Differential Diagnosis of Dizziness using Vestibular and Cerebellar Tests

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Objectives

- List signs and symptoms of vestibular dysfunction
- Name 3 Cerebellar bedside tests
- List at least 3 central signs of dysfunction
- Identify the 3 parts to the H.I.N.T.S. examination

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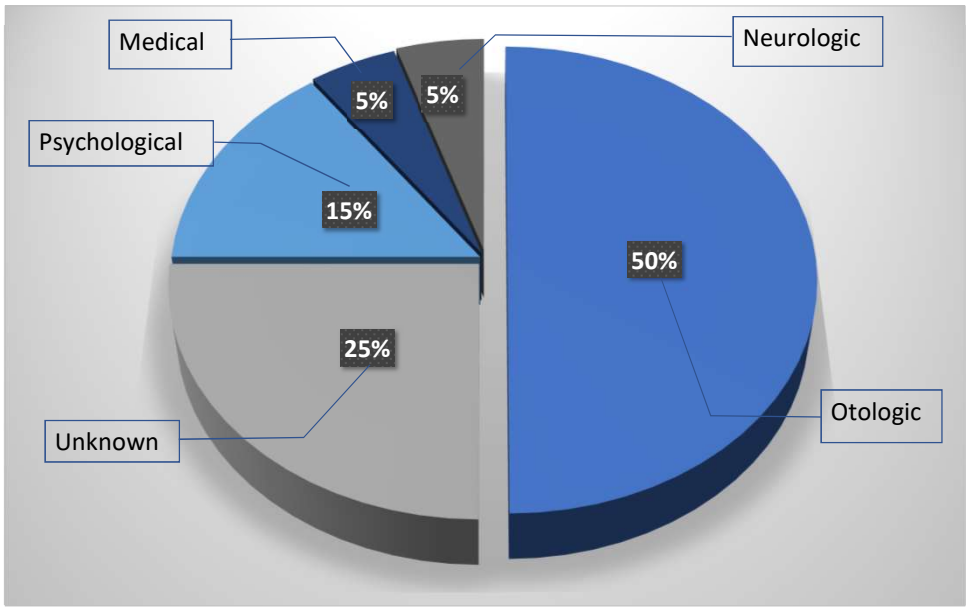
Causes of Dizziness



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Cause of Dizziness	Each Category	Examples
Otologic	50 %	BPPV, Meniere's disease, Vestibular Neuritis/Labyrinthitis, Perilymph Fistula, Bilateral Vestibular Loss, Acoustic Neuroma
"Central" Neurologic	5 %	Stroke, Migraine, Seizures, MS, Cerebellar degeneration, Chiari malformation, Other Cerebellar disorders, Mal de Debarquement, White matter disorders
Medical	5 %	Low BP, Orthostatic Hypotension, Cardiac arrhythmia, Medication side effects
Psychological	15 %	Anxiety, Panic disorder, Malingering, Phobias, Somatization syndrome, Chronic Subjective Dizziness, Phobic positional vertigo
Unknown	25 %	Multisensory disequilibrium of the elderly, Post-traumatic dizziness, Psychogenic dizziness (when used as Dx of exclusion)

Hain, TC. Outline of Causes of Dizziness, Imbalance and Hearing Disorders. *Dizziness-and-Balance.com. Last Updated* 2012

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Why worry about the 5% neurologic?

… Because they are potentially life threatening!

5

Time to consider sinister disorders…

The time to consider a sinister disorder as the cause of dizziness is:

1. When the presentation is atypical for a peripheral vestibular disorder, or
2. When other red flags are identified

Kerber K, Vertigo and Dizziness in the Emergency Department. Emerg Med Clin North Am. 2009 February ; 27(1): 39–viii. doi:10.1016/j.emc.2008.09.002.

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Our Job

When examining a patient with a history of falls, dysequilibrium, or dizziness, our job is to:

1. Examine <u>each system</u> contributing to balance
2. Assist in ruling out central pathology
3. Refer to other medical professionals as needed
4. Reduce the likelihood of falls by addressing systems we can affect, recommending assistive devices as needed, and referring to other professionals to address what we can't

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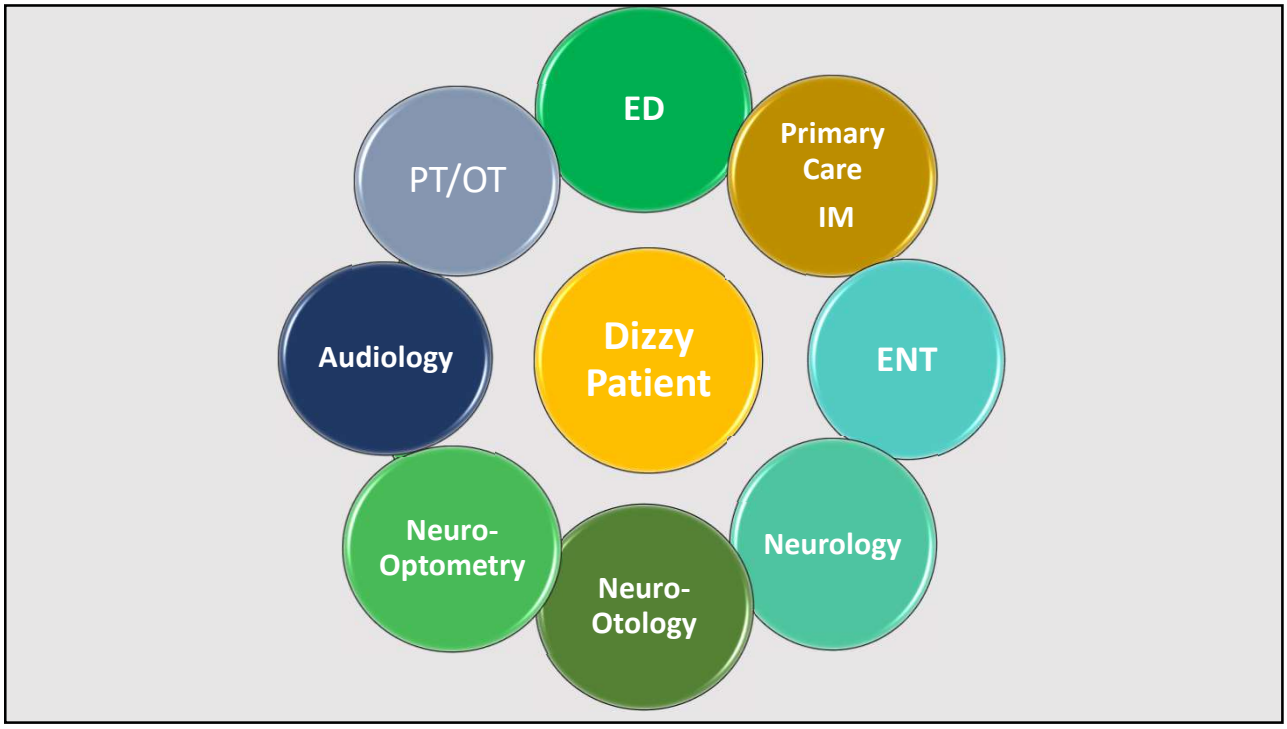

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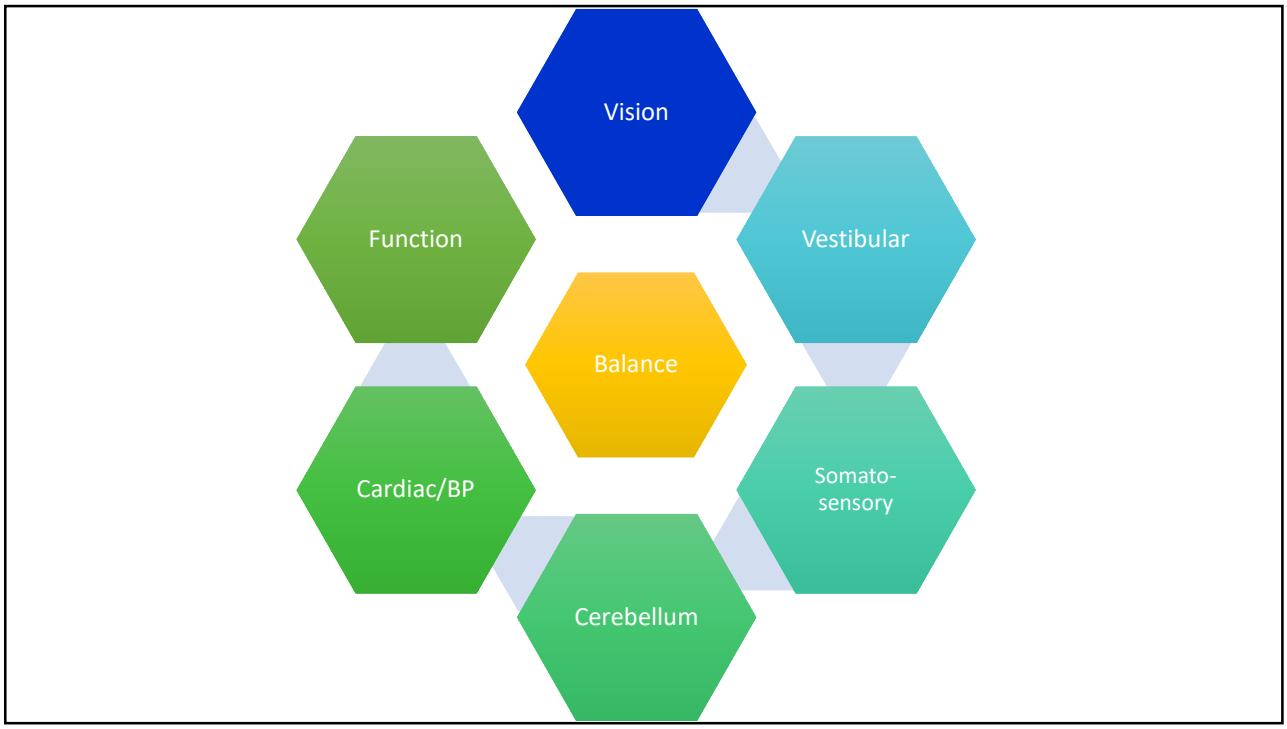

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Statistics – Dizziness

Topic	Statistic/Quote	Source
Vestibular	"From 2001 through 2004, 35.4% of US adults aged 40 years and older (69 million Americans) had vestibular dysfunction."	Agrawal Y, Carey JP, Della Santina CC, Schubert MC, Minor LB. Disorders of balance and vestibular function in US adults. Arch Intern Med. 2009;169(10):938-944.
Dizziness	The prevalence of vertigo and dizziness in people aged more than 60 years reaches 30%, while rising to 50% beyond 85 years	• JonssonR, SixtE,LandahlS, Rosenhall U. Prevalence of dizziness and vertigo in an urban elderly population. J Vestib Res (2004)14:47–52. • BarinK, Dodson EE. Dizziness in the elderly. Otolaryngol Clin North Am (2011) 44:437–54.doi:10.1016/j.otc.2011.01.013
Dizziness	"U.S. doctors reported 5,417,000 patient visits in 1991 because of dizziness or vertigo."	Centers for Disease Control and Prevention. Vital and health statistics, national ambulatory medical care survey: 1991 summary. Washington, DC: National Center for Health Statistics, Public Health Service, US Dept of Health and Human Services; 1994. DHHS publication PHS 94-1777.

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Statistics – Dizziness

Topic	Statistic/Quote	Source
Dizziness	"Disorders of the vestibular system are responsible for 40%-50% of dizziness in patient referred to ENT and Primary Care clinics."	Whitiney S, Alrwaily M. Vestibular Rehabilitation of Older Adults with Dizziness. Otolaryngol Clin North Am. 2011;44:473-96.
BPPV	The most common vestibular disorder is BPPV (1/3 of vestibular diagnosis)	Von Brevern M, 2007; Delminski J, 2010

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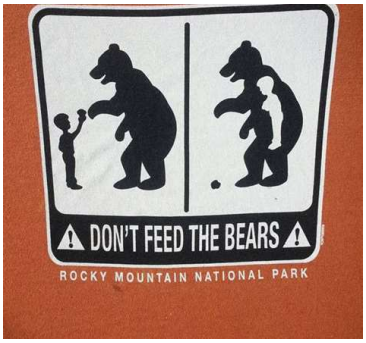

Things we know for certain…

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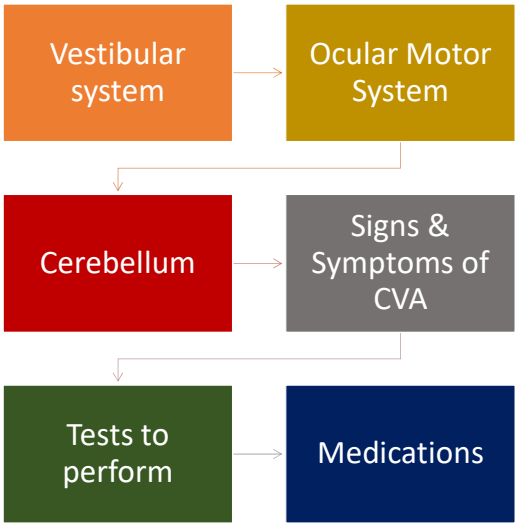
What we need to understand to say what is…

'Likely'



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How we control balance and movement

- **The Frontal Lobe decides** what we want (posture, position, movement)
- **Gather information** from Vision, Vestibular, and Somatosensory systems about our position, motion, and the environment
- **The cerebellum compares** incoming information and decides which systems are most reliable for the current needs. With that information, it coordinates voluntary movements as ordered by the cerebral cortex. It compares our intended movement with actual movements, and makes adjustments as needed.

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Ingredients:

- Oculomotor Exam
- Vestibular Exam
- Somatosensory Exam

Mixer:

- Cerebellum

Pie Pan:

- Musculoskeletal System
- C1-C2 Stability

Taste Test:

Examination of Function

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Useful Terminology

Nystagmus refers to involuntary rhythmic movements of the eyes.

Many Types:

Jerk Nystagmus – Movements alternate between a slow component and a fast corrective component (jerk) in the opposite direction

Pendular Nystagmus – Oscillations are roughly equal in rate in both directions

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The Vestibular System

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Vestibular System Anatomy Functional Roles

Maintains posture & muscle tone
- Senses and perceives position and motion of the head
- Helps to produce muscle contractions to for upright posture

Maintains ocular stability during movement
- Orients the head to vertical
- Produces reflexive eye motions during motion

Maintains equilibrium
- Helps to control the body's 'center of mass'
- Orients the body to vertical

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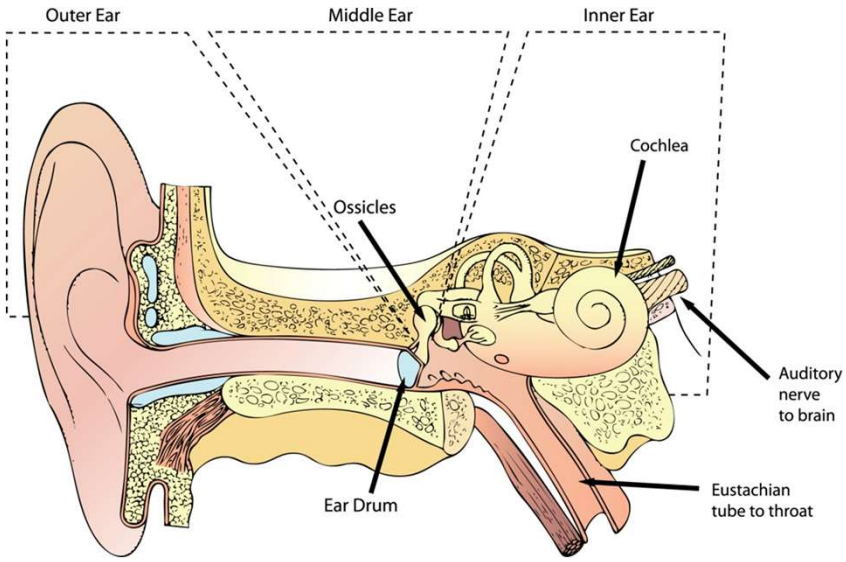
Vestibular System Anatomy

- Found in the Inner Ear next to the Cochlea
- Shares the 8th cranial n. with the Cochlea



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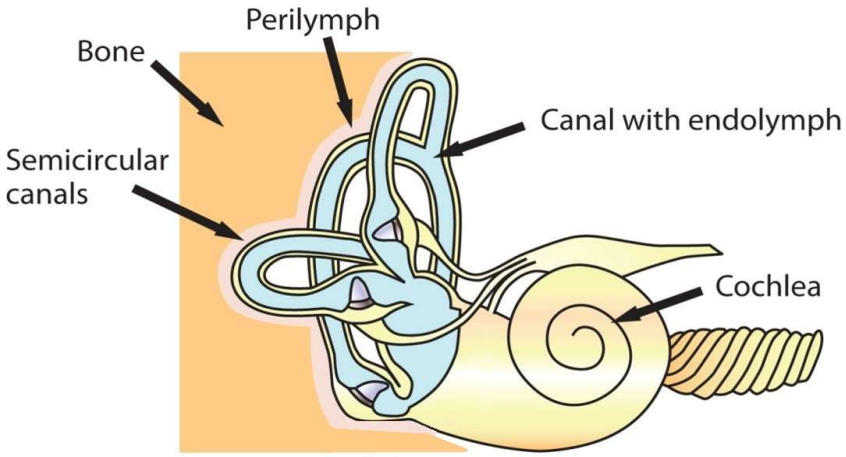
Anatomy: Vestibular System

- Surrounded by bone (3 Bony Labyrinths)
- Membrane (Membranous Labyrinth)
- Floating in fluid (Perilymph)



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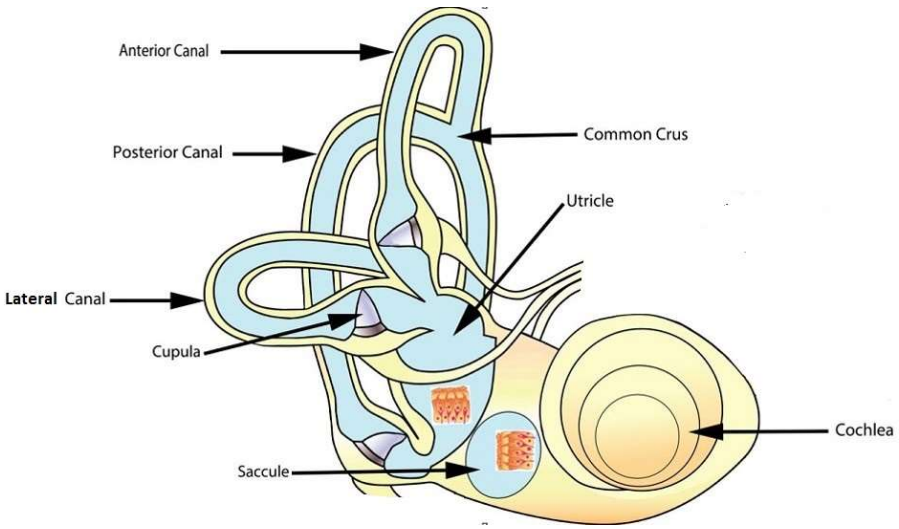
- Two Chambers (Otolith Organs): Utricle & Saccule
- 3 semi-circular canals (Labyrinths)
- Containing fluid (Endolymph)
- Specialized Hair Cells that are attached to nerve cells



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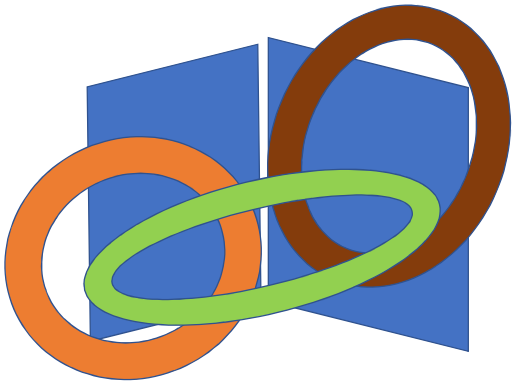
Three semi-circular canals (which originate in the Utricle) named for their orientations:
Anterior
Lateral (Horizontal)
Posterior

Oriented 90° to each other (like the corner of a room)

The anterior portion of the Lateral Canals are pitched up 30 degrees from horizontal

Anterior Canal is superior to (cephalad) to the other canals



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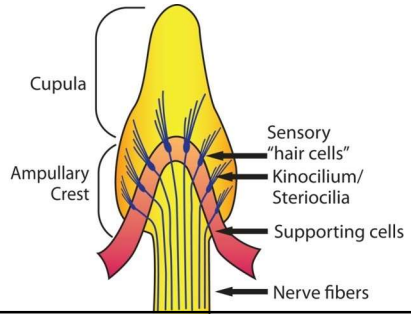
The Canals - Ampullae

- The hair cells in the semicircular canals are located in the ampulla on the Crista Ampullaris
- They stick up into the gelatinous matrix of the cupula
- The cupulae have the same mass of the surrounding fluid and are not effected by gravity



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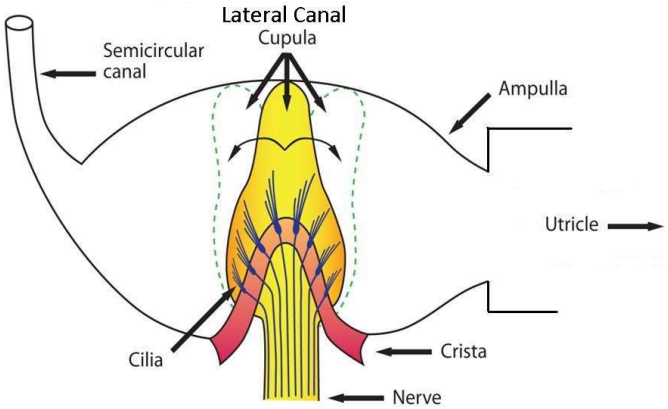
The Vestibular System



- The ends of each of the canals widens to form a bulb called the 'Ampulla' (pl. *Ampullae*) that communicates back to the Utricle
- There are specialized hair cells in each of these bulbs

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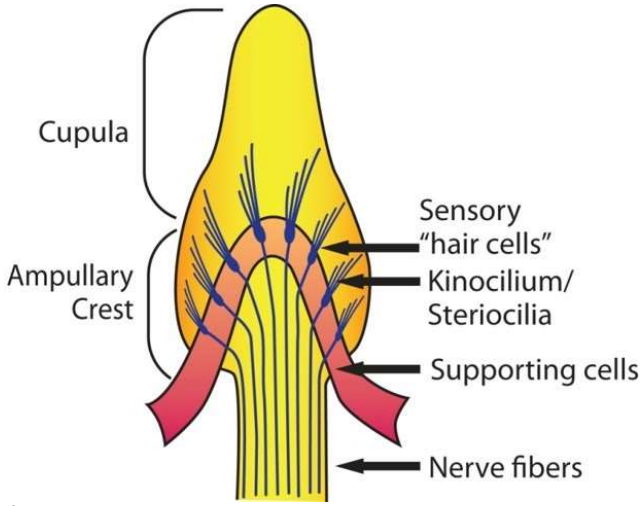
Hair Cells



- Rows of hair cells with the tall Kinocilium, and smaller Stereocilia

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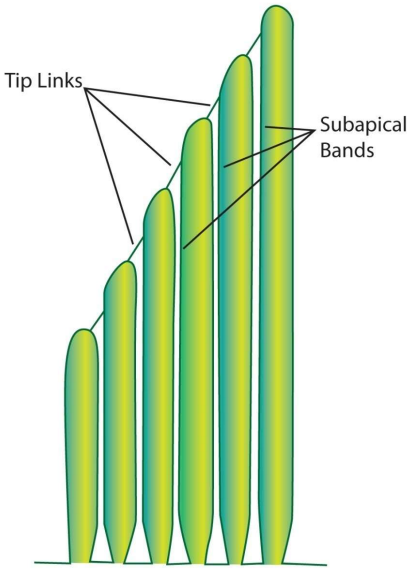
Hair Cells



- Linked together
- Spring-like, prefer resting position
- If one bends, they all bend
- At rest, without movement, there is a steady firing pattern
- Tall Hair named Kinocillium
- All the rest are Stereocilia

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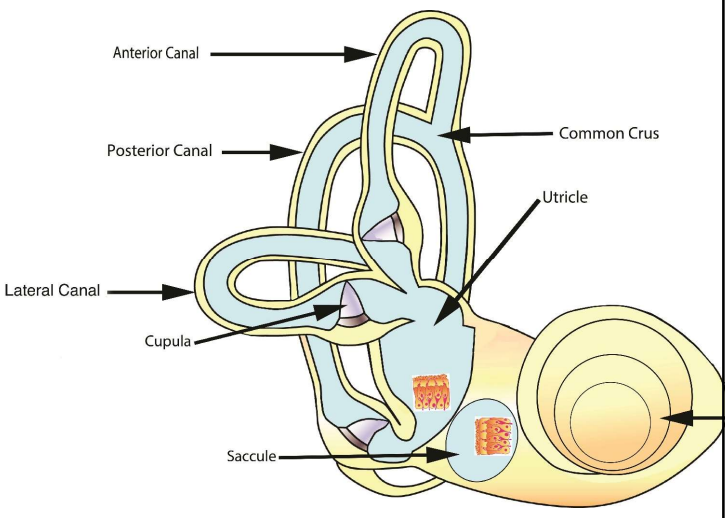
The Canals

- The canals originate in the Utricle
  - Anterior
  - Lateral (Horizontal)
  - Posterior
- Each Canal loops back toward the Utricle
- The canal is completely blocked from the Utricle by a sail-like membrane called the Cupula that is neutrally bouyant



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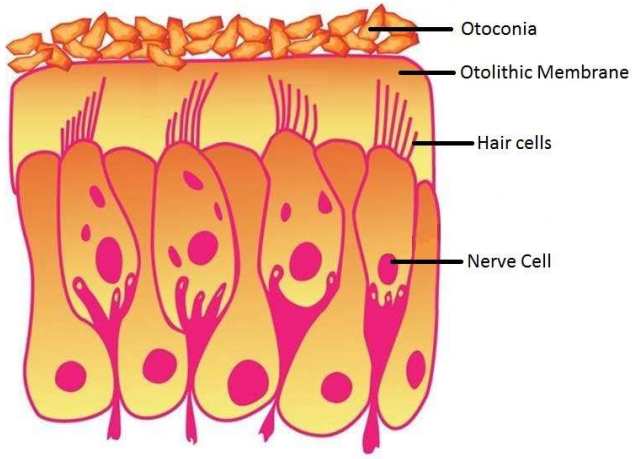
Otolith Organs

Important differences between the Otoliths and the groups of hairs in the canals:

- The matrix does not block off the Utricle or Saccule
- Groups of hair cells do not all face the same way (short to tall)
- The matrix has otoconia (calcium carbonate crystals) held in place on top of it that make the otoliths sensitive to gravity. (when loose may cause BPPV)



Utricle

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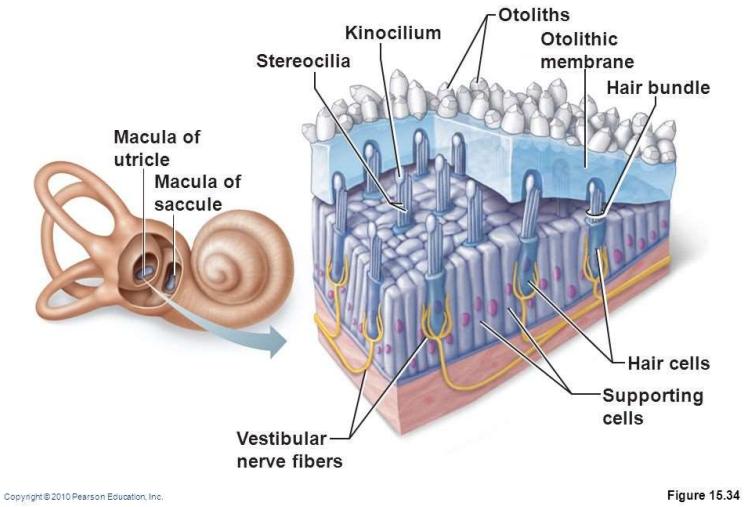

https://fitness.stackexchange.com/questions/34687/why-ear-pain-in-diving

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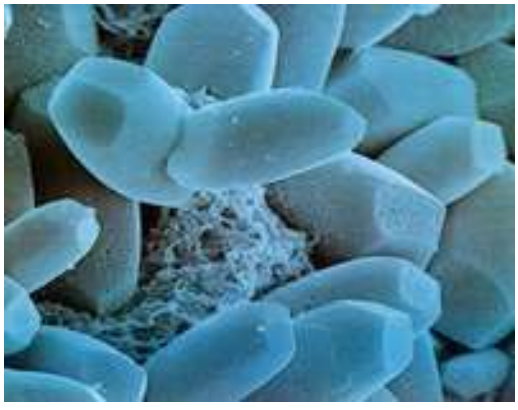
The Otolith Organs

- On top of the Macula rests a gelatinous matrix called the 'Otolithic Membrane'
- Hair cells (Kinocillia and Stereocillia) project from the supporting nerve cells into the Otolithic membrane
- Small Calcium Carbonate crystals called "Otoconia" are glued on top of the membrane, and make the hair cells sensitive to gravity



http://www.med.umn.edu/ent/clinics/otologyneurotology/treatment/benignvertigo/home.html 28 Sept., 2008.

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Anatomy: Otolith Organs

- Utricle: Detects horizontal motions such as accelerating or decelerating in a car or roller coaster

- Saccule: Detects vertical motions, such as moving up or down in an elevator

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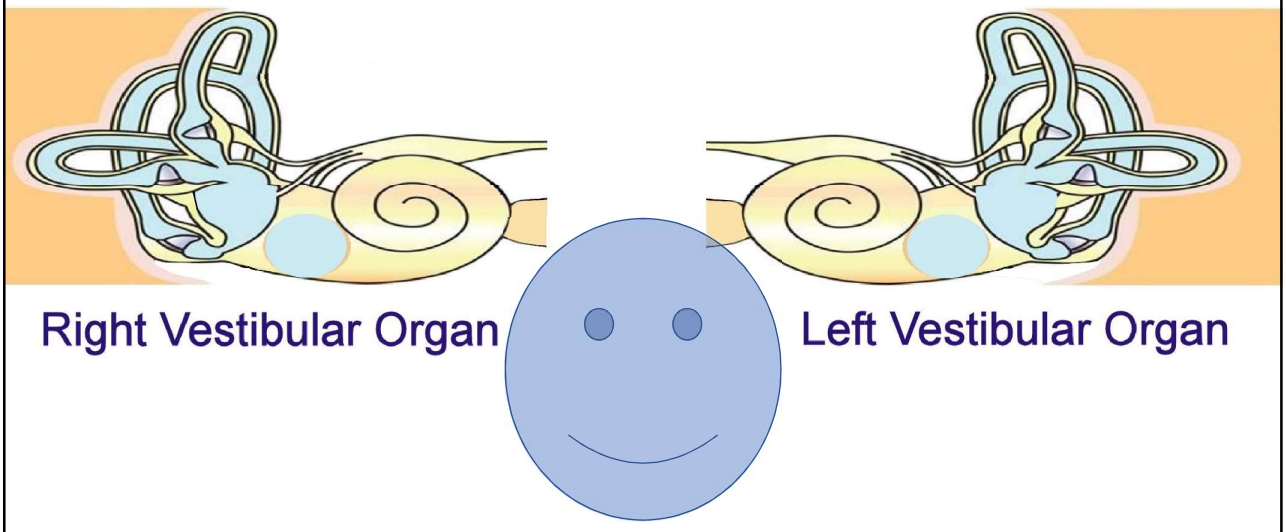

Remember, the peripheral vestibular organ in each ear is a mirror images of the another

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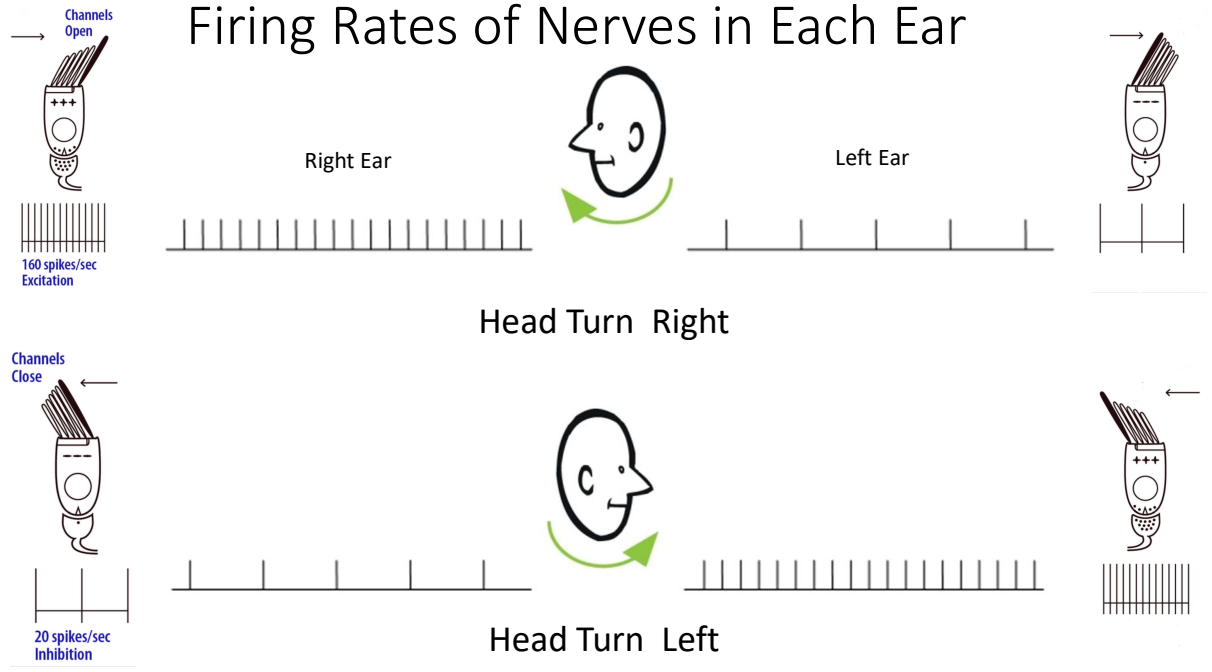
Firing Rates of Nerves in Each Ear



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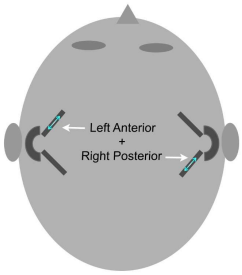
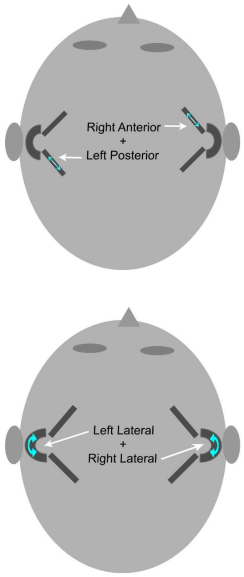
The labyrinth of one ear works with the labyrinth of the other ear that is in the same plane of orientation (called coplanar pairs)

- Right Anterior + Left Posterior (RALP)



- Left Anterior + Right Posterior (LARP)
- Lateral (Horizontal) canals



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The Vestibular System
Interpreting the firing rates

- The brain interprets 'movement' when there is a difference between firing rates

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Vestibular Reflexes

- Vestibular Ocular Reflex (VOR)
- Vestibulospinal Reflex (VSR)
- Vestibulocollic Reflex (VCR)

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Vestibular Reflex	Purpose
VOR	Maintains gaze stability during head motion Vestibular canals work between head frequencies of 0.5 Hz and 5.0 Hz (Best between 1.0 Hz and 5.0 Hz) **Most head motions are between 0.5 Hz and 4 Hz** **VOR Eye motions: 250º-300º/sec** 1 Hz = 360°/sec

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Vestibular Ocular Reflex (VOR)

- The VOR helps us to maintain fixation on a target during head movement
- It is not a sustained reflex. After seconds of constant steady rotation the stimulus for eye movement will die down.
- To be optimal, the VOR needs information from vision (retinal slip), and eye movement information (from oculomotor nuclei, interstitial nucleus of Cajal, and interstitial nucleus of the medial longitudinal fasciculus (MLF) from preoculomotor reticular areas)

http://www.brown.edu/Departments/Engineering/Courses/122JDD//Lcturs/VOR05.html

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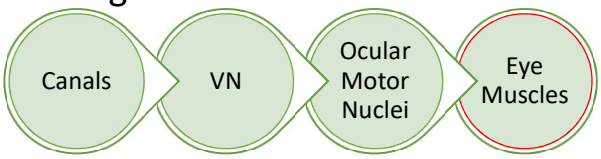
Vestibulo-ocular Reflex (VOR)

- 3 nerve reflex from the peripheral ear to the brainstem to the eye muscles
- Latency of the VOR is ~ 15 msec
- Excitation & inhibition to Medial Rectus and Lateral Rectus on both sides
- For sustained rotations, the VOR is augmented by the Velocity Storage system, and the Optokinetic Reflex, which turns on slower, but doesn't fatigue



http://www.brown.edu/Departments/Engineering/Courses/122JDD//Lcturs/VOR05.html

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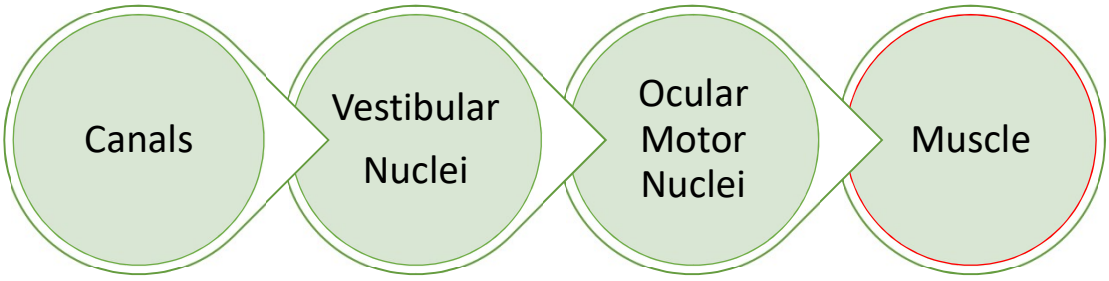
Vestibulo-ocular Reflex (VOR)

- While the cerebellum may influence the VOR, it is not needed to produce the VOR!



http://www.brown.edu/Departments/Engineering/Courses/122JDD//Lcturs/VOR05.html

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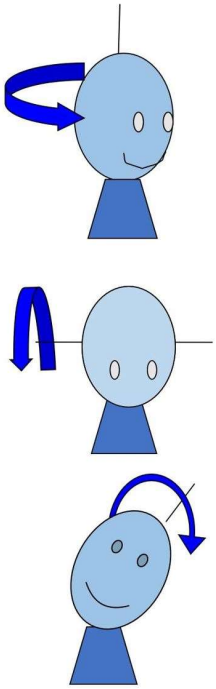
Vestibular Ocular Reflex - VOR

Has 3 main planes of action:

- Yaw – Horizontal rotation
  "No Motion" about a vertical z-axis.
- Pitch – Flexion/Extension
  "Yes Motion" about a horizontal x-axis.
- Roll – Lateral head tilt.
  "Ear to Shoulder Motion"



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- ACTIVITY:
  - Choose a letter on this slide, shake your head "no" as quickly as you can, and keep the chosen letter in focus
  - Repeat the activity, only shaking your head "yes"

    The VOR allows you to accomplish this task

- ACTIVITY:
  - Hold a piece of paper with a number or letter, and move it quickly back and forth (left – right) in front of your eyes

    This uses the 'Smooth Pursuit' system

Which is easier to see?

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Vestibular Reflex	Purpose
VSR	To Stabilize the body Helps mediate eye movements and also muscle tone Increases extensor tone in the limbs on side of stimulation, and flexor tone on contralateral side of the body

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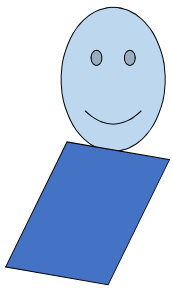
Vestibular Reflex	Purpose
VCR 	Stabilizes the head by acting on neck muscles Reflex head movements counters movement sensed by vestibular system

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Vestibular Causes of Dizziness

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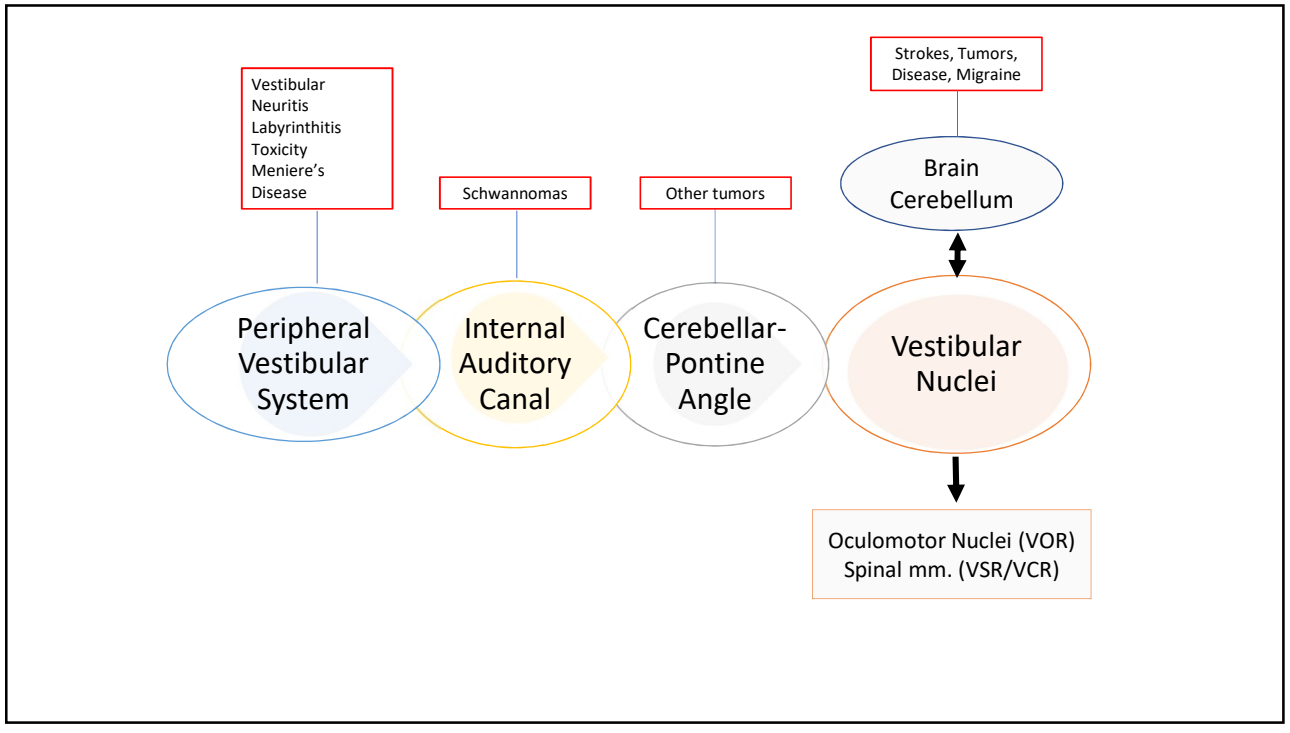

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Vestibular Problems

Damage - 1 Ear
Unilateral Vestibular Loss

Inflammations:
- Vestibular Neuritis
- Labyrinthitis

Physical Damage:
- Dehiscence
- Fistula
- Concussion
- Post-surgery

Tumors:
- Schwannoma (Neuroma)
- Cerebellar-Pontine Angle

Damage – Both Ears
Bilateral Vestibular Loss

Toxicity:
- Medications (Aminoglycosides)
- Cisplatin (chemo.)
- Heavy Metals

Central Etiologies:
- Strokes
- Degenerative Neuro Diseases

One or Both Ears
Abnormal Stimulation

- BPPV

BPPV is usually seen in only one ear at a time, but may be bilateral after trauma

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Damage - 1 Ear
Unilateral Vestibular Loss

Inflammations:
- Vestibular Neuritis
- Labyrinthitis

Physical Damage:
- Dehiscence
- Fistula
- Concussion
- Post-surgery

(Less Commonly)
Tumors:
- Schwannoma (Neuroma)
- Cerebellar-Pontine Angle

Symptoms of Unilateral Vestibular Loss (UVL)

Acute Symptoms:
- Spontaneous (Resting) Nystagmus that increases with gaze away from the affected ear
- Dizziness and nausea that is worse with movement
- May vomit
- Dysequilibrium makes ambulation difficult

Sub-acute Symptoms:
- May only have gaze-evoked nystagmus near ocular end-range, or with vision removed (IR goggles or Frenzel lenses)
- Feel OK while sitting still, but dizzy/nauseous if moving
- Can walk with certainty only without turning head

Chronic Symptoms:
- May not complain if dizziness
- May report dizziness/dysequilibrium while turning quickly

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Damage – Both Ears
Bilateral Vestibular Loss

Toxicity:
- Medications
- Cisplatin (chemotherapy)
- Heavy Metals

Central Etiologies:
- Strokes
- Degenerative Neuro Diseases

Symptoms of Bilateral Vestibular Loss (BVL)

Acutely:
- Extremely dizzy, nauseous and off-balance
- Usually unable to ambulate, and may have difficulty maintaining sitting balance
- No nystagmus are observed
- Reports Oscillopsia with head movement

Chronic:
- Report oscillopsia with head movement
- Typically not dizzy
- Major dysequilibrium
- Ambulates with an assistive device only
- Ambulation is slow and without head turns

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Bilateral Vestibular Loss (Weakness), BVL

- **Symptoms:** Typically Little or no dizziness, but some experience vertigo. May Stand or walk with a wide-base, Tend to move their head and trunk as a unit
- **Etiology:** Aminoglycosides (Gentamycin, Streptomycin) toxicity flattens or damage hair cells in inner ear (most common cause), Chemotherapy, Diuretics, Autoimmune disease, Bilateral Tumors
- **Major problems:** Balancing, Gait Instability, Complain of seeing everything 'bounce' whenever they turn their head or walk (called Oscillopsia), blurred vision when head is moving, may have difficulty reading

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One or Both Ears
Abnormal Stimulation

- BPPV

BPPV is usually seen in only one ear at a time, but may be bilateral after trauma

Symptoms of Benign Paroxysmal Positional Vertigo (BPPV)

Acutely/Chronic:

The patient experiences vertigo during position changes of the head (typically pitches), that are usually noticed when:

- Rolling in bed, Getting in/out of bed
- Bending over to pick up objects
- Bending over to tie shoes
- Washing their hair while showering

No vertigo is noticed while sitting still, however there may be nausea from previous head motions.

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Benign Paroxysmal Positional Vertigo (BPPV)

Crystals (Otoconia) become loose in the Utricle, and float into the semi-circular.

- **Etiology:** Idiopathic, Infections, Head Trauma.
- **Symptoms:** Patient complains of Vertigo (usually spinning) that lasts less than 1 minute while changing positions (sit-supine, rolling in bed, tipping back head to wash hair).
- Nausea may last longer than vertigo. Some vomit.
- Posterior Canal BPPV most common, followed by Lateral Canal, and then Anterior Canal.

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Conditions that Mimic BPPV

Herdman, 2007

Causes	Symptoms
Central Positional Vertigo *with Nystagmus* (Migraine related)	Vertigo & Down-beating Nystagmus, No Torsion & Non-fatiguing
Central Positional Nystagmus *without Vertigo*	Unidirectional Nystagmus: Either Vertical, Horizontal, OR Torsional
Cerebellar Tumor, or Hemorrhage dorsolateral to the 4th ventricle	Vertigo & Down-beating Nystagmus, No Torsion & Non-fatiguing
Vertebral Artery Compression	Vertigo and Nystagmus
Pressure Induced Disorders: • Perilymphatic Fistula • Superior Canal Dehiscence • Hypermobile Stapes	Positional Vertigo & Nystagmus, but usually associated with Hearing Loss
Sensory Mismatch	Vague dizziness during head extension or when rotated to an extreme position

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Medication *Causes of* Dizziness

Medications commonly causing dizziness:

Anticonvulsants

Antidepressants

Anxiolytics

Sedatives (including Hypnotics)

Strong Analgesics

Muscle Relaxants

Antiarrhythmic agents.

- Lawson B. Dizziness in the older person. Rev Clin Gerontol. 2005;15:187-206.
- Furman J,Raz Y., Whitney S. Geriatric vestibulopathy assessment and management. Current Options in Otolaryngology & Head and Neck Surgery. 2010;18:386-391.

56

Medication Class	Examples
Anticonvulsants	Phenytoin, carbamazepine, pregabalin
Antidepressants	Tricyclic antidepressants (e.g., amitriptyline, nortriptyline)
Anxiolytics	Benzodiazepines (e.g. Alprazolam, Diazepam, Lorazepam)
Sedatives	Barbiturates Benzodiazepines Hyptonitic Axiolytics (Estazolam (ProSom), Temazepam (Restoril), Triazolam (Halcion))
Strong Analgesics	Narcotics, Opioids, Morphine
Muscle Relaxants	Baclofen, Carisoprodol (Soma), Chlorzoxazone (Parafon Forte DSC), Cyclobenzaprine (Flexeril)
Antiarrhythmic agents	Amiodarone, Dofetilide, Propafenone, Sotalol

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Other medications *causing* dizziness

Bronstein and Lempert

Mechanism	Class of Drug	Sample Drug
Ototoxicity	Aminoglycosides	Gentamycin Streptomycin
	Glycopeptide antibiotics	Vancomycin
	Alkylating agents	Cisplatin
	Loop diuretics (reversible)	Furosemide Ethacrynic Acid
	NSAIDS (reversible)	Aspirin Ibuprofen
	Antimalarial drugs (reversible)	Quinine Quinidine

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Medications *causing* Falls

Medication side-effects such as sedation may slow reaction times needed to maintain balance. **There is a statistical association between the following drugs and falls:**

- **Benzodiazepines** **(Diazepam, Lorazepam, Clonazepam)**
- **Antidepressants** **(Tricyclic and Serotonin-Reuptake Inhibitors)**

Symptoms may be present with high serum levels, or when permanent damage has been caused by the medications.

Bronstein and Lempert

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The Cerebellum

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Cerebellum	Symptoms of Injury
Important for being able to perform everyday **voluntary** tasks (e.g. walking, writing). Essential to balance and upright posture.	• Loss of balance • Loss of coordinating fine movements • Nystagmus • Tremors • Inability to walk • Slurred speech • Low Muscle Tone (floppy arms/legs)

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Cerebellum

- Cerebellum: Latin for "Little Brain"
- Protrudes from under Posterior Inferior Cerebral Cortex
- 10% of the total weight of the brain
- Contains ~ 50% of the brains neurons

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Cerebellum

Receives Information From:	Sends Output To:	Function:
1. Spinal cord 2. Cerebral Cortex 3. Vestibular nuclei	The Brainstem	• Adjusts motor responses comparing planed movements to sensory data – adjusting movements as needed • Modulates force and range of motion of movements • Involved in motor learning

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Cerebellum

- 3 Cerebellar Lobes
- 3 Cerebellar Zones
- 3 Functional Areas
- 3 Pairs of Peduncles
- 3 Regions - ocular motor control
- 3 Principle cerebellar syndromes

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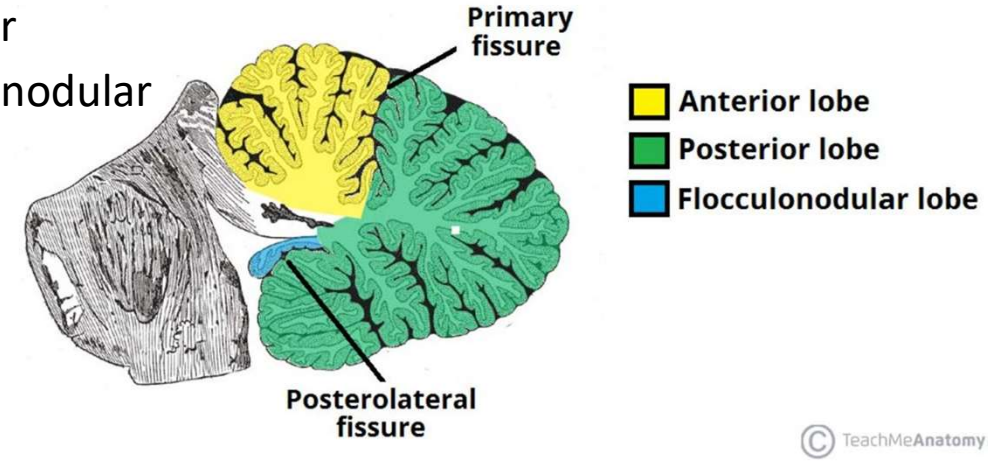
3 Cerebellar lobes:

1. Anterior
2. Posterior
3. Flocculonodular



http://teachmeanatomy.info/neuro/structures/cerebellum/

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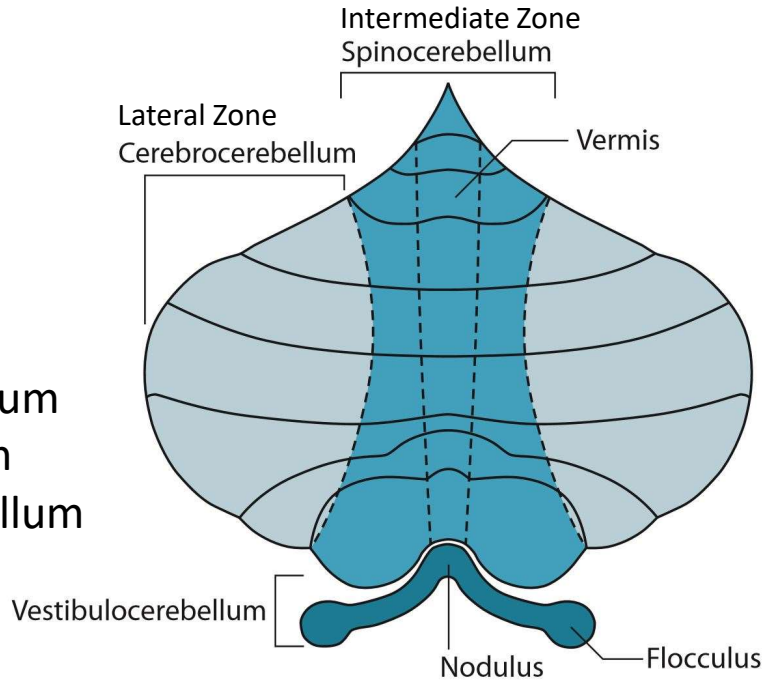
3 Cerebellar Zones:

1.Lateral
2.Intermediate
3.Flocculus

3 Functional Zones

1. Cerebrocerebellum
2. Spinocerebellum
3. Vestibulocerebellum



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3 Cerebellar Functional Areas:

1. **Cerebrocerebellum**

   Comprised: The lateral hemispheres.

   Job: Planning movements and motor learning, coordination of muscle activation, and is important to visually guided movements.

   Receives: Input from cerebral cortex and pontine nuclei

2. Spinocerebellum
3. Vestibulocerebellum

http://teachmeanatomy.info/neuro/structures/cerebellum/

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1. Cerebrocerebellar
2. **Spinocerebellum**

   Compromised: The intermediate zone

   Job: Regulating body movements – error correction

   Receives: limb position and touch/pressure sensations from the spinal cord to compare where limb is in space and where it should be. It modifies motor signals to correct errors in movement.

   Vermis: Runs along the middle. It is involved in posture, limb and eye movements

3. Vestibulocerebellum

http://teachmeanatomy.info/neuro/structures/cerebellum/

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1. Cerebrocerebellar
2. Spinocerebellum
3. **Vestibulocerebellum:**

   Comprised: Flocculonodular lobe and Lingulus

   Job: Controlling balance, posture, and ocular reflexes (mainly fixation on a target)

   Receives: Input from the vestibular system

http://teachmeanatomy.info/neuro/structures/cerebellum/

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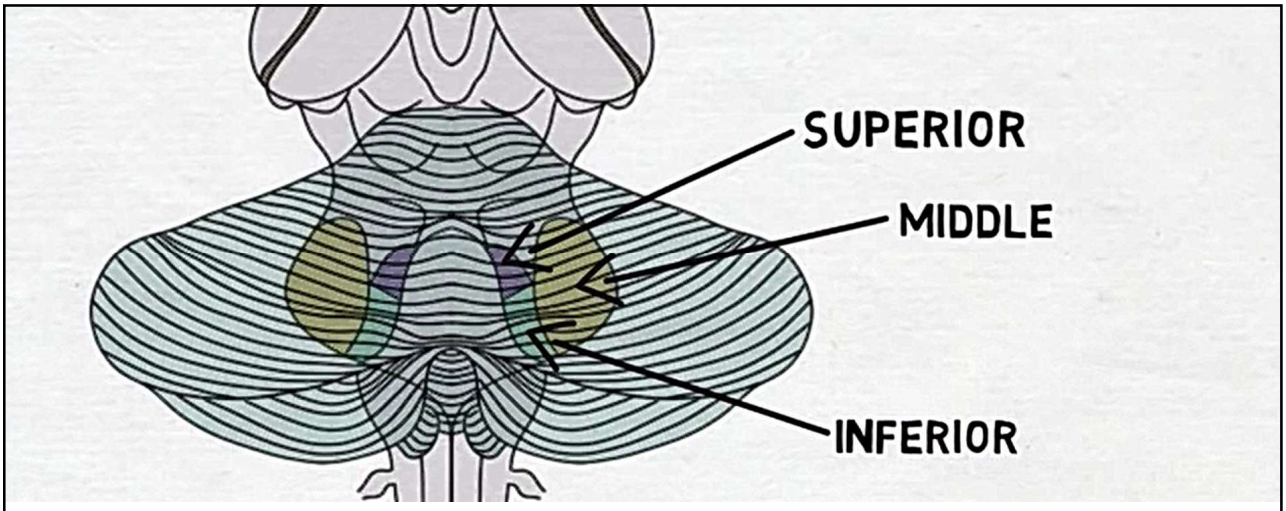

3 Pairs of Cerebellar Peduncles

Part of the Cerebellum that connects there cerebellum to the rest of the nervous system:

2-minute Neuroscience: Cerebellum

70

Peduncles

- Latin for “Little foot”
- Attach to the Pons
- Afferent nerve fibers outnumber Efferent nerve fibers 40:1
- Most afferent tracks enter the cerebellum via the inferior and middle peduncles, with a few entering the superior peduncle.
- The Primary efferent peduncle is the Superior peduncle

Moritani T, Hiwatashi A, Want H, Numaguchi Y, Ketonen L, et al. Anatomy and Pathology of the Cerebellar Peduncle. University of Rochester Medical Center. Rochester, NY.

71

Lesions of the cerebellum and cerebellar peduncles include a variety of pathological conditions:

- Infarction
- Degeneration
- Demyelinating disease
- Toxic metabolic disease
- Trauma
- Tumors

Moritani T, Hiwatashi A, Want H, Numaguchi Y, Ketonen L, et al. Anatomy and Pathology of the Cerebellar Peduncle. University of Rochester Medical Center. Rochester, NY.

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Peduncles

Lesions of the cerebellar peduncles results in clinical symptoms ranging from vertigo or vomiting as the only symptoms to facial palsy, ataxia, nystagmus, diplopia, dysphagia, dysarthria, deafness, contralateral motor weakness, trigeminal sensory loss, dysmetria of the limb, loss of pain and temperature sensation, Horner's syndrome, and Locked-in syndrome.

Moritani T, Hiwatashi A, Want H, Numaguchi Y, Ketonen L, et al. Anatomy and Pathology of the Cerebellar Peduncle. University of Rochester Medical Center. Rochester, NY.

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Lesions of the lateral zone result in errors in the direction, force, speed, and amplitude of movements.

Deficits you may see:

Dysmetria (past-pointing)

Dysdiadochokinesia: Awkward rapid-alternating movements

Rebound phenomena: Lack of force control

Intention Tremor: Tremor noted during movement

Moritani T, Hiwatashi A, Want H, Numaguchi Y, Ketonen L, et al. Anatomy and Pathology of the Cerebellar Peduncle. University of Rochester Medical Center. Rochester, NY.

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3 Regions of the cerebellum involved in all classes of eye movements

1. The oculomotor vermis (lobule VI and VII) and fastigial nuclei
2. The uvula (Inferior portion of the vermis) and nodulus
3. The flocculus and paraflocculus.

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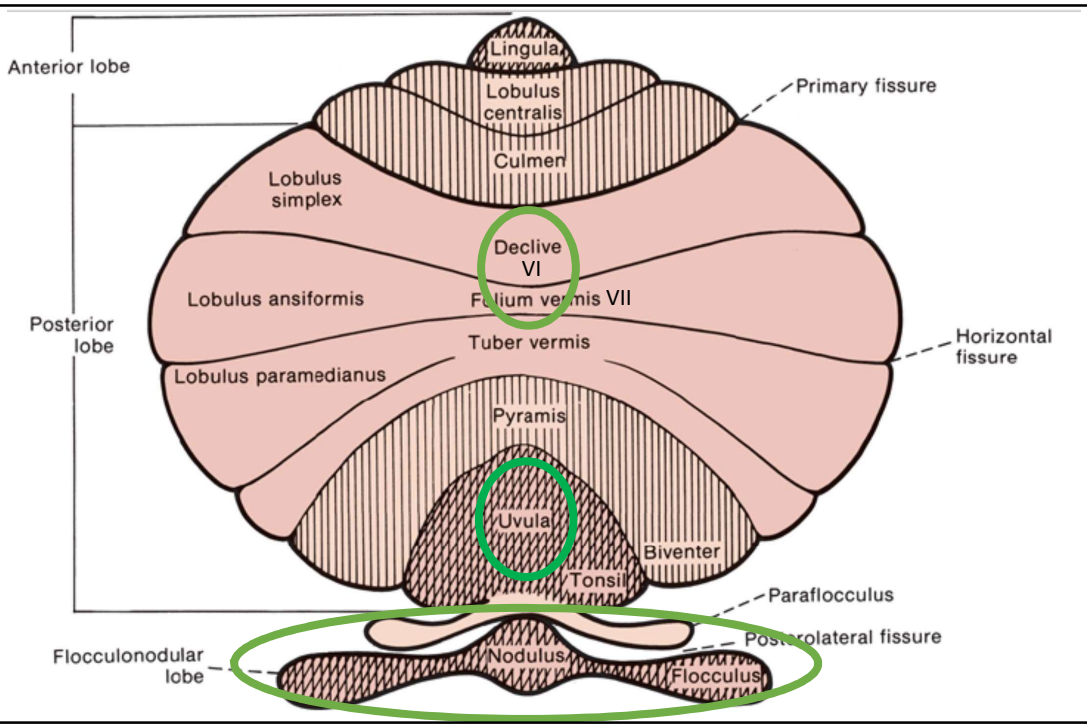

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Functional Differences

Flocculus/Parafloccculus

More concerned with relatively immediate and fast acting ocular motor functions that relate to the needs of holding images steady on the fovea. E.g. Smooth pursuit, VOR, and steady gaze following saccades.

Nodulus and Ventral Uvula

More concerned with generating eye movements that determine the duration and axis of eye rotation in response to low-frequency vestibular stimuli, and to determine the orientation of images on the entire retina.

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Cerebellar disease result in neuro-opthalmic abnormalities that most prominently affect ocular movements

3 Principle cerebellar syndromes:

1. Floccular-parafloccular syndrome,
2. Ventral uvula-nodular syndrome,
3. Oculo-motor vermis-caudal fastigial nuclei syndromes

Zee, D. S., & Walker, M. (2010). Cerebellum and Oculomotor Control. In *Encyclopedia of Neuroscience* (pp. 729-736). Elsevier Ltd. https://doi.org/10.1016/B978-008045046-9.01090-1

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Flocculus/Paraflocculus Syndrome

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Vestibulocerebellum: Flocculus/Paraflocculus

Neurons fire in relation to various features of the stimulus:

- Head Motion
- Visual Target motion
- Position of the eye in the orbit during fixation
- Movements of the eye during smooth pursuit
- Vergence
- Vestibular response to head motion (VOR)

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1st Cardinal Sign of Lesion - Pursuit

With lesions of the flocculus/Paraflocculus smooth tracking is impaired either when the head is still (pursuit) or while moving (VOR cancellation).

Complete lesions of the flocculus/Paraflocculus lead to a steady-state gain (eye velocity/target velocity) of pursuit

There is considerable recovery of pursuit function, even with relatively complete lesions.

81

2nd Cardinal Sign of Lesion - Gaze Holding

Lesions of the flocculus/Paraflocculus cause impaired gaze holding. After eccentric horizontal eye movements, the eyes drift centripetally (toward the center).

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3rd Cardinal Sign of Lesion – Spontaneous Nystagmus

Downbeat nystagmus, in which the eyes drift up slowly (slow phase) and are brought back to the fixation of the target by a corrective downward saccade (quick phase), is a third cardinal sign of Flocculus/Parafloccullus lesion.

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Postsaccadic Drift

Another feature of the floccular/parafloccular syndrome is a brief drift of the eyes, lasting several hundred milliseconds following a saccade.

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Flocculus/Parafloccululs and VOR

The flocculus and Parafloccululs are not critical for the VOR response. The amplitude of the VOR may be normal, increased, or decreased.

The VOR may be misdirected: The axes of the eye rotation and head rotation become misaligned. During head rotation, patients with diffuse cerebellar lesions may show a dynamic upward bias so that the eyes move up and horizontally.

There are also inappropriate torsional components and the responses in the eyes are dysconjugate with more torsion in one eye, and more vertical rotation in the other.

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Flocculus/Parafloccululs Function

The Flocculus/Parafloccululs is responsible for:

- Gaze Holding
- Smooth Pursuit
- VOR Cancellation
- Controlling amplitude and direction of rotational VOR
- Prevent postsaccadic drift

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Ventral Uvula-Nodular Syndrome

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Vestibulocerebellum: Nodulus/Ventral Uvula

The nodulus and adjacent ventral uvula are the most caudal aspects of the cerebellar vermis, and they act upon the 'low-frequency' components of the VOR via projections to a 'velocity storage' mechanism within the vestibular nuclei.

During constant-velocity head rotations, the velocity-storage mechanism extends the duration of the VOR response and slows the decay of nystagmus during constant-velocity head rotations in the dark. It also improves the performance of the VOR at low frequencies of head rotation.

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Lesions of the Nodulus

Lesions of the nodulus result in:

- An increase in the duration of the VOR responses to a constant-velocity input
- A loss of habituation to repetitive stimulation
- Tilt suppression of post-rotary nystagmus (the decay of post-rotary nystagmus is hastened by pitching the head down immediately following the end of a constant-velocity rotation) no longer occurs
- The VOR no longer reorients to the axis of eye rotation that normally occurs with an imposed linear acceleration

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Lesions of the Nodulus / Ulvula

Patients with diffuse cerebellar disease may show abnormally directed slow phases during:

- Low-frequency head rotations around an earth-vertical axis
- During sustained optokinetic stimulation with the head upright
- After horizontal head shaking

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Lesions of the Nodulus / Ulvula

Periodic alternating nystagmus (PAN) is a horizontal jerk nystagmus that may change direction every few minutes and may appear after lesions of the nodulus.

Lesions of the nodulus and/or uvula may also alter smooth pursuit and optokinetic nystagmus, although their exact contributions to these visual-following reflexes is unclear.

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Oculo-motor vermis-caudal fastigial nuclei syndromes

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Dorsal Cerebellar Vermis (Lobules V-VII) and Posterior Fastigial Nucleus

The Dorsal Cerebellar Vermis (also called the oculomotor vermis (OMV)) and the underlying posterior fastigial nucleus (also called the fastigial oculomotor region (FOR)) are Important in the control of saccades and pursuit.

Saccades:

Lesions of the OMV lead to changes in:

- Accuracy
- Latency
- Dynamic properties
- Adaptation

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Dorsal Vermis (Lobules V-VII) and FOR

The Fastigial Nucleus supplies:

- Presaccadic burst for Contraversive saccades (i.e. for right FOR bursts before leftward saccades)
- Braking discharge late during the saccade for ipsiversive saccades

Lesions of the FOR

- Each Fastigial Nucleus facilitates Contraversive hypermetria (overshooting) and Contraversive hypometria (undershooting)
- Bilateral FOR lesions cause hypometric saccades in both horizontal directions

The role of the cerebellum in vertical saccades is not as well localized, and other areas (e.g. paraflocculos) may be important

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Cerebellum's Influence on the Brain Stem

It is proposed that:

- The early influence of the FOR on the initiation of saccades could be mediated through the excitatory burst neurons within the brain step reticular formation
- The neurons could act to brake the saccade by inhibiting the abducens nucleus

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OMV / FOR Lesions and Pursuit

The OMV and FOR participate in the generation of pursuit movements.

Lesions of the OMV lead to:

- Changes in smooth pursuit as well as the ability to undergo pursuit adaptation
- Bilateral OMV lesions produce bilateral pursuit lesions

Lesions of the FOR:

- Contralateral pursuit is impaired
- Bilateral FOR lesions produce symmetrical and relatively intact pursuit

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Multiple Areas…

- There are multiple areas in the cerebellum that are related to pursuit and saccades:
- Uvula and Interposed nucleus, the flocculus/Paraflocculus, and the FOR/OMV complexes all have neurons that discharge with pursuit movements.
- Lesions in the uvula and within the lateral cerebellar hemispheres also have been associated with pursuit deficits.
- The OMV/FOR is concerned with the initiation and termination of the preprogrammed, open-loop, initial portion of pursuit, while the Flocculus/paraflocculus is more concerned with pursuit during sustained tracking.

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Other Ocular Motor Abnormalities

Associated with Cerebellar Dysfunction

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Skew deviations:

Have also been noted with cerebellar dysfunction that cannot be attributed to oculomotor or trochlear nerve palsy, in which the abducting eye is higher.

Esotropia:

Cerebellar abnormalities may cause esotropia, which is usually greater at distance.

Reflexes:

Diffuse cerebellar atrophy may produce dysconjugate vestibular responses, or dysconjugate saccades that are not yoked.

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Cerebellar Syndrome	Features
Flocculus-Paraflocculus Syndrome	• Saccadic smooth pursuit and VOR suppression • Impaired gaze holding: Gaze-evoked nystagmus, Rebound nystagmus • Downbeat nystagmus • Impaired VOR adaptation • Impaired Optokinetic Reflex • Post-saccadic drifts

Nodulus-Ventral Uvula Syndrome	• Impaired velocity storage:
	  • Prolonged post-rotary nystagmus
	  • Failure of tilt suppression of postrotary nystagmus
	  • Perverted head-shaking nystagmus
	  • Periodic alternating nystagmus
	• Positional nystagmus

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Cerebellar Syndrome	Features
Oculomotor Vermis-Caudal Fastigial Nucleus Syndrome: Dorsal vermis damage	• Bilateral hypometric saccades • Saccadic smooth pursuit • Impaired Optokinetic Reflex
Caudal fastigial nuclei damage	• Bilateral hypermetric saccades • Saccadic smooth pursuit • Impaired Optokinetic Reflex
Unilateral dorsal vermis dysfunction	• Ocular contrapulsion • Saccadic contrapulsion: hypermetric contralateral and hypometric ipsilateral saccades • Ipsiversive saccadic pursuit
Unilateral caudal fastigial nuclear dysfunction	• Ocular ipsipulsion • Saccadic ipsipulsion: Hyopmetric contralateral and hypermetric ipsilateral saccades • Contraversive saccadic pursuit

Beh S, Frohman T, Frohman E. Neuro-ophthalmic Manifestations of Cerebellar Disease. Neurologic Clinics. 2014;32(4):1009-1080

101

Cerebellar Lesions…

…do not abolish eye movements but cause them to become coarse, slow, imprecise, and unreliable, leading to degradation in the quality of vision.

It is important to remember:

The cerebellum adapts and compensates for lesions, therefore ocular motor manifestations of acute cerebellar lesions can change, and usually improve with time.

Beh S, Frohman T, Frohman E. Neuro-ophthalmic Manifestations of Cerebellar Disease. Neurol Clin. 2014;32: 1009–1080.

102

Cerebellar Lesions…

Can cause hypotonia.

While hypertonicity (too much muscle tone) is cause by lesions in the motor pathway, the cerebellum helps control tone to prepare for intended movements.

Too little tone (e.g. floppy arms/legs), or too much tone (difficulty moving the limb passively) are signs of a brain lesion.

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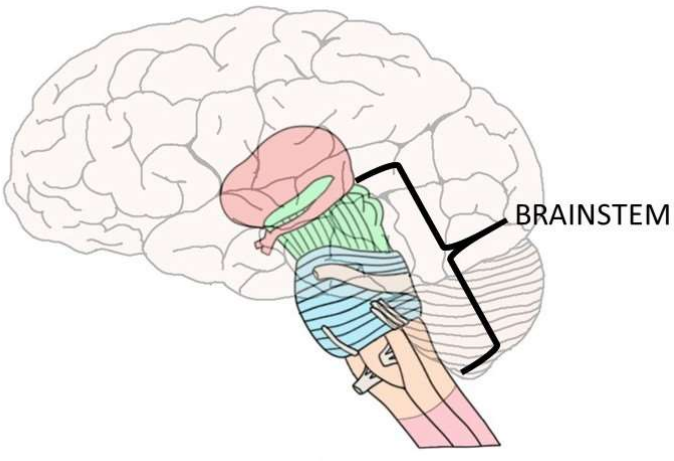
The Brainstem

Midbrain

Pons

Medulla Oblongata



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Brainstem	Symptoms of Injury
Deals with alertness, arousal, breathing, heart rate, blood pressure, and relays information from peripheral nerves and spinal cord to the upper parts of the brain. Parts: Midbrain Pons Medulla Oblongata	• Sleep difficulties (Insomnia, Sleep apnea) • Dizziness, vertigo, nausea • Problems with balance and movement • Decreased breathing capacity crucial for speech • Difficulty perceiving and organizing the environment • Difficulty swallowing food and water • Locked-In Syndrome (Pons)

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Brainstem (Mesencephalon)	Responsibilities
Midbrain	• Acts as a relay center for visual, auditory and motor system information. • Regulates autonomic function (without conscious thought): digestion, heart rate, breathing rate
Pons	• Regulates breathing (amount of air breathed, breathing rate) • Transmission of signals from cerebrum and cerebellum • Involved in sensations of hearing, taste, and balance • Regulates deep sleep • CN: Oculomotor, Abducens, Trochlear, Vestibulocochlear
Medulla Oblongata	• Important to heart rate and blood pressure • Responsible for reflexes: vomiting, sneezing, coughing • Vestibular nuclei (Pontomedullary junction)

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Ocular Motor Assement and the H.I.N.T.S. Exam

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Which tests are important?

1. For the H.I.N.T.S. Exam we need:
   - For completeness, also Cover Tests (Vertical skews indicate a central finding requiring a head scan)
   - Check for Gaze Nystagmus (at 45 degrees and near end range) If possible, check for nystagmus with Infrared Goggles as well
2. Ocular ROM:
   - Lack of upgaze may indicate supranuclear palsy
3. Visual Fields

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Central Signs

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Central Signs During Ocular Exam

Unilateral Gaze Nystagmus when a Head Impulse Test is negative	Only see vertical or rotary nystagmus	Vertical corrective saccades during a Cover Test	Pursuit is saccadic (prior to ~ age 60)	Spontaneous Nystagmus with ocular fixation

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Central Signs During Ocular Exam

Loss of Upward Gaze	Hypometric Saccades > 2 Saccades during saccades testing	Hypermetric Saccades Overshooting saccades during saccades testing	Direction – changing gaze nystagmus	Increased dizziness with fixation Or in light

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Other Central Signs

Abnormal muscle tone
Clonus
Ataxic, uncoordinated movements

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Assessing Vertigo/Dizziness

113

Typical History Question

Is the dizziness a spinning sensation, light headed, or an off-balance sensation?

What does this really tell us?

Both peripheral and central problems may present as light headedness, vertigo, or a feeling of dysequilibrium!

114

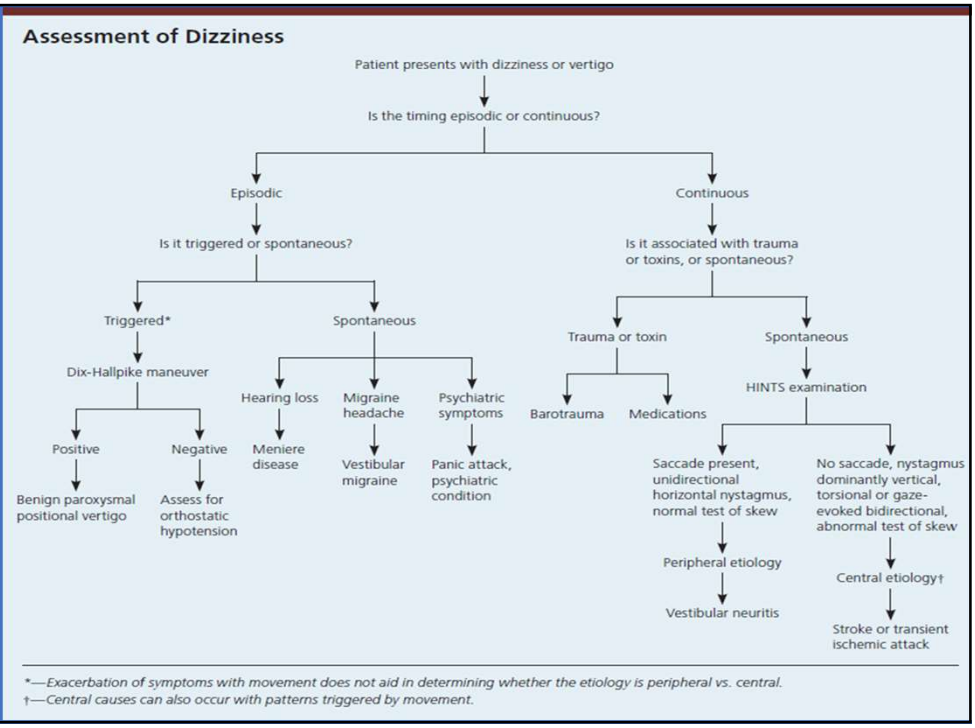
Better questions may be related to:

Episodic vs. Continuous

and

Triggered vs. Spontaneous

Muncie HL, Sirmans SM, James E. Dizziness: Approach to Evaluation and Management. Am Fam Phys. 2017; 95(3):154-162



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How do you assess dizziness?

- History
- Oculomotor Exam
  - Fixation
  - ROM
  - Pursuits
  - Saccades
  - Nystagmus
  - Vergence
  - Perimetry
- Vestibular Exam
  - Head Impulse Test
  - Positioning Tests (BPPV)
  - **VOR-C is NOT a vestibular test**
- Cerebellar Screens
- Cervical Spine Screen
- Somatosensory: Touch and Proprioception
- Muscle Tone
- Standardized Balance Tests
- HINTS Protocol
- Orthostatic BP

116

Dizziness

Vestibular impairments are common after a concussion and may delay recovery.

- Hoffer ME, Gottshall KR, Moore R, Balough BJ, Wester D. Characterizing and treating dizziness after mild head trauma. Otol Neurotol. 2004; 25(2):135–138.
- Naguib MB, Madian Y, Refaat M, Mohsen O, El Tabakh M, Abo-Setta A. Characterisation and objective monitoring of balance disorders following head trauma, using videonystagmography. J Laryngol Otol. 2012; 126(1):26–33.

Vestibular/Oculomotor impairments are reported by 50% of concussed athletes

- Kontos AP, Elbin RJ, Schatz P, et al. A revised factor structure for the Post-Concussion Symptom Scale: baseline and postconcussion factors. Am J Sports Med. 2012; 40(10):2375–2384

Vestibular/Oculomotor issues are not always assessed in the ED

117

Vestibular Testing

Bedside Examination

118

Contraindications to Vestibular Testing or Treatment

Rabie A, 2012

- Acute fractures that prevent the patient from lying down quickly or rolling
- Recent neck fractures, surgery, or instability
- History of vertebral dissection or unstable carotid disease
- Recent retinal detachment

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Screenings

Stability screening tests of the cervical spine prior to testing or treating BPPV or Cervicogenic Dizziness is recommended (Sharp-Purser Test, Alar ligament test)

Many vestibular researchers no longer recommend using Vertebral Artery

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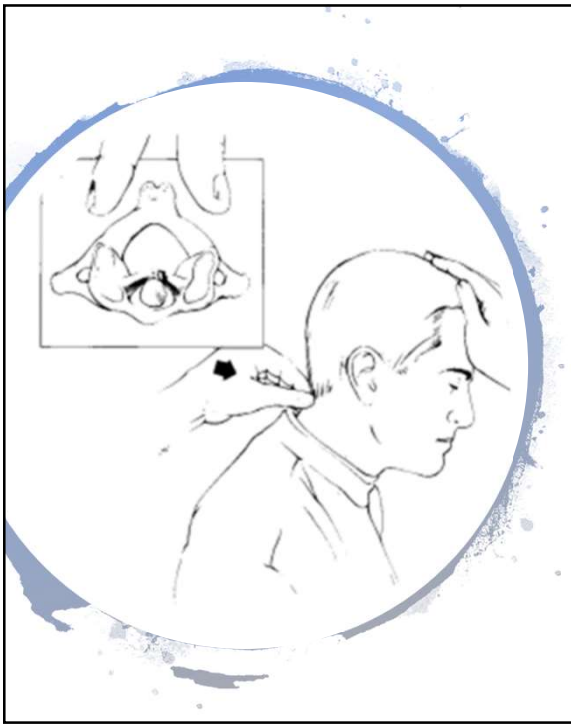
Craniovertebral Hypermobility

Clinical Presentation:

- Patient can give history of flexion injury and has neck pain, occasionally w/ associated head injury
- Diffuse motor loss may occur if the pyramidal tract is affected
- May report a 'lump' in the throat, clumsy hands, numbness to hands/face, ankle clonus, difficulty walking, sphincter control loss
- Headache with sustained flexion
- Vertigo, nausea, tinnitus, and visual disturbances occur with occlusion of the vertebral artery associated with axial rotation of the atlas

Aspinall, W.; Clinical testing for the craniovertebral hypermobility syndrome. J Orthop Sports Phys Ther. 1990;12(2)47-54

121



Cervical Stability Tests

Sharp-Purser Test

Sharp-Purser Test: A test of the Transverse Ligament

The transverse Ligament is the primary restraint to anterior translation of atlas in relation to the lower cervical spine.

- 88% correlation between this test and radiographic findings

http://www.wheelessonline.com/ortho/transverse_ligament_rupture

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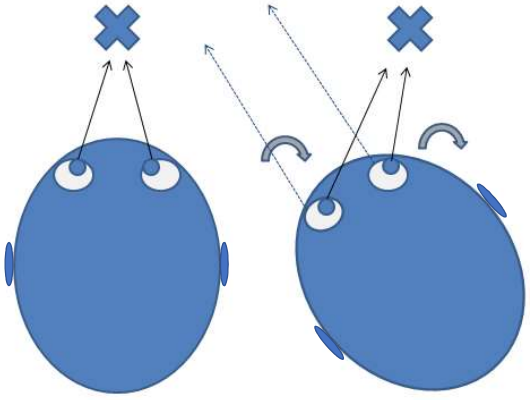
Vestibular Examination: Head Impulse



Left Head Turn,
Eyes move Right

If the Vestibular System is working, the VOR will help move the eyes (in the opposite direction and the same speed of head movement) to stay on a target while the head is turning. During the Head Impulse Test, if the patient has a vestibular weakness, the eyes move with the head, then have to move to find the target when the head stops using a Saccade.

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Head Impulse (Head Thrust) Test

- Hold the patient's head in your hands (above the ears), pitched down 30 degrees
- Give them a visual target (your nose)
- Quickly turn the patient's head toward the ear to be tested at least 10 degrees
  - *Does the patient maintain gaze on the target?*
  - *Observe any corrective saccades*
  - *Test is POSITIVE for direction of Head Thrust if a corrective saccade is seen. E.g.. for a 'Right Head Thrust' that is positive, a Right Unilateral Vestibular Hypofunction would be suspected (usually peripheral)*
- Repeat with head turn to the other side

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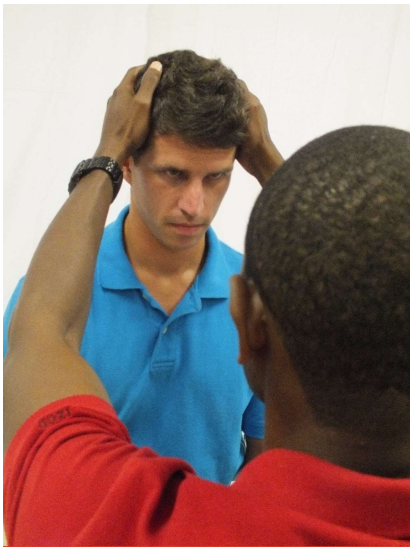

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Head Impulse Test (a.k.a. Head Thrust Test)

- When performing a head thrust test, the patient's head is passively and quickly turned in ONLY ONE DIRECTION while the patient attempts to maintain gaze on a visual target (e.g. your nose). After turning the head, hold it still at the end of the turn (DO NOT turn it back).
- While you turn the head, the vestibular system in one ear is stimulated to create a nystagmus

  (tracking opposite head motion).

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Head Impulse Test (a.k.a. Head Thrust Test)

- The HIT is a powerful clinical tool that helps discriminate peripheral from central lesions in patients with acute vertigo and sustained spontaneous nystagmus (acute vestibular syndrome).
- Video Head Impulse Test (vHIT) allows you to quantify the VOR to stimuli that are high frequency, acceleration, and speed.
- When the head is rotated rapidly to the side of an impaired labyrinth, the slow phase of the VOR is inadequate and requires a compensatory catch-up saccade.

Yacovino D, Akly M, Luis L, Zee D. The Floccular Syndrome: Dynamic Changes in Eye Movements and Vestibulo-ocular Reflex in Isolated Infarction of the Cerebellar Flocculus. Cerebellum. 2017; August 26. Epub ahead of print.

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Head Impulse Test

Specificity and Sensitivity from various studies:

- 100% Sensitivity for complete unilateral vestibular loss (UVL)
- UVL: sensitivity, 36%; average specificity, 97% specific
- Another study: average. sensitivity 46%; average specificity, 75%
- vHIT—specificity 95%, sensitivity 95%

With 30 degrees of flexion and unpredictable timing: Sensitivity:

- 71% UVL, 84% BVL
- Specificity 92%

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Head Impulse Test

References:

Herdman S, Vestibular Rehabilitation. 4th ed. Philadelphia: FA Davis; 2014.

Beyon G., Baguley D.M.A. Clinical evaluation of head impulse testing. Clin Otolaryngol. 1998;23:117-122.

Weber K.P., Aw S.T., Todd M.J., McGarvie L.A., Curthoys I.S., Halmagyi G.M. Head impulse test in unilateral vestibular loss: vestibulo-oculoar reflex and catch-up saccades. Neurology. 2008;70(6):454-463.

Schubert M., Tusa R., Grine L., Herdman S. Optimizing the sensitivity of the Head Thrust Test for identifying vestibular hypofunction. Phys Ther. 2004;84(3)151-158.

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Head Shake Test

1. The patient remains seated, with fixation removed (IR Goggles or Frenzel Goggles)
2. Tilt the patient's head into 30° of cervical flexion
3. The patient closes their eyes while the clinician vigorously shakes the patient's head back and forth for 20 cycles.
4. The patient is instructed to opens their eyes and the head is stopped
   - ***A* Positive *test is when Nystagmus are noted***
   - ***The direction of quick the 'quick phase' is towards the more active ear***

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Nystagmus Grading – Unidirectional beating

Grade I:

Gaze nystagmus in the direction of the healthy ear only

Grade II:

Nystagmus seen in the primary position
Gaze nystagmus in the direction of the healthy ear

Grade III:

Nystagmus seen in the primary position
Gaze nystagmus in the direction of the healthy ear
Gaze nystagmus in the direction of the ear opposite of the healthy ear

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Vestibular Lab

Head Impulse Test

Head Shake Test

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Helpful Equipment

For Vestibular Assessement

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Fixation Removal

Type	Advantages	Disadvantages
Optical	Less expensive ($600 - $1000)	Cannot record images More difficult to see the patient's eye(s)
Video (IR Goggles)	• Available at multiple price points depending on your needs/software • Can record/playback video	• More expensive • Requires a laptop or desktop computer

Observing the eyes with fixation removed is helpful in differential diagnosis, as well as to determine if a vestibulopathy exists.

Frenzel Goggles

There are 2 types: Video Frenzel and Optical Frenzel

Some Video Frenzel's are also called Infrared Goggles

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Fixation Removal

Why remove fixation?

- Patients with peripheral vestibular dysfunction can suppress nystagmus while fixating. When fixation is removed, the nystagmus may appear.
- Observable nystagmus change over time with a peripheral vestibulopathy:
  - Acutely – spontaneous nystagmus at rest
  - Sub-acutely (1-2 weeks) – Nystagmus with gazes at 45 degrees or near end-range
  - Sub-acutely (2+ weeks) – Nystagmus only observed with fixation removed
  - Chronically (4 weeks +) – No observable nystagmus
- Patients with central findings have nystagmus while fixating, but they may disappear in darkness

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Fixation Removal

- With the goggles on, dim the room lights to ensure no light is seen.
- Ask the patient to keep eyes open (allowing them to blink) and observe for spontaneous nystagmus.
- If you have a suppression light, turn it on and ask them to look at it. Did nystagmus subside or slow? If so, it is likely a peripheral issue.
- Ask the patient to look to the Left (observe), and Right (observe)
- Ask the patient to look up (observe), then Upper Left and Right Quadrants
- If you test BPPV, you may also use the goggles

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HINTS Examination

Differentiating Central from Peripheral Causes of Dizziness

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H.I.N.T.S.

H.I. Head Impulse Test, **N.** Nystagmus, **T. S.** Tests of Skew

Using a tests of the ocular motor and vestibular systems, you can determine the likelihood of stroke.

Test for Stroke in Acute Vertigo	Estimated Sensitivity	Estimated Specificity
Brain CT ± contrast CTA	1-42%	98%
MRI with DWI, MRA ± contrast	80% (<24 h)	96%
HINTS Examination	99%	97%

Hyden D, Akerlind B, Peebo M. Inner ear and facial nerve complications of acute otitis media with focus on bacteriology and virology. Acta Otolaryngol. 2006;126(5):460-466.

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H.I.N.T.S.

H.I. Head Impulse Test, **N.** Nystagmus, **T. S.** Tests of Skew

The test is considered positive for stroke when:

- The Head Impulse Test is NEGATIVE, indicating the peripheral vestibular system is intact, **AND…**
- Horizontal direction-changing gaze nystagmus are observed (or Vertical nystagmus are observed)
- **OR**, Vertical Skew is noted on a cover-uncover test (a.k.a. alternate cover test)

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Test	Results	Results	Results	Results
Head Impulse	Negative	Negative	Negative	Positive* (1 ear)
Direction-Changing Nystagmus	Positive	Negative	Positive	Negative
Tests of Skew **(Alternating Cover Test)**	Negative	Positive	Positive	Negative
Suspected Finding	Stroke	Stroke	Stroke	Peripheral (Not Central)

* If the Head Impulse Test is positive for both ears, a CVA is possible

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H.I.N.T.S. Lab

Perform a HINTS Exam

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Cerebellar Tests

One or more may be positive with cerebellar issues

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Cerebellar Screens

Type of Test	Upper Extremities	Lower Extremities
Accuracy of Movements	• Finger to Nose • Finger-Nose-Finger ** Arm fully extended to use as many joints as possible*	• Patellar Tapping • Heel-Shin Slide
Coordinated Movements	• Rapid-Alternating Hands • Hand Clapping	

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Cerebellar Screens

Type of Test	Instructions	Positive Finding
Scanning Speech	Instruct the patient to say something with a lot of consonants, such as '*The American constitution*'	Unable to say clearly
Nystagmus	Instruct the patient to follow your finger into eccentric gaze	See fast-phase nystagmus >3 beats towards side of lesion *Must test vestibular system to rule out peripheral causes

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Cerebellar Screens

Type of Test	Instructions	Positive Finding
Rebound Test	Place a protective arm across the patient. With your other hand instruct the patient to pull on your hand. Let go.	The patient is unable to stop his arm/hand from hitting himself
Pendular Knee Reflex	Strike the patellar tendon with a reflex hammer	The patients leg swings back and forth 3 or 4 times before stopping due to hypotonia

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Cerebellar Screens

Type of Test	Instructions	Positive Finding
Balance	Patient is instructed to stand still (eyes open, then closed)	The patient demonstrates titubation (trunk sways) *The Romberg test is a test of proprioception – patient is still with eyes open but sways with eyes closed
Gait	Ask the patient to ambulate the hallway	Demonstrates a wide-based, ataxic gait. Falls toward the side of lesion.

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Cerebellar Screens

Type of Test	Instructions	Positive Finding
VOR-Cancellation	Alternately move the patients head passively left and right. The examiner moves with the head, and stays in front of the patient. Instruct the patient to keep looking at the tip of the examiner's nose.	The examiner observes catch-up saccades during the motion.

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Cerebellar Screen Lab

Hand Clap
Finger-Nose
Finger-Nose-Finger
Heel-Shin Slide

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Other Central Screens

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Central Screens

Type of Test	Description	Positive Findings
Clonus	*With the patient relaxed, rapidly dorsiflex the ankle*	Repetitive muscle contractions (ankle pumps two or more times)
Muscle Tone	Passively move arms and legs through their full range of motion	• You feel a catch • It is difficult to move the limb (feels like pulling taffy)

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Call 911 or visit Emergency Dept.

- Dizziness after a head injury
- Fever over 101°F, headache, or very stiff neck
- Convulsions or ongoing vomiting
- Chest pain, heart palpitations, shortness of breath, weakness, a severe headache, inability to move an arm or leg, change in vision or speech
- Fainting and/or loss of consciousness

Reference: American Academy of Otolaryngology – Head and Neck Surgery. 2014; online: www.entnet.org/content/dizziness-and-motion-sickeness

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Requires Referral to Neurology and/or Cerebral Imaging

Bronstein A. Oxford Textbook of Vertigo and Imbalance.. Oxford, UK: Oxford University Press; 2013.

- **Prolonged** (≥24 hrs.) **dizziness** *accompanied by* any of the following:

1. **Above 65 years old**
2. **Pre-existing vascular disease**
3. **History of vascular risk factors** *(smoking, DM, HTN, Elevated cholesterol)*
4. **Normal Head Impulse Test** *(Vestibular function screen)*
5. **Any neurologic signs not explained by acute vestibular neuritis** *(which presents with abnormal head impulse test, veering to affected side, and spontaneous nystagmus to the healthy ear)*

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Patient Examination

- Nystagmus – If observed, are they direction-changing?
- Cover Tests – Do you see vertical skews?
- Head Impulse Test – Is it positive?

Other Tests of the Cerebellum:

- VOR- Cancellation – Is it intact?
- Tests of accuracy of motion: Finger-nose, Finger-nose-finger, Heel-shin slide
- Tests of coordination: Hand clap, Rapid-alternating hands, Rapid-alternating feet

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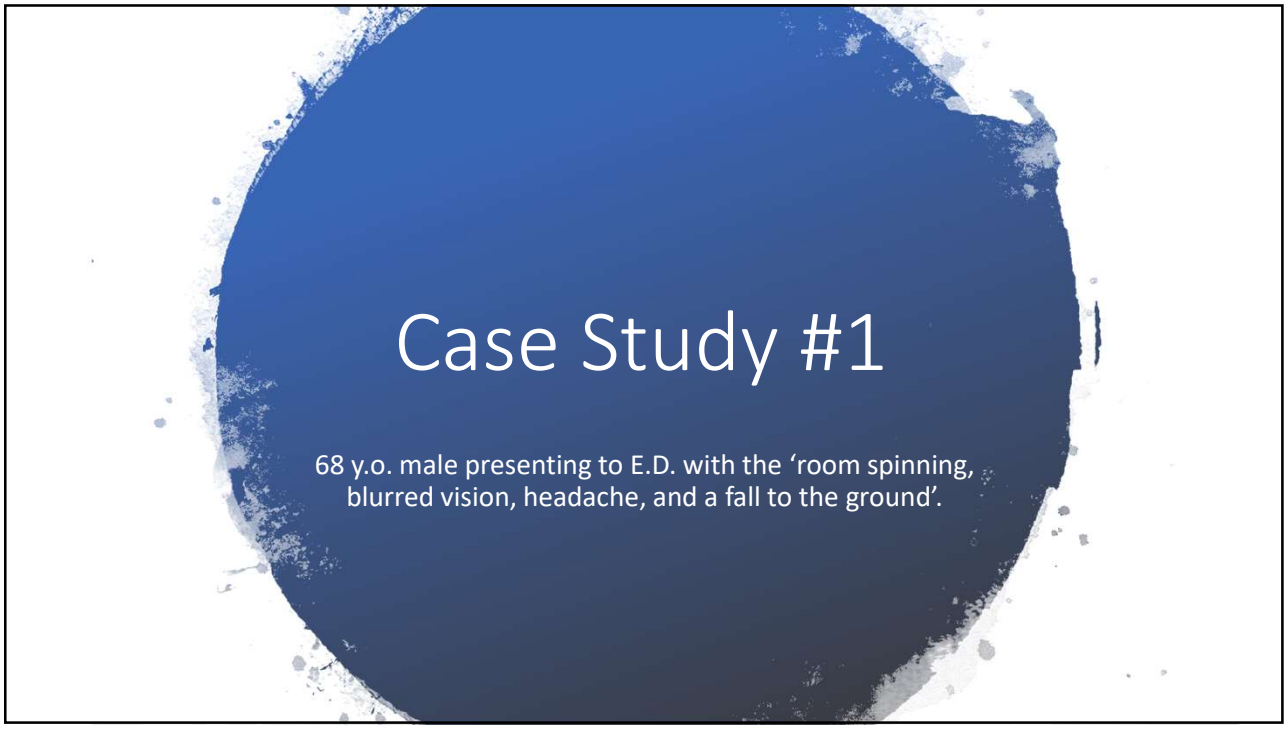

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68 y.o. ♂ presenting to E.D. with the 'room spinning, blurred vision, headache, and a fall to the ground'. Loss of consciousness once on the ground. Denies head trauma. Became diaphoretic, nauseous, and had vomiting. EMS called after 30 minutes. Symptoms are worse with movement. Denies chest pain and shortness of breath. Endorses recent sinus congestion.

PMH: Coronary artery disease, hypothyroidism, asthma, CKD III, HTN, HLD, PVD

Surgical Hx: CABG, Back Surgery

Social Hx: Married, Denies smoking, drinking alcohol, or use of illicit drugs

Admitted to the hospital, the attending Internal Medicine MD requested a 'Vestibular PT' examination

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BP: 167/77, Pulse 77 Resp. 15, SpO2 100%

Orientation: to person, place, and time

Emergency Dept. MD:

- Ordered a CT
  - Mild chronic ischemic changes with no acute intracranial abnormality
  - Mild bilateral ethmoid sinusitis
- Recommended MRI, PT

Hospitalist ordered a Vestibular Evaluation from PT

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Case Study #1 – PT Exam Findings

Ocular Motor Exam

- Spontaneous Nystagmus: [+] up-beating nystagmus, increasing with up-gaze
- Cover Test: WNL
- Alternating Cover Test: [+] Vertical skew (right hypodeviation)
- Vergence: WNL
- Pursuit: Saccadic

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Vestibular Exam

- Head Impulse Test: [+] Bilaterally
- Dix-Hallpike: WNL
- Roll Test: [+] Apogeotropic with Right head roll, Left head roll was WNL

Cerebellar Screens

Hand Clap: WNL

Finger-to-nose: WNL

Finger-nose-finger: WNL

Heel-Shin Slide: Abnormal LLE

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Musculoskeletal Exam

- LE Strength WNL
- Ankle ROM WNL
- UE not assessed secondary to nausea

Somatosensory Screens

Light touch WNL bilateral UE, LE, and Face

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PT recommended an MRI

MRI Results

***

Repeat CT (~24 hours after first CT)

***

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Case Study #2

68 y.o. female. Admitted for episodes of dysequilibrium and unsteady gait, dizziness and vomiting. Tried to sleep, but woke with palpitations.

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Case Study #2 – PT Exam Findings

Subjective

Dizzy while looking at things

Ocular Motor Exam

- Fixation: Intact
- Spontaneous Nystagmus: None
- Eccentric Gaze: [+] Direction-Changing Nystagmus
- Cover Test: WNL
- Alternating Cover Test: [+] Left exophoria
- OKN: WNL
- Vergence: WNL
- Pursuit: Saccadic
- Saccades: Hypermetric vertically (horizontal WNL)
- Finger Counting Confrontation Test: WNL

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Vestibular Exam

- Head Impulse Test: WNL
- Dix-Hallpike: Not tested
- Roll Test: Not tested

Cerebellar Screens

Hand Clap: WNL

Finger-to-nose: WNL

Finger-nose-finger: Dysmetric with Right UE

Heel-Shin Slide: Abnormal RLE

VOR-Cancellation: Impaired

Tone: Ashworth 0 for all extremities (WNL)

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Musculoskeletal Exam

- LE Strength WNL
- Ankle ROM WNL
- UE Strength WNL

Somatosensory Screens

Light touch WNL bilateral UE, LE, and Face

Proprioception: WNL bilateral UE/LE

Gait

Slow gait speed, unsteady – reaching for walls.

Loss of balance x 1 while turning right

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PT recommended an MRI

MRI Results

***

CT

No evidence of acute intracranial process

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Case Study #3

57 y.o. male complaining of vertigo, light headedness and unsteady gait.

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57 y.o. ♂ Complaining of vertigo, light headedness and unsteady gait.

PMH: HTN

Social Hx: Single and lives independently. There are no steps or stairs in his home.

BP: Supine 159/81, HR 70

Sitting 152/87, HR 104

Orientation: to person, place, and time

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Case # 3 Exam Findings

Ocular Motor Exam

- Range of motion is WNL
- Fixation is impaired secondary to nystagmus
- Nytagmus: Grade III right-beating nystagmus
- Near Point of Convergence: WNL
- Cover Test: No vertical skews. Unable to assess alignment secondary to constant nystagmus

Hearing

Grossly intact bilaterally (finger rub)

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Vestibular Exam

- Head Impulse Test: [+] Left
- BPPV Tests: Not tested

Cerebellar Screens

Hand Clap: WNL
Finger-to-nose: WNL
Finger-nose-finger: WNL
Heel-Shin Slide: WNL

Muscle Tone

Ashworth 0 for all extremities

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Musculoskeletal Exam

Strength and ROM WNL for bilateral UE/LE

Somatosensory Screens

Light touch WNL bilateral UE, LE, and Face

Gait

100', Independent, normal gait pattern and speed

Balance

Functional Reach WNL

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MRI

***

Diagnosis

***

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Case Study #4

50 y.o. male complaining of nausea, vomiting, and constant dizziness.

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50 y.o. ♂ Complaining of nausea, vomiting and dizziness.

PMH: HTN, Obesity (340 lbs)

Social Hx: Married. Has 3 steps with a rail in his home. Previously independent with gait and Activities of Daily Living

Orientation: to person, place, and time

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Case # 4 Exam Findings

Ocular Motor Exam

- Range of motion is WNL
- Fixation is intact
- Nytagmus: [+] Direction-changing gaze nystagmus
- Near Point of Convergence: WNL
- Cover Test: Obvious OD exotropia
- Pursuits: Saccadic (abnormal for age)
- Visual Field: Intact to moving confrontation

Hearing

Grossly intact bilaterally (finger rub)

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Vestibular Exam

- Head Impulse Test: [+] Bilaterally
- Dix-Hallpike (BPPV Test): Subjectively positive but without nystagmus
- Roll Test (BPPV Test): WNL

Cerebellar Screens

Hand Clap: WNL
Finger-to-nose: WNL
Finger-nose-finger: WNL
Heel-Shin Slide: WNL

Muscle Tone

Ashworth 0 for all extremities

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Musculoskeletal Exam

Strength and ROM WNL for bilateral UE/LE

Somatosensory Screens

Light touch WNL bilateral UE, LE, and Face

Clonus

Positive Right ankle

Balance

Unsteady static standing requiring minimal assistance

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MRI

***

Diagnosis

***

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Summary

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To differentially diagnose dizziness”

- Get a description (vertigo vs. light headedness), but also inquire about the timing of the dizziness.
- Perform an ocular exam AND a vestibular screen using the HINTS protocol
- You may also check for increased/decreased muscle tone and clonus
- Refer for an MRI if you suspect something central

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Is it central? Complains of dizziness with:

Central Signs	Peripheral Signs
Dizziness worse in the light Dizziness/nystagmus upon fixation Direction changing nystagmus HINTS Exam finds: • Normal Head Impulse Test • AND Vertical Skew • OR, Direction-changing nystagmus Positive Cerebellar Screens Abnormal muscle tone or clonus	Dizziness worse in the dark Nystagmus improves with fixation Unidirectional nystagmus HINTS Exam finds: • Positive Head Impulse Test • No Vertical Skew • Unidirectional or no nystagmus Negative Cerebellar Screens

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Questions?

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