Ontario

Dizziness

Discussion paper prepared for

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This paper includes an Addendum on acute acoustic trauma and dizziness.

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

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 Discussion papers are not peer reviewed.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

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

DIZZINESS

INTRODUCTION

Sense of balance is basic to human function and is complex. The basic organ of balance, the vestibular labyrinth is part of the inner ear. It contains accelerometers within three semicircular canals and the organs of static balance, the utricle and saccule. The labyrinth signals the brain via the vestibular nerve whether the head is accelerating or decelerating and the position of the head in relationship to gravity. That portion of the brain dealing with balance receives and sends modifying signals from the eyes, the muscles and joints of the neck, the trunk, and the limbs. All the inputs are integrated so that a person knows the position of the whole body in space, and whether it is accelerating or decelerating. Visual input is important to balance, and where visual input and balance organ input are out of synchrony a variety of symptoms, including sea sickness, may occur.

THE ORGAN OF BALANCE

The inner ear, contained within the bony labyrinth, consists of a hearing organ, the cochlea; and a balance organ, consisting of three semicircular canals, a utricle and a saccule. The semicircular canals are at mutual right angles to each other, dilated at one end where they contain the sensory organ, the ampulla. Within the bony labyrinth lies a fluid (endolymph) filled membranous sac floating in a different fluid, the perilymph which fills the bony labyrinth. Within the membranous semicircular canal lies at the dilated end, an almost total obstruction, the ampulla - thin, flexible, and filled with nerve endings. As the head accelerates in any direction, the fluid in one or more semicircular canal momentarily lags behind the head movement. This bends the ampulla and stimulates the nerve endings embedded within it and sends an impulse to the brain. The semicircular canals detect only acceleration or deceleration. One certainly recognizes when an airplane is speeding up or slowing down, but there is no sensation of movement when it is at constant cruising speed (or standing still).

The utricle and saccule, mutually at right angles to each other - the utricle in the normal head position flat to the surface of the earth and the saccule at right angles to it - consist of supporting cells and hair cells from which nerves run to the brain. The hairs of the hair cells are embedded in a jelly-like material in which sit very small crystals of calcium salt like grains of sand called otoliths. Depending on the position of the head, the otoliths bend the hairs of the hair cells. If the hairs are bent in one direction, the nervous impulses increase; if they are bent in another direction, they decrease. Thus, the utricle has otoliths pressing directly down on the hair in a neutral position, with the head upright. If the head is tilted forward or backwards, gravity pulls the otoliths forward changing their position, which deforms the hairs and triggers diminished or increased nerve impulses appropriately. The utricle is more active than the saccule, but the saccule works in a similar way.

There is a constant resting input to the brain from the nerve endings of the organ of balance from both the otolith organs and semicircular canals, because change of head position in one way increases the number of nerve impulses coming from the organ, and positions in another way diminish it. Thus, the organ is always active.

The input from the semicircular canals, utricle and saccule, pass by the vestibular nerve to the vestibular nerve to the vestibular nuclei of the brainstem where they are combined with impulses from the organ of vision, from the muscles that move the eye, or oculomotor system, with the proprioceptive fibres from the neck, trunk, and limbs, which rely on stretch receptors and position receptors in the muscles and joints, which indicate the status of the particular joint or muscle and thus position of the joint and body part. The hind brain integrates all these signals so that the body knows its total position in space and the position of its parts at all times.

The vestibular systems can be likened to the paddle wheels of a paddle steamer (after Reading), and the brain to the helmsman at the steering wheel. When both labyrinths are working well, the paddles are turning at the same speed, the rudder is kept in the midline, and the boat progresses on a straight path. If one organ is knocked out i.e. one paddle wheel stops turning, the other one drives the boat towards the side of the stopped paddle wheel. The helmsman sensing this, corrects the rudder of the boat and proceeds back on a straight path with the driving force from one wheel corrected by a new setting of the rudder. Thus destruction or damage to one labyrinth produces diminished output from the semicircular canals, veering or falling towards the side of the lesion, which the brain learns to compensate for by visual and limb input and over a period of weeks resets its central mechanisms (rudder) so that the person once again sees themself to be in a normal position.

If both labyrinths are destroyed, then complete reliance is placed upon the joint and muscle input and particularly visual input - the person sees where the head is and senses balance accordingly. In this instance, the sense of balance is virtually totally lost in darkness, particularly if walking on uneven ground - there is no mechanism of knowing where the head is and, if the ground is uneven, no mechanism for determining the position of the feet and limbs are in relationship to the center of the earth and the trunk.

In turn, the delicate focusing and fixation of the eyes, particularly when moving is dependent on an intact vestibular labyrinth which signals rapidly and continuously small changes in head position, allowing the eyes to remain focused on a target even when the head is moving. Thus, when walking and the head is bobbing up and down, or even more running, it is perfectly possible for someone with intact labyrinths to keep the eyes focused on the horizon or a street sign sufficiently well to read it. If the labyrinths are both lost, this continuing adjustment is usually impossible and everything appears to bob up and down - when driving a car road signs cannot be read, when walking targets cannot be properly fixed (bobbing oscillopsia).

Disturbances of the Sense of Balance

With such a complex system, there are many ways in which the sense of balance can be upset. The peripheral labyrinth, both the gravity receptors and the accelerometers, can fail to work properly and each produces a separate set of symptoms. There can be failure of integration of eye and balance organ producing yet other symptoms; there can be neck muscle and joint problems producing other symptoms again; and more again from limb and trunk nervous diseases.

Symptoms at Balance Disturbance. These consist of vertigo (a sense of spinning in which either the person feels they are spinning in the world or the world is spinning around them), which is frequently but not always in the horizontal plane; staggering and unsteadiness. Staggering and unsteadiness may be modified by visual input. Vertigo may be constant, or of various durations from seconds to days. It usually lasts for a matter of seconds, particularly with changes of position - turning over in bed, moving from lying to sitting or sitting to lying.

The close relationship between sight and balance means that eyes adjust constantly to changes in position. If there are aberrant signals from the balance organ, the eyes make adjustments even when there is no change in head position that necessitate them. These can be seen by an observer and recorded by equipment. These rapid movements of the eye are known as nystagmus.

Test of the Organ of Balance

Most tests of balance rely on detection at nystagmus evoked either by changing the body's position, known as positional nystagmus, or the presence of nystagmus without any evocation, known as spontaneous nystagmus. Nystagmus can also be evoked by the caloric test, used to determine the function of the semicircular canals. In this test, the head is positioned so that the outermost or lateral semicircular canal is in a vertical position. Cold or hot water is irrigated into the ear canal. This warms or cools the air in the middle ear, which in turn warms or cools the fluid within the semicircular canal, setting it in motion by calorie current, upward when warm and downward when cold, bending the ampulla forwards or backwards, increasing or diminishing the static nervous impu1se from it. This makes the makes the head feel it is accelerating and in turn sets off nystagmus, known as caloric nystagmus. This can be seen visually or more usually is detected by electrodes placed around the eye, which senses eyeball movement and is known as *nystagmography*.

Other tests for balance include the sway test in which the person's ability to stand up straight with eyes closed and eyes opened is observed, and it may be quantified electronically by standing on a platform which detects movement. To my knowledge, this equipment is not in general use in Toronto.

A final way in which the sense of balance is tested is by direct acceleration or deceleration in a rotating or oscillating chair with electronystagmographic recordings of the eye movement. This detects nystagmus induced by movement but does not distinguish the balance organ of one side from the other. These tests are sometimes done, but the meaning is not clear.

Disorders of Balance

The sense of balance can be upset by trauma, disease, or degeneration. With such a complex sense, it may be the result of problems with the labyrinth of the inner ear, the nerve of balance, the brain which integrates the sensory input, the eyes, and neck joints, or proprioceptive input. Thus, someone beginning to wear bifocal spectacles often feels a sense of imbalance until the brain adjusts to the new visual input. Eye disease may occasionally produce similar symptoms. Whiplash injury in which the neck or cervical spine joint receptors may be damaged is often cited as a cause of imbalance, although there is controversy about this. Head injury may produce imbalance, either by concussion to the part of the brain which integrates the sense of balance or by direct damage to the labyrinth, either fracture destroying a labyrinth, thus completely eliminating input from one inner ear (the paddle whee1 phenomenon), or by loosening the otoliths and producing positional vertigo. A concussive lesion is more likely to lead to unsteadiness. A fractured temporal bone with destruction of a labyrinth will produce vertigo that usually lasts for a period of some weeks slowing subsiding as it is compensated; and post-traumatic positional vertigo lasting for seconds at a time which with particular changes of head position may last much longer.

Disease of the central nervous system such as multiple sclerosis affecting the vestibular nuclei may produce distressing and persistent vertigo; arteriosclerosis in the elderly may produce vertigo; lack of blood supply to the brain may produce temporary vertigo as experienced quite commonly by people who having sat for a long time suddenly stand up and feel unsteady. This results from deprivation of the balance brain of oxygen. Similar symptoms occur commonly with people on antihypertension treatment.

Disorders of the inner ear or labyrinths are the most common causes of vertigo. They run the full gamut of standard classification of disease; congenital and acquired, and in the latter category traumatic, infective, metabolic, degenerative, and neoplastic.

Traumatic causes include head injury (dealt with above) producing a fracture of the temporal bone (dealt with above), barotrauma from pressure changes caused by flying or diving. The term *diving* includes any work under increased atmospheric pressure, including tunneling, and thus includes certain construction workers as well as professional and amateur divers. Commercial flying rarely causes pressure changes severe enough to produce vestibular disturbances but military flying may do so. Diving is a much more potent cause of traumatic pressure changes than flying and may cause damage in two ways: direct rupture of delicate membranes from excessive movement or by decompression injury (the *bends*). Diving may produce actual rupture of the membrane closing the inner ear from the middle ear - a round window fistula, or produce trauma to the middle ear with dislocation of the stapes - the innermost of the ossicles into the inner ear. This latter is rare unless the stapes has previously been injured (or replaced surgically by prosthesis). Even more rarely, something as vigorous as a hard nose blow may produce a ruptured membrane. The bends (Caisson disease) occurs wherever there is diving or tunneling. When air under pressure is breathed, some Nitrogen is absorbed into the blood stream. When pressure returns to normal, it is released. Usually it is released into the lungs and harmlessly exhaled. If decompression is too swift, it is released *within* the blood stream forming tiny gas bubbles, which interfere with tissue oxygenation. This is particularly a problem in long bones, the brain and inner ear. In the latter, it may produce deafness, vertigo, or both.

Surgical trauma is also a possible cause of vertigo and will be dealt with separately. Noise is also described by some authorities as a possible cause.

Surgical trauma is a potent cause dizziness which is a common and unpleasant sequela of surgery to the middle and inner ear, usually fairly quickly self-limiting but sometimes persistent.

Infections of the inner ear are called labyrinthitis. This may be bacterial or viral, and in the case of a viral labyrinthitis may affect only the balance system and not hearing. Fortunately, acute bacterial labyrinthitis is very rare, producing as it does extremely severe vertigo and hearing loss. Viral labyrinthitis is more common and may be isolated or be part of a more generalized viral infection. It is difficult to distinguish the viral labyrinthitis affecting the vestibular nerve from that affecting the labyrinth itself. It may be an accompaniment of viral infections of the VIIIth and other nerves, such as shingles, some forms of influenza or viral meningitis. An unexplained vertigo is often classified as "viral labyrinthitis," for want of a better descriptor.

Metabolic diseases include Meniere's Syndrome, better described as Labyrinthine Hydrops. In this condition, the fluid balance of the endolymph inside the membranous labyrinth is disturbed. It collects larger quantities than usual, distending the membranous inner ear, frequently producing bearing loss and acute vertigo, which is characterized by spells of nauseating spins and initially intermittent hearing loss and tinnitus. The distention may be sufficiently severe to rupture the membranous labyrinth within the cochlea producing mixing of the endolymph and the perilymph in which the labyrinth floats. This mixing of fluids of different chemical composition poisons the cells of the adjacent inner car producing an acute heating loss, which initially may recover but ultimately becomes permanent. The full blown disorder is characterized by episodic vertigo, hearing loss, tinnitus, nausea, a feeling of fullness of the ear, is common and debilitating. Frequently, it is less severe and not all symptoms are present. There are many causes of Labyrinthine Hydrops. The current belief is that there is an immunological reaction precipitating it in most cases, which may be idiopathic or secondary to bacterial or viral infections (sometimes years before). Where no cause is found, it is known as Meniere's disease. Similar symptoms may occur with bleeds into the inner ear, trauma, etc. ... There are other metabolic causes of dizziness, including some thyroid disorders, nutritional allergies, etc...

Degenerative causes are associated with aging and are complex because aging affects not only the tissues of the labyrinth directly, but also its blood supply; likewise cerebral degeneration with aging may affect the cerebral vestibular nuclei and produce dizziness. Sufficient to say that loss of balance in the elderly is extremely common and may be indirectly lethal because of its propensity to produce falls which break the hips. It is one of the major problems of aging.

Neoplastic disease (tumors) affecting the inner ear or nerve of hearing are a rare cause of dizziness, because the brain adapts to slow steady changes as may be produced by a tumor slowly growing and pressing on the vestibular nerve affecting parts of the inner ear. They may produce acute vertigo if the blood supply to the inner ear is suddenly impaired or if a hemorrhage occurs in the inner ear as may happen with acute leukemia.

The above description applies mainly to disorders of the semicircular canals.

Positional vertigo characterized by acute imbalance produced by change of head position, often something as simple as rolling in bed or lying down or sitting up, is extremely common and usually lasts for a few seconds or a minute or two; and is caused by derangement of the otolith organs. It may be paroxysmal, coming in attacks repeatedly over a period of months or it may occur as just one bout. Aging is an extremely common cause. If some of the otoliths loosen disturbing sense of rotation every time the patient turns because of an aberrant movement, it may result in head injury. Persistent positional vertigo is rare and may indicate neurological disease.

Disorders of the nerve of balance may produce dizziness. The most common is vestibular neuronitis, a viral infection of the vestibular nerve, which produces acute vertigo, which gradually improves over a period of two to three weeks, unassociated with hearing loss. A sense of imbalance without true vertigo may occur as a symptom of tumors of the VIIIth nerve, the most common of which is acoustic neuroma produced by the increasing pressure on the nerve.

There are many central nervous system diseases which produce vertigo, but they are outside the scope of this review, with a single exception. It should be noted that there is an increasing body of literature related to the effects on the central nervous system affects of toxic chemical inhalation (including paint and some industrial organic chemicals), which may present as vertigo or unsteadiness.

Conclusion

Positional vertigo is by far the most common cause of labyrinthine dizziness. It has many causes, the most common of which is aging in which some of the otoliths loosen and lead to a disturbing sense of rotation every time the patient turns over, particularly in one direction. Disturbance of the semicircular canals is also frequent. The three major common causes are: (1) inflammation of the nerve of balance known as vestibular neuronitis, which produces a vertigo which persists and is of sudden onset which diminishes over a period of days or two to three weeks, (2) labyrinthitis (usually viral in which there is an infection of the inner ear usually accompanied by other signs of infection, and (3) Meniere's Syndrome.

Dizziness as a symptom is extremely common - it was the commonest single symptom in a large series of consecutive admissions to the medica1 services of Massachusetts General Hospital. However, it is a very vague and non-specific general descriptor, which is widely used to cover a variety of sensations ranging from a generalized feeling of unwellness to the much less frequent genuine vertigo. Nonetheless, vertigo itself is common, and as shown above, has many causes, only a few of which are related to occupation.

Vertigo Associated With Acute Acoustic Trauma A Medical Controversy

There has long been a suggestion in the literature that exposure to extremely loud sound may produce a transient imbalance, although there is little quantification or justification of this statement. There are also recurrent allusions to chronic balance disturbance resulting from exposure to sound, the evidence for which is also extremely soft.

My colleague Joseph Touma in Huntington, West Virginia, provides an annual update of controversies in noise-induced hearing loss, and lists as one of the controversies "Vestibules and Noise-Induced Hearing Loss". Here he lists seven papers in which these matters are referenced.[1]

Some people may become acutely dizzy when exposed to loud sound. This is known as the Tullio[2] phenomenon, after the man who first brought it to wide public attention, albeit originally in pigeons where he noted extremely loud sound exposure to one ear would produce sway nystagmus. It also occurs in human beings where the standard wisdom is that they must have disease or damage of the middle ear for it to happen. It was common in subjects who had the fenestration operation for otosclerosis, particularly when the stapes was also inadvertently loosened, but it is a medical rarity which in the past I have attempted to pin down in the literature without any great success.

It is generally accepted that an extremely loud sound such as an explosive blast at intensity levels well above those found with industrial noise may produce a transient sense of imbalance as well as temporary or permanent hearing loss. This is frequently described by people who are exposed to a shell blast close by (and survive), or in industry when accidentally too close to a blast in the mines or a quarry. It is generally accepted that "normal" industrial noise (i.e. sounds) below 115 dB do not produce vertigo. The total controversy exists about whether intense sound produces vertigo, and at what sound threshold. The vertigo is usually but not always transient.

There are also repeated reports in the experimental literature of vestibular damage from high levels of sound, usually in guinea pigs. Great care must be exercised in extrapolating this to humans.

Kryter[3] quotes Dixon and Chadwick[4] that a jet aircraft noise must be over 140 dB for a person to feel some disturbance of equilibrium. He quotes Harris and Von Gierke[5] who exposed subjects wearing ear muffs to noise levels of 120, 130, and 140 dB while attempting to balance on a narrow rail, both with eyes open then with eyes closed. Only at 140 dB was any balance impairment found. They did note, however, that if the sound level of the two ears were different, a lower threshold of imbalance was found. Harris has confirmed this observation, but Vanderhel and Loch failed to replicate it.

Pyyko, et al[6] studied the postural ability in 54 Finnish soldiers, who had acute temporary threshold shift from rifle shooting and compared the results to non-noise exposed soldiers and civilians. They found no affect either in body sway nor any acute imbalance. They conclude that impact noise probably does not exert any significant functional effect on the vestibular system in man. They quote two studies by Era[7,8] who substantiates their findings. They do, however, note that Ylikoski[9] found lesions in the vestibular saccule, utricule, and semicircular canals of guinea pigs subjected to small arms fire. In other studies, Henderson and his colleagues have made similar observations.

Man and his colleagues[10] in a study published in 1980 describe the results of vestibular studies performed on 336 patients suffering from acoustic trauma. They found 230 of the patients complained of vertigo. Vertigo is described as "a sensation of spinning that appeared for a few seconds and disappeared again. Sometimes there is momentary nausea but without vomiting. In some patients, vertigo appeared only during noise exposure."

They performed electronystagmographic examinations with and without noise stimulation and believe they found quite clear vestibular changes in a certain proportion of patients, especially if tested in noise. They also found some association between the subjective complaint of vertigo and objective signs of vestibular change. They quote an older paper by Collins in support of their findings, although this paper is misreferenced and I have not yet been able to track it down. Their paper is remarkably vague. It gives details of the studies of the subjects, but almost no information about the nature and type of noise exposure and hearing loss. It does not even give any indication of what proportion of subjects have noise-induced hearing loss.

In my own evaluation *of* subjects with noise-induced hearing loss referred by the Compensation Board of Ontario, where I have now personally asked the question to many hundreds of claimants, the response to the question "Do you have any dizziness?" is usually "no"; and if "yes" is commonly found in people under treatment for hypertension or describing postural vertigo, the common side effect of the therapy for this disorder.

The best total review of the subject I found it, by Oosterveld, et al[11] , who performed intensive vestibular examinations in KLM aircraft maintenance workers exposed to noise and also reviewed the literature in exhaustive detail and credited Urbantschitsch in 1901 with demonstrating nystagmus in humans induced by auditory stimuli, which is now known as the Tullio phenomenon. They make the point that Tullio also described that the sound had to be isolated to one ear alone. They cite jet engine noise as producing vestibular disturbance as well as loud music as causing vertigo and nystagmus and nausea (interestingly, those papers are from the pre-amplified rock music era). They describe balance disorders in shipyard workers where the noise type is similar to the claim in question. They also commented that loud noise exposure may lower the sensitivity of the ear to cause dizziness.

After an extremely detailed and good vestibular study, they concluded "the question remains unanswered whether or not the cochlea and labyrinth undergo damage simultaneously or because of a difference in vulnerability, one precedes the other. The medicolegal aspects of vestibular involvement in noise-induced hearing loss can be some importance. Hearing loss does not usually directly affect work capability, but a vestibular disorder may well do so. In consequence, noise-exposed individuals could be disabled because of vertigo or balance disorder. This is an important but a neglected aspect of noise-induced hearing loss." Nonetheless, it is very difficult to tell from the study whether any of the subjects had a direct balance disturbance resulting from noise exposure.

A brief review of the literature certainty suggests that there may be vestibular disturbance resulting from noise exposure. The usual description is of a momentary imbalance lasting for seconds or a minute or two. The chance of producing vertigo is greater if one ear is exposed to more sounds than the other. The sound has to be extremely intense - preferably 140 dB or above to have a major affect. In small laboratory animals, histological changes have been shown in the vestibule after exposure to intense sound by several investigators in different laboratories. Histological evidence in man is scant - perhaps because it has not been sought.

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