Dizziness and Concussions
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What is a Concussion?

• **Traumatic Brain Injury:**
  - 2.85 million ED visits for TBI/ concussion occur per year.
  - [www.cdc.gov/traumaticbraininjury/](http://www.cdc.gov/traumaticbraininjury/).
  - TBI is caused by a forceful impact to the head (bump or blow) or from penetration to the head (gunshot wound) that disrupts normal function in the brain.
  - The majority of these are mild TBI/concussions (CDC, 2003).
  - TBI can be classified as mild, moderate, or severe.

What is a Concussion?

• **TBI**
  - Mild TBI symptoms include: headache, confusion, lightheadedness, dizziness, blurred vision, tinnitus, bad taste in mouth, fatigue/ lethargy, change in sleeping patterns, changes in mood, memory problems, concentration, attention, processing ([www.ninds.nih.gov](http://www.ninds.nih.gov)).
  - Moderate and severe TBI symptoms include the above symptoms, persistent or worsening headache, nausea, vomiting, convolution, seizures, dilation of pupils, speech deficits, weakness, numbness in extremities, discoordination, confusion, agitation ([www.ninds.nih.gov](http://www.ninds.nih.gov)).
What is a Concussion?

- There are 2 types of damage noted (Finnie and Blumbergs, 2002):
  - Primary damage: tissue deformation at the moment of injury. This results in complex cellular, inflammatory, neurochemical and metabolic alterations. Increased need for glucose but a decreased supply available (Giwa, 2001).
  - Secondary damage: ischemic and hypoxic damage as well as cerebral swelling occur as a result of the primary damage. This causes raised intracranial pressure, hydrocephalus, and infection. These can be the more damaging as it sets off a cascade of metabolic events that increase tissue death.

What is a Concussion?

- Concussion
  - Mild TBI's are synonymous with the term "concussion."
  - Normal recovery from concussion tends to take 7-10 days or more.
  - If symptoms persist longer than this, it is classified as "post-concussive syndrome" or Chronic concussion. The hallmarks of the syndrome are attention and memory deficits as well as fatigue, sleep disturbance, headache, dizziness, irritability, apathy, or personality change (Leddy et al., 2012).
  - Risk of post-concussive syndrome increases with: prior history of concussion, younger age, history of cognitive dysfunction, anxiety and depression (2012).
  - Patients with extended recovery duration tend to have issues with dizziness (Lau et al., 2011)
What is a Concussion?

- Factors that increase concussion risk (Hollis et al, 2011):
  - Type of sport:
    - American football and Australian rugby have the highest incidence for males and soccer for females.
    - Position played (e.g. in football: offensive linemen, linebackers and defensive backs have a higher risk)
    - BMI >27 kg/m² and training time less than 3 hours weekly (Hollis et al, 2011).

What is a Concussion?

- Other factors that can increase risk of concussion include (Ellis, Leddy, Willer, 2016):
  - Previous cervical spine injury
  - Congenital neuroophthalmologic issues such as strabismus and convergence insufficiency
  - History of seizure disorders
  - Structural brain issues (chiari malformation)
  - Hematological conditions (anemia & platelet disorders)

Co-morbidities that can occur with concussion

- A person who has sustained a concussion can also present with (Leddy et al, 2012):
  - BPPV
  - Labyrinthine concussion
  - Central vestibular issues
  - Perilymphatic fistulas
  - Cervicogenic dizziness
CO-MORBIDITIES ASSOCIATED WITH CONCUSSION:

Benign Paroxysmal Positional Vertigo (BPPV)

- Spinning sensation produced by changes in head position relative to gravity
- Patients can alternatively complain of lightheadedness, dizziness, nausea, or generalized imbalance as opposed to true vertigo.
- 50% of patients report subjective imbalance between episodes of vertigo (von Brevern M et al., 2007)
- Benign refers to the condition not being related to a central disorder
- The two most common canals affected are the posterior (85%-95% of all cases) and horizontal (5%-15% of all cases)
- The anterior canal can be affected as well but this is possibly only 1% of all cases

Benign Paroxysmal Positional Vertigo

- BPPV
  - The magnitude of the response to the Hallpike maneuver is based on the total mass of the particles displaced (Rajguru et al., 2004).
  - Latency to the peak response is due to the time needed for the otoconia to move from the ampulla to the posterior apex of the canal (2004).
Benign Paroxysmal Positional Vertigo

- There are 2 types/mechanism of Canal BPPV:
  - Cupulolithiasis: Otoconia adhere to the cupula making it hypersensitive to gravity
  - Canalithiasis: Otoconia are freely mobile in the canal and fall to the lowest point in the canal. Movement causes otoconia to push endolymph causing deflection of the cupula
- Canalithiasis tends to be the more common presentation

Damage to one side of the Vestibular System: Unilateral Hypofunction

- Presentation in acute Unilateral Vestibular Hypofunction:
  - Intense Vertigo and horizontal nystagmus. Spontaneous nystagmus due to a difference in the firing rates of R and L sides. This usually resolves for static positions within the first week.
  - Lateropulsion (leaning/falling toward affected side). This usually is present for about 1 month.
Unilateral Vestibular Hypofunction

- **Ocular tilt reaction** (head tilt to lesioned side).
- **Skew deviation** to the lesioned side (ipsilateral eye lower in its orbit than contralateral eye).
- **Ocular torsion** (Bilaterally the upper pole of the eye rolls toward the lesioned ear).
- These are tested via a subjective visual vertical with a positive test being the line is set too low to the affected side.

Unilateral Vestibular Hypofunction

- **Abnormalities of the VOR: Unilateral Vestibular Loss**.
  - Dynamic sensitivity during head rotation because of no response from the VOR on the lesioned side. This is shown via the head thrust test.
  - **Oscillopsia** with head movements. The world appears to bounce around with movement such as walking.

Unilateral Hypofunction

- **Labyrinthitis** (Verbist, 2012):
  - Inflammation of the labyrinthine organs caused by viral or bacterial infection, inflammatory, or autoimmune origin.
  - The perilymphatic space fills with inflammatory cells.
  - In the acute stages, the symptoms of vertigo and hearing loss can be reversible.
  - Nystagmus occurs spontaneously and is predominantly horizontal.
Bilateral Hypofunction

- **Bilateral Vestibular Loss**
- **Symptoms:**
  - General complaints of dizziness or lightheadedness but not vertigo
  - Gait and postural abnormalities and oscillopsia (world bounces). Gait tends to be wide-based and slow
  - Balance and dizziness are much worse when ambulating in the dark or on grass

Perilymphatic Fistula

- **Perilymphatic fistula** (Hornibrook, 2012):
  - Perilymphatic fistula: Hole between the inner ear and the middle ear caused by Barotrauma, acoustic or head trauma, mastoid or stapes surgery, or vigorous straining.
  - Symptoms of perilymphatic fistula:
    - Transient vertigo or nystagmus with Valsalva maneuver (straining on toilet, coughing, sneezing).
    - Also, the patient can have tulio-phenomenon (vertigo, oscillopsia, nystagmus, ocular tilt reaction, and postural instability) caused by auditory stimulation.
    - Imbalance and aural fullness
    - These typically heal spontaneously.

Perilymphatic Fistula

- **Perilymphatic fistula**:
  - Treatment (Thompson & Amedee, 2009):
    - Usually conservative: bed rest, head elevation, laxatives to avoid strain.
    - Surgery is warranted if symptoms persist
    - Vestibular rehabilitation is not helpful. Refer to an ENT.
Cervicogenic Dizziness

• Cervicogenic Dizziness:
  – Dizziness and dysequilibrium complaints that are associated with neck pain in patients with cervical pathology (Wrisley et al, 2000).
  – It is not considered vertigo but rather imbalance or unsteadiness related to movements or positions of the neck (Reid et al, 2012).
  – Possible etiologies include: trauma such as whiplash, spinal degeneration, and other (Hulse, 1983).

Cervicogenic Dizziness

• Reflexes related to the cervical region:
  • Cervico-ocular Reflex (COR) - signals from neck contribute to eye movement. Information from proprioceptors in the neck give information about neck position.
  • Vestibulo-colic Reflex (VCR) - vestibular signals are relayed to the neck muscles to help stabilize the head.
  • Cervico-colic Reflex (CCR) - aligns the head with the trunk to stabilize the head in space.

Cervicogenic Dizziness

• Cervicogenic dizziness:
  – Symptoms (Brandt & Bronstein, 2001):
    • Lightheadedness or floating
    • Dizziness or feeling of being “off” that increases with neck motion
    • Imbalance or slight ataxia with gait
    • Static balance deficits
    • Neck pain and stiffness/decreased ROM
    • Impaired neck strength
ASSESSMENT FOR CONCUSSION RELATED DIZZINESS

Assessment of Post-Concussion Dizziness

• On field assessment: Vestibular Oculomotor Screen (VOMS)
  – The VOMS is an assessment tool available to screen patient vestibular and ocular motor function resulting from a sport-related concussion (Mucha et al., 2014).
  – It includes: smooth pursuit, horizontal and vertical saccades, vergence, horizontal vestibular ocular reflex (VOR), Visual Motion Sensitivity.
  – VOR and VOMS were most predictive of a concussion.

Assessment of Post concussion dizziness

• Oculomotor screen (in clinic):
  – Smooth pursuit, saccades, VOR cancellation, Subjective visual vertical, convergence/ divergence
  – Gaze stability testing:
    • Bedside tests: head impulse test
    • Measurement tests: Video Head Impulse Test (VHIT), Dynamic visual acuity
  – Cervicogenic Testing
    • Proprioception testing
    • Differentiating cervicogenic from vestibular pathology
Assessment of Post concussion dizziness

- **Postional Testing:**
  - Dix-hallpike, Sidelying, Roll Test

- **Static balance:**
  - mCTSIB (modified computerized test for the sensory integration of balance)

- **Balance with gait test:**
  - Dynamic gait assessment
  - Functional gait assessment

- **Exertional testing:**
  - Buffalo Concussion Treadmill Test

Assessment: Hallpike-Dix Test

- Minimal Detectable Change: Not Established
- Minimal Clinically Important Difference: Not Established
- Normative Data: 57% of patients with traumatic and 19% of patients with benign suffered recurrent attacks of BPPV (Gordon et al, 2004)
- Interrater/Intrarater Reliability: Excellent between 2 assessors (Kappa= 0.92, 95% CI: 0.87-0.98) (Burston et al, 2012)
- 2 Questions that independently predict a positive Dix-Hallpike Test/ diagnosis of BPPV (Noda et al, 2011):
  - 1. Does the dizziness last less than 15 seconds
  - 2. Do you experience dizziness when turning over in bed
Assessment of Post- Concussion Dizziness

- Video Head impulse test
  - Uses as set of goggles with cameras on them that photograph and measure the position of the eye
  - The patient fixates on a stationary target while the examiner performs random quick head movements on no more than 20 degrees rotation
  - The movements are quick (250 degrees/second or greater)
  - The camera measures the amount the eyes deviate off the target and has to refixate (covert saccades)

Evaluation: Medical Tests: vHIT

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Evaluation: Medical Tests: vHIT

- Predictive validity (Unilateral Hypofunction) McDougall et al, 2009):
  - Sensitivity: 100%
  - Specificity: 100%

- Predictive validity (General dizziness) (Harvey et al, 1997)
  - Sensitivity: 35%
  - Specificity: 95%

Assessment of Post concussion dizziness

- Dynamic Visual Acuity
  - Functionally measures the amount of visual loss of acuity due to vestibular deficit.
  - The patient is initially assessed for static head visual acuity.
  - Then the patient rotates their head no more than 20 degrees at 2 Hz or greater while trying to read at the same acuity level.

Evaluation: Medical Tests: DVA
Evaluation: Medical Tests:

DVA

- Predictive validity (BVH, TBI/ concussion) (Rine et al, 2012)
  - Sensitivity: 72%
  - Specificity 69%

Evaluation: Static Balance

- Modified Computerized Test For the Sensory Integration of Balance (mCTSIB).
  - Static balance testing proposed to test the different sensory systems related to balance (visual, vestibular, and proprioceptive)
  - Conditions Tested:
    - On firm ground eyes open/ closed
    - On foam eyes open/ closed
    - Eyes open on firm ground and foam with a visual conflict dome on (this part is optional)

Evaluation: Balance with Gait

- Dynamic Gait Index (DGI)
  - An 8 item test that involves gait with changing speed, head turns, body turns, negotiating obstacles.
  - Highest possible score of 24/24.
  - MDC= 2.9 points (community dwelling elderly) (Romero et al, 2011).
  - MCID= 1.90 (Pardasaney et al, 2012)
Balance with Gait

• Functional Gait Assessment:
  – Measures balance ability with gait under a variety of conditions (eyes open/ closed, narrow base of support, gait with head turns and body turns)
  – MDC = 5 point change clinically (stroke)
  – MDIC = 8 points from admission to follow up
  – A Score of <23/30 predictive of falls (Wrisley & Kumar, 2010).
  – Sensitivity 85%, Specificity 86%

Physical Exertion

• Graded treadmill testing:
    • Based on the Balke Cadiac Treadmill test.
    • Start at 3.6 mph and 0% incline (modify as needed)
    • The incline is increased every minute by 1% until maximum incline is reached or patient unable to continue.

Physical Exertion

• RPE (rate of perceived exertion) and symptoms are rated every minute, while heart rate and blood pressure are measured every 2 minutes.
  • The test is stopped when symptoms rise 3 or greater points on a 1-10 point visual analog scale.

• Depending on demands of sport/ activity:
  – If tolerated/ warranted, perform activities such as sprint and resistance and monitor symptoms.
TREATMENT FOR CONCUSSION-RELATED DIZZINESS

Treatment: General

- Rest primarily during the acute phase, but depending on the symptoms, may include meds for anxiety, depression, blood pressure.
- Monitoring during sessions for HA, fogginess, fatigue.
- Oculomotor training or referral if needed.
- Progressing at a slower rate than patients with other diagnoses.
- Limiting exertion initially with exercise (because of the neuro-metabolic issues).

Treatment: General

- Physical and Cognitive Rest
  - Primarily during the acute phase (1-7 days) with gradual introduction of activity as tolerated after that.
  - Limit activities to non-symptom inducing activities (light stretching, low intensity movement activities, low intensity cognitive tasks) in the acute phase.
  - Limit use television, computer, cell phones, etc.
  - Gradually increase physical and cognitive activities as tolerated.
Treatment: BPPV

- Physical repositioning maneuvers to resolve symptoms.
Treatment: Unilateral hypofunction

- Gaze stability and Compensatory mechanisms for loss of function
- Balance training static initially and functional
- Establishing baseline exertion and physical training parameters

X1 Gaze Stability

Treatment: Cervicogenic Dizziness

Treatment includes:
- Manual therapy
- Cervical strengthening/ flexibility
- Balance training
- Proprioceptive training
- Oculomotor training
Treatment: Vision Therapy

- Vision therapy has been shown to be helpful in the treatment of convergence insufficiency and accommodative insufficiency due to post-concussion syndrome (Gallaway, 2016).
- It also involves the use of oculomotor training and recommendations for light filters. They can also work with Neuro-ophthalmologists for compensatory aides if severe enough (prism lenses, etc.)

Treatment: Lymphedema Therapy

- Lymphatic vessels have been discovered in the dural sinuses.
- They carry fluid and immune cells from csf and are connected to the deep cervical lymph nodes (Louveau et al, 2015).
- Manual lymph drainage is used to decrease edema in the head and cervical regions.

Treatment: Physical Exertion

- **Post-Concussion return to activity guidelines** (International Conference on Concussion in Sport 2008).
  - 1) No activity: exertion 30-40% max heart rate (MHR)
  - 2) Light-moderate: 40-60% MHR
  - 3) Moderately aggressive: 50-60% MHR
  - 4) Sports Performance: 80-90% MHR
  - 5) Sports Performance: Full exertion with contact
Treatment: Physical Exertion

• Post-Concussion return to activity guidelines:
• Presently this paradigm is dated. There is a newer concept of varying the stages by movement and vestibular impairments.
• The idea is to progress the movements and environment in addition to progressing physical exertion (i.e. going from a quiet environment to a busier environment).
• Use of the Buffalo Concussion Treadmill Test to establish a baseline for intensity of walking and progressions.

Case Study A:

• 15 Year old male playing in a basketball. He went up to retrieve a rebound and was impacted by another player consequently landing on his back and head.
• He denies loss of consciousness but complained of dizziness, headache and confusion immediately after.
• He was referred to the ED and released the next day. CT scans and tests were negative.

Case Study A:

• He is referred to outpatient physical therapy 25 days after the onset of injury as he is still complaining of dizziness with all activities but it worsens with head movement, attempting to read, use the computer or his cellphone.
• He also complains of diplopia and headache when he reads and uses electronic devices.
### Case Study A:

- He has not been able to return to practice and finds his symptoms worsen when he does anything more than walk at a slow pace.
- Past medical history includes: history of depression, strabismus when he was a child.
- Upon examination: note that smooth pursuit and saccades are normal but he complains of a symptom increase (4 points of VAS).

### Case Study A:

- Gaze stability testing using the head impulse test denotes overt saccades to the left side.
- Static balance on the mCTSIB notes loss of balance with eyes closed on firm ground and on foam.
- He is symptomatic with the functional gait assessment test and scores 22/30.
- Positional testing is negative.
- He is symptomatic at 4 minutes on the Buffalo Concussion Treadmill Test Heart rate 161 bpm and blood pressure 142/69 mm Hg.

### Case Study A:

- Discussion:
  - He is experiencing visual motion sensitivity.
  - Possible decompensation for pre-morbid strabismus.
  - Gaze instability vestibular hypofunction left side.
  - Visual dominance for balance presently.
  - Exertional intolerance.
Case Study A:

- Plan of care:
  - Decrease visual motion sensitivity.
  - Possible referral to a vision therapist or neuro-ophthalmologist if diplopia doesn’t resolve.
  - Gaze stability exercises
  - Balance training involving eyes closed activities and sensory re-weighting to proprioceptive and vestibular input.
  - Gait with head and body motion
  - Cardiovascular conditioning below symptom-producing intensity with gradual increase as tolerated.

References:

- Finnie JW, Blumbergs PC. Veterinary Pathology November 2002; vol 39 no. 6 679-689.
References:


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References:

- Brandt and Bronstein

References: