Assessing vestibular function: which tests, when?

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Received: 3 October 1999
Accepted: 7 December 1999

Abstract Vertigo and dizziness are common complaints encountered in clinical practice. The patient’s history and a thorough otoneurological evaluation are essential for identifying the specific pathology behind the patient’s complaints. If the patient reports an illusion of movement (vertigo), this most likely indicates an imbalance within the vestibular system. A sensation of rotatory movement together with a spontaneous nystagmus suggests a lesion involving the semicircular canals, while an illusion of linear movement indicates a disturbance of the otoliths. Nystagmus of central origin or caused by a peripheral vestibular lesion can usually be distinguished by other features in the history or on clinical examination. While peripheral vestibular lesions usually lead to a mixed horizontal-torsional or vertical-torsional nystagmus, a pure vertical or pure torsional nystagmus is always caused by a central lesion. With simple bedside tests such as head-shaking nystagmus and rapid head impulses deficits in labyrinthine function can clearly be detected. For a more thorough investigation of vestibular function at the level of individual semicircular canals and the otoliths, modern techniques are now available such as three-dimensional eye movement vector analysis for the evaluation of individual semicircular canal function, measurement of the subjective visual vertical for utricular, and click-evoked myogenic potentials for saccular testing.

Key words Vestibular testing · Vertigo · Nystagmus · Dizziness · Labyrinth

Introduction

The assessment of vestibular function must always begin with the patient’s history, supplemented by a thorough otoneurological evaluation [14], since in many patients with vertigo the symptoms have already subsided when they are first seen by a physician, and there may appear no abnormalities on clinical examination or on laboratory testing. Typical complaints of vestibular disease include: illusions of rotational motion (vertigo), illusions of linear motion (e.g., mal de débarquement), illusions of spatial disorientation (e.g., tilt illusion), oscillopsia, imbalance, drop attacks (e.g., Tumarkin’s otolithic crisis), lateropulsion, and autonomic accompaniments such as malaise, nausea, and vomiting [3, 8, 15].

In addition to describing the symptoms, in distinguishing between various causes it is important to ask for the time course, what triggers the vertigo, and for additional symptoms (see Table 1). This information makes it possible to diagnose several forms of vertigo even from the history. For the further clinical evaluation of a patient with symptoms that suggest vestibular dysfunction it is mandatory to have a good understanding of the basic principles of vestibular physiology.
Physiological principles of the vestibular system

The vestibulo-ocular reflex (VOR) ensures vision during head motion by moving the eyes contrary to the head to stabilize the line of sight in space; vestibulospinal reflexes keep the head and body upright. The semicircular canals (SCCs) sense angular acceleration to detect head rotation; the otolith organs sense linear acceleration to detect both head translation and the position of the head relative to the pull of gravity. The SCCs are arranged in a push-pull fashion with two canals on each side working together: the right and left lateral, the right anterior and left posterior, and the left anterior and right posterior SCC (Fig. 1). If one partner is excited, the other is inhibited and vice versa, while under steady conditions the primary vestibular afferents have a tonic discharge that is exactly balanced between the corresponding canals. During rotation the head velocity corresponds to the difference in firing rate between SCC pairs. Knowledge of the geometrical arrangement of the SCCs and the otolith organs within the head allows one to localize and interpret more accurately certain patterns of nystagmus and ocular misalignment, since stimulation of a single SCC leads via the VOR to slow-phase eye movements that rotate the globe in a plane parallel to that of the stimulated canal.

A central velocity-storage mechanism acts to perseverate peripheral labyrinthine signals and thus extend the low-frequency response of the VOR from a value of about 6–7 s, which is what would be expected from the mechanical properties of the cupula and endolymph, to values above 10 s.

The vestibular nerve is divided into two branches: a superior branch, which runs with the facial nerve in the internal auditory canal and supplies the anterior and lateral SCC and the utricle; and an inferior branch, which runs with the cochlear nerve and supplies the posterior SCC and the saccule. There is a comparable, parallel blood supply that supplies these two parts of the vestibular endorgan.

Projections from the SCCs are predominantly to the rostral positions of the vestibular nuclei complex; those from the otolith organs are predominantly to the caudal portions of the vestibular nuclei complex.

Vestibular symptoms and signs may reflect static (head still) or dynamic (head moving) disturbances; these disturbances must be considered separately.

Bedside examination

Bedside examination of the VOR and vestibulospinal reflex with careful attention to the ocular motor system and other brainstem and cerebellar functions helps the clinician localize the cause of vertigo in many cases and guide the laboratory evaluation. During clinical examination special attention must be given to the eye movements, as the physiological and anatomical substrate of the ocular motor system is intimately connected with the vestibular system. Apart from this, every patient with vestibular symptoms must have a complete neurological examination including cranial nerves, vision, and hearing [17].