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Vibration-induced nystagmus

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Introduction

The influence of proprioceptive neck receptors on equilibrium through the spinocerebellar and spinovestibular tracts has been well known for a long time. Hinoki *et al*.,[1] in 1971, stressed their importance in dizziness due to "whiplash injury".

Vibrations at the level of neck muscles may induce a degree of dizziness and, in some cases, ocular movements[2,3] and a perturbation of the vertical subjective.[4,5]

In 1973, Lücke[6] demonstrated that vibratory stimulation of the mastoid induced nystagmus in patients with a unilateral vestibular lesion.

Hamann[7-9] observed that vibration-induced nystagmus (VIN), usually associated with peripheral vestibular dysfunction, expressed a latent destructive nystagmus and, in 1997, he indicated the importance of this test for the detection of acoustic neuromas.

Halmagyi *et al.*[10] proposed the use of skull taps as a method of vestibular activation.

At the present time, study of the use of VIN is centered mainly in France.[11,12]

Physiology

In 1962, Hood[13] demonstrated that the speed of propagation of the vibratory wave for 100 Hz frequency stimulation through the head is about 100 m/sec.

Consequently, the stimulation of both labyrinths is simultaneous, the transcranial conduction time being about 2 m/sec.

A unilateral vibratory stimulation is not specific to a unilateral vestibular stimulation.

It does not allow demonstration of a unilateral labyrinthine weakness (canal paresis) but can reveal a directional preponderance of the nystagmus.

It is fundamentally different from the caloric test.

In 1977, Young *et al*.[14] demonstrated in monkeys that vibratory stimulation of 125 to 350 Hz of the cranium modified the activation of inner ear cells directly rather than through vibration of the endolymph.

VIN induced by a vibratory stimulus of 100 Hz relates to a frequential zone of stimulation completely different from the physiological stimulation of the rotation of the head, which corresponds to frequencies of 0.05 to 5 Hz.

Method

The method of Dumas *et al*.[11] is proposed:

- stimulator generating vibrations at 100 Hz with an amplitude of 0.2 mm
- subject in sitting position
- three positions for the vibration: vertex, left and right mastoid processes.

Results

To be considered pathological, the VIN must be identical in at least two positions and sustained. The nystagmus is called "apreed".

To be considered abnormal, the slow phase of the VIN must be more than 2.9°/sec.

In the case of nystagmus existing prior to the vibratory stimulation, the slow phase must be significantly enhanced or decreased.

The response to the stimulus appears immediately at the beginning of the stimulation and stops when it ceases.

There is no fatigability in the response.

The rotatory phase is constant.

Vibratory stimulation does not induce vertigo.

In 2004, Magnusson *et al*.[15] demonstrated that EMG responses in the lower leg were evoked by vibratory stimulation of the posterior neck muscles and not through mastoid vibrations.

Dumas *et al*.,[11] Ulmer *et al*.,[12] Ohki *et al*.[16] observe that VIN is inconstant in peripheral labyrinthine lesions. In these cases, when VIN is present, its direction is towards the normal labyrinth.

VIN is also found in central diseases such as cerebrovascular accidents, Arnold-Chiari malformation or spinocerebellar degeneration.

Vibratory stimulation does not modify the results of repositioning manoeuvres in cases of benign paroxysmal vertigo.[17]

Conclusion

VIN permits the demonstration of vestibular asymmetry in a simple

way in patients whose physical state is incompatible with other means of vestibular exploration.

The vibratory test does not differentiate between central and peripheral lesions, nor does it indicate the side affected.

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