Vestibular and Balance Disorders Workshop

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**Introduction**

- **Balance**

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**Balance Disorders**

* 5-10% of all physician visits; affect ≈40% of adults > 40;
  #1 reason for physician visits in pts > 65

1. **Otologic (≈50%)**
   - Neurologic, Cerebrovascular
   - Cardiovascular, Medication Side Effects
   - Metabolic, Orthopedic, Psychiatric, Ophthalmologic, etc.

2. **Non-Otologic (≈50%)**
Outline

- Anatomy & Physiology of Vestibular System
  - Input
    - Vestibular
    - Visual
    - Proprioceptive
  - Motor Output
    - Movement strategies

- History & Physical Examination

- Laboratory Testing
Organization of the Vestibular System

Sensory Input:
- Visual
- Vestibular:
  - Otolithic Organs (linear)
  - Semicircular Canals (angular)
- Proprioceptive
- Auditory
- Other

Central Processing:
- Cerebral Cortex
- Brainstem Nuclei
  - Primary Processor
  - Vestibular Nuclei
- Cerebellum
  - Adaptive Processor

Motor Output:
- Eye Movements
- Motor Neurons
- Postural Control
Vestibular Inputs

- Semicircular canals
  - Detect high velocity and high frequency angular acceleration
  - Help stabilize gaze during impulsive head mvt
  - Prevent oscillopsia by preventing retinal slip
FIG. 2.42 Polarization of cilia, crista. This sketch illustrates ciliary polarization of the semicircular canals. After H. Spoendlin, University of Innsbruck, Austria.
Ewald’s Laws

- Eye movements occur in the plane of the stimulated semicircular canal (SCC) in the direction of endolymphatic flow.

- Horizontal SCC (HSCC) ampullopetal flow → greater response than ampullofugal flow.
  - HSCC ampullopetal flow → stimulatory (ampullofugal flow → inhibitory).

- In vertical canals → reverse is true.
Eye movements evoked by excitatory stimulation of individual semicircular canals

The arrows depict motion of slow-phase components of nystagmus
Figure 164-5. Alexander’s law. With an acute deficit of the right labyrinth, vestibular input drives the eyes to the right (slow-phase component of nystagmus). A defective neural integrator (caused by the unilateral loss) tends to bring the eyes back to neutral position. With the eyes directed toward the lesion, the two effects cancel. With the eyes directed away from the lesion, they add. (From Hullar TE, Minor LB. The neurologic examination. In: Jackler RK, Brackmann DE, eds. Neurotology. 2nd ed. St. Louis: Mosby: 2004:215-227.)
Physiologic Nystagmus
Pathologic Nystagmus - Acute
Pathologic Nystagmus - Early
Pathologic Nystagmus – Partial Compensation
Pathologic Nystagmus – Full Dynamic Compensation

Diagram illustrating the compensation mechanisms and pathways involved in pathologic nystagmus.
Vestibular Organs

- Otolithic Organs
  - Macula of Utricle & Saccule
    - Sense transient linear acceleration & stabilize body position
    - Sense head/body relation to gravity & set muscle tonus
  - Contribute to
    - Linear VOR
    - Ocular counter roll
    - Ocular skew
Encoding of linear acceleration due to tilt and translation

Roll tilt

Interaural translation

Otolith afferent

Onset of tilt

Onset of translation
Visual Inputs

- **Optokinetic reflex**
  - Sensation of relative visual input/movement(s)

- **Saccades**
  - Visual refixation on target(s)
    - Manifest as quick movements to bring eye back to correct fixation after drifting during nystagmus

- **Smooth Pursuit**
  - Specific tracking of desired target(s)
    - Focus stimulus on fovea
    - Inhibits VOR
Optokinetic Nystagmus

Horizontal Eye Position

Vertical Eye Position

Horizontal Eye Position

Vertical Eye Position
Saccades

NEUROSCIENCE, Fourth Edition, Figure 20.11
Smooth Pursuit

[Graph showing eye movement over time for different speeds (10°/s, 15°/s, 20°/s). The graph includes labels for eye movement, target movement, and catch-up saccade.]
Somatosensory Inputs

- Pressure receptors
  - Sense interaction w/ support surface

- Stretch receptors
  - Sense joint alignment and muscle stretch/tension
Central Sensory Integration

- Comparison of Sensory Inputs
- Context – Dependent Hierarchy
- Generate appropriate command → motor system for posture control
Motor Output

- Pyramidal
- Extrapyramidal
- Multisegmental reflex loops
- Muscle conditioning
Movement Strategy

- Ankle Strategy
- Hip Strategy
- Stepping Strategy
Summary

- Balance and Gaze Stability
  - Rely on combination of sensory inputs, central integration and motor output
- VOR
  - Stabilizes gaze during rapid head movement(s)
- VSR
  - Stabilizes posture during transient lineal accelerations or gravity
- Adaptive plasticity and sensory substitution essential to compensation following vestibular injury
Evaluation of Dizzy Patient

- History (70%)

- Physical Exam (10-20%)

- Laboratory Testing (10-20%)
  - ENG/VNG
  - VEMP testing
  - Rotary chair
  - Posturography
  - Other
History

- Questionnaire
- Interview w/ questionnaire as guide
- Focused
  - W/ differential diagnosis in mind
A series of focused questions will differentiate the nature of the pathology in most patients.


<table>
<thead>
<tr>
<th>QUESTION</th>
<th>CLINICAL INFORMATION</th>
</tr>
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<tbody>
<tr>
<td>What does it feel like?</td>
<td>• Is this vestibular / labyrinthine (vertigo) or something else (presyncope, syncope, seizure)</td>
</tr>
</tbody>
</table>
| What other symptoms are associated with it? | • Declining hearing after head trauma (EVA)  
• Tinnitus, hearing loss (hydrops)  
• Dysarthria, diplopia, paresthesias (vertibrobasilar disease)  
• Cranial nerve weakness (skull base, intracranial lesions)  
• Headache, paroxysmal torticollis (migraine, BRVC)  
• Sweating, palpitations, dyspnea (orthostasis, panic, attacks) |
| How long do the symptoms last and how many have occurred? | • Seconds – minutes (BPPV)  
• Hours (TIA, migrant, hydrops)  
• Days – weeks (labyrinthitis, vestibular neuritis) |
| What makes it better or worse? | • Vestibular generated vertigo always worse with movement  
• Rolling, bending (BPPV)  
• Valsalva (PLF) |
| What is the background history? | • Otologic disease (PLF, labyrinthitis, BPPV)  
• SNHL (syndromic/ non-syndromic / congenital vs. acquired /), ototoxic medications, congenital or acquired vestibular hypofunction  
• Neuropathies (peripheral neuropathy)  
• Vascular disease (congenital cardiopulmonary disease, von Hippel-Lindau with, intracranial vascular lesions)  
• Family history neoplasms (NF-2, Gorlin’s syndrome, Costello Syndrome) (acoustic neuroma, medulloblastoma)  
• Anxiety/depression (panic attacks)  
• Motion intolerance (migraine)  
• Family history of balance disorders (periodic ataxias, migraine, hereditary vestibulopathy)  
• Autoimmune disease (autoimmune inner ear disease)  
• Seizure history (temporal lobe seizures)  
• Ophthalmologic disease (Oculomotor anomaly, amblyopia, disorders of acuity, depth perception) |
<table>
<thead>
<tr>
<th>Symptom</th>
<th>Subtype</th>
<th>Likely Cause</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Vertigo</td>
<td>Position-induced</td>
<td>BPPV</td>
<td>If nystagmus does not match BPPV, consider central pathologies. If induced by neck rotation, consider cervical vertigo</td>
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<td></td>
<td>Acute-onset persistent</td>
<td>Stroke</td>
<td>Acute ischemia involving vestibular structures can mimic vestibular neuritis</td>
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<td></td>
<td>with neurologic signs</td>
<td>Tumors</td>
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<td></td>
<td>Acute-onset persistent</td>
<td>Degenerative diseases</td>
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<tr>
<td></td>
<td>without neurologic signs</td>
<td>Labyrinthitis</td>
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<td></td>
<td>Recurrent with no neurologic</td>
<td>Vestibular neuritis</td>
<td>Differential diagnosis is based on presence of hearing loss</td>
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<tr>
<td></td>
<td>signs</td>
<td>Menière's disease</td>
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<td>Migraine</td>
<td>Late-onset Meniere's is possible but not common. Migraines lack progressive auditory symptoms. Transient ischemic attacks should be considered in patients with vascular risk factors</td>
</tr>
<tr>
<td>Disequilibrium</td>
<td>Acute or rapidly progressive</td>
<td>Stroke</td>
<td>Autoimmune or postinfectious diseases should also be considered. May include severe oculomotor abnormalities. Usually includes history of ototoxicity. Hearing loss or oscillopsia may be present</td>
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<td>Worse in the absence of other</td>
<td>Bilateral vestibular loss</td>
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<td>sensory inputs</td>
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<td>Worse in the absence of vision</td>
<td>Proprioception and</td>
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<td>with numbness/weakness</td>
<td>somatosensory loss</td>
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<td>With bradykinesia, rigidity,</td>
<td>Parkinson disease</td>
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<td>tremor</td>
<td>Cerebellar lesions</td>
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<td>With speech disorder, lack of</td>
<td>Disequilibrium of aging</td>
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<td>coordination, intention tremor</td>
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<td>Isolated disequilibrium, gait</td>
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<td>difficulty, lightheadedness</td>
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<td>Presyncope</td>
<td>With blood pressure drop on</td>
<td>Postural hypotension</td>
<td>Associated with reduced blood volume, autonomic disorders, or chronic use of hypertension medications. When 24-h electrocardiogram is abnormal, indicates transient arrhythmia. When 24-h electrocardiogram is abnormal, indicates transient arrhythmia.</td>
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<td>standing</td>
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<td>Abnormal cardiac examination</td>
<td>Heart valve disease</td>
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<td>Induced by fear or anxiety</td>
<td>Arrhythmia</td>
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<td></td>
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<td>Vasovagal attacks</td>
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<tr>
<td>Lightheadedness,</td>
<td>Associated with fear, anxiety,</td>
<td>Psychogenic</td>
<td>Often accompanied by autonomic symptoms</td>
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<tr>
<td>nonspecific</td>
<td>depression</td>
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Components of Physical Exam

- **VOR Exam**
  - Spontaneous nystagmus, Head-thrust, Head – shake, Frenzel’s lenses, dynamic visual acuity

- **Dix-Hallpike**

- **Central Occulomotor Exam**
  - Smooth pursuit, Gaze-evoked nystagmus, Saccades, fixation suppression

- **VSR Exam**
  - Romberg, Tandem Romberg, Foam Romberg

- **Gait**

- **Station (Posture)**
Spontaneous Nystagmus

- Eyes open (visual fixation) v Frenzel lenses (fixation removed)
- Direction fixed v direction changing
- Pendular v jerk nystagmus
- Saccadic intrusions
Left beating nystagmus

Right beating nystagmus
Figure 1. Spontaneous nystagmus in a patient with a recent right peripheral vestibular lesion. Bitemporal leads; upwards pen deflection denotes eye movement.
Classification of Nystagmus - Shape

Jerk Nystagmus

Linear Slow-Phase

Increasing Velocity Slow-Phase

Decreasing Velocity Slow-Phase

Pendular Nystagmus

Torsional Nystagmus
Slow Component of Nystagmus

Utricle

Lateral Canal

Utriculofugal Stimulation of Crista

Cold Water Irrigation
Alexander’s Law

- 1\textsuperscript{st} degree nystagmus
  - Present only in gaze towards fast phase

- 2\textsuperscript{nd} degree nystagmus
  - Present in neutral gaze and stronger towards fast phase

- 3\textsuperscript{rd} degree nystagmus
  - Present in all gazes, strongest in gaze towards fast phase
Gaze Nystagmus

- Drift of eye which is only present for certain directions of gaze
  - Inability to maintain stable conjugated eye deviation away from primary position
- Direction changing
- Failure to maintain eccentric gaze
  - Leaky neural integrator

- Cerebella flocculus

- Common consequence of medication, especially sedatives or anticonvulsants
Smooth Pursuit

- Smooth movement continual tracking of moving object
  - Keep image of an attended moving target near fovea

- Best corrected vision

- Horizontal better than vertical

- Mediated by paramedian pontine reticular formation
  - Frontal eye field region, Cerebellum
Saccades

- Saccadic eye movements (SACs) shift fovea rapidly to a peripheral visual or auditory target

- Conjugate

- Accurate

- Fast

- Minimal delay
Optokinetic Reflex

- Full field retinal stimulation
- Optimal function
  - Large, slow, repetitive target motion
- Augments low frequency VOR
- Non-voluntary reflex
Head – thrust Testing

- Rotate from side to center (or center to side)
- Focus on target (nose)
- Look for corrective saccade(s)
- Can be unilateral or bilateral (if bilateral – suspect bilateral weakness)
Head-shake Testing

- 1-2 Hz for 20 seconds
  - Removal of visual fixation (Frenzel Lenses)
  - Observe for post–headshake nystagmus
  - (usually) beats toward stronger ear
  - Observe for cross-coupling and reversal
Dynamic Visual Acuity

- Normal
  - < 3 line decrease in visual acuity

- Abnormal
  - 3 or more line decrease in visual acuity

- Aphysiologic
  - Decrease w/ torsional mvt
Position Testing

- Rapid Positioning
  - Dix-Hallpike, Bjorab Maneuver, etc.
    - Latency, duration, fatigue, reversal

- Static Positional (Prolonged Positioning) Testing
  - Removal of visual fixation – Use Frenzel lenses
Additional Testing

- Pneumatic otoscopy
- Sound stimulation (Barany Noise Box, etc.)
- Valsalva
- Hyperventilation
- Mastoid oscillation
Limb Testing

- Finger – nose – finger
- Heel to shin
- Rapid alternating movements
- Joint proprioception
- Vibration detection
Posture and Gait

- Romberg
  - Eyes open, Eyes closed
    - Foam balance pad

- Tandem/Sharpened Romberg

- Fakuda Step testing

- Gait analysis
Lab Testing for Vertigo

- VNG
- VEMP
- Rotary Chair
- Posturography