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.

The Founding Father of Vestibular Science

- Robert Barany
  - April 22, 1876 – April 8, 1936
  - Austro-Hungarian Otologist
  - Awarded The Nobel Prize in Physiology or Medicine in 1914
    * "for his work on the physiology and pathology of the vestibular apparatus"
Vestibulo-Ocular Reflex (VOR)

1) Receptors (3 cristae)
2) 1st order neuron at Scarpa's ganglion (CN VIII)
3) 2nd order neuron at the vestibular nuclei
4) 3rd order neuron at motor nucleus of CN III and CN VI
5) Effector organs – Extraocular Muscles

Vestibulo-Ocular Reflex (VOR)

Stimulation of the Ipsilateral Horizontal Semicircular Canal (hSCC)
- Activates: Ipsilateral medial rectus, contralateral lateral rectus
- Inhibits: Ipsilateral lateral rectus, contralateral medial rectus
- Generates a horizontal eye movement with slow phase away from stimulated ear

Comprehensive Vestibular Function Test Battery

- Electro-Videonystagmography (ENG/VNG)
- Rotational Testing
- Vestibular Evoked Myogenic Potentials
  - Cervical (cVEMP)
  - Ocular (oVEMP)

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 system

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

Ewald’s Law

(Stimulation of the SCC causes nystagmus in the plane of that canal)

- Horizontal Canal:
  - Excitation: Ipsilateral medial rectus, contralateral lateral rectus
  - Observation: Horizontal nystagmus
- Posterior Canal:
  - Excitation: Ipsilateral superior oblique, contralateral inferior rectus
  - Observation (right) rightward torsional upbeating nystagmus
- Anterior Canal:
  - Excitation: Ipsilateral superior rectus, contralateral inferior oblique
  - Observation (right) rightward torsional downbeating 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.

Dix-Hallpike Maneuver

Treatment for pSCC BPPV
- Modified Epley
- Semont Maneuver

New Treatment for aSCC BPPV

Horizontal Canal (hSCC) BPPV Diagnosis & Treatment

Roll Test

Patient supine/semi-recumbent at 20-30 degree angle
**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**

![Diagram showing higher and lower SPV with nystagmus traces](image)

**Appiani Liberatory Maneuver**

![Diagram showing Appiani Liberatory Maneuver steps](image)

**BBQ Roll**

From Casani et al., 2002

**Forced Prolonged Positioning**

From Appiani et al., 2001
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).

With ageotropic nystagmus, the debris within the canal can cause either of two scenarios:

1. **Debris free floating in the anterior end of the horizontal canal close to the cupula**
   - In this case, the nystagmus can change to geotropic with simple rolling of the head in a repeated manner from supine (Gufani et al., 1998; Gufani, 2002)

2. **Debris adherent to the cupula on the utricula side**
   - Results in persistent ageotropic nystagmus (Casani et al., 2002)

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**Ageotropic-Anterior Arm hSCC (Canal Side)**

- Lower SPV
- Higher SPV

**Ageotropic-Anterior Arm hSCC (Utricular Side)**

- Lower SPV
- Higher SPV

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**Flow Chart for the Treatment of hSCC BPPV**

- Ageotropic Nystagmus
- Horizontal Head Fall
- Gufoni Maneuver
- From Appiani et al., 2005

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**Gufoni Liberatory Maneuver**

From Appiani et al., 2005
**Brandt-Daroff Habituation Exercises**

**Electro-Videonystagmography: Bithermal Caloric Test**

**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 suggests a Unilateral peripheral deficit
    - Directional Preponderance
    - >28% is abnormal

- **Inter-ear Difference**
  - < 10%

- **Minimum Response**
  - >11 deg/sec from each ear

- **Cost Effective**
  - **Inter-ear Difference**
  - Spontaneous or positional nystagmus
  - Abnormal ocular motility

From: Jacobson et al., 1995; Jacobson & Illes, 1985; Murnane et al., 2009

**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.
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”
(Fron: 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
Nomenclature re: Cervical ( SCM) VEMP (aka cVEMP)

- 1st positive and negative waves are referred to as P13/N23 (or P1/N2).
- Positive waves represent inhibition of EMG
- Negative wave represents excitation of EMG

Acoustical Stimulus Characteristics

<table>
<thead>
<tr>
<th>Stimulus type/s</th>
<th>100 usec click, or, 500 Hz tone burst, 6-10 ms duration, 2-1-2 configuration (Blackman gating)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transducer</td>
<td>ER3a insert earphone or bone conductor</td>
</tr>
<tr>
<td>Rate</td>
<td>5/second</td>
</tr>
<tr>
<td>Intensity</td>
<td>+5 dB re: VEMP response threshold (usually 90-100 dB nHL)</td>
</tr>
</tbody>
</table>

Recording Characteristics

<table>
<thead>
<tr>
<th>Recording Condition</th>
<th>Seated in comfortable reclining chair, or, laying on a table</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-inverting electrode</td>
<td>Ipsi or ipsi &amp; contra middle 3rd of SCM m.</td>
</tr>
<tr>
<td>Inverting electrode</td>
<td>Chin or dorsum of hand</td>
</tr>
<tr>
<td>Ground electrode</td>
<td>Fpz</td>
</tr>
<tr>
<td>Filtering</td>
<td>~5-10Hz – 1500-2000 Hz</td>
</tr>
</tbody>
</table>

How do we activate the SCM? Unilateral Activation/Recording

Subject Variables

Age (elderly: Su et al. 2004)

- VEMP is present in:
  - 98% - < 20 – 40 years
  - 90% - 41-60 years
  - 60% - > 60 years of age
- Decreased amplitudes and increased thresholds begin at 6th decade
  - Mean threshold 20-30 year old = 85 dB nHL
  - Mean threshold 70-80 year old = 96 dB nHL
- No age effect on latency
Subject Variables
Conductive Hearing Impairment.

- **Conductive deficit** (e.g. stapes fixation) makes it difficult for the SPL to be sufficiently intense in order to reach the saccule.
- **Solution:** Bone conduction cVEMP
- **Solution:** Mechanical cVEMP
- Gentle skulls taps with an electric tendon hammer.

Solution: Bone conduction cVEMP
Solution: Mechanical cVEMP
Gentle skulls taps with an electric tendon hammer.

Subject Variable
Magnitude of tissue interposed between recording electrode and SCM

- **Subcutaneous tissue thickness** affects the ability to record the cVEMP response.

Cervical Vestibular Evoked Myogenic Potential (cVEMP)

- The cVEMP is a stimulus-related attenuation of tonic EMG activity.
- If there is no tonic EMG activity the cVEMP will be absent.
- The presence of acceptably high tonic EMG activity and absent cVEMP suggests an impairment affecting (most often) the ipsilateral saccule and/or inferior vestibular nerve.

Normal Limits for VEMP Values
250 Hz Tone Burst (Mayo Clinic data)

<table>
<thead>
<tr>
<th>Pooled Left and Right</th>
<th>Mean</th>
<th>Sd</th>
<th>+2 SD limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>P13 latency, msec</td>
<td>16.90</td>
<td>1.43</td>
<td>20.47</td>
</tr>
<tr>
<td>N23 latency, msec</td>
<td>25.24</td>
<td>1.63</td>
<td>29.31</td>
</tr>
<tr>
<td>P1-N1 amplitude, uV</td>
<td>180.71</td>
<td>120.42</td>
<td></td>
</tr>
</tbody>
</table>

From: Zapala & Brey, 2004
**Clinical Utility of the cVEMP**

- Effective in diagnosing disorders of the saccule and/or inferior vestibular nerve.
- Sensitive in assisting with the diagnosis of early-onset Meniere’s Disease.
- Useful in the diagnosis and follow-up care for patients with Superior Semicircular Canal Dehiscence (SCD).

**Acoustical Ocular VEMP (oVEMP)**

- Identifies thresholds present at reduced stimulus intensities (i.e., <70dBnHL).
- Confirms threshold normalization after superior canal plugging.

Welgampola et al. 2008

**cVEMP versus oVEMP**

- The OVEMP waveform is negative/positive (i.e., going from EMG “off” to EMG “on”) instead of positive/negative (i.e., going from EMG “on” to EMG “off”).

**Ocular VEMP (oVEMP)**

- Represents the synchronous evoked extraocular muscle activity associated with the vestibulo-ocular reflex (VOR).
  - Does not represent movements of the eyes (i.e., they are short latency responses e.g. 10 msec)
- EOMs have properties that allow them to be activated with precision at short latencies for fine motor control of eye movements.

**Evidence that cVEMP and oVEMP have Different Peripheral Origins**

- Electrical stimulation of utricular afferents cause activation of ipsilateral superior oblique m. and contralateral inferior oblique m.

**Evidence that oVEMP Originates from the Utricle**

- Placement of electrodes beneath an averted contralateral eye places the inferior oblique m. beneath an active electrode.
  (Curthoys, 2010)
oVEMP Pathway (after Curthoys, 2010)
- oVEMP pathway
  - Utricle
  - Sup. Vestib. Nerve
  - Medial Longitudinal Fasciculus
  - Motor Nucleus of Contra CN III
  - CN III
  - Contra Inferior Oblique m.

Stimulus Characteristics - oVEMP
Same as cVEMP
- Stimulus type/s: 500 Hz tone-burst, 2-1-2 cycle, Blackman gating
- Transducer: ER3a insert earphone or bone conductor
- Rate: ~5/second
- Intensity: +5 dB re: VEMP response threshold (usually 90-100 dB nHL)

Recording Characteristics - oVEMP
- Electrode locations: Infraorbital (non-inverting), 2 cm inf. (inverting) or chin (inverting), Fpz (ground)
- Gain: 100,000x (versus 5000x for cVEMP)
- Sampling rate: ~3000Hz
- Recording Epoch: 100+ msec including a 10-20 msec prestim period
- Gaze: Supra-medial (up, midline)
- Artifact Reject: On, 40 uV
- Filtering: 1-10 to 1000-2000 Hz

2-Channel oVEMP Recording Version 2
- Ground
- Active (+)
- Reference (-)

oVEMP Pathway is Bilateral (Response Predominates Contralaterally)
From Iwasaki et al. 2008
- Contralateral response: Present consistently
- Ipsilateral response: Present inconsistently

oVEMP Normal Response
- In response to 500 Hz tone burst
  - N1: ~10 msec
  - P1: ~15 msec
- Contralateral response occurs slightly earlier and is larger than ipsilateral response (contralateral pathway is faster)
Subject Variables and the oVEMP Age Effects:

- Amplitude of the oVEMP response decreases with increased age.
- N1/N10 Latency increases with increased age.
- oVEMP Threshold increases with increased age.
- 25% of otologically/neurologically intact >60 population do not generate oVEMP response (500Hz) – Piker et al. 2011.

Breaking News! Effects of Age on the Frequency Tuning of the cVEMP and oVEMP

- Piker et al. 2012 (currently submitted for peer-review publication)
- Investigated the best frequencies to record the VEMP in young, middle-age and older adults.
- For older subjects (i.e. >60 years of age) the optimal toneburst stimulus is 750Hz & 100Hz.
- Decreased mass or increased stiffness of the otoliths alters resonant frequency thus requiring higher frequency stimulus to generate the VEMP response.

Evidence that cVEMP and oVEMP have Different Peripheral Origins

- The cVEMP, oVEMP and caloric test results may vary independently for patients with various peripheral vestibular system impairments (e.g. MD, neuritis)
  - In inferior vestibular neuritis the ipsilesional cVEMP was absent, the oVEMP was present (Manzari et al. 2010)
  - In superior vestibular neuritis the oVEMP was absent, the ipsilateral cVEMP was normal and the ipsilateral cVEMP was normal (Manzari et al. 2010, Govender et al. 2011)
Evidence that cVEMP and oVEMP have Different Peripheral Origins

- In Meniere’s Disease abnormalities in the cVEMP occurred more often than in the oVEMP (temporal bone studies show more extensive damage in the saccule than in the utricle in MD) (Taylor et al. 2010, Huang et al. 2011).

- Patient with right-sided vestibular schwannoma had normal caloric and cVEMP exam but an abnormal oVEMP exam (Murofushi et al. 2010).

Diagnostic Significance of cVEMP, oVEMP and caloric test Abnormalities

- There is an overlap within the pathways assessed through the caloric and oVEMP tests.

- The primary overlap is the superior vestibular nerve.

- Therefore, if impairment affects the superior vestibular nerve only, abnormalities should occur for both caloric and oVEMP tests while the cVEMP should be normal.

- If impairment affects the inferior vestibular nerve only, abnormalities should occur for the cVEMP test only.

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

Possible Patterns of Impairment and Their Significance

<table>
<thead>
<tr>
<th>Type</th>
<th>Caloric</th>
<th>cVEMP</th>
<th>oVEMP</th>
<th>Impairment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>NI</td>
<td>NI</td>
<td>NI</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>Abn</td>
<td>Abn</td>
<td>Abn</td>
<td>Large end organ, or inf. and sup. vestibular nerve</td>
</tr>
<tr>
<td>3</td>
<td>NI</td>
<td>Abn</td>
<td>NI</td>
<td>Saccule or inf. vestibular nerve</td>
</tr>
<tr>
<td>4</td>
<td>Abn</td>
<td>NI</td>
<td>Abn</td>
<td>Sup. vestibular n.</td>
</tr>
<tr>
<td>5</td>
<td>Abn</td>
<td>NI</td>
<td>NI</td>
<td>hSCC</td>
</tr>
<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>
<td>NI</td>
<td>hSCC and/or inf. vestibular nerve?</td>
</tr>
<tr>
<td>9</td>
<td>Abn*</td>
<td>Abn*</td>
<td>NI</td>
<td>SSCD Syndrome</td>
</tr>
</tbody>
</table>

* = Abnormally reduced VEMP thresholds
Type 1: Normal Patient
Monothermal Warm Caloric Test
Monothermal Caloric Asymmetry = 10%

Type 1: Normal Patient
cVEMP
P13 amplitude asymmetry = 11%

Type 1: Normal Patient
oVEMP
N10 amplitude asymmetry = 12%

Type 2
History

- 43 yo male
- Diagnosis of left Meniere’s d.
- 1995 underwent a definitive procedure (vestibular nerve section)
- For 6 mos. slightly off balance
- Denies vertigo, nausea, vomiting
- Brain MRI is normal

Type 2
Audiogram
Type 2
Caloric Test
Caloric asymmetry = 100%

Type 2
cVEMP
P13 amplitude asymmetry = 100%

Type 2
cVEMP
N10 amplitude asymmetry = 100%

Type 2
Profound End Organ, or, Nerve
Impairment

- Abnormal
Caloric Test
- Abnormal
cVEMP
- Abnormal
oVEMP

Type 3
History
- 46 yo female with diagnosis of left Meniere’s disease.
- Over a 10 year period attacks of vertigo occurred q 3 months.
- More recently attacks have occurred 1-2x/week.
- Conservative medical management (LSD, Valium, transtympanic dexamethasone).
- MRI is normal.

Type 3
Audiogram
Type 3 Caloric Test
Caloric Asymmetry = 16%

Type 3 cVEMP
P13 amplitude asymmetry = 50%

Type 3 oVEMP
N10 Amplitude asymmetry = 6%

Type 3 Impairment of Either or Both the Saccule and/or Inferior Vestibular Nerve
- Normal Caloric Test
- Abnormal cVEMP
- Normal oVEMP

Type 4 History
- 50 yo female
- 6 week history of true vertigo with nausea and vomiting with sudden onset
- Augmented with changes in head position
- No auditory symptoms
- Medical history is otherwise unremarkable

Type 4 Audiogram
Type 4
Caloric Test
Caloric asymmetry = 43%

Type 4 cVEMP
P13 amplitude asymmetry = 5%

Type 4 oVEMP
N10 Amplitude asymmetry = 35%

Impairment affecting at least the Superior Vestibular Nerve
• Abnormal Caloric Test
• Normal cVEMP
• Abnormal oVEMP

Type 5
Caloric Test
Caloric Asymmetry = 68%
Right UW

Type 5 cVEMP
Type 5 oVEMP

Vanderbilt Bill Wilkerson Center

Type 5 Horizontal SCC Impairment

- Abnormal Caloric Test
- Normal cVEMP
- Normal oVEMP

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Type 6 Audiograms

1/7/11 2/18/11 7/15/11

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Type 6 Caloric Test (1/26/11)
Caloric Asymmetry = 5%

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Type 6 cVEMP (1/26/11)
Amplitude Asymmetry = 9%

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Type 6 Ovemp (1/26/11)
Amplitude Asymmetry = 47% Right

Vanderbilt Bill Wilkerson Center
**Type 6: Impaired Utricle**

- Normal Caloric Test
- Normal cVEMP
- Abnormal oVEMP

**Current Quantitative Method to Assess Function of the Utricle**

- Unilateral centrifugation and measurement of the subjective visual vertical
  - Expensive
  - Time-consuming
  - Uncomfortable
  - Psychophysical technique

**oVEMP vs. SVV**

- Valko et al. (2010) found the oVEMP to be superior to most measures of subjective visual vertical.
- oVEMPs are quick and less invasive with respect to SVV.

**Type 7: Impaired Utricle + Saccule and/or Inferior Vestibular Nerve**

- Normal Caloric Test
- Abnormal cVEMP
- Abnormal oVEMP

**Type 8: Impaired hSCC + Saccule and/or Inferior Vestibular Nerve**

- Abnormal Caloric Test
- Abnormal cVEMP
- Normal oVEMP

**Type 9: Superior Semicircular Canal Dehiscence**
**Superior Semicircular Canal Dehiscence Audiogram**

**Caloric Test**
- Caloric Asymmetry = 5%

**cVEMP**
- Amplitude
  - Right = 526.61 uV
  - Left = 398.68 uV
  - Asymmetry = 14%
- cVEMP Threshold
  - Right = 70 dBnHL
  - Left = 70 dBnHL

**oVEMP**
- Amplitude
  - Right = 46.69 uV
  - Left = 30.50 uV
  - Asymmetry = 21%
- oVEMP Threshold
  - Right = 65 dBnHL
  - Left = 65 dBnHL

**oVEMP absolute amplitude found to be superior to cVEMP thresholds in diagnosis of SCD**

**Sensitivity & Specificity of VEMP predictive of SCD**
- oVEMP absolute amplitude:
  - ≥ 17.1 uV
  - Predicts diagnosis of SCD and warrants CT scan of the temporal bones.

**Ocular vs. Cervical VEMPs in the Diagnosis of SCD**
- Ocular VEMPs
  - Absolute amplitudes yielded 90%–100% sensitivity & specificity for SCD in all age groups (30–66 years)

**Table: Ocular vs. Cervical VEMPs Predictive of SCD**

<table>
<thead>
<tr>
<th>VEMP Type</th>
<th>Threshold</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Click-ocular VEMP</td>
<td>57.5</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Click-ocular VEMP</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Click-ocular VEMP</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Click-ocular VEMP</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Click-ocular VEMP</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
</tbody>
</table>

(Zuniga et al., 2012)
VEMP Sensitivity & Specificity for SCD by Decade of Life

Zuniga et al., 2012

Summary

- Combined results of the Caloric Test, cVEMP and oVEMP is a powerful diagnostic tool in identifying site of lesion.
- When VEMP responses are absent to a 500Hz stimulus in older patients (i.e. ≥60), adjust stimulus frequency to 750Hz and/or 1000Hz.

Innsensitivity 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 RTSBFCCS offers poor sensitivity (<55%) in the detection of vestibular system impairments.
- The RTSBFCCS 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