Dizziness, Vertigo, and Syncope: Assessment and Treatment

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Disclosure

• No real or potential conflict of interest to disclose.
• No off-label, experimental or investigational use of drugs or devices will be presented.

Objectives

• Having completed the learning activities, the participant will be able to:
  – Differentiate among causes of syncope, near syncope, vertigo, and ataxia including adverse drug effects.
  – Analyze differences between central and peripheral vertigo.
  – Evaluate management strategies for the disorders presented.
Assessment of Dizziness

- Ataxia
  - Inability to maintain balance
- Near-syncope
  - A sense of “sinking” without actual loss of consciousness

Assessment of Dizziness (continued)

- Vertigo
  - Sense of the rotational movement of self or surroundings
- Syncope
  - Actual loss of consciousness

Ataxia

- Balance and coordination are first affected.
- Other symptoms occur later.
  - Loss of fine motor coordination
  - Slurred speech
  - Difficulty swallowing
- Both hereditary and spontaneous forms exist.
Hereditary Forms

<table>
<thead>
<tr>
<th>Autosomal Dominant</th>
<th>Autosomal Recessive</th>
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<tr>
<td>• Spinocerebellar ataxia includes</td>
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<td>– Cognitive defects</td>
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<td>– Dementia</td>
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<td>– Neuropathy</td>
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<td>– Extrapyramidal features</td>
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<td>• Adult onset forms exist</td>
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<td>• Friedreich’s ataxia includes</td>
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<td></td>
<td>– Cerebellar sx</td>
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<td>– Corticospinal sx</td>
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<td>– Sensory loss</td>
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<td>– Wheelchair dependence</td>
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<td>– Average death age 38 years</td>
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### Identifying Ataxia

- When the chief complaint is “dizziness,” the evaluation proceeds to differentiate among actual symptom type.
- When the primary problem is ataxia, the diagnostic evaluation should proceed to rule out hereditary vs. sporadic vs. transient causes.

### Pharmacologic Implications

- Drugs that can produce ataxia
  - Antiepileptics
  - Dextromethorphan
  - Fibric acid derivatives
  - Metformin
  - Levodopa
  - Methotrexate
  - Thiazide diuretics

### Near-syncope

- Patients may be describing near-syncope when the chief complaint is “dizziness.”
- Near-syncope is generally the result of transient, decreased blood flow to the brain.
- Patients describe a general sense of “sinking” of “almost fainting” or “fainting.”
Near-syncope (continued)

- Characteristic (but not prerequisite) in the description of symptoms is that it occurred when the patient was upright and resolved when they went supine/prone.

Near-syncope (continued)

- Causes of near-syncope break down into four different categories.
  - Cardiogenic
  - Neurocardiogenic
  - Neurologic
  - Psychiatric
- The diagnostic evaluation is directed by your impression of category

Vertigo

- Described by the patient as the sense of the patient or the room “spinning”
- Often accompanied by other symptoms
- Occurs in any position
- Is the cardinal symptoms of vestibular disease
- Virtually always exacerbated by head movement and never continuous.
Diagnostic Evaluation of Vertigo

- Vertigo is almost always evaluated effectively by the history.
- Rule out systemic/metabolic causes.
  - Medications
  - Psychogenic
  - Infection
  - Hypoxia

Drugs that Cause Vertigo

- Similar to those causing ataxia
  - AEDs
  - Sedative hypnotics
  - Narcotic analgesics
  - Antibiotics
  - Salicylates
  - Miscellaneous

Central or Peripheral

- After ruling out metabolic causes, need to determine whether vertigo is central or peripheral
- Consider the statistics
  - 50% of vertigo is benign paroxysmal positional vertigo (BPPV).
  - 25% of vertigo is vestibular neuronitis.
  - 10% of vertigo is Meniere’s disease.
- 85% of vertigo will be peripheral
Central Vertigo

• Cerebellar disease accounts for most cases of central vertigo.
• Brain stem ischemia
• Multiple sclerosis

Central Vertigo (continued)

• Associated symptoms increase likelihood of central disease.
  – Hiccups in coordination
  – Visual or sensory loss
  – Diplopia
• Neoplastic disease accounts for only 1%.
  – Onset is insidious.

Central Vertigo (continued)

• Vascular disease accounts for remainder of central vertigo.
  – Other symptoms of vascular disease are usually present.
  – Onset of vertigo more acute and lasts for minutes.
  – Other neurologic symptoms may be present.
Central Vertigo Associated Findings

- Diplopia
- Autonomic symptoms
- Nausea
- Dysarthria
- Dysphagia
- Focal weakness

Central Vertigo Associated Findings (continued)

- Unable to ambulate during acute episodes
- Dysdiadochokinesis (DDK or ataxia in cerebellar disease)
- Sensory/motor symptoms in CNS disease

Peripheral Vertigo

- A variety of conditions that are external to the brain stem and cerebellum are considered peripheral vertigo.
Peripheral Vertigo  
(continued)

• Causes
  – Benign paroxysmal positional vertigo (BPPV); otoliths
  – Meniere’s disease
  – Recurrent vestibulopathy
  – Labyrinthitis
  – Traumatic vertigo
  – Perilymphatic vertigo

Peripheral Vertigo  
(continued)

• Associated symptoms contribute to diagnosis.
  – Hearing loss
  – Pain in the ear
  – Tinnitus

Characteristics of Peripheral Vertigo

• Sudden onset and vivid memory of vertigo usually a product of inner ear etiology
• Onset and time course help distinguish among peripheral causes.
• Associated or exacerbated with head or body movement, position changes.
BPPV (Otoliths)

- The most common cause of vertigo.
- Calcium crystals are inappropriately displaced into the semicircular canals of the labyrinth of the inner ear.
- When detached, head movement causes otoliths to move; this stimulates hair cells.
- Result is vertigo

Meniere’s Syndrome

- Clinical diagnosis of vertigo, hearing loss, tinnitus
- Cause is unknown.
- Distention of the endolymphatic compartment of the inner ear
- It is chronic.
- Sensorineural hearing loss

Recurrent Vestibulopathy

- Meniere’s syndrome without the auditory symptoms
- Most patients will go on to develop the auditory symptoms.
- Increased incidence in migraine sufferers
Labyrinthitis

- Transient vertigo
- Acute and short-lived
  - Typically a matter of days
- Often associated with bacterial or viral infection
- Associated tinnitus and hearing loss
- Rapid head movement will provoke vertigo for weeks.

Less Common Causes of Peripheral Vertigo

- Positional vertigo
  - Vertebrobasilar insufficiency
  - Triggered by position change – Sx occur 10–60 seconds later
- Traumatic vertigo – Follows fx
- Periphymphatic vertigo
  - Linked to head trauma, barotrauma, Valsalva maneuver

Remember…

- Vertigo is never continuous.
- Vertigo is always exacerbated by head movement.
- If both of these are not present, the patient is not having vertigo.
Physical Examination

• Romberg test
• Evaluate gait
• Nystagmus
• Vision/hearing
• Provoking maneuvers
  – Valsalva maneuver
  – Nylen-Barany maneuver

Nystagmus Assessment

• Description of nystagmus should include
  – Provocative factors
  – Direction
  – Latency
  – Fatigue
  – Suppression by visual fixation
  – Accompanying vertigo

Nystagmus

• Peripheral vertigo
  – Usually rotary
  – Jerk nystagmus
  – Most evident by removing visual fixation
  – Can fatigue if elicited by head movement
  – Does not change direction with change of gaze
  – Diminishes with fixation
Nystagmus (continued)

- Central vertigo
  - Purely horizontal or vertical; vertical nystagmus is considered specific to central vertigo
  - Not suppressed by visual fixation
  - Can change direction with gaze
**Vertical Nystagmus**

**Hallpike Maneuver**
- Nystagmus and vertigo occur when diseased side turned downward.
- Peripheral nystagmus fatigues with repeated maneuvers.
- Central nystagmus does not change with repeated maneuvers.

**Medical Management of Central Vertigo**
- **Histamine₁ receptor antagonists**
  - Decreases excitability of inner ear labyrinth and blocks conduction in inner ear vestibular-cerebellar pathways
- **Anticholinergic agents**
  - Blocks action of acetylcholine at parasympathetic sites in CNS
  - Antagonizes histamine and serotonin action
Medical Management of Central Vertigo
(continued)
• Benzodiazepines
  – Potentiate effects of (GABA) and facilitates inhibitory GABA neurotransmission and other inhibitory transmitters
• Phenothiazines
  – Blocks postsynaptic mesolimbic dopaminergic receptors in brain and reduces stimuli to brainstem reticular system

Medical Management of Peripheral Vertigo
• Medications not always recommended; depends upon the type
• Acute vertigo – Bedrest
• Chronic vertigo – Activity

Medical Management of Peripheral Vertigo
(continued)
• Vestibular neuronitis
  – Vertigo without auditory symptoms, lasts several days to one week; frequently followed by several weeks of BPPV
  – 1/3 of patients develop chronic sx
  – Likely of viral etiology
Medical Management of Peripheral Vertigo
(continued)

• Vestibular neuronitis (cont.)
  – Brief course of antiemetics and vestibular suppressant in acute phase
  – Corticosteroids can improve long-term outcomes.

Medical Management of Peripheral Vertigo
(continued)

• BPPV
  – May be symptomatic of another condition or idiopathic
  – Brief vertigo with position change
  – Treatment involves dispersing otoliths.
  – Treated with Epley’s maneuver

http://www.merck.com/mweb/nri Benz/figures/PMPE_00908600.png
Medical Management of Peripheral Vertigo (continued)

• The traditional cocktail for symptom management
  – Benzodiazepine
  – Antiemetic
  – Antihistamine

Management of Peripheral Vertigo

• Meniere’s disease
  – Low salt diet and diuretics helpful for 80% of patients.
    • Thiazides are the most common type used.
    • No strong evidence-based support for efficacy of diuretic therapy

Management of Peripheral Vertigo (continued)

• Meniere’s disease (cont.)
  – Alcohol, caffeine, nicotine avoidance
  – Corticosteroids for severe episodes
  – Injection of gent/bicarb only in ear with no serviceable hearing
  – Surgical therapies very controversial
Syncope

- Multiple causes of syncope
- Generally divided among four categories
  - Neurological/neurogenic
  - Cardiogenic
  - Postural
  - Others

Neurogenic Syncope

- Need to rule out seizure
  - Post episode disorientation
  - Bowel/bladder incontinence
  - Tongue biting
- Small cerebral bleeds/infarcts
- Assess for focal neurological deficits
- Not affected by position

Cardiogenic Syncope

- Generally characterized by an absence of premonitory symptoms
- Can include any cardiogenic cause of decreased cerebral blood flow
  - Dysrhythmia
  - Valvular disease
  - Atrioventricular block
Postural Causes

- Hypotension
  - Consider new vasoactive meds
- Baroreceptor abnormalities
  - Common in the elderly
- Dehydration
- Neurocardiogenic syncope (vasovagal)
  - Emotional factors
  - Physiologic factors

Syncope Risk Factors

- Cardiovascular disease
- Diabetes
- Offending drug therapy
- Age
- Neurologic disease

Signs and Symptoms

- Loss of consciousness is the significant one.
- Presence of a prodrome suggests neurocardiogenic.
- Signs and symptoms of predisposing disease may be present.
Diagnostic Studies

• Dictated by the history of the event
• Positive tilt test suggests autonomic dysfunction
• More invasive procedures as indicated when cause remains elusive

Management of Syncope

• When organic cause identified, treat as appropriate
• Therapies for recurrent neurocardiogenic syncope
  – Non-cardioselective beta blockade
  – Fludrocortisone (Florinef®)
  – SSRIs
  – Disopyramide phosphate (Norpace®)
• Adjust medications that may be offending

References

References (continued)