VESTIBULAR AND VISUAL DYSFUNCTION AFTER CONCUSSION

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GOALS

- Define commonly used terms related to post-concussive vestibular and visual deficits
- Outline the incidence and prevalence of post-concussive vestibular and visual deficits
- Discuss the anatomic and pathologic correlates of post-concussive vestibular and visual deficits
- Develop the framework for interventions for post-concussive vestibular and visual deficits
DEFINITIONS: CONCUSSION

- **Concussion** is a complex pathophysiologic process induced by traumatic forces secondary to direct or indirect impulsive forces to the head that disrupts the function of the brain.

- Concussion is defined as an alteration or loss of consciousness for up to 30 minutes with associated loss of memory surrounding the event (post-traumatic amnesia) for up to 24 hours.

- Transient (<24 hours) neurologic sequelae may also be present, including numbness, dizziness, cognitive deficits, discoordination, and alterations in special senses.

- This disturbance of brain function is typically associated with normal structural neuroimaging findings.
mTBI = Concussion

Concussion = mTBI
In addition to traditional concussive injury, there is an increasing “awareness” of potentially concussive events (PCE).

A PCE is defined as an impulsive force to the head of sufficient intensity that results in acute or chronic symptoms in some individuals, but remain asymptomatic (“subclinical”) in others - no demonstrable neurologic or symptomatic effect.

The term PCE is often applied to all traumatic events that either could have caused a concussion (with altered/lost consciousness) but did not and those that did produce a concussion.
DEFINITIONS:
POST-CONCUSSIVE SYMPTOMS

- Persistent physical, cognitive, emotional, and/or sleep-related symptoms occur in more than half of concussions, but usually resolve in 1-4 weeks.

- Symptoms presenting in the first 1-2 weeks after a concussion are commonly ascribed to the concussion.

- Continued symptoms after 3 months may be labeled as Post-Concussive Syndrome and may occur in up to 30% of injuries.
DEFINITIONS: POSTCONCUSSIVE SYMPTOMS

- Ongoing symptoms are either a prolonged version of the concussion pathophysiology or a manifestation of other processes, such as cervical injury, migraine headaches, depression, chronic pain, vestibular dysfunction, visual dysfunction, or some combination of conditions.

- The pathophysiology of ongoing symptoms from the original concussion injury may reflect multiple causes: anatomic, neurometabolic, and physiologic.

- Neurometabolic Cascade
  - Release of neurotransmitters- Glutamate
  - Calcium influx blocking oxygen preventing cellular respiration
  - K+ efflux cause vasoconstriction
  - This prevents fuel, glucose, from getting to the cells.
  - This leads to cellular death and dysfunction
RISK FACTORS FOR PERSISTENT CONCUSSIVE SYMPTOMS

- Presenting with four or more symptoms was associated with double the risk for concussive symptoms ≥1 week for both football and non-football players.
- History of prior concussion was associated with double the risk for concussive symptoms ≥1 week in football players only.
- Several symptoms were associated with concussive symptoms ≥1 week in all athletes: drowsiness, nausea and concentration difficulties.
- Sensitivity to light and noise was associated with concussive symptoms ≥1 week in non-football players only.
- Amnesia was associated with concussive symptoms ≥1 week in males, but not females.
- Loss of consciousness was not significant.

The body maintains balance with sensory information from three systems:
- Vision
- Proprioception
- Vestibular system

Sensory input from these three systems is integrated and processed by the brainstem. In response, feedback messages are sent to the eyes to help maintain steady vision and to the muscles to help maintain posture and balance.
DEFINITIONS: IMBALANCE

- Dizziness, vertigo and disequilibrium are common terms used to describe vestibular dysfunction as well as other difficulties.

- They are all symptoms that can result from a peripheral vestibular disorder (a dysfunction of the balance organs of the inner ear) or central vestibular disorder (a dysfunction of one or more parts of the central nervous system that help process balance and spatial information).

- Although these three symptoms can be linked by a common cause, they have different meanings, and describing them accurately can mean the difference between a successful diagnosis and one that is missed.
DEFINITIONS: IMBALANCE

- **Disequilibrium** means unsteadiness, imbalance, or loss of equilibrium that is often accompanied by spatial disorientation.

- **Dizziness** describes a sensation of lightheadedness, faintness, or unsteadiness. Dizziness does not involve a rotational component.

- **Vertigo** has a rotational, spinning component, and is the perception of movement, either of the self or surrounding objects.
DEFINITIONS: VISUAL DEFICITS

- Visual field defects
- Versional - Disturbances of gaze stabilization
  - Saccades - Quick, simultaneous movements of both eyes in the same direction.
  - Fixation - maintaining the gaze in a constant direction
  - Smooth Pursuit - tracking by the eyes of a slowly moving object at a steady coordinated velocity
- Vergence – Disturbances of focus
  - Blurred vision/Accommodation
  - Diplopia/Ocular motility
DEFINITIONS: VISUAL/AUDITORY “PAIN”

- Photophobia/Photosensitivity – Light sensitivity
- Hyperacusis – Sound sensitivity
- Tinnitus – Perception of sound when no sound is present
- Unclear peripheral and/or central neurologic etiologies.
DEFINITIONS: VISUAL AND AUDITORY PERCEPTUAL DEFICITS

- **Central Auditory Processing Deficit (CAPD)** – Inaccurate perception, interpretation or understanding of auditory stimuli, due to cortical injury of Primary Auditory Cortex.
  - Bilaterally at upper sides of the Temporal Lobes on the superior temporal plane, within the lateral fissure and comprising parts of Heschl's gyrus and the superior temporal gyrus, including planum polare and planum temporale (Brodmann areas 41, 42 and, and partially 22)

- **Central Visual Perceptual Deficits** - Inaccurate perception, interpretation or understanding of visual stimuli, due to cortical injury of Primary Visual Cortex.
  - Visual cortex includes areas of occipital, temporal and parietal lobes, as well as areas of the limbic cortex. The left cortex plays a major role in recognizing the meaning of common objects.
EPIDEMIOLOGY: CONCUSSIONS

- The CDC estimates that there are 3.5 million civilian concussions in the United States annually.
  - Up to 30% of individuals with concussion will continue to be symptomatic at 3 months (i.e., post-concussion syndrome) and 5% or more will demonstrate abnormalities on testing or by symptom report at one year post-injury.

- The DoD/VA estimates that 16% of all service members who served in the OEF/OIF conflicts experienced at least 1 concussion.

- 9% of all OEF/OIF Veterans seen for care have persistent symptoms related to these concussions.
The long term effects of a subclinical PCE’s, single concussion, multiple concussions (whether separated by a short or long period of time) are not know, nor is the impact of persistent post-concussive symptoms.

Recent reports suggest that a subset of individuals who sustain concussion are at an elevated risk to develop degenerative neurologic disorders (i.e., dementia, Parkinson’s disease, behavioral dysfunction).
EPIDEMIOLOGY: IMBALANCE

- 35% adults aged 40 years or older in the United States—approximately 69 million Americans—have experienced some form of vestibular dysfunction.

- 4% (8 million) of American adults report a chronic problem with balance, while an additional 1.1% (2.4 million) report a chronic problem with dizziness alone.


Epidemiology: Imbalance

- Eighty percent of people aged 65 years and older have experienced dizziness, and BPPV, the most common vestibular disorder, is the cause of approximately 50% of dizziness in older people.

- Overall, vertigo from a vestibular problem accounts for a third of all dizziness and vertigo symptoms reported to health care professionals.

Ator GA. Vertigo—Evaluation and Treatment in the Elderly.


EPIDEMIOLOGY: POST-CONCUSSIVE IMBALANCE

- Nearly one-quarter of the patients with acute concussion present with dizziness. Reports of 80% after blast injury.

- Causes include;
  - Inner ear disorders
  - Benign Paroxysmal Positional Vertigo
  - Labyrinthine concussion
  - Perilymphatic fistula
  - CNS Disorders
  - Post-traumatic migraine
  - Brainstem concussion
  - Autonomic dysregulation (Orthostatic Hypotension)
  - Occulomotor abnormalities
  - Seizures
  - Psychological disorders
  - Musculoskeletal disorders
EPIDEMIOLOGY: VISUAL DISTURBANCES

- Sensorimotor vision symptoms after mTBI are reported in frequencies ranging from 10 to 85%, depending upon the nature of the vision deficit and the criteria used in the study.

- Subtle visual deficits are often overlooked, but may have subtle to profound impacts on basic (reading, walking) to advanced (driving, sports, working) functional tasks.


EPIDEMIOLOGY: VISUAL DEFICITS

- Visual field defects (35% of all TBI, rare with mTBI)
- Versional - Disturbances of gaze stabilization (40-80% mTBI)
  - Saccades - Quick, simultaneous movements of both eyes in the same direction.
  - Fixation - maintaining the gaze in a constant direction (rare with mTBI)
  - Smooth Pursuit - tracking by the eyes of a slowly moving object at a steady coordinated velocity
- Vergence – Disturbances of focus
  - Blurred vision/Accommodation (10-40% of mTBI)
  - Diplopia/Ocular motility (40-56% mTBI)
ANATOMY OF BALANCE

- The vestibular system contributes to balance and the sense of spatial orientation.

- As movements consist of rotations and translations, the vestibular system comprises two components:
  - the semicircular canal system, which indicate rotational movements
  - the otoliths, which indicate linear accelerations.

- The vestibular system sends signals primarily to the neural structures that control eye movements (the anatomical basis of the vestibulo-ocular reflex) and to the muscles that keep one upright.
The vestibular system contains three semicircular canals in each labyrinth.

They are approximately orthogonal (right angles) to each other, and are called the horizontal (or lateral), the anterior semicircular canal (or superior) and the posterior (or inferior) semicircular canal. Anterior and posterior canals may be collectively called vertical semicircular canals.

Movement of fluid within the horizontal semicircular canal corresponds to rotation of the head around a vertical axis (i.e. the neck), as when doing a pirouette.

The anterior and posterior semicircular canals detect rotations of the head in the sagittal plane (as when nodding), and in the frontal plane, as when cartwheeling. Both anterior and posterior canals are orientated at approximately 45° between frontal and sagittal planes.
While the semicircular canals respond to rotations, the otolithic organs sense linear accelerations.

Humans have two of these organs on each side, one called utricle, the other saccule. Otoconia crystals (otoliths) float within the organs.

Most of the utricular signals elicit eye movements, while the majority of the saccular signals projects to muscles that control our posture.

The otoconia crystals in the otoconia layer rest on a viscous gel layer, and are heavier than their surroundings. Therefore they get displaced during linear acceleration, which in turn deflects the ciliary bundles of the hair cells and thus produces a sensory signal.
CENTRAL PROCESSING
CENTRAL PROCESSING

- Signals from the semicircular canals and otolithic organs project via Cranial Nerve VIII to the 4 main vestibular nuclei in the brainstem.

- The vestibular nuclei on either sides of the brain stem exchange signals regarding movement and body position. These signals are sent down the following projection pathways.
  - Cerebellum: Signals sent to the cerebellum are relayed back as muscle movements of the head, eyes, and posture. The VOR is also modulated here.
  - Nuclei of Nerves III, IV, and VI through MLF: Signals sent to these nerves cause the vestibular-ocular reflex. They allow for the eyes to fix on a moving object while staying in focus.
CENTRAL PROCESSING

- The vestibular nuclei on either sides of the brain stem exchange signals regarding movement and body position. These signals are sent down the following projection pathways.
  - Reticular Formation: Signals sent to the reticular formation signal the new posture the body has taken on and how to adjust circulation and breathing due to body position.
  - Spinal Cord: Signals sent via medial; and lateral vestibulospinal tracts to the spinal cord allow quick reflex reactions to both the limbs and trunk to regain balance.
  - Thalamus: Signals sent to the thalamus allow for head and body motor control as well as being conscious of body position.
Central Processing

- Autonomic: Signals sent through efferent projections onto the solitary nucleus of cranial nerves IX and X coordinate vestibulo-sympathetic reflexes that modulate autonomic changes in blood circulation, heart rate and respiratory rate.

- Projections from the Vestibular Nuclei also go to areas of the cortex, however their role is unclear.

- Persistent dizziness and vertigo with acute injury, disease or dysfunction of the vestibular system may therefore be due to:
  - Permanent damage and hypofunction/dysfunction of the vestibular system
  - Dysfunction of the VIII cranial nerve
  - Dysfunction of the visual or peripheral sensory system
  - Dysfunction of the central structures
  - Recalibration of the cerebellum (? or cortex)
ANATOMY OF VISION

- Light passes through the refractive components (i.e., the cornea, aqueous chamber, crystalline lens, and vitreous) to reach the retina.
- The photoreceptors at the retina transmit signals at the level of the optic nerve via retinal ganglion cell (RGC).
- The RGCs maintain long axons that comprise the optic nerve, leaving the retina and proceeding to the optic chiasm.
- At the chiasm, the visual information separates into right and left hemi-fields of visual space; this divided information travels post-chiasm as optic tracts to the lateral geniculate nucleus (LGN), ultimately becoming optic radiations, post-LGN.
CENTRAL PROCESSING

- The vast majority of the axons continue onwards to the occipital cortex (V1).
- At V1, the neural signals for the primary visual pathway begin the transformation into a high-resolution, neural image with appropriate form, color vision, and contrast, in addition to maintaining peripheral vision integrity.
- Parallel processing begins at this level with a ventral pathway and dorsal pathway.
  - The ventral visual pathway is predominantly for object identification, with its axons eventually reaching the posterior-inferior temporal lobe.
  - The dorsal visual pathway is predominantly for spatial representation and visually-guided action, with its axons eventually reaching the parietal lobe.
The vestibulo-ocular reflex (VOR) is a reflex eye movement that stabilizes images on the retina during head movement by producing an eye movement in the direction opposite to head movement, thus preserving the image on the center of the visual field. For example, when the head moves to the right, the eyes move to the left, and vice versa.

Since slight head movements are present all the time, the VOR is very important for stabilizing vision: patients whose VOR is impaired find it difficult to read, because they cannot stabilize the eyes during small head tremors. The VOR reflex does not depend on visual input and works even in total darkness or when the eyes are closed.
The optokinetic reflex (OKR) responds to visual motion stimulation. It is observed when one follows a moving object with their eyes. Once that object moves out of their field of vision, their eyes move back to the original position when first seeing the object.

The cervico-ocular reflex (COR) is a rotational eye reflex elicited by neck musculature and cervical spine proprioception that works in conjunction with the VOR and OKR.

The vestibulocolic reflex (VCR) is involved in head stabilization in space.
### Central vs. Peripheral Imbalance

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<thead>
<tr>
<th></th>
<th>Central</th>
<th>Peripheral</th>
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</thead>
<tbody>
<tr>
<td><strong>Onset</strong></td>
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<td>Sudden (though some latency)</td>
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<tr>
<td><strong>Frequency</strong></td>
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<td>No</td>
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<td><strong>Fatigueable</strong></td>
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<td>Yes</td>
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<tr>
<td><strong>Visual Supression</strong></td>
<td>No</td>
<td>Yes</td>
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<td><strong>Photophobia</strong></td>
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<td><strong>Phonophobia</strong></td>
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<td><strong>Hearing Loss</strong></td>
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PERIPHERAL VESTIBULAR DISORDERS

- BPPV
- Perilymph Fistula (PLF)
- Labyrinthine Concussion
- Post-Traumatic Endolymphatic Hydrops
  - Meniere’s-like presentation
- Vestibular Hypofunction/Dysfunction
- Cervical vertigo
- Eighth nerve complex
BPPV: ETIOLOGY

- BPPV is the most common vestibular disorder
  - 2.4% of all people will experience it at some point in their lifetimes.

- BPPV accounts for at least 20% of diagnoses made by physicians who specialize in dizziness and vestibular disorders, and is the cause of approximately 50% of dizziness in older people.

- The most common cause of BPPV in people under age 50 is head injury and is presumably a result of concussive force that displaces the otoconia.
BPPV: DEFINITION

- Sudden, intermittent and intense episodes of vertigo lasting for seconds and triggered by certain movements of the head.

- BPPV consists of brief, intermittent episodes of nystagmus, lightheadedness and/or vertigo without hearing loss; the episodes are provoked by head movement in the same planes as the semi-circular canals, dislodging displaced otoconia.

- >80% time, it is the posterior canal that is affected. In certain head positions, these particles shift and create a fluid wave which displaces the cupula of the canal affected, which leads to dizziness, vertigo and nystagmus.
BPPV: PATHOLOGY

- BPPV occurs as a result of otoconia, tiny crystals of calcium carbonate that are a normal part of the inner ear’s anatomy, detaching from the otolithic membrane in the utricle and collecting in one of the semicircular canals.

- When the head is still, gravity causes the otoconia to clump and settle.

- When the head moves, the otoconia shift. This stimulates the cupula to send false signals to the brain, producing vertigo and triggering nystagmus (involuntary eye movements).
BPPV: TESTING

- BPPV is diagnosed based on medical history, physical examination, the results of vestibular and auditory (hearing) tests, and lab work to rule out other diagnoses.

- Vestibular tests include the Dix-Hallpike maneuver and the Supine Roll test.

- These tests allow a physician to observe the nystagmus elicited in response to a change in head position.

- The problematic semicircular canal can be identified based on the characteristics of the observed nystagmus.
Frenzel goggles, especially of the type using a TV camera, are sometimes used as a diagnostic aid in order to magnify and illuminate nystagmus.

If electronystagmography (ENG) is employed to observe nystagmus with position changes, it is important that the equipment used is capable of measuring vertical eye movements.

Magnetic resonance imaging scan (MRI) may be used to rule out other problems such as a stroke or brain tumor, but such scans are not helpful in diagnosing BPPV.

Auditory tests may help to pinpoint a specific cause of BPPV, such as Ménière’s disease or labyrinthitis.
A perilymph fistula is a tear or defect in one or both of the small, thin membranes between the middle and inner ears. These membranes, the oval window and the round window, separate the middle ear from the fluid-filled inner ear.

The changes in air pressure that occur in the middle ear (for example, when your ears “pop” in an airplane) normally do not affect your inner ear.

However, when a fistula is present, changes in middle ear pressure will directly affect the inner ear, stimulating the balance and/or hearing structures within and causing typical symptoms.
PERILYMPH FISTULA

- The symptoms of perilymph fistula may include:
  - Dizziness
  - Vertigo
  - Imbalance
  - Nausea
  - Vomiting.
  - Ringing or fullness in the ears
  - Hearing loss

- Most people with fistulas find that their symptoms get worse with changes in altitude (elevators, airplanes, travel over mountain passes), air pressure (weather changes), and with exertion/activity.
If symptoms are severe and have not responded to conservative treatment (bed rest), or if progressive hearing loss has occurred, surgical repair of the fistulas may be required. This procedure involves placing a graft over the fistula defect in the oval and/or round window.

Persons with fistulas should avoid lifting, straining, bending over, or any activity that would increase head pressure, since all of these will worsen symptoms and prevent the fistula from healing. It is also important to avoid air pressure changes as these changes will tend to worsen symptoms.
LABYRINTHINE CONCUSSION

- Posttraumatic vertigo that resolves spontaneously over time, after other diagnoses have been excluded, is known as labyrinthine concussion.

- Typically presents with abrupt hearing loss and persistent vertigo following head trauma in the absence of a temporal bone fracture.

- Symptoms generally can improve over a few days and sensations of persistent vertigo will eventually transition to movement-induced vertigo.

- The pathophysiology of labyrinthine concussion is not well-defined or distinguished from BPPV.
The mechanism of posttraumatic Ménière syndrome, aside from the disruption of the endolymphatic duct secondary to a temporal bone fracture, is thought to be caused by bleeding into the inner ear followed by a disturbance of fluid transport.

In a normal inner ear, the endolymph is maintained at a constant volume and with specific concentrations of sodium, potassium, chloride, and other electrolytes. This fluid bathes the sensory cells of the inner ear and allows them to function normally.

In an inner ear affected by hydrops, these fluid-system controls are believed to be lost or damaged.
This may cause the volume and concentration of the endolymph to fluctuate in response to changes in the body’s circulatory fluids and electrolytes.

Symptoms typical of hydrops include pressure or fullness in the ears (aural fullness), tinnitus (ringing or other noise in the ears), hearing loss, dizziness, and imbalance.

Compared with primary (viral/idiopathic), secondary (post-traumatic) Meniere’s disease tends to have continuous symptoms, that are less violent in nature and less likely to cause long-term damage to hearing and balance.
VESTIBULAR HYPOFUNCTION/DYSFUNCTION

- Vestibular hypofunction (VH) is a general term used to describe inability of vestibular system to adequately detect and convey signals to brainstem.
EIGHTH NERVE COMPLEX

- The eighth nerve complex is at risk for injury, even in cases of mild trauma, because of the shearing effect on the root entry zone of the nerve to the brainstem.

- Also known as brainstem concussion.

- This mechanism has been demonstrated in experimental models and in autopsy reports.
VISUAL FIELD DEFICITS

- Rare with concussion; if present review imaging (MRI).

- Testing:
  
  Hemianopic or quadrantanopic field defects on confrontation testing
  
  Abnormal results with draw-a-clock test
VISUAL ACUITY DEFICITS – BLURRED VISION

- Deficit of accommodation – maintaining clear image or focus on an object as its distance varies. Unclear relationship to TBI, but seems related to difficulty with vergence.

- Seen in 10-40% of TBI

- Blurred vision
  - constant or intermittent
  - evident when changing viewing distances (i.e., near-far and/or far-near blur)

- Eyestrain, brow-aches, eye fatigue evident after brief periods of sustained near vision work

- Dizziness, nausea, or motion sickness during or following a vision-based task
VISUAL ACUITY DEFICITS – DIPLOPIA

- Deficit of vergence – Disjunctive changes in eye position as one attends to objects at varying distances in the visual field. Seen in 40-56% of TBI

- Findings
  - Double vision
  - Eliminated with occlusion
  - Constant or intermittent
  - At any viewing distance
  - More evident in one position of gaze than another
  - Eyestrain, eye fatigue, closing or squinting one eye after a brief vision-related task
  - Avoidance of prolonged vision-related tasks
  - Dizziness, nausea, and motion sickness during or following vision-based tasks
VISUAL ACUITY DEFICITS – VERSIONAL

- Disturbances of gaze stabilization (40-80% mTBI).
- Findings:
  - Reading-related difficulty
  - Slower reading speed
  - Loss of place while reading/ skipping lines
  - The print appears to “float”/ “swim”
  - Avoidance of prolonged vision-related tasks
  - Difficulty shifting to/ tracking objects
  - Dizziness/nausea/motion sickness during or following vision-based tasks
VISUAL ACUITY DEFICITS – VERSIONAL

- **Saccades** - Quick, simultaneous movements of both eyes in the same direction.
  - noticeable undershooting or overshooting of the target on saccadic testing

- **Fixation** - maintaining the gaze in a constant direction.
  - nystagmus on fixation assessment

- **Smooth Pursuit** - tracking by the eyes of a slowly moving object at a steady coordinated velocity.
  - restriction of eye movements on pursuit testing
VISUAL-VESTIBULAR DYSFUNCTION: MANAGEMENT STRATEGIES

- Stand on one foot...
- Use a chair if you need to.
- Tai Chi
- Balance walk
VISUAL-VESTIBULAR REHABILITATION

- Limited strict scientific evidence in concussed populations demonstrating efficacy or identifying specifics of visual and vestibular rehabilitation programs. Extensive literature for geriatric imbalance.

- No role for medications other than acute sedation/nausea management.

- Typically recommended if persistent symptoms after 2-4 weeks.

- Focuses on specific deficits with sensory integration of vestibular, visual, proprioceptive, touch/pressure and hearing systems.
VISUAL-VESTIBULAR REHABILITATION

- Types of Sensory Reintegration:
  - Oculomotor training
  - Eye-Head coordination
  - Balance training
  - Visual motion sensitivity training
  - Neuromuscular control
  - Body mechanics and posture
SUMMARY

- The majority of balance deficits seen acutely after mTBI are related to injury of the peripheral vestibular system. Early return to upright mobility is crucial.

- The majority of visual deficits seen acutely after mTBI are related to central coordination of gaze version and vergence.

- Vestibular rehabilitation focuses on re-integrating the labyrinthian organs with the proprioceptive, visual, and muscular systems.

- Visual rehabilitation focuses on progressively optimizing the brain’s coordination and perception of eye movements.