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

The Ear

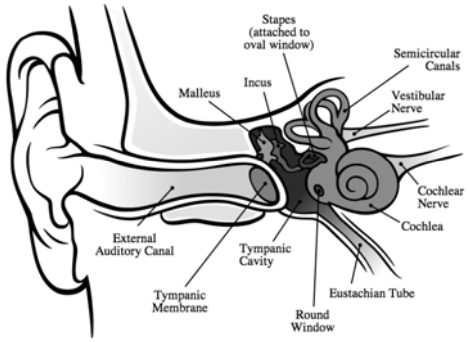


Image from Wikipedia: http://www.wikipedia.org

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

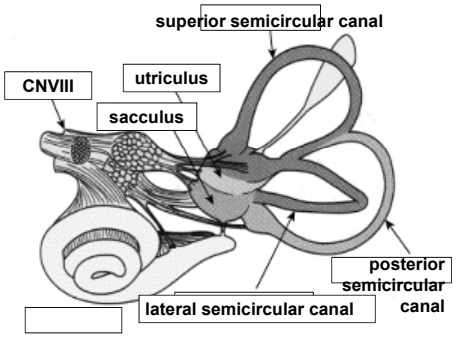


Image from Wikipedia: http://www.wikipedia.org

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

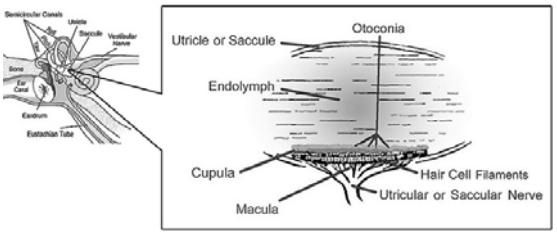
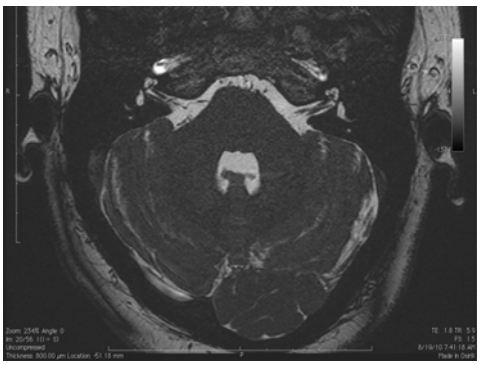


Image from Wikipedia: http://www.wikipedia.org

Vestibular System Anatomy



Otolith Organs

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)

Vestibuloöcular Reflex (VOR)

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

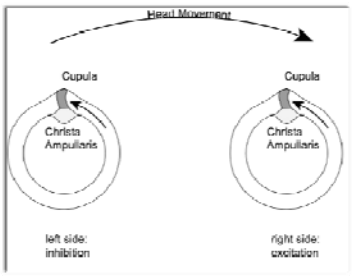


Image from Wikipedia: http://www.wikipedia.org

Vestibular System Function

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

Vestibuloöcular Reflex (VOR)

Keeps vision clear and stable during locomotion

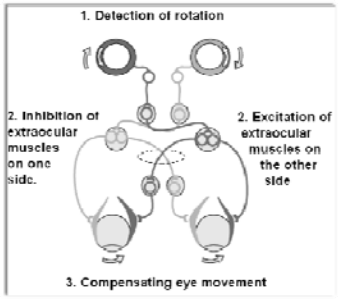


Image from Wikipedia: http://www.wikipedia.org

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

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?

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)

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

The vertigo subsides but the dizzy symptoms persist
Head movements exacerbate the dizzy sensation
Accompanying imbalance
Vestibular suppressants do not work (treat only motion sickness)
When avoidance becomes the behavior, look out!

Benign Paroxysmal Positional Vertigo (BPPV)

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

Case 2

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

Cervicogenic dizziness

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

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

Circa 1969

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

Circa 1962

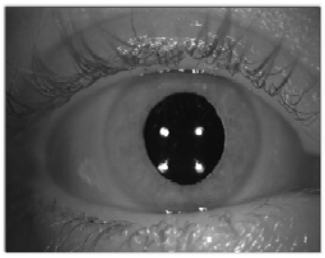
Image from Wikipedia: http://www.wikipedia.org

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

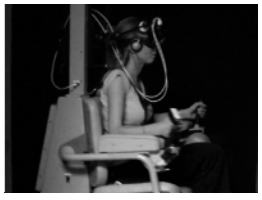
Defining a course of treatment

Ruling out vestibular disorders in complicated cases

Helps define complex cases

Provides triage for further investigations (neuroimaging studies, Otoneurology consultation)

Neurovestibular Testing at OSU



Used with permission from http://www.neuro-kinetics.com

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) 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

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%**

Alboni et al. *J Am Coll Cardiol*. 2001;37(7):1921.

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

Neuroautonomic regulation

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

Arrhythmias

- Sinus node dysfunction
- Atrioventricular block
- Supraventricular tachycardia
- Ventricular tachycardia

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

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

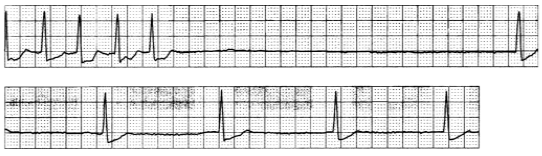
- Excellent for detecting associated cardiac disease
  - LVEF, wall motion abnormalities
  - Valvular disease
  - HCM
- Provides key data affecting prognosis and further evaluation

Additional EP Testing

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

ECG Monitoring

- Telemetry
- Holter or event monitoring



Neurocardiogenic Syncopal Syndromes

Vasovagal Syncope

Situational Syncope

Carotid Hypersensitivity

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

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**

Tilt Table Test

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

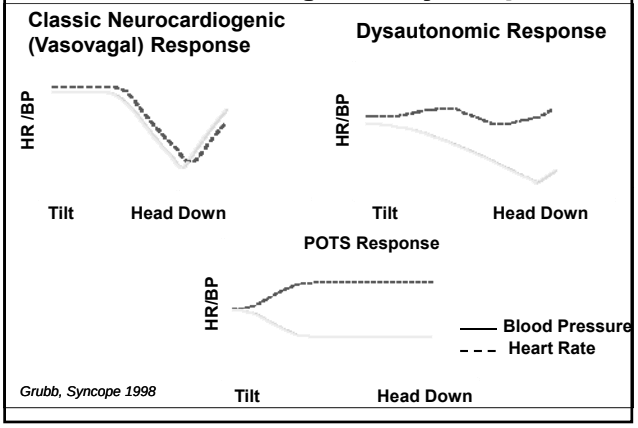
- Isoproteronol
  - Single isuprel stage induced syncope more frequently than standard passive HUT (56% vs 32%) and reduced time with lower specificity
  - Modest decrease is BP in non-specific
  - Contraindicated in pts with severe CAD
- Nitrates
  - May shorten test duration; increases false positives

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



Grubb, Syncope 1998

Treatment of Vasovagal Syncope

- Protective measures
- Lifestyle modfications
  - 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

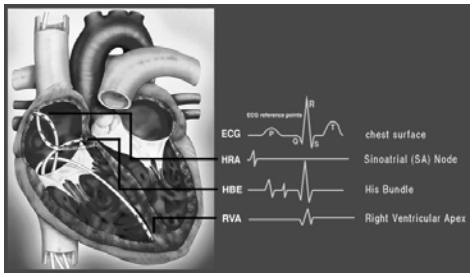
Atrioventricular Block

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

Unexplained Syncope (86pts)

+ EPS 34% | **- EPS 66%**

Tachyarrhythmia	Bradyarrhythmia	+HUT	-HUT
21 VT, 5 SVT	1 SSS, 2 AVB	60%	40%

74% of pts were diagnosed with the combined use of EPS and Tilt Testing

Sra et al. *Ann Intern Med.* 1991; 114(12):1013-9.

Reveal® Plus Insertable Loop Recorder

Medtronic

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