

Sensitivity and specificity of mastoid vibration test in detection of effects of vestibular neuritis

Sensibilità e specificità del test della vibrazione mastoidea nella individuazione degli esiti della neurite vestibolare

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Key words

Vestibular neuritis • Bedside vestibular examination • Mastoid vibratory test

Parole chiave

Neurite vestibolare • Esame vestibolare clinico • Test vibratorio mastoideo

Summary

Aim of this study was to determine sensitivity and specificity of the mastoid vibration test in patients who had suffered an attack of vestibular neuritis. Results were compared with the caloric test and two bedside tests of vestibular function (head shaking test and head thrust test). Results are reported in 28 patients who had a residual vestibular deficit 6 months after acute neuritis and in 25 healthy subjects. Mastoid vibration nystagmus was evoked in 21 patients but not in controls. In these patients, mastoid vibration test had a sensitivity of 75% and specificity of 100%. Since one patient had inverted mastoid vibration nystagmus, specificity of identification on the pathological side was 95%. Sensitivity of the test increased with increasing severity of the vestibular lesion. Indeed, mastoid vibration nystagmus was induced in 93% of patients with caloric paralysis and in 58% of those with caloric paresis. Nystagmus could usually be modulated or elicited by stimulation of either mastoid. In the few patients in whom mastoid vibration nystagmus was elicited only from one side, or when there was a clear difference in intensity of the nystagmus induced on the two sides, the stimulated side was more often the affected side. Four patients still showed spontaneous nystagmus. The caloric test was abnormal in 26/28 patients (93%) with paralysis in 16 and paresis in 12; 71% of patients had a head shaking induced nystagmus: 64% had an asymmetrical response in head thrust test. In conclusion, mastoid vibration test was overall more sensitive than head thrust test. Mastoid vibration test was slightly less sensitive than head shaking test in patients with severe residual deficit and more sensitive in patients with partial deficit. Mastoid vibration test, a valid, low cost clinical screening test for rapid detection of asymmetrical vestibular function, does not cause patient discomfort. It is suggested that this test be included in the diagnostic workup of all patients with suspected vestibular dysfunction.

Riassunto

Lo scopo di questo studio è stato quello di determinare la sensibilità e la specificità del test vibratorio mastoideo (MVT) in pazienti affetti da esiti di neurite vestibolare. I risultati sono stati confrontati con quelli ottenuti dalla stimolazione calorica e da due test dinamici della funzione vestibolare: l'head shaking test (HST) e l'head thrust test (HTT). Riportiamo in questo studio i risultati di 28 pazienti che presentavano un deficit residuo della funzione vestibolare a distanza di sei mesi dalla fase acuta, confrontati con 25 soggetti normali. Il nistagmo da vibrazione mastoidea (MVN) è stato evocato in 21 pazienti ed in nessuno dei soggetti del gruppo di controllo. Pertanto, nei nostri pazienti il MVT ha una sensibilità del 75% e una specificità del 100%. Poiché un paziente ha presentato un MVN invertito, la specificità nella identificazione del lato lesso è del 95%. La sensibilità del test aumenta con la gravità della lesione vestibolare. È stato infatti possibile rilevare MVN nel 93% dei pazienti con paralisi calorica e nel 58% di quelli con paresi calorica. In genere il nistagmo vibratorio viene evocato o modulato dalla stimolazione di entrambe le mastoidei. Nei pochi pazienti in cui il MVN compariva solamente con la stimolazione di una mastoide o quando era rilevabile una evidente differenza di intensità del nistagmo tra le due stimolazioni, il lato stimolato corrispondeva più frequentemente a quello affetto. A distanza di sei mesi dall'esordio, quattro pazienti presentavano ancora un nistagmo spontaneo mentre il test calorico risultava alterato in 26/28 pazienti (93%), con 16 paralisi e 12 paresi. Il 71% dei pazienti presentava un head shaking nystagmus. Nel 64% dei casi era evidente una risposta asimmetrica al HTT.

In conclusione il MVT è risultato costantemente più sensibile dell'HTT. Rispetto all'HST, il MVT si è rivelato leggermente meno sensibile in pazienti con un deficit residuo severo e più sensibile nei soggetti con deficit parziali. I nostri risultati confermano la validità del MVT quale test di screening per una rapida individuazione di una asimmetria della funzione vestibolare. Il test non causa alcun disagio per il paziente, è di facile esecuzione ed interpretazione e richiede una strumentazione di basso costo. Per tali motivi suggeriamo di includerlo all'interno delle procedure diagnostiche di routine per tutti i pazienti con una possibile disfunzione vestibolare.

Introduction

Vibration on the bone behind the ear may elicit nystagmus in patients with vestibular problems. The mastoid vibratory test (MVT) was first introduced in 1973 by Lücke as a simple bedside test to detect static anomalies in peripheral vestibular function¹. In 1993, Hamann demonstrated that MVT elicited nystagmus in 75% of patients with unilateral vestibular deficit and 10% of patients with central vestibular disorders². The usefulness of MVT in the identification of unilateral vestibular loss was confirmed in later studies^{3,4}. A vibratory stimulus has been applied not only to the mastoids but also to the vertex and neck^{4,5}. In 1999, Hamann and Schuster⁶ found that the sensitivity of the test was increased using a frequency of 60 Hz instead of 100 Hz. In 2004, Dumas et al.⁷ reported complete concordance between MVT, the head shaking test (HST) and the caloric test for detecting the side with total vestibular loss. It was also suggested that MVT elicits vibratory nystagmus (VN) in healthy subjects^{5,8}.

It is not clear how mastoid vibration causes nystagmus in patients with peripheral vestibular deficit. Vibration has been shown to excite semicircular canals and otolithic afferents in various animal species⁹⁻¹¹ and in humans^{4,6,12,13}, indicating that the effects on eye movement can be attributed to direct stimulation of intact vestibular receptors. Kalberg et al., in particular, showed that vibration to the mastoid bone induces eye movements similar to those seen acutely, without vibration, after unilateral vestibular loss and that the eye rotation axis of vibration-induced nystagmus is related to the extent of the unilateral vestibular deficit¹². The vibratory test, according to some authors, is not unlike the simultaneous caloric test⁷. Vibration also seems to increase afferent activity from stimulated muscles¹⁴, suggesting that the effects may be due to interaction between neck proprioceptors and the vestibular system^{15,16}.

The aim of the present study was to determine the sensitivity and specificity of MVT in patients with damage caused by vestibular neuritis, comparing results with those of the caloric test, the current gold standard for identifying unilateral deficit in vestibular function. MVT was also compared with the head shaking test (HST) and with the head thrust test (HTT), other conventional bedside tests used in the detection of vestibular asymmetry.

Methods

SUBJECTS

The cohort used in this study was a patient-based clinical population seen between January 2002 and January 2004. Patients were seen in the acute stage of

the disease (1-3 days after the beginning of symptoms) and all were eligible for inclusion in the study. Inclusion criteria were: 1) acute vertigo lasting at least 24 hours, 2) horizontal unidirectional spontaneous nystagmus lasting at least 24 hours, 3) no hearing loss, 4) no additional neurological signs or symptoms (and normal brain imaging, if performed), 5) unilateral caloric weakness (caloric paresis or paralysis). Overall, 57 patients met these criteria.

Patients were asked to return for follow-up after 1, 3 and 6 months. Approximately 50% of the patients recovered, since no spontaneous nystagmus was detectable, caloric response was balanced and no asymmetry was seen with bedside vestibular tests.

Results of the vibratory test performed in 28 patients with residual deficit in vestibular function, six months after onset of the disease, are reported. Vestibular function was considered impaired if patients had a caloric weakness and/or head shaking nystagmus and/or heat thrust sign and/or spontaneous nystagmus.

CLINICAL TESTING

At each clinical examination, patients underwent a complete bedside clinical examination. Spontaneous nystagmus (SN), head-shaking-induced nystagmus (HSN) and mastoid vibration-induced nystagmus (MVN) were sought with fixation removed using Frenzel goggles.

The dynamic performance of the vestibulo-ocular reflex was evaluated using manually-induced head thrusts: abrupt, rapid, high acceleration head rotations of about 20 degrees¹⁷. The patients were instructed to fix on the examiner's nose. The HTT was considered abnormal if there was an obvious corrective saccade supplementing an inadequate slow phase and only the corrective movement was seen, or was considerably larger, with rotation towards one (the affected) side (Head Thrust Sign).

HST was performed according to Kamei et al.¹⁸. The patients were placed in a sitting position with the head flexed down to 30°, the eyes closed and covered with Frenzel glasses. The head was then shaken horizontally by the examiner around the jaw axis, 20 times for about 10 sec (2 Hz) with amplitude of about 45 degrees to either side. When the head movement was stopped, patients were instructed to open their eyes and the examiner recorded the direction, frequency and duration of any nystagmus. HSN was considered present if at least three or more consecutive beats of nystagmus appeared in patients without SN. In patients with SN, the test was considered positive when an evident increase in frequency was observed. Patterns of nystagmus response were classified as parietic HSN when the fast phase was directed towards the healthy ear and reversed HSN if it was directed towards the affected side^{19,20}.

Caloric testing was performed with hot, cold and ice water. Maximum speed of the slow-phase of nystagmus evoked by each ear was analyzed for unilateral weakness and directional preponderance as determined by Jongkees formulae²¹. Caloric *paresis* was defined when there was some response on both sides but the difference between the two ears was 30% or more. Caloric *paralysis* was defined when there was no response to ice water irrigation on one side. Patients were considered to have recovered when asymmetry dropped to <30%.

MVT was applied with a battery-powered device designed for physiotherapy. It has a circular contact surface, 4 cm in diameter (Fig. 1), which is placed perpendicular to the skin just behind the ear. Fundamental frequency, measured with the aid of a phonometer, was 100 Hz (60-120 Hz). Vibratory stimulation was applied in the mastoid region for about 10 sec, first on one side and then on the other. The test was considered positive when persistent horizontal or horizontal torsion nystagmus was evoked for the duration of the stimulus and if the phenomenon was repeatable. In patients with spontaneous nystagmus, the test was regarded as positive when nystagmus showed an evident increase in frequency. We also performed MVT in the control group of 25 normal subjects.



Fig. 1. Mastoid vibrator.

Results

Mean age of the 28 patients (23 male, 5 female) was 57 years. The right side was affected in 14 patients and the left in 14. Results of the tests are shown in Table I.

Spontaneous nystagmus was still present in four patients, with the fast phase always directed towards the healthy ear. The caloric test was positive in 26/28 patients (93%), 12 of whom had caloric paresis, 14

Table I. Results of bedside vestibular tests and caloric test.

	Present	Absent
SN	4 (14%)	24 (86%)
MVN	21* (75%)	7 (25%)
HSN	20† (71%)	8 (29%)
HTS	18 (64%)	10 (36%)
Caloric weakness	26 (93%)	2 (7%)

SN: spontaneous nystagmus; MVN: mastoid vibration-induced nystagmus; HSN: head shaking-induced nystagmus; HTS: head thrust sign

* One patient showed reversed MVN

† Two patients showed reversed HSN

caloric paralysis and two symmetrical caloric response. These two patients were considered still pathological due to the presence of HSN in both. In one, parietic HSN was detectable, in the other, reversed nystagmus, beating towards the affected ear. The patient with a parietic HSN also showed MVN beating towards the affected ear.

HSN was evoked in 20/28 patients (71%), being parietic in 18, with the fast phase towards the healthy ear, while in two it was reversed.

The head thrust sign was evident in 18 patients (64%), always congruent with the affected ear.

MVN was elicited in 21 patients (75%), being reversed in one, with the fast phase beating towards the affected ear. In 16/21 patients, MVN was elicited by vibration of both mastoids. In three of these patients, MVN was stronger, with vibration of the mastoid on the affected side. In the other 13, no difference in nystagmus intensity was seen. In 5/21 patients, MVN was evoked only unilaterally. In three of these cases, this was provoked by vibrating the affected side, in the other two with contralateral stimulus. In none of these cases was the direction of the nystagmus reversed by changing the side of stimulation. Mastoid vibration did not evoke nystagmus in any of the subjects in the control group.

MVN was evoked by MVT in 21/28 patients, thus a sensitivity of 75%. Since none of the normal subjects manifested MVN, the test has a specificity of 100% for patients with effects of vestibular neuritis. One patient experienced reversal MVN indicating that the specificity of identifying the pathological side is 95% (20/21 positive vibration tests).

When the study population was divided into three groups on the basis of the results of the caloric test, MVN was evoked in 13/14 patients with caloric paresis (93%), 7/12 patients with caloric paresis (58%) and one of the two patients without caloric imbalance i.e., the patient with reversal MVN. Results are outlined in Table II.

Table II. Relationship between bedside vestibular tests and caloric results.

	Paralysis	Paresis	Symmetric response
Caloric test	14	12	2
SN	3 (21%)	1 (8%)	/
MVN	13 (93%)	7 (58%)	1* (50%)
HSN	14 (100%)	4† (33%)	2† (100%)
HTS	13 (93%)	5 (42%)	/

SN: spontaneous nystagmus; MVN: mastoid vibration-induced nystagmus; HSN: head shaking-induced nystagmus; HTS: head thrust sign
 * One patient showed reversed vibration-induced nystagmus
 † One patient showed reversed head shaking-induced nystagmus

When the probability of identifying residual deficit in vestibular function was determined only by means of bedside clinical tests, without the caloric test, 15/24 patients (54%) were found to be positive with all three tests (MVT, HST, HTT), 5/28 (18%) with at least two and 4/28 (14%) with only one. The tests were negative in 4/28 patients (14%) with an abnormal caloric test (Table III).

Discussion

Vestibular neuritis may cause sudden unilateral loss of vestibular function. Approximately 50% of patients recover this function spontaneously or after treatment^{22 23}. While clinical and/or other signs of deficit persist in the remaining 50%. The caloric test is still the method of choice to detect vestibular loss with a sensitivity of 93%. Dynamic tests of peripheral vestibular deficit, such as HST and HTT, are simple and rapid to perform and do not require specific instruments. Mastoid vibration is another simple method used in the detection of unilateral vestibular deficit.

In the present study, 28 patients were selected with

vestibular neuritis who still had evidence of residual vestibular deficit, as indicated by caloric weakness and/or dynamic vestibular signs, 6 months after the acute episode. In these patients, MVN was detected in 75%, HSN in 71%, HTS in 64%, and caloric weakness in 93%. These results confirm those of other studies, namely that the vibratory test shows good sensitivity for identifying compensated vestibular loss^{2 4 5 7}. Considering only patients with vestibular neuritis, MVT sensitivity, in the literature, ranges from 75 to 90%, with differences probably due to the fundamental frequency of the applied stimulus (60-100 Hz) and the method used in the detection of nystagmus (Frenzel glasses or videonystagmography)^{4 6 7}. The sensitivity of MVT increases with increasing severity of the vestibular lesion. Indeed, it was possible to induce MVN in 93% of our patients with caloric paralysis. This finding is in keeping with those of Michel et al.⁵ and Dumas and Michel⁴ who obtained sensitivity of MVT of 100% and 96%, respectively, in patients undergoing vestibular neurectomy. When we considered patients with caloric paresis, sensitivity of MVT was only 58%. Comparison with the other two bedside vestibular tests showed that MVT was consistently more sensitive than HTT. Compared with HST, MVT was slightly less sensitive in patients with severe residual deficit (caloric paralysis) and more sensitive in those with partial deficits. Our results are in contrast to those of Hamann and Schuster⁶ who reported a much higher sensitivity of MVT with respect to HST (80% vs. 35%) for identifying peripheral vestibular deficit. This difference could be due to the different patient populations and timing of the tests. In our patients, only one with vibration-induced nystagmus beating towards the affected side was identified. Reversed MVN has been reported in other studies with a higher incidence than in our study⁷. As with HST, identifying the affected side with MVT is not always reliable. Though a rare possibility in vestibular neuritis, and without any satisfactory pathogenetic explanation, it should, nevertheless, be borne in mind as a limit of this method. The side stimulated by vibration does not seem to af-

Table III. Number of patients with one or more positive bedside tests.

MVN	MVN	MVN	HSN				
HSN	HSN	HTS	HTS	MVN	HSN	HTS	Caloric weakness only
HTS							
15/28	3/28	1/28	1/28	2/28	1/28	1/28	4/28

MVN: mastoid vibration-induced nystagmus; HSN: head shaking-induced nystagmus; HTS: head thrust sign

fect the capacity to generate or modulate nystagmus. In most of our patients, MVN was elicited by stimulating on the right and left without any significant difference in response. In 8 patients, who showed MVN when only one mastoid was stimulated, or when there was an evident difference in intensity of the vibration-induced nystagmus, the side stimulated was more often the affected side (6/8 patients). Nystagmus changing direction was not detected in any of these patients as occurs in other labyrinth disorders, such as Menière's disease.

We did not find MVN, in any of the 25 control subjects, that persisted for the duration of the vibration or was repeatable. This is in keeping with the findings of Hamann and Schuster⁶ but not with those of others^{5,8}. One reason could be that our vibratory stimulus was applied exclusively to the mastoids and that nystagmus was observed only with Frenzel glasses and not recorded by videonystagmography. In this case, our results indicate that simpler methods of stimulation and detection make the test more specific and less subject to false positives. We prefer to apply the stimulus to the mastoids, avoiding stimulation of the neck and vertex muscles, to simplify the test and avoid possible interactions or interference with cervical afferents.

The mechanism underlying MVN appears to reside in the capacity of the vibratory stimulus to excite the semicircular canals and otolithic afferents^{9,10}. The effects caused by eye movements appear, therefore, to be due to direct stimulation of intact vestibular receptors on the healthy side^{4,6,12,13}. Some

Authors compare it to a simultaneous caloric test which exploits the peculiar stimulation. However, it is not clear why the response is not more intense when vibration is applied to the mastoid on the healthy side.

Table III illustrates an important practical aspect. Using exclusively clinical tests, without the aid of the caloric test, it was possible to identify a vestibular deficit in 86% of patients. Thus for patients with a perforated ear drum or who refuse the caloric test, there is still a high probability of making a diagnosis. Moreover, the few cases in which MVT, HST and HTT were not useful for diagnosis, were all found to have a partial, rather than severe, vestibular deficit. On the other hand, the negative caloric tests in two patients who certainly still had a vestibular deficit emphasizes the need to carry out also other clinical tests.

In conclusion, the present results suggest that MVT is a rapid, comfortable and valid clinical screening test for detecting the presence of asymmetric vestibular function. We propose that it be included, together with dynamic vestibular and positional tests, in the diagnostic work-up of patients with vertigo.

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