Part I

Brain

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1 Sports-Related Head Injury

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Summary
Traumatic Brain Injury (TBI) is a significant public health concern. There are various classification systems used to describe injury severity. The majority of TBI are mild both in the general population as well as in athletes. Injuries occur more commonly in contact sports. In this chapter we describe the classification and pathophysiology of TBI and review complications and rehabilitation strategies to manage patients with an emphasis on mild, sports-related injuries.

Keywords: brain injury, sports, concussion

1.1 Traumatic Brain Injury Overview

1.1.1 Classification

Traumatic brain injury (TBI) is defined as an "alteration in brain function, or other evidence of brain pathology, caused by an external force."[1] Clinically, TBI severity is often based on the Glasgow Coma Scale (GCS). Mild injuries make up the overwhelming majority of sports-related injuries both in the general population engaging in recreational sports and professional athletes.[2] There are numerous scales to classify severity of brain injury, many of which incorporate some combination of the GCS level, duration of post-traumatic amnesia (PTA), duration of loss of consciousness (LOC), alteration of consciousness (AOC), and focal neurologic deficits.[3,4,5] Based on these scales, patients can be classified as having mild, moderate, or severe brain injury (▶ Table 1.1). Given the inconsistency of accurate reporting for both PTA and LOC, GCS scores are most commonly used clinically for assessing severity.[6] However, it is important to recognize that GCS alone provides insufficient information for capturing both extremes of severity and does not correlate well to outcomes.[7,8] Even with additional demographic information and examination findings, the accuracy of predicting outcomes is below 40%.[6]

The diagnostic criteria for mild TBI vary among different guidelines.[5,9,10] Within the world of sports, mild TBI is often used interchangeably with concussion. The consensus statement from the international Concussion in Sport Group (CISG) defines sports-related concussion (SRC) as an alteration of brain function caused by an external force that usually has "rapid onset of short-lived impairment of neurological function that resolves spontaneously," though signs and symptoms may "evolve over a number of minutes to hours." LOC is not required but possible.[11]

Table 1.1 Classification of injury severity

	Mild	Moderate	Severe
GCS	13–15	9–12	< 8
PTA	< 24 hours	24 hours–7 days	> 7 days
LOC	< 30 min	30 min–24 hours	> 24 hours

Abbreviations: GCS, Glasgow Coma Scale; PTA, post-traumatic amnesia; LOC, loss of consciousness.

Source: Adapted from Appendix C, Definition of mTBI from the VA/DOD Clinical Practice Guideline for Management of Concussion/Mild Traumatic Brain Injury (2009). In: O'Neil ME, Carlson K, Storzbach D, et al. Complications of Mild Traumatic Brain Injury in Veterans and Military Personnel: A Systematic Review [Internet]. Washington (DC): Department of Veterans Affairs (US); 2013. Available from: https://www.ncbi.nlm.nih.gov/books/NBK189784/

1.1.2 Epidemiology

Global estimates of TBI show an annual incidence of 69 million of which over 80% are mild.[12] Sports and recreational activities may be responsible for over 1.6 million TBIs with a similar predominance of mild injuries.[2] Severe brain injuries in sport are more often related to cycling and equestrian sports. In both groups, more severe injuries are seen in unhelmeted riders.[13] A study by Winkler et al further illustrates the importance of proper helmet use. They noted that winter board sports such as skiing and snowboarding where helmet use is high had lower mortality than roller sports such as skateboarding where helmet use is very low.[14]

Given that the majority of TBIs in sports are mild, databases have been set up looking at SRC across various sports associations. During the National Collegiate Athletic Association (NCAA) seasons 2014/15 to 2018/19, there were 4.13 mild TBIs per 10,000 athlete-exposures (AEs) reported for 23 sports, showing a decrease from 4.47 per 10,000 AEs in 2009/10 to 2013/14 reported for 25 sports.[15,16] Across both men's and women's collegiate sports, men's ice hockey had the highest rate of SRC at approximately 7.35 per 10,000 AEs, followed by women's soccer with 7.15 per 10,000 AEs. Men's track and field had 0.33 per 10,000 AEs and women's track and field reported 0.17 in 1,000 AEs.[15] An analysis of high school sports during the 2013/14 to 2017/18 school years found 4.17 concussions per 10,000 AEs across 20 sports.[17] American football had the most reported concussion at 10.40 per 10,000 AEs and girls' soccer registered 8.19 per 10,000 AEs.[17]

The highest reported mechanism of injury was player contact, which accounted for approximately 85.1% of concussions in collegiate men's American football and 83.8% in men's basketball.[15] Most SRC in women's sports was attributed to equipment/apparatus contact in collegiate and high school sports, although collegiate women's ice hockey (45.9%), women's soccer (42.5%), and women's basketball (64.1%) had a greater incidence of player contact.[15] Across both sexes, and disciplines, the majority of SRC occurred during competition, with 54.5% of concussions happening in the second half or third or fourth quarter.[15,17,18]

Partitioning for sex, there was a higher incidence of SRC in collegiate soccer, basketball, and softball/baseball in women relative to men.[15,19] A similar trend was demonstrated in high school, with higher SRCs in girls than boys at 3.35 versus 1.51 per 10,000 AEs, respectively. High school boys and collegiate

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men's ice hockey had the highest prevalence of recurrent concussion at 14.4 and 10.6%, respectively.[15,17]

1.1.3 Pathophysiology

Injuries to the brain can be described both by timing as well as by location. Patients can have injuries focally at the site of impact as well as diffusely due to global force from acceleration and deceleration. Examples of focal injuries include skull fractures, contusions, and hemorrhage.[20] Focal injuries cause specific functional deficits depending on laterality and lobe. The structure of the skull puts the temporal and frontal lobes at higher risk of injury. Damage to these areas is responsible for the neurobehavioral profile often associated with more severe TBI.[21]

Diffuse injuries include axonal injury. The different tissue densities within the brain makes the axons in white matter particularly susceptible to damage from rotational acceleration. Damage initially affects the axon membranes at nodes of Ranvier, causing disruption of ion transport, subsequent swelling, and potential retraction of the axon. More severe injuries can cause structural damage. Given the role of axons in connecting various regions of the brain, diffuse axonal injury (DAI) can cause a wide range of impairments, and the severity of DAI correlates with the duration of LOC.[22]

Diffuse and focal injuries present at the time of impact are referred to as primary injury and these trigger cascades in the following minutes to days causing secondary injury.[20] TBI initially causes disruption of cell membranes leading to glutamate and potassium release as well as accumulation of intracellular calcium. This is followed by energy consumption and a drop in glucose levels. The degree of ionic disruption and glucose depletion correlates with injury severity.[23] These changes occur in the immediate minutes to hours following injury but can lead to further downstream dysfunction in the following days from edema and oxidative stress.[20] Focal injuries such as ischemia and edema can also trigger these secondary cascades in a more localized fashion.[24]

Mild Injury

Mild TBI involves a less severe injury to the brain but as per the definition of TBI above, the force incurred needs to be of sufficient strength to alter brain activity.[25] Although many of the aforementioned diffuse processes occur to a degree, mild TBIs are usually without focal injuries. Some classification systems identify the presence of focal injury as consistent with moderate TBI whereas others label these cases complicated mild TBI.[25,26] Axonal injury is hypothesized to be the main mechanism of initial symptoms[27]; however, ion dysregulation is usually not severe enough to progress to secondary injuries. Therefore, recovery is possible before structural damage occurs.[22,24] This pathophysiology informed the CISG definitions and expected clinical course of SRC as already mentioned.[11]

1.2 Management Principles

The principles of management for TBI are different for mild injuries compared to moderate and severe injuries. In general, all patients require an initial neurologic examination to determine the level of severity and assess the need for further immediate care and stabilization. Additionally, depending on the mechanism of injury a full body trauma examination is often indicated to find concurrent injuries, especially in more severe cases.

1.2.1 Workup and Treatment

Moderate and Severe

Patients with more severe injuries will need prolonged observation or admission to an acute hospital to continue stabilization started in the outpatient setting. For patients with a GCS of < 15, a computed tomography (CT) scan is recommended. A major concern during the acute period is control of edema and ischemia by monitoring cerebral perfusion pressure (CPP) through the surrogate of intracranial pressure (ICP). Patients with GCS below 9 or significant vital sign or neurologic abnormalities are at the highest risk and should be monitored by invasive means.[6]

Moderate and severe TBI patients have a risk of seizure between 2 and 17% in the first week after TBI.[28] It is recommended that patients be given 7 days of seizure prophylaxis to reduce complications from seizure during this often critical time of medical instability. Prophylaxis is meant to address early seizures but does not prevent the development of late seizures after the first week. Early studies were done using older antiepileptics which have fallen out of favor due to their side effect profile.[29] Many practitioners now utilize levetiracetam, though higher quality studies are still needed.[30]

Once a patient is medically and surgically stable, assessment of the functional status is important to guide the next level of care. This is best done via a multidisciplinary rehabilitation team, even in the acute hospital, consisting of a physiatrist, physical and occupational therapists, and speech-language pathologists. Depending on the deficits, this team can work on medications and therapies to improve function and recommend the next level of care including acute inpatient rehabilitation where specialized teams can continue this work.

Given the heterogeneous population, there are not many medical interventions that are recommended for all TBI patients. Some randomized controlled trials have demonstrated efficacy for specific medications in specific instances including amantadine for disorders of consciousness.[31] Beyond this, the main guidance for medications in brain injury medicine is the adage "start low and go slow," in that medications of various drug classes can be used for cognitive and behavioral sequelae of injury but are started at lower doses and gradually increased.[32] Providers need to be aware that certain classes of medications can lead to delayed neurorecovery including benzodiazepines, drugs with anticholinergic effects, and antipsychotics with typicals often having greater impairment than atypicals.[33]

Mild

Baseline Assessment

A baseline assessment can aid in recognition of comorbid conditions and assist in diagnosis and return to play decisions.[11,34] One such validated tool is the Sports Concussion Assessment Tool (SCAT), which was originally developed in 2004 by the

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CISG for medical professionals to evaluate SRC for individuals 13 years or older.[35] Participants between the age of 5 and 12 years old utilize the Child SCAT5. Additionally, computerized neuropsychological testing can be performed preseason and can aid health care providers in tracking SRC and recovery.[34]

On-Field Assessment

Since its early conception, the SCAT underwent multiple revisions with the latest edition updated in 2016 as SCAT5. The test comprises immediate on-field assessments of red flag symptoms, observable signs, memory assessment questions, GCS, and cervical spine assessment. Athletes with signs of LOC, impact seizure, tonic posturing, gross motor instability, confusion, or amnesia should be removed from play.[34] Given the heterogeneity of concussive injury, combined with low sensitivity and specificity of assessment tests, close attention should be given to the athlete's symptoms.[36] For laypersons, the Concussion Recognition Tool 5 (CRT5) can be used to detect signs and symptoms of possible concussion.[37] Ultimately, if there are any concerns for SRC, the athlete should be removed from play for further evaluation. If the athlete is diagnosed with concussion, he or she must be excluded from play that day.

Off-Field Assessment

Additional tests off the field in a quiet, controlled environment consists of modified balance error scoring system (mBESS) to assess for postural stability, vestibular/ocular motor screening (VOMS) to evaluate oculomotor functions, neurocognitive function questions, and post-concussion symptoms scale from the SCAT5.[38] Clinicians should be mindful of an attenuation effect to detect SRC after 3 days following an inciting event.[35] During the off-field assessment, athletes should be evaluated for comorbid conditions such as attention deficit disorders, prior concussion, migraines, depression, and anxiety which may prolong recovery as detailed in the following.[39]

Routine use of CT, magnetic resonance imaging (MRI), or skull radiograph are not recommended without high clinical suspicion for intracranial structural injuries.[34] Clinical decision rules such as the Canadian CT Head Rule for patients older than 16 and the Pediatric Emergency Care Applied Research Network algorithm can help guide imaging decisions.[40,41]

Given the initial ion exchange and glutamate excitotoxicity following head injury, research is being done on the role of utilizing fluid biomarkers to aid in the diagnosis of concussion and prognosis.[38] Currently, there is no scientific consensus to recommend the use of biomarkers for management or diagnosis.[42] Recently, use of glial fibrillary acidic protein (GFAP) and ubiquitin C-terminal hydrolase L1 (UCH-L1) has been shown to aid prognosis in patients presenting with same-day injury with GCS of 3 to 12 and may be able to predict imaging findings.[43,44]

At this time, since there are no imaging or laboratory tests that can confirm the occurrence of concussion, clinicians need to use their judgment to assess the likelihood a concussion occurred. If LOC or posturing following trauma to the head or rapid deceleration with subsequent return to normal neurologic functioning is witnessed, one can be reasonably diagnose a concussion with certainty. In cases without such findings, the degree of force and chance of other diagnoses needs to be carefully considered to ensure the correct diagnosis, though it is always safer to manage acutely as if the patient did indeed suffer a mild TBI.[45] Allowing athletes to continue playing increases the risk of a feared complication of second impact syndrome. Second impact syndrome occurs when patients sustain a second concussion prior to the resolution of the first one, resulting in disruption of the brain's autoregulatory mechanism which can lead to severe brain injury, disability, or death.[46] Previous studies have identified young athletes (13 to 24 years old) and male gender to be associated with second impact syndrome.[46] This issue rose to prominence with the passing of a law in Washington state after the injury of a middle school student who suffered severe sequalae after returning to play with a concussion. The Washington state's Lystedt law which went into effect in 2009 requires athletes with suspected head injury or concussion to be removed from play and not return to practice or competition until evaluated by a medical provider.[47]

Rest

Relative cognitive and physical rest after initial injury has been the mainstay treatment for SRC to allow for optimal recovery.[11] Following the first 24 to 48 hours after the event, patients are encouraged to participate in light aerobic activities as symptoms permit to decrease delayed recovery (greater than 30 days of symptoms).[48] Any aerobic activities which exacerbate post-concussive symptoms scores by two or more points on the post-concussive symptom scale, a 22-question Likert scale with higher scores indicating greater severity, should be halted. Exercise tolerance testing, such as the Buffalo Concussion Treadmill Test, can be used to gradually promote athletes' return to activity.[48]

Rehabilitation

A multidisciplinary team approach is beneficial to identify deficits and address impairments. Common symptoms such as headache or dizziness may arise because of autonomic, cervicogenic, vestibular, or cognitive dysfunction.[49] Up to 81% of patients report dizziness within the first day following concussion.[50] Vestibular rehabilitation has been shown to be beneficial in patients with persistent impairments such as dizziness, gait abnormalities, and balance deficits.[50] Exercises consist of gaze stabilization, standing balance, walking with balance challenge, as well as canalith repositioning maneuver. Therapies aimed at addressing cervicogenic pain include manual treatment, cervical proprioception, and motor control.[49]

Given the transient nature of concussion pathophysiology, conservative symptom management is preferred for most symptoms. Headache can be managed with medications such as nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen. Athletes and parents should be educated on warning signs such as vomiting, changes in baseline mentation, and acutely worsening headaches, which should prompt immediate medical attention and consideration of advanced imaging.[51] Other pharmacological supplements have limited in vivo analysis with no FDA regulations, leading to their low recommendations for treatment.[34]

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Return to School

Although students report improvement in their learning ability with slow integration back to academics, many report initial challenges with poor attention, fatigue, and difficulty understanding materials.[52] On average, students are absent for 3 to 5 days following a concussion, with older students missing for a longer period.[53] Earlier return to school has been associated with lower symptom burden at 2 weeks post injury likely due to socialization, maintaining sleep–wake cycle, lower stress from missed work, and physical activities.[53,54]

A customized learning plan in collaboration with health care providers, students and parents, and the school is important for students to reintegrate into their academic environment.[51] Accommodations include appropriate workloads, extended time for tests, quizzes, and assignments, along with adequate breaks and an environment which does not exacerbate symptoms. Symptoms should be monitored, and adjustments of learning activities should progress such that students are back to full academic workload without symptom exacerbation. In this way, although it is not as easily measured, return to school should mirror return to play with increasing difficulty as the student masters activities without symptom exacerbation.

Return to Play

The Consensus Statement on Concussion in Sports characterizes a stepwise gradual return to sports protocol in six stages. Athletes graduate through each stage if their symptoms are not exacerbated. These stages consist of starting with symptom limited activity, light aerobic exercise, sports-specific exercise, noncontact training drills, full contact practice, and subsequent return to sport. If athletes experience exacerbation of their concussive symptoms in any of the stages, they must stop physical activity, rest for 24 hours, and resume the previous stage.[11] Individualized time frame may vary depending on age, sports, and medical history. Furthermore, athletes must demonstrate psychological readiness with progression in return to play protocol for final clearance.[34]

1.2.2 Complications

Mild

Majority of individuals with mild TBI have resolutions of symptoms, but the time course of recovery is unclear. In a study by the World Health Organization, most patients seemed to recover over the span of 3 months to 1 year.[55] Clinical recovery from concussion often precedes physiological recovery, and there is no single test that can be used to definitively mark the time of full recovery from concussion.[56] Many children recover from concussion within 4 to 12 weeks, and adults generally recover in 2 months.[11,57,58,59] The median time for return to work for adults is 1 to 2 weeks.[60]

In contrast, McCrea and colleagues studied 570 athletes with concussion. Of these, only 10% had a prolonged recovery as measured by persistent elevation of Graded Symptom Checklist score at the 1-week mark; however, their cognitive and balance testing returned to normal limits. These individuals had higher symptom scores early on as well.[61] The faster return compared with the general adult population may be due to differences in responsibilities and life stressors, general health at the time of injury, as well as the potential for underreporting symptoms in order to return to play. Underreporting may also explain why female athletes have higher rates of delayed recovery than males.[62]

There are specific underlying comorbidities that predispose patients to a prolonged recovery. These include prior concussion, ADHD, anxiety, learning disorder, and migraines.[39,63,64] Additionally, a prior history of mental health diagnoses increases one's risk for persistent symptoms. Similarly, initial symptoms including headaches or depression are a risk factor for concussion symptoms to be present for greater than 1 month.[62]

For those patients with delayed or incomplete recovery, the term post-concussive syndrome (PCS) or post-concussional disorder (PCD) has been used. The terms are defined differently by the Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV) and the International Classification of Diseases 10 (ICD-10), the biggest difference being the DSM definition of PCD requiring cognitive impairment to be present.[65] Neither definition provides a mechanism for why the symptoms persist beyond the usual recovery period.

Further highlighting that these symptoms are not clearly linked to brain pathology are studies which have looked at the rates of occurrence of PCS symptoms in healthy controls.[66,67] Based on this, the clearest guidance from Silverberg et al in their Synthesis of Practice Guideline for managing symptoms that persist after an initial head injury is to utilize best practices for those symptoms from the general population and to prioritize sleep, headaches, and mood issues. They reserve referral to specialty treatment teams once symptoms last for a month despite appropriate management of these key symptoms.[60]

Moderate and Severe

The long-term management of more severe brain injuries is complex and relates to the various sequelae of injury these patients may deal with. One main guiding principle is that acute changes in mental status or neurologic function require a thorough workup as multiple complications can arise at variable times post injury. This can include lab work for metabolic or infectious etiologies, repeat imaging, and patient and family interviewing. Some of the most common complications are seizures, urinary tract infections, pneumonia, and central nervous system infections, particularly in patients with history of neurosurgical intervention or skull fractures, hydrocephalus, or intraventricular hemorrhage. Patients are also at risk of other neurologic sequelae of injury including movement disorders, spasticity, and paroxysmal sympathetic hyperactivity.[6,68] The specific workup and management of these issues are beyond the scope of this chapter and readers are referred to the citations and brain injury medicine textbooks.

Preventative screening is possible for some other sequelae of TBI. Around 30% of patients have injury to the pituitary which can cause reversible symptoms that overlap with impairment from TBI.[69] An endocrine panel should be obtained if symptoms are noted. For a preventative screen, there is some debate on the timing and what lab work to include, but a screen at 3 to 6 months is reasonable.[69,70,71]

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Another common complication after TBI is sleep dysfunction. Some estimates suggest that every patient will experience sleep disturbance, at least initially after TBI.[72] Overall sleep disorder prevalence decreases over time after injury, but a large proportion of patients report it as a chronic issue.[72,73] Most commonly diagnosed are insomnia, obstructive sleep apnea, and circadian rhythm disorders. Sleep hygiene should be discussed to optimize recovery. Melatonin, an over-the-counter supplement, can be used to help facilitate sleep. If patients continue to have sleep disturbance, a referral to a sleep specialist is warranted.[51]

Lastly, depression is a common occurrence after TBI affecting 25 to 60% of patients. Screening can be done to allow for early intervention and prevent symptom progression.[33] This unfortunately is not always conducted and not always successful, and the rates of suicide in this population are 3 to 4 times higher than the general population.[74]

1.3 Clinical Pearls

- TBI is broadly defined as an alteration in brain function caused by force to the brain which can be a result of direct impact or significant acceleration and deceleration forces.
- Most injuries in the general population and in sports are mild TBI, which has a greater focus on outpatient management. SRC is defined as an alteration of brain function caused by an external force that usually resolves on its own.
- The most important aspect of SRC is identifying its occurrence to remove athletes from play and avoid potential further injury from subsequent impacts before the concussion has resolved.
- More severe brain injuries occur in fast sports with unhelmeted participants.

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