Mild Traumatic Brain Injury:
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By Marc Andrews, MD [1] and John Bruns, Jr, MD [2]

Traumatic brain injury may occur without visible head injury; it manifests as confusion, focal neurologic abnormalities, an altered level of consciousness, or subtle changes on neuropsychological testing. The initial evaluation includes assessment of the patient's airway and respiratory, circulatory, and neurologic status.

About 1.4 million incidents of traumatic brain injury (TBI) are reported in the United States each year,1 of which 75% are classified as "mild."2 Mild TBI (MTBI) results from a number of causes, including falls, interpersonal violence, and motor vehicle collisions.3 Many cases are sports-related (Box); football and wrestling in men and soccer and basketball in women are the primary sources.4 Patients with MTBI may present with varying neurologic findings. Intoxication, preexisting conditions, polypharmacy, and dementia are frequently encountered confounders during the evaluation. Management decisions may be challenging in the patient who appears well during the evaluation but who was lethargic, confused, or amnestic at the time of the injury. Patients may also present days or weeks after the event with postconcussive symptoms, such as headache, sleep disturbances, memory and concentration problems, and emotional lability.

In this article, we present a pragmatic approach to the patient with MTBI, using evidence-based guidelines when available. CLASSIFICATION OF MTBI

MTBI describes a condition in which there is little or no change from the patient's neurologic baseline after the traumatic event. Although the term "head injury" is often used interchangeably with TBI, this usage is inappropriate. Head injury is defined as clinically evident trauma above the clavicles, such as scalp lacerations, periorbital ecchymoses, and forehead abrasions. TBI refers to injury to the brain itself; it may occur without visible head injury. It manifests as confusion, focal neurologic abnormalities, altered level of consciousness, and/or subtle changes on neuropsychological testing. It may also appear as an abnormality on cranial CT or MRI scans or during intracranial surgeries.

"Concussion" is another term that is used interchangeably with MTBI and defined in various ways in the literature, often in the context of sports injuries.5 Historically, MTBI refers to head injury patients with resolving neurologic symptoms and a Glasgow Coma Scale (GCS) score between 13 and 15; concussion refers to head injury patients with loss of consciousness (LOC) or amnesia of varying durations. For consistency, we will use the terms "TBI" and "MTBI" unless we refer to a scale or a table using the term "concussion."

There is no evidence-based definition of MTBI; inclusion and exclusion criteria vary by classification scheme. The Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine defines MTBI according to the following criteria6:

- Grade 1: Any alteration in mental state at the time of injury (eg, feeling dazed, disoriented, or confused).
- Grade 2: Any loss of memory of the event immediately before or after the injury, with post-traumatic amnesia of less than 24 hours.
- Grade 3: Any period of LOC of less than 30 minutes followed by a GCS score of 13 to 15.7

MTBI may not be clinically evident during the initial medical and neurologic evaluations, whether because of symptom resolution or the use of insensitive assessments in very mild cases. The GCS is the most widely used system of global neurologic evaluation and one of the few systems that assigns an evaluation label that is associated with outcome (Table 1).7
Initially developed to describe patients who were in a coma after head injury, the GCS score facilitates communication of neurologic status among medical personnel. A score of 13 to 15 typically suggests mild injury; 9 to 12, moderate injury; and less than 9, severe injury. Some experts believe that patients with a GCS score of 13, who have a significantly higher rate of neurosurgical findings on CT, belong in the moderate category. Others include in this category only patients with a GCS score of 15 at the time of evaluation.

Although the GCS score is frequently used to grade TBI, the timing of when to determine the score varies considerably in the literature. A single GCS score determination is not sufficient for assessing the severity of a head injury. To optimize its utility, serial GCS measurements are required; the prognosis is worse if the score fails to normalize over time. On the other hand, the initial GCS score is a critical reference point when comparing patient groups and making an initial prognostic judgment.  

The GCS score should be obtained through direct interaction with the patient, after the initial assessment and cardiopulmonary resuscitation (if necessary) but before the administration of sedatives or analgesics that may alter the score. To identify the progression or resolution of TBI, a neurologic assessment, including GCS score, should be performed at regular intervals and when the patient's clinical status changes.

**PATHOPHYSIOLOGY**

MTBI can result from a direct blow to the head or from sudden deceleration or rotational forces that do not involve impact. Alteration in the patient's sensorium and cognitive dysfunction stems from the direct injury to neurons and the surrounding vasculature, with subsequent damage attributable to ischemia and metabolic perturbations.
Early studies comparing CT with MRI demonstrated that patients without pathologic findings on head CT could exhibit structural abnormalities on MRI, specifically in the cortex of the frontal and temporal lobes. These findings correlated with behavioral and neuropsychological abnormalities. Diffuse axonal injury at gray-white matter interface has also been demonstrated on MRI. Subsequent research using dynamic neuroimaging, including positron emission tomography (PET) and single photon emission CT (SPECT), shows impaired cerebral glucose utilization after injury. Decreased cerebral blood flow after injury has also been reported. The current model of brain injury proposes a state of excitatory neurotransmitter toxicity, whereby injury sets off a cascade of neurochemical and metabolic factors resulting in cellular glycolysis with lactate production. These perturbations cause an initial excitatory, followed by a regional metabolic, depression and manifest as functional abnormalities. It is likely that metabolic derangement in the injured brain contributes significantly to the prolonged period of morbidity experienced by many patients who do not have detectable structural lesions on CT or MRI scans. Some functional abnormalities that persist long after the initial injury are not identified with these anatomic imaging modalities (see "Postconcussive Syndrome" below). Future investigations that use more sensitive imaging tests and functional MRI for diffuse axonal injury may clarify the mechanisms and physiology in patients with persistent symptoms.

**INITIAL EVALUATION**

The condition of patients with TBI may deteriorate rapidly after an ostensibly innocuous presentation. Therefore, an immediate and methodical evaluation in accordance with Advanced Trauma Life Support protocols is recommended. This includes an assessment of the patient's airways; respiratory, circulatory, and neurologic status; and immobilization of the cervical spine. After this assessment, a more detailed survey and history taking may proceed. If the patient is unable to provide a reliable history, try to secure information from witnesses or family members. Early transfer to an appropriately staffed and monitored emergency department (ED) is critical and should be considered in those with an altered level of consciousness or focal neurologic deficits. Patients with intoxication or bleeding diathesis and those older than 60 years merit special attention because neurologic deficits may not be readily apparent.

Important historical aspects of the injury include the mechanism, duration of LOC, post-traumatic amnesia, and other injuries. One potential pitfall in the management of the patient with TBI is to assume that this injury is entirely responsible for the overall clinical picture. A comprehensive approach requires initial consideration of the reversible causes of altered mental status, including hypoperfusion, hypotension, hypoxemia, hypoglycemia, and drug toxicity. Strong consideration of possible antecedent events, such as syncope, transient ischemic attack, orthostasis, or dysrhythmia, is prudent, particularly if the patient is elderly.

Evaluation of the patient with dementia may be particularly challenging, especially when an attempt is made to determine whether an acute cognitive deficit is compounding the chronic disease. Cerebrovascular disease and Alzheimer disease may prevent a patient from full participation in the neurologic evaluation. Do not assume that abnormal findings reflect the patient's baseline status or that they are the result of an inability to communicate or participate. A consultation with the caregiver or long-term-care facility may help clarify the patient's preinjury functional status.

**IMAGING STUDIES**

**Cervical spine evaluation.** The potential for cervical spine injury is sometimes overlooked in a well-appearing patient with MTBI. Any patient with acute TBI is considered to have an associated spinal injury until proved otherwise. The American Association of Neurological Surgeons' guidelines state that radiographic assessment of a cervical spine injury is not required in trauma patients who are awake, alert, and not intoxicated; who have no neck pain or tenderness; and who do not have significant associated injuries that detract from their general evaluation. If these criteria are not met, the patient should have a cervical collar placed immediately. For further evaluation, obtain 3 views of the cervical spine with plain film radiography or cervical spine CT. The modality chosen depends on the patient's concomitant injuries; ability to participate; and preexisting conditions, such as degenerative disease or arthritis.

**Skull radiography.** This modality is neither sensitive nor specific for MTBI and therefore has no role in the evaluation of a child or adult with this condition. Although there is an increased likelihood of intracranial hemorrhage in patients with skull fracture, the prevalence of acute pathology in patients with a positive plain skull film (positive predictive value, 0.41) is low. Normal skull radiographs may be falsely reassuring. Although in one meta-analysis the negative predictive value of skull radiography was 0.94, low sensitivity precludes its use for intracranial pathology screening.

**Pediatric cerebral neuroimaging.** Children with head injury whose mental status is normal at
presentation require a thorough history and physical and neurologic examination. If the results are normal, observation for 24 hours by a competent adult in any combination of locations (clinic, office, ED, or home) is recommended for children with minor closed head injury and no LOC. In the absence of LOC, head CT or MRI is not required. After the visit, parents should be alert to any new headache, vomiting, drowsiness, balance problems, confusion, emotional lability, seizures, and drainage from the ears or nose of the child.

Infants have a higher proportion of intracranial injury with head trauma than do older children. Keep in mind that historical signs and symptoms of intracranial pathology are often absent in infants with such injuries. A conservative approach and low head CT threshold are recommended in this age group, particularly if significant scalp hematoma is present. Cranial CT and careful observation may be used in the initial evaluation of children with MTBI and brief LOC (less than 1 minute). If the child's condition deteriorates during observation, perform a thorough neurologic examination and request a cranial CT scan immediately after the patient is stabilized.

**Adult cerebral neuroimaging.** The indications for CT in adults with MTBI have been reviewed. According to evidence-based guidelines of the American College of Emergency Medicine, urgent head CT is indicated in patients with headache, vomiting, drug or alcohol intoxication, short-term memory deficits, post-traumatic seizure, coagulopathy, or physical evidence of trauma above the clavicle, and in those older than 60 years.

### POSTCONCUSSIVE SYNDROME

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<th>Cognitive</th>
<th>Affective</th>
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<td>Impaired attention, concentration, and memory</td>
<td>Anxiety</td>
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Symptoms such as headache, dizziness, anxiety, and impaired cognition and memory may persist after MTBI (Table 2). This constellation of symptoms, known as postconcussive syndrome (PCS), affects more than of 60% of patients 1 month after the injury, and 15% at 1 year. Besides being distressing to the patient, family, and the primary caregiver, PCS is a significant economic burden. Patients miss an average of 4.7 workdays after MTBI as a result of PCS symptoms; about 20% of patients are unemployed at 1 year. These deficits do not typically have correlating radiographic features on CT or MRI scans. The cognitive impairments may have a profound impact on younger, high-achieving persons; deficits in memory and planning have been detected in amateur athletes as young as high-school age after MTBI.

It is difficult to predict which patients will progress to PCS. Improvement at follow-up has been inversely related to the number of symptoms exhibited at hospital discharge. Most symptoms of uncomplicated MTBI display a linear decline during the first post-traumatic year. In one study of 69 patients with MTBI who presented to an ED, predictors of PCS were female sex, non-sports-related mechanism (such as motor vehicle accident or fall), and poor performance on certain neurobehavioral tests (specifically a score of less than 9 on the Digit Span Forward test for women, and a score of less than 11.5 on the Hopkins Verbal Learning test). Whether the cause of these long-term impairments is structural or functional is unclear; there is a correlation with PCS symptoms and lesions in the hippocampus and temporal lobe detectable on
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Regardless of the cause, the patient's complaints must be recognized as a clinical entity for which treatment options exist. Identify community resources for patients at risk for PCS. If necessary, arrange for follow-up with a neurologist or rehabilitation specialist. A multidisciplinary team approach to follow-up is recommended. FOLLOW-UP

The patient with MTBI who has normal results on the neurologic examination and no indications for cerebral or cervical spine neuroimaging criteria may be discharged. Ideally, a patient should be discharged with written instructions provided to a responsible adult who will be able to check the patient during the subsequent 24 hours. Encourage patients to avoid alcohol for the next several days. Indications for immediate medical attention include new severe headache, vomiting, confusion, emotional lability, drowsiness, seizures, difficulty with coordination or balance, and drainage from the nose or ears. Advise patients with MTBI that symptoms of PCS that last weeks to months may develop.

References:


13. Hovda DA, Lee SM, Smith ML, et al. The neurochemical and metabolic cascade following brain...


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