Dizziness

Discussion paper prepared for

The Workplace Safety and Insurance Appeals Tribunal

October 1992

Prepared by:

Dr. Peter W. Alberti
Otolaryngologist

Revised: September 2016 by:

John Rutka MD FRCSC

Professor Department of Otolaryngology, |
University of Toronto

Staff Otologist/Neurotologist

University Health Network (UHN)

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

Dr Rutka is a professor of Otolaryngology-Head and Neck Surgery at the University of Toronto. Following fellowships in otoneurological surgery/medicine at the University of Toronto (1984-5) and Cambridge University (1985-6), he returned to Toronto to a subspecialty practice focused primarily in medical and surgical otology/neurotology. He was awarded the Sir William Osler Clinician Scientist award in 1987.

Dr Rutka is the chief fellowship mentor for the TWJ Foundation (UK) and the Peter and Melanie Munk Foundation (Canada) for subspecialty training in otology/neurotology. He is the co-director of the UHN Center for Advanced Hearing and Balance Testing and the UHN Multidisciplinary Neurotology Clinic. His chief academic interests have been

in areas of topical and systemic ototoxicty, canal based surgery for dizziness, the conservative management of patients with vestibular schwannomas, time trends in the surgery for chronic ear disease and recently the evaluation of dizziness in the head injured patient. His CV lists over 100 peer-reviewed publications and 4 text books in addition to numerous book chapters/ supplements/non-peer reviewed articles.

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 Discussion papers are not peer reviewed.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).

Table Contents

ix. Specific Causes of Vertigo/Dizziness Identified or Postulated to have a Traumatic Link from the UHN Neurotology WSIB Data Base

    a. Diagnoses from the UHN WSIB Neurotology Data Base (N=3,438)

    b. Sub-Group Analysis of the UHN WSIB Neurotology Data Base 1998-2014

x. Common and Controversial Causes for Posttraumatic Vertigo

    a. Common recognized causes for dizziness associated with trauma

        Posttraumatic BPPV
        Delayed endolymphatic hydrops (DEH)
        Traumatic vestibular loss from direct inner ear injury

    b. Controversial diagnoses for posttraumatic dizziness

        Cervicogenic vertigo
        Posttraumatic vestibular migrane
        Perilymphatic fistula formation
        Posttraumatic Meniere's disease and posttraumatic hydrops

xi. Issues that have been considered by the Tribunal

    a. What is the relationship between Meniere's disease/syndrome and trauma?

    b. What is the relationship if any between noise exposure and Meniere's disease and dizziness?

    c. Can there be a delay of onset of Meniere's disease following acute noise exposure?

    d. How severe a force (blow) is required to cause dizziness?

    e. What is the relationship if any between exposure to toxins e.g. mercury/solvents and dizziness?

xii. References

xiii. Selected Appendices for Further Reading

    Appendix 1: Evaluation of Vertigo (*from Evaluation of Vertigo in Office Based Surgery in Otolaryngology 1998; 71-78. Thieme. New York*)

    Appendix 2 :Physiology of the Vestibular System (*from Ototoxicity. Roland P and Rutka J ed. 2004: 20-27.BC Decker Inc. Hamilton, London.*)

    Appendix 3: Vestibular and Balance Disorders. (*from K.J. Lee's Essential Otolaryngology Head & Neck Surgery. Chan & Goddard Ed. 2016 11th Edition Vestibular and Balance*)

xiv. Glossary of Terms

Definitions

The terms "*dizziness*" and "*vertigo*" are used interchangeably in public. To the physician however, there are important distinctions between the two. It is important to realize that while *"all vertigo is dizziness, not all dizziness is vertigo"*

When used in the colloquial sense "*dizziness*" represents a feeling that virtually everyone has or will experience in their lifetime. It can encompass feelings such as light-headedness, a floating feeling, a feeling as if one is going to faint, a sense of giddiness etc. While dizziness does include "*vertigo*" physicians consider this term to have a special meaning that typically represents a hallucination/illusion of movement (often described as a spinning and circling sensation, a feeling of being pushed or pulled, that the ground is moving etc). The major physiologic distinction is that vertigo unlike dizziness is generally caused by an abnormality involving peripheral (inner ear and/or vestibular nerve) or central vestibular pathways. [1]

A Brief Description of Vestibular Physiology [2,3,4]

An individual's orientation (or balance) in 3 dimensional space is dependent on information gathered from vision, vestibular and proprioceptive (muscle, joint and sensory) receptors integrated centrally in the brainstem. Our balance to a large degree is automatic and exists on a seemingly subconscious level until pathology intervenes causing us to experience its ill effects.

The inner ear has specialized neural receptors for hearing (the cochlea) and for balance perception (the vestibular labyrinth). See Figures 1a,b. While the function of the cochlea will be largely understated in this Discussion Paper on Dizziness the reader is advised to see the previously published WSIAT Discussion Paper on Hearing Loss and Tinnitus (2013) should further information in this area be required.

Fig. 1a   Cochlea and Vestibular Apparatus

Superior & Inferior
Vestibular (VIII) nerves

Facial (VII)
nerve

Cochlear (VIII)
nerve

Cochlea

Superior
Semicircular Canal

Posterior
Semicircular
Canal

Lateral
Semicircular
Canal

Oval window

Round window

Fig. 1b   Vestibular Apparatus

Superior
Semicircular Canal

Posterior
Semicircular Canal

Utricle

Lateral
Semicircular Canal

Saccule

Cupula of Posterior
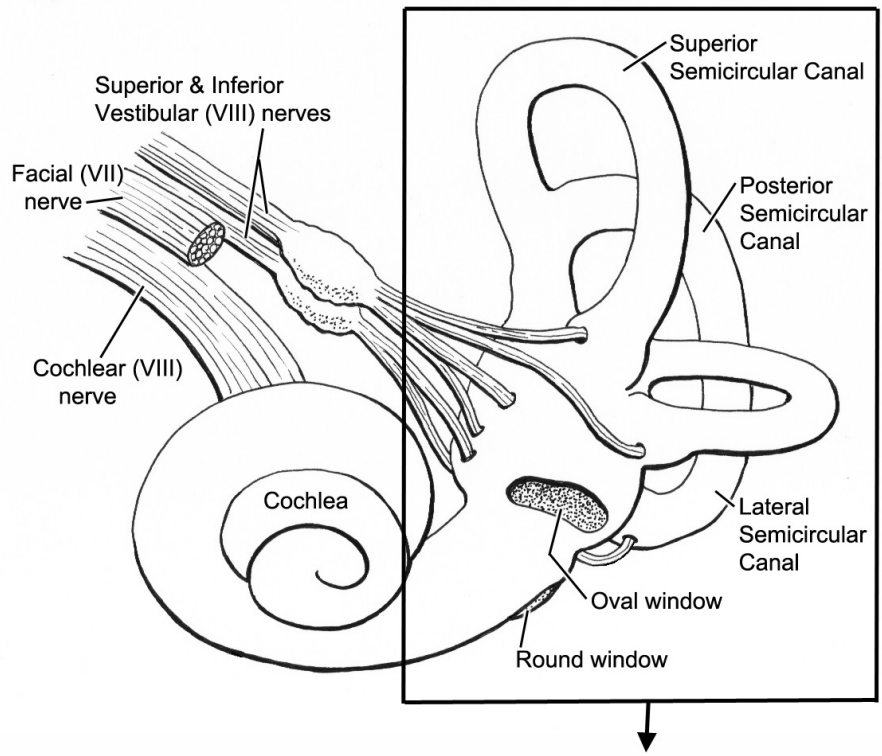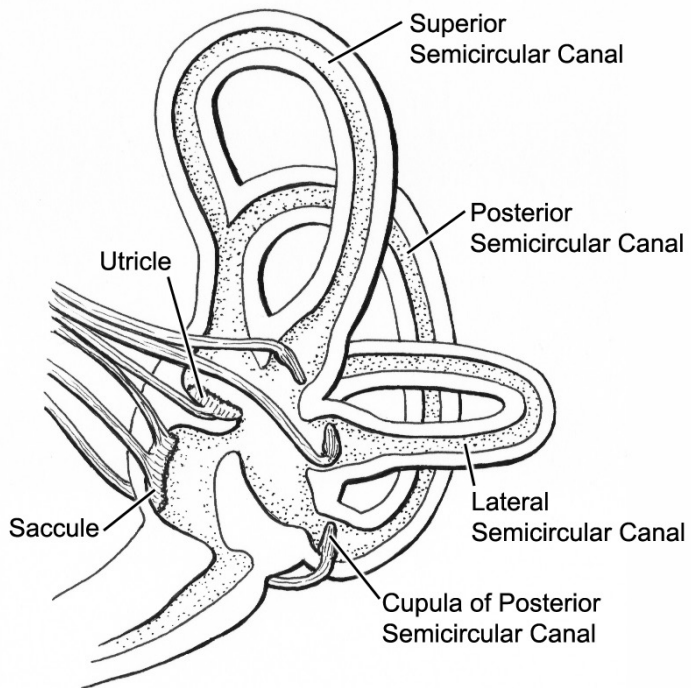Semicircular Canal

It is important to appreciate that the vestibular system is complex and often difficult to understand even for medical professionals at times.

The vestibular system has two broad functions – the maintenance of balance and the maintenance of stable gaze. The vestibular end organs comprise of the otolithic organs, (the utricle and saccule) and the three semicircular canals (lateral, superior and posterior). The semicircular canals are activated during rotational head movement and the otolithic organs during linear movements (or forces such as gravity).

The semicircular canals are paired structures. While the lateral canals are paired with each other, the superior canal on the left is functionally paired with the posterior canal on the right and vice versa. Corresponding equally paired (or conjugate) eye movements are produced in the plane of the stimulated canals. Stimulation of the semicircular canal beyond its resting discharge rate occurs when the cupula sitting atop the end organ of the ampulla is deflected as a result of endolymph (fluid) movement within the canal (remaining relatively still as a result of its inertia) when the head is moved. A similar type phenomenon occurs when linear accelerations cause shifts in the calcium carbonate lining of the otolithic organs. See Figures 2a,b and 3a,b

Fig. 2a   Cupula of Semicircular canal
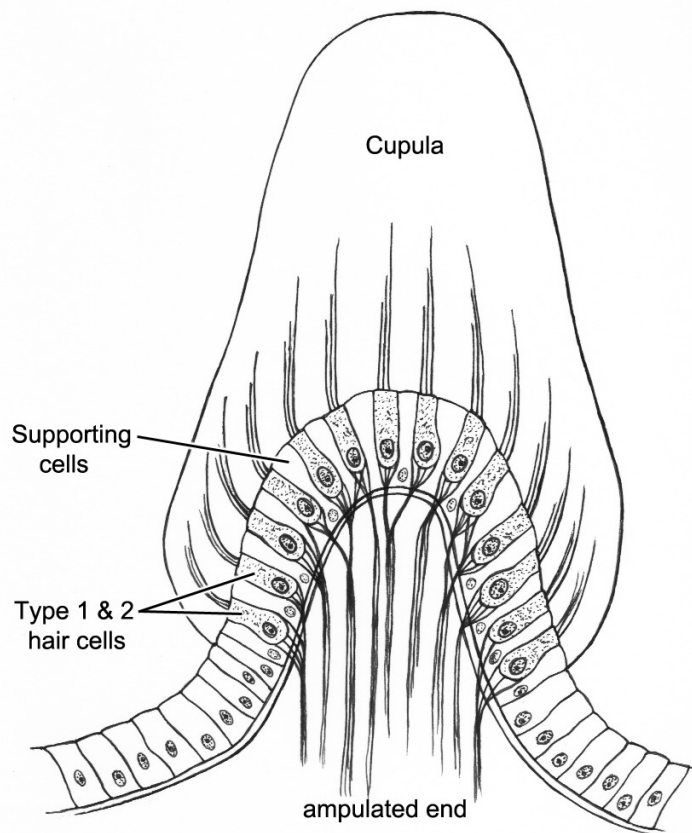
Fig. 2b   Otolithic Organs at Rest

Calcium carbonate crystals

Kinocilium

Stereocilium

Type I & II hair cells
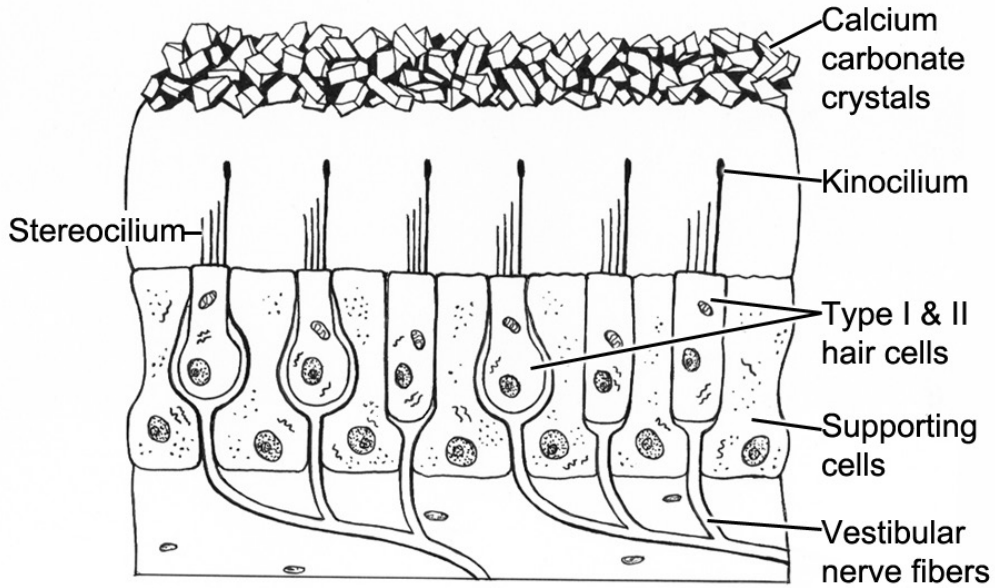
Supporting cells

Vestibular nerve fibers

Fig. 3a   Cupula Displacement in Semicircular Canal Following Movement

Plane of Movement

Cupula

Cupula displaced

Neural firing at rest

Neural firing with movement

Fig. 3b Otolithic Organs with Movement

Calcium carbonate crystals

Kinocilium

Stereocilium

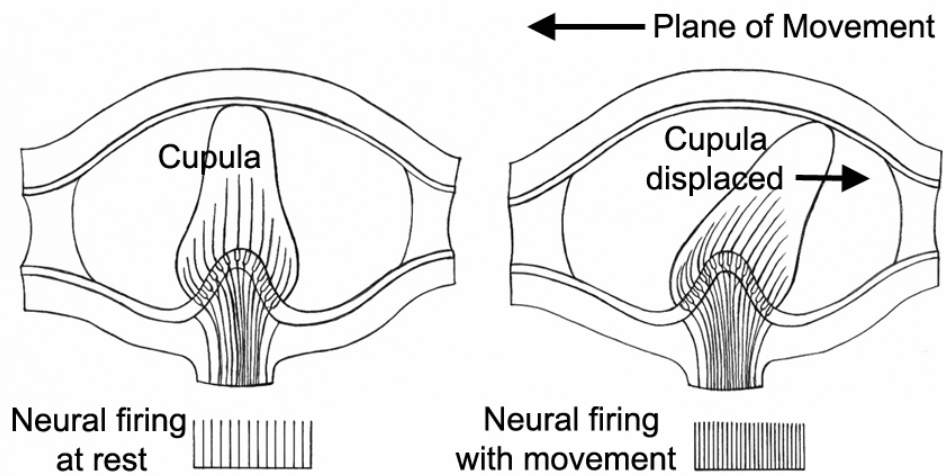Type I & II hair cells

Supporting cells

Vestibular nerve fibers

Movement of Otoconia

Plane of Movement
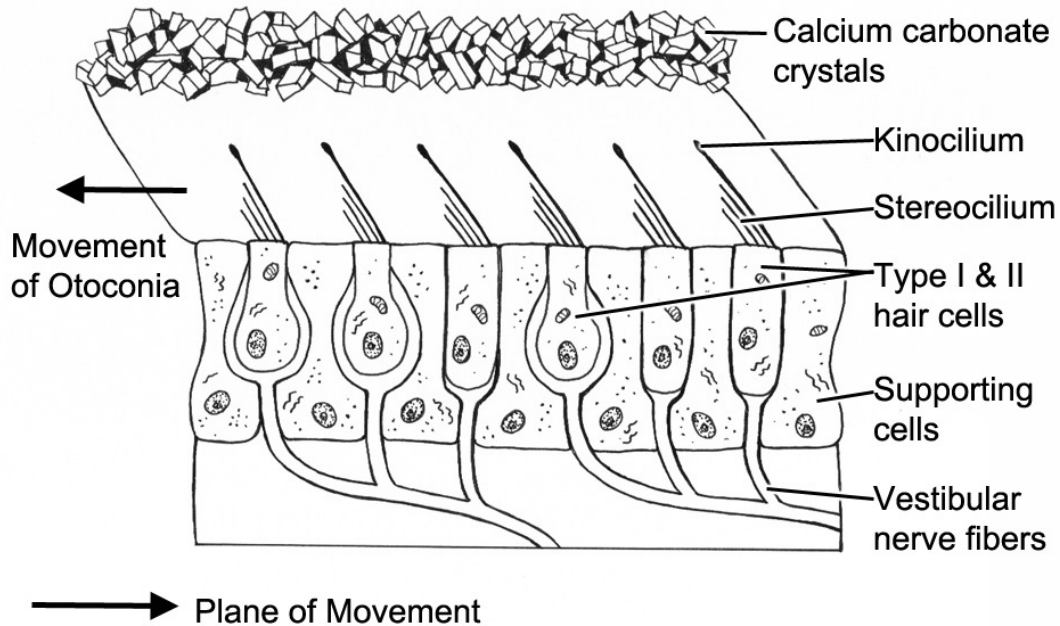
Complex electrical evoked activity both tonic (spontaneously generated) and dynamic (from a stimulus such as a head movement) generated electrical information is sent to the central vestibular nucleus in the brainstem via both the superior and inferior divisions of the vestibular nerve. Interconnecting neural pathways then traverse the medial longitudinal fasciculus of the brainstem on either side to land on the oculomotor nuclei of the eye movement nerves (the oculomotor, trochlear and abducens). Eye movement corresponding to head movement forms the basis for the vestibulo-ocular reflex (VOR) that serves to stabilize one's gaze with equal but opposite eye to head movement. The otolithic organs (saccule and utricle) provide information from linear acceleration perception that provides the substrate for our vestibulospinal tracts (VST). This allows us to generate the protective righting reflexes that prevent falls when we slip for example. Vestibulocerebellar and direct pathways to the emetic centers round off the neurological pathways that form the vestibular system. See **Vestibular Pathways** diagram below.

Vestibular Pathways
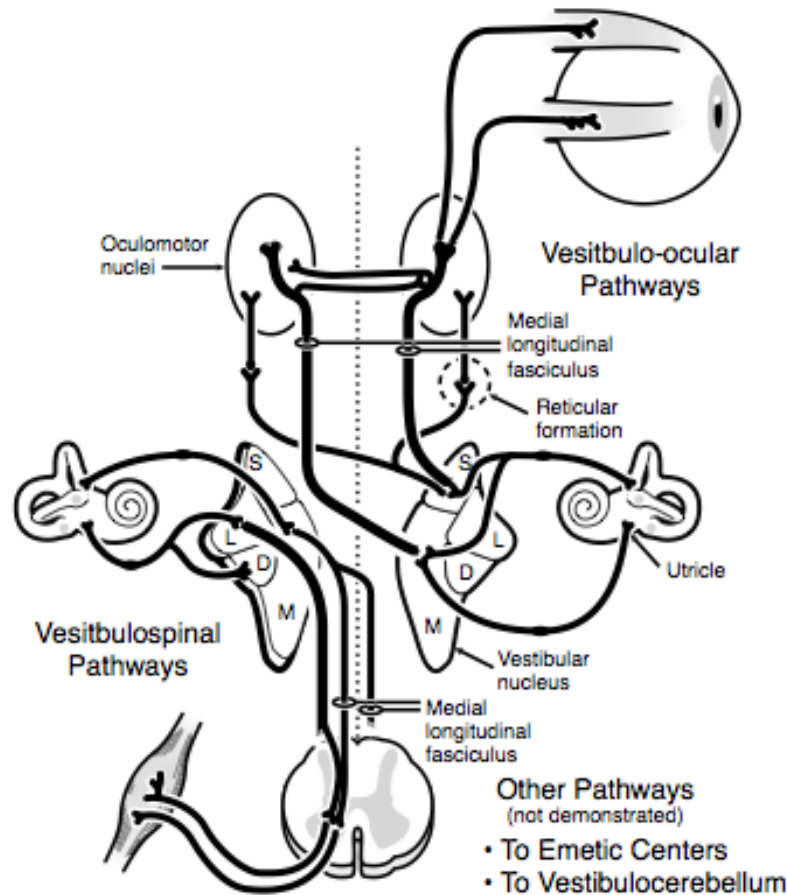
Vestibulo-ocular and Vestibulospinal Pathways (Taken from Rutka JA. Evaluation of vertigo. In:Binder WJ. Office Based Surgery in Otolaryngology. New York: Thieme; 1998,71-78.) [1]

Eye movements (also known as oculomotor movements) unfortunately do not have the ability to move the eyes fast enough to produce the quick compensatory eye movements necessary to stabilize gaze on the back (macula) of the retina. Defects in VOR function results in reduced dynamic visual acuity (DVA) from so called 'retinal slip' which in turn can lead to visual blurring during head movement.

When the head is stable, hair cells within the semicircular canals and otolithic organs have a baseline spontaneous (tonic) firing rate. Using the paired lateral (horizontal) canals as an example, when an individual's head turns to the left the ipsilateral (same sided) lateral semicircular canal will increase its firing rate while the right (opposite or contralateral) lateral semicircular canal will correspondingly decrease its firing rate. This difference in electrical activity is registered in the brainstem. Over the course of

an individual's life these differences have been associated with a head movement. Conflict occurs when pathology involving the vestibular system alters steady state balance or its function. As a result an individual at rest may feel as if they are moving when they are not. This is the classic description of vertigo- an illusion or hallucination of movement. In the setting of an acute vestibular loss one would also expect to see the physical sign of nystagmus and abnormalities in other vestibular generated pathways.

Over time the brain can compensate for changes in the baseline firing rate for a loss of vestibular function. However should weakness persist between sides then an individual never completely compensates and may complain of transient imbalance to a fast head movement for example.

In general terms an individual's actual *"balance"* or perceived sense of *"imbalance"* is a usually a much more complicated physiological process to assess. While vestibular function (both central and peripheral) plays a role in an individual's balance, information is also provided from visual and proprioceptive pathways (the ability to feel one's feet for example) centrally co-ordinated within the brain. Defects in one or more of the sensory senses above or if central pathology exists it can affect an individual's balance. To confound matters more, complaints of *"imbalance"* can often be subjectively magnified by psychological and/or functional features in the absence of significant physical pathology.

To summarize, the otolithic organs of the utricle and saccule are primarily responsible for upright balance and the perception of linear accelerations (both translational along the horizontal plane- think of braking quickly in a car and in the vertical plane-think of gravity perception and the space sickness that astronauts experience in its absence). The vestibulospinal tracts (responsible for the righting reflex that prevents you from falling) are predominantly otolithic in nature. The semicircular canals are primarily responsible for sensing angular acceleration movement of the head. This forms the basis for the vestibular ocular reflex necessary to stabilize gaze with active head movement.

Clinical Presentation

Dizziness and vertigo are common findings post head injury. While the pathophysiology of inner ear trauma leaves many unanswered questions, the labyrinth can be directly injured not only from fractures through its hard otic capsule (ie temporal bone fractures) but also from the shearing effects/pressure waves that can physically damage the delicate lining membranes and end organs. The effects of haemorrhage, subsequent inflammatory change and biochemical alterations affecting inner ear function also require consideration. Shearing injuries can also damage nerve root entry zones, cause diffuse axonal damage and result in petechial hemorrhages within the brainstem including the vestibular nuclei. Magnetic resonance imaging (MRI) is well positioned to look at the brain for these findings.

a.Unilateral Vestibular Loss

Loss of inner ear vestibular function acutely (ie following a basal skull fracture involving the otic capsule or hard bony lining of the inner ear causing direct mechanical injury) results in the disruption of vestibular pathways within the brain associated with specific signs/symptoms.

In an acute traumatic cochleovestibular (inner ear) loss not only will the patient experience hearing loss of varying degrees and/or tinnitus (unwanted head noise), they will also be vertiginous as well. Disruptions of vestibular ocular pathways will cause blurring of vision and the physical observation of nystagmus (ie a rapid rhythmical movement of the eyes with the fast phase of eye movement typically directed away from the side of lesion- this is known as a deafferentative type of nystagmus). The patient will seem ataxic, unco-ordinated and possibly impaired/drunk on the involved side from disruption of vestibulocerebellar pathways. When attempting to walk the patient will fall towards the side of lesion due to disruption of vestibulospinal pathways. By lying still they can avoid any movement that will make them nauseous to the point of vomiting from the adverse influence of the brainstem emetic pathways receiving vestibular input.

Over the course of time and under the influence of a healthy contralateral inner ear, brainstem vestibular nuclei and functioning vestibulocerelbellum, the process of compensation occurs. The individual becomes less vertiginous upon movement and nausea subsides. They become more active. If their vestibular loss is permanent however they may always feel a sense of transient imbalance with quick movement. Patients should be encouraged to be as active as possible in order to promote the compensation process- a hallmark indication for vestibular rehabilitation therapy.

Unfortunately should pathology involve the contralateral inner ear, the brainstem or the vestibulocerebellum then the compensation process may be incomplete resulting in the patient experiencing a permanent sense of "dizziness and imbalance".

b. Bilateral Vestibular Loss

Loss of bilateral vestibular function synchronously typically does not cause an individual to experience vertigo. The major complaint is *oscillopsia* or visual blurring with head movement (due to bilateral VOR impairment) and disequilibrium/imbalance (due to involvement of the otolithic pathways needed to stabilize upright balance).

Bilateral vestibular dysfunction is not common and appears idiopathic much of the time. It would be rarely expected to occur following trauma. It however can be seen in the context of Meniere's disease and from ototoxicity (from medications that are harmful to the inner ear especially).

Assessment of the Dizzy Patient

A relevant history and physical examination tailored to the vestibular system remain the most important elements in the assessment of a dizzy patient. Laboratory vestibular testing provides complementary information but must be always correlated to the patient's presentation and physical findings. Rarely will a lab test on its own provide a diagnosis.
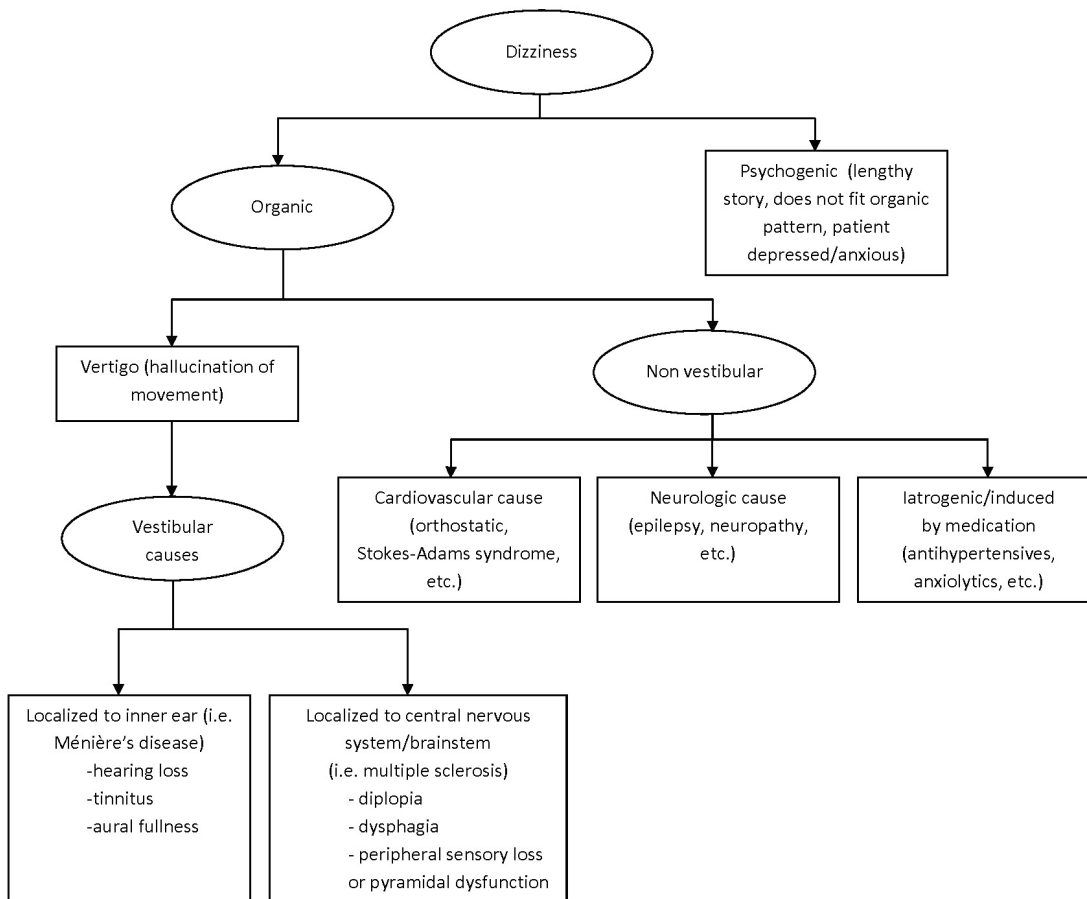
a. History

The history taking begins by asking what "dizziness" means by encouraging the individual to describe their experience (importantly avoiding the word "dizziness" if possible). As mentioned above dizziness can mean many things from a sense of giddiness or light-headedness to a sense of true spinning or tilting of one's environment. From here the examiner gathers an initial impression of whether the dizziness is organic or non-organic (ie psychogenic) primarily.

As a general rule most individuals with peripheral (inner ear or the vestibular nerve related pathology) vestibular dysfunction describe their attacks in a crisp well-defined fashion regarding onset, duration and the association with certain provocational factors (for example positional induced). The association with inner ear dysfunction requires enquiry for symptoms such as tinnitus (unwanted head noise), aural pressure/discomfort and hearing loss (permanent or fluctuant). When dizziness is primarily non-organic/psychogenic the history is typically lengthy and difficult for the examiner to appreciate, even understand. There are often many medically unexplained symptoms that cannot be tied together by known anatomical and physiological processes.

Vertigo can also arise from central nervous system (CNS) dysfunction involving central vestibular pathways (albeit less likely). The proximity of the nearby cranial nerve nuclei in the brainstem stresses the importance of asking for symptoms representative for focal neurological dysfunction (diplopia (double vision), dysphagia (difficulties swallowing), paresis (focal weakness), paresthesia (numbness of tingling) and incontinence of bowel or bladder activity). Loss of consciousness would not be expected and when present requires exclusion of other central cause(s) such as epilepsy or a cardiac arrhythmia. Common medication side effects often include dizziness (especially anti-hypertensives, anti-depressants, anxiolytics, anti-epileptics etc). The patient's medication profile should be documented and especially whether there was a temporal association when a new medicine commenced. See Evaluation of Vertigo flowchart below.

Evaluation of Vertigo Flowchart

Flow-chart of the history of a dizzy patient. (Taken from Rutka JA. Evaluation of vertigo. In:Binder WJ. Office Based Surgery in Otolaryngology. New York: Thieme; 1998,71-78.) [1]

b. The Neurotological Examination

A combined otological and neurological examination with specific attention to eye movements and vestibular pathways is required in an individual with dizziness.

The ears are examined primarily to assess the status of the tympanic membranes and to exclude external or middle ear pathology that might affect inner ear function. Should loud noise or forced straining (ie Valsalva manoeuvre, sneezing, coughing) by history cause vertigo/disequilibrium then it would be appropriate to perform the so-called "fistula" tests.* The Tullio phenomenon refers to dizziness resulting from loud hard

---

* A fistula is thought to arise when this is an anatomical or physiological abnormality involving either the surrounding bone of the otic capsule or the delicate lining membranes of the inner ear.

sound exposure. The Hennebert's phenomenon occurs when physical alterations in air pressure within the external or middle ear cause vertigo to occur. The key objective finding is the presence of nystagmus (simply defined as the presence of rapid and rhythmical fast and slow conjugate movements of the eyes) to the stimulus whether it be sound or pressure stimulation.

The neurological portion of the examination primarily assesses cranial nerves, cerebellar and oculomotor function. Eye movement pathways involved in smooth pursuit, saccades, vergence (convergence/accommodation), ocular fixation (visual stabilization) and VOR suppression are of particular interest as their neural substrates tend to be diffuse within the brain and their function can be affected by central pathology (structural, metabolic, degenerative etc). The eyes are also carefully examined for spontaneous nystagmus (see paragraph above) that is the cardinal sign for vestibular dysfunction, both peripheral or central. Gait (regular and tandem) should also be checked with eyes open and closed.

Where relevant one can also assess deep tendon reflexes, muscle strength, vibratory sense and proprioception.

Special clinical tests of vestibular function that should be performed in all patients with dizziness include:

- **The head thrust (or Halmagyi) manoeuvre**- This physiologic test of high velocity/acceleration VOR activity determines whether the eyes move in an equal but opposite direction to a fast head movement. A VOR defect/loss is typically suspected when the eyes demonstrate the presence of re-fixation *(overt)* saccades required to match the head movement. It however should be remembered that a normal head thrust manoeuvre however does not necessarily imply that the VOR is normal. Certain individuals for example can insert a mid-head rotation quick eye movement (a so-called *covert* saccade) that may be imperceptible to the observer.

- **Headshake test-** Rapid horizontal shaking of the head for approximately 20-25 seconds may result in the finding of transient nystagmus directed away from the ear involved by pathology. It is best seen by examining the patient with Frenzel's glasses on their eyes to prevent visual fixation from occurring. In general terms post headshake nystagmus is typically related to the degree of vestibular loss on the involved side in a complex physiologic process that also involves a region called the central velocity storage area in the brainstem.

- **Dynamic Visual Acuity Testing**- The ability to focus during active head movements depends on an intact VOR. When the VOR is defective bilaterally there is visual degradation during head movement leading

to oscillopsia (in this context oscillopsia is defined as visual blurring with head movement). An individual's dynamic VOR can be assessed while comparing the lines clearly seen at rest while reading a Snellen (a conventional eye) or a Logmar chart compared to those clearly seen during active head movement. Loss of visual acuity greater than 5 lines (static vs dynamic testing) would imply the presence of bilateral vestibular pathology.

- **Hallpike's manoeuvre**- Known alternatively as the Barany-Nylen manoeuvre in other parts of the world, this test remains an important one to perform on any dizzy patient owing to the relatively high prevalence of Benign Paroxysmal Positional Vertigo (BPPV). In the manoeuvre the patient's head is taken from the sitting to the lying position on one side. The patient's eyes are then examined for the presence of nystagmus and whether they are symptomatic for dizziness. They are then brought upright quickly and their eyes are again examined for nystagmus and/or whether they experienced subjectively dizziness. The same manoeuvre occurs on the other side. With a positive test result comes a confident diagnosis for BPPV. See figure 4.

Fig. 4   Right Hallpike's Manoeuver
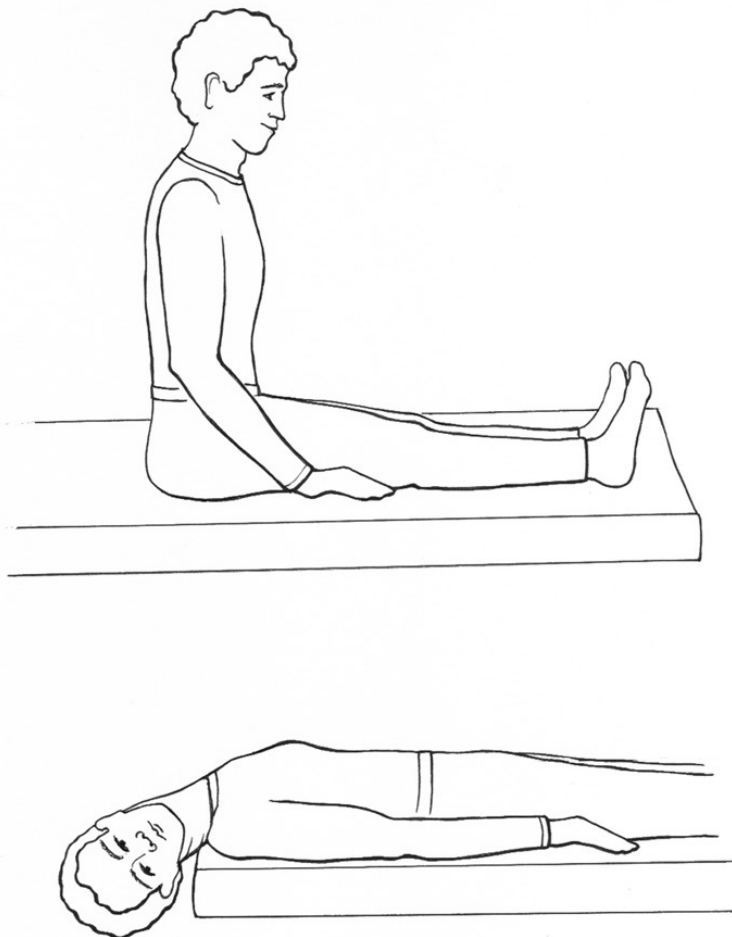
- **Vestibular ocular reflex suppression (VORS)**- Inability to suppress physiological nystagmus during rotation suggests the presence of a defect in the vestibulocerebellum (the phylogenetic oldest developed part of the cerebellum consisting of the flocculus, nodulus, uvula and para-uvula)

Relevant Laboratory Audiological and Vestibular Testing

Laboratory testing results should always be clinically correlated to the presentation of the dizzy patient. Rarely will a laboratory test independently make a diagnosis that cannot be made on clinical grounds. Results of laboratory testing however can be used to quantify function and further establish the diagnosis where required.

Audiometry

a. Audiogram - The standard test for assessing an individual's hearing can be recorded on a graph or digital format. In conventional pure tone audiometry an individual's hearing at certain frequencies is measured against the minimal intensity of sound (in dB) necessary to hear the presented tone.

Other components of the audiogram include the measurement of both the *speech reception threshold (SRT)* and *speech discrimination scores (SDS).* In SRT testing complex words with equal emphasis on both syllables (so-called spondaic words such as "hotdog", "uptown", "baseball" etc.) are measured 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. For SDS testing 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) approximately 40dB above their speech reception threshold (SRT) in the tested ear. Most individuals with normal sensorineural hearing should get over 80% of the words correct. When speech discrimination scores are especially poor a lesion involving the cochlear nerve may be present (ie acoustic neuroma/vestibular schwannoma) and further investigation is necessary.

b. Evoked Response Audiometry

The ability to measure minute electrical potentials following sound stimulation of the cochlea forms the basis of an evoked response that provides information concerning electrical activity within the cochlea (electrocochleography), along the cochlear nerve and in the brainstem (the auditory brainstem response or ABR). An experienced tester, typically an audiologist, is required to perform these technically demanding tests.

i. Electrocochleography (ECoG)

Electrical activity in the cochlea is measured during the first 2 msec of cochlear stimulation. ECoG's chief value lies in its ability to identify wave 1 of the auditory brainstem response (ABR) and whether waveform morphology is suggestive for changes that might occur in Meniere's disease and less commonly in canal dehiscent syndromes or a perilymphatic fistula. Increased summating potential to action potential (SP/AP) ratios typically > 0.30 appear to correlate with the phenomenon of endolymphatic hydrops (the pathophysiologic change in Meniere's disease).

ii. Auditory Brainstem Response (ABR)

Synonymous with the term auditory brainstem evoked potentials (BEPs) this test measures electrical waveforms during the first 10 msec following cochlear stimulation. Waveform morphology is thought to arise initially in the cochlear nerve and then from various relay stations in the brainstem with the electrical response travelling along to the higher cortical centres of audition. Changes in waveform morphology and latency of the ABR can be quite helpful in the assessment of an individual where an asymmetric sensorineural hearing loss is present if an acoustic neuroma/vestibular schwannoma is suspected. The integrity of intrinsic brainstem function is also indirectly tested as pathology (such as multiple sclerosis) can affect the electrical transmission of the ABR.

Vestibular Testing [4]

Formal vestibular testing may help in the identification of the site, side and quantification of vestibular function not identified from history or on physical examination. Its secondary roles also help identify those likely to benefit from vestibular rehabilitation therapy, to assess recovery of vestibular function and whether there might be a contraindication from interventions (both chemically ablative or those involving surgical deafferentation). Vestibular testing may also help determine if an intervention that was expected to result in vestibular deafferentation has been successful.

a. Electronystagmography/Videonystagmography (ENG/VNG)

This common performed test of vestibular function records eye movements with conventional recording electrodes around the eyes (the basis for the ENG) or directly from an infrared video camera with digital video capture technology (the basis for the VNG).

Measurement activities include an assessment for/of:

- spontaneous or gaze evoked nystagmus

- oculomotor function (pursuit, saccade, optokinetic and fixation pathways) which is an indirect measure of CNS integrity

- positional and positioning nystagmus and

- the caloric response

From the metrics provided the caloric test allows for comparison of lateral semicircular canal activity by comparing one ear to the other following thermal stimulation. In bithermal caloric testing, water of different temperatures (alternatively at 44°C and 30°C or 7°C above and below mean body temperature) irrigates the ear canals. The mnemonic COWS (cold opposite , warm same) is used to describe the expected direction of nystagmus generated from thermal stimulation. The maximum velocity of nystagmus generated is recorded and studied by the Jongkee's formula which looks specifically at the excitability difference (the % difference between sides is assumed to be reflective of relative vestibular function). It is defined by the following formula:

$$\frac{[(44R + 30R) - (44L + 30L)]}{44R + 30R + 44L + 30L} \times 100\%$$

In many laboratories an excitability difference > 20% would be considered compatible with a significant difference in activity between ears. While the test is not without concern (ie it is not truly a physiologic test of how the inner ear functions, the results can be affected by pathology involving the ear canal, middle ear and mastoid, the assumption that if lateral canal function is equal then this implies normal inner ear activity in its other parts etc) it still remains the workhorse for conventional vestibular testing

b. Vestibular Evoked Myogenic Potential (VEMP) testing

When brief loud sounds (0.1ms, >100dB) are presented to the ear they would be expected to primarily stimulate the cochlea. Basic science research has also identified that they can stimulate the otolithic organs producing a myogenic response in nearby muscles. Short latency responses from the anterior neck and extraocular muscles to sound stimulation have been collectively known as vestibular evoked myogenic responses or VEMP's.

Surface recording electrodes placed on the sternocleidomastoid muscle in the neck can be used to record a cervical VEMP (cVEMP) response. When placed around the lower rim of the orbit in close proximity to the inferior oblique muscle they can record an ocular VEMP (oVEMP) response. cVEMP responses are thought primarily to arise from the saccule and oVEMP responses from the utricle.

The clinical advantage in cVEMP and oVEMP testing rests in their ability to provide comparative information concerning the otolithic organs and to also indirectly assess the integrity of the vestibular nerve (both superior and inferior divisions). While both tests are technically easy and relatively quick to perform rectification (for unwanted muscle bias) is often required to increase reliability.

Unfortunately age, an inability to generate sufficient muscle tone in the neck and any form of middle ear pathology resulting in a conductive hearing loss may result in increased threshold or absent VEMP responses. Nevertheless increased amplitude responses together with decreased thresholds for stimulation may be identified in individuals with Meniere's disease, a superior canal dehiscent syndrome (SCDS) or a perilymphatic fistula. [5]

c. Video Head Impulse Testing (vHIT)

Lightweight goggles with built in high speed recording cameras capture eye movements that are coupled to a gyroscope measuring head rotation. This forms the basis of this test used to assess horizontal semicircular VOR function with the head thrust manoeuvre. It is a physiologic study of how the eyes move relative to a fast head movement. Under normal circumstances this should be unity (eye movement/ head movement=1). Reduced gains to the rotated side would imply a defect in the VOR. The technology also captures the presence of refixation saccades. Overt (*"catch-up"*) saccades are visible to the naked eye clinically. Covert saccades are embedded in a corresponding eye to head movement that occurs so quickly they are difficult to see (there is speculation they may indicate a better degree of central compensation for the underlying VOR impairment).

Other advantages of the vHIT test include its not being affected by middle ear pathology (unlike caloric and VEMP testing) and being able to test vertical canal function (both superior and posterior) additionally. Disadvantages are usually technical in nature as there may be goggle slippage and associated artefact from head impulses/rotations > 200-300°/s.

d. Magnetic Scleral Search Coil Testing (MSSCT)

In what would be considered the gold standard for VOR testing a silicone contact lens with an embedded copper wire is placed in direct contact with the cornea and surrounding conjunctiva under topical anesthesia. When an individual is placed in the environment of a magnetic field, movements of the eyes cause distortions that can be recorded.

The technique accurately records extremely high velocity eye movements (> 300°/s) and therefore is considered ideal for capturing ultra high velocity VOR function. It may also be applied to rotational chair testing (described below) when oscillating frequencies > 2 Hz.

MSSCT however is an invasive (there are minor risks for eye irritation/injury) and labour intensive study requiring expensive technology and is not utilitarian when compared to ENG or VEMP testing. For this reason testing it is usually available only in tertiary centres often under research settings. (6,7)

e. Rotational Chair Testing

In this study conventional ENG recording electrodes or VNG recording devices capture the horizontal VOR during sinusoidal rotations in a chair. Information from both ears can be recorded simultaneously. Other paradigms include further testing for constant angular acceleration, impulse angular acceleration and what is called a velocity step test. Phase measurements (looking at chair velocity to the maximum slow phase velocity of nystagmus generated) and the symmetry/asymmetry of recorded eye movements are used to identify if a defect in the horizontal VOR exists. New off-vertical axis rotation technology has been used additionally to assess otolithic activity.

While the test is more physiologically based (when compared to caloric testing), the equipment is expensive and most chairs are limited in sinusoid rotations up to 2 Hz which might miss higher velocity VOR dysfunction better captured on vHIT or scleral coil studies.

f. Clinical Posturography and Computerized Dynamic Posturography (CDP)

Home modified clinically developed testing or trademark patented (ie Equitest ®) technology is available for the overall assessment and quantification of balance function. A balance platform measures centre of pressure changes and measures of body sway evaluate an individual's balance in a series of tests involving visual, somatosensory and vestibular inputs. Information is provided through sensory organization tests (evaluating anterior-posterior body sway with eyes open or eyes closed) on a fixed or sway referenced surface (usually a foam surface), motor control tests (which assesses an individual's ability to recover from external provocation such as forwards/backwards platform movement) and adaptation tests (which assesses motor adjustments made when the platform tilts up or down).

While not particularly specific/sensitive for vestibular dysfunction on its own, functional limitations in performance identified during testing seem to correlate well with the Dizziness Handicap Inventory (DHI) - a well-validated tool reflective for how dizziness may affect an individual's quality of life. Posturography may also be used during the rehabilitation process to provide further information on a patient's progress. Trademark patented technologies however are typically expensive which is often the rate limited step preventing more widespread clinical adoption.

What Specialists Commonly See in Clinical Practice-The Multidisciplinary Neurotology Experience at the University Health Network [8,9]

To provide further insight into the common causes of dizziness seen in clinical practice, results from the internationally recognized UHN Multidisciplinary Neurotology Clinic are presented.

While dizziness can be a common complaint in mild head injury (along with headaches and emotive/cognitive change) objective evidence for vestibular impairment (both central or peripheral) is not always identified (see Head Injury and Dizziness section). Although the causes for dizziness will vary from centre to centre (whether it is in the GP's office or the emergency department for example) results from highly specialized and dedicated multidisciplinary tertiary referral clinics do provide a wealth of well studied demographic information.

Previously published longitudinal cohort studies from the University Health Network (UHN) Multidisciplinary Neurotology Clinic in Toronto have revealed the following diagnostic categories for dizziness at presentation.

Diagnosis (N=1000)	%
Peripheral vestibular (inner ear related)	65 %
Central vestibular (CNS related)	7.1 %
Mixed (both central and peripheral)	5.4 %
Psychogenic (functional or non-organic)	8.3 %
Unknown	13.3 %

Inner ear disorders accounted for the majority of visits to this specialized clinic. The incidence of presentation demonstrated that well over 50% of patients presented with either the condition of benign positional paroxysmal vertigo (BPPV) or a recurrent vestibulopathy (RV). While Meniere's disease and vestibular neuronitis (acute viral labyrinthitis) have the most spectacular of clinical presentations their incidence is relatively low by comparison.* Continued study of additional patients referred to the multidisciplinary clinic have demonstrated relative consistency in presentation over the years.

Common Inner Ear Disorders (N=650)*	%
Benign positional paroxysmal vertigo (BPPV)	33 %
Recurrent vestibulopathy (RV)	25 %
Vestibular Neuronitis (VN)	12.1 %
Meniere's Disease	5.7 %
Others	7.4 %*
Unknown	16.8 %

*For the clinical descriptions of BPPV, RV, VN and Meniere's disease please see the section "Common Peripheral (or Inner Ear) Disorders Associated With Vertigo"

An important clinical concept to appreciate in these studied individuals was the presence of more than one peripheral vestibular disorder in the same person over time. In the first 1000 patients studied approximately 12 % had more than one recognizable yet distinct inner ear disorder identified. For example it would not be

unusual for an individual following a vestibular neuronitis to evolve into an RV and then into BPPV over time.

The **Others** category referenced above consisted of relatively rare but interesting causes for peripheral vestibular dysfunction which included ototoxicity (both systemic and topical), delayed endolymphatic hydrops or DEH (considered to be a variant of Meniere's disease), drop attacks (the so - called otolithic crisis of Tumarkin), complications of chronic ear (ie mastoidectomy) or stapes surgery and neurosyphilis.

Central nervous system causes for vertigo for the most part were relatively rare by comparison but heterogeneous in presentation. The presentation of certain CNS disorders however appear age related to a large degree (ie cerebrovascular disease and multiple sensory deficit disorders tended to affect most individuals after the 7th decade of life primarily)

Common CNS Disorders (N=71)	%
1. Idiopathic vestibulocerebellar degeneration**	27.5%
2. Cerebrovascular disease (CVA, TIA, VBI)	21.0%
3. Multiple sclerosis	14.5%
4. Multiple Sensory Deficits (MSD)	10.9%
5. Others (encephalitis, PSP, tumors etc)	26.0%

Key: CVA=cerebrovascular accident, TIA= transient ischemic attack, VBI=vertebrobasilar insufficiency, PSP=progressive supranuclear palsy

** The term cerebellar ataxia with bilateral vestibulopathy (CABV) has been recently coined to describe individuals with evidence for both peripheral and central vestibular degeneration.

Common Peripheral (or Inner Ear) Disorders Associated With Vertigo

While causes for dizziness can be exhaustive in any given text, it should be understood that "common things are common" Table 1 below describes the common types of peripheral vestibular disorders usually seen in clinical practice. They represent a simple yet practical guide to the most common presenting disorders seen. The disorders can be differentiated in general terms by the duration of vertigo and whether they are associated with auditory dysfunction (ie hearing loss, tinnitus and/or aural fullness)

Table 1

Five common causes of inner ear dysfunction [1]

Disorder	Duration of vertigo	Hearing loss	Tinnitus	Aural fullness
BPPV	seconds	-	-	-
Meniere's disease	minutes to hours	Fluctuant SNHL*	+	+
Recurrent vestibulopathy	minutes to hours	-	-	+/-
Vestibular neuronitis (VN)	days to weeks	-	-	-
Acoustic neuroma (Vestibular Schwannoma)	"imbalance"	Progressive loss with poor speech discrimination	+	-

*SNHL=sensorineural hearing loss

a. Benign Positional Paroxysmal Vertigo (BPPV) [10]

Known as benign position vertigo (BPV) additionally, BPPV is the most common cause for vertigo identified clinically. In its classic description patients experience short-lived attacks of vertigo (typically lasting for 5-30 seconds) associated with certain provocative head movements (ie looking up, bending over, rolling over in bed to the affected side). Most of the time BPPV occurs spontaneously but can be frequently identified following head injury, following viral disorders involving the inner ear and after ear surgery (mastoid and stapes surgery especially). It can be unilateral or bilateral in its presentation.

The pathophysiology for this condition is generally thought to arise when particulate debris (usually thought to represent displaced otoconia from the utricle and saccule) enters the posterior semicircular canal and moves freely within the canal following head movement (canalolithiasis) or fixes itself to the cupula of its ampullated end (cupulolithasis). Both horizontal and superior semicircular canals can be affected by the same pathological process but to a lesser degree (the posterior semicircular canal is the most gravity dependent canal in the upright position).

In classic posterior canal based canalolithiais (which seems the best fit for BPPV overall) the Hallpike's manoeuvre on the affected side causes an individual to experience a sense of vertigo following a latent time frame of a few seconds. There is an associated geotropic (towards the ground) rotatory beating nystagmus that lasts 5-30 seconds which crescendos and then decrescendos over this timeframe. The patient is often nauseous to the point of vomiting. When brought upright from the head hanging position a reversal of the nystagmus is found (an ageotropic reversal) lasting a shorter duration. Again the patient may be vertiginous and nauseous. Repeat testing generally seems to fatigue the response.

Most cases of BPPV tend to resolve spontaneously although recurrence is not uncommon (especially in the post traumatic setting). Physical therapy exercises have proven effective in the management of this condition. Commonly performed therapies include the Epley manoeuvre (or particle repositioning procedure), Brandt-Daroff exercises for habituation/adaptation and the Semont's libertary manoeuvre.

Should BPPV not resolve spontaneously or prove recalcitrant to physical therapy manoeuvres then surgical options may be considered. Procedures such as labyrinthectomy (if the affected individual has no useful hearing in a deafened affected ear), vestibular neurectomy and singular neurectomy have all had their advocates historically. At present posterior semicircular occlusion surgery has largely superseded all these procedures for the safe/efficacious management of this condition. [11]

b. Meniere's Disease

Meniere's disease can vary in its presentation (not always presenting with all the typically expected symptoms together). In its classic description patients experience recurrent attacks of episodic vertigo lasting minutes to hours associated with tinnitus, fluctuant sensorineural hearing loss (typically in the lower frequencies initially) often associated with a sense of pressure or fullness in the affected ear. Established variants include the Lemoyez syndrome (resolution of hearing loss and tinnitus with the onset of a vertiginous attack), the otolithic crises of Tumarkin (sudden unexplained falls or drops from otolithic dysfunction affecting vestibulospinal tracts) and cochlear hydrops (fluctuation of hearing, tinnitus and/or aural fullness without a vertiginous attack).

Meniere's disease is estimated to occur in 1/2500 people with peak incidence between the ages of 30-60 years. Estimates however are invariably obscured by the lose entry criteria applied in most published epidemiologic studies. Unilateral involvement is typical but over a patient's lifetime it its estimated that bilateral involvement may occur upwards of approximately 50%. Previous demographic studies from the UK suggest that approximately 95% of patients will present with all 3 symptoms for Meniere's disease within 5 years of initial symptom onset. Periods of remission and exacerbation remain the rule. [12]

The pathophysiology is generally thought to arise from idiopathic endolymphatic hydrops (distention of the delicate lining membranes in the inner ear). This in turn leads to membrane ruptures within the inner ear resulting in an attack. The Na+-K+ theory from the admixing of inner ear fluids (both perilymph and endolymph) with different ionic constituents is often used to explain the physiologic basis for an acute Meniere's attack. This theory has some merits if one looks at findings during electrocochleography and observations regarding the direction changing nature of nystagmus during an acute attack. Nevertheless it still requires to be proven that hydrops is the actual pathophysiologic cause as opposed to a non-specific inner ear pathologic change arising from many other causes potentially.

In order to standardize the diagnostic clinical criteria for Meniere's disease, the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) in 1995 made the following recommendations for clinical reporting. [13]

a. Certain Meniere's disease

Definite Meniere's disease plus histopathologic confirmation at autopsy.

b. Definite Meniere's disease

Two or more definitive spontaneous episodes of vertigo 20 minutes or longer
Audiometric documentation of hearing loss on at least one occasion
Tinnitus or aural fullness in the treated ear
Other causes excluded

c. Probable Meniere's disease

One definitive episode of vertigo
Audiometric documentation of hearing loss on at least one occasion
Tinnitus or aural fullness in the treated ear
Other causes excluded

d. Possible Meniere's disease

Episodic vertigo of the Meniere's type without documented hearing loss.

Sensorineural hearing loss, fluctuating or fixed by history with dysequilibrium but without definitive episodes.

Other causes excluded

Overall the majority of patients with Meniere's require symptomatic treatment and reassurance mostly as their attacks are relatively infrequent. Preventative measures stress dietary modification (low salt diets, avoidance of caffeine etc) especially. Medical management is based on the prevention of hydrops (using diuretics, anti-secretory factor therapies etc) or minimize neurochemical responses (vasodilator and histamine agonists (ie betahistine), Ca^{2+} channel blockers etc). Intratympanic middle ear therapies involve aminoglycoside (primarily gentamicin) or steroid (primarily dexamethasone) injections. Surgery is generally undertaken when unilateral disease fails either to go into spontaneous remission or respond to medical management. These procedures can include surgery on the endolymphatic sac, tentotomy surgery, vestibular neurectomy or labyrinthectomy.

c. Recurrent Vestibulopathy (RV) [14]

Recurrent vestibulopathy (RV) is a descriptive term and was the 2nd most common cause for vertigo (after BPPV) identified in the UHN Multidisciplinary Neurotology Clinic. By definition, RV refers to recurrent attacks of episodic vertigo (similar in duration to those found in Meniere's disease) without hearing loss, tinnitus or focal

neurological dysfunction. The cause for RV remains known but there is speculation that it arise from transient vestibular dysfunction, possibly viral mediated.

Natural history studies have demonstrated that over an 8½ longitudinal follow-up 60% of patients went into spontaneous remission and only 10% continued to have active attacks of vertigo. Evolution to classic Meniere's disease occurred in 15% and BPPV in 10% while another 5% had another suspected form of peripheral vestibular dysfunction. There was no development of CNS dysfunction. The neurotological examination and audiometry was normal for the most part. In 22% of presenting patients a caloric reduction was noted on ENG.

Treatment is purely symptomatic.

d. Vestibular Neuronitis (Neuritis)

Often synonymous with the term "labyrinthitis" at the level of the family practitioner, this condition probably arises from an acute viral induced deafferentation of the vestibular nerve (herpes simplex and zoster viruses have been implicated). The affected individual experiences an acute attack of vertigo typically lasting days to weeks not associated with hearing loss, tinnitus or evidence of focal neurologic dysfunction. Caloric testing in the acute setting reveals a profound or absent caloric response on the affected side. In approximately 50% of patients the caloric response returns to normal within a 6 month timeframe. CNS compensation usually occurs in those whose caloric responses do not recover spontaneously.

Treatment is generally symptomatic with vestibular sedatives provided during the acute vertiginous phase. Oral corticosteroid trials have demonstrated mixed results according to a recent systematic meta-analysis [15]. Vestibular rehabilitation therapy has proven effective by promoting vestibular compensation and neuro-plasticity. Should acute vertigo last for over 2 weeks duration it may be necessary to consider imaging studies to exclude a CNS cause.

e. Vestibular Schwannoma (acoustic neuroma) [16]

Benign tumors of the vestibular nerve remain an uncommon cause for vertigo. Nevertheless it would be important not to miss one. While the term acoustic neuroma is often used interchangeably on histopathological grounds they are really vestibular schwannomas (arising from the schwann cells that cover vestibular neurons). They are rare (perhaps 1/10,000 population), although one suspects their incidence may be on the increase primarily from our ability to detect smaller and at times often incidental tumors on routine intracranial MRI scanning. Bilateral involvement usually implies the condition of NF-2 (an autosommal dominant genetic disorder arising from an abnormality involving chromosome 22).

When small VS's usually present with an asymmetric unexplained sensorineural hearing loss and/or tinnitus. As the tumor grows it may compress structures in the

cerebellopontine (CP) angle causing multiple cranial nerve palsies, eye movement abnormalities, nystagmus and hydrocephalus.

An important maxim to remember is that *"an unexplained asymmetric sensorineural hearing loss is an acoustic neuroma until proven otherwise"*. Tests used to confirm or exclude an acoustic neuroma include ABR audiometry, CT and MRI scanning. Of these MRI is typically considered to be the "gold standard". Management paradigms include a trial of conservative management (*"wait and scan"* approach), stereotactic radiosurgery or microsurgical removal.

f. Other causes

Rare causes for peripheral vestibular dysfunction include ototoxicity (systemic and topical), delayed endolymphatic hydrops (DEH), drop attacks (the otolithic crisis of Tumarkin), complications of chronic suppurative otitis media (CSOM) and its surgery (ie mastoidectomy), canal dehiscent syndromes and neurosyphilis.

Demographics from the UHN WSIB Neurotology Data Base 1988-2014

As an overriding statement the published literature associating head trauma to vertigo/ dizziness is sparse and not that well appreciated despite its common occurrence after minor head injury. The UHN WSIB Neurotology Data Base collection from 1988 to 2014 represents one of the largest prospective databases available that has assessed more than 3,400 head injured workers. All had presenting complaints of dizziness following a work related head injury. All underwent a thorough neurotological assessment which included a formal neurotological examination and laboratory inner ear testing. The results described below provide a unique window with which to view large data in order to identify what types of neurotological dysfunction occur post head injury.

Males were at greater risk for head injury in the workplace compared to females which is probably reflective of their prevalence involving physical work (ie construction). Demographic changes would be expected in future with females assuming greater participation in what were once considered typical male professions. (Table 2) There is a greater prevalence for a work related injury seen between ages 35-65 years. (Figure 5)
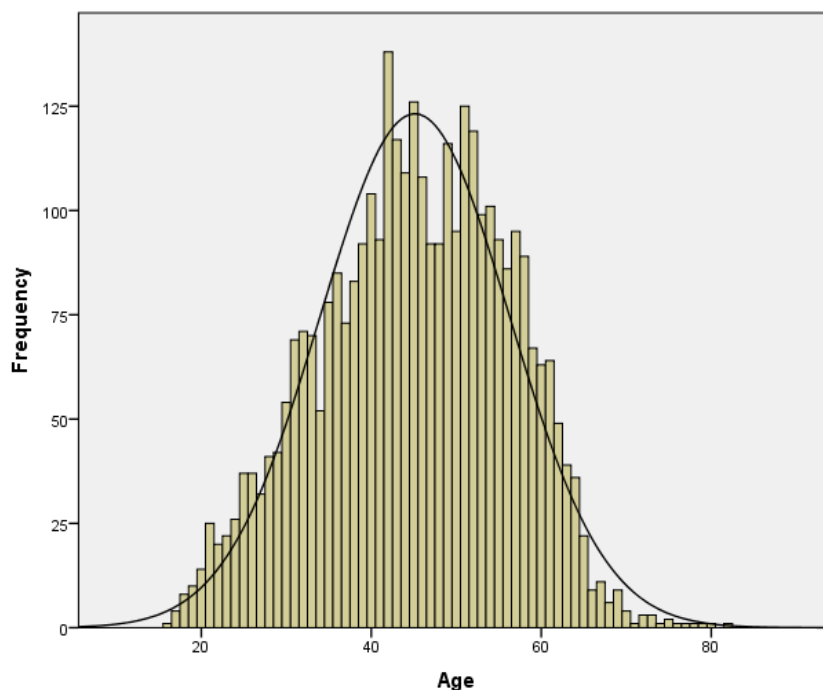
Demographic Information: UHN WSIB Neurotology Data Base 1988-2014

Table 2

N=3438	Cases	%
Female	987	28.70%
Male	2451	71.30%

28

Figure 5

Age Distribution: UHN WSIB Neurotology Data Base 1988-2014

From the UHN WSIB Neurotology database, the majority of work related injuries (approximately 77%) occurred as a result of either a fall (39.4%) or a contusion (38%). Motor vehicle accidents (10.2%) usually involved commercial truck drivers. Assaults in the workplace had a higher prevalence for affecting health care professionals (nurses and personal support workers (PSWs) rather than security or policing professions). Rare causes for workplace trauma over the 25 years of assessment included injuries from explosions (typically tire mechanics) and electrical injuries (electricians/ construction workers). (Table 3, Figure 6)

Table 3

Mechanism of injury (n=3438) UHN WSIB Neurotology Data Base 1998-2014

Mechanism of Injury	Number	Percent
Fall	1356	39.4%
Contusion	1308	38.0%
MVA	352	10.2%
Mixed Mechanism	149	4.3%
Assault	142	4.1%
Explosion	52	1.5%

Mechanism of Injury	Number	Percent
Other Mechanism	44	1.3%
Electrocution	26	0.8%
Asphyxiation	5	0.1%
Unknown	4	0.1%
Total	3438	100.0%

Figure 6

Mechanism of injury (n=3438) UHN WSIB Neurotology Data Base 1998-2014
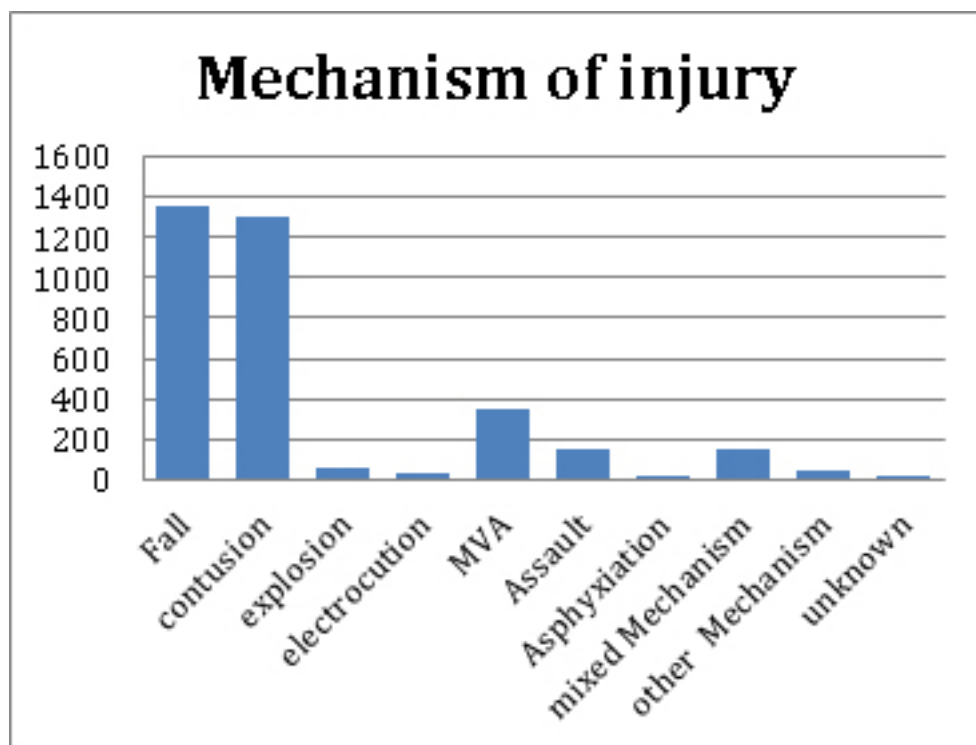
A spectrum of injury severity from falls and contusions ranged from mild to severe. Falls could involve simple slips on an icy surface to a fall from a height while on a ladder/construction site for example. Contusions could involve a simple bump on the head without a loss of consciousness from hitting an overhead cupboard door to a severe closed head injury associated with life threatening injuries/permanent neurological sequelae. Overall the majority of head injuries were deemed minor (77.98%)* Closed head injuries on their own or with skull fractures were identified in 7.65% and 10.99% respectively. Posttraumatic cerebral spinal fluid (CSF) leaks were rare and were usually associated with a skull fracture. A skull fracture was not always radiologically identified. (Table 4a). The majority of injuries (66.3%) were not associated with a loss of consciousness (LOC) but when present it was typically less than 5 minutes. A LOC > 24 hours tended to be associated with a more severe head injury and permanent neurological sequelae (Table 4b, Figure 7).

Tables 4 a,b UHN WSIB Neurotology Data Base 1998-2014

a. Severity of Injury (from clinical/imaging evidence)

| 1	Minor head injury	2681	77.98%
2	Closed head injury	263	7.65%
3	Closed head injury+ skull fracture	378	10.99%
4	Open/compound skull fracture	23	0.67%
5	Minor head injury + CSF leak only	2	0.06%
2+5	Closed head injury + CSF leak	1	0.03%
3+5	Closed head injury+skull fracture + CSF leak	22	0.64%
4+5	Open skull fracture + CSF leak	6	0.17%
	Unknown	62	1.80%
	Total	3438	100.00%

---

* For the purpose of definition a minor head injury (mild traumatic brain injury (mTBI)) is commonly defined as a head injury with a Glasgow Comma Scale score of 13-15, resolution of posttraumatic amnesia within 24 hours and a loss of consciousness for less than 30 minutes. [17]

b. Duration of loss of consciousness (LOC)

Time	Number of Workers	Percentage
None	1419	41.3%
Less than 5 min	861	25.0%
5-60 min	229	6.7%
1-24 hours	74	2.2%
Over 24 hours	102	3.0%
Unknown	753	21.9%
Total	3438	100.0%

Figure 7

UHN WSIB Neurotology Data Base 1998-2014
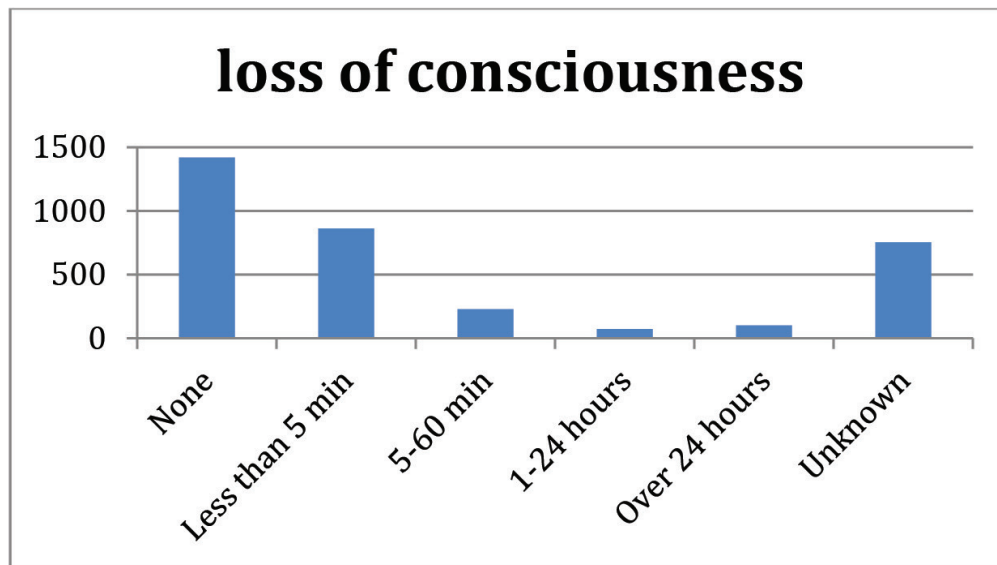
Specific Causes of Vertigo/Dizziness Identified or Postulated to have a Traumatic Link from the UHN WSIB Neurotology Data Base

Diagnoses from the UHN WSIB Neurotology Data Base 1998-2014 (N=3,438)

From a neurotologic perspective all head injured workers had subjective complaints of dizziness as their entry point into the UHN WSIB Neurotology Database. The most common complaint was a subjective history for benign positional paroxysmal vertigo (BPPV) (17.7%) or objective findings for BPPV confirmed in the Hallpike's manoeuvre (3.5%). A small percentage had atypical nystagmus patterns in the Hallpike's

manoeuvre for a posterior semicircular localization that could possibly have reflected involvement from other semicircular canals (superior or lateral) or from otolithic contributions (1.4%).

Approximately 18.5% of head injured workers demonstrated some loss of cochlear or vestibular function on laboratory testing only. Peripheral vestibular diagnoses generally included a spectrum of inner ear clinical diagnoses (the majority had a stable fixed vestibular loss from their injury requiring time for the compensation process to occur). Presumed psychogenic (non-organic or medially unexplained dizziness) featured prominently in this database (27.5%) and was the single largest diagnostic category. A significant number of individuals had complaints of dizziness that may have had some history/finding suggestive for a peripheral vestibular cause but remained unknown (20.3%). Approximately 6.1% could not be diagnosed with confidence regarding a diagnosis. See Table 5.

Table 5

a. Diagnosis		N	%
BPPV			
	Typical BPPV	119	3.5 %
	Atypical BPPV	49	1.4 %
	Hx for BPPV	609	<u>17.7 %</u>
			22.6 %
Cochlear +/- Vestibular Loss*		801	18.5 %
Peripheral Vestibular Diagnosis		149	4.33 %
Central Vestibular		24	0.7 %
Psychogenic (non-organic)		947	27.5 %
Unknown		698	20.3 %
No Diagnosis		208	6.1 %

Sub-Group Analysis of the UHN WSIB Neurotology Data Base 1998-2014

Subgroup analysis of individuals with cochleovestibular dysfunction and the peripheral vestibular diagnoses are illustrated in Tables 6a,b respectively.

Tables 6 a,b

a. Cochlear +/- Vestibular Loss* (N= 950, 27.2% of data base)

Cochlear +/- Vestibular Loss	N	%
Cochlear loss	333	9.7%
Vestibular loss	418	12.2%
Cochleovestibular loss	199	5.8%

b. Peripheral Vestibular Diagnosis (N= 149, 4.33% of data base)

Peripheral Vestibular Diagnosis	N	%
Recurrent Vestibulopathy (RV)	34	0.99%
RV-Otolithic based	18	0.52%
Meniere's**	11	0.32%
Delayed Endolymphatic Hydrops (DEH)	5	0.15%
Drop attacks	4	0.12%
Other	77	2.24%
Total	149	4.33%

** Of which 6 head injured workers had definite Meniere's and 5 had possible Meniere's (by 1995 AAO-HNS CHE reporting guidelines in Diagnosis and Evaluation of Response to Therapy in Meniere disease)

Overall 950 head injured workers had laboratory evidence for a cochlear, vestibular or a mixed (cochleovestibular) loss from laboratory testing. This suggested that approximately 25% had abnormalities that could be identified on audiological and vestibular laboratory testing. Clinical correlation however would be required on an individual basis when trying to determine if the injury were itself causative, whether the findings were related to a pre-existent loss or might even be considered spurious on technical grounds.

The most common peripheral vestibular diagnostic subset involved individuals who sustained a chronic fixed vestibular loss from their labyrinthine trauma. Episodic attacks of vertigo were diagnosed as being compatible with either a recurrent vestibulopathy (RV) or RV with prominent otolithic features primarily determined when their history was suggestive for semicircular (ie a spinning and circling sensation) or otolithic (floating and rocking sensation) involvement. Diagnoses for posttraumatic Meniere's disease, delayed endolymphatic hydrops and drop attacks were decidedly rare in this data base. (See Common and Controversial Causes for Posttraumatic Vertigo Section below).

Common and Controversial Causes for Posttraumatic Vertigo

1. Common recognized causes for dizziness associated with trauma

1. Posttraumatic BPPV
2. Delayed endolymphatic hydrops (DEH)
3. Traumatic vestibular loss from direct inner ear injury

2. Controversial diagnoses for posttraumatic dizziness

1. Cervicogenic vertigo
2. Posttraumatic vestibular migrane
3. Perilymphatic fistula formation
4. Posttraumatic Meniere's disease and posttraumatic hydrops

Common Accepted Causes for Dizziness Associated with Trauma

a. Posttraumatic BPPV

It has been well established that BPPV can arise following head injury. Mechanisms for posttraumatic BPPV are generally based on the shearing affects of the trauma upon the inner ear that results in direct cellular injury or more likely liberates otoconia covering the saccule or utricle. Particulate debris then gravitates into the circulation of the posterior semi-circular canal. The terms *canalolithiasis* and *cupulolithasis* are used to describe where the particulate debris is found and to explain the pathophysiological basis for the nystagmus generated.

Some distinguishing characteristics for posttraumatic BPPV versus its spontaneous variant include being:

• more likely to persist and not go into spontaneous remission

• more likely to be bilateral

• less responsive to physical therapy manoeuvres

• more likely to require surgical intervention (ie PSCC canal occlusion) for definitive treatment in those cases deemed incapacitating by the patient. [11]

b. Delayed Endolymphatic Hydrops [18]

While trauma might result in an individual losing their hearing completely it does not necessarily imply that all vestibular function has been similarly lost. Under the circumstances the phenomenon of delayed endolymphatic hydrops (DEH)

is diagnosed when recurrent attacks of vertigo, similar in duration to Meniere's disease occur on the backdrop of an ear that has experienced a profound unilateral sensorineural hearing loss often many years prior. A traumatic injury may be one such possible cause for hearing loss.

According to earlier Japanese literature approximately 30% of patients following a profound sensorineural hearing loss (regardless of cause) developed recurrent attacks of episodic vertigo over the course of their lifetime. [19] A somewhat more rare clinical variant of DEH is postulated for contralateral DEH where patients experienced signs and symptoms of classic Meniere's disease in the opposite ear.

When attacks of vertigo are incapacitating surgical options can be considered should medical treatment or intratympanic therapies (aminoglycoside or steroid based) prove ineffective. Total osseous labyrinthectomy is generally recommended and appears 100% successful in the elimination of vertigo provided there is no contraindication (ie the deafened ear being the only ear with vestibular function). Unlike Meniere's disease, development of symptoms post surgery in the contralateral non-operated ear is considered extremely rare. [20]

c. Traumatic Vestibular Loss from Direct Injury [17]

Loss of vestibular function can occur from direct trauma to the inner ear. Central vestibular pathway injury is less likely but when present would typically imply severe brainstem injury with hemorrhage and diffuse axonal injury.

Compensation for a chronic stable unilateral vestibular loss is generally the expected norm. The compensation process however can be adversely affected especially by immobility and the continued use vestibular sedatives post injury Vestibular rehabilitation therapy by qualified therapists can play an important role in promoting both the compensation process and vestibular plasticity.

Failure to improve should evoke consideration whether there could be pathology involving the contralateral inner ear or whether any adverse psychological dynamics post head injury need further attention. Treatment of an underlying mood disorder (ie depression, anxiety) cannot be stressed enough.

Controversial Diagnoses in Post Traumatic Vertigo

Some diagnoses in the head injury literature pertaining to dizziness/vertigo generate controversy in the world literature despite being frequently cited. They are controversial often because they involve patient series where more likely explanations exist or where no objective parameters for diagnosis exist. A few of these according to the author's experience are mentioned below.

a. Cervicogenic vertigo

Many disciplines independent from neurology/neurotology have sought to evoke injury to the cervico-ocular reflex (COR) from neck proprioceptors as a cause for a patient's dizziness. Proponents of vertigo arising from a whiplash associated disorder (WAD) caused by flexion/extension injury to neck proprioceptors is not infrequently mentioned. Controversy however exists on the anatomic basis that the COR appears rudimentary in humans when compared to afoveate animals (ie rabbits, birds), that no specific diagnostic test is available to independently confirm and that the pathology/degree of injury to neck proprioceptors is not well defined nor does it seem proportional to the stated injury (ie the unexplained discrepancy between patients with severe neck pain without vertigo and patients complaining of moderate vertigo with mild neck pain). An excellent review in 2001 by Brandt and Bronstein highlight the controversies and lack of convincing evidence for a cervical mechanism where alternative explanations are almost always possible. [21]

b. Posttraumatic Vestibular Migrane

Generalized headaches including migrane are commonly reported following head injury. The association of migrane and vertigo (migranous vertigo) has been reported outside head injury. Post head injury dizziness in conjunction with migrane more often is described as a rocking sensation, a feeling of floating /disequilibrium or drunkenness. Rarely is true vertigo described. The association of both conditions to some degree could reflect the commonality of both migrane and dizziness in the general population.

Whether trauma is actually causative cannot be answered as there have been no clinical trials addressing posttraumatic migranous vertigo specifically.[17] The poor correlation between trauma severity and the development of migrane specifically suggests that other mechanisms such as the emotive changes in an individual post head injury in conjunction with established predisposing factors for migrane (ie family history, a history for motion sickness, "brain-freeze" symptoms while eating ice cream etc) may be more important than the actual injury itself.

In the context of a good history for dizziness being associated with the development of frequent and debilitating migrane, a trial of migrane prophylactic therapy would not seem unreasonable.

c. Perilymphatic Fistula (PLF)

Rupture of the round window membrane (other sites theoretically could include the oval window and through defects in the otic capsule) following trauma would allow for leakage of perilymph from the inner ear into the middle ear space. While rare perilymphatic fistulae have been reported following barotrauma, penetrating trauma, from surgical procedures (especially stapes surgery) and following straining or physical exertion. [22] Controversy exists not so much in the acute presentation following a

traumatic episode but whether a chronic recurrent form of the condition exists from intermittent fluid leakage giving rise to episodic attacks of vertigo and/or disequilibrium. [23] A significant confounder would include traumatic induced inner ear fistulae between the endolymphatic and perilymphatic spaces alternatively.

Symptoms have been reported to mimic those of Meniere's disease at times. Findings of positive fistula tests to positive pressure in the ear canal (Hennebert's sign), loud noise (Tullio phenomenon) and straining (Valsalva manoeuvre) in addition to elevated SP/AP ratios (>0.30) on ECoG were at one time thought to be pathognomonic for a PLF. They however can also be present in Meniere's disease, superior canal dehiscence sydromes and neurosyphilis involving the otic capsule.

In the acute situation (especially following penetrating trauma or post surgery) treatment is generally conservative with bed rest and head elevation. The majority of fistulae will heal spontaneously. Persistent worsening of hearing or vestibular function however would be an indication for surgical exploration to repair/seal the leakage site

d. Posttraumatic Meniere Disease and Post-traumatic hydrops

The association of Meniere's disease with trauma has a number of uncertainties especially as Meniere's Disease typically arises without a known cause. For this reason the term "Meniere's syndrome" has often been applied when a causative link is postulated. The same holds for individuals who experience recurrent attacks of episodic vertigo without auditory symptoms similar in essence to a recurrent vestibulopathy (RV). On the backdrop of a prior traumatic event, the phrase posttraumatic hydrops is often applied.

Consideration that trauma might induce microstructural changes within the inner ear leading to endolymphatic hydrops (the suspected pathophysiologic basis for Meniere's disease) has been suggested by some authors. Pulec's 1972 paper concerning the etiology for Meniere's disease is often quoted but in the 120 patients reported less than 3% had a convincing history for prior trauma. [24] Paparella and Mancini's 1983 paper reported on a series of 37 patients who developed features of a Meniere's syndrome (related to acoustic trauma in 18 and physical trauma in 19 patients respectively). The series included the histopathologic findings of endolymphatic hydrops involving the temporal bone of one patient at necropsy. [25]

Issues that have been considered by the Tribunal

a. What is the relationship between Meniere's disease/syndrome and trauma?

It is somewhat curious that few reports have attempted to broach this association over the past 30 years when literature searches are performed. The great difficulty is that Meniere's disease typically arises spontaneously without an identifiable cause. Any

delay from the injury to the development of symptoms diagnosed as being compatible with Meniere's disease/syndrome often takes place in the remote sense temporally.

The results from the extensive UHN WSIB Neurotology Database suggest that recurrent attacks of episodic vertigo associated with a Meniere's disease/syndrome with or without auditory dysfunction are decidedly uncommon. There is a stronger basis for the relationship between delayed endolymphatic hydrops where a prior hearing loss associated with trauma might cause recurrent attacks of vertigo.

Eleven (11) patients in a database of 3,438 head injured workers were diagnosed with Meniere's disease (either definite (N=6) or possible (N=5) according to the 1995 AAO-HNS guidelines for the diagnosis for Meniere's disease). This diagnosis represented approximately 0.032% of the entire database. See Table 7a,b. In trying to determine whether Meniere's disease is related to trauma one would reasonable have to look at local incidence and prevalence rates. Unfortunately Meniere's disease seems to show considerable worldwide variability in incidence and prevalence. A recent epidemiological table comparing national statistics prepared by Timothy Hain (2015) clearly demonstrates this variability.[12] (This makes it difficult to know if one is looking at a causal relationship to the trauma or if it represents the spontaneous presentation in the population at large independently (see section on relationship between noise exposure and Meniere's disease below)). See Table 8.

Tables 7a and b: UHN WSIB Neurotology Data Base 1998-2014

a. Data for Meniere patients (N=11. 032% of database diagnoses)

Meniere's	N	Age-average	Age-range	Injury to as-sessment	F/U since initial assessment (av)	Total Duration of F/U (av)
Definite	6	49.2	39-70	11 months	15.3 months	26.3 months
Possible	5	49.4	44-58	14.2 months	13.8 months	28.0 months

b. Sub-category Analysis of Meniere's Diagnoses

Meniere's Diagnosis		Injury type			Injury severity		LOC	
	N	Fall	Contusion	MVA	Minor head injury	Midface fracture	None	< 5 min
Definite	6	2	3	1	5	1	3	3
Possible	5	1	2	2	5	0	4	1

Table 8 [12]

Incidence and Prevalence of Meniere's disease in the world literature.

Country	Prevalence/100,000	Incidence	Mystery number	Reference
USA	218	15.3		Wladislavosky-Waserman et al, 1984
USA	190			Harris and Alexander, 2010
England			180	Cawthrone and Hewlett (1954)
England			100	Harrison and Naftalin, 1968
England	56			Goodman (1957)
Ireland		10-20		Wilmot (1983)
Japan	21-36			Shojaku and Watanabe (1997)
Sweden(Uppsala)			45	Stahle (1973)
Finland	43	4.3		Kotimaki et al, (1999)
Finland	513			Havia et al (2005)
Italy	205	8		Celestino and Ralli (1991)

As previously stated above all one can say is that if there is a relationship with trauma it is decidedly rare and that further longitudinal study would be required over years if not decades. The same holds true especially with individuals who were diagnosed to have a posttraumatic recurrent peripheral vestibulopathy (episoidic vertigo without hearing loss/tinnitus) as one might cautiously predict evolution to classic Meniere's disease over time in some.

b. What is the relationship if any between noise exposure and Meniere's disease and dizziness?

Some authors have presented cases studies of Meniere's disease/syndrome following noise exposure. All are retrospective cohort studies with little in the way of prospective study. The publication from Segal et al (2003) is of particular interest in this matter. An epidemiological study of 17,245 Israeli Defence Force (IDF) veterans documented the presence of typical Meniere's disease in 11 veterans (4 had experienced prior noised induced hearing loss and 7 had experienced acute acoustic trauma). The average period between first documentation of the hearing loss and Meniere's disease was 15.8 years (standard deviation $\pm$ 6.6 yrs). A prevalence of 1.9:100,000 of Meniere's disease was identified in a population with acoustic or noise induced hearing loss which was a figure comparable to that in the general population. The conclusion supported there was no causal relationship for Meniere's disease from acoustic trauma or noise induced hearing loss. [26]

One is left with the impression that any association would be relatively rare and might be best explained on the basis of a delayed endolymphatic hydrops type phenomenon.

c. Can there be a delay of onset of Meniere's disease following acute noise exposure?

As mentioned above there appears to be a significant delay from the diagnosis of acute noise induced hearing loss to reported Meniere's disease/syndrome in most reported series. Paparella and Mancini documented Meniere's syndrome from acute acoustic trauma in 3 patients over 3.67 years on average. When combined with chronic noise exposed individuals the mean duration in 18 patients was 5.3 years. [25]

In most instances there seemed to be a delay from when the acoustic trauma happened to when the dizziness began.

d. How severe a force (blow) is required to cause dizziness?

The severity of such a force required to study vestibular injury has not been studied directly in humans. We know that dizziness however in conjunction with mild traumatic brain injury (that can be defined as a head injury with or without transient loss of consciousness (< 30 min), a Glasgow Coma Scale score of 13-15 and resolution of posttraumatic amnesia within 24 hrs) is common. Imaging investigations (especially CT) rarely demonstrate significant structural change although MRI would likely be considered the better investigation. Radionucleide techniques such as single photon emission CT (or SPECT) scanning or functional MRI can provide evidence for brain injury but rarely would an individual have a pre-injury study for comparison. These studies would also be unable to determine if injury had occurred to the inner ear. This primarily leaves the diagnosis of a blow causing injury to be identified on history, a formal neurotological examination and on laboratory vestibular testing.

e. What is the relationship if any between exposure to toxins e.g. mercury/ solvents and dizziness.

The association of workplace toxins has been relatively well studied as it affects the cochlea and in turn hearing. The same however is has not been studied to any large degree in the vestibular system despite complaints of dizziness on presentation (as one of a number of non-specific complaints including headaches, abdominal pain, cognitive impairments etc). While there would be no reason to doubt vestibular system involvement from toxin deposition, exposure to toxins would be unlikely to result in acute vertigo as vestibular end organ function would be affected presumably similarly on both sides. In assessing an individual with suspected toxin exposure one would also need to consider the generalized toxin effects on nerve transmission, neurotransmitter activity in the synapses and neurochemical activity within the brain itself.

Disappointingly the author's recent Medline search under the engines of vestibulotoxicity, heavy metal poisoning and solvent toxicity in occupational diseases failed to demonstrate any published papers in this matter.

References

1. Rutka JA: Evaluation of Vertigo in Office Based Surgery in Otolaryngology 1998; 71-78. Thieme. New York

2. Rutka JA. Physiology of the Vestibular System. In Ototoxicity. Roland P and Rutka J ed. 2004: 20-27. BC Decker Inc. Hamilton.London.

3. Lai P, Pothier D, Rutka JA. Balance Disorders. In K.J. Lee's Essential Otolaryngology Head & Neck Surgery. 2012;102-116. McGraw Hill. New York.

4. Falls C, Lai P, Pothier D, Rutka JA. Vestibular and Balance Disorders. In K.J. Lee's Essential Otolaryngology Head & Neck Surgery. Chan & Goddard Ed. 2016 11th Edition Vestibular and Balance

5. Manzari L, Burgess AM, McGarvie LA, Curthoys IS. An indicator of probable semicircular canal rders 295 – 313. 5. Manzari L, Burgess AM, McGarvie LA, Curthoys IS. An indicator of probable semicircular canal dehiscence: ocular evoked potentials to high frequencies. Otolaryngol Head Neck Surg 2013; 149(1): 142-45.

6. Prepageran N, Kisilevsky V, Tomlinson D, Ranalli P, Rutka J. Symptomatic High Frequency/Acceleration Vestibular loss: Consideration of a New Clinical Syndrome of Vestibular Dysfunction. Acta Otolaryngol 2005: 125: 48-54.

7. Kessler P, Zarandy M, Hajioff D, Tomlinson D, et al. The Clinical Utility of Search Coil Vestibular-Ocular Reflex Testing. Acta Oto-Laryngologica 2008; 128: 29-37.

8. Heaton J, Barton J, Ranalli P, Tomlinson D, Rutka J. Evaluation of the Dizzy Patient: Experience from a Multi-Disciplinary Neurotology Clinic. J.Laryngol Otol. 1999: 113: 19-23.

9. Bath AP, Walsh, RM, Ranalli P, Tomlinson D, Rutka J. Experience from a Multidisciplinary "Dizzy" Clinic. Am J Otol 2000; 21 (1): 92-9.

10. Marom T, Oron Y, Watad W, Levy D et al. Revisiting benign positional vertigo pathophysiology. Am J Otolaryngol Head Neck Surg 2009; 30(4): 250-255.

11. Kisilevsky, V. Bailie, NA, Dutt, SN, Rutka, JA. Lessons Learned from the Surgical Management of Benign Paroxsymal Positional Vertigo: The University Health Network Experience with Posterior Canal Occlusion Surgery (1988-2006). J Otolaryngol 2009; 2:212-221.

12. http://www.dizziness-and-balance.com/disorders/menieres/men epi.html

13. Committee on Hearing and Equilibrium guidelines for the diagnosis and evaluation of therapy in Meniere's disease. American Academy of Otolaryngology-Head and Neck Foundation, Inc. OtolaryngolHead Neck Surg. 1995 Sep;113(3):181-5.

14. Rutka, J., Barber, HO. "Recurrent Vestibulopathy -Third Review." J Otolaryngol 1986; 15:105-107.

15. Goudakos, JK, Markou KD, Franco-Vidal V, Vital V et al. Corticosteroids in the Treatment of Vestibular Neuritis: A systematic Review and Met-Analysis. Otol Neurotol 2010; 31:183-189

16. Hajoff D, Raut V, Walsh R, Bath AP et al. Conservative management of vestibular schwannomas. 3rd review of a prospective 10 year study. Clin Otolaryngol 2008; 33: 255-264.

17. Fife TD, Giza C. Posttraumatic Vertigo and Dizziness. Semin Neurol 2013; 33(3): 238-243.

18. Schuknecht HF. Pathophysiology of endolymphatic hydrops. Arch Otorhinolaryngol 1976; 212: 253-256.

19. Kamei T. Delayed Vertigo. In JD Hood (ed), *Vestibular Mechanisms in Health and Disease*. London: Academic Press, 1978:369-374.

20. Yazdi AK, Rutka JA: Results of Labyrinthectomy in the Treatment of Meniere's Disease and Delayed Endolymphatic Hydrops. J Otolaryngol 1996; 25(1):26-31.

21. Brandt T, Bronstein AM. Cervical Vertigo. J Neurol Neurosurg Psychiatry 2001; 71:8-12.

22. Goodhill V. Ben H. Senturia lecture. Leaking labyrinth lesions, deafness, tinnitus and dizziness. *Ann Otol Rhinol Laryngol*. 1981 Mar-Apr. 90(2 Pt 1):99-106.

23. Friedland DR, Wackym PA. A critical appraisal of spontaneous perilymphatic fistulas of the inner ear. *Am J Otol*. 1999 Mar. 20(2):261-76; discussion 276-9.

24. Pulec JL. Meniere's Disease: Results of a Two and One Half Year Study of Etiology, Natural History and Results of Treatment. Laryngoscope 1972; 1703-1715.

25. Paparella MM, Mancini F. Trauma and Meniere's Syndrome. Laryngoscope 1983: 93:1004-1012

26. Segal S, Ephraim E, Berenholz L Kessler A et al. Is There a Relation Between Acoustic Trauma or Noise-Induced Hearing Loss and a Subsequent Appearance of Meniere's Disease?: An Epidemiologic Study of 17,245 Cases and a Review of the Literature. Otol Neurotol 2003; 24:387-391.

Acknowledgement

Suggested Appendices and Glossary For Further Reading

Appendix 1

Evaluation of Vertigo (from *Evaluation of Vertigo in Office Based Surgery in Otolaryngology* 1998; 71-78. Thieme. New York)

Appendix 2

Physiology of the Vestibular System (from *Ototoxicity*. Roland P and Rutka J ed. 2004: 20-27. BC Decker Inc. Hamilton .London.)

Appendix 3

Vestibular and Balance Disorders. (from K.J. Lee's *Essential Otolaryngology Head & Neck Surgery*. Chan & Goddard Ed. 2016 11th Edition Vestibular and Balance)

A. Glossary of Terms

a. Vertigo: An illusion or hallucination of movement either of one's self (subjective vertigo) or one's environment (objective vertigo). It may have a rotatory, linear, rocking, floating or even tilt like sensation depending on what part of the inner ear is involved.

b. Disequilibrium: A sensation of inability of body positions either walking, standing or sitting.

c. Oscillopsia: Visual blurring with head movement generally reflects a bilateral impairment of the vestibular-ocular reflex from inner ear pathology.

d. Lightheadedness: Sense of an impending faint (pre-syncopal feeling) often synonymous with the term giddiness.

e. Physiological Dizziness- A response to a physiological stimulus such as motion (motion sickness), heights (acrophobia), enclosed spaces (claustrophobia) etc where dizziness is a prominent complaint.

f. Multisensory deficit (MSD)- An altered sensation from the cumulative deterioration/degeneration in the multiple sensory systems responsible for one's balance (ie vision, proprioception and vestibular and its central integration) that can be affected by age and pathology (ie diabetes, stroke, tumors etc)

g. Tullio phenomenon- Dizziness resulting from sound stimulation. Can occur with an anatomic defect involving the otic capsule such as a superior canal dehiscence or from intralabyrinthine disorders such as Meniere's disease. Objective evidence would require the presence of observable nystagmus to sound stimulation.

h. Hennebert's phenomenon- Dizziness related to alteration of pressure within the   middle or inner ears with straining, coughing, sneezing, during pneumatic otoscopy (seeing how the TM moves under pressure). Causes for this are similar to the those found for the Tullio phenomenon etc.

i. 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).

j. 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).

k. 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.

l. 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.

m. 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.

n. Nystagmus- A rapid rhythmical to and fro movement of the eyes. In the absence of a medication affect (from sedatives, alcohol, anit-convulsants etc) it is the cardinal sign of a vestibular disorder involving peripheral or central vestibular pathways. Nystagmus can be peripheral or central in origin, congenital or acquired in etiology, spontaneous or gaze evoked, positional induced or positioning in nature etc.

B. __Audiological and Vestibular Testing 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/cm^2. Because of the large numbers involved in sound pressure measurement dB scales have been created for convenience (ie 100 Bel =10^2 Bel = 0.02 dynes/cm^2 =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:

   __i. Auditory Brainstem Response (ABR)__- Synonymous with the term BERA (brainstem evoked response audiometry) or auditory BEP's (brainstem evoked potentials). This test measures 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.

   __ii. 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).

__Electronystagmography/Videonystagmography__ - This utilitarian test is commonly preformed in the assessment of the dizzy patient. Changes in the corneal retinal potential from eye movements are recorded from conventional recording electrodes placed around the eyes testing in electronystagmography (ENG). In videonystagmography (VNG) records eye movements are recorded directly with an infrared video camera and digital video image technology.

Measurement activities include an assessment for/of:

* spontaneous or gaze evoked nystagmus

* oculomotor function (pursuit, saccade, optokinetic and fixation pathways) which is an indirect measure of CNS integrity

- positional and positioning nystagmus and

- the caloric response

Caloric Testing - Thermal stimulation of the ear canals stimulates the inner ear causing nystagmus as an evoked response. The stimulus can be with air or water of different temperatures. Water calorics are usually performed as they are quantitatively more reliable. Comparison of the nystagmus (from hot and cold water stimulation typically at 44 C and 30C) generated in one ear versus the other is extrapolated to the overall inner ear activity. The mnemonic "COWS" (cold opposite/warm same) is often used to describe the expected direction of nystagmus from thermal stimulation.

Excitability Difference – Determined by the

$$\text{Jongkees Formula} \frac{[(44R + 30R) - (44L + 30L)]}{44R + 30R + 44L + 30L} \times 100\%$$

provides comparison of caloric test results between sides. An excitability difference of >20% is usually significant for a relative loss of vestibular activity. Excitability differences from 20-25% are considered mild, 25-50% moderate, 50-90% severe and no response (NR) profound.

Vestibular Evoked Myogenic Potentials - Short latency responses from the anterior neck and extraocular muscles to sound stimulation have been collectively known as vestibular evoked myogenic responses or VEMP's. The responses are thought to be generated from the otolithic organs: the cervical VEMP(cVEMP) response from the saccule and the ocular VEMP (oVEMP) response primarily from the utricle. The tests have been of value in confirmation of a symptomatic superior canal dehiscence, Meniere's disease and PLF.

Vestibular Head Impulse Testing (vHIT)-The development of light weight infrared red recording glasses and an embedded gyroscope measuring head velocity has allowed for high velocity horizontal VOR function to be assessed. vHIT measures specifically how the eyes move relative to a fast head movement which under normal circumstances should be unity (eye movement/head movement=1). It is a physiologic measure of VOR activity that can be applied to vertical VOR testing additionally.

Magnetic Scleral Search Coil Testing (MSSCT) - High velocity VOR activity is recorded following placement of a contact lens embedded with a copper wire. When placed in a magnetic field movements of the eye with the contact lens causes pertubations of the magnetic field that can be recorded in the advanced setting. Overall MSSCT would be considered the gold standard for high velocity head movements >200os.

Posturography and Computerized Dynamic Posturography- In a test primarily designed for the overall assessment and quantification of balance function a balance platform measures center of pressure changes and measures of body sway evaluate an individual's balance in a series of tests involving visual, somatosensory and vestibular inputs. Information is provided through sensory organization tests (evaluating anterior-posterior body sway with eyes open or eyes closed) on a fixed or sway referenced surface (usually a foam surface), motor control tests (which assesses an individual's ability to recover from external provocation such as forwards/backwards platform movement) and adaptation tests (which assesses motor adjustments made when the platform tilts up or down).