

Ministry of Defence

Synopsis of Causation

Vertigo

Author: Dr John Irwin, Ninewells Hospital and Medical School, Dundee
Validator: Professor Linda Luxon, UCL Institute of Child Health and Great Ormond Street
Hospital for Children, London

September 2008

Disclaimer

This synopsis has been completed by medical practitioners. It is based on a literature search at the standard of a textbook of medicine and generalist review articles. It is not intended to be a meta-analysis of the literature on the condition specified.

Every effort has been taken to ensure that the information contained in the synopsis is accurate and consistent with current knowledge and practice and to do this the synopsis has been subject to an external validation process by consultants in a relevant specialty nominated by the Royal Society of Medicine.

The Ministry of Defence accepts full responsibility for the contents of this synopsis, and for any claims for loss, damage or injury arising from the use of this synopsis by the Ministry of Defence.

1. Definition

- 1.1. Vertigo is defined as an illusion of movement or disorientation in space. The movement is typically, but not invariably, a rotation in the horizontal plane. This can be either a sensation of the world moving around a stable individual or the individual revolving within a stable world.
- 1.2. Other illusions of movement include rotation in the vertical plane, swaying, a sensation as if on a boat at sea, and the feeling that whatever is beneath the subject is sloping or moving in other ways.
- 1.3. Vertigo is one form only of both imbalance and dizziness. Rather than being a disease process in itself, vertigo is a common symptom of many varied conditions affecting the vestibular structures of the inner ear, the vestibular nerve, the vestibular nuclei in the brain stem, the cerebellum, the temporal cortex, and the blood and metabolic supply to these structures.
- 1.4. Dizziness is a non-specific lay term used by people to describe a variety of conditions ranging from light-headedness and unsteadiness to vertigo. Dizziness can be described as painless head discomfort with many possible causes including disturbances of vision, the brain, balance system of the inner ear, the cardiovascular system and the gastrointestinal system. This synopsis deals with the specific topic “vertigo”.

2. Clinical Features

- 2.1. These depend upon the underlying cause of the symptom.
- 2.2. In order to differentiate between causations, it is important to ascertain the following:
 - 2.2.1. Was the symptom triggered by an event? Post-traumatic or post-infective vertigo are examples of this.
 - 2.2.2. What is the time course of the symptom? This can range from sudden, very brief episodes associated with transient interruptions in blood supply caused by increased platelet aggregation; through episodes lasting a few hours in migraine and Ménière's disease; to disabling vertigo, lasting many days or weeks, in sudden peripheral vestibular failure from any cause.
 - 2.2.3. Are there any other symptoms associated with the vertigo? Examples include: palpitations in panic disorders and arrhythmias; visual disturbance in migraine and posterior fossa disorders; changes in hearing and tinnitus in inner ear and 8th nerve conditions; and discharge from the ear in chronic middle ear disease.
 - 2.2.4. Is there anything that precipitates the symptom, for example looking upwards or turning in bed as occurs in paroxysmal positional vertigo, or standing up from a chair as in postural vertigo?
 - 2.2.5. Is there anything that alleviates the symptom? For example, is the symptom better lying very still on one particular side in an acute vertiginous attack?
 - 2.2.6. Are there any abnormalities on examination of the ears, central nervous system, blood pressure (lying and standing), heart, or balance?
 - 2.2.7. What medications is the subject taking? Dizziness is a very common side effect of medication and vertigo can also occur.
- 2.3. Whatever the cause, vertigo is a very common symptom.
 - 2.3.1. In the UK, 5 out of every 1,000 people consult their general practitioner each year because of vertigo.¹
 - 2.3.2. The prevalence of vertigo rises with increasing age and dizziness is the most common cause of presentation to the primary physician in people over 74 years old.² Not all of these patients will have vertigo but there are no good data with which to estimate the numbers (this statement also holds for 2.3.3 and 2.3.4 below).
 - 2.3.3. Self-report of *current* dizziness in the community is 17%.³
 - 2.3.4. Over 25% of 50-64 year olds state that they currently suffer dizziness.⁴
 - 2.3.5. By the age of 80, two-thirds of women and one-third of men will have experienced episodes of vertigo.⁵

- 2.3.6. The prevalence of conditions causing vertigo varies depending upon the population studied. Underlying conditions include cardiovascular, otological and neurological disorders.

3. Aetiology

- 3.1. Vertigo can be a symptom of various conditions affecting many different body systems. In these conditions, vertigo is usually only one part of the overall symptom complex.
- 3.2. **Cardiovascular system**
 - 3.2.1. **Cerebrovascular and coronary artery disease.** Raised cholesterol, smoking, high blood pressure, and family history are known vascular risk factors. Cerebrovascular disease can affect blood flow to the brain and cause vertigo. Coronary artery disease can lead to reduced cardiac output and heart failure. This can also lead to reduced blood flow to the brain and vestibular structures.
 - 3.2.2. **Postural hypotension** also reduces cerebral blood flow. This can be a side effect of medication, a result of carotid sinus disease, or of unknown cause. It also occurs in Parkinson's disease, peripheral and autonomic neuropathies, and spinal cord pathologies. The diagnosis is made on the history and on measuring the lying and standing blood pressure.
 - 3.2.3. **Mechanical dysfunctions**, such as aortic stenosis, heart failure, angina and myocardial infarction can all cause vertigo, as well as other forms of dizziness and imbalance.
 - 3.2.4. **Cardiac dysrhythmias**, from whatever cause, can also reduce cardiac output. A history of palpitations accompanying or preceding the vertigo is often, but not always, present. Sometimes it is possible to confirm this diagnosis by 24-hour ECG recordings.
 - 3.2.5. **The subclavian steal syndrome** occurs when there is a blockage of one of the subclavian arteries. Use of the affected limb means that there has to be an increase in blood flow to the muscles. As the subclavian artery is blocked, blood flow in the vertebral artery is reversed siphoning blood from the vertebrobasilar system into the distal subclavian artery.
- 3.3. **Haematological and metabolic abnormalities**
 - 3.3.1. **Hyperviscosity syndromes, anaemia and polycythaemia** may all be associated with vertigo.
 - 3.3.2. **Hypoglycaemia** is usually a result of insulin overmedication but, rarely, is secondary to Addison's disease, hypopituitarism, or insulinoma. Vertigo can be one of the features of these conditions.
- 3.4. **Anxiety and vertigo**
 - 3.4.1. Anxiety and vertigo are linked in many ways.⁶
 - 3.4.2. Vertigo is one of the features of acute anxiety and panic attacks. In panic attacks, symptoms may be precipitated during examination by hyperventilation and eased by re-breathing.

- 3.4.3. The experience of the unpleasant sensation that is vertigo can also lead to understandable feelings of panic attacks, avoidance behaviour, anxiety and depression. This may be explained by documented connections between the autonomic nervous system, the vestibular system, and cognitive centres in the brain.
- 3.4.4. Following acute vestibular failure, anxiety can prolong the normal central compensation process (see section 3.6.2 (d) and (e)).
- 3.4.5. Patients with persistent or recurrent dizziness/vertigo may report an exacerbation of their symptoms when they are physically or emotionally stressed.

3.5. **Neurological conditions**

- 3.5.1. **Stroke.** As well as the temporary problems discussed in section 3.2.1, completed strokes in the vertebrobasilar vascular system include the condition known as lateral medullary syndrome or Wallenberg's syndrome. This is due to occlusion of the posterior inferior cerebellar artery. Features include vertigo, sensory loss on the face and ataxia on the side of the stroke with loss of pain and temperature sensation on the other side of the body.
- 3.5.2. **Epilepsy.** Vertigo can occur as part of the aura preceding temporal lobe epilepsy, or as a symptom of generalised epilepsy.
- 3.5.3. **Cerebellar pathology.** The cerebellum is involved in coordination and control of movement and balance. Most cerebellar syndromes do not present with vertigo, but symptoms of unsteadiness are common. Acute cerebellar haemorrhage and infarction are associated with acute vertigo.
- 3.5.4. **Multiple sclerosis.** Vertigo usually features at some time during the course of multiple sclerosis,⁷ and is the presenting feature in up to 5% of cases.⁸
- 3.5.5. **Brainstem tumours.** Dizziness and/or vertigo can be early or presenting features in 25% of brainstem tumours.⁹
- 3.5.6. The diagnosis of the above conditions is made by careful history and examination together with appropriate specialised investigations including magnetic resonance imaging.
- 3.5.7. **Migraine**
 - a) Definite migrainous vertigo in an individual is defined as episodic vestibular symptoms of at least moderate severity (i.e. sufficient to interfere with, but not to impede daily activities). In addition, the presence is required of at least one of the following symptoms during at least 2 attacks: migrainous headache (according to the International Headache Society (IHS) criteria¹⁰), photophobia, phonophobia, visual or other aura. Other causes should be ruled out.

- b) Probable migrainous vertigo is defined as episodic vestibular symptoms of at least moderate severity (i.e. sufficient to interfere with, but not to impede daily activities). In addition, the presence is required of at least one of the following: migrainous symptoms during vertigo, migraine-specific precipitants of vertigo, response to anti-migrainous drugs. Other causes should also be ruled out.
- c) Symptoms of vertigo in people with migraine are much more common than previously thought¹¹ (see 3.5.7 (d) to (g) below).
- d) Migraine affects as many as 15-20% of the general population, and it has been estimated that about 25% of patients with migraine experience spontaneous attacks of vertigo and ataxia.
- e) Phonophobia is the most common auditory symptom, but fluctuating hearing loss and acute permanent hearing loss occur in a small percentage.
- f) So-called "vestibular Ménière's disease" (i.e. symptoms of Ménière's disease without changes in hearing or tinnitus) is now more commonly considered to be migrainous in origin.
- g) Motion sensitivity with bouts of motion sickness occurs in about two-thirds of patients with migraine.
- h) There is an association between Ménière's disease and migraine. Ménière himself suggested this association in 1861¹² and the lifetime prevalence of migraine in patients with Ménière's disease is 56%.¹³

3.6. **Otological conditions**

3.6.1. These can involve the inner ear, the 8th nerve, or the central nervous system. These conditions are too numerous to list in full here. Some of the more common syndromes are described below.

3.6.2. **Acute unilateral vestibular failure**

- a) There is usually sudden and dramatic onset of vertigo, often with nausea and vomiting. Any movement exacerbates the symptoms.
- b) Common aetiologies are post-infective (where the condition usually starts 7-10 days into the systemic illness) vascular supply interruptions, and trauma.
- c) The average time course of events is that the sufferer has to spend 2-3 days in bed. It is 2-3 weeks before walking is easy and it may be 2-3 months before a return to work is possible.
- d) Patients with this condition recover by means of a number of mechanisms associated with cerebral plasticity collectively known as central compensation. Impairment of the sensory inputs required for balance (vision, proprioception and vestibular input), psychological disorders, neurological disorders, and systemic disease are the most common reasons for failure of compensation.

- e) Patients may experience minor or major relapses over the course of the next 2-3 years. These are sometimes precipitated by tiredness, intercurrent infection or stress, although they may occur without any obvious precipitating event.

3.6.3. **Recurrent vertigo**

- a) The terminology in this area can be confusing. Labyrinthitis should be reserved for conditions affecting the whole of the labyrinth. In this case, symptoms would include hearing loss and tinnitus. If the symptom is vertigo alone, then vestibular neuritis is generally used, even though it may not be the vestibular nerve that is involved.
- b) Although most people with acute vestibular failure of any cause (see section 3.6.2) have one episode and recover, some people have recurrent episodes. These are very similar to the initial acute episode but frequently reduce gradually in intensity and duration. They may or may not have triggers, which vary between cases, and usually cease eventually. This pattern suggests recurrent decompensation.

3.6.4. **Benign paroxysmal positional vertigo (BPPV)**

- a) This is a clinical presentation where patients experience frequent but brief episodes of vertigo, usually of less than three minutes duration. Each episode is triggered by a specific movement. There is a good discussion of this topic in a recent volume of *Audiological Medicine*.¹⁴
- b) BPPV is the most common cause of vertigo on neck extension, such as looking upwards, and has often been misdiagnosed in the past as vertebrobasilar insufficiency. However, in BPPV there are no other symptoms of interruption to the posterior circulation blood supply.
- c) Vertigo also occurs on first lying down in bed at night, first getting out of bed in the morning, bending forward and turning in bed, usually to one side only.
- d) BPPV is caused by an accumulation of otoliths within a semicircular canal, and the pathophysiological mechanism of generation of symptoms is considered to be canalolithiasis.^{15,16} This otoconial debris is free to move within the lumen of the canal, an effect that occurs during the postural movements described above. The movement of otoliths causes fluid movement and is perceived as vertigo.
- e) Fifty percent of cases are of unknown aetiology but known causes include infection, vascular problems, and trauma.

3.6.5. **Ménière's disease**

- a) Despite this being an uncommon cause of vertigo, it is included here as it is well known.

- b) This is another condition where patients experience bouts of recurrent vertigo. In this case, they are intermediate in duration between BPPV and acute vestibular failure, usually lasting for less than 24 hours.
- c) By definition this is a condition of unknown aetiology. The same symptom complex can occasionally occur following acute vestibular failure or in congenital hearing loss when the condition is called delayed or secondary Ménière's syndrome (rather than Ménière's disease).
- d) Episodes are preceded by a prodrome of a feeling of fullness in the affected ear, a drop in hearing, and either onset or change in tinnitus. This usually lasts for 10-15 minutes but can be shorter and occasionally may precede vertigo by several days.
- e) The vertigo typically lasts for 2-4 hours. This is variable but symptoms should not exceed 24 hours.
- f) Patients often feel "washed out" for 1-2 days following episodes and headache is common at this time.
- g) Individual episodes can occur without warning but some people can identify specific triggers. These include stress, infection, and food triggers.
- h) There is no evidence-based management strategy in Ménière's disease but simple measures may include weight loss where necessary, stress management, a salt free diet, and caffeine exclusion. Other measures including diuretics should only be considered after this.
- i) There is an association between Ménière's disease and migraine (see 3.5.7 (h) above).

3.6.6. **Bilateral vestibular failure.** The symptoms of symmetrical bilateral vestibular failure do not include vertigo, but patients suffer severe instability, particularly in the dark and on uneven/unstable ground, and "bobbing oscillopsia" (movement of the visual world) on walking.

3.7. Trauma

- 3.7.1. There are several different types of trauma that can affect the balance mechanisms. These involve both acute injuries, such as blast injury, direct trauma and barotrauma, and chronic and/or repetitive damage, such as microvascular damage in professional divers.
- 3.7.2. The injuries can be directly to the ear, for example, in skull fracture, or at distant sites such as the neck and cervical spine.
- 3.7.3. Vertigo can be caused by the trauma itself, or may be part of post-concussional syndromes, or part of post-traumatic stress disorder.
- 3.7.4. **Skull fracture.** The vestibular apparatus sits within the temporal bone of the skull. Fractures of this bone can directly damage this structure

and lead to acute vestibular failure (see section 3.6.2). In one series of over 100 patients with gunshot wounds to the head, 25 had temporal bone injuries, and 3 (25%) of the survivors with temporal bone injuries had vertigo.¹⁷

3.7.5. **Noise induced hearing loss and Ménière's disease**

- a) There have been suggestions that late complications of chronic noise trauma can include secondary Ménière's syndrome. These are mainly case reports,¹⁸⁻²⁰ for example, one study gives details of 18 cases in senior army officers.²¹
- b) More recently, this hypothesis has been questioned. A large review of the published literature from 1872 to 1958 concluded that there was no convincing evidence to support this.²² A large study of 17,245 Israeli defence force veterans also found no evidence to support this.²³

3.7.6. **Barotrauma**

- a) This can occur in divers and fliers and is more likely in people with Eustachian tube dysfunction.
- b) At altitude, aircraft cabin pressure is less than atmospheric. On ascent the pressure change is quite gradual but the return to atmospheric is more rapid on descent.
- c) Divers experience rapid pressure change on ascent where the middle ear pressure is higher than atmospheric.
- d) In either case, trauma to the inner ear can result in acute vertigo. Very rarely this can be in the form of the Tullio phenomenon, when patients suffer vertigo when exposed to loud noises.²⁴

3.7.7. **Blast injury**

- a) As well as the loud noise, explosions consist of a compression pressure wave followed by a rarefaction wave, often of greater intensity. This reduction in pressure can cause perforation by pulling the ear drum outwards. This can also cause rupture of the round window membrane and in extreme cases can pull the stapes out of the oval window.
- b) Vertigo is a common symptom in survivors of blast injury. In one study, 60% of 147 soldiers who had been involved in explosions had vertigo,²⁵ and in another study, 7 of 13 survivors of an explosion on a bus also had vertigo.²⁶ Blast injury is another cause of the extremely rare Tullio phenomenon.

3.7.8. **Other causes of vertigo in divers and aircrew**

- a) Acute decompression sickness may be associated with vertigo. This may be due to labyrinthine and/or central problems.

- b) Problems with breathed gasses, such as nitrogen narcosis, oxygen toxicity, hypercarbia and hypoxia can also cause vertigo through central effects.²⁷
- c) People who dive professionally often have evidence of central nervous system damage.
- d) One of the features of chronic vestibular disturbance is spatial disorientation.²⁸ This is a cause of potential and actual serious aircraft mishaps and is a cause of permanent grounding of aircrew.
- e) Acute problems in the posterior circulation have been reported during flight resulting in vertigo. These need to be managed before the aircrew flies again.²⁹

3.7.9. **Neck trauma**

- a) The pathophysiology of vertigo in this condition is not well understood, although various hypotheses, including ischaemia and proprioceptive damage have been suggested. Trauma to the neck can interfere with blood supply either to the posterior circulation through the vertebral arteries or via the internal carotid arteries.
- b) Whiplash injury is common in road traffic accidents and in other trauma with forced flexion and extension of the cervical spine. This may be related to damage to the proprioceptor mechanisms, or the normal vestibulospinal reflex arc. In either case, vertigo and other balance disturbances may occur.
- c) A late whiplash injury syndrome has been reported which develops in 15-20% of cases. This syndrome includes headache, vertigo, instability, tinnitus and nausea as features.³⁰ However, not all authors accept this as a diagnosis.

3.7.10. **Post-concussion syndrome**

- a) Minor head trauma that does not result in skull fracture or brain injury can have persistent and disabling effects.
- b) The syndrome has the following features: dizziness, vertigo, headache, memory loss, perceptual changes, paraesthesiae, tinnitus and psychological disturbances. These are not all present in every case.
- c) Those patients with vertigo will usually have 1 of 4 diagnoses: post-traumatic migrainous vertigo, benign paroxysmal positional vertigo (see section 3.6.4), post traumatic labyrinthine concussion, or post-traumatic spatial disorientation.³¹ The remainder are difficult to classify.

3.7.11. **Brain injury.** More severe head trauma resulting in brain injury may cause vertigo depending on the site of damage.

4. Prognosis

- 4.1. Prognosis depends upon the cause of the vertigo.
- 4.2. In general, if there is no ongoing damage to the vestibular system, no ongoing systemic illness and no psychological dysfunction, the individual should be able to compensate for the damage done. In these situations, decompensation may occur when the system is under stress. This can be due to infection, anxiety and tiredness.
- 4.3. People who have compensated well to their vestibular damage may still have chronic instability (rather than vertigo). This may be sufficient to make their previous employment impossible. For example, post-traumatic spatial disorientation may mean permanent grounding for a pilot. In these situations, objective evidence is usually required.
- 4.4. The vertigo associated with general medical conditions may improve as the general condition is managed.
- 4.5. Good early management of any psychological aspects causing or related to vertigo will improve habituation to any vestibular damage that is present. Once behaviour patterns are established, it can be difficult for intervention to be successful.
- 4.6. Chronic repetitive episodes of illness such as migraine, Ménière's disease and recurrent vestibular decompensation present different problems. These conditions tend to resolve with time but this may take many years.
- 4.7. Ménière's disease usually causes permanent damage to the inner ear as episodes occur. As well as recurrent vertigo, there will be other problems associated with permanent hearing and balance disorder.
- 4.8. Migraine tends not to cause permanent damage, but patients may not get more than 10-15 minutes warning that attacks are going to occur. This may lead to reassessment of employment.
- 4.9. Similarly, recurrent vestibular decompensation tends not to cause permanent damage but sufferers will need regular, and unpredictable, time off work. This may be for 2-3 months at a time.
- 4.10. Acute vestibular failure, whether post-infective, post-traumatic or vascular, is usually a one-off illness with dramatic improvement in a relatively short time. However, this may take some time to resolve fully, especially with high frequency, high acceleration head movements. This can take up to 3 years, during which time, the patient is habituating to the damage done to the vestibular system. Where there is permanent damage, the employment status of the patient may have to be reassessed.
- 4.11. Complete recovery is usual from benign paroxysmal positional vertigo, whatever its cause. Particle repositioning manoeuvres, such as the Epley,¹⁶ are very efficient in moving the otoconial debris out of the semicircular canal and, thus, stopping the symptom. However, patients often suffer recurrent episodes over time, requiring further Epley manoeuvres.

- 4.12. At present, there is a great deal to learn about how the vestibular system works. Research in this area and into better investigation of vertigo should improve diagnosis and management.
- 4.13. There is still a significant group of patients where despite careful history taking, examination and investigation, no specific diagnosis can be made. The prognosis in these cases is variable.

5. Summary

- 5.1. Vertigo is a common symptom, experienced by up to 17% of the population at any one time.
- 5.2. Vertigo is a symptom of dysfunction in the vestibular system. It can be due to many conditions of the inner ear, the vestibular branch of the 8th cranial nerve, the brain stem and cerebellum, and rarely the vestibular cortex and its connections.
- 5.3. Any condition affecting the blood supply or metabolic status of these structures can also cause vertigo.
- 5.4. There is an association between psychological disturbance and vertigo. Anxiety prolongs habituation to acute vestibular insults. The sensation of vertigo is very unpleasant and can lead to anxiety and panic. Vertigo can also be a symptom of panic disorder and phobias.
- 5.5. Vertigo is one of the features of both post-traumatic stress disorder and the post-concussion syndrome.
- 5.6. In most cases, the symptom of vertigo settles with time but there may be residual damage that can affect quality of life and employment.
- 5.7. One of the most common vestibular syndromes is benign paroxysmal positional vertigo. This may occur after head or neck trauma but it is usually completely curable, once the diagnosis is made.

6. Related Synopses

Blast Injury of the Ear

Epilepsy

Migraine

Stroke

Whiplash

7. Glossary

Addison's disease	A disease of the adrenal glands characterised by low blood pressure, loss of appetite, weight loss, and weakness. Without replacement hormonal therapy, it is usually fatal.
ataxia	Difficulty in movement, especially co-ordination of movement.
aura	A sensation or phenomenon (e.g. flashing lights, an odour, or the feeling of a breeze) which often precedes a paroxysmal attack, such as an epileptic seizure or migraine.
hypercarbia	An excess of carbon dioxide in the blood.
hypopituitarism	Abnormally decreased secretion of hormones from the anterior pituitary gland.
hypoxia	Insufficient oxygen levels in the blood.
inner ear	Part of the ear that contains both the organ of hearing (cochlea) and the organ of balance (labyrinth).
insulinoma	Insulin-producing tumour of the pancreas. The increased production and blood level of insulin resulting from these tumours can cause low blood sugar (hypoglycaemia).
labyrinth	A system of fluid passages in the inner ear, comprising the semicircular canals and the vestibule, which provides the sense of balance.
neuritis	Inflammation of nerves; in this context the vestibular nerve (q.v.).
otolith	A particle of calcium carbonate found in the inner ear of vertebrates and involved in sensory perception.
phonophobia	Intolerance of sound.
photophobia	Intolerance of light.
polycythaemia	An overabundance of red blood cells; the opposite of anaemia (a lack of red blood cells).
posterior fossa	The part of the skull containing the brain stem and the cerebellum.
prodrome	An early or warning symptom of a disease.

stenosis	The narrowing of a duct or blood vessel.
temporal cortex	A lobe of the brain which is located laterally and below the cerebrum.
Tullio phenomenon	The phenomenon where loud noise provokes vertigo. This can occur following severe injury to the labyrinth or in a condition where the bony wall of the semicircular canal is eroded.
vertebrobasilar system	A circulatory system consisting of the 3 main blood vessels that provide circulation to the back of the brain.
vestibular nerve	Nerve that carries information relating to body orientation from the vestibular system (q.v.) to the brain.
vestibular system	Organ of the inner ear that is involved in balance and body orientation. Contains the 3 semicircular canals, as well as the saccule and utricle.

8. References

1. Royal College of General Practitioners and Office of Population Census and Surveys (RCGP/OPCS). Morbidity statistics from general practice. London: HMSO; 1986.
2. Baloh R. Dizziness in older people. *J Am Geriatr Soc* 1992 Jul;40(7):713-21.
3. Kroenke K. Symptoms in medical patients: an untended field. *Am J Med* 1992 Jan 24;92(1A):3S-6S.
4. Stephens SDG. Auditory and vestibular rehabilitation in the adult. *Horizons in Medicine* 1990;2:220-8.
5. Luxon L. Vertigo in old age. In: Dix MR, Hood JD, editors. *Vertigo*. Chichester: Wiley; 1984. p. 291.
6. Yardley L. *Vertigo and dizziness*. London: Routledge; 1994.
7. Rudge P. *Clinical neuro-otology*. Edinburgh: Churchill Livingstone; 1983.
8. McAlpine D, Lumsden CE, Acheson ED. *Multiple sclerosis: a reappraisal*. Edinburgh: Churchill-Livingstone; 1972.
9. Barnett HJ, Hyland HH. Tumours involving the brainstem: a study of 90 cases arising in the brainstem, fourth ventricle and pineal tissue. *Q J Med* 1952 Jul;21(83):265-84.
10. Olesen J. Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. *Cephalalgia* 1988;8(Suppl 7):9-96
11. Neuhauser H, Leopold M, von Brevern M, Arnold G, Lempert T. The interrelations of migraine, vertigo and migrainous vertigo. *Neurology* 2001 Feb 27;56(4):436-41.
12. Ménière P. Pathologie auriculaire: memoirs sur une lésion de l'oreille donnant lieu a des symptoms de congestion cérébrale apoplectiforme. *Gazette Médecine du Paris* 1861;16:597-601.
13. Radtke A, Lempert T, Gresty MA, Brookes GB, Bronstein AM, Neuhauser H. Migraine and Ménière's disease: is there a link? *Neurology* 2002 Dec 10;59(11):1700-4.
14. Various authors. Benign positional vertigo. Update. *Audiological Medicine* 2005;3(1):1-68.
15. Hall SF, Ruby RR, McClure JA. The mechanism of benign paroxysmal vertigo. *J Otolaryngol* 1979 Apr;8(2):151-8.
16. Epley JM. New dimensions of benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg* 1980 Sep-Oct;88(5):599-605.
17. Stack BC Jr, Fariior JB. Missile injuries to the temporal bone. *South Med J* 1995 Jan;88(1):72-8.

18. DiBiase P, Arriaga MA. Post traumatic hydrops. *Otolaryngol Clin North Am* 1997 Dec;30(6):1117-22.
19. Roitman R, Talmi YP, Finkelstein Y, Silver S, Sadov R, Zohar Y. Acoustic trauma-induced Meniere's syndrome. *ORL J Otorhinolaryngol Relat Spec* 1989;51(4):246-50.
20. Paparella MM, Mancini F. Trauma and Meniere's syndrome. *Laryngoscope* 1983 Aug;93(8):1004-12.
21. Ylikoski J. Delayed endolymphatic hydrops syndrome after heavy exposure to impulse noise. *Am J Otol* 1988 Jul;9(4):282-5.
22. van der Laan FL. Noise exposure and its effects on the labyrinth, Part I. *Int Tinnitus J* 2001;7(2):97-100.
23. Segal S, Eviatar E, Berenholz L, Kessler A, Shlamkovitch N. Is there a relation between acoustic trauma or noise-induced hearing loss and a subsequent appearance of Ménière's disease? An epidemiological study of 17425 cases and a review of the literature. *Otol Neurotol* 2003 May;24(3):387-91.
24. Ildiz F, Dundar A. A case of Tullio phenomenon in a subject with oval window fistula due to barotrauma. *Aviat Space Environ Med* 1994 Jan;65(1):67-9.
25. Roth Y, Kronenberg J, Lotem S, Leventon G. Blast injury of the ear. *Harefuah* 1989 Nov 15;117(10):297-301.
26. Cohen JT, Ziv G, Bloom J, Zikk D, Rapoport Y, Himmelfarb MZ. Blast injury of the ear in a confined space explosion: auditory and vestibular evaluation. *Isr Med Assoc J* 2002 Jul;4(7):559-62.
27. Farmer JC. Diving injuries to the inner ear. *Ann Otol Rhinol Laryngol Suppl* 1977 Jan-Feb;86(1 Pt 3 Suppl 36):1-20.
28. Bellenkes A, Bason R, Yacavone DW. Spatial disorientation in naval aviation mishaps: a review of class A incidents from 1980 through 1989. *Aviat Space Environ Med* 1992 Feb;63(2):128-31.
29. Grossman A, Chapnik L, Ulanovski D, Goldstein L, Azaria B, Sherer Y, Barenboim E. Acute cerebellar vertigo in a fighter pilot. *Aviat Space Environ Med* 2004 Oct;75(10):913-5.
30. Claussen CF, Claussen E. Neurotological contributions to the diagnostic follow up after whiplash injuries. *Acta Otolaryngol Suppl* 1995;520 Pt 1:53-6.
31. Hoffer ME, Gottshall KR, Moore R, Balough BJ, Webster D. Characterising and treating dizziness after mild head trauma. *Otol Neurotol* 2004 Mar;25(2):135-8.