

Concussion Clinical Mastery

Complete Reference Text

Understanding, Assessing & Managing Concussion

Background

Welcome to the Concussion Training Course, developed for health professionals, educators, and rehabilitation specialists seeking a deeper understanding of concussion pathophysiology, diagnosis, and management. This course integrates the latest evidence-based practices with a strong focus on clinical reasoning, inter-professional care, and ethical responsibility.

Course Purpose

To equip participants with the skills, knowledge, and professional standards required to assess, document, and manage concussion presentations in acute and post-acute settings, including return-to-play, return-to-learn, and return-to-work pathways.

Compliance & Professional Standards

AHPRA's Code of Conduct for Registered Health Practitioners ASQA Principles of Assessment and Rules of Evidence Current guidelines for concussion management including SCAT/SCoAT6 and RTP protocols Best practices for documentation, risk management, and ethical decision-making

Objectives

By the end of this course, participants will be able to analyse the mechanisms and physiological impact of concussion and mild traumatic brain injury (mTBI) to inform accurate diagnosis in 90% of clinical cases.

They will apply structured clinical assessments, including SCAT/SCOAT6 and vestibulo-ocular testing, to achieve a diagnostic accuracy of at least 85% in acute concussion settings. Participants will also differentiate red flags and common concussion mimics (e.g., whiplash, vestibular dysfunction) to reduce misdiagnosis rates by 20%.

They will develop and implement evidence-based management plans aligned with Concussion in Sport Australia (CISA) guidelines, improving patient recovery rates by 15%.

They will collaborate effectively with multidisciplinary teams to manage post-concussion syndrome (PCS), ensuring 100% adherence to referral protocols.

They will adhere to AHPRA's Code of Conduct and ASQA's assessment principles in documentation, referral, and duty of care, achieving 100% compliance in clinical records.

About the instructor

Zac Lewis Osteopath B.Clin.Sci.,M.Ost.Med

Osteopath of 10 years clinical experience with a clinical focusing on neurological injuries, the last 5 of which being directed towards concussion management.

Clinical work spans Australia, New Zealand and Canada, including roles with national and professional ice hockey organisations. Currently pursuing a PhD in neuroscience with research focused on diagnostic and prognostic tools in concussion.

Accessibility & Learning Support

This course is designed to accommodate all learners. Supports include:

- Large print materials and digital formats for visual impairments.
- ~~Wheelchair~~ accessible venues with sign language available on request. Visual and audio aids, including closed captioning for videos. Case studies featuring diverse demographics (e.g., Indigenous, CALD, paediatric, elderly patients) to promote culturally safe practice.
- Translated materials in major community languages (e.g., Arabic, Mandarin) available on request.
- Peer discussion groups and one-on-one instructor support for complex topics.
- Remediation plans for learners who do not meet assessment criteria, including additional practice sessions.

Competency Framework Alignment

Module 1	Introduction to Concussion and Brain Injury	Mechanisms of brain injury, anatomical and neurophysiological impacts, injury classification
Module 2	Concussion Diagnosis and Initial Assessment (Theory)	Use of diagnostic tools (SCAT6, Child SCAT), symptom identification, theoretical assessment
Module 3	Practical Assessment & Acute Concussion Management	Acute symptom tracking, return-to-play/work protocols, identifying critical cases
Module 4	Post-Concussion Syndrome and Long-Term Management	Chronic symptom management, multidisciplinary referrals, neurodegenerative risk
Module 5	Multidisciplinary Approach in Concussion Management	Team-based care planning, inter-professional communication, rehabilitation strategies
Module 6	Return to Play/Return to Work Protocols	Application of return-to-activity plans, special population considerations, patient education
Module 7	Rehabilitation Pathways in Concussion Management	Phenotype mapping, clinical prescription tasks + and rehabilitation programming
Module 8	Legal, Ethical, Communication and Documentation Considerations	Patient education strategies, health literacy, communication across age/cognitive groups

Training Manual Contents:

- 1. Introduction:**
 - Course goals and objectives.
 - Instructor biography, experience.
 - Concussion facts and statistics.
- 2. Module 1: Introduction to Concussion and Brain Injury:**
 - Differentiate between linear, rotational, and blunt trauma mechanisms using clinical case examples, and classify the resulting traumatic brain injuries accordingly.
 - Anatomical, biochemical and neurophysiological impact of TBI.
 - Severity differentiation and investigative tools.
 - Current imaging use cases and value.
- 3. Module 2: Concussion Diagnosis and Initial Assessment THEORY:**
 - Apply current diagnostic criteria (e.g., SCAT/SCOAT6/Child SCAT) to evaluate suspected concussion cases across age groups.
 - Identify the components of a concussion assessment and describe clinical presentation.
 - In-depth theoretical understanding of physical and cognitive assessments (balance, memory, coordination).
 - Limitations of current theoretical understanding.
- 4. Module 3: Practical Assessment & Acute Concussion Management :**
 - Common recovery, symptom progression
 - Step-by-step management guidelines.
 - Return-to-play/work protocols.
 - PCS management flowchart.
 - How to ID unresponsive, undiagnosed.
- 5. Module 4: Post-Concussion Syndrome and Long-Term Management:**
 - Defining PCS and its timeline.
 - Key symptoms: headaches, dizziness, cognitive impairment, mood changes.
 - Multidisciplinary approach to managing long-term symptoms.
 - When to refer for neuropsychological assessment or rehabilitation.
 - Evidence of CTE, risk of further neurodegen (Alzheimer's, parkinsons).
- 6. Module 5: Multidisciplinary Approach in Concussion Management**
 - Describe and differentiate the roles of various healthcare professionals in concussion care.
 - Develop a multidisciplinary management plan for a post-concussion patient, including appropriate referral pathways and role-based interventions from GP, physio, OT, and neuropsychology.
 - Rehabilitation strategies for post-concussion syndrome.
 - Management steps: physical rest, cognitive rest, and monitoring.
 - Treatment and rehabilitation approaches.

7. Module 6: Return to Play/Return to Work Protocols

- Apply return-to-activity protocols and design progression plans for physical and occupational re-engagement post-concussion.
- Graduated return-to-play protocols.
- Return-to-work guidelines and adjustments.
- Role of rehabilitation professionals in concussion recovery.
- Considerations for special populations (e.g., children, elderly).
- Education for patients and families.

8. Module 7: Rehabilitation Pathways in Concussion Management

- Understand the legal responsibilities regarding concussion diagnosis and management.
- Apply ethical guidelines in complex return-to-play or return-to-work decisions.
- Legal requirements for managing sports-related concussions.
- Ethical considerations in return-to-play and return-to-work decisions.
- Documentation and record-keeping for concussion cases.

9. Module 8: Legal, Ethical, Communication and Documentation Considerations

- Learn a 4-step framework to explain concussion in simple, structured terms
- Use analogies and visual tools to enhance patient understanding
- Tailor explanations to different age groups and cognitive levels
- Guide recovery expectations with empathy and clarity
- Practice clear, jargon-free communication through roleplay scenarios

Appendix:

- Assessment overview matrix
- Additional resources (websites, journals).
- Recommended reading list.
- Evaluation form for course feedback.

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Introduction: Welcome to the Course

Before starting, review these key facts & concussion myths

Quick Stats Discussion:

- **3.8 million concussions annually in U.S. sport & recreation** (Centers for Disease Control and Prevention, 2021)
- **30–50% of athletes do not report symptoms**, often due to cultural factors or lack of awareness of concussion symptoms (McCrea et al., 2004)
- **Recovery times vary**: In adults, recovery often ranges from **10 to 14 days**, while in children it can be **4+ weeks** due to their developing brains and unique pathophysiology (Lovell et al., 2004)
- **Neuroimaging**: Standard imaging often shows no abnormalities despite the presence of clinical symptoms, making symptom-based assessment critical (Giza & Hovda, 2001)

Concussions Frequently Go Unreported

- Studies show that 30–50% of athletes do not report suspected concussions due to fear of removal from play, lack of symptom recognition, or a belief that it's "not serious."
- Underreporting is especially common in male adolescent athletes.

Concussion Without Head Contact

- A concussion is a mild traumatic brain injury (mTBI) caused by biomechanical forces.
- It may result from direct or indirect forces to the head (e.g., whiplash, body checks) that cause acceleration-deceleration of the brain within the skull.
- Rotational forces are particularly associated with diffuse axonal injury at the microscopic level.

Neuroimaging is Usually Negative

- Concussion is a functional brain injury, not a structural one.
- CT and MRI are typically unremarkable in concussion and are used primarily to rule out more serious pathologies such as skull fractures or intracranial bleeding.
- Advanced imaging (e.g., DTI, fMRI) may show subtle changes but is not yet standard for diagnosis.

Second Impact Syndrome (SIS)

- Rare but potentially fatal, SIS occurs when a second concussion is sustained before the brain has recovered from the first.
- It leads to rapid cerebral edema and brain herniation, usually in adolescents.
- Prevention through proper return-to-play protocols is critical.

Pediatric and Adolescent Brains Are More Vulnerable

- Children experience slower recovery and are more susceptible to cognitive and behavioral disturbances post-injury.
- The developing brain has higher metabolic demands and a longer period of vulnerability to neurometabolic disruption.

Non-Sport Causes Are Equally Significant

- Falls, motor vehicle accidents, bicycle crashes, and occupational injuries are leading causes of concussions outside of sport.
- In older adults, falls account for over 60% of TBIs requiring hospitalisation.

Economic and Societal Burden

- The direct and indirect costs of concussion and mild TBI are estimated in the billions annually due to lost productivity, healthcare costs, and long-term disability.
- Missed school and work, cognitive impairments, and mental health sequelae add to long-term impact.

Clinical Diagnosis is Paramount

- Diagnosis relies on clinical history, observed signs, symptom checklists, and structured tools like SCAT/SCOAT6, VOMS, and BESS.

Currently there is no single objective biomarker for concussion, making clinician expertise essential.

What Is Unclear About Current Guidelines?

- **What confuses or remains unclear about concussion management?**
 - Address neurobiological mechanisms (e.g., excitotoxicity, ion imbalance) that affect cognitive and physical recovery.
 - Explore emerging research on post-concussion syndrome (PCS) and persistent symptoms.
- **Have you encountered cases that challenge your understanding?**
 - Discuss cases with prolonged recovery or atypical symptoms, focusing on differential diagnosis (e.g., vestibular dysfunction, psychiatric sequelae).
- **Topics of Interest for Deeper Dive:**
 - Diagnostic tools and protocols (e.g., SCAT/SCOAT6, ImPACT testing).
 - Advances in concussion biomarkers and their clinical applicability.

Case Discussion

Case Study:

"A 14-year-old boy presents 3 days post-football collision. No LOC. Complains of feeling 'off,' concentration difficulty, neck pain."

Group Questions:

1. What stands out to you about this case?

- Highlight age-specific considerations, such as the greater susceptibility of children to second-impact syndrome and prolonged symptoms (Giza & Hovda, 2001).
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2. What would be your initial assessment?

- Discuss the importance of a thorough history and symptom checklist, including potential red flags like neck injuries or loss of consciousness.
 - Consider cognitive, physical, and vestibular assessment.
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3. What might be missed in a typical clinic?

- Explore common missed diagnoses, such as vestibular dysfunction or cervicogenic headaches, and how to identify them.
 - Review the concept of "silent" concussions, where symptoms may not appear until later, particularly in non-contact sports (Brenner et al., 2016).
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Reading list:

- Bain, A. C., & Meaney, D. F. (2000). The need for improved models of brain injury: the challenge of concussion. *Journal of Neurotrauma*, 17(1), 1-8.
<https://doi.org/10.1089/neu.2000.17.1>
- Brenner, J. S., et al. (2016). Concussions in youth sports. *Pediatrics*, 138(1), e20160903.
<https://doi.org/10.1542/peds.2016-0903>
- Giza, C. C., & Hovda, D. A. (2001). The new neurometabolic cascade of concussion. *Neurosurgery Clinics of North America*, 12(1), 1-8.
[https://doi.org/10.1016/S1042-3680\(05\)70101-1](https://doi.org/10.1016/S1042-3680(05)70101-1)
- Lovell, M. R., et al. (2004). Recovery from mild concussion in high school athletes. *Journal of Neurosurgery*, 99(3), 453-457. <https://doi.org/10.3171/jns.2003.99.3.0453>
- McCrea, M., et al. (2004). Acute effects and recovery time following concussion in collegiate football players. *Journal of the American Medical Association*, 291(19), 2302-2307. <https://doi.org/10.1001/jama.291.19.2302>
- McCrory, P., et al. (2017). Consensus statement on concussion in sport—The 5th International Conference on Concussion in Sport held in Berlin, October 2016. *British Journal of Sports Medicine*, 51(11), 837-847.
<https://doi.org/10.1136/bjsports-2017-097699>

[Concussion Clinical Mastery]

Concussion Education Australia

 This course contributes to Continuing Professional Development (CPD) requirements for a range of AHPRA-registered professionals.



Module 1: Introduction to Concussion and Brain Injury

From Impact to Injury: The Science Behind Concussion

A deep dive into the anatomical, biomechanical, and neurochemical foundations of traumatic brain injury.

Learning Objectives:

- Describe mechanisms of injury and classify diagnostic categories of TBI.
- Anatomical, biochemical and neurophysiological impact of TBI.
- Severity differentiation and investigative tools.
- Current imaging use cases and value.

Activities & Clinical Knowledge Checks

This module includes interactive quizzes and evidence-based discussions to challenge misconceptions, assess understanding of brain network dysfunction, and consolidate foundational knowledge of concussion and brain injury. Activities are assessed and trainer-marked in real time.

Learning outcomes

1. Analyse the pathophysiology of concussion and its impact on neurological function.
2. Compare the biomechanical forces and mechanisms responsible for concussion.
3. Locate and explain the role of key anatomical structures affected by TBI (e.g., frontal lobe, cerebellum, brainstem).
4. Explain the stages of the neurometabolic cascade following concussion and apply this knowledge to guide early symptom monitoring and rest strategies.
5. Differentiate between concussion and more severe traumatic brain injuries.

 **Common Concussion Myths Quiz**

Instructions: Mark each statement as True (T) or False (F). You'll review answers during the group debrief.

	Statement	True / False
1	A person must lose consciousness to be diagnosed with a concussion.	
2	Most concussions show up clearly on standard CT or MRI scans.	
3	Concussion symptoms usually resolve within 24–48 hours in all cases.	
4	Only contact sports athletes are at risk of concussion.	
5	A clear head CT scan rules out a concussion.	
6	Children recover more quickly from concussion than adults.	
7	You have to “rest completely” (no activity at all) for two weeks post-concussion.	
8	Helmets and mouthguards can prevent concussion.	
9	It’s safe to return to work or sport once symptoms subside, even without clearance.	
10	Concussion only affects cognition — emotional and physical symptoms are unrelated.	

Current understanding

Mechanism of injury¹

1. Linear Acceleration (Coup-Contrecoup Injuries)

- Head is rapidly moved in a straight line, either back-and-forth or side-to-side, leading to the brain shifting within the skull.

2. Rotational (Angular) Acceleration

- Rotational acceleration refers to the forces acting on the brain that cause it to rotate or twist within the skull, often leading to shearing forces on brain tissue.

3. Blunt Impact Trauma

- Blunt impact trauma involves direct physical contact with an object or surface, such as being struck by a ball, a blow to the head, or striking the head against a hard surface (e.g., ground or vehicle).

4. Whiplash

- Sudden acceleration and deceleration of the head and neck, typically seen in rear-end vehicle collisions, resulting in injury to the brain.

Mechanism of Injury	Commonly Affected Brain Regions	Typical Symptoms
Linear Acceleration	Frontal lobe, occipital lobe, brainstem (coup–contrecoup forces)	Headache, blurred vision, disorientation, loss of consciousness, visual disturbances
Rotational Acceleration	Corpus callosum, midbrain, cerebellum, frontal and temporal lobes	Dizziness, nausea, balance loss, executive dysfunction, emotional lability, slowed processing
Blunt Impact Trauma	Focal cortex (impact site), frontal and temporal poles	Headache, confusion, localized deficits, mood changes, speech or motor disturbances
Whiplash (CAD Injury)	Cervical spinal cord, brainstem, vestibular nuclei, proprioceptive pathways	Neck pain, dizziness, cervicogenic headache, visual tracking issues, balance problems

¹ McKee, A. C., & Daneshvar, D. H. (2015). The neuropathology of traumatic brain injury. *Handbook of Clinical Neurology*, 127, 45–66. <https://doi.org/10.1016/B978-0-444-52892-6.00004-0>

Mechanism of Injury Classification

Closed Head Injury:

- Definition: The brain is injured without a break in the skull. The brain may move within the skull due to acceleration/deceleration forces or blunt trauma.
- Example: Whiplash, sports injuries, or falls.
- Symptoms: Concussion, contusions, diffuse axonal injury.

Open (Penetrating) Head Injury:

- Definition: The skull is broken or fractured, allowing foreign objects or bone fragments to penetrate the brain tissue.
- Example: Gunshot wounds, shrapnel injuries, or severe trauma from accidents.
- Symptoms: Bleeding, risk of infection, focal brain damage.

Coup-Contrecoup Injury:

- Definition: A type of closed head injury where the brain is damaged at the site of impact (coup) and on the opposite side of the brain (contrecoup) due to rapid acceleration or deceleration.
- Example: A person's head hitting the windshield in a car accident.
- Symptoms: Swelling, hemorrhaging, and contusions at both sites.

Diffuse Axonal Injury (DAI):

- Definition: A type of injury where widespread damage occurs to the brain's axons due to rotational or acceleration forces. This is a severe form of brain injury often associated with high-speed accidents.
 - Example: High-impact collisions or violent shaking, such as in shaken baby syndrome.
 - Symptoms: Coma, cognitive deficits, and motor impairments.
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Structural Classification

Focal Injury:

- Definition: Injury confined to a specific area of the brain, such as a contusion, hematoma, or brain laceration.
- Example: A brain contusion from a direct blow to the head.
- Symptoms: Localized motor, sensory, or cognitive deficits depending on the region of the brain involved.

Diffuse Injury:

- Definition: Widespread damage across multiple areas of the brain, often caused by rotational forces or rapid acceleration/deceleration.
- Example: Diffuse axonal injury (DAI) resulting from a severe car accident.
- Symptoms: Extensive cognitive, motor, and behavioral impairments.

Secondary Injury Classification

Primary Injury:

- Definition: Immediate brain injury caused by the trauma, such as bruising, bleeding, or tearing of the brain tissue.
- Example: Skull fracture, contusion, or hemorrhage.

Secondary Injury:

- Definition: Cascade of pathological events that occur after the primary injury, including swelling, increased intracranial pressure (ICP), ischemia, and biochemical changes.
 - Example: Brain swelling, reduced cerebral blood flow, or cerebral edema.
 - Symptoms: Worsening neurological function, increased risk of long-term disability.
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Neuroimaging Classification

Concussion (Functional Injury):

- Definition: A functional disturbance of the brain without significant structural damage, often undetectable using standard neuroimaging techniques like CT or MRI.
- Example: Symptoms of dizziness, confusion, and cognitive dysfunction after a mild impact.

Structural Brain Injury:

- Definition: Damage visible on neuroimaging, such as hemorrhage, contusion, or tissue loss.
 - Example: Intracranial hemorrhage (ICH) visible on CT, brain contusion seen on MRI.
 - Symptoms: Visual evidence of injury on brain scans correlating with cognitive or motor dysfunction.
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TBI Classification

Severity Classification

Mild TBI (Concussion):

- Definition: A TBI characterized by a brief loss of consciousness (if any), confusion, or disorientation lasting less than 30 minutes, with a Glasgow Coma Scale (GCS) score of 13–15.
- Symptoms: Headache, dizziness, nausea, sensitivity to light, and temporary cognitive changes.

Example: Concussions, which are common in sports and minor accidents.

Moderate TBI:

- Definition: A loss of consciousness lasting between 30 minutes and 6 hours, with a GCS score of 9–12. Individuals may experience a longer duration of confusion and memory loss.
- Symptoms: Prolonged confusion, cognitive impairments, emotional changes, and possibly motor impairment.

Example: Falls, car accidents, or contact sports injuries resulting in moderate brain damage.

Severe TBI:

- Definition: A loss of consciousness lasting more than 6 hours, with a GCS score of 3–8. This classification is associated with extensive damage to brain tissue and a higher likelihood of long-term cognitive, motor, and emotional impairments.
- Symptoms: Severe cognitive dysfunction, physical disability, possible coma, and persistent vegetative states.

Example: High-impact trauma such as serious car accidents, violent assaults, or severe falls.

Grading²

1. Concussion Grading (Mild, Moderate, Severe) is Outdated

- The old "Grade 1, 2, 3" classification (from Cantu & Colorado Medical Society models) is no longer recommended because concussion is a functional brain injury rather than a structural one.
- Current best practice: Concussions are now graded based on clinical severity and recovery time, rather than acute loss of consciousness (LOC) or symptom duration alone.
- Berlin (2016) Consensus: Concussions should be managed individually rather than categorized by arbitrary severity levels.

2. Subtypes of Concussion: Some Are Valid, Some Lack Strong Evidence

The concept of concussion subtypes (e.g., vestibular, oculomotor, cognitive, affective) is increasingly recognized in clinical research, but some terms used here are not formally accepted classifications in evidence-based medicine. Here's a breakdown:

Evidence-Based Concussion Phenotypes³

Vestibular Concussion – Recognized in research (balance, dizziness, vertigo)

Ocular Concussion – Often referred to as **oculomotor dysfunction** in concussion

Cognitive Concussion – Cognitive symptoms like **brain fog, processing delays** are common

Migrainous Concussion – Migraine-like symptoms are documented in post-concussion syndrome

Affective Concussion – Emotional dysregulation (e.g., depression, anxiety) is common in concussion recovery

3. Post-Concussion Syndrome (PCS) & Second Impact Syndrome (SIS)

PCS – Recognized condition where symptoms persist for **weeks to months** post-injury.

SIS – Rare but documented condition where a second impact causes rapid brain swelling.

² McCrory, P., Meeuwisse, W. H., Aubry, M., Cantu, R. C., Dvořák, J., Echemendia, R. J., ... & Turner, M. (2017). Consensus statement on concussion in sport: The 5th International Conference on Concussion in Sport, Berlin 2016. *British Journal of Sports Medicine*, 51(11), 838-847. <https://doi.org/10.1136/bjsports-2017-097699>

³ Craton, N., Ali, H., & Lenoski, S. (2017). COACH CV: The Seven Clinical Phenotypes of Concussion. *Cureus*, 9(9), e1771. <https://doi.org/10.7759/cureus.1771>

Musculoskeletal involvement⁴

Cervical spine disorders

- **Whiplash-Associated Disorders (WAD):** Neck pain and stiffness often occur due to sudden acceleration-deceleration forces during the concussion event.
- **Cervicogenic Headaches:** Headaches arising from neck dysfunction, particularly due to irritation of the upper cervical joints and muscles
- **Facet Joint Dysfunction:** Pain and limited mobility in the cervical spine due to joint irritation or misalignment
- **Myofascial Pain Syndrome:** Trigger points and muscle tightness, especially in the trapezius, sternocleidomastoid, and suboccipital muscles.

Vestibular and Postural Instability

- **Balance Impairments:** Changes in proprioception and coordination can lead to an increased risk of falls and joint injuries.
- **Benign Paroxysmal Positional Vertigo (BPPV):** Dislodged otoliths in the inner ear cause dizziness and imbalance, increasing injury risk.

Shoulder and Upper Limb Dysfunction

- **Scapular Dyskinesis:** Altered movement patterns in the shoulder blade due to impaired neuromuscular control.
- **Rotator Cuff Dysfunction:** Muscle imbalances and coordination deficits may lead to shoulder pain and impingement.

Low Back and Pelvic Dysfunction

- **Core Stability Deficits:** Concussions can impair deep core muscle activation, leading to low back pain and instability.
 - **Pelvic Alignment Issues:** Postural compensations may contribute to sacroiliac joint dysfunction and hip pain.
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⁴ Schneider, K. J., Iverson, G. L., Emery, C. A., McCrory, P., Herring, S. A., & Meeuwisse, W. H. (2013). The effects of rest and treatment following sport-related concussion: a systematic review of the literature. *British Journal of Sports Medicine*, 47(5), 304–307. <https://doi.org/10.1136/bjsports-2013-092190>

Neuroanatomy

Cortical & Subcortical Gray Matter

- Prefrontal Cortex
- Orbitofrontal Cortex
- Temporal lobes
- Occipital lobe

White Matter Tracts (Axonal Injury)

- Corpus Callosum
- Corticospinal Tracts (Motor Pathways)
- Uncinate Fasciculus (Limbic Connectivity)

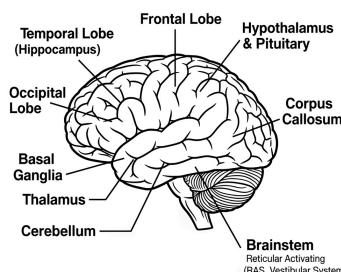
Deep Brain Structures

- Thalamus
- Basal Ganglia (Striatum, Globus Pallidus, Substantia Nigra)
- Hypothalamus & Pituitary Gland

Brainstem & Cerebellum

- Reticular Activating System (RAS, in Brainstem)
- Vestibular & Oculomotor Regions (Cerebellum, Midbrain)

Region	Function	Clinical Symptoms
Frontal Lobe	Executive function, impulse control	Poor concentration, mood changes
Temporal Lobe (Hippocampus)	Memory, emotions	Memory loss, emotional instability
Occipital Lobe	Vision	Blurred vision, light sensitivity
Corpus Callosum	Interhemispheric communication	Slowed processing, motor issues
Thalamus	Sensory relay, consciousness	Fatigue, sensory issues
Basal Ganglia	Motor control, procedural learning	Tremors, fine motor deficits
Brainstem (RAS, Vestibular System)	Arousal, balance	Drowsiness, dizziness, nausea
Cerebellum	Coordination, balance	Poor motor control, vertigo
Hypothalamus & Pituitary	Hormonal regulation, autonomic function	Fatigue, disrupted sleep-wake cycles



Loss of consciousness (LOC)

Reticular Activating System⁵

- Neuronal network in brainstem maintaining consciousness and arousal
- Projects to and stimulates thalamus and cerebral cortex

Diffuse axonal injury

- Axonal stretching/shearing distorts neurons limiting brain region communication

Neurotransmitter disruption

- Maladaptive neurotransmitter release causing imbalance and aberrant firing

Cerebral blood flow change

- Swelling and hypoxia

⁵ Taran, S., Gros, P., Gofton, T., Boyd, G., Neves Briard, J., Chassé, M., & Singh, J. M. (2023). The reticular activating system: a narrative review of discovery, evolving understanding, and relevance to current formulations of brain death. *Journal of Intensive Care*, 11(1), 22. <https://doi.org/10.1007/s12630-023-02421-6>

Biochemistry⁶

Chemical cascade

Ion Imbalance

- Neuronal membrane damage
- K+ **efflux** from voltage-gated channels
- Widespread neuronal **depolarization**
- Excessive **glutamate** (excitatory) release increasing ATP demand
- Na+/K+ **ATPase pump** works to exhaustion to regain homeostasis

Calcium (Ca²⁺) Influx and Mitochondrial Dysfunction

- Excessive extracellular glutamate causes NMDA overstimulation
- Excessive Ca²⁺ **influx**
- Oxidative phosphorylation impairment in mitochondria reduces ATP production
- Cytotoxicity, apoptotic activation and further neuronal damage

Sodium (Na⁺) Dysregulation and Edema Formation

- Na+ **influx** depletes ATP and causes neuronal swelling due to altered osmotic gradient
- Hyperexcitation causes cognitive changes and sensory hypersensitivity
- Glucose dysregulation
- Glial changes, waste accumulation

Transient Cerebral Hypoperfusion

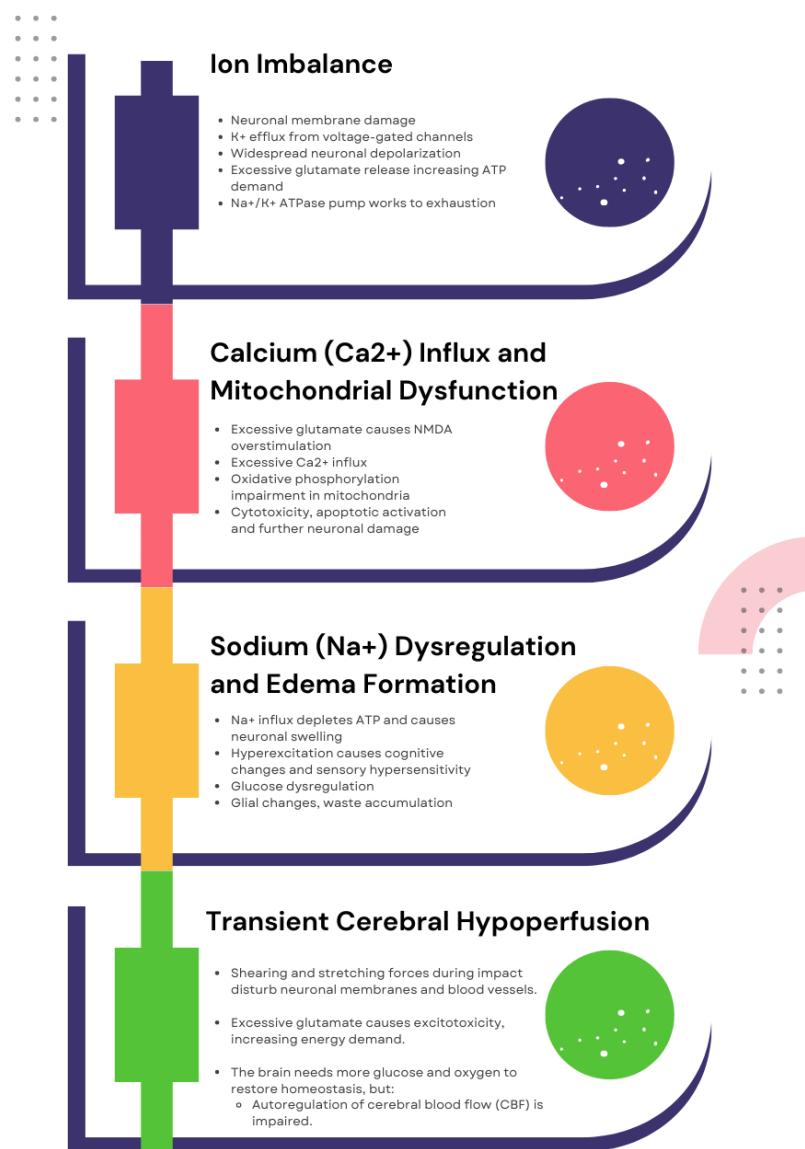
- Cerebral blood flow (CBF) decreases by 20-30% post-injury due to cerebrovascular dysregulation and neurovascular uncoupling ⁷
- Hypoxia
- Oxidative stress
- Reduced waste clearance/microglia and astrocyte activation

⁶ MacFarlane, M. P., & Glenn, T. C. (2015). Neurochemical cascade of concussion. *Brain Injury*, 29(2), 139–153. <https://doi.org/10.3109/02699052.2014.965208>

⁷ Maugans, T. A., Farley, C., Altaye, M., Leach, J., & Cecil, K. M. (2012). Pediatric sports-related concussion produces cerebral blood flow alterations. *Pediatrics*, 129(1), 28–37. <https://doi.org/10.1542/peds.2011-2083>



Chemical Cascade



Physiology

Symptom presentation

Cognitive dysfunction

- Reduced CBF, neuroinflammation, neurotransmitter imbalance, DAI, vascular damage can all contribute to changes in memory, mood, alertness and ADL difficulty.

Oculomotor distress

- The autonomic nervous system (ANS) is impaired, leading to
- Altered heart rate variability (HRV) and baroreceptor dysfunction
- Postural orthostatic tachycardia syndrome (POTS)⁸

Sleep disturbance

- Hyperarousal, autonomic dysfunction, impaired GABAergic inhibition
- Brain energy depletion, orexinergic dysfunction
- Circadian rhythm disruption, melatonin dysregulation
- Brainstem dysfunction affecting respiratory control
- REM disturbance (cholinergic system disruption)⁹

Mood

- Depression
 - Anxiety
 - Irritability
 - Emotional dysregulation,
 - Increased stress sensitivity
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⁸ Gall, N. (2021). Postural orthostatic tachycardia syndrome – An ‘invisible condition’ with far-reaching consequences. *Journal of Internal Medicine*, 290(4), 693–695. <https://doi.org/10.1111/jim.13265>

⁹ Purkayastha, S., Stokes, M., & Bell, K. R. (2019). Autonomic nervous system dysfunction in mild traumatic brain injury: A review of related pathophysiology and symptoms. *Brain Injury*, 33(9), 1129–1136.
<https://doi.org/10.1080/02699052.2019.1631488>

Physiological impact

Default Mode Network

The default mode network (DMN) is a large-scale brain network comprising the medial prefrontal cortex, posterior cingulate cortex, and angular gyrus that exhibits high metabolic activity during resting states and decreases during task-focused conditions. It is characterized by high functional connectivity between these regions during rest and is implicated in self-referential processing, episodic memory retrieval, and theory of mind operations.

Anatomy

- Medial Prefrontal Cortex (mPFC)
- Posterior Cingulate Cortex (PCC) & Precuneus
- Inferior Parietal Lobule (IPL)

Functional change

- Acute functional Hyperconnectivity (up tp 7 days)¹⁰
- Chronic functional hypoconnectivity¹¹
- Impaired task-related deactivation¹²
- Disrupted cross-network dynamics

Symptomatology

- EEG studies show increased theta/delta oscillations during cognitive tasks¹³
- Inability to shift from resting to active cognitive state generates symptoms such as:
- Concentration difficulties and distractibility
- Slower response times
- Mental fogginess

Long term implications:

- Chronic DMN hypoconnectivity (memory, attention, executive function)
- Mood disorders/anxiety
- Neurodegeneration risk (CTE, Alzheimer's)¹⁴

¹⁰ Slobounov, S., Slobounov, E., & Newell, K. M. (2011). Functional brain networks in healthy and mild traumatic brain injury subjects: A graph theory study. *NeuroImage*, 54(3), 2197–2208. <https://doi.org/10.1016/j.neuroimage.2010.10.058>

¹¹ Zhou, Y., Milham, M. P., Lui, Y. W., Miles, L., Reaume, J., Sodickson, D. K., Grossman, R. I., & Ge, Y. (2012). Default-mode network disruption in mild traumatic brain injury. *Radiology*, 265(3), 882–892. <https://doi.org/10.1148/radiol.12120748>

¹² McKee, A. C., Daneshvar, D. H., & Cantu, R. C. (2015). The pathology of chronic traumatic encephalopathy. *PLOS ONE*, 10(8), e0134019. <https://doi.org/10.1371/journal.pone.0134019>

¹³ Buchanan, D. M., Ros, T., & Nahas, R. (2021). Elevated and slowed EEG oscillations in patients with post-concussive syndrome and chronic pain following a motor vehicle collision. *Brain Sciences*, 11(5), 537. <https://doi.org/10.3390/brainsci11050537>

¹⁴ McKee, A. C., Cantu, R. C., Nowinski, C. J., Hedley-Whyte, E. T., Gavett, B. E., Budson, A. E., Santini, V. E., Lee, H.-S., Kubilus, C. A., & Stern, R. A. (2009). Chronic traumatic encephalopathy in athletes: Progressive tauopathy after repetitive head injury. *Journal of Neuropathology & Experimental Neurology*, 68(7), 709–735. <https://doi.org/10.1097/NEN.0b013e3181a9d503>

Physiological impact

Autonomic Nervous System

Autonomic Dysfunction & Disrupted Homeostasis

- Hypothalamus
- Brainstem (Medulla, Pons, Midbrain)
- Insular Cortex
- Cingulate Cortex & Prefrontal Cortex (PFC)

Sympathetic Dominance & Parasympathetic Withdrawal

- Hyperadrenergic State → Excessive norepinephrine¹⁵
- Vagal Dysfunction → Reduced parasympathetic tone and ACh release (Purkayastha, 2019)
- Tachycardia, GI dysfunction, heightened stress response, orthostatic intolerance

Sleep Disturbance

- Parasympathetic disturbance → Sympathetic dominance, reduced PNS tone¹⁶
- Circadian rhythm disruption¹⁷
- Reduced slow-wave sleep and REM → Increased risk of migraines and light sensitivity¹⁸

Cerebral Blood Flow (CBF) Dysregulation

- Hypoperfusion → Cognitive fatigue, headaches, dizziness.
 - Hyperperfusion during stress/exercise → Worsened post-concussion symptoms.
 - Vasospasm risk → Increased risk of migraines¹⁹
 - Endothelial dysfunction → increased blood brain barrier permeability
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¹⁵ Goodman, B., Vargas, B., & Dodick, D. (2013). Autonomic nervous system dysfunction in concussion (P01.265). *Neurology*, 80(7 Supplement), P01.265. https://doi.org/10.1212/WNL.80.7_supplement.P01.26

¹⁶ Galea, M. P., Ramanan, S., & Wightman, W. (2013). Sleep disturbances in mild traumatic brain injury: Role of the autonomic nervous system. *Journal of Neurotrauma*, 30(14), 1213–1220. <https://doi.org/10.1089/neu.2012.2746>

¹⁷ Ayalon, L., Borodkin, K., Dishon, L., Kanety, H., & Dagan, Y. (2016). Circadian rhythm sleep disorders following mild traumatic brain injury. *Journal of Clinical Sleep Medicine*, 12(10), 1373–1379. <https://doi.org/10.5664/jcsm.6160>

¹⁸ Donahue, C. C., & Resch, J. E. (2024). Concussion and the sleeping brain. *Sports Medicine - Open*, 10, 68. <https://doi.org/10.1186/s40798-024-00736-2>

¹⁹ Gao, Y., Li, K., Li, X., Li, Q., Wang, J., Zhang, S., & Zhang, J. (2022). Exploration of cerebral vasospasm from the perspective of microparticles. *Frontiers in Neuroscience*, 16, 1013437. <https://doi.org/10.3389/fnins.2022.1013437>

Typical Clinical Presentation of Post-Concussive Autonomic Dysregulation

A 22-year-old college athlete presents 3 weeks after sustaining a concussion during soccer practice. While her initial acute concussion symptoms (headache, confusion) have largely resolved, she now reports:

Chief complaints:

- Severe fatigue that worsens throughout the day
- Dizziness when standing up from bed or chairs
- Heart racing with minimal activity like walking to class
- Difficulty sleeping despite exhaustion

Physical examination findings:

- Resting heart rate: 95 bpm (baseline was 60 bpm as an athlete)
- Orthostatic vitals: HR increases from 95 to 130 bpm upon standing, with minimal BP change
- Pupils reactive but slightly sluggish response to light
- Appears fatigued but neurologically intact otherwise

Reported symptoms:

- Cannot tolerate her usual exercise routine - heart rate shoots up to 160+ with light jogging
- Feels overheated easily and sweats excessively with minor exertion
- Nausea, especially in the mornings
- "Brain fog" and difficulty concentrating in classes
- Anxiety symptoms she never had before the injury
- Sleep onset insomnia followed by unrefreshing sleep

Functional impact:

- Had to reduce course load from full-time to part-time
- Unable to participate in athletics
- Requires frequent rest breaks during daily activities
- Uses elevator instead of stairs due to symptoms

This presentation shows the classic triad of exercise intolerance, orthostatic symptoms, and sleep disruption that characterizes post-concussive autonomic dysfunction, significantly impacting her return to normal activities despite resolution of initial concussion symptoms.

Quiz: Symptoms of Default Mode Network (DMN) Dysfunction

Instructions:

Select the most appropriate answer for each question. This quiz is designed to test your understanding of how concussion can affect the default mode network (DMN) and its associated symptoms.

1. Which of the following is a common symptom associated with dysfunction of the default mode network (DMN)?

- a) Auditory hallucinations
 - b) Poor memory and attention
 - c) Increased heart rate
 - d) Inability to speak
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2. DMN dysfunction can lead to which of the following cognitive impairments?

- a) Impaired short-term memory
 - b) Impaired coordination and motor control
 - c) Reduced tactile sensitivity
 - d) Inability to recognize faces
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3. The default mode network is primarily active when an individual is:

- a) Engaged in focused tasks
 - b) At rest or mind-wandering
 - c) Processing external sensory information
 - d) Performing complex motor tasks
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4. Dysfunction of the DMN in concussion can lead to which of the following psychological symptoms?

- a) Depression and anxiety
 - b) Increased physical strength
 - c) Enhanced visual perception
 - d) Increased motivation
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5. Which of the following brain regions is a key component of the DMN?

- a) Occipital lobe
 - b) Prefrontal cortex
 - c) Cerebellum
 - d) Brainstem
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6. Which of the following is least likely to be associated with DMN dysfunction in concussion?

- a) Disrupted self-reflection
 - b) Difficulty with planning and decision-making
 - c) Heightened awareness of external stimuli
 - d) Problems with memory recall
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Physiological impact

Oculomotor dysfunction

Saccades²⁰

- Rapid eye movements used to shift gaze
- Damage to brainstem nuclei and cortical regions involved in saccadic planning and execution
- Concussed individuals may exhibit increased saccadic latency (delay in initiating saccades) and reduced saccadic velocity

Smooth Pursuit (Murray, 2020)

- Superior colliculus: Visual input → motor output used to track moving objects
- Cerebellum neural integrator function connecting vestibular and proprioceptive inputs to calibrate intended eye movements

Vergence²¹

- Midbrain nuclei containing saccade-vergence burst neurons (SVBNs)
- Information from cerebellum and vestibular systems to aid binocularity
- Convergence - inward movement to near objects
- Divergence - outward movement to far objects
- Accommodation - focus via lens curvature

Vestibulo-ocular Reflex (VOR)²²

- Mismatch between vestibular input and visual input due to damage to vestibular organs (inner ear), CNVIII or brainstem nuclei
 - Gaze stabilization, orientation (dizziness, vertigo) of visual images on the retina during head movements by producing compensatory eye movements in the opposite direction
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²⁰ Murray, N. G., Szekely, B., Islas, A., Munkasy, B., Gore, R., Berryhill, M., & Reed-Jones, R. J. (2020). Smooth pursuit and saccades after sport-related concussion. *Journal of Neurotrauma*, 37(2), 340–346. <https://doi.org/10.1089/neu.2019.6595>

²¹ Mays, L. E. (1984). Neural control of vergence eye movements: Convergence and divergence neurons in midbrain. *Journal of Neurophysiology*, 51(5), 1091–1108. <https://doi.org/10.1152/jn.1984.51.5.1091>

²² Somisetty, S., & Das, J. M. (2023). Neuroanatomy, vestibulo-ocular reflex. In *StatPearls*. StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK545297/>

Severity

Acute

<72hrs

- Headaches, dizziness, nausea, confusion, memory disturbance, nausea, light and sound sensitivity, and sleep disturbances
- Immediate cellular changes such as neuroinflammation and altered metabolic activity in the brain

Subacute

72hrs - 1-2 weeks

- Signs of recovery, reduced frequency and intensity of symptoms
- Continued neuroinflammation and neurochemical dysfunction
- Persistent cognitive difficulty

Chronic²³

70-80% fully recover >3 months

- Ongoing neuronal changes such as chronic neuroinflammation, neurodegeneration, and structural brain abnormalities
- Tau/S100b protein accumulation secondary to inability to clear waste

Post Concussion Syndrome

<3 months - Years

- Neural damage, neurotransmitter imbalances, and psychological factors
 - Persistent changes in brain activity, neuroinflammation, and altered brain network connectivity
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²³ Haarbauer-Krupa, J., Pugh, M. J., Prager, E. M., Harmon, N., Wolfe, J., & Yaffe, K. (2021). Epidemiology of chronic effects of traumatic brain injury. *Journal of Neurotrauma*, 38(15), 1-16. <https://doi.org/10.1089/neu.2020.7541>

Post Concussion Syndrome (Persistent Post Concussive Symptoms PPCS)

Psychogenic Factors²⁴

Persistent post-concussion symptoms can be amplified or perpetuated by psychological comorbidities, often creating a complex interplay between neurological injury and mental health. These may include:

- Anxiety: Heightened vigilance and somatic symptom focus can exacerbate headache, dizziness, and fatigue. Patients may also avoid activities due to fear of symptom provocation, leading to deconditioning and increased sensitivity.
 - Depression: Associated with anhedonia, fatigue, poor concentration, and sleep disruption — symptoms that overlap with PCS, often complicating diagnosis.
 - Post-Traumatic Stress Disorder (PTSD): Especially common in patients whose concussion was sustained in emotionally charged events (e.g., motor vehicle accidents, assault). PTSD may lead to chronic arousal, avoidance behavior, intrusive thoughts, and autonomic instability, all of which worsen PCS recovery trajectories.
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Structural Brain Changes²⁵

Advanced neuroimaging techniques, especially MRI-based morphometric analysis, have revealed subtle yet persistent structural alterations in some individuals with prolonged concussion symptoms:

- Cortical Thinning: Studies have demonstrated reduced cortical thickness in the prefrontal cortex, anterior cingulate cortex, and medial temporal lobes — areas involved in executive function, emotional regulation, and memory.
- White Matter Abnormalities: Diffusion Tensor Imaging (DTI) frequently reveals:
 - Decreased fractional anisotropy (FA), suggesting demyelination or axonal injury.
 - Increased mean diffusivity (MD), indicating extracellular fluid accumulation or axonal degeneration.

These findings support the theory of microstructural disruption as a contributor to persistent cognitive and sensorimotor symptoms following concussion.

²⁴ Iverson, G. L. (2019). Network analysis and precision rehabilitation for the post-concussion syndrome. *Frontiers in Neurology*, 10, 489. <https://doi.org/10.3389/fneur.2019.00489>

²⁵ Dean, P. J. A., Sato, J. R., Vieira, G., McNamara, A., & Sterr, A. (2015). Long-term structural changes after mTBI and their relation to post-concussion symptoms. *Brain Injury*, 29(10), 1211–1218. <https://doi.org/10.3109/02699052.2015.1035334>

Autonomic Dysregulation²⁶

Concussions can disrupt central autonomic regulatory networks, particularly those involving the brainstem, hypothalamus, and insula, resulting in symptoms consistent with autonomic nervous system dysfunction (dysautonomia).

Key manifestations include:

- Abnormal Heart Rate Variability (HRV): Reduced parasympathetic tone and exaggerated sympathetic activation, especially during exertion or orthostatic stress. This may contribute to:
 - Lightheadedness
 - Tachycardia (including POTS – Postural Orthostatic Tachycardia Syndrome)
 - Fatigue and exercise intolerance
 - Cerebral Blood Flow (CBF) Dysregulation: Dysfunctional neurovascular coupling may lead to:
 - Hypoperfusion in frontal/temporal lobes → impaired cognition, headaches
 - Hyperperfusion during cognitive load → symptom exacerbation and delayed recovery
 - Vasospasm and Endothelial Dysfunction: These can impair the blood-brain barrier (BBB), increasing vulnerability to neuroinflammation and post-injury neurotoxicity.
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Altered CSF Dynamics^{27 28}

Emerging research has identified cerebrospinal fluid (CSF) flow impairment as a potential contributor to prolonged symptoms:

- Ependymal Ciliary Loss: Traumatic Brain Injury (TBI) can damage the ependymal cilia that line the ventricles, reducing CSF propulsion and circulation.
- CSF Stasis: Impaired CSF clearance may contribute to:
 - Neuroinflammation
 - Waste accumulation (e.g., tau, S100B protein)
 - Head pressure symptoms (e.g., persistent headache, cognitive fog)

²⁶ Mercier, L. J., Batycky, J., Campbell, C., Schneider, K., Smirl, J., & Debert, C. T. (2022). Autonomic dysfunction in adults following mild traumatic brain injury: A systematic review. *NeuroRehabilitation*, 50(1), 1–30. <https://doi.org/10.3233/NRE-210243>

²⁷ Xiong, G., Elkind, J. A., Kundu, S., Smith, C. J., Antunes, M. B., Tamashiro, E., Kofonow, J. M., Mitala, C. M., Cole, J., Stein, S. C., Grady, M. S., Einhorn, E., Cohen, N. A., & Cohen, A. S. (2014). Traumatic brain injury-induced ependymal ciliary loss decreases cerebral spinal fluid flow. *Journal of Neurotrauma*, 31(16), 1396–1404. <https://doi.org/10.1089/neu.2013.3110>

²⁸ Fultz, N. E., Bonmassar, G., Setsompop, K., Stickgold, R. A., Rosen, B. R., Polimeni, J. R., & Lewis, L. D. (2022). Increased cerebrospinal fluid flow with deep breathing in humans. *Scientific Reports*, 12(1), 11678. <https://doi.org/10.1038/s41598-022-15034-8>

Imaging

Computed Tomography (CT) Scan

- Structural injuries to skull
- Intracranial hemorrhage (subdural/epidural hematoma)
- General brain tissue contusion

Advanced Magnetic Resonance Imaging (MRI)

- **fMRI** - blood oxygenation levels to evaluate brain activity during cognitive tasks and identify areas of reduced activity or abnormal neural responses after concussion²⁹
- **Diffusion Tensor Imaging (DTI)** - integrity of white matter by measuring the diffusion of water molecules along axonal fibers. DTI can detect microstructural changes such as axonal injury that are often missed by conventional MRI³⁰

Magnetic Resonance Spectroscopy (MRS)³¹

- Biochemical composition of brain tissue.
- Measures concentration of metabolites such as N-acetylaspartate (NAA), choline, and creatine.
- Changes in these metabolites can indicate neuronal injury, metabolic dysfunction, and inflammation.

Single Photon Emission Computed Tomography (SPECT)³²

- 3D images by detecting the gamma rays emitted by radioactive tracers injected into the bloodstream.
- SPECT can assess cerebral blood flow (CBF) and detect areas of hypoperfusion, which may be indicative of concussion-related brain dysfunction.

Positron Emission Tomography (PET)³³

- Radioactive tracer to assess the brain's metabolic activity and detect areas of abnormal energy consumption.
- More sensitive than other imaging techniques in detecting changes in brain function following concussion, such as alterations in glucose metabolism or the presence of neuroinflammation.

²⁹ Bigler, E. D. (2015). Neuroimaging biomarkers in mild traumatic brain injury (mTBI). *Neuropsychology Review*, 25(1), 15–34. <https://doi.org/10.1007/s11065-015-9289-x>

³⁰ Hulkower, M. B., Poliak, D. B., Rosenbaum, S. B., Zimmerman, M. E., & Lipton, M. L. (2013). A decade of DTI in traumatic brain injury: 10 years and 100 articles later. *American Journal of Neuroradiology*, 34(11), 2064–2074. <https://doi.org/10.3174/ajnr.A3395>

³¹ Henry, L. C., Tremblay, S., Leclerc, S., Khiat, A., Boulanger, Y., Ellemborg, D., & Lassonde, M. (2010). Metabolic changes in concussed American football players during the acute and chronic post-injury phases. *BMC Neurology*, 10, 105. <https://doi.org/10.1186/1471-2377-10-105>

³² Jacobs, A., Put, E., Ingels, M., Bossuyt, A., & Goffin, J. (1996). One-year follow-up of technetium-99m HMPAO SPECT imaging in mild head injury and cognitive dysfunction. *Journal of Nuclear Medicine*, 37(10), 1605–1609.

³³ Heretich, R. M., & Sager, T. N. (2020). Positron emission tomography in mild traumatic brain injury: A systematic review. *Brain Imaging and Behavior*, 14(6), 1441-1454. <https://doi.org/10.1007/s11682-020-00338-5>

Near-Infrared Spectroscopy (NIRS)³⁴

- Non-invasive method used to measure cerebral oxygenation and hemodynamic changes in the brain.
 - Uses infrared light to assess the absorption of light by oxygenated and deoxygenated hemoglobin
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³⁴ Urban, K., Irwin, J., Sheffield, M., & Viano, D. C. (2021). Near-infrared spectroscopy for assessing cerebral hemodynamics following sports-related concussion: A systematic review. *Brain Injury*, 35(6), 722–736.
<https://doi.org/10.1080/02699052.2021.1912356>

Identifiable pathology

Diffuse Axonal Injury (DAI)³⁵

Pathophysiology:

- **Cytoskeletal damage** (spectrin and neurofilament breakdown)
- **Impaired axoplasmic transport**, resulting in axonal swelling and eventual disconnection
- **Secondary neuroinflammation**, leading to neurodegeneration

Imaging Detection:

- **Diffusion Tensor Imaging (DTI):**
 - Reduced **fractional anisotropy (FA)** in major white matter tracts (e.g., corpus callosum, internal capsule, superior longitudinal fasciculus)
 - Increased **mean diffusivity (MD)** indicating axonal degeneration and edema
- **Susceptibility-Weighted Imaging (SWI):**
 - Identifies **microhemorrhages** at the gray-white matter junction, commonly seen in DAI

White Matter Disruptions and Myelin Damage³⁶

Pathophysiology:

- Oligodendrocyte dysfunction, leading to **demyelination** and impaired synaptic transmission.
- Secondary ischemia exacerbates white matter injury, resulting in **axonal degeneration** and cognitive dysfunction.

Imaging Detection:

- **DTI and Neurite Orientation Dispersion and Density Imaging (NODDI):**
 - Decreased FA and increased MD in **major white matter tracts**
- **Magnetization Transfer Imaging (MTI):**
 - Detects reductions in magnetization transfer ratio (MTR), a marker of myelin integrity

³⁵ Hulkower, M. B., Poliak, D. B., Rosenbaum, S. B., Zimmerman, M. E., & Lipton, M. L. (2013). A decade of DTI in traumatic brain injury: 10 years and 100 articles later. *AJNR: American Journal of Neuroradiology*, 34(11), 2064–2074.
<https://doi.org/10.3174/ajnr.A3395>

³⁶ Mayer, A. R., Ling, J. M., & Mannell, M. V. (2010). Diffusion abnormalities in pediatric mild traumatic brain injury. *Journal of Neuroscience*, 30(32), 10963–10970. <https://doi.org/10.1523/JNEUROSCI.3379-12.2012>

Microvascular Injury and Cerebral Perfusion Abnormalities³⁷

Pathophysiology:

- Concussion disrupts the neurovascular unit, leading to capillary damage, pericyte dysfunction, and endothelial barrier breakdown.
- This results in ischemia, oxidative stress, and hypoperfusion, contributing to persistent post-concussion symptoms.

Imaging Detection:

- **Perfusion-Weighted MRI (PWI) & Arterial Spin Labeling (ASL):**
 - Detects cerebral hypoperfusion in the frontal and temporal lobes
 - PWI uses a contrast agent where ASL uses a magnetic label
- **Susceptibility-Weighted Imaging (SWI)**
 - Identifies microhemorrhages in deep white matter and periventricular areas
 - Sensitive to substances with different magnetic signatures within the blood (O₂, calcium, iron, hemosiderin)

Blood-Brain Barrier (BBB) Disruption³⁸

Pathophysiology:

- Tight junction disruptions in the endothelial layer, leading to BBB permeability.
- Increased permeability allows inflammatory cytokines, excitotoxic glutamate, and autoantibodies to enter the CNS, promoting neuroinflammation.

Imaging Detection:

- **Dynamic Contrast-Enhanced MRI (DCE-MRI):**
 - Identifies regions of increased BBB permeability, correlating with symptom severity
 - **PET Imaging with Translocator Protein (TSPO) Ligands:**
 - Detects activated microglia and neuroinflammation
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³⁷ Kou, Z., Wu, Z., & Benson, R. R. (2010). The role of advanced MR imaging findings as biomarkers of traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 25(4), 267–282. <https://doi.org/10.1097/HTR.0b013e3181e54793>

³⁸ Marchi, N., Bazarian, J. J., & Janigro, D. (2016). Consequences of repeated blood-brain barrier disruption in football players. *PLoS ONE*, 8(3), e0156805. <https://doi.org/10.1371/journal.pone.0156805>

Cortical Thinning and Structural Atrophy³⁹

Pathophysiology:

- Chronic neurodegeneration following concussion leads to gray matter atrophy, particularly in the prefrontal cortex, hippocampus, and cingulate cortex.
- Atrophy is associated with memory deficits, executive dysfunction, and emotional dysregulation.

Imaging Detection:

- **Voxel-Based Morphometry (VBM) with MRI:**
 - Detects gray matter volume loss
- **Cortical Thickness Analysis:**
 - Reveals progressive thinning in concussion-prone regions

Cerebral Metabolic Dysfunction⁴⁰

Pathophysiology:

- Concussion disrupts **glucose metabolism**, causing a "neurometabolic cascade" involving:
 - **ATP depletion**, mitochondrial dysfunction, and oxidative stress
 - **Impaired glutamate clearance**, leading to excitotoxicity
 - **Delayed metabolic recovery**, persisting for weeks to months

Imaging Detection:

- **Magnetic Resonance Spectroscopy (MRS):**
 - Reduced **N-acetylaspartate (NAA)/Creatine ratio**, indicating neuronal damage
- **Fluorodeoxyglucose Positron Emission Tomography (FDG-PET):**
 - Detects regional **hypometabolism**, particularly in the **cingulate gyrus and medial temporal lobes**

³⁹ Wang, X., Xie, H., Cotton, A. S., Tamburino, M. B., Brickman, K. R., Lewis, T. J., McLean, S. A., & Liberzon, I. (2015). Early cortical thickness change after mild traumatic brain injury following motor vehicle collision. *Journal of Neurotrauma*, 32(7), 455–463. <https://doi.org/10.1089/neu.2014.3492>

⁴⁰ Henry, L. C., Tremblay, S., Leclerc, S., Khiat, A., Boulanger, Y., Ellemborg, D., & Lassonde, M. (2011). Metabolic changes in concussed American football players during the acute and chronic post-injury phases. *BMC Neurology*, 11, 105. <https://doi.org/10.1186/1471-2377-11-105>

Default Mode Network (DMN) Dysfunction⁴¹

Pathophysiology:

- The DMN, crucial for attention, memory, and executive function, becomes hyper connected *and* disconnected post-concussion.
- This leads to brain network inefficiency, affecting cognitive recovery.

Imaging Detection:

- **Resting-State fMRI (rs-fMRI):**

- Shows abnormal connectivity patterns in the posterior cingulate cortex and medial prefrontal cortex

⁴¹ Johnson, B., Zhang, K., Gay, M., Horovitz, S., Hallett, M., Sebastianelli, W., & Slobounov, S. (2011). Alteration of brain default network in subacute phase of injury in concussed individuals: Resting-state fMRI study. *Brain Imaging and Behavior*, 5(3), 295–303. <https://doi.org/10.1007/s11682-011-9133-2>

Biomarkers

Glial Fibrillary Acidic Protein (GFAP)⁴²

- **What It Is:** GFAP is a protein expressed by astrocytes and is released into the bloodstream after brain injury. Elevated levels of GFAP indicate glial injury, a common feature of concussion and traumatic brain injury (TBI).
- **Why It Is Used:** Studies have demonstrated that GFAP can be a sensitive biomarker for detecting concussion, particularly in the acute phase.

S100B Protein⁴³

- **What It Is:** S100B is a calcium-binding protein predominantly found in astrocytes. It has been proposed as a marker for brain injury because its levels increase in response to neurodegeneration.
- **Why It Is Used:** Increased levels of S100B have been observed in patients with concussion and mild TBI, and it may provide an indication of brain injury, although its specificity is debated.

Ubiquitin C-Terminal Hydrolase L1 (UCH-L1)⁴⁴

- **What It Is:** UCH-L1 is a neuronal protein involved in the regulation of ubiquitin-proteasome pathways and is released after neuronal injury.
- **Why It Is Used:** Elevated levels of UCH-L1 in the blood have been associated with concussion and are suggested as an early biomarker for detecting TBI.

Tau Protein⁴⁵

- **What It Is:** Tau is a microtubule-associated protein that stabilizes the microtubule structure in neurons. Abnormal tau protein release into the bloodstream has been linked with neurodegeneration.
 - **Why It Is Used:** Elevated tau levels following concussion may suggest axonal injury and neurodegeneration. Tau is often used as a marker of more severe brain injuries, including chronic traumatic encephalopathy (CTE).
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⁴² Huibregtse, M. E., Bazarian, J. J., Shultz, S. R., & Kawata, K. (2021). The biological significance and clinical utility of emerging blood biomarkers for traumatic brain injury. *Frontiers in Neurology*, 12, 703118. <https://doi.org/10.3389/fneur.2021.703118>

⁴³ Thelin, E. P., Jeppsson, E., Frostell, A., Svensson, M., Mondello, S., Bellander, B.-M., & Nelson, D. W. (2016). Utility of neuron-specific enolase in traumatic brain injury: relations to S100B levels, outcome, and extracranial injury severity. *Critical Care*, 20(1), 82. <https://doi.org/10.1186/s13054-016-1450-y>

⁴⁴ Spitz, G., Hicks, A. J., McDonald, S. J., Dore, V., Krishnadas, N., O'Brien, T. J., O'Brien, W. T., Vivash, L., Law, M., Ponsford, J. L., Rowe, C., & Shultz, S. R. (2023). Plasma biomarkers in chronic single moderate-severe traumatic brain injury. *Brain*, 146(3), 1030-1039. <https://doi.org/10.1093/brain/awae255>

⁴⁵ McKee, A. C., Daneshvar, D. H., Alvarez, V. E., & Stein, T. D. (2014). The neuropathology of sport. *Acta Neuropathologica*, 127(1), 29-51. <https://doi.org/10.1007/s00401-013-1257-3>

Neuroimaging Biomarkers⁴⁶

- **What They Are:** Advanced neuroimaging techniques, such as functional MRI (fMRI), diffusion tensor imaging (DTI), and positron emission tomography (PET), are used to detect brain changes associated with concussion.
- **Why They Are Used:** These imaging techniques can identify alterations in brain activity, white matter integrity, and changes in blood flow, which may provide insight into the structural and functional consequences of concussion.

MicroRNA (miRNA) Biomarkers⁴⁷

- **What They Are:** miRNAs are small non-coding RNA molecules that regulate gene expression. Certain miRNAs are associated with brain injury and inflammation after concussion.
- **Why They Are Used:** Elevated levels of specific miRNAs in the blood may be indicative of concussion and could provide an early biomarker for diagnosing and monitoring recovery from brain injury.

Neurofilament Light Chain (NfL)⁴⁸

- **What It Is:** NfL is a structural protein found in neurons that is released into the blood following axonal damage.
- **Why It Is Used:** Elevated levels of NfL are associated with axonal injury, making it a promising biomarker for detecting concussions and monitoring recovery.

⁴⁶ Gajawelli, N., Lao, Y., Apuzzo, M. L. J., Romano, R., Liu, C., Tsao, S., Hwang, D., Wilkins, B., Lepore, N., & Law, M. (2013). Neuroimaging changes in the brain in contact vs. non-contact sport athletes using Diffusion Tensor Imaging. *PLOS ONE*, 8(10), e76616. <https://doi.org/10.1371/journal.pone.0076616>

⁴⁷ Di Pietro, V., Yakoub, K. M., Scarpa, U., Di Pietro, C., & Belli, A. (2020). MicroRNA signature of traumatic brain injury: From the biomarker discovery to the point-of-care. *Frontiers in Neurology*, 11, 570. <https://doi.org/10.3389/fneur.2020.00570>

⁴⁸ Kulbe, J. R., & Geddes, J. W. (2015). Current status of fluid biomarkers in mild traumatic brain injury. *Journal of Neurotrauma*, 32(6), 315-324. <https://doi.org/10.1089/neu.2014.3780>

Module 1 Summary: Introduction to concussion and brain injury

Learning Objectives Achieved ✓

Mechanism Recognition: Identifies primary biomechanical forces and associated brain regions/symptoms.

Classification Application: Applies modern functional vs. structural and phenotype-based concussion classifications.

Pathophysiology Link: Connects neurometabolic cascade processes to clinical presentations.

Phenotype Identification: Categorises concussion presentations into evidence-based phenotypes for targeted management.

Evidence-Based Practice: Differentiates current active rehabilitation approaches from outdated management practices.

Assessment Proficiency: Performs core neuromusculoskeletal, vestibular, ocular, and autonomic screening.

Recovery Awareness: Describes expected recovery phases, risk factors, and urgent referral red flags.

Essential Mechanisms & Classifications

Primary Injury Mechanisms

Mechanism	Brain Regions Affected	Key Clinical Symptoms
Linear Acceleration	Frontal & occipital lobes, brainstem	Headache, blurred vision, LOC
Rotational Forces	Corpus callosum, midbrain, cerebellum	Dizziness, balance loss, emotional instability
Blunt Impact	Focal cortex, frontal/temporal poles	Confusion, localized deficits
Whiplash	Cervical cord, brainstem, vestibular nuclei	Neck pain, cervicogenic headache

Modern Classification Framework

- Functional vs Structural: Concussion = functional injury without visible imaging damage
 - Primary vs Secondary: Immediate trauma vs subsequent swelling/metabolic disruption
 - Severity Grading: Moved from outdated scales to individualized phenotype-based assessment
-

Neurophysiological Cascade: From Impact to Symptoms

The Neurometabolic Cascade

Immediate (0-72 hours):

- K^+ efflux & glutamate excess → Hyperexcitable brain state
- Ca^{2+} influx → Mitochondrial dysfunction → ↓ ATP production
- Na^+ dysregulation → Cellular edema → Axonal signal delays

Ongoing Effects:

- Cerebral Blood Flow: Hypoperfusion → Reduced oxygen/glucose delivery
- Neuroinflammation: Microglial activation → Connectivity disruption
- Default Mode Network (DMN): Hyperconnectivity (acute) → Hypoconnectivity

Clinical Symptom Correlation

Brain Region	Primary Function	Concussion Symptoms
Frontal Lobe	Executive function, attention	Poor focus, impulsivity, mood changes
Temporal Lobe	Memory, emotion	Memory loss, irritability
Corpus Callosum	Interhemispheric communication	Processing delays, coordination issues
Brainstem (RAS)	Arousal, balance	LOC, nausea, dizziness
Cerebellum	Motor coordination	Ataxia, vertigo

Clinical Phenotypes for Targeted Management

Evidence-Based Phenotype Approach

Phenotype	Primary Symptoms	Clinical Focus
Vestibular	Dizziness, balance problems, vertigo	Balance rehabilitation, vestibular therapy
Ocular	Visual disturbances, tracking difficulties	Vision therapy, oculomotor rehabilitation
Cognitive	Brain fog, slowed processing, memory issues	Cognitive rest and retraining
Migrainous	Headache with migraine features	Migraine-specific management
Affective	Emotional dysregulation, anxiety	Psychological support

Outdated Practices to Avoid

- Complete rest until symptom-free: Evidence supports gradual activity after 24-48 hours
 - Total screen avoidance: Gradual reintroduction as tolerated reduces isolation
 - Waiting for full resolution: Light activity is now part of active rehabilitation
-

Diagnostic & Imaging Considerations

Advanced Neuroimaging Applications

Modality	Primary Use	Key Findings
fMRI	DMN connectivity assessment	Altered network connectivity patterns
DTI	White matter integrity	↓ Fractional Anisotropy (axonal damage)
MRS	Metabolic dysfunction	↓ NAA/Creatine ratio (neuronal damage)
PET/SPECT	Perfusion & metabolism	↓ Regional blood flow/glucose uptake

Biomarker Integration

- GFAP: Sensitive astrocyte injury marker
- UCH-L1: Early neuronal injury detection
- NfL: Axonal damage monitoring
- Tau: Long-term neurodegeneration risk



Integrated Clinical Assessment Framework

Neuromusculoskeletal Screening (5-minute protocol)

1. Cervical ROM with overpressure → Identify WAD/cervicogenic headache
2. BPPV testing (Dix-Hallpike) → Vestibular dysfunction
3. Modified BESS → Balance/proprioceptive deficits
4. Scapular control assessment → Upper limb compensation patterns
5. Core stability testing → Lumbopelvic dysfunction

Autonomic Assessment Indicators

- Heart Rate Variability: Reduced HRV → Sympathetic dominance
- Orthostatic Testing: POTS-like symptoms common post-concussion
- Cerebral Blood Flow: Neurovascular coupling dysfunction

Recovery Trajectory & Complications

Expected Timeline

- Acute (<72 hrs): Rapid metabolic/inflammatory changes
- Subacute (3 days-2 weeks): Symptom reduction begins
- Chronic (>3 months): 70-80% full recovery; 20-30% persistent symptoms

Post-Concussion Syndrome (PCS) Risk Factors

- Psychogenic: Pre-existing anxiety/depression amplifies symptoms
- Structural: Cortical thinning, white matter disruption
- Autonomic: HRV dysfunction, CBF dysregulation
- CSF Dynamics: Impaired waste clearance, neuroinflammation persistence

Clinical Red Flags Requiring Immediate Referral

- Progressive neurological deterioration
- Signs of increased intracranial pressure
- Severe autonomic dysfunction
- Psychiatric crisis or suicidal ideation

Clinical Pearl: Modern concussion management emphasizes individualized, phenotype-driven approaches rather than one-size-fits-all protocols. Early identification of specific symptom clusters enables targeted interventions and optimizes recovery outcomes.

APPENDIX: Clinical Reference Tools

Quick Phenotype Identification Guide

Vestibular Cluster: Dizziness + Balance issues + Nausea
→ *Focus:* BPPV assessment, VOR testing, balance rehabilitation

Ocular Cluster: Blurred vision + Reading difficulty + Screen sensitivity
→ *Focus:* Saccade/pursuit testing, convergence assessment, vision therapy

Cognitive Cluster: Brain fog + Slowed processing + Memory issues
→ *Focus:* Cognitive load management, graduated return to mental tasks

Migrainous Cluster: Headache + Light sensitivity + Nausea
→ *Focus:* Migraine protocols, trigger identification, pharmacological support

Neuromusculoskeletal Assessment Checklist

- Neck mobility (active ROM + overpressure)
- Cervicogenic headache (C1-C3 palpation, suboccipitals)
- BPPV screening (Dix-Hallpike or head roll test)
- Balance testing (modified BESS: eyes open/closed on foam)
- Scapular control (wall push-up or resisted abduction)
- Core engagement (supine transverse abdominis with draw-in)

Oculomotor Function Screen

Saccades: Rapid gaze shifts between targets (30cm apart)
Positive: Delayed initiation, overshooting, "sticky" movement

Smooth Pursuit: Follow pen in slow "H" pattern
Positive: Jerky tracking, inability to maintain smooth pursuit

Vergence: Pen from arm's length to nose
Positive: Convergence failure, diplopia, eye strain

VOR (Gaze Stability): Head movement while fixating on target
Positive: Image blur, visual jumping, dizziness

DMN Dysfunction Clinical Indicators

- Task-switching difficulties: Cannot shift from rest to active engagement
- Sustained attention deficits: Mind-wandering during focused tasks
- Mental fatigue disproportionate to effort: Cognitive exhaustion with minimal load
- EEG findings: ↑ Theta/delta activity during cognitive tasks

Imaging Decision Tree

Normal CT/MRI + Persistent cognitive symptoms → Consider MRS for metabolic dysfunction
Emotional lability + Chronic symptoms → VBM for gray matter assessment
Suspected BBB disruption → PET with TSPO ligand for neuroinflammation
Balance/visual tracking issues → DTI for white matter integrity
Real-time oxygenation monitoring → NIRS during exertional testing

Recovery Phase Management

Acute (0-72 hrs): Relative rest, symptom monitoring, avoid exertion

Subacute (3 days-2 weeks): Gradual activity progression, phenotype-specific therapy
Chronic (>3 months): Comprehensive interdisciplinary approach, PCS protocols

Module 2: Concussion Diagnosis & Initial Assessment Theory

Learning Objectives:

- Display knowledge of current diagnostic criteria to evaluate suspected concussion cases across age groups.
- Identify the components of a concussion assessment and describe typical clinical presentation.
- Explain physical and cognitive assessment approaches including balance, memory, and coordination (balance, memory, coordination).
- Critically evaluate the limitations of current diagnostic tools and theoretical models in accurately identifying concussion and its mimics.

Activities & Clinical Skill Application

This section focuses on practical exercises and case-based learning to reinforce theoretical knowledge related to concussion diagnosis and initial assessment. Participants will engage in activities that enhance their ability to identify concussion symptoms, utilize diagnostic tools like SCAT/SCOAT 6, and apply clinical reasoning for initial concussion evaluation. The activities are designed to improve diagnostic accuracy, symptom recognition, and immediate clinical response in the acute phase following concussion.

Learning outcomes

1. Identify and interpret the clinical signs and symptoms of concussion across age groups.
2. Demonstrate understanding of the SCAT, SCOT6 and Child SCAT structure and components.
3. Discuss limitations and proper clinical contexts for using standard assessment tools.
4. Explain the relevance of vestibular and oculomotor function in concussion assessment.
5. Recognize red flags that necessitate emergency referral or imaging.
6. Interpret clinical findings in the context of differential diagnoses (e.g., migraine, whiplash, vestibular dysfunction).

Current tools

Acute / on field

1. Immediate On-Field/Scene Evaluation (Primary Assessment)⁴⁹

This phase focuses on identifying Initial/obvious signs, red flags of severe brain or spinal injuries before proceeding with concussion-specific testing.

A. ABCs (Airway, Breathing, Circulation) – Life-Saving First

- Ensure **airway** is clear.
- Check **breathing** and pulse (**C**).
- If there is concern about a spinal injury, immobilize the neck.

B. Red Flag Symptoms – Immediate Emergency Referral

- Loss of consciousness (LOC) > 1 minute
- Seizures
- Worsening headache, vomiting, or increasing confusion
- Weakness or numbness in limbs
- Unequal pupil size or fixed pupils

If any of these are present → Immediate Emergency Medical Referral (Call EMS, Hospital Transfer).

2. Rapid Neurological and Cognitive Screening (Sideline or Acute Setting)

If the player/individual is conscious and stable, a focused concussion assessment begins.

A. Glasgow Coma Scale (GCS)⁵⁰

- Assesses consciousness level (score 3-15)
- Mild TBI (concussion) = GCS 13-15
- Scores ≤12 require immediate medical referral

⁴⁹ Broglio, S. P., & Guskiewicz, K. M. (2009). Concussion in sports: The sideline assessment. *Sports Health*, 1(5), 361–369. <https://doi.org/10.1177/1941738109343158> PMC+1PubMed+1

⁵⁰ Brain Injury Association of America. (n.d.). *Glasgow Coma Scale*. <https://biausa.org/brain-injury/about-brain-injury/diagnosis/hospital-assessments/glasgow-coma-scale>

Glasgow Coma Scale

Category	Response	Score	Description
Eye Opening (E)	Spontaneous	4	Opens eyes naturally
	To speech	3	Opens eyes when spoken to
	To pain	2	Opens eyes only to pain stimulus
	None	1	No eye opening
Verbal Response (V)	Oriented	5	Normal speech, aware of surroundings
	Confused	4	Coherent but disoriented
	Inappropriate words	3	Random speech, not conversational
	Incomprehensible sounds	2	Moaning, no words
	None	1	No verbal response
Motor Response (M)	Obeys commands	6	Follows movement instructions
	Localizes pain	5	Reaches towards pain stimulus
	Withdraws from pain	4	Moves away from pain
	Abnormal flexion (Decorticate)	3	Arms flex inward
	Abnormal extension (Decerebrate)	2	Arms extend outward
	None	1	No movement response

Scoring

Total Score	Severity	Clinical Significance
13 – 15	Mild	Minor concussion, full recovery likely
9 – 12	Moderate	Significant injury, requires monitoring
3 – 8	Severe	Coma, critical condition

B. Maddocks Questions (Quick Orientation Test)

Used to assess short-term memory and orientation. Examples:

- "Where are we right now?"
- "What half/quarter is it?"
- "Who scored last?"

A wrong answer indicates potential concussion but should be confirmed with further tests.

3. In-Depth Concussion Screening (If Stable and No Red Flags)⁵¹

At this stage, sideline or medical evaluation continues with standardized concussion tests.

A. Symptom Assessment (SCAT/SCOAT6 or Similar Checklist)

- Headache, dizziness, nausea, vision issues, confusion, memory problems, light/sound sensitivity, etc.

B. Cognitive Testing (SCAT/SCOAT6 / SAC - Standardized Assessment of Concussion)

- Immediate memory recall: Repeat a list of words.
- Concentration: Count backward or say months in reverse order.
- Delayed recall: Repeat the words again after 5 minutes.
- **Note SAC is cognitive only** - (Orientation, immediate memory, concentration)
 - Maddocks, word repeat, reverse number sequence

C. Balance & Coordination (BESS – Balance Error Scoring System)⁵²

- Tests postural stability in various stances.
- Increased errors indicate balance impairment, common in concussions.

D. King-Devick Test (If Available)⁵³

- Assesses eye movements and visual tracking speed.
- Delays suggest possible brain injury.

⁵¹ Patricios, J. S., et al.. (2023). Consensus statement on concussion in sport: The 6th International Conference on Concussion in Sport—Amsterdam, October 2022. *British Journal of Sports Medicine*, 57(11), 695-711. <https://doi.org/10.1136/bjsports-2023-106898>

⁵² Bell, D. R., Guskiewicz, K. M., Clark, M. A., & Padua, D. A. (2011). Systematic review of the Balance Error Scoring System. *International Journal of Sports Physical Therapy*, 3(3), 105-112. <https://doi.org/10.1177/1941738111403122>

⁵³ Galetta, K. M., Liu, M., Leong, D. F., Ventura, R. E., Galetta, S. L., & Balcer, L. J. (2015). The King-Devick test of rapid number naming for concussion detection: Meta-analysis and systematic review of the literature. *Concussion*, 1(1), 37-49. <https://doi.org/10.2217/cnc.15.8>

4. Clinical Decision – Remove from Play or Monitor?⁵⁴

- If concussion is suspected → REMOVE FROM PLAY (per international guidelines, "If in doubt, sit them out.")
- Monitor for delayed symptom worsening in the next 24-48 hours.
- Consider emergency evaluation if symptoms deteriorate.

⁵⁴ <https://www.cdc.gov/heads-up/response/index.html>

SCAT6

Immediate On-Field Assessment (Acute Phase)

Purpose:

- To quickly determine if an athlete has sustained a concussion and whether they should be removed from play.

Use:

- Conducted on the sidelines or in the locker room after a head impact.
- Identifies red flag symptoms (e.g., loss of consciousness, seizures, neck pain).
- Uses observable signs and Maddocks Questions to detect disorientation.
- Helps decide if immediate emergency care is needed.

Post-Injury Clinical Evaluation (Within 72 Hours)

Purpose:

- To perform a more detailed neurological and cognitive assessment after a suspected concussion.

Use:

- Administered in a medical clinic, sports medicine facility, or emergency department.
- Evaluates symptom severity, balance, coordination, memory, and cognitive function.
- Includes Glasgow Coma Scale (GCS), cognitive tests, and balance assessments (BESS).
- Helps confirm or rule out a concussion and guides initial management.

Serial Monitoring (Recovery Tracking)

Purpose:

- To monitor symptoms over time and assess recovery progression.

Use:

- Used in follow-up assessments over the first few days post-injury.
- Tracks symptom improvement or worsening.
- Helps decide when an athlete can begin gradual return-to-play protocols.
- Identifies persistent symptoms that may require specialist referral (neurologist, concussion expert).

Return-to-Play and Rehabilitation Decisions

Purpose:

- To determine when it is safe for an athlete to resume sport activities.

Use:

- SCAT/SCOAT6 results help guide graduated return-to-play protocols.
- If symptoms persist, further testing (e.g., ImPACT testing, vestibular exams, neuropsychological assessment) may be needed.

- Ensures athletes do not return to play prematurely, reducing risk of Second Impact Syndrome (SIS).

Baseline Testing (Optional, Pre-Season)

Purpose:

- To establish an individualized baseline for comparison if a concussion occurs later.

Use:

- Conducted before the season begins for athletes in high-risk sports (e.g., football, rugby, hockey).
 - Helps provide a comparison point for post-injury SCAT/SCOAT6 assessments.
-

Additional Information on SCOAT 6 and Child SCAT:

- **SCOAT 6 (Sport Concussion Assessment Tool - 6)** is a streamlined, concise version of SCAT 6 designed for quicker clinical use in adolescent and adult athletes. It focuses on core symptom evaluation, cognitive screening, and balance testing but with fewer items to facilitate rapid assessment in busy or time-limited clinical environments.
 - **Child SCAT** is an adaptation of the SCAT 6 specifically tailored for children aged 5 to 12 years. It includes age-appropriate language, simplified cognitive tasks, and incorporates caregiver/parent input for symptom reporting. This helps account for developmental differences and ensures more reliable assessment in younger athletes who may have difficulty self-reporting.
 - Both **SCOAT 6 and Child SCAT** are used alongside or as alternatives to SCAT 6 depending on the athlete's age, clinical setting, and need for rapid versus detailed assessment.
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Key Differences Between SCAT 6, SCOAT 6, and Child SCAT

Feature / Aspect	SCAT 6	SCOAT 6	Child SCAT (Ages 5–12)
Intended Age Group	Adolescents & Adults	Adolescents & Adults	Children aged 5 to 12
Assessment Length & Detail	Comprehensive, detailed	Shorter, streamlined for clinical efficiency	Simplified for developmental stage
Symptom Reporting	Self-report	Self-report	Child self-report + caregiver/parent input
Cognitive Tasks	Standardized Assessment of Concussion (SAC) with multiple domains	Core cognitive screening, fewer items	Age-appropriate cognitive tests, simplified
Balance Testing	Balance Error Scoring System (BESS)	BESS or similar simplified balance test	Modified balance tasks for younger children
Use of Maddocks Questions	Yes	Yes	Adapted for child understanding
Setting	Sideline, clinical, emergency department	Clinical/rapid assessment setting	Sideline and clinical, child-friendly
Baseline Testing Use	Recommended for pre-season baseline	Optional, quicker baseline assessment	Recommended with parent input
Purpose	Detailed concussion diagnosis and monitoring	Rapid clinical concussion screening	Pediatric concussion identification and monitoring

Total Score Interpretation (Indicative, Not Diagnostic):

Score Range	Interpretation	Notes
Higher Scores	Suggest fewer or no concussion symptoms; better cognitive function and balance.	Typically consistent with mild or no concussion.
Moderate Scores	May indicate presence of concussion symptoms such as cognitive difficulties, headache, or balance problems.	Warrant close monitoring and conservative management.
Lower Scores	Indicate pronounced cognitive and balance impairments, consistent with significant concussion.	Immediate medical evaluation recommended.

Note: SCAT 6, SCOAT 6, and Child SCAT scores should always be interpreted in the context of baseline scores (if available), overall clinical presentation, and other diagnostic findings. Scores are guides to assist clinical decisions and monitor recovery but do not solely define concussion severity.

MSK / Cervical spine assessment

Medicolegal Disclaimer:

This course provides training in musculoskeletal and cervical spine assessment relevant to concussion management and rehabilitation. It is not intended as emergency medicine training and does not cover the management of acute traumatic cervical spine injuries. In cases of suspected serious cervical spine trauma, unstable injury, or compromised airway/circulation, immediate emergency medical intervention and transport (by ambulance) to appropriate medical facilities are imperative. Practitioners must always follow local emergency protocols and refer accordingly.

1. Primary Survey and Scene Assessment⁵⁵

- **Ensure Safety:** The first step is to ensure that the environment is safe and there are no immediate threats to the athlete or responder. If the athlete is unconscious or there's a significant concern for **cervical spine injury**, **do not move the athlete** unless there is an immediate danger (e.g., fire, further injury).
- **Establish ABCs** (Airway, Breathing, Circulation): This is part of the **primary survey** to ensure that the athlete has an open airway and is breathing. If there is a **severe cervical injury**, manual in-line stabilization should be used to prevent further movement of the neck or spine.

2. Immediate Neurological Screening⁵⁶

Before continuing with a detailed cervical spine assessment, a quick **neurological screen** should be conducted to assess for signs of **neurological compromise** that could indicate cervical spine injury. Look for:

- **Consciousness:** Assess whether the athlete is alert and oriented.
- **Cognitive Function:** Check for confusion or memory problems that could indicate a concussion (e.g., questions about the incident).
- **Motor Function:** Observe for any **paralysis** or **weakness** in the limbs. Any signs of motor deficits can point to cervical spine injury.
- **Sensory Function:** Check for **numbness** or **tingling** in the limbs, which can be a sign of nerve compression or injury in the cervical spine.
- **Pain:** Determine if the athlete complains of any **neck pain** or discomfort, which could indicate a cervical injury.
- **Alertness/Orientation:** Always assess whether the athlete is **alert**, **oriented** to time, place, and situation (this is crucial for distinguishing between a concussion and potential cervical injury).

⁵⁵ https://www.rch.org.au/clinicalguide/guideline_index/Head_injury

⁵⁶ Cheever, K., Kawata, K., Tierney, R., & Galgon, A. (2016). Cervical injury assessments for concussion evaluation: A review. *Journal of Athletic Training*, 51(12), 1037–1044. <https://doi.org/10.4085/1062-6050-51.12.15>

3. Manual Cervical Spine Stabilization

In the event of a suspected cervical spine injury or a player who is **unconscious, manual cervical spine stabilization** should be performed immediately by trained personnel. This involves maintaining **in-line spinal stabilization** of the athlete's head and neck to prevent any movement that might cause further injury.

- **In-line Stabilization:** One responder should stabilize the head by holding it in a **neutral position**, avoiding excessive flexion, extension, or rotation. The **cervical spine** should be kept as straight as possible.
- **Do not attempt to remove a helmet** or equipment unless trained to do so, as this could worsen a potential cervical spine injury.

4. Detailed Cervical Spine Assessment:⁵⁷

Once the athlete is stabilized and cervical spine injury is not suspected, the following steps are performed to evaluate the neck and spine further:

4.1 Inspection

- **Look for Deformities:** Inspect for any obvious **contusions, abrasions, or lacerations** around the neck area. Also, look for any **asymmetry** in the neck and head position that could suggest a dislocation or fracture.
- **Posture:** Check for abnormal posturing, such as **head tilt** or a **forward head position**, which could indicate muscle spasm, strain, or neurological involvement.
- **Abnormal Muscle Spasms:** Look for signs of **muscle spasm** that may result from cervical injury.

⁵⁷ Mohai, A., Gifford, J., Herkt, R., Parker, A., Toder, A., Dixon, D., & Kennedy, E. (2022). A scoping review of cervical spine evaluation in standardized clinical concussion evaluation tools. *Physiotherapy Theory and Practice*, 38(12), 1805–1817.
<https://doi.org/10.1016/j.ptsp.2022.07.010>

4.2 Palpation

- **Palpate the Cervical Spine:** Gently palpate the neck, starting from the **C7 vertebra** (the base of the neck) and moving upward to **C1** (the top vertebra). Look for:
 - **Tenderness:** Pain or tenderness along the spinous processes may indicate fracture, ligament injury, or muscle strain.
 - **Deformity:** Check for any **abnormal lumps** or **step-offs** in the vertebrae, which could suggest fractures or dislocations.
 - **Spasm:** Palpate for muscle **tightness** or **spasms**, which can indicate soft tissue injury or strain.
 - **Midline Tenderness:** This is a **critical test**—if the athlete experiences pain in the **midline of the cervical spine**, it suggests the possibility of a **fracture**, and they should not be moved further without proper immobilization and imaging.

4.3 Range of Motion Testing

Range of motion (ROM) testing should only be performed if there is no indication of a **fracture** or **dislocation**, and the athlete does not complain of **acute pain**. It involves testing for the ability to move the head in the following directions:

- **Flexion:** Gently ask the athlete to touch their chin to their chest.
- **Extension:** Ask the athlete to look up at the ceiling.
- **Rotation:** Ask the athlete to turn their head left and right.
- **Lateral Flexion:** Ask the athlete to tilt their ear toward their shoulder on each side.

If the athlete has **limited range of motion**, significant pain, or **increased muscle tension**, this may indicate cervical spine injury.

4.4 Neurological Assessment

A **neurological exam** is essential to assess the integrity of the **nerves** within the cervical spine:

- **Motor Function:** Assess the strength of the upper and lower limbs by asking the athlete to push or pull against resistance. Any weakness could indicate nerve compression.
- **Sensory Function:** Ask the athlete about **numbness** or **tingling** sensations in their hands, arms, or legs, which could suggest nerve involvement or injury in the cervical spine.
- **Reflexes:** Check **deep tendon reflexes** in the upper and lower extremities (e.g., biceps, triceps, patellar, and Achilles reflexes). **Hyperreflexia** or **hyporeflexia** can indicate nerve root irritation or damage.
- **Sensation Testing:** Testing for sensations like **light touch** and **pinprick** is essential. Loss of sensation or abnormal sensations (such as "**pins and needles**") may indicate **nerve root impingement** or spinal cord injury.

4.5 Special Tests (If Applicable and Safe)

There are some special tests that can help identify **cervical spine injury**, but these should only be performed if there is no suspicion of a significant fracture or dislocation, and the athlete is fully conscious and stable:

- **Spurling's Test:** This test involves extending and rotating the neck toward the side of the symptoms to provoke nerve root irritation. It's typically used to diagnose **cervical radiculopathy**, where nerve roots are being compressed.
- **Compression and Distraction Tests:** Compression (pressing down on the head) and distraction (pulling up on the head) tests can reveal pain associated with cervical nerve root issues.

4.6 Decision-Making:

Based on the findings from the assessment, determine whether:

- The athlete is clear of significant cervical spine injury and can proceed with concussion evaluation.
- The athlete is experiencing symptoms that suggest a potential cervical spine injury (**pain, neurological deficits, deformity, muscle spasm, etc.**), and immediate medical attention is required.

If there is **any doubt** regarding the safety of moving the athlete, it is critical to call for **advanced medical help** and avoid any movement of the neck.

5. Transport to Medical Facility

If a **cervical spine injury** is suspected, the athlete should be **immobilized** and transported via **spinal board**, with **manual in-line stabilization** maintained, to a medical facility for further imaging (CT/MRI) and evaluation.

6. Cervico-neurological testing (expanded in practical component) (Cheever et al, 2016)

- Cervical joint reposition error test - **Muscle spindles**
 - Proprioceptive dysfunction
- Smooth-pursuit neck torsion test - **Cervico-ocular reflex**
 - Oculomotor dysfunction
- Head-neck differentiation test - **Cervicocollic reflex**
 - Cervical afferent, excluding vestibular component
- Cervical flexion-rotation test - **Afferent cervical proprioception**
 - C1-2 specific mechanical dysfunction
- Flexor-extensor motor control test - **Vestibulocollic reflex**

Upper Cervical Syndrome⁵⁸

Upper Cervical Syndrome (UCS), also referred to as **Cervical Vertigo**, is a collection of symptoms arising from dysfunction or pathology in the upper cervical spine, specifically affecting the atlantoaxial joint (C1-C2) and the associated structures such as the cervical muscles, ligaments, and neural elements. This condition can result in a range of neurological, musculoskeletal, and autonomic symptoms.

Pathophysiology:

The upper cervical spine (C1-C2) plays a crucial role in maintaining the stability and function of the head and neck. It supports the weight of the head, enables head rotation, and is involved in proprioceptive feedback related to balance. The cervical spine has a dense network of nerves, including the cervical plexus and the vagus nerve, which can influence autonomic functions. Dysfunction in this region can lead to cervical proprioceptive dysfunction, causing dizziness, vertigo, and other symptoms.

Several key mechanisms can contribute to the onset of UCS:

1. **Joint Dysfunction or Subluxation:** Misalignments or dysfunction in the C1-C2 segment can impair normal proprioceptive input, causing inaccurate sensory signals to the brain. This can affect balance and spatial orientation, resulting in dizziness or vertigo.
 2. **Muscle Spasm or Tightness:** Tension in the suboccipital and upper cervical muscles (e.g., the splenius capitis, semispinalis, and suboccipital group) may contribute to referred pain and autonomic dysfunction, particularly influencing the vestibular system.
 3. **Vascular Impairment:** The vertebral artery, which supplies the brainstem and cerebellum, passes through the upper cervical spine. Any compression or irritation of this artery due to cervical dysfunction could lead to vertebral insufficiency, resulting in dizziness, visual disturbances, and even more severe symptoms like syncope.
 4. **Nerve Root Compression:** Compression or irritation of the cervical nerve roots, especially C1-C3, can cause pain, tingling, or numbness, with a potential referral of symptoms to the head, face, or upper extremities.
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⁵⁸ Brandt, T., & Bronstein, A. M. (2001). Cervical vertigo. *Journal of Neurology, Neurosurgery, and Psychiatry*, 71(1), 8-12. <https://doi.org/10.1136/jnnp.71.1.8>

Clinical Presentation:

The symptoms of UCS can be varied, and patients may present with a combination of the following:

- Vertigo or Dizziness: This is often described as a feeling of imbalance or spinning, particularly during head movements or prolonged neck positions.
- Headache: Often located at the base of the skull, referred to as a cervicogenic headache, which may radiate to the forehead, eyes, or the back of the head.
- Neck Pain and Stiffness: Limited range of motion in the cervical spine, with pain or discomfort in the suboccipital region.
- Visual Disturbances: Blurred vision, diplopia, or difficulties with focus, which may be related to the impaired proprioceptive input affecting the vestibular system.
- Tinnitus: Ringing in the ears can occur if there is vascular compression or neural involvement.
- Nausea and Autonomic Symptoms: These may be triggered by head movements or changes in position.

Diagnosis:

Diagnosis of UCS involves a comprehensive clinical evaluation, including:

1. History: A detailed history focusing on trauma (e.g., whiplash injury), long-standing postural issues, and the onset of symptoms.
2. Physical Examination:
 - Range of Motion (ROM): Restricted neck movement, especially in rotation and flexion, may indicate cervical dysfunction.
 - Palpation: Tenderness or muscle tightness in the suboccipital region and upper cervical spine can indicate UCS.
 - Neurological Examination: To rule out other causes of symptoms, including vestibular tests, cranial nerve examination, and assessment of autonomic function.
3. Imaging:
 - X-rays or MRI can be used to identify structural abnormalities, such as subluxations, disc degeneration, or other pathologies.
 - CT or MRI of the cervical spine can assess soft tissue and vascular issues related to the vertebrobasilar system.
4. Provocative Tests:
 - Spurling's Test: Used to identify cervical radiculopathy by applying pressure on the neck while extending and rotating the head.
 - The Dix-Hallpike Maneuver: Although more commonly associated with benign paroxysmal positional vertigo (BPPV), it can also aid in distinguishing whether vertigo is of vestibular or cervical origin.

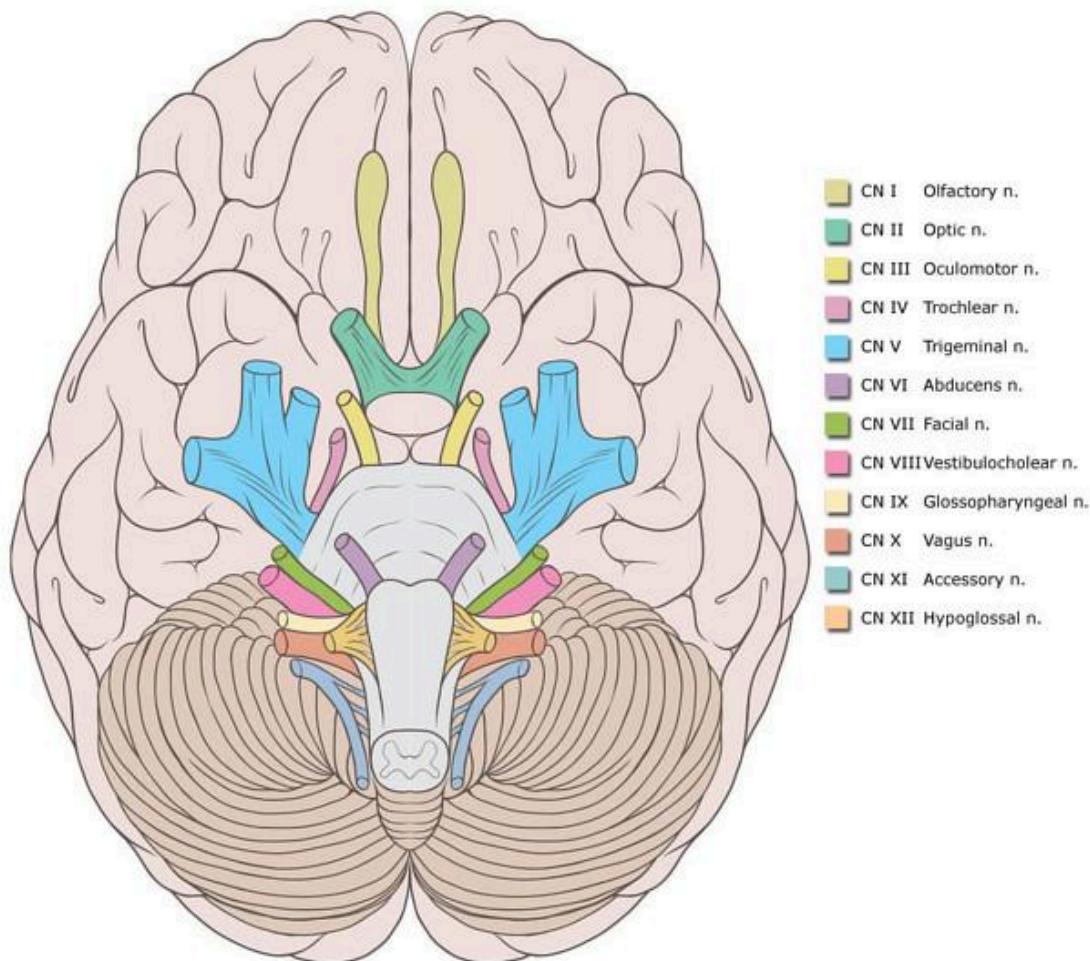
Treatment:

The management of UCS typically involves a multidisciplinary approach:

1. Physical Therapy: Focused on improving posture, range of motion, and strengthening the muscles supporting the cervical spine. Techniques such as manual therapy, cervical traction, and proprioceptive retraining may help to restore normal function.
 2. Medications: Nonsteroidal anti-inflammatory drugs (NSAIDs) or muscle relaxants may help alleviate pain and muscle spasm. In some cases, vestibular suppressants or antihistamines may be prescribed to manage dizziness or vertigo.
 3. Massage Therapy: Targeted soft tissue manipulation can relieve muscle tension and spasm, especially in the suboccipital and upper cervical regions.
 4. Surgical Intervention: In severe cases where structural deformities or compression are present, surgical options may be considered, such as fusion of the upper cervical spine or decompression procedures.
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Cranial nerve (CN) examination⁵⁹

12.6% of head-injured patients (half with mild TBI [mTBI]) had cranial nerve injuries, with CN VII, CN III, and CN II most often injured. In patients with mild injuries, CN I was most often injured followed by CNs VII, III, and IV, while greater than 20% of patients with CN injury had multiple CN injuries.



⁵⁹ Matuszak, J. M., McVige, J., & Leddy, J. (2016). A practical concussion physical examination toolbox: Evidence-based physical examination for concussion. *Sports Health: A Multidisciplinary Approach*, 8(3), 214-222. <https://doi.org/10.1177/1941738116641624>

Cranial Nerve I: Olfactory Nerve (Smell)

- **Function:** Sensory (smell).
 - **Anatomy and Physiology:** The olfactory nerve (CN I) is composed of sensory neurons that are located in the olfactory epithelium of the nasal cavity. These sensory fibers project to the olfactory bulb, and then to the olfactory tract, which transmits signals to the olfactory cortex in the temporal lobe, and also to the limbic system, which explains the strong connection between smell and memory/emotion.
 - **Test:**
 - **Test for Anosmia:** Close the patient's eyes and ask them to occlude one nostril while sniffing a familiar scent like vanilla or coffee. The test is repeated for the other nostril.
 - **Abnormal Findings:** Anosmia (loss of smell) can be caused by nasal obstruction, head trauma, neurodegenerative diseases (e.g., Parkinson's, Alzheimer's), or damage to the olfactory bulb or tract (e.g., tumors or viral infections).
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Cranial Nerve II: Optic Nerve (Vision)

- **Function:** Sensory (vision).
- **Anatomy and Physiology:** The optic nerve (CN II) transmits visual information from the retina, which contains photoreceptors (rods and cones). The axons of retinal ganglion cells form the optic nerve and cross at the optic chiasm. After the chiasm, fibers are referred to as the optic tract and synapse in the lateral geniculate nucleus (LGN) of the thalamus before traveling to the visual cortex (occipital lobe).
- **Test:**
 - **Visual Acuity:** The Snellen chart tests the resolution power of the retina.
 - **Visual Fields:** The confrontation test examines the field of vision of each eye by having the patient look directly at the examiner and move their fingers into the peripheral field.
 - **Pupillary Light Reflex:** Light shone in one eye should cause constriction in both the illuminated eye (direct response) and the opposite eye (consensual response).
 - **Fundoscopy:** Examination of the retina allows assessment of the optic disc for signs of papilledema, which suggests increased intracranial pressure.
 - **Abnormal Findings:** Optic neuropathy, retinal artery or vein occlusion, or diseases like glaucoma can cause vision defects, such as monocular blindness, hemianopia (loss of half the visual field), or scotomas (blind spots). The presence of a relative afferent pupillary defect (RAPD) may indicate optic nerve damage.

Cranial Nerve III: Oculomotor Nerve (Eye Movement, Pupillary Reflex)

- **Function:** Motor (most eye movements), parasympathetic (pupillary constriction and accommodation).
- **Anatomy and Physiology:** The oculomotor nerve (CN III) originates from the midbrain and controls the superior rectus, inferior rectus, medial rectus, and inferior oblique muscles for eye movement. It also innervates the levator palpebrae superioris (eyelid) and contains parasympathetic fibers that control the sphincter pupillae (pupil constriction) and ciliary muscles (accommodation).
- **Test:**
 - **Pupillary Light Reflex:** A normal reaction is constriction of both pupils when light is shone in one eye. Damage to the oculomotor nerve results in a dilated pupil that doesn't constrict in response to light.
 - **Accommodation Reflex:** The pupil constricts and eyes converge when focusing on a near object. Failure to accommodate indicates dysfunction of the parasympathetic fibers.
 - **Eye Movements:** The patient should follow a target in all directions (H-pattern). This tests all the muscles innervated by CN III.
 - **Abnormal Findings:** Ptosis (drooping eyelid) and a fixed, dilated pupil are indicative of oculomotor nerve damage. Paralysis of eye movement can occur in cases of aneurysms, brainstem strokes, or diabetic neuropathy.

Cranial Nerve IV: Trochlear Nerve (Eye Movement)

- **Function:** Motor (innervates the superior oblique muscle for downward and inward eye movement).
 - **Anatomy and Physiology:** The trochlear nerve (CN IV) originates from the trochlear nucleus in the midbrain. The superior oblique muscle controls downward and inward eye movement. It is unique among cranial nerves in that it is the only one to exit the brainstem dorsally and decussate (crosses to the opposite side before innervating the muscle).
 - **Test:**
 - Ask the patient to follow a target in a inferomedial direction. The movement should be smooth and symmetric.
 - **Abnormal Findings:** Damage to CN IV causes difficulty with downward gaze (especially when reading or walking downstairs), resulting in vertical diplopia (double vision).
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Cranial Nerve V: Trigeminal Nerve (Sensory and Motor)

- **Function:** Sensory (sensations from the face) and motor (muscles of mastication).
 - **Anatomy and Physiology:** The trigeminal nerve (CN V) has three branches:
 - **Ophthalmic (V1):** Provides sensation to the forehead, cornea, and scalp.
 - **Maxillary (V2):** Provides sensation to the cheek, upper lip, and nasal cavity.
 - **Mandibular (V3):** Provides sensation to the lower lip, chin, and jaw, and motor function to the muscles of mastication (masseter, temporalis, and pterygoids).
 - The sensory fibers transmit information from the face to the trigeminal nerve ganglion, then to the pons. The motor fibers originate from the pons and control the muscles of mastication.
 - **Test:**
 - **Sensory Function:** Test light touch, sharp/dull sensation, and temperature sensation on all three branches.
 - **Corneal Reflex:** A gentle touch to the cornea should cause bilateral blinking. This tests the sensory input (V1) and motor output (facial nerve).
 - **Motor Function:** Ask the patient to clench their teeth and palpate the masseter and temporalis muscles for asymmetry or weakness.
 - **Abnormal Findings:** Sensory loss in one of the branches suggests a lesion in the corresponding nerve root or peripheral nerve. Loss of corneal reflex points to a lesion in either CN VI or CN VII. Weakness in chewing may indicate dysfunction of the mandibular branch or brainstem involvement.
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Cranial Nerve VI: Abducens Nerve (Eye Movement)

- **Function:** Motor (innervates the lateral rectus muscle for lateral eye movement).
 - **Anatomy and Physiology:** The abducens nerve (CN VI) arises from the abducens nucleus in the pons. It controls the lateral rectus muscle, which abducts the eye.
 - **Test:**
 - Ask the patient to follow a target laterally. The eyes should both move symmetrically.
 - **Abnormal Findings:** Damage to the abducens nerve results in an inability to abduct the affected eye, causing horizontal diplopia. This is often due to intracranial pressure or brainstem lesions.
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Cranial Nerve VII: Facial Nerve (Facial Expression, Taste)

- **Function:** Motor (facial expression) and sensory (taste from the anterior two-thirds of the tongue).
 - **Anatomy and Physiology:** The facial nerve (CN VII) originates from the pons and controls the muscles of facial expression. It also carries parasympathetic fibers to the lacrimal and salivary glands and sensory fibers for taste from the anterior two-thirds of the tongue.
 - It enters the facial canal and divides into several branches, including the temporal, zygomatic, buccal, mandibular, and cervical branches.
 - **Test:**
 - **Motor Function:** Ask the patient to raise both eyebrows, close their eyes tightly, smile, and puff out both cheeks.
 - **Taste:** Taste sensation can be tested on the anterior two-thirds of the tongue (if clinically indicated).
 - **Corneal Reflex:** A test of both the trigeminal and facial nerves.
 - **Abnormal Findings:** Bell's palsy causes unilateral weakness of the entire side of the face. Loss of taste in the anterior two-thirds of the tongue suggests a lesion in the facial nerve or its pathway.
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Cranial Nerve VIII: Vestibulocochlear Nerve (Hearing and Balance)

- **Function:** Sensory (hearing and balance).
- **Anatomy and Physiology:** The vestibulocochlear nerve (CN VIII) has two components:
 - **Cochlear Branch:** Transmits sound information from the cochlea to the brainstem.
 - **Vestibular Branch:** Carries information about balance from the semicircular canals, utricle, and saccule of the inner ear.
- **Test:**
 - **Hearing:** Perform the Rinne and Weber tuning fork tests. Rinne compares air to bone conduction, and Weber assesses for lateralization of sound.
 - **Balance:** Assess for any signs of vertigo or unsteadiness. The Dix-Hallpike test can be used for more specific vestibular examination.
 - **Abnormal Findings:** Sensorineural hearing loss (Rinne negative) and issues with balance (e.g., vertigo) can point to vestibulocochlear nerve dysfunction. Conditions like Meniere's disease, acoustic neuroma, or labyrinthitis can cause these abnormalities.

Cranial Nerve IX: Glossopharyngeal Nerve (Taste, Swallowing, Gag Reflex)

- **Function:** Mixed (sensory and motor); sensory function for taste (posterior third of the tongue), pharyngeal sensation, and parasympathetic innervation to the parotid gland; motor function for swallowing.
- **Anatomy and Physiology:** The glossopharyngeal nerve (CN IX) originates from the medulla oblongata and supplies both sensory and motor fibers. Its sensory fibers carry taste information from the posterior third of the tongue and sensation from the oropharynx, tonsils, and middle ear. The motor component innervates the stylopharyngeus muscle, which assists in swallowing, and the parasympathetic fibers innervate the parotid gland for salivation.
 - The nerve also contributes to the gag reflex via sensory input and participates in the regulation of blood pressure through the carotid sinus reflex.
- **Test:**
 - **Gag Reflex:** Use a tongue depressor to gently stimulate the back of the throat on each side. Both sides of the throat should contract symmetrically. The glossopharyngeal nerve provides the sensory component (afferent limb), and the vagus nerve provides the motor component (efferent limb).
 - **Palate Elevation:** Ask the patient to say "ah" and observe the uvula. It should rise symmetrically. If the glossopharyngeal nerve is affected, the uvula will deviate toward the opposite side of the lesion.
 - **Taste:** In a clinical setting, you can test the taste sensation in the posterior third of the tongue with substances like salty or sour.
 - **Abnormal Findings:** Weak or absent gag reflex, asymmetric palate elevation, or loss of taste sensation in the posterior third of the tongue may indicate glossopharyngeal nerve damage. Conditions such as glossopharyngeal neuralgia, tumors, or brainstem lesions can affect this nerve.

Cranial Nerve X: Vagus Nerve (Speech, Swallowing, Gag Reflex)

- **Function:** Mixed (sensory and motor); motor innervation to the pharynx, larynx, and soft palate, and sensory function to the pharynx and larynx. It also provides parasympathetic innervation to the heart, lungs, and digestive system.
- **Anatomy and Physiology:** The vagus nerve (CN X) originates from the medulla oblongata and travels through the neck, thorax, and abdomen. It supplies motor innervation to the muscles of the soft palate, pharynx, and larynx, which are essential for speech and swallowing. It also provides sensory input to the mucosa of the larynx and pharynx. The parasympathetic fibers regulate the autonomic functions of the heart, lungs, and gastrointestinal tract.
 - The vagus nerve plays a critical role in maintaining homeostasis and regulating the gag reflex.
- **Test:**
 - **Palate Elevation:** Ask the patient to say “ah” and observe for asymmetry of the uvula. The uvula should rise symmetrically in the midline. If the vagus nerve is damaged, the uvula will deviate away from the side of the lesion (towards the unaffected side).
 - **Voice Quality:** Ask the patient to speak. Hoarseness, or a weak voice, may indicate vagal nerve dysfunction, particularly if the recurrent laryngeal nerve is involved.
 - **Gag Reflex:** Similar to testing for the glossopharyngeal nerve, the gag reflex assesses the vagus nerve's motor component.
 - **Abnormal Findings:** Uvula deviation, hoarseness, dysphagia (difficulty swallowing), or absent gag reflex points to vagus nerve dysfunction, which can be caused by brainstem lesions, tumors, or other neurological conditions.

There is emerging evidence to suggest the Vagus nerve is uniquely affected by concussion.

Autonomic Dysfunction⁶⁰: Concussions, especially repetitive ones, may lead to dysregulation of the autonomic nervous system (ANS).

The vagus nerve helps maintain balance between the sympathetic and parasympathetic nervous systems, and damage or dysfunction can lead to postural orthostatic tachycardia syndrome (POTS) or other symptoms of autonomic dysfunction.

Heart Rate Variability (HRV): One of the primary functions of the vagus nerve is regulating HRV. A decrease in HRV is often associated with concussion and may be used as an indicator of autonomic dysfunction. Studies have found that concussion patients, particularly those with persistent post-concussive symptoms (PPCS), show altered HRV patterns. This suggests a possible dysfunction in vagal tone and parasympathetic nervous system regulation (Shaffer & Ginsberg, 2017).

⁶⁰ Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. *Frontiers in Public Health*, 5, 258. <https://doi.org/10.3389/fpubh.2017.00258>

Cognitive and Emotional Impact⁶¹: Vagus nerve dysfunction has been implicated in mood disorders, such as anxiety and depression, which are common in post-concussion syndrome. The vagus nerve has a significant role in mood regulation through its influence on neurotransmitter release (e.g., serotonin) and inflammatory pathways. Impaired vagal function may exacerbate emotional symptoms in concussion patients.

Inflammation⁶²: The vagus nerve also plays an important role in controlling inflammation through the cholinergic anti-inflammatory pathway. After a concussion, neuroinflammation is common, and altered vagal activity might fail to adequately regulate this response. This could contribute to prolonged post-concussion symptoms and slower recovery.

⁶¹ McCrory, P., Meeuwisse, W. H., Dvořák, J., & et al. (2017). Consensus statement on concussion in sport: the 5th international conference on concussion in sport, held in Berlin, October 2016. *British Journal of Sports Medicine*, 51(11), 838-847. <https://doi.org/10.1136/bjsports-2017-097699>

⁶² Borovikova, L. V., Ivanova, S. M., Zhang, M., Yang, H., & Tracey, K. J. (2000). Vagus nerve stimulation attenuates the systemic inflammatory response to endotoxin. *Nature*, 405(6785), 458–462. <https://doi.org/10.1038/35013070>

Cranial Nerve XI: Accessory Nerve (Shoulder Shrug, Head Turn)

- **Function:** Motor; innervates the sternocleidomastoid and trapezius muscles.
- **Anatomy and Physiology:** The accessory nerve (CN XI) has both a cranial and spinal root. The cranial root contributes to the vagus nerve and innervates the muscles of the soft palate, pharynx, and larynx. The spinal root innervates the sternocleidomastoid and trapezius muscles, which are responsible for head rotation and shoulder shrugging, respectively.
 - The spinal portion of the accessory nerve originates from the upper cervical spinal cord (C1-C5) and ascends to join the vagus nerve at the brainstem.
- **Test:**
 - **Shoulder Shrug:** Ask the patient to shrug both shoulders against resistance. Weakness in one side may indicate accessory nerve damage, commonly seen in conditions like trauma or surgery.
 - **Head Turn:** Ask the patient to turn their head to both sides against resistance. Weakness or asymmetry may indicate dysfunction of the sternocleidomastoid muscle, innervated by CN XI.
 - **Abnormal Findings:** Weakness or asymmetry in shoulder shrugging or head turning may indicate accessory nerve damage due to trauma, neck surgery, or spinal cord lesions (particularly in the C1-C5 regions).

BONUS TEST SCH (SHIMIZU)⁶³

The scapulohumeral reflex involves a muscle stretch reflex in the upper extremity.

- The Shimizu variant specifically refers to the method of eliciting this reflex by tapping on the spine of the scapula and the acromion in a caudal (downward) direction.

Clinical Significance:

- A hyperactive scapulohumeral reflex (SHR) is often associated with lesions in the high cervical spinal cord (above the C3 vertebral level).
- It can be a useful indicator of conditions like spinal cord compression in the craniocervical or upper cervical region.
- It is a good screening tool for upper cervical spinal cord lesions.
- **Testing Maneuver:**
 - The reflex is elicited by tapping the tip of the spine of the scapula and the acromion in a downward direction.
 - A positive reflex is indicated by an elevation of the scapula or abduction of the humerus.

⁶³ Shimizu T, Shimada H, Shirakura K. Scapulohumeral reflex (Shimizu). Its clinical significance and testing maneuver. Spine (Phila Pa 1976). 1993 Nov;18(15):2182-90. PMID: 8278829.

- **Involved Muscles:**
 - The primary muscles involved in the SHR are the upper portion of the trapezius, the levator scapulae, and the deltoid.
- **Neurology:**
 - Clinically, the reflex center is presumed to be located between the posterior arch of C1 and the caudal edge of the C3 vertebral body.

Cranial Nerve XII: Hypoglossal Nerve (Tongue Movement)

- **Function:** Motor; innervates the muscles of the tongue.
- **Anatomy and Physiology:** The hypoglossal nerve (CN XII) originates from the hypoglossal nucleus in the medulla oblongata. It controls the intrinsic and extrinsic muscles of the tongue, which are responsible for tongue movements, including protrusion, retraction, and side-to-side motion. The nerve helps with articulation, swallowing, and the voluntary movement of food in the oral cavity.
- **Test:**
 - **Tongue Protrusion:** Ask the patient to stick out their tongue. It should protrude symmetrically. Observe for any atrophy, fasciculations (twitching), or asymmetry.
 - **Tongue Movements:** Ask the patient to move their tongue from side to side. The tongue should move without difficulty or weakness.
 - **Abnormal Findings:** Atrophy, fasciculations, or asymmetry in tongue movements suggest hypoglossal nerve damage, which could be caused by stroke (unilateral), brainstem lesions, or motor neuron diseases like amyotrophic lateral sclerosis (ALS).

 **Cranial Nerve Quiz**

Type: Mixed (Multiple Choice & Short Answer)

Number of Questions: 7

Suggested Time: 10–15 minutes

Questions

1. Which cranial nerve is responsible for innervating the lateral rectus muscle of the eye?

- A. CN II – Optic
 - B. CN III – Oculomotor
 - C. CN IV – Trochlear
 - D. CN VI – Abducens
-

2. A patient presents with loss of pain and temperature sensation on the left side of the body and right-sided facial droop. Which cranial nerve and brainstem region is most likely affected?
(Short Answer)

3. Which of the following is a correct pairing of nerve and function?

- A. CN IX – Motor to sternocleidomastoid
 - B. CN V – Taste from the anterior two-thirds of the tongue
 - C. CN XII – Motor control of tongue movement
 - D. CN I – Vision
-

4. Describe the clinical signs of a right trochlear nerve (CN IV) palsy.

(Short Answer)

5. A patient fails to elevate their right shoulder against resistance. Which nerve is likely impaired, and which muscle is involved?

(Short Answer)

6. The gag reflex involves which two cranial nerves?

- A. CN IX and CN X
 - B. CN V and CN VII
 - C. CN X and CN XI
 - D. CN VII and CN IX
-

7. What is the clinical significance of testing cranial nerve III during a concussion assessment?
(Short Answer)



Vestibulo-ocular motor screening (VOMS)

Smooth Pursuit⁶⁴

Assess oculo-motor control and visual tracking ability.

- **Procedure:** The patient is asked to follow a **target (typically a pen or finger)** that moves smoothly in horizontal and vertical directions. The test should be done in a controlled manner without jerking the target. The examiner watches for **saccadic movements or loss of smooth pursuit** (jerky eye movements).
- **Neurology:** **Parieto-occipital regions** of the brain, with additional input from the **cerebellum**, specifically the **flocculonodular lobe** which is crucial for fine motor control in visual tracking. Impairment of this system can occur after concussion due to disruption of **cerebellar function and cortical processing** of visual stimuli.
- **Symptoms of Dysfunction:** Inability to smoothly track moving objects, leading to **visual blur or eye strain**, often causing **dizziness**.

Saccades⁶⁵

Assess rapid eye movements that shift the focus from one point to another.

- **Procedure:** The patient is instructed to **quickly shift their gaze** between two targets placed in different positions (e.g., left and right). The examiner observes whether the eyes are able to **accurately and quickly** move between the two positions.
- **Neurology:** **Frontal eye fields** and **brainstem**, specifically the **superior colliculus** and **paramedian pontine reticular formation (PPRF)**. Concussion often leads to **impaired saccadic control**, which can manifest as **delayed saccades** or **overshooting** (known as **hypermetria**).
- **Symptoms of Dysfunction:** Inaccuracy, delay, or difficulty in making the eye movements. This can lead to **visual disturbances**, such as **blurry vision** or **difficulty focusing**.

⁶⁴ Rizzo, M., Shapiro, D., & Hovda, D. A. (2014). Smooth pursuit eye movements and their role in concussion assessment. *Journal of Neurotrauma*, 31(15), 1357-1367. <https://doi.org/10.1089/neu.2013.3149>

⁶⁵ Debacker, J., Ventura, R., Galetta, S. L., Balcer, L. J., & Rucker, J. C. (2019). Neuro-ophthalmologic disorders following concussion. In M. L. Shapiro & J. D. Ray (Eds.), *Concussion: Mechanisms, Diagnosis, and Management* (pp. 141-151). Elsevier. <https://doi.org/10.1016/B978-0-444-63954-7.00015-X>

Convergence⁶⁶

Binocular vision and the ability of both eyes to converge or move toward each other to focus on a near object.

- **Procedure:** The patient is asked to focus on a **near target**, such as a pen or finger, and slowly move it toward the bridge of the nose. The examiner notes the point at which the patient experiences **symptoms** or **inability** to maintain focus.
- **Neurology:** **Oculomotor nerve (CN III)** and requires precise coordination between the **medial rectus muscles** of both eyes. **Convergence insufficiency** is common in concussion patients, particularly when there is a disruption to the **brainstem pathways** or **oculomotor function**.
- **Symptoms of Dysfunction:** The patient may report **blurred vision**, **double vision**, or **headaches** as they experience difficulty maintaining focus at near distances.

Vestibular-Ocular Reflex (VOR) Testing⁶⁷

Vestibular system and ocular stabilization during head movement.

- **Procedure:** The patient is asked to **rotate** their head horizontally or vertically while focusing on a fixed target. The examiner watches for signs of **dizziness** or **symptom provocation** such as nausea or imbalance.
- **Neurology:** The **VOR** allows the eyes to maintain a stable visual field during head movement by coordinating the **vestibular system** and **ocular muscles**. The vestibular system, particularly the **semicircular canals**, detects rotational head movements, which are then integrated with visual inputs by the **brainstem**. Disruption of this system in concussion can result in **dizziness** or **vertigo** during head movement.
- **Symptoms of Dysfunction:** Dizziness, nausea, or blurred vision when moving the head while focusing on an object. Impaired VOR indicates a dysfunction in the **vestibular system** or **cerebellum**.

⁶⁶ Pearce, K. L., Sufrinko, A., Lau, B. C., Henry, L., Collins, M. W., & Kontos, A. P. (2015). Near point of convergence after a sport-related concussion: Measurement reliability and relationship to neurocognitive impairment and symptoms. *American Journal of Sports Medicine*, 43(12), 3055-3061. <https://doi.org/10.1177/0363546515606430>

⁶⁷ Broglio, S. P., Cantu, R. C., Gioia, G. A., Guskiewicz, K. M., Kutcher, J., Palm, M., & Valovich McLeod, T. C. (2014). National Athletic Trainers' Association position statement: Management of sport concussion. *Journal of Athletic Training*, 49(2), 245–265. <https://doi.org/10.4085/1062-6050-49.1.07>



Nystagmus⁶⁸

Nystagmus refers to **involuntary, rhythmic eye movements** that can be horizontal, vertical, or rotational (torsional). It typically results from dysfunction in the **vestibular system, ocular motor control, or brainstem structures** that control eye movements. Nystagmus can be a **sign of neurological or vestibular disorder**, including concussion, and is often observed during a neurological or vestibular examination.

Types of Nystagmus

1. **Horizontal Nystagmus:** Movement of the eyes from side to side.
2. **Vertical Nystagmus:** Upward or downward eye movements.
3. **Torsional Nystagmus:** An involuntary, rhythmic eye movement in which the eyes **rotate around the anteroposterior (visual) axis**, causing a **circular or spiral motion of the iris** (clockwise/counterclockwise).
4. **Pendular Nystagmus:** Both eyes move back and forth symmetrically in a pendulum-like manner, often associated with congenital conditions or MS.
5. **Jerk Nystagmus:** Characterized by a slow phase (in one direction) followed by a rapid corrective phase (in the opposite direction).

Beat phase

Up-beat nystagmus may be **constant** or **intermittent** and is typically observed when the patient is gazing in an upward direction.

The **slow phase** is **downward**, and the fast phase is **upward**.

This type of nystagmus can be a key indicator of a **central nervous system** problem, particularly in the **brainstem** or **cerebellum**.

Down-beat nystagmus can occur **spontaneously** or in response to gaze toward extreme **vertical positions** (such as looking up or down).

The slow phase is **upward**, and the fast phase is **downward**.

This type of nystagmus is less common than up-beat nystagmus but can also indicate a central nervous system problem, particularly affecting the cerebellum.

⁶⁸ Tubbs, N., & Bishop, H. (1973). The incidence of post-concussional nystagmus. *Brain Injury*, 7(4), 393-396.
[https://doi.org/10.1016/S0020-1383\(73\)80093-2](https://doi.org/10.1016/S0020-1383(73)80093-2)

Key Points:

- The **fast phase** direction is critical in determining the type and cause of the nystagmus.
- **Peripheral nystagmus** typically shows **fatiguing** characteristics (e.g., reduces with fixation), whereas **central nystagmus** may be persistent and **non-fatiguing**.
- **Vertical nystagmus** and **torsional nystagmus** are more commonly associated with **central lesions in the brainstem or cerebellum**.
- **Horizontal nystagmus**, especially if **fatigued** and is associated with **positional changes**, is commonly seen in **benign paroxysmal positional vertigo (BPPV)**.

Mechanism

Nystagmus often arises from **vestibular dysfunction**, and is linked to the **vestibulo-ocular reflex (VOR)**, which helps stabilize vision during head movements.

The **VOR** relies on input from the **semicircular canals** of the inner ear, which detect head motion and help the eyes compensate for it. When there is damage or dysfunction in the vestibular system or neural pathways (e.g., brainstem or cerebellum), the eye movements become uncoordinated and nystagmus can develop.

Pathophysiology of Nystagmus in Concussion

In a concussion, the vestibular system or neural circuits involved in eye movement control (especially the brainstem or cerebellum) can be disrupted.

Post-concussion nystagmus may present as part of **vestibular dysfunction**, manifesting with increased symptoms such as dizziness, visual disturbances, or imbalance. Concussions may specifically impact the brain areas that integrate sensory input, leading to abnormal eye movements.

Nystagmus in Vestibular and Ocular Motor Testing

Nystagmus is often tested during the **Vestibular/Ocular Motor Screening (VOMS)** or other neurological exams. When nystagmus is present or detected, it may indicate dysfunction in the **vestibular or ocular motor pathways**.

Positive Findings of Nystagmus in Examination

When nystagmus is observed in an examination, the **positive findings** are indicative of underlying neurological or vestibular dysfunction. Below are specific examples of how nystagmus is evaluated in a clinical setting:

1. During Smooth Pursuit Testing

Test Procedure: The patient is asked to follow a target (e.g., a pen or finger) with their eyes in a smooth, slow motion (both horizontally and vertically). The examiner monitors for **jerky** or **abnormal eye movements**.

Positive Findings:

- **Nystagmus during smooth pursuit:** If the patient shows **jerky eye movements** or **involuntary oscillation** when attempting to follow a target, it may indicate dysfunction in the **brainstem** or **cerebellum**. Specifically, **nystagmus** during smooth pursuit testing can suggest problems with **ocular motor control** or **vestibular processing**.

2. During Saccades Testing

Test Procedure: The patient is instructed to quickly shift their gaze between two targets placed about **12-18 inches** apart. The examiner monitors for **rapid, controlled eye movements**.

Positive Findings:

- **Nystagmus during saccades:** If the patient has difficulty making smooth, rapid shifts between targets or shows **jerky eye movements** during the test, it may indicate **dysfunction in the frontal lobe** or **brainstem**. In particular, **nystagmus** seen during saccades testing is indicative of abnormal eye control mechanisms, often related to **cerebellar** or **brainstem** abnormalities.

3. During VOR (Vestibulo-Ocular Reflex) Testing

Test Procedure: The patient is asked to keep their eyes fixed on a target while the examiner moves their head side to side or up and down. This tests the function of the **VOR** in stabilizing vision during head movement.

Positive Findings:

- **Nystagmus with VOR:** The presence of nystagmus during **VOR testing** suggests **vestibular dysfunction**, particularly in the **semicircular canals** or **brainstem**. In concussion, this might manifest as **post-concussion dizziness**, or an inability to stabilize vision during head motion. **Vertical nystagmus** can indicate severe vestibular dysfunction or central nervous system involvement, which warrants further evaluation.

4. During Dix-Hallpike Maneuver (For BPPV Assessment)

Test Procedure: The Dix-Hallpike maneuver is used to evaluate **benign paroxysmal positional vertigo (BPPV)**, a vestibular disorder that causes dizziness. The patient is quickly moved from a sitting to a supine position with the head tilted backward to assess **nystagmus** and vertigo triggered by head movements.

Positive Findings:

- **Positional Nystagmus:** In **BPPV**, nystagmus triggered by specific head movements (e.g., head tilting) is a positive finding. **Horizontal or torsional nystagmus** that is **persistent** (lasting less than 60 seconds) following a positional change is indicative of **BPPV**.
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Types of Nystagmus and Their Clinical Significance

1. **Peripheral Nystagmus** (Vestibular):
 - Often **horizontal** or **rotational**.
 - Typically **fatigues** (lessens) when the head is held in the same position.
 - Usually **improves** with fixation (focus on a target).
 - Associated with **vestibular system dysfunction**, such as **BPPV** or **labyrinthitis**.
2. **Central Nystagmus** (Brainstem/Cerebellar Dysfunction):
 - Can be **vertical**, **torsional**, or **horizontal**.
 - Does not **fatigue** with repeated testing.
 - May **not improve** with fixation.
 - Suggests **brainstem**, **cerebellar**, or **central nervous system** involvement.
 - Can be **persistent** and is more concerning in the context of severe concussion or neurological injury.

Conclusion and Clinical Relevance

Nystagmus is a critical finding in the evaluation of concussion and neurological dysfunction. When nystagmus is observed during tests such as **smooth pursuit**, **saccades**, **VOR**, or **positional maneuvers**, it can indicate **vestibular** or **neurological** dysfunction that may be linked to concussion. Abnormal nystagmus patterns often suggest **central nervous system involvement**, particularly in cases of **brainstem**, **cerebellar**, or **vestibular** dysfunction. Prompt recognition of nystagmus and appropriate management are essential for guiding treatment and recovery strategies following concussion.



Balance Error Scoring System (BESS)⁶⁹

1. Somatosensory System:

- a. The somatosensory system involves proprioception and the ability to detect body position in space via sensory receptors in the skin, muscles, and joints. On the **firm surface**, proprioceptive feedback is more reliable because the surface provides stable sensory input. On the **foam surface**, proprioceptive feedback is compromised, which is why the challenge to balance is greater. This is particularly important after concussions, where proprioceptive input may be disrupted.

PNS Sensory Pathways	Function
Lateral spinothalamic	Pain and temperature
Ventral spinothalamic	Pressure and crude touch
Dorsal column	Vibration, proprioception, two-point discrimination
Spinocerebellar/Cuneocerebellar	Proprioception in joints and muscles
Spinotectal	Tactile, painful, thermal stimuli
Spinoreticular	Integration of stimuli from joints/muscles into the reticular formation
Spino-olivary	Additional information to the cerebellum
Corticospinal	Voluntary, discrete, skilled motor
Reticulospinal	Regulation of voluntary movements, reflexes
Rubrospinal	Promotion of flexor and extensor muscle activity
Vestibulospinal	Inhibition of flexor, promotion of extensor muscle activity
Tectospinal	Postural movements from visual stimuli

⁶⁹ Bell, D. R., Guskiewicz, K. M., [...], & Padua, D. A. (2011). Systematic review of the Balance Error Scoring System. *Sports Health: A Multidisciplinary Approach*, 3(3), 287-295. <https://doi.org/10.1177/1941738111403122>

2. Visual System:

- a. Vision plays a key role in balance by providing feedback about the body's position in space. In the **eyes closed** condition, vision is removed as a sensory input, forcing the other systems (vestibular and somatosensory) to take over. This test challenges the vestibular and somatosensory systems, as visual input is absent, and thus the individual's reliance on vestibular and proprioceptive feedback is emphasized.

3. Vestibular System:

- a. The vestibular system detects changes in head position and helps maintain balance and spatial orientation. The **foam surface** specifically challenges the vestibular system, as the unstable surface forces constant corrections to maintain stability. After concussion, this system can be affected, leading to balance deficits.

Testing Conditions

- **Firm surface:** A stable base that provides reliable sensory feedback.
- **Foam surface:** An unstable base that disrupts proprioception and enhances the challenge to balance.
- **Stance conditions:**
 - **Double-leg stance**
 - **Single-leg stance**
 - **Tandem stance**

Each of these stances is performed on both the firm and foam surfaces, with the eyes closed, and the participant is asked to maintain balance for 20 seconds.

Foot dominance

The single-leg stance in BESS is performed on the non-dominant foot (the foot opposite the dominant one).

- Testing on the non-dominant foot is important because it is usually **less stable and more sensitive** for detecting subtle balance impairments.
 - The tandem stance involves placing the dominant foot behind the non-dominant foot in a heel-to-toe position.
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Mechanism and Purpose

The BESS test is used to quantify balance deficits often resulting from concussions or neurological injuries. By evaluating errors in balance, the test provides insight into how these systems work together and how they may be impaired after an injury. It is particularly useful in concussion assessment, monitoring recovery, and determining when an individual may safely return to physical activity.

Cerebellar component to BESS⁷⁰

The cerebellum integrates the above systems to coordinate precise movements and adjust posture.

1. Processing Proprioceptive Feedback:

- The cerebellum receives input from proprioceptors (sensory receptors in muscles, joints, and skin) that provide information about the body's position in space. It then processes this information to adjust postural control.
- When proprioceptive input is altered (e.g., standing on an unstable surface like foam), the cerebellum must rapidly adjust muscle tone and motor control to maintain balance.

2. Vestibular Processing:

- The cerebellum integrates signals from the vestibular system that detect head position and movement. This helps the body maintain upright posture, especially when visual input is removed (eyes closed) during the BESS test.
- The cerebellum works with the vestibular system to initiate compensatory movements when there are shifts in body position, especially on unstable surfaces.

3. Coordination and Fine-Tuning of Motor Movements:

- The cerebellum coordinates movements by fine-tuning the activity of muscles to produce smooth, controlled postural adjustments. If the cerebellum is impaired (e.g., after a concussion), it can result in poor coordination and difficulty maintaining balance during tasks like those in the BESS test.
- This may lead to errors such as swaying, stepping, or falling during the test, particularly when proprioceptive or vestibular cues are disrupted by foam surfaces or closed eyes.

⁷⁰ Jang, S. H., & Kwon, H. G. (2023). Cerebellar peduncle injuries in patients with mild traumatic brain injury. *Journal of Integrative Neuroscience*, 22(5), 121. <https://doi.org/10.31083/jin2205121>

Cerebellar Dysfunction and Balance Errors in BESS

- Cerebellar dysfunction (such as after a concussion) can lead to deficits in balance, coordination, and postural control. When the cerebellum is impaired, it struggles to process sensory input effectively and make the necessary adjustments to maintain balance, leading to increased errors during the BESS test.
- Typical balance errors in BESS that could be linked to cerebellar dysfunction include:
 - Increased sway or instability: The inability to keep a steady posture.
 - Frequent loss of balance: The person may need to step or shift their weight often to prevent falling.
 - Failure to hold the stance: Inability to maintain the required positions due to poor motor coordination.

Key differences in presentation during the BESS test:

Presentation Aspect	Cerebellar Involvement	Non-Cerebellar Involvement
Balance and Coordination Issues	Significant balance difficulties; uncoordinated movements; trouble adjusting posture.	Balance issues may occur but coordination is typically less affected.
Postural Control	Exaggerated sway; frequent errors in stance; poor compensation for shifts.	Some errors or sway may occur, but with better overall control.
Recovery from Errors	Slower recovery due to impaired coordination and error correction.	Quicker recovery and better ability to regain stability.
Sensation of Dizziness or Disorientation	More pronounced dizziness or disorientation due to impaired sensory processing.	Dizziness may still occur but typically less severe and due to other factors (e.g., vestibular).

Module 2 Summary: Concussion Diagnosis & Initial Assessment

Learning Objectives Achieved ✓

Diagnostic Criteria Mastery: Current assessment approaches across age groups

Clinical Presentation Recognition: Identifying typical concussion symptoms and signs

Assessment Tool Proficiency: SCAT6/SCOAT6/Child SCAT6 structure and application

Critical Evaluation Skills: Understanding limitations of current diagnostic tools

Red Flag Recognition: Emergency referral criteria and differential diagnoses

Acute On-Field Assessment Protocol

Primary Assessment Framework (Life-Saving First)

Priority	Assessment	Action Required
1. ABCs	Airway, Breathing, Circulation	Basic life support if needed
2. Spinal Precautions	C-spine immobilization	Manual stabilization if suspected
3. Red Flags	Emergency indicators	Immediate EMS transfer

Critical Red Flags (Emergency Referral)

- Loss of consciousness >1 minute
- Seizures or convulsions
- Worsening headache, vomiting, confusion
- Limb weakness/numbness
- Unequal/fixed pupils
- Midline cervical tenderness

Rapid Neurological Screening (If Stable)

Maddocks Questions: "Where are we?", "What period is it?", "Who scored last?" **Symptom Checklist:** Headache, dizziness, nausea, vision issues, confusion **Balance Assessment:** BESS test for postural stability **Cognitive Screen:** Memory recall, concentration tasks, King-Devick test

Cervical Spine & MSK Integration

Cervical Assessment Decision Tree

Conscious + No neck pain + Full motor function + Oriented → Proceed with concussion assessment

Tingling in extremities → Cervical precautions

Unconscious or lying awkwardly → Manual stabilization, EMS

Midline tenderness → Immobilization required

Upper Cervical Syndrome (UCS) Recognition

C1-C2 Dysfunction Signs:

- Vertigo and dizziness
- Cervicogenic headache
- Visual changes and tinnitus
- Autonomic symptoms

Assessment Approach:

- History of mechanism
 - ROM limitations
 - Palpation findings
 - Vestibular vs cervical differentiation
-

Glasgow Coma Scale (GCS) Application

Three-Component Assessment Severity Classification

Eye Opening - /4

Verbal Response - /5

Motor response - /6

- **GCS 13-15:** Mild TBI (concussion)
 - **GCS 9-12:** Moderate TBI
 - **GCS 3-8:** Severe TBI
-

SCAT6 Tool Family & Applications

Assessment Tool Selection

Tool	Target Population	Setting	Key Features
SCAT6	Adolescents/Adults	Sideline, clinic, ED	Full comprehensive assessment
SCOAT6	Adolescents/Adults	Time-limited clinic	Streamlined for efficiency
Child SCAT6	Ages 5-12	Pediatric settings	Age-appropriate, caregiver input

Clinical Application Phases

- **Immediate (On-Field):** Red flags, observable signs, Maddocks questions
- **Post-Injury (0-72hrs):** Comprehensive SCAT6 evaluation
- **Serial Monitoring:** Symptom tracking and recovery progression
- **Return-to-Play:** Combined assessment with clinical judgment

Score Interpretation Framework

- **High Scores:** Few symptoms, good cognition/balance → Mild or no concussion
- **Moderate Scores:** Some symptoms, mild impairments → Monitor closely
- **Low Scores:** Severe symptoms, clear deficits → Significant concussion, refer

Cranial Nerve Assessment in Concussion

High-Yield Cranial Nerves Post-TBI

CN	Name	Function	Test	Concussion Relevance
I	Olfactory	Smell	Familiar scents	Anosmia common post-TBI
II	Optic	Vision	Acuity, fields, pupils	Visual disturbances
III	Oculomotor	Eye movement, pupils	H-pattern, pupil response	Diplopia, ptosis
IV	Trochlear	Downward/inward gaze	Inferomedial tracking	Vertical diplopia
VI	Abducens	Lateral gaze	Lateral eye movement	Horizontal diplopia
VII	Facial	Facial expression	Symmetry, eye closure	Bell's palsy-like symptoms
VIII	Vestibulocochlear	Hearing, balance	Rinne, Weber, Dix-Hallpike	Dizziness, hearing loss

Clinical Significance

- **12.6% prevalence** of CN injuries in head trauma
 - **Multiple CN involvement** in >20% cases
 - **CNs I, II, III, IV, VII** most commonly affected
-

Vestibulo-Ocular Motor Screening (VOMS)

Five-Domain Assessment

Test	Purpose	Neurological Basis	Dysfunction Signs
Smooth Pursuit	Eye tracking ability	Parieto-occipital cortex, cerebellum	Jerky movements, poor tracking
Saccades	Rapid eye movements	Frontal eye fields, superior colliculus	Over/undershooting, delays
Convergence	Binocular near focus	CN III, medial rectus coordination	Early diplopia, eye drift
VOR	Gaze stability with head movement	Semicircular canals, brainstem	Dizziness, visual blur
VMS	Visual motion sensitivity	Cortex, vestibular nuclei, cerebellum	Motion-induced symptoms

Clinical Application Indications

- **Visual symptoms:** Blurred vision, diplopia, reading difficulties
- **Vestibular complaints:** Dizziness, motion sensitivity, spatial orientation issues
- **Timing:** Most effective >72 hours post-injury for subacute assessment

Nystagmus Recognition & Significance

Beat Phase Direction: Named for fast corrective movement

Peripheral vs Central:

- Peripheral: Horizontal, fatigues with fixation, improves with gaze fixation
- Central: Vertical/torsional, non-fatiguing, constant

Clinical Importance: Vertical or direction-changing nystagmus suggests central pathology requiring urgent evaluation

Balance Error Scoring System (BESS)

Sensory System Integration

Sensory Input	Function	BESS Challenge
Somatosensory	Proprioception from joints/muscles	Foam surface disrupts input
Visual	Spatial orientation feedback	Eyes closed removes visual cues
Vestibular	Head position/movement detection	Combined challenges test compensation

Testing Conditions & Interpretation

Firm Surface: Baseline performance with full sensory input

Foam Surface: Challenges proprioception and vestibular systems

Eyes Closed: Tests remaining systems' compensation ability

Cerebellar vs Non-Cerebellar Patterns

Cerebellar Involvement:

- Significant balance difficulties
- Exaggerated sway, frequent errors
- Slower error recovery
- More pronounced disorientation

Non-Cerebellar:

- Milder balance issues
- Better overall postural control
- Quicker stability recovery
- Less severe dizziness

Clinical Decision-Making Framework

Differential Diagnosis Considerations

- **Migraine:** Headache with photophobia, phonophobia
- **Whiplash:** Neck pain, cervicogenic symptoms
- **Vestibular dysfunction:** Isolated dizziness, hearing changes
- **Psychological factors:** Anxiety, depression amplifying symptoms

Return-to-Play Progression

1. **Symptom resolution** at rest
2. **Graduated exertion** without symptom return
3. **Sport-specific activities** clearance
4. **Full medical clearance** before contact

Emergency Referral Triggers

- **Progressive deterioration**
 - **Persistent red flags**
 - **Severe cognitive impairment**
 - **Neurological focal signs**
-

APPENDIX: Clinical Reference Tools

Quick Red Flag Checklist

- LOC >1 minute
- Seizures
- Worsening headache/vomiting
- Limb weakness/numbness
- Pupil abnormalities
- Midline cervical tenderness
- Progressive confusion

Maddocks Questions (Sport-Specific)

- "What venue are we at today?"
- "Which half/period is it now?"
- "Who scored last in this match?"
- "What team did you play last week/game?"
- "Did your team win the last game?"

VOMS Screening Protocol

Pre-test: Record baseline symptoms (0-10 scale)
Smooth Pursuit: Horizontal/vertical tracking for 30 seconds
Saccades: Rapid shifts between targets × 10 repetitions
Convergence: Slow approach to nose, note break point
VOR: Head rotation while fixating target × 30 seconds
VMS: Moving visual stimulus exposure × 30 seconds
Post-test: Re-rate symptoms, note ≥2 point increase

BESS Testing Positions

Double leg stance: Feet together, hands on hips
Single leg stance: Non-dominant foot, opposite leg flexed
Tandem stance: Heel-to-toe, non-dominant foot behind *Each position: 20 seconds × 2 conditions (firm/foam surface)*

GCS Quick Reference

Eyes: Spontaneous(4), Speech(3), Pain(2), None(1)
Verbal: Oriented(5), Confused(4), Words(3), Sounds(2), None(1)
Motor: Commands(6), Localizes(5), Withdraws(4), Flexion(3), Extension(2), None(1)

Cranial Nerve Red Flags

- **Anosmia** (CN I): Common but often overlooked
- **Visual field defects** (CN II): May indicate serious pathology
- **Pupil inequality** (CN III): Requires urgent evaluation
- **Vertical diplopia** (CN IV): Suggests brainstem involvement
- **Facial asymmetry** (CN VII): Document baseline vs new
- **Hearing loss** (CN VIII): Often accompanies vestibular symptoms

Assessment Tool Selection Guide

SCAT6: Full assessment when time permits, baseline comparison available
SCOAT6: Time-limited clinical settings, efficient screening
Child SCAT6: Pediatric populations, requires caregiver input
VOMS: Visual-vestibular symptoms present, >72 hours post-injury
BESS: Balance complaints, available foam surface, quick screening

Clinical Pearl: "When in doubt, sit them out" remains the gold standard. No assessment tool replaces clinical judgment, and conservative management prevents Second Impact Syndrome. Serial monitoring often reveals patterns that single assessments miss.



Module 3: Practical Assessment & Acute Concussion Management

From Sideline to Clinic: Assessing and Managing Acute Concussion

Hone your hands-on skills and develop effective return-to-play protocols with real-time assessment tools.

- **Learning Objectives:**

- Demonstrate physical testing protocols and explain their clinical relevance.
- Apply rest and activity modification strategies in early concussion rehabilitation.
- Evidence-based testing protocols in current use.
- Testing order and value of information.
- Relevance of testing components to presentation and injury grading.

Activities & Clinical Skill Application

This module includes practical assessments, clinical reasoning checkpoints, and structured simulations designed to build proficiency in acute concussion screening and physical examination techniques. Learners will demonstrate hands-on skills such as cranial nerve testing, VOMS, BESS, and cervical screening, while applying evidence-based decision-making. Activities are observed and trainer-marked in real time to assess clinical competence and safe practice.

Learning outcomes

1. Demonstrate proficiency in administering SCAT/SCOAT6 and Child SCAT assessments.
2. Perform clinical vestibulo-ocular screening (e.g., VOMS, saccades, convergence tests).
3. Conduct and interpret results of basic balance and gait assessments (e.g., tandem walk, BESS).
4. Assess cervical spine contributions to symptom presentation (e.g., joint position error testing).
5. Implement early symptom-guided activity and rest strategies.
6. Apply safe techniques for initiating symptom-tolerated aerobic exercise.

Cervical Dizziness & Headache Assessment Flowchart

MSK Screen



Active / Passive ROM

Spurling's Test



Positive → Cervical radiculopathy
Negative → Continue

Cervical Flexion-Rotation Test



↓ Decreased rotation + headache → C1–C2 dysfunction / Cervicogenic headache
Normal rotation → Continue

Joint Position Error (JPE) Test



↓ Overshoot / dizziness / poor accuracy → Cervical proprioceptive dysfunction
Accurate repositioning → Continue

Smooth Pursuit Neck Torsion Test



↓ Jerky only during trunk rotation → Cervical-origin (COR dysfunction)
↓ Jerky in all positions → Likely vestibular or central issue

Head-Neck Differentiation Test



↓ Symptoms with trunk rotation only → Cervical cause (COR/proprioceptive)
↓ Symptoms with head rotation only → Vestibular cause (VOR-related)

Vestibulo-Ocular Reflex (VOR) Test



Symptoms reproduced (blur/dizziness) → Confirms vestibular dysfunction
No symptoms → Reassess for central signs or fatigue factors

Musculoskeletal (MSK) Testing Protocol in Concussion

Cervical Spine Assessment

A concussion can involve concomitant cervical spine dysfunction, including cervicogenic headaches, whiplash-associated disorders (WAD), and proprioceptive impairments.

Red Flags (Emergency Referral Criteria)

- Cervical spine fractures or instability
- Significant midline tenderness over the vertebrae
- Neurological deficits (weakness, sensory loss, or reflex abnormalities)
- Severe range of motion (ROM) limitations

Symptoms indicative of vertebral artery dissection (dizziness, nystagmus, difficulty swallowing)

Clinical Examination

Active and Passive ROM

Assess cervical flexion, extension, lateral flexion, and rotation. Any restriction or pain indicates dysfunction.

Finding	Interpretation
Pain or restriction in cervical flexion	Possible cervical spine dysfunction or muscle tightness
Pain or restriction in cervical extension	Likely dysfunction in the upper cervical spine or facet joints
Pain or restriction in lateral flexion	May indicate muscle strain or facet joint dysfunction
Pain or restriction in rotation	Possible cervical radiculopathy or facet joint dysfunction
No pain, but limited ROM in any direction	May suggest mechanical restrictions, such as joint stiffness or muscular tightness
Pain with motion in all directions	Could indicate generalized cervical spine dysfunction or inflammatory conditions

Spurling's Test

Compression applied in cervical extension and lateral flexion to reproduce radicular pain (indicating cervical radiculopathy).

Testing procedure

Position the Patient - Seated, neutral neck position (head facing forward, shoulders relaxed).

Inform the Patient - Explain the test and potential discomfort. Warn about sharp, shooting pain.

Cervical Extension - Guide the head into extension (tilting back), keeping the neck neutral.

Lateral Flexion - Tilt the head to one side (suspected symptomatic side), keeping it controlled.

Apply Axial Compression - Apply gentle, downward axial compression on the head, aligned with the spine.

Observe for Symptoms - Ask the patient about pain, tingling, or radiating pain down the arm.

Repeat on Opposite Side - Perform the same steps on the opposite side for comparison.

Interpret Results

- Positive: Radiating pain indicates cervical radiculopathy.
- Negative: No symptoms or only localized neck pain suggests no radiculopathy.

End the Test - Release compression and return the head to neutral position. Inform the patient of results.

Finding	Interpretation
Reproduction of radicular pain (sharp, shooting pain radiating down the arm)	Suggestive of cervical radiculopathy due to nerve root compression (e.g., herniated disc or degenerative changes)
No pain or symptoms reproduced	Negative test, unlikely to have cervical radiculopathy, though further testing may be needed
Pain localized to the neck without radiation down the arm	Possible cervical muscle strain or joint dysfunction, but not indicative of radiculopathy
Pain worsening with further compression	Indicates nerve root compression or exacerbation of symptoms, supporting a diagnosis of cervical radiculopathy
Pain relieved with release of compression	Nerve root irritation or mechanical compression, with pain resolving upon decompression, suggesting radiculopathy

Cervical flexion-rotation test

Afferent cervical proprioception - C1-2 specific mechanical dysfunction

Testing procedure

Position the Patient – Supine on the treatment table, head supported, eyes closed or relaxed.

Inform the Patient – Explain the purpose of the test and advise them to report any dizziness, pain, or discomfort.

Full Cervical Flexion – Gently guide the patient's head into full cervical flexion by bringing the chin toward the chest. This locks out lower cervical motion.

Rotation in Flexion – While maintaining full flexion, rotate the head passively to the left and then to the right, assessing the available range.

Observe for Limitation – Estimate end range on each side. Normal is around 45°. Restricted motion under 32°, or a side-to-side difference over 10°, is considered abnormal.

Note Symptom Reproduction – Ask the patient if symptoms like dizziness or pain are provoked during rotation.

Interpret Results

- Positive: Reduced rotation and/or symptom reproduction suggests C1–C2 dysfunction or cervicogenic involvement.
- Negative: Full range with no symptoms suggests normal upper cervical mobility.

End the Test – Gently bring the head back to neutral and inform the patient of the findings.

Finding	Movement Range	Interpretation
Normal rotation, no symptoms	~45° each side	Normal upper cervical mobility and proprioception
Reduced rotation on one or both sides	<32°, or >10° asymmetry	Likely C1–C2 segmental dysfunction
Symptom reproduction during rotation	Any	Suggests cervicogenic origin of headache or dizziness
Reduced range and symptom reproduction	<32° with pain/dizziness	Indicates both mobility restriction and abnormal cervical afferent input
Normal range but dizziness or disorientation	~45°	Possible cervical proprioceptive mismatch contributing to symptoms

Joint Position Error (JPE) Test

Used to assess cervical proprioception and differentiate cervical vs vestibular vs central causes of dizziness or headache.

Testing procedure

Position the Patient – Seated, with the head and neck in a neutral position, shoulders relaxed.

Inform the Patient – Explain the purpose of the test and the possible discomfort that may arise.

Eyes Closed – Ask the patient to close their eyes to eliminate visual input.

Starting Position – Place the head in a neutral position, ensuring alignment.

Rotate Head to One Side – Gently rotate the head to one side (about 45°), while keeping the neck aligned.

Reposition the Head – Passively return the head to neutral and ask the patient to actively return it to the same position.

Observe for Errors – Note any discrepancies or inability to return to the exact starting position.

Repeat on Opposite Side – Perform the same steps on the opposite side for comparison.

Interpret Results

- **Decreased rotation + headache:** Likely cervicogenic headache (C1-C2 origin).
- **Decreased rotation + dizziness:** Cervical proprioceptive dysfunction (afferent issue).
- **Normal rotation + dizziness:** More likely vestibular or central dysfunction.

End the Test – Return the head to a neutral position and inform the patient of the results.

Finding	Interpretation
Decreased rotation + headache	Likely cervicogenic headache (C1-C2 origin)
Decreased rotation + dizziness	Cervical proprioceptive dysfunction (afferent issue)
Normal rotation + dizziness	More likely vestibular or central dysfunction

Smooth-pursuit neck torsion test

Cervico-ocular reflex / Oculomotor dysfunction

Testing procedure

Position the Patient – Seated upright, in a neutral head position with eyes at eye level.

Inform the Patient – Explain the test, emphasizing the need to follow a moving target with only their eyes.

Establish Baseline – Hold a pen or small target at arm's length. Ask the patient to track the target smoothly left to right, and right to left, keeping the head still.

Observe Baseline Pursuit – Watch for smoothness of eye movement, looking for any saccades (jerky corrections).

Neck Torsion Setup – Gently rotate the patient's body (shoulders and trunk) approximately 45° to one side while the head remains facing forward (neutral).

Repeat Smooth Pursuit – Repeat the horizontal tracking while in the torsioned body position.

Observe Pursuit in Torsion – Look for differences in smoothness, accuracy, or symptom reproduction (e.g., dizziness, discomfort).

Repeat on Opposite Side – Perform the same procedure with the body rotated to the opposite side for comparison.

Interpret Results

- Positive: Jerky pursuit in torsion only suggests abnormal cervical afferent input (COR dysfunction).
- Positive: Jerky pursuit in both neutral and torsion suggests central oculomotor or vestibular dysfunction.
- Negative: Smooth pursuit in all conditions indicates normal COR and oculomotor function.

End the Test – Return the patient to a neutral position and provide feedback on findings.

Condition	Eye Movement Quality	Interpretation
Head neutral	Smooth pursuit normal	Baseline normal
Neck torsion causes symptoms or jerky pursuit	Decreased accuracy or saccadic intrusions	Suggests abnormal cervical afferent input affecting COR
Jerky pursuit in both neutral and torsion	Poor tracking in all positions	Suggests central (vestibular or oculomotor) dysfunction

Head-neck differentiation test

Cervicocollic reflex (COR) - Cervical afferent, excluding vestibular component

Testing procedure

Position the Patient – Seated/standing comfortably in a chair with the head and neck in a neutral position.

Inform the Patient – Explain the test, noting you'll be rotating either the body or head while they maintain focus on a target.

Condition 1: Head Fixed, Body Rotated – Ask the patient to fix their gaze on a stable target. Gently rotate their torso (shoulders and trunk) left and right while keeping their head still, facing forward.

Observe Symptoms – Ask if they experience dizziness, disorientation, or other symptoms during movement.

Condition 2: Body Fixed, Head Rotated – Now keep the body stationary. Ask the patient to rotate their head left and right while keeping their gaze on the same target.

Observe Symptoms Again – Note if symptoms occur again, and whether they differ from the first condition.

Interpret Results

- Positive in Condition 1 only: Suggests cervical origin (CCR dysfunction).
- Positive in Condition 2 only: Suggests vestibular or central origin.
- Positive in both: Suggests mixed cervico-vestibular involvement.

End the Test – Return the patient to neutral, document findings, and explain outcomes to the patient.

Condition	Symptoms Provoked?	Interpretation
Head Fixed, Body Rotated	Yes	Cervical (CCR-related)
Body Fixed, Head Rotated	Yes (but not in condition 1)	Vestibular or central source
Both Conditions	Yes	Possible mixed (cervico-vestibular) origin

Flexor-extensor motor control test - Vestibulocollic reflex (VOR)

Testing procedure

Position the Patient – Seated or standing with a neutral head and neck position. Shoulders should be relaxed, and the spine upright.

Inform the Patient – Explain the test and emphasize the importance of slow, controlled movement. Encourage them to report any dizziness or discomfort.

Begin Cervical Flexion – Instruct the patient to slowly tuck the chin and flex the neck forward in a controlled motion, avoiding quick or jerky movements.

Observe Alignment – Watch for head and neck alignment throughout the motion. Look for stability and smoothness.

Return to Neutral and Extend – Guide the patient to return to neutral and slowly extend the neck. Again, assess for control and coordination.

Identify Compensations – Note any excessive chin tucking, forward head movement, shaking, or poor motor control.

Monitor Symptoms – Ask if the patient experiences dizziness, discomfort, or disorientation during the test.

Interpret Results

Positive: Jerky motion, poor alignment, symptom reproduction, or compensatory patterns suggest deep neck flexor/extensor dysfunction or VCR impairment.

Negative: Smooth, stable motion without symptoms indicates normal vestibulocollic reflex and motor control.

End the Test – Assist the patient back to a comfortable neutral posture and provide feedback.

Finding	Motor Control Quality	Interpretation
Smooth, controlled flexion/extension	Stable head and neck alignment	Normal deep cervical flexor/extensor control; intact vestibulocollic reflex (VCR)
Jerky, uncoordinated movement	Poor control, visible shaking or compensation	Impaired deep neck muscle function; potential VCR dysfunction or sensorimotor deficit
Chin tucks excessively or pokes forward	Altered movement pattern	Over-recruitment of superficial muscles (e.g., SCM); poor deep cervical control
Unable to maintain neutral during movement	Loss of head/neck positioning	Likely impairment in proprioceptive integration and reflex-driven stabilization
Symptoms (dizziness, discomfort) during test	Discomfort, disorientation	Possible cervico-vestibular mismatch or cervical afferent hypersensitivity

 **Checkpoint 1: Cervical Spine Screening & ROM**

1. What symptom indicates vertebral artery involvement?

2. What is a positive finding in Spurling's test?

3. When should you stop cervical ROM testing immediately?

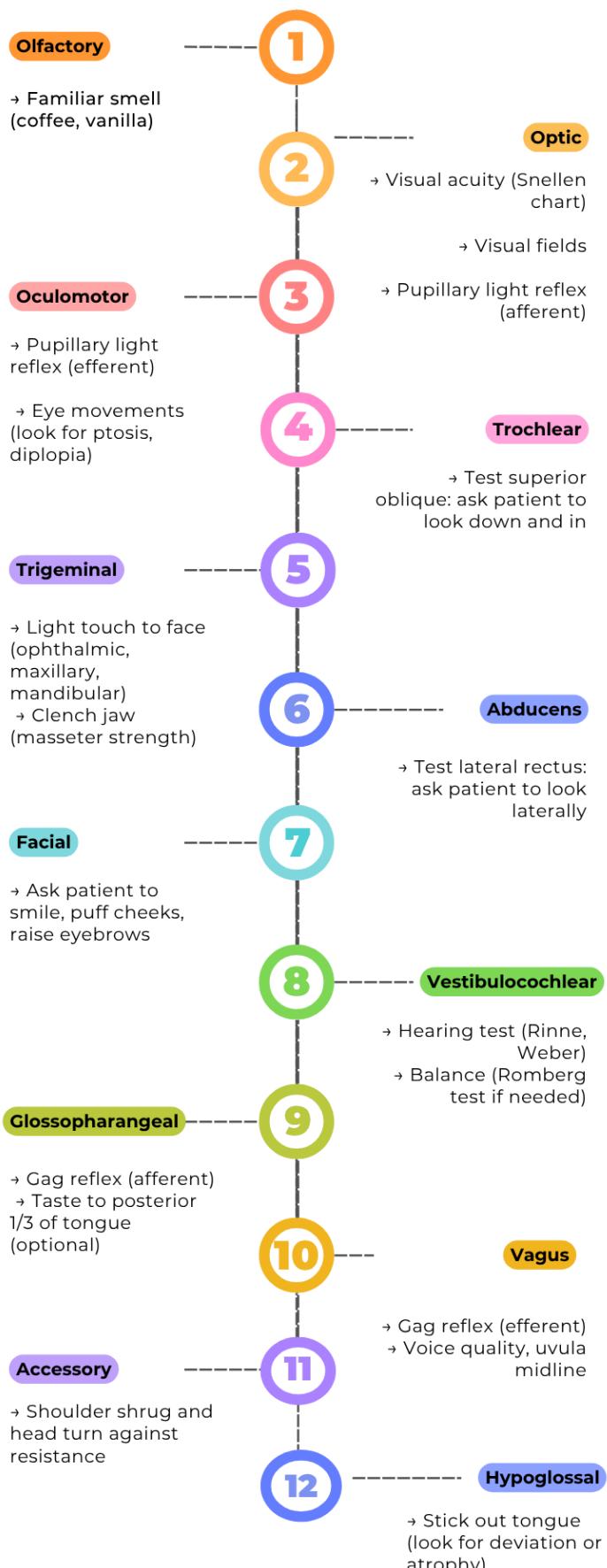
 **Checkpoint 2: Cervicogenic vs Vestibular Differentiation**

1. What does it suggest if a patient has dizziness with decreased cervical rotation?

2. Which test isolates C1–C2 joint mobility and proprioception?

3. What finding in the smooth-pursuit neck torsion test indicates central dysfunction?

CRANIAL NERVE SCREEN



Cranial Nerve Screen⁷¹

Verbal examination

Question	Nerve examined
Have you had any recent change in smell, hearing, taste or vision?	I, II, III, IV, VI, VII (ant 2/3), VIII (cochlear), IX
Have you had any issues chewing, swallowing or speaking?	V (motor), X, XII
Have you had any problems with balance or shoulder, neck movement?	VIII (vestibular), XI

I: Olfactory Nerve (Smell)

- Function: Sensory (smell).
- Test:
 - Ask the patient to close their eyes and occlude one nostril.
 - Place a familiar scent (e.g., coffee, vanilla) near the open nostril.
 - Ask the patient to sniff and identify the smell.
 - Repeat the test for the other nostril.
 - Abnormal Findings: Anosmia (loss of smell), hyposmia (reduced smell), or a distorted sense of smell can indicate damage to the olfactory nerve or other neurological conditions.

II: Optic Nerve (Vision)

- Function: Sensory (vision).
- Test:
 - Visual Acuity: Test each eye separately using a Snellen chart or similar tool.
 - Visual Fields: Perform a confrontation test. Have the patient cover one eye and describe movements of your fingers in the periphery of their visual field. Repeat for the other eye.
 - Pupillary Light Reflex: Shine a light into each eye and observe for a direct and consensual response (constriction in the eye the light is shined into, and the opposite eye).
 - Fundoscopy: Examine the retina for any abnormalities, such as papilledema (swelling of the optic disc), which can indicate increased intracranial pressure.
 - Abnormal Findings: Vision loss, field cuts, or abnormal pupillary reactions suggest optic nerve damage, or neurological conditions like glaucoma, optic neuritis, or stroke.

⁷¹ Reese, V., Das, J. M., & Al Khalili, Y. (2023). Cranial nerve testing. In *StatPearls*. StatPearls Publishing. Retrieved from <https://www.ncbi.nlm.nih.gov/books/NBK585066/>

III: Oculomotor Nerve (Eye Movement, Pupillary Reflex)

- Function: Motor (most eye movements, pupil constriction).
 - Test:
 - Pupillary Light Reflex: Shine light into each eye and observe for constriction (both direct and consensual responses).
 - Accommodation Reflex: Ask the patient to focus on a near object, and then on a distant object. Normal response is constriction and convergence of the eyes.
 - Eye Movements: Ask the patient to follow your finger in H-pattern to assess for smooth eye movement in all directions.
 - Abnormal Findings: Ptosis (drooping eyelid), pupil dilation, or failure of accommodation or eye movements can indicate oculomotor nerve damage, which might be caused by conditions like brainstem lesions, aneurysms, or diabetes.
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IV: Trochlear Nerve (Eye Movement)

- Function: Motor (controls superior oblique muscle, which moves the eye downward and laterally).
 - Test:
 - Ask the patient to follow your finger as you move it downward and laterally.
 - Observe for any eye movement abnormalities, such as vertical diplopia (double vision).
 - Abnormal Findings: Difficulty with downward and lateral gaze may suggest trochlear nerve dysfunction, often seen in conditions like head trauma or brainstem lesions.
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V: Trigeminal Nerve (Sensory and Motor)

- Function: Sensory (sensory information from the face) and motor (muscles of mastication).
 - Test:
 - Sensory Function: Test light touch, pain, and temperature sensation on all three branches of the trigeminal nerve (ophthalmic, maxillary, and mandibular). Have the patient close their eyes and use a cotton wisp or pinprick to test sensation on each side of the face.
 - Corneal Reflex: Lightly touch the cornea with a cotton wisp and observe for bilateral blinking. This tests the sensory function of the ophthalmic branch and the motor function of the facial nerve.
 - Motor Function: Ask the patient to clench their teeth and palpate the masseter and temporalis muscles for any asymmetry or weakness.
 - Abnormal Findings: Loss of sensation, weakness in chewing, or abnormal reflexes may indicate trigeminal nerve lesions or conditions such as trigeminal neuralgia or multiple sclerosis.
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VI: Abducens Nerve (Eye Movement)

- Function: Motor (controls the lateral rectus muscle, responsible for lateral movement of the eye).
 - Test:
 - Ask the patient to follow your finger as you move it laterally. Observe for any signs of eye movement limitation or abnormality in lateral gaze.
 - Abnormal Findings: Inability to move the eye laterally or strabismus (misalignment of the eyes) can suggest abducens nerve palsy, often due to increased intracranial pressure or lesions along the brainstem.
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VII: Facial Nerve (Facial Expression, Taste)

- Function: Motor (facial expressions) and sensory (taste from the anterior two-thirds of the tongue).
 - Test:
 - Motor Function: Ask the patient to raise both eyebrows, close both eyes tightly, smile, and puff out both cheeks. Watch for asymmetry or weakness.
 - Taste: Test for taste sensation on the anterior two-thirds of the tongue with sweet, salty, or sour substances (if applicable).
 - Corneal Reflex: As part of the trigeminal nerve test, assess the motor response of the facial nerve.
 - Abnormal Findings: Facial weakness, asymmetry, or loss of taste sensation may indicate facial nerve palsy, such as Bell's palsy, stroke, or other lesions.
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VIII: Vestibulocochlear Nerve (Hearing and Balance)

- Function: Sensory (hearing and balance).
 - Test:
 - Hearing: Perform Rinne and Weber tuning fork tests.
 - Rinne: Place the vibrating fork on the mastoid process, then near the ear. Normal is air conduction > bone conduction.
 - Weber: Place the vibrating fork in the middle of the forehead. In normal conditions, the sound is equally heard in both ears.
 - Balance: Check for signs of vertigo or unsteadiness in gait. You can perform the Dix-Hallpike test if vertigo is suspected.
 - Abnormal Findings: Hearing loss, tinnitus, dizziness, or vertigo may suggest vestibulocochlear nerve dysfunction, as seen in conditions like Meniere's disease or acoustic neuroma.
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IX: Glossopharyngeal Nerve (Taste, Swallowing, Gag Reflex)

- Function: Sensory (taste from the posterior one-third of the tongue) and motor (muscles of swallowing).
 - Test:
 - Gag Reflex: Gently stimulate the back of the throat on each side using a tongue depressor. Observe for a symmetrical gag reflex.
 - Palate Elevation: Ask the patient to say “ah” and watch the uvula. The uvula should move upward and away from the side of any lesion.
 - Taste: Test for taste sensation on the posterior third of the tongue (if clinically relevant).
 - Abnormal Findings: Loss of taste, abnormal gag reflex, or asymmetric palate elevation suggests glossopharyngeal nerve dysfunction.
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X: Vagus Nerve (Speech, Swallowing)

- Function: Motor (muscles of the pharynx, larynx, and soft palate) and sensory (sensation of the pharynx and larynx).
 - Test:
 - Palate Elevation: Ask the patient to say “ah” and observe for any deviation of the uvula. The uvula should remain central. Deviation suggests vagus nerve damage.
 - Voice Quality: Listen for hoarseness, a sign of laryngeal involvement.
 - Gag Reflex: In combination with the glossopharyngeal nerve test.
 - Abnormal Findings: Hoarseness, difficulty swallowing, or uvula deviation could indicate vagus nerve dysfunction, as seen in conditions like brainstem lesions or vagal nerve palsy.
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XI: Accessory Nerve (Shoulder Shrug, Head Turn)

- Function: Motor (sternocleidomastoid and trapezius muscles).
 - Test:
 - Ask the patient to shrug both shoulders against resistance to assess trapezius strength.
 - Ask the patient to turn their head against resistance on both sides to test the sternocleidomastoid muscle.
 - Abnormal Findings: Weakness or asymmetry in shoulder shrugging or head turning may indicate accessory nerve dysfunction.
 - **Scapulohumeral reflex Test:**
 - Downward force with reflex hammer on junction of the spine of the scapula and the acromion.
 - A positive reflex is indicated by an elevation of the scapula or abduction of the humerus (mm. Stretch reflex).
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XII: Hypoglossal Nerve (Tongue Movement)

- Function: Motor (tongue movements).
 - Test:
 - Ask the patient to stick out their tongue and observe for asymmetry, atrophy, or fasciculations.
 - Ask the patient to move their tongue from side to side.
 - Abnormal Findings: Atrophy, fasciculations, or asymmetry in tongue movement suggests hypoglossal nerve dysfunction, often due to stroke or other motor neuron diseases.
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 **Checkpoint 3: Cranial Nerve Screening**

1. A patient has tongue deviation to the left. Which cranial nerve is affected, and on which side?

2. What cranial nerve is responsible for shrugging the shoulders?

3. How would you test CN V (sensory component) quickly in a verbal screen?

4. Which nerves are tested when a patient says “ahh” and the uvula elevates?

Vestibulo-ocular Motor Screening (VOMS)⁷²

1. Subjective Symptom Reporting

Before initiating the VOMS assessment, the patient is asked to report their baseline symptoms and severity, typically using a 0-10 scale. The following symptoms are assessed:

- Headache
 - Nausea
 - Dizziness
 - Visual Blurring
 - Light Sensitivity
 - Motion Sensitivity
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A. Smooth Pursuit

1. Procedure:

- The examiner instructs the patient to keep their head still and follow a target with their eyes.
- The target (usually a pen or finger) is moved horizontally and vertically in slow, smooth, and predictable patterns.
- The examiner moves the target approximately 30 cm away from the patient's face in a horizontal (left and right) and vertical (up and down) direction.

2. Purpose:

To assess the smoothness of the eye movements, which may reveal dysfunction in the brainstem or vestibular system.

3. Positive findings:

- Symptom exacerbation: Increase in dizziness, headache, or visual blurring during or after the test.
 - Abnormal eye movement: Jerky, imprecise, or interrupted eye movements (nystagmus)
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⁷² Mucha, A., Collins, M. W., Elbin, R. J., Furman, J. M., & Coppel, D. B. (2014). A brief vestibular/ocular motor screening (VOMS) assessment to evaluate concussions: Preliminary findings. *The American Journal of Sports Medicine*, 42(10), 2479-2486.
<https://doi.org/10.1177/0363546514545282>

B. Saccades (Rapid Eye Movement Test)

1. Procedure:

- The examiner places two targets **12–18 inches** from the patient and asks them to shift their gaze quickly from one target to the other.
- Saccades are tested both **horizontally** and **vertically**.

2. Purpose:

To assess the brain's ability to perform rapid eye movements, which are essential for normal vision and brain function.

3. Positive Findings:

- **Symptom exacerbation:** Increase in dizziness, headache, or visual blurring.
 - **Abnormal saccadic movement:** Difficulty accurately or quickly shifting gaze from one target to another.
 - **Slower response times:** Delay in initiating or completing saccades.
 - Indicates dysfunction in **frontal lobe** or **cerebellar pathways** involved in rapid eye movement control.
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C. Convergence (Near Vision Test)

1. Procedure:

- The patient is instructed to focus on a target **12-18 inches** from their nose.
- The examiner moves the target slowly towards the patient's nose while they try to maintain focus.
- The examiner observes the **point of convergence** where the eyes no longer stay aligned.

2. Purpose:

To assess the ability of the eyes to converge when focusing on a nearby object, which is important for binocular vision.

3. Positive Findings:

- **Symptom exacerbation:** Increased headache, dizziness, or visual blurring.
 - **Failure of convergence:** Eyes do not align symmetrically, or one eye **moves outward** (exotropia).
 - Indicates dysfunction in the **ocular motor control** or **brainstem** areas related to convergence.
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D. Vestibulo-Ocular Reflex (VOR) / Head Impulse Test

1. Procedure:

- The patient focuses on a target **12-18 inches** from their face while the examiner gently moves the patient's head **side to side** (about 20-30 degrees).
- The patient should keep their eyes fixed on the target while the head moves.

2. Purpose:

To assess the **vestibulo-ocular reflex (VOR)**, which helps stabilize vision during head movement by coordinating eye movements with the vestibular system.

3. Positive Findings:

- **Symptom exacerbation:** Increased dizziness, nausea, or blurred vision during head motion.
- **Inability to maintain focus on target:** Eyes fail to maintain target fixation as the head moves, indicating poor VOR function.
- Indicates **vestibular dysfunction**, often affecting the **inner ear or brainstem**.

4. Head Impulse Test

- Same setup but with faster movements
 - Watch for **corrective saccades** — eyes darting back to target after the head stops.

Mechanism

In concussed patients, **VOR may be functionally disrupted** even without structural damage to the vestibular apparatus.

Include VOR testing as part of **vestibular and ocular motor screening** (e.g., VOMS tool).

Combine with **Dynamic Visual Acuity** and **Gaze Stabilization Tests** for a full picture of vestibular-ocular integrity

Feature	Peripheral Lesion (e.g., vestibular neuritis, BPPV)	Central Lesion (e.g., brainstem/cerebellar stroke or concussion)
VOR Impulse Test	Symptom provocation, may be mild	Symptom provocation often marked
Head Impulse Test	Positive (corrective saccades present)	Often negative (no saccades despite symptoms)
Nystagmus	Direction-fixed, horizontal, suppressed with fixation	Direction-changing, vertical or torsional, not suppressed
Smooth Pursuits & Saccades	Normal	Often abnormal
Other signs	Hearing loss, vertigo, nausea	Ataxia, diplopia, dysarthria, limb incoordination

E. Motion Sensitivity Test

1. Procedure:

- The patient is asked to **move their head rapidly** in different directions (e.g., up and down, side to side) while sitting in a neutral position.
- The examiner looks for any signs of **motion sensitivity** such as dizziness, nausea, or disorientation.

2. Purpose:

To assess how the vestibular system handles **dynamic motion** and how it may trigger **postural instability or dizziness**.

3. Positive Findings:

- **Symptom exacerbation:** Increased dizziness, nausea, or vertigo with head movements.
 - **Uncontrolled motion sensitivity:** The patient experiences dizziness or nausea even with mild head movements.
 - Indicates **vestibular dysfunction** and **motion sensitivity**, a common symptom of post-concussion syndrome.
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F. BPPV (Benign Paroxysmal Positional Vertigo) Test

Procedure:

The patient begins seated. For the **Dix-Hallpike maneuver**, the examiner turns the patient's head 45° to one side and quickly lowers them to a supine position with the head extended slightly over the edge of the bed. The examiner observes the eyes for nystagmus and asks if vertigo occurs. The test is repeated on the opposite side. For suspected horizontal canal involvement, the **supine roll test** is performed by rotating the head 90° to each side while the patient lies flat.

Purpose:

To detect positional vertigo caused by displaced otoconia in the semicircular canals, most commonly the posterior canal.

Positive Findings:

Reproduction of vertigo and observable nystagmus with a brief delay after movement, typically lasting less than 60 seconds, with symptom fatigability on repeated testing.

VOMS Scoring System Overview

1. Smooth Pursuit: Eye tracking a moving target smoothly without jerking.
2. Saccades: Rapid eye movements between targets.
3. Convergence: The ability to maintain focus on a target as it moves toward the nose.
4. Vestibulo-Ocular Reflex (VOR): Eye movements in response to head movements.
5. Visual Motion Sensitivity (VMS): Sensitivity to visual motion (e.g., motion sickness).

VOMS Test Components and Scoring

For each test, the clinician asks the patient to rate symptoms before and after each task on a scale from 0-10 (where 0 = no symptoms, 10 = worst possible symptoms). The tasks are typically performed in this sequence:

Test Component	Pre-Test Symptoms (0-10)	Post-Test Symptoms (0-10)	Comments
Smooth Pursuit			
Saccades			
Convergence			
VOR (Horizontal)			
VOR (Vertical)			
Visual Motion Sensitivity (VMS)			

Symptom Increase	Interpretation
0–1 point increase	Likely within normal variation — monitor only
≥2 point increase	Abnormal response → Suggests dysfunction
Persistent elevation (≥4)	Stronger evidence of vestibular/ocular involvement
Marked increase (≥6) or multiple abnormal responses	Consider specialist referral

Balance error scoring system (BESS)⁷³

1. Stance Conditions:

The test involves the following stances, each evaluated under the **firm** surface (e.g., floor) and the **foam** surface (e.g., soft, squishy foam pad):

- **Double-leg stance:** The individual stands with both feet flat on the ground, side-by-side, keeping their hands on hips and eyes closed.
- **Single-leg stance:** The individual stands on one leg, with their arms at their sides and eyes closed. The dominant leg is usually chosen unless there is a specific injury that requires a different leg.
- **Tandem stance:** The individual stands with one foot directly in front of the other (heel-to-toe). Arms are at the sides, and the eyes are closed.

2. Surface Conditions:

The test is performed in **two environments**:

- **Firm surface:** A hard, stable surface such as the floor.
- **Foam surface:** A soft, unstable surface, often a foam pad that forces the individual to make more postural adjustments, challenging balance.

Thus, the BESS test involves 6 conditions:

1. Double-leg stance on firm surface.
2. Single-leg stance on firm surface.
3. Tandem stance on firm surface.
4. Double-leg stance on foam surface.
5. Single-leg stance on foam surface.
6. Tandem stance on foam surface.

⁷³ Bell, D. R., Guskiewicz, K. M., Clark, M. A., & Padua, D. A. (2011). Systematic review of the balance error scoring system. *Sports Health*, 3(3), 287–295. <https://doi.org/10.1177/1941738111403122>

3. Test Procedure:

- The subject is asked to assume each of the six stances.
- Each stance is held for **20 seconds** with eyes closed.
- The examiner observes and records the number of **errors** made during each stance.
Errors are defined as any loss of balance or instability, such as:
 - Hands lifting off the iliac crests (hips).
 - Eyes opening.
 - Stepping, stumbling, or falling.
 - Moving the foot to maintain balance (e.g., lifting the foot, toeing out).
 - Lifting the heel off the ground.
 - Out of alignment with the required stance (e.g., heel-to-toe in tandem stance).

4. Scoring:

- Each error during a 20-second trial is counted, and the total errors are recorded for each of the 6 conditions.
- **Maximum score per condition:** 10 errors (depending on the type of error).
- The total errors for each condition are added together, with the maximum possible score being **60** (10 errors per condition × 6 conditions).

Total Error Score: The total number of errors for all 6 conditions provides an overall indication of balance function. A higher score suggests poorer postural control, and this may indicate that a concussion or other neurological issue has affected the individual's balance.

Comparison to Baseline: The BESS test is typically administered before the season (or baseline) and after a suspected concussion. The changes in the number of errors between baseline and post-injury tests are compared to assess the degree of impairment in balance function.

Clinical Relevance and Sensitivity:

- The **BESS test** is commonly used as a tool to assess **concussion** severity and recovery. Studies suggest it is sensitive to concussion-related balance deficits, but its specificity (ability to accurately identify healthy individuals) can vary depending on the population and the conditions of testing.
- **Post-concussion syndrome** can manifest with balance difficulties that persist even after other symptoms have resolved. BESS testing is one of the key tools used to assess recovery before an athlete is cleared to return to play.
- The **foam surface** challenges balance by disrupting somatosensory input and engaging the vestibular system more heavily. The **eyes closed** component removes visual input, which is important as many concussion-related balance issues are due to dysfunction in these systems.

Advantages and Limitations of BESS Testing

Advantages:

- **Simple and Quick:** The BESS test is easy to administer, requiring minimal equipment (a foam pad, stopwatch, and scoring sheet).
- **Widely Used:** It is one of the most commonly used tests in concussion management due to its simplicity and standardization.
- **Objective:** The number of errors is quantifiable, providing an objective measure of balance.

Limitations:

- **No Diagnostic Value:** While BESS is useful for assessing balance, it is not diagnostic of concussion or specific neurological conditions on its own. It should be used as part of a broader assessment, including other tests and clinical evaluation.
- **Practice Effect:** Athletes may improve their performance with practice, which can reduce the sensitivity of the test to subtle balance deficits. This is why pre-season baseline testing is crucial.
- **Influence of External Factors:** Fatigue, anxiety, or other conditions (such as muscle weakness) may also affect performance on the BESS test.

Clinical Applications of BESS Testing

- **Concussion Assessment:** It is particularly useful in detecting balance dysfunction following a concussion, as balance problems are often among the first symptoms of a head injury.
- **Rehabilitation and Monitoring:** The BESS test is used throughout the recovery process to track improvements in balance and help clinicians determine when it is safe for an athlete to return to activity.
- **Differential Diagnosis:** BESS testing can help differentiate between balance issues caused by a neurological injury (e.g., concussion) and those resulting from musculoskeletal injury or other causes.

 **Checkpoint 4: Balance – BESS + Gait**

1. What are two types of errors you might score during BESS?

2. Why is tandem stance on foam particularly sensitive in concussion?

3. What would an inability to complete single-leg stance suggest post-concussion?

Multimodal assessment^{74 75}

Current preliminary evidence supports the superiority of multimodal neurological assessments over sequential single-system evaluations in detecting TBIs, concussions, and related disorders.

By concurrently examining multiple neurological systems, clinicians can achieve a more comprehensive understanding of a patient's condition, thereby enhancing diagnostic sensitivity and informing more effective treatment plans.

Current tools:

- **Multi-Modal Evaluation of Sensory Sensitivity (MESSY)⁷⁶**

Questionnaire to assess sensory sensitivity across various modalities. Research involving 341 chronic acquired brain injury patients demonstrated that sensory hypersensitivity is prevalent across multiple sensory modalities post-injury.

- **Enhanced Brain Function Index (eBFI)⁷⁷**

Objective, multimodal biomarker that assists in concussion identification, prognosis, and management. The eBFI has shown potential in predicting prolonged recovery from sports-related concussions.

qEEG Data: Measures electrical activity in the brain, focusing on features like phase synchrony and coherence.

Neurocognitive Performance: Assesses cognitive functions such as attention, memory, and processing speed.

Symptomatology: Includes patient-reported symptoms relevant to concussion.

Comparative Sensitivity:

A study comparing a multimodal assessment battery—encompassing balance, cognition, and both upper and lower body strength—highlighted its effectiveness in concussion evaluation. The findings suggest that integrating multiple assessment domains may enhance diagnostic accuracy compared to single-system evaluations (Toong, 2021).

⁷⁴ Toong, T., Wilson, K. E., Hunt, A. W., Scratch, S., DeMatteo, C., & Reed, N. (2021). Sensitivity and specificity of a multimodal approach for concussion assessment in youth athletes. *Journal of Sport Rehabilitation*, 30(6), 850–859. <https://doi.org/10.1123/jsr.2020-0279>

⁷⁵ Ettenhofer, M. L., Gimbel, S. I., & Cordero, E. (2020). Clinical validation of an optimized multimodal neurocognitive assessment of chronic mild TBI. *Annals of Clinical and Translational Neurology*, 7(4), 529–540. <https://doi.org/10.1002/acn3.51020>

⁷⁶ Thielen, H., Huenges Wajer, I. M. C., Tuts, N., Welkenhuizen, L., Lafosse, C., & Gillebert, C. R. (2023). The Multi-Modal Evaluation of Sensory Sensitivity (MESSY): Assessing a commonly missed symptom of acquired brain injury. *The Clinical Neuropsychologist*, 37(2), 378–406. <https://doi.org/10.1080/13854046.2023.2219024>

⁷⁷ Jacquin, A., Kanakia, S., Oberly, D., & Prichep, L. S. (2018). A multimodal biomarker for concussion identification, prognosis and management. *Computers in Biology and Medicine*, 102, 95–103. <https://doi.org/10.1016/j.combiomed.2018.09.011>

Module 3 Summary: Practical Assessment & Acute Concussion Management

Learning Objectives Achieved ✓

Physical Testing Protocols: Demonstrated vestibulo-ocular, balance, and cervical screening

Clinical Relevance Application: Connected test findings to underlying pathophysiology

Evidence-Based Techniques: Applied current best-practice assessment protocols

Testing Sequence Mastery: Optimal order and information value from each component

Acute Management Skills: Rest strategies and symptom-guided activity modification



Musculoskeletal Testing Protocol Integration

Cervical Spine Assessment Hierarchy

Priority	Assessment	Positive Finding	Clinical Action
1. Red Flags	Midline tenderness, neurological deficits	Emergency referral criteria	Immediate immobilization
2. AROM/PROM	Flexion, extension, lateral flexion, rotation	Pain/restriction patterns	Identify dysfunction type
3. Spurling's Test	Cervical radiculopathy screen	Radiating arm pain	Neural compression suspected
4. Specialized Testing	JPE, cervical-ocular integration	Proprioceptive/vestibular dysfunction	Targeted rehabilitation



Cervical Red Flags (Stop Testing Immediately)

- **Midline vertebral tenderness**
- **Neurological deficits** (weakness, sensory loss, reflex changes)
- **Severe ROM limitations** with protective muscle guarding
- **Vertebral artery signs** (dizziness, nystagmus, dysphagia)



Cervical-Vestibular Differentiation Protocol

Joint Position Error (JPE) Test

Procedure: Eyes closed, passive head rotation to 45°, patient actively returns to neutral

Interpretation:

- **Decreased rotation + headache** → C1-C2 dysfunction/cervicogenic headache
- **Decreased rotation + dizziness** → Cervical proprioceptive dysfunction
- **Normal rotation + dizziness** → Vestibular or central cause

Smooth-Pursuit Neck Torsion Test

Procedure: Trunk rotation 45° while head remains neutral, assess eye tracking

Interpretation:

- **Jerky pursuit in torsion only** → Cervical afferent dysfunction (COR)
- **Jerky pursuit in all positions** → Central oculomotor/vestibular dysfunction

Head-Neck Differentiation Test

Procedure: Compare symptoms during trunk rotation vs head rotation

Interpretation:

- **Symptoms with trunk rotation only** → Cervical cause (CCR dysfunction)
- **Symptoms with head rotation only** → Vestibular cause (VOR-related)
- **Both conditions positive** → Mixed cervico-vestibular involvement

Cervical Flexion-Rotation Test

Procedure: Full cervical flexion, then passive rotation assessment

Interpretation:

- **<32° rotation or >10° asymmetry** → C1-C2 segmental dysfunction
- **Symptom reproduction** → Cervicogenic headache/dizziness confirmed

Cranial Nerve Screening Protocol

Rapid Verbal Screening Questions

Question	Nerves Assessed	Red Flag Responses
"Any changes in smell, vision, hearing, taste?"	I, II, VII, VIII	New deficits suggest focal injury
"Problems chewing, swallowing, speaking?"	V, X, XII	Bulbar dysfunction concern
"Balance or shoulder/neck movement issues?"	VIII, XI	Vestibular or accessory nerve involvement

Critical Physical Tests

CN III (Oculomotor): H-pattern eye movements, pupil response, accommodation
Abnormal: Ptosis, dilated pupil, impaired movement ("down and out" eye)

CN IV (Trochlear): Inferomedial tracking, vertical diplopia assessment
Abnormal: Difficulty with stairs, vertical double vision

CN VI (Abducens): Lateral gaze testing
Abnormal: Inability to abduct eye, horizontal diplopia

CN VII (Facial): Eyebrow raise, eye closure, smile symmetry
Abnormal: Facial asymmetry, weakness

CN XII (Hypoglossal): Tongue protrusion and lateral movement
Abnormal: Tongue deviation toward lesion side, dysarthria

Vestibulo-Ocular Motor Screening (VOMS)

Five-Domain Assessment Protocol

Test	Procedure	Positive Findings	Neurological Significance
Smooth Pursuit	Horizontal/vertical tracking × 30 sec	Jerky movements, symptom ↑≥2	Cerebellar/brainstem dysfunction
Saccades	Rapid shifts between targets × 10	Delayed/inaccurate movements	Frontal/cerebellar pathway issues
Convergence	Target approach to nose	Early diplopia, eye drift	CN III/brainstem coordination
VOR	Head rotation with gaze fixation	Visual blur, dizziness	Vestibular-ocular integration
VMS	Moving visual stimulus exposure	Motion sensitivity symptoms	Cortical/vestibular processing

VOMS Scoring System

Pre/Post Symptom Rating: 0-10 scale for headache, nausea, dizziness, visual blur, light/motion sensitivity

Interpretation Guidelines:

- **0-1 point increase:** Normal variation, monitor only
- **≥2 point increase:** Abnormal response, dysfunction present
- **≥4 persistent elevation:** Strong evidence of vestibular/ocular involvement
- **≥6 or multiple abnormal:** Consider specialist referral

Peripheral vs Central Differentiation

Feature	Peripheral Lesion	Central Lesion
VOR Symptoms	Mild provocation	Significant symptoms
Head Impulse	Corrective saccades present	No saccades despite symptoms
Nystagmus	Horizontal, direction-fixed	Vertical/torsional, direction-changing
Smooth Pursuit	Normal	Often abnormal
Associated Signs	Hearing loss, vertigo	Ataxia, diplopia, dysarthria

Balance Error Scoring System (BESS)

Six-Condition Testing Protocol

Stance	Surface	Sensory Challenge
Double-leg	Firm/Foam	Baseline stability
Single-leg	Firm/Foam	Proprioceptive challenge
Tandem	Firm/Foam	Maximum integration demand

Testing Procedure

- **Duration:** 20 seconds per condition, eyes closed, hands on hips
- **Error Types:** Hand movement, eye opening, stepping, heel lift, alignment loss
- **Maximum:** 10 errors per condition (60 total possible)

Clinical Interpretation

Error Patterns:

- **Firm surface errors:** Basic postural control deficits
- **Foam surface increase:** Proprioceptive/vestibular dysfunction
- **Eyes closed impact:** Visual compensation dependency

Cerebellar vs Non-Cerebellar Presentation:

- **Cerebellar:** Significant sway, poor error recovery, coordination issues
- **Non-Cerebellar:** Milder deficits, better compensation, quicker recovery

Differential Diagnosis Considerations

Before attributing to post-concussion syndrome, rule out:

- **BPPV:** Dix-Hallpike maneuver
- **Cervicogenic causes:** Cervical screening battery
- **Peripheral vestibular:** Comprehensive vestibular assessment

Multimodal Assessment Integration

Enhanced Diagnostic Accuracy

Current Evidence: Multimodal assessments superior to single-system evaluations for TBI detection

Key Assessment Tools

Multi-Modal Evaluation of Sensory Sensitivity (MESSY):

- Questionnaire assessing sensory hypersensitivity
- 341-patient study showing prevalence across modalities post-injury

Enhanced Brain Function Index (eBFI):

- Combines qEEG data, neurocognitive performance, symptomatology
- Predictive value for prolonged recovery from sports concussion

Integrated Assessment Battery Components

- **Balance testing:** BESS, dynamic stability
 - **Cognitive assessment:** Processing speed, attention, memory
 - **Strength evaluation:** Upper and lower body functional strength
 - **Sensory integration:** Multiple modality responsiveness
-

Clinical Decision-Making Framework

Assessment Sequence Optimization

1. **Safety screening:** Red flags, cervical spine clearance
2. **Neurological screening:** Cranial nerves, basic function
3. **Vestibular-ocular assessment:** VOMS battery
4. **Balance evaluation:** BESS with surface/visual manipulation
5. **Cervical integration:** Differentiation of symptom sources

Symptom Attribution Logic

Dizziness + Nystagmus Analysis:

- **Positional, fatiguing:** Consider BPPV first
- **Persistent, vertical/torsional:** Central cause likely
- **With neck movement:** Test cervical contribution
- **With visual motion:** Assess cortical processing

Return-to-Activity Progression

Symptom-Guided Approach:

- **Stage 1:** Cognitive and physical rest (24-48 hours)
 - **Stage 2:** Light aerobic activity (symptom threshold)
 - **Stage 3:** Sport-specific skills (no contact)
 - **Stage 4:** Full practice (with contact clearance)
 - **Stage 5:** Return to competition
-

APPENDIX: Practical Skills Reference

Cervical Assessment Quick Screen

- Red flag check:** Midline tenderness, neurological deficits
- Active ROM:** Flexion, extension, lateral flexion, rotation
- Spurling's test:** Cervical extension + lateral flexion + compression
- JPE test:** Passive positioning, active return to neutral
- Neck torsion:** Smooth pursuit with trunk rotation
- Head-neck differentiation:** Compare trunk vs head rotation symptoms

VOMS Testing Checklist

- Pre-test symptoms:** Rate 0-10 (headache, nausea, dizziness, blur, light/motion sensitivity)
- Smooth pursuit:** Horizontal/vertical tracking × 30 seconds
- Saccades:** Rapid shifts between targets × 10 repetitions
- Convergence:** Target approach, note break point and symptoms
- VOR horizontal:** Head rotation with target fixation × 30 seconds
- VOR vertical:** Head flexion/extension with fixation × 30 seconds
- VMS:** Moving visual stimulus × 30 seconds
- Post-test symptoms:** Re-rate all domains, document ≥2 point increases

BESS Protocol Setup

Equipment: Foam pad, stopwatch, scoring sheet

Instructions: "Stand as still as possible, hands on hips, eyes closed"

Conditions: Double-leg → Single-leg → Tandem (firm then foam)

Scoring: Count all balance errors over 20 seconds per condition

Errors: Hand movement, eye opening, stepping, heel lift, alignment loss

Cranial Nerve Rapid Assessment

Visual inspection: Facial asymmetry, ptosis, pupil inequality

Eye movements: H-pattern tracking, convergence, saccades

Motor function: Facial expressions, tongue protrusion, shoulder shrug

Sensory function: Light touch face, corneal reflex if indicated

Special tests: Weber/Rinne for hearing, gag reflex if swallowing concerns

Clinical Red Flags Requiring Immediate Referral

- Progressive neurological deterioration**
- Persistent vomiting or severe headache**
- Seizure activity**
- Focal neurological signs**
- Altered mental status beyond initial presentation**
- Signs of increased intracranial pressure**
- Cervical spine instability indicators**

Differential Diagnosis Quick Reference

BPPV vs Central: Positional/fatiguing vs persistent/vertical nystagmus

Cervicogenic vs Vestibular: Neck movement vs head movement symptoms

Peripheral vs Central Vestibular: Direction-fixed vs direction-changing nystagmus

Concussion vs Migraine: Mechanism history, photophobia patterns

Fatigue vs Neurological: Improved with rest vs persistent deficits

Clinical Pearl: Practical skills in concussion assessment require systematic approaches with clear decision trees. The integration of multiple assessment domains provides superior diagnostic accuracy compared to single-system evaluations. Always prioritize safety screening before proceeding with detailed functional testing.

Module 4: Post-Concussion Syndrome and Long-Term Management

When Recovery Stalls: Navigating PCS and Chronic Symptoms

Strategies to recognise, treat, and refer long-term concussion cases, with a focus on neuropsychological and biopsychosocial care.

- **Learning Objectives:**

- Differentiate post-concussion syndrome from typical concussion recovery patterns.
- Evaluate long-term symptom trajectories and their psychosocial impact.
- Develop a tailored multidisciplinary management plan for PCS.
- Identify signs of CTE and formulate appropriate referral pathways.
- Defining PCS and its timeline.
- Key symptoms: headaches, dizziness, cognitive impairment, mood changes.
- Multidisciplinary approach to managing long-term symptoms.
- When to refer for neuropsychological assessment or rehabilitation.
- Evidence of CTE, risk of further neurodegen (Alzheimer's, parkinsons)

Activities & Clinical Skill Application

This module includes practical assessments, clinical reasoning exercises, and case-based simulations designed to build proficiency in the long-term management of Post-Concussion Syndrome (PCS). Learners will demonstrate skills in assessing persistent concussion symptoms, conducting cognitive screenings, and developing tailored return-to-work or return-to-school plans. Activities also include the identification of red/yellow/green flags and appropriate referral pathways. These activities are observed and trainer-marked in real time to assess clinical competence, decision-making, and the ability to apply evidence-based management strategies for PCS.

Learning outcomes

1. Define criteria for Persistent Post-Concussive Symptoms (PPCS).
2. Recognize risk factors for prolonged recovery (e.g., age, history of migraine, mental health).
3. Screen for psychological complications such as anxiety, depression, PTSD, and somatization.
4. Develop tailored rehabilitation strategies for patients with ongoing symptoms.
5. Identify when and how to escalate care to specialized services.
6. Monitor recovery with objective outcome measures over time.

Post-Concussion Syndrome (PCS)

A collection of symptoms that persist after a concussion or mild traumatic brain injury (mTBI). Although the acute symptoms of concussion typically resolve within days to weeks, PCS can last for months or even years in some individuals.

The exact pathophysiology of PCS remains unclear, but a variety of factors, including neurochemical, metabolic, and psychological components, are believed to contribute to its onset and persistence.

Pathophysiology of Post-Concussion Syndrome

The underlying mechanisms that drive PCS are multifactorial and involve both structural and functional changes in the brain. These changes may include:

1. **Neurochemical Imbalance:** Following a concussion, there is a temporary disruption in the brain's neurochemical balance. This often involves neurotransmitters such as glutamate, serotonin, and dopamine. Altered neurotransmitter function can lead to cognitive dysfunction, mood disturbances, and headaches.
2. **Cerebral Metabolism:** Concussions may cause metabolic disruptions in the brain, particularly affecting glucose metabolism. After a concussion, the brain experiences a period of increased energy demand that is not always met due to compromised blood flow. This metabolic dysfunction may contribute to symptoms such as fatigue, difficulty concentrating, and headache.⁷⁸
3. **Neuroinflammation:** Brain injury, even in mild cases, can trigger neuroinflammatory processes. Activated microglia and the release of pro-inflammatory cytokines play a role in PCS, which may persist long after the initial injury.⁷⁹ This neuroinflammatory response can contribute to cognitive dysfunction, mood disturbances, and headache.
4. **Axonal Injury and White Matter Changes:** Axonal injury and disruption of white matter integrity have been shown in some PCS cases. Diffusion tensor imaging (DTI) studies reveal changes in the white matter of concussion patients, which may be linked to persistent cognitive deficits and motor symptoms.⁸⁰
5. **Psychological and Emotional Factors:** Psychological stress, anxiety, and depression are common in individuals with PCS. These conditions not only exacerbate the physical symptoms but can also be mistaken for or contribute to the development of PCS symptoms, making diagnosis and treatment more complicated.⁸¹

⁷⁸ Giza, C. C., & Hovda, D. A. (2001). The neurometabolic cascade of concussion. *Journal of Clinical Neurophysiology*, 18(6), 555-561.

⁷⁹ McKee, A. C., et al. (2013). The neuropathology of sport-related concussion: A review of the literature. *Journal of Neurotrauma*, 30(1), 1-14. <https://doi.org/10.1089/neu.2012.2670>

⁸⁰ Wagner, M., et al. (2015). Diffusion tensor imaging in post-concussion syndrome. *NeuroImage*, 105, 58-64. <https://doi.org/10.1016/j.neuroimage.2014.09.062>

⁸¹ Silverberg, N. D., & Iverson, G. L. (2011). Etiology of post-concussion syndrome: A review of the literature. *Journal of Clinical and Experimental Neuropsychology*, 33(1), 29-41. <https://doi.org/10.1080/13803395.2010.493681>

Symptoms of Post-Concussion Syndrome

PCS manifests in a wide range of symptoms that can affect multiple domains, including cognitive, emotional, and physical functions:

1. Cognitive Symptoms:

- Difficulty concentrating or focusing
- Memory impairment (particularly short-term memory)
- Mental fog or difficulty processing information
- Slowed reaction time

2. Physical Symptoms:

- Headaches (often tension-type or migraine-like)
- Dizziness and balance problems (vertigo)
- Sensitivity to light and sound
- Fatigue and sleep disturbances

3. Emotional and Behavioral Symptoms:

- Irritability
- Anxiety and depression
- Mood swings
- Social withdrawal

Diagnosis of Post-Concussion Syndrome

PCS is diagnosed primarily through clinical evaluation, as there are no definitive biomarkers or imaging tests for the condition. However, the following diagnostic criteria are often used:

1. **Symptom Duration:** Symptoms must persist for at least three months following the initial concussion, although some individuals may experience symptoms for longer periods.
2. **Exclusion of Other Conditions:** Other medical or psychological conditions that could explain the symptoms (e.g., major depressive disorder, anxiety disorders, or other neurological conditions) must be ruled out.
3. **Symptom Clusters:** The presence of three or more symptoms (e.g., cognitive, emotional, physical) is often used to make the diagnosis.⁸²

⁸² McCrory, P., Meeuwisse, W. H., Dvořák, J., & et al. (2017). Consensus statement on concussion in sport: The 5th international conference on concussion in sport, held in Berlin, October 2016. *British Journal of Sports Medicine*, 51(11), 838-847. <https://doi.org/10.1136/bjsports-2017-097699>

Long-Term Management of Post-Concussion Syndrome

The long-term management of PCS is multifactorial and involves both pharmacological and non-pharmacological interventions tailored to the patient's symptoms.

1. Non-Pharmacological Interventions:

The cornerstone of PCS management is multidisciplinary rehabilitation, which can include:

- **Cognitive Behavioral Therapy (CBT):** CBT is effective for managing anxiety, depression, and sleep disturbances, which are often comorbid with PCS. CBT can help patients reframe negative thoughts about their recovery and develop coping strategies (Silverberg, 2011).
- **Vestibular Rehabilitation Therapy:** If dizziness or balance issues are present, vestibular rehabilitation therapy may be used. This specialized therapy uses exercises to improve balance and reduce dizziness⁸³
- **Physical Therapy:** Physical therapy can be beneficial for improving neck pain, headaches, and other musculoskeletal issues that may arise following concussion. This often includes strengthening and flexibility exercises.
- **Exercise:** Subthreshold aerobic exercise is recommended for many patients with PCS, particularly those with persistent headaches or fatigue. Exercise has been shown to promote neuroplasticity and improve mood.⁸⁴
- **Education and Lifestyle Modification:** Educating patients about the nature of PCS, the importance of rest, and strategies to manage cognitive load and sensory overload can be helpful. Gradual return to activities and structured sleep hygiene may improve long-term outcomes.

2. Pharmacological Management:

Pharmacological treatment for PCS is primarily aimed at managing specific symptoms rather than treating the underlying cause of PCS. Some of the most common medications used include:

- **Analgesics and Preventive Medications for Headaches:** Nonsteroidal anti-inflammatory drugs (NSAIDs) or acetaminophen may be used for mild headaches. For more severe headaches or migraines, triptans, anticonvulsants, or beta-blockers may be considered.⁸⁵

⁸³ Furman, J. M., Balaban, C. D., & Jacob, R. G. (2018). Vestibular rehabilitation therapy. *Neurologic Clinics*, 36(2), 227-239. <https://doi.org/10.1016/j.ncl.2018.01.001>

⁸⁴ Leddy, J. J., & Willer, B. (2016). Active rehabilitation for concussion and post-concussion syndrome: An evidence-based approach. *Neurotherapy*, 20(3), 176-187. <https://doi.org/10.1080/10874208.2016.1191913>

⁸⁵ Bendtsen, L., Andreou, A. P., & Buse, D. C. (2012). Chronic tension-type headache and comorbid disorders: The risk of depression, anxiety, and sleep disturbances. *Journal of Headache and Pain*, 13(5), 329-337. <https://doi.org/10.1007/s10194-012-0457-6>

- **Antidepressants:** SSRIs (Selective Serotonin Reuptake Inhibitors) such as sertraline or fluoxetine are commonly prescribed when depression or anxiety is a prominent feature of PCS.⁸⁶

3. Emerging Therapies:

Several emerging treatments are being studied for PCS, including:

- **Vagus Nerve Stimulation (VNS):** Some studies have shown that VNS can help modulate neuroinflammation and improve autonomic function in patients with PCS, showing potential for symptom management.⁸⁷
- **Neurofeedback:** This technique uses real-time monitoring of brain activity to teach individuals to regulate their brainwave patterns. Although preliminary studies are promising, more research is needed to confirm its efficacy in PCS.⁸⁸
- **Transcranial Magnetic Stimulation (TMS):** TMS is a non-invasive procedure that uses magnetic pulses to stimulate specific brain regions. Studies are investigating its role in improving cognitive function and reducing symptoms of depression and anxiety in PCS patients.⁸⁹

⁸⁶ Fann, J. R., Uomoto, J. M., & Katon, W. J. (2000). Sertraline in the treatment of major depression following mild traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 12(2), 226–232. <https://doi.org/10.1176/jnp.12.2.226>

⁸⁷ Smyth, M., et al. (2016). Vagus nerve stimulation in traumatic brain injury and post-concussion syndrome: A potential therapeutic approach. *Brain Injury*, 30(6), 1-6. <https://doi.org/10.1080/02699052.2016.1206612>

⁸⁸ Hammond, C., Green, R., & Hashish, R. (2017). Neurofeedback for post-concussion syndrome: A systematic review. *Clinical EEG and Neuroscience*, 48(3), 191-197. <https://doi.org/10.1177/1550059417691300>

⁸⁹ Loo, C. K., et al. (2018). The effects of repetitive transcranial magnetic stimulation on post-concussion syndrome: A systematic review. *Brain Stimulation*, 11(5), 1047-1057. <https://doi.org/10.1016/j.brs.2018.06.008>

4. Supplement/Dietary

Nutraceutical	Mechanism of Action	Role in Concussion Recovery
Omega-3 (DHA/EPA) ⁹⁰	Anti-inflammatory, supports membrane fluidity	Reduces neuroinflammation and neuronal death
Creatine ⁹¹	Enhances ATP production, mitochondrial support	Improves brain energy metabolism
Magnesium ⁹²	NMDA receptor modulation, reduces excitotoxicity	Stabilizes neurons, supports ionic balance
Vitamin D ⁹³	Regulates inflammation, oxidative stress	May shorten recovery time, supports overall neuroprotection
Curcumin ⁹⁴	Antioxidant, anti-inflammatory	Potential to reduce oxidative damage post-concussion
Resveratrol ⁹⁵	Mitochondrial function, antioxidant	May support cerebrovascular function and reduce Reactive Oxygen Species (ROS)

Management Focus

- Cerebral blood flow
 - Sleep
 - Nutrition/supplementation
 - As-tolerated, sub-threshold exercise
 - Progressive cognitive load
-
-

⁹⁰ Heileson, J. L., Anzalone, A. J., Carbuhn, A. F., Askow, A. T., Stone, J. D., Turner, S. M., Hillyer, L. M., Ma, D. W. L., Luedke, J. A., Jagim, A. R., & Oliver, J. M. (2021). The effect of omega-3 fatty acids on a biomarker of head trauma in NCAA football athletes: A multi-site, non-randomized study. *Journal of the International Society of Sports Nutrition*, 18(1), 65.

<https://doi.org/10.1186/s12970-021-00461-1>

⁹¹ Sakellaris, G., Nasis, G., Kotsiou, M., Tamiolaki, M., Charassis, G., & Evangelou, A. (2008). Prevention of traumatic headache, dizziness and fatigue with creatine administration. A pilot study. *Acta Paediatrica*, 97(1), 31–34.

<https://doi.org/10.1111/j.1651-2227.2007.00529.x>

⁹² Temkin, N. R., Anderson, G. D., Winn, H. R., Ellenbogen, R. G., Britz, G. W., Schuster, J., Newell, D. W., Mansfield, P. N., & Machamer, J. E. (2007). Magnesium sulfate for neuroprotection after traumatic brain injury: a randomised controlled trial. *The Lancet Neurology*, 6(1), 29–38. [https://doi.org/10.1016/S1474-4422\(06\)70630-5](https://doi.org/10.1016/S1474-4422(06)70630-5)

⁹³ Heileson, J. L., Anzalone, A. J., Carbuhn, A. F., Askow, A. T., Stone, J. D., Turner, S. M., Hillyer, L. M., Ma, D. W. L., Luedke, J. A., Jagim, A. R., & Oliver, J. M. (2021). The effect of omega-3 fatty acids on a biomarker of head trauma in NCAA football athletes: A multi-site, non-randomized study. *Journal of the International Society of Sports Nutrition*, 18(1), 65.

<https://doi.org/10.1186/s12970-021-00461-1>

⁹⁴ Alizadeh, M., & Kheirouri, S. (2019). Curcumin reduces malondialdehyde and improves antioxidants in humans with diseased conditions: a comprehensive meta-analysis of randomized controlled trials. *Biomedicine & Pharmacotherapy (Taipei)*, 9(4), 23.

<https://doi.org/10.1051/bmecn/2019090423>

⁹⁵ Leonard, S. S., Xia, C., Jiang, B.-H., Stinefelt, B., Klandorf, H., Harris, G. K., & Shi, X. (2003). Resveratrol scavenges reactive oxygen species and effects radical-induced cellular responses. *Biochemical and Biophysical Research Communications*, 309(4), 1017–1026. <https://doi.org/10.1016/j.bbrc.2003.08.105>

Progression of PCS and risk of CTE^{96 97 98}

The transition from Post-Concussion Syndrome (PCS) to Chronic Traumatic Encephalopathy (CTE) is not universally observed, but both conditions involve disruptions in brain anatomy, physiology, and biochemical homeostasis.

PCS is considered an acute and sometimes prolonged response to traumatic brain injury (TBI), while CTE is a progressive neurodegenerative disorder resulting from repeated head trauma. This discussion will explore the anatomical, physiological, and biochemical mechanisms underlying the progression from PCS to CTE.

Anatomical Considerations

Post-Concussion Syndrome (PCS):

PCS primarily affects cortical and subcortical structures, involving both grey and white matter:

- **Cortical Involvement:** The frontal and temporal lobes are often affected due to their proximity to the rigid skull base, leading to contusions, microhemorrhages, and impairments in executive function, mood regulation, and memory.
- **White Matter Disruptions:** Diffuse axonal injury (DAI) is common in PCS, particularly in the corpus callosum, internal capsule, and brainstem, disrupting communication between brain regions.
- **Vascular Disruption:** Damage to small blood vessels and capillaries leads to blood-brain barrier (BBB) dysfunction, increasing neuroinflammation (Blennow et al., 2012; Smith et al., 2013).

Chronic Traumatic Encephalopathy (CTE):

CTE features progressive atrophy in key brain regions:

- **Cortical and Subcortical Atrophy:** Advanced stages show shrinkage in the medial temporal lobes, hippocampus, amygdala, and cerebral cortex.
- **Tauopathy Localization:** Pathological tau protein accumulates starting in the depths of the sulci in the frontal and temporal lobes, spreading to the diencephalon, brainstem, and cerebellum.
- **Ventricular Enlargement:** Brain atrophy leads to dilation of the lateral and third ventricles, increasing cerebrospinal fluid volume and altering intracranial pressure (McKee et al., 2013).

⁹⁶ Blennow, K., Hardy, J., & Zetterberg, H. (2012). The neuropathology and neurobiology of traumatic brain injury. *Neuron*, 76(5), 886–899. <https://doi.org/10.1016/j.neuron.2012.11.021>

⁹⁷ McKee, A. C., Stern, R. A., Nowinski, C. J., Stein, T. D., Alvarez, V. E., Daneshvar, D. H., ... & Cantu, R. C. (2013). The spectrum of disease in chronic traumatic encephalopathy. *Brain*, 136(1), 43–64. <https://doi.org/10.1093/brain/aws307>

⁹⁸ Smith, D. H., Johnson, V. E., & Stewart, W. (2013). Chronic neuropathologies of single and repetitive TBI: Substrates of dementia? *Nature Reviews Neurology*, 9(4), 211–221. <https://doi.org/10.1038/nrneurol.2013.29>

Physiological Considerations

PCS Pathophysiology:

PCS results from a cascade of neurometabolic dysfunction following the initial injury:

- **Cerebral Energy Crisis:** An initial hyperacute phase of neuronal depolarization causes excessive glutamate release, excitotoxicity, impaired mitochondrial ATP production, and disrupted intracellular calcium homeostasis.
- **Blood-Brain Barrier Dysfunction:** Mechanical injury disrupts the BBB, allowing immune cell infiltration, microglial activation, and inflammatory cytokine release (IL-1 β , IL-6, TNF- α), promoting neuroinflammation.
- **Autonomic Dysfunction:** Impairments in the vagus nerve and brainstem autonomic centers cause symptoms such as dizziness, headaches, and blood pressure instability (Blennow et al., 2012; Smith et al., 2013).

CTE Pathophysiology:

CTE progresses through chronic neuroinflammation, tauopathy, and neurodegeneration:

- **Tau Hyperphosphorylation:** Repeated TBI causes excessive tau phosphorylation and neurofibrillary tangles in neurons and glia, disrupting microtubule integrity and axonal transport.
- **Chronic Neuroinflammation:** Persistent activation of microglia and astrocytes maintains a pro-inflammatory state with elevated cytokines (TNF- α , IL-6, IL-18), promoting oxidative stress and neuronal apoptosis.
- **Amyloid Deposition:** Unlike Alzheimer's disease, β -amyloid plaques are not always present in CTE but can appear in some cortical and perivascular regions (McKee et al., 2013; Smith et al., 2013).

Biochemical Considerations

PCS Biochemistry:

The immediate post-concussion period features neurometabolic changes:

- **Mitochondrial Dysfunction:** Impaired ATP production results from increased mitochondrial Ca $^{2+}$ influx, causing energy deficits.
- **Oxidative Stress:** Elevated reactive oxygen species (ROS) cause lipid peroxidation, DNA damage, and neuronal apoptosis.
- **Neurotransmitter Imbalance:** Glutamate excitotoxicity overactivates NMDA receptors, while serotonin (5-HT) dysregulation contributes to mood instability (Blennow et al., 2012).

CTE Biochemistry:

Chronic alterations in protein metabolism, oxidative stress, and neurotransmitter function characterize CTE:

- **Pathological Tau Accumulation:** Hyperphosphorylated tau disrupts axonal function, leading to synaptic failure.
- **Persistent Oxidative Stress:** Chronic oxidative damage exacerbates lipid peroxidation and protein misfolding.
- **Cholinergic Deficiency:** Reduced acetylcholine levels in later stages correlate with cognitive decline, similar to Alzheimer's disease (McKee et al., 2013).

CTE Biochemistry:

CTE results from chronic alterations in protein metabolism, oxidative stress, and neurotransmitter function:

1. **Pathological Tau Accumulation:** Hyperphosphorylated tau disrupts axonal function, leading to neuronal dysfunction and eventual synaptic failure.
2. **Persistent Oxidative Stress:** Chronic oxidative damage exacerbates lipid peroxidation and protein misfolding, worsening neurodegeneration.
3. **Cholinergic Deficiency:** Later stages of CTE show reduced acetylcholine (ACh) levels, similar to Alzheimer's disease, leading to cognitive decline.

Conclusion: PCS to CTE Progression

PCS and CTE share overlapping but distinct pathophysiological mechanisms. **PCS is an acute, potentially reversible dysfunction**, while **CTE is a progressive, irreversible neurodegenerative disorder**. The key factors driving the transition from PCS to CTE include:

1. **Repeated Head Trauma:** The most significant risk factor for CTE is repeated concussions or subconcussive blows.
2. **Chronic Neuroinflammation:** Prolonged activation of microglia and cytokine production sustains a pro-inflammatory environment that promotes tauopathy.
3. **Failure of Neuronal Repair Mechanisms:** Inadequate resolution of oxidative stress and mitochondrial dysfunction predisposes the brain to neurodegeneration.

Indications of progression:

1. Worsening Cognitive Symptoms

- **Memory Impairment:** Increasing difficulty with short-term and long-term memory, forgetfulness, and disorientation.
- **Cognitive Decline:** Difficulty with problem-solving, executive functions (planning, organizing), and processing information. Tasks that were once easy become more challenging.
- **Attention Issues:** Increased difficulty focusing, staying on task, and multitasking.

2. Behavioral and Emotional Changes

- **Mood Swings:** Heightened irritability, depression, or rapid shifts in mood that worsen over time.
- **Aggression and Impulsivity:** Increased impulsive behavior, irritability, or aggression, often without an obvious trigger.
- **Apathy:** Loss of interest in activities once enjoyed, social withdrawal, and general emotional numbness.
- **Depression and Anxiety:** Feelings of hopelessness, sadness, and nervousness that become more persistent and severe.
- **Suicidal Thoughts or Behaviors:** In advanced stages, individuals with CTE may experience suicidal ideation or behaviors, often linked to depression and mood instability.

3. Motor Symptoms

- **Parkinsonism:** As CTE progresses, motor issues such as tremors, stiffness, difficulty walking, and slowness of movement may develop. This is similar to Parkinson's disease.
- **Balance and Coordination Issues:** Difficulty walking, maintaining balance, and performing tasks that require fine motor control (e.g., writing, buttoning clothes).
- **Dysphagia:** Difficulty swallowing due to motor control issues.

4. Progressive Brain Dysfunction

- **Confusion and Disorientation:** Increasing episodes of confusion, forgetfulness, and disorientation about time, place, or person.
- **Severe Cognitive Impairment:** In later stages, the individual may develop dementia-like symptoms, unable to care for themselves or perform daily activities without assistance.

5. Increased Sensitivity to Stress or Stimuli

- **Heightened Sensitivity:** The individual may become more sensitive to environmental stimuli such as noise, light, or stress. This can lead to heightened emotional responses or panic attacks.

6. Social and Occupational Decline

- **Loss of Occupational Functioning:** As cognitive and emotional difficulties increase, individuals may struggle to maintain their professional responsibilities or find it difficult to return to work after a concussion.
- **Relationship Strain:** The behavioral changes (aggression, apathy, mood swings) often put a strain on personal and family relationships, which can further contribute to isolation and mental health challenges.

Understanding these mechanisms is critical for developing preventive strategies and therapeutic interventions to mitigate long-term neurological damage following concussions.

Activity: PCS Case Study and Management Plan

Objective:

To assess your ability to evaluate, manage, and create a long-term care plan for a patient with Post-Concussion Syndrome (PCS). You will apply symptom management strategies, conduct cognitive screenings, and develop a return-to-work or return-to-school plan based on your assessment.

Case Study:

Patient: Emily, a 23-year-old female, sustained a concussion 6 weeks ago during a soccer match. She reports persistent symptoms of headaches, dizziness, difficulty concentrating, and sensitivity to light and noise. She has not returned to work or school due to her symptoms, and she's unsure of how to move forward with her recovery.

- Current symptoms:
 - Persistent headaches (rated 6/10)
 - Fatigue and dizziness after minimal activity
 - Difficulty concentrating at work and in class
 - Sensitivity to bright lights and loud noises
- Current status:
 - Emily has seen a doctor and was diagnosed with PCS after 4 weeks of persistent symptoms.
 - She has not participated in physical activity since the concussion.
 - Her doctor advised a gradual return to school/work but left the specifics to be determined.

Assessment Criteria:

- Thoroughness and accuracy of symptom assessment and cognitive screening.
- Appropriateness and safety of the symptom management plan.
- Realistic and structured approach to return-to-work/school planning.
- Identification of red/yellow/green flags and timely referral pathways.
- Reflection on the decision-making process and areas for improvement.

1. Complete the Following Tasks:

- Step 1: Conduct a thorough PCS symptom assessment by evaluating the patient's symptoms (e.g., headaches, dizziness, cognitive difficulties, sensitivity to light/noise).

- Step 2: Perform a cognitive screening (e.g., Mini-Mental State Examination or another suitable cognitive test).

- Step 3: Develop a symptom management strategy, including rest, pacing, and graded activity.

- Step 4: Create a return-to-work or return-to-school plan that includes any necessary adjustments (e.g., modified workload, gradual re-entry, environmental changes).

- Step 5: Identify red/yellow/green flags for complications that may require further intervention or referral to a specialist.

- Step 6: Reflect on your approach and identify any areas for improvement based on the patient's response to the plan and your ability to adjust as needed.

Module 4 Summary: Post-Concussion Syndrome and Long-Term Management

Learning Objectives Achieved ✓

- Differentiate post-concussion syndrome from typical concussion recovery patterns
 - Evaluate long-term symptom trajectories and their psychosocial impact
 - Develop tailored multidisciplinary management plans for PCS
 - Identify signs of CTE and formulate appropriate referral pathways
-

Post-Concussion Syndrome (PCS) Definition

PCS represents a collection of symptoms persisting beyond the usual recovery period after concussion/mTBI. While most symptoms resolve in days to weeks, PCS can persist for months or years, requiring specialized long-term management approaches.

Diagnostic Criteria:

- Symptoms persist ≥3 months post-concussion
- Presence of ≥3 symptoms across cognitive, physical, and emotional clusters
- Other causes excluded through clinical assessment

Pathophysiology of PCS

Multiple interacting mechanisms contribute to persistent symptoms:

Neurochemical Disruption

- Altered neurotransmitters (glutamate, serotonin, dopamine)
- Results in cognitive problems, mood disturbances, headaches

Metabolic Dysfunction

- Increased brain energy demands with reduced blood flow
- Manifests as fatigue, concentration difficulties, headaches

Neuroinflammation

- Activated microglia and cytokine release
- Contributing to persistent cognitive and mood symptoms

Structural Changes

- Axonal injury and white matter alterations
- Linked to ongoing cognitive and motor deficits

Clinical Presentation

Symptom Domains

Cognitive Symptoms:

- Difficulty concentrating and short-term memory impairment
- Mental fog and slowed processing/reaction times

Physical Symptoms:

- Headaches (tension or migraine-like patterns)
- Dizziness, balance problems, vertigo
- Light/sound sensitivity, fatigue, sleep disturbances

Emotional/Behavioral Symptoms:

- Irritability, anxiety, depression, mood swings
 - Social withdrawal and behavioral changes
-

Management Strategies

Pharmacological Interventions

Symptom-targeted approach:

- **Headaches:** NSAIDs, acetaminophen, triptans, anticonvulsants, beta-blockers
- **Mood symptoms:** SSRIs (e.g., sertraline) for depression/anxiety

Non-Pharmacological Approaches

Multidisciplinary rehabilitation is fundamental:

Cognitive Behavioral Therapy (CBT)

- Addresses anxiety, depression, sleep issues
- Provides coping strategies and symptom management

Physical Interventions

- Vestibular rehabilitation for dizziness/balance
- Physical therapy for neck pain/musculoskeletal symptoms
- Subthreshold aerobic exercise for neuroplasticity

Education & Lifestyle

- PCS education, pacing strategies, sleep hygiene
- Gradual activity progression with monitoring

Emerging Therapies (Research Phase)

- **Vagus Nerve Stimulation (VNS):** Modulates pain pathways
 - **Neurofeedback:** Brain wave regulation training
 - **Transcranial Magnetic Stimulation (TMS):** FDA-approved for acute migraine
-

Nutritional Support

Evidence-Based Nutraceuticals

 **Omega-3 (DHA/EPA):** Anti-inflammatory, reduces neuroinflammation  **Creatine:** Enhances ATP production, mitochondrial support  **Magnesium:** NMDA modulation, reduces excitotoxicity  **Vitamin D:** Regulates inflammation, may shorten recovery  **Curcumin:** Antioxidant properties, reduces oxidative damage  **Resveratrol:** Supports mitochondrial function, reduces ROS

PCS vs. CTE Progression

Key Differences

PCS: Temporary response with cortical/subcortical involvement **CTE:** Progressive neurodegenerative disease from repeated trauma

Warning Signs of CTE Progression

Red Flags:

- Progressive cognitive decline (memory, executive function)
 - Severe behavioral changes (aggression, suicidal ideation)
 - Motor symptoms (Parkinsonism-like features)
 - Severe brain dysfunction (confusion, dementia-like symptoms)
 - Social/occupational deterioration
-

Clinical Application Framework

Assessment Protocol

1. **Comprehensive symptom evaluation** across all domains
2. **Cognitive screening** using validated tools (SCAT6, MMSE-style)
3. **Risk factor identification** (age, migraine history, mental health)
4. **Psychosocial impact assessment**

Management Planning

1. **Symptom-specific interventions** with multimodal approach
2. **Graded return-to-activity** protocols (work/school/sport)
3. **Environmental modifications** and accommodation strategies
4. **Regular monitoring** with objective outcome measures

Referral Pathways

Specialized Services:

- Neuropsychological assessment for cognitive concerns
 - Vestibular rehabilitation for balance/dizziness
 - Mental health services for psychological complications
 - Rehabilitation medicine for complex cases
-

Key Takeaways for Practice

Early Recognition is Critical: Identifying PCS early allows for timely intervention and better outcomes.

Holistic Approach: Successful management requires addressing physical, cognitive, and emotional domains simultaneously.

Individualized Care: Treatment plans must be tailored to each patient's specific symptom profile and functional needs.

Prevention Focus: Avoiding repeated head trauma is essential to prevent progression to CTE.

Multidisciplinary Collaboration: Optimal outcomes require coordinated care across multiple specialties.

"Understanding the underlying mechanisms of PCS guides evidence-based, holistic treatment combining targeted medications, rehabilitation therapies, education, and lifestyle modifications."

APPENDIX: Clinical Tools & Quick Reference

PCS Assessment Checklist

Cognitive Domain Assessment

- [] Concentration difficulties
- [] Short-term memory problems
- [] Mental fog/clouded thinking
- [] Slowed processing speed
- [] Attention/focus issues

Physical Domain Assessment

- [] Headache frequency/severity (rate 1-10)
- [] Dizziness/balance problems
- [] Light sensitivity (photophobia)
- [] Sound sensitivity (phonophobia)
- [] Fatigue levels
- [] Sleep disturbances

Emotional/Behavioral Domain Assessment

- [] Irritability/mood changes
- [] Anxiety symptoms
- [] Depression screening
- [] Social withdrawal
- [] Behavioral changes

Red/Yellow/Green Flag System

RED FLAGS (Immediate Referral)

- Progressive cognitive decline
- Suicidal ideation
- Severe behavioral changes
- Motor symptoms (tremor, rigidity)
- Confusion/disorientation

YELLOW FLAGS (Monitor Closely)

- Persistent symptoms >3 months
- Functional decline at work/school / Social isolation
- Moderate mood changes
- Sleep disturbances

GREEN FLAGS (Positive Indicators)

- Symptom improvement over time
- Good treatment compliance
- Strong support system
- Motivation for recovery
- Functional improvements

Graded Return-to-Activity Template

Week 1: Foundation Phase

- **Activity Level:** 25% normal capacity
- **Duration:** 2-4 hours daily with frequent breaks
- **Environment:** Controlled, low-stimulus settings
- **Monitoring:** Daily symptom tracking

Week 2: Building Phase

- **Activity Level:** 50% normal capacity
- **Duration:** 4-6 hours daily with scheduled breaks
- **Environment:** Gradual introduction of normal stimuli
- **Monitoring:** Bi-weekly assessment

Week 3+: Integration Phase

- **Activity Level:** 75-100% normal capacity
- **Duration:** Approaching full schedule
- **Environment:** Normal conditions with accommodations
- **Monitoring:** Weekly follow-up

Medication Quick Reference

Headache Management

- **Acute:** NSAIDs, acetaminophen, triptans
- **Preventive:** Beta-blockers, anticonvulsants
- **Consider:** Magnesium supplementation

Mood/Anxiety

- **First-line:** SSRIs (sertraline, escitalopram)
- **Adjunct:** CBT, mindfulness training
- **Monitor:** Suicidal ideation, side effects

Sleep Disturbances

- **Non-pharmacological:** Sleep hygiene, CBT-I
- **Pharmacological:** Melatonin, trazodone (short-term)

Emergency Contacts & Referrals

- **Neuropsychology:** Cognitive assessment/rehabilitation
- **Vestibular Therapy:** Balance and dizziness management
- **Mental Health:** Psychological support and therapy
- **Rehabilitation Medicine:** Complex case management
- **Neurology:** Progressive symptoms or CTE concerns

Patient Education Points

1. PCS is a real medical condition with biological basis
2. Symptoms typically improve with proper management
3. Pacing activities prevents symptom exacerbation
4. Recovery is often non-linear with good and bad days
5. Team approach maximizes recovery potential

Module 5: The Multidisciplinary Approach in Concussion Care

Team-Based Recovery: Building Interdisciplinary Concussion Pathways

Collaborative care planning across physio, neuropsychology, general practice, OT, and more.

- **Learning Objectives:**

- Cultural safety in concussion management
- Describe the roles of healthcare professionals and differentiate when to refer to each discipline in concussion care.
- Develop a multidisciplinary management plan for a post-concussion patient, including appropriate referral pathways and role-based interventions from GP, physio, OT, and neuropsychology.
- Rehabilitation strategies for post-concussion syndrome.
- Management steps: physical rest, cognitive rest, and monitoring.
- Treatment and rehabilitation approaches.
- Demonstrate collaborative clinical reasoning and staged multidisciplinary planning in the context of real-world access limitations.

Activities & Clinical Skill Application

This module includes scenario-based simulations, clinical reasoning checkpoints, and applied practice tasks focused on implementing a multidisciplinary approach to concussion management across recovery phases. Learners will demonstrate the ability to assess and monitor patient progress, collaborate with healthcare professionals from different disciplines, and adapt care strategies based on evolving clinical presentations. Key skills include conducting multidisciplinary case assessments, applying evidence-based protocols, and fostering interprofessional communication. Activities are observed and trainer-marked to ensure clinical safety, competence, and ethical decision-making in managing concussion recovery in various settings, such as sports, education, and rehabilitation.

Learning outcomes

1. Describe the roles of different professionals in concussion care (GPs, physiotherapists, neuropsychologists, occupational therapists, educators).
2. Coordinate care pathways using a team-based approach.
3. Refer patients appropriately based on clinical findings and recovery trajectory.
4. Communicate effectively with patients, families, and other stakeholders.
5. Collaborate with schools and employers to implement cognitive and environmental accommodations.

Multidisciplinary Approach in Concussion Management: Legal Framework in Australia

This protocol outlines the roles of General Practitioners (GPs), sports doctors, and allied health professionals (including physiotherapists, occupational therapists, osteopaths, and chiropractors) in concussion management, with a focus on the legal responsibilities and scopes of practice within Australian healthcare regulations. This includes who is legally allowed to diagnose, refer for imaging, write doctors' notes, and sign return-to-activity documents.



Multidisciplinary Roles in Concussion Care

Effective concussion management requires coordinated care across different phases of recovery. Below is a breakdown of practitioner responsibilities across the **acute**, **sub-acute**, **recovery**, and **full recovery** phases.

Treatment and rehabilitation

Evidence-Based Treatment and Rehabilitation for Concussion Patients in a Multi-Disciplinary Sports Medicine Clinic

Concussion management requires a comprehensive, multi-disciplinary approach involving initial symptom management, progressive return to activity, and targeted interventions such as manual therapy and exercise rehabilitation. Evidence-based guidelines emphasize individualized care based on symptom presentation and recovery trajectory.⁹⁹

⁹⁹ McCrory, P., Meeuwisse, W., Dvorak, J., Aubry, M., Bailes, J., Broglio, S., Cantu, R. C., Cassidy, D., Echemendia, R. J., Castellani, R. J., Davis, G. A., Ellenbogen, R., Emery, C., Engebretsen, L., Feddermann-Demont, N., Giza, C. C., Guskiewicz, K. M., Herring, S., Iverson, G. L., ... Vos, P. E. (2017). Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016. *British Journal of Sports Medicine*, 51(11), 838-847. <https://doi.org/10.1136/bjsports-2017-097699>

Phase 1: Acute Symptom Management (0-48 hours)

Goals: Reduce symptom burden, prevent worsening of the injury, and provide education on recovery.

- Cognitive and physical rest: The patient should avoid strenuous activity, screen exposure, and cognitive overload.¹⁰⁰
 - Symptom monitoring: The SCAT/SCOAT6 or similar assessment tools can track symptoms and help guide activity restrictions.¹⁰¹
 - Manual Therapy (if indicated): Gentle soft tissue techniques for cervical dysfunction and vestibular-ocular symptoms can be beneficial.¹⁰²
-
-
-

Phase 2: Gradual Symptom-Guided Activity (48 hours - 1 week)

Goals: Controlled reintroduction of cognitive and physical activities within symptom tolerance.

- Aerobic exercise: Light, symptom-limited activity (e.g., stationary cycling at 50-60% HR max) improves cerebral autoregulation and recovery speed (Leddy et al., 2019).
 - Vestibular and Oculomotor Rehabilitation: Exercises targeting gaze stabilization, saccades, and convergence are prescribed if dizziness, blurred vision, or balance deficits persist.¹⁰³
 - Manual Therapy: If persistent headaches or neck pain are present, cervicogenic headache treatment such as joint mobilizations and soft tissue release may be applied (Reneker et al., 2018).
-
-

¹⁰⁰ Leddy, J. J., Haider, M. N., Ellis, M. J., & Willer, B. (2019). Exercise is medicine for concussion. *Sports Health*, 11(3), 210-217. <https://doi.org/10.1177/1941738119838413>

¹⁰¹ Echemendia, R. J., Meehan, W. P., McCrea, M. A., Broglie, S. P., Kutcher, J. S., & Giza, C. C. (2023). The SCAT/SCOAT6: A standardized approach to assessing concussions. *British Journal of Sports Medicine*, 57(6), 369-376. <https://doi.org/10.1136/bjsports-2022-106391>

¹⁰² Reneker, J. C., Hassen, A., Phillips, R. S., & McLeod, T. C. V. (2018). The effectiveness of manual therapy in the management of musculoskeletal disorders and concussion: A systematic review. *Sports Medicine*, 48(2), 453-467. <https://doi.org/10.1007/s40279-017-0809-0>

¹⁰³ Alsalaheen, B., Mucha, A., Morris, L. O., Whitney, S. L., Furman, J. M., Camiolo-Reddy, C. E., Collins, M. W., & Kontos, A. P. (2013). Vestibular rehabilitation for dizziness and balance disorders after concussion. *Journal of Neurologic Physical Therapy*, 37(2), 75-80. <https://doi.org/10.1097/NPT.0b013e31828d9939>

Phase 3: Structured Exercise Progression (1-3 weeks post-injury)

Goals: Increase cardiovascular tolerance, address movement impairments, and restore full vestibular function.

- Exercise Progression: Gradual increase in intensity, incorporating sport-specific movement patterns (Leddy et al., 2019).
 - Neuromuscular Control & Balance Training: Dual-task training, dynamic balance exercises, and reaction drills help reintegrate cognitive-motor function.
 - Vestibular & Oculomotor Rehab: More advanced exercises incorporating head movement, gaze shifts, and visual tracking (Alsalaheen et al., 2013).
 - Manual Therapy: Targeted cervical spine mobilizations and postural correction (Reneker et al., 2018).
-
-

Phase 4: Return to Play (3+ weeks, based on symptoms and assessment results)

Goals: Full neurological recovery, confidence in movement, and sport-specific reintegration.

- Return-to-play protocol: A stepwise progression following international guidelines (McCrory et al., 2017), ensuring no symptom recurrence with increasing intensity.
 - High-level cognitive-motor integration: Agility drills, reactive movements, and sport-specific decision-making tasks.¹⁰⁴
 - Clearance Testing: A combination of exertion testing, vestibular function assessment, and neurocognitive testing is used to confirm readiness for full return (Echemendia et al., 2023).
-
-

Monitoring & Measured Progression

- Symptom scales (SCAT/SCOAT6, Post-Concussion Symptom Scale)
- Heart rate thresholds for exertional testing
- Balance error scoring system (BESS) for neuromotor control

Vestibular/Ocular Motor Screening (VOMS) for functional improvements

¹⁰⁴ Howell, D. R., Osterrieg, L. R., Chou, L. S., & Van Donkelaar, P. (2018). Dual-task gait performance in female and male adolescents following concussion. *Gait & Posture*, 61, 143-147. <https://doi.org/10.1016/j.gaitpost.2018.01.020>

Clinician Roles

1. Acute Phase (0–48 hours)

General Practitioner (GP)

- **Role:** First-line assessment, diagnosis, and initial management.
- **Key Actions:**
 - Diagnose concussion using clinical tools (e.g., SCAT/SCOAT6).
 - Order imaging (CT/MRI) if red flags are present (e.g., suspected brain bleed).
 - Issue medical certificates for rest from work/school.
 - Provide early symptom management and advice.
 - Sign initial return-to-activity (RTA) documents (excluding sport-specific RTP).

Sports Doctor

- **Role:** Acute sport-related concussion diagnosis and return-to-play planning.
 - **Key Actions:**
 - Confirm diagnosis in athletic settings.
 - Order imaging if severe injury is suspected.
 - Issue sport-specific rest notes and guide early management.
 - Begin return-to-play (RTP) planning and document clearance once safe.
-

2. Sub-Acute Phase (48 hours – 1 week)

General Practitioner (GP)

- **Role:** Symptom monitoring, secondary assessments, and coordination of referrals.
- **Key Actions:**
 - Monitor symptom progression.
 - Refer for imaging if new symptoms arise.
 - Continue issuing rest/work modifications.
 - Coordinate return-to-school/work plans (non-sport).

Allied Health Professionals (*Physiotherapists, Osteopaths, OTs, Chiropractors*)

- **Role:** Early rehabilitation and symptom-specific management.
- **Key Actions:**
 - Assess balance, neck dysfunction, and vestibular symptoms.
 - Provide targeted rehabilitation and symptom education.
 - Refer to GP/sports doctor for imaging or complications.
 - Submit reports but cannot write official medical certificates or sign RTA forms.

3. Recovery Phase (1–4 weeks)

General Practitioner (GP)

- **Role:** Ongoing care, PCS screening, and referral to specialists.
- **Key Actions:**
 - Identify prolonged symptoms and screen for PCS.
 - Refer to neurologists or neuropsychologists as needed.
 - Continue issuing medical certificates.
 - Clear patient for low-risk daily activity when symptom-free.

Allied Health Professionals

- **Role:** Support recovery and functional rehabilitation.
- **Key Actions:**
 - Continue vestibular, cervical, and cognitive rehab.
 - Monitor readiness for physical or academic reintegration.
 - Recommend adaptations but cannot approve formal clearance.

4. Full Recovery Phase (4+ weeks)

General Practitioner (GP)

- **Role:** Final medical clearance for non-sporting activities.
- **Key Actions:**
 - Confirm complete symptom resolution.
 - Finalize medical notes and return-to-work/school forms.
 - Refer to specialist if any new issues emerge.

Sports Doctor

- **Role:** Final clearance for athletes returning to sport.
- **Key Actions:**
 - Ensure full recovery through graded RTP protocol.
 - Sign off on RTP forms.
 - Provide return-to-training and competition guidance.

Allied Health Professionals

- **Role:** Optimize performance and prevent re-injury.
- **Key Actions:**
 - Finalize rehab and functional testing.
 - Identify lingering impairments and refer back if needed.
 - Communicate progress but not authorized to sign off on official documents.

Multidisciplinary Referral Flow Example

1. **Initial GP assessment** → suspect concussion
2. → Refer to Physio for balance/vestibular rehab
3. → Refer to Psychologist for mood/sleep disturbance
4. → Notify School Liaison for academic supports
5. → Neurology referral if symptoms >4 weeks or red flags

💡 Ensure communication between all professionals through shared notes or a case coordinator (e.g., allied health team leader).

Rehab flow chart



Cultural Safety in Concussion Management

Culturally safe practice is essential in concussion management. Practitioners must:

- Acknowledge historical trauma when working with Aboriginal and Torres Strait Islander peoples.
- Engage in shared decision-making with patients and families.
- Be aware of culturally-specific expressions of distress (e.g., silence, withdrawal).
- Use interpreters where language barriers exist.

Role Play: “Referral, Reality, and Responsibility”

Duration: 12–15 minutes

Module: Multidisciplinary Approach in Concussion Management

Format: Pairs or Triads (2–3 participants)

Learning Goal: Apply multidisciplinary knowledge to a case where practitioner access, funding, and patient readiness are limited.

Scenario:

You're a clinician (e.g. osteopath, physio, EP, OT, GP) seeing a 32-year-old female schoolteacher, 10 days post-MVA with a confirmed concussion.

She presents with:

- Intermittent headaches, visual tracking issues
- Anxiety about returning to the classroom
- Light sensitivity and mental fatigue after ~30 mins of screen use
- Poor sleep and emotional fragility

She's self-referred. She works full time, has limited private health, and can only afford to see 1–2 clinicians over the next month.

Roles:

Each participant plays one clinician from a relevant scope (GP, osteo, physio, EP, OT, psychologist, etc.).

 **Task:**

You have 8 minutes to discuss as a small group or pair:

1. Triage the symptoms – What’s your working hypothesis about what’s causing the ongoing symptoms?
 2. Prioritize care – Given budget and time constraints, who should she see *first*? What outcome do you expect from that consult?
 3. Plan staged referrals – If she improves, who’s next? If not, how would you communicate that to her?
 4. Draft a short interprofessional email – (2 sentences max) to another practitioner you wish to refer her to. Include:
 - Clinical concern
 - Why this referral is important now
 - What you need from them
-

 **Group Debrief (4–6 mins):**

- What was hardest to decide?
-
-
-
-
-
-
-
-

- Did anyone feel their profession was under-prioritized? Why?
-
-
-
-
-
-
-
-

- How can clinicians better collaborate *when they don't share a practice?*

- How do you explain referral urgency and value to a hesitant or overwhelmed patient?

Upper cervical and suboccipital management

Emerging research indicates that manual therapies targeting the upper cervical and suboccipital regions can be beneficial in the rehabilitation of concussion patients, particularly those experiencing persistent symptoms such as headaches, dizziness, and neck pain.

- Reneker et al. (2021) examined the outcomes of 38 patients with post-concussion symptoms who underwent a rehabilitation protocol focusing on early cervical manual therapy and canalith repositioning (Epley), followed by integrated vision and vestibular therapy.¹⁰⁵
 - The study reported significant improvements in both clinical assessments and patient-reported outcomes, suggesting that addressing cervical dysfunction early in the rehabilitation process may enhance recovery (Reneker et al., 2021)
-

- De Iovanna et al. (2021) evaluated various manual therapy techniques for cervicogenic headaches, which are often associated with concussion.¹⁰⁶
 - The review found that spinal manipulative therapy, Mulligan's Sustained Natural Apophyseal Glides, and muscle techniques targeting the cervical region were effective in reducing headache intensity and frequency.
-

- Marshall et al. (2015) discussed the role of the cervical spine in post-concussion syndrome and presented cases where patients received both active rehabilitation and passive manual therapy of the cervical spine.¹⁰⁷
 - The management approach resulted in significant symptom reduction, highlighting the potential benefits of incorporating manual therapy into concussion rehabilitation
-

- Dry needling of the suboccipital muscles has demonstrated clinical benefits in reducing symptoms commonly associated with post-concussion syndrome, such as cervicogenic headaches, dizziness, and reduced cervical mobility. These symptoms are frequently linked to upper cervical dysfunction, particularly at the C0–C2 levels. Multiple studies and case series have shown that targeting myofascial trigger points in the suboccipital region can decrease symptom severity, improve range of motion, and restore

¹⁰⁵ Reneker, J. C., Phillips, R. S., Leddy, J. J., & Stearns, S. C. (2021). Sequencing and integration of cervical manual therapy and vestibulo-oculomotor therapy for concussion symptoms: Retrospective analysis. *International Journal of Sports Physical Therapy*, 16(1), 79–89. https://doi.org/10.26603/001c_18638IJSPORT+1PMC+1

¹⁰⁶ De Iovanna, N., Castaldo, M., & de Iovanna, N. (2021). Effectiveness of manual therapy in the treatment of cervicogenic headache: A systematic review. *Headache: The Journal of Head and Face Pain*, 61(3), 441–456.

<https://doi.org/10.1111/head.14278> American Headache Society

¹⁰⁷ Marshall, C. M., Vernon, H., Leddy, J. J., & Baldwin, B. A. (2015). The role of the cervical spine in post-concussion syndrome. *The Physician and Sportsmedicine*, 43(3), 274–284. <https://doi.org/10.1080/00913847.2015.1074533> Complete Concussions

sensorimotor control, all of which may aid in concussion recovery when cervical involvement is suspected.¹⁰⁸

- While high-level evidence directly assessing dry needling in concussion-specific populations remains limited, existing research from cervicogenic headache and dizziness populations—conditions with strong clinical overlap with post-concussion symptoms—supports the use of this modality. Case series, pilot studies, and systematic reviews suggest that dry needling can be a valuable adjunct to concussion rehabilitation, particularly when symptoms persist beyond the acute phase and are suspected to be of cervical origin.

At home management¹⁰⁹

The **Montreal Virtual Exertion (MOVE) Protocol** is a graded exertion test designed to assess and guide the return to physical activity in individuals recovering from concussion. Developed to be administered virtually, the MOVE Protocol offers a practical alternative to traditional in-person assessments, facilitating remote monitoring and rehabilitation.

Structure of the MOVE Protocol:

The protocol comprises seven stages, each involving specific bodyweight and plyometric exercises performed for 60 seconds. These stages are designed to incrementally increase cardiovascular demand and assess the individual's tolerance to physical exertion post-concussion. The exercises are selected to progressively challenge the patient's aerobic capacity and neuromuscular control while monitoring for symptom exacerbation.

Implementation and Findings:

The MOVE Protocol was evaluated for safety and feasibility among both healthy children and those with subacute concussion.

The study involved 20 healthy children who completed the protocol virtually via Zoom, and 30 children with subacute concussion who were randomized to either the MOVE Protocol or the Buffalo Concussion Treadmill Test (BCTT).¹¹⁰

The results indicated no adverse events and demonstrated that the MOVE Protocol met all feasibility criteria in both cohorts. Notably, increases in heart rate and perceived exertion were comparable between the MOVE Protocol and the BCTT, suggesting that the MOVE Protocol is a viable alternative for exertional testing in concussion management.

¹⁰⁸ Escaloni, J., Vaccaro, J., Ramey, R., & Garber, M. (2018). Use of Dry Needling in the Diagnosis and Treatment of Cervicogenic Dizziness: A Case Series. *Journal of Manual & Manipulative Therapy*.

¹⁰⁹ Teel, E., Alarie, C., Swaine, B., Cook, N. E., Iverson, G. L., & Gagnon, I. (2023). An at-home, virtually administered graded exertion protocol for use in concussion management: Preliminary evaluation of safety and feasibility for determining clearance to return to high-intensity exercise in healthy youth and children with subacute concussion. *Journal of Neurotrauma*, 40(15-16), 1730–1742. <https://doi.org/10.1089/neu.2022.0370PubMed>

¹¹⁰ Haider, M. N., Leddy, J. J., Wilber, C. G., Viera, K. B., Bezerano, I., Wilkins, K. J., Miecznikowski, J. C., & Willer, B. S. (2019). The predictive capacity of the Buffalo Concussion Treadmill Test after sport-related concussion in adolescents. *Frontiers in Neurology*, 10, 395. <https://doi.org/10.3389/fneur.2019.00395PubMed+1PMC+1>

Clinical Implications:

The MOVE Protocol's virtual compatibility allows healthcare providers to remotely assess a patient's readiness to resume physical activities, making it particularly useful in telehealth settings or when in-person visits are not feasible. Its structured, stepwise approach ensures that patients engage in physical activity without exacerbating concussion symptoms, thereby supporting a safe and individualized return-to-activity plan.

Rehabilitation monitoring

Identifying rehabilitation failures in concussion management is crucial to ensure patients receive appropriate care and support. Rehabilitation failure may be indicated by the persistence or worsening of symptoms, development of new symptoms, or lack of progress in recovery. Key indicators include:

1. **Persistent Post-Concussive Symptoms (PPCS):** Symptoms such as headaches, dizziness, fatigue, irritability, anxiety, depression, and cognitive difficulties that continue beyond the typical recovery period may signal rehabilitation failure. These prolonged symptoms can interfere with daily functioning and quality of life.¹¹¹
2. **Exacerbation with Exertion:** If symptoms that were present early after the injury are exacerbated with physical or cognitive exertion but improve with rest, it may indicate ongoing concussion pathophysiology. Conversely, symptoms that are aggravated by minimal activity and do not respond to rest might reflect psychological factors related to prolonged inactivity and frustration.¹¹²
3. **Development of Secondary Issues:** The emergence of secondary problems such as sleep disturbances, mood disorders, or musculoskeletal pain during rehabilitation can indicate that the current treatment plan is ineffective or incomplete.¹¹³
4. **Failure to Progress in Rehabilitation Phases:** Inability to advance through structured rehabilitation phases, such as graded exertion protocols, due to symptom exacerbation or intolerance suggests a need to reassess and modify the rehabilitation strategy.¹¹⁴
5. **Impact on Daily Activities:** Continued difficulty in resuming work, school, or social activities due to persistent symptoms indicates that rehabilitation goals are not being met, necessitating a reevaluation of the treatment approach (Mayo Clinic, 2023).

Regular monitoring and open communication between patients and healthcare providers are essential to detect these signs early. If rehabilitation failure is identified, it is important to reassess the treatment plan, consider alternative therapies, and address any underlying issues impeding recovery.

¹¹¹ Mayo Clinic. (2023). Persistent post-concussive symptoms (Post-concussion syndrome).
<https://www.mayoclinic.org/diseases-conditions/post-concussion-syndrome/symptoms-causes/syc-20353352>

¹¹² Marshall, S., Bayley, M., McCullagh, S., Velikonja, D., Berrigan, L., Ouchterlony, D., & Weegar, K. (2012). Clinical practice guidelines for mild traumatic brain injury and persistent symptoms. *Canadian Family Physician*, 58(3), 257–267.
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3435903/>

¹¹³ Johns Hopkins Medicine. (n.d.). Rehabilitation after traumatic brain injury.
<https://www.hopkinsmedicine.org/health/treatment-tests-and-therapies/rehabilitation-after-traumatic-brain-injury>

¹¹⁴ Leddy, J. J., Haider, M. N., Ellis, M. J., & Willer, B. S. (2019). Exercise is medicine for concussion. *Current Sports Medicine Reports*, 18(8), 268–275. <https://doi.org/10.1249/JSR.0000000000000616>



Case Study (Discussion-Based): Elijah – A Complex Recovery After School Sports Injury

Background:

Elijah is a 15-year-old Aboriginal student who sustained a concussion during a rugby game at school. He was briefly unconscious and experienced dizziness, headaches, and blurred vision for several hours after the injury. He was taken to the emergency department where a GP confirmed a concussion diagnosis using the SCAT/SCOAT6. No imaging was required at the time.

◆ Phase 1: Acute (Day 0–2)

- Practitioner Involved: General Practitioner (GP)
 - Decisions Made:
 - Medical certificate issued for time off school
 - _____
 - _____
 - Cultural Safety Consideration:
 - _____
-

◆ Phase 2: Sub-Acute (Days 3–7)

- Symptoms: Ongoing dizziness, neck pain, fatigue
 - Referrals initiated:
 - Physiotherapy for: _____
 - Occupational therapy for: _____
 - New Issue Identified:
 - Sleep disruption and irritability
 - Discussion Point:
 - Who else should be involved at this stage and why?
 - How should communication occur between team members?
-

◆ **Phase 3: Recovery (Week 2–3)**

- Improvements Seen: _____
- Remaining Barriers to Full Participation:

- OT Actions: _____
- Physiotherapy Focus: _____
- Referral for psychological support made? (Yes/No – Discuss why or why not)

◆ **Phase 4: Full Recovery (Week 4+)**

- Final clearance by which practitioner? _____
- What tool was used to assess readiness for return to activity?

- Final considerations before return-to-play:
 - _____
 - _____
- What role did the school or family play in this phase?

◆ **Group Questions:**

- Which steps in Elijah’s care required the most collaboration?
- How might cultural misunderstandings impact Elijah’s care plan?
- What are the risks of missing or delaying referrals in this case?

Module 5 Summary: The Multidisciplinary Approach in Concussion Care

Learning Objectives Achieved ✓

- Describe roles of healthcare professionals and differentiate when to refer to each discipline
 - Develop multidisciplinary management plans with appropriate referral pathways
 - Demonstrate collaborative clinical reasoning within real-world access limitations
 - Integrate cultural safety principles in concussion management
-

Legal Framework in Australian Healthcare

Scope of Practice Responsibilities

Medical Practitioners (GPs/Sports Doctors)

- **Diagnosis:** Legal authority to diagnose concussion
- **Imaging:** Can order CT/MRI when clinically indicated
- **Documentation:** Medical certificates, return-to-activity clearances
- **Referrals:** Coordinate specialist and allied health referrals

Allied Health Professionals

- **Assessment:** Comprehensive symptom evaluation within scope
 - **Treatment:** Specialized interventions (physio, OT, psychology)
 - **Monitoring:** Progress tracking and symptom management
 - **Communication:** Report findings to referring practitioner (no official clearance authority)
-

Evidence-Based Phased Management Framework

Phase 1: Acute Symptom Management (0-48 hours)

Primary Goals: Reduce symptoms, prevent worsening, educate patient

Key Actions:

- Cognitive & physical rest (avoid screens, exertion)
- Symptom monitoring via SCAT6/SCoAT6
- Gentle manual therapy if cervical or vestibular symptoms present

Lead Practitioners:

- **GP:** Diagnose, order imaging if needed, medical certificates, initiate RTA plans
- **Sports Doctor:** Confirm diagnosis in athletes, sport-specific rest protocols

Phase 2: Gradual Activity (48 hours - 1 week)

Primary Goals: Controlled return to activity within symptom limits

Key Actions:

- Light aerobic exercise (50-60% HR max)
- Vestibular/oculomotor rehab for persistent dizziness/vision issues
- Manual therapy for headaches or neck pain

Lead Practitioners:

- **GP:** Monitor symptoms, coordinate return-to-school/work plans
- **Allied Health:** Assess balance, neck, vestibular symptoms; educate and treat

Phase 3: Structured Exercise (1-3 weeks)

Primary Goals: Improve fitness, neuromotor control, vestibular recovery

Key Actions:

- Gradual intensity increase, sport-specific movements
- Dual-task and balance training
- Advanced vestibular/oculomotor exercises
- Cervical spine mobilizations and posture correction

Lead Practitioners:

- **GP:** Screen for PCS, refer specialists, clear low-risk activities
- **Allied Health:** Ongoing vestibular, cervical, cognitive rehabilitation

Phase 4: Return to Play (3+ weeks)

Primary Goals: Full recovery and sport reintegration

Key Actions:

- Stepwise RTP protocol (no symptom return at each stage)
- High-level cognitive-motor drills
- Clearance testing (exertion, vestibular, neurocognitive)

Lead Practitioners:

- **GP:** Final work/school clearance, documentation
 - **Sports Doctor:** Final RTP clearance and guidance
 - **Allied Health:** Complete rehabilitation, identify lingering issues
-

MOVE Protocol: A Framework for Concussion Management

The MOVE protocol is a structured approach designed to assess and manage athletes recovering from concussion. It emphasizes a multidisciplinary strategy, integrating physical, cognitive, and emotional evaluations to ensure a comprehensive recovery process.

1. M – Medical Evaluation

The initial step involves a thorough medical assessment by a healthcare professional trained in concussion management. This evaluation includes:

- **History Taking:** Documenting the mechanism of injury, symptom onset, and any previous concussions.
- **Physical Examination:** Assessing for signs such as balance disturbances, visual changes, or neurological deficits.
- **Cognitive Testing:** Utilizing tools like the SCAT6 to evaluate cognitive function and symptom severity.

2. O – Observation

Continuous monitoring is crucial during the acute phase post-injury. Athletes should be observed for:

- **Symptom Progression:** Noting any worsening of symptoms such as headaches, nausea, or confusion.
- **Neurological Deterioration:** Monitoring for signs of increased intracranial pressure or other serious complications.
- **Behavioral Changes:** Being alert to mood swings, irritability, or cognitive impairments.

Regular assessments help determine the appropriate timing for progression through recovery stages.

3. V – Vestibular and Ocular Screening

Given the high incidence of vestibular and ocular symptoms following concussion, specialized assessments are performed to identify dysfunctions:

- **Vestibular Ocular Motor Screening (VOMS)**: Evaluates symptoms related to visual motion sensitivity, convergence, and gaze stability.
- **Balance Error Scoring System (BESS)**: Assesses postural stability under different conditions.
- **Neurocognitive Testing**: Tools like ImPACT or CogSport measure cognitive functions such as memory, attention, and processing speed.

Identifying deficits in these areas allows for targeted rehabilitation strategies.

4. E – Exertion and Return-to-Play Protocol

Once an athlete is symptom-free at rest and has normal results on vestibular, ocular, and cognitive assessments, a graded exertion protocol is initiated:

- **Stage 1**: Light aerobic exercise (e.g., stationary cycling).
- **Stage 2**: Sport-specific exercise (e.g., running drills).
- **Stage 3**: Non-contact training drills.
- **Stage 4**: Full-contact practice (if applicable).
- **Stage 5**: Return to competition.

Each stage lasts 24 hours, and progression is contingent upon the absence of symptoms. If symptoms recur, the athlete returns to the previous asymptomatic stage.

Summary

The MOVE protocol offers a systematic approach to concussion management, emphasizing:

- **Comprehensive Assessment**: Incorporating medical, vestibular, ocular, and cognitive evaluations.
- **Symptom Monitoring**: Ensuring continuous observation for any changes.
- **Targeted Rehabilitation**: Addressing specific deficits identified during assessments.
- **Structured Return-to-Play**: Following a graded exertion protocol to safely reintegrate athletes into their sport.

Multidisciplinary Team Roles

General Practitioner (GP)

Primary Responsibilities:

- Initial diagnosis and medical management
- Ordering investigations (imaging, specialist referrals)
- Medical certificates and return-to-activity documentation
- Coordinating care across phases
- Managing comorbidities and complications

Allied clinician

Specialized Focus:

- Cervical spine assessment and treatment
- Vestibular rehabilitation for dizziness/balance
- Exercise prescription and progression
- Manual therapy for headaches and neck pain
- Movement re-education and biomechanics

Neuropsychologist

Cognitive Specialization:

- Comprehensive cognitive assessment
- Treatment of cognitive symptoms
- Return-to-school/work cognitive planning
- Psychological support for mood/anxiety
- Family and educator consultation

Occupational Therapist (OT)

Functional Focus:

- Daily living skills assessment
- Workplace/school accommodation planning
- Cognitive rehabilitation strategies
- Environmental modification recommendations
- Fatigue management and pacing strategies

Sports Medicine Physician

Athletic Specialization:

- Sport-specific injury management
 - Advanced RTP protocols
 - Complex case management
 - Performance optimization post-recovery
 - Preventive strategies for athletes
-

Monitoring Tools & Assessments

Standardized Assessment Instruments

- **SCAT6/SCOAT6:** Comprehensive symptom evaluation
- **Post-Concussion Symptom Scale:** Symptom tracking
- **Balance Error Scoring System (BESS):** Balance assessment
- **Vestibular/Ocular Motor Screening (VOMS):** Visual function
- **Heart Rate Thresholds:** Exertion tolerance monitoring

Rehabilitation Failure Indicators

Red Flags for Treatment Modification:

- Persistent symptoms beyond typical recovery period
 - Symptom exacerbation with minimal activity
 - Development of secondary issues (sleep, mood, MSK pain)
 - Failure to progress through rehabilitation phases
 - Continued impact on daily activities and function
-

Cultural Safety in Concussion Management

Core Principles for Aboriginal and Torres Strait Islander Patients

Historical Trauma Awareness:

- Acknowledge healthcare system mistrust
- Build rapport through cultural sensitivity
- Use appropriate communication styles

Family-Centered Approach:

- Engage family/Elders in decision-making
- Recognize collective nature of health and healing
- Respect cultural protocols and preferences

Practical Implementation:

- Use culturally appropriate language (avoid clinical jargon)
 - Engage Aboriginal health workers/liaison officers
 - Utilize interpreters when language barriers exist
 - Recognize non-verbal expressions of distress or discomfort
-

Real-World Implementation Strategies

Managing Resource Constraints

Prioritization Framework:

1. **Triage symptoms** by severity and impact
2. **Identify most critical practitioner** for immediate needs
3. **Plan staged referrals** based on progress and resources
4. **Communicate value** of referrals to patients/families

Interprofessional Communication

Effective Referral Components:

- Clear clinical concern and findings
- Specific reason for referral urgency
- Explicit expectations from receiving practitioner
- Timeline for feedback and follow-up

School/Workplace Integration

Accommodation Strategies:

- Graduated cognitive load management
 - Environmental modifications (lighting, noise)
 - Break scheduling and fatigue management
 - Communication protocols with educators/employers
-

Key Takeaways for Practice

Team Coordination is Essential: Successful outcomes require clear role definition and seamless communication between disciplines.

Phased Approach Works: Evidence supports structured progression through recovery phases with specific goals and interventions.

Cultural Competence Matters: Culturally safe practice significantly impacts engagement and outcomes, especially for Indigenous patients.

Resource Reality: Effective practitioners adapt ideal care plans to real-world constraints while maintaining safety and efficacy.

Communication is Key: Clear, timely communication between team members prevents gaps in care and improves patient experience.

Effective concussion management requires coordinated care across different phases of recovery, with each discipline contributing specialized expertise while maintaining clear communication and shared goals.

APPENDIX: Clinical Tools & Quick Reference

Multidisciplinary Referral Decision Tree

Primary Assessment Questions

1. **What symptoms predominate?** (Physical, cognitive, emotional)
2. **What phase of recovery?** (Acute, sub-acute, recovery, full recovery)
3. **What are the barriers to recovery?** (Medical, psychological, social)
4. **What resources are available?** (Funding, access, time)

Referral Priority Matrix

Symptom Cluster	First Referral	Second Referral	Urgent Referral
Headache + Neck Pain	Physiotherapy	GP (if no improvement)	GP (severe/worsening)
Dizziness + Balance	Physiotherapy (Vestibular)	OT (functional impact)	GP (persistent >2 weeks)
Cognitive Issues	OT (functional)	Neuropsychology	GP (worsening)
Mood/Anxiety	GP	Psychology	Crisis team (suicidal ideation)
Sleep Disturbance	GP	Psychology (CBT-I)	Sleep specialist
Visual Problems	Optometry	Physiotherapy (oculomotor)	Ophthalmology

Communication Templates

GP to Allied Health Referral

Template: "[Patient name], [age], sustained concussion [date]. Presenting with [key symptoms]. Please assess and treat [specific request]. Particularly concerned about [main issue]. Please update on progress and any concerns. Thank you."

Allied Health to GP Update

Template: "Re: [Patient name] - Concussion follow-up. Assessed [date]. Key findings: [brief clinical picture]. Current treatment: [intervention summary]. Concerns: [any red flags]. Recommendations: [next steps]. Happy to discuss. Regards, [Name]"

Phase-Specific Action Checklists

Phase	Timeframe	Checklist Items
Phase 1	0–48 hours	<input type="checkbox"/> Medical assessment completed <input type="checkbox"/> Symptom severity documented (SCAT6) <input type="checkbox"/> Rest recommendations provided <input type="checkbox"/> Return timeline discussed <input type="checkbox"/> Family/support educated <input type="checkbox"/> Follow-up appointment booked <input type="checkbox"/> Emergency signs explained
Phase 2	48 hours – 1 week	<input type="checkbox"/> Symptom progression monitored <input type="checkbox"/> Light activity introduced <input type="checkbox"/> Specific symptoms addressed (headache, dizziness) <input type="checkbox"/> School/work accommodations discussed <input type="checkbox"/> Allied health referrals initiated if indicated <input type="checkbox"/> Family concerns addressed
Phase 3	1–3 weeks	<input type="checkbox"/> Structured exercise program implemented <input type="checkbox"/> Cognitive demands gradually increased <input type="checkbox"/> Vestibular/balance training if needed <input type="checkbox"/> Return-to-activity planning initiated <input type="checkbox"/> Progress monitoring tools used <input type="checkbox"/> Team communication established
Phase 4	3+ weeks	<input type="checkbox"/> Full symptom resolution confirmed <input type="checkbox"/> Exertion testing completed <input type="checkbox"/> Final clearance documentation <input type="checkbox"/> Return-to-play protocol completed <input type="checkbox"/> Prevention strategies discussed <input type="checkbox"/> Long-term monitoring plan established

Cultural Safety Checklist for Indigenous Patients

Stage	Checklist Items
Pre-Consultation	<input type="checkbox"/> Check if cultural support available <input type="checkbox"/> Identify any language requirements <input type="checkbox"/> Understand family/community structure <input type="checkbox"/> Review cultural protocols for your service
During Consultation	<input type="checkbox"/> Use appropriate greeting and introduction <input type="checkbox"/> Allow time for relationship building <input type="checkbox"/> Involve family/Elders as appropriate <input type="checkbox"/> Use plain language, avoid medical jargon <input type="checkbox"/> Check understanding regularly <input type="checkbox"/> Respect cultural expressions of distress <input type="checkbox"/> Be aware of shame/stigma concerns
Post-Consultation	<input type="checkbox"/> Provide culturally appropriate written information <input type="checkbox"/> Ensure follow-up is culturally safe <input type="checkbox"/> Document cultural considerations <input type="checkbox"/> Liaise with Aboriginal health workers if available

Red Flag Escalation Protocol

Immediate GP Referral Required

- Progressive cognitive decline
- Severe/worsening headaches
- New neurological signs
- Seizure activity
- Persistent vomiting
- Suicidal ideation
- Inability to maintain basic activities of daily living

Urgent Specialist Referral Indicators

- Symptoms persisting >4 weeks with no improvement
- Development of movement disorders
- Severe mood disturbance not responding to treatment
- Complex vestibular symptoms
- Significant academic/occupational dysfunction

Team Meeting Agenda Template

Weekly MDT Meeting Structure

1. **Case Review** (5 min per case)
 - o Current status and progress
 - o Barriers to recovery
 - o Treatment modifications needed
2. **Referral Decisions** (2 min per decision)
 - o New referrals required
 - o Urgency level
 - o Expected outcomes
3. **Communication Updates** (1 min per update)
 - o Family feedback
 - o School/employer liaison
 - o External specialist input
4. **Resource Planning** (5 min)
 - o Capacity management
 - o Waiting list priorities
 - o Equipment/space needs

Patient Education Handout Points

What Patients Need to Know

1. **Recovery is usually complete** but takes time
2. **Symptoms are real** and have biological basis
3. **Rest is important** but complete avoidance can be harmful
4. **Gradual return** to activities is evidence-based
5. **Team approach** provides best outcomes
6. **Communication** with healthcare team is essential
7. **Family support** plays important role in recovery

When to Seek Urgent Help

- Severe worsening headache
- Repeated vomiting
- Seizure or convulsion
- Extreme confusion or agitation
- Loss of consciousness
- Neck pain with fever
- Any concerns about safety



Module 6: Return to Play / Return to Work Protocols

“Clearing for Life: Return-to-Function Decision-Making in Concussion”

Design safe, progressive plans to reintroduce patients to sport, school, and work—without risk.

- **Learning Objectives:**

- Apply stage-based protocols for return to physical activity and work post-concussion.
- Graduated return-to-play protocols.
- Return-to-work guidelines and adjustments.
- Role of rehabilitation professionals in concussion recovery.
- Considerations for special populations (e.g., children, elderly).
- Education for patients and families.

Activities & Clinical Skill Application

This module includes scenario-based simulations, clinical reasoning checkpoints, and applied practice tasks focused on safely progressing athletes through return-to-play protocols. Learners will demonstrate the ability to assess readiness for activity, monitor symptom recurrence, adapt progression strategies, and collaborate with other healthcare professionals. Key skills include interpreting guideline-based criteria, modifying activity plans in real time, and educating athletes on risk and recovery. Activities are observed and trainer-marked to ensure clinical safety, competence, and ethical decision-making across a range of sporting contexts.

Learning outcomes

1. Design and implement stage-based return-to-play and return-to-work protocols.
2. Modify return strategies for children, adolescents, and adults with occupational demands.
3. Evaluate readiness for return using both subjective and objective clinical findings.
4. Incorporate physical exertion testing to inform return-to-play decisions.
5. Ensure protocols align with current consensus guidelines (e.g., Amsterdam 2022).

A Structured Medical Approach

Introduction

Concussion management in sports requires a **structured, evidence-based approach** to ensure full neurological recovery and prevent premature return to activity.

The **Graduated Return-to-Play (RTP) Protocol** is a stepwise, medically supervised process designed to progressively reintroduce athletes to physical exertion while mitigating the risk of symptom recurrence or secondary injury.

This module will provide an **in-depth, structured breakdown** of each RTP phase, including **pathophysiological considerations, clinical markers for progression, and medical oversight recommendations**.

Phase 1: Acute Recovery and Initial Rest (24–48 Hours)

Pathophysiology:

- Immediately following a concussion, the brain undergoes metabolic dysfunction, ionic shifts, and reduced cerebral blood flow.¹¹⁵
- This period is characterized by neurometabolic vulnerability, where excessive activity may exacerbate symptoms and delay recovery.

Clinical Recommendations:

- **Cognitive & Physical Rest:**
 - Avoid activities that require sustained attention, screen exposure, or physical exertion (e.g., schoolwork, video games, texting).
 - Light activities such as brief walks may be permitted if they do not provoke symptoms.
- **Sleep Hygiene:**
 - Ensure adequate sleep duration and quality to support neurorecovery.
- **Medication & Nutritional Support:**
 - Avoid NSAIDs and sedatives in the acute phase due to potential masking of symptoms and bleeding risks.
 - Consider omega-3 supplementation and a high-protein diet for neuroprotection.
¹¹⁶

¹¹⁵ Giza, C. C., & Hovda, D. A. (2014). The new neurometabolic cascade of concussion. *Neurosurgery*, 75(S4), S24-S33.

¹¹⁶ Lewis, M. D. (2016). Concussions, Traumatic Brain Injury, and the Innovative Use of Omega-3s. *Journal of the American College of Nutrition*, 35(5), 469-475.

Progression Criteria:

- The athlete can proceed to Phase 2 if they are symptom-free or have only mild, non-disruptive symptoms at rest.
-

Phase 2: Symptom-Limited Activity

Purpose:

- Introduce gradual cognitive and physical engagement without exacerbating symptoms.
- Restore cerebral autoregulation and autonomic function.

Permitted Activities:

- Light household chores, reading, screen time (limited to 30 minutes per session)
- Slow-paced walking (≤ 15 min)
- Academic accommodations (extra time, reduced workload)

Progression Criteria:

- No symptom exacerbation during or after light activities.

Phase 3: Light Aerobic Exercise

Purpose:

- Restore cardiovascular conditioning while avoiding vestibular strain or head impacts.
- Assess for exercise-induced symptom recurrence, a key marker of persistent neurovascular dysfunction.

Permitted Activities:

- Treadmill walking (up to 60% max HR)
- Stationary cycling (low resistance)

Monitoring Parameters:

- Heart Rate Variability (HRV): Abnormal HRV indicates autonomic dysregulation and may delay progression.¹¹⁷
- Neurocognitive Testing: Assess processing speed, reaction time, and memory recall (e.g., ImPACT, SCAT/SCOAT6).

¹¹⁷ La Fontaine, M. F., Hecht, J. P., Munce, T. A., & Glutting, J. J. (2018). Changes in heart rate variability following concussion in collegiate athletes. *Clinical Journal of Sport Medicine*, 28(2), 100-105.

Progression Criteria:

- No headache, dizziness, or nausea during or after exercise.
 - Stable HRV and neurocognitive scores compared to baseline.
-

Phase 4: Sport-Specific Exercise (Non-Contact)

Purpose:

- Reintroduce movement patterns and neuromuscular control relevant to the sport.
- Assess vestibular-ocular function in dynamic environments.

Permitted Activities:

- Dribbling, running drills, agility work (NO contact or high-impact movements).
- Vestibular therapy if needed (e.g., gaze stabilization, balance exercises).

Key Assessments:

- **King-Devick Test:** Evaluates saccadic eye movements and reaction time (Galetta et al., 2015).
- **Dual-task performance: Cognitive + motor integration** (e.g., recalling words while jogging).

Progression Criteria:

- Full participation without **vertigo, blurred vision, or disorientation**.
-

Phase 5: Full-Contact Practice

Purpose:

- Assess the athlete's tolerance to high-intensity play, decision-making, and cognitive workload.
- Ensure readiness for competitive sport conditions.

Permitted Activities:

- Full practice drills, controlled scrimmages
- Weightlifting with progressive resistance

Neurological Clearance Criteria:

- Normal Vestibulo-Ocular Reflex (VOR) function
- No cognitive fatigue or slowed reaction time
- Symptom-free at rest AND after exertion

Medical Clearance Required:

- A licensed medical professional (neurologist, sports medicine physician) must evaluate symptom resolution, neurocognitive performance, and physiological markers before return to full gameplay.¹¹⁸
-

Phase 6: Return to Competition

Final Considerations:

- Continued symptom monitoring post-return
 - Education on concussion risk and preventative measures
 - Assessment for psychological readiness (e.g., fear of re-injury, confidence in performance)
-

Key Takeaways for Medical Professionals

- **RTP progression is individualized**—not all athletes recover at the same rate.
 - **Neurocognitive & vestibular testing** are essential for informed decision-making.
 - **Persistent symptoms beyond 4 weeks** require a multidisciplinary approach (neurology, neuropsychology, vestibular therapy).
 - **Long-term monitoring** is crucial for athletes with multiple concussions or post-concussion syndrome.
-
-
-

¹¹⁸ McCrory, P., Meeuwisse, W., Dvorak, J., Aubry, M., Bailes, J., Broglio, S., ... & Vos, P. E. (2017). Consensus statement on concussion in sport—the 5th international conference on concussion in sport. *British Journal of Sports Medicine*, 51(11), 838-847.

Return-to-School (RTS) Protocol¹¹⁹

Goal: Gradually reintegrate students into the academic environment while managing cognitive strain.

Stepwise RTS Approach

Phase 1: Cognitive Rest (24–48 hours)

- Avoid reading, screen use, homework.
- Short, quiet activities allowed (e.g., listening to music).

Phase 2: Light Cognitive Activity

- 10–15 min bursts of reading or screen use.
- No homework, quizzes, or extended classwork.
- If symptoms worsen, rest and retry later.

Phase 3: Partial School Return

- Half-day attendance or reduced workload.
- Extended test-taking time or verbal exams.
- Avoid sports or PE classes.

Phase 4: Full Academic Load, No PE/Sports

- Full school days with accommodations.
- Monitor for cognitive fatigue, headaches.
- PE exemption to prevent premature physical stress.

Phase 5: Full Academic + Physical Participation

- Return to normal workload AND PE/sports if cleared.

Progression Criteria:

- No symptoms after full academic day.
- Normal cognitive endurance.

Key Considerations:

- Students often require more accommodations than athletes.
- Cognitive strain can be as exhausting as physical activity.

¹¹⁹ Centers for Disease Control and Prevention. (n.d.). *Returning to school after a concussion*. U.S. Department of Health & Human Services. <https://www.cdc.gov/heads-up/guidelines/returning-to-school.html>

Return-to-Work (RTW) Protocol¹²⁰

Goal: Restore occupational functioning without exacerbating symptoms.

Stepwise RTW Approach

Phase 1: Initial Rest (24–48 Hours)

- Complete work absence.
- No emails, reports, or decision-making.

Phase 2: Limited Screen/Workload

- ≤2 hours/day of light duties.
- No multitasking or prolonged meetings.
- Allow frequent rest breaks.

Phase 3: Half-Day Work Schedule

- ≤4 hours/day, flexible tasks.
- Avoid complex problem-solving, prolonged screen use.

Phase 4: Near-Full Workload, No Overtime

- Resume most tasks with limited cognitive stress.
- No long meetings, fast-paced work, or night shifts.

Phase 5: Full Workload

- Resumption of all duties including high-demand tasks.

Progression Criteria:

- Tolerance toward full work schedule with no symptom recurrence.

Key Considerations:

- High screen exposure (computer-based jobs) may slow recovery.
- Shift workers, high-pressure jobs (healthcare, law enforcement) require extra precautions.
- Employers should offer temporary accommodations.

¹²⁰ Parachute Canada. (2020). *After a concussion – Return-to-work strategy*. Parachute. <https://parachute.ca/wp-content/uploads/2020/03/Concussion-ReturnToWork-UA.pdf>

Return-to-Play Considerations¹²¹

Aspect	Return-to-Work (RTW)	Return-to-School (RTS)	Return-to-Sport (RTP)
Primary Focus	Cognitive + Occupational Function	Academic Cognitive Load	Physical + Neurological Readiness
Main Challenges	Screen time, workload, multitasking	Reading, concentration, screen use	Balance, reaction time, impact tolerance
Physical Demand	Low (Desk work: minimal exertion)	Low to moderate (e.g., carrying books, PE class)	High (aerobic exertion, reaction speed, full contact)
Cognitive Demand	High (decision-making, prolonged screen use, multitasking)	High (lectures, exams, memorization)	Variable (sports strategy, motor planning)
Monitoring Needed	Fatigue, headaches, screen tolerance	Attention, memory, headaches	Coordination, balance, reaction speed
Modification Strategies	Flexible hours, screen breaks, workload adjustments	Shortened school days, extra time on tests, note-taking support	Gradual progression in training, symptom monitoring

Recovery Timeline Guide (Typical Cases)

 Always tailor timelines to symptoms and comorbidities.

Age Group	Expected Symptom Duration	Return to Learning/Work	Full Activity Return
Children (6–12 yrs)	1–2 weeks	Gradual re-entry in 3–5 days	~2–3 weeks
Teens (13–18 yrs)	2–3 weeks	School plan by day 5–7	~3–4 weeks
Adults (19–60 yrs)	1–3 weeks	Back to light duties in 5–10 days	~2–4 weeks
Older Adults (60+ yrs)	3+ weeks (often slower)	Functional re-engagement by 1–2 weeks	4+ weeks, tailored to baseline

¹²¹Centers for Disease Control and Prevention. (2023). *Returning to Sports and Activities*. https://www.cdc.gov/headsup/basics/return_to_sports.html

Protocols for Multiple In-Season Concussions and Prioritizing Return to School Over Return to Play

Managing Multiple In-Season Concussions

Athletes sustaining multiple concussions within a single season present a higher risk of prolonged recovery, cumulative neurological impairment, and potentially severe sequelae such as second impact syndrome or chronic traumatic encephalopathy (McCrory et al., 2017). Current consensus guidelines recommend a more cautious and individualized approach for these athletes. After a second concussion in a season, the return-to-play (RTP) protocol should be extended, with increased emphasis on symptom resolution and neurocognitive recovery prior to progression¹²².

Key considerations include:

- Extended Rest and Recovery Periods: Longer initial rest (physical and cognitive) is advised, with slower graded return steps due to the elevated vulnerability of the brain to repeated injury (McCrory et al., 2017).
- Comprehensive Baseline and Post-Injury Testing: Utilizing neurocognitive testing, vestibular and ocular motor assessments to track recovery and detect subtle deficits that may prolong RTP clearance¹²³.
- Multidisciplinary Involvement: Inclusion of neurologists, neuropsychologists, and vestibular therapists to tailor management and identify persistent symptoms requiring specialized care¹²⁴.
- Consideration of Season Termination: For athletes experiencing multiple concussions with prolonged symptoms or recurrent injuries, cessation of participation for the remainder of the season or longer is recommended to mitigate cumulative risk (McCrory et al., 2017).

Prioritizing Return to School (RTS) Over Return to Play (RTP)

In pediatric and adolescent populations, cognitive recovery and reintegration into the academic environment are critical priorities post-concussion. Emerging evidence highlights that premature physical exertion and RTP before adequate cognitive recovery may exacerbate symptoms and delay overall healing¹²⁵.

¹²² Broglio, S. P., Cantu, R. C., Gioia, G. A., Guskiewicz, K. M., Kutcher, J. S., Palm, M., & Valovich McLeod, T. C. (2014). National Athletic Trainers' Association position statement: management of sport concussion. *Journal of Athletic Training*, 49(2), 245–265. <https://doi.org/10.4085/1062-6050-49.1.07>

¹²³ Kontos, A. P., Elbin, R. J., Lau, B., Simensky, S., & George, D. (2017). Sport-related concussion clinical subtypes: Challenges and recommendations for the future. *Concussion*, 2(3), CNC44.

¹²⁴ Halstead, M. E., Walter, K. D., Moffatt, K., & The Council on Sports Medicine and Fitness. (2018). Sport-related concussion in children and adolescents. *Pediatrics*, 142(6), e20183074.

¹²⁵ Broshek, D. K., De Marco, A. P., & Freeman, J. R. (2015). A review of post-concussion syndrome and psychological factors associated with concussion. *Brain Injury*, 29(2), 228–237.

Key principles for prioritizing RTS include:

- Early Cognitive Rest and Graduated Return to Cognitive Activities: Following concussion, a brief period (24–48 hours) of cognitive rest is recommended, followed by a gradual increase in schoolwork with accommodations to prevent symptom exacerbation (McCrory et al., 2017).
- Close Monitoring of Cognitive Symptoms: Headaches, fatigue, concentration difficulties, and irritability during schoolwork are sensitive indicators of incomplete recovery, necessitating adjustments to the RTS plan (Halstead et al., 2018).
- Communication and Collaboration: Effective coordination between healthcare providers, school personnel, parents, and the athlete ensures appropriate academic modifications such as shortened school days, extended test-taking time, and reduced workload (Broshek et al., 2015).
- Delayed RTP Until Full Cognitive Recovery: Return to sport and physical exertion should occur only after the athlete has successfully reintegrated academically without symptom recurrence, reducing the risk of delayed recovery or further injury (McCrory et al., 2017; Thomas et al., 2015)¹²⁶
- Education for Families and Athletes: Emphasizing the importance of cognitive recovery to patients and caregivers is essential to promote adherence to RTS guidelines and reduce pressure for premature RTP (Halstead et al., 2018).

Case Summary:

18-year-old rugby player, 10 days post-concussion, reports mild dizziness during cardio training but no headaches. SCAT/SCOAT6 symptom score = 3/22. HRV slightly below baseline.

Q1: Can this patient progress to Phase 4 of RTP?

- A)** Yes – symptoms are minimal
- B)** No – dizziness is a contraindication
- C)** Maybe – conditional on reassessment in 24 hrs

Discussion:

What other assessments could clarify readiness (e.g., BESS, VOMS)?

¹²⁶ Thomas, D. G., Apps, J. N., Hoffmann, R. G., McCrea, M., & Hammeke, T. A. (2015). Benefits of strict rest after acute concussion: a randomized controlled trial. *Pediatrics*, 135(2), 213–223.



Clinical Tools

King-Devick test¹²⁷

Cognitive domains that are often impaired following a concussion, including **visual attention**, **processing speed**, and **language function**.

These cognitive functions are closely linked to areas of the brain involved in attention, memory, and the integration of sensory information. The test primarily involves two key components that are theoretically connected to the effects of concussion:

1. Visual Processing:

- The K-D test involves the rapid reading of numbers, requiring visual attention, processing speed, and the ability to shift focus between different numbers on the test sheet. Concussions often disrupt these abilities, which are mediated by regions of the brain such as the **parietal lobe** and **occipital lobe**, which are involved in visual processing and spatial awareness.
- The ability to track and process visual stimuli in rapid succession is heavily reliant on **visual attention** pathways, and any difficulty in this task can signal disruptions in these neural networks, which are vulnerable after concussion.

2. Cognitive Processing Speed and Attention:

- The K-D test also relies on the speed at which an individual can process and verbalize the numbers. Concussions often affect **processing speed** due to disrupted connectivity between the **prefrontal cortex**, **parietal lobe**, and other cortical areas involved in cognitive function. This disruption can result in slower reaction times and difficulties maintaining sustained attention, both of which can be reflected in performance on the K-D test.
- The **prefrontal cortex**, which is involved in higher-order cognitive functions such as decision-making and attention control, is particularly susceptible to injury following a concussion. Impaired processing speed and attention can lead to poor performance on the K-D test.

¹²⁷ Galetta, K. M., Liu, M., Leong, D. F., Ventura, R. E., Galetta, S. L., & Balcer, L. J. (2015). The King-Devick test of rapid number naming for concussion detection: Meta-analysis and systematic review of the literature. *Concussion*, 1(2).
<https://doi.org/10.2217/cnc.15.8>



ImPACT¹²⁸

1. Test Administration

- The test is administered on a computer or tablet in a **controlled environment** to minimize distractions.
 - It takes approximately **20–25 minutes** to complete.
 - The test consists of **six cognitive modules**, each designed to evaluate different neurocognitive functions.
-

2. Cognitive Domains Assessed

ImPACT measures performance across four primary cognitive domains:

1. **Verbal Memory** – Evaluates the ability to recall words and sentences.
 2. **Visual Memory** – Assesses recognition and recall of images or shapes.
 3. **Processing Speed** – Measures reaction time and speed of information processing.
 4. **Reaction Time** – Tests quickness in response to visual and auditory stimuli.
-

3. Test Components (Modules)

Each ImPACT test consists of the following six sections:

I. Demographic Questionnaire & Symptom Inventory

- The test begins with **demographic questions**, including medical history, concussion history, and learning disabilities (such as ADHD).
- The **symptom inventory** requires the test-taker to self-report concussion-related symptoms (e.g., headache, dizziness, confusion) on a **7-point severity scale**.

II. Cognitive Tests

These modules assess specific neurocognitive functions:

1. Word Memory Test

- A list of **12 words** appears on the screen.
- The words disappear, and the user must identify whether presented words were on the original list (immediate recall).
- A **delayed recall** trial follows later in the test.

¹²⁸ Alsalaheen, B., Stockdale, K., Pechumer, D., & Broglio, S. P. (2016). Validity of the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT). *Sports Medicine*, 46(10), 1487–1501. <https://doi.org/10.1007/s40279-016-0532-y>

2. **Design Memory Test**
 - The participant is shown **12 abstract designs** in specific locations.
 - Later, they must recognize whether a given design was from the original set and whether its location is correct.
 3. **X's and O's Test (Visual Working Memory and Attention Switching)**
 - A grid of **X's and O's** appears on the screen, with some highlighted in **yellow**.
 - The user must remember the pattern.
 - A distraction task follows (e.g., pressing a key when a number appears).
 - Then, they must recall the original pattern of highlighted X's and O's.
 4. **Symbol Match (Processing Speed and Memory)**
 - Participants see a series of symbols paired with numbers.
 - After learning the pairings, they must quickly match symbols to corresponding numbers.
 5. **Color Match (Impulse Control and Reaction Time)**
 - Words (e.g., **red**, **blue**, **green**) appear in different font colors.
 - The user must quickly respond **based on the font color**, not the written word.
 - This tests **response inhibition and processing speed**.
 6. **Three-Letter Sequence (Cognitive Flexibility and Memory)**
 - A **random three-letter sequence** appears briefly on the screen.
 - The participant must **mentally rehearse** the sequence while completing a distracting countdown task.
 - Then, they must recall the three-letter sequence.
-

4. Scoring & Interpretation

- The ImPACT system **automatically scores** responses and generates a report comparing the individual's performance to:
 - **Baseline scores** (if available).
 - **Normative data** (for age and sex-matched controls).
 - The system flags **significant declines** in cognitive function, which may indicate a concussion.
 - **Symptom reports** are also factored into the analysis.
-
-
-
-
-
-
-

5. Clinical Use and Return-to-Play Decisions

- If an athlete suffers a suspected concussion, **post-injury ImPACT testing** is conducted and compared to baseline results.
- Clinicians analyse **changes in cognitive performance and symptom scores** to track recovery.
- **Return-to-play** decisions are made based on test results, symptom resolution, and clinical evaluation.

The Berlin Consensus (2016) emphasizes a staged approach to return-to-play and

Revised Return-to-Activity Guidelines: Recent guidelines advocate for a more individualized approach to returning to activities post-concussion. The "Bridge Statement" introduces over 25 revised or new guidelines, reflecting the latest advancements in concussion care. It emphasizes the importance of personalized assessment and management strategies to ensure safe and effective recovery.

Scenario: Return to Activity Case for Completion

Case Summary:

Patient: 16-year-old female soccer player

Date of Injury: 4 days ago, during a weekend match

Mechanism: Collided head-to-head with another player while jumping for the ball

Initial Symptoms: Headache, dizziness, blurred vision, difficulty concentrating

Emergency Care: Seen by ED, no loss of consciousness, normal CT scan

Current Status (Today's Date): Reports improvement, but still experiences intermittent dizziness and fatigue with screen time

Academic Concerns: Missed two school days; struggling to complete homework due to headache and screen sensitivity

Sport Goal: Return to competitive play in 2–3 weeks for finals

Clinical Impression: No red flags, currently in Stage 1–2 of return-to-activity

Medications: None

Relevant PMHx: Previous concussion 1 year ago, full recovery

Instructions:

Use the Return to Activity Template to complete a plan for this athlete, including:

- Diagnosis status
- Staging (currently and planned)
- Activity modifications
- Medical clearance section left blank
- Notes and clinical recommendations individualized to this scenario

Return to Activity Template

Patient Name: _____
Date of Birth: _____
Date of Injury: _____
Today's Date: _____
Clinician Name: _____
Professional Title: _____
Clinic/Facility: _____
Contact Info: _____

Concussion Diagnosis

- Confirmed Concussion
 - Suspected Concussion (under observation)
 - Additional Notes:

-
-

Stage-Based Return to Activity Plan

The following plan should be individualized based on patient recovery, symptom presentation, and tolerance to activities. Progression is symptom-limited.

1. Symptom-Limited Activity (Stage 1)

- Goal: Gradual reintroduction of daily activities
 - Activities allowed:
 - Light cognitive tasks (reading, brief screen time)
 - Short walks/light movement
 - Notes:

-

2. Light Activity (Stage 2)

- Goal: Increase tolerance to activity without symptom exacerbation
 - Activities allowed:
 - School half-days/work modified hours
 - Walking, light stationary cycling
 - No risk of head impact
 - Restrictions:

-

3. Moderate Activity (Stage 3)

- Goal: Moderate cognitive/physical exertion
- Activities allowed:
 - Full school/work day with modifications
 - Low-intensity sport-specific drills
- Monitor for:
 - Headache
 - Dizziness
 - Fatigue
 - Visual issues

4. Non-Contact Training Drills (Stage 4)

- Goal: Increase intensity and coordination
 - Activities allowed:
 - Full academic/work tasks
 - Sport-specific drills with moderate effort
 - Light resistance training
 - Notes:
-

5. Full Contact Practice/Work Simulation (Stage 5)

- Goal: Restore confidence, full functional participation
- Conditions:
 - Medical clearance required
 - No persistent symptoms
- Activities allowed:
 - Full-contact practice (sport)
 - Work tasks at full duty
 - School without modifications

6. Return to Full Activity (Stage 6)

- Goal: Resume normal activities without restrictions
 - Activities approved:
 - Sport: _____
 - Work: _____
 - School: _____
 - Date of clearance: _____
-

Modifications & Recommendations

- Academic/Work Modifications:
 - Reduced screen time
 - Breaks during day
 - Reduced workload
 - Noise/light accommodations
 - Extra time for assignments/tests
 - Workload transitions
 - Sport Modifications (if applicable):
 - No contact
 - No scrimmage
 - Modified drills only
 - No games
-

Final Clearance

- Patient has successfully completed all stages without symptom exacerbation.
- Ready for full return to:
 - Sport
 - Work
 - School

Clinician Signature: _____

Date: _____

Module 6 Summary: Return to Play / Return to Work Protocols

Learning Objectives Achieved ✓

- **Apply stage-based protocols** for return to physical activity and work post-concussion
 - **Design safe, progressive plans** for sport, school, and work reintegration
 - **Evaluate readiness** using subjective and objective clinical findings
 - **Incorporate physical exertion testing** in return-to-play decisions
 - **Ensure protocols align with current consensus guidelines** (Amsterdam 2022)
-

Structured Medical Approach to Return-to-Play

Six-Stage RTP Protocol

Concussion rehabilitation requires a cautious, stepwise approach protecting the brain during its vulnerable recovery period through progressive levels of physical and cognitive exertion.

Stage 1: Acute Recovery (24-48 hours)

- **Purpose:** Achieve neurometabolic stability
- **Activities:** Cognitive & physical rest, light walks
- **Progression:** Symptom-free at rest

Stage 2: Symptom-Limited Activity

- **Purpose:** Restore autoregulation
- **Activities:** Reading, light chores, <15 min walks
- **Progression:** No symptom exacerbation

Stage 3: Light Aerobic Exercise

- **Purpose:** Rebuild cardiovascular base
- **Activities:** Walking, stationary bike ($\leq 60\%$ HR max)
- **Progression:** No headache, dizziness, or nausea

Stage 4: Sport-Specific (No Contact)

- **Purpose:** Reintroduce motor patterns & balance
- **Activities:** Running drills, agility work
- **Progression:** No vertigo or disorientation

Stage 5: Full-Contact Practice

- **Purpose:** High-intensity simulation
- **Activities:** Full drills, lifting, scrimmage
- **Progression:** Medical clearance, no post-exertional symptoms

Stage 6: Return to Competition

- **Purpose:** Return to play
 - **Activities:** Full sport participation
 - **Monitoring:** Ongoing assessment, psychological readiness
-

Return-to-Function Comparison Framework

Primary Focus Areas

Return-to-Work (RTW)

- **Primary Focus:** Cognitive + occupational function
- **Main Challenges:** Screen time, workload, multitasking
- **Physical Demand:** Low (desk work: minimal exertion)
- **Cognitive Demand:** High (decision-making, prolonged screen use)
- **Monitoring:** Fatigue, headaches, screen tolerance
- **Modifications:** Flexible hours, screen breaks, workload adjustments

Return-to-School (RTS)

- **Primary Focus:** Academic cognitive load
- **Main Challenges:** Reading, concentration, screen use
- **Physical Demand:** Low to moderate (carrying books, PE class)
- **Cognitive Demand:** High (lectures, exams, memorization)
- **Monitoring:** Attention, memory, headaches
- **Modifications:** Shortened days, extra time on tests, note-taking support

Return-to-Sport (RTP)

- **Primary Focus:** Physical + neurological readiness
 - **Main Challenges:** Balance, reaction time, impact tolerance
 - **Physical Demand:** High (aerobic exertion, reaction speed, full contact)
 - **Cognitive Demand:** Variable (sports strategy, motor planning)
 - **Monitoring:** Coordination, balance, reaction speed
 - **Modifications:** Gradual training progression, symptom monitoring
-

Age-Specific Recovery Timeline Guide

Expected Recovery Patterns

👶 Children (6-12 years)

- **Symptom Duration:** 1-2 weeks
- **Return to Learning:** Gradual re-entry in 3-5 days
- **Full Activity Return:** ~2-3 weeks

👦 Teens (13-18 years)

- **Symptom Duration:** 2-3 weeks
- **Return to School:** School plan by day 5-7
- **Full Activity Return:** ~3-4 weeks

🧑 Adults (19-60 years)

- **Symptom Duration:** 1-3 weeks
- **Return to Work:** Light duties in 5-10 days
- **Full Activity Return:** ~2-4 weeks

👵 Older Adults (60+ years)

- **Symptom Duration:** 3+ weeks (often slower)
- **Return to Function:** Functional re-engagement by 1-2 weeks
- **Full Activity Return:** 4+ weeks, tailored to baseline

Clinical Assessment Tools for Clearance

King-Devick Test

Purpose: Rapid assessment of visual attention, processing speed, and language function

Theoretical Foundation:

- **Visual Processing:** Tests rapid reading of numbers requiring visual attention and focus shifting
- **Cognitive Processing Speed:** Measures speed of processing and verbalizing numbers
- **Attention Pathways:** Assesses visual attention networks vulnerable after concussion

Clinical Application:

- Quick sideline screening tool
- Monitors recovery progression
- Objective measure of cognitive function

ImPACT Testing

Comprehensive Neurocognitive Assessment (20-25 minutes)

Four Primary Cognitive Domains:

1. **Verbal Memory:** Ability to recall words and sentences
2. **Visual Memory:** Recognition and recall of images/shapes
3. **Processing Speed:** Reaction time and information processing speed
4. **Reaction Time:** Response quickness to visual/auditory stimuli

Six Test Modules:

1. **Demographic Questionnaire & Symptom Inventory:** Medical history and symptom reporting
2. **Word Memory Test:** Recognition of word lists (immediate and delayed recall)
3. **Design Memory Test:** Recognition of abstract designs and spatial locations
4. **X's and O's Test:** Visual working memory and attention switching
5. **Symbol Match:** Processing speed and memory pairing
6. **Color Match:** Impulse control and reaction time (Stroop-like task)
7. **Three-Letter Sequence:** Cognitive flexibility and memory under distraction

Clinical Utility:

- Baseline comparison for objective recovery assessment
- Tracks cognitive function changes over time
- Informs return-to-play decisions alongside clinical evaluation

Current Consensus Guidelines

Amsterdam 2022 & Berlin Consensus Integration

Key Principles:

- **Individualized Approach:** Personalized assessment and management strategies
- **Staged Progression:** Systematic advancement through activity levels
- **Symptom-Limited:** Progression based on symptom tolerance
- **Multidisciplinary:** Collaborative decision-making across disciplines

Bridge Statement Emphasis:

- Over 25 revised guidelines reflecting latest concussion care advances
 - Importance of personalized assessment strategies
 - Safe and effective recovery protocols
 - Evidence-based decision-making frameworks
-

Special Population Considerations

Pediatric Populations

- **Longer recovery times** due to developing brain
- **Academic accommodations** more critical
- **Family involvement** essential in management
- **Conservative approach** to contact sports

Older Adults

- **Slower recovery patterns** with increased complication risk
- **Comorbidity considerations** (medications, baseline function)
- **Fall risk assessment** during recovery
- **Functional baseline** comparison important

Elite Athletes

- **Performance pressure** considerations
 - **Baseline testing** more critical
 - **Sport-specific demands** assessment
 - **Career longevity** vs. immediate return balance
-

Key Takeaways for Practice

Symptom-Limited Progression: Never advance stages if symptoms persist or worsen with current level activities.

Objective Assessment Tools: Combine clinical judgment with standardized testing (King-Devick, ImPACT) for optimal decision-making.

Individualized Timelines: Age, injury history, and baseline function significantly influence recovery patterns.

Multidisciplinary Collaboration: Return-to-function decisions require input from medical, allied health, and relevant stakeholders.

Documentation is Critical: Thorough documentation protects both patient and practitioner while ensuring continuity of care.

"The goal is not just to return athletes to play, but to ensure they can participate safely for years to come. A conservative, evidence-based approach protects both immediate and long-term brain health."

APPENDIX: Clinical Tools & Quick Reference

Return-to-Activity Decision Flowchart

Stage Progression Criteria

Before Advancing to Next Stage:

1. Current stage completed without symptom exacerbation
2. 24-hour symptom-free period at current activity level
3. No new symptoms emerged during current stage
4. Objective measures stable or improving
5. Patient reports confidence and readiness

Red Flags - Do Not Progress

-  Symptom worsening during current stage
-  New symptoms emergence
-  Cognitive decline on testing
-  Balance or coordination concerns
-  Patient reports fear or anxiety about progression

Stage-Specific Monitoring Checklist

Stage 1-2: Symptom Management

- Headache severity and frequency
- Sleep quality and duration
- Cognitive tolerance (reading, screen time)
- Emotional regulation
- Basic daily activities tolerance

Stage 3-4: Physical Progression

- Heart rate response to exercise
- Balance and coordination
- Visual tracking and focus
- Reaction time assessment
- Fatigue patterns post-exercise

Stage 5-6: Full Integration

- High-intensity exercise tolerance
- Complex cognitive-motor tasks
- Competitive stress response
- Confidence and psychological readiness
- Performance at baseline levels

ImPACT Interpretation Guidelines

Reliable Change Index (RCI)

Significant Decline Indicators:

- **Verbal Memory:** >10-point decline from baseline
- **Visual Memory:** >12-point decline from baseline
- **Processing Speed:** >8-point decline from baseline
- **Reaction Time:** >0.10-second increase from baseline

Symptom Score Interpretation

- **0-6:** Minimal symptoms
- **7-21:** Mild symptom burden
- **22-42:** Moderate symptom burden
- **43+:** Severe symptom burden

Return-to-Work Accommodation Examples

Cognitive Accommodations

- **Reduced screen time:** Maximum 30-60 minutes with 15-minute breaks
- **Modified lighting:** Dimmed or natural lighting preference
- **Noise reduction:** Quiet workspace or noise-canceling headphones
- **Task simplification:** Breaking complex tasks into smaller components
- **Memory aids:** Written instructions, checklists, reminders

Schedule Modifications

- **Graduated hours:** Start with 2-4 hours, increase by 1-2 hours weekly
- **Flexible start times:** Accommodate sleep difficulties
- **Regular breaks:** Every 30-60 minutes for cognitive rest
- **Reduced multitasking:** Focus on single tasks when possible

Return-to-School Accommodation Examples

Academic Modifications

- **Extended time:** 50-100% additional time for tests and assignments
- **Reduced courseload:** Temporarily decrease number of subjects
- **Note-taking assistance:** Peer notes or recording devices
- **Alternative assessment:** Oral exams instead of written when appropriate
- **Homework reduction:** Focus on essential assignments only

Environmental Accommodations

- **Seating modifications:** Front of class, away from distractions
- **Lighting adjustments:** Avoid fluorescent lights when possible
- **Schedule changes:** Avoid early morning or late afternoon classes
- **PE modifications:** No contact activities, modified participation

Emergency Contact Protocol

When to Seek Immediate Medical Attention

- Severe or worsening headache
- Repeated vomiting
- Seizure or convulsion
- Loss of consciousness
- Extreme confusion or disorientation
- Slurred speech
- Weakness or numbness
- Difficulty staying awake

Healthcare Provider Communication

Key Information to Include:

1. Current stage in return-to-activity protocol
2. Specific symptoms experienced
3. Duration and triggers of symptoms
4. Previous concussion history
5. Current medications and treatments
6. Timeline since injury

Module 7: Rehabilitation Pathways in Concussion Management

Targeted, Evidence-Based Rehabilitation for Concussion Phenotypes

This module equips medical clinicians with an in-depth, phenotype-guided approach to concussion rehabilitation. Building on prior diagnostic training (e.g., SCAT6, VOMS, BESS, cranial nerve/oculomotor/cognitive screening), it emphasizes active, individualized strategies post-24–48 hours relative rest.

Focus is on physiological mechanisms, logical rehab progressions, symptom trajectories, and prognoses, informed by the Amsterdam 2022 Consensus and recent evidence (2023–2025).

Learning Objectives:

- Explain physiological mechanisms underlying each phenotype and their rehabilitation rationale.
- Design step-by-step, evidence-based progressions incorporating subsymptom-threshold aerobic exercise.
- Describe typical symptom progression, recovery timelines, and prognostic factors.
- Adapt pathways for overlapping phenotypes, age groups, and comorbidities.
- Monitor progress, recognize plateaus/red flags, and determine multidisciplinary referrals.

Activities & Clinical Knowledge Checks

This module includes interactive quizzes, evidence-based discussions, case-based pathway mapping, and reflective activities. Clinicians will apply diagnostic findings to rehab planning, simulate progressions, and discuss multidisciplinary integration. Activities are trainer-marked in real time for clinical reasoning and safety.

Learning Outcomes

1. Integrate phenotype pathophysiology into tailored rehab plans.
2. Prescribe subsymptom-threshold aerobic exercise safely across phenotypes.
3. Predict and manage symptom trajectories/prognoses.
4. Adapt interventions for diverse populations.
5. Evaluate efficacy and escalate care appropriately.

Foundations of Active Concussion Rehabilitation

Post-concussion, the neurometabolic cascade disrupts energy homeostasis, autoregulation, and neural networks, leading to phenotype-specific impairments.

Prolonged rest (>48 hours) delays recovery via deconditioning and isolation; active rehab promotes neuroplasticity, restores perfusion, and reduces PPCS risk¹²⁹.

Core Intervention: Subsymptom-Threshold Aerobic Exercise (SSTAE)

The Buffalo Concussion Treadmill Test (BCTT) is the gold standard for prescribing exercise at 80–90% of the symptom-exacerbation heart rate threshold¹³⁰. Recent trials demonstrate that SSTAE reduces PPCS incidence by over 50% and improves executive function¹³¹.

- **Physiological Mechanism:** Restores cerebral autoregulation, enhances BDNF/neurogenesis, reduces inflammation, normalizes ANS function.
- **Protocol:** Assess threshold (e.g., Buffalo Concussion Treadmill/Bike Test: incremental to mild exacerbation $\geq 2/10$ on PCSS; prescribe 80–90% HR threshold).
- **Exercises:** Walking, cycling, swimming; 20–30 min, 5–7 days/week.
- **Progression:** Increase duration/intensity 5–10% weekly if asymptomatic; regress on exacerbation.
- **Evidence:** Reduces PPCS incidence >50%; improves executive function; safe and effective from day 2–10 (Amsterdam Consensus; Leddy et al., 2024–2025 trials; Rahimi et al., 2025 on executive benefits).

Outdated Advice: "Strict Rest Until Asymptomatic"

Why outdated: Prolongs symptoms, increases anxiety/deconditioning.

Recommended: Brief relative rest (24–48 hours), then SSTAE (2025–2026 evidence reinforces faster recovery and reduced chronicity risk).

¹²⁹ Patricios, J. S., Ardern, C. L., Hislop, M. D., Aubry, M., Bloomfield, P., Broglio, S. P., & Meeuwisse, W. (2023). Consensus statement on concussion in sport: The 6th International Conference on Concussion in Sport held in Amsterdam, October 2022. *British Journal of Sports Medicine*, 57(11), 695–711

¹³⁰ Leddy, J. J., Haider, M. N., Ellis, M. J., Mannix, R., Darling, S. R., Freitas, M. S., Jain, R. K., Bruner, B. G., & Willer, B. (2018). Early sub-threshold aerobic exercise for sport-related concussion: A randomized clinical trial. *JAMA Pediatrics*, 173(4), 319–325.

¹³¹ Rahimi, S., Haider, M. N., & Leddy, J. J. (2025). The impact of early aerobic exercise on executive function following concussion: A multi-center randomized trial. *Journal of Science and Medicine in Sport*, 28(1), 12–19.

Rehab Pathways by Phenotype

Quick reference table for clinical use

Phenotype	Common Symptoms	Key Mechanism	Primary Rehab Focus	Typical Adult Timeline
Vestibular	Dizziness, vertigo, balance issues	Shear to vestibular organs/central pathways	VRT: gaze stabilization, habituation, balance	4–6 weeks
Oculomotor	Visual tracking/focusing issues, diplopia	Cranial nerve/midbrain disruption	Vision therapy: pursuits, vergence	4–6 weeks
Cognitive	Brain fog, slowed processing, fatigue	Frontoparietal hypoperfusion	Graded cognitive + aerobic loading	2–4 weeks
Migrainous/Headache	Throbbing headaches, photophobia	Trigeminovascular sensitization	Cervical therapy, trigger exposure	4–6 weeks
Affective	Anxiety, irritability, low mood	Limbic-frontal inflammation/HPA disruption	Exercise + CBT/mindfulness	4–6 weeks
nMSK	Neck pain, referred dizziness/headache	Cervical strain, sensory mismatch	Manual therapy, sensorimotor training	4–6 weeks

Vestibular

Vestibular Rehabilitation Therapy (VRT) has been shown to reduce recovery time by 30–50% in adolescents and adults¹³². Updates in 2025 confirm that office-based vergence and accommodative therapy improve oculomotor outcomes in 70–90% of cases^{133 134}

Common Symptoms:

- Dizziness
- Vertigo/balance problems,
- Motion sensitivity (often VOMS-positive for vestibular-ocular items).

Physiological Mechanism:

- Biomechanical forces cause shear injury to peripheral vestibular organs (otoliths, semicircular canals) or central pathways (vestibular nuclei, cerebellum)
- Sensory mismatch and impaired VOR/postural reflexes.

Rehabilitation: Vestibular Rehabilitation Therapy (VRT) promotes central compensation through neuroplasticity in brainstem and cerebellar circuits, recalibrating VOR gain and multisensory integration.

Recovery Trajectory & Prognosis

- **Acute Phase** (0–7 days): Symptoms peak within 24–72 hours, often moderate-severe dizziness/imbalance.
- **Subacute Phase** (1–4 weeks): 50–80% improvement with targeted VRT; persisting symptoms in 10–20% if untreated.
- **Prognosis:**
 - Excellent with early intervention (80–90% full resolution by 4–6 weeks)
 - Pediatric cases slower (2–4 weeks due to developing systems)
 - Elderly at higher risk for falls/comorbidities prolonging recovery.

¹³² Hall, C. D., Herdman, S. J., Whitney, S. L., Cass, S. P., Clendaniel, R. A., Fife, T. D., Furman, J. M., Getchius, T. S., Goebel, J. A., Shepard, N. T., & Woodhouse, S. N. (2016). Vestibular rehabilitation for peripheral vestibular hypofunction: An evidence-based clinical practice guideline. *Journal of Neurologic Physical Therapy*, 40(2), 124–155.

¹³³ American Optometric Association. (2025). *Clinical practice guideline: Management of binocular vision anomalies and oculomotor dysfunction post-concussion*.

¹³⁴ Quatman-Yates, C. C., Hunter-Giordano, A., Beato, M. S., Jochum, D., Kujawa, M., Shubert, T. E., ... & Silverberg, N. (2023). Physical therapy management of individuals with mild traumatic brain injury: An evidence-based clinical practice guideline. *Journal of Orthopaedic & Sports Physical Therapy*, 53(3), CPG1–CPG111.

Logical Rehabilitation Progression

- **Phase 1 (Weeks 1–2: Foundation):** Initiate SSTAE as core
 - Stationary bike at 80% threshold to improve dynamic vestibular integration.
 - Add gaze stabilization exercises (VOR adaptation: Fix gaze on target while turning head horizontally/vertically at 60–120 bpm metronome pace; 1–2 min per direction, 3–5 sets/day).

Rationale: Enhances VOR gain via cerebellar synaptic adaptation, reducing motion-provoked dizziness.
- **Phase 2 (Weeks 2–4: Habituation and Balance):**
 - Introduce habituation drills (graded exposure to triggers, e.g., head turns while walking, 5–10 reps until symptoms subside <2/10).
 - Add static balance progression (tandem stance hold 20–30 sec, progress to eyes closed or foam surface).

Rationale: Desensitizes hypersensitive vestibular pathways through repeated exposure and brainstem habituation; strengthens postural reflexes.
- **Phase 3 (Weeks 4+: Advanced Integration):**
 - Incorporate dynamic balance (single-leg stance with perturbations or dual-task, e.g., head turns while counting backward).
 - If BPPV suspected (from Dix-Hallpike in diagnostic phase), perform Epley manouvre

Sessions: 3–5/week, 30–45 min; include home program with patient education on self-monitoring. Monitoring: Reassess VOMS/BESS weekly; advance if symptoms <2/10 on exertion; regress if exacerbation >mild and >1 hour.

Rationale: Promotes multisensory reweighting (proprioception + vestibular) for real-world stability.

Oculomotor

Common Symptoms:

- Visual disturbances, difficulty tracking/focusing
- Eye strain, diplopia (VOMS oculomotor/convergence positive).

Physiological Mechanism:

- Trauma affects cranial nerves III/IV/VI, midbrain/superior colliculus, or cortical eye fields, impairing saccades, pursuits, vergence, and accommodation.
- Rehabilitation drives neuroplasticity in parietal/frontal eye fields and midbrain, improving binocular coordination and neural synchrony.

Recovery Trajectory & Prognosis

- **Acute Phase** (0–7 days): Deficits in 60–70% of cases, often resolving 50% spontaneously.
- **Subacute Phase** (1–4 weeks): Persist in 20–40%; rehab resolves 70–90% by 4–6 weeks.
- **Prognosis:**
 - Excellent with targeted vision therapy (80–95% resolution)
 - Pediatric cases leverage higher plasticity but monitor academic impact
 - Adults/elderly may have slower vergence recovery due to age-related lens changes.

Current evidence highlights office-based therapy reversing convergence insufficiency faster than waiting for natural symptom resolution.

Logical Rehabilitation Progression

- **Phase 1 (Weeks 1–2: Foundation):**

SSTAE to support visual-vestibular integration.

Basic saccadic exercises (rapid eye shifts between two targets 30–50 cm apart, horizontal/vertical; 10–20 reps per set, 3–5 sets/day).

Rationale: Enhances rapid refixation via superior colliculus plasticity, reducing tracking strain.

- **Phase 2 (Weeks 2–4: Pursuit and Vergence):**

Add smooth pursuit drills (track slow-moving target in H-pattern or circles, head still; 1–2 min per direction, progress to faster speeds or VOR cancellation with head movement).

Introduce convergence training (pencil push-ups: focus on tip approaching nose until double, then refocus; 10–20 reps).

Rationale: Strengthens parietal/cerebellar pursuit pathways and midbrain fusional vergence amplitude.

- **Phase 3 (Weeks 4+: Advanced):**

Incorporate accommodation flips (alternate focus near 10 cm/far 3 m cards, 10 reps) and dynamic integration (vergence during walking or with head turns).

Use **Brock string** for feedback.

Rationale: Trains ciliary muscle and accommodative pathways for real-world tasks like reading/driving. Sessions: Daily, 10–20 min bursts to avoid fatigue; collaborate with optometrist for lenses if needed. Monitoring: Near point of convergence (goal <6 cm sustained); King-Devick test weekly.

Cognitive

Common Symptoms:

- Brain fog, slowed processing, memory/attention issues, mental fatigue (cognitive screening deficits on ImPACT/SCOAT6).

Physiological Mechanism:

- Neurometabolic crisis disrupts frontoparietal networks and hypoperfusion
- Fatigue from mitochondrial inefficiency and energy mismatch.

Rehab enhances synaptic strengthening, cerebral blood flow, and executive function via Brain Derived Neurotrophic Factor /upregulated plasticity.

Recovery Trajectory & Prognosis

- **Acute Phase (0–7 days):** Peak cognitive slowing/fatigue 3–7 days.
- **Subacute Phase (1–4 weeks):** 70–80% resolve 10–14 days with active intervention.
- **Persisting (>4 weeks):** 10–20%, often linked to high initial load or pre-injury factors; good prognosis (80% recovery by 3 months). 2023–2025 evidence shows early cognitive activity predicts better outcomes, with persisting symptoms impacting Quality of Life.

Logical Rehabilitation Progression

- **Phase 1 (Weeks 1–2: Foundation):**

SSTAE as primary (e.g., walking to improve perfusion/BDNF). Introduce paced cognitive loading (short sessions of memory games/apps like Lumosity, 10–15 min with 5-min breaks). Rationale: Gradual stimulation prevents overload while promoting prefrontal plasticity.

- **Phase 2 (Weeks 2–4: Loading):**

Add dual-task training (cognitive + physical, e.g., walking while counting backward by 7s; 10–20 min). Incorporate metacognitive strategies (daily planners, task chunking). Rationale: Reintegrates divided attention networks, reducing fog.

- **Phase 3 (Weeks 4+: Integration):**

Progress to complex simulations (e.g., work/school tasks with pacing). Rationale: Builds compensatory efficiency for real-world demands. Sessions: 4–6/week, 20–30 min; monitor for fatigue. Monitoring: ImPACT/computerized tests weekly; advance if accuracy >80%.

Migrainous/Headache

Management of post-traumatic headache (PTH) is guided by the American Headache Society's emphasis on non-pharmacological interventions. For affective symptoms, integrated Cognitive Behavioral Therapy (CBT) and exercise are superior to isolated treatments¹³⁵.

Common Symptoms:

- Headache with migraine features (throbbing, photophobia, nausea).

Physiological Mechanism:

- Central sensitization of trigeminovascular pathways; often cervicogenic from upper cervical dysfunction.

Rehab activates descending inhibition (opioid/endocannabinoid systems) and reduces nociceptive input.

Recovery Trajectory & Prognosis

- **Acute Phase (0–7 days):** Headaches in 80%, peak 24–72 hours.
- **Subacute Phase (1–4 weeks):** 50–70% reduction; persisting in 15–30%.
- **Prognosis:** 60–80% resolution with phenotype-driven care; higher chronic risk if pre-migraine history. Current evidence supports supplements/CGRP for faster recovery.

Logical Rehabilitation Progression

- **Phase 1 (Weeks 1–2: Foundation):**

SSTAE + trigger identification (logs for sleep/hydration/stress). Rationale: Reduces chronicity via anti-inflammatory effects.

- **Phase 2 (Weeks 2–4: Cervical/Relaxation):**

Cervical strengthening (chin tucks/isometrics, 10–15 reps, 3 sets/day). Add diaphragmatic breathing (10 min/day). Rationale: Stabilizes C1–C3, reduces referred pain.

- **Phase 3 (Weeks 4+: Exposure):**

Graded light/noise exposure; biofeedback if available.

Rationale: Desensitizes sensitized pathways. Sessions: Daily, 20–30 min. Monitoring: Headache diary; escalate to CGRP if >3 days/week (Ashina et al., 2024).

¹³⁵ Ashina, H., Terwindt, G. M., Steiner, T. J., Silberstein, S. D., Lipton, R. B., Ashina, M., & Jensen, R. H. (2024). Post-traumatic headache: Epidemiology, diagnosis, and management. *The Lancet Neurology*, 23(3), 295–308.

Affective

Common Symptoms:

- Emotional dysregulation, anxiety, depression.
- Irritability, mood swings, emotional lability, apathy.
- Sleep disturbances, fatigue tied to mood alterations.
- Somatic complaints like appetite changes or psychomotor agitation/retardation overlapping with depressive phenotypes.

Physiological Mechanism:

- Limbic-frontal disruption, inflammation affecting neurotransmitters
- Cycles of fear-avoidance.
- Inflammation affecting neurotransmitters: Post-injury neuroinflammation disrupts serotonin, dopamine, and norepinephrine balance, contributing to mood instability and anxiety.
- Secondary effects: Disrupted hypothalamic-pituitary-adrenal (HPA) axis from stress response, exacerbating cortisol dysregulation and emotional symptoms¹³⁶

Rehabilitation: Integrated exercise and psychological interventions boost BDNF/neurogenesis, rewire amygdala-prefrontal connectivity, and restore HPA axis balance through neuroplasticity and anti-inflammatory effects.

Recovery Trajectory & Prognosis

- **Acute Phase (0–7 days):** Symptoms often emerge 3–7 days post-injury (prevalence 20–40%).
- **Subacute Phase (1–4 weeks):** Typically resolve within 2–4 weeks with appropriate management.
- **Prognosis:**
 - Good overall (70–90% resolution by 4–6 weeks)
 - Persisting symptoms in 10–25%, significantly higher with pre-existing mood disorders or multiple prior concussions
 - Adolescents at elevated risk for prolonged affective symptoms; elderly may overlap with age-related mood changes.

¹³⁶ Iverson, G. L., et al. (2023). "The Role of Premorbid and Post-injury Mental Health in Recovery from Sports-Related Concussion." *British Journal of Sports Medicine (BJSM)*.

Logical Rehabilitation Progression

- Phase 1 (Weeks 1–2: Foundation):

Initiate SSTAE as core (e.g., walking/cycling at 80% symptom threshold, 20 min/day) to elevate BDNF and endorphins for early mood stabilization¹³⁷

- Phase 2 (Weeks 2–4: Psychological Integration):

Introduce basic CBT techniques (thought challenging, cognitive reframing, 10–15 min/day) and mindfulness practices (guided body scans or breathing focus).

- Phase 3 (Weeks 4+: Activation and Reintegration):

Emphasize behavioral activation (scheduled pleasant/social activities) and graded social exposure. Sessions: 3–5/week, 20–30 min. Monitoring: GAD-7/PHQ-9 weekly; advance if scores improve >20%; regress if mood worsens.

Rationale

SSTAE in Phase 1 provides rapid mood boost via endorphin release and BDNF upregulation, laying foundation for psychological work.

Phase 2 CBT/mindfulness targets cognitive distortions and amygdala hyperactivity, rewiring prefrontal regulation.

Phase 3 behavioral activation breaks fear-avoidance cycles, promoting dopamine reinforcement and social reintegration for sustained recovery.

¹³⁷ Leddy, J. J., et al. (2024). "Early Sub-Symptom Threshold Aerobic Exercise for Sport-Related Concussion: A Randomized Clinical Trial." *JAMA Pediatrics*.

Neuromusculoskeletal Involvement in Concussion

The Cervicogenic & Peripheral Interface

While the "Neurometabolic Cascade" explains the primary brain injury, the Neuromusculoskeletal Phenotype involves the mechanical and reflex-driven impairments of the neck, spine, and peripheral joints.

This phenotype often acts as a "great masquerader," producing symptoms like dizziness and headaches that mimic vestibular or cognitive phenotypes.

Physiological Mechanism: Mechanical forces exceed the threshold for cervical tissue injury , causing shear and strain on ligaments, facet joints, and the **trigeminocervical nucleus**.

Disruptions to cervical mechanoreceptors cause a "sensory mismatch" between the neck, eyes, and inner ear.

Recovery Trajectory & Prognosis

- **Acute Phase (0–7 days):** Neck pain/stiffness present in >80% of patients; positional vertigo (BPPV) may emerge within 24–48 hours.
- **Subacute Phase (1–4 weeks):** 60–70% resolve with targeted manual therapy and motor control training.
- **Prognosis:** Excellent (80-90%) with phenotype-driven care. Higher risk of chronicity if there is a history of prior neck trauma or if kinesiophobia (fear of movement) develops. 2024–2025 evidence shows early NMS rehab reduces the risk of PPCS by nearly 50%¹³⁸.

Logical Rehabilitation Progression

- **Phase 1 (Weeks 1–2: Foundation):** Manual therapy for upper cervical (C1-C3) mobility; gentle craniocervical flexion; canalith repositioning (Epley) if BPPV-positive.
- **Phase 2 (Weeks 2–4: Sensorimotor Integration):** Joint Position Error (JPE) training with laser trackers; DNF endurance; scapular setting; core stabilization (TA/Multifidus).
- **Phase 3 (Weeks 4+: Dynamic Loading):** Perturbation training; dual-task balance (cognitive + motor); return to sport-specific kinetic chain loading (e.g., throwing/tackling mechanics).

Rationale: Rehab reduces nociceptive input to the trigeminocervical nucleus, recalibrates cervical mechanoreceptors for balance, and restores the "top-down" motor control of deep stabilizers to prevent secondary limb injuries.

¹³⁸ Schneider, K. J., et al. (2023). "What interventions facilitate recovery after concussion? A systematic review." *British Journal of Sports Medicine*.

Cervical Spine & Headache Syndromes

The cervical spine is the primary driver of NMS symptoms, often acting as a "great masquerader" by mimicking vestibular or cognitive phenotypes through the **trigeminocervical nucleus**.

- **Whiplash-Associated Disorders (WAD):** Rapid acceleration/deceleration strains cervical ligaments and musculature, causing immediate or delayed neck pain and stiffness.
- **Cervicogenic Headaches (CGH):** Referred pain from the C1-C3 segments. These are typically unilateral, non-throbbing, and exacerbated by specific neck postures (Schneider et al., 2023).
- **Facet Joint Dysfunction:** Trauma to the zygapophyseal joints leads to localized pain, protective muscle guarding, and reduced segmental mobility.
- **Myofascial Pain Syndrome:** Development of hyperirritable trigger points in the **upper trapezius, SCM, and suboccipitals**.

These muscles often "splint" the head to provide stability, inadvertently causing referred "tension-type" pressure across the forehead and temples.

Vestibular & Postural Instability

NMS involvement disrupts the vital "bottom-up" feedback required for balance.

- **Balance Deficits:** Trauma to cervical mechanoreceptors causes "noisy" afferent input, leading to impaired proprioception. Patients may pass vestibular tests but fail the **Balance Error Scoring System (BESS)** due to poor cervical proprioception¹³⁹
- **Benign Paroxysmal Positional Vertigo (BPPV):** Biomechanical impact can dislodge otoliths into the semicircular canals, causing brief, intense room-spinning vertigo during head position changes (e.g., rolling in bed).

¹³⁹ Treleaven, J., et al. (2024). *Cervicogenic Dizziness and Postural Instability: The Role of Cervical Mechanoreceptor Dysfunction in Concussion Recovery*.

Shoulder & Upper Limb Dysfunction

The kinetic chain often compensates for cervical guarding or neural irritation.

- **Scapular Dyskinesis:** Altered neuromuscular activation of the serratus anterior and trapezius leads to "winging" or poor scapular rhythm, increasing the risk of secondary impingement¹⁴⁰
 - **Rotator Cuff Dysfunction:** Post-concussive changes in muscle tone and posture can lead to imbalances, resulting in shoulder pain during reaching or lifting tasks.
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Lumbopelvic Dysfunction

Concussion induces a "top-down" disruption of motor control, affecting core stability.

- **Core Instability:** Studies show delayed activation of deep stabilizers like the **transversus abdominis (TA) and multifidus** post-concussion, which increases the risk of subsequent lower extremity injuries¹⁴¹.
 - **Pelvic Misalignment:** Postural compensations for neck pain or dizziness can lead to shifted weight-bearing patterns, manifesting as sacroiliac joint (SIJ) or hip pain.
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¹⁴⁰ Lamba, M., et al. (2024). The Kinetic Chain in Concussion: Scapular Dyskinesis and Upper Limb Dysfunction Secondary to Cervical Proprioceptive Impairment

¹⁴¹ Howell, D. R., et al. (2023). *Delayed Neuromuscular Activation and Core Instability Post-Concussion: Implications for Lower Extremity Injury Risk*.

Targeted Rehabilitation Guide

Subsymptom-Threshold Aerobic Exercise (SSTAE)

- **Procedure:** Assess threshold via the **Buffalo Concussion Treadmill Test**. Prescribe exercise at **80–90% of the heart rate (HR)** reached at the point of symptom exacerbation (ex. 2/10 increase).
- **Purpose:** Restores cerebral autoregulation, upregulates **Brain-Derived Neurotrophic Factor (BDNF)** for neurogenesis, and normalizes the Autonomic Nervous System (ANS).
- **Evidence:** Early SSTAE (within 2–10 days) reduces the risk of Persistent Post-Concussive Symptoms (PPCS) by over **50%** (Leddy et al., 2024).

Positive findings:

- Symptom reduction: Decrease in dizziness/vertigo <2/10 on VOMS.
 - Improved balance: BESS score normalization.
 - Indicates central compensation in brainstem/cerebellum.
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Gaze Stabilization (VOR Adaptation)

- **Procedure:** VOR x1 exercises—fixing gaze on a target while rotating the head at **60–120 bpm** via metronome.
- **Purpose:** Recalibrates the **Vestibulo-Ocular Reflex (VOR)** gain through cerebellar synaptic adaptation.

Positive Findings:

- Reduced symptom exacerbation during head motion.
 - Smooth eye movements without nystagmus.
 - Indicates brainstem/cerebellar plasticity.
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Habituation & Motion Integration

- **Procedure:** Graded exposure to provocative movements (e.g., Optokinetic stimulation or head turns while walking).
- **Canalith Repositioning:** If Dix-Hallpike is positive, perform the **Epley Maneuver** to resolve BPPV by repositioning displaced otoliths.
- **Visual Motion Sensitivity (VMS):** Integrating complex visual environments (e.g., grocery store aisles) with balance tasks to resolve sensory mismatch.

Positive Findings:

- Tolerance to motion without vertigo/nausea.
 - Reduced visual motion sensitivity.
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Dynamic Balance & Sensorimotor Training

- **Procedure:** Progress from static tandem stance to dynamic dual-tasking (e.g., balancing on foam while performing mental arithmetic).
- **Cervical Component:** Utilizing laser head-trackers for **Joint Position Error (JPE)** training to recalibrate cervical proprioceptors (Treleaven, 2024).

Positive Findings:

- Improved postural control on BESS.
 - No imbalance during activity.
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Module 7 Summary: Rehabilitation Pathways

"From Rest to Recovery: The Medical Clinician's Guide to Active Concussion Rehab" *Design, prescribe, and progress targeted interventions for physiological, mechanical, and neurological restoration.*

Learning Objectives Achieved ✓

- Integrate phenotype pathophysiology into tailored, evidence-based rehab plans.
 - Prescribe Subsymptom-Threshold Aerobic Exercise (SSTAE) using BCTT data.
 - Master the "Cervicogenic-Vestibular" differential diagnosis for clinical clearance.
 - Apply kinetic chain assessments (Lamba et al., 2024) to prevent secondary injury.
 - Implement integrated CBT-Exercise protocols for Affective phenotypes.
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1. Foundations: The Physiological Paradigm Shift

The Neurometabolic Rationale for Movement We no longer treat concussion as a structural injury to be "healed" by rest, but as a functional metabolic gap to be "closed" by graded physiological stress.

- Autonomic Dysregulation: Concussion impairs the relationship between Mean Arterial Pressure (MAP) and Cerebral Blood Flow (CBF). SSTAE re-trains the autonomic nervous system.
 - The BDNF Effect: Controlled exercise upregulates Brain-Derived Neurotrophic Factor, facilitating synaptic repair and reducing the "Metabolic Brain Gap."
 - The "Rest is Toxic" Threshold: Prolonged rest (>48 hrs) is a predictor of Persistent Post-Concussive Symptoms (PPCS) due to deconditioning and placebo effects.
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2. Core Intervention: The SSTAE Prescription

Buffalo Concussion Treadmill Test (BCTT) Mastery Clinicians must move beyond "walking" to specific heart rate (HR) dosages.

Step	Action	Metric
1. Assessment	Increase incline 1% every minute at 3.2–3.6 mph.	Stop at symptom flare ≥ 2 points.
2. HR Threshold	Identify the HR at the point of symptom exacerbation.	e.g., 150 bpm.
3. Prescription	Prescribe at 80% to 90% of that threshold.	Script: 120–135 bpm.
4. Frequency	20 minutes daily (stationary bike/walking).	No contact, no high-impact.
5. Progression	Increase HR by 5–10 bpm every 2 weeks.	Only if asymptomatic post-exercise.

3. Phenotype-Specific Rehabilitation Pathways

A. The Vestibular & Oculomotor Pathway

- Mechanism: Sensory mismatch between the visual, vestibular, and somatosensory systems.
- Target: Recalibrating the Vestibulo-Ocular Reflex (VOR).
- Progressive Loading:
 - Level 1: Gaze Stabilization (VOR x1) – seated, 60 bpm.
 - Level 2: Habituation – moving targets, complex backgrounds (Visual Motion Sensitivity).
 - Level 3: Dynamic Integration – balance drills on foam while performing saccades.

B. The Neuromusculoskeletal (NMS) Pathway

- Mechanism: Trigeminocervical Nucleus "priming" via C1-C3 mechanical irritation.
- Target: Restoration of "Bottom-Up" proprioceptive feedback.
- Key Reference: Treleaven et al. (2024) – Cervical mechanoreceptor "noise" causes BESS failure even when the vestibular system is intact.
- Intervention:
 - Manual therapy (segmental glides C1-C3).
 - Joint Position Error (JPE) training with laser head-trackers.
 - Deep Neck Flexor (DNF) endurance training.
- Mechanism: Neuroinflammation and HPA-axis disruption (Iverson et al., 2023).
- Target: Rewiring amygdala-prefrontal connectivity.
- Intervention:
 - Early SSTAE (BDNF boost).
 - Integrated Cognitive Behavioral Therapy (CBT) for "Fear-Avoidance" cycles.
 - Social Reintegration: Scheduled "Low-Noise" social exposures.

4. The Kinetic Chain: Preventing Secondary Injury

Lamba, Howell, and Reneker Frameworks Concussion is a whole-body event. Failure to address the kinetic chain leads to MSK injury upon Return-to-Play.

- Scapular Dyskinesis (Lamba et al., 2024): Cervical guarding inhibits the serratus anterior, leading to winging and shoulder impingement.
 - Lumbopelvic Instability (Howell et al., 2023): "Top-Down" motor control disruption causes delayed activation of the Transversus Abdominis (TA), increasing ACL/Ankle injury risk.
 - Pelvic Misalignment (Reneker et al., 2025): Postural compensation for dizziness leads to SIJ weight-shifting and hip pain.
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5. Recovery Trajectory Guide (Typical Cases)

Age Group	Expected Symptom Duration	Subacute Rehab Focus	Full Clearance
Children (6–12)	1–2 weeks	Academic pacing + NMS	~3 weeks
Teens (13–18)	2–3 weeks	VRT + Social Integration	~4 weeks
Adults (19–60)	1–3 weeks	SSTAE + Work Simulation	~2–4 weeks
Seniors (60+)	3+ weeks	Fall prevention + Vestibular	6+ weeks

6. Clinical Clearance & Documentation Checklist

The "Medical Shield" for Practitioners Before clearing a patient to move from Rehab (Module 7) to Full Return (Module 8), you must document:

- Physiological Recovery: Patient reached 90% Max HR on BCTT without symptom flare.
- Vestibular Recovery: VOMS provocation scores back to baseline (≤ 2 point change).
- NMS Recovery: Joint Position Error (JPE) within 4.5 degrees of center.
- Cognitive Endurance: Patient tolerating full school/work day without "rebound" headache.
- Kinetic Chain: Normal scapular rhythm and core engagement during dynamic movement.

7. Key Takeaways for Clinical Practice

1. Symptom-Limited, Not Symptom-Free: We no longer wait for 0/10 symptoms to start rehab. We load up to the "Threshold."
2. Dampen the Fire First: If a patient has an active Migraine and Vestibular dysfunction, stabilize the Migraine (NMS/Affective/Medical) *before* loading the Vestibular system.
3. Core Stability is Neurological: If a post-concussion patient cannot engage their TA, it is a brain-output issue, not a "weak muscle" issue.
4. Multidisciplinary Threshold: If a patient plateaus for >2 weeks, escalate to a Neuro-Ophthalmologist or Neuropsychologist.

APPENDIX: Module 7 Quick Reference Tools

- The 80% HR Formula Template
 - Joint Position Error (JPE) Scorecard
 - VOMS Scoring & Interpretation Guide
 - Phenotype Overlap Logic Flowchart
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"The goal of rehabilitation is not simply the absence of symptoms, but the restoration of the brain's capacity to handle the physiological and mechanical demands of the patient's specific life."

Module 8: Legal, Ethical, Communication and Documentation Considerations

“Do No Harm: Legal Duty and Ethical Clarity in Concussion Cases”

Navigate medico-legal risk, record-keeping, and ethical dilemmas with clarity and compliance.

- **Learning Objectives:**

- Summarise legal responsibilities and documentation standards for concussion cases.
- Apply ethical and professional guidelines in return-to-activity decision-making.
- Legal requirements for managing sports-related concussions.
- Ethical considerations in return-to-play and return-to-work decisions.
- Documentation and record-keeping for concussion cases.

Rapid Clinical Decision-Making in Concussion Cases

Making the Call: Tools and Reasoning in Concussion Diagnosis

Develop diagnostic confidence through structured tools and real-world clinical reasoning.

By the end of this module, participants will be able to:

- Navigate **complex concussion cases** with confidence.
- Apply **clinical reasoning** to manage **uncertainty** in diagnosis and recovery.
- Decide when to **defer to caretakers** or caregivers for symptom management.
- Identify situations that require **immediate referral** for further evaluation.
- Make informed decisions to **monitor** or **review diagnoses** based on evolving symptoms.
- Use evidence-based strategies to guide patients' **return-to-activity plans**.

 Choose the **best** next step based on limited information. Multiple answers may seem **plausible**—use your clinical judgment.

Return-to-Play Protocols: In Australia, return-to-play protocols (such as the **Concussion in Sport Australia (CISA)** guidelines and **Sporting Club Concussion Management Plans**) require a **doctor's clearance** for an athlete to return to play after a concussion. This is typically outlined in national sports policy and is reinforced by state-based regulations.

Legal Responsibilities: Duty of Care in Concussion Management

As a healthcare practitioner, you owe a legal duty of care to your patients. This includes both the timely recognition and appropriate management of concussions.

Key Principles:

- Immediate and Appropriate Action: “If in doubt, sit them out.” This protects athletes and minimises risk.
 - Use of Guidelines: Follow evidence-based tools like the Australian Concussion Guidelines for Youth and Community Sport.
 - Communication Obligations: Inform athletes, parents, and relevant parties about the diagnosis, risks, and plan of care.
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Ethical Responsibilities in Clinical Concussion Decisions

Clinical care involves ethical decision-making, particularly under external pressure (e.g. coach, parent, or athlete desire to play).

Core Ethical Duties

1. Patient Welfare Primacy

- **Competitive demands never outweigh health considerations**
- Long-term brain health takes precedence over immediate performance
- Professional judgment must remain independent of external pressures

2. Informed Consent

- **Comprehensive education** on concussion risks and consequences
- **Include patients and guardians** in decision-making process
- **Document consent discussions** thoroughly

3. Confidentiality and Privacy

- **Maintain medical privacy** according to healthcare regulations
- **Share information only** with authorized individuals
- **Respect patient autonomy** in disclosure decisions

Managing External Pressures

Common Pressure Sources:

- Coaches demanding immediate return-to-play
- Parents prioritizing academic or sporting commitments
- Athletes minimizing symptoms for competitive advantage
- Employers requesting rapid return-to-work

Ethical Response Framework:

1. **Reaffirm clinical independence** and professional obligations
2. **Educate stakeholders** on concussion risks and recovery
3. **Document all pressure incidents** and responses
4. **Consult colleagues** when facing difficult decisions
5. **Prioritize long-term patient outcomes** over short-term pressures



Documentation Standards in Concussion Cases

Accurate, detailed records protect both the patient and the practitioner. They are critical for legal, ethical, and clinical reasons.

What to Include:

Must-Have Elements	Examples
Date/time of incident	“3:15pm during match on 12/06”
Observed and reported symptoms	“Dazed, slurred speech, reported headache”
Assessment tools used	“SCAT6: Balance off, orientation intact”
Management and referral actions	“Referred to GP same day; advised rest”
Informed consent discussions & RTP/RTW plans	“Explained risk; parent signed RTP form”
Clearance documentation	“Signed by Dr Jane Smith, 14/06”

Communicating Concussion with Clarity

Goal: Equip clinicians with the tools to confidently explain concussion using plain language, appropriate metaphors, and supportive communication tailored to each patient's context.

- Translate clinical terms into accessible patient-friendly explanations
 - Use a simple structure to describe concussion and its expected recovery
 - Reframe patient fears with reassuring, evidence-based analogies
 - Adapt communication to suit age, cognitive level, and context
 - Manage expectations around recovery with confidence and clarity
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Section 1: The 4-Step Explanation Framework

Use this structured approach to ensure every patient leaves the clinic with a clear mental model of their injury and a roadmap for recovery.

Step	Clinical Strategy	Patient-Friendly Language Example
1. Define It Simply	De-escalate the "Brain Injury" label while acknowledging the injury.	"A concussion is a temporary change in how your brain works. It's a functional injury—a 'glitch' in the system—rather than structural damage like a bruise or a bleed."
2. Normalize & Reframe	Validate symptoms as part of a natural process.	"It is completely normal to feel foggy, tired, or irritable right now. These aren't signs of permanent damage; they are signs that your brain is using its energy to recover."
3. Practical Do's & Don'ts	Move from "Rest" to "Pacing."	"Don't sit in a dark room; that can actually make you feel more anxious. Do stay active at a level that doesn't flare your symptoms by more than 2 points."
4. Set Expectations & Red Flags	Set safety boundaries while providing a positive prognosis.	"Most people feel significantly better within 2 weeks. However, if you experience worsening weakness or a worsening headache, we have a clear plan for that."

Section 2: Analogies That Work

Analogy help patients visualize the "invisible" injury. Choose the one that best fits your patient's lifestyle.

- The Dimmer Switch: "Your brain is still on—just a bit dimmed. Gradually, the lights come back up. If we turn them up too fast, the system flickers, so we find the right pace."
 - The Ankle Sprain for the Brain: "If you sprained your ankle, you wouldn't run a marathon the next day. You'd do rehab exercises first. Concussion is the same—rehab first, then return."
 - Software, Not Hardware: "It's like a software glitch—your hardware (the brain) is intact, but the software (how cells talk) needs a reboot."
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Section 3: What to Emphasize vs. Avoid

Language can either foster a "Fear-Avoidance" cycle or promote "Active Recovery."

Focus On (The Green Zone)	Avoid (The Red Zone)
Symptom monitoring without obsessing.	Hyper-vigilance: Tracking every minor fluctuation.
Balanced rest and pacing.	Total cocooning: Dark rooms and social isolation.
Routine, reassurance, and activity.	Catastrophizing: Focusing on permanent damage.
Stepped return to normal function.	Indefinite removal from work/school/sport.

Section 4: Clinical Scripts Across the Lifespan

1. The Teenager (13–18 years)

Context: Concerned about school, sport, and social life; high risk of minimizing symptoms.

- Plain Language Script: "Your brain got shaken up, like a snow globe. Things feel weird now—foggy, dizzy—but that's your brain healing. We're giving it time to settle, like a sprained ankle. That doesn't mean bed rest forever, but we'll ease back into school and sport step-by-step."
- Focus On: Stepped return to learning; social reassurance ("You aren't falling behind"); co-creating the recovery plan.
- Avoid: Jargon like "neurocognitive dysfunction"; overloading with rigid restrictions.

2. The Working Adult (19–60 years)

Context: Concerned about productivity, income, and "Brain Fog" affecting professional responsibilities.

- Plain Language Script: "You're used to high-speed processing, but right now your brain's 'battery' is smaller. If you try to run heavy programs (like 4-hour meetings) all at once, you'll crash. We're going to use a 'pacing' strategy—work for 25 minutes, rest for 5. This isn't a loss of intelligence; it's an energy management issue."
- Focus On: Sub-threshold work tasks; screen-time pacing; objective recovery markers (BCTT/VOMS) to show progress.
- Avoid: Vague timelines; suggesting "total time off" if light duties are available.

3. The Older Adult (65+ years)

Context: Worried about balance, memory, stroke, or permanent cognitive decline.

- Plain Language Script: "This is a mild brain injury. You may feel tired or dizzy—that's expected. Because of your medications and other health factors, we'll keep a close watch. This isn't a stroke; it's a bruise to your system that needs a few extra weeks of patience. You'll get a written plan, and we'll check in regularly."
- Focus On: Fall prevention; medication review; written (large print) instructions to support memory.
- Avoid: Fast-paced or jargon-filled explanations; assuming self-management without a caregiver present.

Section 5: Handling Difficult Questions

Question	Expert Clinical Response
"Nothing showed up on my scan—why do I feel like this?"	"Scans look for 'Hardware' issues like bleeding. They don't see 'Software' issues—the way your cells use energy. Your symptoms are real."
"Google says this causes CTE—should I be worried?"	"CTE is from repetitive exposure over years, not a single managed concussion. Managing this recovery correctly is the best way to protect your health."
"Can I just push through for my finals?"	"Pushing through 'red zone' symptoms is like running on a broken leg. It slows down healing. We will find a 'yellow zone' where you can study safely."
"I'm still not right after months—am I damaged?"	"No. Persistent symptoms usually mean a 'pathway' (like your neck or inner ear) is still out of sync. You just need a 'tune-up' to get things back in line."

Ethical Considerations for Practice

- The Nocebo Effect: Recognize that your words can literally slow down a patient's recovery. Use positive, active, and temporary language.
- Cultural Sensitivity: Adapt metaphors (e.g., "recharging a battery" vs. "software glitch") to fit the patient's cultural and linguistic background.
- Clarity vs. Complexity: Our ethical duty is to ensure the patient is an active participant in their care. If they can't explain the plan back to you, the explanation has failed.

The goal of communication is to move the patient from a state of 'vulnerability' to a state of 'agency.' When they understand the mechanism, they can master the recovery.

Module 8 Summary: Legal, Ethical, Communication and Documentation Considerations

Main Goal

Equip clinicians to explain concussion using plain language, appropriate metaphors, and supportive communication tailored to each patient's context.

Key Learning Objectives

- Translate clinical terms into patient-friendly explanations
- Use simple structure to describe concussion and recovery
- Reframe patient fears with evidence-based analogies
- Adapt communication to age, cognitive level, and context
- Manage recovery expectations with confidence

Core Communication Framework

4-Step Explanation Structure:

1. Define It Simply
2. Normalize & Reframe
3. Do's and Don'ts
4. Set Expectations & Red Flags

Key Analogies

- **Dimmer Switch:** "Brain is still on—just dimmed, lights gradually come back up"
- **Ankle Sprain for Brain:** "Wouldn't run on sprained ankle—same rehab principle"
- **Software Not Hardware:** "Software glitch—brain intact, needs reboot"

Age-Specific Approaches

- **Teenagers (15):** Focus on school/sport return, social reassurance
- **Adults (35):** Work pacing, productivity concerns, family responsibilities
- **Older Adults (65):** Safety, medication interactions, family involvement

Communication Principles

- **Focus On:** Symptom monitoring, balanced rest, routine reassurance, stepped return
- **Avoid:** Medical jargon, over-reassurance without structure, dismissing concerns

Legal, Ethical, and Documentation Considerations

Learning Objectives Achieved ✓

- **Summarize legal responsibilities** and documentation standards for concussion cases
 - **Apply ethical and professional guidelines** in return-to-activity decision-making
 - **Navigate medico-legal risk**, record-keeping, and ethical dilemmas with compliance
 - **Advocate for patient-centered care** under external pressures
 - **Ensure professional accountability** and risk mitigation through accurate documentation
-

Legal Responsibilities: Duty of Care Framework

Fundamental Legal Obligations

Healthcare practitioners owe a **legal duty of care** to patients that encompasses both timely recognition and appropriate management of concussions.

Core Legal Principles

1. Immediate and Appropriate Action

- **"If in doubt, sit them out"** - Primary protective principle
- Minimizes risk to athletes and practitioners
- Legally defensible conservative approach

2. Evidence-Based Practice

- Follow **Australian Concussion Guidelines for Youth and Community Sport**
- Use validated assessment tools (SCAT6, King-Devick)
- Document adherence to established protocols

3. Communication Obligations

- **Inform all relevant parties:** athletes, parents, coaches, employers
- **Clear explanation** of diagnosis, risks, and care plan
- **Written documentation** of all communications

Australian Legal Framework

Return-to-Play Requirements:

- **CISA Guidelines** mandate medical clearance for return-to-play
- **Sporting Club Concussion Management Plans** require doctor's clearance
- **State-based regulations** reinforce national sports policy
- **Professional liability** for practitioners who fail to follow protocols

Key Takeaways for Practice

Documentation is Your Legal Shield: Comprehensive, contemporaneous records protect both patient care quality and practitioner liability.

Clinical Independence is Non-Negotiable: Professional judgment must remain free from external pressures, regardless of source.

Education Prevents Conflicts: Proactive education of stakeholders reduces pressure situations and improves compliance.

Conservative Approaches are Legally Defensible: "When in doubt, sit them out" is both ethically sound and legally protective.

Professional Development is Essential: Staying current with guidelines and best practices is both a legal and ethical obligation.

"The intersection of law, ethics, and clinical care in concussion management requires practitioners to balance competing interests while never compromising on patient safety and professional integrity."

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Appendix

Appendix A - Assessment overview matrix

Module	Unit of Competency	Assessment Method	Learning Outcome Assessed
Module 1: Introduction to Concussion	HLTAAP002 – Recognise healthy body systems	Short-answer quiz + labelled diagram	Identify and explain mechanisms of TBI and affected neuroanatomy
Module 2: Diagnosis & Initial Assessment	HLTAAP003 – Analyse and respond to client health information	Case-based SCAT/SCOAT6 + symptom checklist analysis	Apply structured clinical tools to assess and document concussion
Module 3: Physical Testing & Acute Management	HTLWHS002 – Follow safe work practices for direct client care	Practical demonstration + SOAP note write-up	Demonstrate balance testing (e.g. BESS), VOR testing, and management plan creation
Module 4: Post-Concussion Syndrome (PCS)	CHCCCS020 – Respond effectively to behaviours of concern	Management plan + reflective case response	Formulate a long-term care strategy considering cognitive and emotional symptoms
Module 5: Multidisciplinary Management	CHCPRP003 – Reflect on and improve own professional practice	Team-based case planning + collaboration reflection	Differentiate roles of multidisciplinary professionals in concussion recovery
Module 6: Return to Work/Play Protocols	CHCDEV002 – Analyse impacts of sociological factors	Protocol design + patient education script	Design RTP/RTW plan including special populations and educational support
Module 7: Rehabilitation Pathways in Concussion Management	HLTAAP003 – Analyse and respond to client health information	Clinical prescription task + phenotype mapping and rehabilitation	Design and progress physiological and mechanical rehab plans (SSTAE, VRT, NMS)
Module 8: Legal, Ethical, Communication and Documentation Considerations	CHCLEG003 – Manage legal and ethical compliance	Communication roleplay + documentation audit	Apply ethical reasoning, simplify complex clinical concepts, and maintain compliant medical records

CPD Point attribution

Module	Assessment Type	Delivery Format	CPD Hours	Competency Code
Module 1: Introduction to Concussion	MCQ + Labelled Diagram	Written (In-person/Online)	1.0	HLTAAP002
Module 2: Diagnosis & Assessment Theory	Case-based SCAT Analysis	Group & Self-Assessment	1.5	HLTAAP003
Module 3: Physical Testing & Management	Practical Demonstration (BESS/VOR)	Facilitator-Marked Practical	2.0	HLTWHS002
Module 4: PCS & Long-Term Care	Reflective Case Write-Up	Written / Verbal Discussion	1.5	CHCCCS020
Module 5: Multidisciplinary Collaboration	Team-based Case Planning	Workshop Task	1.5	CHCPRP003
Module 6: Return to Play/Work Protocols	RTP/RTW Protocol Design	Written + Facilitator Check	2.0	CHCDEV002
Module 7: Rehabilitation Pathways	Clinical Prescription Task (SSTAE)	Written + Phenotype Mapping	2.5	HLTAAP003
Module 8: Legal, Ethics & Documentation	Ethics Quiz + Communication Roleplay	Facilitated Role-Play & Written Audit	2.0	CHCLEG003
TOTAL	—	—	14.0	—

Appendix B – Case Study Template (Blank)

Client Name: _____

Age/Gender: _____

Mechanism of Injury: _____

Initial Symptoms: _____

Red Flags Identified: _____

SCAT/SCOAT6 Summary:

- Cognitive Score: _____
- Balance Errors: _____
- VOMS Findings: _____

Initial Management Plan: _____

Referral Pathways: _____

Follow-up Plan: _____

Appendix C – Return-to-Play Protocol Template

Day 0–2: Rest (physical + cognitive)

Day 3–4: Light aerobic activity

Day 5: Sport-specific drills (no contact)

Day 6: Non-contact training drills

Day 7: Full contact practice (if cleared)

Day 8: Return to sport

Each stage requires 24 hours symptom-free before progression. Adjust as needed for paediatrics or non-sporting populations.

Appendix D – Practical Skills Observation Checklist

Skill	Demonstrated Competence	Notes
Performs SCAT/SCOAT6 orientation and memory test	<input type="checkbox"/> Yes <input type="checkbox"/> No	
Administers tandem stance and BESS	<input type="checkbox"/> Yes <input type="checkbox"/> No	
Conducts VOR/head impulse/smooth pursuit tests	<input type="checkbox"/> Yes <input type="checkbox"/> No	

Recognises abnormal findings for referral Yes No

Uses appropriate documentation format (SOAP) Yes No

Assessor Signature: _____
Date: _____

Appendix E – Marking Rubric Example: Case Study Assessment

Criteria	Excellent (5)	Competent (3)	Not Yet Competent (1)
Identifies red flags and differential diagnoses	Thorough and accurate	Mostly accurate	Incomplete or incorrect
Clinical reasoning aligns with current guidelines	Evidence-based and logical	Generally sound	Lacks coherence or outdated
SCAT/SCOAT6 or assessment tool used correctly	Fully complete and correctly interpreted	Minor omissions	Significant errors or omissions
Management plan addresses physical and cognitive domains	Well-structured and appropriate	Mostly clear	Vague or inappropriate
Referral and communication pathways are relevant	All appropriate disciplines included	One or more omissions	Lacks team-based approach

Minimum average score of 3 required for competency.

Appendix F – Concussion Case Documentation Checklist

- Date and time of injury
 - Client demographics and injury mechanism
 - Presence/absence of red flags
 - Cognitive and physical assessments (e.g., SCAT/SCOAT6, VOR)
 - Initial management plan
 - Consent obtained and documented
 - Referral pathways initiated
 - Follow-up plan scheduled
 - Progress notes in SOAP format
 - Documentation meets legal and ethical standards (AHPRA Code)
-

Appendix G – Example: Completed SCAT/SCOAT6 Summary Page (Template)

(Include a sample completed SCAT/SCOAT6 form — or insert a blank with guided instructions.)

Appendix H – Course Evaluation Form

Please rate the following on a scale of 1 (Poor) to 5 (Excellent):

1. Relevance to your scope of practice
2. Clarity and structure of the content
3. Usefulness of case examples and clinical tools
4. Confidence gained in managing concussions
5. Overall satisfaction with the course

Optional feedback:

Suggestions for improvement:

Would you recommend this course to a colleague? Yes No

Appendix I – CPD Skills Log

Date	Skill Practised	Brief Description	Supervisor (if applicable)	Signature
	VOR testing	Practice with peer + instructor feedback	Facilitator	_____
	SCAT/SCOAT6 T6 use	Simulated case form completed	Self-directed	_____
	RTP protocol	Created plan for return to school scenario	Facilitator	_____

Appendix J – Glossary of Terms

- **SCAT/SCOAT6:** Sport Concussion Assessment Tool, 6th Edition
- **PCS:** Post-Concussion Syndrome
- **DAI:** Diffuse Axonal Injury
- **VOR:** Vestibulo-Ocular Reflex
- **SOAP:** Subjective, Objective, Assessment, Plan
- **RTW/RTP:** Return to Work / Return to Play
- **BESS:** Balance Error Scoring System
- **CTE:** Chronic Traumatic Encephalopathy

Appendix K – Key References & Resources

Useful Links:

- <https://www.ahpra.gov.au> – AHPRA Code of Conduct
- <https://bjsm.bmj.com> – SCAT/SCOAT6 official tools
- <https://braininjuryaustralia.org.au> – National advocacy and support
- <https://concussionfoundation.org> – CTE and long-term management
- <https://returntoplay.org.au> – RTP protocols and flowcharts

Additional Resources (Web + Clinical Tools)

- **SCAT/SCOAT6 Clinical Tool** – Download SCAT/SCOAT6 from CISA
 - **VOMS Protocol Summary** – Documents package
 - **BESS Balance Test Protocol** – Documents package
 - **Return to Learn Framework (Children)** – [Insert Dept. of Education concussion guideline link]
 - **Neuropsych Referral Decision Tree** – [Visual tool, include or reference appendix diagram]
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Recommended Reading List

- Giza & Hovda (2001). *The Neurometabolic Cascade of Concussion*.
- McCrory et al. (2017). *Berlin Consensus Statement on Concussion in Sport*.
- Schneider et al. (2013). *Systematic Review of Concussion Treatment Strategies*.
- Iverson (2019). *Network Analysis in Post-Concussion Syndrome*.
- Bigler (2015). *Neuroimaging Biomarkers in mTBI*.
- Lovell et al. (2004). *Recovery Time in Pediatric Concussion*.
- Purkayastha et al. (2019). *ANS Dysfunction Post-Concussion*.

SCAT6 & Clinical Tools Checklist

"Concussion Clinical Assessment Checklist"

Tool	Purpose	When to Use	Notes
SCAT6 (Adults)	Structured acute assessment	Sideline & clinic (within 72 hrs)	Must include symptom scale, memory, balance
SCOAT6 (Child/Adolescent)	Age-adapted SCAT	All <18yo patients	Symptom anchors are modified
VOMS	Vestibulo-ocular dysfunction	Any dizziness/blurred vision	Perform slowly, track symptom reproduction
BESS	Balance testing	Clinic baseline & return-to-play	Best when eyes closed, firm/foam surfaces
Red Flag Screen	Rule out structural injuries	Always pre-assessment	LOC, vomiting, neck pain = refer immediately
Symptom Diary	Daily tracking	First 7–14 days post-injury	Use to monitor subacute symptom resolution