Dizziness:
An Otoneurologist’s Approach

John G. Oas, MD
Associate Professor-Clinical
Department of Neurology
Otoneurology Division
The Ohio State University’s Wexner Medical Center

Learning Objectives

1. Discuss two common vestibular disorders that cause dizziness
2. Learn how modern neurovestibular testing can identify vestibular disorders and direct the treatment of dizziness

Vestibular System Anatomy

Image from Wikipedia: http://www.wikipedia.org
Vestibular System Anatomy

Sophisticated Peripheral Vestibular Sense Organs
- Otolith Organs: sacculus and utriculus
- Semicircular Canals: superior, posterior, lateral

Cochleovestibular Nerve (CN VIII)
- Vestibular (Scarpa’s) ganglion (superior and inferior)
- Cochlear nerve is quite separate but adjacent
- Shares space with the facial nerve (CN VII) in the internal auditory canal and cerebellopontine angle

Otolith Organs

Sensors of gravity and head accelerations

Confined within a sac – utriculus, sacculus
Where otoconia (ear stones) are made, held, and resorbed
Maculae (otolith membranes) act as gravity sensors and a translational head accelerometers with 3-D resolution
Utriculus is the source of the wayward otoconia that cause benign paroxysmal positional vertigo (BPPV)
Differential Diagnosis: Dizziness

- Otogenic (inner ear – trauma, infection, toxicity)
- Cervicogenic (altered upper cervical spine biomechanics)
- Neurogenic (stroke, cerebral neoplasia, migraine)
- Neurocardiogenic (Dr. Rhodes to review)
- Psychogenic (psychophysiologic, phobic, hypervigilance)

Vestibular System Function

- Maintains clear vision during all head movements using the vestibuloocular reflexes
- Determines head position, speed and direction of movements
- Generates postural adjustments/reflexes to maintain balance
- Provides spatial orientation information necessary for coördination/locomotion

Vestibuloocular Reflex (VOR)

Head movement creates an eye movement that is equal and opposite in order to achieve gaze stabilization

Vestibuloocular Reflex (VOR)

Keeps vision clear and stable during locomotion

Image from Wikipedia: http://www.wikipedia.org
History of Symptoms

Nothing replaces a history chronologically defined
The more unique their description, the less error in diagnosis (clinical correlation)
Inquire about associated hearing, headache, neck issues
Ask about any similar illness in family
Time invested here is precious but challenging in these times

Two Common Vestibular Disorders

Residual dizziness due to incomplete recovery or permanent loss after a bout of vestibular ganglionitis
Otolith dysfunction or cervicogenic dizziness residual after a bout of benign paroxysmal positional vertigo (BPPV)

Case 1

54 year old farmer with vertigo goes to the local ER on day 1
You see him in the office on day 2: Valacyclovir days 2-12 (zoster oticus protocol); tapering course of methylprednisolone days 2-23 (NEJM protocol)
Day 24: still ‘dizzy’
What do you say?
What’s your next move?

Vestibular Ganglionitis

Dramatic vertigo that continues beyond 24 hours
Acute care – use Zoster doses of valacyclovir, acyclovir, or famciclovir (if less than 48 hours), rehydration, antiemetics, and vestibular suppressants (no longer than 9 days)
Consider pulse of corticosteroids (if less than 72 hours after onset) cautiously (NEJM protocol)
Caused by reactivation of the alpha-HHV family (herpes simplex, varicella zoster) dwelling in the vestibular ganglia
## Incomplete Recovery: peripheral vestibular system loss/dysfunction

<table>
<thead>
<tr>
<th>The vertigo subsides but the dizzy symptoms persist</th>
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</thead>
<tbody>
<tr>
<td>Head movements exacerbate the dizzy sensation</td>
</tr>
<tr>
<td>Accompanying imbalance</td>
</tr>
<tr>
<td>Vestibular suppressants do not work (treat only motion sickness)</td>
</tr>
<tr>
<td>When avoidance becomes the behavior, look out!</td>
</tr>
</tbody>
</table>

## Benign Paroxysmal Positional Vertigo (BPPV)

<table>
<thead>
<tr>
<th>Positional vertigo (usually on arising or turning over in bed) that lasts only seconds to a few minutes</th>
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</thead>
<tbody>
<tr>
<td>If it persists for days or weeks it's not so benign</td>
</tr>
<tr>
<td>Use vestibular suppressants for no longer than 9 days</td>
</tr>
<tr>
<td>Gentle forms of self-repositioning techniques</td>
</tr>
<tr>
<td>Consider referrals to physiotherapists for repositioning protocols when persists for more than a few days</td>
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</tbody>
</table>

## Case 2

<table>
<thead>
<tr>
<th>69 year old retired teacher awakens with vertigo, goes to the local ER on day 1</th>
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</thead>
<tbody>
<tr>
<td>You see her in the office on day 2: document the nystagmus of BPPV on Dix-Hallpike positioning</td>
</tr>
<tr>
<td>Try your hand at repositioning; or hand out self-repositioning exercises; or refer to a local PT for particle repositioning therapy</td>
</tr>
<tr>
<td>Day 24: vertigo is gone but still ‘dizzy’ Dix-Hallpike positioning does not provoke vertigo but makes her dizziness worse</td>
</tr>
<tr>
<td>What do you say? What’s your next move?</td>
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</tbody>
</table>

## Cervicogenic dizziness

<table>
<thead>
<tr>
<th>Long-term complication of vestibular ganglionitis or BPPV</th>
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</thead>
<tbody>
<tr>
<td>Enigmatic, refractive, frustrating, persists for months/years</td>
</tr>
<tr>
<td>Cervicogenic or tension-type headache comorbidity</td>
</tr>
<tr>
<td>Neurovestibular testing (OSU) helps define the problem quite well (older 1960's-style testing often misses the cause)</td>
</tr>
<tr>
<td>Needs special rehabilitation – not all physiotherapists are trained to treat this disorder</td>
</tr>
</tbody>
</table>
**Otolith loss/dysfunction**

- Long-term complication of vestibular ganglionitis or BPPV
- Gravity sense becomes distorted: imbalance occurs with movement
- Head position changes: tilts (causes a biomechanical stress to the upper cervical spine)
- Frequent cause of vestibular physical therapy failure
- Requires sophisticated physiotherapy, not medication

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**Circa 1962**

Image from Wikipedia: http://www.wikipedia.org

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**Circa 1969**

Image from Wikipedia: http://www.wikipedia.org

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**Neurovestibular Testing at OSU**

Uses technology developed after the 1960s
Test facility located at OSU CarePoint Gahanna
Comprehensive testing – both otogenic and precise neurophysiological testing designed and interpreted by an Otoneurologist
Allows for otolith testing
Eye Movement Tracking

Done in total darkness (infrared illumination)

Why do Neurovestibular testing?

- When the diagnosis is in question
- Defining a course of treatment
- Ruling out vestibular disorders in complicated cases
- Helps define complex cases
- Provides triage for further investigations (neuroimaging studies, Otoneurology consultation)

Neurovestibular Testing at OSU

Important Points

- The vertigo from a bout of vestibular ganglionitis abates over time
- Dizziness that persists after vertigo abates is still a vestibular disorder
- It is not always possible to differentiate an otogenic source from others (cervicogenic, neurogenic, neurocardiogenic, psychogenic) based on the history alone (refer for testing)
### Important Points

| BPPV is defined by brief vertigo, triggered by gravitational forces that act upon the ear with head position changes. | Dizziness after BPPV is either cervicogenic dizziness or otolith dysfunction. |
| Complicating neurological issues can evade neuroimaging studies and only be evident with careful (neurovestibular) testing. |  |

### Learning Objectives

- Review the etiologies of syncope
- Discuss the cardiac evaluation of syncope
- Discuss the evaluation and treatment of vasovagal syncope
- Discuss the approach to syncope following a negative evaluation

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### Dizziness

**Cardiac Electrophysiologist’s Approach**

| Troy E. Rhodes, MD, PhD |
| Assistant Professor of Internal Medicine |
| Division of Cardiovascular Medicine, Electrophysiology |
| The Ohio State University’s Wexner Medical Center |

### Presyncope

Prodromal state of fainting or a near faint; may be associated with lightheadedness, visual blurring, warmth, diaphoresis, and nausea.
Syncope

Abrupt and transient loss of consciousness associated with loss of postural tone, followed by complete and spontaneous recovery

Causes of Syncope

A prospective study of 341 patients found the following causes:
- Reflex -- neurally, vasovagal mediated – 58%
- Cardiac disease, most often a brady or tachyarrhythmia – 23%
- Neurologic or psychiatric disease – 1%
- Unexplained syncope – 18%


Syncope

Common Clinical Problem
- Occurs in up to 20% of the population
- Responsible for 3% of all US ED visits
- Benign or only warning prior to SCD
- Injuries in one-third of patients

Causes of Syncope

- Neurocardiogenic syncope
  - Reflex
  - Situational
    - Cough syncope
    - Swallow syncope
    - Micturation syncope
    - Defecation syncope
    - Syncope associated with pain
    - Carotid sinus hypersensitivity

- Mechanical CV Disease
  - Aortic stenosis
  - Mitral stenosis
  - Obstructive cardiomyopathy
  - Atrial myxoma
  - Pulmonary vascular disease
  - Prosthetic valve dysfunction
  - Cerebrovascular and neurologic
    - Vertebrobasilar ischemia
    - Migraine
    - Subclavian steal syndrome
    - Seizure disorders
  - Orthostatic hypotension
  - Hypovolemia
  - Autonomic insufficiency

Arrhythmias
- Sinus node dysfunction
- Atrioventricular block
- Supraventricular tachycardia
- Ventricular tachycardia
### Syncope

| **High Risk** | Structural heart disease  
| | Decreased EF  
| | Conduction disease  
| | Long QT, Brugada  
| | FH of sudden death  
| | Abrupt onset, injury |

| **Low Risk** | Typical VVS prodrome  
| | Multiple episodes  
| | Young age, no heart disease  
| | Orthostatic trigger |

### Initial Evaluation

- History & Physical
- Orthostatics
- Carotid sinus massage
- Screening labs
- ECG
- Echocardiogram

### History

- Prodrome, residual symptoms
- Activity, posture
- Palpitations
- Seizure Activity
- Related Injury
- Prior Episodes
- FH
  - Syncope, Sudden Death, Cardiac Disease

### ECG

- Preexcitation
- Conduction Defects
- Q waves
- LVH
- Repolarization abnormalities
  - LQTS, Brugada Syndrome
**Echocardiogram**

- Excellent for detecting associated cardiac disease
  - LVEF, wall motion abnormalities
  - Valvular disease
  - HCM
- Provides key data affecting prognosis and further evaluation

**Additional EP Testing**

- Tilt table testing
- EP testing
- Implantable loop recorders (ILR)

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<tr>
<th><strong>ECG Monitoring</strong></th>
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<tr>
<td>- Telemetry</td>
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<tr>
<td>- Holter or event monitoring</td>
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<table>
<thead>
<tr>
<th><strong>Neurocardiogenic Syncopal Syndromes</strong></th>
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<tbody>
<tr>
<td>Vasovagal Syncope</td>
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<tr>
<td>Situational Syncope</td>
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<tr>
<td>Carotid Hypersensitivity</td>
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</table>
Vasovagal Syncope

**Setting**
- young patients, no structural HD
- painful, frightening situation
- hunger, fatigue, hot room
- standing position

**Prodrome**
- nausea, blurred vision
- warmth, diaphoresis
- pallor, yawning

**Syncopal Event**
- white, pale
- may be aborted by becoming supine

**Residua**
- nausea, diaphoresis, fatigue

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Tilt Table Testing

- Supine for 5 minutes, obtain baseline HR & BP
- **Passive** head up tilt, 60-70 deg, 20 min+
- HR, BP, symptom monitoring
- Loss of consciousness or postural tone in association with significant fall in BP or HR
- Returned to supine position

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**Tilt Table Test**

![Tilt Table Test Image](www.aafp.org)

**Tilt Table Testing**

- **Provocative** head up tilt
  - Isoproteronol – 1-3 mcg/min to increase HR 20-25%
  - NTG – 300-400 mcg
Provocative TTT

- Isoproterenol
  - Single isuprel stage induced syncope more frequently than standard passive HUT (56% vs 32%) and reduced time with lower specificity
  - Modest decrease in BP in non-specific
  - Contraindicated in pts with severe CAD
- Nitrates
  - May shorten test duration; increases false positives

Reflex Arcs in Neurally Mediated Syncope

Alterations in autonomic activation
- Cardioinhibitory response
  - Increased parasympathetic activation → sinus bradycardia, asystole, AV block
- Vasodepressor response
  - Decreased sympathetic activity → hypotension
- Mixed response
- Serotonin

Neurocardiogenic Syncope

<table>
<thead>
<tr>
<th>Classic Neurocardiogenic (Vasovagal) Response</th>
<th>Dysautonomic Response</th>
</tr>
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<tbody>
<tr>
<td>HR/BP</td>
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</tr>
<tr>
<td>Tilt Head Down</td>
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</tr>
</tbody>
</table>

Treatment of Vasovagal Syncope

- Protective measures
- Lifestyle modifications
  - 4 L per day, >4 g salt per day
  - Avoid caffeine, alcohol, diuretics
- Physical counterpressure
- Tilt training
- Compression stockings
Treatment of Vasovagal Syncope

- Beta-blockers
- Midodrine
- Fludrocortisone
- SSRIs
- Cardiac pacing

Arrhythmias

- Sinus Node Dysfunction
- Atrioventricular Block
- Supraventricular Tachycardia
- Ventricular Tachycardia

Arrhythmias and Syncope

Typical Placement of Diagnostic EP Catheters

http://mykentuckyheart.com
Combined Use of EP and Tilt Table Testing for Syncope

<table>
<thead>
<tr>
<th>Unexplained Syncope (86pts)</th>
<th>+ EPS 34%</th>
<th>- EPS 66%</th>
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</thead>
<tbody>
<tr>
<td>Tachyarrhythmia</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>21 VT, 5 SVT</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Bradyarrhythmia</td>
<td>+HUT</td>
<td>-HUT</td>
</tr>
<tr>
<td>1 SSS, 2 AVB</td>
<td>60%</td>
<td>40%</td>
</tr>
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74% of pts were diagnosed with the combined use of EPS and Tilt Testing


Undiagnosed Syncope

Further workup

- Neuro: EEG / MRI - seizure
- Vascular: Angiography - VBI / drop attacks
- Psych: Tilt with EEG - conversion rxn
- Cardio: Loop recorder - external / implantable

Summary

- History, ECG, Echo
- Vasovagal syncope most common cause
- Tilt table testing → EPS
- + EPS → Device therapy
- Negative work-up → ILR