Diagnosis and Management of Acute Concussion

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Abstract

Mild traumatic brain injury (mTBI), or concussion, constitutes a significant percentage of the millions of TBIs sustained in the United States each year. Symptoms are typically short-lived, and may correlate to physiologic changes in the acute period after injury. There are many available tools that can be utilized on the sideline as well as in the clinical setting for assessment and diagnosis of concussion. It is important to use validated tests in conjunction with a thorough history and physical examination. Neurocognitive testing may be helpful in the subacute period. Management should begin with removal from risk if a concussion is suspected, and once diagnosis is made, education and reassurance should be provided. Once symptoms have resolved, a graded return-to-play protocol can be implemented with close supervision and observation for return of symptoms. Management should be tailored to the individual, and if symptoms are prolonged, further diagnostic evaluation may be necessary.

Keywords

- mild traumatic brain injury
- concussion
- diagnosis
- return-to-play

Traumatic brain injury (TBI), a major cause of death and lifelong disability, is a significant growing health problem. Approximately 75 to 80% of TBIs are mild in severity, resulting from closed head injury, sometimes referred to as concussion, and is especially prevalent in sports and among military personnel.[1,2] Between 1.6 and 3.8 million sport-related concussions are diagnosed each year, and recent studies have projected that 15 to 30% of those deployed in the conflicts in Afghanistan and Iraq (Operation Enduring Freedom/Operation Iraqi Freedom [OEF/OIF]) have sustained injuries to the head and neck region.[3–6] The majority of TBI occurs in males 15 to 24 years of age, reflecting those populations in which risk for injury is high.[1] Mild TBI (mTBI) accounts for over one million emergency room visits, with 40% of individuals suffering from long-term disability, thus making it a significant public health issue.[7,8] The Centers for Disease Control estimates that 5.3 million Americans are living with TBI-related disability.[9] Most patients recover completely from their mTBI, but up to 30% may continue to suffer symptoms long term with somatic, cognitive, and emotional issues including headaches, dizziness, memory difficulties, mood disorders, and anxiety.[10,11]

Traumatic brain injury is defined as "an injury to the head as a result of blunt trauma or acceleration or deceleration forces that result in one or more of the following conditions: any period of observed or self-reported: transient confusion, disorientation, or impaired consciousness; dysfunction of memory around the time of injury; or loss of consciousness lasting less than 30 minutes."[12] Traumatic brain injury severity is based primarily on the duration of loss of consciousness, duration of posttraumatic amnesia, and the Glasgow Coma Scale (GCS) score at time of evaluation. Mild TBI is characterized by a GCS of 13 to 15, with loss of consciousness (LOC) less than 30 minutes and duration of posttraumatic amnesia less than 24 hours.[13] The incidence of mTBIs presenting to EDs has significantly increased, indicating the importance of understanding the diagnosis and management of this important growing problem.[14,15] mTBI and concussion are overlapping terms that describe TBI on the less severe end of the spectrum. Concussion, as defined by the 4th International Conference on Concussion in Sport in 2012, "is a brain injury and is defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces." The injury may result from

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a direct blow, or may be "an 'impulsive' force transmitted to the head. Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously…may result in neuropathological changes… and results in a graded set of clinical symptoms that may or may not involve loss of consciousness." This definition also states that "no abnormality is seen on standard structural neuroimaging studies," and "in some cases symptoms may be prolonged."[16] For the purpose of this review, we will use the terms "mild TBI" and "concussion" interchangeably. However, there are some who consider these two terms to be different, showing that in children, better outcome is correlated with the term "concussion."[17]

Specific populations requiring special consideration with regards to mTBI include athletes and military personnel. These groups sustain a high number of injuries that may involve the brain as well as other parts of the body. They also make up a significant proportion of those who suffer brain injuries in the United States. Sports-related injuries account for approximately one-fifth of the TBIs in the United States,[15] with 55% receiving outpatient care and 34% receiving no medical care.[1,15] Most sports-related TBIs fall into the mild or moderate category. Studies of deployed military forces to Iraq and Afghanistan estimate that 20% of have suffered a head injury, with those sustained in combat most often due to blast injury.[18] However, studies show that TBI sustained in the military are more likely due to other mechanisms such as motor vehicle accidents, falls, training, or sports and assaults.[19] The prevalence of those who have suffered a mTBI ranges from 4.2 to 23%.[20] Often, symptoms of TBI and posttraumatic stress disorder (PTSD) coexist in armed forces personnel. In both of these populations (athlete and military), there may be less willingness to report an injury, as an athlete may not want to be taken out of his or her sport, similar to a soldier wanting to remain on duty. Therefore, it becomes especially critical to have adequate diagnostic tools to accurately determine the occurrence of mTBI in these groups.

Pathophysiology

Symptoms after mild TBI may be attributable to pathophysiological changes that occur in the brain after injury. Acceleration–deceleration of the brain within the skull results in a stretch and strain upon the white matter and directly to cortical areas. Our knowledge of mTBI pathophysiology derives primarily from studies in animal models, as well as from clinical studies of severe human TBI using invasive monitoring. Animal models characterizing pathological changes and functional correlates, particularly behavioral and cognitive impairments, include fluid percussion, controlled cortical impact, and weight drop injuries.[21] In addition, there are newer models of repeat mTBI/concussion that focus on functional impairment with minimal histological injury.[22–24] After impact occurs, a cascade of intracellular and extracellular processes occurs including neurotransmitter release, alteration in cerebral blood flow, mitochondrial dysfunction, and free-radical formation. Neuronal cell membrane disruptions and axonal stretching occur, with resulting indiscriminate movement of ions and neurotransmitters across the disturbed membrane.[25] Glutamate is released from presynaptic terminals and activates N-methyl-D-aspartate (NMDA) receptors, further exacerbating these ion shifts. Potassium is released extracellularly,[26,27] and the Na^+/K ATP-dependent pump attempts to re-establish ionic equilibrium with resulting depletion of energy stores. Extracellular potassium increases lead to further neuronal depolarization.[28,29] Calcium accumulation occurs intracellularly, leading to mitochondrial calcium overload with resulting mitochondrial dysfunction and oxidative stress.[30] This calcium accumulation has been shown in animal models to correlate with persistent cognitive deficits as detected on Morris water maze testing.[31,32]

In addition to the ionic and neurotransmitter dysregulation that occurs, changes in cerebral glucose metabolism have also been shown both in animal and human studies. There is an initial rapid increase in glucose uptake, which is followed by prolonged glucose metabolic depression.[33] This increase may be secondary to cellular energy needs to restore ionic balance.[34–36] Increased cerebral glucose metabolism is followed by a period of decreased glucose metabolism, and is proportional in magnitude and duration to injury severity in rat models[37] as well as in TBI patients.[38,39] This glucose metabolic depression may be due to multiple reasons, including decreased cerebral blood flow, reduced demand, and impaired glucose transporter function. Animal studies have shown a decrease in cerebral blood flow during the acute phase after injury, with a resultant mismatch between needs and glucose availability.[40] Additionally, there may be decreased expression of the GLUT1 transporter.[41]

Increased free-radical production may occur after injury, which causes free-radical scavengers to be overwhelmed, leading to oxidative damage.[42,43] This free-radical production may be the result of intracellular calcium accumulation that activates free-radical-producing enzymes concomitantly with decreased availability of reducing equivalents. The combination of increased intracellular calcium and reactive oxygen species leads to mitochondrial dysfunction, which in turn reduces energy production, thus potentially initiating cell death through apoptotic and necrotic pathways.[44–46]

In addition to the chemical changes occurring throughout the brain, stretching and shearing of axons throughout the white matter leads to diffuse axonal injury (DAI), particularly in the brainstem, corpus callosum, and frontal lobes.[47,48] Cell membrane permeability, membrane potential, and cytoskeletal disruption occurs, with axonal transport disturbance. Buildup of transport organelles leads to edema, and secondary axotomy may occur leading to "retraction balls." However, there is some debate as to whether DAI may or may not be the primary mechanism of symptomatology behind postconcussive symptoms.[49]

More recently, use of multimodal magnetic resonance imaging (MRI) techniques such as 1H-magnetic resonance spectroscopy (MRS) has furthered the understanding of metabolic changes that occur after mTBI. N-acetylaspartate (NAA) diminishes after mTBI, and may represent neuronal and/or mitochondrial dysfunction; however, its exact function is not completely understood.[50–52] MRS studies have

shown that this decrease in NAA can recover 30 days after injury.[50] However, the temporal range of decreased NAA has been shown to range from days to months to years after injury.[50,53–57] Interestingly, postconcussion reductions in NAA are exacerbated (greater reductions and longer duration—45 days) after repeated concussions.[50]

The pathophysiology of military-related TBI may be different than other TBI sustained from falls, blunt trauma, and sports-related concussions. In a retrospective study of inpatient admission from OIF, Bell et al found that of the 1,513 consultations made by the neurosurgery population, blast injury accounted for almost 60% of military-related TBI.[58] However, other estimates maintain that mTBI sustained in the military looks mechanistically and demographically like sport-related concussion.[19] Many characteristics of the blast determine the resulting effects, including the distance from the blast, whether the blast occurred in an open or closed space, pressure waves that may be reflected off surrounding surfaces, and characteristics of the improvised explosive device (IED).[59] Blast injury results from the transmitted acoustic wave through the brain (primary blast wave) and accompanying blast winds.[60] Vascular damage with hemorrhage and sometimes vasospasm may be triggered, which can lead to ischemia and further clinical deterioration.[61–63]

Assessment and Diagnosis

Defining a concussion or mTBI is difficult due to lack of objective measurements on which to base a diagnosis. Typically, history, symptom report, and clinical assessment are used to make the diagnosis. Ancillary testing may include imaging and cognitive or neuropsychological assessment. As defined above, TBI is an injury to the brain that results from either a direct blow or transmitted force to the brain that results in neurologic deficits. In terms of diagnosing a mTBI, these are injuries that result in less than 30 minutes of LOC, are associated with less than 24 hours of posttraumatic amnesia, and have a GCS at the time of initial evaluation of 13 to 15. One less clear consideration is whether intracranial injury should be taken into account when characterizing a TBI as mild. Studies have shown that those with complicated mild TBI have different outcomes from those with uncomplicated mTBI. However, defining complicated mild injuries is challenging, as early studies suggest that complicated mTBI should be defined as that with space-occupying lesions. More recently, researchers have suggested that complicated mTBI should be defined strictly as those injuries that have intracranial findings and/or a depressed skull fracture.[64–66] Even more vagueness occurs in defining intracranial injury. Furthermore, finding the intracranial injury requires imaging, which may include computed tomography (CT) and/or MRI, and there is no consensus as to which should be required to characterize the intracranial findings.[67,68] Early studies outlining outcome after mTBI characterize complicated mTBI as those with GCS of 13 to 15 and a space-occupying lesion, including contusion, hemorrhage, or hematomas.[69] Those patients as defined by CT with a space-occupying lesion showed greater cognitive deficit beyond one month after injury.[69] The Transforming Research and Clinical Knowledge in Traumatic Brain Injury (TRACK-TBI) study has focused on defining patients by utilizing a Marshall CT classification of 5 or 6 (indicative of complicated mTBI with pathological head CT findings) and determining long-term outcome.[70] Other recent studies also show that not only is long-term outcome worse with a complicated mTBI compared with uncomplicated mTBI, but that this outcome may be related to cognitive reserve.[68]

Concussion Assessment Tools

The Sports Concussion Assessment Tool (SCAT) is based on expert consensus from those attending the serial Concussion in Sport Conferences (CISs) as an acute measure to assess sport-related concussion on the sideline, or for other evaluation in the immediate period after injury.[16,71–73] It has been revised 3 times based on expert consensus and scientific evidence.[16,72–74] The Third CIS Conference produced a PocketSCAT2 that was designed as a brief sideline tool, whereas the SCAT2 is a longer version, intended for tracking recovery serially.[16] The Fourth CIS Conference produced the most recent version of the SCAT3 and differs in scoring (omission of 100-point scoring and scoring of symptoms and balance) and the addition of tandem gait in addition to, or in place of the Modified Balance Error Scoring System (M-BESS).[73,74] Components of the SCAT3 include a symptom questionnaire (Graded Symptom Checklist [GSC]), cognitive assessment (Standardized Assessment of Concussion [SAC]), rating of physical signs, a coordination exam, and a balance examination (M-BESS), as described in more detail below.[73] These components have been shown to be reliable, sensitive, and specific.[74] In addition to an updated SCAT3, the Fourth CIS Conference produced a Child-SCAT3 designed for children between 5 to 12 years of age.[73] However practical, the Child-SCAT3 is not yet validated.

The Military Acute Concussion Evaluation (MACE) is a correlative screening test used in the military setting.[75] It is comprised of historical and objective sections, and has been used to some extent to make return-to-duty decisions.[76] The historical portion essentially covers the data regarding the injury and any acute signs and symptoms including the presence of amnesia and LOC, as well as the presence or absence of nine symptoms. The symptom score alone has been shown to have poor accuracy; furthermore, it does not improve diagnosis when used with the SAC.[77] The objective portion is made up of the Standardized Assessment of Concussion (SAC), which is described below. There are several limitations to the MACE, including inconsistent obtainment of baseline evaluations in service members, and therefore reliance on an absolute score of < 24 to diagnose a concussion.[78] A 2010 study showed that the MACE lacked sensitivity and specificity, and therefore was not valid if administered more than 12 hours after injury.[78]

Maddocks Sideline Questions

One component of the SCAT3 designed to quickly assess attention and memory is the Maddocks test.[79] Prior to the development of this test, concussion diagnosis relied on the

length of LOC and posttraumatic amnesia to determine severity, failing to acknowledge the large number of sport-related concussions that do not result in LOC. Maddocks proposed that traditional orientation questions were not sensitive enough to diagnose the milder injuries sustained in sports, and suggested that game-oriented questions may be more appropriate.[79,80] In a study of 28 players diagnosed as having suffered a concussion, he determined that questions specific to the game/sport, such as "Which quarter?," "Last goal?" "Who was the opponent last week?" may be more sensitive than traditional orientation questions for the diagnosis of mild concussion sustained in sport.[79]

Graded Symptom Checklist/ Postconcussion Symptom Scores

Symptoms after a concussion may vary, and measurement depends on self-report, making accurate quantification difficult. These symptoms typically resolve in the first week after injury for the majority who sustain mTBI.[81–83] The Graded Symptom Checklist (GSC) on the SCAT versions is a subjective measure that can be documented for an individual, useful for comparing pre- and postinjury scores as well as monitoring symptoms throughout recovery.[73] The symptom score is made up of a 22-item scale using a 7-point Likert scale. The scale has been shown to be reliable and valid, and may be used in the acute assessment as well as serially during recovery to assess improvement.[84–86] On the Child-SCAT3, there are both self and parental reports of symptoms.[73] However, these have not yet been validated.

Standardized Assessment of Concussion

The Standardized Assessment of Concussion (SAC) was developed for use immediately after an injury, and has been validated for evaluation in a sport-related concussion context.[87] Studies show sensitivity in the first 48 hours following a concussion.[82,88] The validation studies rely on changes from baseline rather than one-time scores to diagnose impairment after injury.[89–91] When used shortly after an event, the SAC has good sensitivity and specificity for distinguishing cognitive problems after concussion (sensitivity 80–94%, specificity 76–91%).[89,90] Kennedy et al showed that the SAC was only useful in the first 6 hours after injury, and in a military population did not predict outcome.[92] The SAC is a useful screening tool for detecting mTBI-related cognitive impairment, and responses to the psychologically grouped symptoms may even help to predict persistent postconcussive symptoms.[77]

Balance Error Scoring System

Measurement of postural stability using brief sideline assessment tools has been one of the diagnostic methods proposed for objective evaluation of mild head injury. Maintenance of balance requires multiple sensory inputs and outputs to the muscles. A clinical, practical, and cost-effective method, the BESS, has been developed as a standardized, quick sideline measurement of postural stability. The patient holds three different stances (double leg, single leg, tandem) on a firm surface and a medium density foam pad, each for 20 seconds with hands on hips and eyes closed. Errors are counted and summed to a maximum error score of 10 per trial.[93] The SCAT3 relies on a M-BESS, only assessing an individual's balance by evaluating the three stances on the flat ground.[73] The full BESS (including foam pad) has good specificity (91%), but only moderate sensitivity (34–64%) for diagnosis of concussion.[89,94] The M-BESS however, has no published data documenting its sensitivity. Luoto et al found that in an adult sample of patients with mTBI, their M-BESS scores did not differ significantly from controls.[77] Additionally, balance has been suggested to recover sooner than other deficits that may persist longer after concussion.[77,82] Balance testing by NeuroCom International Smart Balance Master System or sensory organization test (SOT) utilizes a force plate to measures stability and sway in multiple sensory conditions that are increasingly more difficult. A score is calculated for each trial, and a composite score determines overall postural stability.[95] Correlations between the BESS and force platform sway or SOT measures with good intertester reliability have been shown in controls, and differences between acutely injured and controls have also been demonstrated.[96,97] However, some issues include interrater reliability, environmental effects such as noise or anxiety, and orthopedic injuries resulting in functional ankle or lower limb instability.[98–102] In addition, there appears to be a practice or learning effect in studies in which subjects were tested repeatedly over short periods.[103]

Reaction Time

Impaired reaction time has been shown to be one of the most sensitive indicators of persistent deficit after injury. Reaction time has traditionally been measured by computerized testing (RTcomp), usually assessed by pushing a key in response to a prompt. Another method that has been developed to objectively evaluate reaction time of individuals on the sideline, and without a computer, is the clinical reaction time test (RTclin).[104–106] Considerable research has shown that reaction time is prolonged immediately after injury, and improves gradually until returning to baseline.[104] Reaction time may even persist beyond resolution of symptoms, thus making it a useful tool in objectively assessing recovery to be used in conjunction with a patient's self-reported symptoms. The pilot study using the clinical RT tool/stick showed a positive correlation between the RTclin and RTcomp in athletes whose effort was deemed valid by the built-in integrity check of the CogState/Axon Sport computerized test. Additionally, the data showed less variability in the RTclin test, suggesting that motivation may be increased in this assessment as compared with computerized testing.[105] In follow-up studies, Eckner et al demonstrated consistent results across seasons as well as correlative deficits in RTclin and RTcomp in athletes after concussion.[104,107] Recent studies have shown practice effect with improvement in time in similar clinical reaction time tests, such as the ruler-drop test.[108]

King-Devick Test

Like the SCAT versions, the King-Devick test (K-D) was proposed as a sideline tool to detect sport-related concussion.

The K-D is a brief (< 1 minute) test requiring saccadic eye movements on a fixed target to read a series of numbers on three test cards. Numerous processes are required for oculomotor function, including sensory and motor components, as well as cognitive processes such as concentration, attention, and spatial memory.[109,110] In a test of professional ice hockey players, Galetta et al found that K-D scores correlated with lower scores of immediate memory on the SAC at preseason baseline.[111] They proposed that this may be due to function specifically in the dorsolateral prefrontal cortex as this brain region plays a role in saccadic eye movement control and working memory. They also found increased time (worse scores) in two athletes who had sustained concussion, consistent with findings in prior studies showing increased K-D time scores after concussion in contact and collision sport athletes.[112–114] Similar to other available tests, athletes showed statistically significantly improvement between pre- and postseason testing.[113] But the clinical significance of a 2.8-second difference is not clear.[113] Despite studies correlating K-D results to the SAC and MACE, no longitudinal studies assessing validity or generalizability have been performed.[112]

Neuropsychological Testing

Neuropsychological testing can be divided into traditional paper-and-pencil testing with a neuropsychologist and computerized cognitive tests. These tests were designed to identify cognitive impairments that frequently occur after injury, and may be able to identify deficits in those individuals who are reporting an absence of symptoms.[115–118] Traditional paper-and-pencil neuropsychological testing administered by a neuropsychologist may be used in the postinjury period to assess cognitive issues that frequently plague individuals after TBI. Typically, referrals for neuropsychological testing are made when a patient reports particular commonly experienced cognitive issues, such as problems with memory, concentration, and attention, but may also be used when mood symptoms are prominent. Pencil-and-paper testing may be more comprehensive, and as a dynamic testing situation, may test additional domains with the ability to distinguish between effects of a concussion and premorbid conditions.[73,118,119]

However, because formal neuropsychological testing is not always practical, particularly in the athletic and military settings, computerized neurocognitive testing has been utilized frequently. As paper-and-pencil testing requires accessibility to a neuropsychologist, a quiet testing environment, and a significant amount of time, this type of cognitive testing may not be readily available in many arenas when evaluating a head injury. Computerized test batteries are objective methods for testing large groups, are generally brief, and can be used in follow-up for tracking recovery. In these populations, computerized neurocognitive assessments may be administered at baseline so that in the case of injury, there is a standard to which postinjury tests can be compared. With athletes as well as military personnel, return to baseline determinations are particularly important, as these individuals are at a higher risk for a repeat injury, and additionally may be at risk for a more catastrophic injury.[120] The advantages over traditional neuropsychological testing include ease of administration (time of testing, decreases need for additional personnel), alternate test forms, and reduction of practice effects (alternate forms, randomized test stimuli). However, a single baseline neurocognitive test may not provide an accurate representation of an individual's cognitive status. More recently, there is growing evidence that neuropsychological testing can also be used effectively in the absence of a baseline result.[115,121]

There are several commonly used computerized neurocognitive tests available for use. Each test utilizes multiple tasks that when analyzed, comprise several different metrics of cognitive ability. These include but are not limited to the Immediate Post Concussion Assessment and Cognitive Testing (ImPACT), CogState/Axon, CNS Vital Signs, and Automated Neuropsychological Assessment Metrics (ANAM). The most commonly administered test is the ImPACT, which evaluates different cognitive modalities across a variety of tests. Multiple studies have shown good sensitivity and specificity in athletes, 81.9 to 91.7% and 69.1 to 89.4%, respectively.[85,122–124] However, multiple studies assessing reliability over a range of populations have yielded mixed results over varying time intervals, ranging from 1 month to 2 years.[123,125–127] A more recent study has suggested that this may be attributable to different versions of the test used (i.e., desktop vs. online). However, results may be difficult to interpret when an intrapatient baseline comparison is not available. There is normative data established for some of these tests.[115,128–133] In a recent study using the CogState/Axon test that compared the sensitivity and specificity of baseline and normative methods of comparison to determine concussion in elite adult, male athletes, it was found that the baseline method had a higher sensitivity and specificity than normative methods.[132]

In 2008, Congress mandated that U.S. military service members have pre- and postdeployment computerized cognitive testing due to increasing awareness and concern for the risk of brain injury during deployment. The most commonly used test used in the military setting is the ANAM, whose normative data has been published.[134] Roebuck-Spencer et al determined that the determination of atypical individuals based on normative data from the ANAM4 was inconsistent between pre- and postdeployment tests within the same individual. Furthermore, they saw that a large number of those classified as atypical were no different in postdeployment from their baseline, suggesting that using normative standards falsely identified these individuals as atypical.[135]

However, due to ease of administration, some athletic programs are solely utilizing computerized neurocognitive tests for making decisions regarding return to activity. All current evidence- and consensus-based sports concussion recommendations advise against having a single test to diagnose or manage concussion, and that these tests should be used in conjunction with other evaluation modalities to make diagnostic and management decisions. Additional issues with computerized neurocognitive testing include premorbid learning disabilities that are not discernable on

computerized testing, underreporting of prior concussions, language issues, administration of testing in a suboptimal environment (including an unsupervised condition), and appropriateness of the test for the age of the injured individual.[136] Although many of these concerns are addressed by comparing baseline and postinjury assessments within the same individual, this likely limits the use of computerized testing for assessment solely after injury, as a single administration postinjury may not be sufficient as an independent diagnostic tool. Other factors may play a role in performance on the exam, particularly relevant in the athlete and military populations, such as dehydration, motivation, fatigue, and other physical factors.[137,138] Although one study has shown no difference in dehydration in ANAM scores,[137] another study has shown that performance suffers when individuals have urinary urgency on the CogState/Axon test.[139] Hutchison et al found that acutely after injury, collegiate athletes had cognitive impairment on the ANAM test battery with either orthopedic injury or concussion.[140] Additionally, environmental factors, such as testing conditions and time of day, can also influence performance.[141] Evidence of practice effects has led some groups to recommend repeat baseline testing to minimize the effect of learning.[142–145] However, other groups have shown no stability in test-retest reliability in several computerized neurocognitive tests.[123] An additional difficulty in utilizing computerized testing is that some individuals may perform perfectly, suggesting a ceiling effect, which limits the ability of the test to detect minor changes, especially in those who perform at the top.[146] Similarly, a floor effect may also limit the usefulness of a test for determining changes in an individual who already performs poorly at baseline.[142]

Imaging

Use of computed tomography (CT) to evaluate sports concussions specifically is only sparsely reported in the literature; however, multiple studies examining CT use for mTBI presenting in the emergency department (ED) have been conducted.[147–151] Computed tomography has been historically used as the standard of care for assessment of TBI in ED settings, but radiation exposure may present its own risks, particularly in children.[152,153] Jagoda et al recommended that noncontrast head CT should be obtained in an adult with TBI if LOC or posttraumatic amnesia (PTA) was present with headache or vomiting, intoxication, short-term memory deficits, posttraumatic seizure, GCS < 15, a focal neurologic deficit, coagulopathy, supraclavicular trauma or age > 60 years.[147] The Pediatric Emergency Care Applied Research Network (PECARN) proposed validated clinical prediction rules for clinically important TBI for children younger than 2 years and older than or equal to 2 years of age.[148] They identified six clinical predictors for each group. For those in the younger group, the predictors of intermediate risk for clinically important TBI are nonfrontal scalp hematoma, LOC ≥ 5 seconds, severe mechanism of injury, or abnormal behavior per parent. Higher risk predictors are altered mental status or palpable skull fracture. Predictors of intermediate risk for those 2 years old or older include any LOC, vomiting, severe injury mechanism, or severe headache. Predictors of higher risk are altered mental status or clinical evidence of basilar skull fracture. The PECARN group categorized those at lowest risk in either group as those who did not have any of the clinical predictors.[148] In a follow-up study, they suggest that those with isolated severe injury mechanisms without any other risk predictors may be observed before deciding if CT is needed.[149,151] Validation of these prediction rules was recently published in a cohort of children from two EDs.[150]

Magnetic resonance imaging (MRI) has been suggested to be useful in determining outcome after injury. Previous studies using CT showed a correlation between intracranial hemorrhage and long-term deficits.[154,155] Magnetic resonance imaging is more sensitive for elucidating small, focal intracranial lesions, and thus may be more sensitive in determining those individuals at risk for prolonged recovery. The TRACK-TBI group studied mTBI patients presenting to the ED with CT and MRI, and found the presence of intracranial findings (cortical contusion or four or more microhemorrhages on MRI) predicted 3-month outcome, specifically for determining which patients may be those to develop persistent dysfunction.[156] Using the Extended Glasgow Outcome Scale (GOS-E) at 3 months postinjury to grade outcome, they determined that using MRI improves outcome prediction. Additionally, in a later study, Yuh et al determined that diffusion tensor imaging (DTI) findings could predict outcome at the 3- and 6-month postinjury time point. Specifically, they found that reduced fractional anisotropy (FA) in at least one region of interest (ROI) predicted worse Extended Glasgow Outcome Scale (GOS-E) scores at both time points, and MRI was better than other predictors at determining outcome at 3 and 6 months.[157,158]

Management

For those individuals at higher risk of injury, such as those playing contact/collision sports or military service members, it is critical to have education regarding concussion risks as well as signs and symptoms of a concussion so they may remove themselves from further risk if a suspicious head injury is sustained. Education may be provided by a licensed health care professional, trained in concussion evaluation and management.[94]

The initial step in concussion management once a head injury occurs is removal from risk for evaluation and management according to the diagnosis. In the case of the athlete, this means he or she should be evaluated on the sideline, and if concussion is diagnosed, then he or she must be removed from the game or practice for treatment and recovery. In the military setting, this may be more difficult, as in War Theater, extraction of the military service member with suspected TBI may not be possible immediately but should be done as quickly and safely as possible. In either sports or the military, full assessment should be performed before he or she is allowed to return-to-play or duty. This is a critical step, as it is necessary to protect the individual from additional injury. An individual who sustains a concussion may demonstrate a slower reaction time, impaired balance, worsened visual

tracking, and cognitive difficulties, which would increase the risk for further injury. Published studies suggest a prior concussion increases the risk for a subsequent concussion by three- to sixfold.[159–161]

At the collegiate level, the National Collegiate Athletic Association (NCAA) has more recently promoted increased brain health and safety, specifically adopting a concussion policy and legislation in 2010.[162] Each stakeholder school is mandated to have a concussion management plan, including education, removal from play if a concussion is suspected, no same-day return if a concussion is suspected, and medical clearance prior to return-to-play determined by a physician or designee of the physician.[162] There are more than 450,000 athletes affected by this.[163] Using a survey of coaches, clinicians, and administrators evaluating the implementation and effectiveness of the policy, Baugh et al found institutional concussion management plans were in place at 92.7% of the schools, with team physicians and/or athletic trainers primarily having final responsibility for return-to-play decisions.[164] However, in the younger age groups, where the majority of athletes participate, there is no unifying policy regarding concussion education and management, often due either to a lack of a national organizing body, or to multiple different youth sports organizations with differing policies.

Once immediate risk is eliminated, physical and cognitive rest until acute symptoms have resolved is typically recommended.[73,94,165] However, the evidence for this protocol is limited, and in particular, there is no consensus period for which rest should be prescribed, and the exact recommendations of "rest" are not clear.[166–169]

When discussing the recovery process, reassurance and education of the individual must be provided. This brief psychological intervention has been termed *cognitive structuring*; in a pediatric setting it is akin to anticipatory guidance. Cognitive restructuring has been shown to be effective in mTBI as a preventative measure for the development of persistent symptoms, and specifically may be helpful in sleep issues and mood disorders as suggested by studies in uninjured youth.[170–172] This type of anticipatory guidance may also involve a reattribution of symptoms, which may decrease the risk of developing chronic postconcussion syndrome.[94]

For return to sports, the CIS conferences have developed a consensus-based graded return-to-play protocol, designed to allow athletes to return to physical activity in a stepwise fashion.[16,73] Though ideally one should be asymptomatic before returning to activity, in cases of chronic symptoms, sometimes returning to noncontact, low-risk physical activity can be beneficial. But he or she should not return to contact-risk until a licensed health care professional has determined that the concussion has resolved. A special consideration in the pediatric and collegiate populations is return-to-learn prior to return-to-play. With these student-athletes, the authors recommend that he or she should be back to full schoolwork without accommodations before returning to full physical activity. These individuals may proceed through returning to school slowly with accommodations as needed, until they are able to participate fully in school. As younger athletes are slower to reach symptom resolution, the return-to-play protocol should be implemented more conservatively.[94,173,174] In addition, because validation of frequently used concussion assessment tools is primarily in the adult population, these tests should be used cautiously in the pediatric population for assessment of recovery after concussion, and there is an urgent need for better validated concussion assessment tools for use in the youth population.

The military has adopted strategies for return to duty based on sport-related return-to-play protocols for active-duty service members, with acute management demanding at least a 24-hour rest period and off-duty until asymptomatic.[75] Additionally, with two documented mTBIs in one year, the injured personnel should remain off active duty for at least 7 days after symptom resolution. After three documented mTBIs within one year, he or she must undergo comprehensive evaluation including neurologic exam, neuroimaging, and neuropsychological and functional assessments if necessary.[75]

Eighty-ninety percent of individuals have symptoms that resolve in the acute period (within 3 weeks) and are self-limited. Frequently reported categories of symptoms include somatic symptoms (such as headache, nausea, and dizziness), cognitive symptoms such as (decreased attention/concentration, difficulty with memory), mood symptoms (such as sadness, irritability, nervousness/anxiety), and problems with sleep. Initial management should include rest from both cognitive and physical activities acutely, and a gradual increase in these activities as tolerated. Management of acute headache may be achieved with traditional over-the-counter remedies such as acetaminophen, ibuprofen, or naproxen. Typically, more-aggressive management is not needed acutely. In some cases, headaches may persist and require further interventions depending upon the headache diagnosis (migraine, tension, medication-overuse, cervicogenic, etc.). Although no pharmacological intervention for the treatment of postconcussive symptoms is approved by the U.S. Food and Drug Administration, treatment of specific, prolonged symptoms may be approached as if in isolation.[73,94,165] Acute management of headaches with migrainous features may include triptans, and should the headache be frequent and persist past 3 to 4 weeks, treatment with prophylactic interventions such as topiramate, magnesium, tricyclic antidepressants (amitriptyline, nortriptyline), or β-blockers may be necessary. Similarly, treatment of other concurrent postconcussive symptoms in the subacute period may include selective serotonin reuptake inhibitors or other antianxiety or antidepressants for mood disorders, melatonin for sleep, and psychotherapy or cognitive-behavioral therapy for various cognitive or behavioral issues.

Though rest is recommended initially, prolonged rest may in fact be detrimental. Therefore, if symptoms are persistent, athletes may benefit from return to some activity without contact risk while waiting for complete symptom resolution. Those who are isolated from their teams or not undertaking any cardiovascular activity may experience increased depression and anxiety, which can cloud the evaluation of recovery of postconcussive symptoms. Low-level, monitored activity may be beneficial even in those athletes who continue to be

symptomatic, as suggested by studies in individuals with migraines and mood disorders.[175–177] Leddy et al suggested that controlled exercise training at a subsymptom threshold level improved postconcussive symptoms compared with no treatment in a small case series.[178] Furthermore, they demonstrated functional MRI activation similar to controls in those patients with postconcussive symptoms who received exercise treatment.[179]

In the military population, concurrent mTBI and PTSD occurs in almost a third of all those with probable concussion.[180] Posttraumatic stress disorder is a relatively common problem among service members, with a conservative prevalence of 16,000 cases in deployed personnel in 2011.[181] However, this number is likely a significant underestimate due to the restrictive terms of diagnosis.[182] The association is greater in those service members who experienced blast injury and did not have a concurrent injury.[183] In this population, postconcussive symptoms may easily be confused with other psychiatric conditions such as PTSD, mood disorders, and chronic pain. Furthermore, these types of symptoms are reported even by service members who were not injured in Theater.[184] Addressing PTSD is a critical piece of improving recovery after injury in this population, as studies suggest that PTSD and postconcussive symptoms are associated with poorer prognosis.[182] Treating psychiatric comorbidities is particularly important given that the suicide rates have been rising over the last decade, and those with TBI are at an increased risk of both suicidal ideation and suicidal attempts/death.[76,185–187]

For those individuals who have sustained multiple concussions, retirement from the offending activity (contact sport, military) may be recommended. When an individual reports chronic cognitive or behavioral issues, he or she may be referred for formal neuropsychological assessment. Retirement should be definitively recommended for those demonstrating dementia/degeneration, permanent or lasting neurocognitive impairment, or intractable pain. Additionally, those individuals who sustain injuries at a decreased interval between injuries, who have longer recovery after subsequent concussions, or who demonstrate a lower impact threshold for concussion may be counseled regarding the risk of further injury in terms of persistence of symptoms.[94]

Summary

Accurate concussion diagnosis and appropriate management are critical, as prior concussions predispose athletes to sustaining an additional concussion. Furthermore, repeat concussion may result in worse long-term outcomes, although the extent remains unclear. There are numerous diagnostic tools available to clinicians to aid in assessing individuals with a suspected concussion. On the sideline, the Maddocks questions are often used in sports settings; the MACE serves as a screening tool in the military. In the acute/subacute setting, preferably in a quieter setting (locker room, clinic, etc.) the SCAT3 may be used to assess symptoms, cognition, and balance, and takes 10 to 12 minutes. Computerized or traditional cognitive testing may have a role subacutely, or when attempting to determine return to duty/play as symptoms resolve, but require proper testing conditions and take more time (up to 20–30 minutes). Emerging tools include the clinical reaction time stick and the King-Devick test. Both are quick and relatively inexpensive, with varying specificity and sensitivity, but may play an increasing role, particularly in screening for acute concussion. Many more complex tools are worthy of continued research investigation, but are not yet ready for prime-time clinical usage, like visual tracking devices, advanced neuroimaging, or electrophysiological monitoring. Undoubtedly, some of these will be added to the concussion assessment armamentarium in the future. No single tool is sufficient to diagnose brain injury, and evaluation must include a thorough history, including symptom report, and neurologic examination in addition to validated adjunctive testing for balance, reaction time, visuomotor skills, and cognitive assessment.

Clinical management starts with identification of suspected concussion. In those scenarios, the individual should be removed from contact risk. For athletes, this means removal from play without return the same day, and formal evaluation by an experienced health care provider. For military personnel, this removal from risk is more problematic, but should be attempted when possible. Experienced clinical personnel should use the above tools to determine if a concussion occurred—conditions like migraine, anxiety, dehydration, hyperthermia, and others may mimic concussion symptoms and need to be treated differently. Once a concussion is diagnosed, education, reassurance, and cognitive restructuring should be implemented, as well as continued protection from contact risk. Clinical symptoms and neurologic function should be monitored and activity gradually increased as recovery ensues. Medications that mask symptoms should be weaned. Careful assessment of symptoms, cognition, balance, reaction time, and neurologic function are important to determine full recovery and eventual return to full activity and potential contact risk. Most return to play/duty protocols use a stepwise plan, with careful observation for symptoms or neurologic exacerbation at each step before advancing. For individuals who are having a prolonged recovery, further investigation and diagnostic testing may be needed to determine other etiologies and modify the treatment plan. Overall, management of concussion/mTBI is individualized to the specific patient. Multidisciplinary teams are optimal, particularly for those patients with more complex or prolonged recovery.

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