ENT Causes of Vertigo

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Introduction

The principle causes of vertigo related to ear disease are: Benign Paroxysmal Positional Vertigo, Meniere's Disease, Vestibular Neuritis, and Labyrinthitis. Each has its own characteristic symptoms and signs and, with a little patience and practice, it is usually possible to differentiate one from the others by history. Physical examination also helps greatly – especially in the acute phase of the disease provoking vertigo.

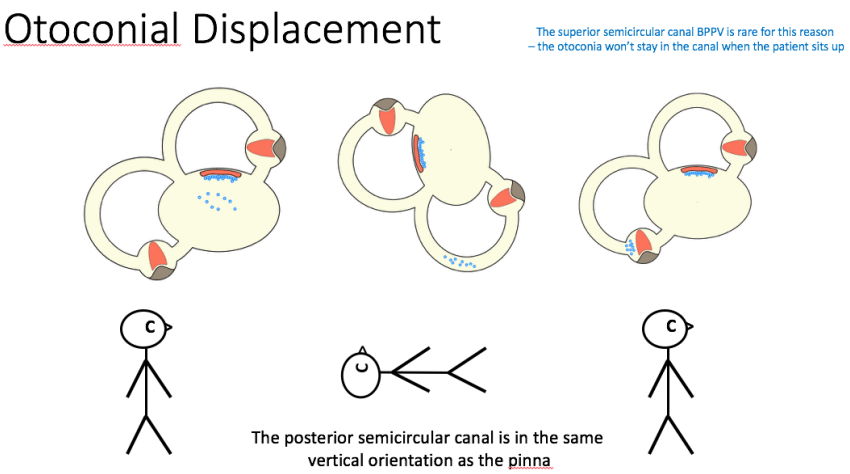
Please be sure to read the section on vestibular physiology before continuing with this tutorial.

BPPV

This is the commonest of the diseases causing vertigo that arise from the inner ear. It is a surprisingly important disease as, not only is it the most common cause of vertigo, it is also the most curable.

Pathophysiology

In BPPV the otoconia attached to the macula of the Utricle become displaced into the endolymph (left image below). While floating in the endolymph of the Utricle they cause no problems but when they become trapped in the semi-circular canals they induce nystagmus and vertigo of short duration following turns of the head or body.



Classically they become lodged in the posterior semi-circular canal. They do this because of the geometry of the canal. When the patient lies down at night the effect of gravity pulls the free floating otoconia into the mouth of the PSCC (middle image above). On sitting up the following

morning gravity pulls them further into the canal until they become lodged behind the cupula in the ampulla (right image above).

In the diagram the otoconia are coded as blue.

The posterior canal is by far the most frequently affected (over 94% of cases of BPPV). You can see that if the otoconia were to drift into the canal of the superior canal they would simply fall out again when the patient sat up.

The posterior canal is wired up to the superior oblique and the inferior rectus muscles of the orbit. Over activity in the canal causes a characteristic nystagmus that is predominantly rotatory.

Symptoms and Signs

BPPV is a disease of otoconia so there is no hearing loss or tinnitus associated with the vertigo. The vertigo lasts for seconds only and is provoked by specific positioning manoeuvres. It is a disease that is usually idiopathic but it also commonly follows head trauma and becomes commoner as patients age. Interestingly it may also follow other causes of vertigo - Meniere's Disease and Vestibular Neuritis.

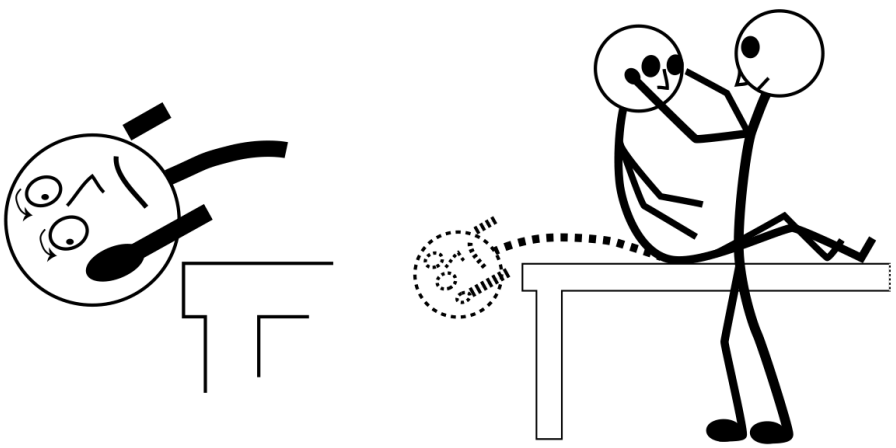
Patients describe their vertigo as brief and provoked by:

1. Rolling over in bed
2. Looking up or down
3. Bending over and straightening up
4. Lying down into bed or in a dentist's chair

These are typical provocations. There is no hearing loss or tinnitus associated with the disease (but beware that these symptoms are common in the general population and may co-exist).

Diagnosis

This is made by history and a particular examination called a Dix-Hallpike (DH) manoeuvre. The DH manoeuvre is sensitive and specific for posterior canal BPPV (the commonest form of the disease).



The DH manoeuvre moves the posterior canal in its own plane through 100 degrees of arc. It is performed with the patient sitting up on a bed with their head turned 45 degrees. The patient is then laid down such that their head extends off the edge of the table slightly.

This movement moves the fluids in the posterior semi-circular canal maximally and provokes both vertigo and nystagmus. The vertigo and nystagmus have particular features and these must be observed for to make an accurate diagnosis.

The nystagmus:

1. Has latency – its starts after a few seconds
2. Is geotrophic and brief – its fast phase is towards the ground and it lasts 20-30 seconds
3. Is tortional – the 12 'o' clock position is directed fast towards the ground
4. Is reversible – on sitting up the nystagmus moves the opposite way (but is hard to see)
5. Is fatigable – lying the patient down again makes the nystagmus less intense

The vertigo:

1. Has latency – it doesn't start immediately
2. Lasts as long as the nystagmus does – 20 – 30 seconds
3. Happens again on sitting up
4. Is fatigable - happens less when the patient lies down again

Course

It is a condition that remits and relapses. Although the spinning only lasts for seconds the patient will describe periods when it is present most days and periods when it is absent. It affects men and women equally. It recurs after successful treatment in 30% of patients.

Treatment

The three options here are:

1. Conservative
2. Particle liberatory manoeuvre or similar
3. Surgery

Conservative management is preferred by some patients as they find the vertigo too distressing to allow active treatment. However, these are the minority and most patients opt for a liberatory manoeuvre of some kind.

Epley Manoeuvre

This is by far the most researched and useful manoeuvre. It is simple to perform and successful in 85% of BPPV sufferers. The Semont manoeuvre can be used and so can Brandt-Daroff exercises. How to perform the Epley is in a separate tutorial.

Meniere's Disease

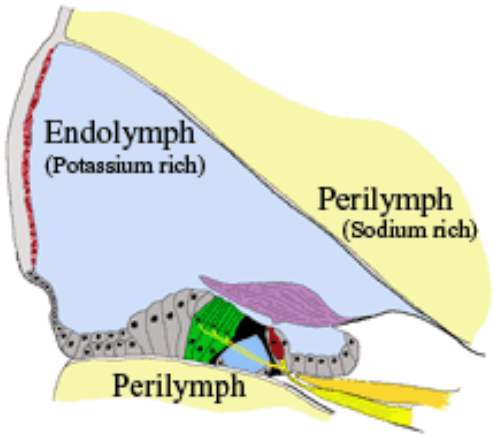
Meniere's disease is a condition that affects the endolymphatic space. As such it provokes symptoms in both the balance portion of the inner ear and in the hearing position. The symptoms are: recurring bouts of vertigo, tinnitus and hearing loss. These settle after some hours but recur at times in the future.

Pathophysiology

One theory of Meniere's disease has it that the function of the endolymphatic sac is impaired and that there is a relative build-up of endolymph within the endolymphatic space. This is sometimes referred to as endolymphatic hydrops.

Remember that the fluids around the endolymphatic space - perilymph - is low in potassium and high in sodium. Remember also that normal function of the delicate neuroepithelia of hearing and balance is dependent on ionic stability within the endolymph.

Now let us focus on the cochlea only. This spiral organ consists of a central endolymphatic space containing the hearing neuroepithelium. This is surrounded by the perilymph space



This diagram is a cross section through one of the turns of the cochlea and show the neuroepithelium bearing endolymphatic space surrounded by perilymph.

The endolymph is high in potassium and this is essential for normal hearing function.

One theory of the events that unravel during an episode of endolymphatic hydrops is that the endolymphatic portion becomes progressively more distended and that finally it ruptures with admixture of endolymph with perilymph. This causes an ionic disequilibrium that causes stimulation and damage to the neuroepithelium of hearing - causing hearing loss and tinnitus.

Later the ionic disequilibrium spreads to include the balance portion of the endolymphatic sac and this, in turn, causes damage and vertigo.

(An alternate theory has it that it is the rupture that signals the end of the Meniere's attack. Much is uncertain in this disease!)

It is true that this is a simplification of the real cause but it does explain the sequence of symptoms in a typical Meniere's attack and is in keeping with histological evidence in patients known to have suffered with Meniere's Disease.

Once the membrane has healed the ionic distributions return to normal and the symptoms regress and finally stop.

Symptoms and Signs

The triad of symptoms that characterize Meniere's Disease are: hearing loss, tinnitus and vertigo. To this triad is usually added 'aural pressure' but it's presence is variable.

In a typical attack the patient will describe a sensation of 'fullness' or 'pressure' in one ear. Sometime later they will notice tinnitus and then hearing loss. These symptoms herald the onset of vertigo.

The vertigo lasts for hours and may be associated with nausea and vomiting. Eventually it eases off and the hearing loss and tinnitus disappear. The attack is over and the patient returns to normal.

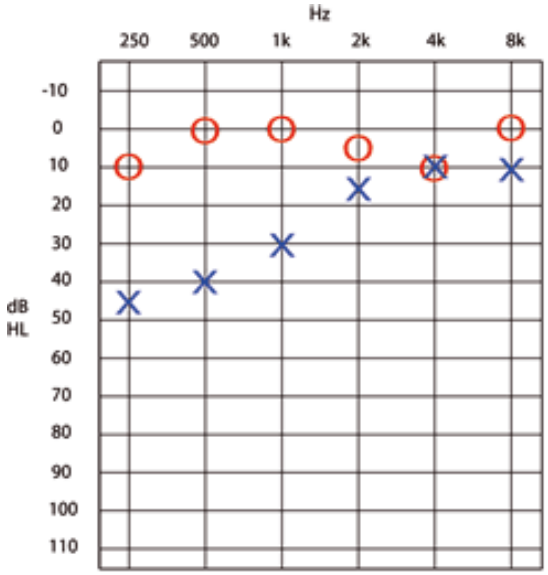
This sequence recurs for years in some. With time the periods between attacks of vertigo are marred by persisting hearing loss and tinnitus in the affected ear. Eventually, as all function in the ear becomes progressively damaged, the attacks stop.

It is unusual to see a patient during an attack of Meniere's disease and so they have very few signs when you examine them. If you were to see someone during the attack you would see typical peripheral nystagmus beating first towards the affected ear and later away from it.

Diagnosis

As always a careful clinical history will give you the diagnosis in most cases of Meniere's Disease. However, there are a few investigations that are required to rule out other causes.

1. Pure Tone Audiogram. This typically shows a fluctuating low frequency hearing loss. Later in the disease the hearing loss becomes permanent and progressive
2. MRI of the posterior cranial fossa and Internal Acoustic Meatus. This is done to rule out acoustic neuroma. A CT scan can be used but is less sensitive
3. FBC, Glucose, and thyroid function are done to exclude systemic diseases



The audiogram shows a low frequency left-sided sensorineural hearing loss.

Were one to plot hearing tests over time one would find that the thresholds improved and worsened at times.

Treatment

This is a very variable disease and, therefore, assessing the success of treatment is difficult. The list below summarises management:

1. Salt restriction
2. Betahistine TID every day as prophylaxis
3. Antiemetic during attacks only
4. Surgery - conservative and destructive

Betahistine (Serc) is the only medication that has any evidence of success in preventing attacks and that evidence is very weak. Antiemetics are used only during attacks.

Surgical options are divided into those that spare hearing function (conservative) and those that destroy it (destructive). The operation that is chosen depends to some extent on how much hearing the patient has left in their ear. If the hearing is good then conservative options are tried:

1. Grommet. Nobody really knows why this works but it does in a large proportion of patients.
2. Methylprednisolone intra-tympanic injection
3. Gentamicin instillation. Here gentamicin is instilled into the middle ear. It passes into the inner ear and destroys balance function while sparing hearing - mostly.
4. Saccus decompression. In this operation, the endolymphatic sac is opened to drain endolymph out.
5. Vestibular nerve section. While this aims to preserve hearing function it is nonetheless a major operation.

Destructive options are limited. The main one is labyrinthectomy. In this operation, the function of the inner ear is totally destroyed by drilling out the inner ear. It is a very successful operation but will destroy all hearing.

Vestibular Neuritis and Labyrinthitis

Background

These two diseases are considered together because the vertigo they provoke is similar. However, they differ in one fundamental way: vestibular neuritis does not cause hearing loss and tinnitus. Labyrinthitis does.

Both of the diseases cause acute vestibular failure. This failure is characterised by a rapid onset of vertigo which may be very severe and associated with nausea, vomiting, pallor, sweatiness and diarrhoea (vegetative symptoms).

Initially the patient will find it difficult to get out of bed or move at all as this will provoke the symptoms. Hence they tend to want to lie still. This phase of the condition lasts for one to two days. After this they are able to move gently and sit in bed but any quick movements will provoke further vertigo and vegetative symptoms.

After a few days they are able to stand and the following few weeks will see a gradual return to normal function without symptoms even on quick movements.

This process of recovery is called compensation and is achieved by the brain by using other senses, adjusting vestibular activity within the vestibular nuclei and a number of other neural activities.

Compensation is the norm, however, there are certain circumstances where it fails to be completed and the patient is left with residual symptoms. Elderly patients, patients on drugs or with other diseases that affect mobility may not ever make a full recovery and even patients who appear to have made a complete recovery may still experience symptoms at a later date if they become ill, start medication or become over fatigued.

Note: the vertigo of labyrinthitis and vestibular neuritis lasts for days or weeks. This is in distinction to Meniere's vertigo that lasts hours or a day and BPPV which lasts seconds.

What is the difference between these two diseases? Labyrinthitis is a disease of the whole labyrinth. Thus, since the labyrinth contains the neuroepithelia of hearing as well as balance, the patient suffers balance and hearing symptoms: vertigo, deafness and tinnitus. Vestibular neuritis is a disease of the vestibular nerve and, therefore, only causes vertigo.

Pathophysiology

You should review the section on vestibular physiology before considering the pathophysiology of these diseases. In particular, you should note the section that details the genesis of nystagmus and the sensation of spinning.

The cause of vestibular neuritis is most likely to be viral and possibly of the Herpes family. However, some may be caused by vascular occlusion - recall that the inner ear is supplied by end arteries and that there is no collateral flow. Whatever the cause it results in a reduction of neural activity in the vestibular nerves on one side and this is what causes the nystagmus and sense of vertigo. The condition is painless and there is no cochlear upset.

Labyrinthitis is also most likely viral in origin although occasionally bacteria can be the cause (for example when a cholesteatoma has eroded into the inner ear or when an acute otitis media has spread inwards). The hearing loss associated with labyrinthitis is permanent and tinnitus often follows but the vertigo will eventually settle.

Symptoms and Signs

Patients with acute vestibular failure as in labyrinthitis and vestibular neuritis are often pale and sweaty and are reluctant to move for fear of worsening their symptoms of nausea and vomiting. They will have nystagmus in addition. This is almost always towards the opposite ear.

Examination early on will reveal this nystagmus in all directions of gaze. Later, as compensation starts to occur, the nystagmus will only be seen when the patient looks in the direction of the fast phase of the nystagmus. If there is associated hearing loss tuning fork tests will suggest that this is of a sensorineural type.

Note that the patient will not be suffering with other signs or symptoms of posterior fossa disease: dysarthria, diplopia, facial weakness, dysphagia, loss of vision, facial or limb paraesthesia, limb weakness. These posterior fossa symptoms and their associated signs point to a circulatory disturbance as the cause and the patient will require emergency brain imaging.

Examination of the ear canal and drum will be normal in all cases unless a middle ear infection or cholesteatoma is the culprit.

Management

The acute management is directed at easing nausea and vomiting. This is done by giving an antiemetic such as prochlorperazine and resting the patient. Hydration is important.

Once the acute phase is over and the patient has begun to mobilize prochlorperazine must be stopped so that full central compensation can occur. The patient is encouraged to gently increase their activities until they return to normal. A hearing aid may be required. Occasionally, where the patient remains disturbed by imbalance, a vestibular rehabilitation program may be offered.

NB. If there is labyrinthitis in the presence of a cholesteatoma or acute middle ear infection the patient should be hospitalised and considered for emergency surgery to remove disease and prevent septic meningitis. Thus examination of the eardrum is mandatory.

Summary of Diseases

	BPPV	Meniere's	Vestibular Neuritis	Labyrinthitis
Vertigo	Yes, seconds	Yes, hours	Yes, days	Yes, days
Hearing loss	No	Yes, hours	No	Yes, permanent
Tinnitus	No	Yes, days	No	Yes, permanent
Trigger	Turning in bed Looking up or down	Stress?	Viral infection	Yes, viral or bacterial ear infection