

# Clinical diagnosis of bilateral vestibular loss: three simple bedside tests

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**Abstract:** Bilateral vestibular loss (BVL) may present with or without vertigo and hearing loss. Amongst the causes of BVL are vestibulotoxic antibiotics, autoimmune ear diseases, Menière's disease and meningitis. Clinical diagnosis of BVL is based on the result of three simple bedside tests: a positive head impulse test, reduced dynamic visual acuity and a positive Romberg test on foam rubber. With these signs, diagnosis of severe BVL is usually straightforward to establish.

**Keywords:** bilateral vestibular loss, dynamic visual acuity, head impulse test, vertigo

## Introduction

Patients with bilateral vestibular loss (BVL) may present with or without vertigo and hearing loss. They usually complain about oscillopsia during head movements and about unsteadiness, especially while walking in the dark [Dandy, 1941; Crawford, 1952]. Common causes of BVL are vestibulotoxic antibiotics (especially gentamicin), even after short periods of administration, autoimmune ear diseases such as Cogan's syndrome, Menière's disease and meningitis [Zingler *et al.* 2007]. BVL may also be associated with multiple system atrophy, neurofibromatosis type 2, hereditary ataxias such as Friedreich's ataxia [Fahey *et al.* 2008], spinocerebellar ataxia [Gordon *et al.* 2003] and episodic ataxia type 2. A combination of vestibular loss, peripheral neuropathy and bilateral vestibular areflexia is known as CANVAS syndrome [Szmulewicz *et al.* 2011].

Clinical diagnosis of BVL is based on the result of three simple bedside tests: a positive head impulse test (HIT) [Halmagyi and Curthoys, 1988], reduced dynamic visual acuity (DVA) during head shaking [Demer *et al.* 1994] and a positive Romberg test on foam rubber [Halmagyi *et al.* 1994]. With these three clinical signs, diagnosis of severe BVL is usually straightforward to establish but mild BVL remains a diagnostic challenge, as clinical tests might not be conclusive and data from laboratory tests, including caloric irrigation, rotational chair and video head impulse testing,

show considerable overlap between patients and normal subjects [Weber *et al.* 2009]. Vestibular outcome seems to be independent of age, gender, time course of manifestation and severity of BVL [Zingler *et al.* 2008]. Roughly 80% of patients with BVL do not improve and, thus, the prognosis seems less favourable than assumed previously [Zingler *et al.* 2008].

## Head impulse test

Owing to its short latency (about 10 ms), the vestibulo-ocular reflex (VOR) keeps the eyes on target during head movements with equal eye rotations opposite to the direction of head rotations [Aw *et al.* 1996]. The image is efficiently stabilized on the retina as long as eye and head velocities are equal. In BVL, the VOR is impaired or absent bilaterally and gaze is only stabilized by visual reflexes, which react with considerable delay, i.e. a latency of 100 ms or more [Viirre and Demer, 1996].

To perform a HIT, the examiner stands in front of the patient seated at the bedside or on a chair. The examiner asks the patient to focus on a target (e.g. the examiner's tip of the nose). The examiner then manually delivers brisk, passive horizontal head rotations of approximately 10–20° amplitude (Figure 1). Normally, the patient's eyes keep focusing on the examiner's nose. Instead, in a patient with BVL, where the VOR is impaired or

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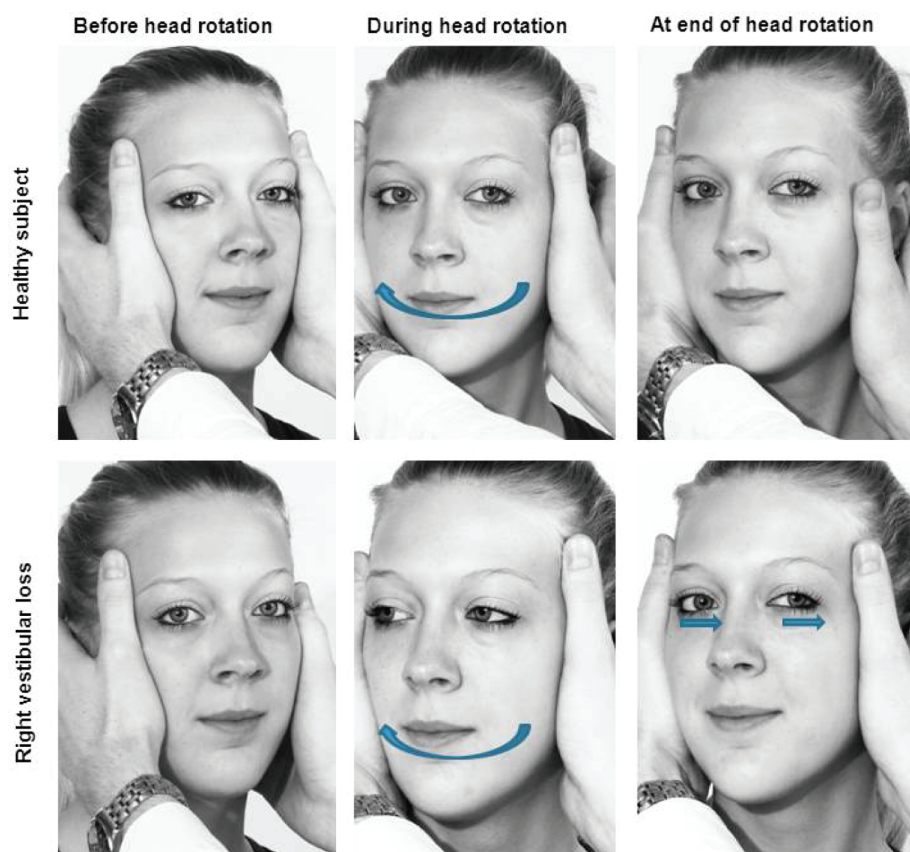
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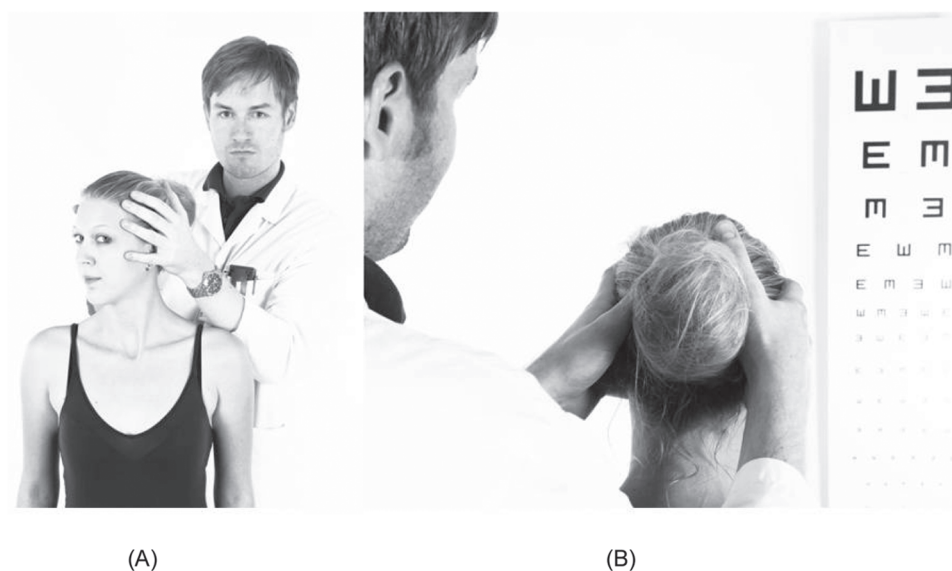
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**Figure 1.** Head impulse test. While the patient is asked to fixate on a target, the examiner briskly rotates the patient's head to the right or left and observes their eye movements. Top row: In a healthy subject, the vestibulo-ocular reflex will keep the eyes on the target. Bottom row: In a patient with bilateral vestibular loss (re-enacted scene), the eyes will move with the head during head impulses to both sides, so that the patient will have to make catch-up saccades after the head movements to bring the eyes back on target.



**Figure 2.** Dynamic visual acuity. (A) The examiner oscillates the patient's head at about 2 Hz in the horizontal or vertical plane. (B) The patient is asked to read the optotypes on a visual acuity chart while the head is moving. A patient with bilateral vestibular loss will typically lose three or more lines compared with static visual acuity.

absent, the eyes drift off the target to the side to which the head is rotated, so that the patient has to make a catch-up saccade to move them back to focus on the examiner's nose. Overt saccades after head rotation are the telltale sign of vestibular loss, whereas covert saccades during head rotation remain imperceptible to the naked eye [Weber *et al.* 2008]. While catch-up saccades appear with head impulses to either side in BVL, they indicate the affected side in patients with unilateral vestibular loss (UVL) whereas the patient's eyes keep focusing on the examiner's nose when the head is rotated to the healthy side.

Sensitivity of bedside HIT is adequate and therefore clinically useful in the hands of both neuro-otological experts and nonexperts [Jorns-Haderli *et al.* 2007]. In a group of subjects with UVL and BVL, its sensitivity was significantly lower (63% *versus* 72%) and its specificity was significantly higher for experts than nonexperts (78% *versus* 64%) when a quantitative HIT measurement with scleral search coils was used as a reference [Jorns-Haderli *et al.* 2007]. In other words, neuro-otological experts were inclined to trade off sensitivity for specificity of the HIT, in order to avoid false-positive results. In another study, the accuracy of the bedside HIT for identifying BVL was comparable with 84% sensitivity and 82% specificity [Schubert *et al.* 2004].

The bedside HIT can identify patients with severe and moderate BVL. However, identification of mild BVL remains a challenge and covert saccades may conceal BVL even in patients with total vestibular loss [Weber *et al.* 2009]. In cases of an inconclusive bedside test, it has now become practical to measure HIT with high-speed video goggles in order to quantify the deficit of the VOR and detect covert saccades [MacDougall *et al.* 2009].

### Dynamic visual acuity

DVA assesses a subject's ability to perceive objects accurately while the head is moved passively. To measure DVA clinically (Figure 2), the examiner oscillates the patient's head horizontally or vertically at 0.5–2 Hz and asks the patient to read optotypes on a visual acuity chart [Demer *et al.* 1994; Longridge and Mallinson, 1984]. In computerized DVA, optotypes are usually displayed during directional head impulses above a velocity threshold of 120–150°/s [Herdman *et al.* 1998; Vital *et al.* 2010]. In patients with BVL, the VOR is no longer able to stabilize gaze, while the



**Figure 3.** Romberg test on rubber foam. The patient is asked to stand on a mat of rubber foam with their feet together and eyes closed. Without visual and disrupted proprioceptive input, a patient with total bilateral vestibular loss will fall off the mat. The examiner must be ready to prevent the fall.

movements are too fast for the smooth pursuit system to keep the eyes on target. As a consequence, visual acuity decreases compared with

what the patient is able to read when the head remains still. In healthy subjects, visual acuity may decline by one or two lines on the optotype chart. A decline of more than two lines is considered abnormal [Fife *et al.* 2000; Sargent *et al.* 1997], but severely affected BVL patients may show a decline of five or more lines. Subjects with UVL may also have abnormal DVA, especially at higher head oscillation frequencies [Dannenbaum, 2009]. DVA based on directional head impulses also indicate the side of the lesion in UVL [Herdman *et al.* 1998; Vital *et al.* 2010]. DVA might display false-negative results when other mechanisms such as augmented cervico-ocular and visual reflexes compensate at least partially for the retinal instability during head movements [Chambers *et al.* 1985; Vital *et al.* 2010]. However, computerized DVA testing has good sensitivity (94.5%) and specificity (95.2%) in subjects with unilateral and bilateral vestibular loss [Herdman *et al.* 1998].

#### Romberg test on rubber foam

Postural control depends on visual, proprioceptive and vestibular input. In patients with BVL, postural control is impaired due to a loss of vestibulo-spinal reflexes [Horak *et al.* 2002]. A simple way of diagnosing ataxia is the Romberg test [Khasnis and Gokula, 2003]: the examiner observes postural stability with the patient placing his feet together, initially with eyes open and then with the eyes closed (Figure 3). The Romberg test is positive when a patient is able to stand with feet together and eyes open, but sways or falls with eyes closed [Lanska and Goetz, 2000]. However, this test is not specific for vestibular loss and does not help to distinguish between UVL and BVL, but also detects cerebellar and proprioceptive impairment. The test's sensitivity can be increased by having the patient stand on foam rubber, which disrupts proprioceptive inputs [Lanska and Goetz, 2000; Shumway-Cook and Horak, 1986]. Foam posturography with eyes closed proved to be very sensitive (up to 79%) and specific (up to 80%) to detect patients with both UVL and BVL [Fujimoto *et al.* 2009].

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#### Conflict of interest statement


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