INTRODUCTION

- Definition of vertigo: perception of movement (rotational or otherwise) when it does not exist
- In general, there are 4 kinds of “dizziness”
  - Vertigo: the focus of this chapter
  - Near syncope: impending loss of consciousness
  - Disequilibrium: usually in the elderly, due to loss of multiple sensory input – hearing, sight, etc.
  - Other: psychiatric and anxiety syndromes

PATHOPHYSIOLOGY

- “Balance” is the result from multiple sensory inputs (visual, vestibular and proprioception) that CNS processes
- Vertigo results from a mismatch of sensory inputs
- Vestibular system end-organ is the assembly of semi-circular canals: movement in these canals (i.e. due to movement of the head) is translated into electrical energy that is sent to the brainstem via cranial nerve VIII
- CNS coordinates this input with the eyes, so that we maintain a visual “fix” while movement occurs.
- Dominant clinical finding in vertigo is nystagmus: rhythmic movement of the eyes
  - Pure vertical or direction-changing nystagmus should be presumed brainstem until proven otherwise.
- Vertigo is more common in the elderly.
- “Physiological vertigo” – the sense of vertigo one feels, for example, in watching a film that captures rapid motion.

CLINICAL FEATURES

- Vertigo usually divided into “peripheral vertigo” and “central vertigo” (see Table 223-2). Important features:
  - Peripheral: caused by abnormalities of the end-organs (semicircular canals and 8th cranial nerve)
    - Explosive onset, intense symptoms, worse with moving head, associated with nausea/vomiting; hearing loss/tinnitus may occur; fatigable; rotatory or horizontal nystagmus.
  - Central: originates form the cerebellum or brainstem.
    - Slow onset, ill defined/less intense; no change with moving head, little or no nausea/vomiting; not fatigable; vertical nystagmus.
  - Important clinical clues: hearing loss with vertigo usually signifies a peripheral cause, presence of other CNS dysfunction by history or exam (diplopia, dysarthria, visual problems) suggests a central cause.

DIAGNOSIS

- History
  - First, try to categorize patients into 1 of the 4 kinds of dizziness noted in INTRODUCTION.
  - Best question: “what do you mean by dizzy?” Without careful attention to this first point, diagnostic evaluation becomes illogical and bogged down in lots of tests without much direction or purpose
  - Once the history suggests vertigo, further questions held to determine peripheral or central
- Physical Examination
  - Physical exam may disclose hypotension, valvular disease → possible non-vertigo causes of dizziness
  - Focused examination of ear, vestibular and neurological systems: external canal, TM, hearing test
  - Neurological examination:
    - Cranial nerves in detail, especially nerves of the cerebellopontine angle (CN V, VII, VIII)
    - Gait: patients with central vertigo often CANNOT walk (must distinguish from mere difficulty walking)
  - Nystagmus: the principle objective sign of vertigo.
    - May be spontaneous (on primary gaze) or positional (only evoked by changes in head position)
    - A few beats of horizontal nystagmus can be normal on extreme lateral gaze.
  - Other tests
    - Dix-Hallpike/Nylan-Barany: Patient rotates head 45° to one side, and then lies back with head hanging below bed and off stretcher. Vertigo, vomiting, nystagmus suggests benign peripheral vertigo
- Ancillary Tests
  - ECG, blood work, brain imaging, Holter monitoring as needed on case-by-case basis
SYMPTOMATIC TREATMENT

• Discussion of treatment organized by condition for ease of reference

• Peripheral Disorders
  o BPPV: scopolamine patch and antihistamines discussed in text, but this is antiquated advice; Epley maneuver curative in 85% of cases, photos or videos helpful to learn maneuver
    § Step 1: Perform Dix-Hallpike, wait until symptoms and/or nystagmus resolve (about 1 minute)
    § Step 2: Move the patient’s head 90° to the other side, wait until symptoms resolve
    § Step 3: Rotate patient 90° (patient now on stomach wait until symptoms resolve
    § Step 4: Have the patient sit up and stay upright for at least 20 minutes.
    § May repeat if Dix-Hallpike is not back to normal.
  o Meniere’s disease: treatment coordinated by ENT; antihistamines, diuretics, calcium channel blockers, salt restriction, intra-tympanic injections of steroids and gentamicin.
  o Peri-lymphatic fistula: treated by the ENT surgeon; some resolve without surgery.
  o Vestibular neuritis: text says to treat symptomatically; however recent well-done studies suggest that steroids lead to much better outcomes; refer to ENT.
  o Vestibular ganglionitis: antiviral therapy aimed at herpes zoster.
  o Labyrinthitis: symptomatic treatment, steroids vestibular neuritis, antibiotics if bacterial, ENT referral.
  o Post-traumatic vertigo: symptomatic treatment, refer to ENT or neurologist if symptoms persist
  o Ototoxicity: stop the offending drug and refer to ENT.
  o Eighth nerve lesions and CPA tumors: refer to neurosurgeon.

• Central Disorders
  o Cerebellar stroke: emergent consultation and referral to neurologist and/or neurosurgeon.
  o Wallenberg syndrome: emergent consultation and referral to neurologist and/or neurosurgeon.
  o Vertebrobasilar insufficiency: aspirin, urgent neurologist or internist consultation
  o Vertebral artery dissection: emergent referral to a neurologist and/or neurosurgeon; heparin; stenting
  o Multiple sclerosis: consider admission or urgent referral to neurologist after, at minimum, phone discussion
  o Tumor: emergent referral to a neurosurgeon.
  o Migraine: anti-migraine treatment and referral to a neurologist

DISORDERS CAUSING PERIPHERAL VERTIGO

• Benign paroxysmal peripheral vertigo (BPPV)
  o One of the most common causes of peripheral vertigo; women more likely to be affected than men
  o Nausea often present, vomiting unlikely; no hearing loss or tinnitus
  o Otoliths in semicircular canals cause movement of endolymph when head is moved.
  o Reviewers note: diagnosis of BPV usually clear; Epley maneuver (see below) may be curative.

• Meniere’s disease
  o Episodic disorder associated with excessive endolymph in the cochlea and labyrinth
  o Symptoms usually start unilaterally, then become bilateral; last several hours per attack
  o Associated symptoms are fullness behind ear, “roaring” tinnitus, nausea/vomiting.

• Perilymphatic fistula
  o Opening in round or oval window that allows pressure changes in middle ear to be transmitted to inner ear.
  o Trauma, infection or rapid pressure changes can cause the tear
  o Suspected in vertigo with SCUBA diving, coughing, sneezing, flying, coughing, sneezing, straining.

• Vestibular neuritis
  o Viral inflammation of CN VIII causing debilitating vertigo lasting for days and requiring bedrest
  o Usually decreases dramatically and does not recur (unlike BPV or Meniere’s disease, which can recur)

• Vestibular ganglionitis
  o Viral inflammation of vestibular ganglion; may be reactivated after initial infection
  o Herpes zoster oticus (Ramsay Hunt) an example of vestibular ganglionitis
  o Antivirals appropriate if diagnosed early

• Labyrinthitis
  o Infection of labyrinth producing peripheral vertigo and hearing loss
  o In bacterial disease, TM is usually red and there may be mastoid tenderness

• Ototoxicity
  o Ototoxic drugs rarely cause vertigo; recreational drugs (i.e. phencyclidine) may cause ataxia and nystagmus
VERTIGO AND DIZZINESS, PAGE 3; DISORDERS CAUSING PERIPHERAL VERTIGO, continued

- **BPV of childhood**
  o Disorder related to migraine headaches, occurs in children under 3 years of age
- **Eighth-nerve lesions and cerebellopontine angle tumors**
  o Meningiomas, acoustic schwannomas and neurofibromas can involve CN VIII at CPA or along its course
  o Typical findings may include ipsilateral facial weakness, lost corneal reflex, deafness and ataxia.
- **Post-traumatic vertigo**
  o May be associated with temporal bone fracture; vertigo immediately following trauma frequently needs brain imaging to exclude hematoma.
  o Head injury can also lead to BPPV (trauma loosens small otolith), but the pattern of vertigo and physical findings are otherwise typical for that diagnosis.

DISORDERS CAUSING CENTRAL VERTIGO

- **Cerebellar hemorrhage and infarction**
  o Emergent neurosurgical intervention is often required for hemorrhage
  o Classic presentation is headache (often occipital), nausea/vomiting and ataxia
  o Patients may complain of a rocking or to-and-fro motion rather than traditional spinning
- **Wallenberg syndrome**
  o Vertigo due to lateral medullary infarction
  o Ipsilateral findings: facial numbness, lost corneal reflex, Horner syndrome, dysphagia and dysphonia
  o Contralateral findings: loss of pain/temperature in the limb and trunk
  o Cranial neuropathy (VI, VII, VIII) may also occur
- **Vertebrobasilar Insufficiency (VBI)**
  o Isolated vertigo unusual; associated diplopia, dysarthria, long tract signs, bilateral visual loss common
  o May be positional if turning the head occludes a vertebral artery
  o If both arteries involved, reticular activating system ischemia may produce LOC (“drop attacks”).
- **REVIEWERS NOTE:** Also consider Vertebral Artery Dissection
  o Symptoms are same as VBI, often with headache or neck pain.
  o Precipitating factors include sudden neck movements (including deceleration from MVCs, diving injuries; also just coughing, sneezing, and chiropractic adjustments.
  o Sometimes there are no risk factors.
- **Multiple sclerosis**
  o Vertigo usually lasts hours to days to weeks.
  o Nystagmus usually prominent; ataxia and/or optic neuritis may also be seen
  o Diagnosis is confirmed by MRI and/or evoked potentials; neurology consultation is important.
- **Neoplasms**
  o Tumors, especially in the region of the 4th ventricle, may cause vertigo
  o Diagnosis is by imaging, preferably MRI; neurosurgical consultation is indicated
- **Migraine-related dizziness and vertigo**
  o Vertigo and motion sickness are strongly associated with migraine
  o May occur as an aura, associated with or independent from headache
- **Physiologic vertigo**
  o “Visual Vertigo” from watching films of motion or looking at complex patterns

OTHER CONDITIONS

- **Disequilibrium of aging:** ill-defined “dizziness” and gait unsteadiness with advancing age; polypharmacy may exacerbate, meclizine may worsen; treatment orchestrated by internist or geriatrician
- **Near syncope:** long differential diagnosis, including issues with the heart (valves, dysrhythmias), blood vessels and their autonomic innervation (vasovagal, Shy-Drager), and hypovolemia. Treatment depends on cause
- **Convulsive disorders:** some patients with non-convulsive status epilepticus will have non-vertiginous dizziness. Neurology consultation along with finding and treating the cause of the seizure are indicated.
- **Hyperventilation syndrome:** reproducible with hyperventilation; treatment is explanation and reassurance
- **Psychiatric dizziness:** common in anxious and depressed patients; important to try to establish this in the initial history; patients benefit from reassurance and referral to a psychiatrist or internist.
DISPOSITION

- Most patients with peripheral vertigo will be discharged from the ED, with internist or an ENT referral
- Those with central vertigo are dispositioned based on diagnosis
  - Migraine related vertigo: discharge and refer (an illustration that not all central causes are life-threatening)
  - Posterior fossa lesion: brain imaging, consultation with a neurologist and/or neurosurgeon in the ED.
- Some patients will need outpatient (or sometimes inpatient, or in ED) referral or consultation with a cardiologist, ENT specialist or psychiatrist.