Peripheral vertigo in general practice

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Balance is maintained in a patient by the complex interpretation by the CNS integrative apparatus (the vestibular nuclei and cerebellum) of information from the labyrinth (balance organ), eyes, skin pressure receptors and muscle and joint proprioceptors. A balance disturbance may occur when there is pathology in any of these systems and there is abnormality in the sensory input into the CNS.

There are many descriptions of balance problems such as unsteadiness, dizziness, giddiness, light headedness, fainting etc. but the principle symptom of labyrinthine pathology is vertigo – a sensation of rotation or spinning, an illusionary movement either of self or of the environment.

A detailed history is therefore crucial to differentiate between the different balance disorders. This paper is limited to vertigo as a result of common pathologies affecting the peripheral vestibular system (labyrinth and vestibular nerves) and how they can be managed in general practice.

Basic anatomy and physiology

The labyrinth consists of three semicircular canals, a utricle and saccule as well as the cochlear organ. This contains two fluid compartments; the endolymph within the membranous labyrinth and the perilymph.

Rotatory head movements set up fluid movements within the canals stimulating hair cells embedded in a jelly-like cupula contained in the ampulla of each canal. Linear forces, e.g. gravity, affect the otoconia (calcium gluconate granules) contained in a gelatinous substance in the maculae of the otolith organs (utricle and saccule) stimulating mechanoreceptor hair cells.

The key to clinical assessment of the vestibular system is the vestibulo-ocular reflex. The vestibular system stabilises the visual system and any pathology disturbing this equilibrium results in nystagmus. In general, the eyes slowly drift towards the relative hypofunctioning labyrinth with an opposite fast phase cortical correction giving the direction of the nystagmus. Vestibular nystagmus (as opposed to central causes) can usually be suppressed by visual fixation, which can be removed using Frenzel glasses (20 diopter lenses preventing image focus).

Benign paroxysmal positional vertigo

This is the most common cause of peripheral vestibular vertigo and is the most easily treated.

Pathogenesis

Intact or degenerate utricular otoconia become displaced to lodge in the cupula in the ampulla of the posterior semicircular canal (cupulolithiasis) or lie free within the canal itself (canalithiasis), which is probably the more common scenario. Abnormal vestibular signals lead to conflicting and confusing central information causing symptoms of vertigo.

The cause is usually idiopathic but it is also seen following head injury, vestibular neuronitis and post-surgery.

History

This is typical and describes a brief episode (usually seconds) of severe vertigo precipitated by specific head positions, e.g. rolling over in bed.

Examination

Producing vertigo and nystagmus with the Dix-Hallpike test[1] is diagnostic. With the patient sitting on a couch, hold their head, eyes open, and lie them flat with the head extended 30° over the edge – to align the posterior semicircular canal in the vertical position. In the same movement, rotate the head 45° to test the undermost ear. Both sides are tested with separate manoeuvres and careful observation of any nystagmus is made. The nys-

tagmus is mixed torsional and vertical with the following characteristics:

I. Usually begins after latency of few seconds
II. Short duration (usually less than one minute)
III. Reverses direction on return to upright position
IV. Fatigable (i.e. gets less with repeated manoeuvres).

Management

- Medication has no value.
- The condition often resolves spontaneously after a few months.
- Labyrinthine fatiguing exercises. The principle is to repeat the manoeuvre causing the vertigo to promote CNS adaptation. (Often useful when the history suggests BPPV but Dix-Hallpike test is negative for nystagmus).
- Canalith repositioning manoeuvre (Epley[2]). The aim is to relocate free-floating debris from the posterior semicircular canal back into the utricle. The patient is placed in the supine position with head rotated 45° to produce the nystagmus as in the Dix-Hallpike test, i.e. the affected ear is dependent. Once the nystagmus settles, the head is rotated slowly through 90° to the opposite side and then the body is turned so the head looks directly downwards for about 15 seconds. The patient is slowly brought into the seated position and should try and remain quiet for 48 hours and sleep with head elevated. (The Sermont manoeuvre[3] requires abrupt head movements and is uncomfortable for patient).
- Surgery – rare: includes singular neurectomy, vestibular neurectomy and posterior semicircular canal occlusion.

Vestibular neuronitis

Pathogensis

This is an infection of the vestibular nerves by a neurotrophic virus, particularly herpes. It is often preceded by an URTI characterised by a sudden onset of severe vertigo, lasting several days before gradual improvement over weeks; the later stages may resemble BPPV (occasionally lasting several months). The disease often occurs in mini-epidemics and vomiting can be severe enough to require hospitalisation for rehydration. As the vestibular nerve is involved there is no hearing loss or tinnitus (i.e. should not be diagnosed as labyrinthitis).

Diagnosis

Requires careful history and examination. In the acute phase, brisk horizontal nystagmus towards the uninvolved ear is present and audiology is normal. Past pointing and the Romberg test are often positive towards the side of the lesion.

Management

- Symptomatic with labyrinthine suppressants;
- Systemic steroids (prednisone) and antivirals (acyclovir) in the acute phase may lessen the severity of symptoms;
- May benefit from vestibular retraining exercises in recovery phase.

Ménière's disease

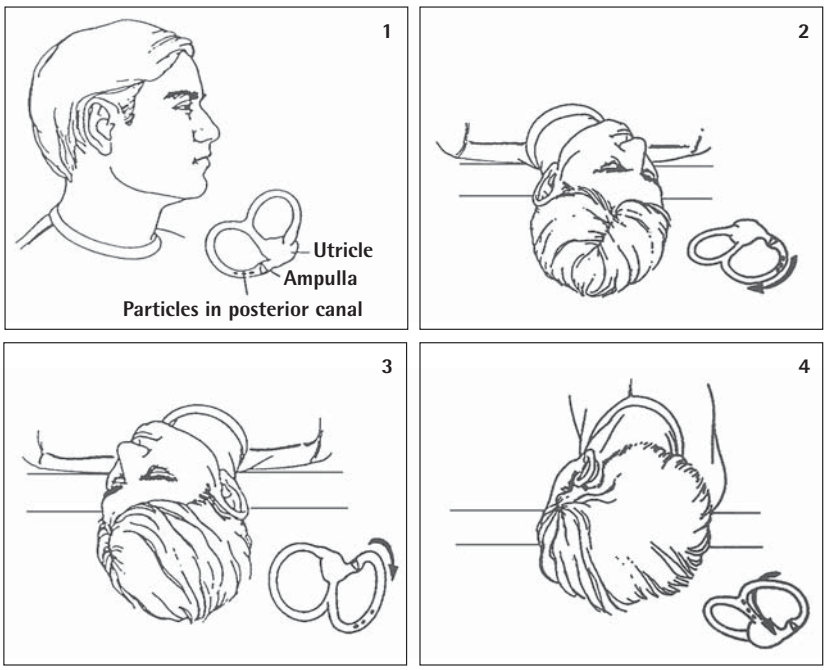
Pathogenesis

Endolymphatic hydrops may occur secondarily to many disease processes, for example, viral labyrinthitis and neuronitis, auto-immune mechanisms, syphilis etc. and is called Ménière's syndrome.

Usually however, no cause is identifiable and the exact pathogenesis is unknown. This primary condition is called Ménière's disease and is characterised by severe vertigo, sensorineural hearing loss and increased tinnitus (typically described as whistling or roaring). There may also be a 'full' sensation in the affected ear.

Ménière's disease usually affects the 40–60 age group, either sex and is occasionally bilateral. 'Attacks' tend to come in clusters and last minutes to several hours before settling down to periods of inactivity. The hearing loss is fluctuating but there is a progressive fall-off with each attack; particularly in the low frequencies. Permanent labyrinthine damage is suspected as being caused by rupture of the membranous layer with decompression of potassium rich endolymph discharging into the peri-

Figure 1. Principle of canalith repositioning using the Epley manoeuvre



lymph – this event is thought to correspond with resolution of the attack.

The clinical course is very variable. There is a range between single sporadic attacks to periods of unrelenting recurrent attacks, from mild inconveniences to complete incapacitation.

Diagnosis

This requires a meticulous history, examination of the ears, tuning fork tests and audiometry. An MRI is often useful to completely exclude retrocochlear pathology (acoustic neuroma). It is important to explain that there is poor understanding of endolymphatic hydrops and that each patient tends to run a different clinical course.

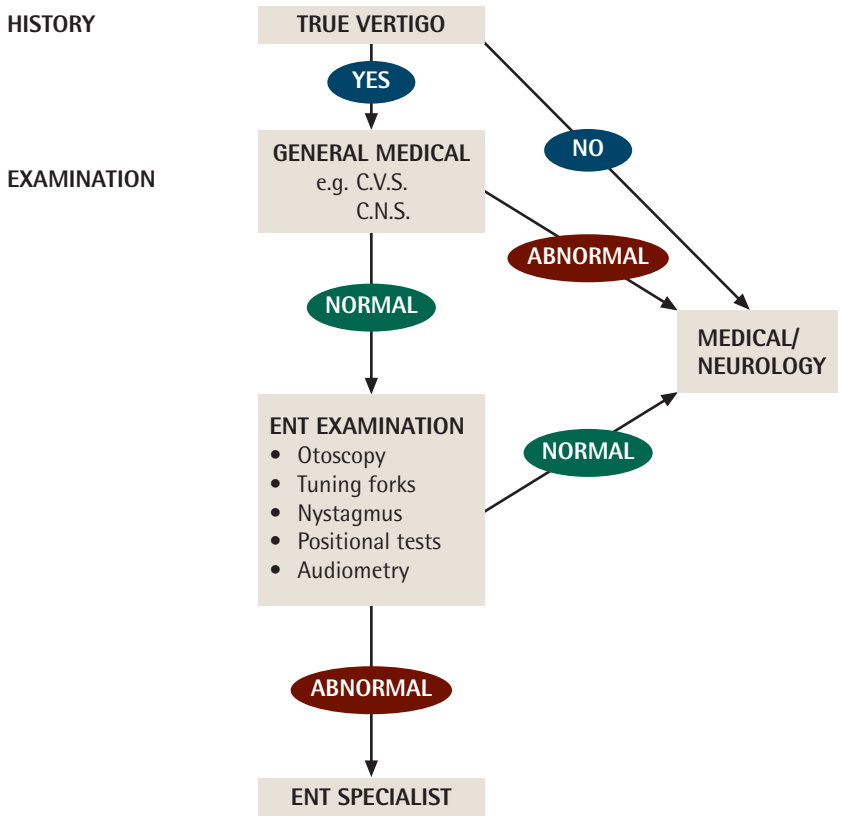
Management

This is individual and primarily aimed at relieving acute attacks and increasing the period of remission.

- Attempts should be made to identify any triggering event, e.g. stress, allergy, foods (chocolate, coffee etc.);
- Salt restriction and diuretics often help as first line medical therapy;
- Betahistine (an oral histamine so avoid with atopy) is used as a vasodilator; some work suggests hydrops is a result of strial ischaemia. Patients learn to titrate the dose to best manage the symptoms;
- Prochlorperazine should be used only for acute attack, not as prophylaxis;
- There is no documented scientific evidence to show that acupuncture, naturopathy, or hypnosis has benefit but anecdotal stories suggest some help in some patients.

Referral to an otologist is important if there is incapacitating vertigo not responding to the above measures. Consideration will be given to grommet insertion, intra-tympanic gentamicin, endolymphatic sac decompression or vestibular neurectomy. These procedures are controversial and subject to evolving philosophies.

Figure 2



C.V.S. Cardiovascular system
C.N.S. Central nervous system

Dizziness

As this paper is designed to help general practitioners with practical management strategies to tackle vertigo, a short section on dizziness is appropriate. Many referrals to ENT Departments get a low priority because of inadequate history, examination and failure to differentiate dizziness, which encompasses a wide range of balance disorders, from true aural vertigo.

Dizziness includes unsteadiness, fainting, near-syncope, lightheadedness and non-specific disequilibrium. It may be physiological, e.g. motion sickness or pathological, incorporating a wide spectrum of medical problems. Examples include CNS pathology, cardiovascular disease, metabolic disorders, vasovagal syncope, cervical arthritis, defective vision, migraine and anxiety/depression causes.

In general terms, patients with chronic dizziness without other otological symptoms are unlikely to have a peripheral vestibular disorder.

Physiological dizziness

This may occur when there is increased stimulation of the vestibular system, e.g. repeatedly turning round and round in circles. Another form occurs when there is decreased sensory input e.g. height dizziness when there is loss of visual clues and increased distance from nearest stationary background.

Motion sickness occurs during travel (land, sea and air) when there

is conflicting sensory stimulation of the CNS from the visual and vestibular systems. Looking inside the car, e.g. reading, leads to visual vestibular imbalance as the visual environment is stationary for the person but the labyrinth registers movement. Tips to manage motion sickness include improving the match between visual and vestibular signals by sitting in the front seat and looking out the window.

Anti-emetics e.g. Avomine, Dramamine, Sea-Legs are effective in controlling motion sickness especially prophylactically in patients prone to this malady.

Space sickness is an interesting variant experienced by 50% of astronauts. Lack of gravity leads to a mismatch between the otoliths, semicircular canals and visual signals leading to symptoms similar to motion sickness. Most adapt within two to three days and some recur with a milder form on returning to earth before vestibular recalibration occurs.

Disequilibrium of ageing

This, to a varying degree, is a common symptom in the elderly. It usually presents as an unsteadiness on walking or moving and is often part of the 'multisystem degenerative' spectrum.

Medical conditions need to be investigated and treated. It is generally inappropriate to send these patients to an ENT specialist. The exception is the patient with an asymmetrical sensorineural hearing loss in which case an MRI scan may be indicated to exclude an acoustic neuroma (more accurately called a vestibular schwannoma).

GP approach to vertigo

The flow chart shown in Figure 2 may prove useful in the practical management of a patient with a balance disorder in general practice.

Key Points

- It is important to distinguish dizziness and unsteadiness from true aural vertigo.
- BPPV (canalithiasis) is best treated with the canalith repositioning manoeuvre of Epley.
- Most important causes of peripheral vertigo:
  - BPPV
  - Vestibular neuronitis
  - Endolymphatic hydrops (Ménière's).
- Patients with chronic dizziness without other otological symptoms are unlikely to have a peripheral vestibular disorder.

Competing interests

None declared.

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3. Semont A, Freyss G, Vitte E. Curing the BPPV with a liberatory maneuver. Adv Otorhinolaryngol. 1988; 42:290-3.

Further reading

Cummings CW et al. Otolaryngology – Head and Neck Surgery: 2nd Ed. Volume 4.
Chapters 141: Pages 2525–2548; 144: Pages 2604–2643; 146: Pages 2652–2683; 181: Pages 3152–3177