Ontario

Hearing Loss and Tinnitus

Discussion paper prepared for

The Workplace Safety and Insurance Appeals Tribunal

February 2003

Prepared by:

John Rutka MD FRCSC

Associate Professor
Department of Otolaryngology, University of Toronto
Staff Otoneurologist
University Health Network(UHN)

Co-director UHN Center for Advanced
Hearing and Balance Testing,
Multidisciplinary Neurotology Clinic

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 Discussion papers are not peer reviewed.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).

Version 2.1

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).

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. Menière'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

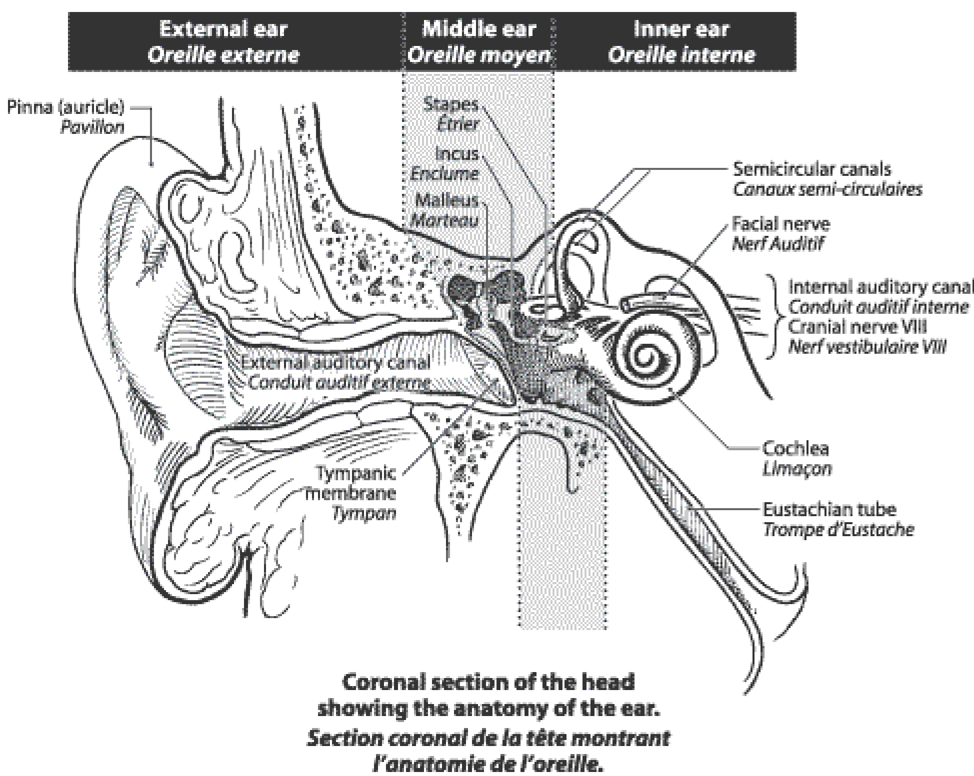

Figure 1: Anatomical drawing of the ear

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 potential 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 and smoking.

Of interest, one near 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

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 typically affects higher frequency hearing than those frequencies essential for communication (i.e. 500, 1000 and 2000 Hz) initially.
6. Four major factors determine the effects of exposure to noise overall:
    a. Overall noise levels.
    b. Spectral composition of noise.
    c. Duration and distribution of exposure during a typical workday.
    d. 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 ½ 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).
    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.

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 years. Around age 55-60 years 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 usually but not always.

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 presbycutic 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) and so on and so forth.

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 destroyed by trans-tympanic instillation of gentemicin with preservation of hearing while in the last resort the whole inner ear and its neurological connections to the brain can be destroyed surgically by a procedure called a labyrinthectomy.

4. Cochlear Otosclerosis

Otosclerosis usually results in a conductive hearing loss from stapes footplate fixation due to new bone growth in this area. 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. cisplatinum) 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 cisplatinum 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. 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 steroid).

Audiology

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

The following audiometric tests form the foundation for most assessments of hearing. These include what is called *conventional audiometry* and *impedance(immitance) 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.

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.

Impedence 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 (ie 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 (ie 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 2):

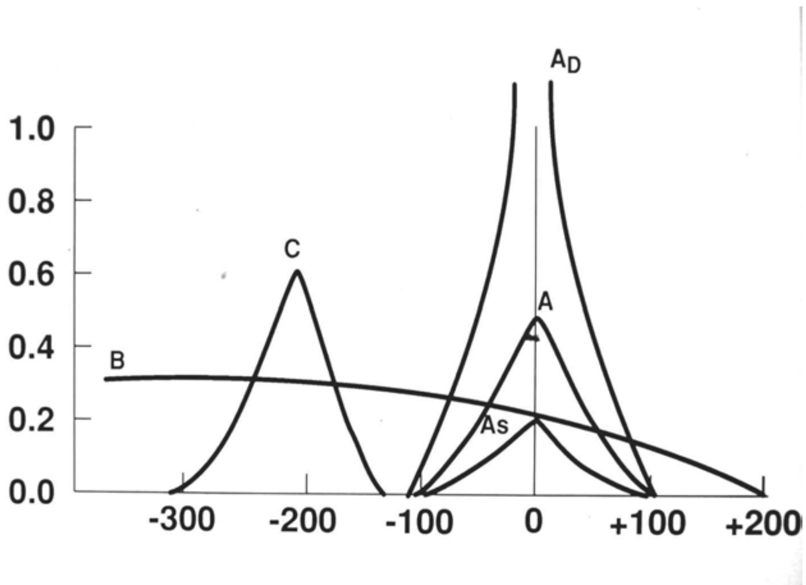

Figure 2: Schematic representation of tympanometry curves

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 followed 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.

Common Audiometric Configurations in Certain Disease Pathologies

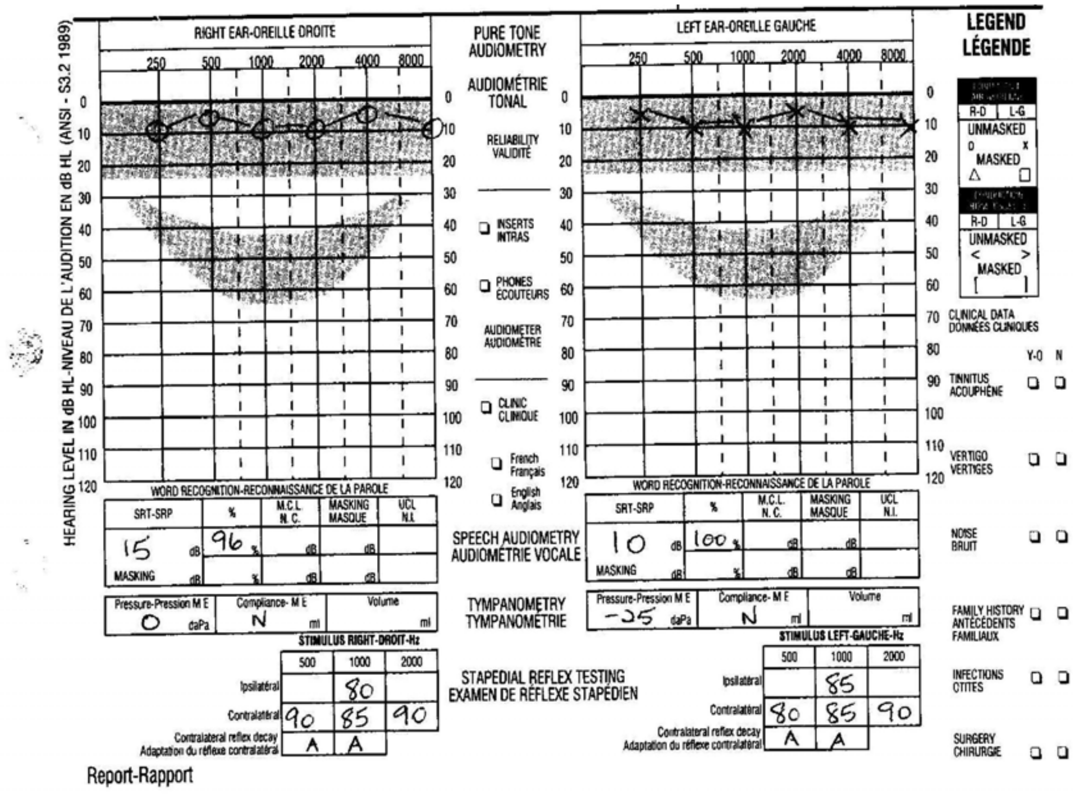

Figure 3: Normal audiogram

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, stapedial reflexes present and a normal A tympanogram noted.

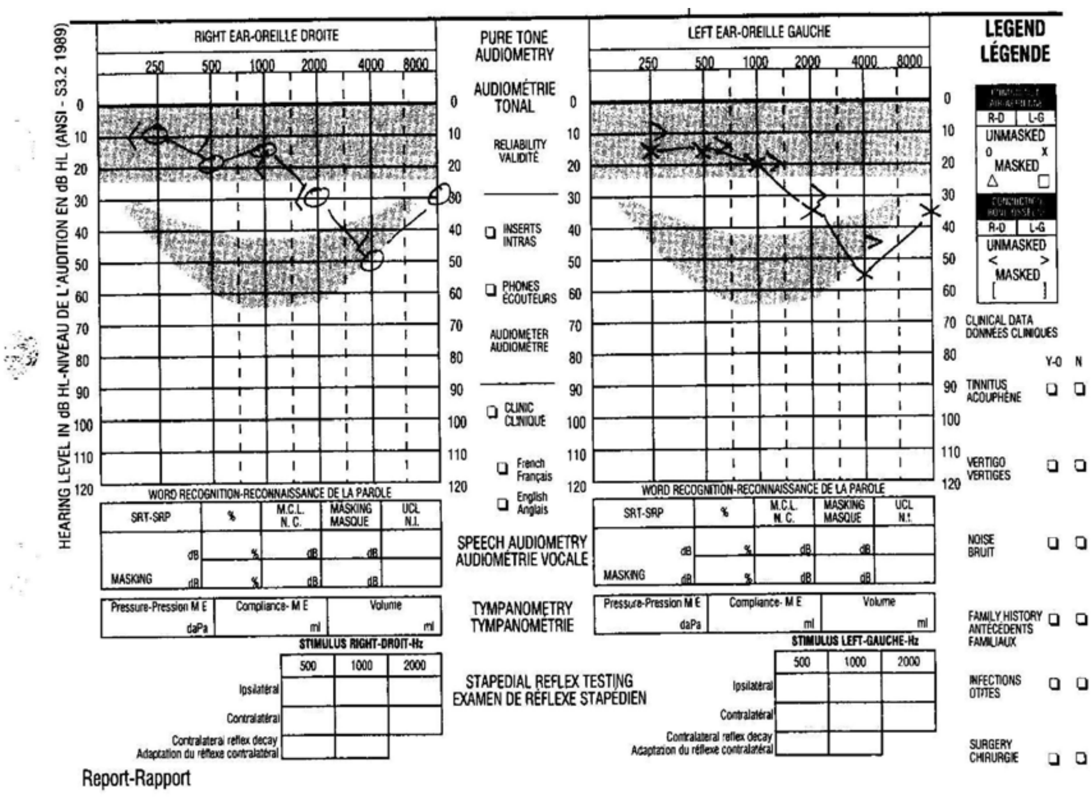

Figure 4: Audiogram in NIHL case

2. Presbycusis

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

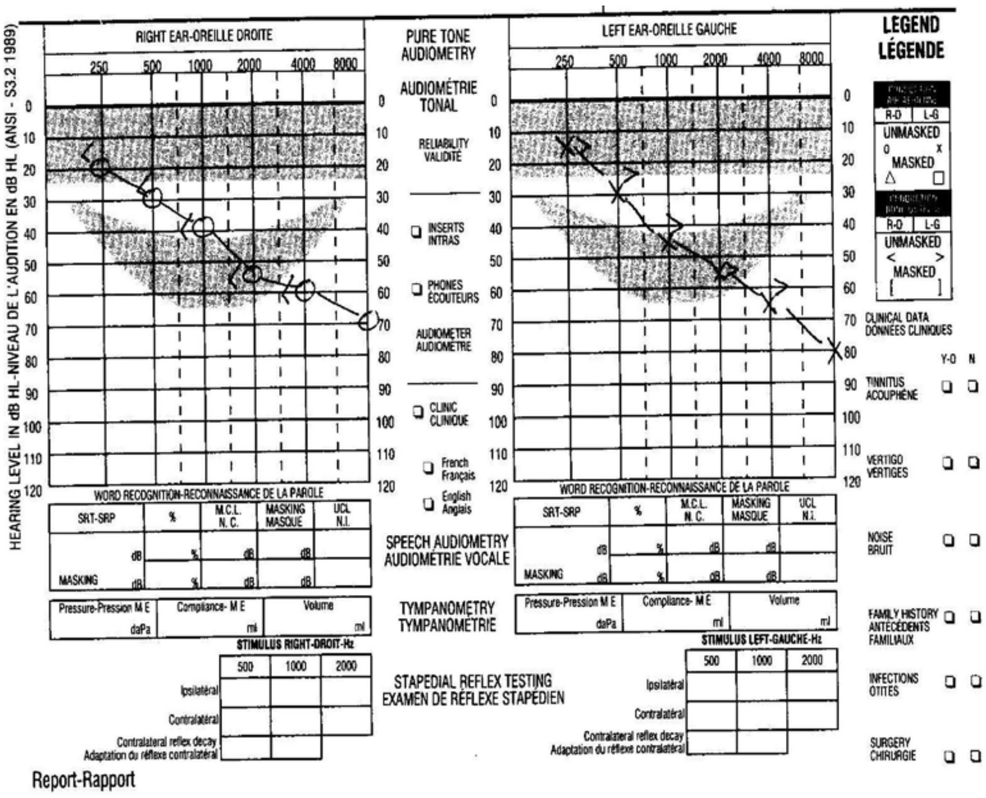

Figure 5: Audiogram in presbycusis case

3. Menière's Disease

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

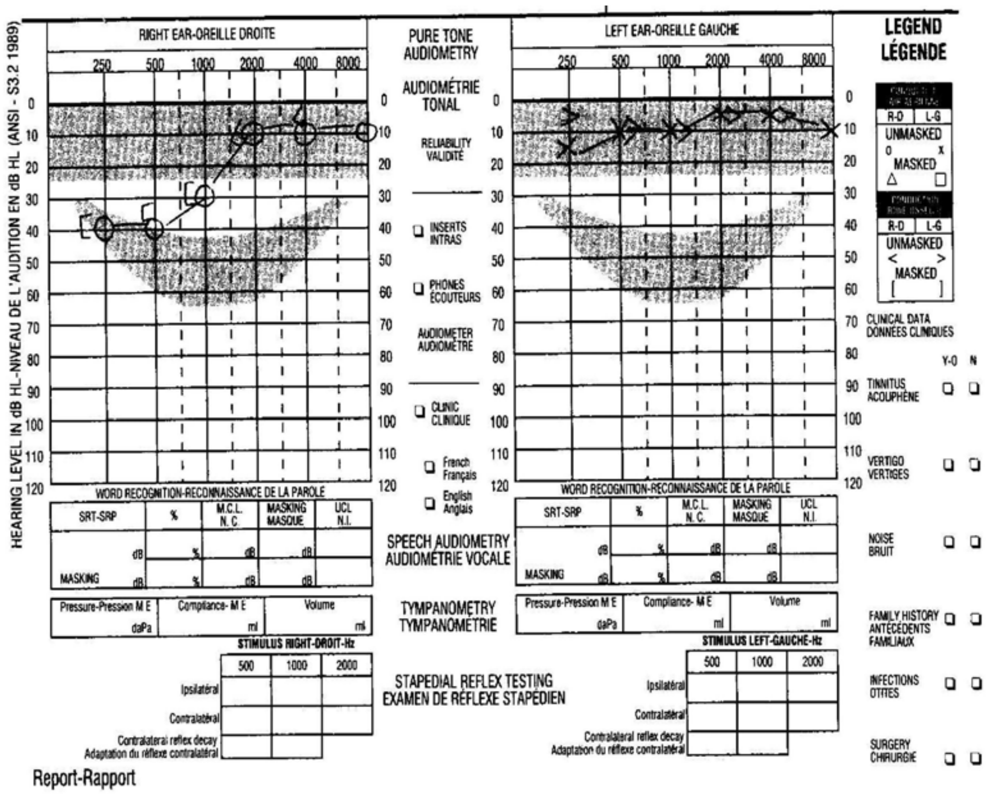

Figure 6: Audiogram in Menière's Disease (Right side affected)

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.

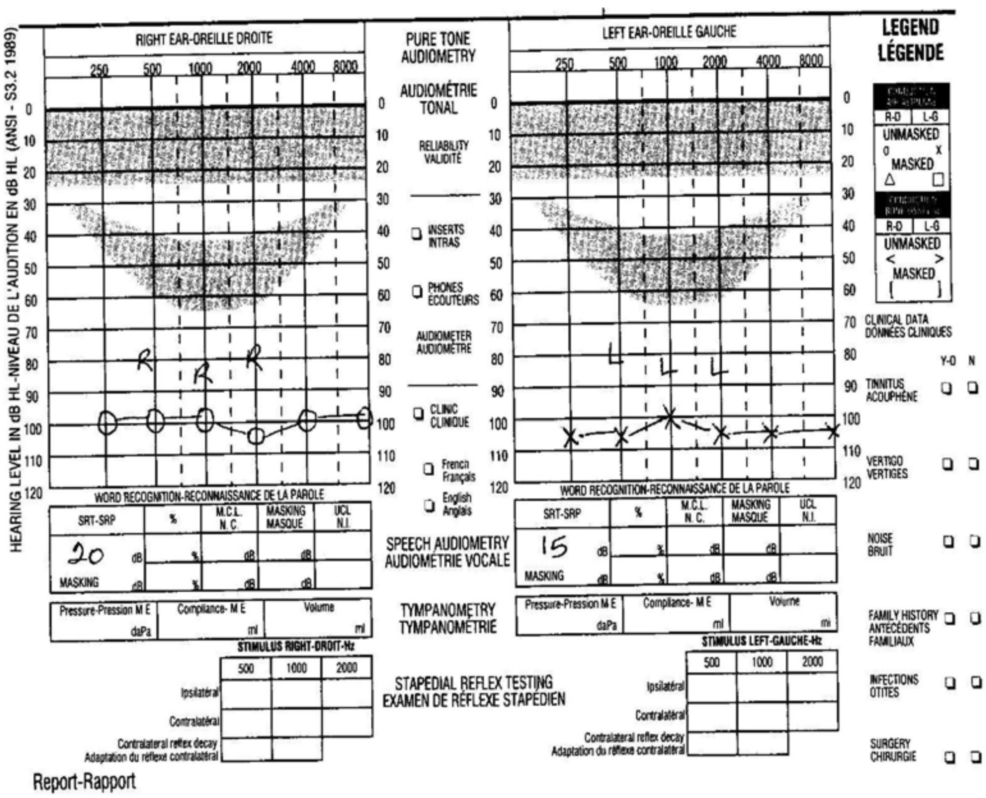

Figure 7: Audiogram in exaggerated hearing loss case

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 an 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.

References

Selected Texts

1. American Academy of Otolaryngology-Head and Neck Surgery Foundation Subcommittee on the Medical Aspects of Noise. Evaluation of People Reporting Occupational Hearing Loss 1998.
2. American Academy of Otolaryngology-Head and Neck Surgery Foundation: Guide for Conservation of Hearing in Noise. Edited by David Osguthorpe 1988.
3. American Medical Association: Guidelines to the Evaluation of Permanent Impairment. 3rd Edition 1990
4. Occupational Hearing Loss. Robert Thayer Sataloff and Joseph Sataloff. Marcel Dekker Inc. New York 1987
5. Occupational Medicine: State of the Art Review. Occupational Hearing Loss. Volume 10(3):July-September, Philadelphia, Hanley and Belfus, 1995
6. Noise Induced Hearing Loss by Lonsbury-Martin BL, Martin GK and Telischi FF, Volume 4, Chapter 126 in Otolaryngology-Head and Neck Surgery 3rd Edition. Charles Cummings, John Fredrickson et al, Mosby Press 1998.
7. Medicolegal Evaluation of Hearing Loss by Robert Dobie. Van Nostrand Reinhold, New York 1993
8. Diseases of the Ear: Clinical and Pathologic Aspects by Michael Hawke and Anthony Jahn. Lea and Febiger, Philadelphia 1987.

Selected papers and Supplements

1. Borg E, Canlon B Engstrom B. Noise Induced Hearing Loss. Scand Audiol 1995; Suppl 40:1

2. Lutman ME, Spencer HS. Occupational Noise and Demographic Factors in Hearing. Acta Otolaryngol (Stockh) 1991; Suppl 476: 74-84
3. Hinchcliffe R. The Age Function of Hearing-Aspects of the Epidemiology. Acta Otolaryngol (Stockh) 1991; Suppl 476: 7-11.
4. David AC, Ostri B and Parving A. A Longitudinal Study of Hearing. 1991; Acta Otolaryngol (Stockh) 1991; Suppl 476: 12-17.
5. Clark WW: Hearing: The Effects of Noise. Otolaryngol Head Neck Surg 1992;106: 669-676.
6. Segal s, Harrell M, Sharar A et al. Acute Acoustic Trauma: Dynamics of Hearing Loss Following Cessation of Exposure. Am J Otol 1998; 9(4): 293-298.
7. Corso JF. Age and Sex Difference in Pure Tone Threshold. Arch Otolaryngol 1963; 77: 385-392.
8. Corso JF. Support for Corso's Hearing Loss Model Relating Aging and Noise Exposure. Audiol 1992; 31: 162-167.
9. Rosler G. Progression of Hearing Loss Caused by Occupational Noise. Scan Audiol 1994; 23: 13-37.
10. Dobie RA. The Relative Contributions of Occupational Noise and Aging in Individual Cases of Hearing Loss. Ear and Hearing 1992; 13(1): 19-27.