Positioning the Audiologist in a Management Role for Patients with Vertigo

Richard A. Roberts, Ph.D.
Disclosures

Employed by Vanderbilt University Medical Center/Vanderbilt University School of Medicine.

Member, Board of Trustees of the American Academy of Audiology Foundation.
Learning Objectives

After participating in this session, attendees will be able to:

1) discuss the role of audiologists in management of patients with common causes of vertigo

2) identify some of the more common causes of vertigo

3) list appropriate interventions for many patients with vertigo
Overview

- Introduction
- BPPV
- Vestibular Neuronitis
- Vestibular Migraine
- Summary
Impact of Vertigo

• **30-35%** of people will experience rotary/postural vertigo/vestibular dysfunction at some point in their lives 
  *(Neuhauser, 2007; Agrawal et al., 2009)*

• **69,000,000** people 40+ in the U.S. will experience vestibular dysfunction *(Agrawal et al., 2009; VEDA)*
Strupp, Dietrich, & Brandt, 2013

### TABLE 1

<table>
<thead>
<tr>
<th>Form of vertigo</th>
<th>Frequency n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign paroxysmal positional vertigo</td>
<td>3036</td>
<td>17.1</td>
</tr>
<tr>
<td>Somatoform phobic vestibular vertigo</td>
<td>2661</td>
<td>15.0</td>
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<tr>
<td>Central vestibular syndromes</td>
<td>2178</td>
<td>12.3</td>
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<tr>
<td>Vestibular migraine</td>
<td>2017</td>
<td>11.4</td>
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<tr>
<td>Menière’s disease</td>
<td>1795</td>
<td>10.1</td>
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<tr>
<td>Vestibular neuritis</td>
<td>1462</td>
<td>8.3</td>
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<tr>
<td>Bilateral vestibulopathy</td>
<td>1263</td>
<td>7.1</td>
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<tr>
<td>Vestibular paroxysmia</td>
<td>655</td>
<td>3.7</td>
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<tr>
<td>Psychogenic vertigo (other)</td>
<td>515</td>
<td>2.9</td>
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<tr>
<td>Perilymphatic fistula</td>
<td>93</td>
<td>0.5</td>
</tr>
<tr>
<td>Vertigo of unknown origin</td>
<td>480</td>
<td>2.7</td>
</tr>
<tr>
<td>Other(^2)</td>
<td>1563</td>
<td>8.8</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>17,718</strong></td>
<td><strong>100.00</strong></td>
</tr>
</tbody>
</table>

\(^1\) 1988–2012: Vertigo clinic of Ludwig Maximilian University and the German Center for Vertigo and Balance Disorders

\(^2\) Includes, among others, nonvestibular vertigo in neurodegenerative diseases, nonvestibular oculomotor disorders in myasthenia gravis, and peripheral ocular muscle paresis
Among the most common

- BPPV (17.1%)
- Vestibular Neuronitis (8.3%)
- Vestibular Migraine (11.4%)

These three issues account for 36.8% of vestibular issues.
U.S. Population Impact

• Estimated 69,000,000 people 40+ will experience vestibular dysfunction

• BPPV (17.1% = 11,799,000)
• Vestibular Neuronitis (8.3% = 5,727,000)
• Vestibular Migraine (11.4% = 7,866,000)

• 25,392,000 million lives!
• >50% required **5 months** or longer to reach a diagnosis
• Only **20%** felt received accurate diagnosis/management
• **75%** had an MRI
• **55%** reported a fall related to their Sx
Impact of BPPV

- $2,000 to diagnose (Li et al., 2000)
- Total healthcare costs exceed $2 Billion annually (Bhattacharyya et al, 2008; 2017)
- 86% of patients have interrupted daily activities/missed work days
- Health-related Quality of Life similar to HIV/AIDS, macular degeneration, Hepatitis B (Roberts et al, 2009)
Management of BPPV

• $2,000 but can often treat this in 10-20 minutes

• **Posterior** Canal: 41 - 95% *(Bhattacharyya et al., 2008; 2017)*

• **Horizontal** Canal: 38.4 - 93% *(Oron et al., 2015)*

• **Anterior** Canal: >75% *(Anagnostou et al., 2015)*
<table>
<thead>
<tr>
<th>Reference</th>
<th>Time Point of Assessment</th>
<th>Treatment</th>
<th>Control</th>
<th>End Point</th>
<th>P Value</th>
<th>Odds Ratio (95% CI)</th>
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<tbody>
<tr>
<td></td>
<td>1 mo</td>
<td>92.00b</td>
<td>42.50b</td>
<td>Negative Dix-Hallpike: Epley vs BD exercises</td>
<td>&lt;.001</td>
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<tr>
<td>Bruintjes (2014)</td>
<td>12 mo</td>
<td>20 of 22 (91)</td>
<td>10 of 22 (45)</td>
<td>Negative Dix-Hallpike: Epley vs control or placebo</td>
<td>&lt;.001</td>
<td>12.00 (2.24-64.28)</td>
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<td></td>
<td>1 mo</td>
<td>21 of 22 (96)</td>
<td>8 of 22 (36)</td>
<td>Negative Dix-Hallpike: Epley vs control or placebo</td>
<td>&lt;.001</td>
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<td>Froehling (2000)</td>
<td>1-2 wk</td>
<td>16 of 24 (67)</td>
<td>5 of 26 (19)</td>
<td>Negative Dix-Hallpike: Epley vs control or placebo</td>
<td>&lt;.020</td>
<td>3.20 (1.00-10.20)</td>
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<tr>
<td>Liang (2010)</td>
<td>7 d</td>
<td>42 of 43 (98)</td>
<td>34 of 44 (77)</td>
<td>Cured*: Epley vs control or placebo</td>
<td>&lt;.05</td>
<td>12.35 (1.51-101.36)</td>
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<td>Lynn (1995)</td>
<td>2 wk</td>
<td>16 of 18 (89)</td>
<td>4 of 15 (27)</td>
<td>Negative Dix-Hallpike: Epley vs control or placebo</td>
<td>&lt;.033</td>
<td>22.00 (3.41-141.73)</td>
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<tr>
<td>Mazoor (2011)</td>
<td>1 wk</td>
<td>22 of 30 (73) Epley</td>
<td>21 of 30 (70) Semont</td>
<td>Negative Dix-Hallpike: Epley vs Semont</td>
<td>.08</td>
<td>1.18 (0.38-3.63)</td>
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<tr>
<td></td>
<td>4 wk</td>
<td>28 of 30 (93) Epley</td>
<td>25 of 30 (83) Semont</td>
<td>Negative Dix-Hallpike: Epley vs Semont</td>
<td>.30</td>
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<tr>
<td>Munoz (2007)</td>
<td>Immediate</td>
<td>13 of 38 (34)</td>
<td>6 of 41 (14)</td>
<td>Negative Dix-Hallpike: Epley vs control or placebo</td>
<td>.04</td>
<td>3.03 (1.01-9.07)</td>
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<td>von Breven (2006)</td>
<td>24 h</td>
<td>28 of 35 (80)</td>
<td>3 of 31 (10)</td>
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<td>37.33 (8.75-159.22)</td>
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<td>Xie (2012)</td>
<td>7 d</td>
<td>54 of 58 (93)</td>
<td>11 of 45 (24)</td>
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<td>&lt;.05</td>
<td>41.73 (12.29-141.65)</td>
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<td>Yimtae (2003)</td>
<td>1 wk</td>
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<td>13 of 20 (65)</td>
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<td>.005</td>
<td>3.95 (0.87-17.99)</td>
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<td></td>
<td>4 wk</td>
<td>16 of 25 (64)</td>
<td>7 of 20 (35)</td>
<td>Negative Dix-Hallpike: Epley vs control or placebo</td>
<td>.336</td>
<td>3.3 (1.0-11.3)</td>
</tr>
</tbody>
</table>

Abbreviations: BD, Brandt-Daroff; OR, odds ratio.

*All randomized controlled trials completed in secondary or tertiary care otolaryngology settings except where designated.

*Raw values not given in article.

*Cured-outcomes reported as a composite measure of symptom resolution and Hallpike test result.

*Primary care setting.
<table>
<thead>
<tr>
<th>Canal</th>
<th>Nystagmus Fast Phase</th>
<th>Paired Extraocular Muscles</th>
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</thead>
<tbody>
<tr>
<td>Posterior</td>
<td>Rotary up-beating</td>
<td>Ipsilateral superior oblique</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Contralateral inferior rectus</td>
</tr>
<tr>
<td>Horizontal (lateral)</td>
<td>Horizontal</td>
<td>Ipsilateral medial rectus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Contralateral lateral rectus</td>
</tr>
<tr>
<td>Anterior (superior)</td>
<td>Rotary down-beating</td>
<td>Ipsilateral superior rectus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Contralateral inferior oblique</td>
</tr>
</tbody>
</table>

Diagnosis of BPPV

Hallpikes or Side-lying
- Posterior: Rotary Up-beating (Torsional)
- Anterior: Rotary Down-beating (Torsional)
  OR Down-beating
- Horizontal: Horizontal
  Geotropic - side with most intense response
  Ageotropic - side with least intense response

Roll Test or Head Right/Left
- Horizontal: Horizontal
  Geotropic - side with most intense response
  Ageotropic - side with least intense response

Deep Head-Hanging
- Anterior: Rotary Down-beating (Torsional)
  OR Down-beating
Lateral Canal BPPV, Geotropic variant

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<table>
<thead>
<tr>
<th>Study</th>
<th>n</th>
<th>PC</th>
<th>HC</th>
<th>AC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herdman, Tusa, &amp; Clendaniel (1994)</td>
<td>59</td>
<td>63.6</td>
<td>1.3</td>
<td>11.7</td>
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<tr>
<td>Fife (1998)</td>
<td>424</td>
<td>91</td>
<td>6</td>
<td>3</td>
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<tr>
<td>Wolf, Boyev, Manokey, &amp; Mattox (1999)</td>
<td>107</td>
<td>95.3</td>
<td>1.9</td>
<td>2.8</td>
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<tr>
<td>Honrubia, Baloh, Harris, &amp; Jacobson (1999)</td>
<td>292</td>
<td>93.5</td>
<td>5.1</td>
<td>1.4</td>
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<td>Ruckenstein (2001)</td>
<td>86</td>
<td>96.5</td>
<td>2.3</td>
<td>1.2</td>
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<td>Korres &amp; Balatsouras, (2004)</td>
<td>122</td>
<td>90.2</td>
<td>8.2</td>
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<td>Cakir et al. (2006)</td>
<td>169</td>
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<td>11.8</td>
<td>1.2</td>
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<td>Moon et al. (2005)</td>
<td>1,692</td>
<td>60.9</td>
<td>31.9</td>
<td>2.2</td>
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<td>Jackson, Morgan, Fletcher, &amp; Krueger (2007)</td>
<td>260</td>
<td>66.9</td>
<td>11.9</td>
<td>21.2</td>
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<tr>
<td>Celebisoy, Polat, &amp; Akyurekli (2008)</td>
<td>157</td>
<td>87.9</td>
<td>9</td>
<td>1.3</td>
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<tr>
<td>De Stefano et al. (2011)</td>
<td>412</td>
<td>70.9</td>
<td>27</td>
<td>2.4</td>
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<tr>
<td>Soto-Varela, Rossi-Izquierdo, &amp; Santosh-Pérez (2013)</td>
<td>614</td>
<td>88.4</td>
<td>6.4</td>
<td>5.2</td>
</tr>
</tbody>
</table>

**PC: 61-97%  HC: 1-32%  AC: 1-21%**

Treatment of BPPV

**Posterior**
- Canalith Repositioning Maneuver (Epley)
- Semont Liberatory Maneuver
- Gans Repositioning Maneuver

**Horizontal**
- Geotropic - 360° Roll (away from involved ear)
- Ageotropic - 360° Roll (away from involved ear)
- Geotropic - Gufoni (away from involved ear)
- Ageotropic - Casani (same side as involved ear)

**Anterior**
- Canalith Repositioning Maneuver (Epley)
- Reversed Maneuvers
- Yacovino Maneuver
Semont Liberatory Maneuver

Canalith Repositioning (Epley) Maneuver

Gans Repositioning Maneuver

HC – BPPV
(Gufoni) Appiani et al., 2001
HC – BPPV (Ageotropic Nystagmus)

Casani et al., 2002
BBQ Roll

Hx & Sx:
- 42 y.o. Male
- 6 Years
- Episodic Vertigo with duration < 1 min
- No Auditory Sx
- Multi MRI, CT Unremarkable

Always provoked with looking upward
Sometimes lying Supine
Dx BPPV few yrs ago—no mgmt.
No Imbalance or nausea
Frequent migraine/aura
Audio WNL
DHI = 16
No BPPV with Hallpikes

- HC-BPPV can provoke in Hallpikes but does not always
- Patient reports looking straight up always provokes vertigo
- Bertholon et al. (2002) “straight head-hanging”
Bertholon et al. (2002)

• Positional DBN in 50 Patients
  – 38 (76%) CNS involvement
  – 12 had Sx consistent with AC-BPPV
  – 2 only provoked AC-BPPV with straight, head-hanging

• “The straight head-hanging manoeuvre should be carried out in all patients with a history of positional vertigo and a negative Dix-Hallpike manoeuvre.”
BPPV Case

- Sounds like BPPV
- Dx with BPPV at one point
- Multiple MRIs
- Followed by neurology for frequent migraine
- Safe to try Deep Head-hanging
Deep Head-hanging
“Deep Head-hanging”

Yacovino, Hain, and Gualtieri (2009)
Reversed Epley

• Zapala (2008)
• Case with Hx of multicanal BPPV
• No apparent neurologic involvement
• Side-lying with nose-down provoked
• Used Reversed Epley to clear

• We also used Reversed Semont
BPPV Case

Hx & Sx:
- 42 y.o. Male
- 6 Years
- Episodic Vertigo with duration < 1 min
- No Auditory Sx
- Multi MRI, CT Unremarkable

Always provoked with looking upward
Sometimes lying Supine
Dx BPPV few yrs ago—no mgmt.
No Imbalance or nausea
Frequent migraine/aura
Audio WNL
DHI = 16

Dx:
- VOR VNG
- VSR mCTSIB
- VCR cVEMP

Deep Head Hanging
Reversed Semont

DBN w/ slight right movement & transient vertigo
Deep Head-hanging

Audio WNL

Dx: Anterior Canal
BPPV likely on right
1) slight rightward beats
2) BPPV more common on right
BPPV Case

• "Amazingly better"
• DHI = 8

One week Post-treatment

Deep Head-hanging
BPPV Summary

- BPPV is most common cause of vertigo
- $2 Billion economic impact
- HRQoL similar to HIV/AIDS, macular degeneration, Hepatitis B
- Highly treatable
- Even if we decreased costs by half, could potentially save $1 Billion!
Impact of Vestibular Neuronitis

- **5,727,000** people
- Fairly easy to identify acute event
- Difficulty is in identification and management of the uncompensated status
  - Up to one year before VRT recommended
    (Herdman et al., 2000)
  - More falls in patients with vestibular hypofunction
- Using BPPV cost assumptions
  - Total healthcare costs may exceed **$97 Million** annually
Pathophysiology of VN

- Latent virus in vestibular ganglion
- Replication causes inflammation and edema
- Edema cannot be contained in narrow bony channel
  - Damage to vestibular ganglion cells and axons

*(Arbusow et al., 1999; Strupp & Brandt, 2009)*
Pathophysiology of VN

• Compromised immune system may lead to activation of virus
  – HSV-1 often implicated
  – Upper respiratory infection
  – Colds
  – Allergies (seasonal)
  – Stress
Acute Phase

- Intense Vertigo
- Nausea
  - Emesis
  - Diarrhea
- Imbalance
- Hours to Days
- No noticeable change in hearing status
- Treated with medication
  - Antiemetics, vestibular suppressants, steroids
Uncompensated Phase

- Dizziness with head/body movement
- Imbalance
- Blurred vision (oscillopsia)
- Mild nausea
- BPPV

- May lead to decreased activity, work absence, reduced HRQoL
- Increased anxiety
Common Findings

- Superior vestibular branch affected most often
  - Lateral canal, Superior canal, Utricle
  - Caloric weakness
  - Abnormal oVEMP
  - Abnormal VHIT for Superior (anterior) and Lateral (horizontal)
  - Spontaneous nystagmus, enhances with headshake
  - Abnormal dynamic visual acuity
  - Abnormal postural stability
Less Common Findings

• Hx & Sx consistent with Vestibular Neuronitis

• **BUT**
  – Robust & Symmetric Calorics
  – Normal oVEMP
  – Abnormal cVEMP
  – Abnormal VHIT for Posterior canal
  
  – Spontaneous nystagmus, enhances with headshake
  – Abnormal dynamic visual acuity
  – Abnormal postural stability

• **Inferior vestibular nerve branch involvement**
Vestibular Rehabilitation for Peripheral Vestibular Hypofunction: An Evidence-Based Clinical Practice Guideline

FROM THE AMERICAN PHYSICAL THERAPY ASSOCIATION
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ABSTRACT

Background: Uncompensated vestibular instability, visual blurring with head movements, and impaired balance and gait are common complaints of people with peripheral (unilateral or bilateral) vestibular hypofunction. The purpose of this guideline is to provide evidence-based recommendations for the effective management of peripheral vestibular hypofunction. This guideline was developed by the American Physical Therapy Association Neurology Section Peripheral Vestibular Hypofunction Task Force.

Therapeutic Intervention for Persons With Peripheral Vestibular Hypofunction

A. Action Statement 1: EFFECTIVENESS OF VESTIBULAR REHABILITATION IN PERSONS WITH ACUTE AND SUBACUTE UNILATERAL VESTIBULAR HYPOFUNCTION. Clinicians should offer vestibular rehabilitation to patients with acute or subacute unilateral vestibular hypofunction. Evidence quality: I; recommendation strength: strong

B. Action Statement 2: EFFECTIVENESS OF VESTIBULAR REHABILITATION IN PERSONS WITH CHRONIC UNILATERAL VESTIBULAR HYPOFUNCTION. Clinicians should offer vestibular rehabilitation to patients with chronic unilateral vestibular hypofunction. Evidence quality: I; recommendation strength: strong

C. Action Statement 3: EFFECTIVENESS OF VESTIBULAR REHABILITATION IN PERSONS WITH BILATERAL VESTIBULAR HYPOFUNCTION. Clinicians should offer vestibular rehabilitation to patients with bilateral vestibular hypofunction. Evidence quality: II; recommendation strength: strong

D. Action Statement 4: EFFECTIVENESS OF SACCADE OR SMOOTH-PURSUIT EXERCISES IN PERSONS WITH PERIPHERAL VESTIBULAR HYPOFUNCTION (UNILATERAL OR BILATERAL). Clinicians should offer sacadic or smooth-pursuit exercises as specific exercises for gaze stability in patients with unilateral or bilateral vestibular hypofunction. Evidence quality: II; recommendation strength: strong

E. Action Statement 5: EFFECTIVENESS OF DIFFERENT TYPES OF EXERCISES IN PERSONS WITH ACUTE OR CHRONIC UNILATERAL VESTIBULAR HYPOFUNCTION. Clinicians may provide targeted exercise techniques to address specific impairments and functional limitations. Evidence quality: II; recommendation strength: strong

F. Action Statement 6: EFFECTIVENESS OF SUPERVISED VESTIBULAR REHABILITATION. Clinicians may offer supervised vestibular rehabilitation to patients with unilateral or bilateral peripheral vestibular hypofunction. Evidence quality: I-II; recommendation strength: moderate

G. Action Statement 7: OPTIMAL EXERCISE Dose of TREATMENT IN PEOPLE WITH PERIPHERAL VESTIBULAR HYPOFUNCTION (UNILATERAL AND BILATERAL). Clinicians may prescribe a home exercise program of gaze stability exercises consisting of a minimum of 3 times per day for a total of at least 12 minutes per day for patients with acute/subacute vestibular hypofunction and at least 20 minutes per day for patients with chronic vestibular hypofunction. Evidence quality: II; recommendation strength: strong

H. Action Statement 8: DECISION RULES FOR STOPPING VESTIBULAR REHABILITATION IN PERSONS WITH PERIPHERAL VESTIBULAR HYPOFUNCTION (UNILATERAL AND BILATERAL). Clinicians may use a decision rule to establish the need for the presence of symptom resolution or time to progress in treatment progressions for stopping rehabilitation. Evidence quality: II; recommendation strength: expert opinion

I. Action Statement 9: FACTORS THAT MODIFY REHABILITATION OUTCOMES. Clinicians may consider factors that modify rehabilitation outcomes. Evidence quality: I-II; recommendation strength: weak to strong

J. Action Statement 10: THE HARM/BENEFIT RATIO FOR VESTIBULAR REHABILITATION IN TERMS OF QUALITY OF LIFE/PSYCHOLOGICAL STRESS. Clinicians should consider the harm/benefit ratio for rehabilitation in terms of quality of life/psychological stress. Evidence quality: II; recommendation strength: strong

These guidelines were issued in 2016 on the basis of the scientific literature published between January 1985 and February 2015. These guidelines will be considered for review in 2020, or sooner if new evidence becomes available. Any updates to the guidelines in the interim period will be noted on the Neurology Section of the APTA website: www.apta.org
Basis of VRT Exercises

- Adaptation: recalibration of the VOR and VSR to achieve central compensation
  - residual vestibular function
  - Exercises provoke symptoms related to the uncompensated peripheral vestibulopathy
  - provide a range of experiences that allow the CNS to compensate to the provoking activities
  - carry over to normal activities occurring in the patient’s everyday life

*Figure 5. Patient is shown bouncing on exercise ball and reading Snellen chart. This task incorporates vestibular rehabilitation aspects of adaptation, habituation, and gaze stabilization.*

*(Roberts, 2009)*
Basis of VRT Exercises

- Habituation: Some include within Adaptation.
  - Exercises are completed repetitively to desensitize the patient to the provoking stimulus.
  - Not yet clear that this occurs through the same underlying processes as adaptation

- Gaze stabilization: Often placed under the category of Adaptation
  - Allowing the CNS to recalibrate to the existing vestibular information to limit the slippage of the target image on the fovea
  - The CNS must experience the slippage for this process to occur
  - On the other hand, patients with BVD also can demonstrate an improvement in gaze stabilization which may reflect the recruitment of other eye movement systems to substitute for the lack of vestibular information.

(Roberts, 2009)
Basis of VRT Exercises

- Substitution: encourage the patient to use non-vestibular sources of information to support VOR and VSR
  - Cervico-ocular reflex (COR)
  - other visual system processes (saccades, smooth pursuit, “pre-programmed” eye movement
  - increased weighting of somatosensory information
  - Useful for patients with all types of uncompensated vestibular function, but greater role for patients with BVD

*Standing on Foam With Eyes Closed*

Goal: Develop improved postural stability on dynamic surfaces by incorporating extra-vestibular systems (substitution).

*Figure 8. Patient is shown standing on dense foam with eyes closed. For patients with no vestibular function, this task is one of substitution. For patients with partial vestibular function, vestibular rehabilitation aspects of adaptation also are used.*

*(Roberts, 2009)*
Common Recommendations

- Only use vestibular suppressants for **ACUTE** symptoms
- Resume normal activities
- Self-directed VRT
  - Clinician-directed for more complex
- Follow-up by phone in 2 weeks
- Follow-up in office 4 weeks
These should be completed *twice each day*

**Targets**  (seated)

Sit with visual target to the right, to the center, and to the left. More visual detail is better. Look at the right target and make certain image is in focus, then move to center target and make certain image is in focus, move to left target and make sure image is in focus. Then, sweep all the way back to right target. You completed one rep. Complete 15-20 times. Next, switch direction by starting to the left, center, right, and then sweep all the way back to the left. Do this 15-20 times. You can also do this with vertical targets.

**Head Movement Eyes Closed**  (seated)

Horizontal, Vertical, Diagonal, Head Circles each direction. Complete 10-15 times for each movement with eyes closed. If this is too provoking complete with eyes open and then progress to eyes closed. This should be completed in a seated position.

**Walking with Head Turns**

Walk forward in hallway. Start with head to the right, take two steps, then head to the left. Repeat this every couple of steps. Complete 10 times. Once it is no longer provoking inside home, move outside to grass, gravel, or sand for increased difficulty.
Hx:
- 38 y.o. Male
- Active duty Pilot
- 2 Attacks
- Both after eating
- MRI/MRA clear
- EKG/Tilt Table WNL
- DHI = 22 (Mild)
- Acyclovir

Sx:
- Constant sensation similar to alcohol “buzz”
- Frequent after-sensation to normal head/body movements
- Better in a.m./worse p.m.
- Moving sidewalk sensation
Caloric Test Summary

Spontaneous Pre-Irrigation - Horizontal Channel

Right Ear: [Graph]
Left Ear: [Graph]

Right Cool - Horizontal Channel

Right Ear: [Graph]
Left Ear: [Graph]

Left Cool - Horizontal Channel

Right Ear: [Graph]
Left Ear: [Graph]

Right Warm - Horizontal Channel

Right Ear: [Graph]
Left Ear: [Graph]

Left Warm - Horizontal Channel

Right Ear: [Graph]
Left Ear: [Graph]

Unilateral Weakness

Directional Preponderance

Graphic representations of results for each condition.
Vestibular Neuronitis

**Hx:**
- 38 y.o. Male
- Active duty Pilot
- 2 Attacks
- Both after eating
- MRI/MRA clear
- EKG/Tilt Table WNL
- DHI = 22 (Mild)
- Acyclovir

**Sx:**
- Constant sensation similar to alcohol “buzz”
- Frequent after-sensation to normal head/body movements
- Better in a.m./worse p.m.
- Moving sidewalk sensation

**Dx:**
- Sway on foam EC
- Absent Left

**Self-Directed VRT**
- Adaptation
- Habituation
- Substitution
- High-level Mods
3-weeks Post-VRT

- DHI = 0
- No further symptoms at all!
- cVEMP Absence led to VRT program
Vestibular Neuronitis Summary

- Uncompensated state more difficult to identify and manage than BPPV

- $97 Million economic impact

- VRT (including self-directed) very effective
  - Shortens uncompensated period
  - Return to work

- Even if we decreased costs by half, could potentially save $48.5 Million!
Impact of Vestibular Migraine

• **7,866,000** people

• Arguably more difficult to identify than BPPV
  – No specific diagnostic test \((Honaker \& Samy, 2008)\)
  – Inconsistent results \((Dietterich \textit{et al}, 2016; Sohn, 2016)\)

• Using BPPV cost assumptions
  – Total healthcare costs may exceed **$1.33 Billion** annually

• HRQoL poorer for vestibular migraine than migraine only \((Wang \textit{et al}., 2016)\)
Pathophysiology of VM

- **Cortical Spreading Depression**
  - May happen in cerebellum
  - Does not explain peripheral vestibular issues

  - Canal paresis
  - BPPV
  - Meniere’s Disease

(Bisdorff, 2011; Sohn, 2016; Dieterich et al., 2016)
Pathophysiology of VM

- Ion Channel
- Mutated gene coding for Ca Channel
  - Familial Hemiplegic Migraine
  - Episodic Ataxia Type 2
- No gene yet ID’d for vestibular migraine

http://membranereceptors.com/transduction-process/ion-channel-linked-receptors/

(Bisdorff, 2011; Sohn, 2016; Dieterich et al., 2016)
Pathophysiology of Vestibular Migraine

- **Neurotransmitters**
  - Calcitonin
  - Serotonin
  - Noradrenaline
  - Dopamine
- Implicated in pathogenesis of migraine
- Important in central and peripheral vestibular neural activity

(Bisdorff, 2011; Sohn, 2016; Dieterich et al., 2016)
Pathophysiology of VM

• Trigeminal and vestibular nervous system connections
  – Spontaneous nystagmus triggered in migraine patients with painful trigeminal nerve stimulation—not seen in controls (Marano et al., 2005)

• Increased metabolism in temporo-parieto-insular areas and bilateral thalami during attack
  – Indicates activation of vestibulo-thalamo-cortical pathway

• Structural differences inferior temporal gyrus, cingulate cortex, posterior insula in patients with VM
  – Cortical processing of vestibular and nociceptive (including pain) information

(Bisdorff, 2011; Sohn, 2016; Dieterich et al., 2016)
Vestibular Migraine

A. At least 5 episodes fulfilling criteria (C) and (D)
B. A current or past history of either migraine without aura OR migraine with aura
C. Vestibular symptoms of moderate or severe intensity, lasting between 5 min and 72 h
D. At least 50% of episodes associated with at least 1 of the following 3 migraine features:
   1) Headache with at least 2 of the following 4 characteristics: unilateral headache, pulsating quality, moderate to severe intensity, or aggravation by routine physical activity
   2) Photophobia and phonophobia
   3) Visual aura
E. Not better accounted for by another ICHD-3β diagnosis or by another vestibular disorder

*International Classification of Headache Disorders - 3β (2013)*
Probable Vestibular Migraine

A. At least 5 episodes with vestibular symptoms of moderate or severe intensity, lasting between 5 min and 72 h

B. Only 1 of criteria (B) and (D) for Vestibular Migraine fulfilled
   1) Migraine history with or without aura
   2) Migraine features during the episode
      1) Headache with at least 2 of the following 4 characteristics: unilateral headache, pulsating quality, moderate to severe intensity, or aggravation by routine physical activity
      2) Photophobia and phonobia
      3) Visual aura

C. Not better accounted for by another ICHD-3β diagnosis or by another vestibular disorder

(Sohn, 2016)
Ocular and Cervical Vestibular Evoked Myogenic Potentials in Patients With Vestibular Migraine

Makowiec, Kathryn F.; Pikker, Erin G.; Jacobson, Gary P.; Ramadan, Nabil M.; Roberts, Richard A.

Otology & Neurotology: August 2018 - Volume 39 - Issue 7 - p e661–e667
doi: 10.1097/MAO.0000000000001680
Vestibular Disorders

Abstract

Objective: To evaluate the relationship between normal and abnormal ocular vestibular evoked myogenic potentials (oVEMP) and cervical vestibular evoked myogenic potentials (cVEMP) in patients with and without vestibular migraine (VM).

Study Design: Retrospective review of oVEMP and cVEMP results in patients with vestibular disorders who were assessed clinically and completed vestibular function studies. Data were extracted from a deidentified RedCap Repository.

Setting: Tertiary care multispecialty medical center.

Patients: Subjects were 212 consecutive adults meeting prespecified inclusion criteria who were evaluated in the Balance Disorders Clinic at Vanderbilt University Medical Center between 2011 and 2017. Patients with bilaterally absent VEMPs were excluded from the study.

Intervention(s): None.

Main Outcome Measure(s): Proportions of subjects with or without VM in one of the following four test outcomes: normal cVEMP/normal oVEMP, abnormal cVEMP/abnormal oVEMP, abnormal cVEMP/normal oVEMP, and normal cVEMP/abnormal oVEMP.

Results: There was a significant relationship between VM and cVEMP and oVEMP test outcomes.

Conclusion: Patients with VM are more likely than subjects with vestibular disorders other than migraine to exhibit normal cVEMP responses in the presence of unilaterally abnormal oVEMP responses. Such a VEMP pattern may be a biomarker of VM and further supports a possible pathophysiologic relationship between the utriculo-ocular reflex and VM.
Pharmacologic Management

• Quick relief: calcium antagonist (flunarizine, verapamil)
  – Sedation, weight gain, extrapyramidal, depression
• VM + hypertension: betablocker
  – Bronchospasm, bradycardia
• VM with prominent headache: anticonvulsant
  – VM + Headache + obesity: topiramate
  – VM + Headache: valproate or betablocker
• VM + poor sleep + anxiety: amitryptiline, nortryptiline
  – Prominent anxiety: SSRI, benzodiazepine
• VM with headache rare: anticonvulsant (lamotrigine)
• Acetazolamide may be helpful: support for use with episodic Ataxia 2 and Familial Hemiplegic migraine

(Bisdorff, 2011)
What about us?

• “The appropriate treatment plan is similar to that for classic migraine. This includes lifestyle changes, such as stress reduction, nutritious diet, avoiding nicotine, or avoiding irregular sleep patterns.” (Honaker & Samy, 2008)

• “General recommendations for migraine headache prophylaxis, such as diet, sleep hygiene, avoidance of trigger factors, are also probably beneficial for migrainous vertigo.” (Bisdorff, 2011)

• “Nonpharmacological treatment options for VM such as avoiding triggering factors, getting regular sleep and meals, and exercising may also be helpful for VM and should be considered as preventative measures, as with general migraine.” (Sohn, 2016)
Migraine Triggers

- Stress
- Hormones
- Poor Exercise Habits
- Foods
  - Skipping meals
- Poor Sleep
Acceptance of Diagnosis

• Poor temporal correlation with headache (Bisdorff, 2011)
  – No headache at all in 30-50%
• May not have “bad headache”
• Dx criteria for VM include current OR past history
  – Headache not mandatory if other migrainous features are present
• Acceptance is key to compliance with recommendations
Tougher to Manage

- **Stress**
- **Exercise**
  - May feel to poorly
  - Helps with stress
  - Weight reduction
- **Hormones**
  - Oral Contraception
  - Hormone Replacement Therapy
Importance of Sleep

• Sleep is a key factor

• any disturbance from the normal routine of sleep is potential trigger
  – staying up too late
  – getting up earlier than usual
  – change in activity pattern due to shift work
  – jet lag from crossing to many time zones
  – oversleeping
  – erratic sleep schedule
  – sleep disorder such as obstructive sleep apnea

Moskowitz, 2016
• **Food sensitivities**
  – Immunoglobulin antibodies
  – Eliminating reactive foods 84% had complete or partial elimination of headache *(Arroyave et al., 2007)*
  – Less response in some other studies because of poor control/compliance

• **Chemicals**
  – Ethanol, caffeine, sodium nitrate, phenylethylamine, tyramine, MSG, benzoic acid, theobromine, sodium metabisulfite

• **68-93% of patients improved on various types of diets** *(Di Lorenzo et al., 2015; Egger et al., 1983; Wantke et al., 1993)*
Acceptance = Adherence

“An important component of effective management for people with frequent or severe migraine is to identify the trigger factors and aggressively neutralize them. The major challenge is convincing the migrainer to make the often disruptive life adjustments that are needed to manage these triggers, and to give the changes enough time to assess their efficacy.” Moskowitz, 2016

Patients with Chronic Migraine (22%) exhibited less compliance with regular lifestyle behavior than patients with Episodic Migraine (69%) (Woldeamanuel & Cowan, 2016)
Non-Medical Management of Vestibular Migraine

• Commit to 60-day rigid pattern
• Start exercising
• Sleep/wake at same times daily
• Eat at same times

Common Triggers for Migraine

Stress
Alcohol (red wine, beer)

Foods
  Chocolate
  Aspartame (artificial sweetener)
  Cured/Aged meats
  Cheese
  Yeast
  Canned Soup
  Monosodium Glutamate (MSG)
  Pickled, fermented and marinated foods

Poor sleep habits
Poor exercise habits
Irregular meal times

Adapted from Gans & Roberts “Unraveling the mystery of migraine.” Hearing Health, Fall 2004.
**Hx & Sx:**

- 9 y.o. Male
- Onset 2 mos. prior
- Rotary vertigo with nausea and imbalance weekly
- Duration = min-hours
- Dark room with cool compresses
- Same strategies he uses for his migraines (2-3 x/month)
- Developmental milestones WNL
- New glasses
- Zofran four times since onset

<table>
<thead>
<tr>
<th>MRI WNL</th>
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<tbody>
<tr>
<td>EEG indicated frontal lobe seizures</td>
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<tr>
<td>Started anti-seizure meds</td>
</tr>
<tr>
<td>Unaware of migraine triggers</td>
</tr>
<tr>
<td>Little physical activity but new bicycle for Christmas</td>
</tr>
<tr>
<td>DHI = 48</td>
</tr>
</tbody>
</table>
mCTSIB

NORMAL  NORMAL  NORMAL  NORMAL

CDVAT

Total 96%  V:Total 95%  H:Total 96%
**Hx & Sx:**
- 9 y.o. Male
- Onset 2 mos. prior
- Rotary vertigo with nausea and imbalance weekly
- Duration = min-hours
- Dark room with cool compresses
- Same strategies he uses for his migraines (2-3 x/month)
- Developmental milestones WNL
- New glasses
- Zofran four times since onset

**Dx:**
1. Vestibular Migraine
2. Seizures

**Lifestyle Modifications with Elimination Diet**

**MRI WNL**
EEG indicated frontal lobe seizures
Started anti-seizure meds
Unaware of migraine triggers
Little physical activity but new bicycle for Christmas
DHI = 48

**VOR/VNG/CDVAT**

**VSR/mCTSIB**

**VCR/cVEMP**

VNG WNL
DVA WNL

Normal
Migraine Case

• “Gas Station Food”

• Two weeks later
  – Mother stopped into office amazed
  – No more vertigo and no more headaches

• Anti-seizure medication
Vestibular Migraine Summary

- More difficult to identify and manage than BPPV
- **$1.33 Billion** economic impact
- Some evidence HRQoL worse for VM than migraine
- Evidence that dietary changes alone can decrease symptoms of migraine in **68 – 93%** of individuals
  - Should achieve greater improvement with other lifestyle modifications
- Acceptance of diagnosis and compliance are key
- Even if we decreased costs by half, could potentially save **$665 Million**!
Overall Summary

• Management is in our scope
• 10-20 minutes
• 25,392,000 million lives
• Conservatively could save: $1,713,500,000
Dear Dr. Richard Roberts,

I meant to write much sooner to thank you. I did the exercises you prescribed and it fixed my problem completely. I am back to doing my Jillian Michaels workouts with no problem. I rarely think about my pin predilection other than to say “Thank God that Dr. Roberts cured me!” Please know that you make a difference in the lives of your patients. I, for one, am very grateful to you.

God bless you.