Clinical Management of lateral (horizontal) Canal Benign Paroxysmal Positional Vertigo (BPPV)

Anne K. Galgon PT, PhD, NCS
Wendy Webb Schoenewald PT OCS
Janene Holmberg PT, DPT

OBJECTIVES:

• Upon completion of this course, the participant will be able to:
  • Apply knowledge of the pathophysiology of lateral Canal BPPV to interpreting the clinical presentation of geotropic and apogeotropic lateral canal BPPV.
  
  • Utilize observation skills in selected test and measures to make appropriate intervention choices for individuals with lateral canal BPPV.

  • Recognize nystagmus patterns that are not consistent with lateral Canal BPPV and determine when a referral to another health professional is appropriate.
Outline of Program

I. Introduction to session

II. Presentation of current research on Lateral Canal BPPV
   i. Physiology and Pathophysiology
   ii. Examination and Diagnostic Issues
   iii. Intervention: alternative approaches

III. Clinical Decision making
   I. Case study presentation I
   II. Case study presentation II
   III. Case study presentation III
   IV. Case study Presentation IV

Labyrinth Orientation

http://otocore.wustl.edu/portals/10/Bohne%20eDocs/ANATOMY%20OF%20HUMAN%20TEMPORAL%20BONE.pdf

Physiologic principles for exciting and inhibiting hair cells in vestibular system

Who is Ewald and why does he matter?

- Ewald’s Laws of labyrinthe Function

  1st: The axis of nystagmus parallels the anatomic axis of the SSC that generated it

  2nd: Ampullopetal endolymphatic flow produces a stronger response than ampullofugal flow in the lateral canals

  3rd: The opposite is true of the vertical canals.
Ewald’s 2nd law

• Excitation-inhibition asymmetry of the lateral (horizontal) Semicircular canals

• Rotation toward the excitatory direction will elicit a greater response
  • Higher afferent firing rate

• Rotation toward the inhibitory direction will elicit less of a response
  • Can only go to zero firing rate

Physiology of BPPV

• Otoconia dislodge from the utricle and become displaced into SCC

• Results in SCC becoming sensitive to gravity
  ➢ Vertigo
  ➢ Nausea
  ➢ Disequilibrium

https://www.verywell.com/benign-paroxysmal-positional-vertigo-bppv-1048316
Physiology of BPPV

- **Canalithiasis:**
  - The otoconia are free floating in the canals within the endolymph.

- **Cupulolithiasis:**
  - The otoconia are adherent to the cupula of one of the SCC.

![Mechanisms of benign paroxysmal positional vertigo (BPPV).](image)

(Galgon & Anderson, 2013)

---

**Percentage of Canal Involvement**

<table>
<thead>
<tr>
<th>Reference</th>
<th>N</th>
<th>PC (%)</th>
<th>AC (%)</th>
<th>LC (%)</th>
<th>Other findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Steenerson et al, 2005</td>
<td>923</td>
<td>91%</td>
<td>NR</td>
<td>9%</td>
<td>Torsional nystagmus in Dix-Hallpike identified as PC BPPV; 4 of CRM, 2.88, PC 3.34 and HC (log roll) 3.1</td>
</tr>
<tr>
<td>Jackson et al, 2007</td>
<td>260</td>
<td>66.9%</td>
<td>21.2%</td>
<td>11.9%</td>
<td>Head trauma with 36.4% of AC, but only 9-10% in other canals of CRM to treat the same for all canals: 1.32 AC, 1.49 PC and 1.34 HC</td>
</tr>
<tr>
<td>Dlugaczyk et al, 2011</td>
<td>74</td>
<td>87.8%</td>
<td>6.8%</td>
<td>10.8%</td>
<td>Idiopathic 85% and post traumatic 15%; Post traumatic group incidence of AC or combined AP/PC was higher than idiopathic group</td>
</tr>
<tr>
<td>De Stefano et al, 2011</td>
<td>412</td>
<td>70.87%</td>
<td>2.44%</td>
<td>26.99%</td>
<td></td>
</tr>
<tr>
<td>Tusa &amp; Herdman, 2007</td>
<td>200</td>
<td>76%</td>
<td>13%</td>
<td>5%</td>
<td>Undecided in 6%</td>
</tr>
<tr>
<td>Korres et al, 2007</td>
<td>204</td>
<td>80%</td>
<td>3%</td>
<td>9.3%</td>
<td>7.8% multiple canals</td>
</tr>
<tr>
<td>Vannucchi &amp; Pecci, 2010</td>
<td>471</td>
<td>65%</td>
<td>4%</td>
<td>19%</td>
<td>12% multiple canals</td>
</tr>
</tbody>
</table>
Galgon, Tate, Webb Schoenewald, 2014
• Retrospective chart review of 240 cases

(Supine or Head) Roll Test

for Lateral (horizontal) Canal BPPV
• Patient is laid supine and the head is then quickly turned to one side—held for 30-60 sec then return to start position. Then turn the head quickly to opposite side for 30-60 sec.
• Look for symptoms and horizontal nystagmus

➢ Geotropic nystagmus—canalithiasis
  ➢ Right roll/right beating - Left roll/left beating
➢ Apogeotropic nystagmus—(cupulolithiasis?)
  ➢ Right roll/left beating – Left roll/right beating

No specificity/sensitivity
Generally Consider Standard test
Bhattacharyya et al, 2008; Yetiser & Ince, 2015
4 Potential Pathophysiological States in Lateral Canal BPPV

CU: Cupulolithiasis
CUu: Utricular Side
CUc: Canal Side
CA: Canalithiasis
CAa: Anterior Arm
CAp: Posterior Arm

Vannucchi & Pecci, 2010; Riga et al, 2013

Sit to Supine and Roll Test Responses

<table>
<thead>
<tr>
<th>Left Roll</th>
<th>Supine</th>
<th>Right Roll</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CU:</strong> Cupulolithiasis</td>
<td><strong>CUu:</strong> Utricular Side</td>
<td><strong>CUu:</strong> Utricular Side</td>
</tr>
<tr>
<td><strong>CUc:</strong> Canal Side</td>
<td><strong>CUc:</strong> Canal Side</td>
<td><strong>CUc:</strong> Canal Side</td>
</tr>
<tr>
<td><strong>CA:</strong> Canalithiasis</td>
<td><strong>CA:</strong> Canalithiasis</td>
<td><strong>CA:</strong> Canalithiasis</td>
</tr>
<tr>
<td><strong>CAa:</strong> Anterior Arm</td>
<td><strong>CAa:</strong> Anterior Arm</td>
<td><strong>CAa:</strong> Anterior Arm</td>
</tr>
<tr>
<td><strong>CAp:</strong> Posterior Arm</td>
<td><strong>CAp:</strong> Posterior Arm</td>
<td><strong>CAp:</strong> Posterior Arm</td>
</tr>
</tbody>
</table>

CAp: Geotropic < 1 minute More intense
CAa: Apogeotropic < 1 minute Less intense
CU: Geotropic > 1 minute Less intense
CUu: Apogeotropic > 1 minute More intense

Ewald’s second law
The system is excited when otoconia moving toward ampula → more response
The system is inhibited when otoconia are moving away from ampula → less response
Issues with Testing for Lateral Canal BPPV

- Symmetrical Intensity of nystagmus and symptoms in Supine Roll Test.
  - 11.5-16 % of LC BPPV cases (Lee et al, 2007, Lee et al, 2010, Choung et al, 2007)

- If side is not determined, direction of CRT/ CRM will be a guess

- ↑# of maneuvers/ length of intervention/ duration of symptomatic

- Other tests that may help identify side of involvement

Summary of additional tests to determine side of involvement

<table>
<thead>
<tr>
<th>Form of LC BPPV</th>
<th>Positional test</th>
<th>Direction of horizontal nystagmus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Geotropic (Canalithiasis)</td>
<td>Spontaneous (pseudo) nystagmus</td>
<td>Away from side of involvement</td>
</tr>
<tr>
<td></td>
<td>Bow &amp; Lean (head pitch/bending)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Up pitch (Lean)</td>
<td>Away from side of involvement</td>
</tr>
<tr>
<td></td>
<td>Down pitch (Bow)</td>
<td>Toward the side of involvement</td>
</tr>
<tr>
<td></td>
<td>Sit to Supine Test</td>
<td>Away for side of involvement</td>
</tr>
<tr>
<td>Apogeotropic (Cupulolithiasis)</td>
<td>Spontaneous (pseudo) nystagmus</td>
<td>Toward the side of involvement</td>
</tr>
<tr>
<td></td>
<td>Bow &amp; Lean (head pitch/bending)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Up pitch (Lean)</td>
<td>Toward the side of involvement</td>
</tr>
<tr>
<td></td>
<td>(or Pitch) Test</td>
<td>Away from the side of involvement</td>
</tr>
<tr>
<td></td>
<td>Down pitch (Bow)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sit to Supine Test</td>
<td>Toward the side of involvement</td>
</tr>
<tr>
<td></td>
<td>Supine Null Point</td>
<td>Toward the side of involvement</td>
</tr>
<tr>
<td></td>
<td>Rotated to side of involvement</td>
<td>No nystagmus</td>
</tr>
</tbody>
</table>

Any of these finding may be seen in an individual with HC BPPV, but typically not all (Califona et al, 2010; Riga et al 2014).
Sit to Supine Test

STS test may be best method to identify side of involvement when intensity could not be distinguished with the SRT,
Supine

Null Point Supine

Rotate to side of involvement
Nystagmus disappears
Changes direction when rotate further

Video

- Geotropic nystagmus with sit to supine and supine roll tests

- Apogeotropic nystagmus with sit to supine and supine roll test
Other Diagnostic Issues

- Central Positional (apogeotropic) nystagmus (CPN)
  - Due to loss of inhibition or imbalance of inputs
    - prolonged post rotatory inputs of SCC
    - direction of CPN was aligned with the vector sum of the rotational axes of the SCC
  - Cerebellar Vermal or nodular lesions.
    - Choi et al, Neurology, 2015 16 individuals with CPN,
    - 81% (13) PN show horizontal nystagmus in supine head turn. All apogeotropic; 9 persistent.
    - 94% had more than one type of nystagmus. (Most common down beating in B Dix-Hallpike or straight head hanging)

- Persistent geotropic nystagmus
  - Light Cupula phenomena

Other Diagnostic Issues

- Multiple causes of horizontal nystagmus: Lechner et al, J of Neurology, 2014
- 60 individuals positional vertigo and horizontal positional nystagmus
  - Lateral canal BPPV 31 canalithiasis/ 9 cupulolithiasis
  - 13 Vestibular migraine: geotropic or apogeotropic symmetrical, persistent and low slow phase velocities (SPV<20 degree/sec)
  - 2 Meniere's disease: apogeotropic symmetrical, persistent and low SPV
  - 4 (3 unilateral, 1 bilateral ) Vestibular hypofunction: geotropic/ apogeotropic symmetrical and Low SPV
  - 1 vestibular schwannoma: asymmetric apogeotropic
# Treatment Options

<table>
<thead>
<tr>
<th>Roll Approach</th>
<th>Sidelying Head Rotation Approach</th>
</tr>
</thead>
<tbody>
<tr>
<td>Geotropic BPPV</td>
<td>Geotropic BPPV</td>
</tr>
<tr>
<td>Lampert Roll Maneuver, BBQ Maneuver, 270-360 log roll Maneuver.</td>
<td>Contralateral Sidelying Head down (Gufoni, Appiani)</td>
</tr>
<tr>
<td>Apogeotropic BPPV</td>
<td>Apogeotropic BPPV</td>
</tr>
<tr>
<td>Modified Log Roll (Kim, 2011)</td>
<td>Ipsilateral Sidelying Head Up/Down (mod Gufoni, Casani)</td>
</tr>
</tbody>
</table>

## What is the log roll (BBQ) approach?

- 270 to 360 degree head rotations starting toward the involved ear and then rolling away
- Takes advantage of inertial and gravitational forces to move the debris along and out of the LC.
- Used to treat both geotropic and apogeotropic forms.
- Probably most effective for canalithiasis (Riga et al 2013).

### Advantages

- Well known to most therapists
- Easy to control the head movements and progress slowly to decrease severity of symptoms
- All maneuvers start on side of involvement and move toward health side (Same as PC maneuvers)
- May reduce variability in therapist application

### Disadvantages

- Need to know side of involvement
- May not be as effective in all forms of apogeotropic
  - 50% in White, 2001
  - May not dislodge Otoconia
- May be difficult for individuals with poor mobility
**Log Roll or BBQ Roll Maneuvers**

Lempert, 1994; Lempert et al, 1997

---

Position 1:
Cap otocoria moves anteriorly
- transient geotropic

Position 2:
Cap otocoria moves posteriorly
- transient away from involvement

Position 3:
Cap otocoria moves medially
- transient geotropic

Position 4:
Cap otocoria moves anteriorly to Utricle
- transient away from involvement

---

“Moderately effective” 38%-88% in 1-3 maneuvers
(Kim et al, 2012; Casani et al, 2011; Koress et al, 2011; Echar et al, 2007)

---

**Modified Roll for Apogeotropic**

Kim et al, 2011

---

Position 1:
CUu: Head rotated 135° down
- Persistent apogeotropic
cU: persistent bending of cupula
- Persistent apogeotropic
cUc & cAa: moves laterally and posterior
- Transient apogeotropic
cUc & common crus: moves medially toward common crus
- Transient geotropic

Position 2:
CUa: moves anterior into utricle
- Transient toward involved side
cUs & CAa: moves anteriorly into common crus
- Transient away from involved side

Position 3:
USu: Less bending of cupula
- Persistent toward involved side
cUc & CAa: moves medially toward common crus
- Transient away from involved side

Position 4:
CUu: detach and move toward utricle
- persistent apogeotropic disappears with vibration
cUc & CAa: moves medially toward common crus
- Transient geotropic

Position 5:
CUu: moves anterior into utricle
- Permanent toward involved side
cUs & CAa: moves anteriorly into common crus
- Transient away from involved side

---

61% 1 maneuver
97% ≤ 6 maneuvers
Kim et al, 2011
What is a sidelying head rotation approach?
- Uses inertial and gravitational forces by moving the patient from sitting to sidelying and then rotating the head 45 degrees down or up.
- May resolve either geotropic or apogeotropic.
- Brisk deceleration of the head in the rapid movements may detach the otoconia in cupulolithiasis

Advantages
- More tolerated for individuals with mobility restrictions
- Less steps – less time to perform
- Mechanical resolution for apogeotropic not clear, but convert to geotropic – which may be easier to treat

Disadvantages
- Need to determine side of involvement
- Speed of movements and deceleration for Apogeotropic may be hard to attain.
- Conversion rate is high causing increase symptoms
- Poor understanding of fluid dynamics, non responsiveness and/or conversions may lead to confusion (increase # of maneuvers or delayed resolution)

Contra lateral Side lying
Head Down
“Gufoni” or “Appiani”

“Moderately effective for Geotropic” 38%-93% in 1-3 maneuvers
Clinical Decision Making

- Diagnosis of type, location and side is Important!

- If unresolved, consider switching starting direction: Side of involvement may be misdiagnosed or side of cupula may be miss assumed

- Careful observation of eyes during maneuvers should help in making the next step in treatment if unresolved
History EB

- 59 year old woman who had a history of a vestibular neuronitis on 12/05/13
- She reported three day of disability and gradual recovery and return to work with occasional instability
- Presented on 02/05/14 with acute vertigo attacks consistent with BPPV
  - Normal oculomotor exam with and without fixation
  - CTSIB- normal except Fell with foam EC
- IR Lenses Exam:
  - positive head shaking with right beating nystagmus
  - Positive upbeating and torsional nystagmus in left HPD test
  - No nystagmus in any other positions only symptoms in left roll test
- Treated effectively with Left CRT
- discharged on 2nd visit recheck without symptoms or nystagmus
History clinical findings

• 3/22/14 awoke with vertigo, nausea and instability
• Returned to clinic 3/27/14 for assessment
• Normal oculomotor exam and CTSIB all 4 conditions
• BPPV exam:
  • Sit to supine LB <1’
  • Right roll test geotropic 30 sec
  • Midline LB
  • Left roll test geotropic 30 sec
  • Right HPD geotropic 20 sec, most symptoms
  • Left HPD geotropic 20 sec

Initial testing
Diagnostic dilemma ???

- **Left Horizontal canal canalithiasis (CAp)**
  - Equal symptoms and nystagmus in roll test
  - Prior L PC Canalithiasis and L neuronitis (headshaking)

- OR

- **Right Horizontal canal canalithiasis (CAp)**
  - Sit to supine –LB (away from involved side)
  - Right>left symptoms and nystagmus on HPD

- What could I have done to confirm side?
  - Bow and lean or sit to supine testing

- Now I would recognize the change in presentation during the Left BBQ roll and reverse

---

Left Log Roll maneuver (BBQ)
Left Log Roll Maneuvers, if Right Side Involvement

Position 1:
- CAp otococia moves medially
- transient geotropic/Left beating

Position 2:
- CAp otococia moves posteriorly medially
- transient away from involvement /Left beating

Position 3:
- CAp otococia moves anteriorly towards cupula
- transient geotropic/Right Beating

Position 4:
- CAp otococia move anteriorly toward the cupula (if into of close to ampula conversion)
- transient away from involvement Right Beating switch to Left beating

Treatment approach for Left HC Canalithiasis

- Began with left BBQ roll (CAp)
  - Changed from geotropic to apogeotropic
    - Persistent in nature
    - Likely CUc
  - Indications that crystals were moving in the wrong direction in position 3/4 confirms chose wrong side –left beating then changed to right beating
- Clues for correct diagnosis HC CAp on the right
  - Supine LB (away for side of involvement )
  - Prone position (change of direction from right to left beating)
  - Sitting up strong RB (bow–toward side of involvement)
- Hypothesis: Conversion to apogeotropic
  - R CAp converted to R CUc ? CUa
Recheck with log roll- confirms apogeotropism

Right modified log roll (Cupulolith- Kim)
Right Modified Roll for Apogeotropic

- Position 1: Head rotated 135° down
  - CUC: detaches and moves laterally
  - CAA: moves laterally - Transient apogeotropic Left Beating

- Position 2: CUC & CAA: moves laterally and posterior
  - Transient apogeotropic Left Beating

- Position 3: CUC & CAA: moves laterally and posteriorly
  - Transient away from involved side Left Beating

- Position 4: CUC & CAA: moves medially toward common crus
  - Transient ? geotropic Left Beating

- Position 5: CUC & CAA: moves anteriorly into common crus to utricle
  - Transient ? away from involved side Left Beating

• Performed Right Cupulolith maneuver (Kim)
  • LB throughout maneuver indicates now moving away from the cupula
• Recheck supine roll test
  • Changed back to geotropic
• Repeated Right BBQ to clear BPPV
• She returned next visit without any nystagmus or symptoms
Teaching points- how do we determine sides?

- Localizing side – patient history, symptoms, intensity of nystagmus
- rechecked
  - Sit to supine test
  - Bow and lean- if I had stopped after the roll test and done bow and lean.....
- Symptom intensity
  - Geotropic-worse on involved side
  - Apogeotropic –worse on uninvolved side
  - During the maneuver of the L Roll the intensity of symptoms changed

What does the intensity/ speed of the horizontal nystagmus tell us?

**Lower grade < 20 (5-10)**
- Migraine
  - geotropic or apogeotropic, direction-fixed
- Cupulolithiasis
- Chronic/Compensated Unilateral Vestibular Loss
  - Meniere’s Disease
  - Vestibular Schwannoma (VS)
  - Vestibular Neuritis
- Central
  - Persistent, no latency

**Brisker/ vigorous >20 (40-50)**
- Canalithiasis
  - Paroxysmal
  - Latency
  - Asymmetry on roll
- Acute/Uncompensated Unilateral Vestibular Loss
  - Vestibular Neuritis
  - Post surgical VS
  - Meniere’s
  - Direction-fixed
- Light Cupula
  - Geotropic
- Central
Low intensity/speed

Moderate intensity/ speed
Paroxysmal nystagmus
Case Studies
Differential Diagnosis

WARNING:
Not all positional nystagmus is BPPV

Case #1: Bryan, 64 year old

• Primary c/o
  • Horrific week, “stomach-flu” with throwing up, dizziness/vertigo lasted for 3 days (in bed)
  • Seen 6 months previously at the clinic with findings consistent with neuritis (64% Unilateral vestibular loss and direction-fixed nystagmus)
• Feels ear fullness w/questionable change hearing but MD confirms stable hearing (day before)
• Severe neck pain/head fullness
• Denies any head ache or migraine history but admits to “lifetime of visual phenomenon that can last for hours”
• Admits to definite light/sound sensitivity during severe exacerbation
Fixation-Removed Examination

Findings

- Severe persistent Apogeotropic Direction Changing Nystagmus in Dix-Hallpike (DH) and Roll testing (relatively asymptomatic)
  - Some transient LT/UB on Left DH
- Supine: Marked Upbeating !!!
- Bow testing: Definite Downbeating nystagmus!!!
- Sitting Flexion Rotation Testing (cervical Torsion testing): apogeotropic as with rolling, negative for cervical influence
Assessment

• Left Posterior Canal BPPV possibly
• Primary finding a severe DCN (Direction Changing Nystagmus) that DOES NOT FOLLOW LAWS OR CANNOT be explained by isolated BPPV
• Presentation/Disability supportive of/consistent with VESTIBULAR MIGRAINE
• Evidence:
  • Migraine “Travelers” (light/sound sensitivity)
  • Past history of episodic ocular migraines
  • Positional nystagmus NOT accountable with BPPV or peripheral end organ
  • Cervicogenic triggering factor

International Criteria for Migraine


• 1. Vestibular migraine
  • A. At least 5 episodes with vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 hours
  • B. Current or previous history of migraine with or without aura according to the International Classification of Headache Disorders (ICHD)4
  • C. One or more migraine features with at least 50% of the vestibular episodes:
    • headache with at least two of the following characteristics: one sided location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity
    • photophobia and phonophobia
    • visual aura
International Criteria for Migraine


• D. Not better accounted for by another vestibular or ICHD diagnosis

2. **Probable vestibular migraine**

• A. At least 5 episodes with vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 hours

• B. Only one of the criteria B and C for vestibular migraine is fulfilled (migraine history or migraine features during the episode)

• C. Not better accounted for by another vestibular or ICHD diagnosis

Power of a Migraine

• Central findings: i.e. impaired smooth pursuit

• Peripheral vestibular asymmetries
  • Some report usually more mild 20-40% (significant Caloric weakness >25%)

• Nystagmus associated with Migraine (Polensek SH Tusa RJ 2010)
  • Spontaneous nystagmus: 19%
  • Horizontal After headshake: 35%
  • Positional nystagmus: 100% (76% sustained and without latency)
    • Direction-changing (50%) Direction Fixed (25%)
    • Up/down beating (10%) or central nystagmus (10%)
    • Torsional (5%)
    • Usually at lower amplitude 2-7 degrees/second
    • Nystagmus often dissipates in asymptomatic periods or measured at 1-2 degree per second
Management

• Treatment for Left Posterior Canal BPPV
  • Left Paroxysmal nystagmus cleared by week follow-up
• Began Migraine Conservative Treatment
  • Educated on Power of migraine to create dizziness
  • Educated on role of diet, exercise, pacing, hydration, etc.
  • Treatment for significant cervicogenic impairments/comorbidities, exacerbate/trIGGERing factors
  • All nystagmus resolved to insignificant (<5 degrees/second) by 4 weeks
• Referral to Neurologist

Treatment course

• Confirmed Migraine placed on gabapentin for prevention/stabilization
  • Cervical-specific treatments completed
  • Disability steadily resolved over 6 weeks
• Returned to clinic 4 months later
• Reporting “not floating anymore” but distinct return of positional dizziness with looking up, blurry vision, and motion-provoked instability
Reassessment

- Normal velocity and quality of gait
- Normal Romberg but still abnormal CTSIB 5/6
- Negative HIT; Abnormal but improved DVA (3 line loss)
- Cervical ROM without pain
- No spontaneous/gaze-evoked nystagmus

Watch his Eyes now
Assessment

- Resolved Direction Changing Nystagmus (DCN) supine and on rolling testing
- Abnormal left Dix Hallpike:
  - symptomatic left torsional/upbeating nystagmus of 20 second duration

Follow-up

- Significantly better since CRT, but not back to wellness
- Normal Dix-Hallpikes (BPPV in remission)
- Taught self CRT principals should he experience exacerbation of BPPV
Final Evaluation

Discharge Testing

• Dizziness Handicap Inventory (DHI) 24 versus 84
• VAS severity rating of overall dizziness 3 versus 5
• Reports Only bothersome symptoms remaining
• Back to wellness program (stationary bike, not regular)
Discharge Testing

- Functional Gait Assessment (FGA): 29/30 vs 17/30
  - <23 associated with fall risk
- Heel-Toe: 30 seconds eyes open, 16 eyes closed
  - Normal
- Single leg: 30 seconds eyes open and 5-8 seconds eyes closed
  - Normal 60-69: 23 +/- 9 eyes open and 10 +/- 9 eyes closed (Bohannon, 1984)
- CTSIB:
  - Normal sway,
  - No Falls
  - Normal tolerance
- Normal Motion Sensitivity

Final phone contact

- Doing well and exercising 5x/week including racquetball
- Will monitor for exacerbation consistent with BPPV that can’t self manage
- FL-41 sunglasses very helpful
- Understands/know how to treat dizziness related to
  - BPPV
  - Migraine
  - Role of cervical/neck exacerbative factors
Case #2: Sharyl, 72 y.o

- History of Right Posterior Canal BPPV, recurrent and resistive forms
  - seen once every couple of years past 10 years
  - 5 years prior ID with significant Right vestibular hypofunction per VNG (35% caloric reduction)
- c/o return of position dizziness for past 2 weeks that she can’t clear with self Canalith Repositioning
- Onset associated with tripping and falling into the wall

Sharyl, 72 y.o

- Dizziness Handicap Inventory
  - 23/100 Mild
- Subjective Rating Scale
  - 3.4/10 dizzy and 4.5/10 balance
- Highly active (bike trip to Netherlands and around the Alps)
- Denies dizziness associated with speech, sensation loss, weakness, swallowing or fine motor control
- Denies accompany hearing fluctuations, headaches, light/sound sensitivity, and/or significant nausea or vomiting
Initial findings

- Smooth pursuit/saccades normal for age
- HINT’s Negative
- Computerized vHIT gain (ABNORMAL = values <.68, asymmetry >20% and presence of corrective saccades)
  - Lateral: .88 L and .86 R (2%)
  - Posterior: .79 L and .68 R (14%)
  - Anterior: .78 L and .76 R (3%)

Eye movement assessment MOST sensitive/specific test for ID acute stroke
- Inferior cerebellar infarcts can mimic peripheral vestibular insults
- Neuritis vs PICA or labyrinthitis vs AICA (can have + HIT)
- 51% have obvious central signs, 29% isolated ataxia, rest only eye

MRI falsely negative 12% and misleading out to 48 hours after sx onset
- Negative likelihood ratio .02 (1% chance being wrong) vs MRI .21 (17% chance of being wrong)
H.I.N.T.S.

- Head Impulse Nystagmus Test of Skew
  - Direction Changing gaze-evoked nystagmus
- 3 step test
  - Head Impulse testing
  - Gaze-evoked nystagmus test
  - Cover/uncover or Maddox Rod test
- STROKE suspected if horizontal Spontaneous nystagmus (some rotational component) AND ONE or more of the following:
  - Normal Head Impulse Test
  - “Dangerous Oculomotor sign (s)”:  
    - Direction Changing Gaze-evoked nystagmus
    - positive skew deviation (vertical misalignment)

Initial findings (cont.)

- Gait unremarkable
- Clinical Test for Sensory Interaction on Balance (CTSIB): Normal
- Single Leg (eyes open): 30 (normal) and Single Leg (eyes closed): 14 sec
- Heel-toe (eyes open): 30 and Heel-toe (eyes closed): 18-30 sec  
  - Normal for age SOLEO 14 +- 9 eyes open and 4 +-3 eyes closed
- 3 Beats Left beating after headshake nystagmus,
  - questionable Left beating spontaneous
- Mastoid Vibration: marked left beating nystagmus (on right only)
- ABNORMAL Positional Testing
Initial IR exam findings:
Right Dix Hallpike then supine

Initial Exam: Left sidelying and right roll
Initial findings (cont.)

- Right Dix-Hallpike
  - Marked persistent geotropic RIGHT beating nystagmus, not paroxysmal
  - Not her “normal” intensity of BPPV subjective c/o
- Supine: mild left beating nystagmus
- Roll Testing/Dix-Hallpike:
  - Marked persistent Geotropic DCN mild/moderately symptomatic
  - No habituation, not paroxysmal
- Bow testing: Right beating nystagmus
- Null point: 20 degrees to the Right
- CAN’T Account for findings with BPPV, canalithiasis, no evidence for central, migraine, or acute vestibular hypofunction

REMEMBER!!!
Lechner et al, J of Neurology, 2014

- Horizontal Positional nystagmus is NON-LOCALIZING FINDING!!!
  - Most common is BPPV (canalithiasis/cupuliolithiasis)
  - Vestibular migraine
    - geotropic or apogeotropic
    - symmetrical
    - persistent and low slow phase velocities (SPV<20 degree/sec)
  - Meniere's disease
  - Vestibular hypofunction
    - geotropic/ apogeotropic symmetrical and Low SPV
  - Vestibular schwannoma
  - OR......
Light Cupula Proposed Etiology

CH Kim, 2014 Med Hypothesis; T. Imai, 2014 BMJ

• Change in density of the endolymph due to inner ear hypoperfusion

• Trauma from microcirculation/compromise or infection disrupts blood-labyrinth barrier leading to increased proteins in inner ear

• Seen with sudden sensory neural hearing loss

• CONSIDERED: “Self-limiting benign condition”

Light Cupula Criteria

Kim CH 2014

• Persistent horizontal DCPN
  • No latency (paroxysmal quality) on Roll testing
  • Geotropic
  • Presence of Null plane rolled slightly (15-40 degrees) to affected side
  • Horizontal Nystagmus on Bow testing (90 degrees nose down) beating to the affected side
  • Horizontal nystagmus on Lean (supine) testing beating away from affected side
  • Spontaneous nystagmus to unaffected side
  • Other causes ruled out (Central, Unilateral peripheral hypofunction)
Management

- Educated
- Taught VORx1 adaptation
- Returned in a week

Follow-up visit: 5 days later
Follow-up visit

• Crisis event is in COMPLETE remission to subjective report and to objective testing
  • Resolved persistent geotropic nystagmus
• Audiogram normal
  • No support for Meniere’s disease
• Cont ex 3-4 weeks and be informally followed
• Symptoms stayed resolved (>8 months)
• Anecdotally
  • Should steadily resolve within 3-6 weeks
  • VOR adaptation/habituation ?may help? facilitate resolution

Wrap-up

• Diagnostic and treatment dilemma
  • Differentiating not only type of nystagmus (apogeotropic vs geotropic)
  • Apogeotropic that is actually canalithiasis (CaA)
  • Realizing 2 sides of the Cupula that would need to be “freed”
• Understanding the rational for rolling maneuvers versus sidelying maneuvers
• Appreciating NOT all DCN is BPPV
• Using not only quality/direction but importance of the INTENSITY of nystagmus
• Differential of migraine, endorgan hypofunction, light cupula, and of course Central
References:


