Benign Paroxysmal Positional Vertigo: Diagnosis and Management  
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I have no relevant financial relationships to disclose  
and  
I will not discuss products in my presentation  

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• CCSU Faculty  
• Dr. Dan Roberts  
  – Neurologist  
  – UConn/CT ENT/HH  
  – Slides and images
Overview

- Dizziness vs. Vertigo
- Vertigo: Central vs. Peripheral
- Case Studies
- Anatomy (Inner Ear)
- Workup for BPPV
- Treatment for BPPV
- Prognosis/Return to function

Balance Disorders in the Elderly: Epidemiology and Functional Impact

> 65 yrs, 19.6% reported a problem with dizziness or balance in the preceding 12 months.

Balance problems:
- Unsteadiness (68.0%),
- Walking on uneven surfaces (54.8%),
- Faintness (29.6%).

30.9% of elderly persons with balance problems who sought care, 85.6%, 30.3%, 23.9%, and 16.8% saw a general practitioner, internist, neurologist, or otolaryngologist, respectively.

Lin and Bhattacharyya, 2012

Dizziness

- CC: “Dizzy”
- Describe the feeling without using the word “dizzy”
  - Imbalance/unsteady
  - Lightheaded/faint
  - Foggy
  - Wavy
  - Spinning (!)
Dizziness

• General Categories (percentages = “all comers”)
  – Vertigo (50%)
    • False sense of motion, SPINNING
  – Dysequilibrium (15%)
    • Imbalance, unsteady, wobbly while standing/walking
  – Presyncope (15%)
    • Transient, tending passing/blacking out coming
  – Lightheadedness (10%)
    • Vague, disconnected with environment
  – Other/Combined (10%)

Dizziness vs. Vertigo

• Vertigo
  – Involves a hallucinatory sense of motion
  – Spinning
  – Maybe nausea/vomiting
  – “Tilt-a-Whirl”

Vertigo

• Central
  – Migraines
  – Multiple Sclerosis
  – Mal de Debarquement
  – Cerebellar Hemorrhage/Infarct
  – Vertebrobasilar Insufficiency
  – Neoplasms
  – Systemic
  – OTHER!

• Peripheral
  – Benign Paroxysmal Positional Vertigo (BPPV)
  – Ménière’s Disease
  – Labyrinthitis
  – Ototoxicity
  – Superior Canal Dehiscence Syndrome
  – EARS!!
Peripheral Vertigo

- **DURATION** of spinning (not aftermath)
  - Seconds/Minutes (BPPV, Perilymphatic fistula)
  - Hours (Vestibular Migraines, Hydrops)
  - Days (Labyrinthitis, Neuronitis)
  - Months (Trauma, Neoplasm, Ototoxicity)

- **Associated Symptoms**
  - Hearing loss, Tinnitus, Pressure, Drainage, Sinusitis

- **Motion/Fixation**
  - Effects of head position change and eye fixation

Case #1

- 55 yo Athletic Trainer/Nurse/Physician/Coach
- Wakes abruptly at 4:30am
- Hits “snooze” – world goes rotational for 90sec, then “dizzy” for days
- NO HL, tinnitus, pain
- Resolves if head still
- Afraid to leave the bed/house or drive

Case #2

- 19 yo Swimmer
- Returns from training trip, c/o vertigo
- Feels like “still on the boat.”
- Some “popping” in ears
- Hearing muffled
Cases

- BPPV
  - Lasts seconds to minutes
  - NO hearing loss
  - NO tinnitus
  - NO drainage
  - NO pain
  - Stops with fixation
  - Worse with head position changes
  - Comes in waves (weakens)

Robert Bárány (1876-1936)

Evaluation/Testing

- Vestibular Anatomy & Physiology
- Clinical Exam
- VNG
- Rotatory Chair
- Posturography
- VEMP
**Anatomy**

- **Utricle:**
  - Detects motion in the horizontal plane (forward-backward, left-right)

- **Saccule:**
  - Senses motion in the sagittal plane (up-down)

**Otolith Organs**

**Semicircular Canals**

- Detect angular acceleration.

- **Anatomy:**
  1. Coplanar pairs
  2. Ampulla
  3. Cupula
  4. Ampullopetal vs ampullofugal endolymph flow
Vestibulo-ocular reflex (VOR)

- 3 neuron arc designed to stabilize images on the retina during head movements.
- Works by generating slow-phase eye movements that are equal and opposite to head movements.

Figure source: Furman JM, Cass SP. Vestibular Disorders: A Case Study Approach, 2nd ed. © 2003.

CLINICAL EXAM

Peripheral vs. Central Nystagmus

<table>
<thead>
<tr>
<th>Peripheral</th>
<th>Central</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direction fixed horizontal or torsional nystagmus</td>
<td>Vertical nystagmus (upbeating or downbeating)</td>
</tr>
<tr>
<td>Suppression of nystagmus with fixation</td>
<td>No suppression of nystagmus with fixation</td>
</tr>
<tr>
<td>Follows Alexander's law</td>
<td>Direction changing nystagmus in neutral position including:</td>
</tr>
<tr>
<td></td>
<td>(1) congenital nystagmus</td>
</tr>
<tr>
<td></td>
<td>(2) periodic alternating nystagmus</td>
</tr>
<tr>
<td>Fast phase toward intact ear except:</td>
<td>May be present as a result:</td>
</tr>
<tr>
<td>(1) Irritative nystagmus</td>
<td>(1) Pharmacy</td>
</tr>
<tr>
<td>(2) Recovery nystagmus</td>
<td>(2) Alcohol</td>
</tr>
<tr>
<td></td>
<td>(3) Tobacco</td>
</tr>
</tbody>
</table>
Spontaneous Nystagmus

- Observe for nystagmus, repeat with Frenzel lenses.

**Interpretation**
- Normal: no nystagmus
- Peripheral: spontaneous, direction-fixed, horizontal rotary jerk nystagmus with fast phase away from affected ear and enhanced with gaze in direction of fast phase or when wearing Frenzel lenses
- Central: direction-changing horizontal, purely vertical or torsional, or pendular nystagmus that is diminished under Frenzel lenses (loss of visual fixation)

Graboyes and Goebel, 2015

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Gaze Evoked Nystagmus

- Finger 30 deg from nose, pt fixates on examiner finger
- If nystagmus observed with fast phase in direction of gaze, hold for 30 seconds then refixate on finger in center position

Graboyes and Goebel, 2015

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Gaze Evoked Nystagmus

**Interpretation**
- Normal: transient gaze-evoked nystagmus at 30 degrees
- Peripheral: direction-fixed most obvious with gaze in direction of fast phase (Alexander’s law)
- Central: direction-changing
- Brun nystagmus: gaze-dependent, direction-changing nystagmus that is combination central and peripheral due to CPA mass compressing flocculus

Graboyes and Goebel, 2015
Saccades

Head stationary, examiner holds fingertip 15 degrees off midline in horizontal plane; patient looks back and forth between examiner’s nose and fingertip.

Interpretation
- Normal: conjugate, accurate, fast.
- Peripheral: normal
- Central lesion:
  - Disconjugate: medial longitudinal fasciculus lesion or MS.
  - Inaccurate: midline cerebellum lesion
  - Decreased velocity: cortical and brainstem disease
    - Progressive supranuclear palsy, olivopontocerebellar atrophy/spinocerebellar ataxia

Smooth Pursuits

- Head stationary, examiner positions finger in front of patient and moves target at 30-40 degrees/second in horizontal plane.
- Repeat for vertical plane.
- Restrict testing to 60 degrees of visual field (30 degrees left and right, up and down) to avoid GEN.

Interpretation
- Normal: accurate tracking without corrective saccades.
- Peripheral: normal.
- Central lesion: impaired or absent tracking with catch-up saccades.
  - Medications: anticonvulsants, sedatives, EtOH.
  - Parkinson’s disease, Alzheimer’s disease, supranuclear degeneration, cerebellar degeneration.
Headshake test

- Head tilted 30 degrees forward (plane of horizontal SCC) +/- Frenzel lenses
- Examiner rotates patient’s head frequency (>2Hz), low amplitude (20-30 degrees) movement x 20 seconds
- Examiner brings head to abrupt stop

Interpretation:
- Normal: no post-headshake nystagmus
- Unilateral vestibular hypofunction: horizontal, direction-fixed, post-headshake nystagmus with fast-phase away from affected ear
- Bilateral symmetric vestibular hypofunction: no post-headshake nystagmus
- Central lesion: normal, cross coupled nystagmus (vertical nystagmus after horizontal headshake), direction-reversing nystagmus.

Dynamic Visual Acuity Test

- Visual acuity (VA) measured (Snellen chart)
- Examiner rotates patient’s chair (or head) in horizontal plane continuously, without pausing, at 2 Hz oscillations while visual acuity is assessed (by patient reading Snellen eye chart)

Interpretation:
- Normal decrease in VA < 2 lines on Snellen chart
- Unilateral vestibular hypofunction: normal or decrease in VA > 3 lines on Snellen chart
- Bilateral vestibular hypofunction: decrease in VA > 3 lines on Snellen chart
- Central lesion: variable
Vestibuloocular Reflex (Head Impulse Test - “Head Thrust”)

- Head tilted 30 degrees forward
- Head turned while patient fixates on examiner’s nose
- Examiner thrusts head rapidly towards midline while patient maintains fixation
- Repeat x 5-10 times per side to document repeatable fixation failure and refixation saccades

Graboyes and Goebel, 2015

Vestibuloocular Reflex (Head Impulse Test) - “Head Thrust”

**Interpretation:**

- Normal: eyes remain fixated on visual target with no corrective saccades
- Unilateral vestibular hypofunction: corrective saccade with angular head movement towards affected ear

Graboyes and Goebel, 2015

Position Tests

- Place head in left/right Hall pike, left/right lateral, supine
  - Normal: no nystagmus
  - Abnormal: torsional geotropic nystagmus (BPPV), horizontal sustained nystagmus (central or peripheral)

Aural Pressure/Sound Test

- Stimulate ear with positive pressure, loud sound, mastoid vibration
- Normal: no nystagmus
- Abnormal: upward deviation or downbeating nystagmus (SSCD), Horizontal nystagmus (perilymphatic fistula)

Cerebellar Limb Tests

- Finger to nose
- Heel-shin
- Rapid alternating motion
  - Normal: accurate movements
  - Abnormal: dysmetria, dysdiadochokinesia (central)

Posture Tests

- Romberg
- Tandem Romberg
- Foam
  - Normal: minimal sway
  - Abnormal: sway/falls
Cerebellar Limb Tests

- Finger to nose
- Heel-shin
- Rapid alternating motion
  - Normal: accurate movements
  - Abnormal: dysmetria, dysdiadochokinesia (central)

Gait Tests

- Observation
- Fukuda Step Test
  - Normal: <45° rotation on Fukuda
  - Abnormal: wide based gait/ataxic/Parkinsonian; >45° rotation on Fukuda

Testing
VESTIBULAR FUNCTION TESTS

Why order formal vestibular lab tests?

1. To assist diagnosis.
   - Confirm certain provisional diagnoses
   - When diagnosis is uncertain after a thorough history and exam.
2. To quantify degree of peripheral vestibular loss.
3. To provide information regarding degree of compensation.
4. Prior to ablative procedures to ensure normal function on the contralateral side.

Limitations of Vestibular Testing

- It does not measure degree of disability. Patients with similar results can have different functional capacities.
- The standard test battery does not assess function of the vertical canals or otolith organs.
What are we testing?

- VOR (VNG, Rotatory chair)
- VCR (VEMP)
- Central tracking & visual acuity (pursuit, saccade test, OKN test)
- Sensorimotor integration (Posturography)

ENG/VNG

1. Spontaneous nystagmus (with and without fixation)
2. Gaze-evoked
3. Headshaking nystagmus
4. Saccade, smooth pursuit
5. Positional testing (Hallpike)
6. Calories (with test of fixation)

ENG/VNG-Techniques for Recording Eye Movements

<table>
<thead>
<tr>
<th>Variable</th>
<th>EOG / ENG</th>
<th>VOG / VNG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spatial resolution</td>
<td>1 degree</td>
<td>1 degree</td>
</tr>
<tr>
<td>Temporal resolution</td>
<td>60 Hz</td>
<td>60 Hz</td>
</tr>
<tr>
<td>Vertical recording</td>
<td>Possible but confounded by eyelink</td>
<td>Good (can view video of torsional eye movements)</td>
</tr>
<tr>
<td>Setup</td>
<td>Slow</td>
<td>Fast</td>
</tr>
<tr>
<td>Cost</td>
<td>Low</td>
<td>Moderate</td>
</tr>
</tbody>
</table>

VNG: Caloric Testing

Involves irrigation of the EAC with a medium of significantly different temperature compared with core body temperature.

Open-loop water: 44°C / 30°C for 40 seconds.
Closed-loop water: 44°C / 27°C for 40 seconds.
Open-loop air: 50°C / 24°C for 60 seconds.

Pros:
1. Each labyrinth can be evaluated independently.
2. Inexpensive equipment.

Cons:
1. Caloric stimuli are not calibrated.
2. Tests only lateral semicircular canal function.
3. Low-frequency stimulation of vestibular system (~ 0.003 Hz).

Caloric Testing

Caloric Data: Key Formulas

Jongkee’s formula:
- Compares caloric responses from the right ear to left ear.
- Remember to look at slow component velocity values too.

\[
RVR = \frac{UW\%}{100} = \frac{(RW+RC)-(LW+LC)}{(RW+RC+LW+LC)} \times 100 > 25\% \text{ is abnormal}
\]

Directional Preponderance:
- Compares the amount of RB nystagmus vs. LB nystagmus generated during caloric testing.

\[
DP\% = \frac{tot \ RB - tot \ LB}{tot \ RB + tot \ LB} = \frac{(RW+LC)-(LW+RC)}{(RW+LC)+(LW+RC)} \times 100 > 30\% \text{ is abnormal}
\]
Rotary Chair Testing

Rotary Chair

Pros:
1. Rotation is a natural stimulus
2. Well-tolerated
3. Precisely controlled
4. Can be used for serial evaluations

Cons:
1. Tests only lateral SCCs
2. Expensive equipment
3. Non-lateralizing

Figure source: www.NDBC.com

Goebels, 2008

Indications for Rotary Chair Testing

- Test of choice for bilateral peripheral vestibular hypofunction.
- Calorics suggesting b/l paresis
- When the degree of compensation is desired
  - Establish baseline to follow
- When caloric information alone is inconclusive or inconsistent
  - Well compensated lesion on VNG but + clinical sx
  - Calorics may be normal
  - Rotational testing higher sensitivity for peripheral disorders, higher specificity than calorics

Goebels et al., 2008

Rotational Chair

Measure SLOW component eye velocity (SCEV) in response to angular acceleration

• Gain = ratio of peak eye velocity/peak chair velocity
  • Significant gain reduction in bilateral vestibular disorders
• Phase = temporal shift in eye velocity relative to head velocity.
  • Increased phase lead suggest vestibular disorder
• Asymmetry = Compares slow phase eye movement between right and left rotation.

Goebels et al., 2008
Computerized Dynamic Posturography (CDP)

- Is a technique used to quantify and differentiate among the variety of sensory, motor, and central adaptive impairments to balance control.
- It does not provide site-of-lesion info.
- Three protocols:
  1. Sensory Organization Test
  2. Motor Control Test
  3. Adaptation Test

Figure source: www.NBCD.com

Vestibular Evoked Myogenic Potentials (VEMP)

- The vestibular system generates muscle potentials in response to auditory and vibration stimuli
- Loud noise presented
- Record EMG from SCM (c-VEMP) or infraorbital muscles (o-VEMP)

Agrawal, 2015
c-VEMP

Sound stimulates the saccule, which activates the inferior vestibular nerve, lateral vestibular nucleus, medial vestibulospinal tract ipsilaterally, and then the sternocleidomastoid muscle in the neck.

o-VEMP Pathway

Can also record oVEMP in response to tap vibration, e.g. with a reflex hammer.

oVEMPs better to assess for dehiscence in the setting of conductive hearing loss, because stimulus bypasses middle ear.

<table>
<thead>
<tr>
<th>VEMP Type</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Click oVEMP</td>
<td>94%</td>
<td>100%</td>
</tr>
<tr>
<td>Tone-burst oVEMP</td>
<td>100%</td>
<td>100%</td>
</tr>
</tbody>
</table>

oVEMP amplitudes in response to ACS are superior to cVEMP thresholds in the diagnosis of SCDS. oVEMPs in response to ACS offer an excellent one-step screening for SCDS before CT imaging.
Clinical Utility

<table>
<thead>
<tr>
<th>Vestibular Disorder</th>
<th>Total n</th>
<th>Absent n</th>
<th>Amp</th>
<th>Threshold</th>
<th>Abnormal n</th>
<th>Normal n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meniere’s Dz</td>
<td>120</td>
<td>63</td>
<td>13</td>
<td>39</td>
<td>158</td>
<td>162</td>
</tr>
<tr>
<td>Vestibular Schwannoma</td>
<td>306</td>
<td>155</td>
<td>61</td>
<td>0</td>
<td>238</td>
<td>68</td>
</tr>
<tr>
<td>SSCD</td>
<td>64</td>
<td>0</td>
<td>0</td>
<td>64</td>
<td>64</td>
<td>0</td>
</tr>
<tr>
<td>Tullio</td>
<td>13</td>
<td>13</td>
<td>0</td>
<td>12</td>
<td>12</td>
<td>1</td>
</tr>
<tr>
<td>VN/Labyrinthitis</td>
<td>99</td>
<td>44</td>
<td>5</td>
<td>0</td>
<td>49</td>
<td>50</td>
</tr>
<tr>
<td>SNHL</td>
<td>46</td>
<td>1</td>
<td>2</td>
<td>42</td>
<td>42</td>
<td>50</td>
</tr>
</tbody>
</table>

Adapted from Akin & Murnane, 2008.

Testing Summary

Clinical exam mainstay of vestibular evaluation

Clinical exam supported by vestibular tests
- Assist diagnosis
- Quantify degree of peripheral vestibular loss
- Prior to ablative procedures to ensure normal function on the contralateral side

Treatment

- Short Term (Crisis)
  - Meclizine/Antivert
  - Antihistamines
  - Valium
- Ideal – Epley Maneuvers
- Observation (1-2 Mos.)
- Vestibular Rehabilitation
  - Habituation
  - Gaze Stabilization
  - Balance Training

www.med.unc.edu/ent
Treatment

- Aftercare (Epley)
  - Head over heart (no bend) – 24 hours
  - Do NOT sleep on trigger side – several days/week
  - May return w/in few weeks, requiring re-Rx
  - 50% recur later in life
  - Move SLOWLY, fix eyes during attacks
  - Avoid bright lights, driving, heights, etc…

Bibliography

Akin FW, Murnane OD. “Ch. 18: Vestibular Evoked Myogenic Potentials” in Balance Function Assessment and Management. Eds. Jacobson GP and Shepherd NT. (pp. 425).