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# Concussion in the Adolescent Athlete

Matthew F. Grady, MD

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Concussion in the adolescent athlete is a common sports and recreation injury. Traditional management of concussion in this age group has focused on sport return-to-play decisions. However, new research on mild traumatic brain injury has dramatically changed the management of concussion. During the acute healing phase, physical and cognitive rest are crucial for healing. In the school-aged athlete, new concepts, such as complete brain rest, have made school management decisions as important as sport return-to-play decisions. Despite tremendous improvements in the under-

standing of concussion, most of the research has been done in young adults. The lack of prospective studies in early adolescent student athletes limits definitive management recommendations. This article reviews the current understanding of the epidemiology, pathophysiology, and clinical presentation of concussion and discusses the unique factors involved in clinical management of concussion in the adolescent student-athlete.

*Curr Probl Pediatr Adolesc Health Care 2010;40:154-169*

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**A**dolescent concussion is a common sports injury among middle school and high school students<sup>1</sup> that historically has been underappreciated and mismanaged. Although the science of concussion has progressed dramatically over the last 10 years, the existing literature on concussion is still evolving. This article reviews our current understanding of the epidemiology, pathophysiology, clinical presentation, evaluation, and management of concussion.

## Background

Historically, the definition of concussion has not been well defined. The word concussion is derived from the Latin word *concutere* (concussus, past participle of *concutere*, to strike together).<sup>2</sup> Concussion is a type of traumatic brain injury (TBI) that has been frequently used interchangeably with mild TBI in the medical literature. The term “mild” refers more to the initial impact of injury rather than the long-term effects from injury. Concussions frequently occur after

relatively low force impacts that are common in collision sports but can also occur after bicycle and motor vehicle accidents. Previous definitions of concussion narrowly defined concussion as an injury that inherently included a brief loss of consciousness.<sup>3</sup> Nevertheless, as our understanding of the pathophysiology of concussion has evolved, the definition of concussion has expanded. In 1999, the Centers for Disease Control recognized that loss of consciousness occurred in less than 10% of concussions.<sup>4-6</sup> This conceptual shift significantly broadened the definition of concussion and changed concussion reporting data.

In 2002, sports medicine concussion experts convened in Vienna, Austria, in part to develop a universally accepted definition of concussion. From this conference and subsequent follow-up conferences, the mostly widely recognized definition of concussion was developed. The third International Conference on Concussion in Sport (Zurich 2008) has defined concussion as “a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathologic and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include:

1. Concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head.

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From the Sports Medicine and Performance Center, Children's Hospital of Philadelphia, King of Prussia, PA.

*Curr Probl Pediatr Adolesc Health Care* 2010;40:154-169

1538-5442/\$ - see front matter

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doi:10.1016/j.cppeds.2010.06.002

2. Concussion typically results in the rapid onset of short-lived impairment of neurologic function that resolves spontaneously.
3. Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury.
4. Concussion results in a graded set of clinical symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course; however, it is important to note that, in a small percentage of cases, postconcussive symptoms may be prolonged.
5. No abnormality on standard structural neuroimaging studies is seen in concussion.<sup>7</sup>

## Epidemiology

The estimated incidence of concussion in the USA is 1.6-3.8 million concussions per year.<sup>4</sup> Derived from national emergency department (ED) surveillance data, the estimated incidence recognizes that only about 5.5%-13.0% of sports-related TBI are evaluated in the ED. In ED surveillance data from 2001 to 2005, pediatric patients (age 5-18) accounted for 60% of the sports injuries and 65% of the sports-related TBIs. Males accounted for 70% of the visits. The highest reported rates of TBI occurred in the 10- to 14-year-old (first) and 15- to 19-year-old (second) subgroups. Activities associated with the greatest number of TBI-related ED visits in the pediatric age groups included bicycling, football, basketball, playground activities, and soccer.<sup>8</sup>

Concussions in high school athletics are common in contact and collision sports. Football-related concussions have the best prospective studies, with the incidence of documented concussions reported at about 1 in 20 high school players (6.3%) per season.<sup>9</sup> All of these concussions were documented by a team athletic trainer,<sup>10</sup> suggesting that the actual incidence of concussion may be higher due to widespread underreporting of symptoms during the season.<sup>10,11</sup> After the season questionnaires have self-reported concussion symptoms as high as 15%-25% per season in high school football players.<sup>10,11</sup> Studies in junior ice hockey have shown both a similar incidence of concussion and a tendency to underreport symptoms.<sup>12</sup>

There are several reasons concussions and concussion symptoms are underreported. Up to a third of injured athletes may not recognize their symptoms as a concussion.<sup>11,13</sup> Coaches and sideline personnel may not recognize the symptoms of concussion in their athletes.<sup>14</sup> Finally, some athletes deliberately do not report symptoms for fear of being removed from the game or letting their teammates down.<sup>10</sup>

Reported concussion rates in a given sport (soccer, basketball, college ice hockey) tend to be higher in females than males.<sup>1,15,16</sup> The reason for this difference is not entirely clear. Some studies have showed that stronger neck muscles are better able to absorb force and dampen the forces transmitted to the brain.<sup>17,18</sup> Because males usually have more muscle mass than females, this anatomic difference has been suggested as a reason for the difference in reported rates.<sup>19</sup> Other authors have suggested that women are more honest in reporting their concussion symptoms, leading to a higher reported rate.<sup>20</sup> Regardless of the reported rates, concussions are common injuries in high school sports, accounting for about 10% of all injuries in contact/collision sports according to the US high school data reporting.<sup>1</sup>

## Pathophysiology

To better understand the pathophysiology of brain injury, animal models for TBI have been developed. The lateral fluid percussion (LFP) brain injury model has emerged as the model most applicable to concussion and mild TBI.<sup>21</sup> The model, which produces injury to the brain using a device to drive fluid against the intact dura of an exposed brain surface, produces a small focal brain contusion and small amount of surrounding hemorrhage. While the focal contusion may mimic a moderate or severe brain injury pattern (coup-contra coup injury), secondary metabolic effects are also produced at areas distant to the injury. The secondary effects produced by LFP have become the model for mild TBI, including concussion.

Additional models developed by the National Football League have demonstrated that the sheer force and rotational deformity associated with trauma correlate better with concussion symptoms than direct trauma to the brain.<sup>22</sup> Subsequent LFP models in developing animals have demonstrated a concussion pattern injury in the absence of focal cell death.<sup>23</sup> Overall, these newer models have helped establish the validity of the LFP model.

The LFP model begins to explain the pathophysiological processes that occur at the cellular level after a concussion. The concussion injury can be thought of as a 2-part process: a primary insult and a secondary inflammatory response. In rat models, the initial insult results in a pathologic release of excitatory amino acid neurotransmitters (glutamine and aspartate) that lead to loss of cell wall integrity. Subsequent changes in the permeability of the cell wall allow an influx of sodium and an efflux of potassium. The changes in intracellular sodium and potassium alter the pH of the cell, leading to an influx of calcium. Further disruption in concentrations of intracellular ions and pH results in cellular damage. As severely injured cells die, they release cytokines that upregulate the inflammatory process.<sup>22</sup> Secondary injury occurs with the upregulation of the inflammatory response.<sup>24</sup> This cascade of cell injury may explain why concussive symptoms can worsen clinically over the first 6-24 hours after the initial injury.

Axonal stretch injury is also being recognized as a contributing factor to brain dysfunction during a concussive injury. Diffuse axonal stretch injuries are well recognized in child abuse cases, such as “shaken baby syndrome.” In the days following head trauma, diffuse axonal swelling and subsequent dysfunction not present on initial presentation have been observed.<sup>25,26</sup> This type of a delayed sequelae injury is mediated via intracellular signaling mechanisms that occur concurrently with the cytokine-mediated inflammation seen in cellular injury.<sup>21</sup>

Very recent research has started to demonstrate that that the forces associated with mild TBI can also produce axonal injury. In 1 recent study, a dynamic stretch of cortical axons at strains lower than 5% of the breaking point of the axon produced no overt pathologic changes. However, subtle physiological changes were detected. There was increased expression of sodium channels on the axonal membrane surface within 24 hours, suggesting subclinical injury. After a second repetitive injury, pathologic increases in intracellular calcium and subsequent axonal swelling and degeneration were observed.<sup>27</sup> This new study not only demonstrated that mild traumatic forces can cause axonal injury, but suggests that trauma before the “concussive blow” may be a factor in lowering the threshold force to cause a symptomatic concussive injury.

## Metabolic Effects Post Injury

Shortly after a TBI, the metabolic needs of the cell, including glucose requirements, increase.<sup>21,28</sup> Mild and moderately injured cells can upregulate Na/K ATPase-dependent ion membrane transport proteins and restore the intracellular pH balance. These ATPase proteins are fueled by glucose.<sup>28</sup> Similar Na channel upregulation, fueled by glucose-dependent ATPase proteins, is observed following axonal stretch injuries.<sup>27</sup> Glucose delivery via cerebral circulation after a brain injury is thus crucial to restoration of intracellular ion balance and cell membrane healing.

Cerebral circulation is regulated by numerous homeostatic mechanisms. Coupling between cerebral blood flow and metabolism is the most important mechanism. To adjust to cellular metabolic demands, the body can alter cerebral blood flow at both a global level and a regional level. During resting levels, cerebral blood flow corresponds to a basal oxygen extraction rate. However, during increased brain activity, cerebral blood flow increases more than the oxygen extraction rate, implying that cerebral blood flow can be independent of local tissue needs for oxygen.<sup>29-31</sup>

After an injury, the brain should increase cerebral blood flow to increase delivery of nutrients, including glucose, to the injured cells. However, the cellular response to injury restricts cerebral blood flow, and the flow metabolism coupling is disrupted.<sup>29</sup> This has been demonstrated in both juvenile and adult animal models and in adult trauma victims.<sup>21,26,32</sup> If cerebral glucose needs remain low (concept of pharmacologically induced coma), the cerebral perfusion and glucose delivery may be sufficient for the cellular needs. However, if the demand for glucose is high, the body is unable to upregulate cerebral blood flow and the glucose needs of the injured cells are not met.

The “metabolic mismatch” of increased cerebral glucose needs and functionally decreased cerebral blood flow is the fundamental concept in acute concussion management.<sup>33</sup> During the early phase of brain healing, a tenuous equilibrium between glucose use and glucose delivery develops. Recently, this has been coined as a “concussion penumbra,” similar to the ischemic penumbra that occurs following an ischemic stroke.<sup>34</sup> In animal models, the cerebral glucose metabolism remained abnormal for about 2 weeks. This same abnormal cerebral glucose metabolism has been observed in hospitalized TBI patients for weeks,

**TABLE 1.** Different types of brain dysfunction

Symptoms: somatic (headache), cognitive (difficulty concentrating), and/or emotional symptoms (depression)
Physical signs (visual tracking and balance problems)
Behavioral changes (irritability, change in personality)
Cognitive impairment (slowed reaction times, memory problems)
Sleep disturbance (difficulty falling asleep)

Adapted from McCrory P et al.<sup>7</sup> Reproduced with permission.

but duration of abnormalities following mild TBI is unknown.<sup>20</sup>

During the acute healing phase, the brain is more vulnerable to additional stress. Mild physical trauma to the brain may worsen the cell injury cascade and cause significant injury. Cognitive work, such as the intense or prolonged concentration needed with school, will increase the metabolic demands of the cells. Physical activity, including weightlifting, which can change blood pressure and blood flow patterns, will exacerbate symptoms. This concept of brain vulnerability, especially early in the healing phase, is the basis for the concept of complete cognitive (no school) and physical rest while symptomatic.<sup>35</sup> Early concussion management seeks to protect vulnerable cells and axons by minimizing cerebral glucose demands and avoiding additional strains on cerebral blood flow.

## Signs and Symptoms

In defining concussion, the 3rd International Concussion Conference statement deliberately defined concussion broadly enough to encompass different types of brain dysfunction. Impairment of neurological function can present in multiple ways (Table 1).<sup>7</sup>

Presentation after a concussion can be variable and symptoms may not occur for a few hours after the initial trauma. Acute concussion symptoms (Table 2) can mimic migraine or attention deficit disorder symptoms. Loss of consciousness, occurring in about 10% of concussions, is not a reliable marker for defining the occurrence or severity of a concussion.<sup>4,7</sup> Chronic symptoms can present as persistent headaches, mood disorders (depression/anxiety), sleep disorders, or poor cognitive/school function. Occasionally, symptoms are not recognized until cognitive stress, such as school work, makes the attention or memory deficits more noticeable.

**TABLE 2.** Concussion signs and symptoms

Concussion symptoms	Concussion signs
Headache or "pressure" in head	Appears dazed or stunned
Nausea or vomiting	Is confused about assignment or position
Balance problems or dizziness	Forgets an instruction
Double or blurry vision	Is unsure of game, score, or opponent
Sensitivity to light	Moves clumsily
Sensitivity to noise	Answers questions slowly
Feeling sluggish, hazy, foggy, or groggy	Loses consciousness ( <i>even briefly</i> )
Concentration or memory problems	Shows mood, behavior, or personality changes
Confusion	Can't recall events <i>prior</i> to hit or fall (retrograde amnesia)
Does not "feel right" or is "feeling down"	Can't recall events <i>after</i> hit or fall (antegrade amnesia)

Adapted from the Centers for Disease Control Heads Up concussion campaign.<sup>36</sup>

## Evaluation and Diagnosis

Depending on the part of the brain injured, the presentation of concussion can be quite variable, making the diagnosis of concussion difficult. Concussion symptoms often overlap with multiple other medical conditions. Exertional headaches, migraine headaches, and dehydration can all present as a headache after exercise. Anemia, overtraining, or inadequate sleep all can present with fatigue. Attention deficit disorder, learning disabilities, absence seizures, and mood disorders can all present with concentration problems or poor school function. However, the diagnosis of concussion should be suspected in any individual who presents with signs or symptoms of a concussion (Table 2) shortly after a traumatic blow to the head or body.<sup>7</sup>

After a traumatic injury, the on field evaluation should start with the basic ABCDE trauma evaluation. The primary survey includes evaluation of airway, breathing, and circulation. The subsequent disability and exposure evaluation should first evaluate for a cervical spine injury. The cervical spine should be stabilized until it has been adequately evaluated. If the athlete has lost consciousness or is not coherent, then a cervical spine injury should be suspected. In athletes wearing a helmet and shoulder pads, their equipment should not be removed.<sup>7,37</sup> The facemask of a football helmet may be removed with either a screwdriver or bolt cutters to gain access to the airway as needed.<sup>37</sup> The presence of midline cervical tenderness, altered level of alertness, neurologic abnormality, or the

presence of a painful distracting injury are all indications for further cervical spine evaluation.<sup>38</sup> Transport to the nearest ED may be necessary to obtain a complete assessment with appropriate imaging studies.

The initial sideline evaluation should focus on the mechanism of injury, neurologic examination with balance testing, and an assessment of neurological function. An unanticipated collision is likely to transmit more force to the brain and should increase the suspicion for a concussion.<sup>20</sup> Balance problems and visual tracking deficits are common concussion findings but any focal neurological deficits should be referred immediately to the ED.<sup>39</sup> Simple assessment of orientation to person, place, and time is not a reliable screen for a sports-related concussion.<sup>40,41</sup> Cognitive evaluation should include situation relevant questions, such as name of opponent, score of the game, and time remaining in the game. If an athlete clinically does not appear to have a concussion and is returned to the game, he/she should be periodically monitored because concussion symptoms may develop over hours. Athletes removed from the game need to be monitored for progressive neurological decline. The Zurich Concussion guidelines (free download at [http://bjsm.bmj.com/content/43/Suppl\\_1/i76.full](http://bjsm.bmj.com/content/43/Suppl_1/i76.full)) include standardized concussion assessment tools-2 (SCAT2) that can aid in sideline evaluation.<sup>7</sup>

An ED or physician office evaluation should include a thorough history and physical examination. Balance deficits are common and should be included in the assessment.<sup>42,43</sup> A number of techniques have been described to test balance,<sup>8,44-46</sup> but a simple test of heel to toe walking backwards (with eyes closed to add difficulty as needed) can be done in any setting. Computed tomographic (CT) scanning is indicated when the injury pattern and physical examination are concerning for a diagnosis of skull fracture or intracranial hemorrhage. Focal neurological findings, progressive neurological decline, or a high-risk mechanism of injury, such as a motor vehicle accident, are all findings that would support additional imaging.<sup>7</sup>

The need for urgent CT imaging is a slightly controversial topic. From a public health perspective, the indiscriminate use of CT is suboptimal. The lifetime risk of cancer due to 1 head CT is age dependent, with the youngest individuals the most vulnerable. The lifetime risk of cancer for a single head CT is about 1 in 2000 for children under 2 and about 1 in 10,000 for a 15-year-old female.<sup>47</sup> The Pediatric Emergency Care

**TABLE 3.** Low-risk characteristics for clinically important brain injuries

Normal mental status
No loss of consciousness
No vomiting
Nonsevere injury mechanism
No signs of basilar skull fracture
No severe headache

Applied Research Network has developed a prediction rule to avoid doing unnecessary head CT scans in children older than age 2. In a large prospective study, a history and physical with all low-risk characteristics (Table 3) supported not doing a CT scan with a negative predicted value of 99.95% (3798/3800, with the 2 missed injuries not requiring surgical intervention).<sup>48</sup>

Ultimately, the need for imaging and the diagnosis of a concussion remain a clinical decision. There are no tests to “prove” that an individual has sustained a concussion. The diagnosis of concussion implies that a more severe injury, such as a brain contusion or bleed, is not present. It also assumes that there is not another reason for the altered brain function, such as intoxication, infection, anatomic brain problem (Chari malformation, etc), or mass lesion (tumor). Brain imaging, which by definition is normal in a concussion, is indicated if the clinical history and physical examination are concerning for an alternative diagnosis. An individual who has the signs and symptoms of concussion shortly after a traumatic blow can be diagnosed with a concussion if the complete medical evaluation (history, physical examination,  $\pm$  imaging, cognitive testing, laboratories) supports the diagnosis.

## Role of Neuropsychological Testing

The development of neuropsychological testing has given clinicians an additional tool to evaluate head injuries. Traditional neuropsychological testing has been the gold standard in documenting deficits in cognitive function. Neuropsychological testing has been demonstrated to detect deficits following sports-related concussions.<sup>49</sup> It is good for documenting deficits, particularly if the deficits are fixed or stable. However, traditional “pencil and paper testing” is both time-consuming and labor-intensive. Because concussion symptoms may resolve quickly, testing is only practical if it can be done quickly and repeated after a short period.

Computerized testing has been developed to make testing more practical. It is easily administered, takes only about 30 minutes to complete, and can be repeated to evaluate transient neurological deficits. Commercial products are routinely used at the college and professional level. Because most of the sports concussions occur at the high school level, there has been a recent push to use testing at the high school level. Commercial computerized testing is currently available for adolescents and is in the final stages of development for the pediatric age patients (ages 5-12).

The reliability of these computerized tests has been debated.<sup>50-53</sup> Traditional pencil and paper testing can be affected by many factors, including physical fatigue and patient effort. While testing is an imperfect tool, standards to assess test-to-test variability have been developed. When compared with traditional pencil and paper tests, the computerized tests have more test-to-test variability. Greater variability makes the confidence intervals larger, making it more difficult to detect minor differences.<sup>49</sup> Scores above or below a previous score may not be detecting true differences, just test variability. However, emergency trauma evaluation testing has documented deficits on computerized testing that have resolved on follow-up examinations.<sup>54</sup> Computerized testing has also detected deficits in self-reported “asymptomatic” athletes<sup>55,56</sup> that resolved with time.<sup>55</sup>

Optimally, computerized neuropsychological testing is done by the athlete in the preseason before any injury. This establishes a baseline score unique to the athlete. After an injury, the test is repeated. Documentation of cognitive function below the athlete’s baseline helps support but does not make the diagnosis of a concussion.

If baseline preinjury data are not available, then neuropsychological testing can be compared with normative age-matched data. This should be done with caution, as there can be several confounding variables.<sup>57</sup> The obvious problem is that normal scores in those individuals who would score very high on baseline testing may lead to premature clearance. Similarly, individuals who would score below average at baseline may be inappropriately denied clearance. The normative data were derived from healthy individuals, so it may not be applicable to individuals with known learning disorders (dyslexia, etc) or concentration problems (attention deficit hyperactivity disorder).<sup>58</sup> Normative data that do not distinguish between male and females may also be inaccurate.<sup>21,59</sup> Other

medical conditions may affect testing, such as an individual on an epilepsy medication that decreases reaction time.

Other additional factors can affect computerized neuropsychological testing. Some athletes may deliberately not give maximal effort during the baseline test. When they then retake the test after an injury, they may be able to match their previous score despite not being fully healed.<sup>60</sup> The “practice effect” is a concern when tests are repeated over a short time interval.<sup>61</sup> If athletes take a similar test several times in a short period, they may score better on the repeat tests due to “learning” how to take the test. Test improvement would not correspond to neurocognitive improvement. Comparing adolescent athletes to their baseline is valid if the injury occurs shortly after the baseline test is done.<sup>59,62</sup> However, there are no data on the duration of the reliability of the baseline test. Normative raw score data do change at least yearly in adolescent athletes as the brain continues to develop.<sup>63</sup> Additional factors that affect traditional pencil and paper neurocognitive testing, such as sleep deprivation, stimulant use, and exercise before testing, have also been demonstrated to affect computerized testing performance.<sup>64</sup>

In summary, neuropsychological testing is meant to enhance, not supersede, clinical judgment. Neurocognitive testing may detect deficits in reportedly asymptomatic athletes.<sup>55,56</sup> There are multiple variables that may affect testing performance, so all testing should be interpreted in the context of the unique clinical situation. If the clinician accepts the premise that computerized testing will detect some but not all the deficits that traditional pencil and paper testing will detect, then computerized testing can be a valuable tool in concussion evaluation and management.<sup>53</sup>

## Recovery Issues

### Age

Age is often an underappreciated factor in concussion management. It is well accepted that high school aged athletes take longer to heal than older athletes.<sup>65-68</sup> Although it is not understood why younger individuals take longer to heal than adults, animal model data suggest that the developing brain may be more sensitive to the pathologic release of excitatory amino acid neurotransmitters (glutamine and aspartate) following trauma than adult brains.<sup>69</sup>

Adults and professional athletes frequently heal quickly from a concussion, with cognitive testing back to baseline within 3-5 days of initial injury.<sup>66</sup> College athletes take slightly longer to heal, with average recovery times of 5-7 days.<sup>67</sup> High school athletes take even longer to heal with average healing times of 10-14 days on computerized neuropsychological testing.<sup>68,70</sup> Formal studies on the 10- to 14-year age group are lacking, but clinical experience suggests that healing times are at least equal to if not longer than high school athletes.

Average healing times are useful in demonstrating the differences between adults and adolescent patients. However, median healing times are clinically useful, because it is the subset of the concussion patients with prolonged symptoms that require the most clinical management. In high school football players suffering from a concussion (average age 16), more than half of them took longer than 1 week to heal, and 10% took longer than 3 weeks. The healing rate was not linear, with a subset of athletes with prolonged symptoms.<sup>9</sup> Similar studies in full-time adolescent students have demonstrated prolonged healing greater than 6 months in a small (6%) subset of concussed individuals.<sup>71</sup> Leading authorities have supported this observation, reporting that a small subset (10%-20%) of concussed high school athletes report symptoms for more than a month.<sup>72</sup>

In pediatric patients (average age 14) presenting to the ED with mild TBI (Glasgow Coma Scale 14-15 on presentation) who were admitted for observation, more than half still had symptoms at a follow-up visit 2-3 weeks after their injury. In this study at a large urban children's hospital, only 13% of the injuries were from sports (27% were falls). Patients with penetrating injuries were not included in this study.<sup>73</sup> Although not directly applicable to sports injuries, the younger age of the patients in this study suggests that a higher percentage of younger patients will have more prolonged symptoms.

### *Concussion History*

A history of 3 previous concussions has been well documented as a risk factor for both increased recovery time and future reinjury in high school and college athletes.<sup>74</sup> Initial studies documented more severe on-field presentation,<sup>74</sup> delayed recovery times,<sup>9,75,76</sup>

and a threefold risk for future concussion in individuals with 3 or more concussions.<sup>77</sup> Three or more concussions were also associated with long-term neurological deficits or decline in cognitive performance.<sup>76</sup> Individuals with 3 previous concussions who were prospectively followed also showed greater neurocognitive declines following a fourth concussion compared with matched controls who sustained their first concussion.<sup>78</sup> Studies using computerized neuropsychological testing have also reported no cumulative effects after 1 or 2 previous concussions,<sup>79</sup> suggesting a benign recovery after the first and second concussion.

Despite previous evidence that 1 or 2 concussions do not lead to chronic or prolonged deficits, recent studies have begun to diverge from this assumption. Neuropsychological testing in college football players with 2 or more concussions has suggested long-term deficits in executive function.<sup>80</sup> Concussed collegiate athletes

with a history of 2 or more concussions took longer to recover verbal memory and reaction time on computerized neuropsychological testing than athletes without a history of concussion.<sup>81</sup> Concussed soccer players with a history of 1 or more concussions scored worse on computerized neuropsychological testing than those without a history of concussion.<sup>82</sup> Balance testing also has

documented delayed healing times after a second concussion compared with the first.<sup>45</sup> Of note, in this particular balance test study, the deficits were documented at 30 days post injury. These athletes in general had normal computerized neuropsychological testing and had been clinically cleared to return to play.<sup>45</sup>

More studies are documenting subtle deficits in the athlete with apparently normal neuropsychological test scores. One study in female college soccer players demonstrated simple memory and reaction time scores, easily measured on computerized testing, were normal compared with the controls. However, the concussed players still showed deficits of cognitive processing speed on pencil and paper neuropsychological testing 6 months after the first concussion.<sup>83</sup> Finally, 2 recent studies have detected subtle electroencephalography abnormalities in asymptomatic individuals.<sup>84,85</sup> One of these studies detected abnormalities in young indi-

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***It is well accepted that high school aged athletes take longer to heal than older athletes.***

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viduals (age  $19.7 \pm 1.3$ ) with a history of concussion on average 3.4 years before testing. Compared with controls, there were no differences in computerized neuropsychological testing.<sup>84</sup> While the clinical significance of these electroencephalography changes is unknown, preliminary evidence suggests that there may be subtle deficits and a cumulative effect from concussions that are not evident with simple computerized neuropsychological testing after a first or second concussion.

## Gender

Baseline neuropsychological testing has documented that female athletes score differently on neuropsychological testing than males. Females score better on verbal memory testing and worse on visual memory tests compared with male athletes.<sup>59,86</sup> After a concussion, this same trend is noted, with female athletes scoring significantly worse on visual memory tasks than concussed male athletes.<sup>87</sup> Gender differences after concussions have also been noted in concussed athletes with women having greater declines in baseline testing, greater decreases in reaction times, and higher self-reported symptoms compared with males.<sup>88</sup> These studies would support the use of individual baseline neuropsychological testing or, at a minimum, normative data that are gender specific.

Gender differences in incident, severity, and recovery patterns are still being defined. There have been several studies that have found that in similar sports (soccer, basketball, ice hockey) women are at increased risk for concussion compared with men.<sup>14-16,89</sup> Because women have different neurocognitive scores on baseline testing compared with men, it is unclear if their worse neurocognitive performance after concussion reflects a more severe injury or just baseline differences. Adult women, but not adolescent females, have a higher reported rate of post concussion symptoms than men.<sup>90,91</sup> In hospitalized TBI patients, women have worse outcomes compared with men<sup>92</sup> but it is unknown if this is applicable to concussions.<sup>93</sup> Large reviews on this topic have concluded that there is a trend to suggest that gender is a risk factor for concussion severity, but more data are needed to make this conclusion.<sup>6,20</sup>

## Comorbid Conditions

Comorbid conditions, such as migraine headaches, attention deficit disorder, sleep disturbances, depres-

sion, anxiety, and other mood disorders, are being recognized as important factors when evaluating individuals with a concussion.<sup>94</sup> There are some adult data that suggest that individuals with preexisting depression have more acute postconcussion neurocognitive deficits than controls.<sup>95</sup> Clinical experience suggests that individuals with premorbid conditions suffer from greater functional impairment and more symptoms than similar age- and sex-matched peers following a concussion. In addition, patients often experience worsening symptoms of their comorbid conditions following a concussion. It is speculated that individuals with comorbid conditions have less "reserve" and are unable to compensate for concussion deficits as effectively as their peers. However, it is not clear if a concussion worsens a comorbid condition, impairs compensation in individuals with an underlying comorbidity, or a combination of both.

Adult researchers are starting to explore the relationship between mild TBI and comorbid conditions. Previous studies in adults with TBI have reported very high rates of post injury depression (40%) and sleep disturbances (40%-50%).<sup>96,97</sup> Significant limitations to applying these data to pediatric and adolescent patients include older ages of patients, reporting biases among adults with secondary gain issues,<sup>97</sup> and samples that include patients with mild, moderate, and severe TBI. Newer studies are suggesting depression rates of 15%-20% within 1 year following mild TBI.<sup>98,99,100</sup> Researchers using military data on US servicemen returning from Iraq who suffered from mild TBIs in a combat environment are also reporting higher levels of anxiety and depression compared with similar groups who did not suffer a mild TBI.<sup>101</sup>

Unfortunately, the literature is scant regarding the concussed high school age athlete with associated comorbidities. Most concussion research has not been appropriately powered to distinguish recovery differences in individuals with comorbid conditions compared with age- and sex-matched controls. As discussed previously, the use of common neuropsychological testing tools is also limited, because common computerized tests do not include individuals with learning disorders in the normative data.

Until the relationship between concussion and these comorbidities is further understood, clinicians caring for patients who have suffered a concussion should evaluate for comorbidities and continue to monitor for late presentations of comorbid conditions. To date there is no good evidence that comorbid conditions

increase the risk for concussion. It is also unknown if concussions are an “environmental stress” in susceptible individuals that trigger these conditions. There is some evidence to suggest that individuals presenting with some of these symptoms may had a prolonged recovery. High school and college athletes presenting with acute migraine-like symptoms (headache, light/noise sensitivity, nausea) had a longer recovery time when evaluated retrospectively.<sup>102,103</sup> At the current time, these comorbidities should be considered a risk factor, not fully defined, for a more complicated healing course.

## Management

Initial concussion management seeks to minimize the secondary cascade injury that occurs during the previously described metabolic mismatch phase. Early after the initial injury, the severity of the injury can be reduced by limiting cognitive and physical exertion. Because prompt recognition is critical, coaches, athletic trainers, medical personnel, and the athletes themselves must be trained to recognize signs and symptoms of concussion and act appropriately.

Sideline management of a concussion starts as soon as an athlete exhibits signs and symptoms of a concussion. The athlete should be removed from the game and evaluated on the sideline. In the high school age athlete, if the athlete clearly shows signs and symptoms of concussion at the time of initial evaluation, it is never appropriate to return the player back to play the same day as the injury regardless of the skill level or resolution of symptoms while on the sideline.<sup>7</sup>

Initial management in the ED or office setting is often supportive, with behavior modification counseling being the primary intervention. Immediately after a concussive injury, the most effective intervention is cognitive and physical rest. Activities that exacerbate metabolic mismatch, including school work, video games, and physical exertion, should be avoided.<sup>7,104</sup>

Animal models have suggested that early activity may worsen or prolong the initial injury,<sup>105</sup> so early return to school may complicate an initial injury. Once symptoms are minimal at home, the student-athlete can start to transition back to school.

The individual with prolonged symptoms may need a different treatment plan after the first month. Fluid percussion animal models have supported early brain rest during the acute recovery phase of mild TBI (3-4 weeks) followed by increased activity during the subacute phase to promote neuroplasticity.<sup>20,105-107</sup> Two very recent studies have supported this concept of permissive low-intensity exercising during the subacute phase while still symptomatic.<sup>108,109</sup>

In the first study, college students with prolonged post concussive symptoms did treadmill exercise testing while being closely monitored. They were encouraged to exercise to, but not beyond, a threshold where their symptoms started to get worse. Individuals were able to increase their intensity and their time to onset of symptoms over several weeks without any complications or decline in recovery. The athletes in the study were able to return to their sport once they were symptom free.<sup>109</sup>

In the second study, pediatric and adolescent-aged patients with symptoms longer than 1 month were enrolled in a supervised “active” rehabilitation. These symptomatic individuals were encouraged to do submaximal exercise and gradually increasing amounts of cognitive work. They could progress in duration of activity as long as their symptoms did not get worse.

Symptom resolution occurred faster than expected without any significant setbacks.<sup>104</sup>

Overall, general management concepts support early brain rest during the “metabolic mismatch phase.” As the student progresses into the subacute healing phase, gradual reintroduction of well-supervised activity, even while symptomatic, may be appropriate as long as it does not exacerbate symptoms. Specific school management and return to sports concepts are discussed in more detail in the following sections.

## School Management

In the preadolescent and adolescent age group, the concept of brain rest must be balanced with the academic demands of school. In individuals with mild symptoms, a few days of rest at home before returning to school is usually well-tolerated. Because concussion symptoms usually worsen with the increased cognitive strain of school, returning to school is not

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***Early return to school may complicate an initial injury.***

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generally recommended until the symptoms are mild or absent at rest. In individuals with significant symptoms, early return to school likely will not be possible or in their best long-term interests. This concept of brain rest requires a paradigm shift in how schools reintegrate concussed students.<sup>34</sup>

Students with severe symptoms must overcome significant barriers if they return to school while still cognitively impaired. First, severe headaches, impaired memory, or impaired attention span impedes new learning. As they try to concentrate more, their symptoms frequently get worse. The school environment, including requirements for prolonged concentration and noise associated with crowded loud hallways, may also make the symptoms worse. Because the concussed individuals look normal, the teachers frequently expect them to function normally. A student with a leg cast is excused from running in gym class without a second thought, but concussed students are often accused of malingering when they cannot keep up with a normal academic load. Finally, when they return to school after a brief absence, they are often expected to complete all make-up work quickly, effectively doubling the academic load.

Students with severe symptoms should transition back to school gradually. Complete brain rest with no school and avoidance of activities that require concentration, such as video games, are appropriate initially. As symptoms improve, homebound education can allow the students to gradually resume work in short intervals at home in a quiet environment. Once they can concentrate for a few hours without significant symptoms, they can progress to returning to school, often starting with half-day classes. At this point, close collaboration with the school officials, especially the guidance counselor, is required to ensure that teachers' expectations are reasonable. They may need temporary learning accommodations that are given to special needs students, such as untimed tests, help with note-taking, and additional time to complete assignments. They should not be expected to make up all the missed work until they can function well with a normal course load. Returning to school does not clear them for physical activity. Students should not be expected to write papers for gym class (and thus increase their cognitive load) as a substitution for physical activity. Monitoring should continue until the student has returned to regular school and has made up all the old school work.<sup>104</sup>

## Symptom Management

Managing the surrounding environment to limit cognitive and physical stress is currently the most effective treatment of concussion symptoms. The goal is not to eliminate the baseline symptoms, but to minimize environment-induced symptom exacerbation. Clinical experience also suggests that adequate sleep shortly after the concussion helps in the recovery process. Several pharmacologic treatments have been used to help manage the symptoms of concussion. To date, there is no strong evidence to recommend any specific treatments.<sup>110</sup> The most common treatments will be briefly discussed below:

### Calcium Channel Blockers

Because calcium influx is part of the pathologic process that contributes to secondary brain injury, early calcium channel blocker use in TBI patients would have a theoretical benefit. LFP animal models have shown neuroprotective effects with early calcium channel blocker use.<sup>111,112</sup> Unfortunately, these benefits have not replicated in TBI injury patients.<sup>113-115</sup>; thus, calcium channel blocker use is not currently recommended after TBI.<sup>115</sup>

### Amitriptyline

Amitriptyline is a tricyclic antidepressant that is also used by neurologists and chronic pain specialists as a prophylactic medicine to treat migraine headaches. Studies using amitriptyline to treat post traumatic headaches have been mixed.<sup>116,117</sup> Because many concussion headaches have a migrainous nature, amitriptyline may be beneficial to selected populations.

### Melatonin

Although there is anecdotal evidence to suggest melatonin (dose 1-6 mg) helps some concussed individuals with insomnia, there are no prospective studies that evaluate melatonin use in concussion patients.

### Return to Play

The return-to-play protocol (Table 4) published in the Zurich guidelines<sup>7</sup> is widely accepted as the standard of care in management of return to play. In

**TABLE 4.** Graduated return-to-play protocol

1. No activity: Complete physical and *cognitive* rest until asymptomatic at rest. Once an athlete is symptomatic at rest, progress through following stages. Each stage should take a minimum of 24 hours to complete. Progress to the next stage only if asymptomatic with the new activities. If the new stage provokes symptoms, return to the previous stage for at least 24 hours.
2. Low levels of physical exertion as tolerated (symptoms do not get worse or come back during or after the activity). This includes walking, light jogging, light stationary biking.
3. Moderate levels of physical exertion as tolerated. This includes sport-specific exercises such as skating drills in ice hockey, running drills in soccer, but no head impact activities.
4. Noncontact sport specific-training drills including passing drills in football and ice hockey; may start progressive weight training.
5. Full contact practice following medical clearance, participate in normal training activities.
6. Normal game play.

Adapted from McCrory P, Meeuwisse W, Johnston K, Dvorak J, Aubry M, Molloy M, et al. Consensus statement on concussion in sport third international conference on concussion in sport. *Clin J Sport Med* 2009;19:185-200. Reproduced with permission.

general, the athlete progresses from 1 step to the next as long as he remains asymptomatic for 24 hours at each step. If he becomes symptomatic during 1 of these steps, he returns to the previous step for at least 24 hours. Athletes must be off any medications that are being used to treat symptoms to be considered symptom-free at rest.

This experienced-based protocol was designed to return athletes back to sport safely. It was structured to allow sufficient time for the brain to heal. Because recent studies in high school athletes using neuropsychological testing have documented deficits in 10% of the asymptomatic individuals,<sup>54</sup> symptom reporting alone may not be sufficient to document healing.

A recent prospective large volume study has suggested that time after injury should also be considered in return-to-play management. In 635 concussed high school and college football players, 24 had a repeat concussion in the same season. About 60% of the athletes had at least a brief symptom-free period before returning to play, while 40% of these athletes returned to play despite having symptoms. Surprisingly, the group that had at least a brief symptom-free period had a higher rate of reinjury than the group that returned to play while symptomatic. However, most of the concussion reinjuries occurred within 7-10 days of the initial injury. The symptom-free individuals who were reinjured (on average 2.8 days symptom-free) did not follow the duration of the current Zurich guidelines (progressed faster) and were frequently playing within a week of their initial injury. The subset of individuals who had a longer symptom-free waiting period (5.8 days) was more consistent with the Zurich guidelines and returned to play on average at 10 days after their initial concussion actually had the lowest rates of reinjury.<sup>118</sup>

As this study demonstrates, defining asymptomatic is becoming more difficult. Balance testing is detecting deficits more than 1 month after a concussion in “asymptomatic” individuals who had returned to activities at least 2 weeks before testing. Interestingly, the scores in the concussion group were worse when they had to perform a simple mental task, such as reciting the months of the year backward while walking.<sup>119</sup> Perhaps with higher level function testing, subtle differences will be unmasked in “asymptomatic” individuals.

In the absence of a clear asymptomatic period, it has been suggested that athletes not return to play during the early period of increased vulnerability to another injury.<sup>118</sup> Experience from rodent models demonstrates that the cerebral metabolism does not normalize for 2 weeks. Human data demonstrate that 80% of same season concussion reinjuries occur within 10 days of the initial injury. Perhaps future return-to-play guidelines may suggest sitting out a minimum number of days from sport plus having a longer symptom-free period with full exertion before return in younger athletes.

While the Zurich guidelines provide an excellent template for return-to-play decisions, each case should be evaluated individually. Clinicians should use multiple tools, including neurocognitive testing, balance testing, and symptom scores, after physical and cognitive exertion to try to confirm healing before return to play. Age, concussion history, and comorbidities all contribute to return-to-play decisions. As most of these return-to-play studies involve high school and college-aged football players, the 11- to 14-year-old age group should be treated more conservatively.<sup>120</sup> Symptom severity, duration, and dysfunction caused by the concussion and a history of prior concussions, especially recent ones, should also be included in

the decision-making process.<sup>7</sup> Finally, there may be situations where return to play is not recommended or appropriate.

## Long-Term Issues

The different balance recovery times between the first and second concussion in “asymptomatic” athletes with normal neurocognitive tests underscore both the limitations in detecting symptoms<sup>84</sup> and the potentially cumulative effect of the previous concussion. Although no guidelines exist for individuals with multiple concussions, several expert recommendations have been made. Individuals who have suffered a series of concussions in a short period should have an extended period of noncontact activity (3-6 months) while remaining completely symptom-free before returning to contact sports. Individuals who demonstrate cognitive decline or develop structural brain lesions after trauma should not return to contact or collision sports. Finally, individuals who take progressively longer to heal from a concussion or who sustain a concussion from a decreasing amount of force should consider “retiring” from contact sports and transitioning to noncontact sports.<sup>121</sup>

Historically, concussions and mild head trauma have been viewed as brief, self-resolving injuries that have little sequelae for most individuals. Recently, some of these preconceptions have changed, in both the medical literature and the general press.<sup>122-125</sup> Chronic repetitive head injury (chronic traumatic encephalopathy) has been known to cause dementia in boxers since the 1920s, but the concept of chronic repetitive subconcussive injuries in contact and collision sports is only beginning to be explored.

Recent studies have documented premature dementia and elevated rates of mood disorders in ex-professional football players.<sup>126,127</sup> Autopsies of the brains of professional athletes involved in contact and collision sports have demonstrated increased deposits of tau proteins, particularly near the surface of the brain. These proteins are seen in other neurodegenerative conditions. There have also been fewer neurons within the brain parenchyma than expected for age, suggest-

ing premature nerve cell death.<sup>123-125</sup> Although the relationship of increased tau proteins and the premature nerve cell death is unclear, there is concern that the protein deposition is a result of microscopic injuries to the brain’s surface and that the protein deposits cause the cell death. There is speculation from the researchers that this pattern of protein deposition is caused by repetitive microtrauma and not just concussive events.<sup>122</sup>

## Conclusions

Concussion is a common injury in the preadolescent and adolescent student-athlete. The pathophysiology of concussion suggests that *cognitive* and physical rest is critical during the acute healing phase. Management of concussion should emphasize gradual reintroduction to both school and physical activity. All return-to-play decisions should be individualized, taking into account comorbidities and associated risk factors.

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***The pathophysiology of concussion suggests that cognitive and physical rest is critical during the acute healing phase.***

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