

PHYSICAL SIGNS

Vestibular reflexes and positional manoeuvres

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Dizziness and vertigo are some of the more frequently encountered symptoms in neurology clinics. In turn, one of the most common causes of vertigo is benign paroxysmal positional vertigo (BPPV), accounting for a quarter of all patients with dizziness and vertigo. Reviewing the value of the positional manoeuvres available is relevant, particularly in the light of the efficient treatments available for BPPV. In this article I will deal with positional manoeuvres first, and then with how vestibulo-ocular reflexes (VOR) can be tested in the clinic. I will not discuss VOR suppression assessment.

POSITIONAL MANOEUVRES

Positional manoeuvres must be conducted in all patients with episodic vertigo or dizziness, particularly if provoked by head-neck movements or positions. Typical symptoms are vertigo on looking up, on bending over and sitting upright, vertigo on lying down and vertigo on turning over in bed. Any of these symptoms in isolation, but even more if they are in association, should prompt the diagnosis of positional vertigo. If the vertigo is intense but brief—that is, less than a minute—the likelihood of the positional vertigo being actual benign paroxysmal positional vertigo (BPPV) is high. If a patient is dizzy on standing up from the sitting position the likelihood of the symptoms being primarily vestibular in origin is less. The latter challenges vascular orthostatic mechanisms but there is no reorientation of the head with respect to gravity nor any significant head angular movement. Therefore the amount of net vestibular stimulation is small. Patients with vertigo or dizziness, which is not clearly positional, episodic, or paroxysmal, may be exempted from the positional manoeuvre although I do not recommend it. Many patients with BPPV describe what initially sounds like vertigo lasting for several days and this creates the erroneous impression of a vestibular neuritis. Careful interrogation often reveals that what they describe is multiple brief attacks of BPPV, sometimes complicated by nausea.

The purpose of conducting a positional manoeuvre is to try to elicit vertigo and nystagmus. The patient should be made well aware of this, explaining that despite modern technology this is the only way to make a diagnosis of a perfectly treatable condition such as BPPV. On doing the positional manoeuvre, and regardless of whether there is vertigo or not, the examiner should carefully observe the patient's eyes. To fulfil this purpose the following practical measures can be taken: (1) Patients should be warned before hand

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that, even if they feel vertiginous, they should look straight ahead at one point on the examiner's face (that is, the nose, bridge of the nose). If the eyes are not in primary gaze or wandering around the description of the observed nystagmus will be more difficult. (2) At least one eye of the patient can be easily helped to stay wide open by one of the free hands of the examiner, as shown in figure 1. (3) Keep the patient in the head down position for a few seconds. Some patients with BPPV show extremely long latencies, occasionally up to 20 or 30 seconds. So if the suspicion of BPPV is strong you should wait this long. In most cases 10–20 seconds is sufficient, indeed most BPPVs have latencies of about 5–6 seconds.

There are no excuses for not conducting a positional manoeuvre. However, two of the most commonly heard are “the couch in my room is placed awkwardly to do a Hallpike, I just cannot get the patient's head to hang off the couch” and “we have not got Frenzel's glasses in our clinic”. Wrong. A positional manoeuvre can be done with a couch in any position and Frenzel's glasses are definitely not required for any positional nystagmus, BPPV included. Figure 1 shows the conventional Hallpike manoeuvre with the head in the classic hanging position and a recommended alternative to the procedure when, for instance, the couch is placed between walls or cupboards. Examination of these pictures show that the final head position achieved is very similar. As to the use of Frenzel's glasses it must be remembered that all the classic descriptions of BPPV by Dix and Hallpike¹ were carried out without Frenzel's glasses. We do not use them for positional nystagmus. Two distinguished groups in Germany show a very similar diagnostic rate, one without (T Lempert, personal communication) and the other one with Frenzel's glasses.² Presumably, the rationale for using Frenzel's glasses is that when the patient is able to fixate their nystagmus can be potentially suppressed. However, the vast majority of patients either have or do not have BPPV. If they have BPPV the nystagmus is extremely strong and so the patient is unable to suppress it with visual fixation. Furthermore, a critical component in the most common form of BPPV, posterior canal BPPV, is torsional (rotatory) nystagmus. The ability to suppress torsional nystagmus by visual fixation is less than nystagmus in other orientations.³ It is theoretically possible, however, that Frenzel's glasses could aid the diagnosis of a patient with a mild form of BPPV.

What can we expect to see during a Hallpike or variant Hallpike manoeuvre. The most common

Abbreviations: BPPV, benign paroxysmal positional vertigo; VOR, vestibulo-ocular reflex

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Figure 1 Examination technique for a right-sided, posterior canal BPPV. (A) The Hallpike manoeuvre, in which the head (face) is turned about 45° to the right. This is done so that the rotation, down to the head hanging position, takes place in the plane of the right posterior canal. Note how the examiner's right hand can contribute to open the patient's eye widely. The patient's knees can be bent, as shown, to reduce strain on the lower back. (B) "Variant" Hallpike manoeuvre, starting from a sitting position. Note that the head reaches a similar final position despite not actually being off the couch; this manoeuvre is useful when the couch is placed in between walls or cupboards.

form of positional nystagmus is the one attributable to BPPV of the posterior canal. During a left ear down head hanging position one triggers a left posterior canal BPPV. The spatial orientation of the canal and the connections between the canals and the ocular motor system determines the characteristic nystagmus. The main component of the nystagmus is a torsional or rotatory nystagmus (these terms are indistinct) beating clockwise from the observer point of view. This means that the upper pole of the patient's eye will beat towards the patient's left shoulder. Technically speaking it is left beating torsional nystagmus, as expected from activation of the left posterior canal. A secondary up beating component of nystagmus is often observed which is synchronous with the torsional beat. The nystagmus is often accompanied by intense vertigo and the patients' attempt to close the eyes or to sit up, for which the doctor will have hopefully instructed them in advance to resist. The characteristics of posterior canal BPPV are the presence of latency, as discussed in the preceding paragraphs, adaptation, and fatigability. Adaptation refers to the decline and eventual disappearance of the nystagmus within a minute or so, usually less. Fatigability refers to the fact that on repeated positioning, the nystagmus and the vertigo are less with time. The patient can be reassured that usually the intensity of the symptoms will be less as we repeat the manoeuvre.

The positional manoeuvre should be conducted on both sides, particularly if no nystagmus is observed on the first side. On confirmation of the diagnosis, these days many specialists proceed directly to the treatment with particle repositioning manoeuvres like the Epley or Semont manoeuvre. Partly because of fears that a new Hallpike after an Epley or Semont manoeuvre could undo the benefits of the treatment, many doctors do not do the Hallpike manoeuvre on the other side if BPPV has been diagnosed and treated on one side. If all symptoms resolve in the first session that is the end of the story. If symptoms persist the Hallpike manoeuvre will have to be carried out on a separate session on both sides.

Several other types of nystagmus can be observed during the Hallpike manoeuvre, which are not posterior canal BPPV. In principle, a purely vertical nystagmus, be it up beating or down beating nystagmus, should be considered of central ori-

gin. In a recent review of our 50 consecutive cases of positionally induced down beating nystagmus, three quarters of patients had separate evidence of neurological disease.⁴ In the remaining quarter of patients the most probable diagnosis was anterior canal BPPV. Ideally, in anterior canal BPPV, you should see a torsional component added to the positional down beating nystagmus. Of interest, the right/left specificity to trigger anterior canal BPPV seems less than for posterior canal BPPV. Although the presence of a positional down beating nystagmus has been known to occur with cerebellar lesions for a long time, a few observations of clinical interest have arisen from our recent review. Firstly, many patients with positional down beating nystagmus had multiple system atrophy. Patients with Parkinson's disease or progressive supranuclear palsy do not. This indicates that the identification of a positional downbeat nystagmus, can be of value in the differential diagnosis of multiple system atrophy. A second observation was in contrast with our expectations. No patient with positional down beating nystagmus (all of whom had no down beat nystagmus in the upright position), showed an Arnold Chiari malformation. Although the Arnold Chiari malformation is one of the most common causes of down beating nystagmus in the upright position, it is not a common cause of down beating nystagmus selectively induced by positioning.

A purely horizontal nystagmus can also be observed during a Hallpike manoeuvre. In a patient with a recent history of peripheral type positional vertigo, the diagnosis is almost certainly horizontal canal BPPV. As in other canals BPPVs, it is usually attributable to canalolithiasis (free floating particles in the lumen of the canal) and occasionally to cupulolithiasis (particles become adhesive to the cupula). Horizontal canal BPPV produces intense nystagmus and vertigo and this happens when the head is turned both to the side of the lesion and to the opposite direction. The nystagmus usually beats in the direction of the face turn—that is, right ear down right beating horizontal nystagmus. The intensity of the nystagmus is stronger in the direction of the abnormal side—that is, stronger right beating nystagmus with the right ear down suggests right horizontal canal BPPV. Horizontal canal BPPV is a much more self limiting condition than other canal BPPV. I



Figure 2 Techniques for assessing the VOR in the clinic. The general principle is that the examiner moves the patient's head, with two hands (A) and (B) or with one hand as in (C). (A) Dynamic visual acuity. Two measurements of visual acuity are taken, one with the head stationary and one with the head oscillating at about 1 Hz. Reductions in visual acuity of two or more lines indicate bilateral abnormality of the VOR. (B) The examiner observes the patient's eye movements during either slow (doll's head) or fast, sudden head turns (head impulse test). The presence of catch up saccades towards a prescribed target during a head turn indicates severe failure of the horizontal semicircular canal in the direction of the head turn. (C) The head is turned during ophthalmoscopy while the viewing eye maintains fixation on a distant object. If the optic disc remains absolutely steady the VOR is functioning well. If the disc appears jerky during head turns in one direction, the ipsilateral horizontal semicircular canal is probably significantly impaired.

have seen a patient's horizontal BPPV disappear spontaneously between Friday and Monday although usually it takes from a few days to a month to disappear spontaneously. Patient waiting lists longer than a month is the most probable reason why specialists in some countries, like the UK, do not see many cases of horizontal canal BPPV. Lying down on the healthy side for many hours is an effective treatment for horizontal canal BPPV as the debris leave the canal in such positions.³ It is possible that some patients whose symptoms resolved within a few days unwittingly adopted such posture in bed and thus treated themselves.

When a patient gives a positive typical history of BPPV and no nystagmus can be elicited in the Hallpike or similar manoeuvres for posterior canal BPPV, other manoeuvres should be investigated. For horizontal canal BPPV the optimal plane of rotation should be in the plane of the canal. This can be achieved with the head end of the couch raised 20° to 30° above the horizontal, followed by a full head turn about the longitudinal axis of the body in each direction. It can be estimated that the Hallpike (that is, posterior canal) manoeuvre may not identify 20% of horizontal canal BPPVs (mean results from an email survey—respondents were R Baloh, P Bertholon, J Furman, T Lempert, L Lopez, M Strupp). In other words, one in five patients with horizontal canal BPPV will require the specific horizontal canal manoeuvre for diagnosis. For anterior canal BPPV the left head hanging Hallpike position should provoke a right anterior canal BPPV and vice versa. This is attributable to the co-planar orientation of the left posterior canal with the right anterior canal and vice versa. However, a crucial factor in provoking anterior canal BPPV seems to be placing the head as low down as possible and this may be best achieved by taking the patient in one movement from the sitting upright position to the straight back, head hanging position.⁴ That right/left specificity for anterior canal BPPV is not so critical has been confirmed by a recent surgically treated case of anterior canal BPPV.⁶

In summary, many positional manoeuvres exist and BPPV can come from any of the three semi-circular canals. Bilateral cases also exist. At least the Hallpike manoeuvre, or variant, for posterior canal BPPV must be done in all patients with

head-neck movement or position triggered vertigo. If negative, the horizontal and straight back manoeuvre should be applied but the hit rate may be low. A typical nystagmus for posterior canal BPPV, with normal CNS examination, needs no imaging procedures. Successive failures to treatment should prompt imaging. Even in the absence of nystagmus, more than 80% of patients with a typical history improve with particle repositioning manoeuvres for posterior canal BPPV.⁷ Observation of any other positional nystagmus, particularly if it lasts for longer than a month, should undergo detailed MRI investigation of the posterior fossa.

Vestibular ocular reflexes

The vestibulo-ocular reflex (VOR) serves a very specific function, to stabilise gaze in space during head movements. The VOR is what allows us to see clearly when we walk, turn our heads, or look out of the window while in a car. It does so by generating slow phase eye movements of an almost equal velocity, but opposite in direction, to head movement. This is achieved by a three neurone, short latency reflex: a Scarpa ganglion neurone, a vestibular nucleus neurone, and an oculomotor nuclear neurone (III, IV, or VI).

For many years clinicians relied almost exclusively on laboratory examination of the VOR, namely caloric or rotational tests. In the past 20 or 30 years, thanks to our increased understanding of the physiological basis of the vestibulo-ocular system and our improved skills in observing eye movements, we can now assess the VOR in the clinic room. Unilateral and bilateral loss of vestibular function can, in many cases, be identified clinically. As a rule of thumb, clinical detection of vestibular damage will be easier the more severe and the more acute the lesion is. In contrast, a unilateral, partial, longstanding, peripheral vestibular disorder cannot be detected clinically with any degree of confidence and a caloric test is required. The clinical manoeuvres available rely either on (1) a slow doll's head manoeuvre, assessed by (a) direct observation of the eyes, (b) measurements of visual acuity, or (c) ophthalmoscopy, or (2) a fast version of the doll's head, the head impulse test (fig 2).

The doll's head (doll's eye) manoeuvre

Observation of the eyes

Neurologists are familiar with this manoeuvre, in the vertical plane, as used for confirmation of the supranuclear nature of a gaze palsy in the syndrome of Steele, Richardson and Olszewski. Here we discuss how the manoeuvre is used in vestibular disease.

This manoeuvre requires the patient sitting in front of the examiner, close enough to be able to observe the patient's eyes carefully. The patient is instructed to continuously fixate a feature of the examiner face (nose or bridge of nose), or an object across the room. The examiner then oscillates the patient's head from side to side, at a frequency of about 0.5 to 1 Hz. In the absence of VOR, the patient's eye movements will not be smooth but will be interrupted by "catch up" saccades towards the fixation target. This occurs because, at frequencies of 0.5 to 1 Hz, a head oscillation of $\pm 30^\circ$ will generate peak head velocities of between 94–188 degrees per second; too high for the pursuit or cervico-ocular reflex to compensate.⁹ As slow phase eye movements are unable to keep up with the target, catch up saccades are put in—and these can be easily observed if you are close enough. In patients with bilateral, complete or severe (>90%) loss of vestibular function (for example, gentamicin ototoxicity, meningitis, or idiopathically¹⁰), the manoeuvre is positive. Typically these patients complain of unsteadiness in the dark and oscillopsia while moving fast, for example, walking, running, or while in a car on a bumpy road.

Dynamic visual acuity

A similar movement of the head can be used to investigate the VOR while reading a visual acuity chart. A baseline visual acuity measurement is noted, binocularly; for the sake of argument 6/6. Standing behind the patient, the examiner oscillates the patient's head at about 1 Hz while a new visual acuity measurement is taken. A normal subject's visual acuity does not change with respect to the baseline reading or it can deteriorate by one line, say to 6/9. A loss of two lines in visual acuity must be treated as suspicious and when three lines or more are lost the results are frankly abnormal—that is, indicative that the patient's VOR is grossly reduced. False positives may occur if the patient has a spontaneous nystagmus, for example, a down beating nystagmus, which is exacerbated by head movement and/or lateral gaze. However, this nystagmus should have been observed during conventional examination of the eye movements. False negatives occur if the patient himself oscillates the head, as they often "cleverly" stop the head movement and get a snap of the visual acuity chart. In controlled conditions, a correlation between the degree of caloric response loss and dynamic visual acuity decrement was found.¹¹

Although examination of the patient's eye during the doll's head manoeuvre can in some cases detect a unilateral vestibular lesion, this is probably detected more efficiently with the head impulse test (see below). The reading or dynamic visual acuity test, as used clinically, cannot be applied to unilateral lesions.

Ophthalmoscopy

A procedure first proposed by Zee¹² promotes the use of ophthalmoscopy for the assessment of vestibular disorders. The ophthalmoscope is a powerful magnifying glass so that nystagmus of small amplitude can be detected easily. The important thing to remember is that the beat direction of any nystagmus is inverted during ophthalmoscopy (we are looking at the back of the eye). Vestibular nystagmus of peripheral (labyrinthine) origin increases with removal of fixation so it would be useful to do ophthalmoscopy in a completely darkened room. As this is rarely possible in a clinic an alternative is to ask the patient to cover his own, non-examined eye. The patient should be instructed to use a "hol-

low hand" and to cover, not touch, the eye. The patient is effectively devoid of visual fixation; the examined eye is dazzled by the ophthalmoscope and the self covered eye is in darkness.

The ophthalmoscope is not only useful for detecting nystagmus of small amplitude but also specific conditions such as micro-saccadic flutter, a benign condition,¹³ and pendular pseudo-nystagmus, which emerges in patients with absent vestibular function and head tremor.¹⁴ In the latter, patients report oscillopsia or blurred vision at rest, aggravated by stress or physical activity. Although conventional examination of the eye movements is normal, ophthalmoscopy reveals an oscillating retina. This is because the head tremor, usually at about 4 Hz or 5 Hz, cannot be compensated at eye level because of the lack of the VOR. Immobilisation of the head by an assistant produces immediate improvement of the visual symptom and stabilisation of the fundus.

A potentially confusing clinical condition worth bearing in mind is latent nystagmus.¹⁵ Latent nystagmus is a congenital disorder, often associated with strabismus, discovered during the alternate covering test or, unwittingly, during ophthalmoscopy. Covering either eye elicits horizontal nystagmus in the opposite direction, and vice versa, and this can be very disorienting for the ophthalmoscopist.

In addition to the static observation of the fundus, the VOR can be assessed with an ophthalmoscope in the following way.¹² The patient looks at an object across the room with one eye while the opposite optic disc is examined. The examiner turns the patient's head (nose) towards the right ear to probe the right horizontal semi-circular canal. If this canal is healthy, the disk will remain perfectly stable under the ophthalmoscope. If the right semi-circular canal is defective, the disk will appear as jerky (because of the catch up saccades discussed above). In bilateral disorders, naturally, the jerkiness is observed with head turns in both directions. The main problem with this technique is uncooperative patients who change targets. Similarly, in patients with square wave jerks, the observer may find it difficult to know if the optic disk jerkiness is attributable to the square wave jerks or to a defective VOR.

Head impulse test

The value of VOR examination as described in the preceding sections is limited by the fact that they are all carried out with relatively low head velocities. The VOR is only really irreplaceable at high velocities and accelerations of the head. Neither pursuit, optokinetic nor cervical mechanisms can fully take over from the VOR during brisk head movements. For this reason, Halmagyi and Curthoys have popularised the examination of the doll's head manoeuvre with discrete, sudden, brisk, and unpredictable head turns, so that the VOR deficit is more apparent.⁸ An editorial published in this journal¹⁶ provides details. In essence the patient is seated with the examiner at close range so that the eyes can be observed carefully. The patient is instructed to fixate a target, preferably across the room, for example, the video camera lens if you wish to record the findings, or on the examiner's face. The head is turned in discrete steps of ± 10 – 15° across the midline, briskly, by the examiner thus producing head velocities of several hundred degrees per second. A fast right head turn will make a patient with right sided vestibular loss introduce one or more catch up saccade towards the target—that is, towards the left. These catch up saccades can be easily identified by a trained observer. The test is clearly useful for identifying acute unilateral peripheral vestibular deficits, for instance in patients with vestibular neuritis (neuronitis, labyrinthitis). In patients with chronic, compensated, incomplete unilateral lesions the test is often negative or inconclusive. A study comparing caloric testing compared with the head impulse test shows that the overall sensitivity of the head impulse test is 34%.¹⁷ This means that, when taking into account all patients

with canal paresis, the sensitivity is low. However, specificity is high (100%) and all patients with a canal paresis $\geq 87\%$ had a positive head impulse test.¹⁷ You should be conversant with the oculo-motor clinical examination and, like any other clinical manoeuvre, be shown how to do it—at least once. In some patients with spontaneous nystagmus or square wave jerks the test maybe inconclusive (“was that the little saccade I am looking for, or just another square wave jerk?”). According to Halmagyi, the test can be used in the accident and emergency room for the differential between vestibular neuritis and acute cerebellar infarct. Patients with acute vestibular neuritis have a positive head impulse test whereas cerebellar patients do not. This is a plausible claim but to my knowledge it has not been formally validated. As with all other clinical procedures for VOR examination, the head impulse test complements but does not fully replace caloric testing.

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