional to the logarithm of exposure time. It reverses slowly over a period of hours. The practical "cutoff point" for a TTS is approximately 40dB. Below this threshold value recovery is relatively swift; above this it appears delayed.

In a PTS the destruction and eventual cochlear hair cell loss is thought to arise from direct mechanical destruction from high-intensity sound and from metabolic decompensation with subsequent degeneration of sensory elements.

While one would normally expect full recovery of hearing function after a TTS there is one important consideration that needs to be taken into account. Some of this is based on the redundancy principle within the inner ear: *not all hairs cells possibly recover following a TTS but enough do as to prevent hearing loss*. Continued exposure to excessive noise will therefore result in further hearing loss.

8. Following acute acoustic trauma what is the natural history of the subsequent hearing loss?

The position in the world literature continues to support that any progressive hearing loss from acute trauma ceases unless the individual is re-exposed to more acute acoustic trauma or some other factor (s) occurs (i.e. concomitant presbycusis, head injury etc).

The classic studies of acute acoustic trauma by Segal et al (1988) and Kellerhals (1991) are often used to support this position.

In the cohort study by Segal et al, 841 individuals from either the regular US army or military reserve sustained a permanent sensorineural hearing loss from acute acoustic trauma without subsequent re-exposure. Audiometric evaluations were performed on these individuals at 6 months, 1, 2 and 4 years after initial exposure. Hearing remained stable at 1 year in approximately 90-91.3%, improved in 7.7% and appeared to worsen in 2.2%. Follow-up beyond the 1 year mark did not reveal any further deterioration of hearing in any individual. Of interest, in a parallel series of control patients who were further exposed to repeated episodes of acute acoustic trauma during the same study period by the end of 4 years 30% of these individuals had demonstrated significant hearing deterioration.

The importance of the Segal study is that after 1 year one would not expect further worsening of hearing in subjects who ceased to have further noise exposure.

Kellerhals' study reviewed a much smaller series of individuals who were exposed to acute acoustic trauma but looked at them over a 20 year duration. In his study cohort only 1% of individuals following acute acoustic trauma demonstrated deterioration of hearing of more than 20 dB in at least 1 frequency. The findings also indicate that the late progression of hearing loss following a single episode of acute acoustic trauma did not exist.

9. Are certain individuals more predisposed to developing NIHL than others?

Large susceptibilities in NIHL occur in persons exposed to the same noise environment. A review of endogenous factors related to individual sensitivity by Ward did not find any effects other than those related to the acoustic energy reaching the cochlea or the oxygen supply to the cochlea.

While there has been concern that smoking and elevated serum cholesterol levels might play a role by affecting blood flow and hence oxygen delivery to the cochlea this has not been validated. Pigmentation differences in subjects grouped by eye color (on the basis that the pigmentation in the stria vascularis of the cochlea is similar) and race have been inconclusive as well.

Other studies have failed to show any effect on vulnerability caused by diabetes, silicosis or leprosy. There is some slight evidence that Meniere's disease might increase vulnerability. Whether deficiencies in vitamins render an individual more sensitive is also not certain.

The link between diabetes and hearing loss has been further identified in some recent studies. In the 2008 study from the National Health and Nutrition Examination Survey the prevalence of hearing loss was 15% for participants without diabetes and almost double for those with diabetes. The 2009 modelling study from the Veteran's Affairs (VA) National Center for Rehabilitation Auditory Research (NCRAR) identified those with diabetes were more likely to have greater hearing loss in the lower frequencies (<2000Hz) across all ages (from 26-71 years). Interestingly the greatest effect of diabetes on hearing appeared in those patients under age 50 years at frequencies > 8,000 Hz. Connective tissue disorders (i.e. rheumatoid arthritis, lupus, vasculitis with granulomatosis etc.) can also affect sensorineural hearing in some individuals. This is usually thought to be a result of an associated vasculitis affecting inner ear blood supply.

In summary while certain disease processes have been associated with hearing loss, increased susceptibility to noise has not been shown to be dependent on gender, race, any known diseases, mental attitude towards noise, exposure history, pre-exposure hearing loss and even a history of smoking.

10. What would be considered inadequate audiometric data to support or deny a NIHL claim?

An accurate audiogram requires:

- the tester to be technically proficient,
- to be performed in a facility that ideally adheres to the generally comparable American National Standards Institute (ANSI) or International Standards Organization (ISO) facility standards for hearing testing (i.e. topic areas include audiometer calibration, test room noise levels and sound level meters for confirmation) and,
- a compliant patient.

There is general consensus that the most reliable audiogram is best performed by a qualified audiologist who by education and training has specialized skills and expertise in both hearing testing and rehabilitation of hearing loss.

Some examples of how systematic and random errors in audiometry can affect hearing results include:

- audiometric calibration error or drift during testing

- excessive background noise levels in the test room
- interfering signals from the test equipment
- improper earphone placement
- tester bias and examination procedure bias
- improvement in performance due to familiarity with the test procedure
- a temporary threshold shift from recent noise exposure shortly before the time of the examination
- partial or complete obstruction of the ear canal (i.e. wax)
- the presence of tinnitus that competes with the audiometric pure tones that are presented

In the Province of Ontario a weighted pure tone average for the sensorineural hearing thresholds at 500, 1000, 2000 and 3000 Hz is required to determine eligibility for compensable benefits. Failure to measure bone conduction thresholds responses (where necessary) and hearing especially at the 3000 Hz tone represent major technical reasons why audiometry is often considered inadequate to support an OHL claim.

Inconsistency in audiometric testing where different checks are used to determine the accuracy of hearing suggests the hearing loss may be exaggerated or that the individual may be malingering.

11. From a medical assessor's perspective why are many NIHL claims contentious?

Most contentious claims arise for the following reasons that include:

- The absence of a proper pre-employment audiogram.
- The absence of a proper exit audiogram especially when a worker leaves their employment.
- Poor quality audiograms performed either by an individual not appropriately trained or in facilities not up to the ANSI or ISO recommended standards.
- The configuration of the audiogram is not typical for NIHL especially when the 4000 Hz loss is not present, there is a greater than expected loss of hearing in the lower frequencies and when significant asymmetries to hearing loss occur without a reasonable explanation.

- Differing opinions between the treating physician and the medical assessor. It is always important to remember that, “while all NIHL is sensorineural in nature, not all sensorineural hearing loss is occupational”.

12. What if hearing loss is below the WSIB threshold policy?

If the hearing loss is below WSIB threshold policy an affected individual may not qualify for compensation benefits. This does not take away from acknowledging that such an individual will experience some degree of hearing impairment. It does however serve notice that an individual must continue to wear hearing protection if exposed to noise levels injurious to hearing and that industry continues to have a role in making the workplace as safe as possible by the introduction and further refinement of hearing conservation programs.

The rules governing compensation awards for hearing loss from noise are well entrenched in the Province of Ontario. It is largely based on the amount of the loss according to well described criteria applied to each worker who submits an occupational hearing loss claim. In Ontario workers a correction factor for presbycusis of 0.5 dB/year after age 60 is also applied. Further information how a worker’s hearing loss is calculated can be found in the answer to Question 2.

13. Does the type of noise, amount duration and frequency of noise exposure in the workplace predispose an individual to NIHL?

In general terms the following statements broadly apply to noise induced hearing loss:

- Impact/impulse noise is more likely than steady state noise to cause hearing loss

Impact noise (i.e. foundry work, jack hammers, pile drivers etc.) arises when two masses collide under force. Under the circumstances the ears are exposed to extremely loud noise levels that peaks then fatigues quickly. Impulse noise is similar but typically arises when there is a sudden expansion of gaseous pressure (i.e. rifle fire, explosion etc.) that quickly returns to normal atmospheric pressure. Under the circumstances the individual does not have time to protect their hearing nor can the inner ear respond by its built in reflexes to minimize noise induced trauma.

- The greater the exposure to noise levels potentially injurious to hearing the more likely a hearing loss will occur.

This statement forms the basis for all hearing conservation programs that require an individual to wear hearing protection to prevent hearing loss when exposed to noise levels injurious to hearing.

- An individual exposed to continuous noise levels will be more likely to experience a greater degree of hearing loss than one who is intermittently exposed to the same noise levels.

This is based on the identification that an ear with intermittent noise exposure will have time to recover in between events that will cause a temporary threshold shift and prevent permanent threshold shifts with continued exposure from occurring.

14. Does hearing deteriorate after removal from workplace noise exposure?

Workers not infrequently complain that their hearing has continued to deteriorate after they have stopped working. The general consensus however is that hearing loss following workplace noise exposure should stabilize when a worker leaves a noisy environment. Further hearing loss if it occurs is therefore thought to arise from other cause(s), the most common being presbycusis (from age related change).

It is also important to recognize that after age 55 hearing loss from presbycusis seems to accelerate. This is usually around the age when many individuals stop working. Loss of sensorineural hearing from prior noise exposure will be compounded by presbycutic change that not only affects the inner ear but also central auditory pathways in the brain as well.

Hearing would not be expected to improve following removal from workplace noise exposure. This is on the basis that the affected individual was not experiencing a temporary threshold shift (TTS) from noise exposure at the time of testing that improved on later testing.

15. Does the wearing of hearing protection prevent NIHL?

There are numerous studies and outcome analyses that demonstrate the effectiveness of hearing conservation programs in industry. Hearing protection (i.e. ear muffs, ear plugs etc.) however needs to be worn properly and consistently to prevent noise induced hearing loss.

Most ear plugs will provide between 20-25 dB of sound attenuation. Combinations of muffs and plugs might provide upwards of 25-35 dB of sound attenuation. Despite wearing hearing protection some workers could theoretically experience noise induced hearing loss if the exposure levels were consistently greater than 110dB and the hearing protection worn did not provide adequate sound attenuation.