Dizziness and Post Traumatic Vestibular Dysfunction

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Introduction

- Balance disorders and dizziness affect most head injury patients
- Vestibular dysfunction contributes significantly to imbalance
- Vertigo is a symptom, not a diagnosis
- Vertigo occurs in up to 75% mild TBI, almost all moderate TBI

Objectives

- Attendees will be knowledgeable about vestibular system function and anatomy
- Attendees will be able to generate a differential diagnosis for the patient who presents with dizziness after head trauma
- Attendees will be able to initiate vestibular evaluation in the office or at the bedside
Vestibular System

Inertial guidance system in all vertebrate animals:

- Peripheral motion detectors
- Central Nervous System structures in brainstem nuclei and cerebellum
- Cortical interaction
Sensory Input
- Vestibular
- Proprioceptive
- Visual

Central Processing
- Vestibular nuclei in the pons
- Connected to ocular motor nuclei in the brainstem
- Connected to vestibulospinal tracts
- Cerebellum, flocculonodular lobe
- Cortex, parietal lobe
Vestibular System Function

- Detects linear and angular acceleration
- Enable perception of orientation in space
- Converts physical stimuli into neural signals which are relayed to CNS for integration with other sensory input
- Postural stability: coordinate head, eye, and trunk movements
- Gaze stability: limit slippage of images on retina during head movement
Vestibular Receptor Anatomy

- Located in the inner ear
- Complex, fluid-filled membranous labyrinth surrounded by bony labyrinth
- Housed in petrous portion of temporal bone
- Paired organ: right and left vestibular labyrinth mirror each other
Linear Acceleration Sensor
Gravitational Sensor

- Utricle: parallel to earth, sensitive in the horizontal plane
- Saccule: perpendicular to the earth, sensitive in the sagittal plane
- Both contain calcium carbonate crystals (otoliths)
Angular Acceleration Sensor

- Three orthogonal semicircular canals
- One in horizontal plane
- Two in vertical plane
- All contain fluid (endolymph)
- All surrounded by fluid (perilymph)

Figure 1-1. Inner ear orientation.
The semicircular tubes are arranged at approximately right angles to each other, in the roll, pitch, and yaw axes.

**No turning**
No sensation.

**Start of turn**
Sensation of turning as moving fluid deflects hairs.

**Constant rate turn**
No sensation after fluid accelerates to same speed as tube wall.

**Turn stopped**
Sensation of turning in opposite direction as moving fluid deflects hairs in opposite direction.
Physics of Mechanotransduction

- $F = Ma$
- Inertial mass is otolith
- $T = Ja$
- Inertial mass is the semicircular canal endolymph
- Hair cells sense force or torque
Vestibular Neuroanatomy
Vestibulo-Ocular Reflex

- Afferent neurons in CN VIII
- Interneurons in the vestibular nuclei
- Efferent lower motor neurons in the nuclei of CN III, IV, VI
- At head velocity < 75 deg/sec, gaze stability is maintained by smooth pursuit
- At head velocity 75-350 deg/sec, VOR is used
- Intact pathway is responsible for oculocephalic reflex (Doll’s eye movement)
Vestibulospinal Reflex

- Afferent neurons in CN VIII
- Vestibulospinal tracts descend from vestibular nuclei
- Efferents are lower motor neurons in CN XI and the spinal cord to influence axial muscles in neck and trunk
- Intact pathway is responsible for extensor activity on the side to which the head is inclined, and flexor activity on the opposite side
Peripheral Vestibular Dysfunction

- Abnormality of vestibular labyrinth or vestibular nerve
- Injury reduces afferent activity from the affected side to the ipsilateral vestibular nucleus
- Unilateral or asymmetric injury results in asymmetric neural activity in brainstem, cerebellum, vestibulo-ocular, and vestibulo-spinal systems
- Asymmetry interpreted as vertigo
Central Vestibular Disorder

- Abnormal processing of normal peripheral vestibular sensory input
Head Trauma and Dizziness

Complaint in up to 90% of all severity head injuries

- Vertigo: peripheral and/or central vestibular dysfunction
- Light-headedness: orthostatic hypotension, arrhythmia, medications
- Hypoglycemia, hypoxia
- Dysequilibrium: peripheral neuropathy, B12 deficiency
- Visual distortion: extraocular muscle dysfunction, cranial nerve injury, new refractive prescription, corneal disease
- Psychophysiologic dizziness: depression, panic attacks
- Persistent post-concussion symptoms
Traumatic Brain Injury and Vertigo

Central causes of vertigo

- Central injury (brainstem, cerebellum, thalamus)
- Post-traumatic migraine
- Epileptic vertigo
Epileptic Vertigo

- Rare cause of vertigo
- May be only manifestation of complex partial sensory seizure
- Seizure focus is temporal or parietal association cortex with vestibular projections
- May have nystagmus if oculomotor nuclei is stimulated
- Usually only mild nausea, no vomiting
- No loss of consciousness unless seizure becomes generalized
Traumatic Brain Injury and Vertigo

Peripheral (otologic) causes of vertigo
- Benign Paroxysmal Positional Vertigo (BPPV)
- Labyrinth concussion
- Temporal bone fracture with vestibular nerve damage
- Ototoxins (phenytoin, gentamycin, vancomycin): vestibular labyrinth more sensitive than cochlea
- Perilymphatic Fistula
- Post-traumatic hydrops (Post-traumatic Meniere’s Syndrome)
Benign Paroxysmal Positional Vertigo

- Most common peripheral cause of vertigo in general population
- Most common peripheral cause of vertigo after head injury, approx 60% Davies RA. J Neurol 1995; 242:222-30
- Otoliths are displaced from utricle membrane into a semicircular canal
- 90% into posterior semicircular canal Honrubia et al. Am J Otol 1999; 20:465-70

Sunday, May 1, 2011
• Otoliths form clumps that move freely within semicircular canal
• Rarely, otoliths adhere to cupula
• Changing head position displaces otolith clump, which in turn displaces cupula
Presentation

- Delayed if otoliths not completely sheared, but only less adherent to utricular membrane
- Paroxysmal vertigo, nystagmus, ataxia lasting seconds to minutes
- Diagnosis made with Dix-Hallpike maneuver: reclining with head rotated $45^\circ$ to one side
- Mixed vertical-torsional nystagmus usually begins after latency of 5-20 seconds and fatigues with repetition
- Nystagmus towards the undermost ear if that is the affected ear
Labyrinth Concussion

- In context of cerebral concussion
- Membranous structures concussed against bony otic capsule
- No structural or bony pathology
- Produces vestibular hypofunction
- Causes severe acute vertigo, nystagmus
Temporal Bone Fracture

- Comprise 22% of skull fxs
  - Cannon C et al Arch Otolaryngol 1983; 109:
  - 285-288
- 80% longitudinal (to axis of petrous bone)
- 20% transverse
- Vestibular/auditory symptoms in up to 95%
- If CN VIII transected or avulsed, no vertigo
- Damage to vestibular labyrinth structures causes severe acute vertigo, nystagmus
Temporal Bone Fracture

- May have associated tympanic membrane tear, sensorineural hearing loss, facial nerve injury
- May have associated otorrhagia, hemotympanum
- May coexist with BPPV, perilymphatic fistula, post-traumatic hydrops
Perilymphatic Fistula

- Rupture of round or oval windows that separate inner and middle ear
- Perilymph from inner ear enters middle ear
- Presents with vertigo, hearing loss
- Symptoms may fluctuate with valsalva
- Suspected cases treated with bed rest 1-2 weeks: most heal spontaneously
- Not identified by diagnostic tests
- Definitive diagnosis by surgical exploration with patching of round or oval windows
Post-Traumatic Hydrops

- Clinically indistinguishable from idiopathic Meniere’s Syndrome
- May present months to years after injury
- Deregulation of endolymphatic fluid leads to excessive accumulation of endolymph
- Auditory symptoms pathognomonic: fluctuating hearing loss, tinnitus, aural fullness
- Episodic vertigo, ataxia, nausea, vomiting lasting minutes to hours
- Managed like Meniere’s with diuretics, dietary salt restriction
Office Tests

- Gait, station
- Stepping test (Fukuda test)
- Oculomotor exam
- Dix-Hallpike maneuver
- Head impulse tests
- Dynamic visual acuity test
- Fukuda: step in place x 30 seconds with eyes closed. Rotation suggests *unilateral* vestibulopathy
- Dix-Hallpike: pt lies back suddenly with head 45 deg to one side, abnl is pt has vertigo and rotatory nystagmus
- Head Impulse (thrust/heave): pt visually focuses on a target, examiner moves pt’s head briskly in plane of paired semicircular canals; test is nl if eyes stay fixed on target, abnl if eyes have to make compensatory movements (refixation saccade) to reacquire the target.

- Dynamic visual acuity: before and during head shaking, asked to read smallest visible line of Snellen eye chart. Normal is ability to maintain acuity w/in 2 lines of the acuity at rest.
Referral Tests

- Electronystagmography
- Vestibular evoked myogenic potentials
- Computerized platform posturography
Electronystagmography

- Battery of vestibular and oculomotor tests based on neurophysiology
- Head motions or vestibular labyrinth stimulation produces compensatory eye movements
- Eye movements recorded with electrodes or video
- Records eye movements in response to vestibular, visual, caloric, rotational, positional stimulation
- Findings can favor diagnosis of central vs peripheral vestibular disorder
Nystagmus after Peripheral Injury

- Caused by reduced tonic activity to the oculomotor nuclei
- Unidirectional
- Fast-phase of nystagmus directed towards the more neurally active side (usually away from the affected side)
- Increases when gaze directed towards lesion
- Suppressed by visual fixation
Nystagmus after Central Injury

- May or may not be present
- May be vertical, torsional, or horizontal
- Not suppressed by visual fixation
Robert Bárány (1876-1936)

- 1914 Nobel Prize in Physiology or Medicine
- Discovered warm and cold calorics
- Produced vertigo and nystagmus (with rapid movement) away from the ear with cool water instillation
Warm water produced nystagmus (with rapid movement toward) the same ear.

Theorized that direction of thermal convective endolymph flow affects the vestibular organ’s “proprioception.”

Absence of response may indicate vestibular nerve damage.
Standard Treatment of Vertigo

- Rehabilitation
- Vestibulosuppressive medication
Vestibular Rehabilitation

- Used in England since 1930s and in U.S. since 1940s
- Developed for brain-injured soldiers
- Improves both peripheral and central vertigo
- Safe and efficacious
Rehab Components

- Maneuvers to liberate otolith from semicircular canal in BPPV
- Habituation exercises to improve vertigo
- Gait and balance training
Liberatory Maneuvers

- Various techniques
- All combine head and trunk movements in a specific sequence
- Cures BPPV in 64-100% of cases

Habituation

- Head movements that produce the most symptoms are the most effective
- Combine head and eye movements
Gait and Balance Training

Visual and proprioceptive information becomes very important
Central Vestibular Compensation

- Normal process of recovery after static peripheral nerve or CNS injury
- Symptomatic improvement does not parallel recovery of peripheral vestibular function
- Vertigo, nystagmus, nausea, vomiting diminish
Better with intact cerebellum, brain stem, spinal, cortical neural pathways

**Hastened by repeated vestibular stimulation**

Most peripheral disorders resolve in 6-12 weeks due to central compensation

Rate of recovery decreases with age
Vestibulosuppressants

- Antihistamines (Phenergan, meclizine)
- Anticholinergics (scopolamine)
- Monoaminergics (ephedrine)
- D2 anti dopaminergics (Thorazine, Haldol)
- Benzodiazepines (Valium)
Indications

- Acute central or peripheral vertigo with nausea and vomiting
- BPPV: only to suppress vomiting during liberatory maneuvers
- Residual central vertigo: long term option after plateau in vestibular rehab
Review

Traumatic brain injury may be associated with
- peripheral vestibular dysfunction
- central vestibular dysfunction
- or both—mixed vestibular dysfunction

Meds for acute symptoms
Rehab for everyone
References