

Evaluation of the Dizzy Patient

Charleston Vestibular Course
November 6th, 2025
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Professor
Otolaryngology Head and Neck Surgery
Medical University of South Carolina

Changing What's Possible



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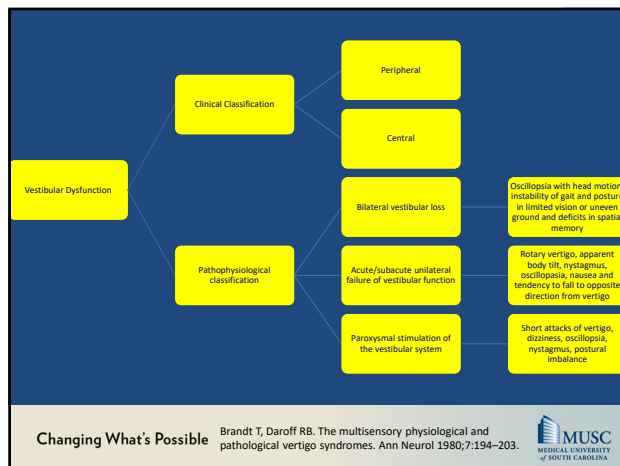
Disclosures

- Spiral Therapeutics
- Vestibular Disorder Association
- None of the above disclosures are relevant to the talk and course

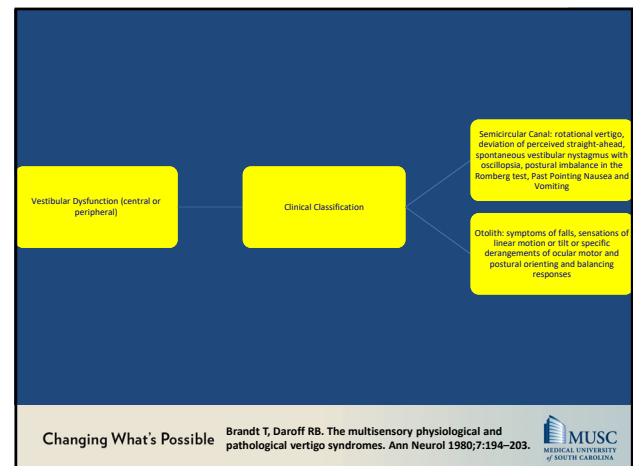
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3



4

Head Posture and Ocular Alignment

- Ocular Misalignment is frequent
- Ocular tilt reaction: triad of head tilt, ocular torsion and skew deviation
- Subjective visual vertical

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Huh YE, Kim JS. Bedside Evaluation of dizzy patients, J Clin Neurol, 2013;9: 203-213



5

Spontaneous Nystagmus

- Ask the patient to fixate on a stationary target in neutral gaze with best corrected vision.
- Repeat exam with Frenzel lenses to cancel fixation
- Description of the nystagmus:
 - Amplitude: grade I-III
 - Direction: horizontal? Vertical? Torsional?
 - Direction of quick phase
 - Fatigability
 - Effect of Fixation

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Goebel JA, The Ten-Minute Examination of the Dizzy Patient, Sem Neurol, 2001, 21: 391-398
Huh YE, Kim JS, Bedside Evaluation of dizzy patients, J Clin Neurol, 2013;9: 203-213



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Brandt T and Strupp M,
General Vestibular Testing,
Clin Neurophys, 2005, 116:
406-426

Measurement of head tilt:

An abnormal head posture to the right or left shoulder or a constant, abnormal tilt is especially observed in patients with (a) paresis of the oblique eye muscles, e.g. in superior oblique palsy, the head is turned to the non-affected side to lessen diplopia, or (b) an ocular tilt reaction due to a vestibular tonus imbalance of the VOR in roll. As a rule, the head is tilted to the side of the lower eye.

- Superior oblique palsy
- **CENTRAL LESIONS+++**
- Initial stage of vestibular neuronitis

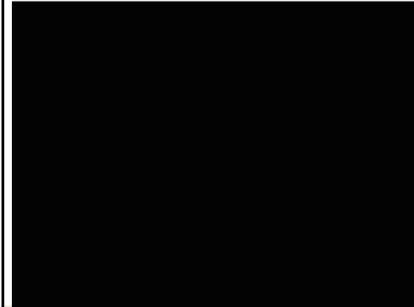


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Skew deviation



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Spontaneous Nystagmus

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Spontaneous Nystagmus

Peripheral lesion (labyrinth and CN VIII)

- Intense
- Diminished with fixation
- Direction fixed
- Horizontal-rotary
- Intensifies when gazes in direction of fast phase: ALEXANDER'S LAW

Central lesion (brainstem, cerebellum, cerebrum)

- Less intense
- Direction-changing or gaze paretic or gaze evoked
- Horizontal, vertical, torsional or pendular
- Can diminish without fixation
- Examples: Periodic Alternating Nystagmus, Congenital Nystagmus, Lesions of the midline cerebellum

*Goebel JA, The Ten-Minute Examination of the Dizzy Patient, Sem Neurol, 2001, 21: 391-398

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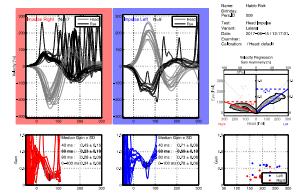
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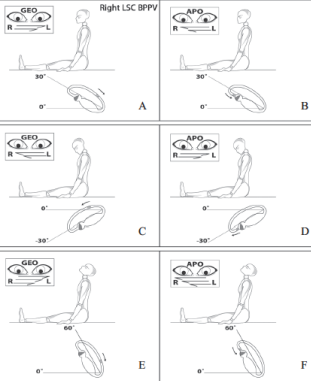
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Asprella-Libonati G, Lateral canal BPPV with Pseudo-Spontaneous Nystagmus masquerading as vestibular neuritis in acute vertigo: A series of 273 cases, J Vest Res, 2014, 24: 343-349

HEAD-PITCH TEST
Spontaneous nystagmus which changed its beating direction by tilting the head forward or backward is in favor of Horizontal Canal BPPV

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Huh YE, Kim JS, Bedside Evaluation of dizzy patients, J Clin Neurol, 2013, 9: 203-213

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Gaze Nystagmus

- Ask patient to gaze at a target 20-30 degrees from the center for 20 seconds
- Look for gaze-evoked nystagmus or change in direction, form or intensity in a spontaneous nystagmus

Goebel JA, The Ten-Minute Examination of the Dizzy Patient, Sem Neurol, 2001, 21: 391-398
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Gaze Nystagmus

Central Gaze evoked nystagmus

- Brainstem and Midline cerebellum (FLOCCULONODULAR LOBES=VESTIBULOCEREBELLUM): Eccentric gaze holding centers
 - If gaze holding fails, the eye drifts toward the midline followed by a refixation toward the target
 - Etiologies: Drug effect (sedatives, antiepileptics), alcohol, CNS tumors and cerebellar degenerative syndromes.

Peripheral cause

- Enhancement of peripheral spontaneous nystagmus (linear slow component velocity)
- No direction change

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Gaze Nystagmus

- Nucleus prepositus hypoglossi and medial vestibular nuclei: neural integrators for horizontal eye movements.
- Interstitial nucleus of Cajal is the neural integrator for torsional and vertical eye movement
- Flocculus/paraflocculus

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Smooth Pursuit

- Move fingers right and left and up and down without exceeding a velocity of 40 degrees per second and total arc of 60 degrees
- Saccadic eye movement suggests cerebellar or brainstem disease
- Abnormal pursuit is usually non localizing in CNS
 - Ipsilateral loss of pursuit is suggestive of ipsilateral parietal lesion
- *Patient can see the target and must be attentive to the task*

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Smooth Pursuit



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Smooth Pursuit

- Visual cancellation of the VOR allows to shift the direction of gaze during head motion
 - Smooth pursuit system allows it.
- Structures responsible of smooth pursuit and eye-head tracking are flocculus/paraflocculus, medial superior temporal area, frontal eye field, dorsal pontine nuclei

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Saccades

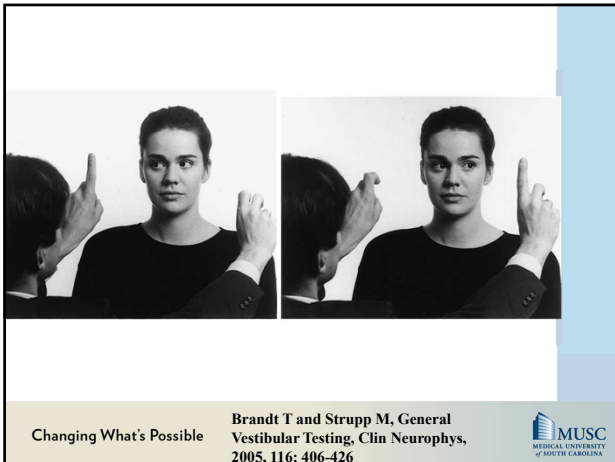
- Ask the patient to look back and forth between two outstretched fingers held 12 inches apart
 - Assess latency of onset, speed, accuracy and conjugate movement
- Frontal lobes: voluntary saccades
- Brainstem reticular formation voluntary and involuntary saccades
- Oculomotor Nuclei III,IV,VI involved in involuntary saccades

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Brandt T and Strupp M, General Vestibular Testing, Clin Neurophys, 2005, 116: 406-426



21

Saccades

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Saccades

- Delayed saccades
 - Cortical and brainstem lesions
- Overshoot saccades
 - Vermis and fastigial nuclei lesions (cerebellum)
- Disconjugate eye movement with slowing of the adducting eye and overshoots of the abducting eye imply MLF:
 - Multiple Sclerosis

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Fixation Suppression Test

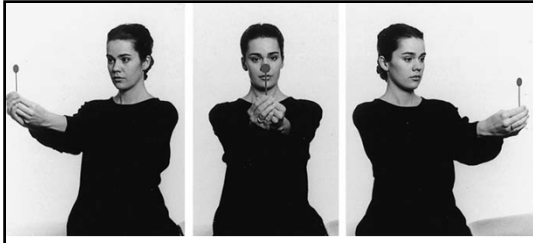
- Ask the patient to fixate his own finger held out at arm's length while the chair is rotating at a frequency of 2Hz
 - Look for a decrease in visual-vestibular nystagmus evoked during chair rotation without ocular fixation
- Cerebellar flocculus modulates this nystagmus

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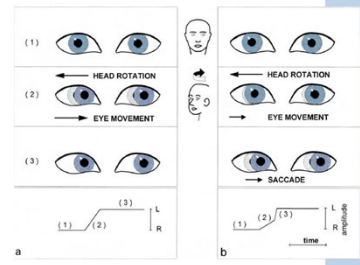
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Brandt T and Strupp M, General Vestibular Testing, Clin Neurophys, 2005, 116: 406-426

Clinical examination of the horizontal vestibulo-ocular reflex (VOR) by the head-impulse test (Halmagyi and Curthoys, 1988). To test the horizontal VOR, the examiner holds the patient's head between both hands, asks him to fixate a target in front of his eyes, and rapidly and arbitrarily turns the patient's head horizontally to the left and then to the right. This rotation of the head in a healthy subject causes rapid compensatory eye movements in the opposite direction (a). In cases of unilateral labyrinthine loss the patient is not able to generate the VOR-driven fast contraversive eye movement and has to perform a corrective (catch up) saccade to refixate the target. Part b explains the findings in a patient with a loss of the right horizontal canal. During rapid head rotations toward the affected right ear, the eyes move with the head to the right and the patient has to perform a refixation saccade to the left; the latter can be easily detected by the examiner.



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Head Thrust Test (bHIT)

- Patient fixates a target on the wall while you move the head rapidly to each side
 - Look for refixation saccades
- If there is a deficit in the VOR, the side to which the head is thrust has a weak vestibular input
- Bilateral positive bHIT is seen in ototoxicity

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Head Thrust Test (bHIT)

- Patient fixates a target on the wall while you move the head rapidly to each side
 - Look for refixation saccades
 - Some people use an ophthalmoscope to look for the papillary disk movement
- If there is a deficit in the VOR, the side to which the head is thrust has a weak vestibular input
- Bilateral positive bHIT is seen in ototoxicity

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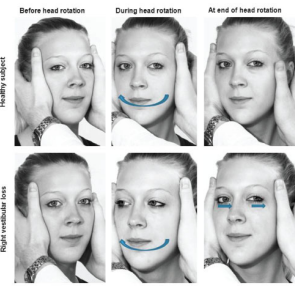
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Head Thrust Test (bHIT)**



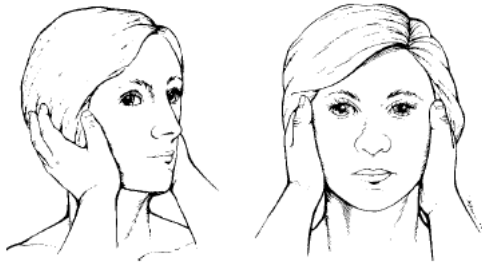
**Petersen JA, Straumann D and Weber K, Clinical Diagnosis of bilateral vestibular loss: three simple bedside tests, Ther Adv Neurol Disord, 2013,6: 41-45

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Head Thrust Test (bHIT)*



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*Goebel JA, The Ten-Minute Examination of the Dizzy Patient, Sem Neurol, 2001, 21: 391-398



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Head Thrust Test (bHIT)

- Patient should not know to which side you are moving the head to avoid anticipatory correction
- If patient has neck problems you can do it from the side to the center

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Goebel JA, The Ten-Minute Examination of the Dizzy Patient, Sem Neurol, 2001, 21: 391-398



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• H.I.N.T.S

- More sensitive than an MRI to predict a stroke in an Acute Vestibular Syndrome

Newman-Toker DE, Kattah JC, Talkad AV et al, H.I.N.T.S to Diagnose Stroke in the Acute Vestibular Syndrome-Three-Step Bedside Oculomotor Exam More Sensitive than Early MRI DWI, Stroke 2009, 40: 3504-3510

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HINTS

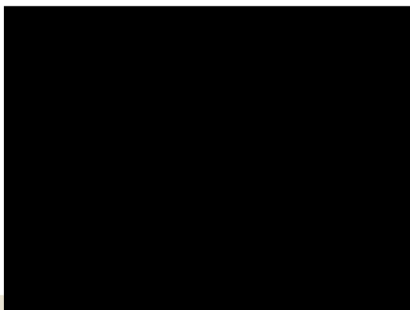
- horizontal-HEAD IMPULSE TEST
 - If negative: more likely central
- Gaze-Evoked Nystagmus
 - Does not follow Alexander's law
- Skew Deviation
 - Herald manifestation of basilar occlusion

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HINTS



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HINTS



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- Dangerous HINTS may be remembered with the acronym INFARCT:
 - Impulse positive Fast Phase Alternating, Refixation on Cover Test
 - 100% sensitive 96% specific for the presence of a central lesion

MRI DWI SENSITIVITY 72% SPECIFICITY 100%

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Postheadshake nystagmus

- Tilt head of the patient 30 degrees forward in horizontal plane and shake at 2 Hz for 20 seconds
 - Patient can keep eyes closed until you stop
 - They have to open their eyes and fixate a target
 - Explanation: Ewald's second law and velocity storage in centers.
- Observe for afterheadshake nystagmus and not direction and reversal
- Can be done in the vertical direction

Goebel JA. The Ten-Minute Examination of the Dizzy Patient, Sem Neurol, 2001, 21: 391-398

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Postheadshake nystagmus

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- Can be done in the vertical direction

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Postheadshake nystagmus

If afterheadshake nystagmus present it means there is an asymmetry in the VOR

Peripheral Cause

- Nystagmus is beating toward the stronger ear

Central origin

- Prolonged duration
- Vertical nystagmus after horizontal headshake (cross-coupling)
- Disconjugate nystagmus

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Huh YE, Kim JS. Bedside Evaluation of dizzy patients, J Clin Neurol, 2013;9: 203-213



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Discussion

- Head-Shaking Nystagmus
 - Shortcomings for lateralization
 - Detects ongoing vestibular, uncompensated lesion
 - Correlated to degree of caloric weakness
 - Sensitivity is affected by experience of clinician

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Discussion

- Head Impulse Test
 - Lateralizing test
 - Normal HIT with suspect peripheral lesion: think of a central lesion
 - Sensitivity is very dependent of clinician experience and of degree of weakness
 - Specificity was very high in this study 91-98%

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Dynamic Visual Acuity

Action

- Have the patient read the lowest line possible on Snellen eye chart
- Repeat while shaking the patient's head at 2 Hz and record number of lines of acuity "lost"

Interpretation

- Vestibular dysfunction will cause loss of foveal fixation
- 2 lines must be lost at least to consider it significant
- Most frequent etiology is
 - Bilateral ototoxicity
 - Aging
 - Poorly compensated unilateral dysfunction but it is harder to identify
- Passive head shake (done by examiner) rather than active done by patient

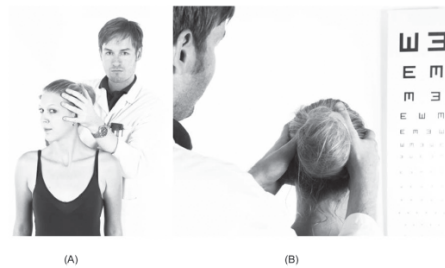
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Dynamic Visual Acuity



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Dix-Hallpike Maneuver

- Turn the patient's head 45 degrees to one side while seated and bring them rapidly back
- No need for hyperextension
- Look for nystagmus and note:
 - Latency
 - Direction
 - Fatigability
 - Habituation
 - Reversal upon sitting up

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Dix-Hallpike Maneuver

- Atypical positioning nystagmus may indicate a cupulolithiasis, multiple canal disease and it can be a central origin

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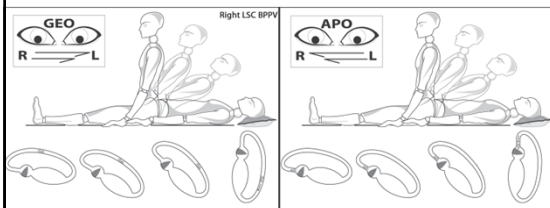
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SUPINE ROLL TEST

Direction changing horizontal nystagmus (geotropic or apogeotropic) in turning the head to either side while supine

Seated to Supine Test

When patient lies back HSC becomes vertical and otoliths are pushed toward utricle in GEO (BEATS TOWARD HEALTHY SIDE) form or float toward Ampulla in APO (beats toward the affected side)



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Asprella-Libonati G, Lateral canal BPPV with Pseudo-Spontaneous Nystagmus masquerading as vestibular neuritis in acute vertigo: A series of 273 cases, J Vest Res, 2014, 24: 343-349



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Horizontal BPPV

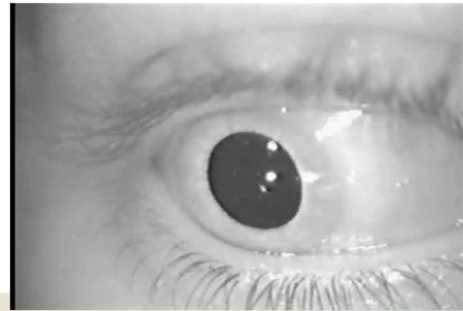


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Superior BPPV



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Posterior Canal BPPV



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Positional Nystagmus: When to look for important causes?

4 U. Büttner et al.

Acta Otolaryngol (Stockh) 119

Table 1. Clinical features of peripheral benign paroxysmal positioning vertigo/nystagmus (BPPV) and central paroxysmal positioning vertigo/nystagmus (central PPV)

Features	BPPV	Central PPV
Latency following precipitating positioning manoeuvre	1-15 sec (shorter in h-BPPV)	0-5 sec
Duration of attack	5-60 sec (longer in h-BPPV)	5- <60 sec
Direction of nystagmus	During stimulation in the plane of the affected canal: torsional/vertical for p-BPPV and a-BPPV; horizontal for h-BPPV	Pure vertical; pure torsional, not attributable to the stimulated canal plane
Fatigability	Typical, rare in h-BPPV	Possible
Course of nystagmus and vertigo in an attack	Crescendo-decrescendo typical, not common in h-BPPV	Crescendo-decrescendo possible
Vertigo	Typical	Typical
Nausea and vomiting	Rare on single precipitating manoeuvres (associated with intense nystagmus), not uncommon after several manoeuvres	Frequent on single precipitating manoeuvres (not necessarily associated with strong nystagmus intensities)
Natural course of the condition	Spontaneous recovery within several weeks in 70-80%	Spontaneous recovery within weeks possible
Associated neurological signs and symptoms	None	None possible, often cerebellar and other oculomotor signs
Brain imaging	Normal	Normal; lesions of the dorsal vermis and/or dorsolateral to the fourth ventricle

a = anterior, h = horizontal and p = posterior canal.

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Positional Nystagmus

• Aschan Types:

- Type I: persistent and changes direction in different head position
- Type II: transitory and direction-fixed
- Type III: BPPV transient: Some call it **Positioning** nystagmus



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HUGH O. BARBER, MD, Toronto, Ontario, Canada

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Positional Nystagmus: When to look for important causes?

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Ch

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Positional Nystagmus

- Type I and II can be peripheral or central
- Type I is either alcoholic positional nystagmus or central

Positional nystagmus Otolaryngol Head Neck Surg 1984, 92

HUGH O. BARBER, MD, Toronto, Ontario, Canada

Positional Vertigo and Nystagmus of Central Origin J Can Sc Neurol, 1981,8

P. WATSON, H.O. BARBER, J. DECK and K. TERNSTROM

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Other classifications: Brandt

- Positional nystagmus I or central positional nystagmus: lasts as long as the head remains in position. No vertigo.
- PN II or BPPV
- PN III central positioning nystagmus with vertigo or Pseudo-BPPV

Acta Otolaryngol (Stockh) 1999; 119: 1-5

Diagnostic Criteria for Central versus Peripheral Positioning Nystagmus and Vertigo: a Review

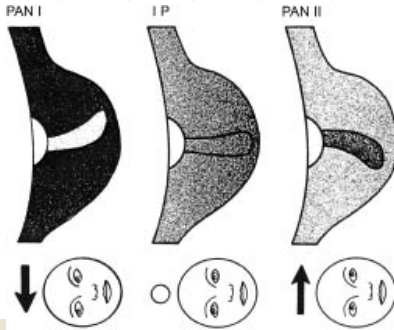
U. BÜTTNER, CH. HELMCHEN and TH. BRANDT
From the Department of Neurology, University of Munich, Munich, Germany

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Buoyancy Cupulopathy



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Canalolithiasis vs Cupulolithiasis

- Schuknecht 1969; Cupulolithiasis theory:
 - SCC becomes a linear acceleration sensor
- Imperfect theory: Brandt 1990
Canalit

Brandt T, Sieddin S (1993) Current view of the mechanism of benign paroxysmal positioning vertigo: cupulolithiasis or canalolithiasis? Vestib Res 3:373-382

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Eur Arch Otorhinolaryngol (2002) 259: 555-566
DOI 10.1007/s00405-001-0790-3

ORIGINAL ARTICLE

Cupulolithiasis of the horizontal semicircular canal

Hiroaki Ichijo

- Horizontal SCC Paroxysmal Positional Vertigo:
 - Geotropic
 - Persistent; Cupulolithiasis Head turned to healthy ear elicits more intense nystagmus
 - Transitory; Canalolithiasis Head turned to the affected ear elicits the symptoms
 - Apogeotropic Always Persistent Always Cupulolithiasis
 - Null position when head turned to the affected side by 10-20 degrees
 - Persistent and Apogeotropic Nystagmus should warrant strong suspicion of Central lesion!!!

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Table 2. Lateralization of benign paroxysmal positional vertigo involving the horizontal canal

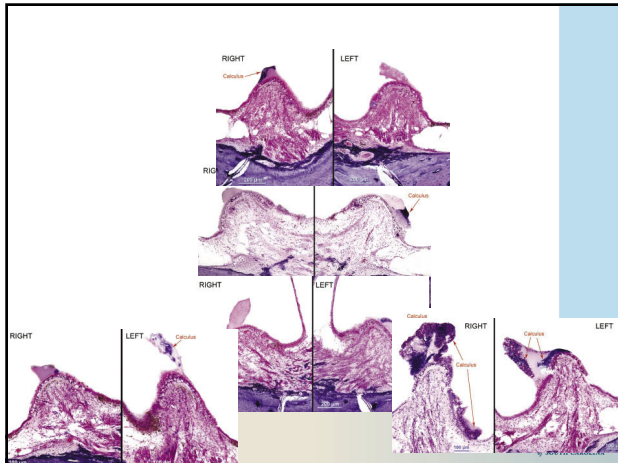
	Lesion side	
	Geotropic nystagmus	Apogeotropic nystagmus
Nystagmus intensity	Stronger side	Weaker side
Lying-down nystagmus	Contralateral	Ipsilateral
Head-bending nystagmus	Ipsilateral	Contralateral
Null point	Uncommon	Ipsilateral

Huh YE, Kim JS, Bedside Evaluation of dizzy patients, J Clin Neurol, 2013,9: 203-213

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Positional tests

- Patient will lie in three positions for 30 seconds
 - Supine
 - Left lateral
 - Right lateral
- Presence of a static positional nystagmus is nonlocalizing
 - A vertical positional nystagmus implies cranio-cervical lesion or IVth ventricle lesion

Changing What's Possible

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 Huh YE, Kim JS, Bedside Evaluation of dizzy patients, J Clin Neurol, 2013;9: 203-213



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Limb Coordination Tests

- Possible Maneuvers
 - Finger to nose
 - Heel-Shin
 - Rapid alternating motion
- Midline or vestibulocerebellar oculomotor dysfunction in presence of dysmetria or dysidiadochokinesia

Changing What's Possible

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Romberg test

- Look for sway with vision present and vision absent
- IT IS A TEST OF PROPRIOCEPTION It compares stability between eyes open and eyes closed

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Romberg test

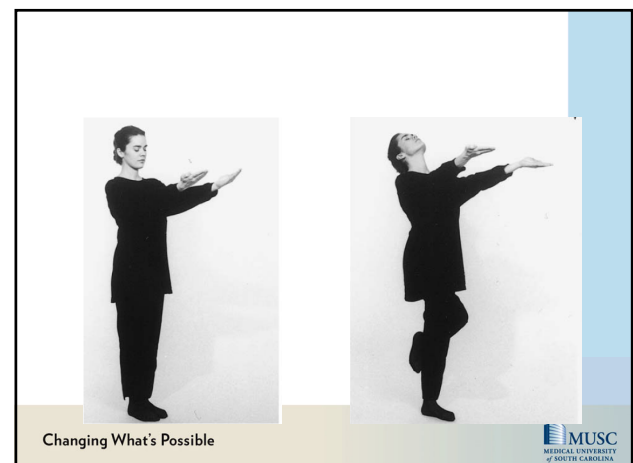
- Modified Romberg
 - TANDEM STANCE OR COMPLIANT FOAM
 - ALTERS PROPRIOCEPTIVE INPUT SUFFICIENTLY TO MEASURE MORE THE VESTIBULAR INPUT
- PASTPOINTING IS A BETTER TEST

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“——Romberg’s test, whose name is perhaps taken in vain more often than any other in medicine”—C. H. Edwards.

Romberg’s test is described on page 227 of the Sieveking translation and it is worth quoting in full:

“I have observed that anaesthesia of the muscles alone, without loss of tactile power, invariably accompanies tabes dorsalis. A simple experiment suffices to determine the fact. If the patient is told to shut his eyes while in the erect posture, he immediately begins to move from side to side and the oscillations soon attain such a pitch that unless supported, he falls to the ground. Even if the trunk is supported, if the patient be sitting and leaning against the back of a chair, the phenomenon takes place to the same extent and he will slip off the chair——. The eyes of such patients are their regulators or feelers”.

Romberg therefore stated that the patient will fall if asked to stand with his eyes closed and the test was described in the context of tabes dorsalis. In this latter condition, the long tracts which subserve proprioception are damaged while the tactile pathways are intact.

Changing What’s Possible



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Romberg on calibrated rubber foam

- Cancels proprioceptive stimuli and unmasks a vestibular weakness



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Petersen JA, Straumann D and Weber K, Clinical Diagnosis of bilateral vestibular loss: three simple bedside tests, Ther Adv Neurol Disord, 2013,6: 41-45

Changing What’s Possible



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Romberg on calibrated rubber foam

- Cancels proprioceptive stimuli and unmasks a vestibular weakness



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Changing What’s Possible



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Therapeutic Advances in Neurological Disorders

Review

Clinical diagnosis of bilateral vestibular loss: three simple bedside tests

Jens A. Petersen, Dominik Straumann and Konrad P. Weber

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- bHIT
- Dynamic visual Acuity
- Romberg on calibrated foam

Changing What’s Possible



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Gait Observation

- Observe initiation of movement, stride length, arm swing, missteps and veering and signs of muscle weakness or skeletal abnormality
- THERE IS NO VESTIBULAR GAIT
- Acute or uncompensated unilateral loss of otolith function: ipsilateral veering of gait

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Changing What’s Possible



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Gait Observation

- A variety of central brainstem, and musculoskeletal lesions cause lateral deviation
- Problems with gait initiation, turns and arm swing reflects extrapyramidal disease
- Gait ataxia implies cerebellar disease
- Exaggerated hip sway, rhythmic deviations, excessive touching of the wall: functional gait disorder

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Changing What’s Possible



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Disturbance of posture and gait control in peripheral vestibular disorders		
Illness	Direction of deviation	Pathomechanism
Vestibular neuritis	Ipsiversive	Vestibular tons imbalance due to failure of the horizontal and anterior semicircular canal and utricle (Strupp, 1999)
Benign paroxysmal positioning vertigo (BPPV)	Forward and ipsiversive	Amphibulge stimulation of the posterior canal due to canalolithiasis that leads to endolymph flow (Brandt and Stedtin, 1993; Brandt et al., 1994)
Attacks of Meniere's disease (Tumarkin's otolithic crisis)	Lateral ipsiversive or contraversive (falls)	Variations of the endolymph pressure lead to an abnormal stimulation of the otoliths and sudden vestibular-ocular reflex failure (Odkvist and Bergenius, 1980; Schuknecht and Gulya, 1983)
Tullio phenomenon	Backward, contraversive, diagonal	Stimulation of the otoliths by sounds of certain frequencies, e.g. in cases of perilymph fistulas or superior canal dehiscence syndrome (Tullio, 1927)
Vestibular paroxysmia	Contraversive or in different directions	Neurovascular compression of the vestibulo-cochlear nerve and excitation (rarely inhibition) of the vestibular nerve (Abusow et al., 1998; Brandt and Dieterich, 1994b)
Bilateral vestibulopathy	All directions, especially forward and backward	Failure of vestibular-spinal postural reflexes, exacerbated in the dark and on uneven ground (Rinne et al., 1995)

Changing What's Possible Brandt T and Strupp M, General Vestibular Testing, Clin Neurophys, 2005, 116: 406-426



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Disturbance of posture and gait control in central vestibular disorders		
Illness	Direction	Pathomechanism
Vestibular epilepsy (rare)	Contraversive	Focal seizures due to epileptic discharges of the vestibular cortex (Brandt and Dieterich, 1993b)
Thalamic ataxia (often overbroad)	Contraversive or ipsiversive	Vestibular tons imbalance due to posterolateral lesions of the thalamus (Mauden and Goeckel, 1988)
Ocular tilt reaction	Contraversive with mesencephalic lesions, ipsiversive with pontomedullary lesions	Torus imbalance of the vestibulo-ocular reflex in the roll plane with lesions of the vertical canals or otolith pathways (Brandt and Dieterich, 1993a)
Paroxysmal 'ocular tilt reaction'	Ipsiversive with mesencephalic excitation, contraversive with pontomedullary excitation or excitation of the vestibular nerve	Pathological excitation of the otolith or vertical canal pathways (VOR in the roll plane) (Dieterich and Brandt, 1993b)
Lateropulsion (Wallenberg's syndrome)	Ipsiversive, diagonal	Central vestibular tons imbalance ('roll and yaw planes') with tilt of subjective vertical (Dieterich and Brandt, 1992)
Downbeat nystagmus syndrome	Backward	Vestibular tons imbalance in the 'pitch plane' (Brandt and Dieterich, 1995)

Changing What's Possible Brandt T and Strupp M, General Vestibular Testing, Clin Neurophys, 2005, 116: 406-426



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Tragal Compression, Pneumatic otoscopy, Tullio Phenomenon, Valsalva with pinched nostrils and closed glottis

- Observe for tonic eye deviations or nystagmus with symptoms of dizziness
- This helps detect a perilymphatic fistula or a third window syndrome

Changing What's Possible Goebel JA, The Ten-Minute Examination of the Dizzy Patient, Sem Neurol, 2001, 21: 391-398



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Tragal Compression, Pneumatic otoscopy, Tullio Phenomenon, Valsalva with pinched nostrils and closed glottis

- EYE ELEVATION WITH INTORSION WITH LOUD SOUNDS OR VALSALVA IS SUGGESTIVE OF SSCD
- ARNOLD CHIARI MALFORMATION OR OTHER CRANIOCERVICAL ABNORMALITIES CAN HAVE DOWNBEAT NYSTAGMUS WITH ANY INCREASE IN ICP

Changing What's Possible Goebel JA, The Ten-Minute Examination of the Dizzy Patient, Sem Neurol, 2001, 21: 391-398



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FUKUDA Step Test

- Ask the patient to march in place with arms and eyes closed for one minute
 - Note degree of lateral rotation
- Normal subjects deviate less than 45 degrees
- Some uncompensated unilateral vestibular dysfunction Deviation more than 45 degrees
 - Inconclusive

Changing What's Possible Goebel JA, The Ten-Minute Examination of the Dizzy Patient, Sem Neurol, 2001, 21: 391-398
Fukuda T, The Stepping Test, Two phases of the Labyrinthine reflex, Acta Otolaryngol, 1959, 50: 95-108



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Fukuda T, The Stepping Test, Two phases of the Labyrinthine reflex, Acta Otolaryngol, 1959, 50: 95-108

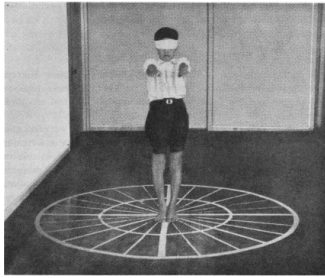
- Unterberger Tretversuch test 1938
- Hirsch Waltzing test 1940
- These were based on gait modification after caloric stimulation
- Fukuda describes in 1959 two tests relative to two stages of labyrinthine responses: Stage of coordination for vertical writing with eyes covered test and Stage of disturbance for the stepping test.

Changing What's Possible



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Fukuda T, The Stepping Test, Two phases of the Labyrinthine reflex, Acta Otolaryngol, 1959, 50: 95-108



Changing What's Possible



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Hyperventilation

- 20 deep breaths in rapid succession and observe for nystagmus
- Hyperventilation causes
 - Cerebrovascular vasoconstriction Can reproduce symptoms if patient is having dizziness and lightheadedness related to hyperventilation syndrome or anxiety
 - Elevates blood pH: irritation of VIIIth nerve and irritative nystagmus if petrous apex lesion, acoustic schwannoma or other lesion causing demyelination of VIIIth nerve

Changing What's Possible

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Mastoid Oscillation

- Vibration source on mastoid tip and observe for nystagmus noting direction, waveform and effect of fixation
- Oscillation stimulates both labyrinths
- If there is an asymmetry it can produce horizontal-rotatory nystagmus toward the stronger ear: similar to afterheadshake nystagmus

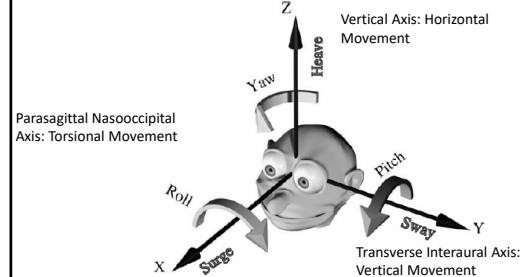
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Huh YE, Kim JS, Bedside Evaluation of dizzy



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How to name eye movements?

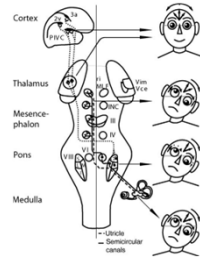


Changing What's Possible



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Brandt T, Dieterich M. Skew deviation with ocular torsion: a vestibular brainstem sign of topographic diagnostic value. Ann Neurol 1993a;33:528-34.



- Yaw Plane Signs:
 - Horizontal Nystagmus
 - Past-pointing
 - Rotational and Lateral Body Falls
 - Horizontal Deviation of perceived straight-ahead
- Roll Plane Signs:
 - Torsional Nystagmus
 - Skew Deviation
 - Ocular Torsion
 - Tilts of the head, body and perceived vertical (ocular-tilt reaction)
- Pitch Plane Signs:
 - Upbeat or downbeat nystagmus
 - Forward or backward tilts and falls
 - Vertical deviation of the perceived straight-ahead

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