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Hands-on Course 15

Bedside examination of the vestibular and ocular motor system (Level 2)

How to examine the vestibular and ocular motor system

Marianne Dieterich + Alexander Tarnutzer
Munich, Germany + Zurich, Switzerland

Email: Marianne.Dieterich@med.uni-muenchen.de
Email: alexander.tarnutzer@ksb.ch

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Prof. Dr. med. Marianne Dieterich (LMU, Munich, Germany) and PD Dr. med. Alexander A. Tarnutzer (KSB, Baden, Switzerland)

Goals:
By a targeted vestibular and ocular motor exam the clinician is able to distinguish between central and peripheral vestibular causes and non-vestibular causes of vertigo and dizziness at the bedside. This is especially true for acute prolonged vertigo or dizziness in association with nausea/vomiting, gait imbalance, motion intolerance and nystagmus (i.e., meeting the diagnostic criteria for an acute vestibular syndrome). Here the distinction between dangerous, potentially life-threatening central causes such as vertebrobasilar stroke and benign, self-limiting peripheral causes such as acute peripheral vestibulopathy is essential. Thus, based on the clinical examination skills demonstrated in this workshop the clinician should be able to decide which patients require an expedited diagnostic workup including neuroimaging and auxillary vestibular testing and which patient should be monitored on a stroke unit.

For the bedside examination of **vestibular** and **ocular motor** function the following five domains should be assessed (for a more detailed description see also [1, 2]): ocular stability for (I) nystagmus and (II) skew deviation, (III) the head-impulse test, , (IV) postural stability (including malleolar vibration sense), (V) ocular motor deficits (of saccades, smooth pursuit eye movements and optokinetic nystagmus).
Testing for possible benign paroxysmal positional vertigo (BPPV) is discussed in a separate session (Prof. Brandt and Prof. Straumann).

(I) Ocular stability → spontaneous or gaze-evoked nystagmus?

Normally the eyes are kept stable in the desired position. This is true both while looking straight-ahead and on eccentric gaze. However, damage to the peripheral or central structures of the vestibular or ocular motor system may result in eye drift ("slow phase"), usually interrupted by compensatory saccadic eye movements ("fast phase"), bringing the eyes back into the desired position. When evaluating such jerk spontaneous nystagmus (i.e., nystagmus elicited in neutral position), several elements should be assessed including the main beating direction of the nystagmus (horizontal vs. vertical vs. torsional), the effect of fixation and its suppression (typically a peripheral-type nystagmus can be suppressed on fixation - thus is increased on fixation suppression - while lacking visual fixation suppression favors a central cause) and its modulation depending on eye position. While predominantly horizontal spontaneous nystagmus (beating away from the lesioned side) is common in both peripheral and central vestibular disorders, vertical and torsional spontaneous nystagmus suggests a central origin.
In a next step the effect of gaze on the nystagmus should be assessed. Varying nystagmus intensity but unchanged beating direction of the fast phase is characteristic of peripheral-type spontaneous nystagmus, while a gaze-dependent changing beating direction (i.e., left beating on left gaze and right beating on right gaze, termed gaze-evoked nystagmus (GEN)) is of central origin (Figure 1). For vertical nystagmus a varying intensity depending on gaze can be depicted as well, with downbeat nystagmus becoming more intense on downgaze and upbeat nystagmus being most prominent on upgaze. Applying horizontal head shaking (at about 2Hz) or vibration to the mastoid bone (both with Frenzel's goggles on to suppress visual fixation) for approximately 10sec may show mostly horizontal nystagmus and thus is useful to identify

(compensated) asymmetries in the vestibulo-ocular reflex (VOR; e.g. in case of a chronic unilateral vestibular deficit), with the nystagmus beating away from the lesioned side. If the direction of the elicited nystagmus is vertical or torsional (termed "perverted nystagmus"), this points to a central pathology. Pendular nystagmus (i.e., nystagmus without a clear distinction between slow and fast phases) is typically of central origin.

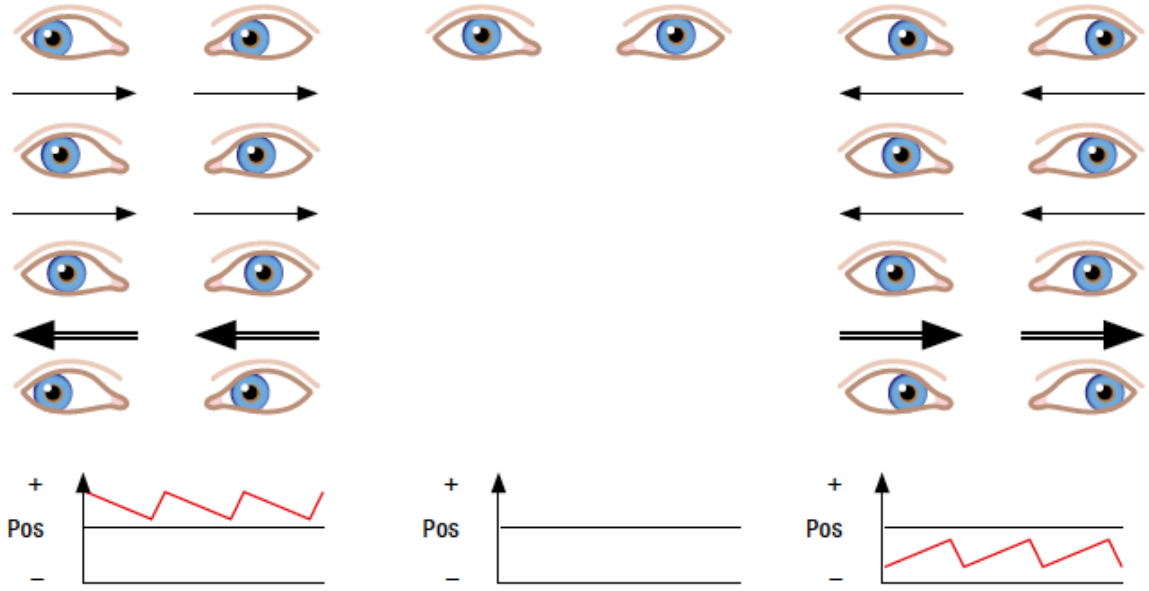

Figure 1: Testing for ocular stability at eccentric gaze. In healthy human subjects, gaze remains stable in eccentric position. In patients with brainstem or cerebellar deficits, the eyes may show centripetal drift (i.e., towards primary gaze, indicated by the thin arrows), which is then compensated by a centrifugal correction saccade (thick arrows), bringing the eyes back to eccentric position. This results in a jerk nystagmus, that changes its beating direction depending on direction of gaze, as shown in the bottom line. Source: [3].

(II) Ocular stability → skew deviation?

Vertical ocular drift on alternating cover test is another sign that should be tested at the bedside. Therefore, the patient is asked to fixate a small visual target (e.g. the tip of a pen). The examiner then looks for compensatory vertical movements of the eye that was just uncovered. Typically, one eye deviates upward and the other downwards on a pathological cover test. Presence of such vertical skew deviation (vertical divergence of the eyes) in the absence of a trochlear nerve palsy suggests a central origin and is part of the **H.I.N.T.S.** bedside examination (see below). Noteworthy, skew deviation may be accompanied by static binocular cyclorotation (no bedside testing possible) and head-tilt, a trias described as ocular-tilt reaction (OTR). An important differential diagnosis of vertical skew is a trochlear nerve palsy. For distinction, testing for vertical ocular deviations both in upright and supine position is recommended, as skew deviation modulates with head-orientation relative to gravity, becoming minimal in supine position, while in case of a trochlear nerve palsy the deviation of the eyes is body-position independent.

(III) Head-impulse test → testing the integrity of the vestibulo-ocular reflex (VOR)

The vestibulo-ocular reflex compensates for head-rotations and thus enables stable gaze on an object in space during head movements. Applying high acceleration, low-amplitude (10-15°) head impulses along the planes of the different semicircular canals allows a side-specific testing of the integrity of the VOR for a single semicircular canal at the bedside (Figure 2). Thereby the examiner asks the subject to fixate an object in space (usually the examiner's nose) while

applying the head rotations. Being one of the fastest brainstem reflexes (duration approx. 7-13msec), compensating for head rotation is finished before perceived by the human eye, thus for the examiner the subject's gaze remains stable. In contrast, interruptions of the VOR will result in a delayed correction for head rotation (triggered by retinal slip), thus such late (latency about 100msec) compensatory eye movements can be visually perceived as "catch-up saccades". It is important to apply the HIT with sufficiently high peak velocities (>150°/sec) in order to achieve a side-specific (ipsilateral) assessment of peripheral-vestibular function. The goal is to drive the frequency of the inhibitory vestibular nerve fibers of the contralateral labyrinth to zero to isolate the contribution of the excitatory pathway of the ipsilateral labyrinth. While testing the horizontal canals can be reliably performed with little training, assessing the vertical canals requires more practice and is often assessed only with video-oculography.
Note that absence of deficits in the efferent portion of the VOR (e.g. an abducens palsy) is a prerequisite for interpreting vestibular function by the HIT. While an abnormal HIT is a characteristic finding of peripheral vestibular deficits, also central lesions to the VOR may result in an abnormal HIT, referred to as "pseudoneuritis". Thus, the HIT alone does not allow a reliable distinction between peripheral and central causes and should be combined with other ocular motor signs. A three-step bedside testing battery called H.I.N.T.S. (**H**ead **I**mpulse, **N**ystagmus, **T**est of **S**kew) includes the head impulse test, testing for gaze-evoked nystagmus and skew deviation. This has been shown to distinguish peripheral from central causes with high sensitivity and specificity [4, 5]. Adding a fourth sign (ipsilateral new-onset hearing loss) even further increases the sensitivity of the H.I.N.T.S. (termed "H.I.N.T.S. plus") [6]. Furthermore, additional testing of the saccade and smooth pursuit systems increases the sensitivity for a central origin.

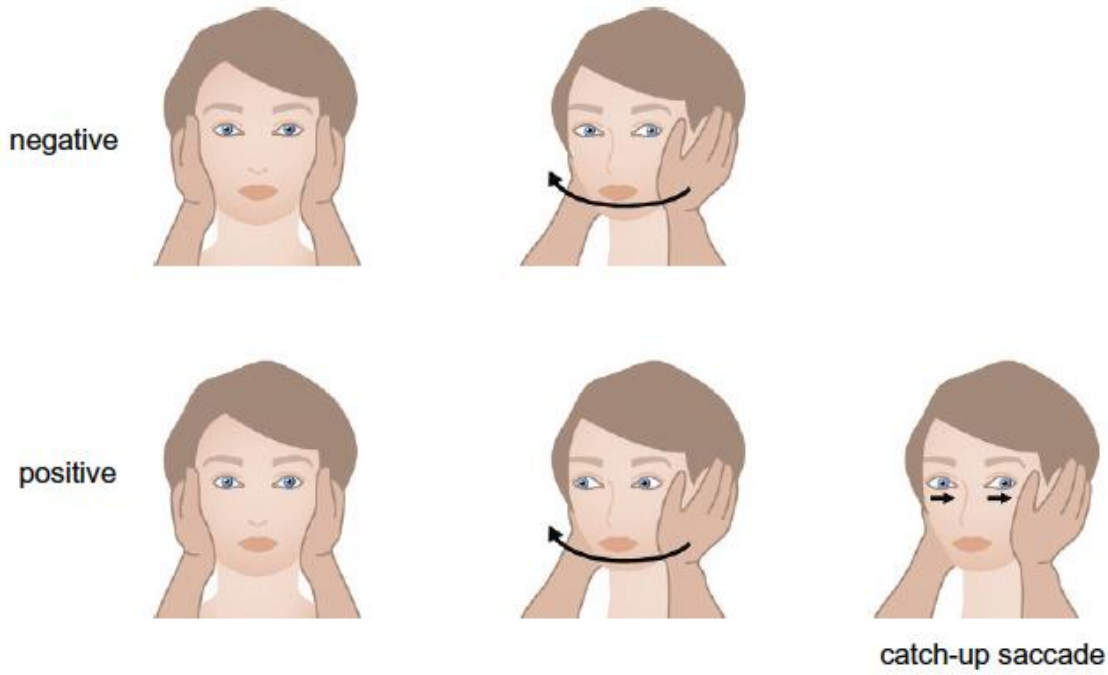

Figure 2: Head-impulse test (HIT) to the right, showing a **normal** (**negative**) response in the top row and an **impaired (positive)** HIT in the bottom row. While the eyes remain stable in space in case of a normal (negative) HIT, they deviate in the direction of the head rotation in case of an abnormal HIT, resulting in delayed (catch-up) saccades. Source: [3].

(IV) Postural testing including Romberg test

Postural control (with eyes closed) relies on both vestibular and proprioceptive input. Damage to peripheral or central vestibular pathways may impair postural control, resulting in body lateropulsion and imbalance. By asking the patient to stand with the feet together, hands by the side and with eyes closed (so-called Romberg test), postural stability is assessed. Sway on Romberg test indicates either vestibular or proprioceptive impairment (or may be functional if improving when distracting the patient). However, for the correct differential diagnosis looking for signs of peripheral neuropathy in the clinical examination is important (reflexes and sensory deficits including malleolar vibration sense).

(V) Eye movement testing for central signs

Shifting gaze onto an object of interest and subsequently keeping gaze on this object is provided by both vestibulo-ocular reflexes and visually-mediated reflexes. Applying slow (0.5 to 1Hz) head oscillations while the patient is fixating a stationary target evokes redundant activity of the VOR and the smooth-pursuit system. Thus, if the patient cannot keep the eyes on the target, both mechanisms are deficient. Targeted testing of the VOR is described further above.

For assessing the smooth pursuit system, the patient is asked to follow a moving target along either the vertical or horizontal plane with constant velocity (at about 20°/sec). If other eye movements (e.g. gaze-evoked nystagmus) interfere with testing of smooth pursuit testing, visual suppression of the VOR (also termed VOR-cancellation) can be used to assess the integrity of the smooth pursuit system while the eyes remain centered and are not moving. Therefore, the patient is asked to look at a head-fixed target (e.g. his/her thumb that is rotating at the same speed as the patient's head). Assuming an intact VOR, the inability to suppress nystagmus during head oscillations and simultaneous fixation of a head-fixed target suggests an impaired smooth pursuit system.

Optokinetic nystagmus can be triggered by a moving visual pattern or a hand-held rotating drum. It allows an assessment of the conjugacy of pursuit eye movements (slow phase of nystagmus) and saccades (fast phase of nystagmus).

For testing fast, saccadic eye movements, the patient is asked to shift gaze quickly between different earth-fixed targets (e.g. the examiner's nose and the tip of a pen). This is usually assessed in the horizontal and vertical plane. Saccades are then evaluated regarding their latency, velocity and accuracy. Slow, delayed or dysmetric (hypo- or hypermetric) saccades point to a central pathology, but have limited lesion localizing value.

Visual fixation may also be interrupted by saccadic intrusions or oscillations. This includes square-wave jerks, ocular flutter and opsoclonus and points to a central pathology.

Performing these five examination steps the sensitivity to find a central origin of vertigo or dizziness is even higher than that by the traditional three-step bedside examination of HINTS which was, indeed, better than early MRI [4].

References

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