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The Ten-Minute Examination of the Dizzy Patient

Joel A. Goebel, M.D., F.A.C.S., Department of Otolaryngology, Division of Head and Neck Surgery, Washington University School of Medicine, Saint Louis, Missouri.

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Abstract and Introduction

Abstract

Dizziness and postural instability are common presenting complaints in patients seen in otolaryngology practice. Unfortunately, these patients frequently do not receive optimal evaluation and therapy because of the difficulty in obtaining a thorough yet pertinent history and the perceived complexity of the physical examination. Over the last 10 years, we have utilized a standardized approach to evaluation of the dizzy patient that is user friendly and maximizes the time and effort spent by the clinician. First, a thorough history is taken from the patient in two forms: (1) a specially designed questionnaire filled out well before the examination and (2) a direct interview using the answers on the questionnaire as a guide. During the physical examination, emphasis is placed on the following subtests: (1) spontaneous nystagmus; (2) central oculomotor function; (3) the vestibulo-ocular reflex (VOR) battery -- headshake, head thrust, dynamic visual acuity, and Hallpike positioning; (4) coordination; (5) posture; (6) gait; and (7) special examinations. This neurotologic examination is completed in about 10 minutes and is performed as a battery of tests following the routine otolaryngologic and/or neurologic examination. We feel that this test sequence is thorough yet easy to perform and ideally will demystify the examination of these challenging patients.

Introduction

Objectives: On completion of this article the reader will be able to perform a thorough screening examination for patients presenting with dizziness, establish at the bedside the difference between central and peripheral mechanisms, and generate an appropriate differential diagnosis.

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The Dizziness Questionnaire and Patient Interview

Prior to the arrival of the patient in the office, the dizziness questionnaire is mailed to the patient's home to be filled out in detail and returned before the visit (Fig. 1). This questionnaire is divided into five main sections:

1. Description of the spell
2. Accompanying symptoms indicative of peripheral etiology
3. Accompanying symptoms indicative of central etiology
4. Accompanying auditory complaints
5. General physical and emotional health

For easy identification, "yes" and "no" options are placed in the left and right margins, respectively, and circled.

Description of the Spell

The hallmark of peripheral dizziness is the definite sensation of relative motion with the visual world, namely vertigo. The sensation is usually described by patients as a "spinning" or "whirling" feeling or the notion that they or their surroundings are moving in a circular fashion. In this first section of the questionnaire, the patient is asked to describe his or her sensation without using the word "dizziness." For most patients with peripheral labyrinthine disorders, the description is brief and very focused on vertigo. Patients with acute central nervous system (CNS) dysfunction may or may not have sensations of vertigo, whereas chronic CNS, cerebrovascular, cardiovascular, and metabolic causes of dizziness seldom produce true sensations of relative motion.

Symptoms Accompanying Peripheral Disease

Patients with peripheral vertigo have distinctive features of onset, duration, and accompanying symptoms in relation to their dizziness. Peripheral vertigo comes in spells and usually lasts seconds (benign positional vertigo), minutes (Ménière's disease), or hours (vestibular neuritis). Hearing loss, tinnitus, and aural fullness are frequent symptoms of peripheral disease. Position changes exacerbate the dizziness, and lying still lessens the symptoms. Benign positional vertigo, for instance, is highly suspected in cases of brief vertigo brought on by a simple position change such as rolling over in bed. In most attacks, the onset is sudden although the offset is less well defined. For the most part, patients feel fine between spells.

Symptoms Accompanying Central Nervous System Disease

Unlike peripheral vertigo, central causes of dizziness produce a more variable picture. The sensation may be described in a variety of ways: spinning, tilting, pushed to one side, lightheadedness, clumsiness, or even blacking out. If documented loss of consciousness is present, a peripheral etiology of the dizziness is rarely if ever at fault. Also helpful for localization is the presence of accompanying signs of neural dysfunction, that is, dysarthria, dysphagia, diplopia, hemiparesis, severe localized cephalgia, seizures, and memory loss. The time course of symptoms is more variable from minutes to hours, and the effect of movement or position change is less predictable. These symptoms lead the clinician to suspect brain stem or cortical rather than labyrinthine sources.

Accompanying Auditory Complaints

The single most useful localizing symptom in a dizzy patient is a unilateral otologic complaint: aural fullness, tinnitus, hearing loss, or distortion. By carefully evaluating these complaints, the clinician frequently can localize both the side and the site of the lesion before any examination or testing is done. Frequent causes of unilateral auditory disease with dizziness include endolymphatic hydrops, perilymphatic fistula, labyrinthitis, vestibular neuritis (slight high-pitched loss with tinnitus), and autoimmune inner ear disease.

General Physical and Emotional Health

Many medical conditions and emotional factors can create a sense of dizziness and imbalance. Hypertension, hypotension, atherosclerotic disease, endocrine imbalances, and anxiety states are common causes of lightheadedness, near syncope, and/or instability but rarely produce a sense of true vertigo. In addition, medication side effects and excessive caffeine, nicotine, and alcohol intake should be investigated as a source of dizziness. Ideally, these conditions have already been addressed by the patient's primary care physician before a referral for formal evaluation by a neurotologist or neurologist.

For further details on history taking in the dizzy patient, the reader is referred to the reviews listed in the reference section.^[1-4]

Performing the Physical Examination

After the history is complete, the clinician performs the routine full head and neck examination. This is important for two reasons: (1) dizzy patients frequently have other ear, nose, and throat pathology and (2) structural problems of the ear, nose, and throat at times cause dizziness or indicate a more widespread process. Common findings on the routine examination related to dizziness include cerumen impaction, otitis media with effusion, chronic otitis with otorrhea, chronic sinusitis with nasal airway obstruction, and

oropharyngeal findings consistent with sleep apnea. Of course, congenital deformities of the pinna, external auditory canal, and face raise the question of labyrinthine involvement.

At the conclusion of the regular examination, the specialized examination for dizziness is performed. Patients are tested with their glasses or contact lenses in place for best corrected vision. The sequence of the examination is as follows:

1. Spontaneous nystagmus
2. Gaze nystagmus
3. Smooth pursuit
4. Saccades
5. Fixation suppression
6. Head thrust
7. Headshake
8. Dynamic visual acuity
9. Hallpike positioning
10. Static positional
11. Limb coordination
12. Romberg stance
13. Gait observation
14. Specialized tests

Spontaneous Nystagmus

Action. Ask the patient to fixate on a stationary target in neutral gaze position with best corrected vision (glasses or contact lenses in place). Observe for nystagmus or rhythmic refixation eye movements. Repeat under Fresnel lenses to observe effect of target fixation.

Interpretation. If nystagmus is observed, particular attention is paid to the amplitude, direction, and effect of target fixation. Lesions of the labyrinth and nerve VIII produce intense, direction-fixed horizontal-rotary nystagmus that is enhanced under Fresnel lenses. The nystagmus also intensifies when gazing in the direction of the fast phase (Alexander's law). This pattern can be seen in both irritative (beating toward the affected ear) and destructive (beating toward the unaffected ear) lesions of the labyrinth, nerve VIII, or (rarely) the vestibular nuclei. In contrast, lesions of the brain stem, cerebellum, and cerebrum cause less intense, direction-changing horizontal, vertical, torsional, or pendular nystagmus that is diminished under Fresnel lenses. Examples include periodic alternating nystagmus (PAN), congenital nystagmus, and lesions of the midline cerebellum.

Gaze Nystagmus

Action. Ask the patient to gaze at a target placed 20 to 30 degrees to the left and right of center for 20 seconds. Observe for gaze-evoked nystagmus or change in direction, form, or intensity in spontaneous nystagmus.

Interpretation. The ability to maintain eccentric gaze is under control of the brain stem and midline cerebellum, particularly the vestibulocerebellum (especially the flocculonodular lobes). When these mechanisms fail to hold the eye in the eccentric position, the eye drifts toward the midline (exponentially decreasing velocity), followed by refixation saccades toward the target. Such gaze-evoked nystagmus is central in origin and always beats in the direction of intended gaze. In contrast, enhancement of peripheral spontaneous nystagmus (linear slow component velocity) occurs without direction change when gazing in the direction of the fast phase. Causes of gaze-evoked nystagmus include a drug effect (sedatives, antiepileptics), alcohol, CNS tumors, and cerebellar degenerative syndromes.

Smooth Pursuit

Action. Ask the patient to follow your finger as you slowly move it left and right, up and down. Make sure the patient can see the target clearly and you do not exceed 60 degrees in total arc or 40 degrees per second.

Interpretation. Normal eye tracking of a slowly moving discrete object generates a smooth eye movement that the examiner can easily see. Cerebellar or brain stem disease can cause saccadic eye tracking in which the patient repeatedly loses the target and then catches up with a small saccade. In most cases, abnormal pursuit is nonlocalizing within the CNS, although ipsilateral loss of pursuit can be ascribed to parietal lobe lesions. The examiner must make sure the patient can see the target and is attentive to the task.

Saccades

Action. Ask the patient to look back and forth between two outstretched fingers held about 12 inches apart in the horizontal and vertical plane. Observe for latency of onset, speed, accuracy, and conjugate movement.

Interpretation. Saccadic eye movements are refixation movements that involve the frontal lobes (voluntary saccades), brain stem reticular formation (voluntary and involuntary saccades), and oculomotor nuclei III, IV, and VI. Delayed saccades are seen in cortical and brain stem lesions, and slow saccades accompany brain stem disease. Inaccurate saccades (especially overshoots) are associated with lesions of the cerebellar vermis and fastigial nuclei. Finally, disconjugate eye movements with slowing of the adducting eye and overshoots of the abducting eye imply medial longitudinal fasciculus pathology frequently associated with multiple sclerosis.

Fixation Suppression Test

Action. Ask the patient to fixate on his or her own index finger held out in front at arm's length. Unlock the examination chair and rotate the patient up to 2 Hz while the patient stares at the finger moving with them. The examiner observes for a decrease in the visual-vestibular nystagmus that is evoked during rotation without ocular fixation.

Interpretation. The modulation of nystagmus invoked by rotation is a CNS phenomenon heavily dependent on the cerebellar flocculus. Failure of fixation suppression in the presence of adequate visual acuity implies floccular dysfunction. This test is similar in nature to the fixation suppression performed after caloric stimulation during electro-oculography.

Head Thrust Test (Head Impulse Test)

Action. Ask the patient to fixate on a target on the wall in front of the patient while the examiner moves the patient's head rapidly ($>2000 \text{ deg/sec}^2$) to each side. The examiner looks for any movement of the pupil during the head thrust and a refixation saccade back to the target (Fig. 2). Either direct observation of pupillary movement or the use of an ophthalmoscope is employed to document eye movement.

Interpretation. Introduced by Halmagyi and Curthoys^[5] in 1988, the head impulse test was described as a reliable sign of reduced vestibular function in the plane of rotation for the ear ipsilateral to the head thrust. The observation of eye movement during the maneuver is a sign of decreased neural input from the ipsilateral ear to the vestibulo-ocular reflex (VOR) because the contralateral ear is in inhibitory "saturation" and cannot supply enough neural activity to stabilize gaze. In such instances, the eye travels with the head during the high-velocity movement, and a refixation saccade is necessary to refoveate the target. Bilateral refixation movements are seen frequently in cases of ototoxicity.

Postheadshake Nystagmus

Action. Tilt the head of the patient forward 30 degrees and shake the head in the horizontal plane at 2 Hz for 20 seconds. Observe for postheadshake nystagmus and note direction and any reversal. Fresnel lenses are preferred to avoid fixation (Fig. 3). The maneuver may be repeated in the vertical direction.

Interpretation. Postheadshake nystagmus is considered a pathologic sign of imbalance in the vestibular inputs in the plane of rotation.^[6] In most instances, a peripheral cause is identified with the nystagmus directed toward

the stronger ear. A small reversal phase is sometimes observed. Signs of central etiology include prolonged nystagmus, vertical nystagmus following horizontal headshake ("cross coupling"), and disconjugate nystagmus.

Dynamic Visual Acuity

Action. Ask the patient to read the lowest (smallest) line possible on a Snellen eye chart with best corrected vision (glasses, contact lenses). Repeat the maneuver while passively shaking the patient's head at 2 Hz, and record the number of lines of acuity "lost" during the headshake.

Interpretation. Excessive retinal slippage during head movement is a sign of vestibular dysfunction. In the clinical examination, the most frequent etiology is bilateral vestibular loss related to ototoxicity or aging. Poorly compensated unilateral dysfunction can also cause loss of dynamic visual acuity but is harder to identify with this clinical test. It is important that the examiner shake the patient's head to avoid pauses during which the patient can see the target.

Dix-Hallpike Maneuver

Action. With the examination chair unfolded like a bed, turn the patient's head 45 degrees to one side while seated and rapidly but carefully have the patient recline. Observe the eyes for nystagmus and, if present, note the following five characteristics: latency, direction, fatigue (decrease on repeated maneuvers), habituation (duration), and reversal upon sitting up.

Interpretation. A positive maneuver is diagnostic for benign position vertigo, which is thought to be due to otoconial debris either floating (canalithiasis) or fixed (cupulolithiasis) within the posterior semicircular canal of the undermost ear. Characteristics of classical positioning nystagmus include geotropic torsional direction, brief latency (5 to 20 seconds), decline with repeated positioning, 30 seconds or less duration, and reversal upon arising. Atypical positioning nystagmus may imply either peripheral or central disease.

Positional Tests

Action. Ask the patient to lie still in three positions -- supine, left lateral, and right lateral -- for 30 seconds and observe for nystagmus. Use of Fresnel lenses is recommended.

Interpretation. The presence of a static positional nystagmus is nonlocalizing by itself and must be interpreted in the light of other physical findings. In general, however, vertical positional nystagmus is central in origin, implying cranial-cervical or fourth ventricle origin.

Limb Coordination Tests

Action. Ask the patient to perform a series of coordination tasks such as finger-nose-finger, heel-shin, rapid alternating motion, and fine finger motion (counting on all digits). Observe for dysmetria or dysrhythmia.

Interpretation. The presence of limb dysmetria or dysdiadochokinesia is a useful indicator of cerebellar cortical disease, which may or may not accompany midline or vestibulocerebellar oculomotor dysfunction.

Romberg Test

Action. Have the patient stand with feet close together and arms at the side with eyes open and then eyes closed. Observe for the relative amount of sway with vision present versus absent.

Interpretation. The Romberg stance is primarily a test of somatosensation and proprioception and not of vestibular input. Patients with compensated bilateral vestibular loss stand normally in both eyes-open and eyes-closed Romberg position because of adequate proprioception from the stable support surface. There are two ways, however, to make this test more sensitive to vestibular deficits -- tandem stance and 3-inch foam. In the tandem stance, the support surface cues are sufficiently altered that vestibular cues play a greater role in maintaining upright posture. Similarly, when the patient stands on a compliant support surface such as 3-inch foam, somatosensory cues are muted and vestibular cues become more important.

Gait Observation

Action. Ask the patient to walk 50 feet in the hall, turn rapidly, and walk back without touching the walls. Observe for initiation of movement, stride length, arm swing, missteps and veering, and signs of muscle weakness or skeletal abnormality (kyphoscoliosis, limb asymmetry, limp).

Interpretation. There is no such thing as a "vestibular gait." If a patient suffers an acute unilateral loss of otolithic function, the patient will tend to veer toward the side of the lesion. However, a variety of central brain stem and musculoskeletal lesions also produce lateral deviation during ambulation. Difficulties with gait initiation and turns and decreased arm swing can be seen in extrapyramidal disease. Gait ataxia implies cerebellar dysfunction and is distinctly different from gait deviation associated with uncompensated peripheral vestibular disease. Finally, exaggerated hip sway, rhythmic deviations, and an excessive reliance on touching the wall during walking may constitute signs of a functional gait disorder.

Specialized Tests

- **Tragal Compression, Pneumatic Otoscopy, Tullio Phenomenon, Valsalva With Pinched Nostrils And Closed Glottis**

Action. With Fresnel lenses in place, observe for nystagmus or tonic eye deviations with symptoms of dizziness under four test conditions: (1) steady tragal compression to increase pressure in the external auditory canal, (2) positive and negative pressure applied with the pneumatic otoscope, (3) presentation of loud tones via tuning fork or impedance bridge, and (4) increased pressure during breath holding against pinched nostrils or closed glottis.

Interpretation. Consistent eye deviations or nystagmus during any of the preceding maneuvers implies abnormal coupling between either the outside atmosphere or the intracranial space and the inner ear. This can occur with abnormal connections between the labyrinth and the middle ear or middle fossa at the following sights: oval window (fistula, excessive footplate movement), round window (fistula), lateral semicircular canal (fistula), and superior semicircular canal (dehiscence). In particular, eye elevation and intorsion with loud sounds or Valsalva maneuver against pinched nostrils is suggestive of superior canal dehiscence syndrome and has been described by Minor.^[7] In addition, cranial-cervical junction abnormalities (Arnold-Chiari malformation in particular) produce vertical downbeat nystagmus with any maneuver that increases intracranial pressure.

- **Fukuta Step Test Action.** Ask the patient to march in place with arms extended and eyes closed for 1 minute. Note the degree of lateral rotation at the end of the maneuver. **Interpretation.** Most normal subjects deviate less than 45 degrees in rotation to one side during the step test, whereas some patients with uncompensated unilateral dysfunction deviate more than 45 degrees toward the affected side. This finding alone, however, is not conclusive for otolith dysfunction.
- **Hyperventilation Action.** Ask the patient to take 20 deep breaths in and out in rapid succession, observe for nystagmus under Fresnel lenses, and record symptoms. **Interpretation.** Hyperventilation has two effects: (1) cerebrovascular vasoconstriction and (2) elevation of blood pH. Vasoconstriction causes lightheadedness and tingling of the hands and lips and may reproduce the symptoms of patients with hyperventilation syndrome or anxiety. More specifically, irritative nystagmus (toward the affected ear) secondary to elevated pH and increased eighth nerve firing is seen in lesions that affect the vestibular nerve such as petrous apex lesions, acoustic schwannoma, and eighth nerve demyelination.
- **Mastoid Oscillation Action.** Place a vibrating source on the mastoid tip and observe for nystagmus under Fresnel lenses. Note direction and waveform and effect of target fixation with removal of lenses. **Interpretation.** Mastoid oscillation acts as an excitatory stimulus to both labyrinths and, in some cases of asymmetry, produces a horizontal-rotatory nystagmus toward the stronger ear. In a sense, this nystagmus is similar in origin to that produced by the headshake maneuver.

Conclusions

1. A thorough history and structured oculomotor and posture-gait examination is crucial in the work-up of patients with dizziness and imbalance.
2. The test protocol mentioned in this review is a succinct yet comprehensive battery that can be added to the standard otologic or neurologic examination.
3. Laboratory tests for dizziness primarily play a confirmatory role following a complete history and examination of these patients.

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Reprint Address

Address for correspondence and reprint requests: Dr. Joel A. Goebel, Department of Otolaryngology, Division of Head and Neck Surgery, Washington University School of Medicine, 660 South Euclid Avenue, Box 8115, Saint Louis, MO 63110.
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