




Vertigo and Balance Issues

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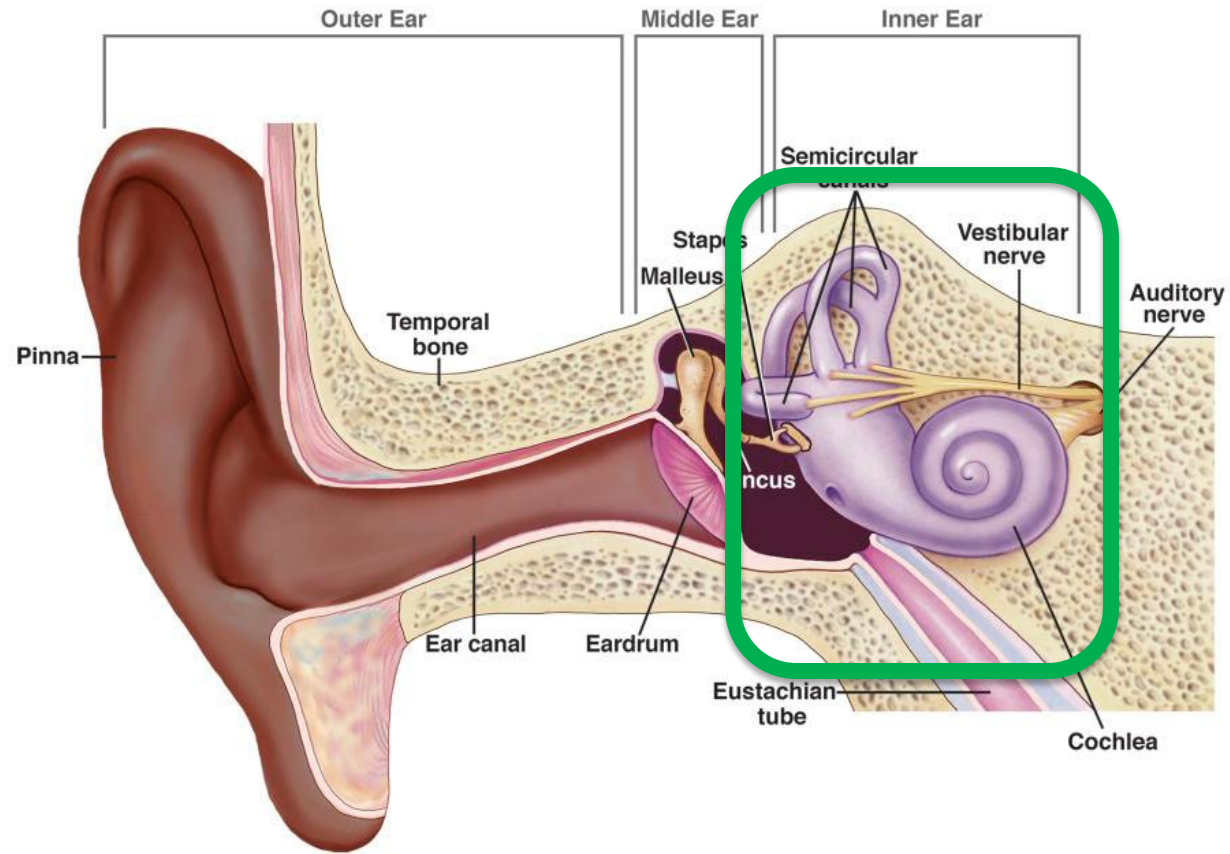
Learner Objectives

1. Describe basic anatomy and physiology related to balance
2. Differentiate symptoms of vertigo and dizziness
3. Identify common vertigo and balance issues related to TBI

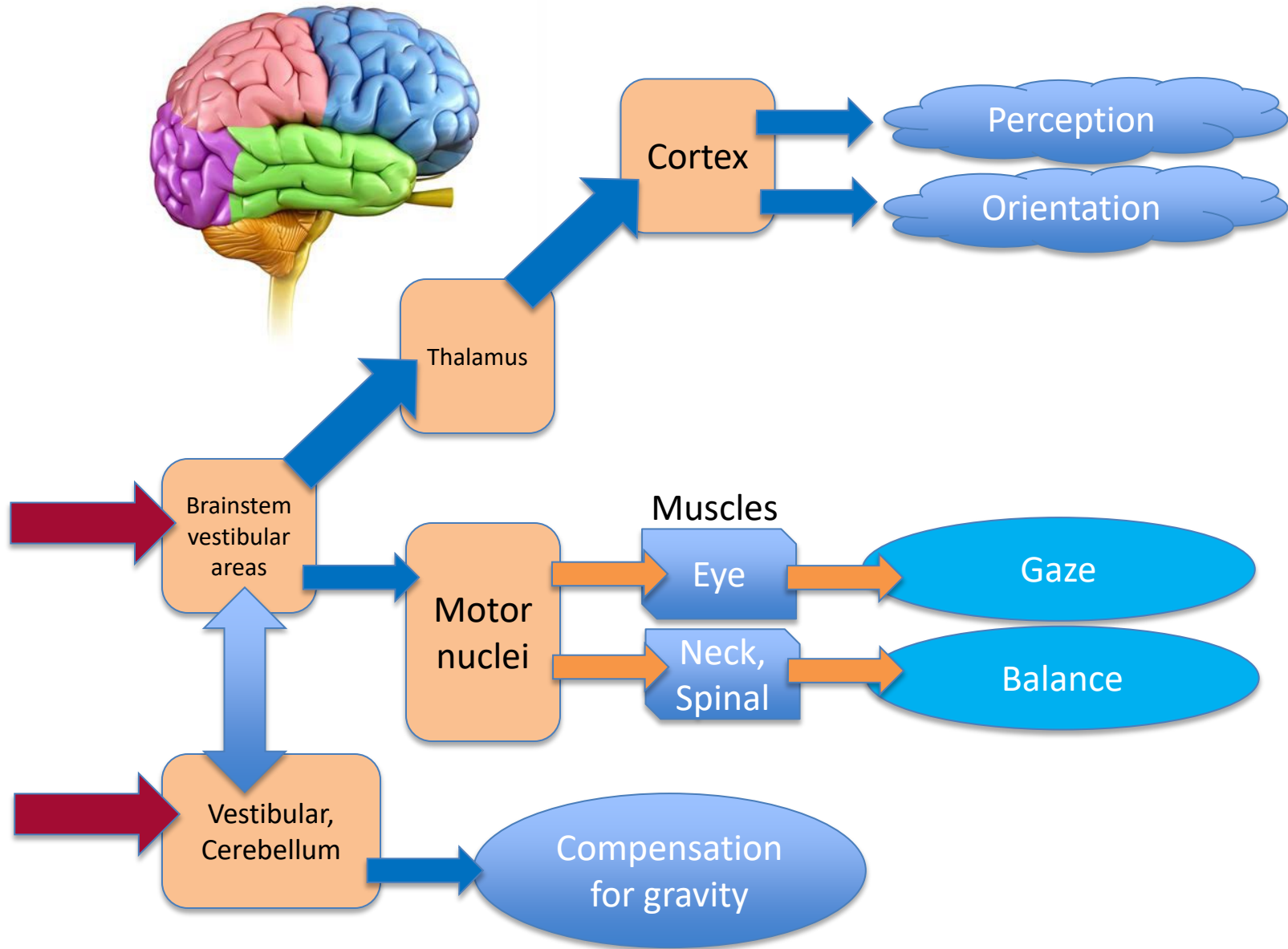
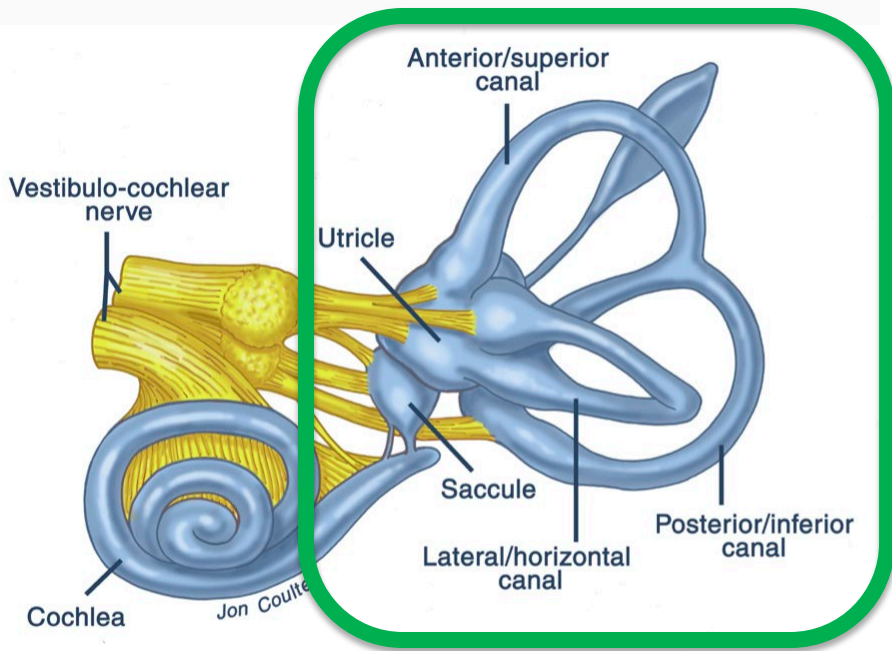


General Anatomy and Physiology Related to Balance

General Ear Anatomy



Vestibular and Balance-Related Systems



BALANCE

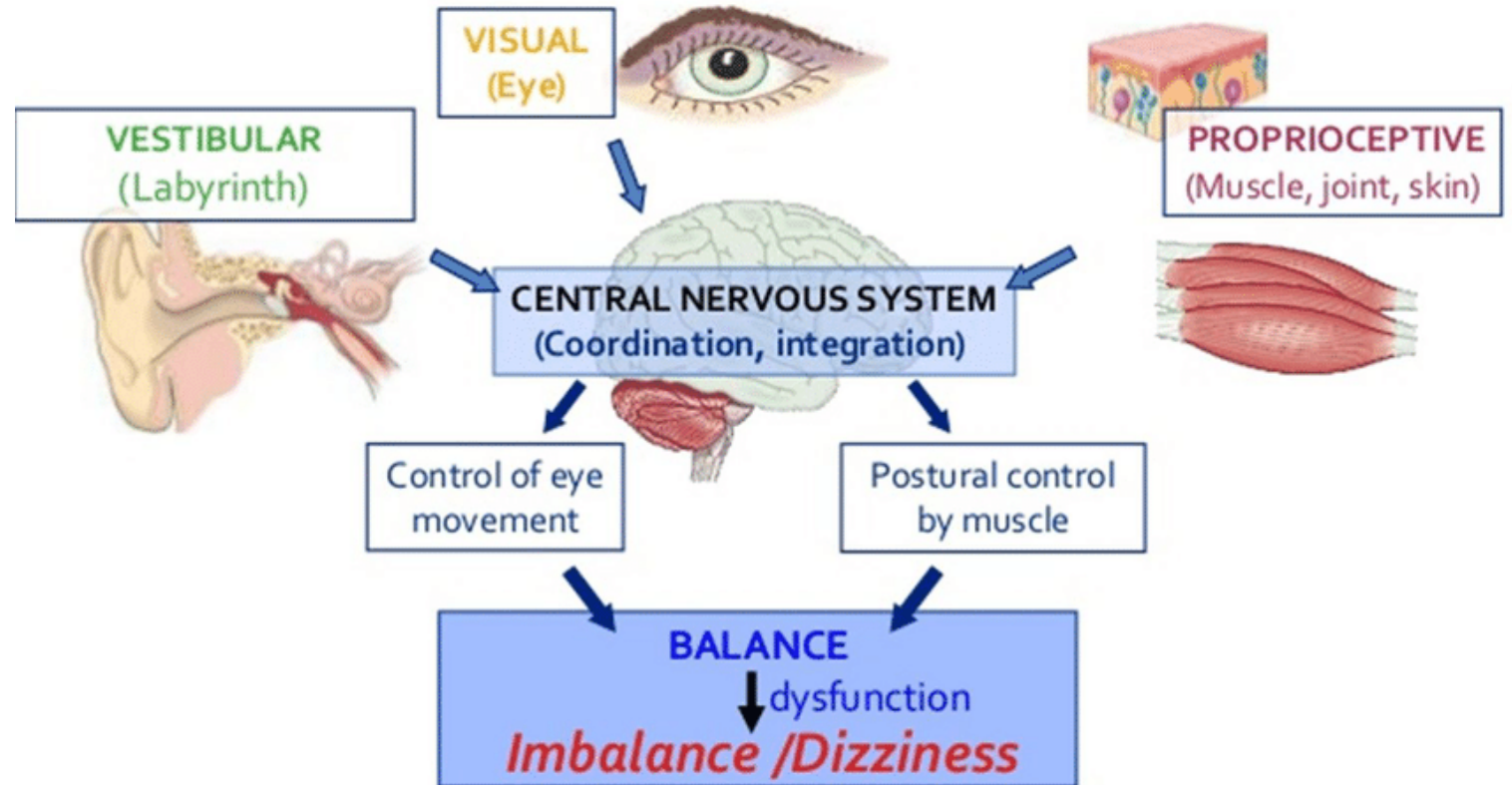
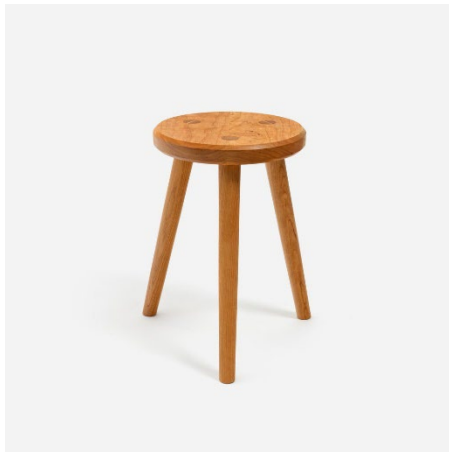
- Where am I going?
- Which way is up?

- Vestibular system key function is balance:
 - measures acceleration forces applied to the head
 - interacts with eyes to help keep visual images stable
 - directs neck and body to help keep a sense of balance

BALANCE CONTROL

Several sensory systems help us maintain balance:

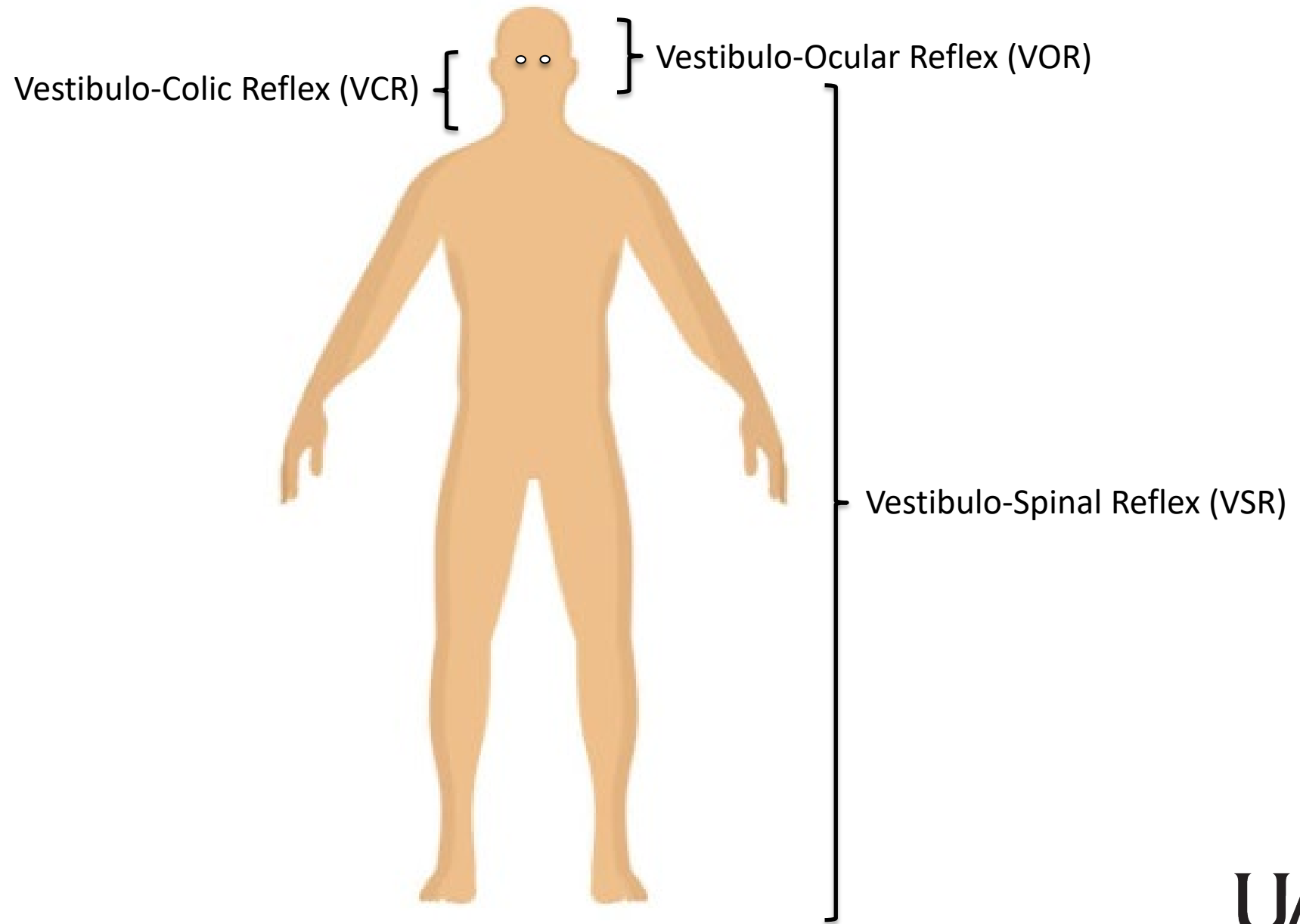
1. Vestibular System
2. Proprioception
3. Visual System



Samuel, Solomon, & Mohan (2015)

Roles of the Various Systems

- Internal stimuli -> Vestibular (acts like a gyroscope)
- External stimuli -> Visual and Somatosensory (proprioceptive)
- CNS -> the integrator of all three modalities (vestibular, visual, and somatosensory), these create the necessary muscular skeletal and motor responses to basically keep us from falling
- It is believed that 2/3 of the information the brain needs comes from the vestibular system, the vestibular system is the most important leg of this tripod



Vestibulo-Ocular Reflex (VOR)

- Allows visual (gaze) stabilization during active head movements
- When someone has peripheral dysfunction, we expect a patient to have oscillopsia, imbalance, and dizziness, especially during changes in head position and head movements in certain planes of stimulation



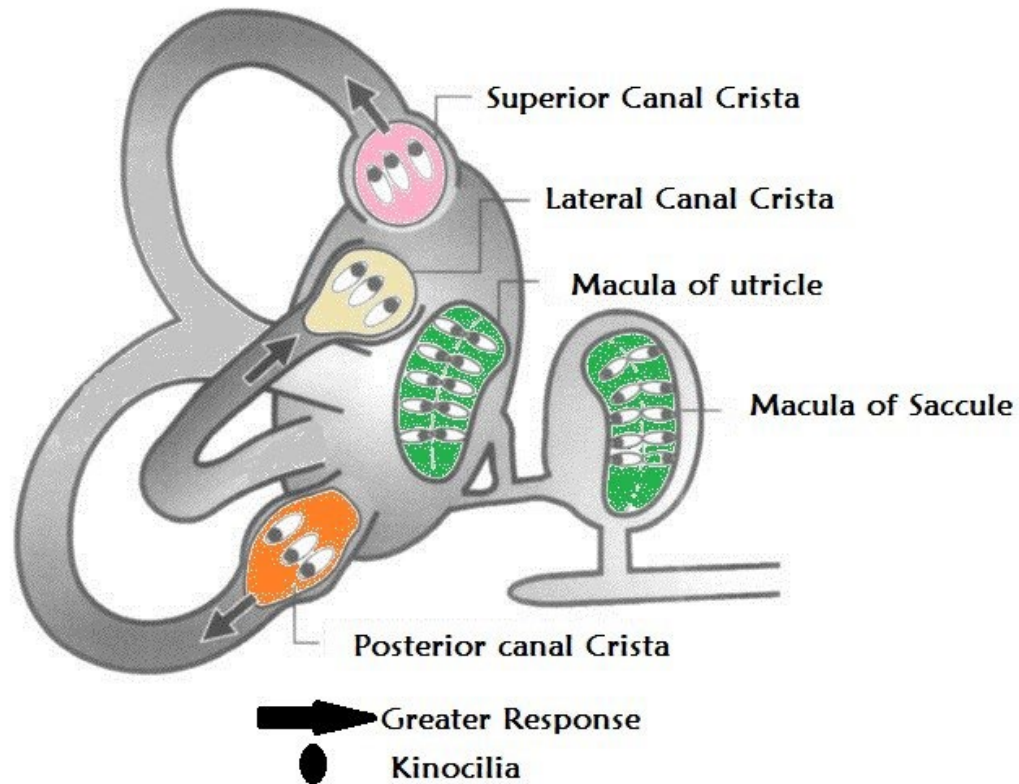
Vestibulo-Spinal Reflex (VSR) and Vestibulo-Collic Reflex (VCR)

- VSR is a reflex that typically helps keep an upright posture with the head centered
 - When the head or body moves, vestibular, vision, and somatosensory inputs help return the head and body to “neutral positions”
- VCR is a head stabilizing reflex
- VOR and VSR are typically far more problematic in dizzy patients compared to the VCR

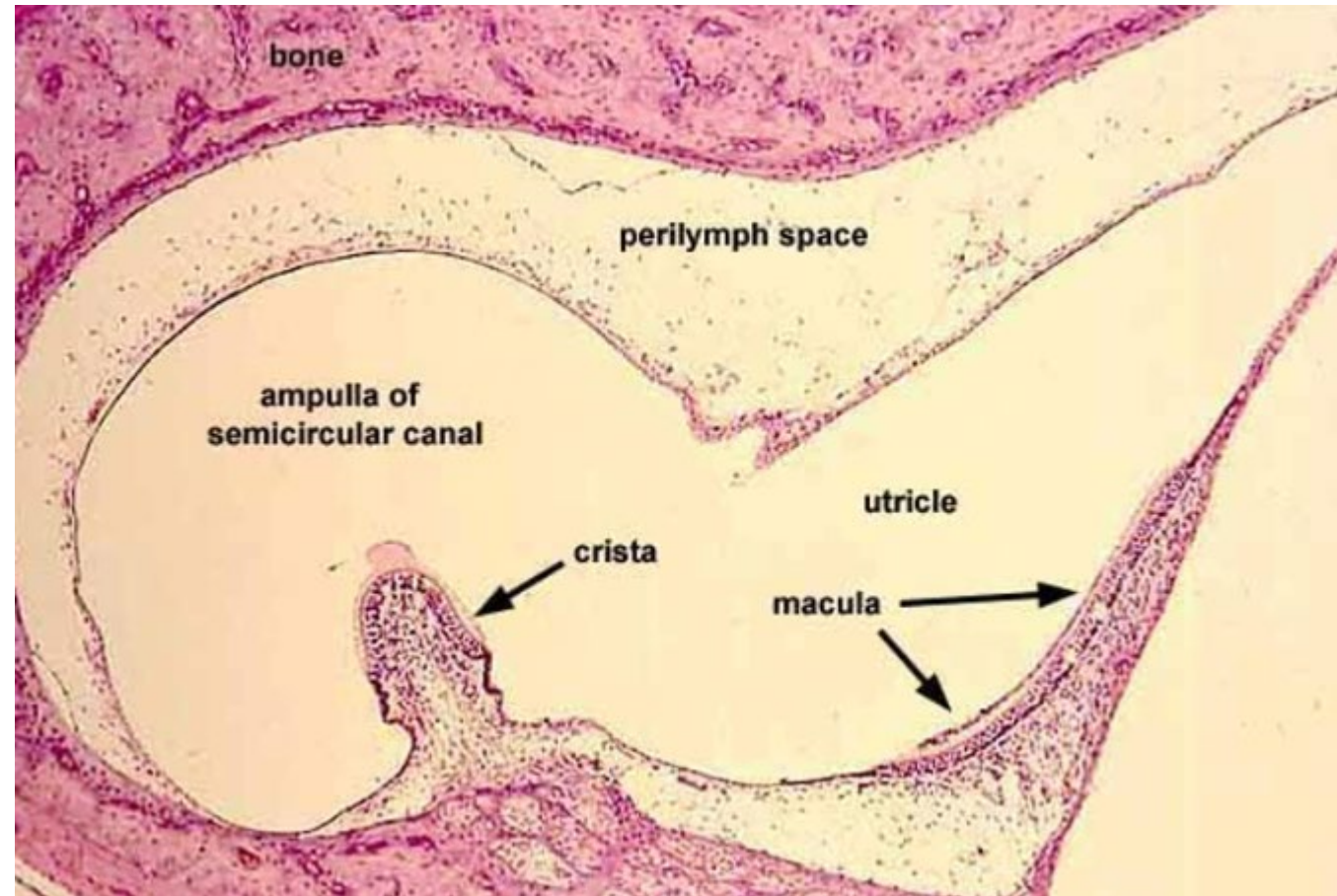


Vestibular Division of the Inner Ear

- Utricle and Saccule are gravity sensors (green)
- Three semicircular canals are velocity detectors (pink, yellow, and orange)

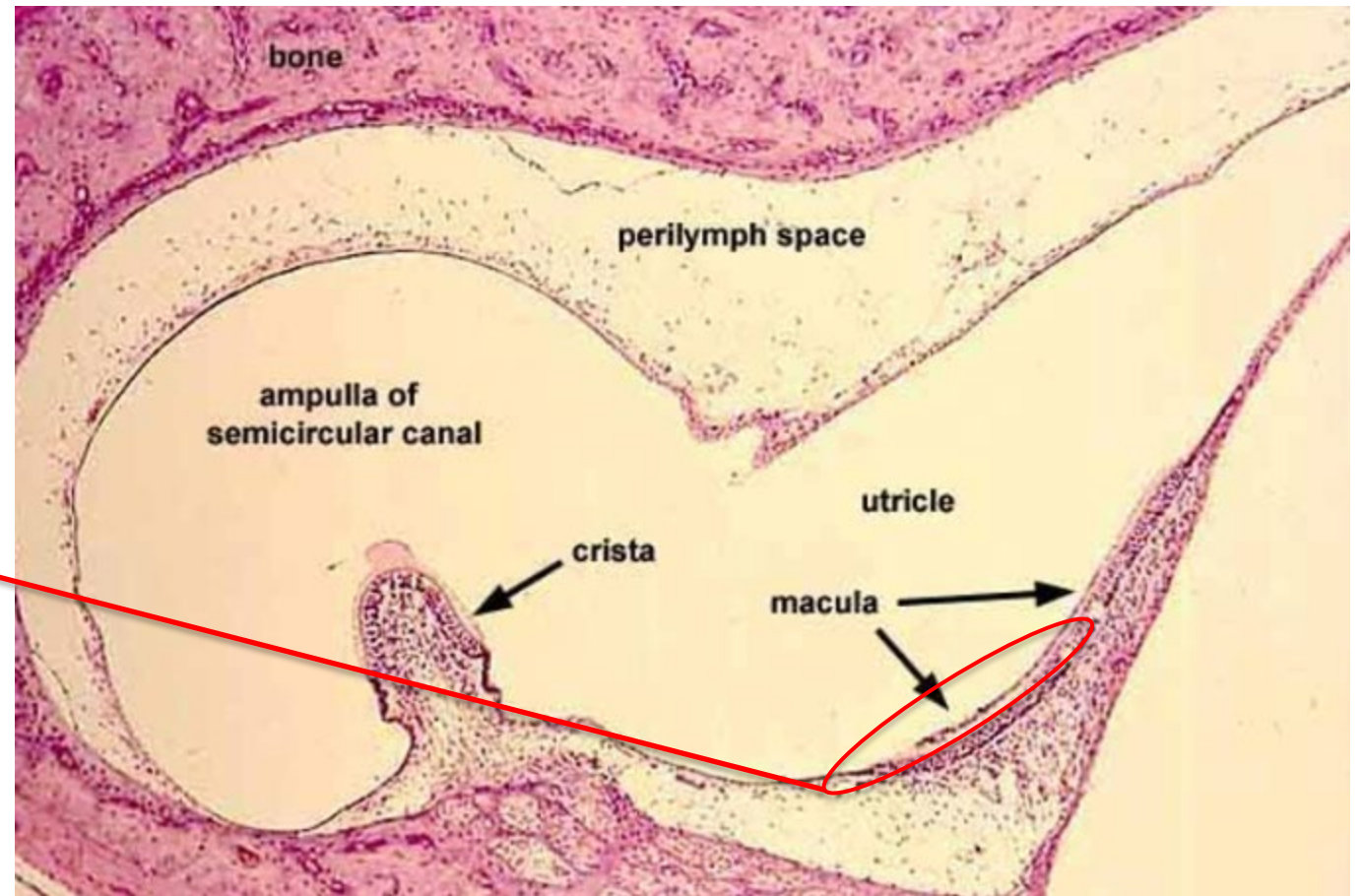
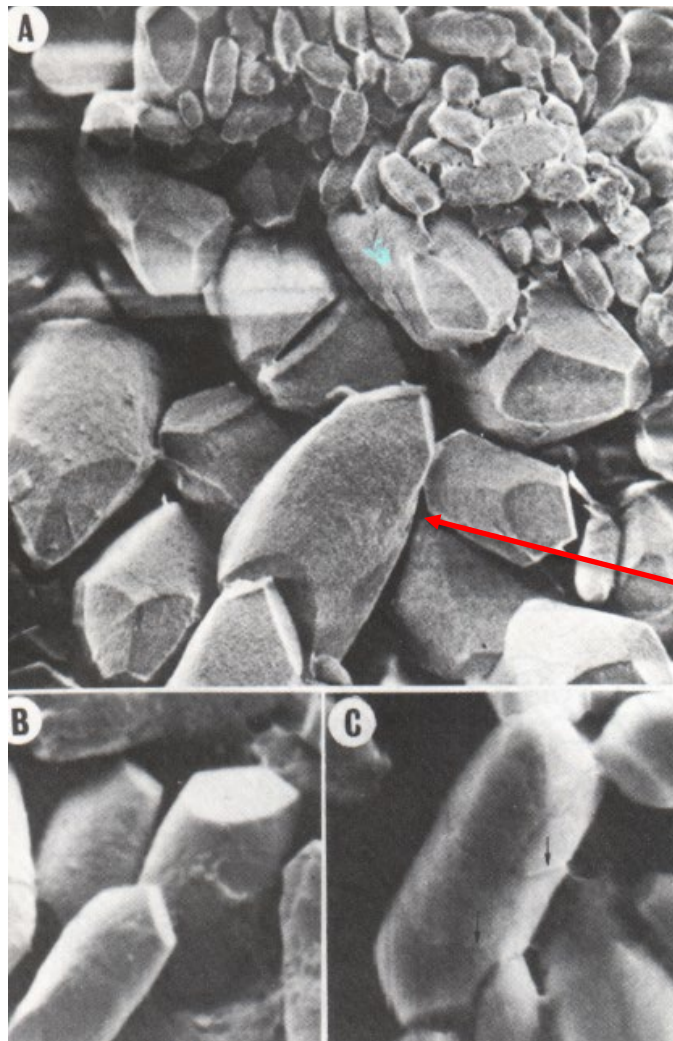


Histology

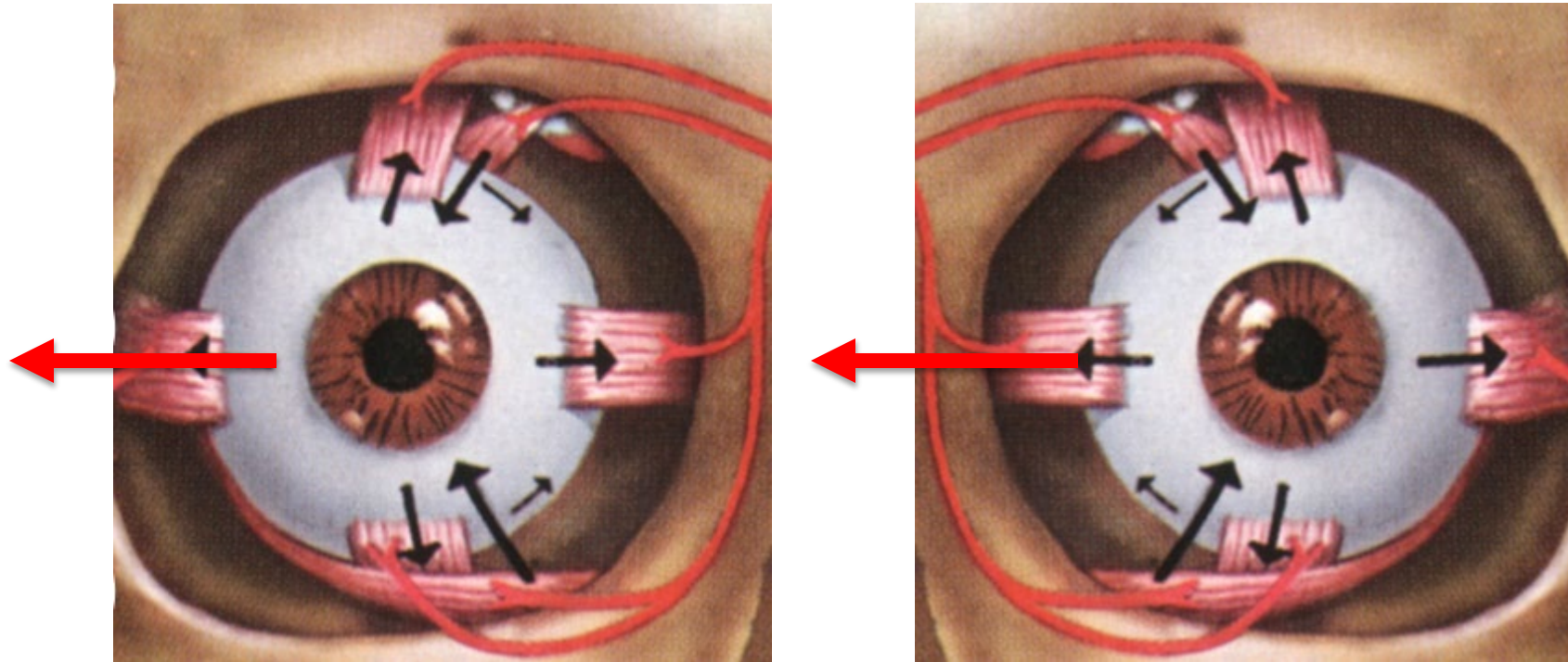


Atlas Wheater's, pgs 414-425, The ear Text Ross and Pawlina, Chapter 25, Ear

Otoconia / Otoliths



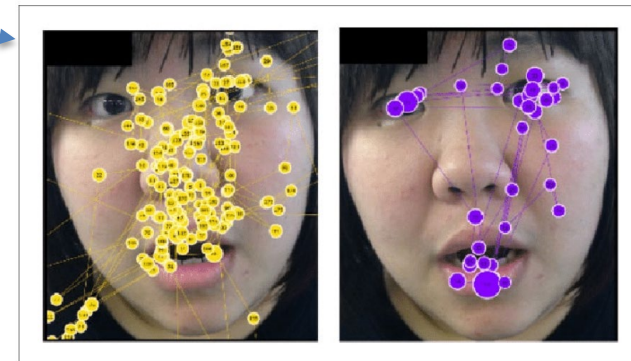
Extraocular Muscles



There are pairs. Lateral rectus of one eye and the medial rectus of the other eye will both contract to move the eye to the right in this figure. Also, consider that the opposite lateral rectus of one eye and the medial rectus of the other eye are relaxed.

What Extraocular Muscles Do

1. Pendular (or smooth) pursuit – following object, no VOR and no vestibular contribution
 - Follow target
 - Voluntary
2. Saccadic pursuit – we tend to use this for reading, or scanning a novel image at different points
 - Rapid repositioning
 - Voluntary
3. Optokinetic pursuit – need to induce optokinetic reflex/nystagmus
 - Perceives motion in visual field
 - Involuntary



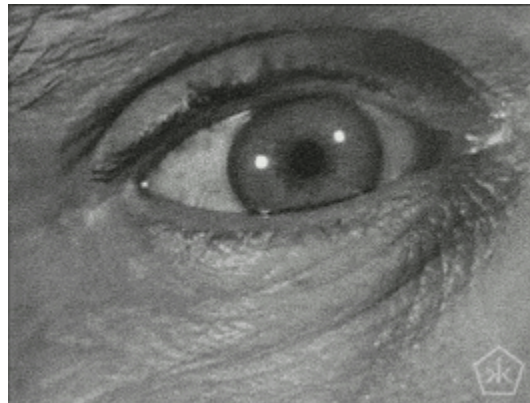
Gaze plots of an individual with ASD (left) and a control (right) viewing a face showing fear.

Corrective Saccade

- Even in the absence of head movement, the eyes constantly adapting to the world around us to keep objects focused on the fovea of the eye.
- The cerebellum is constantly working to keep objects clear on the fovea. Without this, vision would be blurred.
- The ears (vestibular) and eyes (via cerebellum) have to work together. If the ear has an abnormality and doesn't send a message to the brain, the cerebellum tries to step in for the ear with a corrective saccade.
- When the head moves, the vestibular system has equal opposite eye response, telling the eyes how fast, how far it needs to move.

Nystagmus

- Nystagmus – derived, *nystagmo-*, from behavior after drinking too much wine and people's heads would slowly roll back and then snap back up.
- Nystagmus is involuntary and has a fast phase (jerk) and a slow phase (drift), and nystagmus has a beat. It starts slow in one direction, and then quickly jerks back to the other direction.
- Never normal to have a nystagmus, but doesn't mean you can't induce it or it can't be congenital.



From GIPHY.com



Symptoms of Vertigo and Dizziness

What is Dizziness?

Dizziness is a feeling of being lightheaded, unsteady, and faint.

Sometimes the feeling is mild and goes away quickly.

Other times it can be severe and come along with other symptoms, like a headache and throwing up.

What is Vertigo?



The best definition is a hallucination of external rotation or movement. It is a form of dizziness, but it feels like the room is spinning around you and you have trouble staying steady on your feet.



Vertigo is not a diagnosis, it is a symptom.



Vertigo can be a symptom of several health conditions affecting your inner ear and your vestibular system.



Common Vertigo and Balance Issues Related to TBI

Dizziness, Vertigo, Falls...

- Falls are the leading cause of TBI and bone fractures
- Falls are the leading cause of accidental deaths in adults 65+ years
 - 6th leading cause of death for the elderly
 - 60% of fall-related deaths occur in 75+ years
 - 20% who sustain a hip fracture will die within a year
 - Of those, 20% will require placement within long-term care facility
 - 49% of those who sustain hip fracture will die within 6 months
 - Falls among older persons accounted for 2,800,000 emergency room visits

Dizziness, Vertigo, Falls...

- 85% of vertigo and balance dysfunction may be inner ear related, could be a simple mismatch of neural firing rates between the two vestibular systems
- Dizziness is the #1 complaint of persons over age 70
 - 50% of individuals over age 70 will experience BPPV
 - Older adults with BPPV have greater incidence of depression, higher likelihood of anxiety, 3x likelihood of falls, and reduced Activities of Daily Living (ADL)
 - Consider also: polypharmacy, blood pressure, hypo or hyperglycemia, heart problems, lung problems, peripheral arterial issues, etc.

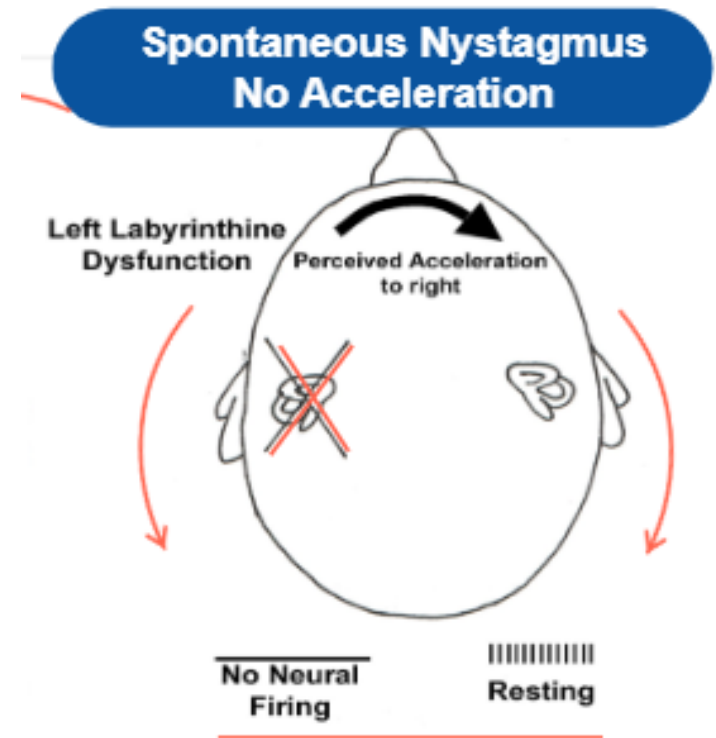
Related to Head Injury

- Vertigo is common symptom for those who have experienced blunt trauma to the head, neck, and craniocervical junction
- Motor vehicle accidents (MVA), falls, assault, and contact sports may cause vertigo
- Great variability among post-trauma patients, and depends on which anatomical structures are affected
- Most common pathologic condition associated with head trauma is BPPV, which occurs in about 28% of individuals with head trauma (Hoffer et al. 2004)

Vestibulopathy



- A vestibulopathy causes resting and stimulated firing rates on the ipsilateral vestibular nuclei to decrease
- The ipsilateral side can no longer inhibit the contralesioned vestibular nuclei
- The contralateral vestibular nuclei is now relatively hyperactive and has more inhibitory power on the ipsilesioned side
- This imbalance causes true vertigo



Can result in oscillopsia, imbalance, fear, avoidance, anxiety, depression, etc.

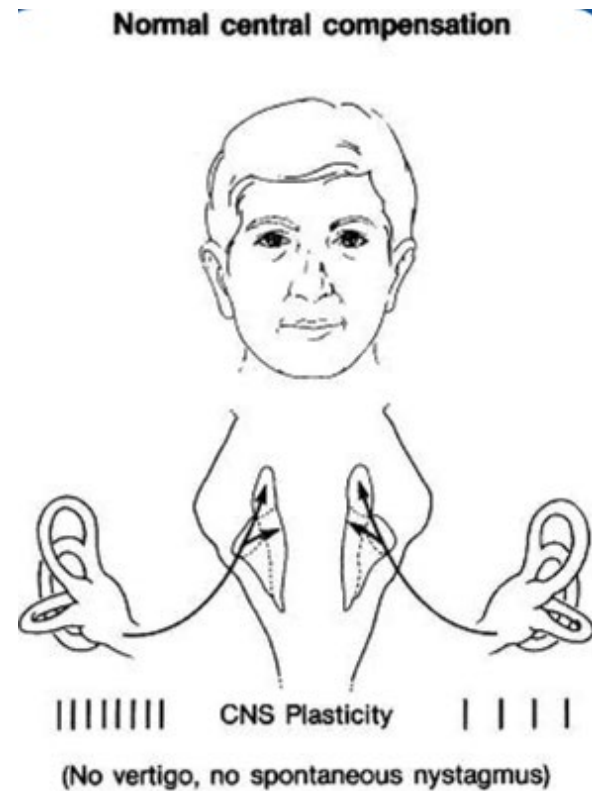
In order to resolve this, the brain needs movement to recalibrate. "The brain can't fix what the brain can't see."

Central Vestibular System

- Central vestibular system begins with the vestibular nuclei
- When we talk about different diseases and disorders, they are mostly destructive. They are vestibulopathies that cannot be repaired. When the ears sustained damage, it stays damaged.
- Thus, vestibular rehabilitation therapy is really about brain therapy, or compensation therapy. **Compensation for vestibulopathy happens in the brain.**

End Result of Central Vestibular Compensation

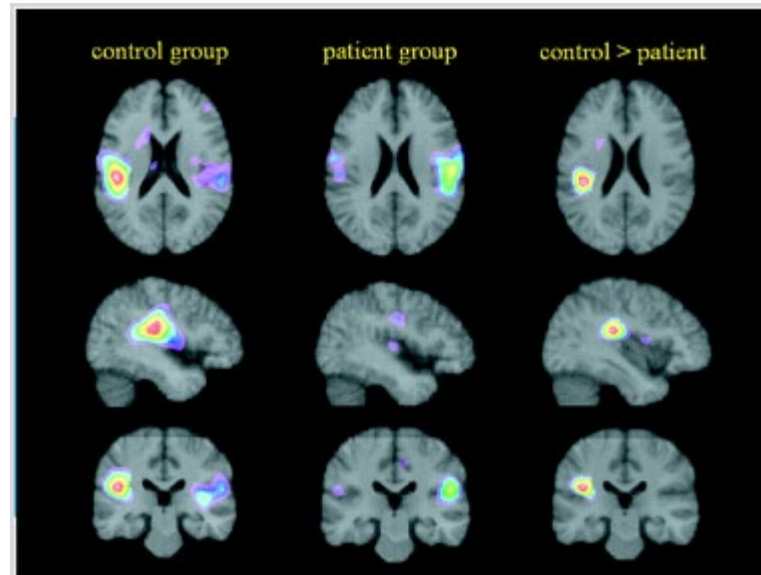
- Although the firing rate of the ipsilesioned vestibular nuclei is lower than the contralateral side, the CNS has deemed this “the new normal”



No change in firing rate, but the brain has compensated!

Recent Research, The Big Picture

- Cortical involvement (concentrated tasks) during vestibular rehabilitation activities has been shown to facilitate changes in brain chemistry, helping promote compensation.
- We can't change the ears, but we can compensate for the ears using the brain.



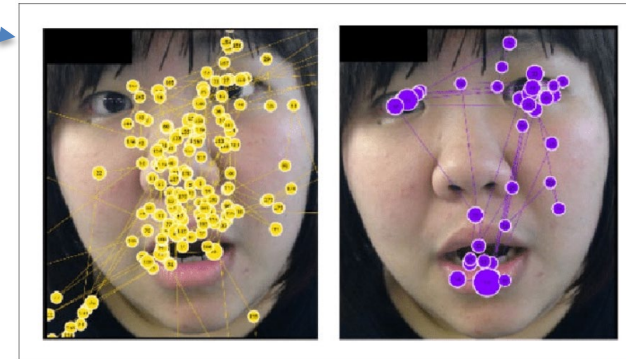
Cortical Projection of Peripheral Vestibular Signaling Miklós Emri, Mihály Kisely, Zsolt Lengyel, László Balkay, Teréz Márián, László Mikó, Ervin Berényi, István Sziklai, Lajos Trón, Ágnes Tóth Journal of Neurophysiology Published 1 May 2003 Vol. 89 no. 5, 2639-2646 DOI: 10.1152/jn.00599.2002

What Extraocular Muscles Do

If there are disorders here, it can be CNS indicators

... could be vestibular or CNS indicators

1. Pendular (or smooth) pursuit – following object, no VOR and no vestibular contribution
 - Follow target
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2. Saccadic pursuit – we tend to use this for reading, or scanning a novel image at different points
 - Rapid repositioning
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Gaze plots of an individual with ASD (left) and a control (right) viewing a face showing fear.

CNS Mediated Causes of Nystagmus

- Can be any eye direction
 - Can be direct-changing
 - Can be vertical
 - Not typically associated with true vertigo, rather internal sensations of motion or no symptoms at all
 - Does not typically fatigue
- A brainstem infarct, however, could be the cause of sudden onset CNS mediated nystagmus, could be tied to other neurological insults, paraplegias, dizziness, confusion, hemiplegias, etc. Seem to have no particular pattern of beating direction. It's the CNS that makes it unpredictable.
 - Downbeat nystagmus example shown with a brainstem stroke, but suspected of having anterior canal BPPV referred by someone else. However, the nystagmus was persistent and non-fatiguing, so it's not BPPV. BPPV treatments won't cause harm, but will have no bearing on this nystagmus. Also, the patient may have zero symptoms or complaints!
 - Positional nystagmus example shown with ageotropic nystagmus. May or may not have dizziness.

NYSTAGMUS PATTERN SUMMARY



Peripheral Nystagmus Patterns	Central Nystagmus Patterns
Direction Fixed (horizontal or torsional)	Vertical Nystagmus (Upbeating or downbeating)
Suppression with Fixation	No suppression with Fixation
Follows Alexander's Law Uni-directional	Direction-changing Nystagmus
Follows Ewald's Law (excitation>inhibition) Vestibular hypofunction-Intact Ear BPPV-Involved Ear	Direction-changing Nystagmus Non-fatiguing Nystagmus

Also, not associated with strong dizziness or vertigo.

Gans, R, Roberts R. Background, Technique, Interpretation, and Usefulness of Positional/Positional Testing. *Balance Function Assessment and Management*. San Diego: Plural, 2008. 173.

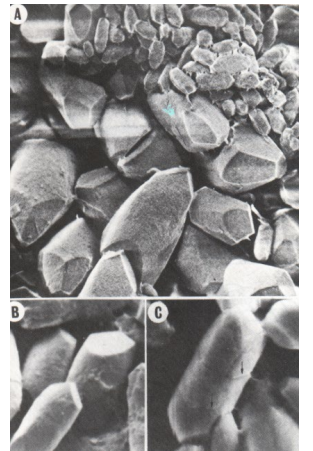
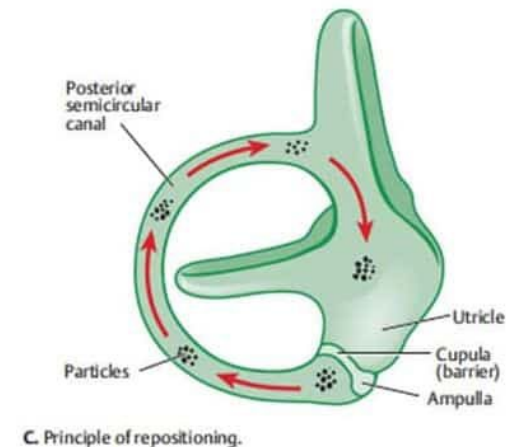


Other Inciting Events Related to Trauma

- Temporal bone fractures
- Concussion
- Anything related to auditory and vestibular insults by head trauma, such as:
 - Noise and acoustic blasts
 - Labyrinthine concussion
 - Benign Positional Paroxysmal Vertigo (BPPV)
 - Vestibular hypofunction
 - Barotrauma
 - Perilymphatic fistulas
 - Superior and Posterior Canal Dehiscence (SSCD and PSCD)
 - Persistent
- Potential cognitive impairment as a result of vestibular dysfunctions

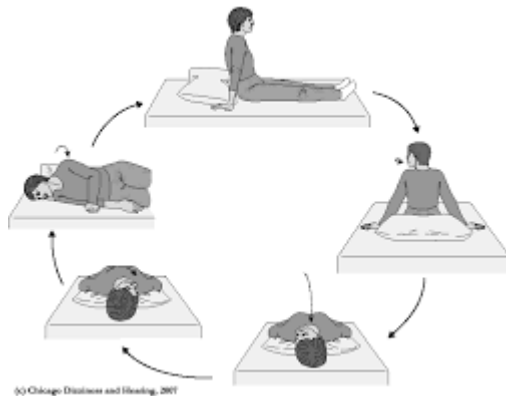
Benign Positional Paroxysmal Vertigo (BPPV)

- Pathophysiology
 - Otoconia dislocated to SSC (not a vestibulopathy!)
 - Causes sensitivity to changes in gravito-inertial vector
 - Most commonly affects posterior-SSC
- The dislodged otoconia causes a pull on the cupula
- Usually lasts about 15-20 seconds and stabilizes until the head moves again
- With untreated BPPV, fall risk goes up 3 fold!



BPPV

- May trouble the patient for years with periods of remission and exacerbation
- May last weeks to months with spontaneous recovery
- Typically about 25% have a spontaneous recovery
- BPPV may have a relationship with diabetes, migraine, cardiovascular, vitamin D3 deficiency



HIGHLY RESPONSIVE TO TREATMENT MANEUVERS

- PC-BPPV
 - Semont Liberatory Maneuver
 - Modified Canalith Repositioning Maneuver (modified Epley/Herdman et al)
 - Gans Repositioning Maneuver
- HC-BPPV
 - Casani Maneuver
 - Appiani Maneuver
 - Horizontal Hybrid Maneuver
 - BBQ/Log Roll Maneuver

Actual differences between these are the body mechanics

Superior Semicircular Canal Dehiscence (SSCD)

- Pathophysiology
 - Caused by defect of bone overlying the superior SSC
 - Located in floor of middle cranial fossa resulting in a “third window”
- Abnormal connection between the inner ear and middle cranial fossa results in a variety of symptoms, including hearing loss, tinnitus, autophony, and vertigo
- Vertigo is triggered by loud sounds

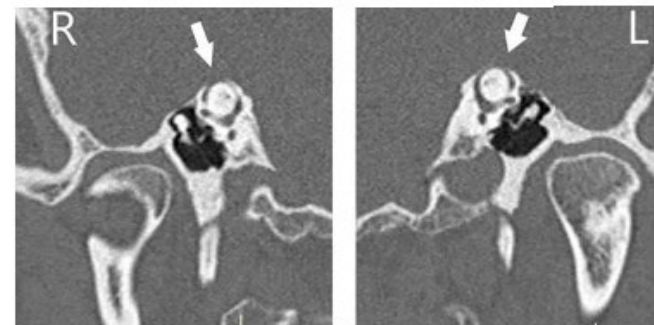


Figure 2: CT scan of the temporal bones of the patient, and images were reformatted in the planes of the right (R) and left (L) SSCs (Pöschl view); bilateral dehiscence (arrows) is noted.

Superior Semicircular Canal Dehiscence (SSCD)

Figure 1. Air- and unmasked bone-conduction thresholds in a 40-year-old female with superior semicircular canal dehiscence syndrome.

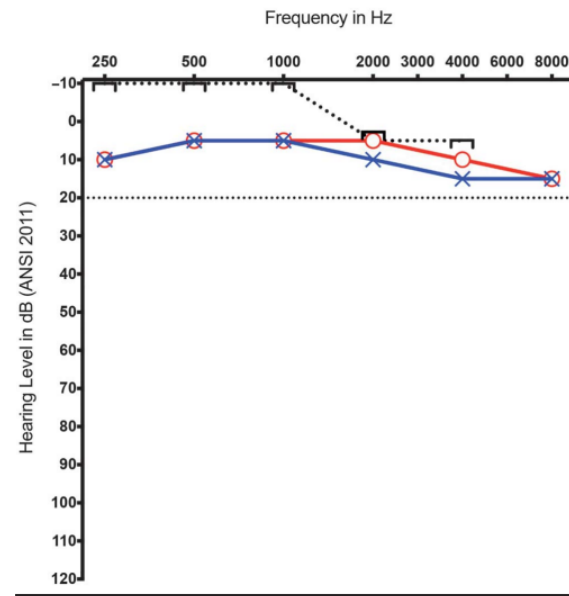


Figure 3. A high-resolution computerized tomography image of a focal deficiency of the right superior semicircular canal (red arrow) compatible with dehiscence in a 40-year-old female.

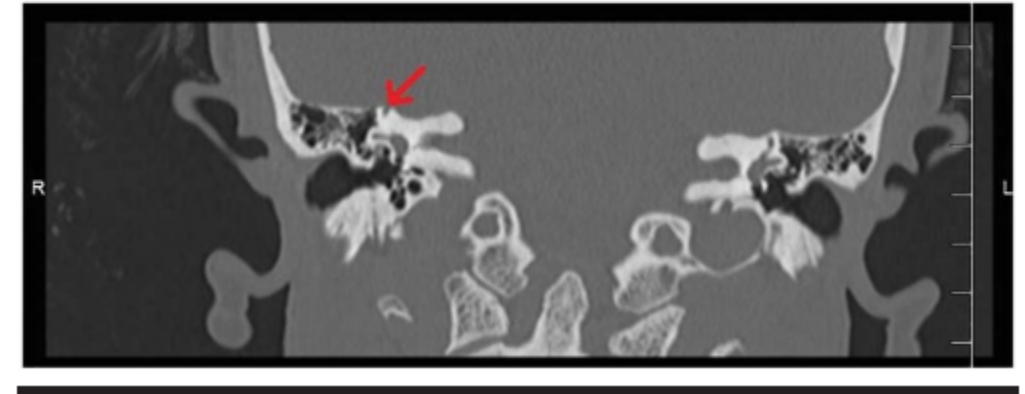
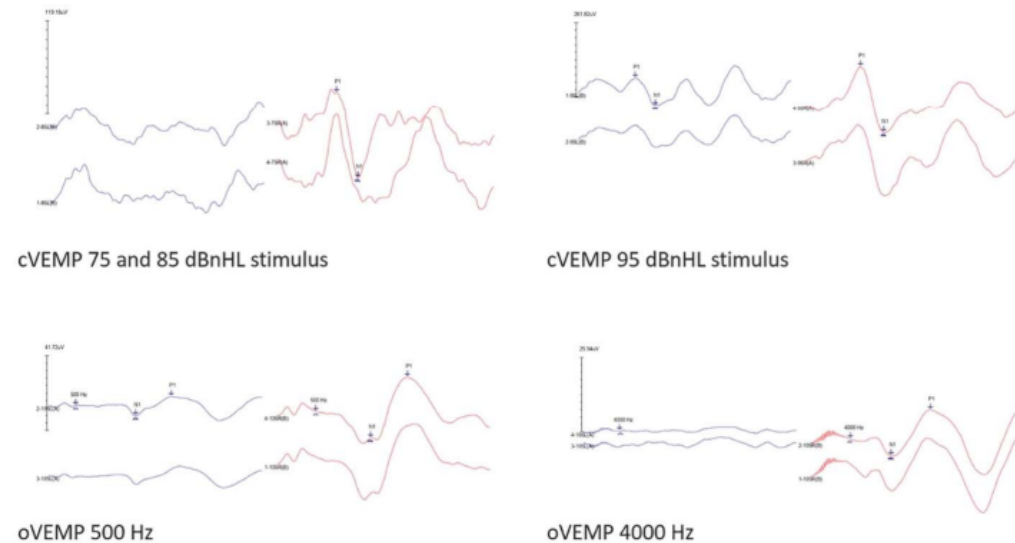


Figure 2. Cervical (cVEMP) and ocular (oVEMP) vestibular-evoked myogenic potential test results in a 40-year-old female with superior semicircular canal dehiscence syndrome.



Persistent Postural-Perceptual Dizziness (PPPD)

- Pathophysiology
 - Not fully understood
 - Follows vestibular insult, medical event, and/or acute psychological distress
 - Changes in posture, motion of self, and visually demanding environments can provoke symptoms of dizziness, unsteadiness, and non-room spinning vertigo
 - Heightened in individuals with high anxiety and body vigilance
 - Traumatic head injury and BPPV known to provoke
- The five criteria to diagnose PPPD:
 - Non-room-spinning, unsteadiness, and difficulties with a balance must be present for most of the days over a 90 period.
 - Symptoms cannot be provoked but can be exacerbated by changes in position and exposure to certain stimuli.
 - PPPD must be preceded by a condition with acute, episodic, or chronic vestibular symptoms.
 - Patients with PPPD must be troubled by their symptoms and must have impairment to some degree.
 - Finally, PPPD cannot be explained by another medical condition or disorder.

Association between saccule and semicircular canal impairments and cognitive performance among vestibular patients (Pineault et al., 2020)

- 54 patients presenting to the Johns Hopkins Neurotology Clinic between August 2017 and March 2018 were enrolled in the study. The patients were 59.3% female, with a mean age of 55.9 ± 15.6 years.

Vestibular Deficits

- cVEMP Test (saccule function)
 - Among 40 patients with cVEMP testing, 31.7%, 14.6%, and 55.8% of patients had bilaterally present, unilaterally absent, and bilaterally absent cVEMP responses, respectively.
- Caloric testing (horizontal semicircular canal function)
 - Among 43 patients with caloric irrigation testing, 55.8%, 32.6%, and 11.6% had normal SCC function, unilateral SCC impairment, and bilateral SCC impairment, respectively.

Cognitive Test Results

- Among patients presenting with dizziness and vertigo to a tertiary care Neurotology clinic, bilateral saccule impairment, determined by bilaterally absent responses on cVEMP testing, was associated with **significantly higher number of errors made on the Benton Visual Retention Test Part-C (BVRT-C).**
- Furthermore, **bilateral SCC impairment was significantly associated with more errors made on the BVRT-C.**
- Importantly, **case-control analyses showed that patients had significantly poorer cognitive performance compared to controls, notably on the Trail Making Test-Part B (TMT-B) test.**

CONCLUSIONS: This study further demonstrates the associations between severity (i.e. bilateral and unilateral) and organ-specific (e.g. saccule and SCC) impairments among patients with vestibular disease and cognitive performance.

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Thank You!

