NEURO-VESTIBULAR EXAMINATION

David E. Newman-Toker, MD, PhD
The Johns Hopkins University School of Medicine
Baltimore, MD

Syllabus Contents

Head Impulse Test (a.k.a. VOR Gain)

What – Overview of the Test

It has been known for over a century that the eighth cranial nerve conveys balance information to the brain, but until the late 1980s, there was no clinical method available to effectively test for vestibular hypofunction in awake patients at the bedside. For many years, comatose patients had been examined using oculocephalic maneuvers (commonly referred to as "doll's eye" maneuvers) or caloric testing to interrogate the vestibular system in coarse fashion at the bedside, without the need for special equipment. However, this was used principally to determine whether the pons was grossly intact or irrevocably damaged. Similar non-quantitative caloric testing could also be performed in awake patients with suspected vestibular disease, but an "intact" response was generally the norm. Partial or relative vestibular hypofunction could only be measured in a laboratory-type setting, using quantitative electro-oculography (EOG) as part of the caloric electronystagmogram (ENG). The doll's eye maneuver in awake patients generally produced no useful results that correlated with quantitative caloric weakness. *In retrospect, the principal problem was that the test was not "challenging" enough for the vestibular system*.

In 1988, the horizontal head impulse test (h-HIT) of vestibulo-ocular reflex (VOR) function was described by Halmagyi and Curthoys as a bedside test for peripheral vestibular disease.[1] This high-acceleration maneuver (which we might think of as "doll's eyes on steroids") proved to be capable of identifying those with vestibular failure in a way that the classic oculocephalic maneuvers could not. Moreover, it became clear that the test was able to interrogate each ear separately, likely due to a quirk of normal vestibular physiology where excitatory responses carry more "weight" than inhibitory ones. This directional asymmetry in vestibular responses of the healthy labyrinth is known to neuro-otologists as *Ewald's second law*. Since its original description, an abnormal h-HIT has repeatedly been shown to correlate with ipsilateral vestibular hypofunction, and, more specifically, to correlate with de-afferentation of the inputs from the ipsilateral horizontal semicircular canal.[1-4]

Although not described in detail here, clinicians should be aware that the h-HIT has since been adapted for use to interrogate function of the two vertical semicircular canals (anterior [a-HIT] and posterior [p-HIT]).[3] A similar test using a linear head motion (the "head heave" test) has also been developed to interrogate function of the linear-acceleration-detecting otolith organs (utricle and saccule).[5]

How – Method of the Test

The horizontal head impulse test (h-HIT) of vestibulo-ocular reflex (VOR) function, as originally described, is a rapid, passive head rotation from a center to lateral (10-20 degrees) position as a subject fixates at a central target (e.g., the examiner's nose). Since the response is linked to high *acceleration* rather than high velocity or amplitude of the head rotation, the technique is properly conducted with a quick "flick" of the examiner's wrists over a very short amplitude. The examiner must warn the patient that the head will be rotated very rapidly, and it is generally advised to rotate the head slowly several times before conducting the high-acceleration maneuver.

The *normal* VOR response to a rapid, passive head rotation as a subject fixates on a central target is an equal and opposite eye movement that keeps the eyes stationary in space (*negative* h-HIT). This is sometimes referred to as a "VOR gain" equal to 1.0 (ratio of head rotation to eye rotation 1:1). An *abnormal* response occurs when the head is rapidly rotated toward the side of a vestibular lesion. The loss of vestibular afferent input results in the inability to maintain fixation during the head rotation ("gain" < 1.0), requiring a corrective gaze shift (re-fixation saccade) once the head stops moving to re-acquire visual fixation on the central target (*positive* h-HIT). A large-amplitude re-fixation saccade indicates a very low VOR gain and substantial vestibular hypofunction.

A common adaptation of the h-HIT is to displace the head laterally first, then rotate the head back to the center position. Although not originally validated with a lateral to center rotation, there is no compelling reason to believe that the results should differ appreciably, since the VOR response is largely independent of the starting position of the head on the neck. Some examiners find the maneuver easier to conduct using this centripetal head motion, and the results are often easier to interpret (since the globes end in the primary position in the orbit, rather than a somewhat laterally-displaced position). Using a centripetal head rotation also reduces any theoretical risk of vertebral artery injury with neck over-rotation by an overzealous, inexperienced examiner.

Note that for the test to work effectively, the patient must be able to fixate steadily on a visual target. This means that if they are visually impaired (e.g., blind, severe refractive error without glasses or contact lenses), cognitively impaired (e.g., comatose, inattentive), or otherwise unable to consistently follow directions (e.g., 2 years old or with a language barrier and no interpreter), then the test cannot be performed. Even awake, alert, and attentive patients must often be reminded to maintain visual fixation during the testing; intermittent attentiveness can result in extraneous re-fixation saccades that can be misinterpreted (i.e., false positives).

Why – Purpose of the Test

The h-HIT interrogates the VOR's disynaptic reflex arc that extends from the horizontal semicircular canal, through the vestibular nerve, into the lateral pons (vestibular nucleus), and from there to the 6th nucleus (and corresponding outputs to the horizontal extra-ocular muscles). The test is abnormal with lesions affecting the labyrinth (e.g., labyrinthitis or aminoglycoside ototoxicity), vestibular nerve (e.g., vestibular neuritis or surgical section), and lateral pons (e.g., AICA stroke or multiple sclerosis plaque). *The h-HIT cannot be used to assess VOR function in the presence of a 6th nucleus lesion or paralysis of horizontal extra-ocular muscles*.

When – Uses of the Test

There are three principal uses of the test, all focused on confirming a peripheral (as opposed to central), cause of vestibular or vestibulo-visual symptoms:

1. **Acute vestibular syndrome**—Patients presenting acutely with severe vertigo, spontaneous nystagmus, nausea or vomiting, head motion intolerance, and gait unsteadiness that has persisted for more than 24 hours have what is known as the "acute vestibular syndrome."[6] This syndrome is most often the result of an acute peripheral vestibulopathy known variably as vestibular neuritis (without auditory symptoms) or labyrinthitis (with auditory symptoms). However, some patients instead harbor dangerous stroke mimics.[7-12] The most common stroke mimic is an inferior cerebellar stroke in the PICA territory which produces a "pseudoneuritis" presentation.[13] Since the three-neuron horizontal VOR arc does not traverse the cerebellum (nor the lateral medulla), PICA strokes mimicking vestibular neuritis can be differentiated from true vestibular neuritis on the basis of a *normal* h-HIT.[11;12] Note that an *abnormal* h-HIT could result from neuritis *or* a lateral pontine stroke.

2. **Chronic dizziness or imbalance**—Some patients who present with chronic vestibular symptoms do not fit an obvious clinical syndrome such as BPPV or Ménière's disease. Some such patients have failed to adequately compensate for a remote unilateral loss of vestibular function (e.g., from prior vestibular neuritis), but may not have a clear history for a prior episode of acute vertigo.[14] Since nystagmus, if present, generally abates within a few weeks following vestibular neuritis, an abnormal h-HIT towards one side may be the only residual bedside sign of prior peripheral vestibular disease. Such a finding would likely push the examiner towards testing or treatment for unilateral 8th nerve or labyrinthine lesions and away from additional testing for central causes of chronic dizziness or imbalance.

3. **Head-motion oscillopsia**—Oscillopsia may be spontaneous and due to intrusive eye movements (e.g., pendular nystagmus, superior oblique myokymia) or associated with head movements and linked to failure (typically bilateral) of the VOR. Confirming a diagnosis of VOR failure in patients with head-motion oscillopsia can be accomplished at the bedside using the h-HIT. Re-fixation saccades in both directions, implying dysfunction of the afferent inputs from both the right and left horizontal semicircular canals, clinches the diagnosis in this context. Causes can then be considered, including aminoglycoside toxicity, carcinomatous meningitis, and neurofibromatosis type 2 with bilateral vestibular schwannomas.

Pearls and Pitfalls

1. **Getting too predictable (false negative)**—It is crucial to remember that for the test to work, the head rotation must be passive (i.e., conducted by the examiner), rather than active (i.e., deliberate head turn executed by the patient).[15] This is because with an active head rotation, the patient's brain will deliberately plan and insert a "predictive saccade" (non-VOR fast eye movement) to correct for a "known" VOR deficit. This predictive saccade may be so well timed and executed during the h-HIT that the end result will appear to be a "normal" VOR response (i.e., the eye appears to stay fixed in space where it is supposed to be, looking at the examiner's nose).[16] A similar false negative response will occur if the examiner attempts several h-HIT cycles (e.g., to "confirm" the presence of an abnormal response), particularly if the cadence of the cycles becomes too consistent or predictable. Thus, an initially abnormal h-HIT response may appear reduced or absent on repeated trials because the patient subconsciously inserts predictive saccades to compensate. An examination technique to minimize this effect is to apply the h-HIT in "unpredictable" fashion (i.e., adjust the timing, direction, or acceleration in a non-patterned way to keep the patient's brain "guessing").

2. **Too little or too much acceleration (false negative or false positive)**—Successful detection of vestibular failure with the h-HIT requires the correct head acceleration. Vestibular hair cell responses are frequency-specific, much the way cochlear hair cell responses are frequency-specific. For the VOR, head acceleration is the "frequency" to which the vestibular hair cells are "tuned." Empirically, the optimal acceleration for the h-HIT appears to be in the range of 2,000-6,000 degrees per second squared.[16] With too little acceleration (reverting towards the classic "oculocephalic"), the test tends to produce false negatives in all but patients with the most severe vestibular failure. With too much acceleration, the test tends to produce false positives, particularly in older patients who may have some asymptomatic (and clinically irrelevant) high-frequency vestibular loss (similar to age-related high-frequency hearing loss).

3. **Over-reliance on a positive (abnormal) h-HIT (overestimated specificity for peripheral disease)**—Because the h-HIT is most often abnormal in peripheral vestibular disease, it is easy to forget that dangerous disorders (e.g., stroke) can also demonstrate a positive h-HIT response. As described above, the most potent bedside predictor of stroke mimic in acute vestibular syndrome appears to be a *negative* h-HIT.[13] Recent studies[10;11] provide evidence that a normal VOR strongly points to a central localization (sensitivity 79%, specificity 93%, likelihood ratio 12). However, these same studies show that an abnormal VOR is less convincing for a peripheral localization (sensitivity 93%, specificity 79%, likelihood ratio 4). The sign's diagnostic utility is diluted because some patients with positive h-HIT (implying vestibular neuritis or labyrinthitis) actually harbor lateral pontine strokes.[11] Many such stroke patients can be identified by other oculomotor signs (skew deviation, direction-changing nystagmus, or impaired vertical smooth pursuit).[10;13]

Corroborative Testing

1. Caloric testing with ENG—Formal, quantitative caloric testing can often corroborate the presence of a peripheral vestibulopathy. However, the h-HIT is "tuned" to slightly higher frequencies than the caloric response, so a dissociation can occur between h-HIT results and caloric test results that does not necessarily indicate a failure of either test.[17]

2. Quantitative h-HIT—The bedside h-HIT can be performed under laboratory conditions using quantitative recording devices such as scleral search coils.[16;18]

VOR Cancellation Test (a.k.a. VOR Suppression Test)

What – Overview of the Test

The VOR is essential for maintaining stable vision on a target when the head is moving, but the brain requires a mechanism for suppressing the VOR during combined eye-head tracking (as in visually pursuing a target moving slowly from right to left off in the distance, where the head and eyes rotate together, rather than in opposition). VOR cancellation is managed by the vestibulocerebellum and, in particular, the flocculus and paraflocculus. However, the task is accomplished by a distributed network of neurons that likely include contributions from the parietal and frontal eye fields, as well as the dorsal pontine nuclei.[19] This network of neurons is either substantially shared with the network serving visual smooth pursuit eye movements or in close anatomic proximity to it.

Although there are many bedside tests of cerebellar function, there are only three that are typically abnormal in diseases predominantly affecting the flocculus and paraflocculus (e.g., Chiari I malformation): (1) eccentric gaze-holding; (2) visual smooth pursuit tracking; and (3) VOR cancellation. Since gaze-evoked nystagmus can result from vestibular disease via a non-cerebellar mechanism, and breakdown of smooth pursuit tracking is often a nonspecific, non-localizing manifestation of age-related brain changes, abnormalities of VOR cancellation are usually more specific markers of vestibulocerebellar dysfunction in patients with dizziness or vertigo.

How – Method of the Test

The bedside VOR cancellation test involves a combined eye-head tracking task. It is typically accomplished as follows: (1) while seated, the patient is asked to extend her arms directly in front of her body and clasp her hands together with thumbs abducted and pointed towards the ceiling; (2) the patient is then asked to visually fixate on her own thumbs and maintain that fixation; (3) the patient's entire body is then rotated "en bloc" from side to side. The rotation should cover an angle sweep of roughly 30 degrees to either side of the starting position and each 60-degree pass lasting approximately 1.5-6 seconds (rotational veocity 10-40 degrees per second). Several passes of increasing velocity should be examined to establish the range within which the VOR cancelling system is able to compensate. This whole-body rotation is most easily accomplished using a swivel chair, although it can also be approximated by asking the patient to twist at the waist from a stationary seat. Particularly in the latter case, the patient must be instructed to keep her head, neck, and shoulders moving together as a unit and her thumbs straight in front of her. Some physical instruction and assistance from the examiner is often required. The test can be adapted for use in interrogating cancellation of the vertical VOR by using the same fixation target (outstretched thumbs), and assisting the patient through several cycles of trunk flexion and extension.

The normal response to the VOR cancellation test is for the eyes to remain stationary and motionless in the primary position of gaze (mid-position within the orbits) throughout the middle of each side-to-side pass. It is not unexpected for normal subjects to slip off the target and demonstrate several nystagmoid re-fixation jerks at the "turns" (i.e., during peak deceleration and acceleration). An abnormal response is for the patient to develop repetitive refixations (nystagmoid jerking) during the middle of each rotational pass (fixed-velocity segment). This will occur at lower velocities and be more prounced in cases with more severe failure of cancellation.

Note that for the test to work effectively, the patient must be able to fixate steadily on a visual target. This means that if they are visually impaired (e.g., blind, severe refractive error without glasses or contact lenses), cognitively impaired (e.g., comatose, inattentive), or otherwise unable to consistently follow directions (e.g., 2 years old or with a language barrier and no interpreter), then the test cannot be performed.

Why – Purpose of the Test

The VOR cancellation test interrogates the circuit that controls visually-mediated override of the VOR during combined eye-head tracking. This is a complex (and incompletely defined) polysynaptic reflex that is managed ultimately by neurons located in the flocculus and paraflocculus. These cerebellar neurons likely receive visual input from direction-specific, parieto-occipital motion-sensing neurons, and a related population of frontal eye field neurons.[19] Inputs likely descend with the parieto- and fronto-pontine fibers in the cerebral peduncle and synapse on a number of small nuclei in the dorsal pons. Relay neurons likely reach the vestibulocerebellum via the middle cerebellar peduncle. As a result, VOR cancellation may be impaired by lesions anywhere along this pathway (including parietal, frontal, and pontine), but are profound and relatively isolated findings with lesions of the

flocculus or paraflocculus (e.g., Chiari I malformation or cerebellar degeneration). *The VOR cancellation test cannot be used to effectively assess VOR cancellation in the presence of primary-position nystagmus*.

When – Uses of the Test

The test is used principally to assess whether patient symptoms are attributable to a vestibulocerebellar lesion:

1. **Dizziness, vertigo, or unsteady gait**—Patients presenting with vestibular symptoms usually have diseases of the vestibulocerebellum or peripheral vestibular system. Distinguishing between the two can be difficult on the basis of nystagmus alone, since few forms of nystagmus are entirely specific to either localization. Central positional nystagmus may be virtually indistinguishable from benign paroxysmal positional nystagmus.[20] The nystagmus of acute cerebellar stroke can be identical to that seen in vestibular neuritis.[11] As a consequence, ancillary signs of cerebellar involvement must be sought. While classical findings of dysarthria or limb ataxia, dysmetria, or dysdiadochokinesis can be relied upon as indicators of a cerebellar localization, these signs are frequently absent in diseases affecting the inferior cerebellum.[13] Accordingly, other central oculomotor signs must be sought. The classic triad of bedside findings with floccular or parafloccular disease is gaze-evoked nystagmus, impaired smooth pursuit, and impaired VOR cancellation. The one least likely to be attributable to peripheral disease or to be a non-specific, unrelated finding is impaired VOR cancellation.

2. **Gaze-evoked nystagmus**—Patients with gaze-evoked nystagmus present a specific challenge with regard to identifying the lesion location. If the gaze-evoked nystagmus is bilateral and symmetric, it is usually due to cerebellar disease causing a failure of normal gaze-holding mechanisms (gaze-holding nystagmus) *or* it is simply a normal physiologic variant. In cases where the nystagmus is relatively mild, a finding of failure of VOR cancellation is likely to corroborate the presence of a disease rather than normal variant. If the gaze-evoked nystagmus is unilateral, it could be the result of either unilateral cerebellar disease (e.g., cerebellar stroke) *or* unilateral peripheral vestibular disease (e.g., vestibular neuritis) that is mild enough (or visually suppressed enough) to be present only in the lateral gaze position,[6] (known as “first degree” peripheral vestibular nystagmus). In these cases, the presence of VOR cancellation failure suggests a central cause. In cases where gaze-evoked nystagmus is brisk, the VOR cancellation task gives an estimate of visual smooth pursuit function, which cannot effectively be tested without being confounded by the gaze-evoked nystagmus.

Pearls and Pitfalls

1. **If you don’t have a VOR, you can’t suppress it (false negative)**—The most common reason for an unexpectedly normal VOR cancellation test is vestibular hypofunction. It is self evident that a “negative” cancellation test is meaningless in a patient with absent VOR function (e.g., post aminoglycoside). Some degenerative diseases affect both the central vestibular structures and peripheral vestibular apparatus. Alternatively, some patients who develop cerebellar disease had pre-existing peripheral vestibular disease, or vice versa. In these cases, there will often be an unexpected dissociation between smooth pursuit deficits and normal VOR cancellation. Thus, it is always advisable to perform h-HIT testing of VOR function prior to assessing and interpreting VOR cancellation test results. Although the most common cause of this dissociation is vestibular hypofunction, there may be certain degenerative conditions in which there is selective loss of smooth pursuit with preserved VOR cancellation without apparent VOR loss.[21]

2. **Over-interpreting during the direction change (false positive)**—Care should be taken not to over-interpret the finding of a few re-fixations at the “turns” during the test. While an ability to maintain fixation during the turns is probably strong evidence of intact VOR cancellation (assuming a normal VOR), the presence of a few nystagmoid jerks during this phase of the test should not be considered pathological if cancellation appears normal during the fixed-velocity (mid-portion) of the rotation (assuming adequate rotational velocity).

3. **Letting the eyes drift out of the primary position (false positive)**—Care should be taken to make sure the patient maintains gaze in the primary position (i.e., eyes in the horizontal mid-position of the orbits) throughout the rotation. If the eyes are allowed to drift eccentrically in the orbits, it may tap into gaze-evoked nystagmus, confounding the test and potentially leading to false positives. This is particularly important when the patient is twisting at the waist from a stationary chair, since they are less likely than those in a swivel chair to accurately maintain an aligned, mid-sagittal placement of their outstretched arms and thumbs.

Corroborative Testing

1. Rotary chair testing with EOG—During formal vestibular function tests with a rotary chair, it is typical for VOR cancellation to be tested using a sinusoidal stimulus at one of several fixed frequencies. Eye movements are measured quantitatively by EOG and subtler deficits than clinically visible can be identified.

Dix-Hallpike Test (a.k.a. Nylen-Bárány Maneuver)

What – Overview of the Test

The Dix-Hallpike test, known by variously-permuted eponymous names[22] (including the Nylen-Bárány maneuver) is a provocative bedside test designed to reproduce symptoms and signs (i.e., nystagmus) in patients with positional dizziness or vertigo. Although forms of positional testing to provoke vestibular symptoms and signs in various clinical contexts have been used since the late 19th century, the modern form of the test was first described in detail by Dix and Hallpike in 1952.[22] With minor adjustments to technique, the Dix-Hallpike test remains the gold standard diagnostic test for the condition now known as benign paroxysmal positional vertigo (BPPV) of the posterior semicircular canal (p-BPPV).[23] The test may also be used in the diagnosis of central positional vertigo syndromes[20] and as a non-specific provocative maneuver to elicit vestibular nystagmus. With rare exception of combined partial vestibular neuritis with BPPV,[24] the test should not be performed in patients who are acutely ill with persistent, continuous vestibular symptoms (vertigo, nausea or vomiting, head motion intolerance). Such patients almost invariably have spontaneous nystagmus at baseline, and exacerbation of symptoms or nystagmus by the Dix-Hallpike test offers no probative diagnostic information in the vast majority.

BPPV is now known to result from canalolithiasis (glibly, “rocks in the head”), likely displaced calcific otoliths that aggregate into mobile concretions or debris. Approximately 90% of cases involve posterior semicircular canal debris (i.e., p-BPPV), but 5-10% involve the horizontal canal (h-BPPV).[25;26] *Note that the Dix-Hallpike test is a specific diagnostic test for p-BPPV and a different diagnostic maneuver (described elsewhere[23]) is required*.

How – Method of the Test

The test method was recently described in an American Academy of Neurology Practice Parameter derived from an evidence-based review of canalith repositioning maneuvers,[23] and is reproduced here (Figure 1):

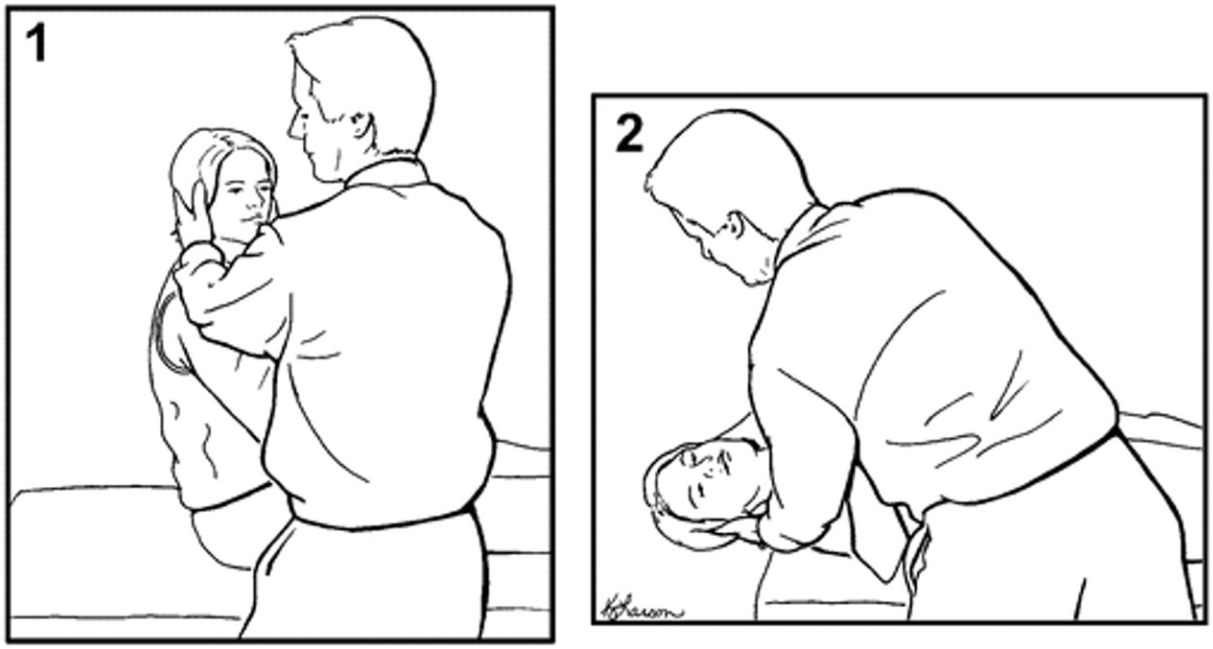

Figure 1 Dix–Hallpike maneuver for diagnosis of right p-BPPV

Step 1 – Seat the patient on a table positioned so they may be taken back to the head hanging position with the neck in slight extension. Stabilize the head with your hands and move the head 45 degrees toward the side you will test. Move the head, neck and shoulders en bloc to the head hanging position (Step 2).

Step 2 – Observe the eyes, hold them open if necessary.

A few clinical points on technique are worth noting. Although some practitioners have been taught to move the head to its final resting position during the transition from sitting to supine or after the patient is supine, it is probably easier and slightly more effective to displace the head 45 degrees *before* the transition. It is often said that the patient should be laid back "quickly." I prefer to use the language "expeditiously but carefully" to make the point that the movement need neither be rough nor as fast as possible; 2-3 seconds is about the correct amount of time to move from upright to the recumbent position. The final head position should be hanging back over the edge of the examining table, rather than flat on the bed (which could lead to a false negative test result); modern clinical beds sometimes have sculpted built-in pillow-like head rests – use the un-sculpted (flat) side for the test.

It is generally advisable to explain to the patient what you are about to do, what they are likely to experience, and what *they* must do. Specifically, they should be warned that they may feel very dizzy, vertiginous, or nauseated, but they should be instructed to keep their eyes open no matter how they feel, since you must observe their eyes to determine the test result. It is a good idea to supply the patient with an emesis basin in easy reach if they have a history of nausea or vomiting with their positional vertigo episodes. *Note that vomiting is rare with a single Dix-Hallpike test in BPPV, and its presence should generally prompt consideration of central mimics*.[20]

Because this is a provocative test, the normal resopnse is no response (no symptoms, no nystagmus). A specific positive response is the development of vertigo or dizziness in conjunction with the classic p-BPPV nystagmus (p-BPPN).[20;27] The classic p-BPPN[28;29] has a mixed vertical-torsional vector with the eyes in the primary position, often with the more dominant vector being vertical in one eye and torsional in the other.[30] The direction of the fast phase is a combined upbeat and ipsi-torsional (i.e., towards the shoulder of the affected ear); thus, the 12 o'clock "pole" of the eyes move towards the right shoulder with a right p-BPPV. Note that if the eyes are deviated laterally during the test, the nystagmus may appear almost purely torsional (looking towards the affected ear) or vertical (looking away from the affected ear), but the direction of the nystagmus fast phase will not change. If the test is performed in the light without Frenzel goggles[31] or another method to block visual fixation, the patient may be able to suppress the vertical vector of the p-BPPN using vision, so the nystagmus may appear almost purely torsional.

Beyond the nystagmus vector and direction are several other nystagmus features said to help discriminate between p-BPPN and central mimics which are often assessed during (or on repeated trials of) the Dix-Hallpike test, although central mimics can be almost indistinguishable on each of these variables.[20] The nystagmus should (a) not begin immediately but after a short latency (1-15 seconds); (b) damp after 5-60 seconds; (c) reverse direction (but not change vector) on arising from the Dix-Hallpike position; and (d) fatigue on repeated trials (i.e., progressively disappear on successive attempts of the Dix-Hallpike test).

Less specific positive responses include the appearance of atypical nystagmus forms with or without symptoms and the development of symptoms without nystagmus. Such findings should be interpreted with caution. Note that finding a few beats of nystagmus as the head reaches its final head-hanging destination should not be considered even a non-specific positive finding unless the head has completely stopped moving. A careful examiner will sometimes notice the tail end of the normal per-rotatory nystagmus as the head comes to a complete stop.

Why – Purpose of the Test

The test is used to confirm a diagnosis of p-BPPV.

When – Uses of the Test

The test is used principally to assess whether patient symptoms are attributable to p-BPPV:

1. **Episodic positional vertigo**—The test is used to confirm a diagnosis of p-BPPV in patients with a typical history of short-lived (<1-2 minute), episodic vertigo or dizziness triggered by characteristic changes in head position with respect to gravity, such as reaching for a top shelf, reclining or arising from bed, and rolling over in bed. The specific nystagmus (p-BPPN, described above) is sought for confirmation prior to treatment by

canalith repositioning maneuvers (described below). Details of the nystagmus are used to identify central mimics (central positional vertigo), which are typically caused by diseases of the cerebellum, including cerebellar degeneration, cerebellar tumors, and small, acute strokes in the inferior cerebellum. Nystagmus features suggestive of central pathology include pure vertical or pure torsional vector, onset without latency, failure to damp within 60 seconds, lack of reversal on arising, and lack of fatigue on repeated trials.

2. **Dizziness, vertigo, or unsteady gait**—The test is sometimes applied by vestibular specialists in patients with ongoing, unexplained dizziness, vertigo, or unsteadiness, particularly if their symptoms have an episodic component or one that might be related to changes in head position relative to gravity. Here, the purpose of the test is two-fold: (1) assess whether there is co-morbid p-BPPV superimposed on a pre-existing vestibular disease (BPPV appears to be more common among those with pre-existing vestibular disease; roughly 50% of BPPV patients have a second disease affecting the vestibular system[27]); (2) determine if there is evidence of central positional nystagmus that might help explain the overall clinical picture.

Pearls and Pitfalls

1. **Not knowing what you're looking for… and when to look for it (false positive or false negative)**—A properly handled Dix-Hallpike test starts with knowing the indications for the test, its purpose, and what to look for… things many physicians are apparently uncomfortable with[32] or mistaken about.[33] The test should be used almost exclusively as a specific test for p-BPPV, which means not using it in patients with acute vestibular syndrome nor in patients with suspected h-BPPV. The examiner must know what p-BPPN should actually look like, including its vector, direction, timing, and reproducibility (described above), facts which sometimes run counter to received wisdom or what is (erroneously) written in textbooks.[32-34] Anything other than classic p-BPPN in the context of a classic history for p-BPPV should spark concern for a possible underlying central disorder. Be particularly wary of symptom-only "positive" Dix-Hallpike results. If the patient has p-BPPV with strong symptoms provoked by position change, there should be strong nystagmus… if there are strong symptoms but no nystagmus, the examiner should entertain an alternative diagnosis.

2. **Poor technique (false negative)**—Although perhaps not as technique dependent as tests such as the head impulse test of VOR function, the Dix-Hallpike test needs to be performed well to achieve optimal sensitivity. This means turning the head 45 degrees before moving the patient, moving the patient quickly to the supine position, making sure the head is hanging off the top edge of the bed, and insisting the patient keep their eyes open throughout (sometimes repeated exhortation is required ("Open your eyes! Open your eyes!"). This all requires some forethought (how to position the bed, what to instruct the patient, and where to stand for the best view of the eyes). If the examiner is lost for 5 or 10 seconds, the nystagmus may already be gone.

3. **Over-interpreting a negative test in a patient with a good history (false negative)**—The typical natural history for p-BPPV involves multiple stereotyped, head-position-provoked episodes that are brief, lasting seconds; these usually occur multiple times per day for days or even weeks during a "bout." The bout eventually remits spontaneously in most patients, presumably because the otolithic debris dissolves or is dislodged through the course of normal activity. Depending on clinic appointment wait times, it is not uncommon for the patient to have suffered their last symptoms a day or more prior to the visit. In such cases, it is typical that the Dix-Hallpike test is negative. There are very few diseases that mimic the classic history for p-BPPV, and the closest mimics (the central positional vertigo syndromes) tend not to remit spontaneously. Accordingly, a negative Dix-Hallpike test should not preclude a well-made historical diagnosis of p-BPPV, but, in such cases, the history of positional triggers and short-lived episodes should be absolutely clear. A history of bouts separated in time (weeks, months, or years apart[35]) is further evidence supporting the diagnosis.

Corroborative Testing

1. Dix-Hallpike with EOG and/or video nystagmography (VNG)—The Dix-Hallpike test can be performed under laboratory conditions with quantitative EOG (or scleral search coil) assessment of nystagmus or video recording that can be "replayed" to assess small details likely to be missed on a single bedside viewing.

Modified Epley Maneuver (a.k.a. Canalith Repositioning Maneuver for p-BPPV)

What – Overview of the Treatment

The modified Epley maneuver is a manual treatment for p-BPPV that involves rotating the head in such a way as to free the canaliths from the posterior semicircular canal.[36] The procedure is one of several canalith repositioning maneuvers (CRPs) that have been described to treat either p-BPPV or h-BPPV.[23] It was the first such maneuver developed[37] and has been studied the most extensively. It has been the subject of numerous randomized clinical trials and at least 6 systematic reviews demonstrating its efficacy in (acutely) curing p-BPPV.[23;38] It is the only therapy recommended with strong evidence by an evidence-based guideline for treatment of p-BPPV.[23]

How – Method of the Treatment

The treatment method was recently described in an American Academy of Neurology Practice Parameter derived from an evidence-based review of canalith repositioning maneuvers,[23] and is reproduced here (Figure 2):

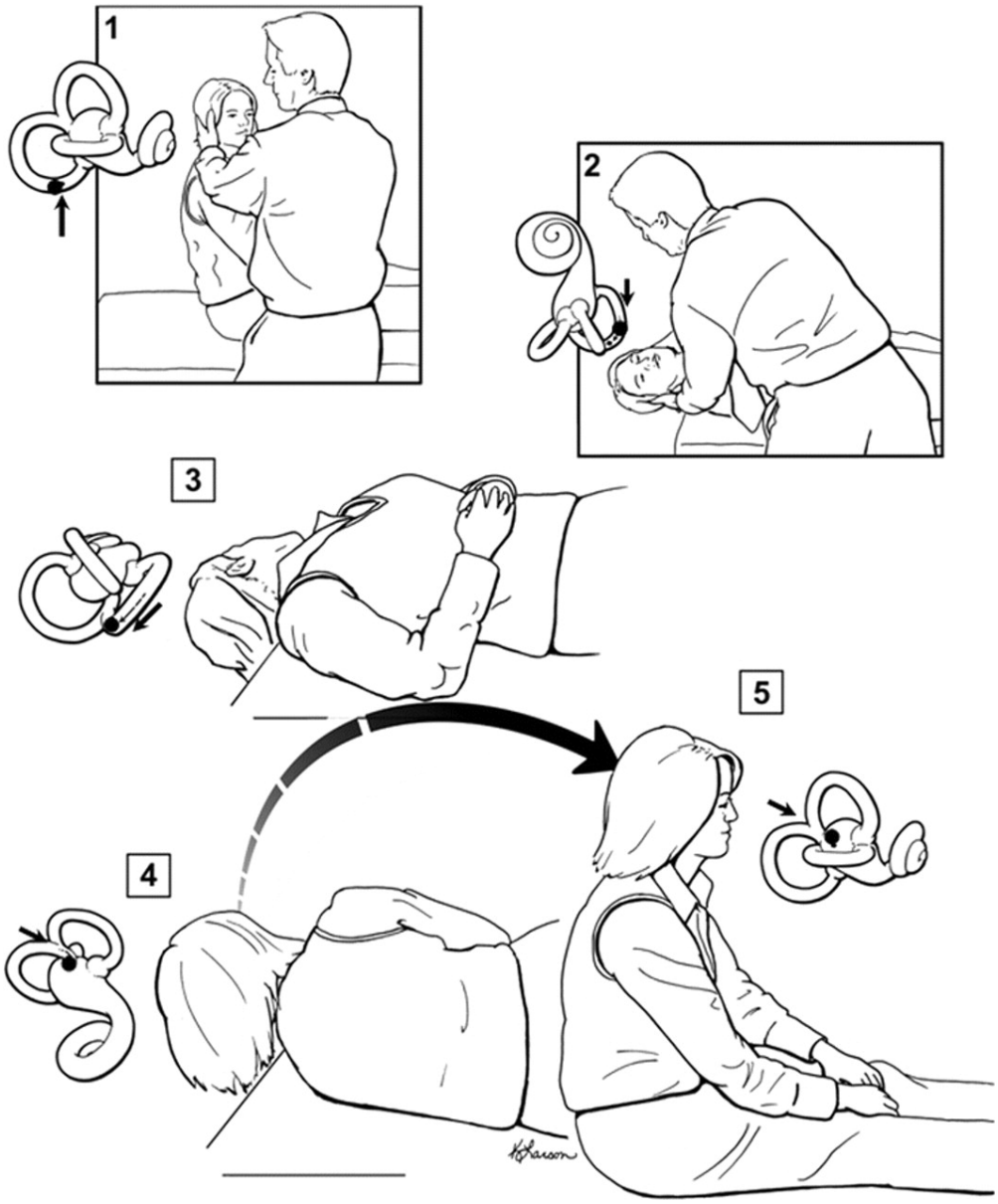

Figure 2. A step-wise method of performing the canalith repositioning procedure for Right BPPV.

Step 1 – Seat the patient on a table positioned so they may be taken back to the head hanging position with the neck in slight extension. Stabilize the head with your hands and move the head 45 degrees toward the side you will test. Move the head, neck and shoulders en bloc to the head hanging position (Step 2).

Step 2 – Observe the eyes, hold them open if necessary. Wait for all the nystagmus to stop and then give it about half as long as it lasted (usually about 10 seconds after it stops).

Step 3 – Keeping the head back with the neck slightly hyperextended, turn the head about 90 degrees toward the opposite side and wait 20-30 seconds. Hold the patient's head to avoid neck strain.

Step 4 – Roll the patient all the way on to his/her side and turn the head to face the ground and hold it there 10-15 seconds. There should be no nystagmus (*if there is, it should beat in the same direction*). If they report a little dizziness, it is usually a favorable sign that the particles are moving and the treatment will be successful.

Step 5 – Keeping the head somewhat in the same position toward the shoulder, have the patient sit up. Hold on to them for a moment because some patients feel a sudden but very brief tilt when sitting up.

REPEAT: After waiting 30 seconds or so, repeat the whole maneuver. If there is no paroxysmal nystagmus or symptom during Dix Hallpike positioning (Steps 1, 2) when repeated, this suggests that CRP has been successful (*be sure to complete the final Epley cycle anyway, lest you accidentally dislodged debris back into the posterior canal during your final Dix-Hallpike test pass; the maneuver may be repeated several times, although it is wise to have an emesis basin nearby for the patient if they remain symptomatic through multiple Dix-Hallpike passes*).

Why – Purpose of the Treatment

The purpose of the modified Epley maneuver is to cure an acute bout of p-BPPV. The treatment offers no known benefit to reduce the long-term risk of symptomatic recurrence (i.e., future bouts).

When – Uses of the Treatment

The treatment should be applied when a patient is suffering from symptomatic p-BPPV and desires relief. Treatment of minimally symptomatic or asymptomatic (incidental) p-BPPN has not been studied.

Pearls and Pitfalls

1. **No treatment is "risk free" (risks to the patient)**—Although the maneuver is non-invasive and thought of as "safe," there is an approximate 12% rate of side effects (nausea, vomiting, fainting) or complications from the procedure (conversion of p-BPPV to h-BPPV or a-BPPV).[23] Conversion can generally be treated by additional (different) maneuvers at the bedside, although one or more additional office visits may be required.

2. **Attempting to treat h-BPPV with the modified Epley maneuver (ineffective)**—The modified Epley maneuver is a specific treatment for p-BPPV. Other maneuvers are available for treating h-BPPV.[23]

3. **Stopping after a single treatment cycle (reduced effectiveness)**—Once a diagnosis of p-BPPV is confirmed by the Dix-Hallpike test and the modified Epley maneuver is performed, it is generally advised to confirm the treatment effect by repeating the Dix-Hallpike test. This can be done through several cycles if Dix-Hallpike induced symptoms or signs (nystagmus) persist and the patient is not suffering ill effects of the treatment. On the final Dix-Hallpike pass, when symptoms or signs have abated, the treatment maneuver (modified Epley) should probably be conducted one final time to ensure the otolithic debris was not "re-inserted" into the posterior canal during the head hanging phase of the diagnostic test (Dix-Hallpike).

Additional Treatment

1. Surgical therapy for intractable p-BPPV—Surgical treatments including singular neurectomy and posterior canal occlusion ("plugging") have been reported to produce remissions in those with intractable symptoms, but evidence of benefit is of insufficient caliber to offer practice recommendations for these procedures.[23]

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