

Management Of Mild Traumatic Brain Injury In The Emergency Department

Abstract

With over 1.7 million people in the United States seeking medical attention for head injury each year, emergency clinicians are challenged daily to screen quickly for the small subset of patients who harbor a potentially lethal intracranial lesion while minimizing excessive cost, unnecessary diagnostic testing, radiation exposure, and admissions. Whether working at a small, rural hospital or a large inner-city public hospital, emergency clinicians play a critical role in the diagnosis and management of mild traumatic brain injury. This review assesses the burgeoning research in the field and reviews current clinical guidelines and decision rules on mild traumatic brain injury, addressing the concept of serial examinations to identify clinically significant intracranial injury, the approach to pediatric and elderly patients, and the management of patients who are on anticoagulants or antiplatelet agents or have bleeding disorders. The evidence on sports-related concussion and postconcussive syndrome is reviewed, and tools for assessments and discharge are included.

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CME Objectives

Upon completing this article, you should be able to:

1. Identify the low- and high-risk criteria for ICI in patients with head trauma.
2. List the indications for imaging in mild TBI.
3. Explain both the short- and long-term sequelae of mild TBI as well as the importance of appropriate follow-up.
4. Recognize the significance of sports concussions.
5. Discuss the assessment of suspected mild TBI in infants and young children.

Prior to beginning this activity, see "Physician CME Information" on the back page.

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Case Presentations

It's 8 PM and you are just getting into the groove of your first in a series of several night shifts. After picking up your fourth head injury chart, you think to yourself, "Good grief, are we having a sale on head injury to-night?" Your patients are:

- *A 16-year-old boy brought in by his parents after head-butting another player during a soccer game. He was confused for several minutes and now has a headache. His coach told his parents that he had a concussion and should go to the ER to be checked out before he can return to play.*
- *A 38-year-old woman who was in a low-speed motor vehicle crash. She states that she "blacked out" for a few seconds but feels fine now.*
- *A 2-month-old brought in by her parents with a bump on her head. They said the babysitter told them the baby rolled off the bed while she was changing her diaper.*
- *A well-known (to you) alcoholic brought in by the police, intoxicated, with an abrasion on his forehead. He has no idea how he hit his head and is asking for something to eat.*

These are 4 cases of what appear to be minor injuries, although you know there is the chance that any of the patients may be harboring a neurosurgical lesion and that all 4 are at risk for sequelae. In your mind, you systematically go through the high-return components of the physical exam of a head-injured patient, the indications for neuroimaging in the ED, and the information needed at discharge to prepare the patients and their families for what might lie ahead. The medical student working with you is very impressed with the complexity of managing these cases, which he thought were so straightforward.

Introduction

Minor head injury, mild traumatic brain injury (TBI, also known as MTBI), and concussion are terms that are often used interchangeably. Regardless of the variation in nomenclature, emergency clinicians can expect to see a number of patients each shift who have sustained some sort of blunt trauma to the head. The clinical approach to these patients varies widely, and, despite the availability of clinical guidelines, most patients will undergo computed tomography (CT) imaging, and the majority will be interpreted as normal. The challenge for emergency clinicians is to quickly screen for the small subset of patients who harbor a potentially lethal intracranial lesion while minimizing excessive costs, admissions, and unnecessary diagnostic testing. Emergency clinicians must accurately document a neurologic baseline for serial examinations and provide discharge instructions that educate patients and families about the potential sequelae of head injury no matter how minor the injury may appear to be.

Further challenges include the rapidly evolving milieu of head injury treatment in the sports arena, with all but 2 states having active or pending laws on return to play for youth sports and full elimination of any same-day return to play after concussive events.¹ Furthermore, with up to 50% of nonactive military personnel seeking care outside of the Veterans Health Administration system,² emergency clinicians can expect to provide care for the increasing numbers of military personnel returning to the United States with postconcussive symptoms. Called the "signature" injury of the Iraq and Afghanistan Wars, military-related mild TBI has affected close to 200,000 soldiers to date,^{3,4} with up to 30% suffering continued postconcussive symptoms.⁵

Critical Appraisal Of The Literature

Appraising the literature is very challenging due to the lack of uniformity—and often impassioned disagreement—regarding the definition of the terms used to describe these injuries. Moreover, studies often lack consistency in the timing of injury assessments, suffer from selection bias, and have conflicting outcome measures. The literature review was performed using PubMed and Ovid MEDLINE® searches for articles on TBI published between 1966 and 2012. Keywords included *traumatic brain injury, concussion, head injury, MTBI, neuroimaging, postconcussive syndrome, sports, and second impact syndrome*. The articles obtained from these searches provided content and background for further manual literature searches. Over 650 articles were reviewed, and 158 of these are included here for the reader's reference.

Additionally, major published guidelines regarding mild TBI were evaluated. These included guidelines published by the Centers for Disease Control and Prevention (CDC), the Brain Trauma Foundation, the American College of Emergency Physicians (ACEP), the American Academy of Neurology, the American Academy of Pediatrics, the Advanced Trauma Life Support® (ATLS®) course, and the Eastern Association for the Surgery of Trauma. Website addresses for several guidelines are provided in **Table 1**.

Definitions

Concussion, a term common in sports medicine, has been used almost interchangeably with *mild TBI* and *minor head injury* to describe a patient who sustains a traumatic force to the head resulting in a transient alteration in cognitive abilities, motor function, or level of consciousness. Fewer than 10% of patients with sports-related concussion sustain a loss of consciousness, and sports concussion is defined by the clinical presence of a rapid-onset, short-lived impairment of neurologic function that resolves spontaneously.⁶ In this article, the term *mild TBI* will be used to describe patients who have suffered

either direct or indirect blunt trauma to the head, have an initial Glasgow Coma Scale (GCS) score of 13-15, and may have somatic, cognitive, or affective symptoms. There is a tremendous research effort underway focusing on both the short-term and long-term implications of mild TBI, and a concise, universal definition is imperative, yet elusive.

Epidemiology

In the United States, 1.7 million people with head trauma seek medical attention each year.⁷ Another 3.8 million people sustain sports and recreation-related head trauma annually, but the vast majority do not seek medical care.⁸⁻¹⁰ TBI most frequently occurs in children and young adults (ages birth to 24 y), with a subsequent peak in incidence occurring in adults > 75 years of age. Males are overrepresented by 3:1 in all subgroups of TBI; however, in some comparable sports, the rate of concussion is higher in females.^{9,11} The 4 leading causes of TBI treated in the emergency department (ED) are:⁹

- Falls
- Motor vehicle-related injury
- Nonintentional strike by / against an object, including sports and recreational injury
- Assaults

Morbidity And Mortality

About 80% of patients with TBI seeking ED care are treated and released.¹² Of those with mild TBI, < 10% will have intracranial injury (ICI) identified on CT and < 1% of patients will require neurosurgical intervention.¹³ Older age (> 65 y of age) comprises the

group of patients with the highest rates of hospitalizations and deaths; age is a much stronger predictor of poor outcome than the specific cause of the injury.^{9,13}

- Motor vehicle-related injuries are the leading cause of TBI-related hospitalizations and deaths, with mortality highest in people ages 20 to 24.
- Falls are the second leading cause of TBI-related hospitalization with mortality highest in people > 65 years old.
- Assaults are the third leading cause of TBI-related deaths, with mortality highest in people ages 20 to 35.

As many as 30% of patients with a discharge diagnosis of mild TBI will have symptoms at 3 months postinjury (known as postconcussive syndrome), and up to 15% will continue to be symptomatic at 1 year postinjury.^{9,14} Direct medical costs and indirect costs (such as lost productivity) of TBI exceed \$60 billion annually in the United States.¹⁵

Pathophysiology

Mild TBI is a complex pathophysiologic process caused by direct or indirect traumatic biomechanical forces to the head. The symptoms largely reflect a functional disturbance rather than a structural injury that can be identified on standard neuroimaging. The precise mechanisms responsible for the clinical features of mild TBI remain unclear, but using functional magnetic resonance imaging (MRI), clinical symptoms can be mapped to specific areas of the brain with axonal injury.¹⁶

Current research suggests that blunt forces causing microscopic neuronal shearing lead to a transient hypermetabolic state that, when paired with alterations in cerebral blood flow and autoregulation, result in the clinical symptoms of mild TBI.¹⁷ Several proteins have been identified that are released from injured central nervous system (CNS) structures and have a potential role as serum biomarkers in patients with mild TBI.¹⁸ Secondary injury occurs from a multitude of complex neurobiological cascades that are thought to be worsened by insults such as hypoxia, hypotension, hyperglycemia, hypoglycemia, and hyperthermia.^{17,19} Typically, these microscopic changes are transient, but repetitive injuries have been shown to have lasting pathobiological effects.¹⁷

About 6% to 8% of patients with a mild TBI will have specific injuries detectable on CT.²⁰⁻²² These injuries include subarachnoid hemorrhage, subdural or epidural hematomas, cerebral contusions, intraparenchymal hemorrhage, and evidence of axonal injury such as edema and petechial hemorrhage.

- Traumatic subarachnoid hemorrhage is caused by tearing of the pial vessels with subsequent tracking of blood in the subarachnoid space into the sulci and cisterns.

Table 1. Major Guidelines On Mild Traumatic Brain Injury

Organization	Website Address
Centers for Disease Control and Prevention	http://www.cdc.gov/concussion/index.html
American College of Emergency Physicians	http://www.acep.org/clinicalpolicies/
Brain Trauma Foundation	http://tbguidelines.org/gliHome.aspx
American Academy of Pediatrics	http://pediatrics.aappublications.org/site/aappolicy/index.xhtml
Zurich Consensus on Concussion in Sports (SCAT2)	http://bjsm.bmj.com/content/43/Suppl_1/i76.full
Defense and Veterans Brain Injury Center (MACE2)	http://www.dvbic.org
National Conference of State Legislatures (return-to-play laws)	http://www.ncsl.org/issues-research/health/traumatic-brain-injury-legislation.aspx

- Subdural hematomas most often occur as a result of shear through the bridging veins, with blood tracking along the brain under the dura.
- Epidural hematomas typically occur when a skull fracture disrupts an artery and blood escaping from the artery pushes the tightly adhered dura away from the calvarium.
- Contusions are areas of punctuate hemorrhages and cerebral edema, and they are typically due to acceleration-deceleration injuries against the bony internal surfaces of the cranium.
- Intracerebral bleeds are caused by a tear of a parenchymal vessel or the coalescence of cerebral contusions.
- Axonal injury occurs due to a rapid rotational or deceleration force that causes stretching and tearing of neurons, leading to petechial hemorrhage and/or edema at the gray-white matter junction, at the corpus callosum, and/or in the brainstem.
- Skull fractures may be linear or comminuted, with varying degrees of depression. They have implications for adjacent anatomical structures in the following ways:
 - Fractures that cross the meningeal artery are often associated with epidural hematomas, while those that cross a dural sinus can cause subdural hematoma and thrombosis.²³
 - Fractures through the base of the skull and carotid canal can cause carotid artery dissection.²⁴
 - Basilar skull fractures are frequently associated with dural tears and cerebrospinal fluid (CSF) leaks.
 - Skull base fractures are associated with damage to the cranial nerves.

Prehospital Care

As in any prehospital encounter, the scene must first be secured to minimize potential risks to bystanders and emergency personnel. Management of an alert patient with head injury should be systematic to ensure that occult injuries are identified.²⁵ Due to the associated risk of cervical spine injury in patients with TBI, management must coincide with the assessment of the cervical spine.²⁶ Although oxygenation, ventilation, and hemodynamic adjuncts are rarely indicated in the patient with isolated mild TBI, episodes of hypoxia, hypercarbia, and hypotension have been shown to worsen outcomes in TBI and must be quickly ruled out.²⁷⁻³⁰

A brief, focused neurological examination should be performed, with specific attention given to the GCS score,³¹ pupillary examination, and overall motor function. Serial GCS score monitoring is a dynamic tool that provides early clinical warning of neurological deterioration.^{32,33} **(See Table 2.)** Patients with a sports-related injury can be assessed using the Sports Concussion Assessment Tool-2 (SCAT2), which documents symptoms and coordination while incorporating components of the Balance Error Scoring System (BESS), the Standardized Assessment of Concussion (SAC), and the Maddocks Score for memory.⁶ **(See Table 3.)** In the military setting, the Military Acute Concussion Evaluation-2 (MACE2) tool is used to document symptoms and assess for memory and concentration deficits.³ Both the SCAT2 and MACE2 are available online. **(See Table 1, page 3.)**

Transport

Emergency medical services (EMS) providers and online medical command clinicians should

Table 2. Glasgow Coma Scale Scoring

Component	Adults	Score	Children	Score
Best Eye Opening	Spontaneous	4	Spontaneous	4
	To verbal stimuli	3	To verbal stimuli	3
	To painful stimuli	2	To painful stimuli	2
	No eye opening	1	No eye opening	1
Best Verbal Response	Oriented	5	Appropriate coo and cry	5
	Confused	4	Irritable cry	4
	Inappropriate words	3	Inconsolable crying	3
	Incomprehensible	2	Grunts	2
	No verbal response	1	No verbal response	1
Best Motor Response	Obeys commands	6	Normal, spontaneous movement	6
	Localizes pain	5	Withdraws to touch	5
	Withdraws to pain	4	Withdraws to pain	4
	Flexion to pain	3	Flexion to pain	3
	Extension to pain	2	Extension to pain	2
	No motor response	1	No motor response	1
	Total	_____	Total	_____

be aware of the indications for transport to a facility with neurosurgical capacity. The Brain Trauma Foundation recommends that all regions in the United States have an organized trauma care system with established protocols to direct transport decisions for patients with TBI.²⁷ Most EMS protocols direct a patient with TBI and a GCS score < 14 to be transported to a Level I or II trauma center. A recent study of 52,000 patients using the National Trauma Database found that those who had a GCS score ≤ 13 in the prehospital setting were 17 times more likely to die than those who had a higher GCS score.³²

Table 3. Components Of The Sports Concussion Assessment Tool-2 (SCAT2)⁶

Symptoms		
Concussion is suspected if any 1 or more are present		
<ul style="list-style-type: none"> • Loss of consciousness • Seizure • Amnesia • Headache • "Pressure in head" • Neck pain • Nausea or vomiting • Dizziness • Blurred vision • Balance problem • Sensitivity to light • Sensitivity to noise 	<ul style="list-style-type: none"> • Feeling slowed down • "In a fog" • "Don't feel right" • Difficulty concentrating • Difficulty remembering • Fatigue or low energy • Confusion • Drowsiness • More emotional • Irritability • Sadness • Nervous or anxious 	
Maddocks Memory Function ³⁴		
<ul style="list-style-type: none"> • "What venue are we at today?" • "Which half is it now?" • "Who scored last in the game?" • "What team did you play last week/game?" • "Did your team win the last game?" 		
Balance Error Scoring System (BESS) ³⁵		
Stand 20 seconds each in 3 different positions:		
Stand with feet together	Stand on nondominant foot and lift up other leg	Stand heel-to-toe with nondominant foot in back
<ol style="list-style-type: none"> 1. For each position, try to maintain stability for 20 sec with hands on hips and eyes closed. 2. If you stumble out of this position, open your eyes and return to the start position and continue balancing. 3. More than 5 errors (lifting hands off hips; opening eyes; lifting forefoot or heel; stepping, stumbling, or falling; or remaining out of the start position for more than 5 sec) may suggest a concussion. 		
Standardized Assessment of Concussion (SAC) ³⁶		
<ul style="list-style-type: none"> • Oriented to month, date, year, day of the week, and time within 1 h. • Repeat back list of 5 words 3 times. • Recite the months of year in reverse. • Repeat strings of numbers in reverse. • Coordination: finger-to-nose, each arm, 5 times. 		

For the full SCAT2 assessment tool, go to http://bjsm.bmj.com/content/43/Suppl_1/i85.full.pdf

Emergency Department Management

Initial Evaluation

Most patients with mild TBI have a straightforward clinical presentation, but some have an unclear history and little or no physical evidence of trauma. Because mild TBI is an almost entirely symptom-based diagnosis, it is imperative that the emergency clinician obtain an accurate history of presenting illness and the mechanism of injury. Clinicians should avoid early diagnostic closure in patients with any degree of altered mental status or possible head trauma; the wide differential necessitates a thorough history and physical examination for accurate and timely diagnosis. Polytrauma is common in patients with TBI, and a systematic approach ensures that occult injuries are identified.²⁵

History

A focused history should include a detailed description of the traumatic event solicited from the patient, family members, and EMS. Witnesses or individuals who know the patient may be helpful in ascertaining the details of the event and environment as well as the patient's normal level of functioning. Key historical data include:

1. The mechanism of injury may provide information regarding associated injuries. Mechanisms that are associated with an increased risk of ICI in adults include pedestrian being struck by a motor vehicle, an occupant ejected from a motor vehicle, or a fall from an elevation of > 3 feet (0.9 m) or 5 stairs.^{21,37} In children, important mechanisms include motor vehicle crash with ejection, death of a passenger, or rollover; being struck by a vehicle; a fall from > 5 feet (1.5 m) (or if < 2 y old, > 3 ft [0.9 m]); or a head struck by high-impact object.³⁸ An inconsistent history suggests the possibility of child abuse.³⁹
2. Symptoms shown to have a significantly high positive likelihood ratio for ICI include seizures, deterioration in mental status, GCS score < 14, repeated vomiting, and focal neurological deficit or history of neurosurgery.^{13,40,41}
3. The presence of loss of consciousness has been shown to increase the risk of ICI, but its absence is only useful as a negative predictor if there are no associated symptoms or high-risk variables.^{22,42} In children, studies have shown that more than half of those with ICI on CT did not have a loss of consciousness.^{38,43}
4. Drug or alcohol use, with either chronic or current intoxication, is associated with ICI in patients with TBI, but it does not have a clear role as an independent predictor of outcome.^{44,45}
5. Anticoagulant or antiplatelet use, hemophilia, or platelet disorders are associated with increased risk of immediate and delayed ICI in patients with TBI.⁴⁶⁻⁴⁸

6. Any CNS surgery, past head trauma, and immediate posttraumatic seizures should be noted, as they are associated with increased risk of ICI in patients with TBI.¹³
7. Patients > 60 years of age have an increased risk of ICI due to mild TBI.¹³ Age has been shown to be an independent predictor of mortality in isolated mild and moderate TBI.⁴⁹
8. In sports, several factors are predictive of poorer outcomes after mild TBI. These include the number of past concussions, the severity and duration of symptoms, and the time elapsed since the last concussion.⁶

Physical Examination

Patients who are alert and clinically stable after mild TBI should undergo a focused physical examination with special attention paid to the neurological evaluation. The general physical examination should include assessment for the following:

- Basilar skull fracture: hemotympanum, periorbital ecchymosis, postauricular ecchymosis, CSF rhinorrhea or otorrhea
- Spinal injury: bony tenderness, paresthesias, incontinence, extremity weakness, or priapism
- Carotid or vertebral artery dissection: bruits, headache, or extremity weakness

Neurological Examination

A focused neurological examination should be performed, with attention to GCS score, cognitive functioning, pupillary examination, and motor and balance function. Serial neurological monitoring has been shown to be useful as a dynamic tool to provide early clinical warning of deterioration.^{32,33}

Glasgow Coma Scale Score

Scoring for each component of the GCS score should be documented separately in order to provide complete information for subsequent measures (eg, GCS score 10 = E3 V4 M3). (See Table 2, page 4.) Deficits in the motor component have the strongest correlation with poor outcome in patients with TBI,^{50,51} and a recent validation of a motor-only score was shown to perform as well as the GCS score.^{52,53}

Cognitive Examination

A recent prospective study of over 1000 patients with mild TBI revealed that ICI on CT does not predict cognitive deficits.⁵⁴ Furthermore, cognitive tests have not been shown to predict abnormalities on head CT.⁵⁵ Nonetheless, several prospective studies have revealed that memory tests can be used to predict postconcussive syndrome.^{12,56,57} A patient with mild TBI can be quickly assessed for cognitive deficits by testing short-term memory (3-item recall, 5-number recall) and concentration (serial sevens, backwards recitation of the months of the year, or

“world” spelled backwards). The SCAT2 includes validated tests of orientation, memory, and concentration.⁶ Basic cognitive testing in the ED acts to expand the focus of care from a search for the rare abnormal head CT to a more patient-focused approach, addressing the neurocognitive symptoms that patients are much more likely to experience. Emergency clinicians must be aware that no test, in isolation, can rule in or out cognitive deficits secondary to mild TBI,⁵⁸ and, to date, the most sensitive and specific approach to testing cognitive function includes a battery of tests that are best administered by a trained neuropsychologist.⁶

Pupillary Reflexes

Pupillary reflexes indicate both underlying pathology and severity of injury and should be monitored serially.⁵¹ Pupillary abnormalities in alert patients are most likely due to etiologies other than TBI. In 2012, a large retrospective study by Hoffmann et al of over 24,000 patients revealed that abnormal pupillary findings in patients with TBI are limited to patients with GCS < 13.⁵¹

- The normal diameter of the pupil is between 2 mm and 5 mm, and > 6 mm is dilated.
- Anisocoria > 1 mm is considered clinically significant.
- Nonreactive pupils have < 1 mm response to direct light, a finding very predictive of poor prognosis in TBI.⁵¹

Motor And Balance Testing

Motor testing should include the evaluation of cranial nerves, gross extremity strength, coordination, and balance. When performing the cranial nerve examination, attention should be paid to cranial nerves IV and VI, as palsies may not be evident until the patient is taken through a careful extraocular examination.⁵⁹ The most common cranial nerves injured after mild TBI are I, VII, and VIII.⁵⁹ Coordination can be assessed using finger-to-nose testing and rapid, alternating hand movements. Gait (straight-line and tandem) is often used in the ED as a marker of balance, although specific balance testing has been shown to detect deficits that may not be picked up by gait assessment alone.^{35,60} Subtle balance and coordination deficits can persist long after other symptoms of mild TBI have resolved. The SCAT2 includes well-validated balance and coordination testing components.⁶ (See Table 3, page 5.)

Diagnostic Testing

Laboratory And Bedside Studies

In general, routine laboratory and bedside studies have little value in the evaluation of uncomplicated ED patients with mild TBI. The following groups of

patients are more likely to benefit from studies:

- All patients with undifferentiated altered mental status should undergo a bedside glucose test and a blood count, an electrolyte panel, and be considered for a blood alcohol level and toxicology screen.
- Patients with a history or clinical evidence of anemia or thrombocytopenia should have a complete blood count with platelets.
- Elderly patients and those with significant comorbid conditions or weakness should have an electrolyte panel, blood count, urinalysis, and electrocardiogram (ECG) performed.
- Patients with known or suspected coagulation disorders, liver disease, or those taking anticoagulants will benefit from coagulation studies.⁶¹
- Patients who sustain a basilar skull fracture can have a dural tear, leading to a CSF leak. Because it may be difficult to distinguish normal nasal secretions from suspected CSF rhinorrhea, several bedside and laboratory tests can be performed.
 - The tau-transferrin test is considered the gold standard for identifying CSF because it is a protein only found in CSF, perilymph, and the vitreous humor.^{62,63}
 - The presence of glucose in secretions has been used to differentiate CSF from nasal secretions because nasal secretions should be free of glucose. A glucose level of > 30 mg/dL is generally considered positive, but false positives can occur due to contamination with blood.⁶³
 - The “halo sign” is seen when bloody fluid on tissue paper reveals a central ring surrounded by a tinted halo of CSF, but false positives can occur.^{64,65}

Radiography

Plain Skull Radiography

As early as 1980, studies demonstrated that plain skull films were neither sensitive nor specific in the identification of patients with ICI.⁶⁶ Some clinicians routinely obtain skull films in suspected child abuse cases on the premise that the pattern of fractures may suggest abuse. This practice may have merit when screening asymptomatic patients with no suspicion of head injury, but plain films do not obviate the need for CT in abuse-related head trauma. A 2010 review by Leventhal et al of a United States database of more than 18,000 children under age 3 demonstrated that in abuse-related TBI, ICI is more common than isolated skull fracture, and in children under the age of 1 year, the finding of ICI or fracture is much more likely to be caused by abuse than in older-aged children.⁶⁷

Computed Tomography

Noncontrast CT is both highly sensitive and specific for the detection of fractures, contusions, epidural and subdural bleeds, and subarachnoid hemorrhages, and it is currently the diagnostic imaging technique of choice in patients with TBI.¹³ A CT interpreted as normal in a neurologically intact person with a normal mental status allows for safe discharge with appropriate instructions and avoids prolonged ED observation or hospital admission.⁶⁸ Disadvantages of CT include its poor sensitivity in basilar skull fractures, areas of axonal injury, and parenchymal lesions located at the base of the brain,⁶⁹⁻⁷¹ as well as the radiation exposure and its potentially carcinogenic risk. Radiation exposure from head CT is relatively small and is inversely related to age; a 40-year-old has a cancer risk of 1:8-10,000, but a 20-year-old has a risk of 1:4-5000.⁷² Disadvantages of CT include cost as well as the added ED throughput time necessary to obtain and result the CT.^{20,72}

Which patients with mild traumatic brain injury benefit from computed tomographic imaging?

Most clinicians agree that CT is high-yield in patients with clear evidence of basilar, depressed, or open skull fracture; penetrating injuries; GCS score < 13; and/or focal neurological deficits. Nonetheless, only about 6% to 8% of patients with mild TBI will have ICI detected on CT, and less than 1% will require neurosurgical intervention.^{13,20,22} This low yield has led to a myriad of studies over the past 2 decades in search of the “holy grail” of clinical criteria to guide in the use of CT in patients with mild TBI.

To date, over 20 clinical decision rules for guiding CT use in the ED have been published,¹³ but the New Orleans Criteria (NOC) and the Canadian CT Head Rule (CCHR) stand out due to their high sensitivity (99%-100%) in repeated external validations.^{20,21,73-75} Both clinical decision rules maintained their original high sensitivity in TBI patients with and without loss of consciousness and in patients with a GCS score of 13 to 15.^{20,21,73-75} (See Table 4, page 8.) In 2008, the CDC and ACEP endorsed the clinical variables from both guidelines in a nationwide campaign to improve the care of patients with mild TBI.^{7,68,76}

Is there such a thing as “clinically unimportant” or “inconsequential” intracranial injury?

The ubiquitous use of CT scanning, along with the improved quality of late-generation CT scanners, has led to the detection of increasingly minute intracranial lesions that are thought to rarely, if ever, require directed interventions. The CDC/ACEP guidelines recommend identifying the mild TBI patients with any intracranial lesion on CT, and they do not limit their focus to only those patients requiring neurosurgical intervention.⁶⁸ This approach can be expected to reduce CT use by no more than 20%,^{73,76,79} but in an attempt to further

reduce the use of CT, some researchers have labeled small, isolated lesions as “clinically unimportant” or “inconsequential” to clinical care. These lesions include: (1) a solitary contusion < 5 mm in diameter, (2) localized subarachnoid blood < 1 mm thick, (3) a smear subdural hematoma < 4 mm thick, (4) isolated pneumocephaly, and (5) a closed depressed skull fracture not through the inner table.^{80,81} Several guidelines are directed toward identifying only patients with clinically significant lesions and disregarding the insignificant lesions, which leads to the question: Is it safe to disregard these “inconsequential” intracranial lesions?^{21,78}

In 2002, using a prospectively collected database of 8000 patients with ICI, Aztema et al studied 155 patients with “clinically inconsequential” intracranial lesions and found that 10% required a neurosurgical intervention, although all could be identified by an abnormal GCS score or altered mental status.⁸² Another review of > 4000 patients with mild TBI with a GCS of 15 found that 80% of those who required a neurosurgical intervention had a decline in GCS within 6 hours or had other symptoms such as altered mental status, vomiting, or severe headache.⁸³ Based on the best evidence to date, we can estimate that about 1 in 1000 patients with mild TBI will have an “inconsequential” lesion

that results in a poor outcome, but it appears that those patients can be identified in the ED during a 6-hour observation period to monitor for a decline in GCS score, altered mental status, repeated vomiting, or severe headache.

Interestingly, the presence of ICI on CT in patients with mild TBI has not been shown to affect the risk of postconcussive symptoms,^{54,84,85} although studies using more-advanced MRI technology have shown a correlation between postconcussive symptoms and white matter lesions not detected on CT.^{86,87} About 25% to 30% of patients with mild TBI can be expected to have continued neurocognitive symptoms beyond the expected 7- to 10-day recovery period.^{8,14}

What about patients with an abnormal Glasgow Coma Scale score that returns to normal in the emergency department?

After TBI, there is an inverse relationship between the GCS score and the incidence of positive findings on CT. In fact, the rate of ICI and need for neurosurgical intervention doubles when the GCS score drops from 15 to 14.^{88,89} Many authors recommend that patients with a GCS score of 13 be classified as moderate instead of mild, due to the higher incidence of ICI and poor outcomes in those patients.^{32,90-92} Few emergency clinicians would hesitate to obtain a CT in the setting of a low GCS score, but what about patients who start off with a GCS score of 13 or 14 and then normalize to 15? There are no studies that specifically address this question, although several studies include this subset of patients in their overall analysis, indirectly demonstrating that no patient had a poor outcome if the GCS score normalized within 2 hours of injury and they had no other associated symptoms.^{21,78,88} A review of > 4000 patients with a mild TBI found that 80% of patients in need of neurosurgical intervention could be identified by worsening or no improvement of symptoms during a 6-hour observation period.⁸³ Based on the best evidence to date, we can expect that an otherwise asymptomatic patient whose GCS score rapidly normalizes will not have a clinically important lesion on CT.

What about patients with no loss of consciousness?

Much of the mild TBI research has been focused on the group of patients who have a history of loss of consciousness. This may have originated from the sports medicine or pediatric literature that equates loss of consciousness with more severe injury.¹⁰ The initial study population in the NOC study included only patients with loss of consciousness, while the CCHR included patients with and without loss of consciousness, and both studies have been validated in patients with and without loss of consciousness.^{21,73,75,77,78} In 2007, Smits et al prospectively studied almost 2500 patients and showed that the need for neurosurgical intervention

Table 4. Clinical Decision Rules In Mild Traumatic Brain Injury In Adults

	New Orleans Criteria ²⁰	Canadian CT Head Rule ²¹
CT if any criteria present	<ul style="list-style-type: none"> • Headache • Vomiting (any) • Age > 60 y • Drug or alcohol intoxication • Seizure • Trauma visible above clavicles • Short-term memory deficits 	<ul style="list-style-type: none"> • Dangerous mechanism of injury* • Vomiting ≥ 2 times • Patient > 65 y • GCS score < 15, 2 h postinjury • Any sign of basal skull fracture • Possible open or depressed skull fracture • Amnesia for events 30 min before injury
Need for neurosurgical intervention	Sensitivity: 99%-100% 20,73,75,77,78 Specificity: 10%-20%	Sensitivity: 99%-100% 21,73,75,77,78 Specificity: 36%-76%
Clinically significant ICI	Sensitivity: 95%-100% ²⁰ .73,75,77,78 Specificity: 10%-33%	Sensitivity: 80%-100% ^{21,73,75,77,78} Specificity: 35%-50%

*Dangerous mechanisms of injury include ejection from a motor vehicle, a pedestrian struck by a motor vehicle, or a fall from a height of > 3 ft (0.9 m) or 5 steps.

Abbreviations: CT, computed tomography; GCS, Glasgow Coma Scale; ICI, intracranial injury.

remains the same regardless of the presence of loss of consciousness and that the criteria used in the NOC and CCHR are largely unaffected by loss of consciousness, supporting the use of both guidelines in patients without loss of consciousness.⁴²

How do guidelines differ for children and infants, compared to adults with mild traumatic brain injury?

Mild TBI in children is common, but decisions for neuroimaging are complicated by the potential need for sedation and the inherent risk of radiation exposure. Depending on their age, children can be up to 10 times more radiosensitive than adults, and the risk of subsequent cancer death can be as high as 1:1000.⁷² Risk stratification in children with mild TBI can be difficult, and there are few studies on children < 2 years of age. The overall rate of ICI and ultimate need for neurosurgical intervention in children with mild TBI is about the same as adults,¹³ although pediatric guidelines have historically included observation as an approach in the management of children with mild TBI.⁹³

In children < 2 years of age, up to 20% of TBI is caused by child abuse,⁹⁴ but as children advance in age, the mechanisms of injury parallel those of adults with TBI.⁷ The highest incidence of ICI in apparently mild TBI is found in infants < 12 months of age.^{13,43,95} More than 10 clinical decision guidelines for the management of mild TBI in children have been published over the past 15 years.^{13,22,38,96-98} The 3 largest studies are the Pediatric Emergency Care Applied Research Network (PECARN), developed in the United States; the Children's Head Injury Algorithm for the Prediction of Important Clinical Events (CHALICE), developed in the United Kingdom; and the Canadian Assessment of Tomography for Childhood Head Injury (CATCH).^{38,96,99} To date, only PECARN (sample size > 40,000, with almost 15,000 undergoing CT) has been prospectively validated at an external site.^{38,98,100} It was found to be highly sensitive in a prospective validation study at an Italian center with over 350 patients.¹⁰⁰ In PECARN, a decision tree directs immediate CT in the presence of any of the high-risk variables (4% risk of ICI) and offers the options of observation or CT in the presence of the lower-risk variables (1% risk of ICI). (See Table 5.) The decision to observe is based on the age of the child (with younger infants at higher risk for ICI), number of symptoms (with more symptoms increasing the risk of ICI), and parent and physician comfort.³⁸ In the Italian validation study, the researchers increased the observation period to 12 hours for infants < 6 months of age.¹⁰⁰

What is the best diagnostic approach in infants with mild traumatic brain injury?

Infants are challenging to evaluate because they often have few or no clinical findings, even in the setting of ICI. Loss of consciousness is not present

in almost 50% of infants with ICI, and many infants have little more than a scalp hematoma on physical examination.^{38,43} PECARN prospectively studied over 10,000 children < 2 years of age, and the criteria were highly sensitive in identifying children that could be evaluated without CT.³⁸ In general, the younger the child, the lower the threshold should be for obtaining imaging studies. The greater the severity and number of signs and symptoms, the stronger the consideration should be for obtaining imaging studies.

Do elderly patients with mild traumatic brain injury have an increased risk of intracranial injury?

Age > 60 years is an indication for CT in the CDC / ACEP guidelines,⁷⁶ and its moderate association with ICI is confirmed by a recent large meta-analysis.¹³ Several studies of patients > 65 years of age revealed a much higher association with ICI and showed that the risk of ICI increases directly with advancing age.^{22,101-104} People ≥ 75 years of age have the highest rates of TBI-related hospitalizations and death,⁷⁶ a trend thought to be due to cerebral atrophy and fragile, less-elastic bridging veins that are prone to disruption in the aged, even in the setting of low-energy trauma. Elderly patients with ICI often have fewer clinical clues, such as loss of consciousness or a serious mechanism of injury, and several studies have shown that the majority of elderly patients with mild TBI who require neurosurgical intervention do not have a history of

Table 5. PECARN Clinical Decision Rule For Children With Mild Traumatic Brain Injury³⁸

<p>CT if any high-risk variable present:</p> <ul style="list-style-type: none"> • GCS score < 15 • Altered mental status: agitation, somnolence, repetitive questioning, verbally slow to respond • Palpable skull fracture or suspected basilar skull fracture 	<p>CT or observe if any present:</p> <ul style="list-style-type: none"> • Loss of consciousness • Severe headache • Vomiting • Nonfrontal scalp hematoma age < 2 y • Not acting normal (per parent) age < 2 y • Severe mechanism of injury: MVC with ejection, death of passenger, rollover, being struck by vehicle, fall > 5 ft (1.5 m) (or > 3 ft [0.9 m] if age < 2 y), head struck by high-impact object
<p>Neurosurgical intervention³⁸:</p> <p>Sensitivity: 100%</p> <p>Specificity: 59%</p>	<p>Intracranial injury³⁸:</p> <p>Sensitivity: 97%</p> <p>Specificity: 58%</p>

Note: In PECARN, n = 42,000.

Abbreviations: CT, computed tomography; GCS, Glasgow Coma Scale; MVC, motor vehicle crash; PECARN, Pediatric Emergency Care Applied Research Network.

loss of consciousness.^{103,105} Emergency clinicians must maintain a low threshold for CT use in elderly patients with mild TBI.

What about patients with bleeding disorders or those taking anticoagulants or antiplatelet agents?

Patients with mild TBI who have a bleeding disorder or who take anticoagulants or antiplatelet agents present a challenge to the emergency clinician. Practice guidelines typically exclude these patients, although research is growing rapidly in this area. Best practices, to date, advocate for immediate CT in this group of patients, without regard to symptoms or loss of consciousness.⁴⁶ A more in-depth discussion on managing anticoagulated patients in the ED is available in the January 2011 issue of *Emergency Medicine Practice*, "An Evidence-Based Approach To Managing The Anticoagulated Patient In The Emergency Department."

Anticoagulants: Warfarin (Coumadin®, Jantoven®) is the most common and the most studied of the anticoagulants in patients with mild TBI. There is significant overlap in the risk of ICI due to advanced age and due to the presence of anticoagulant use; in addition, a significant amount of these patients that have ICI do not have a history of loss of consciousness, altered mental status, or visible evidence of trauma above the clavicles.^{48,103,106,107} There is strong evidence to support the use of immediate CT on all patients with mild TBI taking anticoagulants.^{106,108-110} The risk of ICI is increased in the setting of an elevated international normalized ratio (INR), with the best evidence showing that an INR of 2.4 or more increases the risk of immediate ICI.^{47,61,107} Unfortunately, no specific INR can be used to rule out the risk of ICI with patients at a subtherapeutic INR at risk for ICI, likely due to the overlap of advanced age in this group.¹¹¹ Dabigatran (Pradaxa®) is a new oral anticoagulant that is gaining popularity because it does not need therapeutic monitoring; unfortunately, there are no studies that address its impact on patients with mild TBI.¹¹²

In 2002, concern over delayed ICI after a normal CT in patients on anticoagulants led to the European Federation of Neurological Societies recommending a 24-hour observation period followed by a repeat head CT for all anticoagulated patients with minor head injury.¹¹³ A recent prospective study of 97 patients on warfarin found that although 6% of patients had evidence of a delayed ICI on a repeat CT at 24 hours, only 3% required hospital admission and less than 1% required neurosurgical intervention.⁴⁷ This study also found that an INR of ≥ 3 was associated with delayed ICI.⁴⁷ Several larger studies have shown even lower rates of delayed hemorrhage.^{48,114,115} The largest prospective study to date, with > 700 patients on warfarin, demonstrated that < 1% had delayed ICI after an initially normal CT, with only 0.2% having

a lesion that required neurosurgical intervention.⁴⁸ A retrospective study of > 500 patients subjected to a 6-hour period of observation after an initially normal CT revealed that no patient with a clinically important lesion would have been missed had a repeat CT been withheld.¹¹⁴ The best evidence, to date, demonstrates that anticoagulated patients with mild TBI and a normal initial CT have < 1% risk of delayed hemorrhage and about 2 in 1000 will have a lesion that requires neurosurgical attention.^{47,48} A conservative, risk-stratification approach to anticoagulated patients would include admission for 24-hour observation in only those patients with continued symptoms or an INR ≥ 3 ,^{110,47} while other patients who remain asymptomatic after a 6-hour observation period may be discharged, with close follow-up, in the company of a reliable adult who is educated about the risk of delayed hemorrhage and encouraged to return immediately for a repeat CT for any new or worsening symptoms.^{48,116}

Antiplatelets: Several studies have found aspirin and clopidogrel (Plavix®) to be associated with increased risk of intracranial bleed.^{46,48,117,118} In 2010, Fabbri et al reviewed a database of over 14,000 patients with mild TBI and found a very strong association between aspirin use and increased incidence of ICI.¹¹⁸ In a 2012 multicenter prospective study of almost 300 patients with blunt head trauma taking clopidogrel, Nishijima et al reported that 12% of patients had ICI on initial CT, and no patients had delayed ICI on repeat CT.⁴⁸ Patients on antiplatelet agents should undergo CT after mild TBI.

Bleeding Disorders: Adults and children with bleeding disorders and mild TBI present a challenge in the ED. CT use is very commonly implemented in these patients; in fact, in the PECARN study, children with hemophilia were 20 to 40 times more likely to undergo CT.^{38,119} About 50% of hemophiliacs with mild TBI who harbor an ICI will initially be asymptomatic, and no validated clinical decision rules exist to guide CT use in these patients.^{119,120} Patients with bleeding disorders should undergo CT after mild TBI.

Reversal Agents: Emergency clinicians should have a low threshold for factor replacement or reversal agents in patients with a bleeding disorder or patients who are on antiplatelet agents or anticoagulants.^{121,122} Patients with hemophilia benefit from empiric factor replacement (Factor VIII, cryoprecipitate, or fresh frozen plasma) before CT in the presence of symptoms of TBI or severe hemophilia.^{123,124} Patients on warfarin with an ICI on CT should undergo rapid reversal using fresh frozen plasma or prothrombin complex concentrates, but the role for empiric reversal before

CT is unclear.^{121,125} Vitamin K should be initiated in the ED, but emergency clinicians must be aware that full reversal using vitamin K may take up to 24 hours. To date, platelet transfusions in patients on aspirin or clopidogrel have not been shown to impact outcomes after TBI.¹²⁶ Some clinicians have considered the use of desmopressin in patients with ICI who are on antiplatelet agents, but there are no studies that address this issue. Finally, recent attention is being given to the new oral anticoagulant, dabigatran, because the only readily available reversal agent is emergent dialysis.¹¹²

How should an intoxicated patient with mild traumatic brain injury be evaluated?

Alcohol and TBI are unfortunate bedfellows, with over 20% of mild TBI associated with alcohol use.¹²⁷ Patients with alcohol intoxication can be challenging to assess, and most emergency clinicians can recall feeling alarmed when discovering an unexpected positive scan on the patient “sleeping it off” in the corner of the ED. Intoxicated patients have been shown to have an increased risk of ICI, but it is unclear whether intoxication alone is an independent predictor of ICI.^{45,127} Bracken studied over 3000 intoxicated patients and found only 3 otherwise asymptomatic patients with ICI, and none required a neurosurgical intervention.¹²⁷ Furthermore, recent studies have shown that intoxication has little effect on the GCS score unless the blood alcohol level is > 200 mg/dL.^{128,129} The CDC/ACEP guidelines include intoxication as an indication for CT, although the best evidence to date shows that it is probably safe to closely observe an otherwise asymptomatic patient who rapidly sobers.^{21,22,73,76,78,127}

Magnetic Resonance Imaging

Noncontrast CT remains the gold standard in suspected mild TBI, although MRI has an established role in the elucidation of brain stem lesions, diffuse axonal injury, and nonhemorrhagic lesions.^{70,71} The lesions detected by MRI do not typically influence early neurosurgical intervention and, therefore, MRI is more commonly used as a secondary test for the investigation of persistent symptoms.¹³

Postconcussive Syndrome

Postconcussive syndrome refers to a symptom complex that continues beyond the expected 7- to 10-day recovery period, and it is experienced by 25% to 30% of patients after mild TBI.^{8,14} The syndrome encompasses somatic, cognitive, and affective complaints, and patients commonly report headache, dizziness, difficulty concentrating, and depression. (See Table 6.) There appears to be both psychological and structural components to postconcussive syndrome, as patients with a history of migraines, depression, or

anxiety are more likely to experience postconcussive syndrome.¹⁰ Diffusion-weighted MRI has demonstrated specific structural areas of white matter injury that correlate with a patient’s postconcussive syndrome symptoms,⁸⁶ but the postconcussive syndrome symptom complex is not necessarily specific to TBI; it is also associated with trauma-related anxiety and posttraumatic stress disorder where there has been no TBI.^{4,130} Postconcussive syndrome is more common in patients with negative perceptions about their traumatic episode and in those with pre-existing stress, anxiety, and depression.¹³¹ In the ED, patients with more severe symptoms such as prolonged amnesia, dizziness, headache, anxiety, noise sensitivity, or trouble with verbal recall have been shown to be at a higher risk of developing postconcussive syndrome.^{12,132,133}

Sports-Related Concussion

There are an estimated 3.8 million concussions due to sports and recreational activities each year in the United States.¹¹ Controversy regarding the sideline management of sports-related concussions has led to the development of multiple competing practice guidelines,⁶ largely in response to the premise that allowing an athlete to return to play prematurely could result in prolonged symptoms, long-term cognitive disability, depression, early dementia, or—rarely—death, as exemplified by the second impact syndrome.^{11,134}

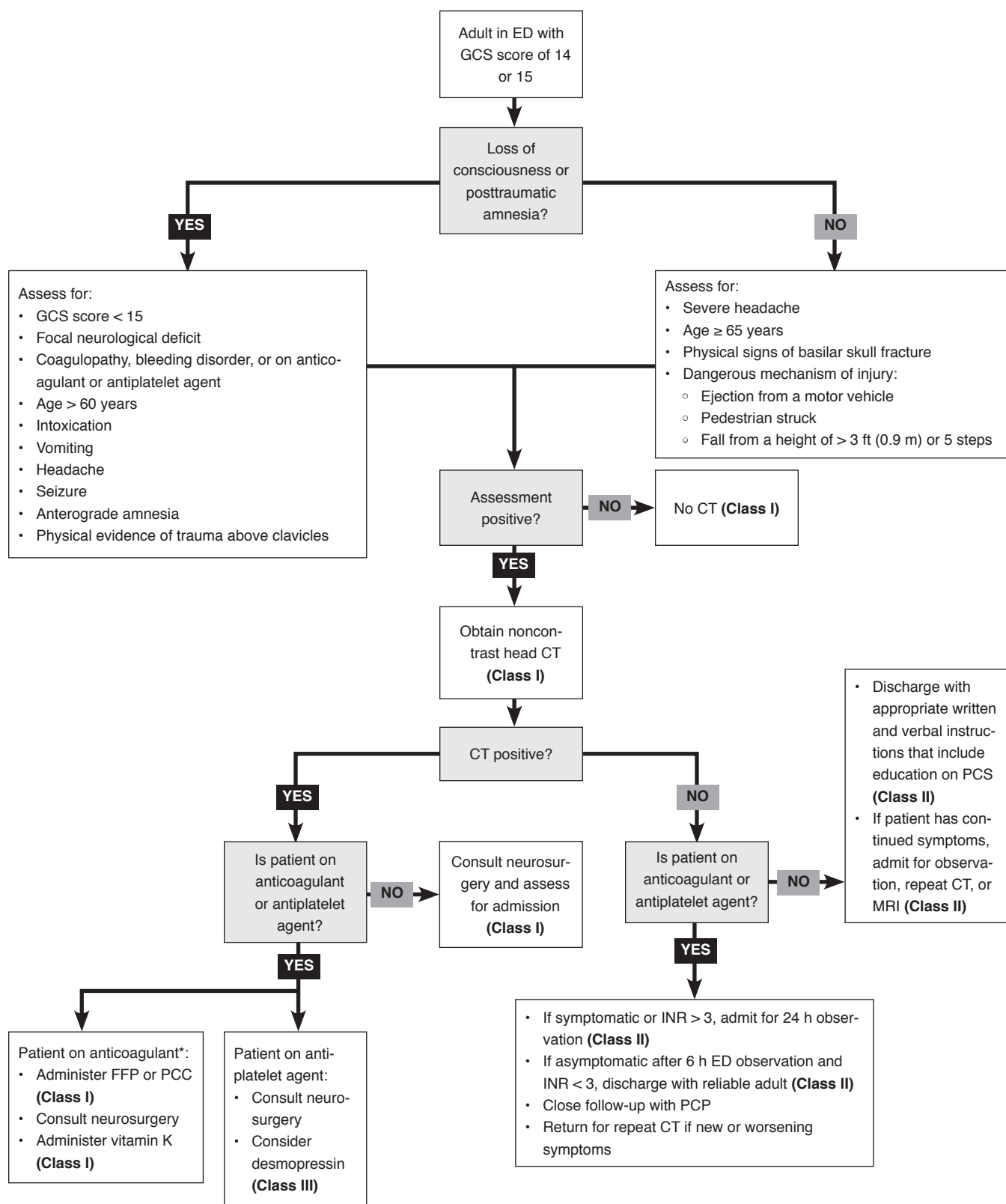
Each year in the United States, almost 10 young athletes suffer a fatal blow to the head, most commonly due to a subdural hematoma.^{135,136} In 2012, McCrory et al challenged the concept of the second impact syndrome, reporting that there is little evidence that the diffuse cerebral edema first reported in second impact syndrome is related to repeated concussions.¹³⁷ Epidemiological studies show that most fatal injuries are associated with an extradural hematoma, and it is unclear whether the history of recent concussion with continued symptoms has a statistically significant association.^{135,136}

The most recent return-to-play guidelines from 2011 dismiss the sideline grading of concussion and

Table 6. Symptoms Seen In Postconcussive Syndrome

Somatic	Cognitive
• Headache	• Attention/concentration problems
• Sleep disturbance	• Memory problems
• Dizziness/vertigo	
• Nausea	Affective
• Fatigue	• Irritability
• Over-sensitivity to noise/light	• Anxiety
	• Depression
	• Emotional lability

Clinical Pathway For Evaluating The Adult With Mild Traumatic Brain Injury

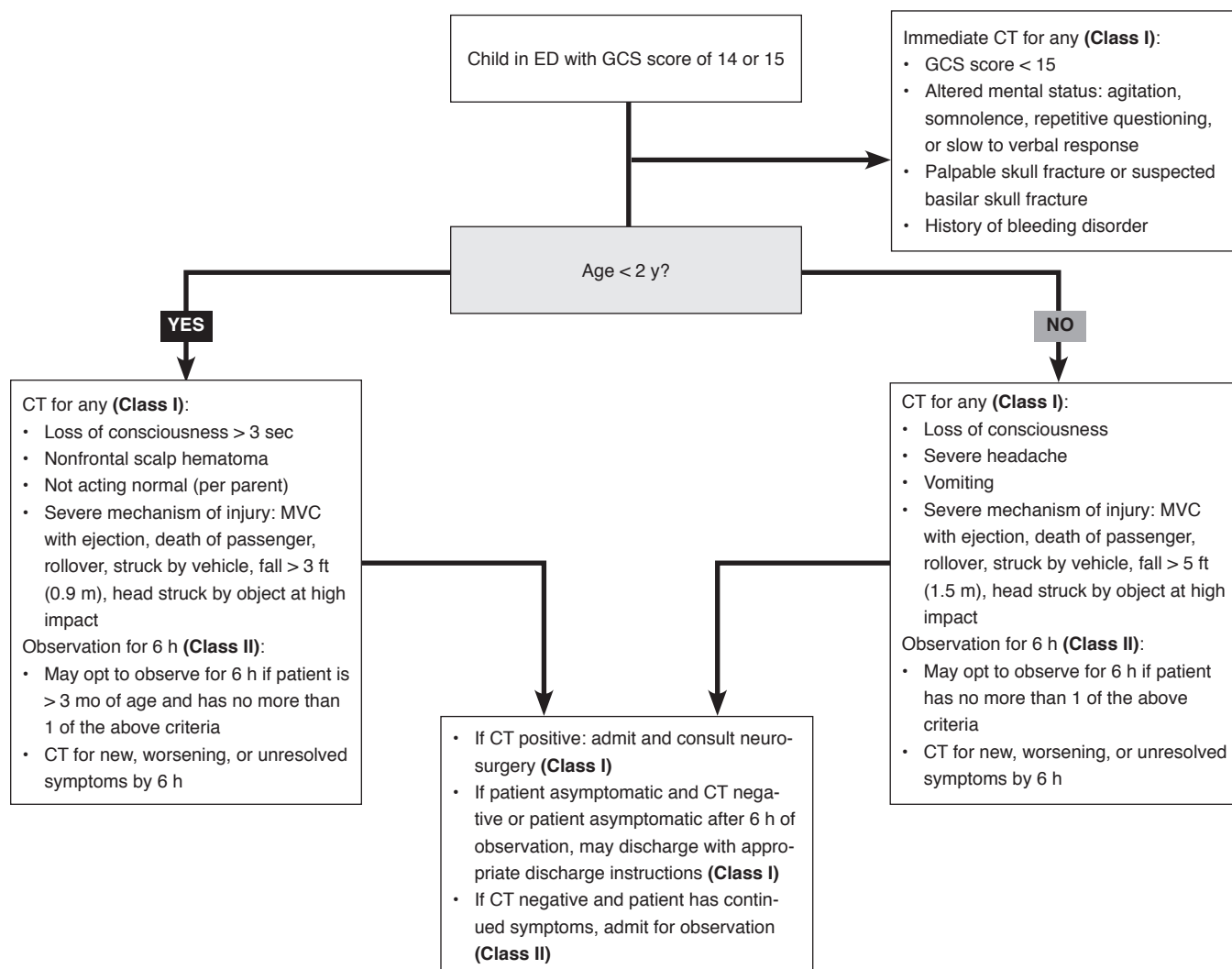


*See text, page 10.

For class of evidence definitions, see page 13.

Abbreviations: CT, computed tomography; ED, emergency department; FFP, fresh frozen plasma; GCS, Glasgow Coma Scale; INR, international normalized ratio; MRI, magnetic resonance imaging; PCC, prothrombin complex concentrate; PCS, postconcussive syndrome; PCP, primary care provider.

Clinical Pathway For Evaluating The Child With Mild Traumatic Brain Injury



*The decision to observe is based on the age of child, the number of symptoms present, and parent and physician comfort. Observation should be for 6 h, and if symptoms continue or worsen, CT is indicated.

Abbreviations: CT, computed tomography; GCS, Glasgow Coma Scale; MVC, motor vehicle crash; TBI, traumatic brain injury.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I

- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:

- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II

- Safe, acceptable
- Probably useful

Level of Evidence:

- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III

- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:

- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate

- Continuing area of research
- No recommendations until further research

Level of Evidence:

- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and represen-

tatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA*. 1992;268(16):2289-2295.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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Risk Management Pitfalls For Mild Traumatic Brain Injury

1. **"The GCS score was normal. How can he have a head bleed?"**

Even in patients with a GCS score of 15, there is a small—but definite—risk for an intracranial lesion. About 6% to 8% of patients with mild TBI and a normal GCS have ICI on CT, and less than 1% will require neurosurgical intervention.^{13,20,22}

2. **"But I told the patient everything at discharge."**

Patients discharged from the ED after mild TBI can be expected to recall no more than 30% to 50% of verbal instructions, and a significant number will suffer from both short-term and long-term postconcussive symptoms.^{8,14} This holds true even for those patients who appear completely neurologically intact. Consequently, all discharge instructions should not only be written down, but also told to a responsible third party.

3. **"But the skull films showed no fracture."**

Numerous studies have demonstrated the low sensitivity of skull films for predicting intracranial lesions. Though the presence of a fracture on a skull film increases the incidence of a traumatic intracranial lesion, the absence of a visible fracture does not decrease the incidence of an intracranial lesion. CT with bone windows is the imaging strategy of choice for patients with suspected TBI.

4. **"The babysitter initially said that the baby fell down the steps, and then changed her story and said the baby fell off the sofa."**

Child abuse is a frequently reported cause of TBI in infants. Emergency clinicians should be on their guard and recall that an inconsistent history is often associated with child abuse.³⁹ When in doubt, it is best to err on the side of caution and involve the proper child protective services.

5. **"But the CT was negative."**

CT is an excellent test for identifying lesions in need of neurosurgical intervention, but it is not very good at identifying brain stem lesions, basilar skull fractures, or nonhemorrhagic injuries. In fact, about 25% of focal axonal injuries,⁶⁹ 50% of brain stem lesions,⁷⁰ and 30% of basilar skull fractures are missed on CT.⁷¹ These injuries typically involve a great deal of energy and are therefore not commonly found in a patient with mild TBI or found in isolation.¹³⁸ It is extremely rare for an initially undetected lesion on CT to evolve into a lesion that requires neurosurgical intervention.¹³⁹ Patients and

families should be given discharge instructions that describe symptoms that require a repeat visit to the ED.

6. **"The patient is malingering. His CT was negative, and the neurologic examination was normal."**

Many patients diagnosed with mild TBI have deficits on cognitive testing despite a normal CT. Most of these deficits resolve within 3 months of the injury, but some do not. It is very stressful for patients with persistent symptoms that do not seem to be supported by objective evidence. Follow-up with a neurologist can be very helpful to determine the need for further neuroimaging or neuropsychological testing.

7. **"The coach asked me if he could play in the tournament tomorrow."**

There is no longer any role for same-day return to play, and the assessment for return to play involves the individual evaluation of the player by his or her primary care or sports medicine physician with consideration to the severity of concussion, past injuries, and expected future impact injuries. Discharge instructions must include both physical and cognitive rest until cleared by the player's physician.

8. **"I thought the patient was just drunk."**

Alcohol users are at increased risk for TBI, and evaluation is made difficult by their intoxication. These patients require serial neurologic evaluations, and if there are any associated high-risk criteria, a CT is indicated.

9. **"He didn't get knocked out. How could he have a subdural hematoma?"**

In many cases of mild TBI, there will be no loss of consciousness, and only about 10% of sports TBI is associated with loss of consciousness. A period of unconsciousness or amnesia to the event is not required for ICI, and the absence of loss of consciousness is not protective against ICI or future symptoms of postconcussive syndrome.

10. **"I know he was on warfarin, but his CT was normal, so I sent him home."**

Delayed hemorrhage is a rare, but important, concern in anticoagulated patients.^{114,115} All patients on anticoagulants must be educated about the risk of delayed hemorrhage and instructed to return for a repeat CT in the setting of any new or worsening symptoms.

admonish that no player should return to play the same day of the concussive insult.¹¹ Thereafter, the assessment for return to play involves the individual assessment of the player by his or her primary care provider with consideration of severity of the concussion, past injuries, and expected future impact injuries.¹⁰ Again, this is not a decision made by the emergency clinician. The website for state laws regarding return to play can be found in **Table 1, page 3.**

Controversies And Cutting Edge

Biomarkers

A simple blood test to rule out ICI in patients with mild TBI would be absolute nirvana for emergency clinicians. In the past 10 years, researchers have evaluated several potential biomarkers, including S100B, glial fibrillary acidic protein (GFAP), myelin basic protein, and neuron-specific enolase. Although some of these markers correlate with injury severity, there are conflicting results. S100B is a calcium-binding protein found in CNS supporting cells and is the most frequently studied biomarker for mild TBI. S100B is also found in chondrocytes and adipocytes, leading to elevated levels in non-CNS injuries, while GFAP has the potential to be more brain-specific than S100B.¹⁴⁰⁻¹⁴² A small prospective study found that GFAP was also more predictive of functional outcome in mild TBI.¹⁴³ Current studies show that the specificity and sensitivity of serum biomarkers as independent predictors of ICI are not superior to the validated clinical decision guidelines, but they may have an important role when used in conjunction with clinical variables.¹⁴⁴⁻¹⁴⁶

Diffusion-Weighted Imaging

In the past few years, diffusion-weighted MRI has come to the forefront in concussion and postconcussive syndrome. Diffusion-weighted MRI is dependent on the molecular movement of water, and it has definitively shown structural change in the white matter at the neuronal level in patients with TBI.⁸⁷ It has been shown to detect minute alterations in white matter after mild TBI, postconcussive syndrome, and even minor impacts such as heading a soccer ball.¹⁴⁷ Its role in patients with mild TBI has yet to be fully elucidated, but in patients with symptoms not explained by CT or MRI, it is allowing neurologists to map white matter injury patterns, even years after the injury.^{87,148} (See **Figure 1.**)

Neuropsychological And Sideline Testing

Neuropsychological testing to assess cognitive function after mild TBI has been studied extensively in the sports medicine, military, and postconcussive syndrome literature. Computerized neuropsychiatric tests are performed 48 to 72 hours to several weeks postinjury,^{12,131} while sideline evaluations by athletic trainers or medics in the military field are used

in the acute setting.^{6,10,11} Limited neurocognitive testing can be performed quickly using a paper and pencil or even using a smart phone application.^{149,150} Most sports and military tests evaluate concentration, reaction times, and information processing.

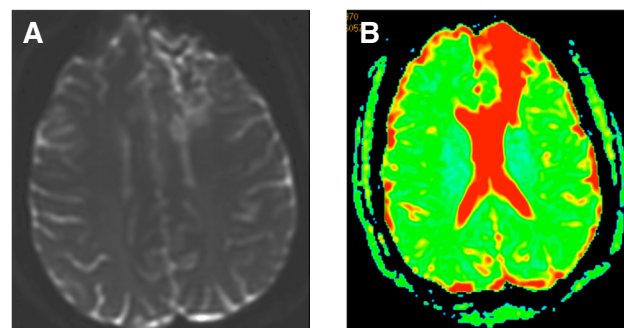
The Zurich Consensus on Concussion in Sports promotes use of the SCAT2 for sideline evaluation of concussed players, which can be downloaded from their website at http://bjsm.bmj.com/content/43/Suppl_1/i85.full.pdf.⁶ In the military setting, the MACE2 tool is used to document TBI symptoms and assess for memory and concentration deficits in deployed soldiers.³ Both the SCAT2 and MACE2 can be used to screen for acute mild TBI and have very little use outside the acute setting.^{6,151}

Computerized neurocognitive testing has been used outside of the acute window to evaluate for ongoing neurocognitive deficits. ImPACT (Immediate Postconcussion Assessment and Cognitive Testing) and ANAM (Automated Neuropsychological Assessment Metrics) are online testing programs designed to measure memory, attention, processing speed, and reaction time, which are then compared to baseline preinjury testing. Both have conflicting results, depending on the setting, and must be used in the appropriate clinical context.^{152,153} ImPACT is used by many national and college-level sports leagues, while ANAM is used extensively by the United States Department of Defense in deployed military soldiers.^{151,153}

Disposition

Disposition of head-injured patients is typically determined by results of clinical examination and neuroimaging studies. A secondary analysis of the PECARN database (> 40,000 pediatric patients) revealed that a period of observation significantly

Figure 1. Diffusion-Weighted Magnetic Resonance Imaging Showing Frontal Injury



Diffusion-weighted magnetic resonance imaging allows clinicians to view the diffusion of water molecules through central nervous system tissue (diffusion-weighted scan, view A) and compare it to the diffusion of water molecules within blood vessels (perfusion scan in view B). Image courtesy of Micelle Haydel, MD.

decreased the use of CT,¹⁵⁵ while a retrospective study of > 17,000 patients with uncomplicated minor head injury concluded that 6 hours of observation allowed clinicians to identify patients that require CT.¹³⁹ Observation that reveals persistent symptoms, abnormal mental status, or abnormal neurological examination should lead to CT.

Both adult and pediatric patients may be discharged to home if their CT, neurological examination, and mental status are all normal.⁶⁸ Delayed ICI in patients with a CT interpreted as normal is exceedingly rare; a retrospective cohort study of > 17,000 children in Canada with a normal CT or asymptomatic 6-hour observation period identi-

fied a delayed ICI in only 0.03% of patients.¹³⁹ Patients with continued symptoms such as short-term memory deficits or repeated vomiting should be considered for admission for further observation, repeat CT, or MRI. If a patient who takes anticoagulants or has a bleeding disorder is not admitted, he or she must be discharged with a reliable adult who can monitor for new or worsening symptoms, and the patient may benefit from a telephone follow-up.^{47,48,103,114}

A prospective study of 200 patients discharged from the ED after mild TBI revealed that patients recall no more than 30% to 50% of verbal instructions.¹⁵⁶ Because cognitive function is frequently

Table 7. Indications For Computed Tomography In Mild Traumatic Brain Injury

Population	Obtain CT			Observation and CT if Worsening or No Resolution of Symptoms
Adults ^{20,21,76}	Immediate CT head for any ⁷⁶ : <ul style="list-style-type: none">GCS score < 15Focal neurological deficitCoagulopathy	Patient with LOC after head trauma, obtain a CT if any present ^{20,76} : <ul style="list-style-type: none">HeadacheEmesisAge > 60 yDrug or ethanol intoxicationSeizureAnterograde amnesia/short-term memory deficitsPhysical evidence of trauma above clavicles (abrasions, contusions, ecchymosis)	Patient with no LOC after head trauma, obtain CT if any present ²¹ : <ul style="list-style-type: none">Severe headacheAge > 65 ySuspected basilar skull fractureDangerous mechanism of injury, including ejection from a vehicle, pedestrian struck by vehicle, fall > 3 ft (0.9 m) or 5 steps	<ul style="list-style-type: none">Consider 6 h observation if the only criterion present for CT is intoxication^{20,21,64,67,69,105}Consider 6 h observation if only criterion is history of GCS score of 14 that returned to normal within 2 h of trauma^{20,69,79}
Children ³⁸	Immediate CT head for any: ³⁸ <ul style="list-style-type: none">GCS score < 15Palpable skull fractureSuspected basilar skull fractureAltered mental status (to include agitation, somnolence, repetitive questioning, verbal slowness to respond)	CT if any present: <ul style="list-style-type: none">History of loss of consciousnessSevere headacheVomitingSevere mechanism of injury: MVC with ejection, death of passenger, rollover, struck by vehicle, fall > 5 ft (1.5 m), head struck by high-impact object If age < 2 y, CT for above, plus: <ul style="list-style-type: none">Nonfrontal scalp hematomaNot acting normal per parentFall > 3 ft (0.9 m)		Consider 6 h observation if age > 3 mo, if only 1 symptom present, and parents and physician comfortable with plan ³⁸
Patients taking anticoagulant or antiplatelet agent or with bleeding disorder	Immediate CT for all			<ul style="list-style-type: none">Admit and give reversal agents for patients with ICIEmpiric reversal agents before CT for severe hemophilia or symptoms of TBIAdmit for continued symptoms or supratherapeutic INR or severe hemophilia.May discharge after 6 h asymptomatic observation, with close monitoring for new symptoms

Abbreviations: CT, computed tomography; GCS, Glasgow Coma Score; INR, international normalized ratio; LOC, loss of consciousness; MVC, motor vehicle crash.

compromised after mild TBI, clear, written instructions should be provided to the patient's family members.

Almost a third of patients will experience headache, dizziness, difficulty concentrating, or depression for up to a month after the injury, which can cause a great deal of anxiety, especially when these symptoms are unexpected.¹⁵⁷ It has been postulated that anxiety caused by inaccurate expectations about recovery after mild TBI plays a role in the development of postconcussive syndrome, and patients have been shown to benefit from early referral for cognitive behavioral therapy.¹⁵⁸ Interestingly, postconcussive syndrome is thought to be less common after sports-related mild TBI because athletes typically have peers or coaching staff who have experienced or witnessed similar symptoms and can explain symptoms that are common after head injury.

The CDC and ACEP have developed a discharge instruction sheet to help patients understand symptoms to expect and when to return to the emergency department.⁷⁶ It is imperative that patients and family be educated about the expected course of recovery and be provided with access to resources in case symptoms persist. The discharge instruction sheet can be downloaded from the CDC website: http://www.cdc.gov/concussion/pdf/TBI_Patient_Instructions-a.pdf. The SCAT2 also includes discharge instruction sheet for patients with sports-related head injury and can be downloaded at http://bjsm.bmj.com/content/43/Suppl_1/i85.full.pdf. Emergency clinicians must be aware of their state's laws governing return to play guidelines. (See Table 1, page 3.)

Key Points For Evaluating And Treating Mild Traumatic Brain Injury

- Obtain a careful history, focusing on loss of consciousness, amnesia, alteration in sensorium, mechanism of injury, vomiting, drug/alcohol use, use of medications (such as warfarin, clopidogrel, and aspirin), bleeding disorders, and any repetitive head injury history.
- Perform a careful physical and neurologic examination, to include GCS score, mental status, pupillary examination, and cranial nerve evaluation, and note any evidence of skull fracture and/or basal skull fracture.
- Obtain CT based on the guidelines in Table 7.
- Observation can be considered in children if they have no high-risk criteria, they are > 3 months of age, they have only 1 symptom present, and the parents and physician are comfortable with the plan. Observe for 6 hours, and if symptoms persist, CT is indicated.³⁸
- Patients whose neurological examination, mental status, and CT are all normal may be discharged to home.⁶⁸

- Patients on anticoagulants or antiplatelet agents should undergo immediate CT. Patients with a normal CT and continued symptoms or a supratherapeutic INR should be admitted for 24 hours. If the CT is normal and the patient is asymptomatic after 6 hours of observation, they may be discharged with a reliable adult. The patient and family must be educated about the risk of delayed hemorrhage and the need for symptom monitoring, and they should be encouraged to return immediately for a repeat CT if any new symptoms should occur.^{47,116}
- Written and verbal discharge instructions must be provided and should include symptoms to expect after a mild TBI, the time course, the overall positive prognosis, activity limitations, and the point at which a patient should seek a neurologist or concussion specialist for further testing. The CDC and ACEP have collaborated to develop a well-written discharge instruction sheet and wallet card for patients that can be downloaded from the CDC website at: http://www.cdc.gov/concussion/pdf/TBI_Patient_Instructions-a.pdf
- Discharge instructions after sports-related injury must stress the need for both cognitive and physical rest until cleared by the patient's primary care or sports medicine physician.⁶ The SCAT2 includes discharge instructions for patients with sport-related head injury and can be downloaded at: http://bjsm.bmj.com/content/43/Suppl_1/i85.full.pdf

Cost-Effective Strategies For Mild Traumatic Brain Injury

1. Skull radiographs are not indicated in patients at risk for TBI; go straight to CT with bone windows.
2. There is no need to observe or admit uncomplicated, asymptomatic adults and children who have a normal CT.
3. Empiric factor replacement (Factor VIII, cryoprecipitate, or fresh frozen plasma) after head injury, before CT, is indicated only in patients with severe hemophilia and symptoms of TBI.¹²⁴
4. Platelet transfusions in patients on aspirin or clopidogrel have not been shown to impact outcomes.¹²⁶
5. Comprehensive written and verbal discharge instructions for mild TBI patients can educate patients and families about follow-up, help them understand their symptoms, and prevent unnecessary return ED visits.

Summary

Clinicians will continue to be faced with patients with mild TBI, and based on the best available evidence, a CT is indicated for all patients with a GCS score < 15, focal neurological deficits, or coagulopathy. The CDC/ACEP guidelines clearly define which other patients should also undergo CT, and following those guidelines will result in a reduction of about 20% of unnecessary scans.⁷⁶ The latest studies have opened the door to observation of lower-risk patients, but the clinician and patient must be aware that observation will not identify all patients with ICI and may miss a rare patient with a clinically important injury. Clinicians must also be aware of their state laws governing return-to-play guidelines as well as the importance of discharge instructions in aiding the 30% of patients who will experience postconcussive symptoms.

Areas In Need Of Future Research

- Identification of the subset of patients whose transient symptoms resolve in the ED who will benefit from CT.
- Determination of the optimal length of time a patient should be observed before making the decision to discharge without CT.
- Identification of patients at highest risk for developing postconcussive syndrome so referrals can be made from the ED.
- Determination of which patients on anticoagulants benefit from admission after a normal CT.
- Determination of which subset of elderly patients may be discharged without a CT.
- Further study of brain-specific serum biomarkers as adjunctive clinical tools.

Case Conclusions

Your 16 year-old soccer champ had no history of loss of consciousness, and while in the ED, his symptoms resolved completely within 2 hours. Using the CDC guidelines, you determined that a CT was not indicated. You discussed this with his parents, and he was discharged home symptom-free 6 hours after his injury. You instructed him and his parents about the importance of physical and cognitive rest (based on the Zurich Guidelines) until cleared by his primary care provider.

The 38-year-old woman in the low-speed motor vehicle crash had a loss of consciousness but no symptoms or risk factors. Based on the CDC guidelines, you do not think a CT is indicated. You discussed with her the very low likelihood of a clinically important ICI, and she was discharged with head injury precautions and information about postconcussive syndrome.

The history on the 2-month old baby was inconsistent, so you suspected abuse. She had a small hematoma in the left parietal region, and you ordered a CT, which

revealed a small subdural. Child Protective Services was called, and the patient was admitted to the PICU.

Your drinking buddy sobered up quickly, but you convinced him to wait for the CT you ordered based on the following CDC criteria: presumed loss of consciousness, intoxication, and physical evidence of trauma above the clavicles. His CT showed atrophy but was otherwise normal. You provided him with follow-up and clear discharge instructions, which he promptly threw in the trash on the way out. Another night in the ED...

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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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- The most appropriate term to use when describing an impact to the head that causes an episode of vomiting, a headache, and a GCS score of 15 is:
 - Minor head trauma
 - Minimal head injury
 - Mild TBI
 - Grade 1 concussion
- Which symptom has not been shown to have a significantly high positive likelihood ratio for ICI?
 - Seizures
 - Deterioration in mental status
 - GCS score of 14
 - Repeated vomiting
- Deficits of which components of the GCS score have the strongest correlation with poor outcomes in patients with TBI?
 - Best eye opening
 - Best verbal response
 - Best motor response
- The tau-transferrin test is the gold standard for distinguishing CSF from normal nasal secretions.
 - True
 - False
- When comparing radiographic modalities after trauma, which of the following is true?
 - CT has a high sensitivity in identifying basilar skull fractures.
 - Diffusion-weighted MRI is indicated as a first-line imaging modality in patients with mild TBI.
 - MRI is more sensitive for all intracranial bleeds immediately after the injury than CT.
 - Diffusion-weighted MRI can show structural damage in the white matter at the neuronal level in patients with TBI.
- Which of the following is true regarding TBI in the elderly?
 - Although the elderly fall more than other groups, they have a lower risk of hospitalization or death due to TBI.
 - The elderly have less fragile, more-elastic bridging veins and are not at risk for more severe injuries.
 - Because the elderly typically have some degree of cerebral atrophy, they are less prone to hemorrhage.
 - Age has been shown to be an independent predictor of mortality in isolated mild and moderate TBI.
- The emergency clinician should have a lower threshold for imaging patients with mild TBI in which of the following groups?
 - Anticoagulated patients
 - The elderly
 - Infants < 2 months of age
 - All of the above
- With regard to postconcussive syndrome, which of the following is true?
 - It is only found in patients who have had an abnormality on CT.
 - Nearly 68% of patients with mild TBI will be symptomatic at 3 months postinjury.
 - The risk of postconcussive syndrome is higher in patients with preexisting stress, anxiety, and depression.
 - Discharge information about postconcussive syndrome is only important when the patient has a positive CT.
- Neuropsychological testing:
 - Should be performed as soon as possible after ED arrival
 - Should be considered on an outpatient basis for patients with continued symptoms after mild TBI
 - Has no role in mild TBI
 - Has never been studied

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