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Short-term and long-term outcome of athletic closed head injuries

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Only gradually has a research literature evolved that differentiates short- versus long-term effects and outcomes in athletic head injuries. The good news of early studies was that symptom number was small and most symptoms resolved for most athletes within 5 to 10 days postinjury [1]. Continued study has revealed that a small percentage of athletes recover more slowly, and an even smaller subset appears to manifest permanent symptoms [2–5]. The somatic symptoms that predominate both short- and long term are headache and confusion. Neuropsychological assessment measures have demonstrated their worth in detecting nonsomatic residuals of injury, primarily diffuse frontal and temporal lobe manifestations, including impaired attention, decision-making, memory, and speed of processing. Determining what factors may predict early versus delayed resolution of mild sport-related head injury symptoms remains the challenge. This article reviews the basic mechanisms of sport-related closed head injury, primary risk factors for differential outcome, and findings of neurocognitive deficit that characterize the immediate, short, and long-term aftermath of athletic head injury, and points toward known or theoretically sound mechanisms that might be candidates for clarifying differences.

Athletic closed head injuries defined

Vigorously played sports, whether considered contact or noncontact, carry with them the potential for injury, including head trauma. Although some sports have equipment or are played in environments that may result in impact to the

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skull and cause penetrating injuries (the sticks in ice hockey and lacrosse, the goal posts in football and soccer, or the terrain in skiing), most traumatic head injuries that arise in the context of sport are closed head injuries (CHI), and the majority of these are termed concussions [6]. Moreover, the majority of concussions fit within the category of mild traumatic brain injury (MTBI) [7]. The typical concussive injury may arise from several sources including: hand, arm or foot blows to the head; body-to-head contacts; collision with fixed objects; and impact with the ground. The common denominator in such injuries is the change in acceleration at the time and point of impact. Although such acceleration injuries may be concomitant with blows to the head, many more will stem solely from the application of acceleration/deceleration forces [7,8]. These forces may be applied so as to affect the head either at the long axis or at a tangent to the direction of head movement or position. It has been suggested that the majority of sport-related closed head injuries are less severe than those occurring in such contexts as automobile accidents because the acceleration changes and the force-mass relationships (in cases of physical blows) are generally of much lower magnitude [7,9,10],

Despite the seeming reassurance conveyed by the term "mild" in describing the majority of these injuries, recent studies caution that the repetitive nature of some sport head injuries may result in much more serious functional, and perhaps structural, outcomes than previously suspected [4,11–15]. For example, although controversial at this time, the prospect that repetitive head-to-ball contacts in soccer might cause both short-term and enduring cognitive impairments has engendered considerable debate and resultant research, while also alerting the soccer community to the potential for harm [13,15,16]. Moreover, the functional definition of "mild" with respect to closed head injury continues to evolve as more studies report lasting effects following so called mild trauma [17,18].

Generally, when reviewing outcome after sport related injuries it is important to use caution in generalizing results from nonsport settings with humans, or from the carefully controlled animal studies. Notwithstanding the current debate over what is mild and what is not, trauma from auto accidents imparts acceleration forces far in excess of what is typically observed in most sport settings. Exceptions might include skiing and snow boarding, the rough events in rodeo, and high force-mass blows in hockey or lacrosse. [19,20] Thus, although studies based upon consecutive presentations in the emergency department (for example) of major hospitals provide much needed data on MTBI, generalizations about sport-related MTBI must be made cautiously. Also, because of the demonstrated capacity of small animals to absorb and endure extremely high acceleration blows with sometimes trivial outcomes, generalizations from animal studies also must be made very conservatively [10].

The typical athletic closed head injury is notable for a period of confusion and disorientation immediately postinjury, and may be accompanied by brief loss of consciousness (LOC) or post-traumatic amnesia (PTA). Forty years ago, Symonds suggested that the effects of even mild concussion were never fully reversible [21]. In contrast, Barth and colleagues showed that for the vast

majority of athletes who suffered mild head injury, complete resolution was obtained in 5 to 10 days [1]. Both Symonds and Barth et al, however, concluded that complete recovery following multiple concussive or even subconcussive blows might be less probable. It is not uncommon for postconcussive symptomatology (PCS) to persist for weeks or months under these conditions in some athletes [17], as has been reported in more general clinical populations [18]. This confusing differential duration of symptoms poses significant difficulties for choosing methods of assessment generally [3], and for differentiation of guidelines for return-to-play in athlete populations [22]. Moreover, the factors that predict which athlete will have quick versus slow symptom resolution have not been identified clearly. Two athletes who suffer similar head trauma may differ widely in their recovery and return to play, despite no obvious differences in injury mechanics, diagnostic imaging, and sideline symptoms, including presence or absence of LOC and PTA, yet other factors such as number and recency of concussions may critically determine symptom resolution [9,23–25].

Sports as a laboratory assessment model (SLAM)

Beginning in 1989, Barth and colleagues described the use of the sport environment as a laboratory assessment model for studying the neurocognitive sequelae of head trauma. This approach has been validated by its application in many subsequent empirical studies [4,5,26,27]. A key element of the sports as a laboratory assessment model (SLAM) involves baseline assessments of neurocognitive functioning, which then serve as the standard for assessing the presence or absence of concussion and its severity, and subsequent readiness for return to play and practice. Such baselining has become the normative approach for evaluating high school, collegiate, and professional sport teams [26,28–30]. The methodological value of this approach is that each player serves as his or her own control, eliminating sole reliance upon average, normative data that describe neurocognitive functioning.

In their groundbreaking studies of closed head injury in college football players, Barth, Macciocchi, and colleagues reported that concussed football players typically had recovered normative baseline functioning within 5 to 10 days following injury. Subsequent studies have built upon the earlier ones, and have described some exceptions to that recovery duration in which impaired function has persisted for longer periods of time [4].

The natural recovery curve

Studies of athletic head injuries most typically report on immediate, short-term, and long-term outcomes for recovery of normal cognitive function and resolution of physical symptoms such as headache and nausea. Immediate is just that. Upon observation or player self-report of head injury, a sideline assessment

is made on the spot. Such assessments typically involve brief mental status questions. The Standardized Assessment of Concussion (SAC) represents the most widely used, standard sideline evaluative method [31]. Based on the player's response and the trainer/physician's observation, return to competition may be almost immediate in the absence of mental/cognitive deficits and neurologic symptoms, or delayed attendant upon further observation and resolution of all symptoms [32]. Because a sideline examination is necessarily brief, clear markers of impairment in mental status represent the primary assessment metrics.

Assuming that an immediate, sideline judgment has been made that a player suffered from concussion, then subsequent neurocognitive assessments are typically initiated within 24 hours in this model. If symptoms still are present, then additional measurement is likely after 3 to 5 days, and again at 7 to 10 days. If symptoms or cognitive functions have not returned to normal within 10 days, it is common to make further observations at regular intervals until symptoms have resolved and the player returns to his or her baseline neurocognitive function. Beginning 24 hours postinjury, preseason neurocognitive tests will be readministered. These will typically include measures aimed at assessing general brain functioning, including reaction time and speed of mental processing, as well as attention, concentration, decision-making, and mental flexibility. Most recently, computer [33,34] and web-based [29,35] neurocognitive tests have been introduced for assessing impairment and recovery. With these very time- and cost-effective methods, the baselining approach introduced more than a decade ago remains not only relevant but gains vitality and new venues for application [36]. Players typically are assessed before their playing season begins or when they have been absent from head knocks for a reasonable period of time. Most instruments, such as the Concussion Resolution Index, rely upon precise measures of simple and complex reaction time, cognitive processing speed during simple and conditional tasks, and tests of immediate and delayed recall [29,35] With this web-based technique, players' postinjury performances can be more easily compared with their baselines and with appropriately developed norms. From both research and clinical vantage points, this within-subject approach results in much clearer understanding of individual functioning. Moreover, even though head–injury-related changes in baseline performance may only be on the order of milliseconds, the differences are highly reliable and valid, and provide a purely quantitative evaluation of an athlete's neurocognitive functioning [29]. These approaches allow clinicians an objective alternative to reliance on subjective symptom complaint for decisions relating to release from treatment and return to play. This is important for two reasons. First, athletes may well minimize symptoms in order to obtain an earlier return decision [37,38]. Second, subjective symptom reports have been shown to be inadequate discriminators between normal and general MTBI populations [39].

Quite commonly, measures of neurocognitive functioning taken at 24 hours postinjury reveal depressed reaction times, speed and accuracy of information processing, attention, learning, and memory. In most instances scores have returned to normal (if the individual's baseline is available) or to normal range

(if normative comparisons are being employed) within 5 to 10 days. This appears to capture the majority of cases as represented in the scientific literature to date, employing many types of measuring instruments across a variety of sports. The somatic symptoms that predominate during these times are headache and confusion or disorientation, which are reported by about 50% to 75% of the athletes [1,4].

Given that 80% or more of athletes have recovered within 5 to 10 days, the remainder continue to report symptoms for variable durations. The physical trauma that created the brain injuries generally appears to be comparable across the short and longer recovery categories [4]. Moreover, the neurocognitive impairments that persist relate most often to memory and attention [1]. Headache, the most common somatic complaint in the early post-injury days, may re-emerge in the long term, particularly during exertion. No commonality in personality changes has been noted, and the frequency of such change appears to be low.

Paniak and colleagues have shown that somatic complaints may not always differentiate among individuals who have suffered MTBI in a general clinical sample [39]. Complaints of memory impairment have been cross validated with computerized measures of reaction time and information processing, however [40]. Nonetheless, because of the variability in symptom report, the possibility either of malingering or of under-reporting, and the lack of predictive validity of markers such as LOC and sideline measures of PTA, the use of neurocognitive testing to determine severity of injury, duration of recovery, and return to play has become standard [30]. It is important that the neuropsychological assessment is broad in scope, and measures all general levels of brain and cognitive functioning.

Injury mechanisms

The considerable variability in recovery following closed head injury demands a mechanism that itself varies, but in ways that are difficult to predict. If simple extent of focal brain insult were the major culprit, an examination of surface area impacted by the blow in interaction with the specific locus would predict reasonably well the nature of postconcussive symptoms and the duration of recovery. Focal injuries in sport-related head trauma, however, are rare [7]. From a theoretical standpoint, interruption in pathways carrying information needed to provide the basis for decision making, or providing the feedback necessary for maintaining attention, should be sufficient to produce the types of deficits that commonly are seen. The most obvious candidate for a major mechanism of these differences is likely to be extent and location of white matter compromise.

More than 60 years ago, Holbourn and Symonds set the stage for looking at white matter involvement to explain concussion effects [21,41]. In his shear/strain model, for example, Holbourn viewed concussion as a disruption in cytoarchitecture within the brain, as opposed to focal injury, which was considered to produce more severe structural and functional outcomes. In

1940, Symonds commented on the seeming perverse resistance of concussion to be pigeonholed regarding a common time course for recovery: "whatever might be the nature of concussion the symptoms could not be made to conform with any arbitrary limit of the time taken for recovery of full consciousness, or indeed with the absence of sequelae" [21]. Their historic lead still provides the most productive model for theorizing the factors that determine injury severity, particularly as measured by latency to recovery of normal functioning. As an example, white-matter dysfunction in the absence of focal or lateralizing neurologic findings may best be predicted as a result of accelerations sufficient to impart injury at the histological level. The greatest risk of linear acceleration-deceleration injury may be from the bony protuberances that impact the ventral surface of the temporal and frontal lobes. Injury resulting from angular acceleration or rotational forces will be most prominent at areas of gray/white tissue differentiation beginning below cortical levels and then descending deeper toward the brain stem [42]. Therefore, we would look for evidence that rotational, as opposed to linearly applied force, has the potential to cause significant neural disruption in the absence of obvious focal damage and serious morbidity. Unfortunately, no clear human demonstrations have been reported. Computer simulations of rotational versus linear heading forces present in soccer play have demonstrated significantly greater risk potential for injury based upon peak rotational acceleration quotients [43] The potential internal changes to the white matter have almost infinite histological and cognitive/behavioral manifestations. For example, if a wide receiver in football is tackled from behind in the small of the back while leaping for the ball, there may be a whiplash effect on the head and brain, causing a linear deceleration injury. If we add a second tackler to the scenario whose shoulder causes a forceful blow at a tangent to the receiver's facemask simultaneous with the first tackle, then a rotational acceleration is added. Although we may speculate theoretically on the differential contributions of these twin blows to a resultant brain injury, and although we might accurately measure the white matter damage in vitro, mechanisms for in vivo clinical evaluation lag far behind [42]. The known mechanisms of diffuse axonal injury from such an event might include transient ischemia of the axon, or tearing and dismemberment of the neurolemma in the white matter, with or without accompanying necrosis of the soma [44,45]. If the soma survives, despite new sprouting from the axon hillock, axonal reconnection is compromised due to rapid gliosis and cytotoxic metabolic mechanisms. Although areas of petechical hemorrhages are highlighted in magnetic resonance imaging (MRI), smaller zones of axonal shear injuries remain invisible with the standard technology [42,45]. Advanced MRI techniques have now been shown to detect the presence of some diffuse axonal injury (DAI) [46]. Although it cannot yet be determined how many instances of athletic head injury may present with DAI, the histological basis for suspecting DAI as a major player in the variability of recovery has been established. Although all available forms of imaging, including techniques of functional MRI (fMRI), positron emission tomography (PET), and single photon emission computed tomography (SPECT), show promise of

unmasking suspected shear-strain injuries, no study has yet reported a direct relationship between such damage and injury severity or latency to recovery of function in sports trauma [7,42]

Risk factors

Cumulative concussive and subconcussive blows

Most of the data in the area of synergistic effects in neuronal dysfunction have come from animal models. In those studies, two or more concussive blows in close succession have produced significantly greater neurologic impairment and resulting neurobehavioral deficits than a simple sum of these singular blows [47]. Much of this research is aimed at identifying the mechanisms of action that may underlie the second-impact syndrome (SIS), the catastrophic outcome following two mild concussions close in time [48]. Indeed, Hovda and his colleagues have elaborated rather elegantly the pathophysiological mechanisms that underlie the synergistic effects of repeated concussions in rats [44,45]. They describe concussion as a neurometabolic cascade of events whereby energy stores are depleted through excitotoxic mechanisms, with accompanying ionic fluxes of great magnitude, and neuronal/axonal impairment and injury. If a second concussive event occurs within this period of metabolic instability and vulnerability, the probability of neuronal mortality increases greatly. Even assuming an injury mechanism in humans analogous to that discovered in rats, the time course of all similar metabolic events would presumably be much extended.

The potential that repeated, subconcussive blows to the head might cause equivalent if not greater damage than a single mild concussion was proposed decades previously [49,50]. A subconcussive event may be defined as an apparent brain insult with insufficient force to cause hallmark symptoms of concussion. The rationale for wrapping subconcussive events into the context of concussion is that such traumatic events may exist on a continuum of histologically based damage. For the very reason that subconcussive events are not so easily marked as are concussions, however, it is conceptually problematic to make the link to any observed impairment. The major impetus for considering subconcussive outcomes is the fact that such events are common in sports such as soccer and football, and in boxing. Indeed, boxing has served as the primary laboratory for assessing the role of multiple concussive and subconcussive blows in producing neurocognitive impairment [51]. The main difficulty in interpretation is determining where the effects of concussion end and those of subconcussion begin. The same difficulty has characterized research that attempts to link soccer heading (subconcussive events) with neurocognitive deficits [15,52,53]. Because known histories of concussion characterize up to 50% of high-level players, the unambiguous role of heading is difficult to conclude. Studies that have reported effects generally have made the link by considering self-reported heading frequency as a categorizing variable, and then observing that more

performance impairments characterize the frequent heading categories. These studies are very suggestive but not conclusive.

APOE genotype

One of the more intriguing outcomes in recent studies has been a succession of findings relating the interaction of head trauma with possession of the apolipoprotein E (APOE) genotype. Already well known as a correlate with incidence of Alzheimer's disease, the combination of the APOE ε4 allele with head trauma has correlated with earlier-onset Alzheimer's disease in older patients [54], poor outcome following head trauma in young patients [55], and increased severity of chronic TBI in experienced boxers [56]. Most recently, Kutner and colleagues reported that currently active, older professional tackle football players who were heterozygous for the ε4 allele, exhibited decreased neurocognitive performance in comparison with their contemporaries who did not possess the allele [57]. It was presumed that number of concussive and subconcussive events increased with playing experience, which was supported by the self-report data on concussion. Because of the small sample size (total of 53, with 14 heterozygous ε4 presentations), the authors considered their findings preliminary but very suggestive of an important risk factor for athletes involved in sports where head trauma also occurred with more than trivial probabilities.

Age

Theoretically, youth athletes should have a higher risk of sports-related concussion than adults, given their lower level of skill and playing experience and less developed brains [58]. Conversely, the heavier weight and greater strength exhibited by adult athletes might suggest that adults rather than youth would be at higher risk, given higher force-mass ratios that would characterize collisions. Many sanctioning bodies for organized youth sports have introduced changes to standard rules to better protect youth participants from potential harm [59,60]. Possibly such protective rules interact with developmental factors to safeguard children but not adolescents. For example, Proctor and Cantu have observed that after about age 12, adolescent head and neck injuries increase as a function of increasing age, particularly in rough sports such as American football [61]. Other studies have reported that a greater percentage of young athletes than older athletes suffered head trauma [62]. In a large-scale study of high school and collegiate football players, Guskiewicz and colleagues reported that high-school athletes were at higher risk of concussion than were most collegiate players (NCAA Division III players were the exception) [23]. Tysvaer and Storli noted the presence of abnormal EEGs in 35% of the active soccer players in their study (ages 15–34), compared with 12% of the control subjects [62]. Older, retired players (ages 35–64) exhibited electroencephalogram (EEG) abnormalities in a similar ratio to their control group. Tysvaer noted that when players from both the active and retired samples were examined as a group, EEG abnormalities

occurred most frequently in the players younger than age 24 [63]. Unfortunately, the EEG abnormalities were not linked to specific concussive events in individual players. Rather, it was assumed that concussion was the mechanism producing the abnormal electrical activity.

There have been several accounts of serious injury and poor outcome in young adults who have experienced repeated mild brain injury over short periods of time [64–66]. Very little research, however, has been reported that directly examined the role of age as a determinant of sports concussion outcome following a single concussive event. As an example, Moser and Schatz reported that considerably more of their high-school level athlete participants (aged 14–19) appeared to have longer lasting neurocognitive and somatic symptoms than would have been expected based upon previous studies of older athletes, but no direct comparison with an older group was possible [17]. Argument for Moser and Schatz's position also relates to the concerns over return-to-play criteria for young athletes whose brains may be at higher risk for SIS than their older counterparts [61]. Although some controversy exists over how well documented the SIS phenomenon may be, there appears to be no argument that the more compelling cases have occurred with youthful individuals [67]. A rapid, generalized cerebral edema that has been reported in some children following MTBI is one possible mechanism that may underlie both SIS and differential concussion outcome based upon age [68].

The final confusion is that between age and playing experience, which could easily mask any differential sensitivity of the young brain to the neurocognitive effects of brain trauma. Several studies have reported that years of experience playing soccer correlated with greater neurocognitive impairment [13,15,52]. Although some controversy exists over whether the effective mechanism in these studies was history of concussion per se or cumulative subconcussive heading incidents, either explanation entails a phenomenon that assumes greater prominence with years of playing experience, much like cumulative blows endured in boxing, or multiple concussions experienced by football players.

Gender

Almost no research on differential outcome by gender following sports-related MTBI has been reported. From a logical standpoint, one could argue convincingly for a greater or lesser risk of injury, and a greater or lesser recovery duration based on gender. For example, it may be that women are at greater risk for more severe injury and postconcussion effects due to lower body mass and smaller neck size and supporting musculature. Conversely, it could be argued that concussion-inducing collisions in women's sports may be less severe because of overall lower body mass entering into the force-mass relationship. Moreover, the general finding that women's brains are less lateralized than men's suggests that neurocognitive outcomes of concussion in women may be ameliorated due to more distributed functional capacity. Historically, more opportunities have existed for men to engage in sports that are likely to correlate with an increased risk of concussion (mainly football). The increased participation by women in

soccer, ice and field hockey, mountain biking, rodeo, and extreme sports, however, suggests that large-participant gender studies are now possible.

In their meta-analysis of gender differences in TBI outcome, Farace and Alves found that women fared worse than men on 85% of the measured variables, primarily somatic symptoms [69]. Although the eight studies that they reviewed did not report sports-related injuries, the consistency of outcome data affirms the hypothesis that sports-related MTBI may also result in more serious postconcussion symptoms with women. In studies reporting on injury risk in sports, women had significantly more mountain-bike related injuries overall than men, but no gender difference in concussion frequency was obtained [70]. Preliminary data comparing head injury occurrences per unit of athlete-exposure hours indicates that women who play intercollegiate soccer are at a slightly higher risk of concussion than are men [71]. Unfortunately, no published findings to date have commented on any differential neurocognitive and symptomatic outcome by gender. A preliminary report of a prospective study by Broshek and colleagues with high school and collegiate athletes (n = 46), however, found no major gender differences in concussion grade, number of sideline symptoms, or duration of recovery. A trend for the women players to have a greater likelihood of impaired reaction times and processing speed (as measured by the web-based Concussion Resolution Index) suggested that further study of this issue is warranted [72].

History of concussion

History of concussion has been reported in many studies to be a significant risk factor for future concussions, with studies estimating variously that a player who suffered one concussion in a season was three to six times more likely to suffer a second concussion than a player who had no history [23,37,73,74]. Although this stunning relationship often is mentioned prominently in arguing for caution in releasing athletes to play and practice [23,75], the mechanism that may determine the nature of the risk remains unclear and goes essentially unremarked. Do multiply-concussed athletes have some genetic neurological predisposition toward concussion? Do previous concussions result in some permanent postural instability that predisposes for such clumsy behavior that subsequent concussions are more likely? Neither of these propositions has any documented empirical support. Do individuals characteristically engage in risky behavior that maintains a high probability of injury? One study has reported that soccer players who head the ball the most frequently also exhibit significantly higher extraversion scores, and report higher levels of aggressive play than players who head less frequently [76]. Because frequent heading may be considered risky with respect to increasing the potential for head trauma, this one hypothesis now has limited support. But clearly, this is one issue that has been under-researched.

The degree to which a history of previous concussion contributes to poorer neurocognitive outcome and recovery remains controversial. For example, Macciocchi and colleagues reported on 24 college football players who had

sustained one or two grade I concussions. Although the players with two concussions initially reported more symptoms at 24 hours postinjury, there were no differences found in the 10-day assessment [25]. Barnes et al and Collins et al, however, reported that soccer and football players, respectively, with a history of multiple concussions, exhibited more neurocognitive deficits than players with one or no concussions [26,77]. Moreover, Collins et al also reported that multiple concussions interacted with history of learning disabilities to result in even more impaired neurocognitive performance. Wilberger and Maroon showed that performance on neuropsychological measures declined relative to the number of mild injuries experienced. Neuropsychological examination of athletes with three, four, or five incidents of mild TBI revealed performance declines on information processing tasks of 25%, 33% and 40%, respectively [78].

Loss of consciousness (LOC) and post-traumatic amnesia (PTA)

Historically, LOC was a defining symptom of concussion [79]. As overall understanding of mild head injury has evolved, however, it has become clear that concussion in the absence of LOC is very common [75]. The remaining controversy relates to the role of LOC as a marker of severity of injury, and as a factor important for limiting return to play. Although some studies suggest that LOC does mark a more severe injury, these findings become controversial when placed into the context of other studies that report LOC not to be predictive of injury severity, as measured by duration and total number of symptoms and neurocognitive performance [24,40].

Similar to LOC, post-traumatic amnesia has occupied a prominent role in the concussion literature, even though the term has been used variously to describe impaired orientation, retrograde amnesia, and anterograde amnesia. For example, PTA was and is a critical determinant of Cantu's concussion grading system, and within that context refers mainly to impaired orientation. When discussed as a short-term marker of concussion, for example, at 24 hour postinjury evaluation, PTA frequently refers to retrograde amnesia. When used in the contest of persistent postconcussive symptoms, PTA more likely refers to anterograde amnesia [75]. PTA has been viewed as an important determinant of both concussion severity and predicted duration of recovery. PTA in that context typically involves impaired orientation, however, as opposed to a persisting memory problem. For that reason, Erlanger et al suggest that the term "post-concussion memory dysfunction" (PCMD) be used to characterize the lingering postconcussion memory complaints that are problematic but that are not of the order of severity of the anterograde memory deficits seen, for example, after stroke. Erlanger et al reported recently that self-reported memory problems at 24 hours postinjury, but not LOC, were very predictive of severity of concussion as measured by overall number of symptoms on the sideline, impairments in neurocognitive reaction time and speed of processing, and by duration of recovery [40].

Role of specific sport or type of injury in determining outcome

It is the rare sport that has not had participants who have incurred head injuries. American football has been the model head-injury sport because of the large number of participants and the relatively high percentage of head injuries that occur [1]. Soccer is the most popular sport in the world, but the reported incidence of head injury and concussion in soccer is somewhat lower than in American football [71] Equestrian sports have high relative percentages of head injuries but many fewer participants overall. Wrestling, rodeo, skiing, snowboarding, Australian rules football, rugby, lacrosse, and ice hockey are other sports where concussions are frequent enough to garner reports in the literature [80]. From the earlier perspective of looking at the potential for prevalent white-matter injuries as a critical factor that might differentiate short- from long-term recovery of function, we might consider the potential of each sport to serve as a host for violent, rotational-force based concussions. For example, linemen in tackle football are less likely to suffer such injuries than are wide receivers and defensive backs, because mostly linear forces act on the former, whereas the potential for angular as well as linear acting forces are more likely for the latter. Ice hockey has the potential for all manner of blows from all angles and should provide a good laboratory for assessing acceleration type as a factor in short- and long-term recovery differences. The rough events in rodeo (bull and bronco riding) may similarly provide such violent rotational accelerations. Soccer players commonly accelerate their heads in an angular movement to strike an oncoming ball.

Return-to-play decisions

The obvious goal of neurocognitive assessment following athletic closed head injury is to determine both the functional impact of the injury and also to guide the clinician, coach, and participant in making return-to-play decisions. Because the standard imaging and other medical assays now available still reveal little structural change in concussive injuries, and because subjective symptom complaint may not reliably discriminate MTBI patients from uninjured individuals [39], neurocognitive and other nonmedical tests (such as balance) are the mainstay for making return-to-play decisions [23,81,82]. The weight of literature, research, and clinical experience suggests that most athletes should be able to return to play within 5 to 10 days after initial concussion, assuming a rapid reduction of symptoms. So the real value of testing is to validate that expectation for most athletes and to identify those individuals whose recovery represents a clear exception to this expectation. Most typically, tests of mental processing speed and accuracy, reaction time, attention and concentration, and learning and memory have provided the most sensitive data for these purposes. Both standard and computer-based neurocognitive tests have proved to be valuable adjuncts to neurologic symptom monitoring in such decision making.

Summary

The continued development of the sport environment as a laboratory for clinical investigation of mild head injury has greatly advanced the use of neuropsychological assessment in evaluating brain-injured athletes, and tracking their symptoms and recovery in an objective manner [30]. The use of neurocognitive baseline measures has become critical in determining whether a brain-injured athlete has recovered function sufficiently to return to play. The rapid growth of computerized and web-based neurocognitive assessment measures provides an efficient, valid technology to put such testing within the reach of most institutions and organizations that field sport teams [29,34]. Moreover, the knowledge of the recovery curve following mild head injury in the sport environment can be generalized to the management of MTBI in general clinical environments where baseline measures are unlikely. What we know today is that sideline assessments of severity are not predictive of which athletes will show the most typical 5- to 10-day recovery period and which will report persistent PCS complaints and exhibit impaired neurocognitive performance for an extended time [24]. The research on mechanisms of brain injury in MTBI suggests that unpredictable, diffuse white-matter damage may control much of the variability in functional impairments and recovery duration [45].

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