Utilizing Vestibular Function Testing to Assess and Treat Vestibular System Dysfunction

Combined Sections Meeting 2013
San Diego CA, January 24, 2013
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Vanderbilt Bill Wilkerson Center Nashville, Tennessee
Course Objectives for Participants

• Discuss challenges faced in PT assessment and management of concomitant BPPV and peripheral hypofunction.
• Identify the most common causes for bilateral vestibular hypofunction.
• Discuss functional impairments and rationale for treatment in patients with bilateral vestibular loss.
• Identify central and peripheral causes of vestibular dysfunction.
Course Objectives for Participants

• Discuss the importance of diagnostic vestibular function testing, in addition to PT examination, identifying and differentiating impairments that impact PT interventions in patients with vestibular dysfunction.

• Discuss the importance of PT examination in conjunction with vestibular function testing for comprehensive assessment of static vs. dynamic vestibular system compensation.
Course Objectives for Participants

• List the primary diagnostic vestibular function procedures used in the identification of peripheral and central vestibular system deficits and what each test tells you in respect to the functional integrity of the vestibular system.

• Differentiate between a compensated vs. uncompensated peripheral vestibular system disorder as well as complete vs. subtotal bilateral peripheral vestibular system hypofunction.
Course Objectives for Participants

• Describe how vestibular rehabilitative therapy goals for treatment will vary based on the type and degree of vestibular dysfunction.
Vanderbilt Bill Wilkerson Center for Otolaryngology and Communication Sciences

- Department of Audiology (Balance Function Testing, Pediatric and Adult Hearing Clinics)
- Pi Beta Phi Rehabilitation Institute (PT, OT, ST, MSW, Driving Program, Seating and Mobility, Neurological PT Clinical Residency Program)
- Department of Otolaryngology (Neurotology, Cochlear Implantation, Voice Center, Head and Neck Cancer, Otology)
- Department of Speech Language Pathology (Adult and Pediatric programs, Mama Lere School)
- Graduate level teaching (MD, SLP, AuD, PT, OT)
COI Disclosure

• We have NO financial relationships to disclose.
Vestibular Function Testing

Sarah L. Grantham, Au.D.
Balance Disorders Laboratories

Rotary Chair

VNG/ENG 1

VNG/ENG 2

VNG/ENG 3

Posturography
Clinical Tests/Programs

- Electro-Videonystagmography (ENG/VNG)
- Rotational Testing
- Vestibular Evoked Myogenic Potentials
  - Cervical (cVEMP)
  - Ocular (oVEMP)
- Computerized Dynamic Posturography
- Pediatric Balance Function Assessment
- Falls Risk Assessment
Objectives

• Present current Vestibular Function Test (VFT) battery

  – Review the neural pathways of:
    • The Vestibulo-Ocular Reflex (VOR)
    • The Vestibulo-Collic Reflex (VCR)

  – Review recording techniques and interpretation of VFT findings.

• Present patterns of abnormality commonly identified during VFT.
ENG/VNG

• Ocular Motility
  – Saccades, Smooth Pursuit, Optokinetics, Spontaneous and/or Gaze-Evoked Nystagmus

• Positional/Positioning Testing
  – Benign Paroxysmal Positional Vertigo (BPPV)
  – Spontaneous & Central Positional Nystagmus

• Bithermal Caloric Test
  – “Gold Standard” for identifying peripheral vestibular system impairment affecting the lateral SCC and/or superior vestibular nerve.
Vestibulo-Ocular Reflex (VOR)

- Stimulation of the Ipsilateral Horizontal Semicircular Canal (hSCC)
  - Activates: Ipsi. Medial Rectus, Contra. Lateral Rectus
  - Inhibits: Ipsi. Lateral Rectus, Contra. Medial Rectus
  - Generates a horizontal eye movement with slow phase away from stimulated ear
Benign Paroxysmal Positional Vertigo (BPPV)

• Screening for BPPV
• Dix-Hallpike Assesses:
  – Posterior SCC (pSCC)
    • Up-beating Nystagmus
  – Anterior SCC (aSCC)
    • Down-beating Nystagmus
• Roll Tests Assess:
  – Horizontal SCC (hSCC)
    • Geotropic vs. Ageotropic Nystagmus
Pathophysiology of BPPV

- Displaced otoconia act as mobile densities within the canal.

- Head movement causes mass of otoconia to shift within the SCC.

- Endolymphatic fluid becomes displaced, deflecting the cupula which elicits nystagmus and vertigo.
Nystagmus Characteristics of h-BPPV
Three Subtypes

• Bilateral Geotropic Nystagmus (Canalolithiasis)
  – Otoconial debris within posterior arm of hSCC

• Bilateral Ageotropic Nystagmus
  – Reverts to Geotropic
  – Otoconial debris within anterior arm of hSCC

• Bilateral Ageotropic Nystagmus (Cupulolithiasis)
  – Persistent ageotropic nystagmus
  – Otoconial debris located on utricular side of hSCC
Geotropic h-BPPV

- Geotropic variant: Rotation of the head results in horizontal nystagmus which beats toward the undermost ear.

  - With geotropic nystagmus, the location of the debris within the canal causes ampullopetal endolymph flow which generates excitatory GEOtropic nystagmus (i.e. the affected side generates the MORE intense nystagmus response).
Geotropic-Posterior Arm hSCC

Higher SPV  Lower SPV
Ageotropic h-BPPV

• Ageotropic variant: Rotation of the head results in horizontal nystagmus which beats toward the uppermost ear.

• With ageotropic nystagmus, the location of the debris within the canal causes ampullofugal endolymph flow which generates inhibitory AGEOtropic nystagmus (i.e. the affected side generates the LESS intense nystagmus response).
Ageotropic-Anterior Arm hSCC (Canal Side)

Lower SPV

Higher SPV
Ageotropic-Anterior Arm hSCC (Utricular Side)

Lower SPV  Higher SPV
Flow Chart for the Treatment of hSCC BPPV

Horizontal BPPV

GEO
- Appiani Log Roll

AGEO
- Gufoni
  - Does not Convert to Geotropic
    - Casani Modified Semont
    - Modified Brandt-Daroff
- Converts to Geotropic
Electro-Videonystagmography: Bithermal Caloric Test

Remember “COWS”:
Cold – Opposite
Warm – Same
Bithermal Caloric Norms and Terms of Use

• Quantification of the VOR:
  – Slow Phase Velocity (SPV)
  – Degrees per Second (deg/sec)

• Total Caloric Response
  – <22 deg/sec suggests a Bilateral deficit.

• Unilateral Weakness
  – >22% asymmetry suggest a Unilateral peripheral deficit

• Directional Preponderance
  – >28% is abnormal
Rotary Chair

- Assesses the VOR over a broader operational range of frequencies (0.01Hz-0.64Hz).
  - Calorics assess VOR at ~0.003Hz (very low frequency response)
- Useful in determining:
  - Central Compensation of Unilateral Peripheral Deficits
  - Degree of Bilateral Peripheral Vestibular System Hypofunction
  - Identification of Central Vestibular System Impairments
**Phase:** Timing relationship between eye and head velocity.

**Gain:** The ratio of peak eye velocity to head velocity.

**Symmetry:** The ratio of rightward versus leftward SPV.
Acutely Uncompensated Unilateral Peripheral Deficit
Profound Bilateral Hypofunction
Acoustical Cervical VEMP (cVEMP)

P13

N23
Acoustically Evoked Sonomotor Response

- The cVEMP is a stimulus-related attenuation of tonic EMG activity.

- An acoustically evoked toneburst (500Hz) stimulus acts as a hydro-mechanical force to move the endolymphatic fluid and, as a consequence, translates otoliths to create transduction.
The Receptor for cVEMP is the Saccule. Halmagyi & Curthoys (1999)

- The Saccule is the vestibular end organ most sensitive to sound.
- Lies under the stapes footplate.
- Neurons from saccular macula respond to tilts and click stimuli.
- Electrical output from the saccule is routed through the inferior vestibular nerve.
Central Connections & Efferent Pathway
“Vestibulocollic Reflex”
(From: Rosengren et al. 2009)

- Saccule (a)
- Scarpa’s ganglion (a)
- Inferior vestibular nerve (a)
- Vestibular nucleus (a)
- Medial vestibulospinal tract (MVST) – (e)
- Spinal accessory nucleus of CN XI (e)
- CN XI (e)

a = afferent, e = efferent
How do we activate the SCM?

Unilateral Activation/Recording

Testing right

Testing left
Acoustical Ocular VEMP (oVEMP)
The oVEMP Pathway
(after Curthoys et al., 2011 and Manzari et al., 2010)

- Utricle
- Sup. Vestib. Nerve
- Medial Longitudinal Fasciculus
- Motor Nucleus of Contra CN III
- CN III
- Contra Inferior Oblique m.
Test-specific Topological Localization

• Caloric Test
  – Horizontal Semicircular Canal
  – Ampullary Branch of the Superior Vestibular Nerve

• oVEMP
  – Utricle
  – Utricular Branch of the Superior Vestibular Nerve

• cVEMP
  – Saccule
  – Inferior Vestibular Nerve

Image from Manzari et al., 2010
Patterns of Abnormality in cVEMP, oVEMP, and Caloric Tests May Provide Topological Information about Vestibular Impairment

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Gary P. Jacobson*
Devin L. McCaslin*
Erin G. Piker*
Jill Gruenwald*
Sarah L. Grantham*
Lauren Tegel*
### Possible Patterns of Impairment and Their Clinical Significance

<table>
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<tr>
<th>Type</th>
<th>Caloric</th>
<th>cVEMP</th>
<th>oVEMP</th>
<th>Impairment</th>
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<td>NI</td>
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<tr>
<td>2</td>
<td>Abn</td>
<td>Abn</td>
<td>Abn</td>
<td>Large end organ, or inf. and sup. vestibular nerve</td>
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<tr>
<td>3</td>
<td>NI</td>
<td>Abn</td>
<td>NI</td>
<td>Saccule or inf. vestibular nerve</td>
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<tr>
<td>4</td>
<td>Abn</td>
<td>NI</td>
<td>Abn</td>
<td>Sup. vestibular n.</td>
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<td>Abn</td>
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<td>NI</td>
<td>hSCC</td>
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<tr>
<td>6</td>
<td>NI</td>
<td>NI</td>
<td>Abn</td>
<td>Utricle</td>
</tr>
<tr>
<td>7</td>
<td>NI</td>
<td>Abn</td>
<td>Abn</td>
<td>Utricle + saccule or inf. vestibular nerve?</td>
</tr>
<tr>
<td>8</td>
<td>Abn</td>
<td>Abn</td>
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<td>hSCC + saccule, and/or inf. vestibular n.?</td>
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<tr>
<td>9</td>
<td>NI</td>
<td>Abn*</td>
<td>Abn*</td>
<td>SSCD Syndrome</td>
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</table>

* = Abnormally reduced VEMP thresholds
Insensitivity of the Romberg Test of Standing Balance on Firm and Compliant Support Surfaces (RTSBFCSS) in Diagnosing Vestibular System Disorders (Jacobson et al., 2011)

• Using the NHANES subject selection criteria with the RTSBFCSS offers poor sensitivity (≤55%) in the detection of vestibular system impairments.

• The RTSBFCSS is a test of “Balance” NOT “Vestibular Function”.
  – Balance requires central integration of multiple sensory inputs and motor outputs.
  – Whereas vestibular function relies specifically on the integrity of the vestibular end organ and their afferent and efferent pathways.
Excellent Reference Materials

Electronystagmography and Videonystagmography

ENG / VNG

Devin L. McCaslin

A Volume in the Core Clinical Concepts in Audiology Series

BALANCE FUNCTION ASSESSMENT AND MANAGEMENT

Gary P. Jacobson
Neil T. Shepard
A Case of Concomitant Vestibulopathy and BPPV

Pat Flemming PT DSc GCS
Background.. Case One

• Dizziness is one of the most frequent clinical drivers for a visit to a physician or other health care provider.
• Treatment may be provided by a MD, nurse practitioner & others in a variety of clinical settings (i.e. ER, out-pt.)
• Common diagnoses referred to PTs specializing in vestibular rehabilitation therapy (VRT) are Benign Paroxysmal Positional Vertigo (BPPV) and Unilateral Peripheral Hypofunction.
• Many times, however, a definitive diagnosis has not been reached; the referring diagnosis is non-specific or misleading, i.e. Dizziness, Ataxia.
Background.. continued

- Sophisticated diagnostic equipment or therapists specializing vestibular rehabilitation with may not be available in all communities.
- BPPV may present alone or in combination with other vestibular dysfunction.
- In some cases, a peripheral hypofunction may go untreated since the most provoking and urgent symptoms are related to BPPV.
- Vestibular function testing can assist in identifying underlying vestibulopathy which may benefit from vestibular rehabilitation.
Purpose

• The primary purpose of this case is to describe a patient presenting with concomitant unilateral peripheral vestibulopathy and BPPV.

• Secondary purposes include:
  – Increase therapist awareness of vestibular co-morbidities in patients they may evaluate and treat
  – Increase therapist awareness of the benefits of audiological vestibular function testing in diagnostics
  – Describe bedside tests that may be utilized by therapists for screening when vestibular function testing is not available in the community
BPPV Etiology and Characteristics

• Often occurs spontaneously; remission can also be spontaneous.
• May follow trauma to the head, labyrinthitis, and/or ischemia.
• Symptoms evoked with looking down or up, rolling over in bed, putting head back for an appointment, i.e. dentist.
• Vertigo often fleeting, but symptoms of imbalance, lightheadedness may persist.

S. Herdman, Vestibular Rehabilitation, Ch.17, 2007.
Vestibular Hypofunction: Etiology and Characteristics

- Vestibular neuritis, and labyrinthitis are common causes.
- Symptoms may include vertigo, dysequilibrium, acute nausea/vomiting, oscillopsia, tinnitus, dizziness, imbalance, blurred vision, falls history, fear of movement, and hearing loss.

Herdman, S. Vestibular Rehabilitation, Ch. 19, 2007.
Diagnostics: Vestibular Hypofunction

- Bithermal caloric test is considered a significant test in diagnosis of vestibular dysfunction.
- A difference of more than 20-23% between responses from the ears is considered clinically significant for a weak vestibular system.
- A unilateral weakness implies vestibular disorder on the side of the decreased response; there is asymmetry in the magnitude of information entering the brainstem, resulting in different nystagmus characteristics from each ear.

Jacobson et al., 1997; Jonkees et al., 1962.
Concomitant Vestibulopathy and BPPV

• Numerous disorders may cause the presence of both disorders.
  – Vestibular neuritis
  – Labyrinthitis
  – Meniere’s Disease
  – Cerebrovascular disease, including CVA, TIA, migraines, basilar artery insufficiency, vertebrobasilar insufficiency
  – Head Trauma

Primary and Secondary BPPV

• Patients without history of otologic pathology have been described as presenting with primary BPPV; with a definite history of otologic pathology, secondary BPPV.

• Prevalence of vestibulopathy in individuals with BPPV has been reported in range of 13 -50%.
Prevalence of Vestibulopathy in Benign Positional Vertigo Patients With and Without Prior Otologic History


- **Purpose:** determine prevalence of vestibulopathy in two groups of participants presenting with posterior canal BPPV; retrospective review of 157 charts.

- Patients divided into two groups based on otological history: 49 patients with positive prior history, 108 with negative history.

- Caloric testing performed on all patients as part of assessment of vertigo.

- Findings: Vestibulopathy present in 53.1% of patients with positive Hx, 30.6% in patients with negative Hx.
Clinical Significance of Concomitant BPPV and Vestibulopathy

• Patients with BPPV and additional vestibulopathy may have a greater incidence of symptoms following BPPV Rx than those with BPPV alone.
• Underlying vestibular disorder may result in dysequilibrium, lightheadedness, increase falls risk.
• Vestibulopathy may be caused by varied conditions: uncompensated peripheral hypofunction, an unidentified vertical canal, otolith, or central vestibular dysfunction.

Roberts et.al, 2005.
Case Description

• 54 y.o. female reported to ER in October via ambulance after 1 week of mild dizziness and lightheadedness. Chief complaint: Onset of constant dizziness. Severe episode of nausea, imbalance, unable to walk alone, vertigo with certain head movements on day presenting to ER. She had vomited x2. Hypertension noted en route. Initial concern: possible CVA.

• MRI at time of ER visit
  – Negative for acute infarct; Old infarct of left centrum semivale; MRA of head/ neck negative for stenosis, dissection/ or aneurysm
Patient History and Hospital Course

- Patient history included heart murmur, + TB skin test (completed INH 20 years prior), G3G4 (twins), hearing loss in L ear, vertigo, tinnitus x1 day
- Employed as RN, married, working one full and one part-time job.
- Neurology consult in ER: “most likely viral acute labyrinthitis or vestibular neuronitis; onset too abrupt with constant symptoms unlikely to be BPPV. Treat with steroids, meclizine, fluids, refer to ENT”. Patient admitted for overnight observation.
Hospital Course.. Continued

- ENT consult during in-patient stay:
  - Audiogram revealed L sided high-frequency SNHL; patient reported long standing hearing loss vs. acute loss
  - Dix Hallpike Test: positive to the left
  - Epley maneuver performed, followed by patient vomiting. After recovery, Dix Hallpike repeated with < vertigo and rotary nystagmus. Canal involvement not documented.
  - Discharge following one overnight; referral for audiologic balance function testing as out-patient.
  - ENT resident assessment “Findings consistent with BPPV”.
Vestibular Function Testing
(28 Days s/p Hospital Discharge)

• Impressions:
  – BPPV affecting the L horizontal semi-circular canal; geotropic nystagmus (treated x1 with Bar-b-q Roll)
  – Caloric testing: L peripheral vestibular system disorder (50% weakness)
  – Absent cVEMP and oVEMP on the left
  – Rotary Chair Test: Uncompensated weakness. Asymmetries to the right consistent with L beating recovery nystagmus.
  – 46/100 Dizziness Handicap Inventory (severe perceived handicap); abnormal anxiety rating (HADS)
Recovery Nystagmus

- Fine, left-beating nystagmus
Roll Tests

Head Left

Geotropic Nystagmus
(Greater SPV Head LEFT)

Head Right
VNG

- NORMAL
  - Ocular motility

- ABNORMAL
  - Fine, left-beating recovery nystagmus
  - Geotropic nystagmus c/w LEFT hSCC BPPV
  - 50% unilateral weakness in the LEFT ear.
Abnormal cVEMP examination
- Absent LEFT cVEMP

Amplitude Asymmetry Ratio:
- 100% to the LEFT
- >47% is Abnormal

Impaired Saccule and/or Inferior Vestibular Nerve
oVEMP

- Abnormal oVEMP examination
  - Absent LEFT oVEMP

- Amplitude Asymmetry Ratio:
  - 100% to the LEFT
  - >33% is Abnormal

- Impairment affecting at least the Superior Vestibular Nerve
Rotary Chair

Multi-frequency Asymmetries (0.01Hz-0.04Hz)
Presence of Multi-frequency asymmetries Indicates INCOMPLETE Compensation
Physical Therapy Initial Evaluation

• Subjective: inability to work currently as RN.
• PT Eval: 4 days post vestibular function testing.
• InVision Testing initiated, but discontinued during Gaze Stabilization Test. Static and dynamic visual acuity then assessed with Snellen chart seated from 10’. 20/16 static visual acuity; 20/50 (5 Line Drop with imposed horizontal head movements.)
• Video goggles utilized to perform Dix Hallpike and Roll Tests: Left beating horizontal nystagmus > 1 min. duration noted with Roll Test bilaterally, indicating L horizontal canal cupulolithiasis.
Physical Therapy Initial Intervention

• Quick bar-b-q Roll initiated from the left, followed by Casani (modified Semont maneuver.)

• Patient instruction : prolonged positioning at night, lying initially on the left, then rolling to the right. Patient instructed to repeat process if out of bed during night or aware she had changed position.

• Patient instruction : Casani maneuver for follow through daily at home.

• VOR 1 viewing and two target eye/ head substitution exercises.
Second PT Visit

• Subjective report: still dizzy, no true vertigo, performing prolonged positioning & Casani
• Negative for BPPV using video goggles
• No subjective report of vertigo
• Left beating nystagmus of lesser degree.
• Progression to VOR 1 view full field stimulus.
• Self canalith repositioning maneuver discontinued due to resolution of BPPV.
Third PT Visit

• Progression to VOR 2 viewing ex. , negative Dix Hallpike/ Roll Tests.

• Completion of InVision Testing.
  – Perception Time: 30 msec.
  – Gaze Stabilization : 83 degrees/ second left; 90 degrees second/ right
  – Dynamic Visual Acuity: .12 Logmar loss to L, .32 Logmar loss to R with horizontal head movements; WNL with vertical head movements
Perception Time Test

Testing Distance: 10.0 feet

Static Acuity: -0.20 logMAR
Testing Settings: 0.00 logMAR

Minimum Perception Time

milliseconds

30
Gaze Stabilization Test

Head Movement: Horizontal
Static Acuity: -0.20 logMAR
Perception Time: 30 msec

Testing Distance: 10.0 feet
Optotype Size: 0.00 logMAR
Optotype Display Interval: 40 - 75 msec

Average Achieved Velocity

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<th>Right</th>
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Task Range

Velocity Symmetry (%)

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Average Achieved Velocity:

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<tr>
<td>83</td>
<td>90 **</td>
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</table>
Dynamic Visual Acuity Test

Perception Time: 30 ms
Optotype Display Interval: 40 - 75 ms
Head Movement: 85 deg/sec Horizontal
Testing Distance: 10.0 feet

DVA Loss

logMAR

Direction

Left  Right

0.12  0.32

DVA Loss Symmetry (%)

Direction

Left  Right

50  50

13

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<tr>
<th>Left Direction</th>
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<td>Snellen Fraction:</td>
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<td>20/13</td>
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<td>logMAR:</td>
<td>-0.08</td>
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Dynamic Visual Acuity Test

Perception Time: 30 ms
Optotype Display Interval: 40 - 75 ms

Head Movement: 60 deg/sec Vertical
Testing Distance: 10.0 feet

DVA Loss

DVA Loss Symmetry (%)
Fourth PT (VRT) Visit

- Subjective report: feeling better, off FMLA, able to return to work part-time. Continuing with gaze stabilization / VOR program. Still reporting dizziness with quick head movements.
- Gaze Stabilization Test: 87 degrees/ second to the left; 127 degrees/ second to right (19% asymmetry).
- Progressed to Week 5/6 of the gaze stabilization program (VOR1 and 2, two target eye/ head).
Gaze Stabilization Test

Head Movement: Horizontal
Static Acuity: -0.20 logMAR
Perception Time: 20 msec

Testing Distance: 10.0 feet
Optotype Size: 0.00 logMAR
Optotype Display Interval: 40 - 75 ms

Average Achieved Velocity

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Task Range

Velocity Symmetry (%)

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Discharge Visit (#5): Patient Outcomes

• 5 PT visits over 10 weeks (December to February)
• 30/30 on the Functional Gait Assessment
• 12/100 Dizziness Handicap Inventory (absent perceived handicap vs. 46/100 initially)
• Snellen Chart: accurate at 2 line drop with imposed horizontal head movements at 2 Hz.
• Subjective report of return to work at 36 hr/ week as RN, looking for supplemental prn work as well
• Instruction in continuing gaze stabilization program over next several months.
Use of Vestibular Function Testing Results: Patient with BPPV and Hypofunction

- > collaboration between healthcare providers
- Vestibular Function Testing alerted therapist to multiple areas of vestibulopathy which could affect patient’s functional status:
  - Absent VEMPs on the left
  - L peripheral system disorder (50% weakness)
  - Left beating recovery nystagmus
  - Identification of canal affected 4 days prior to PT evaluation; Left horizontal SCC BPPV canaliathiasis
  - High perceived dizziness handicap; high anxiety
Benefit of Collaborative Testing

• Without benefit of vestibular function testing, increased possibility of “tunnel vision” by therapist and/or patient, focusing solely on the BPPV.
• Audiologists skilled in performing VEMPs, ENG/VNG, Rotary Chair, etc.
• PTs skilled in assessing postural control, gait, functional balance.
• The patient “wins” with comprehensive assessment/Rx from the vestibular rehab team.
Take Home Message: Case One

• Consider underlying vestibulopathy when a referral for BPPV is received or when you identify a patient with BPPV; consider referral for vestibular function testing.

• Perform “bedside tests” if vestibular function testing is not available in your community: Head Impulse Test, screening with Snellen chart.

• Don’t assume that if the BPPV is resolved, the patient’s functional deficits are resolved.

• Create a vestibular rehabilitation network within your region to provide optimal care for patients.
A Case of Bilateral Vestibular Hypofunction

Cathey Norton, PT
Background

• Bilateral Vestibular Hypofunction (BVH) is less common than unilateral hypofunction and less often diagnosed as a cause of dizziness.
• Imbalance, lightheadedness and oscillopsia are common complaints.
• BVH leads to increased risk for falls, decreased vocational ability, participation in leisure activities, driving and social isolation.
• Since symptoms mimic many nonvestibular causes of dizziness, Vestibular Function Testing is beneficial in determining proper treatment and functional outcomes for patients with bilateral vestibular hypofunction.

Schubert, 2004; Brown et al, 2001
Purpose

• Describe common presenting symptoms and causes of bilateral vestibular hypofunction (BVH)
• Discuss benefits of Vestibular Function Testing results in planning appropriate treatment
• Present appropriate clinical tests and measures
• Describe rationale for treatment of BVH
• Discuss expected outcomes for patients with BVH
Common Causes of BVH

- Ototoxicity
- Meningitis
- Labyrinthine infection / bilateral neuritis
- Otosclerosis
- Paget’s disease
- Bilateral tumors
- Endolymphatic Hydrops
- Polyneuropathy
- Autoimmune Disease
- Congenital
- Ototoxic medications (e.g., aminoglycosides, cisplatin)
- Idiopathic vestibular loss
- Bilateral Meniere’s disease
- Cerebellar ataxia with neuropathy and bilateral vestibular areflexia syndrome (CANVAS)
- Trauma
- Autoimmune disease
- Genetic disease
- Meningitis
- Neurofibromatosis type 2
Causes of BVH

• The cause of BV remains unclear in about half of all patients.
• A large subgroup of these patients have associated cerebellar dysfunction and peripheral polyneuropathy.
• This suggests a new syndrome that may be caused by neurodegenerative or autoimmune processes.
Case Description

- Referral from Otolaryngology Nurse Practitioner.
- Referring Diagnosis - Vertigo, peripheral- 386.10
- Initial evaluation 9/3/09
- 30 year old female
- Onset: 2-3 years ago
- Chief complaints – Patient reports 2-3 years of feeling wobbly in her head with multiple brief episodes of vertigo which only last minutes and does not return for weeks.
- Described as an episode of her eyes shifting "like someone hit me in the head" and feeling off balance in the dark.
Patient History

• Subjective: "I feel off balance and like my eyes will not stay still at times"
• Patient Goals: “I want to have better balance and get back to normal. I need to be able to drive with my kids in the car.”
• Neurology consult: 4/28/09 - Impression: “Recurrent dizziness of uncertain cause. I do not think that she has a vestibular migraine. I do not have a clear explanation for her recent episodes of transient/fleeting visual shift. The comprehensive examination shows no evidence for cerebellar syndrome or benign positional vertigo.”
• Diagnostic testing: MRI 5/7/09 – normal
• Referred to Otolaryngology
• Vestibular Function Testing: 6/2/09
Medical History

- DM - TYPE I since age 3 (has insulin pump)
- UTI 3/04-Cipro
- PYELONEPHRITIS - April 2001
- NPDR OU (non proliferative diabetic retinopathy)
- Graves Disease 2005, I-131 treatment (radioiodine treatment)
- Hypothyroidism after ablation
- Dysuria 4/04
- URI 2/09
- Elevated blood pressure 04-09
- Four wisdom teeth removed
- Renal cyst left drained '99.
VFT Results

- **VNG (ABNORMAL)**
  - Fine spontaneous versus central positional nystagmus
  - Abnormally Reduced Total Caloric Response
    - 9 deg/sec – 2009
    - 11 deg/sec – 2010
    - <22 deg/sec suggests Bilateral Peripheral Vestibular System Hypofunction

- **cVEMP (NORMAL)**
  - Bilaterally Normal Responses
  - INTACT Saccule & Inferior Vestibular Nerve
Rotary Chair

Multi-frequency VOR gain reductions with asymmetries and phase leads (Note preserved high frequency function)
Impression

• Bilateral Vestibular hypofunction based on:
  – Visual disturbances (oscillopsia)
  – Imbalance – especially in the dark
  – Vestibular function testing
  – Normal MRI
Examination

- ROM, Strength, coordination, muscle tone, bed mobility, transfers and ambulation normal
- Sensation testing performed in Diabetic Clinic and reported as normal.
- Oculomotor exam normal
- Head Impulse test: abnormal bilaterally
- Dynamic Gait Index: 22/24
DGI Videos
Sensory Organization Test
(Sway Referenced Gain: 1.0)

Equilibrium Score

Sensory Analysis

Strategy Analysis

COG Alignment
Gaze Stabilization Test

Head Movement: Horizontal
Static Acuity: -0.08 logMAR
Perception Time: 20 msec

Testing Distance: 10.0 feet
Optotype Size: 0.12 logMAR
Optotype Display Interval: 40 - 75 msec

Average Achieved Velocity:

- Left Direction: 75 **
- Right Direction: 155 **

Velocity Symmetry (%):

- Left: 35
Dynamic Visual Acuity Test

Perception Time: 20 ms
Optotype Display Interval: 40 - 75 ms

Head Movement: 85 deg/sec Horizontal
Testing Distance: 10.0 feet

DVA Loss

DVA Loss Symmetry (%)

<table>
<thead>
<tr>
<th>Direction</th>
<th>Left Direction</th>
<th>Static</th>
<th>Right Direction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snellen Fraction:</td>
<td>20/21</td>
<td>20/17</td>
<td>20/21</td>
</tr>
<tr>
<td>logMAR:</td>
<td>0.02</td>
<td>-0.08</td>
<td>0.02</td>
</tr>
<tr>
<td>Average Achieved Velocity(deg/sec):</td>
<td>107</td>
<td>0</td>
<td>104</td>
</tr>
</tbody>
</table>
Intervention

- Patient elected to attend therapy at a facility closer to her home as she is not comfortable driving and lives 25 miles from the clinic.
- She was referred to a facility with a PT who has experience in Vestibular Rehabilitation. The clinic is 5 miles from her home and she feels she will be able to attend therapy there.
Second Evaluation

• 6/29/10 (9 months following initial evaluation)
• Patient did not attend therapy at the local facility
• Chief complaints - She has been unable to drive for the past month due to increase in symptoms of dizziness when driving. She feels dizzy when shopping in the grocery store, at church and in restaurants. She reports nausea when she gets dizzy.
• Physical exam unchanged
• DGI 22/24 remains unchanged
Sensory Organization Test
(Sway Referenced Gain: 1.0)

Equilibrium Score

Composite: 52

Sensory Analysis

Strategy Analysis

COG Alignment
Gaze Stabilization Test

Head Movement: Horizontal
Static Acuity: -0.16 logMAR
Perception Time: 20 msec

Testing Distance: 10.0 feet
Optotype Size: 0.04 logMAR
Optotype Display Interval: 40 - 75 msec

Average Achieved Velocity

<table>
<thead>
<tr>
<th>Degrees/Second</th>
<th>Left</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>128</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Velocity Symmetry (%)

<table>
<thead>
<tr>
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<th>Right Direction</th>
</tr>
</thead>
<tbody>
<tr>
<td>128</td>
<td>142</td>
</tr>
</tbody>
</table>
Dynamic Visual Acuity Test

Perception Time: 20 ms
Optotype Display Interval: 40 - 75 ms

Head Movement: 85 deg/sec Horizontal
Testing Distance: 10.0 feet

DVA Loss

DVA Loss Symmetry (%)

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<th>Right Direction</th>
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<td>20/25</td>
</tr>
<tr>
<td>logMAR:</td>
<td>0.03</td>
<td>-0.16</td>
<td>0.10</td>
</tr>
<tr>
<td>Average Achieved Velocity(deg/sec):</td>
<td>98</td>
<td>0</td>
<td>103</td>
</tr>
</tbody>
</table>
Treatment

- Adaptation exercises to improve use of remaining vestibular function
- Substitution exercises to improve use of alternate sensory systems for improving visual stability and balance.
- Safety and falls prevention education
Treatment

• Adaptation refers to the potential for the remaining vestibular system to adjust its output according to the demands placed on it.

• Achieve long-term change in the neuronal response to reduce symptoms and normalizing gaze and postural stability.

• A critical signal to induce adaptation is retinal slip during head movements.

• VORx1 , VOR x2 (use if some functions remains)
Treatment

• Substitution – use of alternative strategies for missing vestibular function.
• Substitution for the VOR includes the cervical ocular reflex, use of smooth pursuit eye movements, and central preprogramming of eye movements.
• Substitution includes the use of visual cues, somatosensory cues, or both to maintain stability.
Treatment

• Substitution exercises
  – Saccades – two targets central preprogramming
  – Imaginary targets – COR
  – Visual substitution exercises – walk to a target
  – Somatosensory inputs - firm surface, proprioceptive training at ankles

• Precautions
  – Light in home – carry a flashlight
  – Watch uneven terrain
  – Use of assistive device if necessary
Outcomes

• Patient received 9 visits over 2 months (6-29 to 9-2-10).
• SOT composite score declined from 47 to 39.
• DVA unchanged from 0.19 left 0.26 right to 0.11 logMar left, 0.26 logMar right.
• Functional: patient reports decreased symptoms of oscillopsia, when shopping or at church she can use visual fixation to feel less “dizzy”, limited driving, in good weather, close to home and during daylight hours. Does not drive when she is “having a bad day”.
Sensory Organization Test
(Sway Referenced Gain: 1.0)

Equilibrium Score

- Conditions 1, 2, 3, 4, 5, 6
- Composite Score: 39

Sensory Analysis

Strategy Analysis

COG Alignment
Dynamic Visual Acuity Test

Perception Time: 20 ms
Optotype Display Interval: 40 - 75 ms

DVA Loss

LogMAR

- 0.11 (Left)
- 0.26 (Right)

DVA Loss Symmetry (%)

- 9%

<table>
<thead>
<tr>
<th>Direction</th>
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<th>Static</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snellen Fraction:</td>
<td>20/18</td>
<td>20/14</td>
<td>20/25</td>
</tr>
<tr>
<td>logMAR:</td>
<td>-0.05</td>
<td>-0.16</td>
<td>0.10</td>
</tr>
<tr>
<td>Average Achieved Velocity (deg/sec):</td>
<td>96</td>
<td>0</td>
<td>102</td>
</tr>
</tbody>
</table>
Conclusion

- Patients with bilateral vestibular loss improve in their perception of dizziness and imbalance.
- 33% to 55% improved with individualized vestibular physical therapy program.
- Most patients with BVH continue to be at risk for falls.
- Recovery is longer than UVH – up to 2 years.
- Patients should be referred to physical therapist with training in vestibular rehabilitation.
- Some may be able to return to driving.
- Refer for VFT to determine BVL.

A Case of Dizziness With Concomitant Central and Peripheral Dysfunction

Amy Pause, PT, DPT
Background

• Patient’s presenting with vertigo:
  – 75% Peripheral vestibular disorders
  – 25% Central origin
• Most Common Causes of Vertigo:
  – BPPV
  – Vestibular neuritis
  – Meniere’s syndrome
  – Vascular disorders
Background

- Vertigo of vascular origin is usually limited to:
  - Migraine
    - Migraine affects as many as 15-20% of the general population.
    - A quarter of patients with migraine experience spontaneous attacks of vertigo.

Karatas M, 2011
Background

- Transient ischemic attacks
- Ischemic or hemorrhagic stroke
  - 6 - 7% Cerebrovascular disease
  - 1.5 - 3.6% Cardio-circulatory disease
- Cerebrovascular and cardio-circulatory causes are:
  - Relatively uncommon
  - More serious causes of vertigo
  - Lead to various central or peripheral vestibular syndromes with vertigo
Purpose

• Present a patient with symptoms of dizziness of vascular origin.
• Discuss benefits of Vestibular Function Testing to guide clinical tests and measures.
• Describe use of tests and measures to formulate customized treatment interventions to maximize outcomes.
Case Description

• The patient is a 28 year old male who works in audio engineering at a local hotel resort.
• Past medical history includes:
  • Congenital heart disease - double outlet right ventricle
  • Pulmonary valve replacement
  • 1st and 2nd degree AV block with pacemaker placement
  • Atrial tachycardia with ablation procedure
  • Cardioembolic stroke/TIA x 5
  • Psychogenic non-epileptic seizures
  • Suspected migraines
History

• Complex congenital heart disease resulted in multiple surgeries and pacemaker placement.
  – Age 19, experienced a brainstem stroke resulting in:
    • Left-sided facial weakness, extremity weakness, significant dysarthria, difficulty managing his airway, diplopia.
  – Recovered well and discharged
    • Had remaining supranuclear paralysis of left eye with convergence and retraction nystagmus
  – Inpatient rehab for 6 days
  – Outpatient rehab 2x/week
History

– Between ages 20 and 22 years - emergency department for headaches, dizziness, and seizures.
– Age 25, he had a TIA event and prescribed Coumadin.
– Five months later he presented to ED with:
  • left eyelid droop
  • right arm weakness
  • Surgery to remove a 13 cm plastic fragment from his left atrium. This was thought to be the cause of clot formation, and thus his strokes, and eventually he was taken off Coumadin.
History

- Age 26, brief TIA with:
  - Diplopia & Left sided weakness
- Age 27, sudden onset of:
  - Headache, double vision, dizziness, heaviness in left arm
  - Evaluated in the ED
    - CTA of the head and neck was normal and he was discharged.
- Neurology consult with stroke specialist. Started on Coumadin with no further episodes; although, dizziness persisted.
Otolaryngology Consult

- Otolaryngology referral, three months later, for persistent dizziness.
  - Describes dizziness as a sensation of acute disequilibrium.
    - Episodes of dizziness occur in frequency of a ~ 10 times each day
    - Usually last for 5-10 seconds.
    - Dizziness is not related to changes of head position.
Otolaryngology Consult

- Falling towards the left direction.
- History of headaches that are sometimes associated with the dizziness.
- Problems with double vision secondary to his CVAs.
- Has been prescribed Antivert in the past without improvement of symptoms.
- Referred for CT scan and Vestibular Function Testing.
VNG

- NORMAL
  - Ocular motility
  - Positional and positional testing

- ABNORMAL
  - Fine, left-beating spontaneous nystagmus
  - 30% unilateral weakness in the RIGHT ear
  - >22% is Abnormal
VOR Phase, Gain & Symmetry (0.08Hz-0.32) WNL
(Isolated asymmetry at 0.01Hz is in keeping with the presence of spontaneous nystagmus)
cVEMP

- Bilaterally normal cVEMP examination

- Amplitude Asymmetry Ratio:
  - 36% to the LEFT
  - > 47% is Abnormal

- Intact Saccule and Inferior Vestibular Nerve
oVEMP

- Bilaterally normal oVEMP examination
- Amplitude Asymmetry Ratio:
  - 8% to the RIGHT
  - > 33% is abnormal
- Intact Utricle and Superior Vestibular Nerve
Vestibular Function Testing
Overall Impression

• Abnormal VNG examination
  – Bithermal caloric testing yielded a significant (30%) weakness on the right side.
  – Overall results suggest the presence of a peripheral vestibular system disorder affecting the right side.
Neurology Consult

- Referred to neurology for assessment of migraines and central etiology.
  - Started on Gabapentin/Neurontin to help reduce episodes of dizziness and headache.
  - Reports significantly reduced frequency of his episodes over the next few months.
Otolaryngology Follow-up

- Otolaryngology follow-up one year later due to reduced effectiveness of Gabapentin /Neurontin.
  - Increase of dizziness and imbalance
  - Experienced a fall at work due to sudden onset of dizziness
  - Currently on leave from work
  - Referred for vestibular rehabilitation therapy
Subjective Assessment

- Dizziness described as disequilibrium
- Dizziness while riding in a car
  - Magnified with head turns to look out window
- Dizziness in stores
- Dizziness with prolonged walking
- Falls regularly
  - Sudden and without provocation
- On medical leave due to current symptoms
Objective Assessment:

• Gait:
  – No assistive device
  – Holds onto fiancé’s arm and finger touch to wall
  – Minimized head movements
  – Path deviation towards the left
• MMT: Normal
• ROM: Normal
• Sensation: Intact
Objective Assessment

- Oculomotor Exam: normal
- VOR cancellation: intact
- Head impulse testing (Weber KP, 2008): negative
- BPPV assessment:
  - Dix-Hallpike testing: negative
  - Roll testing: negative
Neurocom SMART EquiTest & In-Vision System
Sensory Organization Testing (SOT)

- Composite score of 80
- No deficit in the use of vestibular, visual, or somatosensory cues for balance.
Head Shake SOT

- Head Shake SOT (Horizontal)
  - Equilibrium Score Ratio:
    - Fixed Surface .98
    - SwayRef Surface .90

Mishra A, 2009
Clinical Integration Seminar. 2005, Neurocom International, Clackamas, OR.
Neurocom In-Vision

- **Gaze Stabilization Test:**
  - Maintain visual acuity at velocities up to:
    - 156 deg/sec in leftward
    - 66 deg/sec in rightward
Neurocom In-Vision

- **Dynamic Visual Acuity:**
  - 0.01 LogMar loss of visual acuity with leftward head movements.
  - 0.27 LogMar loss of visual acuity with rightward head movements.
Objective Measure

• Functional gait assessment: 20/30
  • Slower gait speeds with path deviation, pivot turns, gait with narrow BOS, gait with eyes closed.

• Six-minute walk test:
  – 1600 feet with dizziness and imbalance during turns.

• Single limb stance: (Bohannon, 2006)
  – Left LE - 13.06 seconds
  – Right LE - 1.93 seconds

• Motion Sensitivity Quotient:
  – <1% (0-10 = mild) (Herdman, 2007)
Impression

• Unilateral Vestibular Hypofunction based on the following findings:
  – Impaired right gaze stability
  – Impaired right dynamic visual acuity (Herdman, et al, 1998)
  – Increased dizziness with head turns
  – Path deviation with head turns during gait
  – Vestibular Function Testing
Treatment Interventions

• Patient seen for 6 visits over an 8 week period.
  – **Gaze Stabilization Adaptation Exercises** (Herdman, 2007)
    • VOR X1 & VOR X2 viewing
    • Various balance strategies
    • Incorporated into gait with forwards and backwards walking
    • Various visual stimulating backgrounds
  – **Optokinetic Stimulation** (Pavlou M, 2011)
    – Visual stimulating videos
    – Passenger in a car
    – Walking in stores and mall
Treatment Interventions

• **Balance training on compliant surfaces**
  – Foam standing, various stance positions, incorporation of head turns, eyes open and closed
  – Turning 180 & 360 degrees

• **Neurocom Custom Training**
  – Support and surround movement to facilitate various balance strategies

• **Single limb stance**
  – Stepping over objects, negotiating stairs at slow speed
  – Increasing single limb stance times with home program

Giray M, et al., 2009
Treatment Interventions

- Gait training on various surface types
  - Progressive walking program for up to 30 minutes daily
  - Incorporation of head turns
  - Tandem gait activities
  - Dual task:
    - Carrying objects of various sizes and weight to simulate work duties
    - Gait on various surfaces with reading & cognitive skills
- Treadmill training
  - Head turns & speed changes
Outcomes

• Intermittent dizziness
• Occasions of sudden imbalance lasting 10 -15 sec ~4 per wk
• Independent ambulation without UE support on various surfaces.
  – Able to incorporate functional head turns with no significant veering
• Return to work with no visual stimulating dizziness while in the work environment
• Improvement in Functional Gait Assessment score 30/30
• Single limb stance times improved to 30 seconds
In-Vision Results:

- Improvement in gaze stabilization at velocities up to:
  - 186 deg/sec in leftward movement directions
  - 154 deg/sec in rightward movement directions
In-vision Results

- Improved Dynamic Vision
  - 0.18 LogMar loss of visual acuity with leftward head movements.
  - 0.02 LogMar loss of visual acuity with rightward head movements.
Outcome

- Returned to work full time
- Independence with a home exercise program to help maintain functional gains (Byung In Han, 2011)
- Discharged from therapy.
Discussion

- Vestibular Function Testing is important in differential diagnosis of peripheral versus central vestibular involvement.

- Customized vestibular rehabilitation programs can be provided based on diagnosis, presentation, and symptoms.

- When available, develop a working relationship with audiologists in your area to optimize patient care.
Questions
References


References

• Bohannon R. Single Limb Stance Times – A Descriptive Meta-Analysis of Data from Individuals at Least 60 Years of Age. *Top Geriatr Rehabil.* 2006;22(1):70-77.


• Clinical Integration Seminar. 2005, Neurocom International, Clackamas, OR.


References


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