HM513

Vertigo and Dizziness; Vestibular System Disorders - Summary

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I. INTRODUCTION - DIZZINESS

Dizziness is one of the most common presenting symptoms to the primary care physician, and a chief complaints that most physicians are uncomfortable evaluating. Dizziness is an imprecise term used by patients to describe various sensations ranging from whirling to lightheadedness to unsteadiness. However, a working concept of the vestibular system and knowledge of the common disorders presenting as dizziness lead to a practical approach to patients with this symptom.

As with most aspects of human medicine, the history is the paramount component of the evaluation, and the examination serves to confirm the physician's suspicions. The first question to ask any patient with dizziness is "what do you mean by dizziness?" Many patients will use the term dizziness to describe a variety of ailments, and the character of the sensation is a key feature in the history. Dizziness may be separated into:

- **vertigo** - sensation of movement, often rotary, indicating disorder of the vestibular system (see below)
- **non-vertiginous** dizziness such as: imbalance, lightheadedness, syncope, faintness, and other diseases.

  Imbalance may be described as dizziness, however, does not in isolation result from vestibular lesions. Imbalance may be a symptom of cerebellar dysfunction, drug toxicity, extrapyramidal disease (e.g. Parkinson disease), or other non-vestibular disorders.

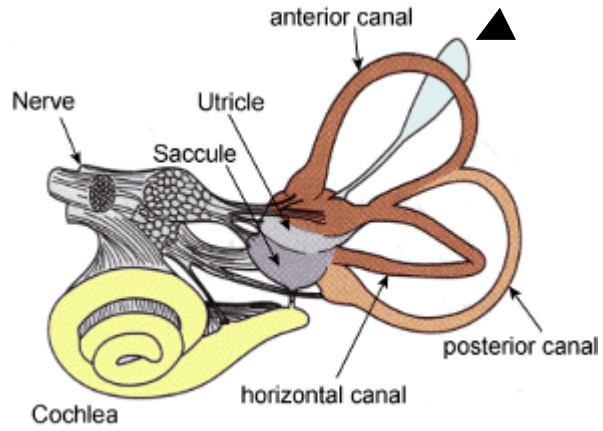
  Lightheadedness, often described as "floating" dizziness or "wooziness" may result from medications or the multiple sensory deficits syndrome. The multiple sensory deficits syndrome results from "de-afferentation"; often patients are elderly who have visual dysfunction (e.g. macular degeneration), balance difficulty (orthopedic or extrapyramidal disease), hearing loss and peripheral neuropathy. Patients are thus effectively cut off from receiving accurate information about the orientation of the environment which often leads to "dizziness". Additionally, certain medications may produce a non-vertiginous sense of dizziness described as lightheadedness.

  Syncope or presyncope often presents with "faint" feelings of dizziness. This sensation results from global hypoperfusion of the brain. Cerebral hypoperfusion may result from hypotension or arrhythmias. Orthostatic hypotension is a relatively common type of syncope/presyncope. This typically presents with faint feeling of dizziness after postural change, such as arising from the seated or lying position. Dehydration, certain antihypertensive medications, or autonomic failure may produce orthostatic hypotension. This is best assessed by taking the patient's pulse and blood pressure in the supine, seated and standing positions; a systolic drop of greater than 15-20 mm Hg is considered significant, while a rise in pulse indicates an intact autonomic nervous system attempting to compensation for decreased cerebral perfusion.

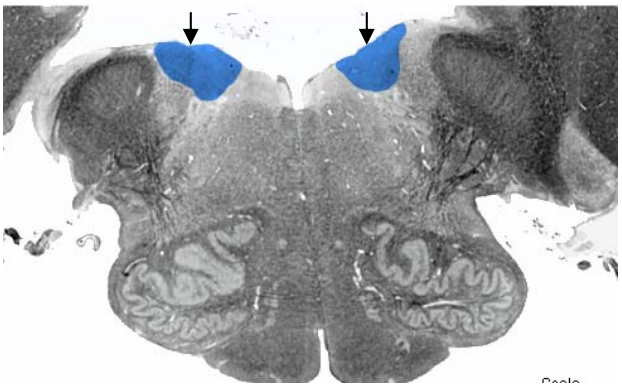
  Other medical conditions such as **endocrine diseases** (especially hypothyroid) may present with non-vertiginous dizziness. Lab tests to include thyroid function and FTA/RPR are often obtained in unexplained cases. Drugs including prescription **medications** may also result in several types of dizziness. Patients with **psychiatric disorders** may describe dizziness.

II. VESTIBULAR SYSTEM OVERVIEW

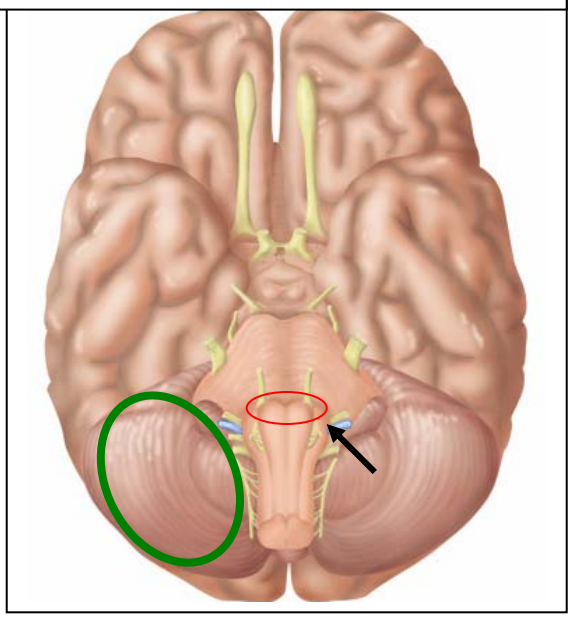
A. Introduction and anatomy: The primary purpose of the vestibular apparatus is to inform the brain of head position and acceleration. The labyrinth performs this through two organelles: the semi-circular canals, and the otolith. The semi-circular canals are responsible for detection of angular head acceleration, while the otolith responds to linear acceleration or gravity effects. Relative inertia of the endolymph within the semicircular canals during angular acceleration displaces hair cells embedded in the cupula, activates the hair cells, and transmits electrical activity to the vestibular division of CN VIII (vestibulocochlear nerve). Linear acceleration results in displacement of the otoliths within the utricle or saccule. This distorts the hair cells and increases or decreases the frequency of action potentials in the vestibular division of CN VIII.



Left: The labyrinth of the inner ear, from the left ear. It contains i) the cochlea - the peripheral organ of our auditory system; ii) the three semicircular canals, which transduce rotational movements; and iii) the otolithic organs (utricle and saccule), which transduce linear accelerations. The pouch (arrowhead) is the endolymphatic sac (duct), and contains only fluid. Courtesy of Wikipedia;http://en.wikipedia.org/wiki/Vestibular_system; Image: VestibularSystem.gif



L, R: courtesy of P.A. Stewart, Functional Neuroanatomy



Left: Arrows indicates vestibular nuclei in medulla. Right: the arrow points to CN VIII (vestibulocochlear nerve) at cerebellopontine angle; the thick circle indicates cerebellar hemisphere; the thin circle in the midline indicates the pons.

The cochlear (acoustic) and vestibular divisions travel together to the internal auditory meatus and then to the brainstem (cerebellopontine angle), each entering the brainstem separately. Axons (first order auditory neurons) in the cochlear division synapse in the dorsal and ventral cochlear nuclei in dorsolateral

medulla. The central auditory pathway includes bilateral connections to inferior colliculi, medial geniculate body, and auditory cortex in the temporal lobe.

First order vestibular neurons synapse in the vestibular nuclei in the dorsolateral medulla. Vestibular nuclei project to cerebellum; to ocular motor nuclei through the ascending medial longitudinal fasciculi (MLF) to subserve VOR (see below); to the spinal cord through the lateral vestibulospinal tracts to mediate postural reflexes; and to the thalamus, which projects to the postcentral gyrus.

B. Vestibular ocular reflexes (VOR) are mediated by vestibular nuclei, MLF, ocular motor nuclei and CN III, IV and VI. The purpose of the VOR is to maintain foveation during head movement - e.g., to maintain steady gaze and focus to read a street sign while walking or driving. This "visual gyroscope" moves the eyes an equal and opposite amount to compensate for head movement - if the head moves 30 degrees to the right, the eyes must move 30 degrees to the left to maintain fixation. The VOR depends on a 3 neuron arc: semi-circular canals via the vestibular nucleus --> internuclear connections to the extraocular motor neurons --> efferent projections along CN 3, 4, and 6 to the eye muscles. A tonic level of activity is present within each vestibular nerve, and the two vestibular organs act in a push-pull fashion with one side increasing activity and one side decreasing activity in response to head acceleration. An imbalance of the vestibular inputs leads to asymmetric information transmitted to the brain, and vestibular nystagmus occurs. The brain is able to compensate for these asymmetries given time (probably involves cerebellum to a large degree) by tuning up or down the three static vestibular inputs. The VOR is the reflex arc assessed with doll's head maneuver or caloric testing (for vertigo or in comatose patients).

C. Nystagmus: Nystagmus is an involuntary rhythmic oscillation of eyes in horizontal, vertical, torsional or mixed direction. Usually, there is slow drift of the eyes in one direction followed by quick jerk in the opposite direction. Nystagmus is named for the direction of the fast component. The normal maintenance of ocular posture and alignment of the eyes with the environment depends on retinal input, labyrinthine input, and central connections including vestibular nuclei and cerebellum. Thus, nystagmus may result from visual disease, labyrinthine disease, or disorders affecting the cerebellum or brainstem.

III. VERTIGO AND VESTIBULAR SYSTEM DISORDERS

A. Definition

Vertigo is defined as the sensation of movement of self or environment, often rotary. Vertigo results from a mismatch of the brain's three primary information systems: visual, vestibular, and sensory (proprioception). Vertigo is the historical hallmark of a vestibulopathy.

B. Clinical Presentation

1. Relevant historical features of dizziness/vertigo include:

- Duration
- Character or description
- Frequency
- Precipitants
- Associated symptoms

Vertigo and time (duration of symptoms): The duration of symptoms is important in consideration of possible causes of vertigo. (Each of the following diseases is discussed in more detail below.)

Duration	Disease
seconds	BPPV, ischemia (TIA)
minutes	ischemia (TIA), migraine
hours	Meniere's, migraine
days	vestibular neuritis, trauma, labyrinthine infarct

Note: "Constant", or 24 hour/day, dizziness is almost never vertigo - central compensation usually occurs. When evaluating patients reporting 24 hour/day dizziness for months, consider non-vertiginous causes, or faulty central compensation.

2. Physical exam features:

Nystagmus: Diseases of the vestibular system present with vertigo and/or oscillopsia (visual illusion of environmental movement). The physical exam hallmark of vertigo is nystagmus. However, this must be examined for in an appropriate fashion; e.g., patients with peripheral vestibular lesions may suppress their nystagmus by visual fixation. The vestibular system controls the vestibular-ocular reflex (VOR), and abnormalities of this system lead to nystagmus. Although nystagmus is named for the fast phase of the eye movement, the slow phase is the most telling regarding the pathophysiology.

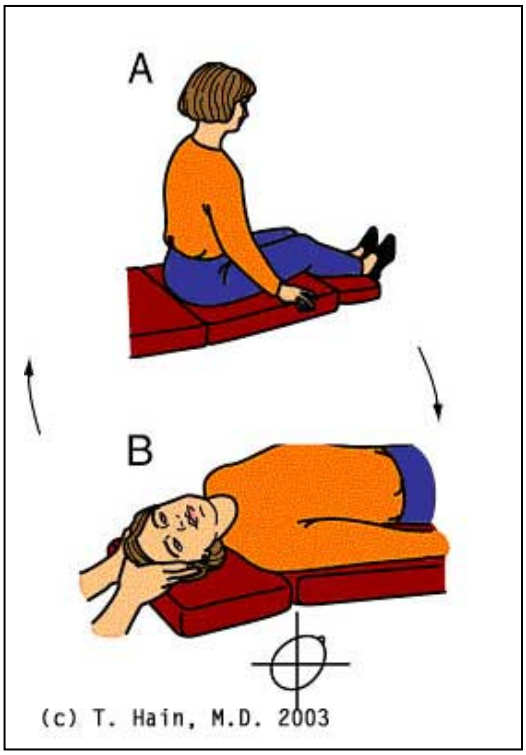
Associated symptoms to distinguish between central and peripheral causes: Both central (vestibular nucleus and brainstem) and peripheral (vestibular nerve and labyrinth) lesions may produce vertigo and resultant nystagmus. Perhaps the company a disease keeps is the most useful way to separate peripheral from central causes. The proper separation of vertigo into central or peripheral origins is useful because this narrows the list of possible pathophysiologies. The nystagmus character and associated features are often helpful in separating the two general locations:

Symptom	Central	Peripheral
nausea	mild- moderate	severe
imbalance	moderate - severe	moderate
hearing loss	rare	common
tinnitus	rare	may occur
oscillopsia	severe	mild
neurologic	common	rare
compensation	slow	rapid
nystagmus	pure vertical unchanged with fixation no fatigability no latency	mixed torsional-vert/horiz damps with fixation fatigability (BPPV) latency post maneuver (BPPV)

Thus, a patient with vertigo, diplopia, dysarthria, weakness and/or numbness probably has a brainstem located lesion, while another patient with vertigo, hearing loss, tinnitus and no other neurologic features of brainstem involvement more likely has a peripheral etiology.

C. Peripheral Causes of Vertigo

1. Benign paroxysmal positional vertigo (BPPV): BPPV is the most common cause of vertigo. This disorder is important to diagnose because (a) it is common, (b) it is easily treated without medications, and (c) correct diagnosis avoids costly and unnecessary testing. The typical history consists of short-lived episodes of vertigo (rotary) precipitated by head movements (especially with neck extension - lying down, looking up - "top shelf" vertigo). The time frame of vertigo is usually less than 45 seconds, although patients may report symptoms for minutes (difficult to time when acute vertigo is present) - but not hours (unless many spells lasting seconds recur over the period of hours). Trauma is a common antecedent event (50%), while other cases are idiopathic or occur following vestibular neuritis. The key physical exam finding is observed on Dix-Hallpike testing (see diagram at right), in which the patient is rapidly moved from a seated position to lying position with the head 45 degrees turned and extended; this maneuver is designed to maximally stimulate the posterior semi-circular canal.



Hain, TC. *http://www.dizziness-and-hearing.com*. 9 Aug 2008 <http://www.dizziness-and-balance.com/images/dix-hallpike-c.jpg>

BPPV nystagmus consists of slow phases directed down with torsion; right BPPV produces slow phases clockwise and down, with fast phases the opposite direction. Nystagmus begins after a latency lasting seconds, and the response may fatigue such that repeated attempts to reproduce it fail. Therapy via repositioning maneuvers is generally 75% effective with a single ~5 minute treatment.

2. Vestibular neuritis: Vestibular neuritis typically produces a monophasic episode of vertigo lasting from days to weeks, often accompanied in the acute phase by severe nausea and vomiting. Acute treatment with vestibular suppressants (meclizine, diazepam, dramamine, lorazepam) is often necessary; however, treatment should be withdrawn as soon as possible to help foster central compensation. Vestibular physical therapy may enhance the rate of recovery from this and other vestibulopathies.

3. Ménière's disease: Meniere's disease appears to be caused by endolymphatic regulatory dysfunction. Spells of vertigo usually last hours, and are classically preceded by ear pressure/fullness, and change in tinnitus and hearing function. It is important to ask patients with vertigo if there is a history of fluctuating hearing loss and tinnitus. Spells may be precipitated by high salt intake. Both medical and surgical treatments are available, depending upon the severity and frequency of symptoms. Medical treatment often includes low salt diet, alcohol abstinence, diuretic medications, and short courses of PRN vestibular suppressants.

4. Trauma: Trauma may produce several types of vertigo including BPPV, direct labyrinth trauma/contusion (persistent imbalance), or non-vertiginous, non-specific "dizziness" as part of the post-concussive syndrome. The details of the history and physical exam will help define the specific diagnosis.

D. Central Causes of Vertigo

1. TIA and brainstem infarction: Brainstem ischemia is an important cause of vertigo because further ischemia in this region may be acutely life threatening. These patients commonly have traditional vascular risk factors (hypertension, diabetes, hyperlipidemia, tobacco use). Brainstem TIAs present with spells characterized not only by vertigo but also combinations of diplopia, dysarthria, numbness, and incoordination; isolated vertigo is rarely due to ischemia. The most common brainstem ischemic syndrome is Wallenberg's lateral medullary syndrome, usually due to occlusion of the vertebral artery or posterior inferior cerebellar artery. Patients may elude correct diagnosis because there is no weakness, which is often expected with a TIA or stroke. Typical patients present with vertigo, diplopia (due to vertical misalignment of the eyes secondary to skew deviation), ipsilateral Horner's syndrome (ptosis, miosis, and anhidrosis), ipsilateral loss of pain/temperature sensation on the face, ipsilateral ataxia, CN 9/10 palsy (palate elevates to the contralateral side), and contralateral loss of pain/temperature sensation on the body (may be reported as numbness).

2. Tumor or mass: Brainstem tumors rarely present with isolated vertigo. Vestibular/acoustic neuromas (nerve sheath tumors of CN 8) typically present with progressive unilateral hearing loss; the slow pace of vestibular loss allows central compensation until very late.

3. Migraine: Approximately 10-30% of patients with migraine cephalgia develop episodic vertigo lasting minutes to hours. Vertigo may or may not occur temporally related to headache. Physical examination and ancillary testing between episodes is normal. Treatment with traditional prophylactic migraine medicine is often successful.

4. Multiple Sclerosis: Demyelinating disease is an important cause of central vestibular lesions in young patients. Approximately 33% of MS patients present with visual symptoms, while an additional 7% present with vestibular symptoms. Exam may evidence central, peripheral or mixed patterns of nystagmus.

Recommended reading:

Baloh and Honrubia - Clinical Neurophysiology of the Vestibular System
Leigh and Zee - The Neurology of Eye Movements
www.dizziness-and-balance.com
http://webiocosm.com/timelapse/bppv/bppvlarge.htm - video of BPPV testing

Practice Questions

1. A patient presents with vertigo, diplopia, dysarthria, weakness and numbness. Which of the following is the most likely location of the lesion?

A. brainstem
B. vestibulocochlear nerve
C. spinal cord
D. labyrinth

2. A patient presents with vertigo, hearing loss, and tinnitus. There are no other neurologic abnormalities. Which is the following is the most likely location of the lesion?

A. brainstem
B. vestibulocochlear nerve
C. spinal cord
D. cerebral cortex

3. A patient describes short-lived episodes of vertigo (rotary) precipitated by head movements, especially when lying down (neck extension). The vertigo usually lasts less than 45 seconds. There are no other neurological symptoms except nystagmus. What is the most likely diagnosis?

A. Meniere's disease
B. Occlusion of the posterior inferior cerebellar artery
C. Multiple sclerosis
D. Benign paroxysmal positional vertigo (BPPV)

4. A patient describes spells of vertigo lasting hours, preceded by ear pressure/fullness, and change in tinnitus and hearing function. There are no other neurological abnormalities. What is the most likely diagnosis?

A. Meniere's disease
B. Occlusion of the posterior inferior cerebellar artery
C. Multiple sclerosis
D. Benign paroxysmal positional vertigo (BPPV)

Answers: 1. A; 2. B; 3. D; 4. A