Dizziness, Vertigo, and Syncope: Assessment and Treatment

Sally K. Miller, PhD, AGACNP-BC, AGPCNP-BC, FNP-BC, FAANP
Senior Lecturer
Fitzgerald Health Education Associates
North Andover, MA
Clinical Professor
Drexel University College of Nursing and Health Professions
Philadelphia, PA
Nurse Practitioner,
Nevada Health Center, Amargosa Valley, NV

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 pharmacologic interventions for the disorders presented.

References

Additional references at end of presentation

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
Autosomal Dominant
• Spinocerebellar ataxia includes
  – Cognitive defects
  – Dementia
  – Neuropathy
  – Extrapyramidal features
• Adult onset forms exist

Autosomal Recessive
• Friedreich’s ataxia includes
  – Cerebellar sx.
  – Corticospinal sx.
  – Sensory loss
  – Wheelchair dependence
  – Average death age 38 years

Sporadic Ataxia
• Diagnosis of exclusion
• SCA is ruled out.
• Adult onset
• No family history
• Symptoms less severe than SCA

Transient Forms
• Short-lived as name implies
• Result from insult to the cerebellum
• Alcohol or drug intoxication are leading differentials.
• Infection: Viral, meningitis, Creutzfeldt-Jacob
• Metabolic imbalance
• Abnormalities in PTH, Vit E malabsorption

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” or “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 will be explored more fully in the discussion of syncope.
  - Cardiogenic
  - Neurocardiogenic
  - Neurologic
  - Psychiatric

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 dz
- 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.

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 dz.
  - 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 sx 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
  – Meniere’s disease
  – Recurrent vestibulopathy
  – Labyrinthitis
  – Benign paroxysmal positional vertigo (BPPV)
  – 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 disease
• Onset and time course help distinguish among peripheral causes
• Associated or exacerbated with head or body movement or position changes

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

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 dizziness

Nystagmus

- Peripheral vertigo
  - Usually rotary
  - 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
  - Not suppressed by visual fixation
  - Can change direction with gaze

Head Tilt Test

- 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

- **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 (continued)

- **Vestibular neuronitis**
  - Vertigo without auditory sx, 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

### 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

(continued)

- Meniere’s disease (cont.)
  - Alcohol, caffeine, nicotine avoidance
  - Steroids 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.
End of Presentation
Thank you for your time and attention.
Sally K. Miller,
PhD, AGACNP-BC, AGPCNP-BC, FNP-BC, FAANP
sally@fhea.com www.fhea.com

References

References (continued)

References (continued)

Images/Illustrations: Unless otherwise noted, all images/illustrations are from open sources, such as the CDC or Wikipedia or property of FHEA or author.
• All websites listed active at the time of publication.

Copyright Notice
Copyright by Fitzgerald Health Education Associates
All rights reserved. No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopy, recording or any information storage and retrieval system, without permission from Fitzgerald Health Education Associates

Requests for permission to make copies of any part of the work should be mailed to:
Fitzgerald Health Education Associates
85 Flagship Drive
North Andover, MA 01845-6184
Statement of Liability

• The information in this program has been thoroughly researched and checked for accuracy. However, clinical practice and techniques are a dynamic process and new information becomes available daily. Prudent practice dictates that the clinician consult further sources prior to applying information obtained from this program, whether in printed, visual or verbal form.
• Fitzgerald Health Education Associates disclaims any liability, loss, injury or damage incurred as a consequence, directly or indirectly, of the use and application of any of the contents of this presentation.
Peripheral Vertigo

A variety of conditions that are external to the brain stem and cerebellum are considered peripheral vertigo.
Redistributed particles

Particles in semicircular canal

The head may be rapidly turned even further to almost face the floor. The patient is returned to the upright position, and the head is rotated back to normal.

The clinician rotates the patient's head toward the affected ear, then lowers the patient backward to the supine position with the head hanging over the table's edge.

The head is turned further, so that the ear is parallel to the floor.

The head is turned to the other side.

http://www.merck.com/media/mmpe/figures/MMPE_08ENT_86_01_eps.gif