Function tests of the otolith or statolith system

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Purpose of review
This review aims to provide an overview of recent advances in tests to evaluate otolith function over the last 2 years.

Recent findings
Over the last 2 years, many papers have focused on the application of the vestibular evoked myogenic potentials (VEMP). Several aspects are under survey: a search for optimal stimuli, search for normative data, search for which labyrinthine function losses and what kind of pathologies induce abnormal VEMPs. The review shows that some fundamental problems still have to be solved to improve reproducibility and to increase sensitivity. Other research and modelling is performed to find out how the brain distinguishes tilts from translations. Several papers support routine implementation of subjective visual vertical (SVV) measurements (in rest and during centrifugation) in the standard vestibular test battery. Interesting reports mention short latency vestibulo-ocular reflex induced by taps and short auditory stimuli. One report mentions the impact of otolith dysfunction upon spontaneous nystagmus and head shaking nystagmus.

Summary
Although validation is still needed and in progress, the state of the art laboratory should consider the following tests for an evaluation of otolith function as relevant: slow tandem gait, VEMP, SVV during centrifugation.

Keywords
diagnostic test, head shaking nystagmus, SVV, thresholds, VEMP

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Abbreviations

EMG electromyography
SVH subjective visual horizontal
SVV subjective visual vertical
VEMP vestibular evoked myogenic potentials
VOR vestibulo-ocular reflex

1350-7540

Introduction

The otolith system (or statolith system, according to the function) detects linear accelerations, by which it senses head translations in three dimensions. The system also detects head tilts relative to the gravity vector that serves as an absolute reference in space. The maculae utriculus and sacculus are, however, unable to discriminate between translations and tilts because both stimulate the hair cells in a similar way. Many experiments have been performed and theories designed to unravel the secret as to how the brain deals with this ambiguity problem that also complicates the design of sensitive otolith tests [1,2].

In different ways, most recent reports indicate that *canal* information is used together with the otolith input in an internal model in the brain [1,2] to discriminate between translations and tilts depending on the motion frequency [1]. The orientation and magnitude of the perceived gravito-inertial vector are used to calibrate (proprioceptive and visual) orientation systems [3•] and serve as an absolute and crucial reference for balance control and spatial orientation. Function losses of the maculae are counterbalanced and partly compensated for by reweighing or enhancing the visual and proprioceptive inputs [3•] which can mask the loss of otolith function in tests of balance and spatial orientation. In many otolith tests, propriocepsis also contributes to the responses leading to complex interpretation and limited test specificity.

Although there is still no consensus, research predicts and supports the theory that 'all situations which provoke motion sickness are characterized by a condition in which the sensed vertical as determined on the basis of integrated information from the eyes, the vestibular system and the nonvestibular proprioceptors is at variance with the subjective vertical as expected from previous experience' [4]. As motion sickness and space sickness are believed to be related to otolith functionality, this points to the clinical relevance in the design of otolith tests that evaluate the ability to perceive and discriminate tilts and translations precisely.

An important limitation of the vestibular labyrinth and thus of the maculae, is that the translation and tilt are detected by accelerometers so that constant velocities are not sensed. In daily life, this imperfection together with the ambiguity problem is partly counteracted by using other sensory input (predominantly visual and proprioceptive).

Recent literature

Over the last 2 years, several developments regarding otolith function and pathology have drawn attention.

Relation pathology of maculae and canals

In their excellent paper about general vestibular testing, Brandt and Strupp [5••] argue that most vestibular syndromes involve semicircular canal and otolith function losses: 'There are several possible reasons why. The different receptors for perception of angular and linear accelerations are housed in a common labyrinth. Their peripheral (VIIIth nerve) and central (e.g. medial longitudinal fascicle) pathways take the same course. Finally, there is a convergence of otolith and semicircular canal input at all central vestibular levels, from the vestibular nuclei to the vestibular cortex.'

Increasingly, however, we recognize symptoms that might arise from pathology that occurs in a specific isolated part of the labyrinth, canal, sacculus or utriculus. Superior canal dehiscence and benign paroxismal positioning nystagmus are examples of isolated local disturbances. Historically, analogous to Brandt and Strupp's suggested relationship between canals and maculae, it was thought that hearing and vestibular deficits should almost always coincide because of their close anatomical relationship. For this reason, many oto-rhino-laryngologists are still unaware that a vestibular examination is the examination of first choice in a dizzy or balance-impaired patient and not audiometry, despite the limited sensitivity of vestibular tests. Many vestibular deficits can occur without any abnormality of the auditory system and many of them are listed by Brandt and Strupp [5••]. But, correlations between hearing deficits do indeed exist, especially between hearing and otolith function; hearing in congenital deaf children appears to be correlated closely with utricular function, but not related to semicircular canal function, but it seems that all combinations of deficits and normal function of the substructures exist [6].

Tests of balance and gait

Patients with assumed vestibular dysfunction [3•,5••] frequently mention problems with gait and balance, falling and sensations 'like walking on pillows or like a drunken person'. In a very well documented review, Basta *et al.* [3•] indicate the importance of the evaluation of balance and gait in the search for otolith deficits, especially by means of the so-called Standard Balance Deficit Test. According to Brandt *et al.* [7], we are better off running than walking in the case of acute vestibulopathy. It is frequently argued that the otolith system is our primary spatial reference during stance and for learning to balance. At higher speeds propriocepsis and vision take over as primary references, while parallel motor activity becomes progressively pre-programmed (hippocampus) and does not need significant vestibular feedback. The clinical message here is that in the case of vestibular deficits, gait and balance under ideal conditions often show normal. In contrast, function loss and fear of falling show up at low speeds and balance is especially impaired during slow tandem gait, when standing or walking on a soft support phase or in a situation where spatial orientation by visual cues is reduced (dark) or misleading (busy traffic).

Frequency range of the otolith system

The optimal frequency range of the canal function is limited roughly to be within about 0.1–10 Hz [8] with the lowest thresholds being about $0.1°/s^2$ (acceleration threshold) or 3°/sec (velocity threshold). [9]. The frequency range of the otolith system has a complicated behaviour especially because of the ambiguity problem described above. There is a high-pass dynamics (> 1 Hz) for translations (linear vestibulo-ocular reflex) but a low-pass characteristic (< 4 Hz) for head roll and tilt as reflected by the induced eye torsion [10]. The theoretical models implying the overall sensitivity for translations, rolls and tilts indicate sensitivities that range from 0 Hz minimum to 40 Hz maximum for the otolith membrane. To my knowledge, not much is yet known about possible differences regarding the frequency sensitivities between the human sacculus and utriculus.

Perception of translation

Measuring the perception of translation for diagnosis of statolith function losses has a long history, starting with the use of simple parallel swings, to sophisticated linear sleds that allow the application of various stimulus profiles with different frequency compositions in the horizontal plane. Despite the inventive constructions and high technology, however, it remains very difficult to eliminate all other sensory cues that might affect the perception thresholds (e.g. vibration). Even more complicated is the consistent finding that the perception thresholds decrease upon repetition by learning and increase again by fatigue, and vary widely among healthy subjects [11•]. As acceleration thresholds vary with the stimulus profile used to determine the thresholds (sinus, parabolic, linear, steps), it is better that thresholds are expressed in terms of velocity that are less variable with the stimulus profile. Velocity thresholds in healthy subjects range from 3.0 to 36.6 cm/s. whereas patients with bilateral vestibular areflexia may show thresholds only slightly higher than the normal range (> 40 cm/s) [11•]. This aspect prevents an easy, rapid and reproducible application in patients. To my knowledge, there are no recent publications regarding the perception thresholds for vertical translations.

Perception of tilt

Flight simulators are a striking demonstration of the ambiguity in the otolith system: sustained tilts are used to simulate translations supported by appropriate simultaneous optokinetic stimulation [4,9]. From this application it was shown that as long as tilt rate is below the 3°s threshold for the canals, no tilt is perceived. Canals are thus more sensitive for dynamic tilts than the otolith system. It was suggested that somatosensory cues are more prominent than vestibular cues in the perception of verticality [12]. Even in the absence of any otolith stimulation, the illusion of motion can occur as we can experience when we are sitting in a train and look through the window to a passing train. This illustrates the strong impact of cognitive factors and other senses and indicates that at least in conditions with minor linear accelerations, the brain tends to rely more on visual and proprioceptive cues to detect translations. This apparent higher sensitivity of propriocepsis and canals for *dynamic tilt* makes detection of the functionality of the otolith system for dynamic tilts difficult.

The perception of sustained tilt, either induced artificially or perceived by an error in the spatial orientation system, can be assessed in various ways. Detection of the subjective proprioceptive horizontal (analogous to the subjective visual horizontal) is one of them. Here, subjects with eyes closed and bare feet, stand on a tilted platform and are asked to adjust the slope of the platform back to horizontal. Healthy subjects are able to do this within ± 1.0°. Patients with vestibular or proprioceptive function losses (mixed polyneuropathies) show errors of more than 3–7°. With the head erect, subjects with an intact otolith system can position a line horizontally (subjective visual horizontal, SVH) or vertically (subjective visual vertical, SVV) around 0.0° within a standard deviation of about 1.1°. Patients after loss of vestibular function initially show a tilt of the SVV towards the affected side of about 10° decreasing to 2–3° after one year [13,14,15•]. The similarity between the SVH and SVV measurements confirms that either test can be used clinically for patients with vestibular lesions [16•].

The limited sensitivity of the test in centrally compensated patients with partial lesions may explain the limited application in the clinic. Some papers suggest that additional head roll of about 30° enhances the sensitivity of the SVV to identify compensated vestibular losses [17], but many factors play a role when additional body or head roll is applied [18•,19]. Literature shows, however, that the SVH and SVV most likely do not only reflect otolith function, but also depend directly and indirectly (via internal references) on canal and proprioceptive function, and are affected by eye torsion upon head roll [20].

Vestibular evoked myogenic potentials

Since their first description, vestibular evoked myogenic potentials (VEMPs) are now being used by investigators worldwide, and characteristic changes observed with ageing and in a variety of peripheral and central vestibulopathies have been described [21••,22•]. In their review [21••], Welgampola and Colebatch indicate that VEMPs evoked by clicks are a robust, reproducible screening test of otolith function. They report various methods of stimulation and results in case of vestibular neuritis, herpes zoster oticus, Ménière's disease, and vestibular schwannomas, superior semicircular canal dehiscence and central vestibulopathy.

Without a doubt, VEMP develops as a sound clinical test. Some critical remarks can, however, be made which might be of value when starting to apply this technique in your clinic, as these problems are not that clearly mentioned in the literature. The middle ear status affects VEMP amplitudes. Using a bone-conduction stimulus as a solution solves that problem, but does not allow assessing side differences anymore. Reproducibility of VEMP amplitudes is good when studied in one single measuring session, but if attained on different days, reproducibility is poor. VEMP amplitude depends on many factors among which not only saccular function but also on air-conduction (via the middle ear to the round window), electrode conduction and electrode location. Moreover, VEMP amplitude linearly increases with the rectified electromyography (EMG) level, which indicates that control of the contraction state of the sterno-cleido mastoid muscle is crucial to acquire reproducible results and reliable comparison between left and right saccular function is based upon VEMP amplitudes. The procedure to obtain a symmetrical and reproducible contraction state of the neck muscles is not yet standardized and still subject for discussion. Using (visual) feedback of the rectified EMG level through a monitor to attain a constant contraction state does improve the reproducibility and stability of the neck muscle contraction state and rectified EMG amplitude, but surprisingly does not improve VEMP amplitude reproducibility upon repetition over days. VEMP thresholds seem to reproduce better, if sufficient contraction of the neck muscles is achieved. Therefore, for a reliable clinical implementation, it seems to be necessary to use only VEMP thresholds and latencies as relevant output parameters (refrain from use of VEMP amplitudes) in combination with feedback and constant load to control the muscle contraction state.

The mentioned (likely) saccular origin of VEMP is based on animal research and interpretation of patient studies using other vestibular test. A recent paper showed that VEMP could still be induced in a patient despite resection of the inferior vestibular nerve which was explained by the assumption that nerve fibres of saccular origin proceed to the brainstem via both the inferior and superior vestibular nerve [23•].

Eye movements and otolith function

Many methods have been proposed in the past to evaluate otolith function by study of eye movements [24]. Measuring the linear vestibulo-ocular reflex during translations is complex due to the large impact of fixation distance and instruction that are difficult to control in darkness [1,2,10]. Quantifying otolith function by measuring the eye torsion induced by head roll or linear translation came into view thanks to the development of 3-D video eye-trackers but showed no great clinical relevance [25] due to large interindividual variability and small responses. Very elegant centrifugation tests have been suggested by Clarke *et al.* [26], especially the unilateral tests of saccular and utricular function. Displacing the head by 3.5–4 cm from the rotation axis, the eccentrically positioned utricle is stimulated unilaterally by a resultant centrifugal force. This paradigm was employed to elicit eye torsion or to permit measurement of the SVV. This procedure, however, requires substantial investment in equipment and expert knowledge for a reliable application and yet still shows limited sensitivities. Fortunately, Clarke showed that also during normal, on-centre yaw axis rotation, asymmetrics of otolith function could be localized and quantified by means of SVV estimation. This test mode can be more easily integrated into routine clinical testing [26].

Short-latency vestibulo-ocular reflex by otolith stimulation

A remarkable recent report from Jombik and Bahyl mentions possible short latency VOR by vibrational stimuli and short tone burst in humans [27••]. Vibrations of the skull were induced with head taps and with a single period of 160 Hz tone burst on the inion, vertex, and the mastoids while the subjects viewed a target. A vestibulo-ocular reflex (VOR) occurred between 5–20 ms, which seemed to be compensatory to the second phase of the sine wave of vibration impulse, and was greatly diminished/absent in patients with bilateral vestibular deficits and ocular palsies. Patients with unilateral vestibular deficits showed enhancement of vertical VOR amplitude on the side of vestibular loss and/or diminution on the healthy side. The authors suggest that the response might arise from the otolith system.

Otolith input modifies spontaneous nystagmus and head shaking nystagmus

Palla *et al.* [28•] recently indicated the relevance of investigating the impact of head orientation upon spontaneous nystagmus and head shaking nystagmus. They describe an increase of this pathological nystagmus in patients with a unilateral deficit when patients lie on their affected ear in case of an additional loss of otolith function. They explain this by an asymmetric suppression of vestibular nystagmus by unilaterally impaired otolith organs. This might point to a relatively simple test to evaluate the involvement of otolith dysfunction in patients with unilateral vestibular losses. It is generally known that nystagmus velocity and frequency increase when gaze direction is in the direction of the fast phase. As it was previously experienced that patients tend to direct their gaze towards the cushion when lying in a supine position, this might lead to a false positive sign of the impact of gravity upon the observed spontaneous or head shaking nystagmus. This is to be taken into consideration before clinical introduction of the test, as performed by Palla *et al.* [28•].

Conclusion

At this moment, the following tests for an evaluation of otolith function can be considered as relevant: slow tandem gait, VEMP and SVV during centrifugation. Interesting developments are that head taps and tone bursts possibly stimulate the otolith organs and induce a short latency VOR. Also, head orientation (otolith load) seems to affect spontaneous nystagmus and head shaking nystagmus.

Despite these positive developments, the current otolith function tests still have limited sensitivity and specificity; this is why a normal outcome does not prove normal function. In this respect, the patient history remains of utmost importance. Clinical signs of a loss of otolith function that may point to an otolith problem are sensations such as ‘like walking on pillows’, ‘feeling drunk’ or ‘tumbling’ [3•], body lateropulsion, falls (drop attacks), illusions of translatory motion or tilt, derangements of ocular motor and postural, orienting and balancing responses, disturbed spatial orientation abilities [5••]. In line with previous reports and in my clinical experience, the patient with severe disorientation may describe symptoms which sound bizarre [25], and in the past raised doubts over the organic basis of the disease. By use of the various tests, however, we increasingly discover otolith dysfunction and pathology as a cause of these ‘bizarre’ complaints.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:
- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 92).

1 Merfeld D, Park S, Gianna-Poulin C, *et al.* Vestibular perception and action employ qualitatively different mechanisms. I. Frequency response of VOR and perceptual responses during translation and tilt. J Neurophysiol 2005; 94 (1):186–198.

2 Green A, Shaikh A, Angelaki D. Sensory vestibular contributions to constructing internal models of self-motion. J Neural Eng 2005; 2 (3):164–179.

3 • Basta D, Todt I, Scherer H, *et al.* Postural control in otolith disorders. Hum Move Sci 2005; 24 (2):268–279.

In this retrospective study in patients with a minor head injury that complained about persistent unsteadiness, slipping away, tumbling, especially in the dark or upon fast body movements, the outcomes of a large series of vestibular tests were evaluated, including specific otolith tests [centrifugation and various (static and dynamic) tests of balance and gait]. The relatively simple Standard Balance Deficit Test, composed of nine different balance and gait components, was found to be the most sensitive indicator of otolith disorders.

4 Bles W, Bos J, de Graaf B, *et al.* Motion sickness: only one provocative conflict? Brain Res Bull 1998; 47 (5):481–487.

5 Brandt T, Strupp M. General vestibular testing. Clin Neurophysiol 2005; 116
•• (2):406–426.
This paper is a compact, but rather complete, state of the art description of many relevant diagnostic techniques currently used in clinical practice. The paper is especially recommended to update any residents, ear, nose and throat specialists and neurologists who are less aware of the recent developments in the field of diagnosis of vestibular disorders.

6 Tribukait A, Brantberg K, Bergenius J. Function of semicircular canals, utricles and saccules in deaf children. Acta Otolaryngol 2004; 124 (1):41–48.

7 Brandt T, Strupp M, Benson J. You are better off running than walking with acute vestibulopathy. Lancet 1999; 354 (9180):746.

8 Tabak S, Collewijn H, Boumans LJ, van der Steen J. Gain and delay of human vestibulo-ocular reflexes to oscillation and steps of the head by a reactive torque helmet. I. Normal subjects. Acta Otolaryngol 1997; 117 (6):785–795.

9 Groen EL, Bles W. How to use body tilt for the simulation of linear self motion. J Vest Res 2004; 14:375–385.

10 Paige GD, Seidman SH. Characteristics of the VOR in response to linear acceleration. Ann NY Acad Sci 1999; 871:123–135.

11 Kingma H. Thresholds for perception of direction of linear acceleration as a
• possible evaluation of the otolith function. BMC Ear Nose Throat Disord 2005; 5 (1):5.
The median thresholds for the perception of direction of linear movement were minimal 6.5 cm/s^2 (acceleration, with a range of 3–23 cm/s^2) and 10.4 cm/s (velocity, range between 4.8–36.6 cm/s). Subjects needed several stimuli to reach the lowest threshold in contrast to, for example, tone audiometry or visual perimetry. The method applied makes it likely that these thresholds reflect otolith function predominantly.

12 Bronstein A. The interaction of otolith and proprioceptive information in the perception of verticality. The effects of labyrinthine and CNS disease. Ann NY Acad Sci 1999; 28 (871):324–333.

13 Tabak S, Collewijn H, Boumans LJ. Deviation of the subjective vertical in long-standing unilateral vestibular loss. Acta Otolaryngol 1997; 117 (1):1–6.

14 Tribukait A, Bergenius J, Brantberg K. Subjective visual horizontal during follow-up after unilateral vestibular deafferentation with gentamicin. Acta Otolaryngol 1998; 118 (4):479–487.

15 Pinar HS, Ardic FN, Topuz B, Kara C. Subjective visual vertical and subjective
• visual horizontal measures in patients with chronic dizziness. J Otolaryngol 2005; 34 (2):121–125.
This paper supports earlier reports that mention that the SVV and SVH are not only abnormal in the acute stage of vestibular loss, but also in many patients with common dizziness aetiologies in the chronic stage. The authors recommend the regular use of the SVV-test in the clinical practice.

16 Hafstrom A, Fransson PA, Karlberg M, Magnusson M. Idiosyncratic compen-
• sation of the subjective visual horizontal and vertical in 60 patients after unilateral vestibular deafferentation. Acta Otolaryngol 2004; 124 (2):165–171.
This paper confirms that both SVV and SVH can be used clinically for patients with vestibular lesions to evaluate otolith function and that the test results of SVV and SVH are highly correlated (rho = 0.74).

17 Aranda-Moreno C, Jauregui-Renaud K. The subjective visual vertical in vestibular disease. Rev Invest Clin 2005; 57 (1):22–27.

18 Kaptein R, Van Gisbergen J. Interpretation of a discontinuity in the sense of
• verticality at large body tilt. Neurophysiol 2004; 91 (5):2205–2214.
The SVV is considered as a test of otolith function and directly related to the perception of head, or even body tilt. Additional head tilt is mentioned to increase the sensitivity of the SVV test. This study, however, shows a clear dissociation between performance in the subjective-vertical task and the sense of body tilt, at large body tilt angles. This fundamental paper is of special interest for those who want to understand how we sense our orientation in space under various conditions.

19 Hoppenbrouwers M, Wuyts FL, Van de Heyning PH. Suppression of the E-effect during the subjective visual vertical test. Neuroreport 2004; 15 (2):325–327.

20 Pavlou M, Wijnberg N, Faldon ME, Bronstein AM. effects of semicircular canal stimulation on the perception of the visual vertical. J Neurophysiol 2003; 90:622–630.

21 Welgampola M, Colebatch J. Characteristics and clinical applications of
•• vestibular-evoked myogenic potentials. Neurology 2005; 64 (10):1682–1688.
This is a well documented review about VEMP covering major publications dealing with the origin of the reflex, the required test conditions, analysis methods, describes the results in patients with vestibular neuritis, herpes zoster oticus, late Ménière's disease, and vestibular schwannomas, superior semicircular canal dehiscence including the Tullio phenomenon, the monitoring of the efficacy of intratympanic gentamicin therapy, central vestibulopathy, differentiation of labyrinthine from retrolabyrinthine lesions.

22 Basta D, Todt I, Ernst A. Normative data for P1/N1-latencies of vestibular
• evoked myogenic potentials induced by air- or bone-conducted tone bursts. Clin Neurophysiol 2005; 116 (9):2216–2219.
VEMP latencies obtained in 63 healthy subjects show no significant differences between female and male volunteers or between air and bone-conducted stimulation, and did not differ with age. Normal latencies (mean $\pm$ 2 SD) are maximal 20.3 ms for P1 and 28.0 ms for N1.

23 Brantberg K, Mathiesen T. Preservation of tap vestibular evoked myogenic
• potentials despite resection of the inferior vestibular nerve. J Vestib Res 2004; 14 (4):347–351.
This paper informs more about the origin of the VEMP. In a patient with a small acoustic neuroma the caloric exitability was preserved after surgery that was believed to spare the superior vestibular nerve function but to abolish inferior vestibular nerve function. In response to sound stimulation there were no VEMPs on the operated side, irrespective of the way sounds were presented. VEMPs were, however, still obtained in response to forehead skull taps suggesting that, at least partly, these depend on the superior vestibular nerve function. These findings still agree with assumption that VEMP is of saccular origin.

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25 Kingma H. Clinical testing of the statolith-ocular reflex. ORL J Otorhinolaryngol Relat Spec 1997; 59 (4):198–208.

26 Clarke AH, Schonfeld U, Helling K. Unilateral examination of utricle and saccule function. J Vestib Res 2003; 13 (4–6):215–225.

27 Jombik P, Bahyl V. Short latency responses in the averaged electro-oculogram
•• elicited by vibrational impulse stimuli applied to the skull: could they reflect vestibulo-ocular reflex function? J Neurol Neurosurg Psychiatry 2005; 76 (2):222–228.
Monoaural air conducted tones elicit bi- or triphasic transient vertical electro-oculography responses with a duration of about 10 ms and a 7–8 ms latency in 16 of 40 tested volunteers and in the patient with congenital deafness. In patients with acoustic neuromas, the responses were induced only by stimuli to the healthy ear. In the remaining 24 healthy subjects, the responses were absent or barely discernible. Although these results need verification by use of the search coil technique to measure the precise eye movements in order to exclude artefacts, the study is impressive, the findings are consistent and should stimulate researchers to explore this short latency evoked VOR.

28 Palla A, Marti S, Straumann D. Head-shaking nystagmus depends on gravity.
• J Assoc Res Otolaryngol 2005; 6 (1):1–8.
In acute unilateral peripheral vestibular deficit, horizontal spontaneous nystagmus increases when patients lie on their affected ear. In this study the authors show an analogue phenomenon: also head shaking nystagmus depends on head position. Using a three-dimensional turntable, patients were oscillated (1 Hz $\pm$ 10°) about their head-fixed vertical axis and head shaking nystagmus was modulated, interpreted as a modulation due to stimulation of the otolith organs by the tumbling gravity vector and allowing the of this paradigm to evaluate otolith function.