For some time it has been recognized that...

• In addition to movement, vestibular afferents may be activated by:
  – Sounds of high intensity
  – vibration and
  – electrical stimulation applied over the mastoid process

Dr. Pietro Tullio (1881-1941)
  – Observed sound-evoked head and eye movements after fenestration of areas of the bony labyrinth
Sonomotor Responses

- Von Bekesy (1935)
  - Observed that high intensity sounds (e.g. 128-134 dB SPL) evoked head displacement toward the stimulated ear.

Background

- Mid 1960's Cody, Bickford et al. showed that a large portion of what were thought to be “neurogenic” auditory evoked responses (MLR 0-50 ms) were attenuated by anesthesia.

Myogenic component
Neurogenic component following paralysis

Background

- The high amplitude responses were, sound-evoked muscle responses (i.e. “sonomotor” responses).
- Near-field recorded
- May or may not represent a high intensity sound-evoked transient reduction in muscle tone.

Sonomotor Responses

Sound-induced Reduction in EMG

Background

- Sonomotor responses include:
  - the acoustic jaw reflex (Meier-Ewert et al. 1974)
  - the postauricular m. (PAM) potential (Kiang, 1963),
  - the ionion potential (Cody et al., 1964) which has become one of a group of contemporary...
  - vestibular evoked myogenic potentials (VEMP)
Reincarnation of the Sonomotor Response - VEMP
Halmagyi and Curthoys, 1990's

- VEMP can be recorded from muscle groups that react to sound: SCM, trapezius, tricep, gastrocnemius and quadraceps muscles etc. etc. (e.g. Ferber-Viart et al. 1998; Rudisill and Hain, 2008)

VEMP is a Sonomotor “Reflex”
What is a reflex pathway?

- Consists of:
  - A receptor (end organ)
  - An afferent pathway
  - Central connections
  - An efferent pathway, and,
  - End muscles

Central Connections & Efferent Pathway
“Vestibulocollic Reflex”
(From: Rosengren et al. 2009)

- Saccule (a)
- Inferior vestibular nerve (a)
- Vestibular nucleus (a)
- Ipsilateral medial vestibulospinal tract (MVST) (e)
- Spinal accessory nucleus of CN XI (e)
- CN XI (e)
- SCM (a)

a = afferent, e = efferent

Numbers of VEMP Articles Published From 1992-2009
Halmagyi, 2010

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
Nomenclature

- 2nd negative-positive complex N34-P44
  - Has a lower stimulus threshold than cVEMP
  - Absent in 30-40% of normal subjects
- Most discussions focus on P13/N23.

Outline

- Historical background
- Anatomic and Physiologic origins
- Conventional stimulus (e.g. intensity, frequency, rate), subject (e.g. age, gender, muscle tone) and recording variables (e.g. filtering, amplification, artifact rejection)
- Normal response (i.e. waveform and measurement parameters)
- Responses from abnormal populations
- Summary

VEMP is a Sonomotor “Reflex”

What is a reflex pathway?

- Consists of:
  - A receptor (end organ)
  - An afferent pathway
  - Central connections
  - An efferent pathway, and,
  - End muscles

Otolith Organs

- When the head tilts out of the upright position, the component of the gravitational vector tangential to the macula creates a shearing force on stereocilia of utricular hairs cells.

Dynamics of the Otoliths
Receptor for cVEMP is the Saccule?
Hamagyi & Curthoys (2000)

- Saccule is vestibular end organ most sensitive to sound
- Lies under the stapes footplate
- Neurons from saccular macula respond to tilts and click stimuli

Receptor is the Saccule?

- Present in patients with:
  - Destroyed SCC’s
  - Deformed cochlea but normal saccules
- Absent in Meniere’s Syndrome patients with surgical destruction of cochlea and saccule

Receptor is the Saccule?

- Stimulation of the utricle and saccule results in generation of IPSPs in ipsilateral SCM motor neurons

Nerve of Afferent Pathway is Inferior Vestibular Nerve?

- Electrical output from the saccule is routed through the inferior vestibular nerve.

Physiological Evidence

From: Curthoys et al. 2012

Response from irregular otolith neuron during stimulation by BC vibration (left) and AC sound (right). Note the synchronization of the increase in discharge rate with sound onset for this irregularly discharging neuron.
Compared to Auditory Nerve Fibers, Vestibular Afferents Show High Thresholds and Broad “Tuning”

Best Frequencies and Thresholds of Response for Cochlear Neurons and Acoustically Responsive Irregular Vestibular Afferents (McCue & Guinan, 1995)

Nerve of Afferent Pathway is Inferior Vestibular Nerve?

- Response is absent for patients with vestibular nerve section and who have had an inferior vestibular neuritis

Receptor is the Saccule?

Halmagyi & Colebatch (1995)

- VEMP is present in deaf patients who have intact vestibular system function (Colebatch et al. 1993)

Outline

- Historical background
- Anatomic origins
- Conventional stimulus, subject and recording variables
- Normal response (i.e. waveform and measurement parameters)
- Responses from abnormal populations
- Summary

Introduction to Stimulus Variables

What stimuli can be used to elicit the VEMP?

- Air conduction VEMP
- Mechanical (tap) VEMP (CHL)
- Bone conduction VEMP (CHL)
- Galvanic VEMP
- Sound (unilateral)
- Vibration (bilateral)
- Electricity (galvanic, bilateral)
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>

Stimulus Variables

- Stimulus Frequency

- Stimulus Variables
  - Intensity = VEMP grows quickly once threshold is exceeded
  - Rate = Optimal ~5 Hz

Key Concept!

- We are using sound only as a pressure stimulus i.e. sound pressure is being used as a hydro-mechanical force to move the endolymphatic fluid and, as a consequence, to transduce otoliths to create transduction.
- A low frequency tends to provide a greater push

Q: How can we evoke a VEMP in a patient with a conductive hearing loss?
A: Bone Conduction VEMP

Bone Conduction VEMP

Two-channel recording with head elevated
Bone Conduction VEMP
Radioear B1 bone vibrator

- Optimum stimulator placement is 3cm post. and 2 cm sup. to EAM

Mechanisms BC-VEMP Generation

- Difference in best frequencies may be due to utricular contributions to the BC VEMP (i.e. w-BC stimulus we are setting entire skull into vibration)

Alternative Stimulus
Mechanical VEMP (Skull Taps)

- Otitis media sample:
  - 59% generate a VEMP to tone bursts,
  - 91% generate a VEMP to light skull taps (Yang and Young, 2003)
- Latencies similar to those obtained for AC signals (but unable to guarantee only the saccule is responding)

Methods
Mechanical VEMP

- Electrode placement identical as that for acoustical stimulation
- Patient supine with head elevated from table
- Light pressure applied to forehead with a gauze pad as patient elevates head to increase muscle tone (isometric activation)

Methods
Mechanical VEMP - Triggering

- Light taps are presented to Fpz, from a reflex hammer that contains an inertial trigger
- EP machine is triggered to signal average every time it strikes the forehead
- Only ~50 stimuli usually are required at a rate of ~3 Hz.
Mechanical VEMP

- Result is a P13/N23 in both SCMs (both saccules are stimulated by the stimulus)
- Tap elicits a 2nd negativity (N2) of unknown significance (utricular?)

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 – 600 Hz</td>
</tr>
</tbody>
</table>
Recording Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amplifier gain</td>
<td>~2500 – 5000</td>
</tr>
<tr>
<td>Sampling rate</td>
<td>~3000 Hz</td>
</tr>
<tr>
<td>Epoch length</td>
<td>~100 msec incl. 10-20 msec prestimulus</td>
</tr>
<tr>
<td>Sweeps per average</td>
<td>~80-120 (30-60 for mechanical stim)</td>
</tr>
<tr>
<td>Waveform replication</td>
<td>X1 minimum</td>
</tr>
<tr>
<td>Artifact rejection</td>
<td>Off</td>
</tr>
</tbody>
</table>

Key Concept!

- 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

“Tonic EMG Level and cVEMP Amplitude”

Methods to Control for EMG Level

- Visual or acoustic feedback
  - Patient can view EMG target amplitude on a CRT during data collection (or hear EMG converted to sound). or,
  - Combine optimized activation method with method for normalizing VEMP

Methods to Control for EMG Level: Method 1

- Visual Feedback of EMG Level

- Representation of EMG activity is displayed on a screen as a horizontally moving line
- Patient is asked to maintain EMG at target level

EMG Standard Deviation (i.e. target width)
How do we activate the SCM?
• Supine and ask patient to lift head at midline against gravity (bilateral activation)
• Turn head away from the ear stimulated (unilateral activation) in a sitting position
• Apply loads to muscle through loop and pulley that changes the traction on the neck muscle
• Lift head and push against a padded bar, or blood pressure cuff

How do we activate the SCM?
Bilateral Activation/Recording
• Recumbent patient is asked to raise their head from the table- (bilateral testing)

How do we activate the SCM?
Unilateral Activation/Recording
• Hybrid: Recumbent head turned to opposite ear and elevated

Comparison of the Head Elevation Versus Rotation Methods in Eliciting Vestibular Evoked Myogenic Potentials
• (Hybrid) rotation + elevation the response rate was 100%
• Rotation only had a response rate of 70% (VEMP was of smaller amplitude)
  – An alternative for those who cannot sustain SCM muscle contraction by head elevation.
  Isaacson et al. 2006, Isaradisaikul et al. 2008

Recording the Acoustic VEMP
Artifact Rejection and Magnitude of Tonic EMG Activity

Common Critical Errors in Recording the cVEMP

- Artifact reject must be disabled
- Spontaneous EMG must be monitored carefully to ensure that amplifier saturation does not occur

Effect of Amplifier Saturation of VEMP

Saturated

Not saturated

P13 = 15.95 μV

P13 = 76.68 μV

Outline

- Historical background
- Anatomic origins
- Conventional stimulus (e.g. intensity, frequency, rate), subject (e.g. age, gender, muscle tone) and recording variables (e.g. filtering, amplification, artifact rejection)
- Normal response (i.e. waveform and measurement parameters)
- Responses from abnormal populations
- Summary

The Normal Response

Quantification of VEMP

- Prominent components:
  - P13/P1 (ms) and
  - N23/N1 (ms)
Measures

- P13 & N23 latency (msec): P13 (> 20.47), N23 (> 29.31)
- Interaural latency difference P13: 3.39 msec
- P13-N23 amplitude (uV): range 15 – 350 uV for tone bursts and 15 – 200 uV for clicks
- Interaural (i.e. left/right) amplitude asymmetry measure (P13-N23)
- P13 threshold: ~90 dB (75-100 dB nHL)

Formula for Computing P13/N23 Amplitude Asymmetry Ratio

\[
\frac{\text{P13 latency right side} - \text{P13 latency left side}}{\text{P13 latency left side}} \times 100
\]

(\text{P13-N23 amplitude right side})

(\text{P13-N23 amplitude left side})

- Asymmetry ratio (amplitude): ≥ 40%
- reported normal upper limit ranges 30% – 47%

Normal Limits for Absolute VEMP Values

500 Hz Tone Burst (Vanderbilt Clinic data)

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Sd</th>
<th>+2 SD limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pooled Left and Right</td>
<td></td>
<td></td>
<td></td>
</tr>
<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: McCaslin et al., 2013

Normal Limits for Left/Right VEMP Asymmetries

500 Hz Tone Burst (Vanderbilt Clinic data)

<table>
<thead>
<tr>
<th>Left/Right Asymmetries</th>
<th>Mean</th>
<th>Sd</th>
<th>+2 SD limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>P13 latency, msec</td>
<td>.09</td>
<td>1.35</td>
<td>3.39</td>
</tr>
<tr>
<td>N23 latency, msec</td>
<td>-.16</td>
<td>1.42</td>
<td>3.54</td>
</tr>
<tr>
<td>P13-N23 amplitude</td>
<td>-.02</td>
<td>1.19</td>
<td>.47</td>
</tr>
</tbody>
</table>

From: McCaslin et al., 2013

More Detail
Re: Significant Variables Affecting the VEMP

- It is possible to record the VEMP in newborn infants.
- Neonates show (compared to adults)
  - increased P13 latencies
  - smaller P13-N23 amplitudes
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 appropriate SPL to reach the saccule
  - Solution: Bone conduction VEMP, or,
  - Solution: Skull tap VEMP

Subject Variable
Magnitude of tissue interposed between recording electrode and SCM

“Measuring Neck Structures in Relation to Vestibular Evoked Myogenic Potentials”

(Huge) Subject Variable
Magnitude of Tonic EMG Activity
- Patient’s ability to generate large and symmetrical tonic EMG for both left and right SCM’s (i.e. rectified RMS EMG of 30-50 uV, Akin et al. 2004)
  - Magnitude of stimulus-related attenuation is index of impairment

“The Influence of Voluntary Tonic EMG Level on the Vestibular-Evoked Myogenic Potential”
McCaslin et al., 2013
Methods to Control for EMG Level

- Visual or acoustic feedback
  - Patient can view EMG target amplitude on a CRT during data collection (or hear EMG converted to sound). or,
- Combine optimized activation method with method for normalizing VEMP

Control for EMG Level: Amplitude Normalization

Unrectified Waveform

Full-wave Rectification

\[ \text{Miyogenic potentials generated by a click-evoked vestibulocerebellar reflex} \]

The process of clipping a signal or waveform such that either the positive or negative portion of it is completely eliminated.

Methods to Control for EMG Level: Method 1
Visual Feedback of EMG Level

- Representation of EMG activity is displayed on a screen as a horizontally moving line
- Patient is asked to maintain EMG at target level

The Gestalt of the Idea

The common problem

- You record a left ear VEMP and it is 3x larger than the right ear VEMP.
- How do you know whether the amplitude difference is due to pathology or because the EMG from which the VEMP was extracted was 3x larger on the left side compared to the right side?

Gestalt of the Idea

The Common Problem

- So how about if we obtain a value that represents the average size of the EMG on the left side during signal averaging of the VEMP. Same for the right side.
- Then we divide into each data point in the left VEMP tracing the mean EMG value and do the same on the right side. In this way we can normalize the traces.

Recent Vanderbilt Study

EMG NORMALIZATION DOES NOT REDUCE SIGNIFICANTLY VARIABILITY IN VEMP NORMATIVE DATA FOR YOUNG NORMAL CONTROLS

McCaslin, Jacobson, Hatton, and Thornton.
Purpose

• To determine whether the use of visual feedback of tonic electromyographic (EMG) activity, and/or the use of amplitude normalization techniques would reduce significantly the variability in amplitude asymmetry data in otologically and neurologically intact young adults.

Four Conditions

• Condition 1: Patient supine, head turned away from the stimulated ear and elevated, without EMG monitoring or amplitude normalization

• Condition 2: Patient supine, head turned away from the stimulated ear and elevated, with a visual target set at 50 μV.

Four Conditions

• Condition 3: Patient supine, head turned away from the stimulated ear and elevated, with amplitude normalization.

• Condition 4: Patient supine, head turned away from the stimulated ear and elevated, with the use of a visual target set at 50 μV and with amplitude normalization.

Patient’s EMG Display

• The EMG display is a direct running on-line display of the RMS of collected in 100ms prestimulus blocks.

• Upper as well as lower rejection limits of EMG can be set to determine the range of EMG to be used in the VEMP averaging.

Patient’s View

Controller View
Amplitude Normalization

- Raw tonic EMG activity was collected over the 100 ms interval preceding each stimulus onset.
- For each of these 100 ms pre-stimulation time blocks, an RMS value representing the EMG level of that 100 ms pre-stimulus interval was calculated.
- At the end of the recording, an average of the individual RMS values from all of the obtained pre-stimulus intervals was calculated.

Obtaining Normalization Value

- Each obtained VEMP waveform has its own specific “normalization value” which is calculated this way:
- Each Epoch used for the averaging of the VEMP waveform has its own 100ms PreStim period. For each of these PreStim periods the RMS voltage is calculated.
- The average of all these PreStim RMS values is the “normalization value” for this VEMP waveform.

Waveforms Prior to Normalization

- RIGHT
  P1 amplitude: 348 uV
  Ave. RMS EMG: 201 uV
- LEFT
  P1 amplitude: 120 uV
  Ave. RMS EMG: 105 uV

Amplitude Asymmetry Value

\[
\text{Amplitude Asymmetry} = \frac{(\text{amplitude P13-N23}_{\text{left}} - \text{amplitude P13-N23}_{\text{right}})}{(\text{amplitude P13-N23}_{\text{left}} + \text{amplitude P13-N23}_{\text{right}})} \times 100
\]

uncorrected

\[
\text{Amplitude Asymmetry} = \frac{(120 \, \text{uV} - 348 \, \text{uV})}{(120 \, \text{uV} + 348 \, \text{uV})} \times 100 = 49\%
\]

After Normalization

- RIGHT
  P1 amplitude: 1.55 fV
  Ave. RMS EMG: 201 uV
- LEFT
  P1 amplitude: 1.055 fV
  Ave. RMS EMG: 105 uV

fV = “Frankenvolts”
Corrected Asymmetry Value

\[
\text{Amplitude Asymmetry} = \frac{(\text{amplitude } P13-N23_{\text{left}} - \text{amplitude } P13-N23_{\text{right}})}{(\text{amplitude } P13-N23_{\text{left}} + \text{amplitude } P13-N23_{\text{right}})} \times 100
\]

\[
(1.05 \text{ fV} - 1.55 \text{ fV})
\]

\[
(1.05 \text{ fV} + 1.55 \text{ fV})
\]

\[
\times 100 = 19\
\]

\[fV = \text{“Frankenvolts”}\]

Participants

- Twenty-five, otologically and neurologically healthy adults (mean age 23.32 [sd = 1.35] 8 male.
- All subjects demonstrated normal audiometric thresholds (i.e. air conduction thresholds that were < 20 dB without air/bone gaps) and did not report a previous history of otologic disease.
- All subjects were able to generate the minimum required EMG activity of 50 uV.

Asymmetry Comparisons

<table>
<thead>
<tr>
<th>Condition 1</th>
<th>Condition 2</th>
<th>Condition 3</th>
<th>Condition 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Monitoring No EMG Normalization</td>
<td>Monitoring No EMG Normalization</td>
<td>No Monitoring EMG Normalization</td>
<td>Monitoring EMG Normalization</td>
</tr>
<tr>
<td>43.82%</td>
<td>37.21%</td>
<td>30.74%</td>
<td>36.87%</td>
</tr>
</tbody>
</table>

Asymmetry Values (Mean +/- 2 SD)

EMG Amplitude Correction and Asymmetry

McCaslin et al., 2013

Conclusions

- Activating the SCM with the patient in semi-recumbent position with head turned away from the stimulated ear and head elevated (the optimal technique) was sufficient to produce the highest amplitude VEMPs with an acceptable amount of variability in young adult subjects.

Conclusions

- The added use of visual targets and amplitude normalization did reduce the variability in amplitude asymmetry measures, however the differences did not reach statistical significance.
Clinical Utility of Amplitude Normalization for Dealing with Asymmetry

McCaslin et al., 2014 JAAA

Design

• Subjects were 20 young adults from whom we recorded cVEMPs at 5 different EMG levels ranging from 0-400 µV.
• The absolute peak-to-peak amplitude of P13-N23, absolute latency of P13, and the left/right amplitude asymmetry of P13-N23 were measured.
• With and without the use of an EMG amplitude normalization techniques.

Participants

• All subjects demonstrated normal audiometric thresholds (i.e. air conduction thresholds that were < 20 dB without air/bone gaps) and did not report a previous history of otologic disease.
• All subjects were able to generate the minimum required EMG activity of 50 uV.

Data Analysis

• Dependent Variables
  – p13 latency.
  – p13-n23 peak to peak amplitude
  – mean RMS EMG activity.
  – standard deviation of the RMS
  – interaural amplitude asymmetry ratio across conditions and age.

Effect of EMG Amplitude Correction on IAA

• The main effect of CONDITION was significant \( p < .001 \).
• McCaslin et al., 2013 \( (p = .389) \).
Effect of Amplitude Correction on cVEMP Asymmetry

<table>
<thead>
<tr>
<th>EMG Target Comparisons</th>
<th>Uncorrected IAA%</th>
<th>Corrected IAA%</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>100 µV - 100 µV</td>
<td>14.12%</td>
<td>13.79%</td>
<td>P = .943</td>
</tr>
<tr>
<td>100 µV - 200 µV</td>
<td>31.04%</td>
<td>18.08%</td>
<td>P = .113</td>
</tr>
<tr>
<td>100 µV - 300 µV</td>
<td>55.81%</td>
<td>17.89%</td>
<td>P = .000***</td>
</tr>
<tr>
<td>200 µV - 200 µV</td>
<td>14.08%</td>
<td>10.11%</td>
<td>P = .304</td>
</tr>
<tr>
<td>200 µV - 300 µV</td>
<td>22.04%</td>
<td>11.31%</td>
<td>P = .015*</td>
</tr>
<tr>
<td>200 µV - 400 µV</td>
<td>29.37%</td>
<td>14.03%</td>
<td>P = .015*</td>
</tr>
<tr>
<td>300 µV - 300 µV</td>
<td>13.76%</td>
<td>11.44%</td>
<td>P = .361</td>
</tr>
<tr>
<td>300 µV - 400 µV</td>
<td>15.98%</td>
<td>12.88%</td>
<td>P = .860</td>
</tr>
<tr>
<td>400 µV - 400 µV</td>
<td>11.94%</td>
<td>12.18%</td>
<td>P = .945</td>
</tr>
</tbody>
</table>

* P < 0.05, ** P < 0.001

Effect of Normalization

Clinical Utility of Normalization and EMG monitoring

• In summary, the results of the investigation support the use of amplitude correction (i.e. amplitude normalization) of the cVEMP using EMG magnitude estimates obtained from the mean RMS of the EMG occurring before stimulus onset.

Conclusions

• For normal subjects who have been asked to create artificial amplitude asymmetries, IAADs are reduced significantly following the application of amplitude correction.
• Following amplitude correction the mean IAAD was between 14% and 17% regardless of the size of amplitude asymmetry that occurs prior to amplitude normalization.

What is the influence on standing balance when a saccular impairment is present?
McCaslin et al., 2012

McCaslin et al., 2011
Purpose

- To investigate the extent to which impairments in the function of the saccule (unilaterally absent VEMP), affects postural stability as measured via the Sensory Organization Test (SOT)
- Determine whether postural stability of individuals saccular impairment was significantly different compared to the postural performance of:
  - normal controls
  - patients with an impairment of the at least horizontal semi-circular canal.
  - patients with an impairment of at least both the lateral SCC and saccule

Participants

- Inclusion criteria
  - Diagnosed with unilateral vestibular system impairment
  - Must have completed the VEMP, Caloric test and SOT
- Exclusion criteria
  - Presence of spontaneous nystagmus
  - Orthopedic/neurologic deficits
  - Had taken a vestibular suppressant within 24 hours

Subject Grouping

<table>
<thead>
<tr>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>Group 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal VEMP; normal Caloric result</td>
<td>Normal VEMP; abnormal Caloric result</td>
<td>Abnormal VEMP; abnormal Caloric result</td>
<td>Normal Control Group</td>
</tr>
<tr>
<td>N = 28</td>
<td>N = 19</td>
<td>N = 15</td>
<td>N=30</td>
</tr>
<tr>
<td>51.50</td>
<td>49.52</td>
<td>65.86</td>
<td>48.43</td>
</tr>
</tbody>
</table>

Conclusion

- In this study, participants with an isolated impairment of the saccule, did not demonstrate significantly different self-perceived handicap when compared to the other experimental groups (i.e groups 2 and 3).
- However, they did demonstrate significantly higher self-perceived handicap when compared to their normal age-matched counterparts (group 4).

Something New

Acoustical Ocular VEMP (oVEMP)
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 VOR.
  - Does not represent movements of the eyes (i.e. they are short latency responses e.g. <10 msec for onset)
- EOMs have properties that allow them to be activated with precision at short latencies for fine motor control of eye movements

Anatomic Origins of oVEMP

Peripheral Generator/s of oVEMP

- Question: What is the peripheral end organ from where the oVEMP originates?
  - Saccule?
  - Utricle?
  - Semicircular canals?
  - All of the above?
- Answer: The utricle

Curthoys, 2012

- (There are) a small group of otolith afferents, which have an irregular spontaneous firing, respond well to dynamic otolith stimulation (motion, vibration, acoustical stimuli)
- Acoustic stimuli activate very few semicircular canal neurons and then only at high intensities
- ...the neural connections in the sacculo-ocular system are relatively weak compared to the neural connections in the utriculo-ocular ...
- ...saccular neurons have a strong projection to neck muscles and a weak projection to the ocularmotor system

From: Curthoys et al. 2012

Response from irregular otolith neuron during air conduction stimulation. Note synchronization of the increase in discharge rate with sound onset.
Evidence that oVEMP Originates from the Utricle

- Placement of electrodes beneath an averted contralateral eye places the inferior oblique m. beneath an active electrode.
- The saccule has weak, and the utricle strong, projections to extraocular muscles (e.g. inf. oblique) (Curthoys, 2010)

From Weber et al, 2012

Curthoys, 2012

- “I put forward the hypothesis that measuring oculomotor responses to ACS and BCV probes predominantly utricular function, whereas measuring neck muscle responses to ACS and BCV probes predominately saccular function.”
- “It is important to stress that this differential probing of utricular and saccular function is possible because of their differential neural projections, not because ACS and BCV are specific stimuli for different otolithic sensory regions; they are not.”

Curthoys, 2012

- “The oVEMP is a crossed, excitatory, ascending, utriculo-ocular response”
- “N10 probably indicated primarily the myogenic potential of the inferior oblique and inferior rectus muscles, because the size of the oVEMP n10 increases as the subject looks up and brings these muscles close to the electrodes”

oVEMP Pathway (after Curthoys, 2010)

- oVEMP pathway
  - Utricle
  - Sup. Vestib. Nerve
  - Medial Longitudinal Fasciculus
  - Motor Nucleus of Contra CN III
  - Contra Inferior Oblique m.
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)
  - Patient with right-sided vestibular schwannoma has normal caloric and oVEMP tests but abnormal cVEMP test (Murofushi et al. 2010)
  - 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 caloric test was abnormal and the ipsilateral cVEMP was normal (Manzari et al. 2010, Govender et al. 2011)
- 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)

Outline

- Vestibular Evoked Myogenic Potential Basics (history, types etc)
- Origins of cVEMP
- Origins of oVEMP
- Methods to record oVEMP and differences compared to cVEMP
- The normal oVEMP
- Abnormal oVEMP in the presence of disease
- Patterns of oVEMP and cVEMP abnormality
- Summary

Stimulus, Subject and Recording Variables for oVEMP

<table>
<thead>
<tr>
<th>Stimulus type/s</th>
<th>500 or 750 Hz tone-burst, 2-1-2 cycle, 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>
Effect of Stimulus Mode on oVEMP: Bilateral Response with Contralateral Predominating

Negativity is plotted as an upward deflection in this Figure

From: Rosengren et al. 2009

Stimulus Mode: oVEMP in Response to Sound and Vibration

From Todd et al. 2009

Sound – Response present 100-800 Hz  
Vibration – Response present 100-200 Hz

Effect of Stimulus Frequency

From Murnane et al. 2011

Best frequency is between 500 Hz and 1000 Hz

Effect of Stimulus Intensity

From Murnane et al. 2011

From: Piker et al. 2012

From: Piker et al. 2012
### Recordability of the cVEMP (top) and the oVEMP (bottom)

(Piker et al. 2012)

<table>
<thead>
<tr>
<th></th>
<th>10 Hz</th>
<th>20 Hz</th>
<th>30 Hz</th>
<th>40 Hz</th>
<th>50 Hz</th>
<th>60 Hz</th>
<th>60 dB SPL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young Adult</td>
<td>80%</td>
<td>87%</td>
<td>80%</td>
<td>80%</td>
<td>77%</td>
<td>80%</td>
<td>80%</td>
</tr>
<tr>
<td>Old Adult (50+)</td>
<td>78%</td>
<td>85%</td>
<td>80%</td>
<td>75%</td>
<td>78%</td>
<td>75%</td>
<td>75%</td>
</tr>
</tbody>
</table>

Recordings consist of 100+ msec epochs including a 10-20 msec prestim period.

### Recording Characteristics - oVEMP

- **Electrode locations:** Infraorbital (non-inverting), 2 cm infraorbital (inverting) or chin (inverting), Fpz (ground)
- **Gain:** 100,000x (versus 5000x for cVEMP)
- **Sampling rate:** ~3000 Hz
- **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

### Filter Types

- **Inf. Oblique m. is Innervated by CNIII**
  - Elevation of the eye places the infraorbital oblique muscles close to recording electrode.

### 2-Channel oVEMP Recording Version 2

- **Ground**
- **Active, +**
- **Reference, -**

### Recording Characteristics-oVEMP

- **Samples per Completed Average**
  - **Samples:** 100-500
  - Since this is a large EP but not as large as the cVEMP more single sweeps are required to improve the SNR e.g. 50-100 sweeps are required for the larger cVEMP
**Outline**

- Vestibular Evoked Myogenic Potential Basics (history, types etc)
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- Patterns of oVEMP and cVEMP abnormality
- Summary

---

**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)

---

**oVEMP: Contralateral versus Ipsilateral**

*Chihara et al. (2007) and Piker et al. (2011)*

Contralateral response is shorter in latency

<table>
<thead>
<tr>
<th>Stimulus: 500 Hz tone burst</th>
<th>Ipsilateral eye</th>
<th>Contralateral eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>Response prevalence (20 ears)</td>
<td>45%</td>
<td>90%</td>
</tr>
<tr>
<td>Amplitude (n1-p1) uV</td>
<td>1.9 (±0.2)</td>
<td>7.0 (±1.0)</td>
</tr>
<tr>
<td>N1 latency (msec)</td>
<td>12.8 (±0.6)</td>
<td>10.5 (±0.1)</td>
</tr>
<tr>
<td>P1 latency (msec)</td>
<td>17.7 (±0.9)</td>
<td>15.9 (±0.3)</td>
</tr>
</tbody>
</table>

---

**Interaural Latency and Amplitude Asymmetry Values**

*Piker et al. 2011*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Upper limit (mean + 2 sd) latency diff.</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1 latency (msec)</td>
<td>2.47</td>
</tr>
<tr>
<td>P1 latency (msec)</td>
<td>3.02</td>
</tr>
<tr>
<td>N1-P1 amplitude asymmetry (%)</td>
<td>33%</td>
</tr>
</tbody>
</table>

---

**Vestibular-evoked extraocular potentials by air-conducted sound**: Another clinical test for vestibular function

Yasuhiko Chihara ***, Shinichi Iwashita ***, Munetaka Usui ***, Yoshinobu Murai***

Response is larger for 500 Hz Tone burst than for a click

<table>
<thead>
<tr>
<th>Click</th>
<th>Response prevalence (%)</th>
<th>Amplitude (n1-p1) uV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsilateral eye</td>
<td>0</td>
<td>1.9</td>
</tr>
<tr>
<td>Contralateral eye</td>
<td>50%</td>
<td>7.0</td>
</tr>
</tbody>
</table>

Response is more prevalent contralaterally

Subjects were instructed to look upward and ipsilateral to stimulation.

---

**Effect of Ipsi versus Contra Recording on oVEMP Amplitude**

*Murnane et al, 2011*

![Graph showing difference in response between ipsi and contra](image)
Subject Variables and the oVEMP

Effect of Age on oVEMP Threshold
Threshold increases by ~5 dB
Piker et al. 2010

From: Piker et al. 2012
Best frequency increases with age

Subject Variables
Amplitude decreases with age and is absent in ~33% of normal elders

Effect of Age on oVEMP Amplitude and Latency
From Piker et al. 2011

Recording Variables and the oVEMP
Effect of Gaze Direction on oVEMP

- The largest oVEMPs were obtained from the inferior electrodes with upward, midline gaze.


Effect of Gaze Direction on oVEMP

Murnane et al. 2011

Why do oVEMPs become larger when you look up? Explaining the effect of gaze elevation on the ocular vestibular evoked myogenic potential

Sally M. Rosengren \textsuperscript{a,b,}, James G. Celio\textsuperscript{b,}, Dominik Stramann\textsuperscript{c,}, Konrad P. Welber\textsuperscript{e,d}

- Gaze elevation significantly increases oVEMP amplitude, while gaze depression decreases amplitude and prolongs latency.
- This gaze effect is primarily due to changes in tonic eye muscle activity, while the contribution of changes in muscle-electrode distance is significant but small.
- oVEMPs recorded from below the eyes originate mainly in the inferior oblique muscle during up-gaze and in the inferior rectus muscle during down-gaze.

Rosengren et al., 2013
OVEMP and Reference Contamination
Best montage is infraorbital-chin bipolar

Current montage
Infraorbital – Infraorb.-Jom
Infraorbital - chin
Reference montage, GJ/DM
Reference contamination
Infraorb.-3 cm – chin

Reference contamination occurred 100% of the time and averaged 30% (range: 18-43%)

Piker et al. 2011

Caveats
Adapted from: Curthoys, Manzari, Smulders, Burgess (2009)

- Asymmetry in cVEMP and oVEMP can occur due to:
  - End organ or nerve impairments
  - Asymmetric muscle function
  - Differential effects of disease on the transmission pathways
  - Technical errors (in placement of electrodes, or, in setting the amplification gains)

Summary Conclusions

- The cVEMP and oVEMP can vary independently of one another
- This suggests that their peripheral anatomic/physiological origins are probably different
  - The cVEMP in response to low frequency tone bursts probably originates from the saccule and inferior vestibular nerve.
  - The oVEMP in response to low frequency tone bursts probably originates from the utricle and superior vestibular nerve
  - The caloric and vHIT response originates from the horizontal SCC and superior vestibular nerve.
- VEMP results when combined with results of the caloric test and vHIT test can provide localizing information about the site or sites of peripheral vestibular system impairment

Should I do a threshold search?

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Caloric C1/C2 (mV)</th>
<th>Caloric C1/C2 (mV)</th>
<th>Caloric C1/C2 (mV)</th>
<th>Caloric C1/C2 (mV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>54</td>
<td>14.0</td>
<td>14.0</td>
<td>14.0</td>
<td>14.0</td>
</tr>
<tr>
<td>55</td>
<td>14.0</td>
<td>14.0</td>
<td>14.0</td>
<td>14.0</td>
</tr>
<tr>
<td>56</td>
<td>14.0</td>
<td>14.0</td>
<td>14.0</td>
<td>14.0</td>
</tr>
</tbody>
</table>

Table 4. Vestibular evoked magnetic potential thresholds and specificities for the diagnosis of superior canal dehiscence syndrome per decade of age

*Should I do a threshold search?*
“Contemporary Concepts in Positioning Vertigo”

Devin L. McCaslin, Ph.D.
Division of Audiology
Division of Vestibular Sciences
Vanderbilt Bill Wilkerson Center for Otolaryngology and Communication Sciences
Vanderbilt University Medical Center

Faculty and Staff for Vestibular Sciences

Gary Jacobson, Ph.D.
Devin L. McCaslin, Ph.D.
Kelley Hatton, Au.D.
Lauren Tegel, Au.D.
Sarah Grantham, Au.D.

“Positional” vs “Positioning” Nystagmus

• Positional: nystagmus present in static head postures
• Positioning: nystagmus induced by head motion (Hallpike Maneuver)
• Represents attempts by eye movement system to stabilize gaze when vestibular system is activated
• Identification of SCC is based on the direction of the slow phase eye movement

Positional Testing Protocol
(From: Barber and Stockwell 1976; Shepard, 2001)

• Sitting
• Sitting, head turned lt
• Sitting, head turned rt
• Body rt
• Body lt
• Supine
• Supine w-head elevated 30 deg.
• Supine head turned lt
• Supine head turned rt
• Head Hanging
Assessment and Treatment of Benign Paroxysmal Positional Vertigo (BPPV)

History and Significance
- Barany (first documented case)
- Margaret Dix and Charles Hallpike (1952)
  - Temporal bone histology
  - Determined it was peripheral
- Flourens showed impaired SCC would cause pigeons to move their heads. SCCs were not for processing sound
- Ewald — applied positive and negative pressures to SCCs — set forth laws 1 and 2

History and Significance
- Wersall showed that each vestibular sensory cell has one kinocilium and many stereocilia.
- Schuknecht (1962) — cupulolithiasis
- Gacek confirmed Schuknecht’s findings — nerve sections
- Cawthorne, Semont, and Brandt-Daroff exercises devised

Incidence of BPPV
- Most common peripheral vestibular disorder (Mizukoshi et al. 1988, Froehling et al. 1991)
- 64,000-100,000 cases/year (Herdman)
- Can occur spontaneously (>50%)
- Reporting frequency is age-dependent
  - 3% age 11-29
  - 20% ages 30-59
  - 30%-50% ages 60-99

History and Significance
- Epley — (1993) developed a technique for free-floating particles using models
- Parnes and McClure (1992) — identified free-floating particles during a posterior canal occlusion — developed their very similar technique

Movements that Provoke Symptoms
- Bending over
- Looking up
- Lying down or getting up
- Rolling over in bed
- Going to the Dentist or getting hair washed at Salon
Pathophysiology

Displaced otoconia are present as mobile densities within the canal.

After a head movement, the mass of otoconia moves within the SCC.

The movement displaces the endolymphatic fluid, deflecting the cupula of the involved canal and elicits the nystagmus and vertigo.

Probability of Canal Involvement

<table>
<thead>
<tr>
<th>Study</th>
<th>Number</th>
<th>PC (%)</th>
<th>HC (%)</th>
<th>AC (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herdman, Tusa, &amp; Ciricoteanu (1994)</td>
<td>18</td>
<td>1.3</td>
<td>1.7</td>
<td>3.5</td>
</tr>
<tr>
<td>Zablotno (1986)</td>
<td>124</td>
<td>2.6</td>
<td>4.0</td>
<td>5.9</td>
</tr>
<tr>
<td>Wolf, Bayou, Mandel, &amp; Matta (1998)</td>
<td>127</td>
<td>5.5</td>
<td>3.9</td>
<td>2.9</td>
</tr>
<tr>
<td>Hombalis, Belchetz, Horns, &amp; Jacobs (1999)</td>
<td>252</td>
<td>7.0</td>
<td>2.4</td>
<td>1.2</td>
</tr>
<tr>
<td>Rudgevend (2001)</td>
<td>86</td>
<td>7.3</td>
<td>3.5</td>
<td>1.2</td>
</tr>
<tr>
<td>Koren &amp; Stabufkine (2004)</td>
<td>112</td>
<td>8.6</td>
<td>6.2</td>
<td>1.8</td>
</tr>
<tr>
<td>Cali et al. (2005)</td>
<td>109</td>
<td>12.0</td>
<td>11.0</td>
<td>1.2</td>
</tr>
<tr>
<td>Jackson, Monger, Pasternak, &amp; Riveiro (2007)</td>
<td>180</td>
<td>11.0</td>
<td>11.0</td>
<td>21.2</td>
</tr>
</tbody>
</table>

PC = posterior canal, HC = horizontal canal, AC = anterior canal

Age

– BPPV is more common in older adults (Herdman and Tusa, 2000)
  • Evidence of weakened linking filaments among otoconia in older rats (Jang et al., 2006)
  • Also, otoliths deteriorate with age (from the inside)

Side of Involvement

– Primarily unilateral – 4% to 15% are bilateral (Longridge and Barber, 1978)
  • Bilateral BPPV occurs 2X more often in head trauma patients (Katsarkas, 1999)
– Right side > left side (Epley, 1992, Korres et al., 2002)
  • Von Brevern et al. (2004) reviewed 18 studies and found that only two studies reported more left than right being 1.41 times more likely. Thought to be sleeping position.

Co-existing Peripheral Vestibulopathy

– 13-50% of patients with BPPV demonstrate a peripheral vestibulopathy (Blessing et al, 1986, Hughes and Proctor 1997)
  • 37%, Roberts et al. (2005)
  • 39%, Jacobson et al. (unpublished observations)
Canal Involvement
– The identification of canal involvement usually is based on the direction of the nystagmus observed when the patient is moved into the provoking position.

Examining Patients with BPPV
– Positioning testing should be done prior to "positional" testing
– The initial positioning maneuver should be directed toward the ear assumed to be affected.

Examining Patients with BPPV
– Frenzel’s or VNG should be used to

Examining Patients with BPPV
– Patients should be counseled re: the underlying mechanisms and characteristics of BPPV, before the maneuver to ensure cooperation and comfort.
– Should be instructed to keep their eyes open.

Maneuvres to Provoke BPPV
– Modified Dix-Hallpike (Posterior/Anterior)
– Sidelying Test (Anterior)
– Roll Test (Horizontal/Lateral)

Identification of Posterior Canal BPPV
Dix-Hallpike
(posterior and anterior SCC)

Maneuver patient from an upright to supine position with the head turned 45 degrees to one side

Head should be extended 20 degrees.

Side-lying Maneuver

– Maneuver if neck hyperextension is contraindicated by VBI or cervical spondylosis
– Cohen (2004) found no significant difference in the ability to identify BPPV when SL was compared to DH
– Head and neck are fully supported by the table
– Do not use with patients who have hip replacements

Diagnostic Criteria-pBPPV

<table>
<thead>
<tr>
<th>Duration</th>
<th>Usually less than 40 s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direction change</td>
<td>Down-beating when returning to the sitting position</td>
</tr>
<tr>
<td>Fatigability</td>
<td>Intensity reduces when the maneuver is repeated</td>
</tr>
<tr>
<td>Temporal course</td>
<td>Initial increase in intensity and then slowly declines</td>
</tr>
<tr>
<td>Direction of nystagmus</td>
<td>Torsional/up-beating when placed in the initial provocative position</td>
</tr>
</tbody>
</table>

McCaslin, 2012

Posterior BPPV
(Canalithiasis)
Diagnostic Criteria

- Temporally, the rate of nystagmus typically begins gently, increases in intensity, and then declines in intensity as it resolves.

Identification of Anterior Canal BPPV

Deep Supine Head-Hanging (anterior SCC)

Deep Supine Head-Hanging (anterior SCC)

Posterior SCC

Deep Supine Head-Hanging (anterior SCC)

aSCC BPPV

- Herdman and Tusa (1996)
  - Is a rare variant that sometimes occurs as a transition from typical p-BPPV to an a-BPPV while patients are undergoing Liberatory (treatment) maneuvers.
  - Should be alert to central causes
Diagnostic Criteria-aBPPV

<table>
<thead>
<tr>
<th>Duration</th>
<th>Usually less than 40 s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direction change</td>
<td>Up-beating when returning to the sitting position</td>
</tr>
<tr>
<td>Fatigability</td>
<td>Intensity reduces when the maneuver is repeated</td>
</tr>
<tr>
<td>Temporal course</td>
<td>Initial increase in intensity and then slowly declines</td>
</tr>
<tr>
<td>Direction of nystagmus</td>
<td>Torsional/down-beating when placed in the provocative position (torsional component may be difficult to identify)</td>
</tr>
</tbody>
</table>

Peripheral Down-Beating  

Anterior BPPV  
(Canalithiasis)

Central Down-beating Nystagmus  
(Cerebellar)

Identification of Lateral SCC BPPV

Roll Test
Clinical Syndrome of hSCC BPPV

<table>
<thead>
<tr>
<th>Duration</th>
<th>Usually less than 60 s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direction change</td>
<td>Reverses direction on lateral head turn</td>
</tr>
<tr>
<td>Fatigability</td>
<td>Intensity reduces when the maneuver is repeated</td>
</tr>
<tr>
<td>Temporal course</td>
<td>Initial increase in intensity and then slowly declines</td>
</tr>
<tr>
<td>Direction of nystagmus</td>
<td>Linear-horizontal (can be geotropic or otopositropic)</td>
</tr>
</tbody>
</table>

McCaslin, 2012

Geotropic hBPPV

- Geotropic type: rotation of the head toward the involved end-organ results in horizontal nystagmus beating toward the undermost (affected) ear

- Rotation of the head toward the unimpaired side results in horizontal nystagmus (lower SPV) which also beats toward the undermost ear

Geotropically Posterior Arm hSCC

Geotropic Horizontal SCC BPPV, Head-roll Left

Geotropic Horizontal SCC BPPV, Head-roll Right

Horizontal Double Drop
“Bow and Lean” Test

Ageotropic H-BPPV
- Ageotropic type: Rotation of the head toward the impaired side results in horizontal nystagmus (lower SPV) which beats toward the uppermost ear
- Rotation of the head toward the uninvolved end-organ results in horizontal nystagmus beating toward the uppermost ear

Ageotropic hBPPV
- Ageotropic nystagmus has been suggested to originate from otocodia located in the anterior part of the lateral SCC in close approximation to the cupula
- Can be on either the canal side or the utricular side

(Casani et al, 2002)

Ageotropic-Anterior Arm hSCC
(Canal Side)

Ageotropic-Anterior Arm hSCC
(Utricular Side)

Ageotropic Horizontal SCC BPPV, Head-roll Right
Ageotropic Horizontal SCC BPPV, Head-roll Left

“Bow and Lean” Test

Diagnostic Criteria for Typical hSCC BPPV
- Latency/Duration:
  - Latencies = < 5s
  - Durations = longer (20-60 s)
- Often more severe than in pSCC BPPV and more frequently associated with nausea
- About 4/10 of the patients show moderate horizontal canal paresis during caloric irrigation of the affected ear

Management of BPPV

Treatment of aBPPV and pBPPV
- Two types of treatments have been found effective for ant/post canal BPPV:
  - 1) the canalith repositioning procedure - Epley
  - 2) the Semont/liberatory maneuver
Canalith Repositioning Maneuver

Canalith Repositioning Procedure
• How many times do you do the CRP during a visit? 1
• In our clinic this has been found to be most effective. Will occasionally bring people back if symptoms persist.

Pre-Treatment (CRP)
• The patient should be counseled that nausea or a sense of falling may arise during the procedure.
• If a patient is known to have severe motion intolerance or a previous history of severe nausea during the identification process may wish to be prescribed an antiemetic.

Modified Epley
1
(posterior and anterior SCC)

Modified Epley
• Epley recommended leaving the patient in each position for only 6-13 seconds based on the latency of onset and duration of nystagmus
• A range of 2 minutes to 4 minutes has been suggested by others. (Ruckenstein, 2001, Herman et al. 1993, Tirelli et al, 2000).
• We like 1.5 min.
Semont – Liberatory Treatment

- Patient is seated in the upright position and the head is turned 45 deg toward/away the canal to be treated
- Patient is then rapidly moved to the side-lying position
- Rapidly move the patient to the opposite side-lying position without stopping and without changing the position of the head.

Semont-Liberatory Maneuver
(Left Posterior Canal)

Self Treatment of BPPV
(Posterior SCC)

Identification of hBPPPV

- In order for treatment to be successful the clinician must identify the involved side
- Identification is based on:
  - Geotropic nystagmus suggests the impaired side is the one with the greater response
  - Ageotropic nystagmus is assumed to the side with the smaller response

Geotropic hSCC BPPV

- Believed to make up 60-83% of the cases involving HC-BPPV. (Honrubia et al, 1999; Steenerson et al, 2005; Cakir et al, 2006; White et al, 2005)
- Otoconia located in the posterior part of the lateral SCC
“BBQ” Roll Maneuver (Geo)

Casani et al., 2002

This treatment has been reported to be effective 75-80% of the time

(Fife, 1998; White et al, 2005; Casani et al, 2002).

Liberatory Maneuver for Geotropic hBPPV

“BBQ Roll Maneuver”

Patient is instructed to:
- Lay down supine
- Rotate the head and body toward the healthy side
- Maintain this position for 8-12 hours

“Forced Prolonged Positioning”
Ageotropic hSCC BPPV

- Believed to make up 17-40% of the cases involving HC-BPPV. (Honrubia et al, 1999; Steenerson et al, 2005; Cakir et al, 2006; White et al, 2005)
- Otoconia located in the anterior part of the lateral SCC

Gufoni Manuever (geotropic)


Case Illustrating Ageotropic (Before Gufoni)

Head Roll Left

Head Roll Right

Case Illustrating Ageotropic (After Gufoni)

Head Roll Left

Head Roll Right
Casani Treatment


McCaslin, 2012

Treatment of Anterior Canal BPPV

Yacovino, Hain, & Guaitieri, 2009

Treatment of Left Anterior Canal BPPV

Kim et al., 2006

Summary (Canalithiasis)

- Posterior BPPV
  - Canalith Repositioning Procedure- CRP (Epley)
  - Liberatory Maneuver (Semont)
  - Brandt-Daroff
- Anterior BPPV
  - Canalith Repositioning Procedure- (Kim et al.)
  - Deep Supine Head Maneuver (Yacovino)
  - Liberatory Maneuver (Semont)
Summary

• Geotropic Horizontal Canal
  – BBQ Roll
• AGeotropic Horizontal Canal
  – Gufoni (Conversion)
  – BBQ Roll
  – Casani

Habituation

Brandt-Daroff Exercises

  – Instructions:
    • Rapidly lie on affected side, wait for dizziness to resolve plus 20 sec
    • Sit-up wait for dizziness to resolve plus 20 sec
    • Lie on opposite side, wait for dizziness to resolve or if no symptoms just 20 sec
    • Repeat 10-20 times, 3x day until the patient has two days in a row with no vertigo (Tusa and Herdman 1998).
  – Rationale: Canalith dispersion or habituation
  – Expectations: Improvement days to weeks

Brandt-Daroff Exercise

  – Indications - Posterior or anterior canal cupulo/canalithiasis
  – Contraindications: Severe back/neck problems

Method of Treatment

  How do habituation exercises, or, repositioning maneuvers “fix” BPPV?
  – Move the displaced otoconia out of the canal
  – Dissolve the displaced otoconia
  – Central nervous system compensation
Canalith Repositioning for Benign Paroxysmal Positional Vertigo

- Compared CRP to groups not treated using meta-analysis techniques.
- 72% of the patients who underwent CRP resolved
- 1/3 had spontaneous resolution

Quality of Life

- Treatment has been shown to increase QOL. (Lopez-Escamez et al, 2003; Gamiz and Lopez-Escamez, 2004)
- There were significant pre-treatment QOL scores between patients with and without BPPV.
- Following treatment there was no difference.

Post-Treatment Instructions

- Research suggests that keeping the head upright for 48 hours does not increase the success of treatment.
  - Massoud and Ireland, 1996
  - Tusa and Herdman, 1996
  - Nuti et al., 2000

Questions