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ORIGINAL COMMUNICATION

Head-shaking tilt suppression: a clinical test to discern central from peripheral causes of vertigo

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Abstract Tilt suppression refers to both tilting the head away from an Earth vertical axis and a reduction of an induced horizontal nystagmus. This phenomenon of reducing an induced horizontal nystagmus involves a circuitry of neurons within the vestibular nuclei and the cerebellum (collectively referred to as velocity storage) and signals from the otolith end organs. Lesions involving this circuitry can disrupt tilt suppression of induced horizontal nystagmus. We investigated the clinical value of combining the horizontal head-shaking nystagmus test with tilt suppression in 28 patients with unilateral peripheral vestibular hypofunction and 11 patients with lesions affecting the central nervous system. Each of the subjects with peripheral vestibular lesions generated an appropriately directed horizontal nystagmus after head shaking that then suppressed the induced angular slow phase velocity on average 52 ± 17.6% following tilt down of the head. In contrast, patients with central lesions had very little ability to suppress post-head-shaking nystagmus (mean 3.4 ± 56%). We recommend tilting the head after head shaking as a useful clinical test to assist in the differential diagnosis of vertiginous patients. In the case of unilateral peripheral vestibular hypofunction, head tilt suppresses the induced nystagmus via influence of the otolith organ. In the case of central pathology, the inability to suppress the nystagmus is from lesions impairing the otolith mediation on the velocity storage circuitry.

Keywords Tilt suppression · Vestibular · Velocity storage · Head shake nystagmus test · Cerebellum · Nodulus

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Introduction

The nodulus, flocculus, and uvula of the cerebellum receive a significant volume of semicircular canal and otolith afference, and comprise the majority of the vestibulocerebellum. The principal projection of utricular afferents toward the cerebellum is to the nodulus with a weaker proportion to the flocculus or uvula. The principal projection of saccular afferents is to the uvula of the cerebellum, with weaker proportion synapsing in the nodulus [1]. A primary function of the nodulus and ventral uvula is the mediation of the velocity storage mechanism of the vestibulo–ocular reflex. The putative purpose of the velocity storage system is to enhance the low-frequency performance of the angular vestibulo–ocular reflex (aVOR) [2, 3], which sustains the duration of an induced horizontal nystagmus during head rotation longer than the time that would be predicted based on the mechanical properties of the semicircular canals [4]. This prolonged duration of nystagmus is measured as the time constant, occurs both for sustained rotation and again after the rotation has stopped (albeit in a different direction), and is judged to be normal within the range of 10–25 s [3]. The velocity

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Table 1 Patients with unilateral peripheral vestibular hypofunction without evidence for central pathology

Patients	Etiology	Age	Gender	vHIT	Direction of post-HSN	SPEV post-HSN—neutral	SPEV post-HSN—flexion	% Suppression
1	Vestibular neuritis	31	M	(+) R	L	8.1	2.8	65
2	Presumed vascular	57	F	(+) R	L	5.1	2.2	56
3	Presumed vascular	77	F	(+) L	R	3.1	0.7	77
4	Vestibular schwannoma	80	F	(+) L	R	2.7	0.8	70
5	Vestibular neuritis	49	F	(+) L	R	3.1	0.6	80
6	Unknown	62	F	(+) L	R	5.6	3.1	44
7	Gentamycin injection	60	M	(+) R	L	8.4	2.2	73
8	Presumed vascular	60	M	(+) L	R	6	1	80
9	Vestibular neuritis	35	F	(+) R	L	3.0	1.5	50
10	Vestibular neuritis	69	F	(+) R	L	23.5	14	40
11	Vestibular neuritis	22	M	(+) R	L	11.6	5.6	51
12	Vestibular neuritis	64	F	(+) R	L	10.3	8.3	19
13	Vestibular neuritis	60	F	(+) L	R	12.3	10.2	17
14	Vestibular neuritis	35	M	(+) R	L	7.1	4.2	40
15	Vestibular neuritis	61	M	(+) L	R	12.1	2.9	76
16	Vestibular neuritis	61	M	(+) L	R	12.1	2.9	76
17	Vestibular neuritis	26	F	(+) R	L	5.7	3.3	42
18	Vestibular neuritis	38	M	(+) L	R	0.3	0.1	66
19	Temporal bone tumor	57	M	(+) R	L	12.5	8.4	32
20	Presumed vascular	60	M	(+) L	R	1.5	0.4	73
21	Superior vestibular neuritis	49	F	(+) L	R	1.3	1.4	-7
22	Vestibular neuritis	36	F	(+) L	R	12	8.8	27
23	Vestibular neuritis	37	M	(+) L	R	12	8.4	32
24	Vestibular neuritis	38	M	(+) L	R	2.4	0.8	66
25	Vestibular neuritis	75	F	(+) L	R	11	6	45
26	Vestibular neuritis	79	F	(+) R	L	12.4	4.7	62
27	Labyrinthitis	34	F	(+) R	L	17.2	8.8	49
28	Vestibular neuritis	46	M	(+) R	L	13.3	4.6	65

vHIT video head impulse test, *SPEV* slow phase eye velocity, *post HSN neutral* number of fast phase beats in neck neutral spine after head shaking, *post HSN flexion* number of fast phase beats after head shaking and then positioning the head into flexion

storage response is suppressed from nodular inhibition on utricular afferents via the utricle-nodular pathways. For example, when the head is tilted away from an Earth vertical position immediately after stopping a constant velocity head-rotation (earth vertical axis), the rotation axis of the post-rotary nystagmus is re-directed toward the gravitational axis (spatial reorientation) with a reduction in the time constant [3].

The head-shaking nystagmus (HSN) test is considered a useful clinical tool for detecting asymmetries between the vestibular labyrinths and provides some insight into the integrity of the velocity storage system [5]. Shaking the head at 2 Hz oscillation for approximately 20 s in the horizontal plane may cause a horizontal nystagmus where the fast phase beats towards the unaffected labyrinth [6]. This finding suggests a peripheral vestibular hypofunction with duration of nystagmus that can last as long as 6 s post-acute stage [6]. The presence of a vertical nystagmus after a horizontal head shaking typically suggests pathology affecting the central vestibular pathways [7]; nystagmus that is downbeating has been reported as the most common direction after horizontal head shaking in patients with migrainous vertigo [8].

Originally described in 1966, the Head Tilt Suppression Test has been used to probe the integrity of the vestibulocerebellum [9]. Using rotational chair testing and healthy control subjects, Han et al. reported a reduction of the time constant by head tilting in both per- and post-rotary nystagmus conditions [10]. Hain et al. expanded the effect of

Table 2 Patients with central vestibular pathology

Patients	Etiology	Age	Gender	vHIT	Direction of post-HSN	SPEV post-HSN—neutral	SPEV post-HSN—flexion	% suppression
1	Cerebellar	73	F	(−)	DBN	39	39	0
2	Cerebellar glioma	40	M	(+) L and (+) R	R	5.9	10.4	−76
3	Central DBN	70	F	(−)	DBN	1.7	2.2	−29
4	Cerebellar degeneration	27	M	(−)	L	1.5	0	100
5	CVA	62	F	(−)	L	5.7	1.5	73
6	AICA right	72	M	(+) R	L	11	7	36
7	Vascular cavernoma cerebellar left bulge	48	M	(−)	L	10	19	−90
8	AICA Left	63	M	(−)	DBN	7	6	14
9	Vascular cavernoma cerebellar peduncle right middle	26	F	(−)	R	23	19	17
10	Cerebellar hemisphere tumor (right)	37	F	(−)	R	19	19	0
11	Cerebellar peduncle vascular lesion (left)	60	F	(−)	R	14	15	−7

Please see Table 1 for abbreviations

CVA cerebrovascular accident, *AICA* anterior inferior cerebellar artery, *PICA* posterior inferior cerebellar artery, *DBN* downbeating nystagmus

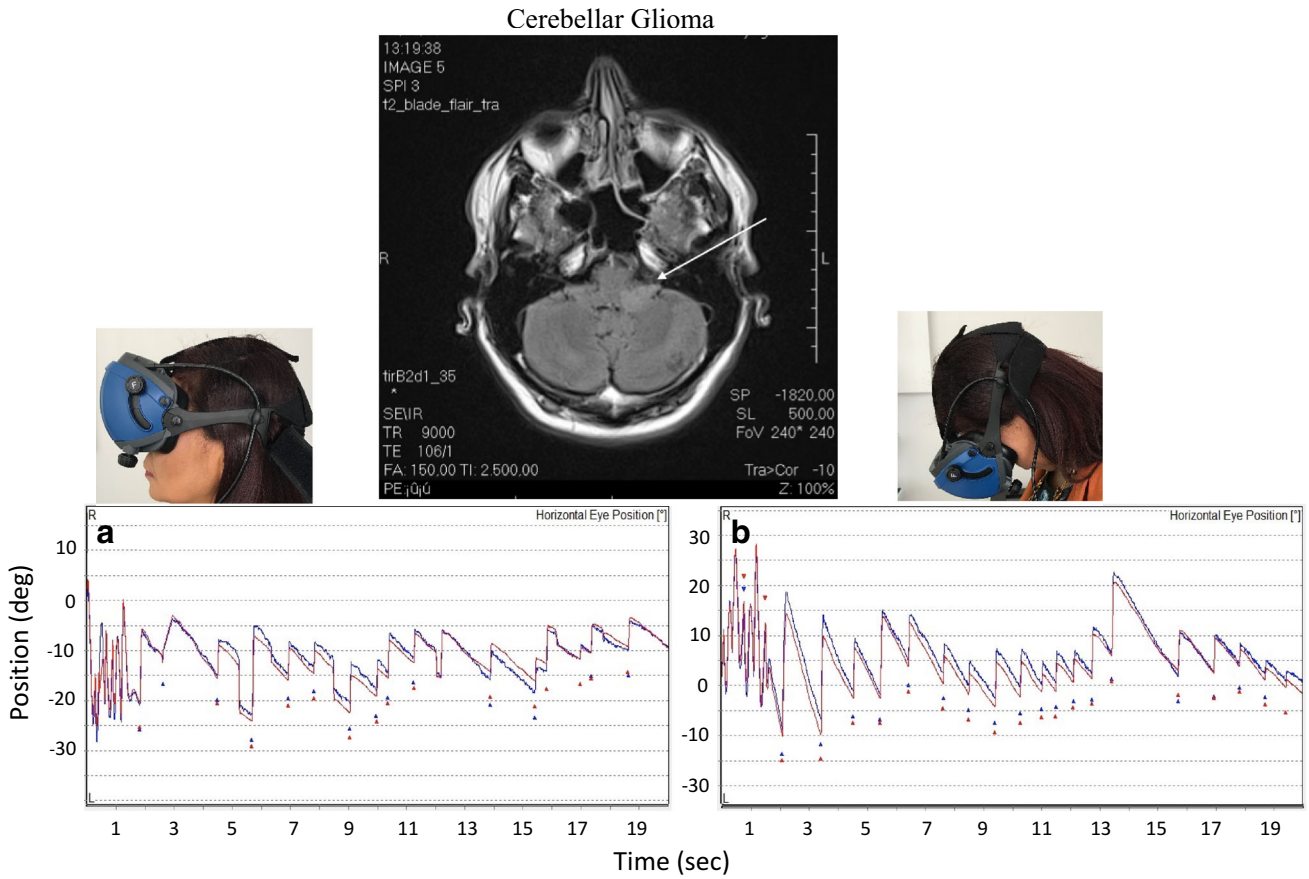

Fig. 1 Limited effect of head tilt on suppressing post-head-shaking nystagmus in cerebellar glioma (*white arrow*) centered in the internal portion of the inferior cerebellar peduncle and extending inferiorly and laterally into the flocculonodular lobule and posterolateral part of the medulla. **a** Right-beating nystagmus post-head shaking with a velocity of 5.9 deg/s. **b** The right beating actually increased to 10.4 deg/s after the HSTS test, illustrating an inability to suppress. The first 2 s illustrate the end of the head shaking

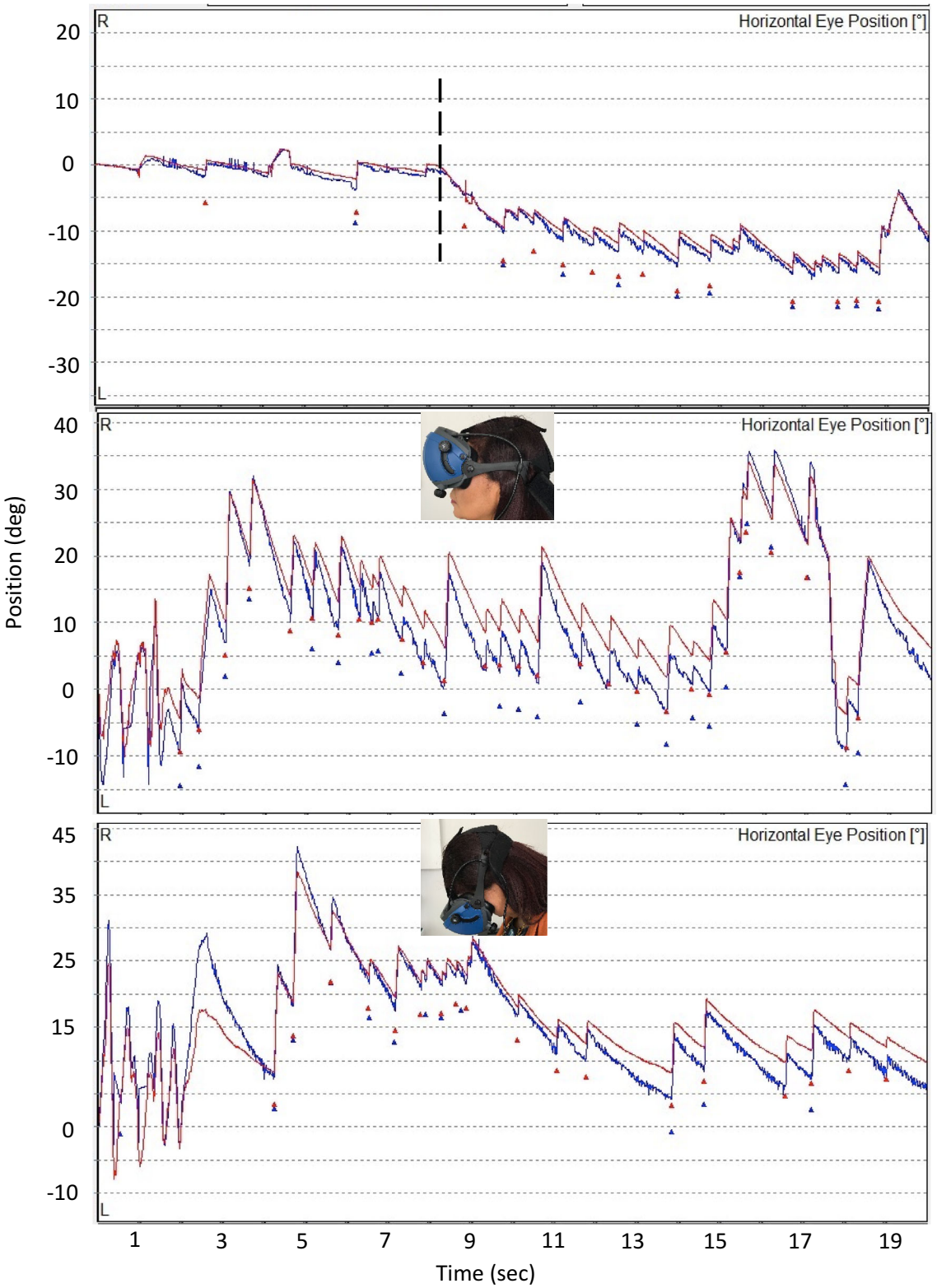

Fig. 2 The spontaneous right-beating nystagmus peaks at 4.5 deg/s once fixation is removed (*vertical stippled line*, *top panel*). After head shaking (*middle panel*), a robust increase in the right-beating nystagmus occurs (13.1 deg/s). After HSTS (*bottom panel*), the right beating is suppressed 42% to 7.6 deg/s. The first 3 s of the *middle* and *bottom panels* illustrate the end of the head shaking

tilt on the time constant of post-rotatory nystagmus in a group of normal subjects and patients with cerebellar lesions. They reported a tilt suppression of 63% in healthy controls, yet patients with lesions near the uvula and nodulus were unaffected by head tilt [11]. Recently, Lee et al. reported that some patients with cerebellar lesions do retain the ability to suppress via tilt, the post rotary chair induced nystagmus (37%), though this was uncommon in those patients with identifiable nodulus and uvular infarction [12].

To date, no study has examined the effect of tilt suppression after head shaking. The aim of the present study was to investigate the clinical utility of combining the HSN test with head tilting to discern the effect of head tilt on an induced nystagmus in two groups of patients; those with peripheral unilateral vestibular hypofunction and those with lesions affecting the central vestibular system. We hypothesized that patients with central pathology would have impairment in head tilt suppression.

Materials and methods

Subjects

Twenty-eight patients (15 female) with peripheral unilateral vestibular hypofunction (UVH) (Table 1) and eleven patients (6 female) with central nervous system lesions (Table 2) were examined using the head-shaking tilt suppression test. Patients with UVH lesions were diagnosed based on:

- Spontaneous nystagmus of a horizontal-only or combined horizontal-rotatory direction independent of the direction of gaze, with fixation removed.
- Absence of benign paroxysmal positional vertigo.
- Reduced vestibular response >25% based on videonystagmography (VNG) or video head impulse test (vHIT). The caloric component of the VNG exam was not performed in all patients.
- Absence of other neurologic findings.
- Audiogram result.

Patients with central nervous system lesions were confirmed on MRI, with the diagnosis aided by clinical and bedside examinations (i.e., spontaneous downbeating nystagmus). All of the central patients had a normal vHIT. Audiograms were used in the differential diagnosis.

All experiments followed the tenets of the Declaration of Helsinki approved by the Brazilian Lutheran University (ULBRA-RS) in Canoas (CAEE 06137012.3.2002.5349). Informed consent was obtained in each subject after the nature and possible consequences of this study had been explained to the participants.

Test procedure: head-shaking nystagmus (HSN) and head-shaking tilt suppression (HSTS)

For the HSN test, the subjects were tested in the sitting position, and their heads were passively rotated in the horizontal plane by an examiner for 10 s at an approximate amplitude of $\pm 10^{\circ}$ between 2 and 3 Hz. Once the head shaking was stopped, the examiner ensured the head remained still for 60 s. For the HSTS test, the patients were instructed to tilt their head forward until the chin rested on their upper thorax, immediately after the passive horizontal head shaking. They remained in this position for 60 s.

A positive test HSN was defined as the presence of at least three beats of nystagmus after stopping the passive head rotation. We determined the induced nystagmus velocity after both HSN and HSTS. We calculated the tilt suppression index (TSI) using the following formula:

$$\mathrm{TSI} = \frac{\text{SPEV after HSN} - \text{SPEV after HSTS}}{\text{SPEV after HSN}} \times 100,$$

where SPEV is the slow phase eye velocity; HSN is the head-shaking nystagmus; HSTS is the head-shaking tilt suppression (flexion of the neck (tilt the head) to rest against the thorax).

Recordings and statistical analysis

Eye and head rotation was recorded using portable video-oculography with capacity to block visual fixation. The high-speed infrared camera (sampling rate of 250 Hz) and goggle frame include a built-in rate sensor (Otometrics, Denmark; Interacoustics, Denmark). Velocity of the induced head-shaking nystagmus was described using conventional descriptive statistics. Differences in the induced nystagmus between HSN and HSTS were analyzed using the two-sample t test assuming unequal variances. Results were considered statistically significant at $\alpha < 0.05$. All statistical analyses were performed in Excel (Microsoft Office 2013).

Results

There was no difference in age between the patients with UVH (mean 52.1 ± 16.8 years) and the patients with central pathology (mean 52.5 ± 17.6 years), $p = 0.96$.

Three of the patients with central pathology developed downbeat nystagmus after the horizontal head shaking that ranged from 1.7 to 39 deg/s, (Table 2). In this group, tilting of the head forward had no effect on the induced downbeating nystagmus (HSN: 15.9 ± 20 deg/s vs. HSTS: 15.7 ± 20.2 deg/s, $p = 0.49$). Another eight patients with central pathology developed either right or left beating

horizontal nystagmus after the HSN test with a mean velocity of 11.2 ± 7.2 deg/s. In this sub-population, tilting the head had no effect in suppressing the induced nystagmus (mean of 11.4 ± 7.9 deg/s, $p = 0.48$). Some patients demonstrated an increased nystagmus after head tilting, Fig. 1. Considering all subjects with central pathology (those with down and horizontal beating post-head-shaking nystagmus), the combined effect of tilting the head on suppressing the nystagmus was limited to 3 ± 56%.

Each of the patients with UVH demonstrated an abnormal head impulse test (mean ipsilesional VOR gain 0.42 ± 0.23; mean contralesional VOR gain 0.85 ± 0.13) and appropriately directed head-shaking nystagmus with magnitudes ranging from 0.3 to 23.5 deg/s (Table 1). The mean HSN velocity for patients with UVH was 8.4 ± 5.4 deg/s. After tilting the head, the patients with UVH had a reduction of nystagmus by a mean of 4.2 ± 3.6 deg/s (Fig. 2). Therefore, the patients with UVH were able to suppress their nystagmus a mean of 52 ± 20% with tilt of the head forward, significantly greater than the suppression in patients with central vestibular pathology ($p = 0.008$).

Discussion

Our data suggest that patients with central vestibular lesions as cause for their symptoms have a reduced ability to suppress nystagmus induced by horizontal head-shaking. Some of the patients with central pathology, however, do retain the ability to suppress the induced nystagmus with head tilting [12]. This suggests a healthier functioning vestibulocerebellum, with lesions elsewhere. Horizontal HSN from central causes is typically due to lesions involving the vestibulocerebellum, which comprise the flocculus/paraflocculus, nodulus and ventral uvula [12]. In monkeys, ablation of the nodulus and uvula leads to prolongation of the velocity storage [13], therefore cerebellar inhibition is impaired. The nodulus and uvula facilitate integration for the aVOR and inhibit the integration that underlies angular velocity storage. The nodulus and uvula are responsible for the reduction in the aVOR time constant that occurs during habituation [3]. Hain et al. reported that patients with a midline cerebellum lesion near the uvula and nodulus showed no decrease in time constant for post-rotatory nystagmus caused by head-forward tilting [11]. However, in that same study normal subjects showed vestibular responses that decayed with a time constant of 19.6 s at upright and 7.2 s after the head was tilted down, for a 63% reduction in nystagmus with tilt, similar to the 56% that we report in patients with healthy cerebellar function yet abnormal peripheral vestibular end organ function. These results suggest that the midline cerebellum regulates neural network signals transmitted from the peripheral end organ to the brainstem.

Conclusion

Our data suggest that tilting of the head forward after first applying horizontal head-shaking (HSTS test) is much less effective at suppressing the induced nystagmus in patients with a central cause for their vestibular-like symptoms. This may have a useful clinical value at the bedside to differentially distinguish vertiginous patients of central vs. peripheral etiologies. The HSTS is a simple test that does not require expensive instrumentation that can be performed by any clinician.

Compliance with ethical standards

Conflicts of interest None of the authors have any conflict of interest to declare.

Ethical standard The studies was approved by the local ethics committee and was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

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