Vertigo, Dizziness, and Giddiness...Oh My!

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Is this **YOU** when a new **DIZZY** patient is on the clinic schedule?
Vertigo
Pre-syncopal / lightheadedness
Disequilibrium (off balance)
Other ( )
20-30% may have vertigo or dizziness in their lifetime.

1.7% of ambulatory medical care visits recorded vertigo or dizziness among CC.
  - 2.2 million people in 2010

Accounted for 2.5% of US ED visits in the past (1995-2004)
Sir, are you spinning around the room or is the room spinning around you?
The meaning of the word “vertigo”

- Blakely and Goebel, 2001

- Objective: To determine whether otologists see further need to define “vertigo” and assess the variety of meanings meant by it.

- Survey questionnaire to 720 AOS and ANS members

- 42% response rate (n = 306 usable responses)
Background: Blakely and Goebel, 2001

- “inner ear disorders do not always cause spinning. If inner ear disorders are mild or if compensation reduced the severity…words like “imbalance, drunkenness, wooziness, swaying…may best describe the sensation”

- AAO-HNS defines vertigo as “sensation of motion when no motion is occurring relative to earth’s gravity”…thus any motion counts?

- Dorland’s Medical Dictionary: A hallucination of movement; a sensation as if the external world were revolving around the patient (objective vertigo) OR …revolving in space (subjective vertigo)

- Mosby’s Medical dictionary: “a sensation of instability, giddiness, loss of equilibrium, or rotation, caused by a disturbance in the semicircular canal of the inner ear or the vestibular nuclei of the brainstem.
The meaning of the word “vertigo”

Less than 50% agree on what it means!

Table 2. Responses

<table>
<thead>
<tr>
<th>Response to the statement, “The word vertigo should refer to dizziness with...”</th>
<th>Number (%) (n = 411)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spinning or turning only</td>
<td>184 (45%)</td>
</tr>
<tr>
<td>Any sensation of movement between the patient and the environment</td>
<td>166 (40%)</td>
</tr>
<tr>
<td>Imbalance or any of the 2 previous sensations</td>
<td>24 (6%)</td>
</tr>
<tr>
<td>Oscillopsia as well as other dizziness</td>
<td>16 (4%)</td>
</tr>
<tr>
<td>All of the above</td>
<td>21 (5%)</td>
</tr>
</tbody>
</table>
It’s vertigo…now what?
# Peripheral vs. Central Vertigo

## Table 2
Differential diagnosis of vertigo

<table>
<thead>
<tr>
<th>Peripheral Causes</th>
<th>Central Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPPV</td>
<td>Migrainous vertigo</td>
</tr>
<tr>
<td>Vestibular neuritis</td>
<td>Intracranial mass</td>
</tr>
<tr>
<td>Meniere’s disease</td>
<td>Cerebrovascular attack</td>
</tr>
<tr>
<td>Perilymphatic fistula</td>
<td>Vertebrobasilar insufficiency</td>
</tr>
<tr>
<td>Herpes zoster oticus</td>
<td>Chiari malformation</td>
</tr>
<tr>
<td>Acoustic neuroma</td>
<td>Multiple sclerosis</td>
</tr>
<tr>
<td>Ototoxicity</td>
<td>Episodic ataxia type 2</td>
</tr>
<tr>
<td>Otitis media</td>
<td></td>
</tr>
<tr>
<td>Semicircular canal dehiscence syndrome</td>
<td></td>
</tr>
<tr>
<td>Posttraumatic vertigo (labyrinth concussion)</td>
<td></td>
</tr>
</tbody>
</table>

Wipperman, J. Dizziness and Vertigo.
<table>
<thead>
<tr>
<th>Encephalitis</th>
<th>Toxicity (Phencyclidine)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma</td>
<td>Vertebrobasilar</td>
</tr>
<tr>
<td>Herpes Simplex</td>
<td>Atherothrombotic Disease</td>
</tr>
<tr>
<td>Labyrinthitis</td>
<td>Wernicke Encephalopathy</td>
</tr>
<tr>
<td>Mastoiditis</td>
<td>Otosyphillis</td>
</tr>
<tr>
<td>Meningitis</td>
<td></td>
</tr>
<tr>
<td>Subarachnoid</td>
<td></td>
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<tr>
<td>Hemorrhage</td>
<td></td>
</tr>
<tr>
<td>Subdural Hematoma</td>
<td></td>
</tr>
<tr>
<td>Thrombolytic Therapy</td>
<td></td>
</tr>
<tr>
<td>Toxicity (Carbon</td>
<td></td>
</tr>
<tr>
<td>Monoxide)</td>
<td></td>
</tr>
</tbody>
</table>
The BIG 4

- BPPV (40%; MOST COMMON)
- Vestibular neuronitis/ labyrinthitis (10%; 2nd MOST COMMON)
- Meniere’s disease (0.5%; NOT EVEN A LITTLE BIT COMMON)
- Migraine vertigo
Benign Paroxysmal Positional Vertigo

- Most common cause of vertigo
- 40% of vertigo diagnosis seen in primary care
- 5th-6th decade; ♀ > ♂
- 10% of adults diagnosed by 80 y.o.a.

Risk factors:
- prior trauma
- prior vestibular disorders,
- osteoporosis/ vit. D def.
- sleep position
Benign Paroxysmal Positional Vertigo

- 2.4% lifetime prevalence
- Loose calcium carbonate debris (otoconia)
  - Motion resolves when debris settles
- 85-90% posterior SSC, 8-10% lateral SSC
- Duration: seconds to a minute
- Nystagmus:
  - Geotrophic, rotatory (torsional) nystagmus TOWARD the affected ear (fast phase toward affected ear)
- Triggers: (position changes)
  - Turning in bed
  - Looking up
  - Bending forward
BPPV Treatment Guidelines

The following recommendations are ALL per the most recent Clinical Practice Guidelines per the AAO-HNS 2008
**Dix-Hallpike Maneuver:**
- Gold standard
- Head movement should be *fairly quick WITH EYES OPEN*
- Head turned 45° while patient upright and neck extended 20°
- Latency 5 -20s
- Crescendo-decrescendo nystagmus up to 60 sec.

**Lopez-Escamez et al. 2000:**
- SN 82%, SP 71% among specialty clinicians

**Hanley and O’Dowd, 2001:**
- PPV 83%, **NPV 52%**
Factors that affect exam:
- Speed of movements
- Time of day
- Vestibular suppressants
- Angle of the plane of the occiput

Relative Contraindications:
- Severe vascular disease
- Cervical stenosis
- Severe kyphoscoliosis
- Limited neck ROM
- Down’s syndrome
- Severe RA
- Spinal cord injuries
- Morbidly obese
- SCI
So, Dix Hallpike was negative...

...*Do I give up on the diagnosis?*

**Answer:** **NO**

Given low NPV – repeat in 1 week or separate visit (avoids FN)

vs.

- Supine roll test

Repeat exam on a separate visit per clinical practice guidelines because of FNs and because failure to diagnose BPPV can lead to a costly diagnostic work up thus it is in the best financial interest of the patient to make sure everything has been done to establish a diagnosis.
Sounds like BPPV but...
Lateral Canal BPPV

- Clinically sounds like BPPV, but Dix-Hallpike is negative.
- 8-10%(15%) prevalence
  - Gets considerably less attention
- Dx with supine roll test
Supine Roll Test

- NO SN/SP data
- NO gold standard test to compare

Supine roll test:
- Nystagmus:
  - Geotrophic: horizontal, beating toward undermost ear
  - Apogeotrophic: less common; horizontal nystagmus toward uppermost ear
  - In both, the side with the most intense nystagmus is the affected ear
BPPV: Treatment

- Epley’s Maneuver aka Canalith repositioning procedure (CRP)
- Semont Maneuver
- Lempert Maneuver (roll maneuver) – lateral canal BPPV
- Self-administered (home) exercises
Epley Maneuver:
-Most commonly attempted procedure
-Head changes settles the loose otoconia from the SCC into the utricle
-Start with affected side (series of 90 degree rotations)

Cochrane Review:
-OR of 4.2 (95% CI) in favor of treatment for *subjective symptom resolution*
-OR of 5.1 (95% CI) in favor for conversion after (+) Dix Hallpike
## CRP Effectiveness

### Table 7
Randomized controlled trials evaluating the effectiveness of CRP for posterior canal BPPV

<table>
<thead>
<tr>
<th>Reference</th>
<th>Improved in treatment group n/N (%)</th>
<th>Improved in control group n/N (%)</th>
<th>Endpoint</th>
<th>Time to assessment</th>
<th>P value</th>
<th>Odds ratio (95% CI)</th>
<th>NNT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lynn 1995</td>
<td>11/18 (61%)</td>
<td>3/20 (15%)</td>
<td>Vertigo resolution</td>
<td>2 weeks</td>
<td>0.033</td>
<td>6.3 (1.29-30.5)</td>
<td>2.2</td>
</tr>
<tr>
<td>Froehling 2000</td>
<td>12/24 (50%)</td>
<td>5/26 (19%)</td>
<td>Vertigo resolution</td>
<td>1-2 weeks</td>
<td>0.020</td>
<td>4.2 (1.2-14.8)</td>
<td>3.3</td>
</tr>
<tr>
<td>Simhadri 2003</td>
<td>19/20 (95%)</td>
<td>3/20 (15%)</td>
<td>Vertigo resolution</td>
<td>1 week</td>
<td>0.001</td>
<td>107.7 (10.2-1135.5)</td>
<td>1.3</td>
</tr>
<tr>
<td>Yimtae 2003</td>
<td>12/29 (41%)</td>
<td>1/27 (4%)</td>
<td>Vertigo resolution</td>
<td>1 week</td>
<td>0.005</td>
<td>18.4 (2.2-154.4)</td>
<td>2.7</td>
</tr>
<tr>
<td>Cohen 2005</td>
<td>*/24 (CRP)</td>
<td>*/25 (CRP)</td>
<td>Vertigo frequency scale (0-10)</td>
<td>4 weeks†</td>
<td>0.021</td>
<td>3.3 (1.0-11.3)</td>
<td>3.4</td>
</tr>
<tr>
<td>von Brevern 2006</td>
<td>28/35 (80%)</td>
<td>4/31 (13%)</td>
<td>Vertigo resolution</td>
<td>24 hours</td>
<td>0.001</td>
<td>27.0 (7.1-109.9)</td>
<td>1.5</td>
</tr>
</tbody>
</table>

CI, confidence interval; CRP, canalith repositioning procedure; LM, Semont’s liberatory maneuver; NNT, number needed to treat.

*Responses were analyzed with multilevel methods and expressed as fitted linear regression graphs, so no discrete numerical expression of the response rates could be determined.

†Time to evaluation was varied, so data presented are based on fitted linear regression curves at 4 weeks.
**Semont Maneuver:**

Per Clinical practice guidelines: Semont maneuver is more effective than no treatment or Brandt-Daroff exercises in relieving symptoms of posterior canal BPPV.

**Figure 4** The Semont maneuver for right-sided BPPV. (1) Patient is seated in the upright position, then the patient’s head is turned 45 degrees toward the left side, and the patient is then rapidly moved to the side-lying position as depicted in position (2). This position is held for approximately 30 seconds, and then the patient is rapidly moved to the opposite side-lying position without passing in the sitting position and without changing the head position relative to the shoulder, resulting in position (3). This position is maintained for 30 seconds and then the patient gradually resumes the upright sitting position. (Adapted from reference 19.)
**Lempert Maneuver:**
- AKA Barbeque roll
- Each position held for 10-30s until symptoms subside
- Up to 100% success for geotrophic variant
- Up to 50% success for apogeotrophic variant
- Start towards unaffected side
**Gufoni Maneuver:**

- For geotrophic variant: sitting position → straight side lying position on unaffected side then quick head turn 45-60° x 2 minutes → sit up with head in same position

- For apogeotrophic variant: sitting position → straight side lying position on affected side then quick head turn 45-60° x 2 minutes → sit up with head in same position

RCT shows statistically more successful than Lempert + FPP – 86% vs. 61%
Patient has BPPV, do I HAVE to...

- ...treat with in office CRP first? What if CRP don’t work initially?

Answer: It is RECOMMENDED but not necessarily...

Per Clinical Practice guidelines 2008:
- Vestibular rehabilitation
- Observation
### Table 8
Symptom resolution rates for observation alone for BPPV*

<table>
<thead>
<tr>
<th>Reference</th>
<th>Resolved n/m</th>
<th>% Resolved</th>
<th>Sham or pure observation</th>
<th>Time to assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>von Brevern 2007&lt;sup&gt;11&lt;/sup&gt;</td>
<td>22/26</td>
<td>84.6</td>
<td>Sham</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Sekine 2006&lt;sup&gt;142&lt;/sup&gt;</td>
<td>48/60</td>
<td>80.0</td>
<td>Observation</td>
<td>1 month</td>
</tr>
<tr>
<td>Imai 2005&lt;sup&gt;49&lt;/sup&gt;</td>
<td>45/70</td>
<td>64.0</td>
<td>Observation</td>
<td>1 month</td>
</tr>
<tr>
<td>Simhadri 2003&lt;sup&gt;177&lt;/sup&gt;</td>
<td>3/15</td>
<td>20.0</td>
<td>Observation</td>
<td>4 weeks</td>
</tr>
<tr>
<td>Yimtae 2003&lt;sup&gt;129&lt;/sup&gt;</td>
<td>7/20</td>
<td>35.0</td>
<td>Observation</td>
<td>1 month</td>
</tr>
<tr>
<td>Sherman 2001&lt;sup&gt;131&lt;/sup&gt;</td>
<td>11/22</td>
<td>50.0</td>
<td>Sham</td>
<td>3 months</td>
</tr>
<tr>
<td>Asawavichianginda 2000&lt;sup&gt;135&lt;/sup&gt;</td>
<td>18/22</td>
<td>81.8</td>
<td>Observation</td>
<td>3 months</td>
</tr>
<tr>
<td>Steenerson 1996&lt;sup&gt;171&lt;/sup&gt;</td>
<td>17/40</td>
<td>42.5</td>
<td>Observation</td>
<td>3 months</td>
</tr>
<tr>
<td>Lynn 1995&lt;sup&gt;128&lt;/sup&gt;</td>
<td>3/15</td>
<td>20.0</td>
<td>Sham</td>
<td>1 month</td>
</tr>
<tr>
<td>Blakley 1994&lt;sup&gt;176&lt;/sup&gt;</td>
<td>19/22</td>
<td>86.4</td>
<td>Observation</td>
<td>1 month</td>
</tr>
</tbody>
</table>

*Endpoint: resolution of vertigo symptoms at the time of assessment.*
Patient is cured!...

Now what?

Answer:

- **Re-evaluate in 1 month**
  - If persistent BPPV
    - Repeat H&P
    - Re-visit DDX
    - Imaging
    - vestibular function tests
  - Surgery? (extremely rare)

- **Educate** – 25% recurrence in 6 mos.
### BPPV Practice Guidelines Summary 2008

Table 4
Outline of evidence-based statements

<table>
<thead>
<tr>
<th>Guideline segment (Evidence-based statement number)</th>
<th>Statement strength</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I. Presumed benign paroxysmal positional vertigo (BPPV)</strong></td>
<td></td>
</tr>
<tr>
<td>a. Diagnosis of posterior canal BPPV (Statement #1a)</td>
<td>Strong recommendation</td>
</tr>
<tr>
<td>b. Diagnosis of lateral canal BPPV (Statement #1b)</td>
<td>Recommendation</td>
</tr>
<tr>
<td>c. Differential diagnosis (Statement #2a)</td>
<td>Recommendation</td>
</tr>
<tr>
<td>d. Modifying factors (Statement #2b)</td>
<td>Recommendation</td>
</tr>
<tr>
<td><strong>II. Diagnostic testing</strong></td>
<td></td>
</tr>
<tr>
<td>a. Radiographic and vestibular testing (Statement #3a)</td>
<td>Recommendation against</td>
</tr>
<tr>
<td>b. Audiometric testing (Statement #3b)</td>
<td>No recommendation</td>
</tr>
<tr>
<td><strong>III. Treatment</strong></td>
<td></td>
</tr>
<tr>
<td>a. Initial therapy of BPPV</td>
<td></td>
</tr>
<tr>
<td>i. Repositioning maneuvers as initial therapy (Statement #4a)</td>
<td>Recommendation</td>
</tr>
<tr>
<td>ii. Vestibular rehabilitation as initial therapy (Statement #4b)</td>
<td>Option</td>
</tr>
<tr>
<td>iii. Observation as initial therapy (Statement #4c)</td>
<td>Option</td>
</tr>
<tr>
<td>b. Medical therapy (Statement #5)</td>
<td>Recommendation against</td>
</tr>
<tr>
<td>c. Reassessment of treatment response (Statement #6a)</td>
<td>Recommendation</td>
</tr>
<tr>
<td>d. Evaluation of treatment failure (Statement #6b)</td>
<td>Recommendation</td>
</tr>
<tr>
<td>e. Education (Statement #7)</td>
<td>Recommendation</td>
</tr>
</tbody>
</table>
Vestibular Neuronitis/ Labyrinthitis

- Acute, prolonged attack of severe vertigo
- Second most common cause of vertigo
- 10% of vertigo diagnosis seen of all patients presenting for dizziness
- 30-50 years of age; ♀ = ♂

Risk factors:
- None well studied
Sudden, persistent, severe
Lasts days (weeks)
Clinical picture: Miserable looking patient with a basin in their arms, moves slow for fear of motion triggered vertigo, eyes closed (sunglasses on).

Nystagmus:
- Unidirectional & horizontal +/- torsional
- Fast phase to NON-AFFECTED ear (unlike BPPV)
- IMPORTANT → Improves with visual fixation
How do I check for fixation...

- When I don’t own a pair of Frenzel glasses?
- Can I try Google® glasses?

ANSWER: Blank paper test
- Viral infection 8th CN
- Increased incidence during viral epidemics
- HSV 1

Fig. 14. Vestibular nerve and ganglion excised from a patient with BPPV and VN who lost hearing following posterior canal occlusion. Viral particles (arrows) enclosed in transport vesicles were found in cytoplasm of ganglion cells. Magnification x46,000.
Diagnosis

- Clinically - **MAINLY**
- Head –thrust test
- Other:
  - Vestibular testing
  - HINTS
  - Imaging
The normal state
Head movement towards a canal (yellow in figure) will cause activation of that canal, and reflex movement of the eyes in the opposite direction - that is, away from the canal.

The pathological state and the basis of the head thrust test
Head movement towards a defunct canal (blue in figure) will result in failure of activation of the vestibular ocular reflex and thus the visual target will be lost from fixation during sudden head movements. In the head thrust test, the examiner turns the patient’s head with a high acceleration but low amplitude head thrust, in this case to the patient’s left. The test is positive when the patient makes a catch-up saccade to re-fixate the visual target (usually towards the examiner’s nose).
<table>
<thead>
<tr>
<th>Table 3</th>
<th>Red flags for a central cause</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History</strong></td>
<td></td>
</tr>
<tr>
<td>Sudden onset</td>
<td></td>
</tr>
<tr>
<td>New, severe headache</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular risk factors</td>
<td></td>
</tr>
<tr>
<td><strong>Nystagmus</strong></td>
<td></td>
</tr>
<tr>
<td>Direction-changing</td>
<td></td>
</tr>
<tr>
<td>Purely vertical or torsional</td>
<td></td>
</tr>
<tr>
<td>Unsuppressed by visual fixation</td>
<td></td>
</tr>
<tr>
<td>Inability to walk</td>
<td></td>
</tr>
<tr>
<td><strong>Negative head-thrust test</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Additional neurologic signs (e.g. aphasia, dysarthria, weakness, sensory loss)</strong></td>
<td></td>
</tr>
</tbody>
</table>
VN/L: Treatment: What’s the goal?

- ANSWER: Purely SYMPTOMATIC
  - Vestibular suppressants (refer to later section)
    - ALL KINDS
  - Vestibular Rehabilitation
Meniere’s Disease

- Aural Fullness
- Tinnitus
- Vertigo
- Fluctuating hearing loss
Endolymphatic hydrops
Meniere’s Disease – idiopathic
Meniere’s Syndrome – known etiology
  • Trauma
  • Iatrogenic/Surgery
  • Otic syphilis
  • Autoimmune
Crisis of Tumarkin – sudden fall; erroneous vertical gravity input.

Endolymphatic hydrops:
Crisis of Tumarkin: A sudden unexplained fall without loss of consciousness or vertigo, attributed to abrupt change in otolithic input, resulting in an erroneous vertical gravity reference which, in turn, generates an inappropriate postural adjustment via the vestibulospinal pathway, resulting in a sudden fall. <2% of MD patients
Meniere’s Disease

- Periodic nature makes epidemiology difficult to assess
  - 0.2\% percent prevalence
  - 4^{\text{th}} - 5^{\text{th}} decade; \& > \sigma
  - Genetic preponderance

- Risk factors:
  - Hx of VN
  - Head trauma
  - Syphilitic otitis
  - And....
Inflammatory disease
Immunologic dysfunction
Infection
Vasculopathy
Disturbances in barometric pressure...osmotic pressure...hydrostatic pressure...or perfusion pressure
Essentially, **anything that impairs inner ear homeostasis**, including:
- Stress
- Sleep deprivation
- Poor diet
- Hormonal changes
- Allergies

Fig. 15. Electron microscopic exam of excised vestibular nerve in a patient with MD revealed viral capsids enclosed in transport vesicles (arrows). M = Mitochondria. This 45-year-old female has been previously reported. Magnification x13,000.
If that’s the case, why make a formal diagnosis of MD?

ANSWER: (per AAO-HNS practice guidelines)

1. Guide Treatment
2. Prognosticate
Meniere’s Disease

Clinical variability
- “Auditory dominant” pattern = HL>>V
- “Vestibular dominant” pattern = V>>HL
- “Mixed” pattern HL=V

Stages:
- Stage I –
  - predominant symptom is episodic vertigo, associated with nausea and vomiting.
  - Attacks may last from 20 minutes to several hours.
  - Between attacks, hearing returns to normal
- Stage II –
  - vertigo accompanied by fluctuating hearing loss, usually affecting the lower pitches.
- Stage III
  - hearing loss ceases to fluctuate but worsens
  - attacks of vertigo diminish
Meniere’s Disease: AAO-HNS

Critical Clinical features:
1. Instability
2. Hearing and balance involvement
ANSWER: Please NOTE – THIS IS A DIAGNOSIS OF EXCLUSION, MOSTLY MADE CLINICALLY but the following are available (supplementary):

- Audiogram
- VNG
- ECOG
- Glycerol dehydration test
- MRI
- VEMP

### Audiometry

Stages of Meniere’s Disease


<table>
<thead>
<tr>
<th>Stage</th>
<th>PTA(^4) (500, 1k, 2k, 3k Hz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt; 25 dB</td>
</tr>
<tr>
<td>2</td>
<td>26 – 40 dB</td>
</tr>
<tr>
<td>3</td>
<td>41 – 70 dB</td>
</tr>
<tr>
<td>4</td>
<td>&gt; 70 dB</td>
</tr>
</tbody>
</table>
25% MD pt’s normal
- Dobie et al., 1982

Weakness found in about 50–60% of MD pt’s
- Oosterveld, 1981
- Meyerhoffetal, 1981
- Pfultz & Malef, 1981
- Dobie et al., 1982

Greatest for establishing laterality
Electrocochleography (ECOG)

- Variant of brainstem audio evoked response (ABR)
  - Examination of wave 1

- Cochlear potentials
  - Resting endolymphatic potential of + 80 mV is present in a normal cochlea.
  - There are at least 3 other potentials generated upon cochlear stimulation:
    - Cochlear microphonic (CM)
    - Summating Potential (SP)
    - Action Potential (AP)
ECOG

- **Cochlear microphonic (CM):**
  - Alternating current (AC) voltage that mirrors the waveform of the acoustic stimulus.
  - OHCs of the organ of corti
  - Proportional to the displacement of the basilar membrane

- **Summating potential (SP)**
  - Direct current (DC) voltage response of the hair cells as they move in conjunction with the basilar membrane
  - Stimulus-related potential of the cochlea

- **Auditory nerve action potential (AP)**
  - Most widely studied component in ECOG.
  - Summed response of the synchronous firing of the nerve fibers.
Meniere’s Disease & ECOG

- Changes in SP seen in MD
  - nonlinear response in Reissner's membrane caused by elevated endolymphatic pressure and distension

- ECOG results are reported as an SP/AP ratio.
  - $\text{SP/\text{AP} > 0.41 c/w MD}$

- Chung et al 2004:
  - SN 71%
  - SP 96%
ECOG: Is It Clinically Useful?

- Nguyen et al. 2010 - Clinical Utility of Electrocochleography in the Diagnosis and Management of Meniere's Disease: AOS and ANS Membership Survey Data

- 143 /344 possible respondents (41.6%) – AOS/ANS members

| Table 1 |

<table>
<thead>
<tr>
<th>Results of agree/disagree items</th>
<th>Agree</th>
<th>Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>A diagnosis of hydrops cannot be made unless the ECoG is abnormal.</td>
<td>3.6%</td>
<td>96.4%</td>
</tr>
<tr>
<td>An abnormal ECoG in the opposite, asymptomatic ear indicates bilateral Meniere’s disease.</td>
<td>23.9%</td>
<td>76.1%</td>
</tr>
<tr>
<td>Abnormal ECoG is a requirement before ablative therapy.</td>
<td>8.6%</td>
<td>91.4%</td>
</tr>
<tr>
<td>Because abnormal ECoG results have been documented in normal research subjects, the test has indeterminate value.</td>
<td>73.2%</td>
<td>26.8%</td>
</tr>
<tr>
<td>ECoG findings fluctuate with the activity of the disorder.</td>
<td>77.9%</td>
<td>22.1%</td>
</tr>
<tr>
<td>When the ECoG reverts to normal, one can predict remission of symptoms.</td>
<td>18.0%</td>
<td>82.0%</td>
</tr>
<tr>
<td>I discount a result that is contradictory to my clinical</td>
<td>82.6%</td>
<td>17.4%</td>
</tr>
</tbody>
</table>
ECOG: Is It Clinically Useful?

![Bar Chart](image)

- ENG/Calories: 57
- VEMP: 27
- Other: 26
- History: 17
- Audio: 13
- CHAMP: 5
- I don't know: 3
Glycerol dehydration test

- First proposed by Klockhoff & Lindblom in 1966 for MD
- Assumes endolympathic volume increase

Dehydration Tests

- Standard Glycerol Test
  - Sensitivity: reported from 47% to 60%
    - highly sensitive to psychological factors of pt
  - Specificity: reported near 100%
    (Snyder, 1974; Klockhoff, 1976; Akiok et al., 1990)
  - Threshold shifts not seen in advanced non-fluctuating HL though discrim may change
  - Greatest uses may be:
    - atypical MD presentations
    - prediction of successful:
      - diuretic Tx
      - surgery
Glycerol dehydration test

Dehydration Tests

- Standard Glycerol test:
  - Loading dose:
    - 1.5 g/k
    - Mix with equal vol. juice
  - Baseline audiogram
  - Serial audiograms every hour x 3 hrs (total 4 audiograms)
    - Max effect: 2-3 hrs
- Interpretation
  - Positive test:
    - 16% word discrim
    - OR
    - At 3 consecutive freq’s a total of combined 25 dB improvement
Purpose: To determine if the saccule and inferior vestibular nerve and central connections are intact and working normally

VEMP = Muscle EMG
**VEMP: Does it help?**

**ANSWER:** *Controversial but it might.*

- “Low amplitude of VEMPs may be found in the affected ear” - Waele, 1999
- “…a substantial proportion of subjects show no VEMP, or a higher threshold” - Rauch et al, 2004.
- “VEMP amplitudes can be increased in early Meniere's disease, as well as fluctuate oppositely to hearing, perhaps due to saccular dilatation” - Young et al, 2002
- Has been proposed that VEMPs that increase on glycerol loading or furosemide injection are suggestive of Meniere's disease - Shojaku et al, 2002 & Seo et al, 2003
Treatment: *What is the goal?*

**ANSWER:** Restoring homeostasis

- **Meniere’s ear has lost regulatory control**
  - Sodium/electrolytes level
    - Diuretic
  - Fluid levels
    - Caffeine & Alcohol
    - Hydration

- **Other challenges to homeostasis:**
  - Stress
  - Hormonal changes
  - Sleep deprivation
  - Barometric changes
  - Allergies
  - Medical co-morbidities
Diet/Lifestyle:
NAS diet/salt restricted diet (1,500-2,000 mg per day)
Single dose per day ETOH or caffeine and limiting chocolate
Exercise, regular sleep, regular daily routine.
Allergy control/Immunotherapy
Vestibular rehabilitation

Medical therapy:
Diuretics
Vestibular Suppressants

Minimally invasive: Meniett pump
OR
Non-ablative:
Intratympanic steroids, ESS

Ablative:
- Intratympanic gentamycin
- Vestibular neuronectomy
- Labyrinthectomy

66% CR (have failed diet/lifestyle)

5-10% of patients; 99% CR
Portable machine that delivers pulses of positive pressure to the middle ear via an ear tube. This theoretically controls sxs by improving endolymphatic drainage. Its evidence toward effectiveness is limited.
Global disturbance of sensory perception arising from abnormal processing of NTS resulting in a broad spectrum of sensory distortions and intensifications.

- Recurrent vertigo +/- Migraine HAs (HA + prodromal sx
...and Meniere’s disease? 

Am I over diagnosing MD?

- 13% gen. pop. have Migraines
  - 25-35% of migraineurs experience vertigo indistinguishable from MD
    = 3.25% gen. pop.

3.25% VM vs. 0.2-0.5% MD = VM 6.5-16.25X more prevalent than MD

TO ADD TO THE CONFUSION → Prevalence of migraine in MD patients is 56% and 85% in bilateral MD patients.
Migraine Treatment

Migraine Vertigo:
1. Migraine Diet (1-2 mo.)
2. Migraine suppressant RXs

Lifestyle
- Sleep
- Regular meals
- Exercise
- General health

Meniere’s Disease
# SUMMARY OF the BIG 4

## Table 1

<table>
<thead>
<tr>
<th></th>
<th>BPPV</th>
<th>Vestibular Neuritis</th>
<th>Vestibular Migraine</th>
<th>Meniere's Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time Course</strong></td>
<td>Recurrent, lasting seconds</td>
<td>Single episode lasting days</td>
<td>Recurrent, lasting minutes to days</td>
<td>Recurrent, lasting hours</td>
</tr>
<tr>
<td><strong>History</strong></td>
<td>Brief, triggered by head movement</td>
<td>Subacute onset of severe, constant vertigo with significant nausea and vomiting</td>
<td>Previous history of migraine. Vertigo accompanied by migraine symptoms</td>
<td>Hearing loss, tinnitus and ear fullness</td>
</tr>
<tr>
<td><strong>Nystagmus</strong></td>
<td>Up-beating torsional</td>
<td>Horizontal or horizontal-torsional</td>
<td>Usually none</td>
<td>Horizontal or horizontal-torsional</td>
</tr>
<tr>
<td><strong>Gait</strong></td>
<td>Normal</td>
<td>Veers toward affected side</td>
<td>Abnormal during vertigo attacks</td>
<td>May have impaired gait and imbalance</td>
</tr>
<tr>
<td><strong>Auditory Symptoms</strong></td>
<td>None</td>
<td>Hearing loss (acute labyrinthitis)</td>
<td>None</td>
<td>Present</td>
</tr>
<tr>
<td><strong>Diagnostic Findings</strong></td>
<td>Positive Dix-Hallpike maneuver</td>
<td>Positive head-thrust test, Nystagmus suppressed by visual fixation</td>
<td>Vertigo attacks resolve with acute migraine medications</td>
<td>Repeat audiometry shows fluctuating, low-frequency hearing loss</td>
</tr>
</tbody>
</table>
What can I give my patient medically to help with vertigo symptoms besides Meclizine?
Pharmacotherapy

ANSWER:

- Antihistamines
- Histaminergics
- Anticholinergics
- Dopamine blockers
- Benzodiazepines
- Calcium antagonists
- Anti-convulsants
- Sympathomimetics
Refer to handout; This enhances a practitioner’s armamentarium for treating vertigo medically/symptomatically.
I can comment a little bit on that. Susan was referring to the 1995 guidelines for the diagnosis of Maniere’s, but right now we’re actually trying to revise that. One of the important factors is to try to determine whether it’s migraine or Meniere’s disease. One important point that’s going to change is that Meniere’s Disease does not extend beyond twelve hours. Migraine does, oftentimes two or three days. So that’s one thing that’s going to be added to the new guidelines. And then you’re right about that most of the people are actually migraine rather than Meniere’s Disease. The other important thing is that the International Headache Association does not want to admit that migraine and vertigo are related.

About the testing, one of those we’re trying to include in the guidelines, but the problem is that not every facility has those, and there’s not a clear cut test that will actually be sensitive enough.
Sources

- Wipperman, J. Dizziness and Vertigo. Prim Care Clin Office Practice; 2014; 41:115-131