Introduction

In 1985, the Royal Belgian Society for Ear, Nose, Throat, Head and Neck Surgery and the Belgian Professional Union of Ear, Nose, Throat, Head and Neck Surgery founded the Otoneurological and Expertise Commission.

Professor R. Boniver has been the president since.

At present, the members of the commission are Professors Christian Desloovere, Naïma Deggouj, Floris Wuyts and Doctors Sarah Casteleyn, Stéphane Dejardin, Anne Englebert, Chantal Gilain, Catherine Hennaux, Christian Van Nechel.

In 1986, the publication in the Acta Oto-Rhino-Laryngologica Belgica of expert recommendations for ear, nose and throat specialists introduced the topic of vestibular exploration.

In the absence of evidence-based medicine for this specific subject and with the encouragement of Doctor Robillard, the commission started in December 2003 to draft guidelines for the exploration and treatment of vertigo and dizziness. Their recommendations are based both on the experience of the members of the commission and on scientific advances relating to vertigo and dizziness.

This supplement contains specialist articles describing tests and examination approaches. Asterisks refer to annexes.

The guidelines are based on more recent oto-neurological findings and will be updated in the future.

References in the annex will give readers the opportunity to explore this subject in greater depth.

We hope this paper will bring you fruitful and enjoyable reading.

Guidelines on vertigo and dizziness

1. Patient history

1.1. *Vertigo or dizziness*

- Description (rotatory vertigo, horizontal or vertical linear sensations, postural imbalance)
- Start, duration, frequency
- Provocative event (e.g. position, orthostatism, spontaneous, Valsalva, Tullio)
- Initial manifestations
- Autonomic symptoms
- Gait: quality and perturbation factors
- Direction of body tilt or imbalance (lateral, posterior)
- Falls: circumstances (current occupations, situation)

1.1.1. Visual influence

- Mobile environment intolerance
- Acrophobia

1.1.2. Agoraphobia, Anxiety (HAD and PHQ scale in annex)

1.1.3. Effect on life quality evaluation (DHI scale in annex)

1.2. *Otological symptoms* (for each symptom, check laterality and temporality with vertigo)

1.2.1. Hypoacousia or hyperacousia, fluctuating hearing, diplacousia, distorsion

1.2.2. Tinnitus: continuous, pulsating, positional

1.2.3. Hearing fullness or pressure

1.2.4. Otalgia

1.2.5. Otorrhea

1.3. *Visual manifestations*

1.3.1. Amaurosis

1.3.2. Horizontal or vertical diplopia

1.3.3. Oscillosia

1.3.4. Visual field inversion

1.3.5. Refraction correction related

1.4. *Neurological manifestations* (precise temporality with vertigo)

1.4.1. Migraines, headache and facial pain

1.4.2. Sensitive and motors manifestations (e.g. precision in movement of upper limbs)

1.4.3. Symptoms related to other cranial nerve disorders

1.4.4. Symptoms related to cervical spine disorders (e.g. cervicalgia)

1.5. *Prior history*

1.5.1. Hereditary (according to current pathology study)

1.5.2. ENT

1.5.3. Neurological

1.5.4. Traumatic

1.5.5. Cardiovascular and vascular risk factors (hypertension, diabetes, cholesterol, smoking)

1.5.6. Metabolic and hormonal

1.5.7. Infectious

1.5.8. Immunological

1.5.9. Locomotor (rheumatological, orthopedic)

1.5.10. Strabismus, amblyopia, multifocal refracted lenses

1.5.11. Gait habits (lack of activity, chronic lying position ...), sport (diving ...)

1.5.12. Occupation

1.5.13. Toxic (drugs, professional, alcohol, smoking)

1.6. *Treatment*

- Current, recent modification
- Prior (ototoxic)

- Physiotherapy, cervical manipulation, vestibular training or repositioning manoeuvres (further details required)

2. Clinical examination

2.1. *Otorhinological*

2.1.1. Otomicroscopic examination

2.1.2. Rhinological examination depending on symptoms

2.2. *Oculomotor and nystagmus*

2.2.1. Visual control test

2.2.1.1. Gaze holding ability

2.2.1.2. Vertical or horizontal ocular misalignment

2.2.1.3. Restriction in ocular amplitude movements

2.2.1.4. Smooth pursuit and saccade testing

2.2.1.5. Inhibitory testing of vestibulo-ocular reflex (VOR)

2.2.2. Halmagyi test

2.2.3. With videoscopic or Frenzel glasses (without fixation)

2.2.3.1. Spontaneous and other gaze holding abnormalities

2.2.3.1.1. Vestibular nystagmus

2.2.3.1.2. Non-vestibular nystagmus

2.2.3.2. Positioning nystagmus (to be conducted at the end of the clinical evaluation)

2.2.3.2.1. Methodology (patient sitting, head to knees, supine, 90° lateral rotation of the whole body and head to the right, and then to the left, supine + head rotating, Hallpike or Brandt and Daroff, Rose, not necessarily in this order)

2.2.3.2.2. Clinical significance (diagnostic criteria)

2.2.3.3. Horizontal and vertical head shaking test

2.2.3.4. Dynamic visual ability

2.3. *Other cranial nerves*

- Face sensitivity defect (if neurinoma is suspected, complete facial sensitivity exploration, front pain sensitivity and corneal reflex included)
- Claude Bernard Horner's sign
- Face and oropharyngolaryngeal sensitivity

2.4. *Limbs*

2.4.1. Cerebellar signs in upper limbs (dysmetria, adiadocokinesia)

2.4.2. Sensation or motor defect in lower limbs

2.5. *Stato-kinetic tests*

2.5.1. Index test, finger pointing test

2.5.2. Romberg's test (standard or enhanced)

2.5.3. Unterberger or Fukuda

2.5.4. Standard gait and star gait tests

2.5.5. Gait exploration

2.5.6. Dynamic Gait Index

3. Diagnostic Progression

3.1. *Isolated Vertigo*

3.1.1. Isolated positioning vertigo

3.1.1.1. Positioning vertigo: 1st episode

3.1.1.1.1. If history evocative of benign paroxysmal positioning vertigo (BPPV):
otomicroscopy and hearing test;
search for the pathological canal;
execution of the repositioning manoeuvre.
After one week, check:
If asymptomatic: end of investigation
If residual symptoms persist after 2 or 3 repositioning manoeuvres: see 3.1.1.1.2.

3.1.1.1.2. If history and clinical presentation "atypical"

Baseline explorations: complete clinical examination (see chapter 3), hearing test, Brainstem Evoked Response Audiometry (BERA), Videonystagmography (VNG) Electronystagmography (ENG) + oculomotricity, subjective visual vertical perception test (SVV), Vestibular Evoked Myogenic Potentials (VEMP)

3.1.1.2. Positioning vertigo: relapse

Baseline exploration (seen in 3.1.1.1.2) + temporal bone scan if conductive hearing loss

3.1.2. Non-positioning isolated vertigo

3.1.2.1. If baseline exploration (see 3.1.1.1.2.) non-contributive: review patient history and test:

metabolic exploration (glycaemia and thyroid)
cardiovascular exploration
psychological exploration (anxiety, phobia ...)
migraine event

3.1.2.2. If baseline exploration suggests labyrinthic pathology (see VNG or ENG criteria)

Study of peripheral vestibular aetiological pathology:
If no result: VEMP to exclude inferior vestibular neuritis.
If cardio-vascular risk: exploration

3.1.2.3. If baseline exploration identifies non-labyrinthic pathology

(see VNG or ENG, BERA, oculomotricity criterias)
neurological exploration
specific neurological imaging

3.2. *Vertigo and hearing signs*

In any case, baseline exploration: hearing test, fistula test, BERA, VNG or ENG + oculomotricity, VVS, VEMP

3.2.1. Conductive hearing loss

tympanometry + acoustic reflex
temporal bone TDM if otosclerosis suspected, aqueduct dilatation, superior canal dehiscence syndrome ...

3.2.2. Perceptive hearing loss

tympanometry + acoustic reflex (level of reflex, "reflex Decay" test RDT)
supraliminar test
otoacoustic emissions

temporal bone and pontocerebellar angle MRI if retro-cochlear lesions suspected
(ECOG if Ménière's disease is suspected)
genetic investigation if familial history (DFNA9)

3.3. *Vertigo and neurological symptoms*

3.3.1. Vertigo and headache or facial algia

3.3.1.1. Patient with unusual vertigo and brutal headache
= Emergency (unusual intensity and localisation)
Exploration should be conducted within hours.

3.3.1.1.1. Latero-cervical pain
Look for vertebral dissection (MRI)

3.3.1.1.2. Occipital pain
Look for:

- expansive lesion of posterior fossa (infratentorial tumor, blood collection ...) (TDM)
- Arnold-Chiari decompensation (MRI)
- basilar aneurism (TDM)

3.3.1.2. Vertigo and usual known headache

3.3.1.2.1. Vestibular migraine
personal and familial history
usual provocative events like migraines

3.3.1.2.2. Anxious tension headache and vertigo
cervicalgia, whiplash
imbalance without vertigo

3.3.2. Vertigo, imbalance and visuals symptoms

3.3.2.1. Ocular disalignment or diplopia

3.3.2.1.1. horizontal

3.3.2.1.1.1. convergent

- nuclear or post nuclear VI nerve lesion
- somewhere near vestibular nuclei
- orbital trauma
- convergent spasm (post-traumatic)

3.3.2.1.1.2. divergent

- mesencephalic lesion or nerve III
- orbital lesion

3.3.2.1.2. vertical

3.3.2.1.2.1. skew, ocular tilt reaction
vertical saccades palsy in sub-thalamic lesions near otolithic pathway

3.3.2.1.2.2. nerve IV lesion (post-traumatic in 30%)

3.3.2.2. Non-vestibular nystagmus and oscillopsia

- gaze-evoked nystagmus
- acquired pendular nystagmus
- flutter, opsoclonus
- congenital nystagmus (idiopathic, latent non-compensated)
- oculomotor palsy (loss of vestibulo-ocular gain)

3.3.2.3. Excessive visual dependence
(generally after vestibular deficiency)

3.3.2.4. Post-refraction change
- multifocal lenses
- major and recent refraction correction

3.4. *Other vertigo*

3.4.1. Child vertigo
As adult specifications but particular focus on:
- serous otitis
- familial history of migraine
- tumours are more frequent
- food
- familial stress
- BPPV less frequent before 10 years of age

3.5. *Imbalance without vertigo*

3.5.1. Imbalance with or without hearing loss, without any neurological sign

3.5.1.1. Drug side-effect or interference (local or general), ototoxicity

3.5.1.2. Haemodynamic disorders
- blood pressure
- arrhythmia

3.5.1.3. Metabolic disorders
- diabetes
- dysthyroidia
- suprarenal dysfunction

3.5.1.4. Genetic (DFNA9 – COCH gene ...)

3.5.1.5. Anxiety, agoraphobia

3.5.2. Combine with neurological defect
Neurological exploration must be conducted

4. Laboratory examination (in accordance with §4 Diagnostic criteria indications)

4.1. *Hearing test*
Tonal, vocal, supraliminar, depending on pathology

4.2. *Tympanomatry/ Stapedial (acoustic) reflex*

4.3. *Auditory brainstem response*

4.4. *Electrocochleography (if Ménière's disease or perilymph fistula suspected)*

4.5. *Otoacoustic emissions*

4.6. *Vestibular evoked myogenic potentials (VEMP)*

4.7. *VNG or ENG* (normative data)

4.7.1. Gaze holding in primary and lateral positions under fixation (20 to 30° maximum)

4.7.2. Exploration for spontaneous and positional nystagmus without fixation

4.7.3. Ocular pursuit

4.7.4. Saccade analysis

4.7.5. Optokinetic pursuit

4.7.6. Rotatory/pendular tests

4.7.7. Caloric test

4.8. *Vertical or horizontal visual perception test*

4.9. *Posturography*

4.9.1. Static

4.9.2. Dynamic

4.10. *Vibratory nystagmus*

4.11. *Otolithic linear and rotatory test*

4.11.1. Excentric rotation test

4.11.2. OVAR

5. Treatment Strategy

5.1. *Medical treatment*

5.2. *Vestibular rehabilitation: soon in B-ENT (Symposium in November 2005)*

5.3. *Psychological approach*

5.3.1. Anxiolytic

5.3.2. Relaxation

5.3.3. Behavioural

5.3.4. Psychotherapy

5.4. *Surgical treatment*

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Head-shaking nystagmus

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Key-words. Head-shaking nystagmus; vestibular dysfunction test

Abstract. *Head-shaking nystagmus.* Head-Shaking Nystagmus (HSN) is a latent spontaneous vestibular nystagmus provoked by rapid passive head shaking around a vertical axis.
HSN is not specific in distinguishing peripheral hypofunction from more central vestibular imbalances.
This test is an excellent bedside test for detecting unilateral vestibular hypofunction but further rotatory and caloric testing will be necessary to clarify the patient's condition.

Introduction

Head-shaking nystagmus is a latent spontaneous vestibular nystagmus. It is provoked in a seated patient by rapid passive head shaking around a vertical axis. Frenzel's glasses in a dark room or a video camera (videonystagmoscopy) show no spontaneous nystagmus after head shaking in normal subjects. In patients with peripheral and central vestibular lesions, however, passive head shaking is a powerful way of activating spontaneous nystagmus.

Methods

HSN is elicited by encouraging vigorous, approximately sinusoidal, head shaking for 15-20 seconds. When patients stop shaking their heads, the nystagmus can be observed with Frenzel lenses. Invariably, a transient (5-20 seconds) but relatively brisk nystagmus is found, with the slow phases being initially directed towards the impaired ear. This nystagmus is followed by a much longer but lower-amplitude nystagmus with slow phases directed away from the impaired ear. Vertical head shaking also induces a horizontal nystagmus but the primary phase will then be directed away from the impaired ear. The reversal phase is small or absent.

Results

HSN has been studied for years. The most important papers were selected from the extensive literature on the subject.

In 1986, Takahashi[1] studied biphasic HSN (b-HSN) in nineteen patients using electronystagmography. Sixteen of these patients had unilateral peripheral vestibular disturbance. As in Kamei's[2] study, the first phase beats towards the healthy side and the second phase towards the damaged side in thirteen patients in this group (81%). In the remaining three cases (19%), the first phase beats towards the damaged side, and the second phase towards the healthy side. This contradicts Kamei's findings, in which b-HSN was also observed in three cases of central vestibular disturbance, indicating that b-HSN occurs not only in cases of peripheral vestibular disturbance but also in cases of central origin.

In 1987, Hain *et al*.[3] used the scleral eye coil technique to study nystagmus, finding HSN in sixty subjects with unilateral peripheral vestibular lesions.

Horizontal head shaking elicited horizontal nystagmus with slow phases that were initially directed towards the side of the lesion and upwards. All subjects showed a prolonged lower-amplitude reversal phase after the initial response following horizontal head shaking.

In 1989, Wei *et al*.[4] studied 108 patients referred for caloric testing and found that HSN is not as powerful a test as canal paresis for the detection of lesions of the 8th nerve.

In 1990, Takahashi *et al*.[5] evaluated horizontal HSN in 85 patients complaining of dizziness and vertigo. This was done by comparing the horizontal head-shaking test with routine rotatory and caloric vestibular testing.

They found that HSN evoked by horizontal head-shaking is a highly sensitive way of detecting unilateral vestibular hypofunction. Except in patients with additional central vestibular imbalance or in patients with Ménière's disease,

the direction of horizontal HSN is highly significant, indicating the side of the lesion, with the fast phase beating towards the intact side. However, horizontal HSN is not specific for distinguishing peripheral hypofunction from more central vestibular imbalances. Peripheral vestibular hypofunction, as well as a central asymmetry of the vestibular velocity storage mechanism, can each produce horizontal HSN, either separately or in combination. So the head-shaking manoeuvre is an excellent bedside test for detecting unilateral vestibular hypofunction, but further rotatory and caloric testing is still necessary to clarify the patient's condition.

In 1991, Burgio *et al*.[6] evaluated HSN in 115 patients with vestibular lesions. The data indicate that, with passive head movement, the head-shaking nystagmus test is neither sensitive nor specific enough for use as a screening test for vestibular loss.

In 1992, Hall *et al*.[7] studied a series of 340 patients and 20 controls to compare the vestibular test data with HSN. HSN appears to reflect the underlying spontaneous nystagmus and its direction has no relationship to the side of the vestibular asymmetry.

In 1993, Fujimoto *et al*.[8] conducted a prospective analysis of a series of patients who underwent a head-shaking test during routine ENG. The incidence of head-shaking nystagmus (HSN) in a dizzy population was relatively high (31.7%) when compared with other "abnormalities" in the routine ENG test battery.

Incidence rates in active and passive head-shaking tests are also similar. When present, different types of HSN were identified (monophasic (76.8%), biphasic (22.7%) and triphasic (0.5%)). In some cases, reversals of the expected "normal" pattern occurred. A high correlation was found between a positive head-shaking test and the presence of spontaneous nystagmus, positional nystagmus and caloric test abnormalities.

In 1997, Asawavichianginda *et al*.[9] analysed a group of 1300 patients with a clinical diagnosis of peripheral vestibular disorder. There was a positive correlation of HSN in patients in the pathology group compared with normal control subjects.

In 1997, Tseng and Chao[10] compared HSN and bithermal caloric tests with ENG to determine the sensitivity of the two tests for vestibular dysfunction in 258 patients. The normal limit adopted for canal paresis was 20%. These authors found HSN to be more sensitive than canal paresis. The sensitivity of HSN for canal paresis was 90%.

In 2000, Katsarkas *et al*.[11] demonstrated that the lability of the direction of the initial phase of HSN is due to the reflection of interactions between two main time constants associated with "velocity storage" and "gaze holding" in the vestibular central processes.

In 2002, Guidetti *et al*.[12] examined 420 patients with vestibular diseases of different origin: peripheral, central, or both central and peripheral. They concluded that the sensitivity of the head-shaking test is actually poor, especially over time, and that it should therefore not be used alone in follow-up for patients with vestibular disease.

In 2004, Iwasaki *et al*.[13] compared the incidence of HSN with the value for canal paresis (CP) obtained from a caloric test. The HSN test is not very sensitive but is acceptable as one of the screening tests for detecting asymmetric vestibular dysfunction.

In 2004, Palla *et al*.[14] demonstrated that HSN in patients with chronic unilateral vestibular deficit following vestibular neuritis is influenced by gravity.

In 2004, Perez *et al*.[15] studied the characteristics of horizontal HSN and its relationship with vestibular dysfunction. They found no correlation between HSN and clinical patterns.

Discussion

Head-shaking nystagmus (HSN) has been recognised for many years. It refers to the observation that patients with vestibular lesions of either peripheral or central origin may show a transient increase in or emergence of a spontaneous nystagmus after a period of vigorous head shaking. This nystagmus has traditionally been ascribed to the activation of a latent vestibular imbalance.

Three processes are invoked to explain the presence and the direction of the two phases of HSN according to Zee.[16]

Ewald's second law is of primary importance. It states that, for high velocities of head rotation, excitation is a more effective stimulus than inhibition. This asymmetric response occurs because vestibular afferents are silenced/driven into inhibitory cut-off at a velocity of head rotation that is lower than that which leads to saturation during excitation. The effect of Ewald's law is most apparent when the head is positioned so that the plane of the particular semicircular canal

being tested is parallel to the plane in which the head is rotating. In the case of an absent labyrinth, the increase in peripheral vestibular activity that is relayed centrally with rotation towards the good ear (the excitatory direction) is greater at high speeds of rotation than the decrease in vestibular activity that is relayed centrally with rotation towards the bad ear (the inhibitory direction). This non-linear property of the labyrinthine response forms the basis for using high-speed rotational stimuli to detect unilateral peripheral vestibular lesions. Furthermore, to probe the function of a particular pair of semicircular canals, the head should be positioned with the plane of the canals parallel to the plane in which the head is rotating.

With rapid head-shaking, the non-linearity described by Ewald's second law leads to a continual, asymmetric increase and decrease in activity that is relayed to the central velocity storage mechanism. Consequently, there is an accumulation of activity for slow phases directed towards the impaired ear. When the head stops shaking, the velocity storage mechanism gradually discharges, leading to a slowly decaying nystagmus with slow phases directed towards the bad ear. To account for the reversal phase of HSN, we postulate a short-term adaptive mechanism, comparable to that which produces the reversal phase of caloric or of post-rotatory nystagmus in normal individuals.

The combination of Ewald's second law, asymmetric velocity storage and adaptation gives a plausible explanation for the pattern of horizontal nystagmus that occurs after head shaking in patients with a unilateral peripheral vestibular loss. Note that this hypothesis predicts that head rotations of low velocities should not lead to HSN, because Ewald's law should only become apparent when the speed of rotation is high. HSN caused by more central lesions, such as asymmetries in the velocity storage mechanism itself, might appear when the speed of head rotation is low. Finally, if velocity storage is relatively ineffective, as indicated by a low VOR time constant, the primary phase of HSN will be shorter and the reversal phase will emerge sooner.

What is the origin of the horizontally directed component of the nystagmus induced by vertical head shaking? The most likely explanation is that, in normal individuals, excitation of the vertical semicircular canals also contributes to the generation of horizontal slow phases of nystagmus. This "cross-coupling" between activities in the vertical semicircular canals and horizontal nystagmus arises from the geometrical arrangement of the semicircular canals within the head.

The lateral canals are pitched upwards about 30° and the vertical canals are tilted backwards correspondingly. Consequently, when the head is oriented in certain directions with respect to the axis of rotation, the vertical canals contribute to the generation of horizontal nystagmus and the lateral canals to the generation of torsional nystagmus. In fact, when the head is pitched 60° upwards, and the body is rotated around an earth-vertical axis, a significant horizontal component of the VOR (about 50% of that with the head upright) is still generated – as it should be – even though peripheral vestibular activity with this head orientation arises almost exclusively from the vertical semicircular canals.

One must also remember that excitation of the vertical semicircular canals leads to ipsilaterally directed horizontal slow phases and that rotation around an earth-vertical axis with the head upright leads to inhibition of activity from the vertical canals in the same labyrinth in which the lateral canal is being excited. Accordingly, during vertical head shaking by a patient with only one functioning labyrinth, activity for horizontal nystagmus accumulates in central velocity storage. After vertical head shaking, there is a transient horizontal component of nystagmus with slow phases directed towards the intact ear.

Conclusion

HSN is useful as a "first-line" examination in the evaluation of dizzy patients, particularly when other vestibular tests are impossible.

It is generally admitted that HSN is not sensitive since it is elicited in only 30-40% of patients with a unilateral vestibular deficit.[15]

HSN is also considered non-specific because the existence of positional HSN has been described in 50% of healthy control subjects, as well as in patients without detectable vestibular asymmetries in functional studies.[7,9]

HSN is also found in cases of central vestibular lesions.

HSN has to be interpreted with caution, as one element among others in the diagnosis of vestibular disease.

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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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Neuro-ophthalmological symptoms in vertigo and dizziness

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Abstract. *Neuro-ophthalmological symptoms in vertigo and dizziness.* The vestibular and visual systems are closely linked in the genesis of vertigo and dizziness. An examination of these two systems is helpful in the search for an aetiological diagnosis. In ENT, this double approach can also help to avoid certain ophthalmological pitfalls such as the mistaken idea that a squint cannot be of vestibular origin, that the absence of diplopia symptoms is enough to exclude any recent oculomotor paresis, or even that eyelid asymmetry is not relevant to diagnosing dizziness.
This paper is intended to help in understanding the neuro-ophthalmological aspects of the guidelines. It is sometimes limited to defining certain terms. However, on the whole, it covers diagnostic procedures.

1. Skew deviation and vertical diplopia of vestibular origin

A vertical diplopia (one image above the other) may be the consequence of a lesion in the vestibular system, including a lesion restricted to the labyrinth. The otolithic system contributes to the control of vertical eye movements and alignment. In mammals with lateral vision, a head tilt induces an upper movement of the ipsilateral eye and a lowering of the contralateral eye. The modification of eye-muscle implantation associated with the shift to frontal vision added a torsional (ocular rotation around the visual axis) action to the vertical muscles. So a head tilt in humans induces a torsional movement of both eyes with an amplitude of a few degrees, the "counter-rolling reflex". Normally, this reflex does not result in any vertical misalignment between the eyes because of accurate balance in the antagonist vertical eye muscles. A dysfunction in any structure associated with this otolithic reflex, from the labyrinth to the eye muscles, through the vestibular nuclei and the mesencephalic nuclei of Cajal, can provoke a vertical misalignment with torsion of the eyes and vertical diplopia if the patient has binocular vision. This vertical misalignment with torsion of the eyes consecutive to a lesion of the vestibular system is called "skew deviation". The lower eye is ipsilateral to a labyrinthic or vestibular nucleus lesion, and contralateral to a lesion located higher on the otolithic pathways. The impairment of the otolithic system means that this skew deviation is often combined with a spontaneous head tilt, body laterodeviation and an error in the estimation of the visual vertical. This complete otolithic syndrome is called "Ocular Tilt Reaction" (OTR).[1]

Horizontal diplopia is never a consequence of a direct lesion of the vestibular system. However, it can result from impaired structures very close to the vestibular pathways and so contribute to the localisation diagnosis.

Let us remember that an ocular misalignment does not necessarily imply diplopia. Monocular amblyopia, alternating fixation or, paradoxically, a significant angle between the ocular axes can eliminate the diplopia. A contralateral head tilt with a normal counter-rolling reflex can also sometimes counteract vertical diplopia.

There are two simple ways of detecting ocular misalignment. The first one consists of observing the reflection of a lamp in both pupils. The position of this reflection should remain relatively stable during gaze deviations. The second consists of placing a coloured filter in front of one of the patient's eyes. To exclude an eye misalignment, we check that the patient does not see two different points in binocular vision, while one point is indeed seen when we hide each eye. This last method can lead to some false positive results when there is phoria or a lack of binocular fusion. These two techniques will allow for the easy detection of a vertical diplopia.

2. Nystagmus

This paper will not provide details about the most likely localisations depending on the manifestations of all the different types of nystagmus. Several syntheses of the contribution of nystagmus to this localisation diagnosis are available.[2,3] From the physiopathological point of view, it should be remembered that, although there are three mechanisms responsible for nystagmus, only the first is of vestibular origin.

a) Nystagmus associated with impaired vestibulo-ocular reflex
These deficits are characterised by both eyes being "pulled in one direction", with this movement corresponding to the slow phase of the nystagmus. This direction depends directly on the site of the lesion. The plane orientations of the semicircular canals are close to those of the oculomotor muscles. Each canal has a privileged relation with the muscle that moves the eye in a direction opposite to the head movement stimulating that canal. Ocular muscle implantation precludes a purely vertical eye movement through the stimulation of a single vertical muscle. The movement will always be around a vertical axis associated with a torsional movement of the eyeball. It is reasonable to assume that this association excludes the possibility of nystagmus with a purely vertical or rotatory slow phase caused by a unilateral peripheral lesion. At the central level it is necessary to keep clearly in mind that the conjugate horizontal eye movements are, in essence, organised at the level of the pons. It is at this level that we find the abducens nucleus (VI), the starting point for the stimulation of the lateral rectus muscle and the ascending pathway (medial longitudinal fasciculus) which stimulates the motoneurons of the medial rectus muscle at the level of the common oculomotor nucleus. Oculomotor nuclei responsible for vertical and torsional eye movements are located higher in the brainstem, at the level of the midbrain. As a result, nystagmus of central origin associated with a lesion situated at the brainstem input level in the vestibular pathways (ponto-medular level) will be horizontal or – less frequently – vertical, whereas a lesion at the mesencephalic level will usually result in vertical, torsional and – rarely – horizontal binocular nystagmus.

b) Nystagmus resulting from inability to maintain one or both eyes in an eccentric position
Here, elastic elements try to return the eye to the primary position, resulting in the slow phase of the nystagmus, which will therefore change direction according to the position of eyes. Accordingly, there will be right horizontal nystagmus when looking to the right, left nystagmus in left gaze, up-beat nystagmus when looking up, and down-beat nystagmus in the reverse gaze direction. The inability to maintain one or both eyes in an eccentric position can result from a muscular paresis or a weakness of the neurological structures commanding ocular muscles. When a nystagmus of this kind is binocular and conjugate, it is generally caused by a failure of the integrator of the horizontal and vertical eye movements and always corresponds to a central lesion. However, it should be kept in mind that the first cause of this failure is associated with medication (psychotropic, anti-epileptic).

c) Nystagmus caused by visuo-motor-loop impairment
Eye fixation and pursuit result from the permanent correction of the position of the eyes to compensate for retinal slip. A change in the associated feedback loops can induce ocular oscillation or drift. The impairment of these loops can affect both perception and motor elements. This group includes congenital nystagmus, acquired pendular nystagmus and non-nystagmic eye fixation instabilities (square waves and ocular flutter, opsoclonus).

3. Vestibulo-ocular inhibition

This is usually evaluated with electro-nystagmography or video nystagmography but it can be also clinically tested by asking patients to stretch out their arms in front of them and to look at their thumbs while rotating the head and the arms "together". Any inability to maintain the gaze on the thumbs is easily detected by the appearance of a nystagmus during the rotation. The diagnosis of a deficit in the inhibition of the vestibulo-ocular reflex is not always correct. It should be remembered that this immediate inhibition results from the genesis of another eye movement in the opposing direction but

at a speed identical to the slow phase of the vestibulo-ocular nystagmus.[3] At the speeds usually tested, a pursuit movement cancels the slow phase of the nystagmus. The inhibition will therefore be impaired if the pursuit system is failing. A central lesion could very well spare this system of eye pursuit. The correct interpretation is therefore that a deficit in vestibulo-ocular reflex inhibition signals a central lesion but the opposite is not necessarily true. The preservation of normal inhibition does not indicate that the deficit is of labyrinthic origin.

4. Tilt of the visual fields (room tilt illusion)

Patients perceive a rotation, often of 90 or 180 degrees, of the visual fields with both eyes. This rotation can occur in the three spatial planes. They are usually brief and found most commonly in brainstem[4] or cerebellar infarcts, in cortical lesions – more particularly during vestibular epilepsies – but also in peripheral lesions.[5] They are a consequence of the faulty integration of visual and otolithic information. This visual tilt is rarely present simultaneously with abnormalities of the subjective visual vertical line because the latter is of otolithic origin and can be corrected by adequate visual information.

5. Visual symptoms associated with an improvement in visual refraction

Vision contributes to balance through two mechanisms. Firstly through the analysis of the content of the visual fields, by extracting vertical or horizontal references and anticipating destabilisation factors (obstacles, escalators). And secondly by allowing subjects to estimate their own stability. This is done by analysing the movement of the projections on the retina of fixed visual targets, or by measuring the eye movements necessary to stabilise this projection on the retina. All the factors that may interfere with the movement of the projection of fixed visual targets on the retina may therefore affect the ability of subjects to estimate their stability on the basis of visual information. The most common disruptive elements include the prismatic effect of lenses, any drastic modification of refraction, for example after cataract surgery, or ocular instabilities associated with abnormal eye movements.

Multifocal lenses merit particular attention. The correction they bring about changes with the vertical direction of the gaze. The prismatic effect, in other words the deviation of light rays caused by the curvature of the glasses, also varies according to the vertical position of the eyes. This modifies the amplitude of the compensatory eye movement for a head movement (vestibulo-ocular gain) as a function of the vertical position of the eyes. In other words, the same 10-degree movement of a visual target will require the eye to turn more than 10 degrees when focusing for close vision, and less than 10 degrees when focusing for remote vision. The stabilisation of the visual environment and the estimation of subject stability from fixed visual targets therefore becomes much more complex because the analysis has to change for every vertical position of the eyes. Although a lot of subjects quickly adapt, others never do.

Similarly, when there is a drastic correction in astigmatism, there may be interference with space perception and particularly of the orientation of vertical or horizontal lines.

If subjects can estimate their own stability by analysing the eye movements required to stabilise the image on the retina, it is clear that any paresis of an ocular muscle or any modification in eye motility may induce vertigo symptoms.

6. Ocular saccade impairments

Two types of ocular saccade abnormalities are particularly significant for dizziness: saccade hypermetria and the slowing of the vertical saccades.

a) Saccade hypermetria
   Saccades are fast and precise movements. These two characteristics make them particularly sensitive to dysfunction in several structures of the brainstem. In particular, lesions in the cerebellar system will impair the precision and sometimes also the speed of the saccades. This loss of saccade precision will appear as saccade amplitudes that are either too weak (hypometric) or too strong (hypermetric). Although dysfunctions in many structures involved in saccade programming can produce hypometria, saccade hypermetria is almost specific to lesions of the cerebellar vermis. Other median cerebellar structures control vestibulo-ocular reflex gain and play an essential role in balance. Hypermetric saccades against a background of vertigo or dizziness are therefore strongly suggestive of the presence of a cerebellar

syndrome. For mechanical reasons associated with the orbit, the probability of detecting hypermetric saccades is doubled when we test the precision of centripetal saccades, in other words when the gaze moves from an eccentric position, returning to the primary position. Clinical diagnosis involves asking patients to target alternately one finger situated between 20 and 30 degrees laterally and the second situated in front of them. The clinician will look for the eyes overshooting each target, followed by a corrective saccade. These hypermetric movements are easily recognisable on saccade recordings in so far as the saccades are unpredictable in terms of amplitude and position, preventing the progressive improvement of precision by anticipating saccades with the same amplitude.

b) Slowing of vertical saccades
A vestibular otolithic syndrome is frequently the consequence of an ischaemic lesion in the terminal territory of brainstem arteries. The lesion is situated in the sub-thalamic region, and extends up to the midbrain. This mesencephalic extension is responsible for the otolithic syndrome, which is often associated with a slowing of the vertical saccades, without any abnormality in horizontal saccades. The prenuclear structures specifically involved in the realisation of vertical saccades (mesencephalic reticular formation and, in particular, the rostral interstitial nucleus of the median longitudinal fasciculus) are near the otolithic afferences of oculomotor nuclei. When the vertical eye misalignment related to the otolithic deficit is clear in the acute phase, the slowing of the vertical saccades may be the only remaining sign several months after the sub-thalamic lesion. This deficit is easily highlighted by simply asking the patient to switch as quickly as possible between two targets (for example the index fingers of the examiner) located one above the other.

7. The subjective visual vertical

The subjective visual vertical (SVV) of a subject is the angle between the physical vertical line (gravitational axis) and the position of a visual linear mark adjusted vertically by the subject. This SVV is probably built up on the basis of sensory vestibular, visual and proprioceptive information which include abdominal sensors. Other perceptions such as the dynamic moments of inertia may also contribute. The subjective visual vertical is not an indicator of the postural vertical (the body axis when a subject thinks he/she is vertical), because the importance of sensory information in the estimation of these two vertical references is different. This explains the discrepancies found between postural deviation and the SVV. The sensitivity of the otolithic organs to gravity suggests that they play an essential role in the estimation of the physical vertical axis orientation. Visual information may, however, modify this perception. The effective use of these otolithic and visual data for postural control does indeed imply a correction relative to the position of the head with regard to the trunk, and the different segments of the body in space. Cervical somaesthetic information, cutaneous, muscular and articular data are therefore needed to estimate the orientation of the physical vertical axis correctly.

The SVV relates only to the visual representation of the vertical axis and is measured in the absence of any visual reference. It seems particularly dependent on the position of the head in space and does not seem to be very sensitive to variations in the position of the cervical column or the body [data submitted for publication]. In binocular measurements, the SVV is less sensitive to eye torsions induced by oculomotor paresis, even though a binocular approach can also reduce its sensitivity to some cases of otolithic dysfunction. With methods of this kind, the SVV can be considered to be an otolithic evaluation. The SVV is frequently impaired after labyrinthic lesions, lesions of the vestibular nerve or vestibular pathways in the brainstem and in the cortical vestibular areas.

The distribution of the normal values obtained for binocular SVV measurements with the head straight using a glowing bar moving in rigorously controlled darkness (Vertical Test) in 81 subjects shows a deviation greater than 2.8° in fewer than 5% of normal subjects.[6]

8. Visual dependence and visual vertigo

Vision contributes to the preservation of balance, not only by allowing us to detect and anticipate obstacles or irregularities in the ground but, in particular, by supplying vertical and horizontal

references which contribute to the adjustment of our perception of the physical vertical. Finally, movements in the projections of fixed visual targets on the retina, or the eye movements needed to stabilise this projection, allow us to estimate and therefore correct our own stability. Problems arise when this visual strategy is used in circumstances where the available visual information is not appropriate.[7] If most of the visual field is occupied by mobile elements, or if all fixed visual landmarks are taken away, the subject must be able to disregard this visual information and use vestibular or somaesthetic information more to control balance. The use of visual information in these conditions will not allow subjects to turn or stabilise themselves correctly in space, and often lead to a sensation of nausea as a result of the activation of the alarm system (parabrachialis nucleus - limbic cerebral cortex system). This persistence in the use of inadequate visual information can be the result of a vestibular deficit or a loss of the ability to select an adequate source of sensory information. This can persist in spite of the recovery of normal vestibular function, when a subject has got used to controlling balance with a visual strategy. Vestibular rehabilitation can be used in an attempt to help these patients regain the ability to select adequate sensory information.

9. Dynamic visual acuity

The dynamic visual acuity (DVA) test is a method for measuring the clinical functioning of the vestibulo-ocular reflex. This test measures visual acuity during horizontal sinusoidal head rotations of at least 2 Hz and greater than 120°/second, which exceeds the limits at which it is possible to prevent compensation with anticipatory slow eye movements and catch-up saccades.

The subjects are asked to identify symbols or letters on a visual acuity chart. This is continued on successive lines until the subject misses three of the five optotypes on a line.

This is done under two conditions: head stationary (static binocular visual acuity - SVA), and with the head being passively rotated sinusoidally, 15° from centre to the left and right, to the beat of a metronome at 2 Hz (dynamic visual acuity - DVA).

A difference greater than two lines between SVA and DVA constitutes a failure score.

This test has been shown to be sensitive for vestibular impairment in adults[8] and children.[9] The results of the study of Rine *et al*.[9] indicate that the clinical DVA test is a reliable and valid test of gaze stability in children, and can be used to screen for vestibular hypofunction in children as young as three years of age.

10. Migraine and vertigo

Vertigo has been found to occur significantly more frequently in patients with migraine than in control subjects. The international classification of migraines (IHS) includes the symptom "vertigo" only as an aura of a basilar migraine. This aura has to consist of at least two of a number of symptoms, including: vertigo, tinnitus, dysarthria, binocular visual symptoms in the nasal and temporal fields, hearing loss, diplopia, ataxia, bilateral paraesthesia, bilateral paresis or a decline of the consciousness level. However, clinical practice suggests that the link between vertigo and migraine is much more frequent, being found outside the group of patients who fulfil these criteria, and that vertiginous monosymptomatic aura is common.

Neuhauser *et al*.[10] assess the prevalence of migrainous vertigo in patients with migraine and in patients with vertigo according to two different diagnoses.

The diagnosis of *definite migrainous vertigo* was based on the following criteria:

1. Episodic vestibular symptoms of at least moderate severity (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance, *i.e*., sensation of imbalance or illusory self or object motion that is provoked by head motion)
2. Migraine according to the IHS criteria
3. At least one of the following migrainous symptoms during at least two vertiginous attacks: migrainous headache, photophobia, phonophobia, visual or other auras
4. Other causes ruled out by appropriate investigations

A separate diagnostic category of *probable migrainous vertigo* was chosen for patients who did not entirely fulfil the above criteria for migrainous vertigo but were still considered to have migrainous vertigo as the most likely diagnosis.

The diagnosis of probable migrainous vertigo was based on the following criteria:

1. Episodic vestibular symptoms of at least moderate severity (rotational vertigo, other

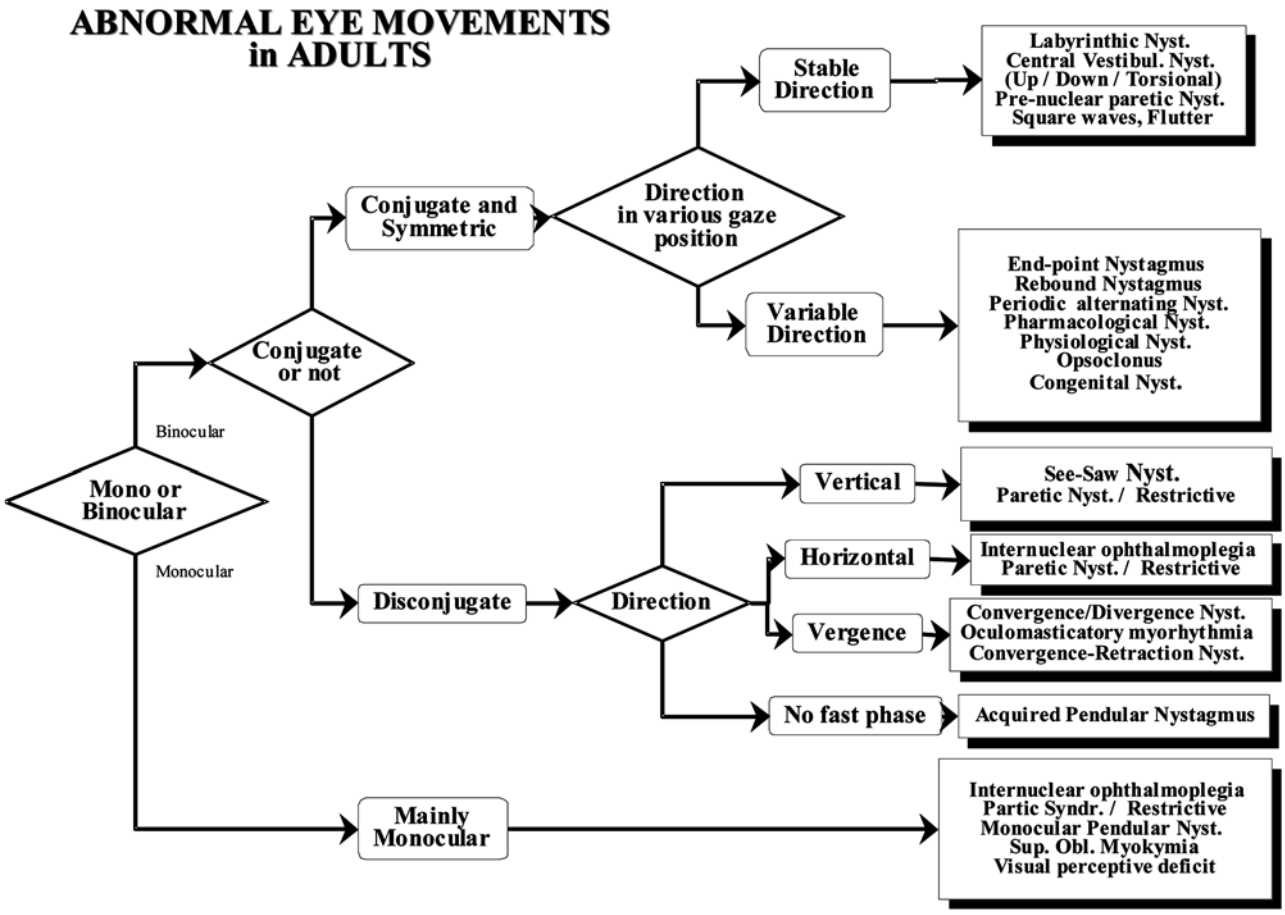

Figure 1

illusory self or object motion, positional vertigo, head motion intolerance)
2. At least one of the following: migraine according to the criteria of the IHS; migrainous symptoms during vertigo; migraine-specific precipitants of vertigo, e.g., specific foods, sleep irregularities, hormonal changes; response to anti-migrainous drugs
3. Other causes ruled out by appropriate investigations

Vestibular symptoms were defined as "mild" if they did not interfere with daily activities, "moderate" if they interfered with but did not impede daily activities, and "severe" if patients could not continue daily activities.

The results of this study show that the prevalence of migraine according to the IHS criteria was higher in the dizziness clinic group (38%) compared with the age- and sex-matched control group (24%, $p < 0.01$). The prevalence of migrainous vertigo was 7% in the dizziness clinic group, and 9% in the migraine clinic group. In 16 of 33 patients, vertigo occurred both with and without headache, and in two patients headache and vertigo never occurred together. The duration of attacks varied from minutes to days.

Moreover, Dieterich *et al.*[11] have effectively treated migrainous patients with vertiginous aura who do not fulfil the criteria for basilar migraine, as well as migrainous patients suffering vertigo without any headaches, and patients with recurring dizziness and without a history of the usual signs of migraine.

Benign paroxysmal vertigo in childhood is also included in the IHS classification of migraine. The criteria are:

1. Episodes of vertigo or disequilibrium without hearing loss or tinnitus
2. Accompanied by visual flashing, nausea/vomiting, pallor, agitation and ataxia. Headache not a usual feature
3. First decade of life, commonly at ages of one to four years
4. Duration: usually minutes, sometimes hours
5. Positive family history of migraine, and many develop

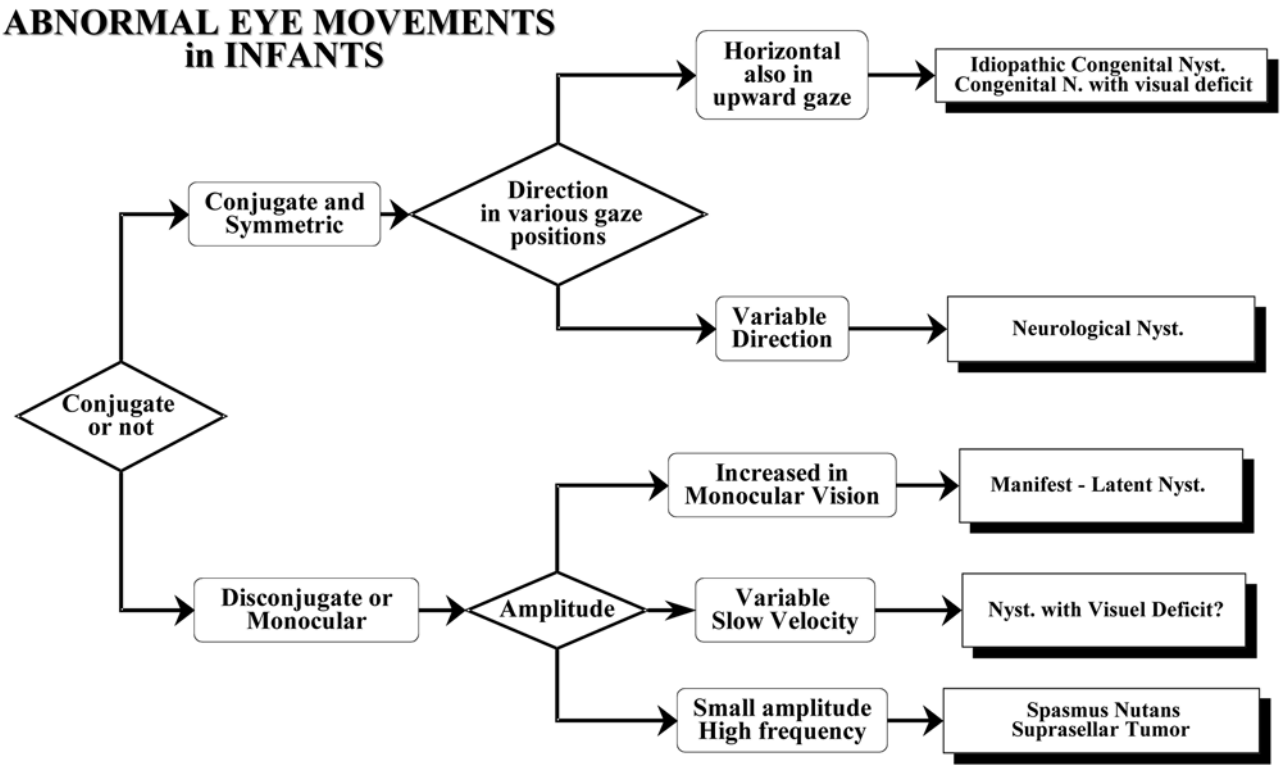

Figure 2

migraine with aura at older ages

6. Other causes ruled out. Differential diagnosis: Ménière's disease, vestibular epilepsy, perilymphatic fistula, posterior fossa tumours and psychogenic disorders

11. The Claude Bernard Horner syndrome

The interruption of the orthosympathetic eye fibres is known as Claude-Bernard-Horner syndrome (CBH). It is characterised by ipsilateral ptosis consecutive to the denervation of the Müller superior eyelid muscle and also by a discreet rise of the lower eyelid caused by denervation of its retractor muscle. In addition this syndrome includes anisocoria with ipsilateral miosis. The anisocoria may be moderate and not exceed 1/2-1 mm. It is clearer in half-light. The anhydrosis of the ipsilateral hemi-face present in CBH syndromes of central origin is not obvious. It is often transitional because it is compensated by denervation hypersensitivity to circulating adrenergic substances. The diagnosis can be confirmed by eye-drop tests from ophthalmologists (cocaine test followed by the hydoxy-amphetamine test). At the ponto-medullar level, the orthosympathetic fibres pass just inside the vestibular nuclei. There is therefore a high probability that a lesion of the vestibular nucleus is associated with CBH syndrome. Only larger lesions will result in the classic syndrome of Wallenberg. The presence of CBH signs can still indicate a central origin months after a vestibular deficit.

In a post-traumatic context, CBH syndrome can result from the impairment of the orthosympathetic fibres at the level of the cervical cord or in the pathway along the carotid arteries. We should also bear in mind that a CBH syndrome can result from a migraine crisis and this can also lead to dizziness and vertigo.

12. Facial sensitivity deficit

In the brainstem, post-synaptic pain sensitivity fibres of the trigeminal nerve extend down to the first cervical levels. They constitute the downward root of the trigeminal nerve and reach the upper spinal cord. At the bulbar level, these fibres are situated just inside vestibular nuclei. They are a part of the same vascular territory as the vestibular nuclei, and are irrigated by the terminal branches of the antero-inferior cerebellar artery. Other branches of the same artery irrigate the internal ear. A sensory deficit of the face

associated with acute dizziness indicates the presence of a lesion at the level of the floor of the fourth ventricle, associated or not with a labyrinthic lesion. Since topography is inverted in this downward root of the trigeminal nerve, it is mostly in the upper part of the face that we find the sensory deficit related to such a lesion.

13. Transient visual obscurations (Visual "Eclipses")

Transient visual obscurations are brief moments of binocular darkening of the vision. They are found in 68% of patients suffering from idiopathic intracranial hypertension[12] but also in cases of orthostatic hypotension, and secondary intracranial hypertension.

14. Oscillopsia

These are not specific and can result from all acquired nystagmus, decompensated congenital nystagmus, non-nystagmic eye oscillations, superior oblic myokimia and spasmus nutans.

An overview of abnormal eye movement in adults and in infants is given in Figures 1,2.[13]

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Principle of the head impulse (thrust) test or Halmagyi head thrust test (HHTT)

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Key-words. Vestibular test; semicircular canal; head impulse test; head thrust test

The head impulse or head thrust test was first described by Halmagyi and Curthoys in 1988.[1] It has acquired an increasingly important place in the clinical examination of the vertigo patient. It detects severe unilateral loss of semicircular canal (SCC) function clinically; it is more sensitive and specific than the traditional Romberg and similar tests; and it is particularly important in the emergency unit, where it can distinguish between vestibular neuritis and cerebellar infarction, which can both generate similar symptoms suggesting an initial attack of severe acute vertigo. The result of the head thrust test is definitely normal in a patient with a cerebellar infarction but abnormal in a patient with vestibular neuritis.

General physiological background: the push-pull principle of the vestibulo-ocular reflex

The peripheral vestibular sensors transmit motion to the brain through frequency encoding. Like FM radios, our brains continuously receive 'frequency modulated' signals. A normal resting discharge rate of approximately 90 spikes per second is modulated such that any increase in this rate corresponds to excitation and a decrease to inhibition. The polarisation of the hair cells in the horizontal semi-circular canal is such that deflection of the stereocilia in the cupula towards the kinocilium (ampullo- or utriculopetal) results in hair cell depolarisation and the activity of the primary afferent neurons therefore increases. Deflection of the stereocilia away from the kinocilium (ampullo- or utriculofugal) results in hair cell hyperpolarisation and decreased primary afferent neuron activity.

The orientation of the left and right semi-circular canals in the head is such that any movement always induces an antagonistic response in both canals. Horizontal head movements in the yaw plane are an example. During rightward head rotation, the endolymph in the lateral semi-circular canals on both sides lags behind, bending the cupula of the right SCC towards the vestibulum (ampullo- or utriculopetal) and simultaneously deflecting the cupula of the left SCC away from the vestibulum (ampullo- or utriculofugal). A key difference is the polarisation of the hair cells. Indeed, since the the hair cells in the right and left canals are implanted in opposing directions (in a mirror image fashion), the deflection on the "leading" right side induces the movement of the stereocilia towards the kinocilium, whereas the movement of the stereocilia is away from the kinocilium in the opposing, "following" ear. As a result of this "push-pull principle", the activity of right lateral SCC primary afferent neurons increases, and, at the same time, the activity of left lateral SCC primary neurons decreases with respect to the normal resting discharge rate.

The activity of the lateral SCC primary afferent neurons is modulated by horizontal head rotation. The firing rate increases in the leading ear (the ear towards the movement is directed) and decreases in the following ear. This is the push-pull principle of the VOR.

The right medial vestibular nucleus in the brainstem receives an increased input from the right lateral SCC primary neurons (no crossing). This excites the activity of type I secondary vestibular neurons. These excitatory neurons drive the leftward compensatory eye movements of the VOR, to ensure gaze stabilisation. However, commissural disinhibition from the left lateral SCC primary neurons also contributes to the excitation of the type 1 neurons. Both excitation of the right SCC and disinhibition of the left SCC are therefore needed for an optimal VOR.

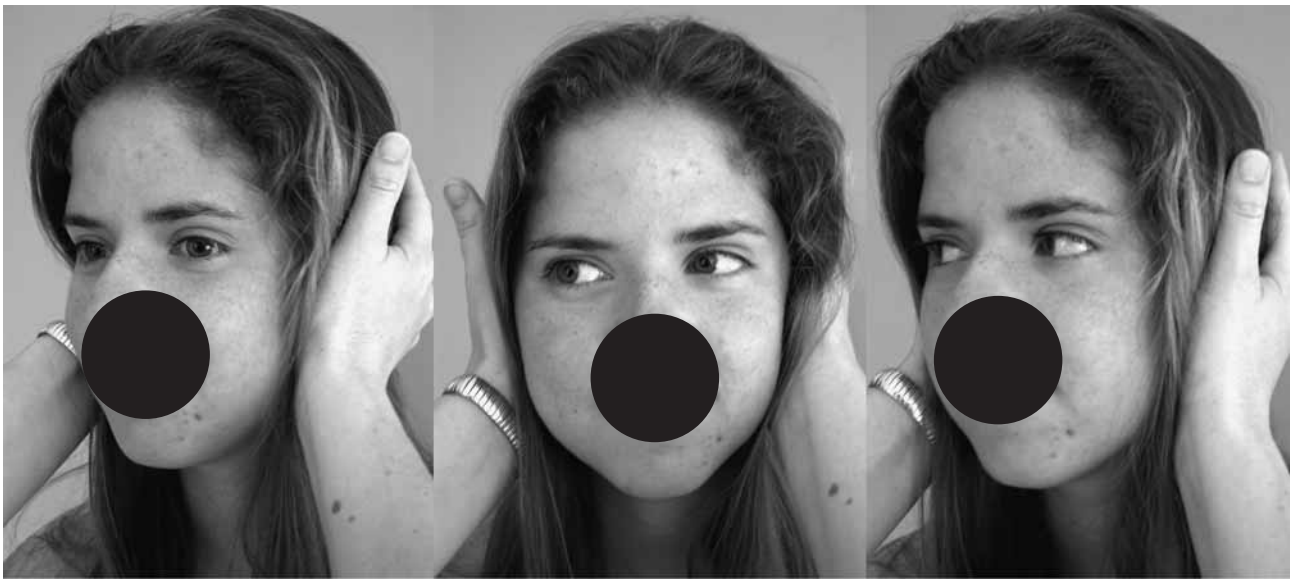

Figure 1
Left: the clinician holds the head of the subject firmly and turns it briskly to the left. Centre: After the rotation to the left, the subject maintains the gaze on the distant fixation point, *i.e.*, the eyes stay stable in space.
Right: After abrupt rotation to her right, the subject moves her eyes with her head and loses the target. A refixation is necessary to fixate the point again (not shown). The side towards the gaze fixation is lost is the deficit side, *i.e.*, the patient's right.

Head thrust test

The head thrust test is primarily based on the fact that inhibition of primary and secondary vestibular neurons cannot produce fewer than 0 spikes per second. Excitation can drive the discharge rate from 90 to 300 or more spikes per second. So when the healthy side is excited for a high acceleration head movement, the healthy side will generate the larger part of the VOR, since the disinhibition of the ipsilateral type-1 neurons by the contralateral SCC contributes relatively little to the VOR. Passive head impulses or thrusts should be typically rapid but with a small amplitude (± 20 degrees). Their velocity ranges up to 180 deg/s but high acceleration is particularly important (3000-4000 deg/s2). They have to be unpredictable since the patient very quickly learns to anticipate and this reduces the sensitivity of the test to a considerable extent. The examiner should therefore thrust the head of the patient firmly from left to right at random and from right to left a little later, *i.e.*, not immediately. The starting position should be such that the patient's head is turned slightly past the midline, and it should then be thrust just past the midline to the opposite side. Here, amplitude is low but acceleration can be considerable. This test demands some training, particularly with respect to the positioning of the hands on the side of the head and holding the head firmly. The instruction to the patient is to fix on a point in the distance behind the examiner.

When the subject's head is turned to the side of the lesion, the VOR is deficient and the eyes will move with the head so that they no longer fix on the point in the distance. The patient therefore needs a refixation saccade just after the thrust. When the head impulse is in the direction of the healthy side, the VOR will maintain the target on the fovea and no refixation saccade will be needed.

The head-thrust test is positive for the side that causes the refixation saccade upon thrust (Figure 1)

It is not only the lateral SCC that can be examined – this is, in a sense, a clinical approximation of the caloric test – but also the other SCC. Here, the patient's head must be thrust in the RALP or LARP planes (Right Anterior – Left Posterior or Left Anterior – Right Posterior SCC).[2]

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Tullio's phenomenon

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Key-words. Tullio; vertigo; sound-induced; Hennebert sign; superior semi-circular canal dehiscence

Abstract. *Tullio's phenomenon.* Tullio's phenomenon corresponds to a pattern of sound-induced subjective and objective responses. The subject may feel sensations of unsteadiness, imbalance or vertigo, associated with disturbances of oculomotor and postural control. Tullio's phenomenon is provoked by very loud sound and should be considered physiological. It is pathological if it is provoked by normal sounds. Changes to the functioning and/or the morphology of the labyrinth should be looked for in patients with the pathological form: decreased thresholds for the acoustically evoked vestibular potentials, semicircular canal dehiscence, traumatic lesions of the labyrinth, ligament hyperlaxity.

Introduction

Tullio's phenomenon (TP) is a pattern of sound-induced imbalance symptoms, motor responses of the eyes (nystagmus), head (myogenic responses) and other spinal neuron synkinesis (postural sway).[1,2] It may be physiological or pathological.

Physiological TP

Very loud sounds (250-500-1000 Hz or clicks at 110 dB) applied mono-aurally (but not binaurally) may elicit postural responses in normal subjects.[1,3,4] This physiological Tullio's phonomenon results in postural sway, increasing with closed eyes, when recorded with posturographic techniques.

It also induces myogenic responses in the sterno-cleido-mastoidian muscles, when recorded with vestibular evoked myogenic potential (VEMP) techniques.[1,4-7] Those vestibulocollic responses (provoked by a physiological TP) are thought to be useful in exploring saccular function.

Pathological TP

This form corresponds to a vestibular hypersensitivity to sound, resulting in perceived vertigo or unsteadiness. The otolothic-like symptoms are elicited with less loud sounds, <70 dB nHL for clicks,[4,5] or by loud pure tones presented binaurally.

Normal sounds provoke acute modifications in oculomotor control, leading to nystagmus. They disturb the postural responses, inducing feelings of unsteadiness and increased postural sway. The posturographic recordings may show a fall in the vestibular and composite postural scores when subjects are exposed to loud sounds. This fall is not observed in neuro-otological patients without a history of TP, or in normal subjects.[3]

Acoustically-evoked vestibular potentials have lower thresholds and increased amplitudes. On the other hand, galvanic-evoked vestibulocollic responses present normal thresholds. It is therefore supposed that sound hypersensitivity in subjects with TP is likely to occur distally to the vestibular nerve.[4]

The mechanisms by which acoustic stimuli act on the vestibular end organs remain unclear. Studies of animals have shown that afferents from all the vestibular end organs could respond to acoustic stimuli.[8] However, some pathological changes lower thresholds, resulting in sono-vestibular symptoms.[8]

Aetiology

The superior semicircular canal dehiscence syndrome is a newly recognised syndrome characterised by vertigo and nystagmus (torsional and down beating) induced by sound (= TP), or pressure changes in the middle ear (= Hennebert sign), or intracranially.[4,5,8,9] The dehiscence renders the canal particularly sensitive to sound and pressure changes, probably because it works like a third window, allowing larger volume, pressure, deflections and displacements at the level of the canal.

Other pathological modifications may be involved in TP. A pathological contiguity of the tympano-ossicular chain and membranous labyrinth may be associated with it. It is observed in cases of dislocated ossicular chain, stapes hyperlaxity, fracture of the footplate or of the labyrinth, fibrotic damping of the ossicular chain, fibrosis of the inner ear, traumatic labyrinth, perilymphatic fistula and endolymphatic hydrops.[1,3-14]

The distance between the footplate and the utriculus is only 0.5 mm in the posterior part of the oval window.[11] Membranous connections exist between the utriculus and the footplate in 25% of subjects.[11] An increase in those connections may favour sonovestibular hypersensitivity.

Diagnosis

When a subject presents symptoms of sonovestibular hypersensitivity, the TP diagnosis must be considered. It is confirmed by different tests like a positive Hennebert sign, sound-induced nystagmus, sound-induced postural responses recorded with posturographic tests and falls in VEMP thresholds.

A morphological modification of the middle and inner ear must be considered. Hyperlaxity may be suspected in cases where there is a fall in the resonance frequency of the ear. A CT scan of the temporal bone should show any semi-circular canal dehiscence.

Conclusion

Pathological Tullio's phenomenon is characterised by subjective and objective sonovestibular symptoms resulting from abnormal hypersensitivity to normal sounds of the vestibular end organs secondary to morphological changes in vibration and pressure transmission between the external and the inner ear.

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The clinical investigation of static and dynamic balance

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Key-words. Vestibular function tests; posture; reference values; reproducibility of results

Abstract. *The clinical investigation of static and dynamic balance.* This article describes the clinical examination of static and dynamic balance. The purpose is to illustrate the guideline for the diagnosis and management of vertigo. For most of the tests, indicative normal values are given and discussed. The paper also looks at the clinical examination of gait.

1. Index test

This test is performed on a seated patient whose eyes are closed. The aim is to look for the presence of past pointing, the tendency for the outstretched arms and fingers to drift unidirectionally. In peripheral vestibular disorders, lateral deviation of the index is directed towards the side of the lesion (or towards the slow phase of the spontaneous nystagmus). Non-harmonious past pointing, the deviation of only one index or vertical deviation suggest a central vestibular disorder. Vertical deviation of the arms and fingers may also result from motor or proprioceptive disorders.

This test is mainly interesting for the diagnosis of acute vertigo. It is useful to compare the direction of the nystagmus (observed with Frenzel spectacles) and the direction of the postural deviations. Furthermore, in the case of acute vertigo, this is the only test that can be performed in a patient confined to bed. On the other hand, in a patient with chronic vertigo, a simple weakness may induce a deviation. Furthermore, this sign disappears progressively as vestibular compensation is established.

A dynamic variation of this test, looking at the tendency for the repetitively elevated and lowered outstretched fingers to drift unidirectionally, may be performed to enhance sensitivity. Another dynamic variation is the finger-pointing test. It is more sensitive than the finger-to-nose test for cerebellar dysmetria or hypermetria. The patient is instructed to follow the finger of the clinician by rapidly pointing towards each new position it takes.

2. Romberg test

The patient stands, feet together with eyes open then closed (to eliminate visual clues) in order to compare static balance in these two states. Normally, there is no body sway or directional fall.

In unilateral peripheral vestibulopathy, the patient slowly deviates towards the side of the lesion. This observation must be reproducible. In neurological pathology, postural balance is less affected by eye closure (except in sensory ataxia). However, this test is not very sensitive, so more difficult variations of the Romberg are described:

- Jendrassik's manoeuvre: the patient is asked to pull both hands in opposite directions with the fingers linked together, resulting in an enhancement of muscular relaxation in the lower members.
- Romberg test in tandem: patient places one foot directly in front of the other (heel to toe): this test is very difficult and few elderly people are able to manage it.
- Push test: the patient is put off balance by an antero-posterior push followed by a lateral push. This variation of the test is often used if malingering is suspected.
- Clinician may distract the patient by writing numbers on his forearm if a psychological disorder or malingering is suspected.

It is interesting to investigate how head position influences the direction of postural deviation as postural reactions initiated by vestibulospinal reflexes are usually opposite to the direction of the fast phase of nystagmus. Patients with right peripheral vestibular lesion will show lateral body deviation towards the right. Asking the patient to turn the head to the right (left) will result in a backward (forward) fall.

Normal values for the Romberg standing test were reported by Nyabenda *et al*.[1] in a sample of 120 healthy subjects broken down into different age categories (ten-year brackets, with each age category including 20 subjects). In this study, postural deviation was measured in standing subjects with eyes closed and arms outstretched as described by Gill *et al.*[2] Lateral drift of the fingers was measured by reference to the vertical axis. Lateral deviation was considered to be significant if maintained during 30 seconds.

Significant index deviation was found in four subjects only: a subject aged 69 years (5 cm, 5% of the age category) and three subjects in the 70-79 age category (3-10 cm, 15% of the age category).[1]

Romberg's standing test was also investigated using craniocorpography (CCG).[3,4] The patient is marked with lights upon both the shoulders and the head by means of a hard hat containing marker lights above the forehead and the occiput. Lights are reflected through a mirror system on the ceiling into a video camera and a computer which receives, analyses and prints the signal (a newer marking method uses ultrasound markers and an ultrasound receiver unit instead of light markers).

Table 1 shows the normal values for the CCG of the standing test.

Romberg's test is a useful method for studying patient with symptomatic falls. If cerebral vascular hypoxia, epilepsy, cerebellar ataxia, intoxication or sensorimotor loss are the main aetiology of pathological falls, vestibular dysfunction is a significant differential diagnosis for these patients. Brandt and Dieterich[5] classified central and peripheral vestibular falls in relation to the preferred direction of falling.

Table 1

Normal values for the longitudinal and lateral sway as measured by craniocorpography during Romberg's test. These data were derived from a neuro-otological data bank with 10,335 normal and neuro-otological cases [Claussen CF: communication at the 30th Annual Meeting of the Neurootological and Equilibriometical Society (NES) Porto – Portugal, April 3-5, 2003]

Parameters for standing CCG	Normal range – Lower border	Normal range – Upper border
Longitudinal sway	1.75 cm	10.53 cm
Lateral sway	1.74 cm	7.06 cm

Peripheral vestibular syndromes

Vestibular neuritis results in slow falling towards the side of the lesion.

Benign paroxysmal positioning vertigo: patients in whom attacks of vertigo are elicited by head tilt exhibit large sway amplitudes, predominantly in the fore-aft direction. Instability decreases progressively in parallel with the reduction of nystagmus and vertigo.

Tumarkin's otolithic crisis: in this particular version of Ménière's disease, patients feel as if they are thrown to the ground without warning. This "drop attack" is not preceded or accompanied by vertigo. Patients remain conscious.

Otolithic Tullio phenomenon: diagonal and backwards towards the unaffected ear.

Bilateral vestibulopathy: instability is multidirectional with the largest amplitude in the fore-aft direction. Patients often complain of oscillopsia associated with head movement or when walking.

Central vestibular syndromes

Several vascular or tumour disorders at the level of the brainstem may involve the central vestibular pathway. Ipsiversive postural deviation usually results from lateral medullar lesions while contraversive postural drift results from pontomesencephalic brainstem lesions. Thalamic lesions involve either ipsi- or contraversive postural deviation.

Postural imbalance is frequently combined with central ocular signs or symptoms (nystagmus with central features, ocular tilt reaction, failure of vertical gaze, lateropulsion of the closed eyes, tilt of perceived visual vertical, Claude Bernard Horner's syndrome, internuclear ophtalmoplegia,…), with sensorimotor signs affecting the limbs or cranial nerves, and with cerebellar syndromes. A careful examination is therefore required, since the main apparent symptom is the patient's inability to maintain an upright posture.

One exception should be noted: in some cases of Wallenberg's syndrome, the nystagmus may be horizontal-rotatory beating in the opposite direction from postural deviations ("harmonious nystagmus").

Lesions of otholitic central pathways or some thalamic diseases may occur without paresis or sensory or cerebellar deficit. In these cases, ocular signs are of particular importance.

Downbeat nystagmus syndrome is often associated with a tendency to fall backwards.

Diagnostic elements

- Backward falls suggest sensory ataxia, especially when the eyes are closed. When the eyes are open, the backward deviation suggests frontal-lobe or fronto-pontine disorders. These features are also observed in a range of degenerative syndromes or in diffuse cerebral arteriosclerosis.
- Fore-aft deviations are often associated with cerebellar ataxia.
- In sensory ataxia, the Romberg test results only in slight unsteadiness when the eyes are open. When patients close their eyes, large and disordered oscillations occur. This contrasts with the slow and progressive deviation observed in patients with peripheral vestibular disorders.

3. Unterberger and Fukuda's stepping test

The stepping test initially described by Unterberger is commonly used to assess individuals with peripheral vestibular dysfunction or balance instability. Patients are required to step on the spot with arms outstretched. In the initial form of this test, normal subjects show no deviation or rotation while patients with peripheral vestibular dysfunction rotate progressively towards the side of their lesion.

In 1956, Fukuda[6] added a spider's web drawn on the floor, within which the patients had to perform their stepping. This makes it possible to quantify displacement after a series of 50 steps. Angle of rotation (spin), angle of displacement and distance of displacement are measured. The most reproducible parameter is the spin: Fukuda considers a rotation of more than 30 degrees while stepping to be pathological.

A common variation of this test is a stepping test with the arms alongside the body. Results are globally similar.[7]

However, the test-retest reliability of the Fukuda's stepping test is a subject of discussion.[8] Several authors have reported that the stepping test does not appear to be useful for the detection of abnormalities in the vestibular system or for distinguishing normal individuals from patients. In a prospective study of 131 normal and pathological subjects, they found considerable inter- and intra-individual variation in direction and width of rotation and in displacement.[9] Others studies reported the same results and conclusions.[10,11]

A quantification study for the Fukuda stepping test has been published.[1] The protocol included 45 steps, with arms alongside the body. Of 120 normal subjects in different age categories, only two presented no deviation and there were four subjects in whom there was no spin. Subject displacement was always forwards, never backwards. The mean values for distance of displacement, angle of displacement and angle of rotation are reported in Table 2. The correlation between age and angle of deviation or angle of rotation was significant ($r = 0.56$, $p < 0.000$). The correlation between age and forward displacement was not significant ($r = 0.17$, $p = 0.06$). Unfortunately, the authors did not report the percentage of healthy subjects (they were all normal in this study) who performed an abnormal test (*i.e.*, more than 30° spin as described by Fukuda), which would be more significant for clinical practice. However, it seems obvious from their table of results that this percentage also increased with the age of the subjects.

In a study of 48 healthy subjects ranging from 20 to 35 years, Wintgens reported mean spin values of 27° (+/- 4° SD) after a 50-step test (communication presented at the Journées de Posturologie, December 6-7, 2002, Brussels). During the first trial of the stepping test, 75% of the healthy subjects performed the classic Fukuda stepping test in the normal range (defined by these authors as 32° spin) and 71% of the subjects performed a normal Fukuda's test with arms alongside the body (Table 3).

Using CCG, Claussen reported a normal spin value of 82° after a 100-step test.[3,4]

As for the reliability of the test, which was also discussed above, Nyabenda reported good test-retest reliability for body spin and angle of deviation.[1] A change in the direction of the rotation from one test to another was only observed for one subject. Reiss *et al.*[12] reported similar results.

Wintgens also reported good test-retest reliability for the mean spin value and for the global percentage of abnormal tests (communication presented at the Journées de Posturologie, December 6-7, 2002, Brussels). A major problem persists: subjects do not present consistently abnormal spin values in the first and second parts of the test (a subject may be normal (spin <32°) in the

Table 2

Mean values (and standard deviation) of distance of displacement, angle of displacement and angle of rotation measured during a stepping test.[1] The protocol included 45 steps, arms alongside the body

	Forward displacement (cm)		**Angle of deviation (d°)**		**Angle of rotation (d°)**	
Age category	Mean	SD	Mean	SD	Mean	SD
20-29	60.7	30.7	19.9	7.8	13.9	6.9
30-39	62.5	26	32	10.1	23.2	10.6
40-49	71.7	35	35	12	26.7	11.3
50-59	73.3	40.7	37.8	13.4	31.9	10.6
60-69	76.2	40.2	39.1	9.8	34.7	11.4
70-79	75	33	41.5	10.9	42.1	10.1

Table 3

Number of subjects in whom spin is within the normal range (here: 32°) after the 50th step in the first and second run and in the two runs, and subjects outside the normal range in the same conditions. Fukuda = arms extended; Fukuda repeat = arms alongside the body. The subjects considered to be normal or abnormal, Fukuda or Fukuda repeat, identical in the first and second run or not, are not necessarily the same in each line

		Number of subjects with abnormal spin		
	Test component	Abnormal left spin	Normal	Abnormal right spin
Fukuda	First	5	36	7
	Second	4	37	7
	Identical	2	29	3
Fukuda repeat	First	9	34	5
	Second	5	40	3
	Identical	2	29	1

first part and abnormal in the second). Table 3 contains more detailed results.

In conclusion, it is not easy to state a normal reference value for the stepping test since the protocols described earlier are dissimilar. Nyabenda's study[1] clearly demonstrates that age is an important factor to take into account when interpreting the Fukuda test. The reliability and the specificity of this test are debated. So clinicians should interpret the results of the stepping test with caution, especially if it is used as a screening tool. Clinicians should make different static and dynamic tests of balance and compare their results in order to arrive at clear conclusions about balance in their patients.

4. Standard gait and star gait (Babinski-Weill test)

The standard gait test was first described by Fregly and Graybiel.[13] The patient is required to walk 3.5 metres, with eyes closed, in three successive runs. Deviation from the straight line is measured. This simple test, which is easier than Fukuda's test or than the star gait test, may be the most useful test for evaluating the evolution of vestibular compensation.

During the star gait test (Babinsky-Weill), subjects are required to walk 3 to 5 steps forwards then backwards, with their eyes closed. The star gait is a result of the systematic unilateral postural deviation that occurs in peripheral vestibular pathology. It is not always easy to conduct the test in practical terms since a large space is required to ensure that subjects cannot orient themselves within the room.

Normative values are difficult to report since the variations in the method are even greater than for the stepping test. To provide an indication, the results of a study by Nyabenda *et al*.[1] are reported in Table 4. There is considerable correlation between age and deviation, as was seen in the stepping test ($r = 0.71$; $p < 0.000$).

5. Examination of walking

Gait examination may sometimes help to determine whether a posture and walking disorder is induced by a vestibular or a central disorder. Of the various neurology syndromes that may induce walking difficulties, falls and dizziness are often associated with central vestibular and cerebellar syndrome, with sensory ataxia, Parkinson's syndrome and frontal lesions. Gait examination starts when the patient enters the examination room. Some gait features would appear to be immediately characteristic.

Clinical examination continues with the patient barefoot. A careful observation of trunk posture, stance and walking is performed. The clinician should notice the overall pattern of body movement during walking, the swinging motion of arms and legs, the

Table 4

Mean values (and standard deviation) of angle of displacement during a standard gait test (patient are required to walk 5 m with their eyes closed in a straight line) and a star gait test (three series of three paces forwards and backwards)

	Standard gait		**Star gait (Babinsky Weill)**	
Age categories	**Mean** (°)	**SD** (°)	**Mean** (°)	**SD** (°)
20-29	10.3	4.8	1.7	0.8
30-39	18	5.6	2.3	0.7
40-49	21	7.2	2.8	1.1
50-59	23.6	10	3.4	0.8
60-69	26.3	14	4.0	1.1
70-79	28	11	4.5	0.9

regularity and the size of the strides, the speed of walking and the synergy of head, trunk and leg movements.

Subtle syndromes may be revealed by asking the patient to stop and go, to walk in a straight line heel-to-toe (tandem gait), to walk and turn quickly. Other special manoeuvres consist of asking the patient to crouch, to sit and to stand up, to walk on their heels and then to walk on tiptoe.

Akinetic-rigid gait

The classic and most common akinetic-rigid gait disorder is seen in Parkinson's disease. Of course, falls are induced by walking difficulties but interestingly, late in the syndrome, patient falls result from the loss of postural and righting reflexes (that do not respond to levodopa medication).

Patients adopt a flexed truncal posture with stooped trunk, shoulders and neck. Gait is slow and rigid with small paces and loss of the swinging of one or two arms. Tremor of the upper limbs might be present but is less commonly observed in the lower limbs. Initiating the first step is difficult so that patients begin walking with a few rapid, very short, shuffling steps (start hesitation); sometimes patients actually step up and down in the same place without any forward progress. Episodes of freezing (complete cessation of movement, "feet glued to the floor") are also typically observed in patients with Parkinson's disease. Freezing may also be present if a doorway or another obstacle is encountered; shuffling and freezing may be revealed if the patient is asked to turn back quickly. To maintain balance when walking, patients may move forwards in a series of very small steps (festination) while bent forwards (subjects look as though they are running after their centre of gravity).

Cerebellar syndrome

Patients adopt a wide base stance. Backward and forward rhythmic swaying occurs. This instability is not influenced by eye closure but is greatly increased if patients bring their feet closer together. Gait is slow, strides are irregular and variable in timing (dyssynergia) and the steps are erratic as if the patients are drunk. This particular gait is often observed in alcoholism (selective damage affecting the cerebellar vermis) where legs and gait are usually affected while ocular movement, speech and upper limbs are spared. With lesions confined to one cerebellar hemisphere, anomalies will be limited to the affected ipsilateral limb and will affect coordination of movement more than balance (if the vermis is not involved). Patients tend to fall towards the side of the lesion and throw their leg on the affected side too high and outwards. Finally, in lesions affecting the vestibular part of the cerebella, symptoms resemble those observed in peripheral unilateral vestibular disease.

Unilateral peripheral vestibular syndrome

Clinicians should be attentive to sudden changes in gait direction or lateropulsion, especially if these abnormalities depend on head movements. Paradoxically, lateropulsion away from the side of the lesion may occur as a result of voluntary efforts to correct balance.

Cautious gait

Putting aside the typical cases described above, it is important to note that patients with any decline in walking ability and balance tend to develop compensation mechanisms that may disguise the underlying problem. Those patients will adopt a slower gait with shorter and shallower steps in order to keep contact with the ground for a longer time (cautious gait). This cautious and guarded gait is often present in the elderly. Factors contributing to a decline in the mobility of the elderly in crude degenerative joint disease, reduced range of limb mobility and limited exercise capacity due to cardiovascular fitness decline. Additional factors might be sensory deficit (vision, vestibular and

proprioceptive function) without any one lesion being severe enough to explain the observed walking difficulty. Another common factor might simply be the fear of falling. Clinicians should not diagnose Parkinson's disease in all these patients, even if some aspects of gait look similar!

In all these cases, gait examination will not immediately lead to a diagnosis and a complete vestibular and neurological examination should be performed.

7. Dynamic gait index

The Dynamic Gait Index was developed by Anne Shumway-Cook[14] and has been used in older adults to determine their likelihood of falling. Scores of 19 or less are related to falls in older adults. The index tests 8 facets of gait and can be used with an assistive device. Self-reported falls in the past six months and Dynamic Gait Index scores have been found to be related in persons with vestibular disorders.[15] The dynamic gait index has also been used to determine the effect of vestibular rehabilitation on the reduction of fall risk in individuals with unilateral vestibular hypofunction.[16]

1. Gait level surface

Instructions: walk at your normal speed from here to the next mark (20')

Grading: mark the lowest category that applies.

(3) Normal: walks 20', no assistive devices, good speed, no evidence of imbalance, normal gait pattern.
(2) Mild impairment: walks 20', uses assistive devices, slower speed, mild gait deviations.
(1) Moderate impairment: walks 20', slow speed, abnormal gait pattern, evidence of imbalance.
(0) Severe impairment: cannot walk 20' without assistance, severe gait deviations or imbalance.

2. Change in gait speed

Instructions: begin walking at your normal pace (for 5'). When I tell you "go", walk as fast as you can (for 5'). When I tell you "slow", walk as slowly as you can (for 5').

Grading: mark the lowest category that applies.

(3) Normal: able to change walking speed smoothly without loss of balance or gait deviation. Shows a significant difference in walking speeds between normal, fast, and slow speeds.
(2) Mild impairment: is able to change speed but demonstrates mild gait deviations, or no gait deviations but unable to achieve a significant change in velocity, or uses an assistive device.
(1) Moderate impairment: makes only minor adjustments to walking speed, or accomplishes a change in speed with significant gait deviations, or changes speed but loses significant gait deviations, or changes speed but loses balance but is able to recover and continue walking.
(0) Severe impairment: cannot change speeds, or loses balance and has to reach for wall or be caught.

4. Gait with horizontal head turns

Instructions: begin walking at your normal pace. When I tell you to "look right", keep walking straight, but turn your head to the right. Keep looking to the right until I tell you to "look left", then keep walking straight and turn your head to the left. Keep your head to the left until I tell you to "look straight" then keep walking straight but return your head to the centre.

Grading: mark the lowest category that applies.

(3) Normal: performs head turns smoothly with no change in gait
(2) Mild impairment: performs head turns smoothly with slight change in gait velocity, *i.e.*, minor disruption to smooth gait path or uses walking aid.
(1) Moderate impairment: performs head turns with moderate change in gait velocity, slows down, staggers but recovers, can continue to walk.
(0) Severe impairment: performs task with severe disruption of gait *i.e.*, staggers outside 15" path, loses balance, stops, reaches for wall.

4. Gait with vertical head turns

Instructions: begin walking at your normal pace. When I tell you to "look up", keep walking straight but tip your head and look up. Keep looking up until I tell you, "look down". Then keep walking straight and turn your head down. Keep looking down until I tell you, "look straight", then keep walking straight, but return your head to the centre.

Grading: mark the lowest category that applies.

(3) Normal: performs head turns with no change in gait.

(2) Mild impairment: performs task with slight change in gait velocity i.e., minor disruption to smooth gait path or uses walking aid.
(1) Moderate impairment: performs task with moderate change in gait velocity, slows down, staggers but recovers, can continue to walk.
(0) Severe impairment: performs task with severe disruption of gait *i.e*., staggers outside 15" path, loses balance, stops, reaches for wall.

5. Gait and pivot turn

Instructions: begin walking at your normal pace. When I tell you to "turn and stop", turn as quickly as you can to face the opposite direction and stop.
Grading: mark the lowest category that applies.
(3) Normal: pivot turns safely within 3 seconds and stops quickly with no loss of balance.
(2) Mild impairment: pivot turns safely in >3 seconds and stops with no loss of balance.
(1) Moderate impairment: turns slowly, requires verbal cueing, requires several small steps to catch balance following turn and stop.
(0) Severe impairment: cannot turn safely, requires assistance to turn and stop.

6. Step over obstacle

Instructions: begin walking at your normal speed. When you come to the shoebox, step over it not around it, and keep walking.
Grading: mark the lowest category that applies.
(3) Normal: is able to step over box without changing gait speed; no evidence of imbalance.
(2) Mild impairment: is able to step over box, but must slow down and adjust steps to clear box safely.
(1) Moderate impairment: is able to step over box but must stop, then step over. May require verbal cueing.
(0) Severe impairment: cannot perform without assistance.

7. Step around obstacles

Instructions: begin walking at normal speed. When you come to the first cone (about 6' away), walk around the right side of it. When you come to the second cone (6' past first cone), Walk around it to the left.
Grading: mark the lowest category that applies.
(3) Normal: is able to walk around cones safely without changing gait speed; no evidence of imbalance.
(2) Mild impairment: is able to step around both cones, but must slow down and adjust steps to clear cones
(1) Moderate impairment: is able to clear cones but has to slow down significantly to accomplish task, or requires verbal cueing.
(0) Severe impairment: unable to clear cones, walks into one or both cones, or requires physical assistance.

8. Steps

Instructions: walk up these stairs as you would at home (i.e., using the rail if necessary). At the top, turn around and walk down.
Grading: mark the lowest category that applies.
(3) Normal: alternating feet, no rail.
(2) Mild impairment: alternating feet, must use rail.
(1) Moderate impairment: two feet to a stair, must use rail.
(0) Severe impairment: cannot do safely.

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Unilateral centrifugation

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Key-words. Unilateral centrifugation

In this test, subjects are rotated about an earth vertical axis at a velocity that is relatively high, e.g., 400 degrees per second. After rotating at a constant velocity for several seconds, the subject is gradually moved 4 cm first to the right, and then to the left, along an interaural axis, to a position at which one utricle and then the other becomes aligned with the axis of rotation. At these points, one utricle is exposed to the combination of gravity and a centrifugal acceleration of 0.4 g, corresponding to an apparent roll-tilt of 21.7 degrees. The other utricle is only exposed to gravity. This stimulus induces ocular counter-rolling, *i.e*., ocular torsion, which is measured using three-dimensional video-oculography (VOG). This test dates from the early 1990s, as described by Wetzig *et al*.,[1] and was further developed by Clarke *et al*.[2] The amount of ocular counter-rolling is a linear function of the apparent gravito-inertial tilt of the head during the lateral translation.[3] Using this method, utricular sensitivity and the preponderance of the right or left utricle can be assessed separately. This method is much more powerful than the simple lateroflexion test because it permits the localisation of the side of utricular dysfunction.[4]

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Vestibular evoked myogenic potentials

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Key-words. Galvanic; tone burst; vestibular evoked myogenic potential; vestibulocollic

Abstract. *Vestibular evoked myogenic potentials.* The testing of Vestibular Evoked Myogenic Potentials has become a well-established approach for the exploration of sacculocollic pathways. Two types of tests have been described: VEMP induced by high intensity sound (click or tone burst) and VEMP induced by galvanic stimulation. We describe a method for recording the VEMP using both types of stimulation, and how to interpret them, and we report several clinical applications. Each of these techniques activates the vestibular afferents in different ways, and is a complementary tool for testing vestibular function at the level of the saccule or the inferior vestibular nerve.

Introduction

Patients suffering from vertigo are usually examined using caloric and rotation tests. These tests are essential, but they only inform the clinician about one of the five pairs of labyrinthic receptors, and they fail to establish a full picture of the functionality of the otolith receptors.

Vestibular evoked myogenic potential (VEMP) testing has become a well-established approach for the exploration of the sacculocollic pathways. This test has several advantages compared with other otolithic tests: it studies saccular function and the sacculocollic pathways selectively and never compensates. It requires the standard equipment for auditory evoked potentials to deliver the triggered sounds as well as EMG equipment.

We describe two types of test: VEMP induced by high-intensity sound (click or tone burst) and VEMP induced by galvanic stimulation. Both of these techniques activate the vestibular afferents in different ways and they are complementary.

VEMP induced by loud sound

Von Bekesy first discovered the sensitivity of the human vestibule to loud sound in 1935 and several studies since have confirmed this. It has been reported that a high-intensity sound induces an inhibitory impulse in the ipsilateral sternocleidomastoid (SCM) muscle.[1] Intense monaural clicks and tones give rise to short latency biphasic positive-negative responses (p13n23) in the ipsilateral SCM muscles if they are activated tonically.[2] These responses are preserved in subjects with severe sensorineural hearing loss and absent in patients who have undergone selective vestibular neurectomy and are therefore vestibular-dependent.[3] Several reports based on animal studies have suggested that these responses originate from the sacculus. Primary saccular afferents, due to their proximity to the footplate of the stapes, may be mechanically stimulated by loud sound[4] or stimulated by means of Eddy currents. It has been reported that the VEMP may travel along the hypothesised response pathway from the vestibular sacculus to the inferior vestibular nerve, vestibular nucleus, lateral (Deiter's) nucleus, and lateral vestibulospinal tract to the SCM muscle.[1]

Methods

VEMPs can be evoked by two kinds of auditory stimuli: clicks (0.1 ms, 100 dB nHL) and tone bursts (250, 500, 750 or even 1000 Hz, tone bursts of 500 Hz are often used, 95 dB nHL, rise/fall time = 2 ms, plateau time = 2 ms, repetition rate = 5.1 Hz). These stimuli are presented through a headphone, and the VEMPs are recorded on the SCM muscle ipsilateral to the stimulated ear.

Electromyographic activity in the SCM muscle is recorded using surface electrodes on the upper half of the SCM muscle with a reference electrode on the upper edge of the sternum and a ground electrode on the forehead. It is essential for the SCM to be in a strong contracted state during the recording. Several methods can be used for this purpose. The seated subject may be required to flex the

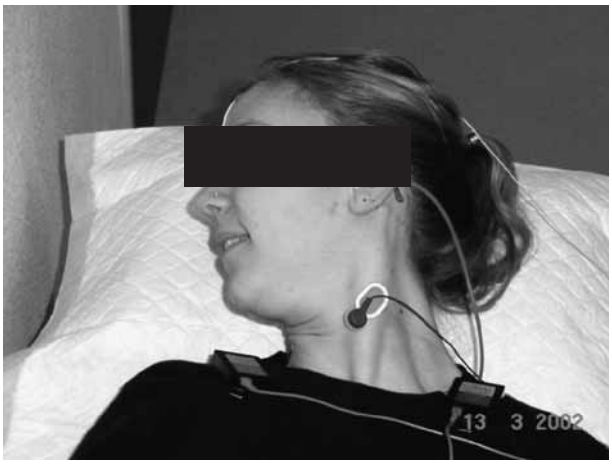

Figure 1
Electrode placement and contraction of the SCM muscle. Insert earphones are used. (Picture UZA).

head approximately 30 degrees forward and rotate it approximately 90 degrees to one side. Alternatively, patients can be placed in a supine position and be instructed to turn and hold their heads as far as possible toward the side contralateral to the stimulated ear to activate the ipsilateral SCM muscle (Figure 1). The vestibulospinal pathways have an inhibitory action on the SCM, and the VEMP records result from a diminution of the EMG activity of the SCM induced by the sound stimulation. EMG responses are amplified, band-pass filtered (20 Hz to 2 kHz), and averaged over a series of approximately 128 stimuli. Each recording should be repeated twice.

Two types of waves are recorded for the SCM muscle ipsilateral to the stimulated ear: short latency waves and long latency waves. The short latency waves (Figure 2) are composed of a first positive wave with a mean latency of 12 ms (p13), followed by a negative wave with a mean latency of 20ms (n23).

These waves originate from the activation of the sacculospinal pathways. Stimulus intensity largely determines the response, as can be seen in Figure 3. The long latency waves appear with mean latencies of 30 ms (n34) and 44 ms (p44) respectively. They originate from cochlear afferents. These last waves appear to be inconsistent, and therefore have no potential clinical application.[5]

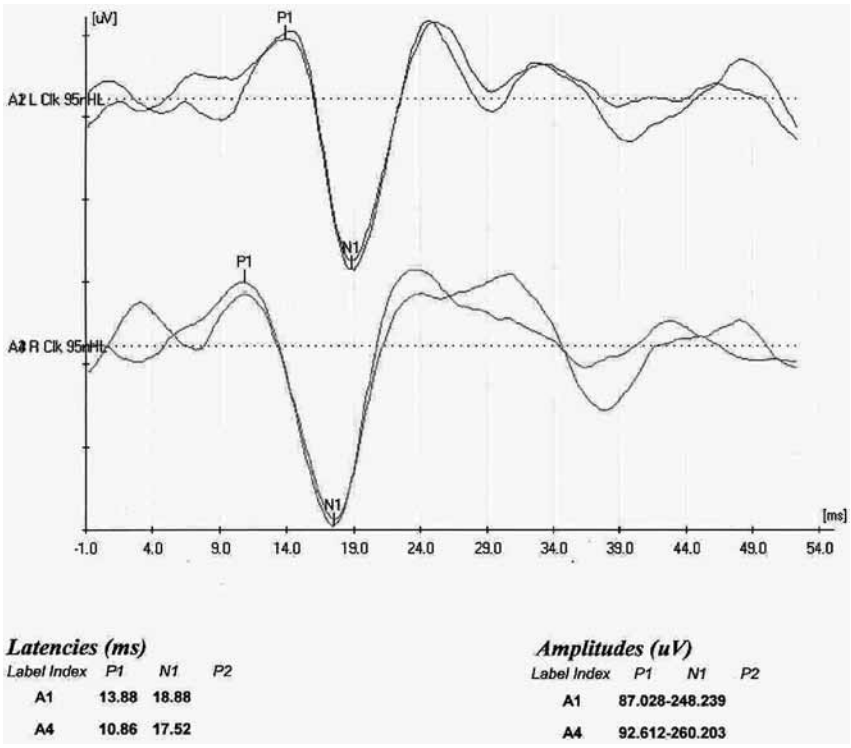

Figure 2
Click-induced VEMPs: the short latency waves are composed of a first positive wave p13 (p1), followed by a negative wave n23 (n1).

Results

VEMP responses are present in all normal subjects under the age of 60.[4] After the sixth decade the amplitude of the reflex decreases progressively by 25-30% per decade, but there are no differences in response amplitudes between the sexes. The absence of VEMP on one side indicates a disorder in the ipsilateral sacculospinal pathway and/or the mobility of the footplate.

It has been suggested that the most cautious interpretation of the potentials seems to be their ability to identify the asymmetry between left and right sides and

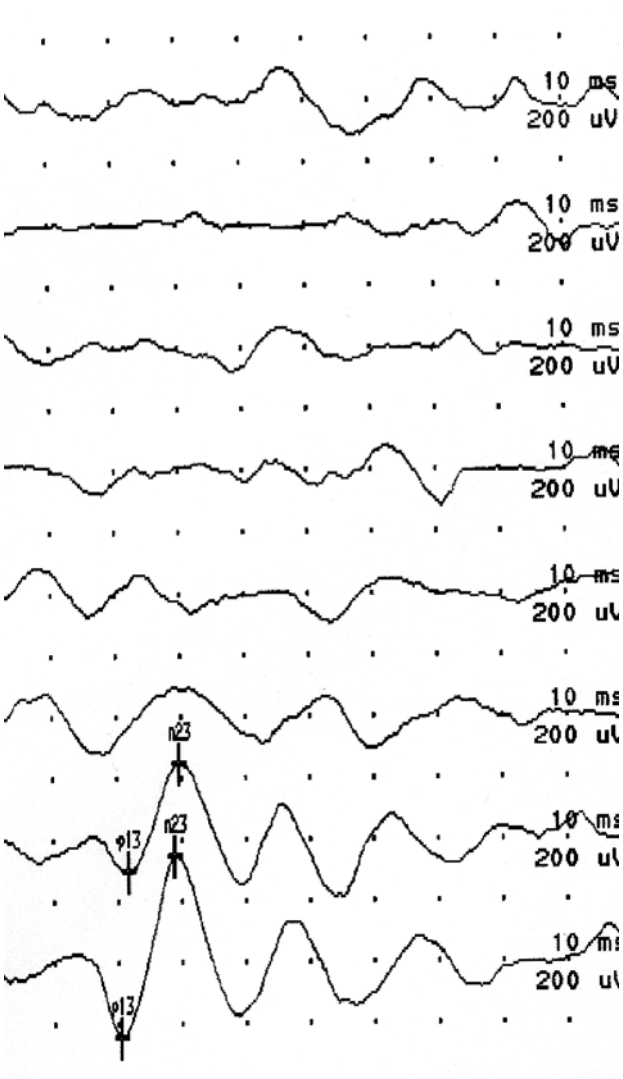

Figure 3
VEMP responses as a function of stimulus intensity (500 Hz tone burst). From top to bottom: 50, 60, 65, 70, 75, 80, 85 and 90 dB nHL. A clear p13-n23 waveform is seen only at intensities above 80 dB.

therefore to suggest the likely side of the pathology.[1] The tracings are too non-specific to allow for a discrimination of the underlying pathology or to suggest a diagnosis. Furthermore, the amplitude of the p13n23 waves varies widely between patients, making it difficult to use this parameter for clinical evaluation. The amplitude of the wave also depends on muscular tension during recording, increasing the difficulties for interpretation and comparison.

Welgampola *et al.*[4] provided reference values in 2001 for subjects below the age of 60.

Since the relationship between the reflex amplitude and the tonic EMG activity has been found to be linear, raw amplitudes were divided by the mean rectified EMG activity for 20 ms prior to stimulus onset to correct for differences in activation (corrected reflex amplitude). All subjects had corrected click-evoked reflex amplitudes of at least 0.5. Side-to-side differences in corrected reflex amplitudes were expressed as an "asymmetry ratio" (AR) calculated as AR = 100 (Al-Ar)/(Al+Ar), and a "side-to-side ratio" (SR), which was Al/Ar. In subjects under the age of 60 years, ARs of up to 35% and SRs of up to 2.1 were found to be normal for click-evoked responses.

The mean p13n23 peak latencies were 12.0 and 20.3 ms, respectively, for clicks. Side-to-side differences in the p13 latency were always less than 3.6 ms and less than 3.8 ms in the n23 latency. Given the strong dependency of amplitude on the SCM contraction state, it is highly advisable to measure the mean rectified voltage during the VEMP recording. It is only if this is done that the differences between left and right VEMP amplitudes can be attributed to a genuine sacculocollic pathway lesion on one side. Otherwise, the difference in VEMP amplitude may be induced by a difference in muscle contraction between the left and right SCM. Furthermore, there must be no air bone gap since this obviously does not deliver the appropriate stimulus to the saccule.

When comparing click and tone-burst evoked responses, Welgampola *et al.*[3] discovered that amplitudes of responses were not significantly different. On the contrary, the average p13 and n23 latencies of responses to an optimal tone burst stimulus were 13.1 and 22.8 ms, both occurring significantly later than the corresponding latencies for clicks.

The clinical application of VEMP has been reported for patients with acoustic tumours, Ménière's disease and vestibular neurolabyrinthitis. De Waele *et al.*[5]

showed that VEMPs were absent in 54% of 60 patients with Ménière's disease. However, it has also been demonstrated that VEMP can be augmented in cases of saccular hydrops.[6]

Since most acoustic tumours are thought to originate from the inferior vestibular nerve, VEMP might be the most useful test for the detection of these tumours.[1] Chen *et al.*[7] reported that 89% of patients with CPA tumours had no VEMP on the affected side. Matsuzaki *et al.*[8] reviewed the files of 33 patients with acoustic tumours, finding two cases with normal ABR and absent VEMP.

They assumed that VEMP might be more sensitive for detecting early acoustic tumours involving only the inferior vestibular nerve. De Waele *et al.*[5] also showed that, after surgery for these tumours, VEMPs were definitely abolished, thereby demonstrating the absence of central compensation for the reflex.

In the case of vestibular neuritis, Ochi *et al.*[1] showed that VEMPs were absent in two cases out of three. Similar results were obtained by De Waele *et al.*,[5] and they concluded that the viral lesion affects the inferior vestibular nerve in two-thirds of cases.

VEMP evoked by galvanic stimulation

Watson and Colebatch[9] showed in 1998 that galvanic stimulation applied to the mastoid process also evoked myogenic responses in the SCM, and that these responses disappeared after selective vestibular nerve section. They therefore seemed to be of vestibular origin, like those evoked by loud sound. Watson *et al* [9] suggested that galvanic stimulation stimulated the most distal portion of the inferior vestibular nerve ("spike trigger zone"), while the sound stimulation acted directly on the saccular receptor. Accordingly, Murofushi *et al.*[10] showed that galvanic-evoked myogenic responses in the SCM are useful in differentiating labyrinthine lesions from retrolabyrinthine lesions in patients with an absence of VEMP evoked by clicks.

Methods

A transmastoid galvanic stimulation of 4 mA and 2 ms is used, and the VEMPs are recorded on the ipsilateral SCM using the method described above for VEMPs evoked by loud sound (the SCM must be tonically activated). With this method, there is often a problem with stimulus artefact, and a technique of trace subtraction has to be used to minimise it:[9] the average EMG when stimulation is applied during relaxation consists of stimulus artefact only, while the average during tonic activation consists of stimulus artefact plus the reflex response; the first trace is subtracted from the second to cancel out the artefact, and to obtain the reflex response alone.

A biphasic response similar to the one observed with loud sound stimulation is observed on the SCM ipsilateral to the galvanic stimulation occurring at similar latency. In addition, a biphasic negative-positive (n12p20) response is observed in the other SCM. Late responses can also be seen, but these are not of vestibular origin.

Results

Welgampola *et al.*[4] have also studied VEMP induced by galvanic stimulation and have provided reference values. In this study, all patients below the age of 60 had detectable reflexes with corrected reflex amplitudes of at least 0.3 and a decrease in reflex amplitude was seen after the seventh decade. These authors propose the following reference values for side-to-side differences in corrected reflex amplitudes: for subjects below the age of 60 years, an AR of up to 41% and an SR of up to 2.4. The mean p13n23 peak latencies were 12.1 and 20.2 ms respectively.

Clinical application of VEMP induced by galvanic stimulation was reported to distinguish labyrinthine from retrolabyrinthine lesions in patients with an absence of VEMP evoked by loud sounds. Murofushi *et al.*[10] recorded galvanic-evoked VEMPs in patients with an absence of VEMP evoked by clicks. All patients who were diagnosed as having Ménière's disease or delayed endolymphatic hydrops showed normal galvanic-evoked responses on the affected side, while 88% of patients who were diagnosed as having cerebellopontine angle tumours showed no response or decreased responses, even to galvanic stimulation.

Conclusion

VEMP testing is a complementary tool for assessing vestibular function at the level of the saccule or the inferior vestibular nerve.

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Normative data in ENG and VNG

F. Wuyts and R. Boniver

Normative data from different papers and reports		
Test items	**Normative limits (μ ± 2s)**	**Reference, number of cases**
Spontaneous nystagmus	α 4°/s	1,2
	α 5°/s	3,4
	α 6°/s	5,6
	α 7°/s	7,8,9
	α 6 beats/10 s	3
Ocular motor testing		
Saccades		
Peak velocity for 20° saccade:	210°/s	4,10
lower limit	252°/s	11, *N* = 20
	283°/s	12, *N* = 38
Latency for 20° saccade	104-365 ms	13, *N* = 34 (IR method)
	128-255 ms	12, *N* = 38
Smooth pursuit gain at 0.3 Hz	>0.80	1,14,15
Optokinetic nystagmus asymmetry	<13%	12, *N* = 38
	<16%	1, *N* = 43

Normative data from different papers and reports		
Test items (μ ± 2s)	**Normative limits number of cases**	**Reference,**
Positional testing	<6°/s	5
Rotatory chair testing		
Sinusoidal harmonic acceleration Test		
Gain		
0.05 Hz, 60°/s	0.20-0.80	16, *N* = 20
0.05 Hz, 60°/s	0.13-0.77	17, *N* = 10
0.05 Hz, 50°/s	0.24-0.85	12, *N* = 38
0.05 Hz, 60°/s	0.38-0.98	18, *N* = 167
Phase		
0.05 Hz, 60°/s	6-14°	16, *N* = 20
0.05 Hz, 60°/s	2-20°	17, *N* = 10
0.05 Hz, 50°/s	-1-18°	12, *N* = 38
0.05 Hz, 50°/s	-1.9-24°	19, *N* = 50
0.05 Hz, 60°/s	0.8-20.2	18, *N* = 167
Directional preponderance		
0.05 Hz, 60°/s	≤15%	18, *N* = 208
0.05 Hz, 50°/s	≤24%	12, *N* = 38
Velocity step 90°/s		
Gain	0.33-0.72	20, *N* = 20
Time constant	11-26 s	20, *N* = 20
Directional preponderance	≤22%	20, *N* = 20
Velocity step 100°/s		
Gain	0.27-0.99	1, *N* = 43
Time constant	5-19.4 s	1, *N* = 43

Very important remarks

- Changing light conditions dramatically alter the calibration factors.
- It is crucial to perform calibrations repeatedly throughout the entire ENG.
- The clinician should never rely blindly on the computer output without inspecting the traces and the calculation results.
- Patients referred for ENG should not take tranquillisers, sedatives or alcohol for 48h before testing.
- Continuous visualisation of the patient and monitoring of the eye recordings are essential for test accuracy as well as for patient comfort and safety.
- Patient alertness has a major effect on the VOR. Ensure optimal alertness of the patient throughout the ENG examination.
- A clinically significant spontaneous nystagmus should appear consistently throughout the ENG test.

Normative data from different papers and reports		
Test items	**Normative limits (μ ± 2s)**	**Reference, number of cases**
Caloric testing		
Labyrinth asymmetry (%)	≤25%	5, *N* = 114
	≤19.8%	3, *N* = 30
	≤19%	12, *N* = 38
	≤15%	21, *N* = 47
	≤22%	1,2, *N* = 43
	≤20%	8, *N* = 58
	≤20%	22, *N* = 49
	≤25%	18, *N* = 167
General labyrinth asymmetry limit	≤22%	Meta-analysis
Directional preponderance (%)	≤23%	5, *N* = 114
	≤22.7%	3, *N* = 30
	≤16%	12, *N* = 38
	≤18%	21, *N* = 47
	≤28%	1,2, *N* = 43
	≤26 %	8, *N* = 58
	≤27%	22, *N* = 49
	≤31.8%	8, *N* = 167
General directional preponderance limit	≤26 %	Meta-analysis

Main parameters of interest of the standard vestibular test protocol	
Protocol item	**Parameters of interest**
Ocular motor screening battery	
Spontaneous nystagmus detection	Nystagmus direction, SCV
Gaze-evoked test (centre, 30° left/right, 15° up/down)	Nystagmus at different positions
Saccades	Velocity, latency, accuracy, binocular asymmetry
Optokinetic nystagmus	Gain, left-right asymmetry
Smooth pursuit	Gain, left-right asymmetry, morphology
Position tests	
Positioning testing	Nystagmus direction, latency, fatigability
Positional testing	Nystagmus SCV, fixation suppression
Vestibular tests	
Rotatory chair test	Gain, phase, time constant, asymmetry
Caloric test	Maximum SCV, labyrinth asymmetry, nystagmus asymmetry, total responsiveness

- Failure in the ocular motor pathways usually leads to consistently abnormal patterns. The patient can therefore either perform the test or not perform the test at all. Decreased alertness interferes with repeatability.
- During rotational testing, let the patient perform mental tasks, such as counting backwards in steps of 3.
- Do not talk continuously to the patient because that provides orientation clues that influence the VOR.
- If the gain is too low, measures of asymmetry or phase are inaccurate, and should be interpreted with caution.

Caloric test

- Maintain alertness throughout the caloric test.
- Calibrate prior to each irrigation since, in particular, the effect of the stimulus changes the impedance of the electrodes (due to sweating etc). Calibration is therefore required before each irrigation.
- Caloric responses should be consistent. If one out of four irrigations does not concur with the others, it should be repeated.

Conclusion

- Every laboratory is strongly advised to establish its own normative data.
- Meta-analysis indicates an upper normal limit for labyrinth asymmetry of 22% and for directional labyrinth preponderance of 26%.
- A dedicated and well-trained technician is crucial for the reliability of vestibular tests.

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Vertigo and psychological disorders

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Abstract. *Vertigo and psychological disorders.* Vertigo may be a symptom of psychiatric illness. Alternatively, vestibular dysfunction or other organic causes of dizziness may trigger psychiatric disorders such as anxiety, depression or panic attacks. Different mechanisms might account for the link between anxiety and vestibular disorders. The ENT specialist must be aware of this reality to take into account the psychological dimension of vertigo in his diagnosis approach. This may lead to different types of treatment depending of the nature of the disorder. Some questionnaires can be useful in this respect.

Introduction

Dizziness caused by vestibular dysfunction, like pain and many other illnesses, involves psychological reactions.[1] These consequences can vary considerably depending on whether or not psychiatric patients are involved. On the other hand, vertigo can be a symptom of a psychiatric disorder with no objective symptoms of a disorder of the vestibular system or of other neurological pathologies. However, this dichotomy in which psychological difficulties are the cause or the consequence of dizziness generally fails to reflect the complex reality and leads to sub-optimal care. Typically, neurotologic disorders and psychiatric disorders coexist and interact.[2]

Discussion

Vertigo can be defined as an illusion of motion. As an illusion, it is a subjective sensation involving the subconscious and depending on the central nervous system status of the patient. The interpretation of a vestibular disorder depends on the emotional condition of the patient. Many factors have to be considered: the personality of the subject, the subject's relationship with the doctor, the subject's understanding of the pathology and the memory of similar sensations experienced before.

The psychological reactions associated with dizziness can be particularly important for different reasons. First of all, vertigo can result in limitations on everyday life (social contacts, professional abilities, mobility). This can contribute to a reduction in self-esteem and further depression. Secondly, vertigo often evolves unpredictably, with fluctuations in the intensity of imbalance. This unpredictable component of vertigo is a factor leading to anxiety and may lead to phobic avoidance responses in specific situations. The role of close relations is very important too. They often have difficulty in understanding what is happening to the patient, particularly in the absence of obvious symptoms. This lack of understanding can lead to poor support and exacerbate the psychological difficulties of the vertigo patient.

The interactions between vestibular disorders and psychiatric disorders can be explained by somatopsychological mechanisms, psychosomatic mechanisms and by neurological linkage mechanisms.[2]

Somatopsychological mechanisms involve the psychological and behavioural consequences of vestibular dysfunction. One possible consequence is anxiety. DSM-IV (Diagnostic Manual of the American Psychiatric Association) lists 11 anxiety disorders.[3] Three of these are "Panic Disorder without Agoraphobia", "Panic Disorder with Agoraphobia" and "Agoraphobia without any history of Panic Disorder". In these disorders, an association with vestibular disorder is likely to be found. Dizziness is often situation-specific. Vestibular disease therefore favours avoidance responses in some situations, and these responses are sometimes confused with psychogenic vertigo. The

mismatch between the three sensorial inputs (vestibular, visual and proprioceptive) can lead to dizziness even in healthy individuals (physiological vertigo). In vestibular disorders, the patient can develop unusual sensitivity to some stimuli or inadequate balance strategies (visual dependence for example).[4] Space and motion sensitivity is a heightened awareness of non-vestibular sensation. This can interfere with social activities and lead to psychological decompensation.

Agoraphobia is defined in DSM-IV as anxiety about being in places or situations in which escape might be difficult or help might not be available in the event of a panic attack or panic-like symptoms. Most, but not all, people with panic disorder develop at least some degree of agoraphobia. In extreme cases, an individual with panic disorder and agoraphobia may be completely unable to leave the house. More typically, people with agoraphobia experience some restrictions in what they are able to do but they are able to leave the house, especially if they are accompanied by someone they know. In some cases, agoraphobia may be explained by a psychological mechanism (panic disorders). In others, it can be thought of as functional dizziness. Agoraphobia may be a reaction to dizziness rather than a cause, a reasonable adaptation to conditions that affect balance in an unpredictable way: open places have neither surfaces that can be used for support nor close visual references.[4] The feeling of dizziness and subjective postural imbalance associated with anxiety felt in wide open spaces and public places may be initiated by the physiological impairment of visual control over body sway linked to the distance to stationary objects in the seen environment.[5]

Acrophobia results when physiological height vertigo induces a conditioned phobic reaction characterised by dissociation between the objective and the subjective risk of falling.[1] It is likely to occur as a post-traumatic neurotic reaction, sometimes initiated by a traumatic lesion of the otoliths. Like agoraphobia, then, it can be seen as functional vertigo.

The syndrome of phobic postural vertigo, which was described by Brandt,[1] is characterised by a combination of situational triggered panic attacks including vertigo and subjective postural and gait instability, and the fear of imminent death. Patients complain of vertigo rather than anxiety and feel physically ill. This syndrome could be explained by the hypothesis that an impairment of the space constancy mechanism leads to partial uncoupling of the efferent copy for active head movements. This triggers phobic attacks. Brandt claims that it represents the third cause of vertigo in specialist consultations. Clinical experience does indicate that there are people with positional vertigo who are conditioned to be dizzy, with or without objective signs of vertigo. At present, this syndrome is of uncertain validity or significance as it lacks a specific test for diagnosis.

Psychosomatic mechanisms involve an alteration in vestibular function as a result of psychiatric conditions. As awareness and somnolence affect the vestibular function (modifying the gain of the VOR), anxiety and hyperventilation can affect vestibular responses. Some patients, after compensation of a peripheral vestibular disease (initial major rotational vertigo), still complain of chronic indefinite dizziness and postural imbalance because of psychiatric disorders (anxiety and phobic postural imbalance).

In addition to psychiatric dizziness and balance disorders without any psychiatric troubles, and the functional overlap between both types of troubles, vestibular disorders and psychiatric disorders in some individuals could be manifestations of a common underlying disorder of the central nervous system. This is called linkage and may apply to some patients with anxiety disorders. The explanation could be that there are shared brain pathways that mediate autonomic responses. The effect of some neurotransmitters (noradrenalin and serotonin) may also play a role.[6]

Many dizzy patients present with psychological decompensation because of vertigo, but some personalities are more at risk: obsessive-compulsive personalities, perfectionist traits, pre-existing anxiety or depressive problems, somatisation.[1,4] Some studies have shown a relationship between a specific psychologic profile and the development of Ménière's disease. Those studies are controversial.[7,8] Some people are predisposed to develop a high degree of anxiety when they experience difficult situations such as dizziness. The borderline between physiological vertigo induced by sensorial mismatches and true kinetosis, for example, is not always evident. Dizziness that occurs after an accident (post-traumatic vertigo) also seems to be associated with a high risk of psychological decompensation.

Vertigo can also be a symptom of mental disorder. In psychiatric

dizziness, the dizziness is part of a recognised psychiatric syndrome: dizziness in panic attack and abnormal gait in conversion hysteria are typical examples. Here, dizziness cannot be explained by vestibular dysfunction.

As doctors, we must be able to distinguish between psychological problems caused by vertigo and true psychiatric disorders. In the latter case, patients should be referred to a specialist service. There should be an adequate otoneurologic assessment beforehand. Unnecessary and prolonged examinations must be avoided, for obvious economic reasons, but more to avoid to reinforcing patient belief in an organic illness.

Putting aside this psychiatric pathology, ENT clinicians should be able to manage therapy for our patients, even if they have psychological problems. This assumes a willingness to listen, which in turn implies providing enough time in our consultations. After a full and thorough examination, patients should receive the most complete information possible about their illness: symptoms, consequences, evolution, and proposed treatment. Where possible, and subject to the agreement of the patient, it can be very useful to provide that information in the presence of close relations. A better understanding of the disorders, organic disorders and psychological reactions will help them to support the patient. A close relationship with the doctor gives patients a reassuring feeling and prevents the development or the aggravation of neurosis. The treatment should include vestibular training. This helps to develop vestibular compensation and to recover adequate balance strategies. It also plays a role in psychological support for the patient. In some cases, pharmacotherapy, psychotherapy and behavioural therapy should be considered. We must remain aware that most of the medical treatments used for anxiety, depression and dizziness interfere with central vestibular compensation.

Because subjective perceptions are very important in the psychological domain, the use of questionnaires in the examination phase can be helpful. Some questionnaires evaluate the handicap caused by vertigo; others are more psychological, tending to evaluate patient levels of depression or anxiety. Repeating the questionnaire later is one way of assessing patient progress and psychological disorders. This can provide information about the efficiency of our therapeutic approach and, depending on the results, about the eventual need for specialist psychiatric advice. Another advantage of the systematic use of such questionnaires is the possibility of including some psychological aspects of the management of vertigo in multi-centre studies. Those questionnaires are particularly useful in this area, but they should never become a substitute for the clinical sense of the practitioner based on professional experience and a good relationship with the patient. The questionnaire structures mean that they provide a picture of situation of the patient at a very specific point in time. This may not be an accurate reflection of reality when there are fluctuations in symptoms. The subjective nature of most of the questions means that the questionnaires are comparable from time to time in the same patient but do not allow for meaningful comparisons between patients.

Three questionnaires have been included at the end of this paper in the English versions. Where translated versions have been validated, they have also been included.

Conclusion

Psychogenic vertigo can be broken down into three forms: vertigo as a symptom (anxiety, depression, hysteria, psychosis, post-traumatic syndrome, stimulation); vertigo as a defined syndrome: agoraphobia, acrophobia, phobic postural vertigo (which is controversial); and the psychological overlay of organic vertigo syndromes in predisposed personalities and manifest psychiatric disorders.

The need to take into account the psychological dimension of pathology in our otoneurologic consultations is now well established. Ideally, the scope of those consultations should include all pathologies. This implies attentive and focused listening from the physician. Adapted questionnaires can be useful, providing information about depression or anxiety and the handicap caused by this pathology.

Questionnaires

HAD (Hospital Anxiety and Depression scale)[9]
For each item select the reply which comes closest to how you have been feeling in the past weeks.
- most of the time
- a lot of the time
- from time to time
- not at all

1. I feel tense or wound up
2. I still enjoy the things I used to enjoy
3. I get a sort of frightened feeling as if something awful is about to happen
4. I can laugh and see the funny side of things
5. Worrying thoughts go through my mind
6. I feel cheerful
7. I can sit at ease and feel relaxed
8. I feel as if I am slowed down
9. I get a sort of frightened feeling like butterflies in the stomach
10. I have lost interest in my appearance
11. I feel restless as if I have to be on the move
12. I look forward with enjoyment to things
13. I get sudden feelings of panic
14. I can enjoy a good book or radio or TV programme

HAD (french version)[10]
Pour chaque item, sélectionnez la réponse qui convient concernant les semaines qui ont précédé.
- la plupart du temps
- souvent
- de temps en temps
- jamais

7 items d'anxiété (A), 7 items de dépression (D), cotation de 0 à 3

A/ 01. Je me sens tendu ou énervé
D/ 02. Je prends plaisir aux mêmes choses qu'autrefois
A/ 03. J'ai une sensation de peur comme si quelque chose d'horrible allait m'arriver
D/ 04. Je ris facilement et vois le bon côté des choses
A/ 05. Je me fais du souci
D/ 06. Je suis de bonne humeur
A/ 07. Je peux rester tranquillement assis à ne rien faire et me sentir décontracté
D/ 08. J'ai l'impression de fonctionner au ralenti
A/ 09. J'éprouve des sensations de peur et j'ai l'estomac noué
D/ 10. Je ne m'intéresse plus à mon apparence
A/ 11. J'ai la bougeotte et n'arrive pas à tenir en place
D/ 12. Je me réjouis d'avance à l'idée de faire certaines choses
A/ 13. J'éprouve des sensations soudaines de panique
D/ 14. Je peux prendre plaisir à un bon livre ou à une bonne émission de radio ou de télévision

DHI (Dizziness Handicap Inventory)[11,12]
The Dizziness Handicap Inventory (DHI) can be used to determine the level of impairment felt by patients with dizziness. It incorporates the measurement of emotional function and the physical impact of the dizziness on the person's life.

Answer by No, Sometimes or Yes.

1. (P) Does looking up increase your problem?
2. (E) Because of your problem do you feel frustrated?
3. (F) Because of your problem do you restrict your travel for business or recreation?
4. (P) Does walking down the aisle of a supermarket increase your problem?
5. (F) Because of your problem do you have difficulty getting into or out of bed?
6. (F) Does your problem significantly restrict your participation in social activities such as going out to dinner going to the movies dancing or going to parties?
7. (F) Because of your problem do you have difficulty reading?
8. (P) Does performing more ambitious activities such as sports, dancing, household chores such as sweeping, or putting dishes away increase your problems?
9. (E) Because of your problem are you afraid to leave your home without having someone accompany you?
10. (E) Because of your problem have you been embarrassed in front of others?
11. (P) Do quick movements of your head increase your problems?
12. (F) Because of your problem do you avoid heights?
13. (P) Does turning over in bed increase your problem?
14. (F) Because of your problems is it difficult for you to strenuous housework or yard works?
15. (E) Because of your problem you are afraid people may think you are intoxicated?
16. (F) Because of your problem is it difficult for you to go for a walk by yourself?
17. (P) Does walking down a sidewalk increase your problem?
18. (E) Because of your problem is it difficult for you to concentrate?
19. (F) Because of your problem is it difficult for you to walk around your home in the dark?
20. (E) Because of your problem are you afraid to stay home alone?
21. (E) Because of your problem do you feel handicapped?
22. (E) Has the problem placed stress on your relationships with members of your family or friends?
23. (E) Because of your problem are you depressed?
24. (F) Does your problem interfere with your job or household responsibilities?
25. (P) Does bending over increase your problem?

(E): emotional items
(F): functional items
(P): physical items

Responses: No = 0 point
Sometimes = 2 points
Yes = 4 points

Sub score for Emotional items = sum of points for questions 2 9 10 15 18 20 21 22 23 /36
Sub score for Functional items = sum of points for questions 3 5 6 7 12 14 16 19 24 /36
Sub score for Physical items = sum of points for questions 1 4 8 11 13 17 25 /24
Total score = sum of points for all 25 items /100
The higher the score is the greatest the handicap.

Questionnaire D.H.I (French translation by J. P. Demanez, Université de Liège, 1991)

Instructions: Le but de ce questionnaire est de déterminer les difficultés que vous éprouvez dans la vie courante par le fait de vos vertiges et de votre déséquilibre. Veuillez répondre à chacune des questions selon le code suivant:

0 = non, jamais.
1 = rarement.
2 = parfois.
3 = souvent.
4 = oui, en permanence.

Pour évaluer votre réponse, tenez compte exclusivement de l'influence éventuelle de vos malaises vertigineux ou de déséquilibre pendant la période des quatre dernières semaines._

1. (p) Le fait de regarder vers le haut accentue-t-il vos troubles?
2. (e) A cause de votre problème, vous sentez-vous découragé(e), désappointé(e)?
3. (f) A cause de vos malaises, limitez-vous vos déplacements professionnels ou de loisir?
4. (p) Vous déplacer dans une allée de grande surface commerciale augmente-t-il vos troubles?
5. (f) Par le fait de vos malaises, avez vous des difficultés à vous mettre ou à sortir du lit?
6. (f) Votre problème limite-t-il votre participation à des activités sociales comme dîner à l'extérieur, aller au spectacle, en soirée ou au dancing?
7. (f) Vos troubles réduisent-ils votre capacité de lire des livres ou des revues?
8. (p) Le sport, la danse ou des tâches ménagères (entretenir la maison, remettre la vaisselle...) accentuent-ils votre problème?
9. (e) A cause de vos malaises, évitez-vous de sortir de chez vous non-accompagné(e)?
10. (e) Votre problème a-t-il été responsable d'une sensation d'embarras face aux autres?
11. (p) Les mouvements brusques de la tête accentuent-ils vos troubles?
12. (f) En raison de vos malaises, évitez-vous les hauteurs, chaise, échelle, balcon?
13. (p) Vos troubles augmentent-ils lorsque vous vous tournez dans votre lit?
14. (f) Eprouvez-vous des difficultés à exécuter des tâches soutenues, dans le ménage ou dans le jardin?
15. (e) Du fait de vos troubles, craignez-vous que l'on vous considère en état d'ivresse?
16. (f) Du fait de votre problème, vous est-il difficile d'aller vous promener seul(e)?
17. (p) Votre malaise s'accroît-il lorsque vous marchez le long d'un trottoir?
18. (e) A cause de vos troubles, éprouvez-vous des difficultés de concentration?
19. (f) Eprouvez-vous des difficultés à sortir autour de votre maison?
20. (e) En raison de votre problème, craignez-vous de rester seul(e) chez vous?
21. (e) A cause de vos malaises, vous sentez-vous physiquement diminué(e)?
22. (e) Vos troubles ont-ils été responsables de relations tendues avec des membres de votre famille ou avec des amis?
23. (e) A cause de votre problème, vous sentez-vous dépressif(ve)?
24. (f) Vos troubles ont-ils une conséquence sur vos responsabilités professionnelles ou familiales?
25. (p) Vous pencher en avant accentue vos malaises?

0 = non, jamais.
1 = rarement.
2 = parfois.
3 = souvent.
4 = oui, en permanence.

FACTEUR
physique: 1, 4, 8, 11, 13, 17, 25 /28
fonctionnel: 3, 5, 6, 7, 12, 14, 16, 19, 24 /36
emotionnel: 2, 9, 10, 15, 18, 20, 21, 22, 23 /36

DHI (Dutch version)[13]

Het doel van deze vragenlijst is te bepalen in hoeverre u moeilijkheden ondervindt door uw probleem van duizeligheid en instabiliteit. Wilt u de vragen beantwoorden met ja, nee of soms. Bij het beantwoorden van de vragen moet u steeds voor ogen houden dat ze betrekking hebben op uw probleem van duizeligheid en instabiliteit. Indien u een situatie die we beschrijven niet hebt ervaren, probeer dan te denken aan een vergelijkbare situatie waarin u zicht hebt bevonden en antwoord voor die situatie.

P1 Neemt uw probleem toe wanneer u naar boven kijkt?
E2 Voelt u zich gefrustreerd door uw probleem?
F3 Beperkt u het reizen door uw probleem (zowel op privé- als op beroepsvlak)?
P4 Neemt uw probleem toe wanneer u in de supermarkt tussen de rekken loopt?
F5 Is het moeilijk om uit bed te komen door uw probleem?
F6 Beperkt uw probleem ingrijpend uw sociale leven (uit eten gaan, naar de film, gaan dansen, ...)?
F7 Wordt lezen bemoeilijkt door uw probleem?
P8 Neemt uw probleem toe wanneer u meer actief bent zoals bij sporten, dansen, het huishouden doen (poetsen, de vaat wegzetten, ...)?
E9 Bent u, door uw probleem, bang om het huis te verlaten zonder dat iemand u vergezelt?
E10 Door uw probleem, voelt u zich beschaamd in bijzijn van anderen?
P11 Neemt uw probleem toe door snelle hoofdbewegingen?
F12 Vermijdt u hoogtes door uw probleem?
P13 Neemt uw probleem toe bij het omdraaien in uw bed?
F14 Door uw probleem, is het moeilijk om inspannend werk te doen in huis of in de tuin?
E15 Door uw probleem, bent u bang dat mensen zouden denken dat u dronken bent?
F16 Door uw probleem, kunt u moeilijk alleen wandelen?
P17 Neemt uw probleem toe bij het wandelen op het voedpad?
E18 Door uw probleem, kunt u zich moeilijk concentreren?
F19 Door uw probleem, hebt u moeilijkheden om in het donker in uw huis te lopen?
E20 Door uw probleem, heeft u angst om alleen thuis te blijven?
E21 Voelt u zich gehandicapt door uw probleem?
E22 Heeft u probleem voor spanning gezorgd in uw relatie met familie of vrienden?
E23 Voelt u zich depressief door uw probleem?
F24 Heeft uw probleem invloed op uw verantwoordelijkheden in uw beroep of uw taken thuis?
P25 Neemt uw probleem toe wanneer u zich bukt?

P: physical
F: functional
E: emotional

PHQ (Patient Heath Questionnaire)[14,15]

The PHQ questionnaire is a very complete questionnaire exploring all fields of health. PHQ-9 is a more concise version, which has been standardised and validated by a lot of studies for the diagnosis and follow-up of depressive problems. This version seems to be the most useful in the management of dizzy patients.

PHQ-9
Name:
Age:
Sex:
Date:

1. Over the last 2 weeks, how often have you been bothered by any of the following problems?

0 not at all
1 several days
2 more than half the days
3 nearly every day

a. Little interest or pleasure in doing things
b. Feeling down, depressed, or hopeless
c. Trouble falling or staying asleep, or sleeping too much
d. Feeling tired or having little energy
e. Poor appetite or overeating
f. Feeling bad about yourself, or that you are failure, or have let yourself or your family down
g. Trouble concentrating on things, such as reading the newspaper or watching television
h. Moving or speaking so slowly that other people could have noticed? Or the opposite: being so fidgety or restless that you have been moving around a lot more than usual.
i. Thoughts that you would be better off dead during or of hurting yourself in some way.

2. If you checked off any problem on this questionnaire, how difficult have these problems made it for you to do work, take care of things at home, or get along with people?

not difficult at all
somewhat difficult
very difficult
extremely difficult

Score of Quick Depression Assessment
1-4 minimal
5-9 mild
10-14 moderate
15-19 moderately severe
20-27 severe

PHQ-9 (Dutch)
Naam:
Leeftijd:
Sex:
Datum:

A. Hoe vaak heeft u in de voorbije 2 weken last gehad van één van de volgende problemen?
   0 helemaal niet
   1 verschillende dagen
   2 meer dan de helft van de dagen
   3 bijna elke dag

a. Weinig interesse of plezier in uw gewone activiteiten.
b. Zich neerslachtig, depressief, hopeloos voelen.
c. Moeilijk inslapen, moeilijk doorslapen of te veel slapen.
d. Zich moe voelen of gebrek aan energie hebben.

e. Weinig eetlust of overmatig eten.
f. Een slecht gevoel hebben over uzelf- of het gevoel hebben dat u een mislukkeling bent- of het gevoel dat u zichzelf of uw familie heeft teleurgesteld.
g. Problemen om te concentreren, bv. om de krant te lezen of om TV te kijken.
h. Zo traag bewegen of zo langzaam spreken dat andere mensen dit zouden kunnen gemerkt hebben. Of integendeel, zo zenuwachtig of rusteloos zijn dat u veel meer rondliep.
i. De gedachte dat u beter dood zou zijn of de gedachte uzelf op een bepaalde manier pijn te doen.

B. Als u minstens één probleem op deze vragenlijst heeft aangeduid, hoe moeilijk maakten deze problemen het dan voor u om uw werk of uw huishouden te doen, of om op te schieten met andere mensen?

helemaal niet
een beetje moelijk
erg moeilijk
extreem moeilijk

PHQ-9 (french)
Nom:
Sexe:
Age:
Date:

A. Durant ces deux dernières semaines, à quelle fréquence avez-vous été gêné(e) par les problèmes suivants ?

0 pas du tout
1 durant plusieurs jours
2 plus de la moitié du temps
3 presque chaque jour

1. Peu d'envie ou de plaisir à faire les choses
2. Se sentir cafardeux (se), déprimé(e) ou désespéré(e).
3. Difficultés à vous endormir, à rester endormi(e) ou dormir trop longtemps.
4. Se sentir fatigué(e) ou avoir trop peu d'énergie.
5. Manque d'appétit ou manger excessivement
6. Vous sentir mécontent(e) de vous, ou avoir le sentiment d'être un(e) raté(e) ou que vous avez déçu votre famille ou que vous êtes déçu(e) de vous même.
7. Difficultés à vous concentrer, par exemple en lisant le journal ou en regardant la télévision.
8. Bouger ou parler si lentement que d'autres pourraient l'avoir remarqué, ou au contraire: être si nerveux(se) ou si agité(e) que vous ne pouvez rester en place.
9. Penser qu'il vaudrait mieux pour vous d'être mort ou de vous faire mal d'une manière ou d'une autre. Difficultés à vous concentrer, par exemple, en lisant le journal ou en regardant la télévision.

B. Si vous avez coché au moins un des problèmes sur ce questionnaire, quel degré de difficultés ce(s) problème(s) ont-ils occasionné dans la réalisation de votre travail, ou pour mener à bien vos tâches à la maison, ou pour fréquenter d'autres personnes?

pas difficile du tout
quelque peu difficile
très difficile
extrêmement difficile

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Medical treatment for vertigo

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Key-words. Vertigo; drug therapy

Abstract. *Medical treatment for vertigo.* An overview of the medical treatment options currently available for vertigo in Belgium is given. There are three sections: drug treatment for acute unilateral vestibular loss, Ménière's disease and motion sickness. The pharmacological properties, contra-indications, side-effects and dosage are discussed for each drug.

Introduction

An overview of the medical treatment options currently available for vertigo in Belgium is given. The pharmacological properties, contra-indications, side-effects and dosage are discussed for each drug. For more detailed information we refer to textbooks on pharmacology.[1-3]

1. Acute unilateral vestibular loss

For acute unilateral vestibular loss, medical treatment is appropriate during the first days to reduce vertigo and secondary neurovegetative signs (Table 1). These drugs should not be continued too long since they may interfere with compensation processes.

Table 1

Drugs for acute unilateral vestibular loss

generic name	dosage
dimenhydrinate	rectal 120-240 mg oral 3×50-80 mg
meclozine	oral 3×25-50 mg
sulpiride	intramuscular 100-200 mg oral 2×50-100 mg
promethazine	intramuscular 50 mg oral 2×25 mg

1.1. *Antihistamines of the benzhydril group*

1.1.1. Dimenhydrinate

Pharmacological properties: Dimenhydrinate belongs to the ethanolamines and has an antihistamine (H1-antagonist) and anti-emetic effect. Maximum plasma levels are reached in 1-2 hours and its half-life is 6 hours. It works after 30 minutes, and the effects last for 8 hours.

Contra-indications: prostatic hypertrophy, glaucoma.

Side-effects: somnolence, in case of prolonged use accommodation problems, dry mouth, urine retention, confusion or excitation in elderly people. In rare cases, tremor and gastrointestinal complaints. Very rare cases of leucopenia, agranulocytosis.

Dosage: For an acute attack of Ménière's disease, suppositories 60-120 mg. For motion sickness, 50-80 mg 30 min before the trip. In prolonged treatment: 50-160 mg before each meal and before sleep.

1.1.2. Meclozine

Pharmacological properties: Meclozine is an H1-antihistamine and anti-emetic drug with a prolonged effect. It works 2 hours after oral intake and the effects last for 8 hours.

Contra-indications: prostatic hypertrophy, glaucoma.

Side-effects: drowsiness

Dosage: adults: 25-50 mg a day preferably before sleep or 1 hour before the trip. Maximum dose 100 mg a day. It is not available for rectal application.

Children: syrup 1 mg/ml, <2 years 4 mg, 2-5 years 6 mg, 5-10 years 12 mg, if necessary 2-3 times a day.

1.1. *Antihistamines of the phenothiazine group*

1.2.1. Sulpiride

Pharmacological properties: Sulpiride is a specific antagonist of the dopamine D2/D3 receptors and belongs to the class of the benzamides. Low doses (150-600 mg) enhance dopamine transmission. High doses block the D2/D3 receptors. After intramuscular injection, maximal plasma levels are reached after 15-30 min. After oral intake the drug works for 4-5 hours. The half-life is 7 hours.

Contra-indications: phaechromocytoma

Side-effects: extrapyramidal symptoms, neuroleptic malignant syndrome (loss of consciousness with evolution to stupor and coma), autonomic symptoms with hyperthermia, tachycardia, peripheral vasoconstriction, transient hyperprolactinaemia which disappears after discontinuing the treatment.

Dosage: during an acute attack of vertigo: intramuscular injection of 100-200 mg.

Prolonged oral treatment: 2×50 mg, maximum 2×100 mg a day.

1.2.2. Promethazine

Pharmacological properties: Promethazine is an H1-antihistamine with a strong anticholinergic effect. It works 2 hours after oral intake, and the effects last for 6-18 hours. A stronger and faster effect is achieved after intramuscular injection.

Contra-indications: prostatic hypertrophy, glaucoma.

Side-effects: pronounced sedation, dry mouth

Dosage: during an acute attack of vertigo: intramuscular injection of 50 mg. Oral treatment: 25 mg twice a day.

2. Ménière's disease

2.1. *Acute attack*

The same drugs can be given as for an acute unilateral vestibular loss.

2.2. Prophylactic treatment

2.2.1. Betahistine

Pharmacological properties: Betahistine is a strong H3-antagonist, facilitating histamine transmission, a weak H1- (calcium release) and H2- (production cAMP) agonist. In animal experiments betahistine has been found to enhance cerebral and cochlear blood supply and improve oxygenation of the inner ear, enhance central compensation and reduce nystagmus in humans, probably through an effect on the vestibular nucleii. There is clinical evidence for an effect in Ménière's disease and an enhancement of compensation after acute unilateral peripheral vestibular loss. Betahistine has a prophylactic effect on Ménière's disease, and there is a limited effect during an attack.

Indications: Ménière's disease and other recurrent vertigo attacks.

Side-effects: gastrointestinal disturbances. Precautions in case of phaeochromocytoma, peptic ulcer and asthma

Dosage: Starting at 3×16 mg a day, increase dose up to 3×32 mg. If the frequency of the attacks declines, the dose can be reduced to 3×6-8 mg a day. If the treatment is effective, it is advised to continue the treatment for at least 3 months before slowly reducing.

2.2.2. Diuretics

The most frequently used diuretics in Ménière's disease are:
hydrochlorothiazide, 25 mg a day
chlorthalidone, 50 mg a day
spironolactone, 50 mg a day
acetazolamide, 250 mg a day.

3. Calcium antagonists, nootropic drugs

3.1. *Cinnarizine, flunarizine*

Pharmacological properties: These are selective calcium entry blockers which belong to group IV of the calcium antagonists. They have also an antihistamine (H1) effect. They inhibit contractions of vascular smooth muscle cells. The cellular influx of calcium is blocked in a tissue selective way without an effect on blood pressure and heart rate. Microcirculation is improved by raising erythrocyte deformability and reducing blood viscosity. Cellular resistance to hypoxia is increased. These drugs inhibit the stimulation of the vestibular system, resulting in a reduction of nystagmus and other autonomic disturbances. Compensation after acute unilateral vestibular loss is stimulated, probably through the inhibition of the reaction on the normal side. Cinnarizine blocks the calcium influx of the endolymph to the vestibular sensory cells.

Maximum plasma levels of cinnarizine are reached 2-4 hours after intake. The half-life is

4 hours and terminal half-life is 16 days. The maximum effect is reached after 1-3 hours, and lasts 6-8 hours.

Maximum plasma levels of flunarizine are reached in 2-4 hours and a steady state is reached at 5-6 weeks. The mean terminal elimination half-life is 18 days.

Indications: Cinnarizine: migraine prophylaxis, cerebrovascular disturbances, vertigo of peripheral and central origin, tinnitus, peripheral circulatory disorders such as intermittent claudication, prophylaxis of motion sickness. Flunarizine: migraine prophylaxis, vertigo of peripheral and central origin. The treatment should be discontinued after 2 months in case of vertigo and after 6 months for migraine.

Contra-indications: Cinnarizine: recent cerebral haemorrhage and extrapyramidal symptoms.

Flunarizine: depressive illness and Parkinson's disease.

Side-effects: Cinnarizine: somnolence and gastrointestinal disturbances, usually transient. In rare cases extrapyramidal symptoms, weight gain, depressive feelings. Flunarizine: drowsiness and/or fatigue (20%), usually transient, and weight gain (11%). Gastrointestinal disturbances. During chronic treatment (>6 months) and in elderly people: depression, extrapyramidal symptoms, insomnia, anxiety, galactorrhoea, dry mouth, muscle ache, skin rash.

Dosage: Cinnarizine: in adults 3x75 mg a day, for motion sickness 3x25-50 mg a day, at least 1 hour before the trip. Flunarizine: 10 mg daily (at night) for patients younger than 65 years of age and 5 mg for those older than 65 years of age.

3.2. *Piracetam*

Pharmacological properties: Piracetam is a nootropic drug, a psychotropic substance, that enhances the function of the higher telencephalic cerebral functions, which play a role in cognitive processes such as learning ability, memory en consciousness. It is thought to restore and protect central neural transmission by influencing membrane phospholipides and through the activation of the turnover of second messengers. Piracetam enhances microcirculation through the inhibition of platelet aggregation and an increase in erythrocyte deformability. It is also thought to antagonise vasospasm. It may have a positive effect on vertigo of central origin, particularly in elderly people. After oral intake, the maximum plasma level is reached after 30 minutes, and the maximum level is reached in cerebrospinal fluid after 2-8 hours. The half-life is 4-5 hours in the blood and 6-8 hours in cerebrospinal fluid.

Indications: Positive effects have been described for: memory loss in elderly people, vertigo, diminished consciousness and concentration, sickle cell anaemia, chronic alcoholism, coma, cognitive disturbances, cortical myoclonus. This is a registered drug for vertigo of central origin.

Side-effects: These are thought to occur in fewer than 5% of patients. Agitation, irritability, anxiety, disturbance of sleep and gastrointestinal complaints have been described.

Dosage: 30-160 mg/kg/day in 2, 3 or 4 doses. For vertigo after cerebral trauma an initial dose of 9-12 g is proposed, followed by a maintenance dose of 2.4 g a day. For vertigo of central origin in elderly people an initial dose of 4.8 g a day is proposed, followed by a maintenance dose of 1.2-2.4 g/day.

3.3. *Co-dergocrine mesilate*

Pharmacological properties: Codergocrine mesilate blocks the adrenergic alpha receptors and stimulates the dopamine and serotonine receptors. A shortening of cerebral circulation time and a vasodilatatory effect on the precapillary sphincters has been observed. This is thought to lead to an improvement in impaired metabolic cerebral function. Codergocrine mesilate may result in some improvement in the impaired mental capacity in elderly people older than 60 years of age.

After oral intake, absorption is only 25%, due to the first-pass effect. Biological availability is only 5-12%. With the FAS (Facilitated Absorption System) form of the product, biological availability improves by one-third compared to the normal form. Codergocrine mesilate works for 3-24 hours.

Indications: Positive effects have been described in case of chronic cerebral insufficiency with improvement of the following parameters: mental alertness, confusion, memory, emotional balance, depression, anxiety, vertigo, somnolence.

Contra-indications: acute or chronic psychosis.

Side-effects: nausea, congestion of the nasal mucosa, orthostatic hypotension.

Dosage: 3 times 1.5 mg a day or one dose of 4.5 mg FAS a day before breakfast.

Table 2
Drugs for motion sickness

generic name	dosage
scopolamine	oral 0.3-0.6 mg transdermal 1.5 mg
dimenhydrinate	before the trip 50-100 mg max. 3x50-100 mg
promethazine	before the trip 25 mg max 2x25 mg
cinnarizine	before the trip 25-50 mg max 25-50 mg

4. Motion sickness

Scopolamine is the most effective monosubstance against motion sickness, but is no longer available due to the side-effects even after transdermal application: drowsiness, blurring of vision, dry mouth. The oral dose for adults is 0.3-0.6 mg with a maximum effect after 30-60 minutes lasting for 4 hours. After local application (patch) the maximum effect is reached after 6-8 hours, lasting for 72 hours.[4]

The dosage of the other drugs for motion sickness is given in Table 2.

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The surgical treatment of vertigo

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Key-words. Surgery; vertigo; labyrinthectomy; neurotomy; fistula

Abstract. *The surgical treatment of vertigo.* We present surgical treatments for vertigo targeting symptoms and aetiology.
The choice of treatment should be guided by the symptoms, the supposed aetiological factors and the age of the subject. Surgery is rarely indicated in the majority of the patients with vertigo.
Surgical solutions should be proposed for cases of chronic incapacitating vertigo that are refractory to medical or physical treatment.
The surgical procedures possible today target aetiological factors or attempt to cancel information coming from the vestibular organ. We will refer to the latter as "symptomatic treatment".

A. Symptomatic surgical treatment

These treatments destroy all or part of the labyrinth, or deconnect it from the vestibular centres.

1. *Labyrinthectomy*

a) Total labyrinthectomy

This procedure may be either chemical or surgical. The intratympanic application of Gentamycin is the gold standard for the treatment of intractable chronic vertigo of the kind found in Ménière's disease.[1-4] Ideally, the aminoglycosides are administered in the round window niche through a paracentesis, a transtympanic tube or sometimes through a pump (not available now). The easiest solution today is to administer it using the Spongostan of a Silverstein tube (Micromedics Inc. Minnesota 5510 USA). This treatment results in good control of the vertigo (>80%) but it may adversely affect hearing. Surgical labyrinthectomy should be avoided. Otologists should aim to preserve cochlear morphology so that cochlear implantation remains possible at a later stage. The destruction of the diseased end organ will eliminate vertigo symptoms but also destroys hearing. This procedure should be reserved for ears with significant hearing loss in old patients.

b) Localised labyrinthectomy

The plugging of a semi-circular canal is an effective treatment with >90% success in vertigo control in cases of intractable benign paroxysmal positional vertigo (BPPV).[5-7] It usually involves the posterior canal and sometimes the horizontal. Incomplete occlusion of the canal may result in recurrence of BPPV and in a catiogenic fistula.[8]

2. *Vestibular neurotomies and neurectomies*

a) Singular neurectomy

This approach is proposed for chronic disabling refractory BPPV. The neurectomy is carried out in the ampullary recess of the posterior semi-circular canal. This technique provides complete relief from BPPV in 96% of subjects.[9] Sensorineural hearing loss is a complication in 3-4%. A positive fistula response may be present for a few months in some patients.

b) Vestibular neurotomy

A vestibular neurotomy in the pontocerebellar angle allows for the deconnection of the vestibular end organ from the vestibular nuclei and the cerebellum. It usually results in a central vestibular compensatory reorganisation, allowing for the recovery of functional equilibrium. This compensation can happen despite the persistent evolutive disease in the ear or the nerve. It is less efficient in the elderly and so this will not be the first-choice surgical option for them.

A vestibular neurotomy results in improvements or the resolution of vertigo in >90%.[10-12] The retrosigmoid approach is simpler, safer and more reliable than the middle or retrolabyrinthine

approaches.[11] The retrosigmoid approach has been used to associate neurotomy and vascular decompression.[13] A recurrence of vertigo after surgery can be observed. This may be due to an incomplete neurotomy or, more frequently, to the passage of vestibular neural fibres into the preserved cochlear nerve.[14]

B. Aetiological surgical treatments

The aim of these treatments is to correct the supposed aetiological factors of vertigo.

1. *Treatments acting on the pressure in the ear*

Those treatments were developed historically to cure or to improve an endolymphatic hydrops (EH), which was presumed to explain the symptoms. The first decompressive surgical treatments consisted of sacculotomy or cochleosacculotomy. They were intended to reduce EH by drainage and therefore improve the symptoms.

They were found to be effective for vertigo in 80%.[15,16] However, the risk of associated deterioration in hearing was higher: 20%.[16]

Decompressive surgery procedures involving the endolymphatic sac continue to be used by many teams.[17-26]

In fact, all these decompressing sac surgeries have been found to be unspecific.[16,19,21] They have the same positive effect on vertigo improvement as a simple mastoidectomy[19] on transtympanic ventilation tube in humans.[15,16] In animal models of EH, the cochleo-sacculotomy was not found to have any effect on the induced EH.[15]

A severe permanent hearing loss may occur in 20% of patients after sacculotomy and cochleosacculotomy.[16]

A transtympanic tube seems preferable to sac surgery in Ménière's disease.

This tube makes it possible to apply low-intensity alternating pressure. The Meniett device seems to be a promising and efficacious treatment for Ménière's subjects with vertigo uncontrolled by other treatments.[27]

2. *Treatment of fistula*

Labyrinthic fistulae may be primitive or secondary to an inflammatory process in the middle ear (cholesteatoma, granulomatosis); to surgery (cholesteatoma, cochlear implantation otosclerosis); to traumatisms; to malformations (Mondini). In the case of symptomatic fistulae, there should be a surgical exploration of the middle ear and the fistula should be packed with periosteum muscle or other tissues.[28-30]

The superior semicircular canal dehiscence is a special type of fistula.[31-33] It must be treated using a combined otological and neurosurgical procedure through the middle cranial fossa.

3. *Other*

Some cases of Tullio's phenomenon are due to abnormal mobility of the stapes or other ossicles. The hypermobility may be stabilised by placing cartilage chips beside the crurae of the stapes.[34] Abnormal scar tissue around the ossicles may be resected during a surgical exploration of the middle ear.

Conclusion

Rapid surgical treatment is indicated when a aetiological factor may be safely corrected. In other cases, the surgical option should be reserved for incapacitating vertigo that does not respond to medical or physical treatment. The choice of treatment must be guided by the symptoms, experience and the age of the subjects.

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