Workplace Safety and Insurance
Appeals Tribunal

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

Dizziness

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This medical discussion paper will be useful to those seeking general information about the medical issues 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 Discussion papers are not peer reviewed.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. Please see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal) [2009] O.J. No. 2080 (Ont. Div. Court)* for reference. For more information about these papers, please consult the WSIAT's *Guide to Medical Information and Medical Assessors*.

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Glossary of Terms

Acoustic Neuroma (Vestibular Schwannoma): benign brain tumour arising 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 vestibular nerve. A more pathologically correct term would be vestibular schwannoma (VS).

Benign Positional Paroxysmal Vertigo (BPPV): the most common inner ear disorder seen in clinical practice where movement of so-called otolithic derived "crystals" in the posterior semicircular canal cause intense, brief episodes of spinning vertigo triggered often by looking up or turning in bed from side to side. Physical therapy manoeuvres such as the Epley manoeuvre are often performed for treatment.

Catastrophization: a cognitive distortion where there is magnification beyond normally expected under the circumstances. Worst case scenarios are persistently ruminated along with a feeling of helplessness. An exaggeration of the severity can leave the affected individual emotionally paralysed and unable to cope with their symptoms

Closed Head Injury: typically a more severe head injury usually but not always associated with a LOC. Imaging demonstrates evidence for anatomic injury including intracranial bleeding (i.e. subarachnoid, subdural, epidural bleeding etc.), intraparenchymal bleeding (i.e. "brain bruising") etc. See Minor Head Injury (mTBI) below.

Cognitive vestibular disorders: a more specific collective term to describe dizziness likely arising from other generators in the brain not involving organic recurrent vestibular dysfunction such as *syndrome du mal de débarquement, persistent postural perceptual dizziness, functional neurological disorders* etc. It may also be considered a subset in those diagnosed with a Functional Neurological Disorder.

Delayed Endolymphatic Hydrops: a variant of Meniere's disease where affected individuals following a profound sensorineural hearing loss years later develop attacks of episodic vertigo lasting minutes to hours. Hearing does not change because of the prior antecedent loss.

Disequilibrium: an abnormal sensation in the perception of body positions either walking, standing or sitting.

Dizziness Handicap Inventory (DHI): a 25-item, self-reported questionnaire that quantifies the functional, emotional and physical impacts of dizziness or unsteadiness on daily life and helps to track the rehabilitation process. It has a high test-re-test reliability and internal consistency making it a standard tool in vestibular assessment.

Endolymphatic Hydrops: a disorder the inner ear where distention of the delicate lining membranes in the cochlear and vestibular system may be associated with episodic attacks of vertigo, fluctuation in hearing, tinnitus and a sense of pressure in the affected ear. When primary or idiopathic the term Meniere's disease is used. The findings are however reflective of injury at some level to the inner ear and can represent a non-specific pathologic change.

Functional Neurological Disorder (FND): refers to neurological conditions caused by changes in how the brain networks work rather than changes in the structure of the brain itself. Physical conditions are perceived as real in the affected individual but cannot be explained by changes in brain structure. Dizziness amongst other complaints can be seen in FNDs. See *Cognitive Vestibular Disorders*.

Hallpike's manoeuvre: the diagnostic clinical test used to identify BPPV and other variants causing positional vertigo. Subjective vertigo and classic nystagmus patterns are found when BPPV is present.

Hennebert's Phenomenon: dizziness related to alteration of pressure in the middle or inner ears with straining, coughing, sneezing. Can be seen in *Meniere's disease* or a *Superior Semi-circular Dehiscent Syndrome*.

Hydrops (endolymphatic): see *endolymphatic hydrops; Meniere's Disease*

Laboratory Vestibular Testing: conventional test battery done to quantify inner ear function that can include videonystagmography (VNG) with caloric testing, vestibular evoked myogenic potentials (VEMP) to assess otolithic activity and video head impulse testing (vHIT) to assess the vestibular ocular reflex (VOR) in all planes of movement.

Lightheadedness: a rather broad term that can imply a sense of impending faint (pre-syncopal), not quite feeling right and often synonymous with the term "giddiness". Can especially be seen from medications typically used to treat hypertension, chronic pain related syndromes, depression/anxiety etc.

Mal de Debarquement Syndrome (MdDS): refers to a sensation of rocking, bobbing or swaying along with unsteadiness, anxiety and a loss of self-confidence that typically follows travel on a boat or airplane. The sensation persists beyond the transient physiologic symptoms that occur in many individuals after travel (i.e. getting "one's land legs" after a cruise). Symptoms worsen with rest and paradoxically seem to improve when in motion (i.e. in a car). While the brain can adapt to a particular motion when the motion stops, in this condition the brain seems unable to adapt to its former state.

Mastoidectomy: a surgical procedure intended to exteriorize disease in the mastoid bone and adjoining middle ear. The procedure is usually done for chronic suppurative otitis media (CSOM) with the intent of providing a safe (to prevent a serious complication), dry and if possible better hearing ear.

Meniere's Disease: the prototypic inner ear disorder is classically associated with fluctuant sensorineural hearing loss, tinnitus (unwanted head noise), a sense of pressure in the affected ear(s) and attacks of vertigo lasting minutes-hours. The pathology is thought to arise from excess fluid accumulation (so called *endolymphatic hydrops*) in the inner ear leading to membrane rupture.

Minor Head Injury (Minor traumatic brain injury (mTBI): a minor head injury (mild traumatic brain injury (mTBI)) is commonly defined as a head injury with a Glasgow Coma Scale score of 13-15, resolution of posttraumatic amnesia within 24 hours and a loss of consciousness for less than 30 minutes. See *Closed Head Injury above*.

Multisensory Deficit (MSD): an altered sensation from the cumulative deterioration/ degeneration in the multiple sensory systems responsible for balance (i.e. vision, proprioception, vestibular and its central integration) that can be affected by age and pathology (i.e., diabetes, stroke, tumors etc.).

Nystagmus: a rapid rhythmical *"to and fro"* movement of the eyes. In the absence of a medication affect (from sedatives, alcohol, anticonvulsants etc.) it is the cardinal sign of a vestibular disorder both peripheral (inner ear or vestibular nerve) or central. It can also be congenital or acquired in etiology. Depending on how the eyes move it can be spontaneous, evoked by eye movement testing (gaze evoked), positionally induced or positioning in nature.

Oscillopsia: visual blurring with head movement that generally reflects a bilateral impairment of the vestibular ocular reflex (VOR) typically from inner ear pathology.

Otolithic Organs: the utricle and saccule are specialized gravity-sensitive structures in the inner ear's vestibular system that detect linear acceleration, tilt and head position sense. They consist of specialized hair cells covered by a gelatinous matrix containing calcium carbonate crystals (otoconia) that shift with motion to trigger nerve signals for balance and spacial orientation.

Ototoxicity: the tendency of certain drugs/substances to cause functional and cellular damage to the inner ear, especially involving the neuroreceptor tissue (or end organs) of hearing and balance. Certain antibiotics (especially the aminoglycoside class), anti-malarials, chemotherapeutic agents and even excessive doses of ASA can be toxic to the inner ear.

Perilymphatic Fistula: an abnormal tear or opening in the membranes of the oval or round window of the inner ear causing a leak of inner ear fluids (perilymph) into the middle ear. Commonly mentioned in the differential diagnosis of those with fluctuating hearing loss, vertigo, tinnitus and aural pressure following trauma. It should however be considered a diagnosis of exclusion. It has been likely over diagnosed in the past much to the detriment of patient care where there may be other more likely causes.

Persistent Postural Perceptual Dizziness (PPPD): a descriptive term for dizziness under the umbrella of a Functional Neurological Disorder. It is characterized by chronic, non-spinning dizziness exacerbated by motion, visual stimulation or upright posture.

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.

Semicircular canals: consists of three, fluid-filled canals that act as an internal gyroscope for angular acceleration perception essential for balance and sensing rotational movement of the head.

Stapedectomy/Stapedotomy: surgical procedures for otosclerosis (an inherited disorder where the stapes footplate becomes fixed by new bone growth). 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.

Superior Canal Dehiscence Syndrome: an interesting but relatively rare condition caused by missing bone over the superior semicircular canal leading to what is a called a “third window effect” where brain linings are in intimate contact with the fluid filled superior canal. Can be seen after minor head injury trauma. Affected individuals have rather unusual symptoms of dizziness triggered by loud noise (the Tulio phenomenon) or sneezing/coughing/straining, pulsatile tinnitus synchronous with their heartbeat, hearing one’s own voice abnormally (i.e., autophony – sometimes described as “Darth Vader-like” sound) and even hearing their eyes move.

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

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

Vestibular cerebellar degenerations: refers to damage/degeneration affecting both the cerebellum and peripheral vestibular pathways leading to severe balance, coordination and eye-movement abnormalities.

When present clinically without additional neurological features it is called **cerebellar ataxia with vestibulopathy** or **CABV**. When a neuropathy involves the lower extremities the diagnosis becomes cerebellar ataxia with neuropathy and the **vestibular areflexia syndrome** or **CANVAS**. Different genetic links have been discovered for both conditions. Symptoms of chronic dizziness, vertigo, unsteadiness, and nystagmus require multimodal therapy, including physical and occupational rehabilitation.

Vestibular pathways: Consists of neural pathways responsible for balance and spacial orientation arising from the inner ear traveling to the brainstem/cerebellum which include the following:

i) **The Vestibular Ocular Reflex (VOR)** which is responsible for compensatory eye to head movements to prevent visual blurring when moving.

ii) **Vestibulospinal reflexes** that co-ordinates reflexes to prevent one from falling which primarily arise from the otolithic organs, and

iii) **Vestibulocerebellar reflexes** which assist with rate, rhythm, speed and accuracy of movement (i.e., co-ordination).

See the Vestibular Pathways Diagram in the *Brief Description of Vestibular Physiology* section.

Vestibular Rehabilitation Therapy (VRT): a specialized, exercise-based physical therapy program designed to reduce dizziness, vertigo, gaze instability and imbalance caused by inner ear or brain disorders. It uses customized techniques – including gaze stabilization, habituation, and balance training – to retrain the brain to process balance signals effectively. VRT is effective for conditions such as BPPV, acute or chronic uncompensated vestibular impairments and concussions, aiming to improve daily functional activity and to reduced risk of falls.

Introduction

Simply put, dizziness is complicated. The shopping list which forms the differential diagnosis for this condition is extensive and arises singularly or in combination with inner ear, neurological, cardiovascular, mental health and iatrogenic causes. It is a condition most physicians feel uncomfortable managing. Where precise history is important for diagnostic purposes there lies a lack of clarity by what is often meant by the terms “dizziness” and “vertigo” and what the individual perceives. Language is unfortunately not as quantifiable as metrics from laboratory testing and the above terms can mean different things to different people.

In this 3rd edition of the discussion paper on *Dizziness* I have tried to make a more readable version for the lay person and at the same time provide some understanding of the basic physiology and research in this field. There remain many unanswered questions especially in the context of dizziness following head injury (i.e. why some patients with the same injury respond differently where sometimes to the extreme they are completely incapacitated).

Over the years I have come to a greater appreciation of the psychological effects of head injury on the mental health individuals with dizziness which can act as a barrier to their recovery. Conversely, we have seen how vestibular rehabilitation therapy can truly help those with a fixed vestibular (balance) impairment and how a knowledgeable and empathetic physiotherapist may be of immeasurable benefit in helping a head injured worker improve.

The glossary of definitions from underlined definitions/phrases of importance has been alphabetically placed in the front for easy reference. *The Physiology of the Vestibular System* section has been made more concise and the language made less technical. For those interested in further technical explanations regarding laboratory testing and the diagnostic criteria for certain clinical conditions, this can be found in Appendix 1.

What is reported in textbooks unfortunately has very little to do with what is commonly seen in clinical practice in many instances. The UHN Hertz Multidisciplinary Neurotology is unique in North America. It is a dedicated specialized clinic where neurotologists, neuro-ophthalmologists, vestibular psychiatrists, advanced practice vestibular physiotherapists, vestibular nurses, fellowship trainees and staff from the UHN Centre for Advanced Hearing and Balance Testing provide a “one stop shop” for patients with routine and complex presentations for their dizziness/imbalance. The results from this clinic can be seen as a proxy for what occurs outside of head injury in the real world and can be contrasted with dizziness/imbalance following head injury from the comprehensive Workplace Safety and Insurance Board (WSIB) Head Injury Neurotology Database. Sections entitled *What Big Data Demonstrates in Head Injury* and *Specific Causes*

of Vertigo/Dizziness Identified or Suspected to have a Traumatic Link present our findings.

The additions of new sections on *Medically Unexplained/Functional Neurological Dizziness* and *Vestibular Rehabilitation Therapy* further provide the reader with new concepts of how one's mental health can affect their perception of dizziness and why there can be further barriers to recovery. We however end on a positive note regarding how vestibular rehabilitation therapy may help many to recover or improve significantly.

Responses on selected topics commonly encountered in cases before the Workplace Safety and Insurance Tribunal (WSIAT) regarding dizziness are also provided.

For those interested more detailed information concerning the Clinical Evaluation of the Dizzy Patient, the Basic Physiology of the Vestibular System and what training ENT residents/fellows are expected to know can be found in Appendix 2 (*Evaluation of Vertigo in Office Based Surgery*), Appendix 3 (*Physiology of the Vestibular System*) and Appendix 4 (*Vestibular and Balance Disorders*) respectively at the end of the discussion paper.

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, inner ear (vestibular labyrinth) and muscle, joint and sensory (i.e. proprioceptive) receptors that is 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 1 a,b.

Fig. 1a Cochlea and Vestibular Apparatus

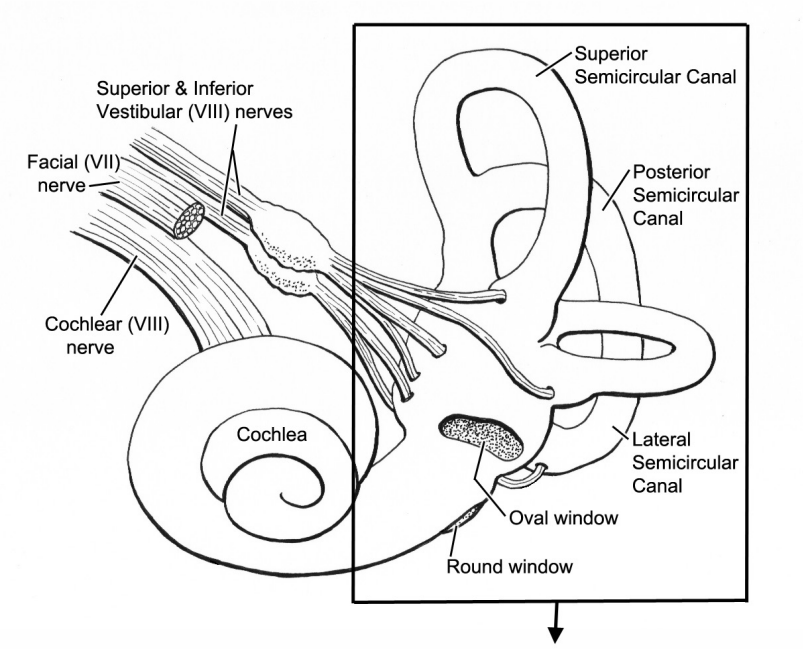
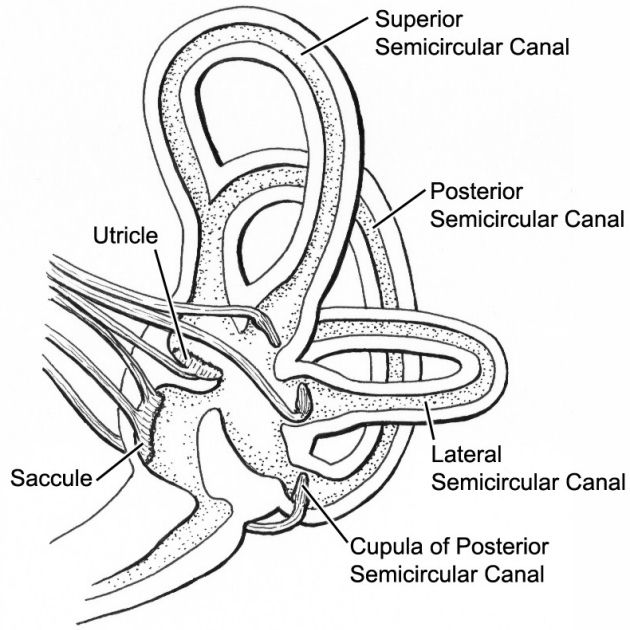


Fig. 1b Vestibular Apparatus



While the function of the cochlea will be largely understated in this medical discussion paper on *Dizziness* the reader is advised to see the previously published WSIAT medical discussion paper on *Hearing Loss and Tinnitus* (2013) should further information be required.

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

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 and resemble internal gyroscopes. Corresponding but equal eye movements are produced in the plane of the canals when they are stimulated. Stimulation of the semicircular canals occurs when the gelatinous hinge joint (or cupula) sitting atop the sensorineural receptor called the ampulla is deflected as a result of inner ear fluid movement within the canals. This typically occurs when the head moves. A similar phenomenon occurs when linear accelerations (i.e. thinking of stopping or starting in a car) cause a shift in the calcium carbonate lining of the otolithic organs (i.e. crystals). See Figures 2 a,b and 3 a,b.

Fig. 2a Cupula of Semicircular canal

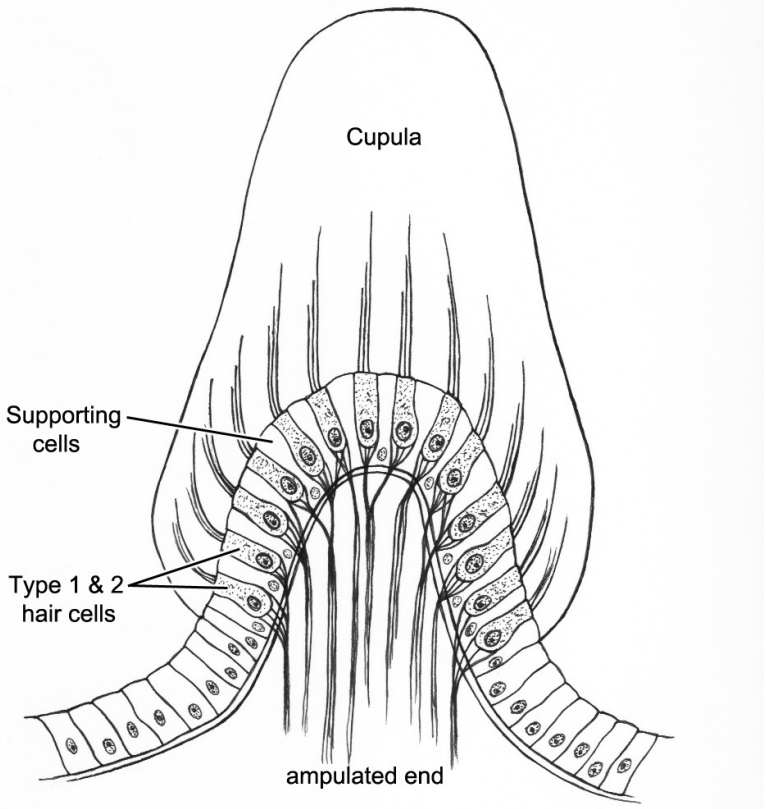


Fig. 2b Otolithic Organs at Rest

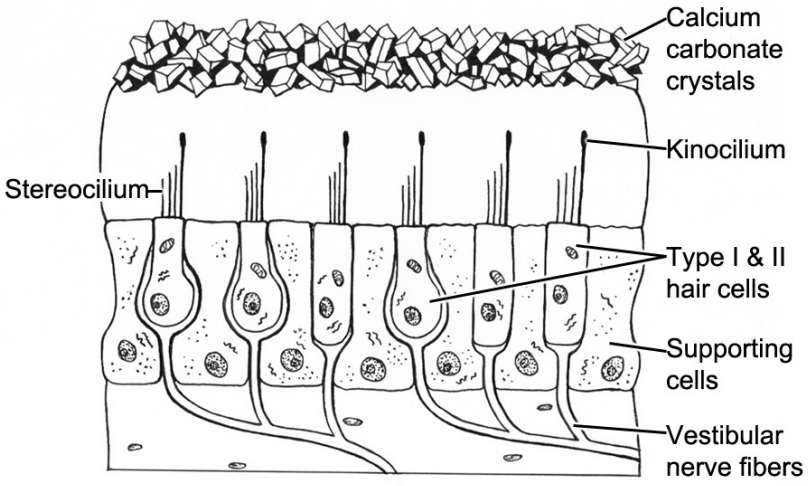


Fig. 3a Cupula Displacement in Semicircular Canal Following Movement

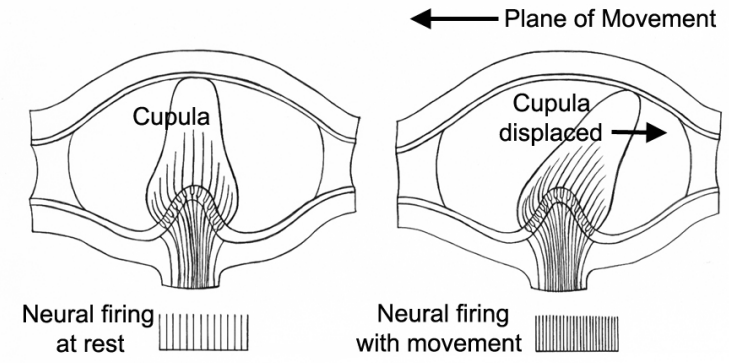
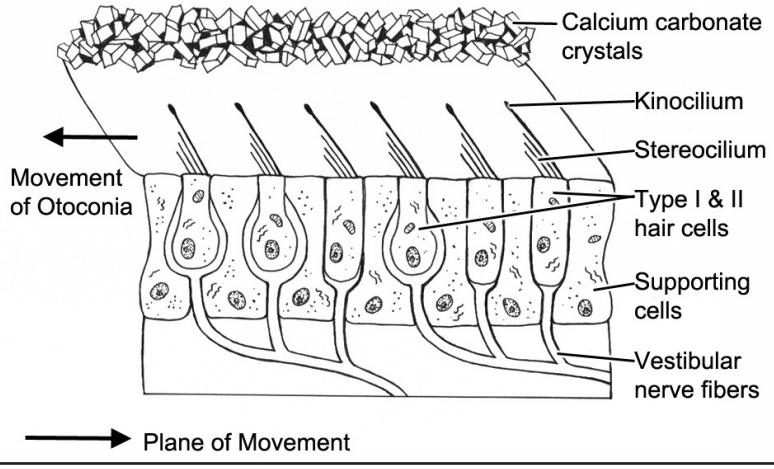
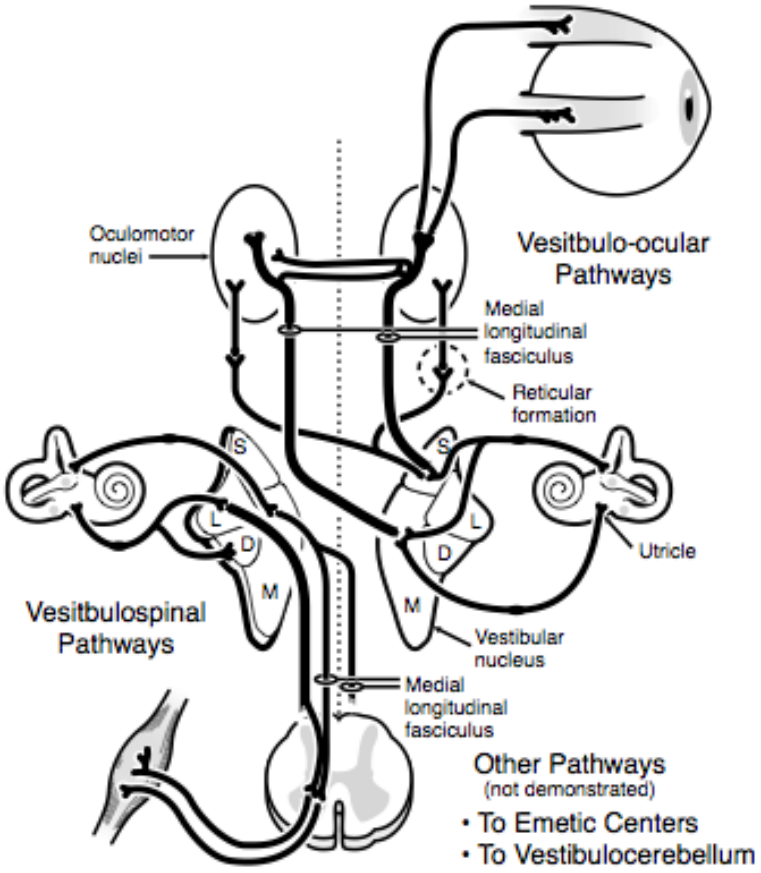


Fig. 3b Otolithic Organs with Movement



Complex neuronal activity generates electrical information from spontaneously generated (tonic) and active stimuli such as a head movement (dynamic) which is sent is sent to the central vestibular nucleus in the brainstem via both divisions of the vestibular nerve (superior and inferior). Interconnecting neural pathways to the eye movement centres cause the eyes to move in an equal but opposite direction to the head movement so our vision is stable when moving. This forms the basis for what is called the **vestibulo-ocular reflex (VOR)**. Conversely the otolithic organs (saccule and utricle) provide information that provides the basis for **vestibulospinal tracts (VST)** important in providing reflexes that prevent falls by steadying our balance when we slip. Vestibulocerebellar pathways (involved in co-ordination) and direct pathways to the vomiting (emetic) centres round off the neurological pathways that form the vestibular system. See Figure 4.

Figure 4. 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 on their own unfortunately do not have the ability to move the eyes fast enough to produce the movements necessary to stabilize gaze which is the role of the inner ear. Defects in VOR activity results in a loss of dynamic visual acuity (DVA) which can lead to visual blurring also known as oscillopsia (visual blurring with head movement)

The brain is able to decode changes in electrical activity depending on how an individual moves their head from the inner ear information provided and associates these changes with a movement. A conflict however arises when pathology involving the vestibular system alters the steady state information coming from the inner ears. 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 (a rapid rhythmical movement of the eyes) and abnormalities clinically in other vestibular pathways.

Over time the brain can compensate for changes in electrical activity between sides for a loss of vestibular function. However, should weakness persist then an individual may never completely compensate and may complain of transient imbalance with 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, a number of vestibular mediated tracts arise from inner ear input to the brain. The otolithic organs (saccular and utricle) are primarily responsible for upright balance and linear acceleration perception (i.e. think of what we feel when braking quickly in a car and gravity perception best recognized paradoxically in astronauts experiencing space sickness in its absence). Vestibulospinal tracks are responsible for the righting reflexes that prevents us from falling and largely arise from the otolithic input. The semicircular canals are primarily responsible for sensing head movement which forms the basis for the vestibular ocular reflex (VOR) helping to stabilize gaze when moving.

Clinical Presentation

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

a. Unilateral Vestibular Loss

An acute loss of inner ear function (i.e. a skull fracture disrupting the hard bony lining (or otic capsule) of the inner ear causing direct mechanical injury) ultimately results in a disruption of the vestibular pathways within the brain and is associated with a number of specific signs/symptoms.

In an acute traumatic inner ear loss (or traumatic cochleovestibular loss) the patient may experience hearing loss of varying degrees and/or tinnitus (unwanted head noise). They will also be vertiginous as well. Disruptions of VOR pathways cause blurring of vision and the physical finding of nystagmus (i.e. a rapid rhythmical movement of the eyes with the fast phase of eye movement typically directed away from the side of lesion). The patient will seem ataxic, inco-ordinated and possibly impaired or “drunk”-like from disruption of vestibulocerebellar pathways. With an attempt to walk they will fall towards the involved side from disruption of vestibulospinal pathways. They tend to lie as still as possible as any movement will make them nauseous to the point of vomiting from disruption of pathways to the emetic centres.

Over time and under the influence of a healthy contralateral inner ear, brainstem vestibular nucleus and a functioning vestibulocerelbellum, the process of compensation occurs. They become less vertiginous upon movement and the nausea subsides. They become more active. If their vestibular loss is permanent however they may always feel as sense of transient imbalance with a 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 (VRT). See discussion in Section on *The Role for Vestibular Rehabilitation Therapy*.

Unfortunately, should pathology involve the contralateral inner ear, the brainstem or vestibulocerebellum then the compensation process may be impaired resulting in a permanent sense of “dizziness” and imbalance.

b. Bilateral Vestibular Loss

Loss of bilateral vestibular function typically does not cause an individual to experience vertigo. Their major complaint is oscillopsia or visual blurring with head movement (due to a 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 rarely be expected to occur following trauma. It however can be seen in the context of Meniere’s disease and 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.

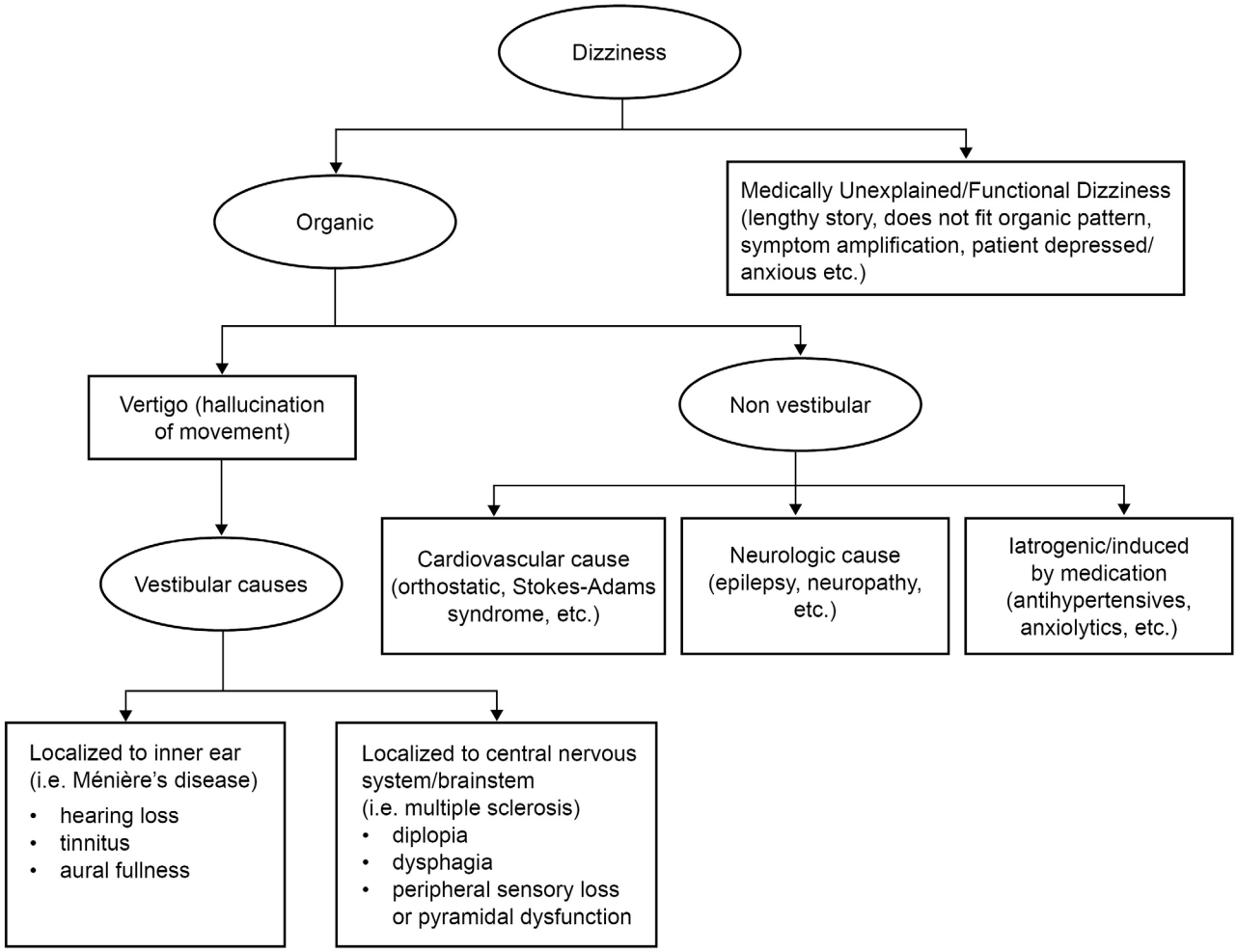
a. History

The history taking begins by asking what "dizziness" means by encouraging the individual to describe their experience with this symptom (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 (i.e. functional or arising from a non-vestibular cause) primarily.

Most individuals with inner ear or vestibular nerve related pathology describe their attacks in a concise, generally consistent fashion regarding onset, duration and the association with certain provocational factors (for example, positional induced). The association with inner ear dysfunction requires enquiry for other symptoms such as tinnitus (unwanted head noise), aural pressure/discomfort and hearing loss (permanent or fluctuant). When dizziness is primarily based on the non-organic or from a functional amplification of symptoms the history is typically lengthy and difficult for the examiner to appreciate, even understand. There are often many unexplained symptoms that cannot be tied together by known anatomical and physiological processes.

Vertigo can also arise from central nervous system (CNS) dysfunction involving the central vestibular pathways (albeit less likely). The proximity of nearby cranial nerve nuclei in the brainstem stresses the importance of asking for symptoms representative for focal neurological dysfunction such as diplopia (double vision), dysphagia (difficulties swallowing), paresis (focal weakness), paresthesia (numbness orf tingling) and incontinence of bowel or bladder activity. Loss of consciousness (LOC) would not be expected and when present requires exclusion of other central mediated 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 the Evaluation of Vertigo Flowchart, Figure 5.

Figure 5: 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.)

b. The Neurotological Examination

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

The ears are examined to primarily 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 (i.e. trying to pop one's ears (Valsalva manoeuvre), sneezing, coughing) by history cause vertigo/disequilibrium then so-

called “fistula* tests” are required to determine if there is an anatomical or physiological abnormality involving the bone of the otic capsule or the delicate lining membranes of the inner ear. Dizziness from loud noise exposure is called the Tullio phenomenon. Alternation of air pressure in the ear canal evoking dizziness is called the **Hennebert’s phenomenon**. A key objective finding is the presence of nystagmus to the stimulus whether it be sound or pressure stimulation.

The neurological portion of the examination primarily assesses cranial nerves, cerebellar and eye movement (or oculomotor) function. Eye movements are of particular interest as their neural pathways tend to be diffuse within the brain and function can be affected by central pathology (structural, metabolic, degenerative etc.). The eyes are carefully examined for nystagmus (a rapid, rhythmical conjugate movement of the eyes with fast and slow components) which is a cardinal sign for vestibular dysfunction, both peripheral (inner ear or vestibular nerve related) 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.

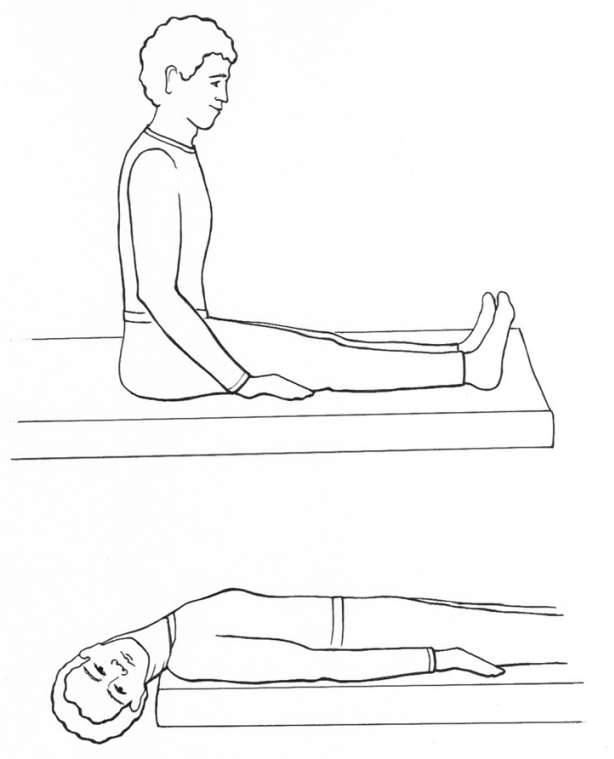
Special clinical tests of vestibular function that should be performed in all patients with dizziness that include:

- **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 continued small movements after the head has stopped. These are called overt or refixation saccades.
- **Head shake test –** Rapid horizontal shaking of the head for approximately 20-25 seconds charge the inner ears “like a battery” and may result in transient nystagmus directed usually away from the ear involved by pathology. This is best seen using Frenzel’s glasses that prevent visual fixation that sometimes suppresses the nystagmus.
- **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 movement leading to oscillopsia (defined as visual blurring with head movement). This is best checked with a routine eye (or Snellen) or Logmar chart used for assessing vision. Loss of visual acuity greater than 5 lines on active horizontal head movements would imply the presence of bilateral vestibular pathology.

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

- **Hallpike's manoeuvre –** This test remains the most important to perform on any dizzy patient owing to the relatively high prevalence of benign positional paroxysmal vertigo (BPPV) especially in the head injured patient. 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. The same manoeuvre is performed on the other side. With a positive test results comes a confident diagnosis for BPPV. See Figure 6.
- **Vestibular ocular reflex suppression (VORS) –** Inability to suppress physiological nystagmus during rotation suggests the presence of a defect in the vestibulocerebellum and is suggestive for a CNS abnormality.

Figure 6: Right Hallpike's Manoeuvre



Relevant Laboratory Audiological and Vestibular Testing

a. Audiometry

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 **speech reception threshold** (SRT) – the lowest intensity of sound an individual can clearly hear a certain word and **speech discrimination scores** (SDS) – the percentage of words an individual can hear from a list of common phonetically balanced words in the English language.

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 or ECoG), along the cochlear nerve and into 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 otic capsule defects such as the superior semicircular dehiscent canal dehiscent (SSCD) syndromes. Increased summating potential to action potential (SP/AP) ratios typically > 0.30 appear to correlate with the phenomenon of endolymphatic hydrops (not infrequently found in Meniere's disease)

ii) Auditory Brainstem Response (ABR)

Synonymous with the term, brainstem evoked potentials (BEP) this test measures electrical waveforms obtained during the first 10 msec of 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 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 a tumor such as a vestibular schwannoma/acoustic neuroma 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.

b. Vestibular Function Testing[4]

Laboratory vestibular function 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 over

time and whether there might be a contraindication to interventions where inner ear vestibular activity is intentionally worsened to paradoxically stop dizzy attacks occurring.

Electronystagmography/Videonystagmography (ENG/VNG)

This commonly performed test of vestibular testing records eye movements with convention 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
- eye movements (oculomotor tasks such as pursuit, saccade, optokinetic and ocular 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 the lateral (or horizontal) semicircular canal activity 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 difference in nystagmus generated between the 2 sides is called the **excitability difference (ED)**. In many testing laboratories an ED > 20% would be considered significant for a vestibular loss.

While the test is not without its concerns (i.e. it is not truly a physiological 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 normal caloric responses generally implies that normal inner vestibular activity is present may not be always entirely accurate etc.) it still remains the workhorse for conventional vestibular testing.

Vestibular Evoked Myogenic Potential (VEMP) Testing

When brief and 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 muscle (or myogenic) response in the nearby muscles of the eyes and neck. Short latency responses from the anterior neck and extraocular muscles to sound stimulation have been collectively known as vestibular evoked myogenic responses or VEMPs.

Surface recording electrodes placed on the sternocleidomastoid muscle in the neck record cervical VEMP (or cVEMP) responses. When placed around the lower rim of the orbit they can record ocular VEMP (or oVEMP) responses. cVEMP responses are thought primarily to arise from the saccule and oVEMP responses from the utricle.

The importance of cVEMP and oVEMP testing resides in their ability to provide comparative information concerning the otolithic organs and to indirectly assess the integrity the vestibular nerve (both superior and inferior divisions).

Unfortunately, age, an inability to generate sufficient muscle tone in the neck and any form of middle ear pathology causing a conductive hearing loss may result in increased threshold or absent VEMP responses. Increased amplitude responses at decreased thresholds for stimulation may be identified in individuals with Meniere's disease, a superior canal dehiscent syndrome (SCDS) or perilymphatic fistula (where there is a fluid leak from the inner ear into the middle ear) making them invaluable at times.[5]

Video Head Impulse Testing (vHIT)

Lightweight goggles with built in high-speed recording cameras can capture eye movements when coupled to a gyroscope that measures head rotation which forms the basis of this test used to measure horizontal and vertical VOR function. It is a physiologic study of how the eyes move relative to a fast head movement that under normal circumstances 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 smaller eye movement(s) necessary to fixate on the target (called refixation saccades). Overt or obvious "**catch-up**" saccades are visible to the naked eye clinically. Covert saccades are embedded in a corresponding eye to head movements that occur so quickly they are difficult to see. On clinical ground they probably indicate a better degree of central compensation for an underlying VOR impairment.[6,7]

The vHIT test is not affected by middle ear patholology (unlike caloric and VEMP testing). Disadvantages are usually technical in nature as there may be goggle slippage with associated artefact with head impulses/rotations > 200-300°/s.

Rotational Chair Testing

Conventional ENG recording electrodes or VNG recording devices capture the horizontal VOR during sinusoidal rotations in a moving chair. Information from both ears can be recorded simultaneously. While this test is more physiologically based (when compared to ENG/VNG caloric testing), the equipment is expensive and most chairs are limited in sinusoid rotations up to 2 Hz (cycles per second) which might miss higher velocity VOR dysfunction better captured on vHIT.

Clinical Posturography and Computerized Dynamic Posturography (CDP)

Home modified or trademark patented (i.e. Equitest ®) technology is available for the overall assessment of balance function which assesses the effects of vision, vestibular and proprioception in overall balance performance. A balance board measures centre of pressure changes and body sway to evaluate balance. Information is provided through what are called **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 sudden movements (from 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 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.[8] 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 has limited their widespread clinical adoption in many centres.

What is Commonly Seen in Clinical Practice – The Hertz Multidisciplinary Neurotology Experience at the UHN [9,10]

Insight into the common causes for dizziness in clinical practice outside head injury and findings from the UHN Hertz Multidisciplinary Neurotology Clinic are presented.

While dizziness can be a common complaint in mild head injury (along with headaches, neuropsychological and neurocognitive complaints) objective evidence for vestibular impairment is not always identified (see the 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 UHN Multidisciplinary Neurotology Clinic in Toronto have revealed the following diagnoses by causation made at presentation to clinic staff. See Table 1 below.

Table 1: Diagnoses by Causation

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

Inner ear disorders accounted for the majority of visits to this specialized clinic. Well over 50% of patients had either the condition of benign positional paroxysmal vertigo (BPPV) or a recurrent vestibulopathy (RV) defined as episodic attacks of vertigo without hearing loss, tinnitus or neurological dysfunction. While Meniere's disease and vestibular neuronitis (VN) had the most spectacular clinical presentations their incidence was relatively low by comparison. See Table 2 below.

Table 2: Common Inner Ear Disorders

Common Inner Ear Disorders (N=650)*	Percentage
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%

The Others category included rare but interesting causes that included ototoxicity (both systemic and topical), **delayed endolymphatic hydrops** (considered to be a variant of Meniere's disease), drop attacks (the so-called otolithic crisis of Tumarkin), complications of ear surgery (i.e. **mastoidectomy, stapedectomy surgery**), **superior semicircular canal dehiscence syndromes** and neurosyphilis.

Continued study of additional patients referred to the Hertz Multidisciplinary Neurotology Clinic over the ensuing year has demonstrated a remarkable consistency in presentation. 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 was the finding that more than one peripheral vestibular disorder could be present in the same person over time. In the first 1000 patients

* 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*."

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 VN to evolve into an RV and then into BPPV over time.

Central nervous system causes for vertigo were relatively rare by comparison but heterogeneous in presentation. The presentation of certain CNS disorders however seemed age related to a large degree. See Table 3 below.

Table 3: Common CNS Disorders

Common CNS Disorders (N=71)	Percentage
1. Idiopathic vestibulocerebellar degeneration CABV/CANVAS)	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%

Cerebellar ataxia with bilateral vestibulopathy (CABV) and **cerebellar ataxia with the vestibular areflexia syndrome (CANVAS)** represent combined bilateral peripheral vestibular and vestibulocerebellar degenerations that are now known to have a genetic linkage.

Cerebrovascular accidents (CVAs) such as strokes can involve the brainstem and especially the vestibular nuclei. Transient ischemic attacks (TIAs) represent short lived reversible episodes of neurological dysfunction on a vascular basis which is often a warning sign for a more serious stroke unless the underlying cause is treated. In vertebrobasilar insufficient (VBI) the posterior circulation supplying the brainstem is affected by hypo-perfusion that can result in syncopal attacks where there is a loss of consciousness. **Multisensory deficits (MSD)** generally reflect a “grumbling” down of all systems response for balance that includes vision, inner ear, proprioceptive and central integrative function.

Common Peripheral (or Inner Ear) Disorders Associated with Dizziness/Vertigo

While causes for dizziness/vertigo can be exhaustive in any given text, it should be understood that “**common things are common**”. A list of commonly seen vestibular disorders frequently mentioned are discussed below.

a. Benign Positional Paroxysmal Vertigo (BPPV)[11]

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 (i.e. looking up, bending over, rolling over in bed to the affected side). Most of the time it occurs spontaneously but is often identified after head injury, following viral inner ear disorders and after ear surgery (**mastoid and stapes surgery** especially). It can be unilateral or bilateral in its presentation.

The cause is thought to arise when released otoconia ("little stones" or "crystals") from the otolithic organs are released and enter into the posterior semicircular canal which is the most gravity dependent of all the semicircular canals. With a provocative head movement vertigo is caused when the "crystals" move in the canal (canalolithiasis) or should the "crystals" be fixed to the cupula (cupulolithiasis). Both cause an abnormal deflection of the cupula causing the associated symptoms. Both horizontal and superior canals can be affected by the same pathological processes but to a lesser degree.

In classic posterior canal based canalolithiais (which seems the best model for BPPV overall) the Hallpike's manoeuvre to the affected side causes an individual to experience a sense of vertigo following a latent time frame of a few seconds that is associated with geotropic (towards the ground) beating rotatory nystagmus lasting 5-30 seconds that crescendos, then decrescendos over this timeframe. The patient is often nauseous to the point of vomiting. When brought upright a reversal of the nystagmus occurs (an ageotropic reversal) lasting shorter duration. Repetitive testing causes the response to fatigue.

Most cases of BPPV tend to spontaneously resolve although recurrence is not uncommon (especially in the post traumatic setting). Physical therapy exercises have proven effective in the management of this condition. Those commonly performed include the Epley manoeuvre (or particle repositioning procedures), Brandt-Daroff exercises for habituation/adaptation and the Semont's libertary manoeuvre.[11]

Should BPPV not resolve spontaneously and prove recalcitrant to physical therapy maneuvers then surgical options may be considered. Procedures such as labyrinthectomy (where the affected ear is surgically removed), vestibular neurectomy (cutting the balance nerve) and singular neurectomy (cutting the nerve that goes to the posterior canal) 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.[12]

b. Meniere's Disease[13-15]

Meniere's disease is protean in presentation (not always presenting with all the typically expected symptoms together). In its classic description patients experience recurrent attacks of episodic vertigo lasting min-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 presentation is typical but over a patient's lifetime it is estimated that bilaterality may occur upwards of approximately 50%. Prior demographic studies from the UK approximately 95% of patients will present with all 3 symptoms of Meniere's disease within 5 years of the initial symptom onset. Periods of remission and exacerbation remain the rule.[13]

The pathophysiology is generally thought to arise from idiopathic endolymphatic hydrops (distention of the delicate lining membranes in the inner ear) leading to membrane ruptures causing an attack to occur. The Na+-K+ intoxication theory from the admixing of inner ear fluids (perilymph and endolymph) with different ionic constituents is widely used to explain the physiologic basis for an acute Meniere's attack.[15]

The majority of patients with Meniere's require symptomatic treatment only and reassurance mostly when their attacks are relatively infrequent. Preventative measures stress and dietary modification (low salt diets, avoidance of caffeine etc.) are usually recommended. Medical management is based on the prevention of hydrops (using diuretics, anti-secretory factor therapies etc.) or minimizing neurochemical responses with vasodilator and histamine agonists (i.e. betahistine or Serc ®), Ca2+ channel blockers etc.). Intratympanic therapies (IT) involve aminoglycoside (primarily gentamicin) or steroid (decadron) injections. Surgery is generally undertaken when unilateral disease fails to go into spontaneous remission or respond to medical management. These procedures can include surgery on the endolymphatic sac, tentotomy surgery (where the tendons to the middle ear bones are cut), vestibular neurectomy or labyrinthectomy.

c. Recurrent Vestibulopathy (RV)[16]

Recurrent vestibulopathy (RV) is a descriptive term and the 2nd most common cause for vertigo (after BPPV) identified in the UHN Hertz Multidisciplinary Neurotology Clinic. By definition it 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 a RV remains unknown but there is speculation in some instances arise from transient vestibular dysfunction, possibly viral mediated.

Natural history studies have demonstrated that over an 8½ longitudinal follow-up 60% of those affected 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.

The recent introduction of delayed contrast gadolinium enhanced temporal bone MRI shows promise in being able to visualize endolymphatic hydrops and possibly predict who will evolve to Meniere's.[17]

For the time being treatment is purely symptomatic unless the involved side can be accurately localized.

d. Vestibular Neuronitis (Neuritis)

Synonymous with "labyrinthitis" at the family practitioner level, this condition probably arises from an acute viral induced inflammation of the vestibular nerve (herpes simplex and zoster viruses have been implicated). The affected individual experiences an acute attack of vertigo lasting days to weeks **not** associated with hearing loss, tinnitus or evidence of focal neurologic dysfunction. Caloric testing especially in the acute setting finds a profound or absent caloric response on the affected side. In approximately 50% the caloric response returns to normal within 6 months. CNS compensation usually occurs in those whose 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[18]. Vestibular rehabilitation therapy has proven effective by promoting vestibular compensation and 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)[19]

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 histology they are really vestibular schwannomas (arising from the Schwann cells that cover vestibular neurons). They are relatively rare (1/10,000 population), although one suspects their incidence may be on the increase primarily as we are able detect smaller and at times incidental tumors on MRI. Bilateral involvement usually implies the condition of NF-2 (an autosomal 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**". Important tests used to confirm or exclude an acoustic neuroma include ABR an CT or MRI scanning. MRI is considered to be the "gold standard". Management paradigms include a trial of conservative management ("wait and scan" approach), stereotactic radiosurgery or microsurgical removal.

Dizziness Following Head Injury

Dizziness, headache and neurocognitive/neuropsychological complaints are commonly reported symptoms after head injury.[20] Most clinicians would not disagree with this statement. That being said, for those further interested an excellent account of symptoms post head injury can be found in the WSIAT medical discussion paper on *Traumatic Brain Injury and Concussion* (2023).

While much has been written about minor traumatic brain injury (mTBI) following concussion and sports related injury, large scale population studies addressing dizziness from workplace head injury do not exist nor have they been appropriately studied. Longitudinal outcomes for dizziness post-head injury are generally not available, excepting in certain select patient series (i.e. post-traumatic BPPV). There remains significant controversy over the terminology used (i.e. the use of dizziness vs. vertigo) and whether certain conditions such as post-traumatic endolymphatic hydrops (i.e. Meniere's-like attacks), cervicogenic vertigo (where injury to cervical neck proprioceptors play a role) and trauma causing leaks of fluid from the inner ear to the middle ear (also known as a **perilymphatic fistulae or PLF**) exist.

To add to the confusion, one suspects that what is reported in the world literature has been inappropriately amplified through a lens of personal bias looking at only

selected conditions. The *What Big Data Demonstrates Section* aims to provide more clarity to controversy.

The interplay of symptoms from a traumatic vestibular disorder and other associated non-organic (i.e. mood change, functional amplification, **catastrophization** etc.) features requires careful consideration. Both may converge and adversely influence each another including other workplace considerations (i.e. return to a toxic work environment, concerns about safety etc.). Persistent symptoms that do not improve with time and directed vestibular physiotherapy (better known as **Vestibular Rehabilitation Therapy (VRT)**) do not follow the anticipated trajectory where compensation is generally the rule.

Euphemistic terms such as medically unexplained dizziness, psychogenic dizziness, non-organic dizziness and chronic non-specific dizziness have all been applied. Predisposing circumstances, persistent dizziness and specific symptoms absent of fluctuant vestibular activity have received diagnoses such as **mal d'embarquement**, chronic phobic dizziness, **persistent perceptual postural dizziness (PPPD)** etc. While a traumatic vestibular injury may have been the epiphenomenon, the persistent downstream complaints strongly indicate another generator site within the brain. In those affected vestibular rehabilitation therapy seems of limited value. Treatment with mood stabilizing medications (i.e. the SSRI or SNRI classes of antidepressant therapy) and interventions such as cognitive behavioral therapy (CBT) seem likely to be recommended. A diagnosis of a **cognitive vestibular disorder** (considered a specific subset of a **Functional Neurological Disorder**) seems best under the circumstances.

What Big Data Demonstrates in Head Injury: Findings from the Comprehensive UHN WSIB Neurotology Database of 4,291 Head Injured Workers (1988-2018)[21-22]

In what is the largest comprehensive prospective database available, from 1988 to 2018, 4,291 head injured workers from the Workplace Safety and Insurance Board (WSIB) of Ontario with dizziness following head injury have been investigated for this complaint. All underwent a thorough neurotological assessment by the senior author including a formal neurotological examination and laboratory inner ear testing. The results provide a unique window with which to view large data to identify what is commonly seen following head injury.

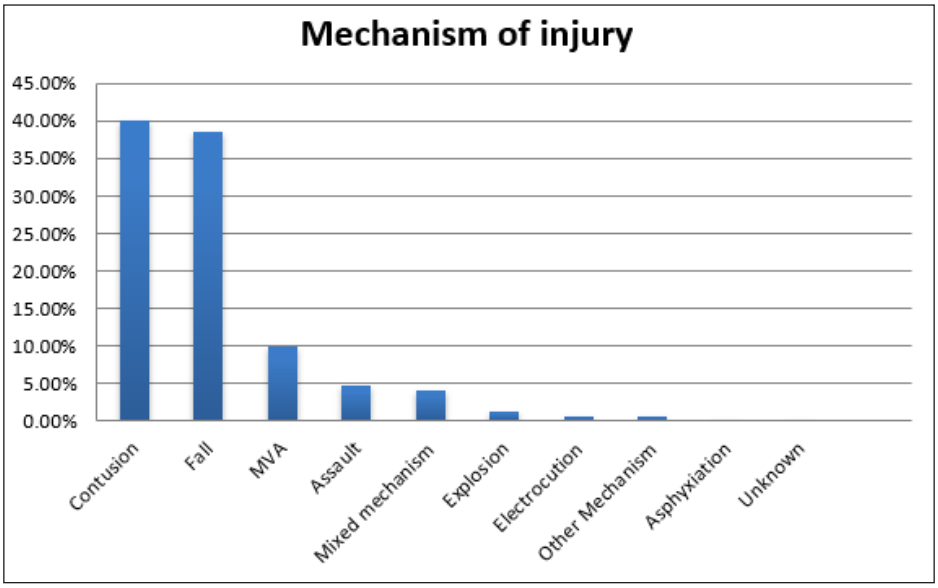
Males were at greater risk for head injury in the workplace compared to females, M:F, 2860 (66.6%):1431(33.3%) which largely is reflective in the prevalence involving physical work (i.e. construction, trades). Demographic changes however are expected in future as more females assume greater participation in what were once considered typical male professions.

The majority of work-related injuries (approximately 79%) occurred as a result of either a fall (38.45%) or blow to the head (38%). Motor vehicle accidents (MVAs) usually involved commercial drivers. Assaults in the workplace had a higher prevalence affecting health care professionals (nurses and personal support workers (PSWs) rather than security or policing professions). Rare causes for workplace trauma over the 30 years of assessment included injuries from explosions (typically tire mechanics) and electrical injuries (electricians/construction workers). See Table 4, Figure 7.

Table 4. Mechanism of Injury (n= 4291) from the UHN WSIB Neurotology Database 1988-2018

Mechanism of Injury	Number	Percentage
Fall	1654	38.45%
Contusion	1718	40.03%
MVA	423	10.2%
Mixed Mechanism	172	4.0%
Assault	205	4.77%
Explosion	55	1.28%
Other Mechanism	44	1.3%
Electrical Injury	29	0.67%
Asphyxiation	5	0.11%
Unknown	5	0.11%
Total	4291	100.0%

Figure 7. Mechanism of Injury (n=4291) UHN WSIB Neurotology Database 1988-2014



A spectrum of injury severity involved falls and contusion injuries from mild to severe. Falls could involve a simple slip and fall on an icy surface to a fall from a height while on a ladder/construction site. Contusions could involve a simple “bump” on the head without a loss of consciousness (LOC) from hitting an overhead cupboard to a severe closed head injury with life threatening injuries/permanent neurological sequelae.

The majority of head injuries were deemed minor (77.98%)*. Closed head injuries** and closed head injuries with fractures were identified in 7.65% and 10.99% respectively. Posttraumatic cerebral spinal fluid (CF) leaks were rare and were usually associated with a skull fracture but not always was a fracture radiologically identified. See Table 5. The majority of injuries (66.3%) were not associated with a 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 with permanent neurological sequelae.

Table 5. Severity of Injury (classified on clinical/imaging evidence) from the UHN WSIB Neurotology Data Base 1988-2014 (n= 4291)

Scale	Type of Head Injury	Number	Percent
1	Minor head injury	3472	80.91%
2	Closed head injury	297	6.92%
3	Closed head injury+ skull fracture	429	9.99%
4	Open/compound skull fracture	23	0.53%
5	Minor head injury+CSF leak only	2	0.04%
2+5	Closed head injury + CSF leak	1	0.02%
3+5	Closed head injury+skull fracture + CSF leak	24	0.55%
4+5	Open skull fracture + CSF leak	6	0.14%
–	Unknown/none	37	0.86%
–	Total	4291	100%

* For the purpose of definition a minor head injury (mild traumatic brain injury (mTBI)) is commonly defined as a head injury with a Glasgow Coma Scale score of 13-15, resolution of posttraumatic amnesia within 24 hours and a loss of consciousness for less than 30 minutes.[23] See Appendix 1 Additional Clinical, Technical and Diagnostic Information: Section 3.

** A closed head injury typically represented a more severe head injury usually but not always with a LOC. Imaging post trauma demonstrates evidence for anatomic injury including intracranial bleeding (i.e. subarachnoid, subdural, epidural bleeding etc.), intraparenchymal hemorrhage (i.e. “brain bruising/bleeding”) etc.

Specific Causes of Vertigo/Dizziness Identified or Suspected to Have a Traumatic Link From the UHN WSIB Database [22-24]

Diagnoses from the UHN WSIB Neurotology Database 1998-2018 (N=4291)

All head injured workers had subjective complaints of “dizziness” as entry criteria into this database. In most instances the initial diagnosis made remained unchanged but in some more than one diagnostic entity could apply simultaneously and there was on occasion evolution from one to another diagnostic condition. There was a heterogeneity in the broad diagnostic categories noted which included:

- Central Vestibular
- Medically Unexplained
- Positional Dizziness (subjective, typical BPPV or atypical BPPV)
- Peripheral Vestibular (Recognized peripheral vestibular disorder or evidence for cochlear +/- vestibular loss)
- Unknown
- No diagnosis

Of those listed above positional dizziness, medically unexplained dizziness and those with an unknown diagnosis seemed most common.

Table 6: Diagnostic categories from the UHN WSIB Neurotology Database

Diagnoses	N	%
Central	28	0.65
Medically Unexplained	1166	27.17
Positional Vertigo (Subjective)	714	16.63
Positional Vertigo (Typical BPPV)	137	3.19
Positional Vertigo (Atypical BPPV)	57	1.32
Peripheral Vestibular Disorders (excluding positional vertigo)	244	5.58
Traumatic Inner Ear Loss {Cochlear and/or Vestibular)	885	20.77
Unknown Diagnosis	804	18.73
No Diagnosis	256	5.96

A common complaint was a subjective history for benign positional paroxysmal dizziness (BPPV) (16.63%). Objective findings confirming BPPV however were relatively uncommon at 3.19%. A small percentage had atypical nystagmus patterns in the Hallpike’s manoeuvre not from posterior semicircular canal involvement but likely from other semicircular canals (superior and lateral) or from otolithic contributions. A recent update by Magos et al of 4829 patients confirmed diagnostic stability. [23]

Approximately 885 (20.77%) head injured workers demonstrated loss of cochlear or vestibular function on laboratory testing. The presence of a cochlear and/or vestibular loss however does not always equate to injury from the accident. Clinical correlation is always required on an individual basis to determine whether the accident was causative, whether the findings were from an unrelated pre-existent injury or might even be considered spurious on technical grounds.

Where the work-related injury was felt to be causative a substantive number of workers had a fixed inner ear loss from their injury that remained unchanged and were felt likely to receive benefit from **Vestibular Rehabilitation Therapy (VRT)** with an advanced practice physiotherapist. See Section on the Role of Vestibular Rehabilitation Therapy (VRT).

Peripheral vestibular diagnoses outside of positional vertigo generally included a spectrum of inner ear clinical diagnoses. Episodic attacks of vertigo were diagnosed being compatible with either a recurrent vestibulopathy (RV) or RV with prominent otolithic features primarily when history was suggestive for semicircular canal (i.e. a spinning and circling sensation) or otolithic (i.e. a floating or rocking sensation) involvement respectively. Diagnoses for posttraumatic Meniere's Disease, delayed endolymphatic hydrops and drop attacks were decidedly rare in this database. (See Common and Controversial Causes for Posttraumatic Vertigo Section below). See Table 7.

Table 7: Peripheral vestibular disorders (n= 244) excluding those with suspected or confirmed positional vertigo.

Main Presentation Diagnosis	No. of Workers	% out of peripheral vestibular pathology (n=1105)	% out of total data (n=4291)
RV	78	7.05%	1.81%
Uncompensated fixed vestibular loss	77	6.9%	1.79%
Meniere's disease	11	0.99%	0.25%
DEH	10	0.9%	0.23%
Drop attacks	9	0.81%	0.20%
SSCD	3	0.27%	0.06%
Undiagnosed peripheral	56	5.0%	1.28%

Key: BPPV: Benign paroxysmal positional vertigo, RV: recurrent vestibulopathy, DEH: delayed endolymphatic hydrops, SSCD: superior semicircular canal dehiscence

Medically Unexplained/Functional Neurological Dizziness

Psychiatrists use the Diagnostic and Statistical Manual (DSM)-5 as the accepted "gold standard" for the diagnosis and classification of psychiatric disorders.[25] As defined in the manual, a Functional Neurological Disorder (FND), involves one or more altered voluntary motor or sensory symptoms that are incompatible with and cannot be better explained by another physical or mental disorder. In the context of a post traumatic functional vestibular disorder, the epiphenomenon was the head injury which may or may not have caused loss of vestibular function. The generator for the persistence of dizziness however is likely arising from other areas of the brain.[26]

The described dizziness is persistent having features of unsteadiness and non-spinning typically made worse with upright posture, movement or with visual stimuli that likely involves maladaptive neural responses to an acute vestibular, medical or psychological trigger.

Psychological stressors can involve panic, anxiety and catastrophization (a maladaptive thought process that involves irrational fear and worry about anticipated or actual symptoms). While symptoms are perceived as real by the affected individual, they appear unlikely to be based on fluctuant vestibular function from known recognized organic (inner ear) causes. Diagnostic labels applied have included persistent postural perceptual dizziness (PPPD), chronic subjective dizziness (CSD), phobic postural dizziness and psychogenic dizziness.

Treatment generally involves:

- Vestibular rehabilitation therapy (VRT) with specialized exercises to reduce hypersensitivity to motion.
- Cognitive behavioral therapy (CBT) to reduce the anxiety and hypervigilance that may perpetuate the dizziness.
- Medication trials with selective serotonin reuptake (SSRI) and selective noradrenergic reuptake inhibitor (SNRI) classes of medication.

Other variants to this theme include the condition of **mal d'embarquement** and **unclassified functional vestibular disorders**.

Symptoms of mal d'embarquement include a persistent sense of motion described as rocking, swaying or bobbing and a sense of disequilibrium, never true vertigo that typically follows prolonged exposure to motions such as on a cruise or with airplane travel. Chronic fatigue is an all-pervasive symptom. The clinical examination and results of laboratory vestibular testing are invariably normal. Curiously symptoms significantly improve when in motions such as driving in a car for example.[27]

Unclassified functional vestibular disorders are characterized by dizziness, imbalance and instability that can be neurophysiologically reversible and not caused by structural lesions in the vestibular system. Individuals often exhibit hypersensitivity to movement, visual stimuli or specific environments such as shopping malls, car rides or in high places.

As many of the conditions described above can overlap with one another it is possible that the umbrella diagnosis of a cognitive vestibular disorder might be best considered.

The Role of Vestibular Rehabilitation Therapy (VRT)[28,29]

The goal of vestibular rehabilitation therapy is to promote compensation for a vestibular impairment that utilizes the principles of habituation (desensitization), adaption (gaze stabilization) and substitution (gaze stabilization and the use of other components of the balance system). It has found itself a role in those with a unilateral chronic vestibular loss, those with a bilateral vestibular loss and especially those with BPPV.

In the opinion of the author, it is best provided by a formally trained advanced practice physiotherapist willing to spend the time, effort and the ability to provide the necessary encouragement. It may be extended to those who have medically unexplained/functional vestibular disorders and even those with recurrent episodic vestibular disorders. The results however are not as predictable.

Barriers to the success in treatment include co-morbidities, the long-term use of vestibular suppressant therapies and those with significant anxiety overlay. Our studies have also identified that those on long term disability (LTD) or seeking a legal claim (LC) are not only less likely to receive benefit for management of their chronic dizziness, underlying mental health complaints and catastrophization but also from VRT.[30,31]

Common and Controversial Causes for Posttraumatic Vertigo

a. 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

b. Controversial diagnoses for posttraumatic dizziness

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

Common Accepted Causes for Dizziness Associated with Trauma

a. Posttraumatic BPPV [11,12]

It has been well established that BPPV can arise following head injury. Mechanisms for posttraumatic BPPV are generally based on the shearing effects of the trauma upon the inner ear that results in direct cellular injury or more likely liberates otoconia (crystals) covering the saccule or utricle. Particulate debris 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 it 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 (i.e. PSCC canal occlusion) for definitive treatment in those cases deemed incapacitating by the patient.

b. Delayed Endolymphatic Hydrops [32]

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.[33] A 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 (i.e. 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.[34]

c. Traumatic Vestibular Loss from Direct Injury

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 psychological dynamics post head injury need further attention. Treatment of an underlying mood disorder (i.e. depression, anxiety) cannot be stressed enough.

Controversial Diagnoses in Posttraumatic 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 there are no objective parameters for diagnosis. 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 are not infrequently mentioned.

Controversy however exists on the anatomic basis that the COR appears rudimentary in humans when compared to afoveate animals (i.e. rabbits, birds), that no specific diagnostic test is available to independently confirm this diagnosis and that the pathology/degree of injury to neck proprioceptors is not well defined nor does it seem proportional to the stated injury (i.e. 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. [35]

b. Posttraumatic Vestibular Migraine

Generalized headaches including migraine are commonly reported following head injury. The association of migraine and vertigo (migrainous vertigo) has been reported outside head injury. Post head injury dizziness in conjunction with migraine is more often 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 migraine and dizziness in the general population.

Whether trauma is actually causative cannot be answered as there have been no clinical trials addressing posttraumatic migrainous vertigo specifically. The poor correlation between trauma severity and the development of migraine specifically suggests that other mechanisms such as the emotive changes in post head injury in conjunction with established predisposing factors for migraine (i.e. 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 associated with the development of frequent and debilitating migraine, a trial of migraine prophylactic therapy would not seem unreasonable however.

c. Perilymphatic Fistula (PLF)[36-37]

Ruptures 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. 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.

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 syndromes 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 In the acute situation (especially following penetrating trauma or post surgery) treatment is generally conservative with bed rest and head elevation.

d. Posttraumatic Meniere Disease and Posttraumatic 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.[38] 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. [39]

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 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.

In an earlier study from our unit, 11 patients in a database then 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).[22] This diagnosis represented approximately 0.032% of the entire database. See Table 8.

Table 8. UHN WSIB Neurotology Database 1998-2014

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

Meniere's	N	Age average	Age range	Injury to assessment	Follow-up (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

2. 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

In trying to determine whether Meniere's disease was related to trauma one would reasonably 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 (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 9.

Table 9. Incidence and Prevalence of Meniere's disease in the world literature.

Country	Prevalence/ 100,000	Incidence	Mystery number	Reference
USA	218	15.3	–	–
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 (episodic 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 + 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. [40]

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.

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.[40] 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 LOC (< 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 how a "blow" causing injury to the vestibular system needs to be identified on a detailed history, a formal neurotological examination and together with laboratory vestibular testing.

e. Dizziness from medications

Many classes of medication can trigger dizziness and lightheadedness. Just ask anyone who has watched US pharmaceutical commercials that discussing the side effects of medications being promoted.

A list from MEDSAFE 2017 (the New Zealand Medicines and Medical Devices Safety Authority) is presented below which is not meant to be exhaustive. With the introduction of new medications (i.e. new biologics (monoclonal antibody therapies) for the treatment of oncologic and rheumatology conditions, weight loss medications etc.) expect to see more.

Medicine Class	Examples
Analgesics	codeine
Antibiotics	aminoglycosides, macrolides, minocyline, nitrofurantoin, sulfamethoxazole
Anticonvulsants	levetiracetam, phenytoin, pregabalin

Medicine Class	Examples
Anti-inflammatories	celecoxib, parecoxib, naproxen, prednisone
Antimalarials	mefloquine, quinine, hydroxychloroquine
Antivirals	oseltamivir, raltegravir
Anti-Parkinson's drugs	lisuride
Cardiovascular drugs	nifedipine, furosemide, indapamide, prazosin, terazosin, glyceryl trinitrate, isosorbide mononitrate, sotalol, timolol
Gastroenterology drugs	omeprazole, lansoprazole, sucralfate
Rheumatology drugs	zolendronic acid, alendronate
Phosphodiesterase type-5 inhibitors	sildenafil, vardenafil
Other medicines	lithium, haloperidol, benzodiazepines, desmopressin, melatonin

It is important to recognize that medications can also be ototoxic causing permanent damage to the inner ears (eg, aminoglycosides, anti-inflammatory medicines, phosphodiesterase type-5 inhibitors, furosemide). When a patient is taking a medicine known to be ototoxic presents with dizziness/imbalance dose reduction or discontinuation of the medicine needs to be considered to prevent irreversible hearing/vestibular loss.

f. Dizziness from carbon monoxide (CO) poisoning

CO is a colorless and orderless gas that binds to hemoglobin to from carboxyhemoglobin (HbCO). It subsequently prevents blood from carrying oxygen (O^2). CO binds to hemoglobin about 200-250 times more strongly than oxygen does. In the process it affects hemoproteins (blood related proteins such as myoglobin, cytochrome P450) and mitochondrial cytochrome oxidases. Common symptoms of CO poisoning are headaches, dizziness, weakness, an upset stomach and mental confusion.

The inner ear is highly metabolically active organ with the vestibular nerve having the highest spontaneous firing rate of any nerve in the body. Acting as an ototoxicant, CO can damage all structures within the inner ear from brain hypoxia, disrupted blood flow, cellular toxicity and from its direct effects on mitochondrial function

Damage to the outer hair cells in the cochlea leads the release of glutamate from the damaged cells which causes an "excitotoxicity" reaction that damages the ganglion cells of the cochlear nerve. This can lead to the phenomenon of cochlear synaptopathy or "hidden hearing loss". Under the circumstances an individual may have what appears to be a reasonable conventional audiometry but loses the ability to hear complex speech and sound because of the specific damage to the cochlear nerve.

The vestibular system is reported to be involved more often and although vertigo is cited in some publications it is difficult to appreciate how a systemic condition affecting both inner ears would cause differences in the electrical activity between sides leading to this symptomatic complaint. A sense of imbalance would be more likely the volunteered complaint as a result of bilateral vestibular loss from damage to the vestibular apparatus or from the demyelination of the vestibular nerve resulting in decreased electrical transmission to the brainstem. The central vestibular nuclei can be affected.

Recovery of vestibular function after CO poisoning is possible but the extent and speed of recovery depends on the severity and the duration of exposure. In the acute setting hyperbaric oxygen (HBO) therapy is recommended. HBO prevents further injury to brain by rapidly eliminating CO, increasing plasma oxygenation, promoting mitochondrial recovery and preventing further brain inflammatory change from lipid peroxidation that can cause a delayed neurological sequelae (DNS) if the intracranial microcirculation is affected.

CO poisoning was relatively uncommon from the comprehensive UHN WSIB Neurotology Database being found in 0.11% or 5 of 4,219 workers assessed. Evidence for CO toxicity to the vestibular system however was unclear as LOC associated falls causing head injury were also found in those assessed.

g. What is the relationship if any between exposure to toxins, e.g., mercury or 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 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 should be affected 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.

1. Exposure to solvents

Volatile organic solvents found in paints/solvents/adhesives, organophosphates in pesticides and heavy metal exposure can all cause damage to the inner ears especially from chronic workplace exposures.

Many commonly used chemicals such as toluene, styrene, n-hexane, xylene etc. when used to dissolve or disperse substances are called solvents. They are used in a wide range of industries including engineering, construction, chemicals, printing, rubber, plastics, pharmaceuticals, footwear, textiles, foods, woodworking, dry cleaning, paint and ink manufacture. They can be found in paints, lacquers, varnishes, paint-removers, cleaning and degreasing materials, adhesives, inks and ink removers, pesticides, and toiletries. Somewhat alarming is the thought that some 10 million workers in the EU and perhaps a similar number in the USA could be exposed to solvents in the occupational workforce yearly.

In certain situations of exposure there may be a synergistic effect with occupational noise exposure and toluene dyes leading to a greater degree of hearing loss than anticipated.

2. Exposure to Heavy Metals

The shopping list of toxic heavy metals toxic to the inner ears is lengthy but lead (Pb) has been perhaps the best studied which is strongly linked to hearing and balance loss. Other ototoxicant metals include Cadmium (Cd), Mercury (Hg), Arsenic (As), Cobalt (Co) and Manganese (Mn).

h. Dizziness related to exposure to mould, multiple chemical exposures and fiberglass

Exposure to Moulds

Moulds and multiple chemical exposures can lead to dizziness from direct neurotoxicity and systemic inflammation that affects inner ear balance mechanisms. Dizziness is thought to arise from several biological pathways. Those with mould toxicity can also develop Multiple Chemical Sensitivity (MCS) where low-level exposure to everyday chemicals like perfumes or cleaning products triggers symptoms such as dizziness, headaches, and nausea.

Biological mechanisms for dizziness are postulated to arise from the adverse effects of mycotoxins (toxic substances produced by mould) directly bypass the blood-brain barrier which can cause,

- Changes in synapse function and even damaging neurons physically
- Disrupting the delicate balance between endolymphatic/perilymph fluids in the inner ear and hair cell function in both the cochlea and vestibular end organ

Diagnosing dizziness induced by mould or multiple chemical exposures is a multi-step clinical process as there is no single, universally accepted lab test that can definitively confirm “mould toxicity”. The diagnosis is primarily in the

realm of environmental physicians who use a combination of the medical history, environmental assessments and specialized biomarkers to come to this conclusion.

Treatment requires managing the immediate neurological symptoms and removing the underlying toxic burden from the affected individual's body and the environment (i.e. professional mould remediation, air filtration with high-efficiency particulate air (HEPA) filters and humidity management to prevent new mould growth).

Fiberglass exposure

Fiberglass exposure on its own doesn't appear to cause vestibulotoxicity. Exposure to styrene however used in the manufacturing process for fiberglass is likely the root cause and has been associated with vestibulotoxicity.

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Selected Appendices for Further Reading

Appendix 1: Additional Technical, Clinical and Diagnostic Information

1) 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 (i.e. 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.

Caloric Testing – Thermal stimulation of the ear canals stimulates the inner ear causing nystagmus as an evoked response. Comparison of the nystagmus (from hot and cold water stimulation typically at 44°C and 30°C) generated in one ear versus the other is extrapolated to the overall inner ear activity. The mnemonic "COWS" (cold opposite/warm same) is used to describe the expected direction of nystagmus from thermal stimulation.

Determination of the caloric difference (the difference between sides) is determined by the Jongkees Formula which takes what is called the maximum slow phase velocity (SPV) of the nystagmus after the each ear has been stimulated from warm and cold caloric irrigations.

Excitability Difference – $\dfrac{[(44R + 30R) - (44L + 30L)]}{44R + 30R + 44L + 30L} \times 100\%$

Normal values for nystagmus generated in our testing laboratory for warm responses are 8-60 °/sec caloric and 6-40 °/sec.

Excitability differences as judged as follows:

- Normal from 0-15%
- Mild peripheral vestibular loss from 16-25%
- Moderate peripheral vestibular loss from 26-50%
- Severe peripheral vestibular loss from 51-90% and
- Profound vestibular loss or no response (NR) from 91-100%

A bilateral vestibular loss is judged to be present when the additive values from warm and cold caloric responses are < 20 °/sec.

Caloric test responses however can be affected by inattention, the sedentary effects of medications, alcohol etc. if taken prior to testing. To counteract any sedentary effect the individual being tested is asked to do mental tasks requiring concentration such as subtracting serial 7s from 100, naming the provinces and provincial capitals of Canada etc.

Magnetic Scleral Search Coil Testing (MSSCT)[1] – Although not specifically reported in the medical discussion paper on *Dizziness*, MSSCT was the first physiologic means of assessing high velocity/acceleration VOR activity in primates and then in humans. It is a technically demanding procedure where high velocity VOR activity is recorded following placement of a contact lens embedded with a copper wire is placed on the corneal under topical anesthesia. When the subject is placed in a magnetic field movements of the eyes are recorded the contact lens. When the eyes move electrical pertubations in the magnetic field can be recorded in an advanced setting.

Overall MSSCT would be considered the gold standard for high velocity/acceleration head movements >200 degrees/s.

Subjective Visual Vertical Testing (SVV)[2] **–** When an acute vestibular loss (i.e. vestibular neuronitis) affects the otolithic organs a conjugate counter rolling of the eyes occurs. This provides in return a feeling as if the environment has tilted. The effect however is short lived as the compensation process seems to kick in relatively quickly. SVV is a diagnostic test that assesses the individual's perception of verticality and to determine if signs of an abnormal tilt that might cause dizziness/vertigo. It is primarily a test of the utricule (one of the two otolithic organs).

There are a few ways to perform the test. One involves sitting in darkened room where the affected individual sits and is asked to adjust a laser-controlled projection line or a Maddox illuminated vertical rod until it is perceived as being in the vertical plane. Sometimes a low tech variant called a **Bucket test** is used where the individual has a bucket placed on their head which is rotated until a line at the bottom of the bucket is rotated until it is perceived as vertical. Deviations from what the actual vertical to what the individual perceives as vertical can be used to determine if a utricular loss is present.

1 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.

2 Riera LR, Caballero-Garcia A, Martin-Mateos AJ et al. Efficacy of the Subjective Visual Vertical Test using a Mobile Application to Detect Vestibular Pathology. J Vest Res; 32(1): 21-27. Doi: 10.3233/Ves-201526

Normal variations in the subjective vs the real visual vertical are usually in the order of 0 ± 2 °. Values > 2.5 ° are suggestive for an otolithic impairment found on the involved side.

Suppression Head Impulse Paradigm (SHIMP) Test[3] **–** Conceptually a difficult test to understand. The purpose however is to determine if there is residual vestibular function in an individual with an apparent bilateral peripheral vestibular loss.

The test measures the VOR by asking the individual to fixate on a target that moves with their head. In a normal healthy individual, large corrective saccades (termed "catch up" or fast eye movements) are made because they are trying **not** to look at the target. The absence of a corrective saccade indicates a complete loss of vestibular function (i.e. there is no vestibular function to suppress).

2) Updated Diagnostic Criteria for Meniere's Disease[4]

Diagnostic criteria for Meniere's Disease (MD) were recently revised by the Classification Committee of the Barany Society in cooperation with several national and international organizations and were later approved by the AAO-HNSF Equilibrium Committee.

Definite MD

- Two or more spontaneous attacks of vertigo, each lasting 20 minutes to 12 hours
- Fluctuating aural symptoms (hearing loss, tinnitus, or fullness) in the affected ear.
- Audiometrically documented fluctuating low- to mid-frequency sensorineural hearing loss in the affected ear on at least 1 occasion before, during, or after 1 of the episodes of vertigo
- Other causes excluded by other tests

Probable MD:

- At least two episodes of vertigo or dizziness lasting 20 minutes to 24 hours
- Fluctuating aural symptoms (hearing loss, tinnitus, or fullness) in the affected ear
- Other causes excluded by other tests

3 Manzari L, De Angelis S, Princi AA, Galeoto G et al. The Clinical use of the Suppression Head Impulse Paradigm in Patients with Vestibulopathy: A Systemic Review. Healthcare (Bassel) 2022; 10(7):1182. Doi: 10.3390/healthcare10071182

4 Lopez-Escamez, JA, Carey J, Chung, WH, Goebel, JA et al. Diagnostic Criteria for Meniere's Disease. J Vest Res 2015; 25(1):1-7. DOI 10.3233/VES-150549

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 (i.e. betahistine), Ca^{2+} channel blockers etc. Intratympanic Therapies (IT) 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.

3) Definitions of a Mild Traumatic Brain Injury (TBI) used in this manuscript[5,6,7]

According to the 2016 US Veterans Administration/Department of Defense (VA/DoD) Clinical Practice Guideline for the Management of Concussion- Mild Traumatic Brain Injury a TBI is defined as a traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force and is indicated by new onset or worsening of at least one of the following clinical signs immediately following the event:

- Any period of loss or a decreased level of consciousness
- Any loss of memory for events immediately before or after the injury (posttraumatic amnesia)
- Any alteration in mental state at the time of the injury (i.e. confusion, disorientation, slowed thinking, alteration of consciousness/mental state)
- Neurological deficits (e.g. weakness, loss of balance, change in vision, praxis, paresis/plegia, sensory loss, aphasia) that may or may not be transient
- Intracranial lesion

Numerous guidelines have been written on the inclusion criteria for diagnosis of a mild traumatic brain injury (mTBI). Most concur that mTBI (while recognizing it as a complex pathophysiological process affecting the brain) clinically is associated with a Glasgow Coma Scale (GCS) of 13–15, resolution of posttraumatic amnesia within 24 h and a loss of consciousness for less than 30 minutes.

5 Veterans Administration (VA)/Department of Defense (DoD) Clinical practice guideline for the management of concussion-mild traumatic brain injury. Version 2.0. 2016.

6 Editorial. The changing landscape of traumatic brain injury research. Lancet Neurol 2012; 11(8):651

7 Marshall S, Bayley M, McCullagh S, Velikonja D, et al. Updated clinical practice guidelines for concussion/mild traumatic brain injury and persistent symptoms. Brain Inj. 2015;29:688–700.

Appendix 2

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

Appendix 3

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

Appendix 4

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