

Vertigo and Dizziness

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Common Complaints

 Springer

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Preface

There are three convincing arguments why it is important to learn about the management of vertigo:

- After headache, it is the second most common complaint of patients, not only in neurology and ENT departments.
- Most syndromes of vertigo can be correctly diagnosed only by means of a careful medical history and physical examination of the patient.
- The majority of these cases have a benign cause, take a favourable natural course, and respond positively to therapy.

Vertigo and dizziness are not disease entities, but rather unspecific syndromes consisting of various disorders with different causes. For this reason, our clinically oriented book is for physicians of different specialisations who treat patients with vertigo and for medical students. To make the book easy to use, we have provided an overview of the most important syndromes of vertigo and dizziness, each with elucidating clinical descriptions and illustrations.

A general chapter deals with how the vestibular system functions, its disorders, the pathophysiological mechanisms involved, diagnostic signs, history taking, examination procedures, laboratory diagnostics and principles of therapy. The most important clinical syndromes of vertigo are treated in individual chapters organised as follows: patient medical history, clinical aspects and natural course, pathophysiology and principles of therapy, pragmatic therapy, ineffective treatments, as well as differential diagnosis and clinical problems. We have put special emphasis on the various drug, physical, operative or psychotherapeutic treatments available. The book is based on the common experience that we have accumulated over many years working in a multi-

regional referral center for dizziness outpatients. Many parts of the text, tables and figures are updated versions of those in a considerably more detailed monograph on the clinical and scientific aspects of vertigo (Brandt T. *Vertigo: its multisensory syndromes*, 2nd ed. Springer, London, 1999). The accompanying DVD presents typical case histories, results of examinations for the individual syndromes, physical examination techniques and laboratory diagnostics. The book is oriented to daily medical practice, and we hope that it will prove helpful by providing readily accessible information. The whole field of vertigo and dizziness, imbalance and eye movement disorders has been considered extremely difficult because of the variety of its manifestations and its resistance to compartmentalisation. We hope that we have succeeded in making these syndromes more understandable by using clear, anatomical categories and clinical classifications.

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Introductory Remarks

1.1 Vertigo and Dizziness: Multisensory Syndromes

Vertigo and dizziness are not unique disease entities. Sometimes vertigo is attributed to vestibular disorders, while dizziness is not (Neuhäuser and Lempert 2004). There is, however, no general agreement, and visual stimuli can cause vertigo (e.g., height vertigo or optokinetic vection), just as central vestibular or otolith disorders can cause dizziness. The two terms cover a number of multisensory and sensorimotor syndromes of various aetiologies and pathogeneses, which can be elucidated only within an interdisciplinary approach. After headache, 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 as a function of age is around 17%; it rises to 39% in those over 80 years of age (Davis and Moorjani 2003). Whether caused by physiological stimulation (motion sickness, height vertigo) or a lesion (unilateral labyrinthine failure, central vestibular pathway lesions), the resulting vertigo syndrome characteristically exhibits similar signs and symptoms despite the different pathomechanisms—dizziness/vertigo, nausea, nystagmus and ataxia (Figure 1.1). Disorders of perception (dizziness/vertigo), gaze stabilisation (nystagmus), postural control (falling tendency, ataxia) and the vegetative system (nausea) are related to the main functions of the vestibular system, which are located in different sites in the brain.

The most important anatomical structure of the vestibular system is the vestibulo-ocular reflex (VOR). The VOR has three major planes of action:

2 1. Introductory Remarks

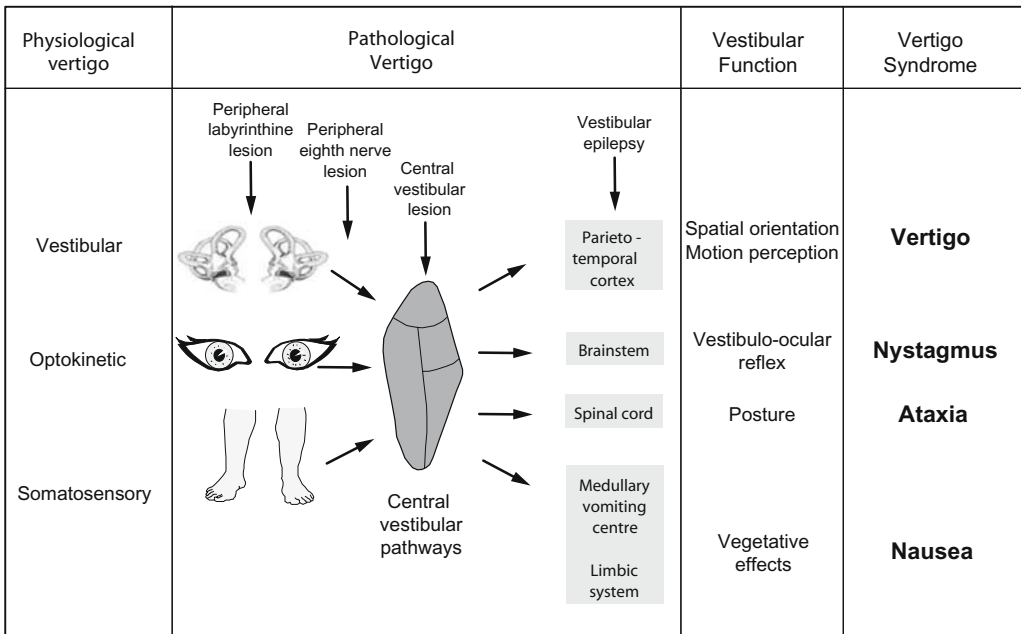


Figure 1.1. Physiological vertigo (motion stimulation) and pathological vertigo (induced by lesion or stimuli) are characterised by similar signs and symptoms that derive from the functions of the multisensory vestibular system (Brandt and Daroff 1980)

- horizontal head rotation about the vertical z-axis (yaw)
- head extension and flexion about the horizontal y-axis (pitch)
- lateral head tilt about the horizontal x-axis (roll).

These three planes represent the three-dimensional (3-D) space in which the vestibular and ocular motor systems responsible for spatial orientation, perception of self-movement, stabilisation 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 (Figure 1.2). The VOR connects a set of extraocular eye muscles that are aligned by their primary direction of pull with the same particular spatial plane of the horizontal, the anterior or the posterior canal. The canals of both labyrinths form functional pairs in the horizontal and vertical working planes. In other words, the canals are excited or inhibited pairwise: the horizontal right and left pair; 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 pairs of canals function as a gauge of rotatory acceleration and react to the rotational movements of the head in the corre-

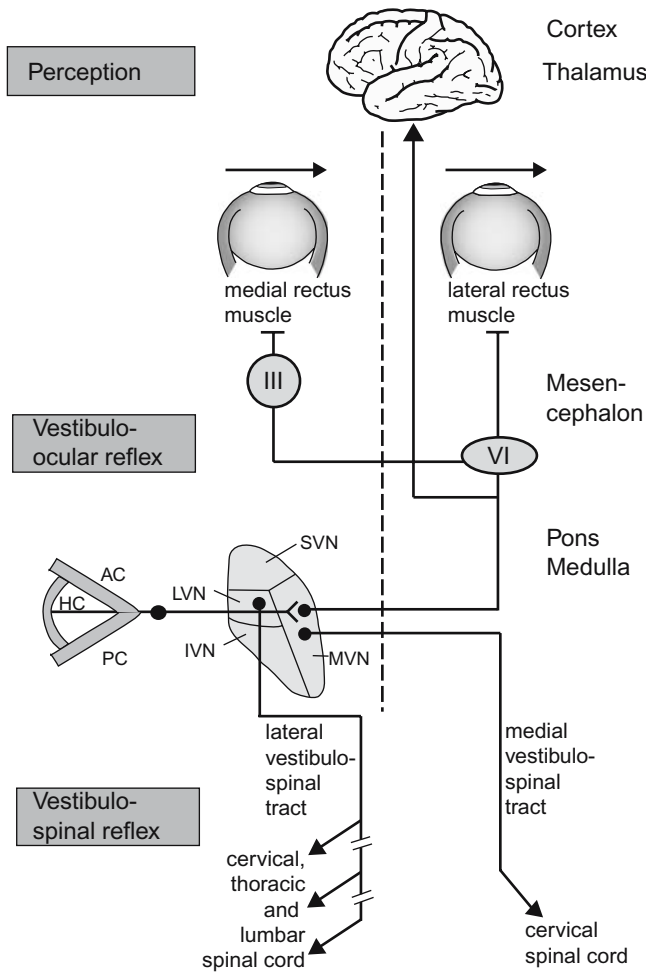


Figure 1.2. Schematic drawing of the horizontal vestibulo-ocular reflex (VOR). The VOR is a part of a complex sensorimotor system, which makes possible perception of head position and motion (connections via the thalamus to the vestibular cortex), gaze stability (three-neuron arc to the nuclei of the ocular muscles), as well as head and postural control (vestibulospinal reflexes). *AC, HC, PC* anterior, horizontal and posterior semicircular canals; *SVN, LVN, IVN, MVN* superior, lateral, inferior and medial vestibular nuclei; *III, VI*, oculomotor and abducens nuclei.

sponding plane. The otoliths function as a gauge of gravity and linear acceleration.

The most frequent forms of peripheral vestibular vertigo are benign paroxysmal positioning vertigo (BPPV), vestibular neuritis and Menière's disease. The neurovascular compression syndrome of the VIIIth cranial nerve (vestibular paroxysmia), bilateral vestibulopathy and perilymph fistulas occur more

rarely. Peripheral vestibular attacks, which affect semicircular canal function, are characterised by a strong rotatory vertigo and spontaneous nystagmus in one direction, a tendency to fall in the other direction, nausea and vomiting.

Central vestibular forms of vertigo arise from lesions at the neuronal circuitry between the vestibular nuclei and the vestibulocerebellum, 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 are clearly defined clinical syndromes of various aetiologies, 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 brainstem or cerebellar disorders allows their definitive localization. On the other hand, central vestibular vertigo can also be a part of a more complex infratentorial clinical syndrome. Then 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. Central forms of vertigo can manifest as attacks lasting for seconds or minutes (basilar/vestibular migraine), for hours up to days (brainstem infarction), or as a permanent symptom (downbeat nystagmus in cases of Arnold–Chiari malformation).

Our first overview of the most frequent forms of vertigo/dizziness was made in an unselected group of in- and outpatients in the early 1980s. It showed that BPPV, vestibular neuritis, phobic postural vertigo and Menière's disease accounted for 85% of cases of vertigo; their respective frequency is according to the order cited. After the establishment of a multiregional neurological unit for vertigo and dizziness, a second overview was made for the years 1989 to 2003 (for 4,790 patients). It revealed that the whole spectrum as well as the relative frequency of the individual diagnoses had shifted (Table 1.1).

Benign paroxysmal positioning vertigo was still the most frequent cause, occurring in 18.3% of the patients. The second most frequent diagnosis was phobic postural vertigo (15.9%), followed by central forms of vestibular vertigo in vascular and inflammatory diseases (lacunar infarcts or multiple sclerosis [MS] plaques) of the brainstem or the cerebellum. Basilar/vestibular migraine has two frequency peaks: one in the second decade and another in the sixth decade, thus it is by no means a disease of only younger women. Now it is the fourth most frequent cause, followed by vestibular neuritis and Menière's disease.

It is difficult to compare the frequency data of various hospitals and medical specialisations, because the definitions of the concept "vertigo/dizziness" differ greatly. Some are broader, others more narrow. Vertigo/dizziness is seen either as a subjec-

Table 1.1. Relative frequency of different vertigo syndromes diagnosed in a dedicated neurological dizziness unit (n = 4,790 patients in 1989–2003)

Diagnosis	Frequency %
Benign paroxysmal positioning vertigo	18.3
Phobic postural vertigo (PPV)	15.9
Central vestibular vertigo	13.5
Vestibular migraine	9.6
Vestibular neuritis	7.9
Menière's disease	7.8
Bilateral vestibulopathy	3.6
Psychogenic vertigo (without PPV)	3.6
Vestibular paroxysmia	2.9
Perilymph fistula	0.4
Various other disorders	12.3
Unknown aetiology	4.2

tive symptom or as a functional vestibular disorder that can be objectified. Both tendencies are dissatisfying, as on the one hand, the symptom of dizziness or vertigo is observed in non-vestibular functional disturbances (orthostatic dysregulation) and, on the other hand, central vestibular functional disturbances (lateropulsion in Wallenberg's syndrome) also occur without any subjective dizziness.

1.2 Patient History

Vertigo is considered either an unpleasant disturbance of spatial orientation or the illusory perception of a movement of the body (spinning and wobbling) and/or of the surroundings. Care is necessary when taking the neuro-otological history of the patient (the usual pre-prepared vertigo questionnaire cannot replace it), especially because the patient's complaint of being "dizzy" is ambiguous. The important criteria for differentiating the various dizziness/vertigo syndromes, the basis for clinical classification, are as follows:

- **type of vertigo:** rotatory vertigo as experienced when riding a merry-go-round (e.g., vestibular neuritis) or postural imbalance, such as during boat trips (e.g., phobic postural vertigo) or numbness (e.g., medication, drug intoxications) (Table 1.2)
- **duration of vertigo:** attacks of vertigo that last for seconds to minutes (vestibular paroxysmia), over hours (e.g., Menière's disease, basilar/vestibular migraine; Table 1.3), persistent

Table 1.2. Numbness as a key symptom

Presyncopal dizziness

- Orthostatic dysregulation
- Vasovagal attacks
- Neurocardiogenic (pre) syncope
- Cardiac arrhythmia and other heart diseases

Psychiatric illnesses

- Hyperventilation syndrome
- Panic attacks
- Agoraphobia
- Acrophobia
- Phobic postural vertigo

Metabolic disorders

- Hypoglycaemia
- Electrolyte disorders (hypercalcaemia, hyponatraemia)

Intoxication

- Alcohol
 - Medications (see Table 6.3, p. 127)
 - Toxic substances (see Table 2.2, p. 79)
-

Table 1.3. Episodic vertigo, diseases with recurrent attacks of vertigo

Labyrinth/vestibulocochlear (VIIIth cranial) nerve

- Menière's disease
- Vestibular paroxysmia
- Perilymph fistula or 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 tumours of the cerebellopontine angle

Central vestibular system

- Transient vertebrobasilar ischaemia

Rare

- "Rotational vertebral artery occlusion syndrome"
- Vestibular epilepsy
- "Room-tilt illusion"
- Paroxysmal ataxia/dysarthrophonia (multiple sclerosis)
- Familial episodic ataxia types 1 and 2
- Paroxysmal "ocular tilt reaction"

Peripheral and/or central

- Basilar/vestibular migraine
 - Benign paroxysmal vertigo of childhood
 - Vertebrobasilar transient ischaemia (e.g., anterior inferior cerebellar artery)
-

Table 1.4. Persistent vertigo or dizziness**Infections**

- *Viral*
 - Vestibular neuritis
 - Herpes zoster oticus
 - Viral neurolabyrinthitis
- *Bacterial*
 - Bacterial meningitis
 - Tuberculous labyrinthitis
 - Syphilitic labyrinthitis
 - Chlamydial labyrinthitis
 - Lyme borreliosis
 - Otitis media (rarely)

Autoimmunological inner ear diseases (see Table 2.2, p. 79)

Tumours

- Vestibular schwannoma
- Meningeoma
- Cholesteatoma
- Epidermoid cyst
- Glomus tumour
- Metastasis
- Meningeosis carcinomatosa

Vascular

- Labyrinthine infarction (anterior inferior cerebellar artery)
- Pontomedullary brainstem infarction
- Vertebrobasilar ectasia
- Hyperviscosity syndrome

Traumatic

- Temporal bone fracture (transverse > longitudinal fracture)
- Labyrinthine concussion
- Post-traumatic otolith vertigo
- Perilymph fistula
- Brainstem concussion

Iatrogenic

- Temporal bone surgery
 - Systemic or transtympanic treatment with aminoglycosides
 - Other ototoxic substances (see Table 2.2)
-

vertigo lasting for days to a few weeks (e.g., vestibular neuritis; Table 1.4), attacks of postural vertigo lasting from minutes to hours (e.g., transient ischaemic attack of the brainstem or cerebellar structures; Table 1.3)

- **trigger/exacerbation of vertigo:** no trigger (e.g., vestibular neuritis), walking (e.g., bilateral vestibulopathy), head turning (e.g., vestibular paroxysmia; Table 1.5), head positioning (e.g., BPPV), coughing, pressing, loud sounds of a certain frequency—as a Tullio phenomenon (e.g., perilymph fistula), or in certain social situations (e.g., phobic postural vertigo).

Table 1.5. Vertigo 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
-

Further questions and investigations should try to identify other possible accompanying symptoms.

Combination of vestibular and audiological symptoms

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

Illusionary movements of the surroundings (oscillopsia)

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