Vertigo

In general, we will not expect of you to have a thorough knowledge of Vertigo. This fairly comprehensive review aims to serve as a reference source for you.

Part I – History and examination

Introduction:

Vertigo and dizziness are complex problems, which is poorly understood by most practitioners. In a busy practice or casualty setting, attending to a dizzy patient can be difficult to say the least. Traditional teaching places an extreme emphasis on distinguishing vertigo from dizziness, unsteadiness, oscillopsia, and presyncope. The problem however is that patients often have no clue as to what this means and would use these terminologies completely incorrectly and interchangeably.

It is estimated that approximately 90% of individuals over 65 years of age have visited their physician at least once for vertigo as their primary complaint, and the lifetime incidence is 30%. A diagnosis can be made in 70% of patients on history alone. Physical examination and special tests will only contribute 10-20% respectively. Vertigo can be successfully treated in 90% of patients. Unfortunately, patients are often over investigated, misdiagnosed and given a cocktail of medications.

My aim is to provide you with some basic anatomy and physiology knowledge, but more importantly a stepwise approach to a dizzy patient. Of course, it is very easy to over-complicate or simplify this topic which is of no value to you. At the end you will be able to distinguish vertigo from other forms of dizziness. You will also be able to differentiate vertigo further into peripheral and central pathologies and advise the correct treatment options. Complex cases should however be identified and referred to a specialized unit.

Anatomy and Physiology:

Each labyrinth consists of the vestibular apparatus and the cochlea. The vestibular apparatus can be divided into three semi-circular canals, and the otolith organ which is composed of the utriculus and sacculus (figure 1).

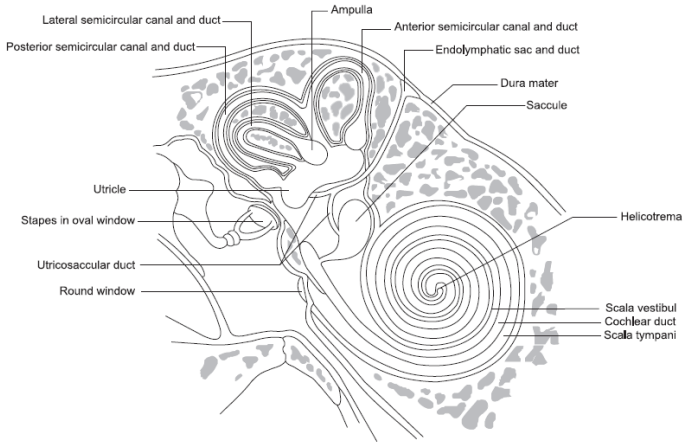

Figure 1. Inner ear anatomy

The semi-circular canals register angular rotations, and the otolith organs linear changes and gravitational direction. The information from the ears, along with visual and proprioceptive information forms the major input to the balance system as depicted in figure 2.

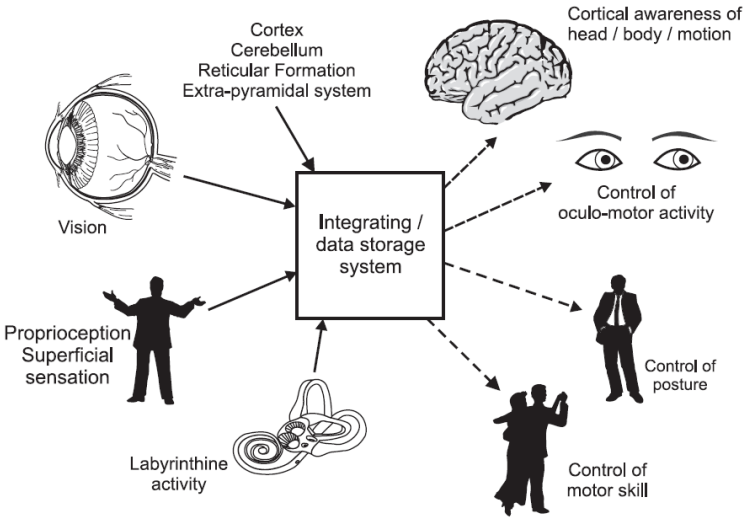

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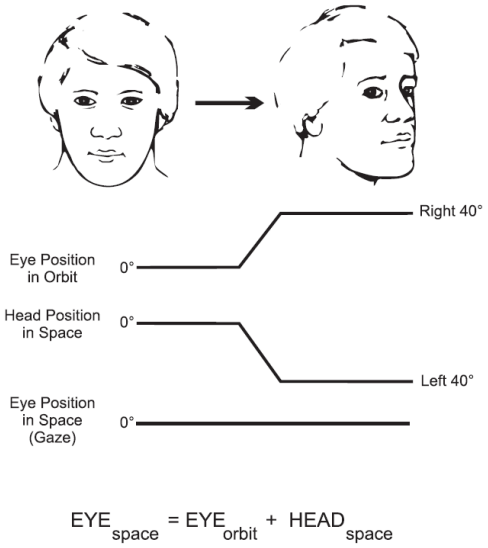
Figure 2. Input and output of vestibular system

The vestibular end organs are dynamic structures. They are not silent until stimulated but constantly discharge at a resting pattern of signals to the brain. The two sets (right and left) of vestibular apparatus are mirror images of one another. With any acceleration / deceleration movement the opposite but equal change in firing rate takes place on the contra-lateral side. The cerebral cortex interprets the change in firing rate as movement of specific direction and speed. The vestibular nuclei have important connections to various regions in the brain, but three of the most important are to the ocular nuclei, cerebellum and spinal tracts. In response to vestibular stimulation, the eyes will move in the opposite direction to retain the field of last gaze, or otherwise known to reduce retinal slip as not to cause blurred vision (see figure 3). This allows for movement without becoming dizzy or feeling off-balance while still maintaining clear vision. This is the basis of what is referred to as the vestibular-ocular reflex (VOR). Examination of the VOR is of vital importance in the dizzy patient as will be later shown. So, at its core, the vestibular organs change mechanical energy to electrical energy and activate the VOR. This produces extremely fast (< 80 milliseconds) compensatory eye movements to keep focus on a target. It is far quicker than conscious eye movements, known as neurological eye movements or ocular-motor signs.



$$EYE_{space} = EYE_{orbit} + HEAD_{space}$$

Figure 3. Vestibular-ocular reflex

Vestibular-spinal tracts will adjust trunk and limb muscles, and the cerebellum adjust muscle tone to meet the new situation. Over time the brain has learned exactly what to expect from the vestibular organs, partly instinctual but mostly learned as a baby learns to walk etc. The vestibular organs are therefore in constant dynamic balance, one checking the other and working as a team.

Disease strikes:

With a sudden pathological dysfunction of one vestibular organ (mostly hypofunction), the two sides discharge at unequal rates. This unbalanced information is interpreted by the brain as a condition of constant motion. This is the basis of our definition of vertigo, namely a hallucination of movement which will be rotatory in most cases, but can be describe as pitching, yawing or rolling in character. The same unbalanced information arrives at the ocular nuclei and spinal tract. The eyes move in response to the stimulation to the last field of gaze and the slow phase of nystagmus is born. Because the eyeball can't turn 360°, it reaches a point of maximal deviation and a signal from the reticular formation return the eyes to their starting position, which forms the fast phase of nystagmus. By international convention, nystagmus is reported in the direction of the fast phase. The same unbalanced information in the spinal tract causes staggering and ataxia.

In a matter of minutes, the cerebellum imposes a shutdown of the unbalanced information from the vestibular nuclei. This alleviates but certainly does not eliminate the immediate problem. Fortunately, over a few days the spontaneous nystagmus and vertigo abate because of plasticity and adaptation within the central nervous system, but the asymmetric VOR persist as long as the impaired labyrinth remains depressed (important to note in diseases like vestibular neuritis). Taking this explanation into consideration there are two important rules; (1) a vestibular crisis of any severity will cause vestibular nystagmus of which the characteristics will be explained later on; (2) if the symptoms last continuously for more than 2 to 3 weeks, the cause is not vestibular.

Bilateral vestibular loss will cause severe oscillopsia with minimal vertigo. Oscillopsia refers to a sensation of bobbing up and down and blurred vision when moving. Patients struggle to read signs while moving, and once they stand still, they are able to do so. These patients tend to make slower head movements by turning the head and body together to avoid this sensation.

In contrast, unilateral loss of otolith input causes postural instability and deviations in upright stance. During acute otolith loss, the head tilts and the body leans towards the impaired side because of loss of extensor tone on the involved side. Usually, the ipsilateral eye counter roll inferiorly and the contralateral eye superiorly. Because proprioceptive input is intact, the patient can still stand. Over time the deviation lessens, and the patient shows little sign of injury. With bilateral loss of otolith input, the patient is deprived of his or her internal sense of gravity. He or she becomes more dependent on proprioceptive and visual cues and has trouble walking in the dark or on unpredictable surfaces (sand, grass, inclines). This disability persists although most patients learn to ambulate under most conditions quite well.

The ability to maintain quiet stance is driven mainly by proprioceptive input. Pressure receptors in the soles of the feet and joint stretch receptor information in the feet, legs, trunk, and neck, all combine to create a rich network of multilevel subcortical and cortical reflex pathways designed to maintain the body's centre of gravity over its base of support at the feet. During vestibular dysfunction, this system becomes more important to make up for loss of labyrinthine information regarding gravity. Diseases such as peripheral neuropathies and corticospinal degeneration interrupt these pathways and create difficulties with posture control and ambulation.

Definitions:

Table 1. Symptoms and Definitions.

Symptom	Definition
Vertigo	Vertigo implies a hallucination of movement. That means a sensation of movement when no movement is occurring. Usually, it is a rotatory sensation with the room spinning around the patient, or rarely the patient spinning around the room. Rarely the sensation of movement can be an up/down or side-to-side.
Dizziness	The sensation of disturbed or impaired spatial orientation without a hallucination of movement

Unsteadiness / Disequilibrium	The feeling of being unstable while seated, standing, or walking without a particular directional preference
Oscillopsia	A bobbing sensation (close your one eye, and press up and down on the lower eyelid of the open eye)
Presyncope	The sensation of impending loss of consciousness
Syncope	Transient loss of consciousness due to transient global hypoperfusion characterized by rapid onset, short duration, and spontaneous complete recovery
Non-specific dizziness	Any other balance related sensation not fitting the prior categories

Approach to a patient:

There are some excellent articles on the examination of dizzy patients [2,3,4]. I would urge all general practitioners to read through these, as they explain specific tests in more detail which I did not include here due to space limitation. We know that a diagnosis can be made in 70% of patients on history alone and physical examination and special tests will contribute 10-20% respectively. History therefore is of vital importance, and there are three approaches to this. The **classical approach** is to differentiate between "vertigo" and "the rest". It assumes that patients are able to describe their symptoms to a high degree. In reality, quite frequently, patients find it very difficult to describe their symptoms. Thus, the **alternative approach** can be used, especially in a casualty setting. Lastly, one can focus on only the **million-dollar questions** to establish a diagnosis.

Below is an outline of the history and examination. It is neither fully comprehensive nor will everything be discussed. However, as you will read along, it will build on the outline given below.

- History
  - Classical approach
  - Alternative approach
  - Million-dollar questions
- Examination
  - General
    - One can form an immediate clinico-pathological picture by assessing the patient's posture and walking when coming into the consulting room. The physician should also look for signs of anaemia, lymphadenopathy, weight loss and any other obvious signs.
  - Head and neck
  - Ear, nose and throat (ENT)
  - Neuro-otological
    - Nystagmus
      - Spontaneous / Induced
      - Fixation – Gaze straight
      - Fixation – gaze in different positions
      - Without fixation
    - Central oculo-motor signs
      - Smooth pursuit
      - Saccades
      - Vergence
      - Visual fixation / gaze holding
      - Optokinetic nystagmus
    - VOR battery
      - Dix Hallpike and Lateral semi-circular canal testing
      - Dynamic visual acuity test (DVAT)
      - Head thrust / Head impulse test
      - Head shake
      - Caloric test

- Rotation testing
- Fistula test
- Fixation suppression test
- Subjective visual vertical

- CNS
  - Higher functions
    - GCS
    - Orientation
    - Intellect
    - Communication
    - Emotional status
  - Cranial nerves
    - I
      - Smell
    - II
      - Vision (Snellen chart)
      - Vision (counting fingers at 1 meter)
      - Eye fields
      - Colour vision
      - Ophthalmoscopy
      - Pupil reflex
        - Direct
        - Indirect
    - III, IV, VI
      - Ptosis
      - Movement
      - Cover up
      - Diplopia
    - V
      - Sensory
      - Motor
      - Corneal reflex
      - Jaw reflex
    - VII
      - Sensory
      - Motor
      - Taste
      - Lacrimation
      - Hyperacusis
    - VIII
      - Cochlear
      - Vestibular
    - IX, X
      - Soft palate sensation
      - Gag reflex
      - Larynx
    - XI
      - Inspection
      - Palpation
      - Motor
    - XII
      - Inspection
      - Palpation
      - Motor

        - Cerebellum
            - Fast tongue movements
            - Ataxia
            - Dysmetria
            - Finger-nose test
            - Dysdiadochokinesia
            - Romberg
            - Heel-shin test
            - Pendular reflexes
            - Nystagmus
        - Motor
            - Inspection
            - Palpation
            - Strength
        - Sensory
            - Pain, temperature and general sensation
            - Light touch, position and vibration
            - Stereognosis
        - Reflexes
        - Gait
        - Coordination
        - Posture
    - It is important to also examine the following systems
        - CVS
            - Arrhythmias
            - Reduced cardiac output
            - Hypovolemia
            - Pericarditis
            - Orthostatic hypotension
            - Autonomic dysfunction
            - Vasovagal syncope
        - Hyperventilation
        - Hypoglycaemia
    - Special examinations
        - VNG
        - VEMP's
        - Posturography
        - Scans
        - Neurologist / Cardiologist / Physician / GP
        - Audiology / audiologist
        - Physiotherapist
        - Bloods

History:
<u>Classical approach</u>

The key history points are:

- Is it vertigo?
- What is the time course?
- Precipitating / Exacerbating factors
- Accompanying symptoms

Vertigo can be an hallucination that the external world is moving relative to an individual or the individual relative to space. Rotational vertigo or other hallucinatory sensations of motion indicate vertigo (vestibular symptoms), whereas a sensation of light headedness, giddiness, drowsiness, or impending fainting implies dizziness of non-vestibular origin. Non-spinning dizziness only when standing or walking usually indicates a neurological gait problem rather than vestibular vertigo.

Vertigo onset is usually sudden and comes in spells varying from seconds or minutes to hours. The offset is less clear with patients feeling unwell for a variable time. Some types of peripheral vertigo is brought on by a change in position and most will improve by lying still. Tinnitus, hearing loss, and aural fullness frequently accompany peripheral disease. Central disease can rarely also have hearing loss and tinnitus if the anterior inferior cerebellar artery is involved.

Unlike peripheral vertigo, central causes of dizziness produce a more variable picture. Patients may describe it as spinning, tilting, forced to one side, light headedness, clumsiness or blacking out. If loss of consciousness is documented, a peripheral aetiology for dizziness is rarely – if ever – at fault. The following symptoms also points to a central cause namely dysarthria, dysphagia, diplopia, hemiparesis, severe localized cephalgia, seizures or memory loss.

Alternative approach

The new approach places an emphasis on categorizing timing and triggers, and four key patterns are recognizable:

- Acute, spontaneous, prolonged symptoms (acute vestibular syndrome)
- Episodic, positional symptoms
- Episodic, spontaneous symptoms
- Chronic unsteadiness
    - With oscillopsia
    - Without oscillopsia

Acute, spontaneous, prolonged symptoms:
This is seen in patients with vestibular neuritis, labyrinthitis (which is extremely rare and will present with hearing loss), or posterior fossa stroke. Differentiating between vestibular neuritis and posterior fossa stroke can be difficult. Only 50% of patients with a posterior fossa stroke will have symptoms such as diplopia, dysarthria, dysphagia, and dysmetria. On the other hand, audiological symptoms such as tinnitus and hearing loss usually points to an ear problem but can also occur in anterior inferior cerebellar artery stroke. The most sensitive way to discriminate between the two conditions on clinical grounds is explained by the acronym HINTS. This stands for head impulse test (HI), nystagmus pattern (NT), and alternate cover test for skew deviation (S). An abnormal head impulse test and unidirectional direction fixed nystagmus will point to vestibular neuritis. Direction changing or weird patterns of nystagmus, and skew deviation will point to a central problem. (HINTS describe below again).

Episodic, positional symptoms:
This is seen in patients with BPPV and central mimics such as central positioning nystagmus. BPPV can be diagnosed almost instantaneously on history taking. Patients will complain of a severe spinning sensation for seconds, when lying down, turning over in bed, looking up, or bending over. As doctor you need to be specific about questioning these patients, as they would often misinterpret the spinning sensation with the unwell, nauseated feeling that follows. BPPV can be confirmed with positional testing and will cause a unidirectional specific type of nystagmus. Central positioning nystagmus will cause a direction changing nystagmus depending on the position.

Episodic, spontaneous symptoms:
Ninety percent can be explained by six disorders (discussed in more detail under diseases):

1. Meniere's disease is characterized by vertigo attacks lasting 20 minutes to hours. The patients would typically complain about tinnitus, aural fullness and hearing loss with an attack. With time the hearing loss becomes permanent (especially the lower tones).

2. Vestibular migraine can cause vertigo lasting minutes to days. There is a female predominance, and most patients will have a history of previous migraine. Most patients will develop migraine symptoms during a vertigo attack as time goes on.
3. Vertebrobasilar TIAs affect older patients with vascular risk factors. Most attacks will last less than 1 hour and can be accompanied by other symptoms of the posterior fossa circulation.
4. Vestibular paroxysmia is caused by vascular compression of the eight' cranial nerve. It is characterized by numerous brief attacks of vertigo lasting seconds, especially when turning the head left or right.
5. Orthostatic hypotension causes brief attacks of dizziness lasting seconds to minutes after standing up. In older patients it may be accompanied by supine hypertension.
6. Panic attacks usually last minutes, occur in specific situations, and are accompanied by choking, palpitations, tremor, heat, and anxiety symptoms.

Less common causes are labyrinth fistulas such as superior semi-circular canal dehiscence, cardiac arrhythmia, otosclerosis, autoimmune inner ear disease, and medication side effects.

Chronic unsteadiness (with or without oscillopsia):
This is seen in patients with persistent postural perceptual dizziness (3PD) (previously chronic subjective dizziness (CSD) and phobic postural vertigo), cerebellar degeneration, bilateral vestibular failure (will produce oscillopsia), spinal cord compression, metabolic disease, or psychiatric problems.

3PD is commonly reported as the second most common diagnosis, after BPPV, among patients with vestibular symptoms. It is characterized by persistent unsteadiness and non-vertiginous dizziness, usually worse when walking, less severe when standing, and absent or minimally present when they are lying down. Other traits have been associated with the disease over the years such as being more common in patients with obsessive compulsive behaviour, it improves with mild intake of alcohol, and the patients can typically still produce complex balance tasks (which is usually automatic) such as running or riding a bicycle.

Million-dollar questions (short cut)

As mentioned, some questions in the history can point to as diagnosis, so to speak "Million-dollar" questions.

- Do you get dizzy just rolling over in bed?
  - Benign paroxysmal positional vertigo (BPPV)
- Are you light sensitive during your dizzy spell or / and had a previous diagnosis of migraine?
  - Vestibular migraine
- Does one ear feel full before or during an attack?
  - Meniere's disease
- Does a loud sound make you dizzy or make your world jiggle?
  - Superior semi-circular canal dehiscence
- Was your first attack severe vertigo lasting hours with nausea and vomiting?
  - Vestibular neuritis
- Are you lightheaded when you get up from a bed or chair for a few seconds?
  - Blood pressure / Cardiovascular disease (CVS)
- Do you pass out completely with your dizziness?
  - CVS

Other symptoms

Presyncope and syncope would prompt a search for cardiovascular, metabolic or central causes. ***Unsteadiness*** without dizziness or vertigo is most commonly seen in patients with sensory loss (e.g., peripheral neuropathy), spinal cord diseases (eg, transvers myelitis, cord compression), and slowly progressive, bilateral cerebellar or vestibular failure. ***Gait unsteadiness*** only with eyes closed, or when walking on an uneven surface or in the dark, is usually bilateral vestibular failure. ***Oscillopsia*** present at rest usually indicates the presence of spontaneous nystagmus (brainstem lesions, drug overdose, or alcohol intoxication). Oscillopsia that occurs

only when walking (head motion) usually indicates bilateral vestibular failure. Nonspecific dizziness would prompt a search for psychiatric or metabolic causes.

Examination:

As mentioned earlier a general, ENT and neuro-otological examinations are done. I will mainly focus on the neuro-otological part. The section below does not follow the natural sequence of events in a consulting room. That is discussed under the heading **"how do I do it"** that follows this section. I will also discuss **"Some HINTS on differentiating between vestibular neuritis and posterior fossa stroke"**. This is certainly repetitive but can be consulted later on as single sections.

Firstly, look for nystagmus with fixation with the gaze straight and then in different positions. Repeat the same without fixation usually using Frenzel glasses (20+ dioptre), although if you do not have access to these, a dimly lit room will aid in reducing fixation. Lastly, look for nystagmus in positional testing (discussed later). Peripheral nystagmus (vestibular nystagmus) will cause a unidirectional, direction fixed nystagmus which will increase when looking in the direction of the fast phase and with loss of fixation. In almost all cases it will be horizonto-rotatory with the eye in the neutral position (important, because patients often would look in the direction you are turning them). All other forms points to a central problem such as direction changing, disconjugate, seesaw, pendular, congenital and gaze nystagmus. Purely vertical and / or tortional nystagmus always points to a central problem. A differential diagnosis for these types of nystagmus is given below.

Next examine specific oculo-motor eye movements. For **smooth pursuit** testing, ask the patient to track your finger / a light in the horizontal and vertical planes. Make sure as not to exceed 40^0/s and more than a 60^0 arc. As a general rule horizontal smooth pursuit is better than vertical, and both will diminish with age. Next ask the patient to look back and forth between two fingers about 20 cm apart without moving their head. This is known as **saccade** testing. Observe the eyes for either under or over correction and also conjugate movement. Abnormalities in smooth pursuit or saccade testing point to a central problem. The other oculo-motor tests won't be discussed but two of them, namely **fixation** and **vergence** are covered when testing the cranial nerves.

The VOR battery of testing follows: The **Dix Hallpike** and **lateral semi-circular canal** testing are specifically aimed at diagnosing the different forms of BPPV. Because posterior canal BPPV causes more than 90% of problems the Dix Hallpike test is discussed in more detail (see figure 4). With the patient sitting up in bed the head is turned 45^0 to the side and the patient is brought into a supine position with the head barely hanging over the edge of the bed. This is not a simultaneous movement and there is also no need to do this briskly. The patient is instructed to keep their eyes in the neutral position and on instruction by the physician to the left and right. Observe the eyes using Frenzel glasses and note any type of nystagmus. In posterior canal BPPV there is usually a brief latency, followed by a geotropic (beating towards the ground) horizonto-rotatory nystagmus lasting less than 60 seconds. Sometimes there is a reversal of the nystagmus pattern when coming up again. Lateral canal testing is done with the patient lying prone with the head flexed 30^0 and then turning their head to the left and right. Again, observe for any nystagmus using Frenzel glasses. Because of the different variations associated with lateral canal BPPV, it is best to refer these patients.

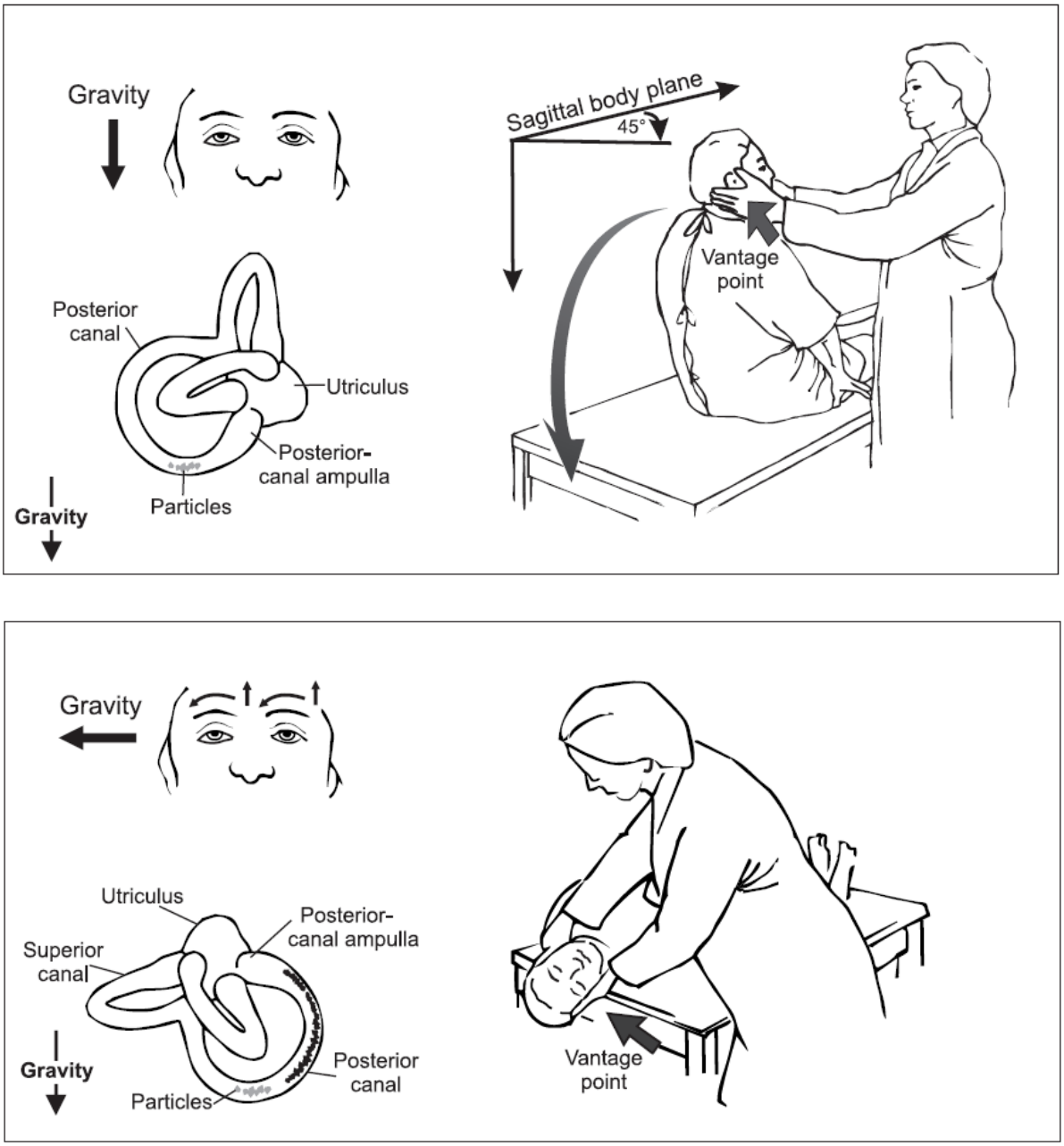


Figure 4. Dix Hallpike test for BPPV showing "crystals" in the posterior SCC.

The most common modification of this is to have the patient sitting up in bed with the feet over the edge. The head is turned 45^0 to a side and the patient is brought into a side lying supine position opposite to the side in which the head was turned. This is commonly used in elderly patients or morbid obese patients. The same process follows to note any nystagmus.

DVAT is an extremely useful test to confirm a peripheral lesion. With the best corrected vision, the patient reads the smallest line possible on a Snellen eye chart (handheld is acceptable). The procedure is repeated shaking the patient's head at 2 Hz by the examiner and record the number of lines lost during headshake. A more than 2-line drop indicates a bilateral vestibular loss or a poorly compensated unilateral loss.

The **head thrust** test is also known as **head impulse test** (HIT). This test has become one of the most important bedside tests for the evaluation of the vestibular ocular reflex (VOR). The patient is instructed to look at the examiner's nose while he quickly turns the head randomly around the horizontal axis between 20^0 - 30^0. Normal patients will have no problem keeping his gaze on the nose. With unilateral vestibular loss the VOR will fail to keep the gaze on the nose and there will be a catch-up saccade. A patient who presents with acute vertigo and has a normal HIT **strongly** points to a central problem. The HIT is illustrated in figures 5-6.

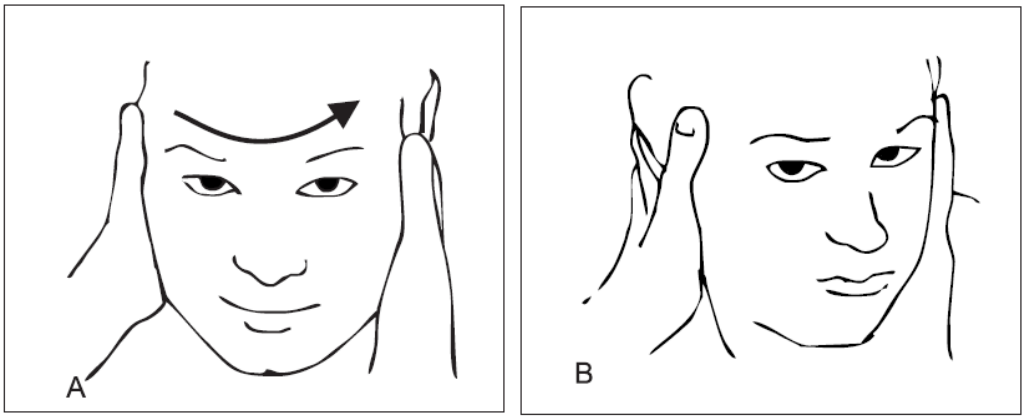


Figure 5. Normal HIT. When turning the head to the left (A), the eyes stay focused on the examiners nose (B).

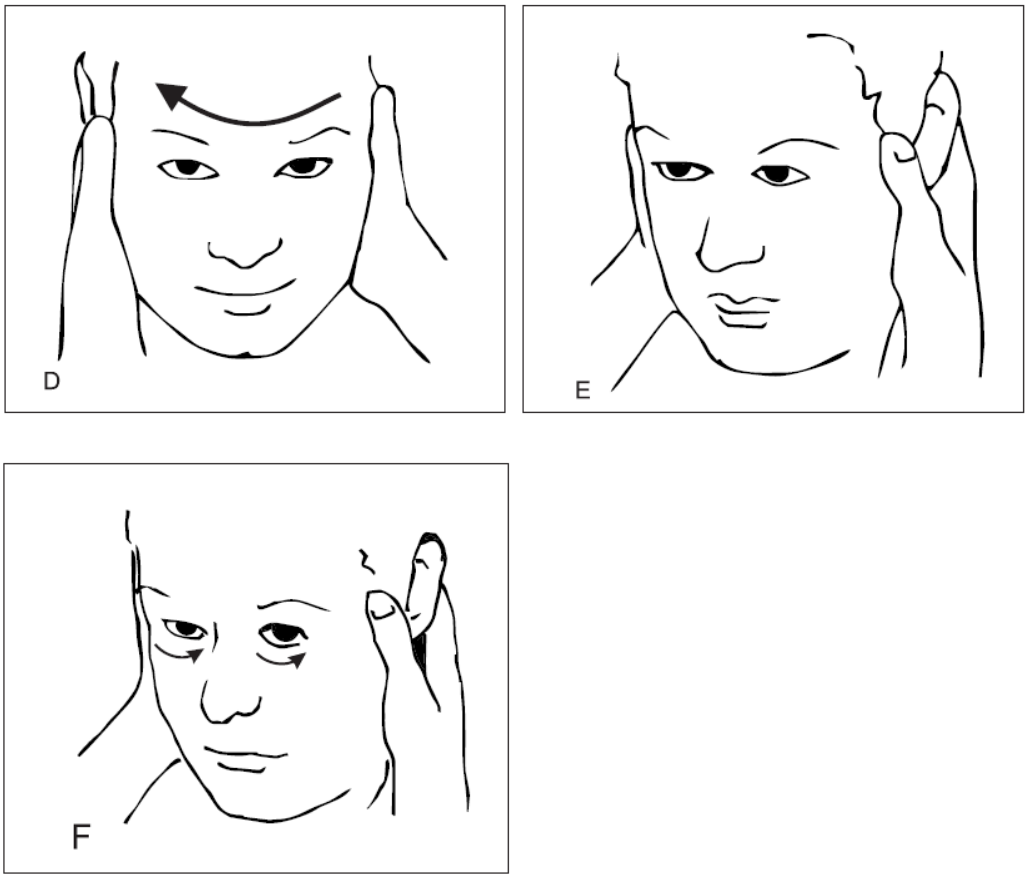


Figure 6. Abnormal HIT. When turning the head to the right (D), the eyes follow the movement of the head (E), and a corrective saccade is necessary to fixate on the examiners nose (F).

In the **head shake test**, tilt the patients head 30^0 forward and shake at 2 Hz for 20 seconds. Observe the eyes with Fentzel glasses for any abnormal eye movements. A peripheral problem will cause post head shake nystagmus with a small reversal component. Central problems may cause prolonged, disconjugate or cross coupling nystagmus (vertical nystagmus following horizontal shaking).

Caloric testing stimulates mainly the lateral semi-circular canal. Various irrigating regimes are described, but I suggest using the mini-caloric test. In this test tap water is used to irrigate the ear for 10 seconds in summer and 8 seconds in winter. The most important aspect of caloric tests is to visualize the tympanic membrane when irrigating. Frenzel glasses are used, and the durations and amplitude of nystagmus are recorded. The caloric test is very important seeing that it is the only vestibular test that can test one side at a time. The acronym COWS

stands for cold water opposite, warm water same side, indicating the direction of nystagmus produced with water. Sometimes, ice water is used in a suspected brain-dead patient to elicit an extreme caloric response. It is important to note that nystagmus occurring in the opposite direction of the cold-water injection, is a **normal** response and indicated a normal functioning vestibular system.

The Romberg test, tests primarily for somatosensory and proprioception and not vestibular input. There are however two variations to make it more sensitive for vestibular input. Firstly, instructing the patient to do a tandem stance Romberg (standing with one foot in front of the other) and secondly to do a Romberg test while standing on 10 cm foam. Observe for any sway with the eyes open and then closed.

The subjective visual vertical test will be abnormal 95% of patients with peripheral or central problems. In an abnormal test patients are unable to vertically align an object like a ruler. It is an extremely easy test to perform in a casualty setting. Close the curtains and dim the lights and ask the patient to align a ruler vertically. If they are out by more than 15 degrees, it confirms a problem. Variations of this test is known as the "bucket test".

The other neurological tests such as cranial nerves examination, cerebellar function, posture and gait will not be discussed but are of extreme importance.

Summary of results:
There are **three main** groups after the examination. Firstly, the group with definite **peripheral vertigo** will have positive signs with some of the following tests: Dix Hallpike test, lateral SCC test, caloric test (absent or abnormal response on affected side), HIT, and DVAT. Patients with **central vertigo** will have positive signs with some of the following: smooth pursuit, saccades, and cerebellar tests. Lastly **patients with dizziness** will have none of the above or sometime bizarre combinations.

The difference between peripheral and central vertigo is summarized in the table 2 below.

Table 2. Central versus Peripheral.

Differentiation of Central versus Peripheral		
	Peripheral	**Central**
Hallucinations of movement	Definite	Less definite
Onset	Usually, paroxysmal	Seldom paroxysmal
Intensity	Usually, severe	Less severe
Duration	Seconds to hours	Weeks to months
Induced by head position	Frequently	Seldom
Nystagmus	Present	Present or absent
Nystagmus pattern	Uni-directional, increased by loss of fixation	Direction changing, no change with fixation, other forms of nystagmus
Autonomous nervous system symptoms	Definite	Less definite or absent
Tinnitus	Frequently present	Seldom present
Hearing loss	Frequently present	Seldom present
Disturbance of consciousness	Absent	More frequently present
Other neurological signs	Usually, absent	Frequently present

In practice "How do I do it":

The examination starts when the patient enters the room, or the physician sees a patient lying in bed. The general examination and a thorough ENT examination are done with the patient in the chair. During the examination of the throat and larynx I would test the function of the IX and X cranial nerves. Next, I usually test all the cranial nerves with the exception of IX and X. While testing the III, IV and VI nerves I would also do all the other special eye movements tests as mentioned under the vertigo examination. Lastly, I would examination the cerebellar

functions that can be examined in the chair namely the past-pointing, dysdiadokokinesis, dysmetria, fast tongue movements and finger-nose test.

Before taking the patient to the examination bed I would take the BP because the patient has been sitting for quite a while, and then instruct the patient to stand up and take the BP again to check for orthostatic hypotension.

The next step would be to take the patient to the examination bed, but before that, with the patient standing I would do the Romberg (and Unterberger tests). On the way to the examination bed, I would instruct the patients to walk in a straight line and turn respectively to the right and then to the left. This will also reveal any forms off central ataxia.

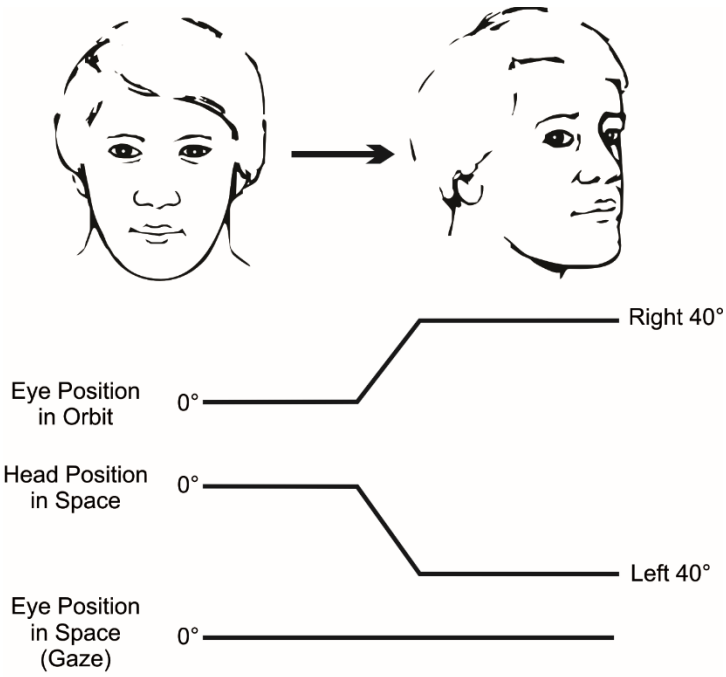
On the examination bed, with the patient lying I usually start off with the rest of the cerebellar tests namely heel-shin. After this I complete my CNS examination by testing the motor, sensory and reflex systems. This I do before examining the vestibular systems, otherwise one tends to skip that part especially in a patient who experienced vertigo and nausea during the vestibular tests.

I would start with the Dix Hallpike test, followed by the lateral canal testing. After this I usually do the head thrust and headshake tests. Lastly, and only when indicated I would do caloric tests, because this usually induces vertigo and nausea in most patients.

Some "HINTS" to differentiate vestibular neuritis from a stroke

Both vestibular neuritis and a posterior fossa stroke presents with symptoms known as acute vestibular syndrome (AVS). Patients complain of vertigo, nausea, vomiting, and unsteadiness. This can be extremely unnerving to differentiate a self-limited disorder, vestibular neuritis, from a potential life-threatening stroke. Only half of the patients with a stroke will complain of diplopia, dysarthria, dysphagia, and focal sensory and motor symptoms. On the other hand, hearing loss is not always a sign of ear disease and can be part of an anterior inferior cerebellar artery infarction. Initial MRI can be falsely negative in 12% of cases. This section will focus on the acronym "HINTS" to try and assist you to differentiate between the two conditions on clinical grounds.

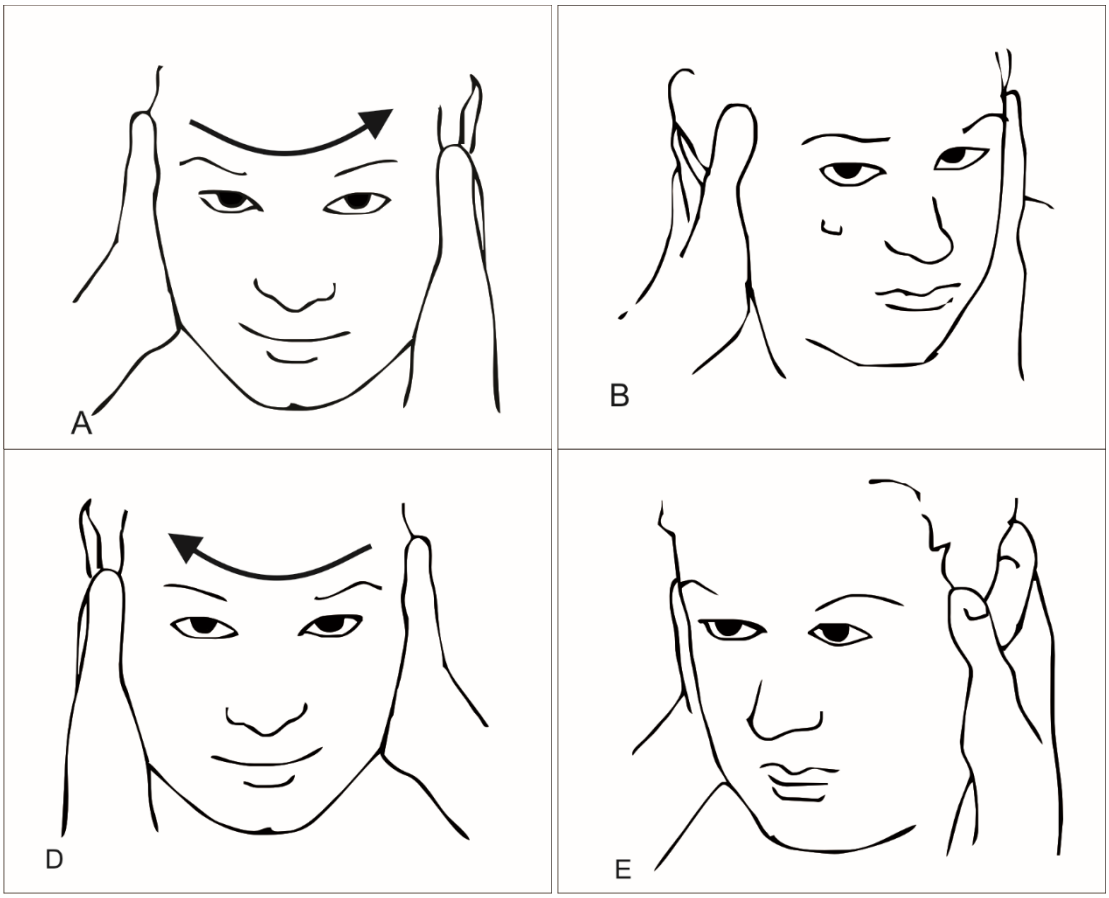
"HINTS" stands for **H**ead **I**mpulse Test, **N**ystagmus Pattern, and **T**est of **S**kew Deviation. The HIT tests the vestibular ocular reflex (VOR), which lets you focus on one spot while moving your head (Figure 7).

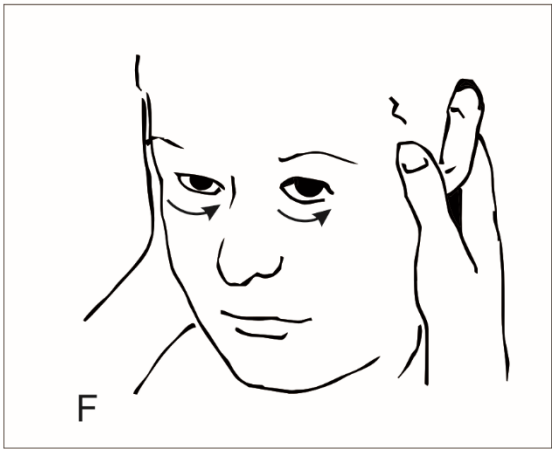


$$EYE_{space} = EYE_{orbit} + HEAD_{space}$$

Figure 7. HIT

In the HIT, you need to instruct your patient to focus on your nose, relax the neck, and then briskly turn the head 20-30° away from the midline in the horizontal plane. This needs to be randomly to the left and right. As examiner you need to see if your patients' eyes stay focussed on your nose, or whether there is a corrective eye movement after turning the head (saccade). The pictures A and B demonstrate a normal and D, E and F an abnormal HIT.





Pictures A-F. HIT testing.

A normal HIT in a patient with AVS strongly indicates a central lesion. An abnormal HIT usually indicates a peripheral problem but can be due to lateral pontine lesions.

Retrospective studies confirm that nystagmus is frequently noted, but in most cases completely inaccurate. A comprehensive overview of nystagmus falls outside the scope of this chapter, but I would like to stress a couple of important points. In these patients the nystagmus will be spontaneous as opposed to induced. As examiner you need to differentiate peripheral from central nystagmus patterns as discussed in table 3.

Table 3. Peripheral versus Central nystagmus.

	Peripheral	**Central**
Nystagmus pattern	Uni-directional horizontal nystagmus	Direction changing, vertical, gaze-evoked nystagmus
Vertigo	Will always complain about vertigo	May or may not complain about vertigo (although nystagmus is present)
Direction	Nystagmus increases when looking in the direction of the fast phase	Variable
Fixation	Nystagmus increased by loss of fixation	Variable. May be induced by fixation

In practice ask the patient to look at your finger. The first thing is to figure out the direction of the fast phase of nystagmus. If this is in one horizontal direction (either to the left or right), and is increased by asking the patient to look in the direction of the fast phase, and the direction of the fast phase is not influenced by the position of the eye, then you have a peripheral nystagmus pattern. For all other practical purposes everything else will be central in origin. Remember that in some cases you might only be able to assess the nystagmus with loss of fixation (Frenzel glasses, dark room, video nystagmography).

Skew deviation is vertical misalignment of the eyes. The alternate cover test will identify this. Sometimes it can be accompanied by a head tilt and ocular torsion. Skew deviations strongly point to a central problem.

Therefore, in vestibular neuritis, the HIT will be abnormal towards the affected side, the fast phase of nystagmus towards the healthy side, and no skew deviation. Patients with strokes will have a normal HIT, weird patterns of nystagmus, and skew deviation (sensitivity 98-100%, specificity 85-96%). Table 4 shows the differences between vestibular neuritis and stroke.

Table 4. The differences and also some additional points to remember.

	Vestibular neuritis	**Stroke**
HIT	Abnormal	Normal
Nystagmus	Uni-directional, horizontal	Varies
Skew deviation	Typically, absent	Present
Other symptoms	Hearing loss with labyrinthitis	Diplopia, Dysarthria, Dysphagia
Nausea and vomiting	In proportion to nystagmus	Either greatly increased or minimal in proportion to nystagmus
Walking and ataxia	Usually able to walk	Severe gait or truncal ataxia. Typically, unable to sit unassisted.
Stroke risk factors	Absent	Present (Smoking, hypertension, hyperlipidaemia, diabetes, atrial fibrillation, eclampsia, hypercoagulable state, recent cervical trauma, prior stroke or myocardial infarct)

A differential diagnosis for central forms of nystagmus

- Purely torsional nystagmus usually reflects intrinsic brainstem involvement within the vestibular nuclei and suggests syringomyelia.
- Downbeat nystagmus in primary position usually reflects disease at the craniocervical junction, such as the Arnold-Chiari deformity or degenerative lesions of the cerebellum.
- Upbeat nystagmus in the primary position occurs with lesions at the pontomedullary and pontomesencephalic junction or within the fourth ventricle.
- Periodic alternating nystagmus is a form of central vestibular nystagmus and is usually caused by lesions in the nodulus of the cerebellum.
- Nystagmus on attempted eccentric gaze and with slow phases that show a declining exponential time course results from an unsustained eye position command. This commonly occurs as a side effect of certain medications such as anticonvulsants, hypnotics and tranquilizers. It can also occur in patients with diseases of the vestibulo-cerebellum or its brainstem connections.
- Nystagmus with accelerating slow phases is typical of congenital nystagmus.
- Acquired pendular nystagmus may be a manifestation of multiple sclerosis, tolune intoxication, or a result of brainstem infarction with inferior olivary hypertrophy. Acquired pendular nystagmus frequently is disconjugate and may even be horizontal in the one eye and vertical in the other.
- Convergence-retraction nystagmus usually occurs with midbrain lesions.
- Seesaw nystagmus, when the one eye goes up and the other down, also occurs in midbrain lesions.

Part II – Clinical Diseases

Differential diagnosis:

Important concepts to remember are the following. Vertigo does not imply peripheral disease; it can be from a central cause as well. Vertigo does imply some form of hallucination of movement, mostly spinning. All other symptoms are grouped under dizziness.

After history and examination, the clinician needs to decide the following:

- Is it peripheral labyrinthine, eight nerve, central vestibular disease, or diffuse?

    - If labyrinthine, is it unilateral or bilateral?
    - Is this acute isolated, episodic spontaneous, episodic positional, or chronic disease?
- If central, is it brainstem or cerebellar disease?

Three typical forms of peripheral vestibular dysfunction can be identified based on their characteristic symptoms and signs

1. Acute or subacute unilateral vestibular failure, characterised by rotational vertigo, oscillopsia, and a tendency to fall toward the affected ear. Usually acute vestibular neuritis.
2. Bilateral peripheral vestibular failure (bilateral vestibulopathy), characterised by instability of gait and posture, and oscillopsia induced by head movement
3. Paroxysmal peripheral vestibular stimulation or inhibition, characterised by attacks of vertigo and oscillopsia, for instance, in BPPV, Meniere's disease, and vestibular paroxysmia.

A differential diagnosis for the most common causes of vertigo is presented in table 5.

Table 5. Vertigo causes.

Vertigo	
Peripheral	Central
Common	*Common*

• BPPV
• Vestibular neuritis
• Meniere's disease
• Bilateral vestibulopathy
• Vestibular schwannoma / Acoustic neuroma
	• Phobic postural vertigo
	• Vestibular migraine
	• Pathological forms of nystagmus for example
	  ○ Down beat nystagmus
	  ○ Upbeat nystagmus
	  ○ Gaze nystagmus

Rare	*Rare*
• Superior semi-circular canal dehiscence (SSCD) • Vestibular paroxysmia (vascular loop compression) • Perilymph fistula • Labyrinthitis • Auto-immune inner ear diseases	• Central positioning vertigo • Dizziness syndromes of unclear aetiology / familial • Episodic ataxia type II • Arnold-Chiari malformation • Psychogenic dizziness
Other problems	
Physiological / pathological stimulation	*Central nervous system diseases / causes*

• Motion disease
• Caloric stimulation
  ○ Water exposure
  ○ Wind exposure
• Rotational stimulation
  ○ Flying
  ○ Driving
• Pressure changes
• Changes in specific gravity
  ○ Alcohol induced vertigo
	• Multiple sclerosis
	• Vascular disease
	• Tumours
	• Epilepsy
	• Infections
	• Medications

A differential diagnosis for dizziness is presented in table 6.

Table 6. Dizziness causes.

Dizziness	
Condition	Causes

Pre-syncope / syncope	• Arrhythmias
	• Reduced cardiac output
	  ○ Hypovolaemia
	  ○ Pericarditis
	• Orthostatic hypotension
	• Autonomic dysfunction
	• Vasovagal syncope
	• Hyperventilation
	• Hypoglycaemia

Central nervous system	• Normal pressure hydrocephalus • Posterior fossa tumours • Primary orthostatic tremor
Cardiovascular system	• Blood pressure • Vascular diseases
Metabolic	• Glucose metabolism • Thyroid hormone production
Medication	• Blood pressure medication • Antibiotics - aminoglycoside • Chemotherapy • Psychotropic mediation • Tranquilisers
Proprioceptive / Somatosensory	• Neck disease • Peripheral neuropathy
Eye	• Poor vision
Psychogenic	

Looking at a clinical correlation it is easy to follow the schematic description below.

- Is it vertigo?
  - Yes
    - Rotatory
      - Vestibular neuritis
      - BPPV
      - Meniere's disease
      - Vestibular migraine
    - Sensation of boat
      - Bilateral vestibulopathy
  - No
    - Dizziness
- Duration
  - Seconds to minutes
    - BPPV
    - Vestibular paroxysmia
  - Minutes to hours
    - Meniere's disease
    - Vestibular migraine
  - Hours to days
    - Vestibular neuritis
  - Varies

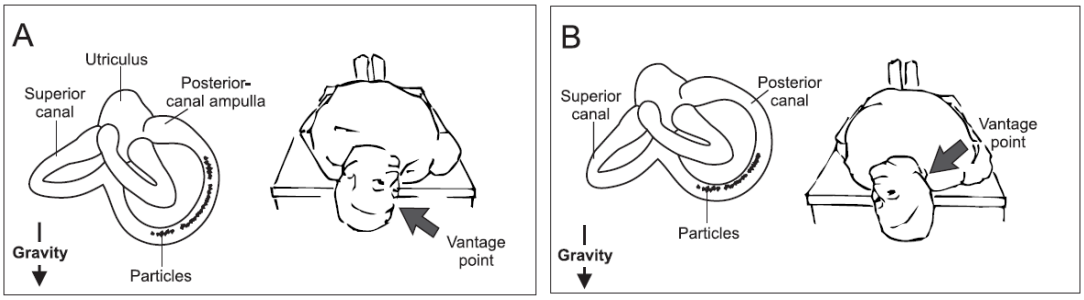
        - Fistula
        - SSCD
- Precipitating / Exacerbating factors
    - Present in rest
        - Vestibular neuritis
    - Worse when walking
        - Bilateral vestibulopathy
    - Precipitated by turning the head to the left and right
        - Vestibular paroxysmia
    - Turning in bed to one side
        - BPPV
    - Coughing / pressing / sounds
        - Fistula
        - SSCD
    - Social or environmental condition
        - 3PD
- Accompanying symptoms
    - Inner ear
        - Tinnitus, hearing loss, aural fullness
    - Central nervous system
        - Diplopia, dysphagia, sensory disturbances, dysarthria, paralysis of arms or legs
    - Headache
        - Vestibular migraine

BPPV

BPPV is the most common cause of vertigo. It affects mainly older patients and has a female predominance. It can also follow after vestibular neuritis, head trauma, prolonged bed rest and Meniere's disease but more than 90% is idiopathic. It is caused by otolith crystals getting stuck in a semi-circular canal. As the name implies this is a benign condition with a spontaneous resolution in weeks, however 30% of cases will persist. As said previously posterior canal BPPV is the most common variant (90%) and will be discussed in more detail.

It is characterized by brief attacks of vertigo after turning in bed, lying down in bed, looking up or bending down. The vertigo usually last less than 1-2 minutes and patients would typically lie still. Some patients confuse the time frame of the vertigo versus feeling unwell that follows the vertigo, but a careful history will clear this up.

It is diagnosed by doing a Dix Hallpike test as described earlier. A corrective turning manoeuvre is done to move the crystals out of the affected canal. There are two manoeuvres namely the Semont or Epley. Both are equally effective with cure rates of more than 95%. The Epley manoeuvre will be describe (see figure 7). This is a turning manoeuvre with 1–2-minute intervals at certain positions. The patient's head is kept in the same position the nystagmus was seen after the Dix Hallpike test for 2 minutes. The head is turned 90^0 towards the opposite side and wait another 2 minutes. The patient is then turned on their side, with head turning a further 90^0 in the same direction. The patient will be facing the ground, and typically will experience vertigo again (which is a good sign). After 2 minutes instruct the patient to keep their chin as far as possible on their shoulder and bring them into an upright position.



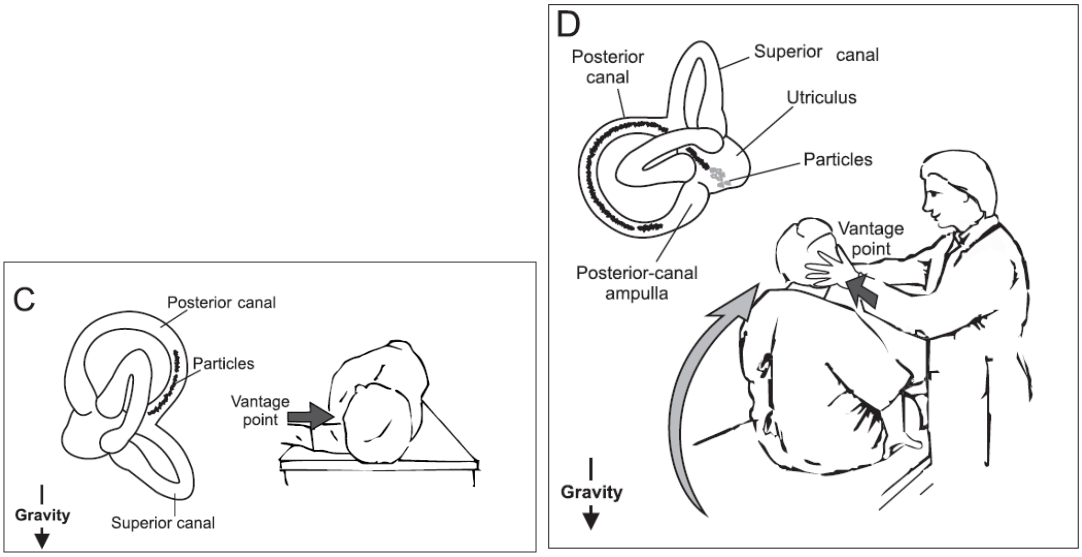


Figure 7. Epley manoeuvre (A-D) for a right sided posterior semi-circular canal BPPV.

Afterwards, I instruct the patients not to do any movements that can trigger the BPPV again such as lying down or any up / down movements for 10 days. I also instruct them not to lie on the affected side or completely flat for 3 days. Some centres instruct their patients to do the manoeuvres at home. Unfortunately, the recurrence rate of BPPV is 15 – 30% per year

Vestibular neuritis

Vestibular neuritis is the second most common cause of peripheral vertigo after BPPV. It is characterized by acute / subacute sustained horizonto-rotatory nystagmus with oscillopsia and imbalance with falls, nausea. The nystagmus is suppressed by visual fixation and there is a pathological HIT. An otherwise normal neurological examination and the ability to stand unassisted without lateropulsion are important in distinguishing vestibular neuritis from a brainstem bleeding or infarction (also see HINTS). There is no hearing loss associated with vestibular neuritis, in contrast to labyrinthitis, which presents the same but is extremely rare. There is sometimes history of upper airway infection and evidence suggests that vestibular neuritis is caused by reactivation of a latent Herpes simplex type I virus.

Although the onset of VN is more predictable, the chronic course of the disease is more variable. In most cases, the initial episode will resolve, and the patient experiences disequilibrium while ambulating and momentary dizziness with rapid head turns lasting up to three months. Clinically this correlates with and impaired HIT. Therefore, as long as a patient has an impaired HIT, he / she will still complain of symptoms of dizziness. Some patients (10%) experience repeated episodes of severe vertigo much like the initial episode. BPPV may follow vestibular neuritis, and the physician should always be on the outlook for this.

Treatment is based on the following:

- Supportive treatment
    - IV fluids
    - Anti-emetic agents
- Vestibular sedatives for 3 days
    - Diazepam or others
- Causative treatment
    - Methylprednisolone 100mg/d and reduced every fourth day by 20 mg
    - Valacyclovir 1gr tds po for 7 days
        - Unfortunately, the administration of antivirals alone or in combination with steroids have no additional benefits
- Improved vestibular compensation

    - There might be some evidence to support the use of high dose betahistidine (Serc®). Unfortunately, the dose of 48 mg tds po is impractical and expensive in South Africa.
- Vestibular exercises
    - Depending on the severity of the patient, this is usually initiated only after 3 days (in mild patients, it can be started earlier)
    - 30 minutes three times per day
    - At first it is a basic VOR exercise
        - Patient focusses on a spot / their own thumb (50cm) and do a horizontal VOR. By implication this means at a slow pace, will keeping focus on the spot / thumb, look left and right.
        - After this, the vertical VOR is introduced on the same principle, and later on any complex head movement while keeping focus.
        - Once the patient is comfortable with this, introduce smooth pursuit, whereby the head is kept still, and the patient tracks his / her own thumb again first horizontally, then vertically, and lastly random patterns.
        - Finally, the fixation suppression test can be done by moving the head and thumbs in the same direction and lastly in opposite directions.

The question always arises whether or not to scan a patient. In a straightforward case there is no need to do a scan, however more often than not one is confronted with an atypical case or a possible cerebellar infarction. These patients should be scanned but also referred to a specialized unit.

Meniere's disease

The diagnosis of Meniere's disease is based on a combination of symptoms namely episodic vertigo lasting minutes to hours, fluctuating low tone hearing loss which becomes permanent, tinnitus, and aural fullness.

The diagnosis can be difficult to make as the classical picture can take years to develop. In contrast to the classical picture, there are also two variants namely:

- Cochlear variant
    - Fluctuating hearing loss, tinnitus and fullness with no vertigo
- Vestibular variant
    - Episodic vertigo with no hearing loss, tinnitus or fullness

There is no single diagnostic test to confirm Meniere's disease, and the diagnosis is made by exclusion. Therefore, an MRI scan is advised on all patients with a possible diagnosis. Meniere's itself can have a variable course, with some patients spontaneously going into remission and other patients developing Meniere's in both ears.

Treatment consists of lifestyle modifications, medical, and surgical options. Lifestyle modifications are best remembered by the acronym **CATS**. This stands for the avoidance of **c**affeine, **a**lcohol, **t**ension and a low **s**alt intake. Usually a maximum of 2 gram/day sodium diet is advised, but in an even distribution. Many other treatments have been published such as allergy and sugar control, diuretics, external ear devices, ventilation tubes, placebo treatments and the psychic of the patient.

An acute attack is treated with sedatives and anti-inflammatory medications, much like vestibular neuritis. Only 10% of patients will require additional treatment to reduce the number of acute attacks. The main factor that needs to be taken into account is the level of hearing loss when deciding on treatment options. Currently the most common would be intra-tympanic steroid injections. Unfortunately, there are minimal treatment options for the hearing loss (except hearing aids) and tinnitus associated with the disease. Because of the difficulty in diagnosis the patients and the various options available, I think it's best to refer these patients.

Bilateral vestibulopathy

As mentioned previously, these patients have minimal vertigo, but presents with postural imbalance, broad based gait, increased gait variability, and oscillopsia. In fact it is the most common cause of postural imbalance in the elderly.

The symptoms will always worsen when the patient needs to rely on the vestibular organs such as closing the eyes, dark environment or walking on uneven ground (typically when going to the bathroom for the elderly patients). On examination there will be an abnormal HIT and Romberg test with eyes closed.

In ¾ of patients one is unable to establish the aetiology, but known causes are antibiotics, Meniere's disease, meningitis, and encephalitis. The treatment consists of balance training (discussed under vestibular neuritis).

Vestibular schwannoma / Acoustic neuroma

This is a rare tumour of the vestibular nerve. In most cases it is sporadic, but there is a genetic component in 5% of cases. It presents with hearing loss (95%), tinnitus (70%), imbalance (50%), and vertigo (20%). In severe cases it can cause brainstem, and cranial nerve V and VII symptoms. Any patient who presents with all three inner ear symptoms, namely hearing loss, vertigo, and tinnitus should rather be referred to a ear, nose and throat surgeon.

Brun's nystagmus is an unusual type of nystagmus which can occur in large cerebellopontine tumours including vestibular schwannoma. It entails slow, large amplitude nystagmus when looking towards the side of the lesion, and quick, small amplitude nystagmus when looking away.

Superior semicircular canal dehiscence syndrome (SSCD)

As the name implies this syndrome is caused by exposure of the superior semi-circular canal to the cerebro-spinal fluid (CSF) surrounding the brain. SSCD is characterized by vertigo induced by noise / pressure, pulsatile tinnitus, autophony, and unilateral hearing loss / fullness. Sometimes a patient gives as history of chronic disequilibrium following minor head trauma.

Examination and special examinations are complex, and it is best to refer these patients.

Vestibular paroxysmia

This is characterized by spontaneous attacks of vertigo lasting seconds to minutes. Attacks occur mostly spontaneously but can but induced by hyperventilation, exercise, and head turn. It is assumed that vascular compression of the 8th nerve is the cause (as in trigeminal neuralgia, hemifacial spasm and superior oblique myokymia). The condition is treated with carbamazepine.

Persistent postural perceptual dizziness (3PD)

PPV is common, accounting for the second most common diagnosis in a tertiary referral balance unit[5]. The patients complain of swaying vertigo, light headedness, and gait unsteadiness that are continually present but fluctuate in severity. Symptoms are often accompanied by anxiety, fear of falling, but without actually falling. This is usually followed by increasing avoidance behaviour especially large open spaces. Typically, the patients have obsessive compulsive disorder (OCD) personalities.

In general, the symptoms are worse during day, improve by taking moderate amounts of alcohol, and exercise. These patients typically have symptoms when standing or walking, but as the balance task gets more difficult, they improve.

A decoupling hypothesis explains the underlying mechanism. Patients are more aware of normal body movements / sway and interpret this as abnormal. Treatment consists of explanation, desensitization, and selective serotonin re-uptake inhibitor (SSRI) will help in a third of cases. In general, three quarters of patients will improve.

Vestibular migraine

Vestibular migraine is the most common cause of central vertigo[6]. Up to 1/3 of all migraine patients have experienced vertigo. It presents with a combination of vertigo and ataxia of stance of gait. The vertigo usually lasts minutes to hours and rarely days. 60 % of patients have auras during vertigo accompanied or followed by head pressure, pain, nausea, and vomiting. In some patients there is no correlation with a headache. Treatment consists of regular migraine medications.

Episodic ataxia type II

Episodic ataxia type II is characterized by vertigo attacks lasting hours and ataxia. It is often provoked by stress or exercise. It is an autosomal dominant disorder and treated with acetazolamide.

Down beat nystagmus (DBN)

Down beat nystagmus is the most common form of central nystagmus. The leading symptoms are postural imbalance and oscillopsia usually worse in the morning and improving during the day. On examination the patient presents with fixation nystagmus with an increase in intensity during lateral and downward gaze and when lying prone with the nose down.

DBN is usually due to bilateral dysfunction of the flocculus of the cerebellum. Its three most common causes are cerebellar atrophy, ischemia, and Arnold-Chiari malformation.

Gaze nystagmus

These patients are unable to sustain eccentric gaze. It is probably the most common form of acquired nystagmus. Known causes are a range of drugs such as anticonvulsants, benzodiazepine, alcohol, and midline cerebellar diseases.

Psychogenic forms of vertigo

The patient describes experiencing frequent postural imbalance or a diffuse feeling of dizziness (a feeling of numbness, light headedness, unsteadiness when walking, a feeling of toppling over) or very rarely rotatory vertigo[9].

Depending on the underlying psychiatric illness, the following additional symptoms can be present: disorders of motivation and concentration, decline in performance, restriction of daily and professional activities, vegetative symptoms (accelerated heartbeat, nausea, sweats, apnoea, fear of suffocating, loss of appetite, weight loss), emotional and mood disorders, sleep disturbances and symptoms of anxiety.

These patients are best referred to the appropriate discipline.

Motion Sickness

Introduction:
Motion sickness traditionally refers to symptoms and signs that occurs during travel by car, at sea, or in the air. Nowadays it also includes concepts such as microgravity (space), large visual environments (cinema), and virtual environments (simulators, video games). Classical symptoms and signs include dizziness, nausea, sleepiness, apathy, cold sweat, increased salivation, pallor, and headache.

Incidence and pathogenesis:
The peak incidence is in children between the ages of 2-12 years, and rarely occurs after the age of 50 years. It's more common in woman, especially during pregnancy and menstruation. There is a strong association with migraine and a genetic polymorphism as well as otolith organ asymmetry has recently been reported.

Since birth visual, vestibular, and proprioceptive information is processed in the brainstem and cerebellum. A blueprint is formed in the cerebellum based on the input from these three systems. As you are able to perform more complex tasks, such as walking or running, the process becomes automatic, and the cerebellum only matches the input of the three systems to a blueprint. At times the cortex will be consciously aware of your balance and gives the okay. This is the reason why motion sickness rarely presents before two years.

Motion sickness develops if there is a mismatch between the visual, vestibular, and proprioceptive input and the blueprint, or if there is no blueprint for that specific stimulation (space flight). Therefore, anybody can develop symptoms or signs depending on the quality and quantity of stimulation, although there is a huge individual variation. It is therefore a normal response to an abnormal situation.

Classical examples of mismatch include:

- Abnormal visual and vestibular stimuli – watching the waves over the side of a ship, looking at the ground through a binocular out of a helicopter
- Visual stimuli without vestibular input – virtual environments, cinema, space
- Vestibular stimuli without visual input – elevator, reading in a car, closed cabin in a ship

Treatment:
The best treatment remains avoidance of the stimulation. If that is not possible the following options are available:

- Behavioural measures:
  - Make a conscious effort to match visual, vestibular and proprioceptive information, for example by looking at the horizon when at sea, sitting at the window when flying or in a train, looking forward out of the window in a car or rather drive. Keep head upright and avoid unnecessary movements when in motion. Other measures include acupuncture (P6 point), avoid tiredness and alcohol.
- Adaptation:
  - It is well known that repeated or continued exposure to motion results in a declining motion sickness response in most individuals. This can be due to a decreased response of the receptor system (adaptation) or decreased neuronal activity (habituation). A classic example is ice skaters being able to perform severe rotation without developing nystagmus.
- Drug treatment:
  - Three neurotransmitters, histamine, acetylcholine, and noradrenaline, play an important role in the neural process of motion sickness. Medication is most effective when taken before exposure.
  - Hyoscine butylbromide (scopolamine) (Buscopan®), an acetylcholine antagonist, is the most effective anti-motion sickness agent. Unfortunately, it can have moderate to severe anticholinergic side effects and also withdrawal symptoms.
  - First generation antihistamines can be very effective, but also has side effects (newer generation antihistamines are ineffective). This group includes diphenhydramine, chlorpheniramine (Allergex®), cinnarizine (Stugeron®), phenothiazine (Vallergan forte®), promethazine (Phenergan®), cyclizine (Valloid®).

- Studies have shown that rizatriptan (Maxalt®) prevented motion sickness symptoms in patients with migraine and vestibular migraine.
- Other medications that have positive effects are phenytoin (Epanutin®), flunarizine (Sibelium®), dextroamphetamine (only used in astronauts), and ginger. When prescribing a medication, one should take the patients age and side effects in consideration.

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