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Head-shaking nystagmus

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Abstract. *Head-shaking nystagmus.* Head-Shaking Nystagmus (HSN) is a latent spontaneous vestibular nystagmus provoked by rapid passive head shaking around a vertical axis.
HSN is not specific in distinguishing peripheral hypofunction from more central vestibular imbalances.
This test is an excellent bedside test for detecting unilateral vestibular hypofunction but further rotatory and caloric testing will be necessary to clarify the patient's condition.

Introduction

Head-shaking nystagmus is a latent spontaneous vestibular nystagmus. It is provoked in a seated patient by rapid passive head shaking around a vertical axis. Frenzel's glasses in a dark room or a video camera (videonystagmoscopy) show no spontaneous nystagmus after head shaking in normal subjects. In patients with peripheral and central vestibular lesions, however, passive head shaking is a powerful way of activating spontaneous nystagmus.

Methods

HSN is elicited by encouraging vigorous, approximately sinusoidal, head shaking for 15-20 seconds. When patients stop shaking their heads, the nystagmus can be observed with Frenzel lenses. Invariably, a transient (5-20 seconds) but relatively brisk nystagmus is found, with the slow phases being initially directed towards the impaired ear. This nystagmus is followed by a much longer but lower-amplitude nystagmus with slow phases directed away from the impaired ear. Vertical head shaking also induces a horizontal nystagmus but the primary phase will then be directed away from the impaired ear. The reversal phase is small or absent.

Results

HSN has been studied for years. The most important papers were selected from the extensive literature on the subject.

In 1986, Takahashi[1] studied biphasic HSN (b-HSN) in nineteen patients using electronystagmography. Sixteen of these patients had unilateral peripheral vestibular disturbance. As in Kamei's[2] study, the first phase beats towards the healthy side and the second phase towards the damaged side in thirteen patients in this group (81%). In the remaining three cases (19%), the first phase beats towards the damaged side, and the second phase towards the healthy side. This contradicts Kamei's findings, in which b-HSN was also observed in three cases of central vestibular disturbance, indicating that b-HSN occurs not only in cases of peripheral vestibular disturbance but also in cases of central origin.

In 1987, Hain *et al*.[3] used the scleral eye coil technique to study nystagmus, finding HSN in sixty subjects with unilateral peripheral vestibular lesions.

Horizontal head shaking elicited horizontal nystagmus with slow phases that were initially directed towards the side of the lesion and upwards. All subjects showed a prolonged lower-amplitude reversal phase after the initial response following horizontal head shaking.

In 1989, Wei *et al*.[4] studied 108 patients referred for caloric testing and found that HSN is not as powerful a test as canal paresis for the detection of lesions of the 8th nerve.

In 1990, Takahashi *et al*.[5] evaluated horizontal HSN in 85 patients complaining of dizziness and vertigo. This was done by comparing the horizontal head-shaking test with routine rotatory and caloric vestibular testing.

They found that HSN evoked by horizontal head-shaking is a highly sensitive way of detecting unilateral vestibular hypofunction. Except in patients with additional central vestibular imbalance or in patients with Ménière's disease,

the direction of horizontal HSN is highly significant, indicating the side of the lesion, with the fast phase beating towards the intact side. However, horizontal HSN is not specific for distinguishing peripheral hypofunction from more central vestibular imbalances. Peripheral vestibular hypofunction, as well as a central asymmetry of the vestibular velocity storage mechanism, can each produce horizontal HSN, either separately or in combination. So the head-shaking manoeuvre is an excellent bedside test for detecting unilateral vestibular hypofunction, but further rotatory and caloric testing is still necessary to clarify the patient's condition.

In 1991, Burgio *et al*.[6] evaluated HSN in 115 patients with vestibular lesions. The data indicate that, with passive head movement, the head-shaking nystagmus test is neither sensitive nor specific enough for use as a screening test for vestibular loss.

In 1992, Hall *et al*.[7] studied a series of 340 patients and 20 controls to compare the vestibular test data with HSN. HSN appears to reflect the underlying spontaneous nystagmus and its direction has no relationship to the side of the vestibular asymmetry.

In 1993, Fujimoto *et al*.[8] conducted a prospective analysis of a series of patients who underwent a head-shaking test during routine ENG. The incidence of head-shaking nystagmus (HSN) in a dizzy population was relatively high (31.7%) when compared with other "abnormalities" in the routine ENG test battery.

Incidence rates in active and passive head-shaking tests are also similar. When present, different types of HSN were identified (monophasic (76.8%), biphasic (22.7%) and triphasic (0.5%)). In some cases, reversals of the expected "normal" pattern occurred. A high correlation was found between a positive head-shaking test and the presence of spontaneous nystagmus, positional nystagmus and caloric test abnormalities.

In 1997, Asawavichianginda *et al*.[9] analysed a group of 1300 patients with a clinical diagnosis of peripheral vestibular disorder. There was a positive correlation of HSN in patients in the pathology group compared with normal control subjects.

In 1997, Tseng and Chao[10] compared HSN and bithermal caloric tests with ENG to determine the sensitivity of the two tests for vestibular dysfunction in 258 patients. The normal limit adopted for canal paresis was 20%. These authors found HSN to be more sensitive than canal paresis. The sensitivity of HSN for canal paresis was 90%.

In 2000, Katsarkas *et al*.[11] demonstrated that the lability of the direction of the initial phase of HSN is due to the reflection of interactions between two main time constants associated with "velocity storage" and "gaze holding" in the vestibular central processes.

In 2002, Guidetti *et al*.[12] examined 420 patients with vestibular diseases of different origin: peripheral, central, or both central and peripheral. They concluded that the sensitivity of the head-shaking test is actually poor, especially over time, and that it should therefore not be used alone in follow-up for patients with vestibular disease.

In 2004, Iwasaki *et al*.[13] compared the incidence of HSN with the value for canal paresis (CP) obtained from a caloric test. The HSN test is not very sensitive but is acceptable as one of the screening tests for detecting asymmetric vestibular dysfunction.

In 2004, Palla *et al*.[14] demonstrated that HSN in patients with chronic unilateral vestibular deficit following vestibular neuritis is influenced by gravity.

In 2004, Perez *et al*.[15] studied the characteristics of horizontal HSN and its relationship with vestibular dysfunction. They found no correlation between HSN and clinical patterns.

Discussion

Head-shaking nystagmus (HSN) has been recognised for many years. It refers to the observation that patients with vestibular lesions of either peripheral or central origin may show a transient increase in or emergence of a spontaneous nystagmus after a period of vigorous head shaking. This nystagmus has traditionally been ascribed to the activation of a latent vestibular imbalance.

Three processes are invoked to explain the presence and the direction of the two phases of HSN according to Zee.[16]

Ewald's second law is of primary importance. It states that, for high velocities of head rotation, excitation is a more effective stimulus than inhibition. This asymmetric response occurs because vestibular afferents are silenced/driven into inhibitory cut-off at a velocity of head rotation that is lower than that which leads to saturation during excitation. The effect of Ewald's law is most apparent when the head is positioned so that the plane of the particular semicircular canal

being tested is parallel to the plane in which the head is rotating. In the case of an absent labyrinth, the increase in peripheral vestibular activity that is relayed centrally with rotation towards the good ear (the excitatory direction) is greater at high speeds of rotation than the decrease in vestibular activity that is relayed centrally with rotation towards the bad ear (the inhibitory direction). This non-linear property of the labyrinthine response forms the basis for using high-speed rotational stimuli to detect unilateral peripheral vestibular lesions. Furthermore, to probe the function of a particular pair of semicircular canals, the head should be positioned with the plane of the canals parallel to the plane in which the head is rotating.

With rapid head-shaking, the non-linearity described by Ewald's second law leads to a continual, asymmetric increase and decrease in activity that is relayed to the central velocity storage mechanism. Consequently, there is an accumulation of activity for slow phases directed towards the impaired ear. When the head stops shaking, the velocity storage mechanism gradually discharges, leading to a slowly decaying nystagmus with slow phases directed towards the bad ear. To account for the reversal phase of HSN, we postulate a short-term adaptive mechanism, comparable to that which produces the reversal phase of caloric or of post-rotatory nystagmus in normal individuals.

The combination of Ewald's second law, asymmetric velocity storage and adaptation gives a plausible explanation for the pattern of horizontal nystagmus that occurs after head shaking in patients with a unilateral peripheral vestibular loss. Note that this hypothesis predicts that head rotations of low velocities should not lead to HSN, because Ewald's law should only become apparent when the speed of rotation is high. HSN caused by more central lesions, such as asymmetries in the velocity storage mechanism itself, might appear when the speed of head rotation is low. Finally, if velocity storage is relatively ineffective, as indicated by a low VOR time constant, the primary phase of HSN will be shorter and the reversal phase will emerge sooner.

What is the origin of the horizontally directed component of the nystagmus induced by vertical head shaking? The most likely explanation is that, in normal individuals, excitation of the vertical semicircular canals also contributes to the generation of horizontal slow phases of nystagmus. This "cross-coupling" between activities in the vertical semicircular canals and horizontal nystagmus arises from the geometrical arrangement of the semicircular canals within the head.

The lateral canals are pitched upwards about 30° and the vertical canals are tilted backwards correspondingly. Consequently, when the head is oriented in certain directions with respect to the axis of rotation, the vertical canals contribute to the generation of horizontal nystagmus and the lateral canals to the generation of torsional nystagmus. In fact, when the head is pitched 60° upwards, and the body is rotated around an earth-vertical axis, a significant horizontal component of the VOR (about 50% of that with the head upright) is still generated – as it should be – even though peripheral vestibular activity with this head orientation arises almost exclusively from the vertical semicircular canals.

One must also remember that excitation of the vertical semicircular canals leads to ipsilaterally directed horizontal slow phases and that rotation around an earth-vertical axis with the head upright leads to inhibition of activity from the vertical canals in the same labyrinth in which the lateral canal is being excited. Accordingly, during vertical head shaking by a patient with only one functioning labyrinth, activity for horizontal nystagmus accumulates in central velocity storage. After vertical head shaking, there is a transient horizontal component of nystagmus with slow phases directed towards the intact ear.

Conclusion

HSN is useful as a "first-line" examination in the evaluation of dizzy patients, particularly when other vestibular tests are impossible.

It is generally admitted that HSN is not sensitive since it is elicited in only 30-40% of patients with a unilateral vestibular deficit.[15]

HSN is also considered non-specific because the existence of positional HSN has been described in 50% of healthy control subjects, as well as in patients without detectable vestibular asymmetries in functional studies.[7,9]

HSN is also found in cases of central vestibular lesions.

HSN has to be interpreted with caution, as one element among others in the diagnosis of vestibular disease.

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