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The head-shaking test in the horizontal plane

PRODUCT INSIGHTS

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That rapid head movement could generate nystagmus was already known to Bárány in 1907, but it was Vogel who introduced head-shaking nystagmus (HSN) into clinical practice more than 20 years later. Head-shaking (HST) is one of the high-frequency tests for the non-instrumental clinical evaluation of vestibulopathic patients.

PURPOSE

To evaluate the asymmetry of the dynamic gain of the vestibulo-oculomotor reflex (dg-VOR) generated by the lateral semicircular canal (CSL) or dysfunction of the central vestibular pathways.

EXPLORED FREQUENCY

High (2 Hz).

INSTRUMENTATION

Frenzel glasses or systems to eliminate visual fixation, but often the intensity of nystagmus is such that it is visible even to the naked eye.

MODE OF EXECUTION

According to Kamei's guidelines (1), the patient is seated and wears Frenzel glasses (or in any case instruments to avoid visual fixation); the head, anteroflexed by approximately 30 degrees, is held firmly by the hands of the operator who subjects it to a lateral-lateral movement approximately twice per second (2 Hz), with an excursion of 45° on each side and for a duration of about 15 seconds. At the end of the shaking, any nystagmic response is observed.

INVOLVED MECHANISM AND PATHOPHYSIOLOGY

To understand the method of execution and correctly interpret the findings it is necessary to remember that the HST is based on the integrated action between Ewald's second law, velocity storage mechanism (VSM) and the nodulus-uvula complex of the cerebellum (cNU).

1. Ewald's second law establishes a directional asymmetry according to which, for the same stimulus, excitation determines a more intense response than inhibition and this is due to the

impossibility for an inhibitory stimulus to reduce the discharge frequency of the vestibular nerve below the zero (2).

2. The VSM stores and prolongs the vestibular signal over time, increases the time constant by approximately three times and improves vestibular performance at low stimulation frequencies (3).
3. Finally, cNU exerts inhibitory control over the VSM and the spatial orientation of the angular vestibulo-ocular reflex, allowing the response to occur in the stimulus plane (4, 5).

THE NYSTAGMUS RESPONSE

HSN is considered significant when at least 5 nystagmus shocks can be detected (6).

The parameters to be taken into consideration are qualitative and quantitative.

The qualitative parameters are represented by the morphology (monophasic or biphasic response), the direction, the plane and the intensity of the nystagmus.

The quantitative parameters are essentially represented by the duration of the response.

The possible responses will now be examined in case of asymmetry of the dynamic gain of the VOR due to peripheral pathology and in case of lesion affecting the central vestibular apparatus (AVC).

HSN IN PERIPHERAL LESIONS, PATHOPHYSIOLOGY AND CLINICAL INTERPRETATION

In case of symmetry of the dg-VOR, the signals generated in the two CSLs during the HS will be of the same entity but of opposite sign, so the VSM cannot be loaded and no HSN will be generated.

On the contrary, in case of asymmetry, the asymmetric canal signals due to the prevalence of one hemisystem will generate a "positive energy balance" which will charge the VSM, which in turn is responsible for the HSN when stimulation stops.

We examine two possibilities: absence or presence of spontaneous nystagmus.

1. In the absence of spontaneous nystagmus, it will be possible to observe a horizontal-torsional nystagmus with a rapid phase directed towards the prevailing hemisystem, a typical finding for example of a vestibulopathy not yet compensated at high frequency: the so-called "paretic" HSN. However, the possibility cannot be excluded that HSN is directed toward the side known to be hypoactive in a kind of vestibular recruitment: as in the case of cochlear recruitment, when the stimulus is particularly intense (as in the case of head-shaking) the partially injured receptor may be activated more than the healthy receptor. Such a finding is discreetly frequent in Meniere's disease, for example.

2. In the presence of spontaneous nystagmus, it may be possible to observe:

    a. an increase in the frequency of the amplitude of the nystagmus, which supports the presence of a dynamic asymmetry of the VOR, the static one being already attested by the presence of spontaneous nystagmus.

    b. the lack of modification of spontaneous nystagmus. Two explanations are possible:
        i. in the acute phase of a peripheral vestibulopathy or in the case of a complete unilateral deficit, the VSM could be deactivated "to protect" the patient (7) since a long duration of the constant VOR time would greatly accentuate the symptoms with even the slightest movement of the head; deactivating the VSM will therefore not allow any HSN to be generated;
        ii. b. the nystagmus does not change because it is determined by a lesion to the AVC: in such cases a peripheral stimulus such as the HSN generally has no effect;

    c. the reversal of the direction of spontaneous nystagmus. At least three explanations are possible:
        i. the nystagmus is of "recovery" type and is directed towards the hypoactive side, while the HSN is directed towards the

healthy side; the "recovery" nystagmus can be the expression of:

1. an imbalance of vestibular nuclei with prevalence of the previously hypoactive hemisystem, which is now the site of static reorganization,
2. the expression of the recovery of the receptor function;

ii. spontaneous nystagmus is of "irritative" type and is directed towards the hypoactive side while the HSN is directed towards the healthy side; this finding is frequent for example in the case of Menière disease where it is possible to observe a spontaneous nystagmus directed towards the affected side, an ipsilateral deficit confirmed by V-HIT and an HSN directed towards the healthy side (personal observation); this "irritative" nystagmus is generated by a mechanic distortion of the cupula due to a translabyrinthine dilational pressure PO in response to hydrops

iii. spontaneous nystagmus beats towards the prevailing side while HSN directed contralaterally toward the hypoactive labyrinth is the result of a labyrinthine recruitment which, like cochlear recruitment, is generated with particularly intense stimuli such as head-shaking and vibration. In this regard, it is important to clarify a concept: although reported in the literature, we tend to exclude that, in such cases, the HSN is of "recovery" type: in fact, it is difficult to understand how a hypoactive hemisystem from a static point of view can instead be prevalent, compared to the contralateral one, from a dynamic point of view.

In each of the possible cases reported, the response may be monophasic or biphasic.

The monophasic response is characterized by the presence of nystagmus that always beats in the same direction for the entire observation time. As already mentioned, the HSN is defined as "paretic" if it is directed towards the side that is prevalent at that specific moment in the patient's clinical history; on the contrary, it is defined as "recovery" if it is directed towards the side considered hypoactive. In our experience, this finding is not frequently encountered; it may in fact be present in a rather narrow time window during the dynamic and high-frequency recovery phase of the previously affected hemisystem which becomes temporarily dominant compared to the healthy hemisystem, still inhibited by the cerebellar structures to reduce the static-dynamic asymmetry.

The biphasic response is characterized by a nystagmus which, after an initial intense phase directed towards one side, spontaneously reverses its direction, giving rise to the second phase. The explanation would be to be found in the fact that the first phase, especially if particularly intense, would determine a functional exhaustion of the peripheral but more likely central vestibular structures and a consequent transitory prevalence of the contralateral structures. In any case, the second phase is not related to the recovery of the peripheral receptor.

HSN IN CENTRAL LESIONS, PATHOPHYSIOLOGY AND CLINICAL INTERPRETATION

In clinical practice, the two central HSN patterns are represented by the perverted HSN (p-HSN) and the minimal stimulus HSN (ms-HSN).

By p-HSN after HS on the horizontal plane we mean the appearance of a nystagmus on the sagittal plane (down beat or up beat nystagmus) or frontal plane (clockwise or anticlockwise torsional) which clearly prevails over any concomitant horizontal nystagmus, or the variation of the plane of a horizontal spontaneous nystagmus. The lesion in this case is to be found in a cNU suffering, with loss of spatial orientation of the angular vestibulo-ocular reflex and inability to match the response plane with the stimulus plane.

By sm-HSN we mean the appearance of a nystagmus after very few cycles of head rotations (even 2-3) performed at a very low speed, a stimulus not capable of generating a "quantity" of energy sufficient to load the VSM. The ms-HSN thus evoked, regardless of the plane in which it strikes, is suggestive of a lesion of the cNU: the loss of the inhibitory control exercised by this complex

on the VSM allows the latter to load and generate even particularly long nystagmic responses with minimal stimuli. Therefore, in the presence of a very strong HSN generated with stimulation of 15 seconds, it is always very important to perform a HS with minimal stimulation.

REFERENCES

Asawavichianginda S, Fujimoto M, Mai M, Rutka J. (1997) Prevalence of headshaking nystagmus in patients according to their diagnostic classification in a dizziness unit. J Otolaryngol.; 26: 20-25.

Baloh, R. W. and V. Honrubia (2001). Clinical Neurophysiology of the Vestibular System, Oxford University Press.

Califano L, Capparuccia PG, Bojano A, Di Maria D, and Villari G (1999) Vertical nystagmus induced by the head-shaking test: 10 cases. Acta Otorhinolaryngol Ital 19: 297-302.

Califano L, Capparuccia PG, Di Maria D, Melillo MG, and Villari G (2001) Sign-report: torsional nystagmus from head-shaking in patients with labyrinth lithiasis of the vertical canal. Acta Otorhinolaryngol Ital 21: 337-340.

Choi KD, Oh SY, Park SH, Kim JH, Koo JW, and Kim JS. Head-shaking nystagmus in lateral medullary infarction: patterns and possible mechanisms. Neurology 68: 1337-1344, 2007c.

Cohen B et al. (2002)The nodulus and uvula: source of cerebellar control of spatial orientation of the angular vestibulo-ocular reflex. Ann N Y Acad Sci. Dec; 978: 28-45.

Demer JL (1985) Hypothetical mechanism of head-shaking nys- tagmus (HSN) in man: asymmetrical velocity storage [ab- stract]. Soc Neurosci; 7:1038.

Fetter M, Zee DS, Koenig E, Dichgans J (1990) Head-shaking nystagmus during vestibular compensation in humans and rhesus monkeys. Acta Otolaryngol 110: 175-181.

Goldberg JM, Fernandez C (1971): Physiology of peripheral neurons innervating semicircular canals of the squirrel monkey: I. Resting discharge and response to constant angular accelerations. J Neurophysiol; 34: 635-660.

Guidetti G, Monzani D, Civiero N. (2002) Head-shaking nystagmus in the follow-up of patients with vestibular diseases. Clin Otolaryngol Allied Sci 27: 124-128.

Hain TC, Fetter M, Zee DS (1987) Head-shaking nystagmus in unilateral peripheral vestibular lesions. American J. Otolaryngology, 8:36-47.

Hain TC, Spindler J (1993) Head-Shaking Nystagmus. in The Vestibulo- Ocular Reflex and Vertigo (Ed. Sharpe JA, Barber HO), Raven.

Hain TC (2007) Head-shaking nystagmus and new technology. Neurology 68: 1333-1334.

Huh, Y. E. and J. S. Kim (2011). "Patterns of spontaneous and head-shaking nystagmus in cerebellar infarction: imaging correlations." Brain 134(Pt 12): 3662-3671.

Humphriss RL, Baguley DM, Moffat DA (2003) Head-shaking nystagmus in patients with a vestibular schwannoma. Clin Otolaryngol; 28(6): 514-519.

Iwasaki S, Ito K, Abbey K, Murofushi T (2004) Prediction of canal paresis using head-shaking test. Acta Otolaryngol 124: 803-806.

Jacobson GP, Newman CW, Safadi I (1990) Sensitivity and specificity of the head-shaking test for detecting vestibular system abnormalities. Ann Otol Rhinol Laryngol 99: 539-542.

Kamei T. (1990) Two Forms of Head-Shaking Tests in Vestibular Examination. In: Deecke L., Eccles J.C., Mountcastle V.B. (eds) From Neuron to Action. Springer, Berlin, Heidelberg.

Kamei T, Takegoshi T, Matsuzaki M. (1995) A quantitative analysis of head-shaking nystagmus of peripheral vestibular origin. Acta Otolaryngol Suppl;520 Pt 1: 216-9.

Katsarkas A, Smith H, Galiana H (2000) Head-shaking nystagmus (HSN): the theoretical explanation and the experimental proof. Acta Otolaryngol; 120: 177-181.

Kim JS, Ahn KW, Moon SY, Choi KD, Park SH, Koo JW (2005) Isolated perverted head-shaking nystagmus in focal cere- bellar infarction. Neurology; 64: 575-576.

Kim CH, Shin JE, Song CI, Yoo MH, Park HJ (2014) Vertical components of head-shaking nystagmus in vestibular neuritis, Meniere's disease and migrainous vertigo. Clin Otolaryngol; 39: 261-265

Kim, H. A., H. Lee, et al. (2011). "Perverted head-shaking nystagmus in focal pontine infarction." J Neurol Sci 301(1-2): 93-95.

Lee, J. Y., W. W. Lee, et al. (2009). "Perverted head-shaking and positional downbeat nystagmus in patients with multiple system atrophy." Mov Disord 24 (9): 1290-1295.

Leigh, RJ and Zee, DS (2015) The Neurology of Eye Movements, 5th ed. Oxford University Press, Oxford.

Mandala M, Nuti D, Broman AT, Zee DS (2008). Effectiveness of careful bedside examination in assessment, diagnosis, and prognosis of vestibular neuritis. Arch Otolaryngol Head Neck Surg 134: 164-169.

Magnusson M, Brantberg K, Pyykkö I, Schalén L. 1989 Reduction of the time constant in the VOR as a protective mechanism in acute vestibular lesions. Acta Otolaryngol Suppl;468: 329-32.

Minagar A, Sheremata WA, and Tusa RJ (2001) Perverted head-shaking nystagmus: a possible mechanism. Neurology 57: 887-889.

Palla A, Marti S, and Straumann D (2005) Head-shaking nystagmus depends on gravity. J Assoc Res Otolaryngol 6: 1-8.

Perez P, Llorente JL, Gomez JR, Del Campo A, Lopez A, Suarez C (2004) Functional significance of peripheral head-shaking nystagmus. Laryngoscope 114: 1078-1084.

Rabbit RD, Boyle R, Highstein SM. Sensory transduction of head velocity and acceleration in the toadfish horizontal semicircular canal. J. Neurophysiol. 72(2):1041–1048, 1994.

Rabbit RD, Boyle R, Highstein SM. Mechanical indentation of the vestibular labyrinth and its relationship to head rotation in the toadfish, opsanus tau. J. Neurophysiol. 73(6):2237–2260, 1995.Takahashi S (1986) Clinical significance of biphasic head-shaking nystagmus. Auris Nasus Larynx.;13 Suppl 2: S199-204.

Takahashi S, Fetter M, Koenig E, Dichgans J (1990) The clinical significance of head-shaking nystagmus in the dizzy patient. Acta Otolaryngol 109: 8-14.

Tseng HZ, Chao WY (1997) Head-shaking nystagmus: a sensitive indicator of vestibular dysfunction. Clin Otolaryngol Allied Sci 22: 549-552.

Waespe W, Cohen B, Raphan T. (1985) Dynamic modification of the vestibulo-ocular reflex by nodulus and uvula. Science; 228:199-202.

Yamauchi A, Rabbitt RD, Boyle R, Highstein SM. Relationship between inner-ear fluid pressure and semicircular canal afferent nerve discharge. J Assoc Res Otolaryngol. 2002 Mar;3(1):26-44. doi: 10.1007/s101620010088. PMID: 12083722; PMCID: PMC3202362.

Yang, TH et al (2020) Clinical implications of head-shaking nystagmus in central and peripheral vestibular disorders: is perverted head-shaking nystagmus specific for central vestibular pathology? Eur J Neurol Jul; 27(7): 1296-1303. doi: 10.1111/ene.14161. Epub 2020 Mar 30.

Zee DS (1988) New Concepts of Vestibular Nystagmus in Vestibular Disorders. In: Barber HO, Sharpe JA, eds. Vestibular disorders. Year Book Medical Publishers, Chicago;189-200.

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