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Pediatric Mild Traumatic Brain Injury in the Acute Setting

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Pediatric mild traumatic brain injuries (mTBI), the majority of which are concussions, are an increasingly common reason for presentation to medical care. Over the past decade, the incidence of diagnosed pediatric mTBI has increased exponentially in the acute and primary care settings, likely due to increased awareness and recognition. Our understanding of the pathophysiology and optimal management of concussed patients has also rapidly evolved. As more and more children present to emergency departments across the country, acute care providers must remain current with the diagnosis and management of mTBI. This article will review the definition, epidemiology, pathophysiology, diagnosis, and management of mTBI in the acute setting.

Definition and Epidemiology

The most widely accepted definition of concussion comes from the Consensus Statement on Concussion in Sport, revised every four years by an international workgroup. The latest revision occurred in 2016, and defines a concussion as a complex pathophysiological process induced by biomechanical forces, the features of which include: “(1) an injury resulting from either a direct blow to the head, face, or neck, or other part of the body with an impulsive force transmitted to the head; (2) the development of short-lived impairment in neurologic function that resolves spontaneously, though symptoms and signs may evolve

over a number of minutes to hours; (3) neuropathological changes, though the acute symptoms largely reflect a functional disturbance rather than a structural injury, and, as such, no abnormality is seen on standard neuroimaging studies; and (4) a range of clinical signs and symptoms that may or may not involve loss of consciousness and typically follows a sequential course, however, in some cases may be prolonged.”¹ While the majority of concussions in the pediatric population are the result of sport,^{2,3} other mechanisms of injury include trauma such as falls, road traffic injuries, and assaults.

Due to expanded awareness, as well as a likely increase in true incidence, the diagnosis of concussion has increased dramatically over the past decade. Recent studies estimate that concussion diagnosis rates have doubled over the past 10 years.⁴ There are nearly 1 million emergency department (ED) visits per year for mild traumatic brain injury for patients of all ages, with the highest reported rates in patients age 12–17 years-old.⁵ The majority of ED visits for pediatric concussion are from male patients (estimates between 55% and 60%).^{2,3} The true incidence of concussion, however, is likely significantly higher than estimated, as studies have shown the majority of pediatric concussion is diagnosed and treated outside of an ED.⁶ The reported incidence will likely continue to rise as, between 2009 and 2014, all 50 states and the District of Columbia enacted legislation mandating that, following a concussion, children be cleared by a health professional prior to returning to sport. Studies have shown that such laws have led to increasing healthcare utilization for concussed patients.⁷

Pathophysiology

Multiple basic science and animal models of mTBI have demonstrated the metabolic cascade that occurs following a concussion. Sheer force and rotational deformity, rather than direct trauma, lead to mechanical damage to neurons and axons.⁸ Excitatory neurotransmitter release then leads to ion flux and membrane depolarization (see Figure 1). An influx of calcium interferes with mitochondrial function and causes temporary cell dysfunction. Repair mechanisms, using adenosine triphosphate (ATP) fueled sodium/potassium pumps, attempt to restore intracellular balance.⁹ During the early stages of the injury, glucose utilization to generate ATP is very high,¹⁰ ultimately increasing neuronal energy demand.⁹ Simultaneously, both pediatric and adult studies have shown cerebral blood flow, which delivers glucose, decreases in response to injury, leading to an imbalance in energy supply and demand.^{11,12} This creates a metabolic mismatch almost immediately after injury, and forms the basis for cognitive and physical rest recommendations very early in the injury process. Finally, axons themselves have been shown to undergo structural damage, as disturbances in membrane homeostasis cause cytoskeletal injury.¹³

Acute History: Signs and Symptoms of Concussion

Concussion symptoms can be classified into three domains: physical, cognitive, and emotional (affective) (see Table 1).¹ Physical symptoms include headache, nausea, vomiting, dizziness, balance problems, vision problems, photophobia, and phonophobia. Cognitive symptoms include difficulty concentrating, difficulty remembering, and feeling mentally slow and foggy. Affective symptoms include emotional lability, irritability, depression, and

anxiety. Physical symptoms tend to present earlier than cognitive or emotional symptoms, and also tend to resolve sooner.^{2,14} There seems to be a distinct entity, post-traumatic migraine, which encompasses prolonged headache, nausea, photophobia, and phonophobia,¹⁵ and can last for several months following the injury. This can occur both in migraine-prone children as well as children without a prior history of migraine headaches. The acute care provider should be aware of the evolution of symptoms in both history-taking and anticipatory guidance.

In addition, during history-taking, the provider must examine elements of the patient's past medical history pertinent to concussion recovery. Several populations have been shown to be at risk for prolonged recovery, including females, teenagers, those with prior concussion, those with underlying ADHD or a learning disability, and those with an underlying mood disorder (see "Special Populations" section below). A social history should ascertain the patient's current status in school to give the provider a sense of the cognitive demand that will be required of the patient during recovery (e.g., a child in elementary school has a very different cognitive demand than a teenager preparing for college entrance exams).

Physical Examination

In addition to a standard neurologic examination, there are specialized physical examination maneuvers to evaluate for vestibular and oculomotor dysfunction, very common post-concussive deficits. One version of the vestibular and oculomotor examination for concussion, the modified vestibular/oculomotor screening examination for concussion (mVOMS), described in Table 2 and Figure 2, includes tests for (1) nystagmus, (2) smooth visual pursuits, (3) fast saccades in both the horizontal and vertical direction, (4) gaze stability testing (the vestibulo-ocular reflex), (5) near-point convergence, (6) monocular accommodation and (7) balance/gait stability. This test has been standardized across providers in primary care, emergency medicine, and sports medicine at one large, tertiary-care pediatric institution, and takes approximately 2–3 minutes to complete.¹⁶ It can be performed in children as young as 6 years old. A similar version of this examination, the vestibular/oculomotor screening examination (VOMS), has been shown to positively correlate with symptom scales, with each individual element showing high predictive value in identifying concussed patients.¹⁷ The examination has shown excellent sensitivity for concussion, as in specialty clinics nearly 80% of concussed patients show at least one vestibular or oculomotor abnormality.^{18,19} In addition to its use in concussion diagnosis, the specialized examination also shows potential for helping prognosticate for patients with mTBI, as those with abnormalities are at risk for prolonged recovery from concussion,^{18,20} and vestibular deficits are more prevalent in children with prolonged symptoms.²¹ Finally, this examination has functional implications, as it mimics the eye tracking demands of children in the school classroom; vestibular and oculomotor deficits can impact school re-entry, and allow the provider to predict potential areas of difficulty in the school-aged population. It should be noted that, while performing a version of the examination is feasible in the office or emergency setting, and the 2016 International Consensus Statement on Concussion recommends performing a version of vestibular and oculomotor testing in this setting,¹ currently administration of the full spectrum of testing is not widespread and is yet to become a standard of care. We do anticipate that, because of its utility for diagnosis and

its prognostic value, more and more clinicians will perform vestibular and oculomotor examinations over the coming years in children with concussion.

Neuroimaging and Laboratory Testing

Diagnosis of mTBI is made by history and physical examination, and in general does not require neuroimaging. Consensus statements agree that imaging via either computed tomography (CT) scan or magnetic resonance imaging (MRI) of the head contributes little to the diagnosis of concussion.¹ In the ED, CT scan should be used when the clinician has suspicion for skull fracture or intracranial hemorrhage. Multiple studies have developed decision models to help rule out clinically significant intracranial hemorrhage, the largest and most widely utilized of which was published by Kuppermann et al in 2009.²² Of note, there is overlap between the higher risk features of traumatic brain injury in this study and symptoms of concussion, and not all patients who fail to meet the very low risk criteria in this study require a head CT.

Like CT, MRI has little role in the acute diagnosis of concussion. The vast majority of MRIs obtained on patients with mTBI are normal.²³ In patients with prolonged symptoms, MRIs are useful if the clinician is entertaining alternative diagnoses beyond post-concussive syndrome (e.g. a space-occupying lesion). More recently, functional magnetic resonance imaging (fMRI) has been used in patients recovering from mTBI, to show subtle abnormalities, even in asymptomatic patients.^{24,25} In the future, fMRI may play a role in helping rehabilitate patients with prolonged recovery, but currently does not play a role in the acute management of concussion.

Previously, there was little role for laboratory testing in the setting of mTBI, though more recently several serum biomarkers have been studied. Glial fibrillary acidic protein (GFAP), an intermediate filament expressed by astrocytes, has been shown to correlate to CT findings in those with more severe TBI in pediatric patients.²⁶ Recently, a pilot study in children with mTBI demonstrated that levels of GFAP in the serum drawn shortly after the injury correlate with symptom burden both initially and one month following the injury.²⁷ This suggests that there may be a role in the future for GFAP, among other potential candidate markers (including S100 calcium binding protein B, myelin basic protein, and ubiquitin c-terminal hydrolase-L1), as potential indicators for more severe concussion.²⁸

Neurocognitive Testing

Many computerized neurocognitive testing batteries are available for children suffering from concussion, which can be completed in a relatively short period of time. Prior studies have shown that such testing is feasible in an ED setting,²⁹ though one must keep in mind many acutely concussed children are too symptomatic to complete neurocognitive testing, and exposing children to these batteries in the acute setting may solely serve to exacerbate symptoms. The utility of neurocognitive testing acutely is somewhat controversial; some studies have shown patients with mTBI perform worse on computerized testing when compared with normal controls,^{29,30} even when asymptomatic, while other studies have shown lower raw scores on neurocognitive testing in the ED do not predict prolonged

recovery.³¹ Likely more useful than raw data is comparison between a patient's baseline testing and their performance following a mTBI. However, even with patients who have baseline testing available, there are limitations; for example, studies show patients do worse on baseline testing with less sleep,³² therefore a similar score between baseline and after a potential injury would not necessarily rule-out concussion. The most important use of such testing is likely in trending scores as a patient recovers, and determining clearance for return to sports.³³ Ultimately, neurocognitive testing, in conjunction with history and physical examination findings, may play a role in concussion diagnosis, but should serve as an adjunct and not a primary diagnostic tool.

Management: Cognitive and Physical Rest

Based on consensus statements and expert opinion, both from the International Conference on Concussion¹ and the American Academy of Pediatrics Council on Sports Medicine and Fitness,³⁴ the standard of care for treatment of mild traumatic brain injury in children and youth is cognitive and physical rest. Given the pathophysiology of concussion, and the metabolic mismatch that occurs early in the course of injury, some degree of rest is likely beneficial during the early recovery phase. However, the optimal dose and duration of rest remains controversial, with equivocal findings in the literature.

Early studies showed improvement in symptoms among those who rested at least one week after injury, though in the largest early study there was no comparison group.³⁵ More recent studies have demonstrated that prolonged rest may actually be detrimental. Thomas et al randomized a group of patients age 11–22 years to either 5 days of strict rest or 1–2 days of rest followed by a stepwise return to activity, and the strict rest group had an increased number and a slower resolution of symptoms.³⁶ Buckley et al randomized 50 college-age athletes to either strictly rest for 48 hours, or to not receive specific post-injury instructions, and the “no-rest” group was symptom-free sooner (5.2 vs. 3.9 days).³⁷ The provider must consider the psychological consequences of prescribing physical and cognitive rest; DiFazio et al have described an “Activity Restriction Cascade,” whereby removing a child from validating life activities, combined with physical deconditioning, contributes to the development of persistent postconcussion symptoms and lengthens recovery.³⁸ In addition, in contrast to prior recommendations that suggested complete abstinence from physical exercise while symptomatic, recent studies have also shown early exercise may not only reduce symptoms but reduce abnormalities in brain activity on fMRI imaging.³⁹

At the same time, the provider must be cautious in recommending early return to full activity, as too much activity too soon may prolong recovery. Brown et al recently showed that those in the highest quartile of cognitive activity within the first three weeks following injury had a more prolonged recovery,⁴⁰ and Silverberg et al showed sharp increases in mental activity lead to an increased risk of symptom spikes (it should be noted, however, that symptom spikes in and of themselves were not a risk factor for a prolonged recovery).⁴¹ Additionally, early cognitive and physical activity should not be equated with early return to activities that may result in another head injury. A second injury, prior to the full recovery of the first injury, may have severe consequences. Both basic science and animal models have shown a re-injury during a vulnerable window, usually within 1–2 weeks of injury, may

result in persistent and possibly permanent injuries,^{42,43} including ion channel dysfunction, structural neuronal damage, and cognitive impairments.^{44–46}

In recommending return to activity in the acute setting, the acute care provider must consider the eye tracking demands required in the classroom. Reading, using a computer screen, taking notes, and viewing objects close to one's face can be difficult if the child still has abnormalities in the vestibular and oculomotor examination (for example, horizontal saccades mimic reading a book, and vertical saccades mimic looking up at a board or screen and taking notes).

The optimal duration of rest after concussion remains unclear, but there is enough evidence to recommend a period of 24–48 hours of rest, followed by graduated return to activity in concert with a primary-care provider, a recommendation supported by the International Consensus Statement.¹ Clinicians can expect these recommendations to be refined in the coming years as more research on the treatment of concussions is undertaken.

Management: Medications

In the acute setting, analgesics including acetaminophen and ibuprofen can provide some pain relief (especially in the setting of a contact-related injury), however they generally are not helpful during the recovery phase and should not be continued after ED discharge. Often such symptoms occur as a result of excessive cognitive activity, and usually only improve with rest. If, during the acute stages of concussion, patients are requiring pain medication to enable participation in physical and cognitive activities, they are likely over-exerting themselves. Sleep disturbances, which can occur shortly after a concussion, can be treated with melatonin in the acute setting.⁴⁷ Other medications have been trialed outside of the acute setting for those patients with persistent post-concussive syndrome, including gabapentin, topiramate, and amitriptyline. While a complete discussion of their efficacy is beyond the scope of this review, amitriptyline has been shown to be most effective for post-concussive headaches.⁴⁸

Special Populations

Numerous studies have shown certain populations are at risk for a prolonged recovery from concussion, and therefore may require closer follow-up.

Age

Compared with college-aged athletes and adults, adolescents have demonstrated prolonged recovery time.^{49,50} Among school-aged children and teenagers, there is controversial evidence regarding which patients are at highest risk for prolonged symptoms. Some studies show the youngest patients take longer to recover,³ whereas others indicate that teenagers are predisposed to a longer duration of symptoms than younger children.^{51,52} There are likely some confounding factors in the differential recovery by age, as younger children may be inclined to report symptoms less frequently than older teenagers; additionally, older teenagers experience a larger cognitive burden upon return to school. In terms of neurocognitive testing, there appears to be a trend of prolonged recovery time by age; adults

(greater than 22 years-old) have been found to return to baseline in 3–5 days; college-aged young adults (age 18–22 years) recover in 5–7 days; and high school students (age 14–17 years) recover in 10–14 days.^{53,54} It has been hypothesized that the pediatric brain may have increased susceptibility to neurotransmitter-mediated excitotoxicity as mechanism for the longer recovery time when compared with adults.⁵⁵

Gender

Multiple studies have shown the females take longer to recover compared with males.^{51,56} While females do report more baseline symptoms than males, they take longer to return to their baseline following a mTBI.⁵⁷ Following concussion, females have been shown to score more poorly on neurocognitive testing.⁵⁸

Prior Concussions

Many studies have shown that a history of multiple (greater than two) prior concussions is an independent risk factor for a prolonged recovery.^{59,60} More recent studies have suggested that any prior concussion may predispose children to prolonged duration of symptoms.⁵⁶ On neurocognitive testing, those with a prior history of at least one concussion perform worse when recovering from a second injury, compared with those who have never suffered a prior concussion.⁶¹

Mood Disorder and Learning Disabilities

Mood disorders (both anxiety and depression) have been shown to increase the risk of development of post-concussive syndrome, as well as the risk for prolonged duration of symptoms and recovery.^{3,62} Children following concussion report significant mood-related symptoms and can develop novel psychiatric diagnoses,⁶³ though it is unclear if these come about a result of the mTBI or represent an underlying diagnosis that is unmasked by the injury. Those with a strong family history of mood disorders are likely at higher risk to develop depression and anxiety following an injury. Additionally, those children with attention deficit hyperactivity disorder (ADHD) have been shown to be at risk for prolonged recovery.⁵⁶ Interestingly, those with ADHD are actually at higher risk to sustain a concussion, in addition to their risk for prolongation of symptoms.⁶⁴

Prolonged recovery or more likely to report symptoms?

Some researchers have proposed that several of these underlying conditions may not necessarily predispose children to a prolonged recovery, but rather, represent conditions whereby children are simply more likely to report symptoms. Iverson et al examined symptom-reporting in non-concussed children, and found that girls, those with mood disorders, and those with migraine headaches were more likely to endorse symptoms resembling concussion than those without these conditions.⁶⁵ While a propensity to over-report symptoms may partially explain the prolonged recovery in these specific groups, there likely is also an element of true prolongation of symptoms as well. Regardless, the acute care provider must be cognizant of the risks in concussed patients with the above underlying conditions.

Follow-Up and Referral

Prior research has estimated symptoms in the vast majority of mTBI (as high as 80–90%) patients will resolve within 4 weeks.⁶⁶ Both studies and position statements have emphasized that most concussions can be managed by primary care providers.¹⁶ Referral to concussion specialists is indicated for patients who experience symptoms unresponsive to traditional rest after three weeks. These specialists can help coordinate education and neuropsychological resources, as well as facilitate referral to specialized physical therapists. Studies have proven that visual and oculomotor therapy can be successful for those with prolonged visual and vestibular complaints.⁶⁷

In providing referral and follow-up, the acute care provider should be aware of the specialized populations mentioned above, as, given their risk to prolonged recovery, they likely will require closer follow-up. Tools exist to aid the clinician in helping prognosticate for families. For example, recently, the Pediatric Emergency Research Canada Concussion Team published a clinical risk score for predicting persistent postconcussive symptoms from the emergency department, which includes points for being teenage, female sex, prior concussion history, migraine history, answering questions slowly, errors in balance testing, and reported symptoms of headache, sensitivity to noise, and fatigue.⁵²

Conclusion

Pediatric mild traumatic brain injury (mTBI) is an increasingly common reason for presentation to medical care. As the incidence of concussion diagnosis continues to rise, it is imperative for acute care providers to have an understanding of the epidemiology and pathophysiology of concussion, an awareness of specialized elements of the history and physical findings when evaluating the potentially-concussed patient, a knowledge of appropriate recommendations for rest and rehabilitation, and an appreciation of special, vulnerable populations at risk for a prolonged recovery.

Abbreviations

ADHD	attention deficit hyperactivity disorder
ATP	adenosine triphosphate
CT	computed tomography
ED	emergency department
fMRI	functional magnetic resonance imaging
GFAP	glial fibrillary acidic protein
MRI	magnetic resonance imaging
mTBI	mild traumatic brain injury

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Target Audience

This continuing medical education activity is intended for physicians, physician assistants, nurse practitioners, and emergency medical service providers who care for pediatric patients.

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Objectives

1. Discuss the pathophysiologic basis for mild traumatic brain injury and the metabolic mismatch that occurs following injury
2. Perform a concussion-specific history and physical examination and articulate their utility in concussion diagnosis and prognosis
3. Identify vulnerable populations at risk for prolonged recovery from pediatric mild traumatic brain injury
4. Describe the evidence base behind cognitive and physical rest following pediatric mild traumatic brain injury and explain appropriate return to activity guidelines to patients and families

Figure 1.

Biochemical cascade following concussion (Adapted from Grady MF, Master CL, Gioia GA. Concussion pathophysiology: rationale for physical and cognitive rest. *Pediatr Ann.* 2012; 41: 377–382; with the author's permission)

a.

b.

Figure 2.

Demonstration of saccadic and gaze stability testing in the vestibulo-ocular examination for concussion (a) To test horizontal saccades, the examiner's fingers are placed shoulder-width apart (horizontal) or forehead-chin distance (vertical), and the patient is asked to look between them for 30 repetitions. (b) To test gaze stability, the patient fixes gaze on an object (or the examiner's thumb) while nodding yes and then shaking head no side-to-side for 30 repetitions. (Adapted with permission, Plant G, Splaton D. Chapter 19: Neuro-ophthalmology. In: Spalton D, Hitchings R, Hunter P. Atlas of Clinical Ophthalmology, 3rd Edition. Elsevier Limited. Oxford, Great Britain. 2005.)

Table 1

Signs and symptoms of concussion

Physical Symptoms	Cognitive Symptoms	Emotional/Sleep Symptoms
Headache	Difficulty concentrating	Irritability
Nausea/vomiting	Difficulty remembering	Mood changes
Balance problems	Feeling foggy	Anxiety
Dizziness	Feeling slowed down	Depression
Vision problems		Emotional lability
Sensitivity to light/sound		Sleep disturbance

Table 2

Vestibulo-ocular examination for concussion

Physical Exam Element	How to Perform Examination	Abnormal Findings
Dysmetria	Finger-nose-finger, examiner's finger moving horizontally, 10 repetitions	Slow reaction time, past-pointing, intention tremor
Nystagmus	Examiner's finger moving horizontally, progressively more rapidly, stopping centrally	>1 beat of nystagmus at center of visual field
Smooth pursuits	Examiner's finger moving horizontally, progressively more rapidly	Red/watering eyes, symptom provocation (headache, dizziness, eye fatigue)
Fast saccades	Examiner's fingers shoulder-width apart (horizontal) and forehead-chin distance (vertical), 30 repetitions	Red/watering eyes, symptom provocation (headache, dizziness, eye fatigue, fogginess)
Gaze stability	Patient fixes gaze on examiner's thumb while nodding yes and then shaking head no side-to-side, 30 repetitions	Red/watering eyes, symptom provocation (headache, dizziness, eye fatigue, fogginess)
Near-point convergence	Patient holds pen with letters at arm's length, brings toward nose until becomes double	Letters become double at >6 cm from tip of nose
Gait/balance testing	Tandem heel-toe gait forward and backward with eyes open and closed	Raises arms for stability or widens gait, extreme truncal swaying