Fortnightly Review

Benign positional vertigo: recognition and treatment

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Dizziness is one of the most common complaints in general practice, and yet doctors often find it difficult to establish a firm diagnosis in individual patients. Benign positional vertigo accounts for about a fifth of the referrals to specialised vertigo clinics[1] and is the most commonly missed treatable condition. Fortunately, it can be readily diagnosed by positional testing. Recent insights into its pathophysiology have provided new means of effective treatment that can be applied in clinics.

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Epidemiology

The incidence of benign positional vertigo is conservatively estimated to be 64 per 100 000 population per year.[2] Therefore a general practitioner is likely to see several new patients every year. Age at onset spans from childhood to senescence, but most patients are over 40.[3,4] Women are affected about twice as often as men.[5]

Symptoms and natural course

Most patients report attacks of rotational vertigo of between 10 and 20 seconds that are provoked by turning over in bed, lying down or sitting up, and occasionally by looking up or bending forward. The paroxysmal nature of the vertigo and its precipitating factors, however, may not be volunteered by patients and should be established by specific questioning. Patients may also feel nauseous, but vomiting is rare. They often become apprehensive, and both vertigo and avoidance of movement may limit their activities considerably.

Several disorders affecting the peripheral vestibular system may precede the onset of benign positional vertigo; viral neuronitis and head trauma are the most common, followed by vascular, inflammatory, and surgical damage to the labyrinth. In about 60% of cases no apparent cause can be identified.[4]

The condition tends to resolve spontaneously after several weeks or months. Some patients, however, experience recurrences months or years later. Variants range from a single shortlived episode to decades of suffering with only short remissions.

Positional testing

The diagnosis is confirmed by positional testing as described by Dix and Hallpike (Hallpike manoeuvre; fig 1).[6] The classic nystagmus of benign positional vertigo occurs when the head is reclined and turned to the affected side. It is characterised by its direction and time course.[4,6,7] For example, when the left ear is affected the nystagmus occurs when the head is hanging to the left. The examiner will see alternating quick eye rotations around the line of sight towards the left (undermost) ear and slow rotations in the opposite direction, which constitute a torsional nystagmus (fig 2). Recognition of torsional nystagmus (which is sometimes called rotatory nystagmus) is facilitated by paying attention to scleral vessels and to the radially oriented structures of the iris. Typically an upward beating nystagmus component is superimposed, resulting in a mixed torsional-vertical eye movement. This pattern of eye movements is characteristic and once seen is easily recognised.

Usually nystagmus occurs around 5 seconds but sometimes as late as 30 seconds after positioning. Its duration varies from a few seconds to almost a minute and parallels the sensation of vertigo.

After patients sit up nystagmus may reappear, now

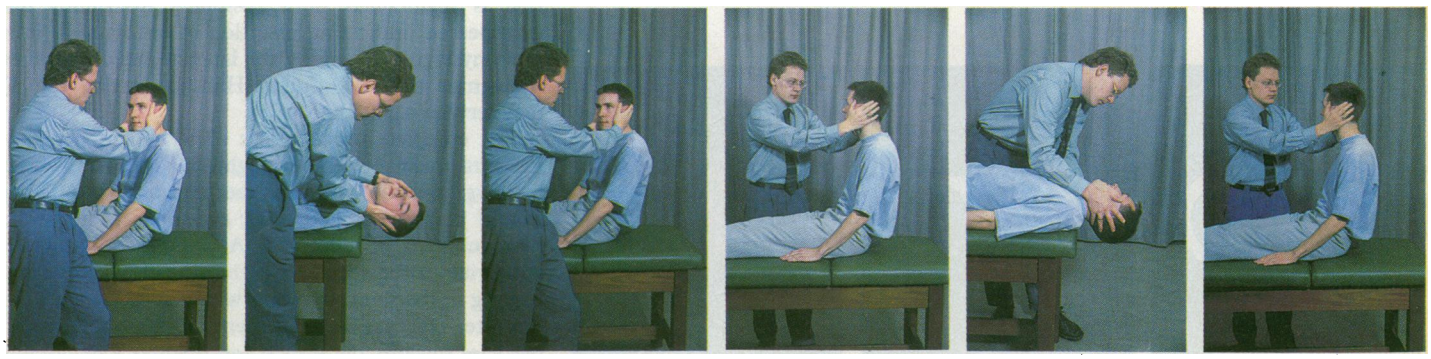


FIG 1—*Hallpike positional manoeuvre for diagnosis of benign positional vertigo. Patients must be warned that transient vertigo may develop in any position. Patients are instructed to keep their eyes open throughout and to stare at examiner's nose. In each position observe eyes closely for up to 30 seconds for development of nystagmus. This manoeuvre can be applied safely to patients with cervical spondylosis if neck is not hyperextended. From left to right: Begin with patient sitting upright on a couch with head turned 45° to left to test left posterior canal. With head in this position, lie patient down rapidly until head is dependent. Return patient to upright position. Approach patient from other side and rotate head to right to test right posterior canal. Lie patient down into opposite head hanging position. Return patient to upright*

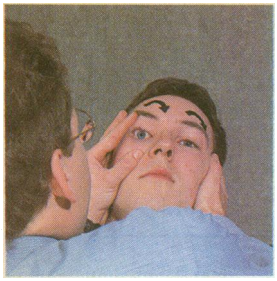


FIG 2—*Patient in left head hanging position. Arrows on brow show direction in which nystagmus will beat. Lids may be retracted for better observation of nystagmus*

beating in the opposite direction; this nystagmus is also accompanied by vertigo. With repeated positioning vertigo and nystagmus diminish and eventually disappear. Although rare, bilateral benign positional vertigo may occur and is characterised by a typical nystagmus in both head hanging positions.

Caloric testing, in which the labyrinth is excited by irrigating the external meatus to provoke vestibular hystagmus, reveals unilateral hypofunction in about one third of patients, mostly on the affected side.[4]

Differential diagnosis

Neurological findings are normal in benign positional vertigo. Occasionally, however, a similar positional vertigo and nystagmus may indicate brain stem or cerebellar disease.[3,7] Central positional nystagmus, however, tends to persist for as long as the provoking position is maintained. It is not as direction specific as benign positional vertigo but may beat in any direction, often downward or horizontally towards the upper ear. For practical purposes, every case of positional vertigo and nystagmus that does not satisfy the criteria for benign positional vertigo should be referred for neurological investigation.

Pathophysiology

Benign positional vertigo results from an inappropriate activation of the ipsilateral posterior semicircular canal of the labyrinth. This can be inferred from both the provocative position and the torsional-vertical nystagmus which reflects the known excitatory connections of the posterior canal with specific eye muscles. Moreover, this condition is abolished by transection of the branch of the vestibular nerve innervating the posterior canal.[8]

The clinical features of benign positional vertigo are consistent with the hypothesis that the posterior canal contains free floating particles that are heavier than the surrounding endolymph.[9-11] After the head is moved in the plane of the canal the material will fall to the undermost portion of the canal, dragging the endolymph in the same direction and leading to deflection of hair cells (fig 3). When the head is moved in the opposite direction the particles will also be shifted in the opposite direction to induce a nystagmus in the reversed direction. The latency of the development of nystagmus may be attributed to the time taken for the particles to be displaced but also to the particles' adherence to the membranous wall of the labyrinth. Adherence may also explain why a brisk head movement is needed to induce the vertigo.

Agglomerated masses within the posterior canal have been observed both at necropsy[12] and during labyrinthine surgery.[13] They are most probably otoconia that may have become displaced from the utricular macula by aging,[14] labyrinthine disease,[15] and head trauma.[16]

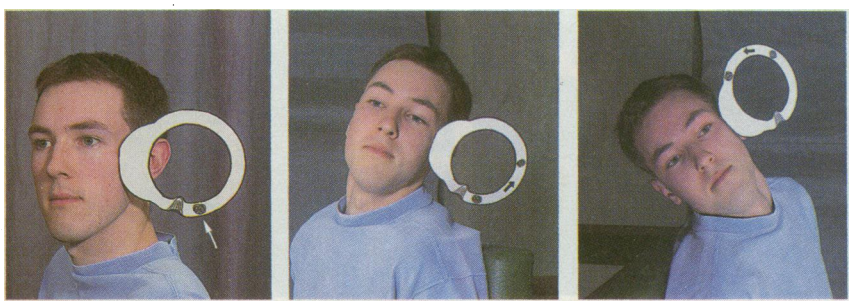


FIG 3—*Canalolithiasis theory of benign positional vertigo. From left to right: Orientation of posterior canal in head upright position with debris shown resting in bottom of canal (arrow). When head is moved to dependent position the debris sinks in the canal under the influence of gravity to cause flow of endolymph. Treatment by positional manoeuvres aims at orienting canal so that debris is directed towards canal opening into utricle*

Summary points

- Benign positional vertigo is one of the commonest causes of dizziness
- It is characterised by short attacks of rotational vertigo that are precipiated by head movements such as looking up, lying down, or turning over in bed
- The diagnosis is confirmed by Hallpike positional testing which shows a characteristic torsional nystagmus when the head is reclined and turned to the affected side
- Benign positional vertigo is probably caused by otoconial debris that is trapped in the posterior semicircular canal and starts to move when head position is changed quickly with respect to gravity. The concurrent flow of endolymph stimulates the hair cells of the affected canal, causing vertigo
- The condition can be treated successfully in most patients by a simple manoeuvre of the head that clears the canal from debris
- Repeated manoeuvres and self guided positional exercises will increase the success rate in those whose condition does not improve after one treatment session

Treatment

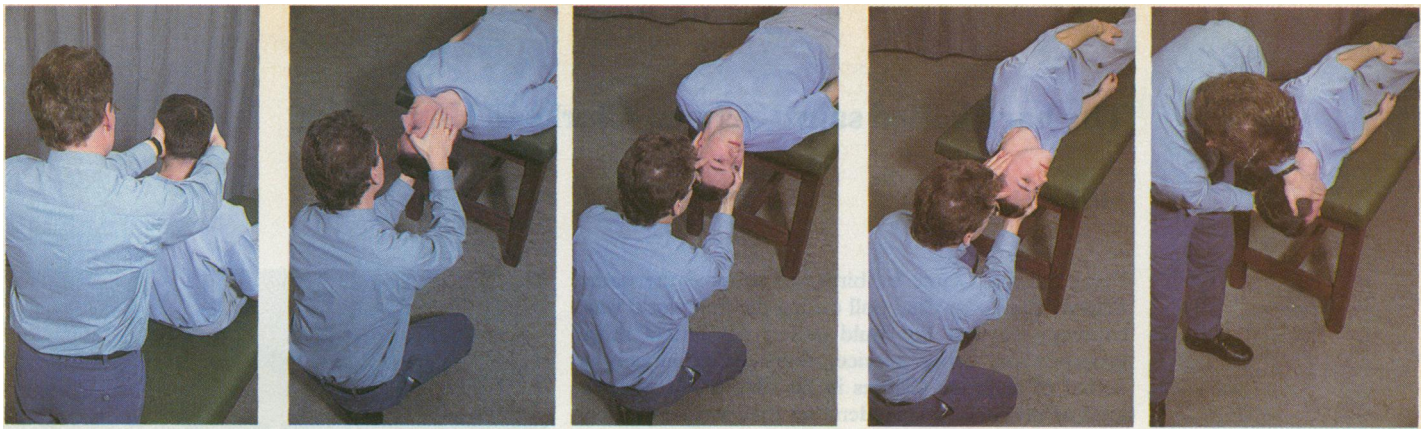
New treatments have been developed with the aim of clearing the posterior semicircular canal from debris on the basis of the canalolithiasis theory described above (fig 3). This approach is unique because it addresses the underlying aetiological mechanism, whereas in other vestibular disorders rehabilitation seeks to promote central compensation of an established vestibular deficit.[17]

Epley's manoeuvre

Epley has introduced a procedure in which the posterior canal is rotated backwards close to its planar orientation, thus directing foreign material out of the canal into the utricle.[10]

The manoeuvre consists of successive head positionings each of about 90° angular displacement. Premedication with a vestibular sedative such as dimenhydrinate or prochlorperazine one hour before treatment is advisable in severely affected patients. Each positioning is performed rapidly and each position is maintained until positional nystagmus has disappeared, indicating cessation of endolymph flow. If no nystagmus is visible the latency and duration of nystagmus observed during Hallpike testing (fig 1) may serve as a guideline.

The complete procedure is illustrated in figure 4. Patients must first be brought to the lateral head hanging position towards the affected side. They should be asked to keep their eyes open to allow the nystagmus to be observed. A positional nystagmus appearing in the second and third head positions and beating in the same direction with respect to the head indicates successive movement of the endolymph particles towards the utricle and predicts a favourable outcome.[18] The manoeuvre should be repeated up to three or four times during one session if nystagmus can still be elicited by Hallpike positioning (fig 1). If positional vertigo persists after the first session the



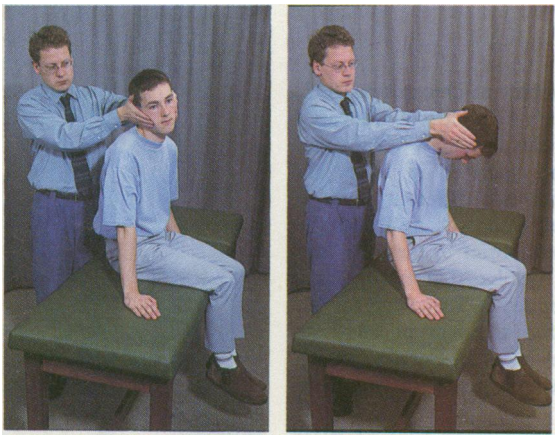


FIG 4—*Epley's manoeuvre. Head movements are guided from behind and each is executed rapidly, within 1 second. Each position is maintained for at least 30 seconds. From left to right: Sit patient upright with head turned 45° to affected side. Lie patient down with head dependent as if performing Hallpike manoeuvre. Rotate head through 90° with face upwards, maintaining dependent position. Ask patient to roll on to side while head is held in this position. Rotate head so that patient is facing obliquely downward, with nose 45° below horizontal. Raise patient to a sitting position while maintaining head rotation. Finally, simultaneously rotate head to central position and move 45° forward*

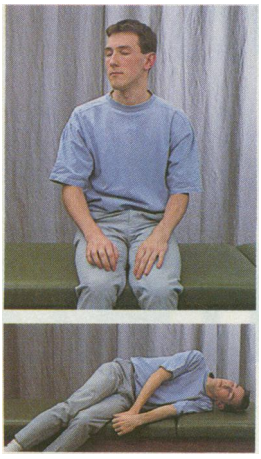


FIG 5—*Self guided positional exercises. One session should include six repetitions to either side, and at least three sessions should be completed daily. Eyes should be closed to reduce vertigo. Sit on a bed with head turned 45° to one side. Quickly lie down to opposite side with head still turned so that region behind ear touches bed. Maintain this and every subsequent position for about 30 seconds. Sit up again. Quickly lie down to other side after turning head 45° towards the other side. Sit up again*

patient should be repeatedly treated at close intervals.

A handheld vibrator placed on the mastoid in each head position may improve the migration of particles by reducing adherence to the membranous walls of the labyrinth.[10] We do not use head vibration routinely in practice, but we have found it helpful in patients with refractory disease.

Usually patients are advised to keep their head fairly upright for 48 hours after treatment[10 18] to prevent particles from re-entering the posterior canal. The usefulness of this measure, however, has not been investigated.

Epley showed effective relief from positional vertigo in 77% of patients after just one treatment session and in another 20% after a second trial a week later.[10] By contrast, spontaneous remission of untreated benign positional vertigo takes an average of 10 weeks after diagnosis.[19] Subsequent trials showed similarly favourable results.[18 20]

Epley's approach was preceded by the liberatory manoeuvre of Semont,[21] which, likewise, has proved valuable.[20] So called Semont's manoeuvre entails rapidly swinging the body from one head lateral position through upright to the contralateral side. Therefore, it is somewhat less easy to perform, especially in elderly people.

As positional manoeuvres are effective in most patients, surgical elimination of posterior canal function[22 23] is restricted to rare cases of longstanding refractory benign positional vertigo. Medical treatment has proved to be ineffective.[24]

Positional exercises

If patients have symptoms after the first treatment they may perform the positional exercises of Brandt and Daroff before returning for a second treatment session.[25] Patients should be provided with an illustrated instruction to ensure proper positioning (fig 5). Again, a vestibular sedative taken one hour in advance may be valuable in severe cases.

Positional testing and therapeutic manoeuvres for benign positional vertigo are safe and easy to perform but should be practised by the physician before they are applied in clinics. Routine adoption will, however, relieve many sufferers of benign positional vertigo.

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