Differential Diagnosis of the Dizzy Patient

Determining vestibular and nonvestibular causes of dizziness

1. The Dizzy patient

a. Dizziness is one of the most common patient complaints, affecting an estimated 20-30% of the population. [1] One study found that 7.5 million patients with dizziness are examined in ambulatory care settings in US each year. [1]

b. "Dizziness" is an imprecise term that can mean different things to different people and therefore can have many different underlying pathophysiologic mechanisms [Figure 1]. [2, 3]

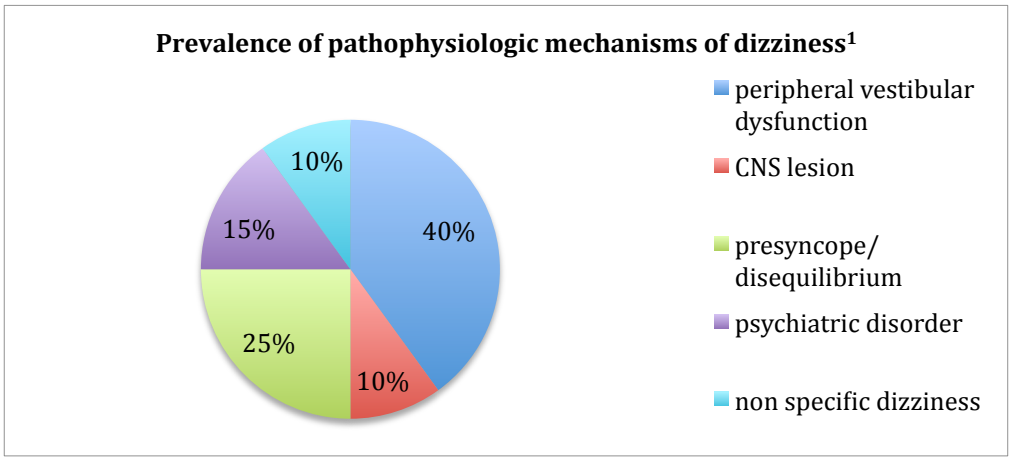

Figure 1.

c. When a patient reports dizziness it is important to determine if it is of vestibular or nonvestibular origin. True vertigo is most likely due to vestibular dysfunction whereas nonvertigo can be do to a variety of CNS, cardiovascular or systemic diseases. [1]

d. If the dizziness is vestibular in nature, one must differentiate between peripheral and central causes because several central causes require immediate medical attention. This second differentiation process is beyond the scope of this document.

2. **Patient History** is the most important part of the evaluation of a dizzy patient. [2, 4]

a. It can be a tedious process. A questionnaire can be used prior to seeing patient to reduce time discussing parts of history that will not assist in differential diagnosis or treatment of patient. (Refer to supplementary document 1 for questionnaire [5] developed by James Cavanaugh, PT, NCS or access questionnaire created by Tim Hain, MD available at: http://www.dizziness-and-balance.com/practice/resources/questcdh2007b.pdf)

b. The tempo, symptoms, circumstances of dizziness should guide patient history. [3, 6] (Refer to supplementary document 2, a basic chart I developed as general overview for classification of dizziness using history. The flowchart is just a guide thought process and is not intended to be used for diagnosis!)

i. **Tempo**: temporal characteristics of symptoms have been shown to be more effective than symptom quality in differentiating forms of dizziness. [7]

Is it acute, chronic, or does it occur in spells?

1. First onset of <u>less than 3 days</u> most likely to be due to acute vestibular neuritis or labyrinthitis. [3]

2. <u>Chronic dizziness</u>
   a. > 3 days could indicate bilateral vestibular, mal de debarquement, oscillopsia, anxiety or depression [3]
   b. >7 days could indicate unilateral vestibular deficit [6]
   c. If relieved by sitting or lying down could indicate disequilibrium [6]

3. <u>Episodes</u> of dizziness can occur in many time courses:
   a. Seconds: Benign proximal positional vertigo [3, 6, 8]
   b. Seconds-minutes: Orthostatic hypotension, vasovagal attacks, cardiac arrhythmias, anxiety [3, 5, 6, 8]
   c. Minutes: Transient ischemic attach (TIA), migraine, panic attack [3, 5, 6, 8]
   d. Hours: Meniere's syndrome, viral neurolabyrinthitis, motion sickness, migraine [1, 6, 8, 9]

4. Ask patient about onset of dizziness (inciting factors, duration and severity). Often if of peripheral cause it is severe enough to remember whereas central vestibular causes often present with more insidious onset. [4, 5]

ii. **Symptoms:** define and expand upon what the patient means by dizziness. [8]
Is it vertigo?

1. Ask patient to describe dizziness using words other than dizzy, but keep in mind that some patients may use incorrect descriptors. [2]

2. Common descriptors and associated pathology:
   a. Impending faint, unsteadiness, falling: <u>pre-syncopal lightheadedness</u> [8]
   b. Spinning of environment or self, motion sick, or tilting: <u>vertigo</u> [8]
   c. Off balance, clumsy, swaying, or unsteady when walking: <u>disequilibrium</u> [8]
   d. Giddy, floating, swimming, rocking, fogginess, or fuzziness: <u>psychogenic, pharmacologic or ill-defined mechanisms</u> [5, 10]

3. Ask if dizziness has a component of movement or not. Vestibular dysfunctions will and non-vestibular typically will not. [10]

4. A feeling of imbalance or unsteadiness <u>un</u>related to abnormal head sensation is most likely nonvestibular. [3, 6]

5. Other symptoms besides dizziness (e.g. nausea and vomiting, vertical diplopia, tinnitus, hearing loss) can be indicative of vestibular vs non-vestibular etiologies. [5]

iii. **Circumstances.** What triggers or exacerbates symptoms often gives information about mechanism behind dizziness. [3]

1. Spontaneous onset could indicate: acute vestibular neuritis, Wallenberg infarct, TIA, labyrinthitis, mal de debarquement, oscillopsia (when eyes open), panic attack, Meniere's disease. [3]

2. If triggered by various types of movement could indicate:
    a. Head movement: acute vestibular neuritis, labyrinthitis, migraine [3]
    b. Body movement: migraine, motion sickness [3]
    c. Eye movement (with head still): anxiety or depression [3]
    d. Postural changes: BPPV, orthostatic hypotension, disequilibrium [3]

3. Do symptoms cluster during specific time of day?

c. System Review: the most frequent abnormalities seen in patients with dizziness are bolded.

i. General: fever, chills, fatigue, sleep disturbance, unexplained weight loss, appetite change [5]
ii. Skin: abnormal coloring, texture, hair growth, new moles, lesions [5]
iii. **Eye:** inflammation, pain, **diplopia, recent change in visual acuity,** new glasses or contacts. [5] Bifocals and trifocals can cause chronic disequilibrium. [4] Vertical diplopia is commonly due to skew eye deviation from peripheral or central otolith dysfunction. [6]
iv. **Ear: hearing loss** (especially unilateral or unexplained), aural pressure, **tinnitus (**which may be indication of inner ear disease). [1, 5]
v. **Cardiopulmonary: palpitations, syncope, chest pain/discomfort/pressure, leg swelling, dyspnea, orthpenea**, sputum, congestion [5]
vi. **GI: nausea, vomiting, dysphagia,** pain, constipation, diarrhea, bleeding [5]
vii. MSK: pain, stiffness, swelling, joint limitation (especially cervical) [5]
viii. Hematologic: bruising, bleeding [5]
ix. **Neurological:** Dizziness is often associated neurological symptoms: [5]
    1. MS, cerebrovascular disorders affecting posterior circulation (cerebellar infarct, hemorrhage or vertebrobasiliar insufficiency), migraines, seizures disorders, meningitis, and peripheral neuropathies all can have dizziness as symptom. [3]
    2. Diplopia, dysarthria, dysphagis and focal, and motor/sensory weakness indicates CNS ischemia. [1]
    3. Ataxic gait and dysdiadochokinesias indicate cerebellar diseases. [1]
    4. Concomitant complaints of headaches may indicate migraine related dizziness. [1, 5]
x. Psychological: history of psychiatric screen or treatment [6]

xi. History of traumas (which can damage labyrinthine membranes), migraines, ear disease, ear surgery, psychiatric screens/treatment, cervical dysfunction or pain, and/or substance abuse. [8] Patients post concussion (MVA, falls, athletics) often present with vestibular complaints. [11]

d. **Medication review**: ask for complete list of prescription, over-the-counter, herbal medicines.

   i. Polypharmacy is common cause of dizziness in elderly patients. [5]
   ii. Medications can cause dizziness due to effect on peripheral vestibular. Some of these include: aminoglycosides, alkylating agents and cyclophosphamide, aspirin, NSAIDs, loop diuretics, and quinines. [4, 5]
   iii. Vestibulotoxity: Aminoglycoside antibiotics can destroy the auditory and vestibular hair cells causing bilateral vestibular loss. [4]
   iv. Medications can cause central vestibular dysfunction with symptoms of nonspecific disequilibrium, fogginess, and/or giddiness. Some of these include: Antihypertensives, quinolones, neuroleptics, antidepressants, sedatives, anticonvulsants. [4, 5]
   v. Vestibular suppressants (e.g. Meclizine hydrochloride and fluoxetine hydrocloride, benzodiazepines) used to decrease symptoms can suppress the vestibular system thereby prolonging symptoms and reducing or delaying effectiveness of vestibular rehabilitation. [4, 11, 12]

3. True Vertigo

a. Definite rotational sensation, patient feels that he/she or the environment is spinning. [10]
b. Tends to be episodic; occurs due to sudden imbalance of tonic neural activity generated from the labyrinth. [6]
c. Vertigo often has incapacitating effect on patient, they are bed-bound at least in beginning with mild nausea, vomiting and anxiety. [10]
d. Common causes of vertigo: BPPV is by far the most common cause (from displaced otoconia from otolith organs into SCCs), acute stage of unilateral vestibular lesion, labyrinthitis, ear infections, Meniere's disease, acute brainstem lesions that affect the root entry zone of peripheral vestibular neurons of vestibular nuclei, and vertebrobasiliar insufficiency (TIA and various infarcts). [8, 13]
e. Oscillopsia: visual hallucination that objects in visual surrounding are moving (only occurs with eyes open). [13] Occurs secondary to vestibular system not generating adequate compensatory eye velocity. [13] Two types: spontaneous and head-induced.
f. Vestibular hypofunction during head movements is a common cause of oscillopsia.

4. Nonvertigo: symptoms usually lack rotary component of movement of either self or surroundings and do not have associated symptoms of nausea and vomiting. [10]

a. **Presyncope**
   i. Syncope is a loss of consciousness due to decreased blood supply that causes disruption of oxygenation of brain. [1]
   ii. Presyncope is more common than syncope. [10] It is the sensation of fainting or near fainting due to momentary loss of blood supply to brain (hypotension). [1, 10]
   iii. Patient may describe it as light-headedness, visual blurring or dimness, diaphoresis, becoming pale, and recovery upon assuming recumbent position. [10]
   iv. If onset of faintness is gradual and is not relieved by lying down it could indicate hypoglycemia or other metabolism disorders. [10]
   v. Sudden alteration of consciousness to dreamy states could indicate temporal lobe seizures. [10]

vi. Common causes of presyncope include: orthostatic hypotension, decreased cardiac output due to cardiac arrhythmias, heart conditions (severe hypertension, recurrent MI, congestive heart failure, severe valvular disease, and peripheral vascular disease), psychogenic disorder (e.g. anxiety), hyperventilation, anemia, hypo and hyperglycemia, vasovagal attacks/syncope and medications. [8, 10]

b. **Disequilibrium**
   i. Sense of unsteadiness, instability and loss of balance involving leg or trunk secondary to a disorder in motor system control. [1, 10]
   ii. If dizziness sensation is not related to head movement. [10]
   iii. Occurs only when a patient is walking or standing and diminishes with sitting down. [6, 10]
   iv. Common causes include: peripheral neuropathy, chronic vestibular disorder, musculoskeletal disorder, motor or cerebellar lesions, gait disorder, Parkinson's disease, decreased or double vision or proprioceptive loss, somatosensory loss and weakness in LEs, and multiple sclerosis. [1, 8, 10, 13]

c. Mal de debarquement
   i. Sensation of rocking or swaying as if on a ship occurring after long sea or air trip. Thought to be secondary to vestibular system's adaptive process to the motion during extensive travel. [6]

d. Motion sickness
   i. Episodic dizziness, diaphoresis, salivation, nausea and occasional vomiting from passive locomotion or motions occurring around person while they stand still. [6]
   ii. Due to sensory mismatch between visual and vestibular cues. [6]

e. Non specific
   i. Patients who have difficulty articulating exact symptom and have no definite illness, "chronic subjective dizziness". [10]
   ii. Vague and subjective lightheadedness, faintness, hypersensitivity to motion cues in absence of vestibular deficits, or disequilibrium. [10]
   iii. Common causes include: psychiatric disorders such as depression, generalized anxiety disorder, panic or phobic disorder, conversation disorder, or obsessive-compulsive disorder), hyperventilation, multiple sensory impairments (secondary to peripheral neuropathy, cervical spondylosis or age related degeneration). [1, 6, 10]

5. Clinical examination components (comprehensive list included below, **bolded** most commonly tested)

a. Subjective reported dizziness:
   i. Dizziness Visual Analog scale (VAS) [14], Modified Borg Rating scale for Dizziness (refer to supplementary document 3), Vertigo symptom scale (VSS) [15]. These are quick, easy assessment that can be completed with each treatment follow-up to assess symptom improvement. [14]
   ii. Subjective visual verticality (SVV): in sitting, patient opens eyes and positions 20cm line in self-perceived vertical. [16] Deviation of 2 or more degrees from true vertical indicates vestibular asymmetry. [16]

b. If patients experiences dizziness during exam/test ask patient if it is "their dizziness", as some may cause dizziness of a different nature. [10]

c. General assessment of patient

- **i.** Signs of CVA or tumor (facial asymmetries, dysarthria, hoarseness) [4]
- **ii.** **Vital signs**: evaluate for orthostatic hypotension, orthostatic intolerance [4]
- **iii.** Peripheral pulse: evaluate tachycardia, bradycardia or arrhythmias [4]
- **iv.** Use of assistive device, glasses [4]
- **v.** **Hearing**: Self-report of deficits of changes. Can utilize tuning fork or complete a Weber and Rinne examination [4]

d. Cardiopulmonary tests: testing for precyncope

- **i.** **Heart rate**
- **ii.** Pulse oximetry to check arterial oxygen saturation levels, static and dynamic [5]
- **iii.** Self-report of valsalva or perform potentiated valsalva maneuver: patient squats for 30 second, then stands and strains against a closed glottis [10]
- **iv.** Hyperventilation: patient breathes deeply for three minutes, if symptoms present, asked if it is "their dizziness" [10]

e. Neuromuscular tests: visual perception and oculomotor tests

- i. Acuity (Snellen eye chart), diplopia, extraocular movements (CN III, IV, VI), visual field, visual neglect [5, 6]
- ii. **Smooth pursuit, saccades** (monitor latency, speed and accuracy), convergence (look for symmetry), response to optokinetic stimulation. [5, 6]

f. Vestibulo-ocular tests

- **i.** **Nystagmus**: spontaneous, gaze evoked (gaze held 30 degrees off center), head shaking (use Frenzel lenses or infrared goggles to test without fixation) [3, 5, 6]
  1. **1.** Test spontaneous nystagmus with and without fixation because peripheral causes of nystagmus can be suppressed with fixation.
  2. **2.** Normal: no nystagmus in light or dark environments.
  3. **3.** Nystagmus is described by direction of the quick phase. [6]
  4. **4.** Nystagmus in acute peripheral vestibular hypofunction: [6]
     - **a.** Primarily horizontal, may be slightly torsional
     - **b.** The quick phase is towards the uninvolved ear
     - **c.** Increases with gaze towards the direction of the fast phase
     - **d.** Decreases with gaze towards the direction of the slow phase

- ii. Ocular alignment; presence of skew deviation (hypertropia) [6]
  1. Eye on side of lesion drops in orbit and eye opposite the side of lesion is elevated secondary to loss of tonic otolith input [6]
  2. Named based on side with elevated eye [6]

- **iii.** **Head Thrust test**: testing VOR gain [3-6, 9]
  1. **1.** Patient fixates on therapist's nose. Therapist observes patient's eyes (with no blinking) after passive head thrust horizontally and vertically. [3, 4, 6]
  2. **2.** Normal: patient's eye remain stable and focus on target [Images A and B in Figure 2 below]. [3, 4, 6]

3. Abnormal: corrective re-fixation saccade required back to target when thrusted toward affected side. [Figure 2: Image C = lose fixation and D= after corrective saccade made back to target). [3, 6]This indicates loss of VOR gain (<1). [3, 4, 6]
4. Typically largest loss of VOR gain occurs for rotations towards affected side. [6]

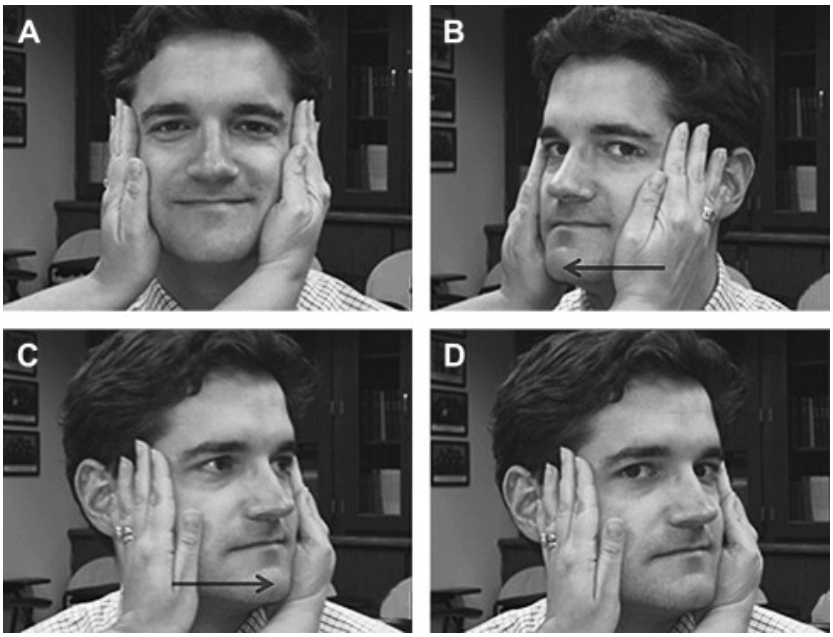

Figure 2. Kutz JW,Jr. The dizzy patient. *Med Clin North Am*. 2010;94: 989-1002.

iv. **Dynamic visual acuity:** Test VOR gain using biocular Snellen acuity with passive head rotations of 60 degrees at 1.5-2 hertz. [4-6, 17]
   1. Normal: decreased visual acuity of 1-2 line [4]
   2. Abnormal: suggested that decreased of visual acuity of 3-4 lines (unilateral loss) or 5-6 lines (bilateral loss). [4, 17]
   3. Computerized version of DVA also available. [18]

v. VOR cancellation: abnormal smooth pursuit system overrides VOR causing saccadic intrusions of eye movement during head movements. [5]

vi. **Dix-Hallpike test**: look for nystagmus and symptoms [3-6, 9]
   1. Normal: no nystagmus or vertigo
   2. Abnormal: with head turned toward affected ear, transient symptoms of vertigo and upbeating or torsional nystagmus with up to a 30 second delay. [9] Video showing positive test: http://www.dizziness-and-balance.com/disorders/bppv/movies/bppv.avi

vii. Other positioning tests/maneuvers if indicated:
   1. Side-lying test: Positive for posterior canal if upbeating and torsional nystagmus produced; positive for anterior canal if test produces downbeating with or without torsional component. [6, 9]
   2. Supine roll test: Positive for lateral canal with geotropic (beats towards earth) or apogeotrophic (beats away from earth) horizontal nystagmus [6, 9]

viii. Motion Sensitivity Quotient: identifies symptom-producing positions; patient rates intensity and duration of dizziness during 16 position changes. [19, 20] (Refer to supplementary document 4).

g. Somatosensory: Proprioception and or vibration, **light touch**, pain/temperature [5, 6]

h. **Cerebellar:** diadokokinesia, heel to shin, finger to nose, rebound. [4-6]

i. Reflexes: DTR, Hoffman and Babinski, spasticity, tone [5, 6]

j. Musculoskeletal

i. Posture, note deviations (scoliosis, kyphosis, lordosis) [5, 6]

ii. C-spine and upper quarter screen, pain, ROM, strength, spasm [5, 6]

iii. TMJ screen (symmetry, joint dysfunction) [5]

k. Balance

i. Sitting balance: weight shift, head righting, equilibrium reactions, abilities to bring trunk to vertical after perturbation. [6]

ii. Static Balance

1. Romberg, Sharpened Romberg [3, 4, 6]

a. Patient stands with feet slight apart, arms folded across chest, first with eyes open then eyes closed, for 30 seconds.

b. Positive test: if patient is able to stand with eyes open but loses balance or demonstrated increased sway with eyes closed.

c. Indicates proprioceptive deficits often secondary to peripheral neuropathy, dorsal root ganglia or dorsal column disease. [3]

d. Rarely positive in patients younger than 50 years old with acute vestibular loss. [3]

2. Single leg stance (eyes open and eyes closed) [6]

3. Standing on foam (eyes open and eyes closed) [6]

4. Perturbations in static stance [6]

iii. Dynamic balance

1. Standing reach test: self-initiated reach forward and to side to assess patient's ability or willingness to move outside base of support. [6] Scores less or equal to 6 inches indicated high risk of falling. [6] Instructions and tool available at http://www.rehabmeasures.org/Lists/RehabMeasures/DispForm.aspx?ID=950

2. Clinical Test of Sensory Interaction and Balance (CTSIB) or Sensory organization test (SOT) using Computerized dynamic posturography (CDP). Instructions and tool available at http://www.rehabmeasures.org/lists/rehabmeasures/dispform.aspx?id=897

3. BEST Test: [21] Instructions and tool available at http://web.missouri.edu/~proste/tool/bestest/index.htm

4. Timed up and Go (TUG) [6, 9] Instructions and tool available at http://www.rehabmeasures.org/Lists/RehabMeasures/DispForm.aspx?ID=903

5. Gait: specifically ask patient to walk at comfortable pace for 20 feet in one direction and make quick turn and then ask to make quick stop before return. [3, 4, 10]
    a. Monitor speed, base of support width, step length, arm swing, trunk lean, path deviations, apraxia and movement of head. [3]
    b. Decreased movement of head is commonly seen with patients with vestibular dysfunction. [3]
    c. Functional gait assessment (FGA): obstacle course, dual-task activities, stairs, grass, etc. Instructions and tool available at http://www.rehabmeasures.org/Lists/RehabMeasures/DispForm.aspx?ID=893
    d. Dynamic Gait Index (DGI): fall risk during gait with head turns, obstacles and varying speeds. Instructions and tool available at http://www.rehabmeasures.org/lists/rehabmeasures/dispform.aspx?id=898

l. Other tests used by non-PT healthcare professionals

i. Imaging when central cause suspected: diffused-weighted MRI (most common) or CT scan (for images of boney labyrinth for SCC fistula or dehiscence) [3]
ii. Blood work to rule out otic syphilis and vasculitis or if cause of dizziness is unclear. [3, 4]
iii. Electronystagmography (ENG) including caloric study (to evaluate SCC function) can quantify extend of vestibular deficit. [3, 18]
iv. Audiogram, if patient complains of hearing loss. [3]
v. Cervical vestibular-evoked myogenic potentials (cVEMP) identifies saccule dysfunction. [6, 9, 18]

References

1. Chan Y. Differential diagnosis of dizziness. *Curr Opin Otolaryngol Head Neck Surg*. 2009;17:200-203.

2. Newman-Toker DE, Cannon LM, Stofferahn ME, Rothman RE, Hsieh YH, Zee DS. Imprecision in patient reports of dizziness symptom quality: a cross-sectional study conducted in an acute care setting. *Mayo Clin Proc*. 2007;82:1329-1340.

3. Tusa RJ, Gore R. Dizziness and vertigo: emergencies and management. *Neurol Clin*. 2012;30:61-74, vii-viii.

4. Kutz JW,Jr. The dizzy patient. *Med Clin North Am*. 2010;94:989-1002.

5. Cavanaugh J. Examining the patient with dizziness of unknown etiology. *Neurology Report*. 1999;23:100-113.

6. Herdman SJ. *Vestibular Rehabiliation*. 3rd ed. Philadelphia, PA: F.A. Davis Company; 2007.

7. Pope JV, Edlow JA. Avoiding misdiagnosis in patients with neurological emergencies. *Emerg Med Int*. 2012;2012:949275.

8. Baloh RW. Dizziness in older people. *J Am Geriatr Soc*. 1992;40:713-721.

9. Hain T, Cherchi M. Information about dizziness, balance and hearing. [serial online]. April 2013;2013:2013. Available from: http://www.dizziness-and-balance.com/.

10. Mukherjee A, Chatterjee SK, Chakravarty A. Vertigo and dizziness--a clinical approach. *J Assoc Physicians India*. 2003;51:1095-1101.

11. Friedman JM. Post-traumatic vertigo. *Med Health R I*. 2004;87:296-300.

12. Black FO, Angel CR, Pesznecker SC, Gianna C. Outcome analysis of individualized vestibular rehabilitation protocols. *Am J Otol*. 2000;21:543-551.

13. Schubert MC, Minor LB. Vestibulo-ocular physiology underlying vestibular hypofunction. *Phys Ther*. 2004;84:373-385.

14. Toupet M, Ferrary E, Grayeli AB. Visual analog scale to assess vertigo and dizziness after repositioning maneuvers for benign paroxysmal positional vertigo. *J Vestib Res*. 2011;21:235-241.

15. Yardley L, Donovan-Hall M, Smith HE, Walsh BM, Mullee M, Bronstein AM. Effectiveness of primary care-based vestibular rehabilitation for chronic dizziness. *Ann Intern Med*. 2004;141:598-605.

16. Gomez Garcia A, Jauregui-Renaud K. Subjective assessment of visual verticality in follow-up of patients with acute vestibular disease. *Ear Nose Throat J*. 2003;82:442-4, 446.

17. Dannenbaum E, Paquet N, Chilingaryan G, Fung J. Clinical evaluation of dynamic visual acuity in subjects with unilateral vestibular hypofunction. *Otol Neurotol*. 2009;30:368-372.

18. Clarke AH. Laboratory testing of the vestibular system. *Curr Opin Otolaryngol Head Neck Surg*. 2010;18:425-430.

19. Hall CD, Cox LC. The role of vestibular rehabilitation in the balance disorder patient. *Otolaryngol Clin North Am*. 2009;42:161-9, xi.

20. Akin FW, Davenport MJ. Validity and reliability of the Motion Sensitivity Test. *J Rehabil Res Dev*. 2003;40:415-421.

21. Horak FB, Wrisley DM, Frank J. The Balance Evaluation Systems Test (BESTest) to differentiate balance deficits. *Phys Ther*. 2009;89:484-498.