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General vestibular testing

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Abstract

A dysfunction of the vestibular system is commonly characterized by a combination of phenomena involving perceptual, ocular motor, postural, and autonomic manifestations: vertigo/dizziness, nystagmus, ataxia, and nausea. These 4 manifestations correlate with different aspects of vestibular function and emanate from different sites within the central nervous system. The diagnosis of vestibular syndromes always requires interdisciplinary thinking. A detailed history allows early differentiation into 9 categories that serve as a practical guide for differential diagnosis: (1) dizziness and lightheadedness; (2) single or recurrent attacks of vertigo; (3) sustained vertigo; (4) positional/positioning vertigo; (5) oscillopsia; (6) vertigo associated with auditory dysfunction; (7) vertigo associated with brainstem or cerebellar symptoms; (8) vertigo associated with headache; and (9) dizziness or to-and-fro vertigo with postural imbalance. A careful and systematic neuro-ophthalmological and neuro-otological examination is also mandatory, especially to differentiate between central and peripheral vestibular disorders. Important signs are nystagmus, ocular tilt reaction, other central or peripheral ocular motor dysfunctions, or a unilateral or bilateral peripheral vestibular deficit. This deficit can be easily detected by the head-impulse test, the most relevant bedside test for the vestibulo-ocular reflex. Laboratory examinations are used to measure eye movements, to test semicircular canal, otolith, and spatial perceptional function and to determine postural control. It must, however, be kept in mind that all signs and ocular motor and vestibular findings have to be interpreted within the context of the patient's history and a complete neurological examination.

Keywords: Vestibular function; Vestibular disorders; Vertigo; Dizziness; Vestibulo-ocular reflex; Neuro-otological examination; Neuro-ophthalmological examination; Electronystagmography; Posturography

1. Introduction

Vertigo and dizziness are among the most frequent presenting symptoms, not only in neurology. According to a survey of over 30,000 persons, the prevalence of vertigo and dizziness over all ages lies around 17%; it rises to 39% in those over 80 (Davis and Moorjani, 2003). Vertigo and dizziness are not unique disease entities. Sometimes vertigo is attributed to vestibular disorders, while dizziness is not (Neuhauser and Lempert, 2004). There is no general agreement, and visual stimuli can cause vertigo (e.g. height vertigo or optokinetic vection) just as otolith disorders can cause dizziness. Furthermore, central vestibular disorders such as lateropulsion in Wallenberg's syndrome may occur without subjective vertigo or dizziness (Dieterich and Brandt, 1992). Vertigo and dizziness are considered either an unpleasant disturbance of spatial orientation or the illusory perception of a movement of the body (spinning, wobbling, or tilting) and/or of the surroundings. Both terms refer to a number of multisensory and sensorimotor syndromes of various etiologies and pathogeneses (Baloh and Halmagyi, 1996; Brandt, 1999; Brandt et al., 2004; Bronstein et al., 2004).

A dysfunction of the vestibular system is commonly characterized by a combination of phenomena involving perceptual, ocular motor, postural, and autonomic manifestations: vertigo/dizziness, nystagmus, ataxia, and nausea (Fig. 1, Brandt and Daroff, 1980). These 4 manifestations correlate with different aspects of vestibular function and emanate from different sites within the central nervous system. Vertigo/dizziness itself results from a disturbance of

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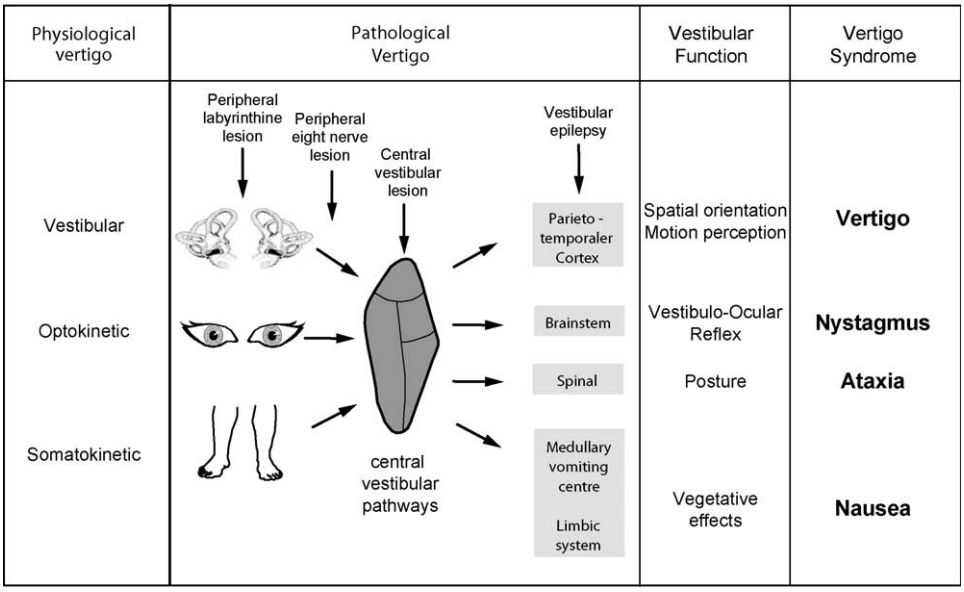

Fig. 1. Physiological vertigo (motion stimulation) and pathological vertigo (induced by lesion or stimuli) are characterized by similar signs and symptoms that derive from the functions of the multisensory vestibular system (Brandt and Daroff, 1980).

cortical spatial orientation. Nystagmus is secondary to a direction-specific imbalance of the VOR, which activates brainstem neuronal circuitry. Vestibular ataxia and postural imbalance are caused by inappropriate or abnormal activation of monosynaptic and polysynaptic vestibular–spinal pathways. Finally, the unpleasant autonomic responses of nausea, vomiting, and anxiety traverse ascending and descending vestibulo-autonomic pathways and activate the medullary vomiting center.

This review on general vestibular testing is organized in 4 parts: first, background concepts about the malfunctioning of the vestibular system; second, approaching the dizzy patient; third, different clinical neuro-ophthalmological and neuro-otological bedside tests, which are clearly illustrated in the figures; and fourth, the laboratory examinations, e.g. the magnetic search coil technique, electronystagmography, video-oculography, vestibular-evoked myogenic potentials, and posturography.

2. Background concepts

2.1. The vestibulo-ocular reflex (VOR) and the classification of central vestibular disorders

The most important anatomical structure of the vestibular system in the brainstem is the VOR. The VOR has 3 major planes of action:

- horizontal head rotation about the vertical *Z*-axis (yaw).
- head extension or flexion about the horizontal *Y*-axis (pitch).
- lateral head tilt about the horizontal *X*-axis (roll).

These 3 planes represent the three-dimensional space in which the vestibular and ocular motor systems responsible for spatial orientation, perception of self-movement, stabilization of gaze, and postural control operate. The neuronal circuitry of the horizontal and vertical semicircular canals as well as the otoliths is based on a sensory convergence that takes place within the VOR. The VOR roughly connects a set of extraocular eye muscles that are aligned with their primary direction of pull with the respective spatial planes of the horizontal, anterior, and posterior canals. The canals of both labyrinths form functional pairs in the horizontal and vertical working planes. In other words, the canals are excited/inhibited in pairs: the horizontal right and left pair, and the vertical anterior of one side along with the posterior canal of the opposite side. The vertical planes of 'pitch' and 'roll' are a result of the wiring connecting the two vertical canals that are diagonal to the sagittal plane in the head. The pair of canals function as a gauge of rotatory acceleration and react to the rotational movements of the head in the corresponding plane. The otoliths function as a gauge of gravity and linear acceleration.

There is evidence that these 3 major planes of action of the VOR allow a useful clinical classification of central vestibular syndromes (Brandt, 1999). The plane-specific vestibular syndromes are determined by ocular motor, postural, and perceptual signs.

Yaw plane signs are horizontal nystagmus, past-pointing, rotational and lateral body falls, horizontal deviation of perceived straight-ahead.

Roll plane signs are torsional nystagmus, skew deviation, ocular torsion, tilts of the head, body, and perceived vertical (ocular-tilt reaction); see, for example, Fig. 2.

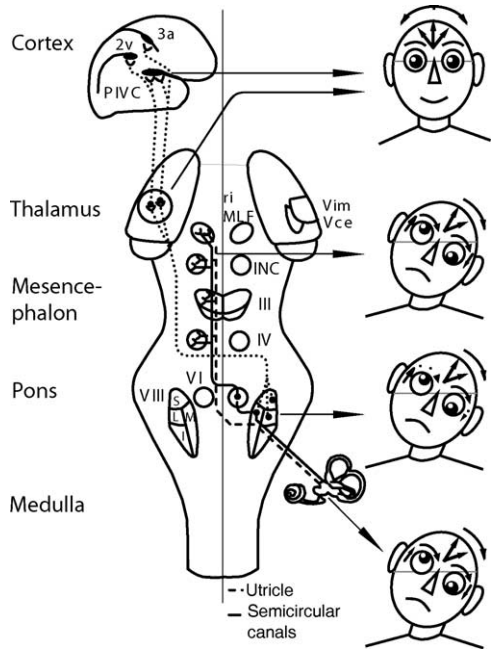

Fig. 2. *Vestibular syndromes in the roll plane:* Graviceptive pathways from otoliths and vertical semicircular canals mediating vestibular function in the roll plane. The projections from the otoliths and the vertical semicircular canals to the ocular motor nuclei (trochlear nucleus IV, oculomotor nucleus III, abducens nucleus VI), the supranuclear centers of the interstitial nucleus of Cajal (INC), and the rostral interstitial nucleus of the medial longitudinal fascicle (riMLF) are shown. They subserve VOR in 3 planes. The VOR is part of a more complex vestibular reaction which also involves vestibular–spinal connections via the medial and lateral vestibular–spinal tracts for head and body posture control. Furthermore, connections to the assumed vestibular cortex (areas 2v and 3a and the parieto-insular vestibular cortex, PIVC) via the vestibular nuclei of the thalamus (VIM, Vce) are depicted. 'Graviceptive' vestibular pathways for the roll plane cross at the pontine level OTR (skew torsion, head tilt, and tilt of perceived vertical, SVV), which is depicted schematically on the right in relation to the level of the lesion: ipsiversive OTR with peripheral and pontomedullary lesions; contraversive OTR with pontomesencephalic lesions. In vestibular thalamic lesions, the tilts of SVV may be contraversive or ipsiversive; in vestibular cortex lesions they are most often contraversive. OTR is not induced by supratentorial lesions above the level of the INC (From Brandt, 1999).

Pitch plane signs are upbeat or downbeat nystagmus, forward or backward tilts and falls, vertical deviation of the perceived straight-ahead.

2.2. Basic forms of vestibular dysfunction

Pathophysiologically, there are 3 basic forms of vestibular dysfunction, each with its typical symptoms and clinical signs:

- *Bilateral (peripheral) loss of vestibular function*. The main symptoms are oscillopsia during head movements (failure of the VOR), instability of gait and posture, which increases in darkness or on uneven ground (reduced or absent visual or somatosensory substitution of vestibular loss), and only recently detected deficits in spatial memory (lack of vestibular input for navigation and spatial memory; Schautzer et al., 2003; Smith, 1997).
- *Acute/subacute unilateral failure of vestibular function* (of the labyrinth, vestibular nerve, vestibular nuclei, or central pathways), which causes a vestibular tonus imbalance. The main symptoms are rotatory vertigo or apparent body tilt (for a few days or weeks), nystagmus, oscillopsia, nausea, and the tendency to fall in a direction opposite to that of vertigo.
- *Paroxysmal stimulation of the vestibular system* of the labyrinth (e.g. benign paroxysmal positioning vertigo), the vestibular nerve (e.g. vestibular paroxysmia due to vascular cross-compression), or the brainstem (e.g. paroxysmal ataxia in MS). The main symptoms and signs are short attacks of vertigo, dizziness, oscillopsia, nystagmus, and postural imbalance.

Clinically, two major relevant areas of vestibular dysfunction should be differentiated anatomically: peripheral vestibular disorders originating from the labyrinth and/or vestibular nerve and central vestibular disorders. Central vestibular forms of vertigo arise from lesions at the neuronal connections between the vestibular nuclei and the vestibular cerebellum as well as those between the vestibular nuclei, the vestibular and ocular motor structures of the brainstem, cerebellum, thalamus, and vestibular cortex. On the one hand, these include clearly defined clinical syndromes of various etiologies, for example, upbeat or downbeat nystagmus (the quick phase of nystagmus beats upward or downward). The occurrence of these typical ocular motor findings in only the central vestibular brainstem or in cerebellar disorders allows a definite topical attribution. On the other hand, central vestibular vertigo can also be part of a more complex infratentorial clinical syndrome. In such cases other signs and symptoms, such as supranuclear or nuclear ocular motor disorders and/or other neurological brainstem signs (e.g. Wallenberg's syndrome), can also be observed. Vertigo/-dizziness can manifest as attacks lasting for seconds or minutes (vestibular migraine), for hours up to days (brainstem infarction), or as a permanent syndrome (downbeat nystagmus in cases of Arnold–Chiari malformation).

2.3. Semicircular canal or otolith dysfunction

There are several possible reasons why most vestibular syndromes involve semicircular canal and otolith function. The different receptors for perception of angular and linear accelerations are housed in a common labyrinth. Their peripheral (VIIIth nerve) and central (e.g. medial longitudinal fascicle) pathways take the same course. Finally, there is a convergence of otolith and semicircular canal input at all central vestibular levels, from the vestibular nuclei to the vestibular cortex.

Thus, most vestibular syndromes are mixed as regards otolithic and canal function. A peripheral prototype of such

a mixture is vestibular neuritis. It is mostly caused by failure of the superior division of the vestibular nerve that subserves the horizontal and the anterior semicircular canals and the maculae of the utricle and the anterosuperior part of the saccule (Balo, 2003; Büchele and Brandt, 1988; Fetter and Dichgans, 1996; Murofushi et al. 2003). A central prototype is Wallenberg's syndrome, which involves the medial and superior vestibular nuclei, where otolith and canal input converge. This typically causes ocular and body lateropulsion, and spontaneous torsional nystagmus (Dieterich and Brandt, 1992).

It is, however, possible to selectively stimulate single canals by caloric irrigation of the external auditory canal or by the head-thrust test devised by Halmagyi and Curthoys (Cremer et al., 1998; Halmagyi and Curthoys, 1988). The prototype of a semicircular canal disease is benign paroxysmal positioning vertigo of the posterior or horizontal canal. Typical signs and symptoms of semicircular canal dysfunction are rotational vertigo and deviation of perceived straight-ahead, spontaneous vestibular nystagmus with oscillopsia, postural imbalance in the Romberg test and past-pointing, and nausea and vomiting, if severe. The three-dimensional spatial direction of nystagmus and vertigo depends on the spatial plane of the affected semicircular canal and on whether the dysfunction is caused by ampullofugal or ampullopetal stimulation or a unilateral loss of afferent information. Malfunction of a single or more than one semicircular canal can be detected by three-dimensional analysis of spontaneous nystagmus (Böhmer et al., 1997; Straumann and Zee, 1995), the head-thrust test with individual semicircular canal plane head impulses (Cremer et al., 1998), or perception of rotation (von Brevern et al., 1997). Central vestibular syndromes may take precedence over semicircular canal or otolith types. In other words, 'dynamic', rotatory vertigo and nystagmus represent (angular) canal function, whereas 'static' ocular-tilt reaction, body lateropulsion, or tilts of the perceived vertical represent (linear) otolithic function.

Although the pathophysiology of otolith dysfunction is poorly understood, a disorder of otolithic function at a peripheral or central level should be suspected if a patient describes symptoms of falls, sensations of linear motion or tilt, or else shows signs of specific derangements of ocular motor and postural orienting and balancing responses (Gresty et al., 1992). A significant number of patients presenting to neurologists have signs and symptoms that suggest disorders of otolithic function. Nevertheless, diseases of the otoliths are poorly represented in our diagnostic repertoire. Of these, posttraumatic otolith vertigo (Brandt and Daroff, 1980) may be the most significant; the rare otolith Tullio phenomenon may be the best studied (Dieterich et al., 1989; Fries et al., 1993). Other examples are vestibular drop attacks (Tumarkin's otolithic crisis; Baloh et al., 1990) and a number of central vestibular syndromes that indicate tonus imbalance of graviceptive circuits (skew deviation, ocular tilt reaction, lateropulsion, or room-tilt illusion; Brandt, 1997; Brandt and Dieterich, 1994a; Brodsky, 2003; Tiliket et al., 1996), some of which manifest without the sensation of dizziness or vertigo.

3. Approaching the patient

About 75% of all patients presenting with vertigo or dizziness in a neurological dizziness unit will have one of the 8 most common syndromes listed in Table 1. A clinician not familiar with dizzy patients can best deepen his knowledge by acquainting himself with these 8 most frequently met but still challenging conditions. The diagnosis of central vestibular disorders, however, comprises a variety of syndromes extending from the brainstem to the vestibular cortex. Although most clinicians welcome the effort being made to develop computer interview systems for use with neuro-otological patients (O'Connor et al., 1989) and expert systems as diagnostic aids in otoneurology (Auramo et al., 1993; Mira et al., 1990), the actual application of these systems in a clinical setting is still quite limited.

Vertigo and dizziness are vexing symptoms. They are sometimes difficult to assess because of their purely

Table 1
Relative frequency of different syndromes diagnosed in a special neurological dizziness unit ($n = 4790$ patients in 1989–2003)

Diagnosis	Frequency (%)
Benign paroxysmal positioning vertigo	18.3 (Brandt and Steddin, 1993)
Phobic postural vertigo (PPV)	15.9 (Brandt, 1996)
Central vestibular disorders	13.5 (Brandt and Dieterich, 1994a)
Vestibular migraine	9.6 (Dieterich and Brandt, 1999; Neuhauser et al., 2001; 2004)
Vestibular neuritis	7.9 (Baloh, 2003)
Menière's disease	7.8 (James and Thorp, 2001)
Bilateral vestibulopathy	3.6 (Rinne et al., 1995)
Psychogenic vertigo (without PPV)	3.6
Vestibular paroxysmia	2.9 (Brandt and Dieterich, 1994b; Jannetta et al., 1984)
Perilymph fistula or superior canal dehiscence syndrome	0.4 (Minor et al., 1998)
Various other disorders	12.3
Unknown etiology	4.2

subjective character and the variety of sensations patients report. The sensation of spinning or rotatory vertigo is much more specific; if it persists, it undoubtedly indicates acute pathology of the labyrinth, the vestibular nerve, or the caudal brainstem, which contains the vestibular nuclei. The diagnosis and management of vertigo syndromes always require interdisciplinary thinking combined with a careful taking of the patient's history. The history is still much more important than recording eye movements and postural sway or using brain imaging techniques.

3.1. Diagnostic criteria

The important diagnostic criteria of vestibular syndromes manifesting with vertigo or dizziness are as follows:

- *Type of vertigo*: Rotatory vertigo as experienced when riding a merry-go-round (e.g. vestibular neuritis) or postural imbalance as experienced during boat trips (e.g. bilateral vestibulopathy) or dizziness/lightheadedness (e.g. intoxication).
- *Duration of vertigo*: Attacks of vertigo lasting for seconds to minutes (e.g. vestibular paroxysmia), over hours (e.g. Menière's disease, vestibular migraine), sustained vertigo for days to a few weeks (e.g. vestibular neuritis), attacks of postural instability from minutes to hours (e.g. transient ischemic attacks of the brainstem or cerebellar structures).
- *Trigger/exacerbation of vertigo*: No trigger (e.g. vestibular neuritis), walking (e.g. bilateral vestibulopathy), head turning (e.g. vestibular paroxysmia), head positioning (e.g. benign paroxysmal positioning vertigo), coughing, pressing, loud sounds of a certain frequency (perilymph fistula or superior canal dehiscence syndrome), or certain social situations (phobic postural vertigo).
- *Vertigo associated with* auditory dysfunction, non-vestibular neurological signs and symptoms, or headache.

3.2. Diagnostic categories

A thorough patient history allows the early differentiation of vertigo and disequilibrium disorders into 9 categories that serve as a practical guide for the differential diagnosis.

3.2.1. Dizziness and lightheadedness (Table 2)

Most of us have experienced presyncopal dizziness at some time when rapidly standing up from a relaxed supine or seated position. Such an experience best exemplifies this category (Baloh and Halmagyi, 1996), which includes orthostatic hypotension and cardiac arrhythmias as well as hyperventilation syndrome and panic attacks.

Table 2
Dizziness and lightheadedness

Presyncopal dizziness
Orthostatic dysregulation
Vasovagal attacks
Neuro-cardiogenic (pre) syncope
Cardiac arrhythmia and other heart diseases
Psychiatric illnesses
Hyperventilation syndrome
Panic attacks
Agoraphobia
Acrophobia
Phobic postural vertigo
Metabolic disorders
Hypoglycemia
Electrolyte disorders (hypercalcemia, hyponatremia)
Intoxication
Alcohol
Medication
Toxic substances

3.2.2. Single or recurrent attacks of (rotatory) vertigo (Table 3)

Recurrent vertigo attacks in children which last several seconds or minutes are most likely due to benign paroxysmal vertigo of childhood, a migraine equivalent. In adults short attacks of rotatory vertigo may occur in Menière's disease, vestibular migraine, or transient vertebrobasilar ischemia.

3.2.3. Sustained rotatory vertigo (Table 4)

Sustained vertigo occurs either with acute unilateral peripheral loss of vestibular function or with pontomedullary brainstem lesion near the vestibular nuclei. Vestibular neuritis is the most frequent cause and its diagnostic

Table 3
Episodic vertigo (diseases with recurrent attacks of vertigo)

Labyrinth/vestibulo-cochlear nerve
• Menière's disease
• Vestibular paroxysmia
• Perilymph fistula
• Superior canal dehiscence syndrome (induced by coughing, pressing, or loud sounds of a specific frequency, i.e. a Tullio phenomenon)
• Benign paroxysmal positioning vertigo (only during changes of head position relative to gravity)
• Cogan's syndrome
• Cysts or tumors of the cerebellopontine angle
Central vestibular system
• Transient vertebrobasilar ischemia
• 'Rotational vertebral artery occlusion syndrome'
• Vestibular epilepsy
• 'Room-tilt illusion'
• Paroxysmal ataxia/dysarthrophonia (multiple sclerosis)
• Episodic ataxia types 1 and 2
• Paroxysmal 'ocular tilt reaction'
Peripheral and/or central vestibular system
• Basilar/vestibular migraine
• Benign paroxysmal vertigo of childhood
• Vertebrobasilar transient ischemia (e.g. AICA)

Table 4
Sustained vertigo or dizziness

Infections
Viral
Vestibular neuritis
Herpes zoster oticus
Viral neuro-labyrinthitis
Bacterial
Bacterial meningitis
Tuberculous labyrinthitis
Syphilitic labyrinthitis
Rarely
Chlamydial labyrinthitis
Lyme borreliosis
Autoimmunological inner ear diseases
Cogan's syndrome
Neurosarcoidosis
Behçet's disease
Cerebral vasculitis
Systemic lupus erythematosus
Polychondritis
Rheumatoid arthritis
Polyarteritis nodosa
Wegener's granulomatosis
Giant cell arteritis
Primary antiphospholipid syndrome
Tumorous
Vestibular schwannoma
Meningeoma
Epidermoid cyst
Glomus tumor
Metastasis
Meningeosis carcinomatosa
Cholesteatoma
Vascular
Labyrinthine infarction (AICA)
Pontomedullar brainstem infarction
Vertebrobasilar ectasia
Hyperviscosity syndrome
Traumatic
Temporal bone fracture (transverse > longitudinal fracture)
Labyrinthine concussion
Posttraumatic otolith vertigo
Perilymph fistula
Superior canal dehiscence syndrome
Brainstem concussion
Iatrogenic
Temporal bone surgery
Systemic or transtympanic administration of aminoglycosides
Other ototoxic substances

hallmark is unilateral hyporesponsiveness to caloric irrigation. Differential diagnosis of the pathologies of sustained central vertigo involves all acute processes of the intraaxial infratentorial structures (involving the root entry zone of the VIIIth nerve or the vestibular nuclei) such as multiple sclerosis, tumors, or brainstem infarctions.

3.2.4. Positional/positioning vertigo (Table 5)

In the majority of patients presenting with this condition, positioning vertigo is due to canalolithiasis in the posterior semicircular canal. All central forms of positional vertigo involve the region around the vestibular nuclei and a neuronal loop to the cerebellar vermis (Büttner et al., 1999a,b).

Table 5
Positional/positioning vertigo and/or nystagmus

Elicited by changes of head position relative to gravity
• Benign paroxysmal positioning vertigo
• Positional alcohol vertigo/nystagmus
• Positional nystagmus with macroglobulinemia
• Positional 'heavy water' nystagmus
• Central positional nystagmus
• Positional down-beating nystagmus
• Central positioning vomiting
Elicited by lateral head rotation
• Vestibular paroxysmia
• Rotational vertebral artery occlusion syndrome
• Compression of the VIIIth nerve due to cerebellopontine angle mass
• Carotid sinus syndrome

3.2.5. Oscillopsia (apparent motion of the visual scene) (Table 6)

Patients with involuntary ocular oscillations (acquired pendular nystagmus, downbeat or upbeat nystagmus) not only report a worsening of visual acuity but also apparent motion of the visual scene (Bronstein, 2004). Patients with extraocular muscle paresis or defects of the VOR are often unable to recognize faces or to read while walking; they may also report oscillopsia. Either the deficiency of compensatory eye movements (due to an inappropriate VOR) or the deficiency of visual fixation (due to ocular

Table 6
Oscillopsia (illusionary movements of the surroundings)

Without head movements
• Spontaneous vestibular nystagmus (e.g. in vestibular neuritis)
• Congenital nystagmus (depending on direction of gaze)
• Downbeat nystagmus
• Upbeat nystagmus
• Acquired pendular nystagmus
• Periodic alternating nystagmus
• Opsoclonus
• Ocular flutter
• Vestibular paroxysmia
• Myokymia of the superior oblique muscle (monocular)
• Paroxysmal 'ocular-tilt reaction'
• Spasmus nutans (infants)
• Voluntary nystagmus
During head movements
• Bilateral vestibulopathy
• Disorders of the ocular motor system (peripheral or central)
• Vestibular paroxysmia
• Benign paroxysmal positioning vertigo
• Central positional/positioning vertigo
• Vestibulo-cerebellar ataxia
• Perilymph fistula
• Superior canal dehiscence syndrome
• Posttraumatic otolith vertigo
• Rotational vertebral artery occlusion syndrome
• Intoxication (e.g. anticonvulsants, alcohol)

oscillation) causes undesired retinal image motion with disturbing oscillopsia and sometimes unsteadiness.

3.2.6. Vertigo-associated with auditory dysfunction (Table 7)

The presence of dizziness, vertigo, or disequilibrium combined with sensorineural hearing loss or tinnitus narrows down the differential diagnosis to certain peripheral vestibular disorders. The rare central vestibular disorders that manifest with audiovestibular symptoms are vestibular epilepsy or caudal brainstem disorders, such as occur in multiple sclerosis. Audiovestibular dysfunction associated with interstitial keratitis indicates infectious or autoimmune disease. Congenital unilateral and bilateral vestibular disorders may be combined with sensorineural hearing loss.

3.2.7. Vertigo associated with brainstem and cerebellar symptoms (Table 8)

Clinical studies of the differential effects of central vestibular pathway lesions have increasingly shown that vestibular syndromes are accurate indicators for a topographic diagnosis. Vestibular pathways run from the VIIIth nerve and the vestibular nuclei through ascending fibers, such as the ipsilateral or contralateral medial longitudinal fascicle, brachium conjunctivum, or the ventral tegmental tract to the ocular motor nuclei, the supranuclear integration centers in the rostral midbrain, and the vestibular thalamic subnuclei. From there they reach several cortex areas through the thalamic projections. Another relevant ascending projection reaches the cortex from the vestibular nuclei via the vestibular cerebellum structures, in particular the fastigial nucleus. In the majority of cases, central vestibular vertigo/dizziness syndromes are caused by dysfunction or a deficit of sensory input induced by a lesion. In a small proportion of cases they are due to pathological excitation of various structures, extending from the peripheral vestibular organ to the vestibular cortex. Since peripheral vestibular disorders are always characterized by a combination of perceptual, ocular motor, and postural signs and symptoms, central vestibular disorders may manifest as ‘a complete syndrome’ or as only single components. The ocular motor aspects, for example, predominate in the syndrome of upbeat or downbeat nystagmus. Lateral falls may occur without vertigo in vestibular thalamic lesions (thalamic astasia) or as lateropulsion in Wallenberg’s syndrome. Most central vestibular syndromes have a specific locus but not a specific etiology. The etiology may, for example, be vascular, autoimmunological (e.g. in MS), inflammatory, neoplastic, toxic, or traumatic.

Table 7
Combination of vestibular and audiological symptoms

Menière’s disease
Perilymph fistula or superior canal dehiscence syndrome
Vestibular paroxysmia
Cerebellopontine angle tumor
Cogan’s syndrome or other inner ear autoimmune diseases
Ear/head trauma
Pontomedullary brainstem infarct
Pontomedullary MS plaque
Labyrinthine infarct (AICA, labyrinthine artery)
Hyperviscosity syndrome
Neurolabyrinthitis
Zoster oticus
Cholesteatoma
Inner ear malformation
Vestibular epilepsy

Table 8
Vertigo with additional brainstem/cerebellar symptoms

Basilar/vestibular migraine
Intoxication
Craniocervical malformations (e.g. Arnold–Chiari malformation)
Lacunar or territorial infarcts
Hemorrhages (e.g. cavernoma)
Inflammation (e.g. MS plaque)
Brainstem encephalitis
Head trauma
Tumors of the cerebellopontine angle, brainstem, or cerebellum
Episodic ataxia type 2
Creutzfeldt–Jakob disease

3.2.8. Vertigo associated with headache (Table 9)

Various peripheral and central vestibular disorders are typically associated with headache, such as basilar or vestibular migraine. One-third of patients with vestibular migraine, however, do not complain of headache associated with vestibular aura deficits (Dieterich and Brandt, 1999; Neuhauser et al., 2001).

3.2.9. Dizziness or to-and-fro vertigo with postural imbalance

Dizziness and to-and-fro vertigo with postural imbalance are non-specific but frequently described symptoms. Differential diagnosis on the basis of such symptoms is very difficult, because central and peripheral vestibular disorders but also non-vestibular syndromes such as visual vertigo, presyncopal faintness, or somatoform phobic postural vertigo are all possible diagnoses in this category.

Table 9
Vertigo with headache

Migraine without aura
Basilar/vestibular migraine
Brainstem/cerebellar ischemia
Vertebrobasilar dissection
Infratentorial hemorrhage
Inner/middle ear infection
Head trauma (especially transverse temporal bone fracture)
Infratentorial tumor
Zoster oticus

4. Clinical neuro-ophthalmological and neuro-otological examinations

The major aim of the neuro-ophthalmological, neuro-otological, and neuro-orthoptic examinations is to differentiate between peripheral and central vestibular forms of dysfunction. Since the disorders underlying vertigo and dizziness are often combined with disturbances of the ocular motor system due to anatomical proximity, ocular motor examination techniques (how to) are also described and an interpretation of the typical findings and their localizing impact or topographic determination of the lesion is given. This non-vestibular system must always be tested in patients suffering from vertigo and disequilibrium. Then the simple and reliable bedside test of the vestibulo-ocular reflex, the most important anatomical and physiological structure of the vestibular system, is presented (Halmagyi–Curthoys head-impulse test) (Halmagyi and Curthoys, 1988; Halmagyi et al., 1990) together with a recently developed test of otolith function ('the head-heave test' (Ramat et al., 2001)). Finally, positioning tests for posterior and horizontal canal benign paroxysmal positioning vertigo (BPPV), the examination with Politzer's balloon for perilymph fistula or superior canal dehiscence syndrome, and the tests of stance and gait are demonstrated. The techniques of the different tests and their typical questions are summarized in Table 10.

4.1. Eye position and nystagmus

Clinical examination of patients with suspected vestibular disorders should begin with the *examination of the eyes in 9 different positions* (i.e. looking straight ahead, to the right, left, up, down as well as diagonally right up, right down, left up, and left down) to determine ocular alignment (for example, a possible misalignment of the eye axes, which may be accompanied by a head tilt, Fig. 3) (Brandt and Dieterich, 1994a), fixation deficits, spontaneous or fixation nystagmus (Serra and Leigh, 2002), range of movement, and disorders of gaze-holding abilities (Büttner and Grundei, 1995). The examination can be performed with an object for fixation or a small rod-shaped flashlight. In *primary position* one should look for periodic eye movements, such as *nystagmus* (e.g. horizontal-rotatory, suppressed by fixation as in peripheral vestibular dysfunction), vertically upward (*upbeat nystagmus*) (Baloh and Yee, 1989; Fisher et al., 1983) or downward (*downbeat nystagmus syndrome*) (Baloh and Spooner, 1981; Böhmer and Straumann, 1998; Glasauer et al., 2003), or horizontal or torsional movements with only slight suppression (or increase) of intensity during fixation as in a central vestibular dysfunction. A (non-vestibular) *congenital nystagmus* (Gottlob, 1998; Maybodi, 2003) beats, as a rule, horizontally at various frequencies and amplitudes and

Table 10
Examination procedure for ocular motor and vestibular systems

Type of examination	Question
Inspection	
Head, body, and posture	Tilt or turn of head/body
Position of eyelids	Ptosis
Eye position/motility	
Position of eyes during straight-ahead gaze	Misalignment in primary position, spontaneous or fixation nystagmus
Cover test	Horizontal or vertical misalignment
Examination of eyes in 8 positions (binocular and monocular)	Determination of extent of motility, gaze-evoked nystagmus, end-position nystagmus
Gaze-holding function: after 10–40° in the horizontal or 10–20° in the vertical and back to 0°	Gaze-evoked nystagmus: horizontal and vertical, rebound nystagmus
Smooth pursuit movements: horizontal and vertical	Smooth or saccadic
Saccades: horizontal and vertical when looking around or at targets	Latency, velocity, accuracy, conjugacy
Optokinetic nystagmus (OKN): horizontal and vertical with OKN drum or tape	Inducible, direction, phase (reversal or monocularly diagonal)
Peripheral vestibular function: clinical testing of the VOR (Halmagyi–Curthoys test): rapid turning of the head and fixation of a stationary target	Unilateral or bilateral, peripheral vestibular (semicircular canal) deficit
Fixation suppression of the VOR: turn of head and fixation of a target moving at same speed	Failure of fixation suppression
Examination with Frenzel's glasses:	
Straight-ahead gaze, to the right, to the left, downward, and upward	Spontaneous nystagmus
Head-shaking test	Provocation-induced nystagmus
Positioning and positional maneuver (with Frenzel's glasses): to the right, left, head-hanging position, turning about the cephalocaudal axis	Peripheral positional or positioning nystagmus, central positional nystagmus
Posture and balance control:	
Romberg's test and simple and difficult posture and gait tests:	Instability, tendency to fall
Open-closed eyes	
With/without reclining the head	
With/without distraction (writing numbers on the skin, doing maths mentally)	Psychogenic/functional components

Fig. 3. *Measurement of head tilt*. An abnormal head posture to the right or left shoulder or a constant, abnormal tilt is especially observed in patients with (a) *paresis of the oblique eye muscles*, e.g. in superior oblique palsy, the head is turned to the non-affected side to lessen diplopia, or (b) an *ocular tilt reaction* due to a vestibular tonus imbalance of the VOR in roll. As a rule, the head is tilted to the side of the lower eye. Acute unilateral lower medullary lesions (e.g. involvement of the vestibular nuclei in Wallenberg's syndrome) or acute unilateral peripheral vestibular lesions cause an ipsiversive head tilt, whereas pontomesencephalic lesions of vestibular pathways cause a contraversive head tilt.

Fig. 4. Clinical examination with *Frenzel's glasses*. The magnifying lenses (+16 diopters) with light inside prevent visual fixation, which could suppress spontaneous nystagmus. Frenzel's glasses enable the clinician to better observe spontaneous eye movements. Examination should include spontaneous and gaze-evoked nystagmus, head-shaking nystagmus (either the examiner turns the subject's head or the patient is instructed to quickly turn his head to the right and to the left about 20–30 times; the eye movements are observed after head shaking), positioning and positional nystagmus, as well as hyperventilation-induced nystagmus. *Spontaneous nystagmus* indicates a tonus imbalance of the vestibulo-ocular reflex; if it is caused by a peripheral lesion—as in vestibular neuritis—the nystagmus is typically dampened by visual fixation. *Head-shaking nystagmus* shows a latent asymmetry of the so-called velocity storage, which can be due to peripheral and central vestibular disorders.

increases during fixation. So-called *square-wave jerks* (small saccades [0.5–5°]) that cause the eyes to oscillate around the primary position increasingly occur in progressive supranuclear palsy or certain cerebellar syndromes (Averbuch-Heller et al., 1999; Rascol et al., 1991; Shallo-Hoffmann et al., 1990). *Ocular flutter* (intermittent rapid bursts of horizontal oscillations without an intersaccadic interval) or *opsoclonus* (combined horizontal, vertical, and torsional oscillations) occur in various disorders (Helmchen et al., 2003; Wong et al., 2001) such as encephalitis, tumors of the brainstem or cerebellum, intoxication, or in paraneoplastic syndromes (Bataller et al., 2003).

The examination of the eyes with Frenzel's glasses (Fig. 4) is a sensitive method for detecting spontaneous nystagmus. This can also be achieved by examining one eye with an *ophthalmoscope* (while the other eye is covered) and simultaneously checking for movements of the optic papilla or retinal vessels (Zee, 1978) even with low, slow-phase velocities/frequencies or *square-wave jerks* (small saccades [0.5–5°] that are often observed in progressive supranuclear palsy or certain cerebellar syndromes) (Leigh and Zee, 1999). Since the retina is behind the axis of rotation of the eyeball, the direction of any observed vertical or horizontal movement is opposite to that of the nystagmus detected with this method, i.e. a downbeat nystagmus causes a rapid, upward movement of the optic papilla or retinal vessels.

After checking for possible eye movements in primary position and the misalignment of the axes of the eyes, the examiner should then establish the *range of eye movements* monocularly and binocularly in the 8 end-positions; deficits found here can indicate, e.g. extraocular muscle or nerve palsy. *Gaze-holding deficits* (Büttner and Grundei, 1995; Leigh and Zee, 1999) can also be determined by examining eccentric gaze position. Use of a small rod-shaped flashlight has the advantage that the corneal reflex images can be observed and thus ocular misalignments can be easily detected (note: it is important to observe the corneal reflex images from the direction of the illumination and to ensure that the patient attentively fixates the object of gaze.) The flashlight also allows one to determine whether the patient can fixate with one or both eyes in the end-positions. This is important for detecting a defect of gaze holding. *Gaze-evoked nystagmus* can only be clearly identified when the patient fixates with both eyes. It is most often a side effect of medication (e.g. anti-convulsants, benzodiazepines) or toxins (e.g. alcohol). *Horizontal gaze-evoked nystagmus* can indicate a structural lesion in the area of the brainstem or cerebellum (vestibular nucleus, nucleus prepositus hypoglossi, flocculus, i.e. the neural eye velocity to position integrator). *Vertical gaze-evoked nystagmus* is observed in midbrain lesions involving the interstitial nucleus of Cajal (Bhidayasiri et al., 2000; Büttner and Grundei, 1995; Leigh and Zee, 1999). A *dissociated horizontal gaze-evoked nystagmus* (greater in

the abducting than the adducting eye) in combination with an adduction deficit points to internuclear ophthalmoplegia due to a defect of the medial longitudinal fascicle (MLF), ipsilateral to the adduction deficit. *Downbeat nystagmus* usually increases in eccentric gaze position and when looking down. To examine for a so-called *rebound nystagmus* the patient should gaze at least 15 s to one side and then return the eyes to the primary position; this can cause a transient nystagmus to appear with slow phases in the direction of the previous eye position. Rebound nystagmus generally indicates cerebellar dysfunction or damage to the cerebellar pathways (Hood, 1981; Hood et al., 1973).

4.2. Smooth pursuit

The patient is asked to visually track an object moving slowly in horizontal and vertical directions (10–20°/s) while keeping his head stationary. Corrective (catch-up or back-up) saccades are looked for; they indicate a smooth pursuit gain that is too low or too high (ratio of eye movement velocity and object velocity). Many anatomical structures (visual cortex, motion sensitive areas MT, V5, frontal eye fields, dorsolateral pontine nuclei, cerebellum, vestibular and ocular motor nuclei) are involved in smooth pursuit eye movements, which keep the image of a moving object stable on the fovea (Büttner and Grundei, 1995; Gaymard and Pierrot-Deseilligny, 1999; Lisberger et al., 1987; Pierrot-Deseilligny and Gaymard, 1992). These eye movements are also influenced by alertness, various drugs, and age. Even healthy persons exhibit a slightly saccadic smooth pursuit during vertical downward gaze. For these reasons a saccadic smooth pursuit as a rule does not allow either an exact topographical or etiological classification. Marked *asymmetries* of smooth pursuit, however, indicate a structural lesion; strongly impaired smooth pursuit is observed in intoxication (anticonvulsives, benzodiazepines, or alcohol) as well as degenerative disorders involving the cerebellum or extrapyramidal system (Gaymard and Pierrot-Deseilligny, 1999; Pierrot-Deseilligny and Gaymard, 1992). *A reversal of slow smooth pursuit* eye movements during optokinetic stimulation is typical for congenital nystagmus (see above).

4.3. Saccades

First, it is necessary to observe spontaneous saccades triggered by visual or auditory stimuli. Then the patient is asked to glance back and forth between two horizontal or two vertical targets. The velocity, accuracy, and the conjugacy of the saccades should be noted. Normal individuals can immediately reach the target with a fast single movement or one small corrective saccade (Botzel et al., 1993). *Slowing of saccades*—often accompanied by *hypometric* saccades—occurs for example with intoxication (medication, especially anticonvulsives or benzodiazepines) (Thurston et al., 1984) or in neuro-degenerative disorders (Troost et al., 1974). *Slowing of horizontal* saccades is generally observed in brainstem lesions; there is often a dysfunction of the ipsilateral paramedian pontine reticular formation (PPRF) (Gaymard and Pierrot-Deseilligny, 1999). *Slowing of vertical* saccades indicates a midbrain lesion in which the rostral interstitial medial longitudinal fascicle (riMLF) is involved, not only in ischemic inflammatory diseases but also in neuro-degenerative diseases, especially progressive supranuclear palsy (Bhidayasiri et al., 2001; Burn and Lees, 2002; Kuniyoshi et al., 2002; Troost and Daroff, 1977). *Hypermetric* saccades, which can be identified by a corrective saccade back to the object, indicate lesions of the cerebellum (especially the vermis) or the cerebellar pathways. Patients with Wallenberg's syndrome make hypermetric saccades toward the side of the lesion due to a dysfunction of the inferior cerebellar peduncle; defects of the superior cerebellar peduncle, conversely, lead to contralateral hypermetric saccades (Helmchen et al., 1994; Robinson et al., 2002). A slowing of the *adducting* saccade ipsilateral to a defective MLF is pathognomonic for internuclear ophthalmoplegia (INO) (Cremer et al., 1999; Zee, 1992). *Delayed onset saccades* are mostly caused by supratentorial cortical dysfunction (Leigh and Zee, 1999).

4.4. Vestibulo-ocular reflex

One bedside test is of special clinical importance: the head-impulse test of Halmagyi and Curthoys (Halmagyi and Curthoys, 1988; Halmagyi et al., 1992); it allows the examination of the horizontal VOR. This test is closely related to the purpose and special properties of the VOR (see above).

Fig. 5 summarizes how to do this test and how to interpret the findings. The test also allows examination not only of the horizontal, but also of the vertical canals, because they can be stimulated in specific planes and sides, e.g. the left anterior semicircular canal can be stimulated by moving the head in the plane of this canal downward and observing the induced eye movements. According to our experience, the head-impulse test is very helpful; if it gives a pathological finding, it is not necessary to do an additional caloric irrigation.

Testing dynamic visual acuity (subject turns his head horizontally to the right and left with a frequency of about 1 Hz and visual acuity is determined by, e.g. the Snellen chart) is also helpful in diagnosing bilateral vestibular failure (Burgio et al., 1992). A decrease of visual acuity by at least 3 lines is pathological and indicates a bilateral deficit of the VOR.

4.5. The head-heave test for otolith testing

This is a bedside test for *utricular function* and the translational VOR (Ramat et al., 2001). The head of the subject

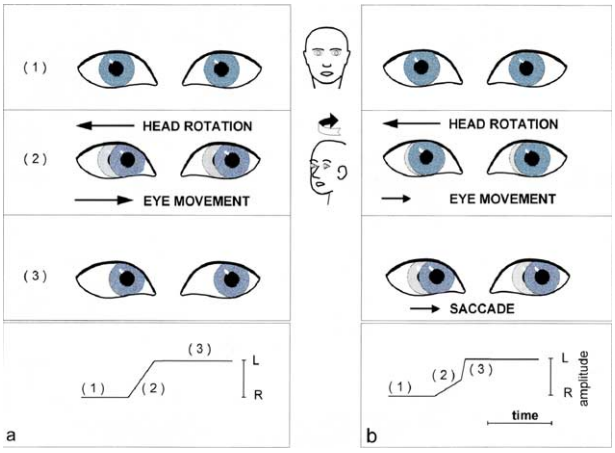

Fig. 5. Clinical examination of the *horizontal vestibulo-ocular reflex* (VOR) by the *head-impulse test* (Halmagyi and Curthoys, 1988). To test the horizontal VOR, the examiner holds the patient's head between both hands, asks him to fixate a target in front of his eyes, and rapidly and arbitrarily turns the patient's head horizontally to the left and then to the right. This rotation of the head in a healthy subject causes rapid compensatory eye movements in the opposite direction (a). In cases of unilateral labyrinthine loss the patient is not able to generate the VOR-driven fast contraversive eye movement and has to perform a corrective (catch up) saccade to re-fixate the target. Part b explains the findings in a patient with a loss of the right horizontal canal. During rapid head rotations toward the affected right ear, the eyes move with the head to the right and the patient has to perform a refixation saccade to the left; the latter can be easily detected by the examiner.

has to be moved manually in a horizontal direction to the right and left with brief but highly accelerated motions ('heaves'). The ocular response to the translation is a compensatory eye movement to keep the target stable on the retina. This eye movement response is asymmetrical in patients with a unilateral peripheral vestibular lesion. For instance, if the patient has a left-sided peripheral vestibular lesion and his head is moved toward the affected side, the examiner can easily observe a corrective saccade to the right. This indicates a deficit of the translational VOR (Ramat et al., 2001) on analogy to the 'head thrust sign' (Halmagyi and Curthoys, 1988).

4.6. Positioning/positional maneuvers

All patients should also be examined with the so-called Dix-Hallpike maneuver (Fig. 6) in order not to overlook the most common form of vertigo, benign paroxysmal positioning vertigo (BPPV) (Brandt and Steddin, 1993; Schuknecht, 1969) of the posterior as well as central positioning/positional nystagmus or vertigo (Bertholon et al., 2002; Brandt, 1990; Büttner et al., 1999a,b). In addition, the 'barbecue-spit maneuvers' should be performed to look for a BPPV of the horizontal canal (Baloh et al., 1993; McClure, 1985; Pagnini et al., 1989; Strupp et al., 1995), which is characterized by a linear horizontal nystagmus beating in most cases to the undermost ear ('geotropic'), but in some cases to the uppermost ear (Bisdorff and Debatisse, 2001).

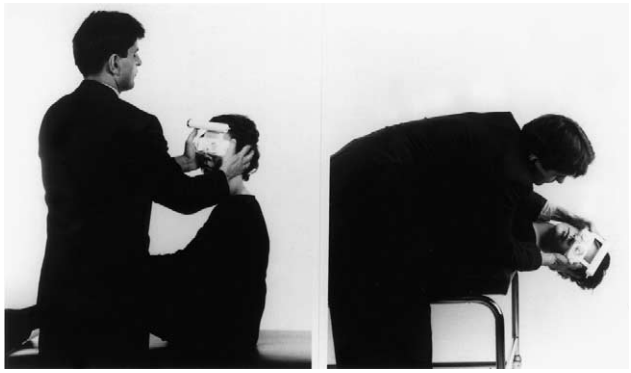

Fig. 6. The so-called *Dix-Hallpike maneuver* is performed to determine whether benign paroxysmal positioning vertigo (BPPV) is present. While the patient is sitting, his head is turned by 45° to one side, and then he is rapidly put in a supine position with head hanging over the end of the examination couch. If a BPPV of the left posterior semicircular canal, for example, is present, this maneuver will induce, with a certain latency, a crescendo–decrescendo-like nystagmus, which from the patient's viewpoint beats counterclockwise toward the left ear and to the forehead. When the patient is returned to a sitting position, the direction of nystagmus will change.

4.7. Miscellaneous

4.7.1. Visual fixation suppression of the VOR

A disorder of visual fixation suppression of the VOR (which as a rule occurs with smooth pursuit abnormalities, as these two functions are mediated by common neural pathways) (Takemori, 1983) is often observed in lesions of the cerebellum (flocculus or paraflocculus) or of the cerebellar pathways and in progressive supranuclear palsy (see above). Anticonvulsants and sedatives can also impair visual fixation of the VOR. Before testing visual fixation suppression of the VOR, it is necessary to confirm that the VOR is intact.

4.7.2. Head-shaking nystagmus

To test for head-shaking nystagmus (HSN), the examiner turns the subject's head by about $\pm 45°$ horizontally about 30 times within about 15 s or the patient does it by himself. HSN is defined as the occurrence of at least 5 beats of nystagmus immediately after the head-shaking maneuver, which should be performed with Frenzel's glasses. There is good evidence that HSN reflects a dynamic (peripheral and/or central vestibular) asymmetry of the velocity-storage mechanism (Hain and Spindler, 1993; Hain et al., 1987). In peripheral lesions, the ipsilateral dynamic VOR deficit leads to an asymmetric accumulation within the velocity storage, the discharge of which determines the direction of HSN, usually toward the unaffected ear (Hain and Spindler, 1993). Head-shaking nystagmus rarely beats toward the affected ear; this may be related to recovery nystagmus ('Erholungsnystagmus') when prior compensation becomes inappropriately excessive as a peripheral function recovers. Central vestibular lesions may also induce an asymmetry in

velocity storage, which itself can produce HSN even though the peripheral vestibular inputs are balanced. Furthermore, horizontal head-shaking may also lead to a vertical nystagmus due to cross-coupling of nystagmus, which is also compatible with a central vestibular origin of HSN (Leigh and Zee, 1999). Head-shaking nystagmus may indicate a 'latent' (compensated) vestibular tonus imbalance, since it has also been found in healthy control subjects (12 of 50) (Asawavichiangianda et al., 1999).

4.7.3. Politzer's balloon: testing for perilymph fistula or superior canal dehiscence syndrome

Patients who report attacks of rotatory or postural vertigo caused by changes in pressure, for example, by coughing, pressing, sneezing, lifting, or loud noises and accompanied by illusory movements of the environment (oscillopsia) and instability of posture and gait with or without hearing disturbances may suffer from perilymph fistula (Nomura, 1994) and should be tested with Politzer's balloon. The balloon can be used to apply positive and negative pressures to the middle ear. One should look for eye movements, especially nystagmus, vertigo, dizziness, oscillopsia, or blurred vision induced by these changes in pressure. A perilymph fistula may be caused by (a) a pathological motility of the membrane of the oval or round window or the ossicular chain with a hypermotility of the oval window (Dieterich et al., 1989) or (b) bony defects in the region of the lateral wall of the labyrinth (toward the middle ear) together with a partial collapse of the perilymphatic space, so-called 'floating labyrinth' (Nomura et al., 1992).

A bony defect toward the epidural space of the anterior canal is the cause of the 'dehiscence of the superior semicircular canal,' in which vertigo and/or eye movements can also be induced by changes in pressure, e.g. by Politzer's balloon (Deutschländer et al., 2004; Minor et al., 1998). The superior canal dehiscence syndrome can be diagnosed by high resolution CT scan of the temporal bone (Hirvonen et al., 2003).

4.7.4. Hyperventilation

Hyperventilation leads to alkalosis and changes in the transmembraneous potential of cells which cause increased excitability. It may induce a transient nystagmus in vestibular paroxysmia, which is characterized by recurrent but short attacks of vertigo due to a neurovascular cross-compression of the VIIIth cranial nerve in the root entry zone as in trigeminal neuralgia (Brandt and Dieterich, 1994b; Jannetta et al., 1984; Moller et al., 1986), and in vestibular schwannoma (Minor et al., 1999). Downbeat nystagmus due to cerebellar lesions may also worsen during hyperventilation (Walker and Zee, 1999).

4.8. Stance and gait

Finally, the patient's balance should be examined under static conditions There are different variations of the *Romberg* and *one-leg stance test*: feet next to each other with eyes first open and then closed (to eliminate visual cues); standing on one foot at a time with the head in normal position or with reclining head (the latter creates extreme imbalance). If a psychogenic disorder is suspected, the examiner distracts the patient by writing numbers on her arm or having her do maths mentally. If there is improvement under the last condition, the stance disorder has a psychogenic-functional origin. Another variation is the Romberg test in tandem, during which the patient places one foot directly in front of the other (the toes of one foot touch the heel of the other). Excessive fore–aft, right–left, or diagonal sway should be looked for. A peripheral vestibular functional disorder typically causes ipsiversive falls; upbeat and downbeat nystagmus syndromes are typically associated with increased body sway forward and backward once the eyes are closed. The analysis of posture and gait

Table 11
Disturbance of posture and gait control in peripheral vestibular disorders

Illness	Direction of deviation	Pathomechanism
Vestibular neuritis	Ipsiversive	Vestibular tonus imbalance due to failure of the horizontal and anterior semicircular canal and utricle (Strupp, 1999)
Benign paroxysmal positioning vertigo (BPPV)	Forward and ipsiversive	Ampullofugal stimulation of the posterior canal due to canalolithiasis that leads to endolymph flow (Brandt and Steddin, 1993; Brandt et al., 1994)
Attacks of Menière's disease (Tumarkin's otolithic crisis)	Lateral ipsiversive or contraversive (falls)	Variations of the endolymph pressure lead to an abnormal stimulation of the otoliths and sudden vestibular–spinal tonus failure (Odkvist and Bergenius, 1988; Schuknecht and Gulya, 1983)
Tullio phenomenon	Backward, contraversive, diagonal	Stimulation of the otoliths by sounds of certain frequencies, e.g. in cases of perilymph fistulas or superior canal dehiscence syndrome (Tullio, 1927)
Vestibular paroxysmia	Contraversive or in different directions	Neurovascular compression of the vestibulo-cochlear nerve and excitation (rarely inhibition) of the vestibular nerve (Arbusow et al., 1998; Brandt and Dieterich, 1994b)
Bilateral vestibulopathy	All directions, especially forward and backward	Failure of vestibular–spinal postural reflexes, exacerbated in the dark and on uneven ground (Rinne et al., 1995)

Table 12
Disturbance of posture and gait control in central vestibular disorders

Illness	Direction	Pathomechanism
Vestibular epilepsy (rare)	Contraversive	Focal seizures due to epileptic discharges of the vestibular cortex (Brandt and Dieterich, 1993b)
Thalamic astasia (often overlooked)	Contraversive or ipsiversive	Vestibular tonus imbalance due to posterolateral lesions of the thalamus (Masdeu and Gorelick, 1988)
Ocular tilt reaction	Contraversive with mesencephalic lesions, ipsiversive with pontomedullary lesions	Tonus imbalance of the vestibulo-ocular reflex in the roll plane with lesions of the vertical canals or otolith pathways (Brandt and Dieterich, 1993a)
Paroxysmal 'ocular tilt reaction'	Ipsiversive with mesencephalic excitation, contraversive with pontomedullary excitation or excitation of the vestibular nerve	Pathological excitation of the otolith or vertical canal pathways (VOR in the roll plane) (Dieterich and Brandt, 1993b)
Lateropulsion (Wallenberg's syndrome)	Ipsiversive, diagonal	Central vestibular tonus imbalance ('roll and yaw planes') with tilt of subjective vertical (Dieterich and Brandt, 1992)
Downbeat nystagmus syndrome	Backward	Vestibular tonus imbalance in the 'pitch plane' (Brandt and Dieterich, 1995)

instability frequently allows differentiation between peripheral (Table 11) and central vestibular disorders (Table 12) (Bronstein et al., 2004; Jacobson et al., 1993).

5. Laboratory examinations

The laboratory examinations measure:

(a) *eye movements* by, e.g. electronystagmography (ENG), video-oculography, and the magnetic search coil technique;
(b) *semicircular canal function* by either caloric irrigation for the horizontal canal, rotational testing or determination of the gain of the VOR for all 3 canals by combining the head-impulse test (see above, Fig. 5);
(c) *otolith function* by assessing the eye position in roll by the laser-scanning ophthalmoscope, ocular counterroll (a test which still has to be established for general vestibular testing), and vestibular-evoked myogenic potentials for saccule function;
(d) *spatial perceptional function*, e.g. by determining the subjective visual vertical; and
(e) *postural control* by means of posturography. Figs. 9–13 and Table 4 summarize the essential neuro-ophthalmological laboratory procedures of examination, give typical findings, indicate how to interpret them, and describe an orthoptic examination.

Table 13 shows the advantages and disadvantages of the different laboratory examinations in comparison with the neuro-ophthalmological and neuro-orthoptic examinations.

Table 13
Neuro-ophthalmological examination and laboratory diagnostics for vestibular and ocular motor disorders

Technique	Features	Advantages	Disadvantages
Neuro-ophthalmological examination	Total range of eye movements, horizontal, vertical (torsional)	No technical requirements, simple, resolution $<1°$	No recording, eye movement velocity cannot be judged
Orthoptic examination	Fundus photography, determination of eye misalignment and psychophysical determination of, e.g. the subjective visual vertical	Precise measurement with documentation, non-invasive, well-tolerated	Expensive apparatuses (e.g. scanning laser ophthalmoscope)
ENG	Measurement range $\pm 40°$, resolution of 1°	Non-invasive, well-tolerated even by children, caloric stimulation possible, widespread method	No measurement of torsional and poor measurement of vertical movements, eyelid artifacts, baseline drift
Video-oculography	Measurement range $\pm 40°$, resolution of 0.1–1°	Non-invasive, well-tolerated, possible to measure torsion	Measurement only possible with eyes open, 3-D analysis is still complicated and expensive
Infrared system	Measurement range $\pm 20°$, resolution of 0.1°	High resolution, non-invasive	Measurements only possible with open eyes, relatively expensive, vertical measurements poor, torsional measurements not possible
Magnetic-coil technique	Measurement range $\pm 40°$, resolution of 0.02°	Best resolution of horizontal, vertical, and torsional movements (research)	Semiinvasive, unpleasant, expensive, only with cooperative patients, maximum of 30 min, local anesthetic necessary
Vestibular-evoked myogenic potentials	Examination of saccular function	Non-invasive, well-tolerated, simple to perform	Still moderate clinical experience has been made, some findings in part still contradictory

To clarify the cause of disorders (differential diagnoses: ischemia, hemorrhage, tumor, or inflammation) additional *imaging techniques* (Casselman, 2002; Mark and Fitzgerald, 1994) are necessary: primarily cranial magnetic resonance imaging with precise sections of the brainstem, the cerebellopontine angle, and the labyrinth; high resolution CT of the temporal bone, e.g. in superior canal dehiscence syndrome or temporal bone fractures; Doppler sonography; and in some cases also a spinal tap, auditory evoked potentials, and audiometry. As a rule, an otolaryngologist gives a hearing test. In connection with the main symptom of vertigo, audiometry is especially important for diagnosing Menière's disease, labyrinthitis, vestibular schwannoma, and other diseases of the vestibulo-cochlear nerve as well as bilateral vestibulopathy. These techniques are not described in this review.

5.1. Measurements of eye movements/eye position

5.1.1. Electronystagmography (ENG)

To quantitatively record eye movements, two electrodes are placed horizontally and vertically on each eye so that the changes in the dipole between the retina and cornea, which occur with eye movements, can be recorded (Fig. 7)

Fig. 7. *Electronystagmography* (ENG). (a) Placement of the electrodes for *monocular recording* of horizontal and vertical eye movements. The electrophysiological basis of the ENG is the corneo-retinal dipole (a potential difference of about 1 μV). The dipole is parallel to the longitudinal axis of the eye, with the retina or the cornea having a negative potential. Changes in this dipole between the horizontal or vertical electrodes are DC-amplified. The ENG allows non-invasive horizontal recordings of ±40° with an accuracy of about 1° and vertical recordings of ±20°. Major disadvantages are susceptibility to eyeblink artifacts, electromyographic activity, and unstable baseline; torsional eye movements cannot be recorded with the ENG. (b) *Rotatory chair and rotatory drum* (with vertical stripes) with an apparatus that projects a laser spot (above the patient). This setup allows recordings of eye movements under static conditions (e.g. test for spontaneous or gaze-evoked nystagmus, saccades, pursuit, and optokinetic nystagmus) and under dynamic conditions (per- and postrotatory nystagmus, fixation suppression of the vestibulo-ocular reflex), as well as positional and positioning testing and caloric irrigation. (c) *Caloric testing by electronystagmography*. By means of caloric testing, the excitability of the individual horizontal canals can be determined and thus whether or not they are functioning. After excluding the possibility of a lesion of the eardrum, the head of the patient is tilted 30° upward, so that the horizontal semicircular canals approach the vertical plane. This allows optimal caloric stimulation. The outer auditory canals on each side are separately irrigated under standard conditions with 30 °C cool and 44 °C warm water. At the same time the horizontal and vertical eye movements are recorded by means of electronystagmography. The irrigation with 44 °C warm water causes excitation of the hair cells of the horizontal canal and slow contraversive eye movements; the 30 °C cool water leads to an inhibition of the slow ipsiversive eye movements.

(Furman et al., 1996; Jongkees et al., 1962). ENG also allows documentation of the findings (important for monitoring the course of the patient) and, for example, exact measurements of saccade velocity and saccade accuracy. In addition, irrigation of the external auditory canal with 30 °C cool and 44 °C warm water (caloric testing) can be used to detect loss of labyrinthine function (horizontal canal). To quantify peripheral vestibular function the maximal velocity of the irrigation-induced eye movements (peak slow phase velocity, PSPV) should be measured; PSPV values less than 5°/s are considered pathological. Since there is considerable interindividual variation of caloric excitability, the so-called 'vestibular paresis formula' of Jongkees (Jongkees et al., 1962) is used to compare the function of both labyrinths: $(((R30°C + R44°C) - (L30°C + L44°C))/(R30°C + R44°C + L30°C + L44°C)) \times 100$, where for instance, R 30°C is the PSPV during caloric irrigation with 30°C cool water. Values of >25% asymmetry between the affected and non-affected labyrinth are considered pathological and indicate, for example, a unilateral peripheral vestibular disorder. This formula allows a direct comparison of the function of the horizontal semicircular canals of both labyrinths and is highly reliable in detecting unilateral peripheral vestibular loss (Fife et al., 2000). This is also a reliable parameter for follow-up or treatment studies (Strupp et al., 2004).

Rotational chair testing requires that the subject sit on a chair that rotates or oscillates with certain velocities/frequencies, while his eye movements are being measured in parallel by electronystagmography (or video-oculography, see below). During longer rotations at constant speed, however, the thus-induced rotational nystagmus resolves (time constant about 20 s). If the rotational chair is then suddenly stopped, postrotational nystagmus (P I) can also be measured. The aim of rotational chair testing is to determine the gain of the VOR, i.e. the slow component eye velocity: head velocity (a gain of 1.0 indicates a perfect VOR). In contrast to caloric irrigation (which tests the VOR at a single, effectively very low frequency of 0.003 Hz), rotational chair testing allows examination of the VOR at different frequencies. Since rotation affects both labyrinths simultaneously, it is not very helpful for diagnosing unilateral vestibular hypofunction. However, it is the only reliable test for bilateral vestibular failure (Baloh et al., 1984a,b; Hess et al., 1985).

All in all, ENG including caloric irrigation and rotational chair testing is the most important and clinically relevant neuro-otological laboratory examination. Therefore, every patient with vertigo or dizziness should be examined at least once by this technique or by video-oculography (see 5.1.2). Although widely used, electronystagmography has certain limitations. The measurement of vertical eye movements is not always reliable, especially due to artifacts of eyelid movements. Other muscle artifacts and electronic noise reduce its sensitivity, and there may be a baseline drift, mainly due to the subject's sweating (Baloh and Honrubia, 1979; Black and Hemenway, 1972).

5.1.2. Video-oculography

Video-oculography or video-nystagmography is another non-invasive method that is now being used more frequently (Vitte and Semont, 1995a,b; Schneider et al., 2002). It has the same clinical relevance as ENG but is cheaper and easier to handle. The eyes are first filmed by one or two video cameras (i.e. monocular or binocular recording) integrated in a mask attached to the head. Then a computer analysis of the image of movements of the pupils and light reflexes is performed to represent the eye movements in two dimensions. This method allows rapid and reliable recording of horizontal and vertical eye movements (without muscle artifacts or unstable baseline). Recording is only possible when the eyes are wide open, and the resolution is limited due to the image repeat frequency of the video camera (today generally limited to 100 Hz). There is a largely linear resolution in the range of $\pm 30°$. The use of 3-D representation of eye movements for research purposes (i.e. additional measurement of torsion) requires an extensive analysis of the image of the iritic structures or of two additional marker dots applied to the sclera (Fig. 8) (Schneider et al., 2002).

5.1.3. Magnetic search coil technique

The magnetic search coil technique (Fig. 9) is the gold standard of scientific eye movement recordings for several reasons (Bartl et al., 1996; Robinson, 1963). It allows the recording of horizontal, vertical, and torsional eye movements, i.e. in all 3 planes, including recordings of head movements (see below). Its sensitivity is <5 min of an arc and the drift is minimal. With the magnetic search coil

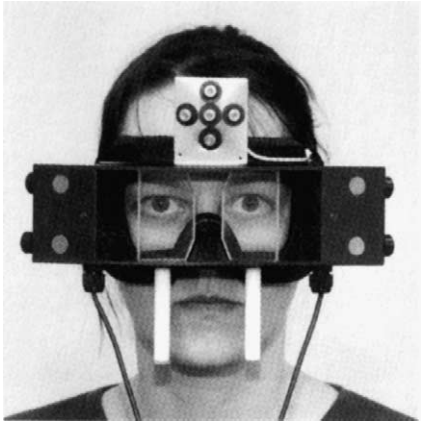

Fig. 8. *Video-oculography* performed with a mask attached to the head in which a camera is integrated. An infrared headlight built into the mask also allows measurement of eye movements in complete darkness. The signal of the integrated camera is transmitted to a normal digital video camera and finally stored on a PC. The pictures are analyzed offline by means of a video-oculography program that calculates the eye movements. A Cajal dot placed on the sclera simplifies the registration or analysis of eye movements in the roll plane.

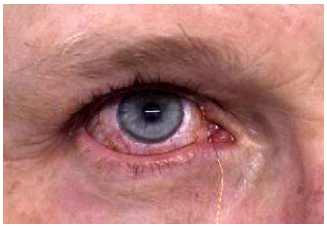

Fig. 9. The magnetic search coil technique. Silastic annulus with imbedded coils of fine wire is put on the cornea. The subject sits in a magnetic field within a cage. Changes of the eye position cause a current that is amplified and recorded, thus allowing the three-dimensional recording of eye movements.

technique, eye movements can be recorded and analyzed three-dimensionally and their trajectory reconstructed, thus, the semicircular canal(s) involved can be determined. This is important for the diagnosis of, e.g. superior canal dehiscence syndrome (Cremer et al., 2000a,b), or detailed analysis of BPPV (Fetter and Sievering, 2000), vestibular neuritis (Fetter and Dichgans, 1996), isolated lesions of certain semicircular canals (Cremer et al., 2000a,b), and alcohol-induced nystagmus (Fetter et al., 1999).

The major disadvantage of this 'semiinvasive' method is that a kind of contact lens has to be used which makes necessary the application of topical anesthetic drops to the cornea. These drops are rarely harmful to the cornea. Recordings longer than 20–30 min should not be performed.

Fig. 10. *Measurement of the eye position in the roll plane*. The scanning laser ophthalmoscope (SLO) can be used to make photographs of the fundus of the eye (examination is also possible with a fundus camera). The rolling of the eye or eye torsion can be measured in degrees on the fundus photographs as the angle between the horizontal and the so-called papillofoveal meridian. The patient sits upright, looks into the SLO, and fixates a dot. (It is not necessary to administer a mydriatic drug; however, it is necessary if the measurement is made with traditional fundus photography). Both eyes of healthy controls exhibit a slightly excyclotropic position in the roll plane, i.e. counterclockwise rotation of the right eye, clockwise rotation of the left eye (from the viewpoint of the examiner). The normal range (± 2 SDs) is from -1 to 11.5 degrees. Values outside this range are considered pathological (e.g. patients with a peripheral vestibular lesion show an ipsiversive ocular torsion).

In general, the magnetic search coil technique is mainly used as a research tool; its clinical relevance is limited.

5.1.4. Measurement of the static eye position in the roll plane

Measurement of ocular torsion by fundus photography or the 'scanning laser ophthalmoscope' (Ott and Eckmiller, 1989) (Fig. 10) is of special importance for the diagnosis of central vestibular disorders that affect graviceptive pathways, e.g. the ocular tilt reaction and for the differentiation of ocular tilt reaction (OTR) (Brandt and Dieterich, 1993a; Dieterich and Brandt, 1993b; Strupp et al., 2003) and trochlear palsy (Dieterich and Brandt, 1993a). This technique has become more and more important in centers specialized in the diagnosis and treatment of patients suffering from vertigo or dizziness.

5.2. Measurements of the gain of the VOR and of otolith function

5.2.1. Three-dimensional analysis of eye movements and the VOR

Ewald's first law predicts that the axis of the nystagmus matches the anatomic axis of the semicircular canal that generated it (Ewald, 1892). This law is clinically useful when diagnosing the detailed pathology of the vestibular end-organ. The magnetic search coil technique (5.1.3 and Fig. 9) in combination with the head-impulse test (4.4 and Fig. 5) allows calculation of the gain of the VOR for each plane and thus, for each semicircular canal (Cremer et al., 1988; Cremer et al., 2000a,b; Della-Santina et al., 2001; Halmagyi et al., 1991; Halmagyi et al., 1992). The involvement of certain canals, for instance, in vestibular neuritis can be determined by this method (Aw et al., 2001).

5.2.2. Measurement of otolith function

5.2.2.1. Vestibular-evoked myogenic potentials (VEMPs). In response to loud clicks, a vestibular-evoked potential can be recorded from sternocleidomastoid muscles. This is called a 'vestibular-evoked myogenic potential' (VEMP) (Fig. 11) (Colebatch et al., 1994; Murofushi et al., 1996; Murofushi et al., 2002). There is evidence that VEMPs originate in the medial (striola) area of the saccular macula (Murofushi et al., 1995). Therefore, they should test for otolith function and also for central lesions, which may show pathological latencies in case of multiple sclerosis (Murofushi et al., 2001b). The findings for individual illnesses are as follows. *Vestibular neuritis*: the VEMP is preserved in two-thirds of the patients. This is due to the sparing of the pars inferior of the vestibular nerve, which supplies the saccule and posterior canal, among others (Colebatch, 2001; Murofushi et al., 1996). *Tullio phenomenon in cases of superior canal dehiscence syndromes or perilymph fistula*: here there is a clearly lowered stimulus threshold, i.e. a stimulus reaction occurs already at low dB values. *Bilateral vestibulopathy*:

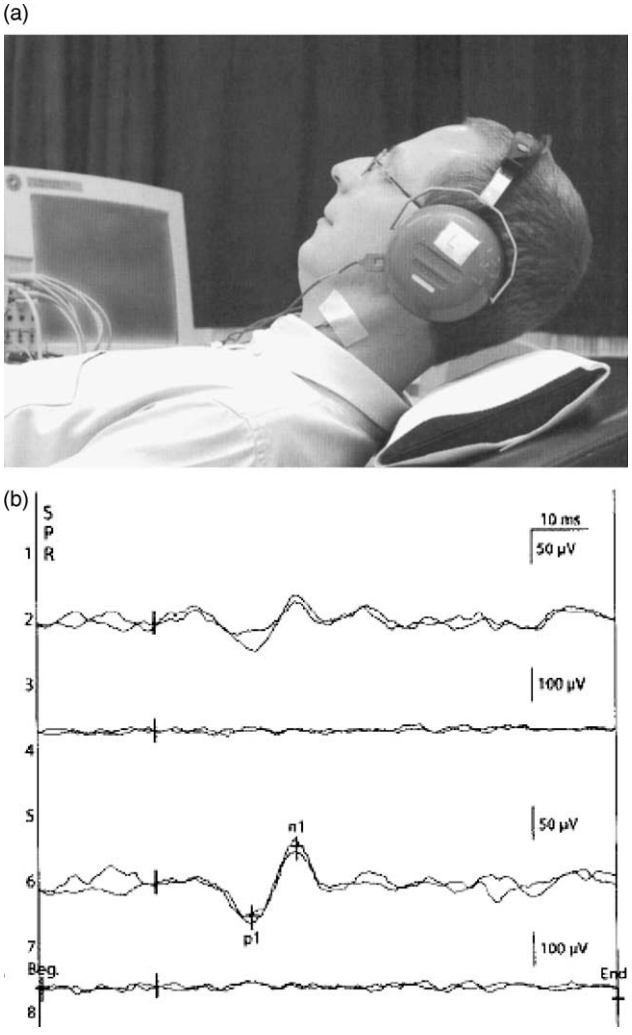

Fig. 11. *Vestibular-evoked myogenic potentials* (VEMPs). The VEMP is used to test the reflex arc of the saccule which extends over the vestibular nerves, vestibular nuclei, interneurons, and motor neurons to the neck musculature (sternocleidomastoid m.). It complements caloric testing, since the latter tests only the canal system and not the otolith function. The prerequisite for VEMP testing is an intact middle ear function; it is not necessary that hearing be preserved, since the 'sensitivity to sound' of the saccule can be used in the VEMP. The reflex is triggered by a loud click. Surface EMG is used to record from both sternocleidomastoid muscles. (a) Healthy subjects first show on the ipsilateral side a positive wave (about 14 s after the stimulus) as well as a negative wave (about 21 ms; lines 1 and 3). (b) The responses can as a rule not be recorded contralaterally (lines 2 and 4). Approximately 50–100 averagings are necessary for the recording. It is important that the musculature is tense, e.g. for this the test person can raise his head from the support surface. Evaluation criteria are the presence of the waves P14 and N21 as well as their amplitude. The absence of these waves as well as a clearly reduced amplitude are considered pathological; the relevance of changes in latency must still be determined (Recording (b) by K. Botzel, Munich).

the VEMP is absent in only a portion of the patients; this should be interpreted as a sign of additional damage to the saccular function (Colebatch, 2001; Matsuzaki and Murofushi, 2001). *Menière's disease*: the VEMPs are frequently reduced or absent (Murofushi et al., 2001a). The clinical relevance of VEMP for general vestibular testing has still to be evaluated.

5.3. Psychophysical procedures

5.3.1. Measurement of the subjective visual vertical

Recent years have witnessed the growing importance of psychophysical examination procedures, in particular the psychophysical determination of the subjective visual vertical (SVV) (Fig. 12) (Böhmer and Mast, 1999; Böhmer and Rickenmann, 1995). The topographical and diagnostic significance of these procedures is particularly evident when differentiating between peripheral and central vestibular or ocular motor lesions and between OTR (Brandt and Dieterich, 1993a) and trochlear palsy (Dieterich and Brandt, 1993a). In our experience the determination of SVV is an important clinical tool for all patients suffering from vertigo, dizziness, or ocular motor disorders. It is easy to handle and the findings are easy to interpret.

5.4. Posturography

Posturography allows the examination and quantification of postural stability under different conditions, such as standing with the eyes open/eyes closed, standing on firm ground, or standing on a foam rubber platform (Fig. 13) and under static or dynamic conditions (Baloh et al., 1998; Black, 2001; Black et al., 1989; Di Fabio, 1996; Furman, 1994; Hamid et al., 1991). From the raw data (measured changes in the center of gravity to the right, left, forward,

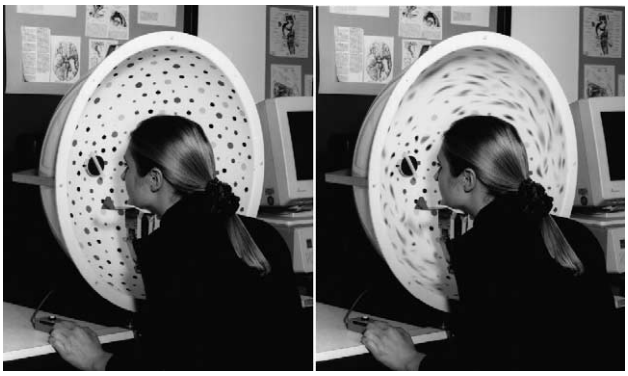

Fig. 12. *The subjective visual vertical (SVV)*. For determination of the SVV, the patient sits upright in front of a hemispheric dome (60 cm in diameter) and looks into it. The surface of the dome extends beyond the limits of the patient's visual field and is covered with a random pattern of dots. This prevents the patient from orienting himself spatially by fixed external structures. The hemispheric dome is connected axially to a motor and can be rotated; a circular target disk (14° of visual field) with a straight line through the center is 30 cm in front of the patient at eye level. The line is also connected with a DC motor and can be adjusted by the patient by means of a potentiometer until he has the subjective impression that the line is 'vertical'. The deviation of the line from the objective vertical axis is measured in degrees and registered on a PC. The mean of 10 measurements equals the SVV. Under these conditions, the normal range (mean ± 2 SDs) of the SVV is $0° \pm 2.5°$. Measurements can be made under static (left) and dynamic (right) conditions.

Fig. 13. *Posturography*. This technique allows the examination of control of postural stability (here a Kistler platform). The parameters include the original registrations of body sway to the right or left, forward or backward, upward or downward; the frequency analysis of the sway (Fourier power spectra); and the so-called sway path values (SP, m/min). The SP is defined as the length of the path described by the center of foot pressure during a given time. Healthy subjects also exhibit body sway as a result of inherent physiological instability when standing on a recording platform; SP is exacerbated in vestibular disorders. The SP values can be derived automatically with a PC for the anteroposterior, mediolateral, and craniocaudal directions and as the sum of both components. These values are calculated as the distances between two consecutive sampling points (measured every 25 ms); the anteroposterior (*sagittal* = *x*) plane, i.e. sagittal sway (calculated as $\sum|\Delta x|$), the mediolateral (*frontal* = *y*) plane, i.e. frontal sway (calculated as $\sum|\Delta y|$), and the craniocaudal (transversal = *z*) plane, i.e. the transversal sway (calculated as $\sum|\Delta z|$) or for all 3 planes as the *total* SP.

backward, upward, and downward directions), different parameters can be calculated, for example, the sway path, sway path histograms for determining the preferential direction of sway, or a frequency analysis can be made (Fourier power spectra). The results of posturography are clinically significant, e.g. for documenting the direction of falls (lateropulsion in Wallenberg's syndrome or thalamic astasia) and for monitoring the course of postural instability in degenerative cerebellar disorders or during rehabilitation. Posturography also plays an important role in clinical research. In the analysis of sway direction, for example, it may indicate the side of lesion (semicircular canal or otolith organs) and the type of dysfunction (excitation or inhibition). It is also used to measure postural sway activity, e.g. in phobic postural vertigo (Holmberg et al., 2003; Querner et al., 2000), during the treatment of vestibular disorders (Strupp et al., 1998), and after the application of pharmacological agents such as nicotine (Pereira et al., 2001) or other agents. Despite its applications in research, posturography is of limited usefulness for general vestibular testing, because the findings are often non-specific for certain disorders and, therefore, it does not help detect the underlying dysfunction. However, there are exceptions, such as the characteristic 3-Hz sway in (alcohol-induced) anterior lobe cerebellar atrophy (Diener et al., 1984) and the pathognomonic 14–18 Hz sway in orthostatic tremor (Yarrow et al., 2001).

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