Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Volume 34, Number 11 / May 19, 2013 www.emreports.com

Authors:

Justin L. Weppner, DO, Lieutenant, Medical Corps, USN, Naval Medical Center Portsmouth, Portsmouth, VA.

James A. McCombs, DO, Lieutenant, Medical Corps, USN, Naval Medical Center Portsmouth, Portsmouth, VA.

Peer Reviewer:

Michael C. Bond, MD, FACEP, FAAEM, Assistant Professor, Residency Program Director, Department of Emergency Medicine, University of Maryland School of Medicine

Take our 2013 Reader Survey! See details on page 131.

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Farel (CME question reviewer) owns stock in Johnson & Johnson. Dr. Schneider (editor) is a retained consultant for Logical Images. Dr. Stapczynski (editor), Dr. Weppner (author), Dr. McCombs (author), Dr. Bond (peer reviewer), Ms. Mark (executive editor), and Ms. Hamlin (managing editor) report no financial relationships with companies related to the field of study covered by this CME activity.

AHC Media

Minor Trauma Management: Part I

Over the past few years, we have learned that seemingly minor injuries can cause significant morbidity and, in some cases, even contribute to mortality. Astute emergency providers have learned to look beyond minor complaints and watch for signs of significant problems. Mild traumatic brain injury or concussion is one of the best examples of an injury that has been often dismissed. Post-concussive syndrome was overlooked for many years. Athletes in particular are prone to repeated injury, which we now know contributes to later cognitive impairment and even in some cases death.

This is a two-part series covering minor injuries that are, in fact, not minor. In this first part, the authors discuss mild traumatic brain injury with special emphasis on the judicious use of testing. Part two will concentrate on neck and thoracic injuries.

— Sandra M. Schneider, MD, Editor

Introduction

Determining how to manage minor injuries such as mild traumatic brain injury and minor neck and chest trauma can be difficult due to the trivial outward appearance of such injuries. In cases of minor traumatic injuries, emergency physicians must consider injuries that do not seem apparent upon first glance and must proactively identify and treat unseen injuries before they become catastrophic. Emergency physicians should be cognizant of the potential pitfalls in order to safely and effectively manage patients with minor trauma. This evidence-based review assesses the literature and reviews current guidelines and decision rules on mild traumatic brain injury.

Mild Traumatic Brain Injury

Traumatic brain injury (TBI) is defined as the impairment of brain function due to mechanical force. TBI is an important public health problem in the United States and is frequently referred to as the "silent epidemic" because the complications from TBI, such as changes affecting thought, sensation, language, or emotions, may not be readily apparent.[1] The clinical approach to these patients varies widely, and despite the availability of clinical guidelines, many patients undergo computed tomography (CT) imaging, with the majority being interpreted as normal. The challenge for emergency physicians is to quickly screen for the small subset of patients who have potentially lethal intracranial lesions while minimizing excessive cost and radiation.

Each year, an estimated 1.7 million people sustain a head injury in the United States, with 1.3 million of those undergoing emergency evaluation, of which 80% are considered mild injury.[1,2] Men are overrepresented at a ratio of 3:1 in all subgroups of TBI, but in some comparable sports, the rate of mild TBI is higher in women.[3,4] The four leading causes of TBI treated in the emergency department (ED) are: falls, motor vehicle–related injury, unintentional strike by or against an object (which includes sports and recreational injury), and assaults.[3]

Mild TBI may result in a predominantly metabolic insult, as opposed to a

Executive Summary

- Patients who sustain a mild traumatic head injury and meet the ACEP criteria — symptomatic, older, on anticoagulants, short-term memory loss, seizures, etc. — should receive a CT scan of the head.
- Post-concussive syndrome can occur after a head injury, even one which did not cause a loss of consciousness.
- Patients with a mild traumatic head injury and a normal CT who are not on anticoagulants are at very low risk for an adverse outcome due to the injury.
- While a single concussion may cause some symptoms such as post-concussive syndrome, a second concussion can cause permanent cognitive damage or even death.

structural one.[5-7] The neuropathology involved in producing the signs and symptoms of minor TBI may remain at the neurobiochemical level, without damage to the microstructure.[8] Disruption of enzymatic pathways and accumulation of lactate and nitric oxide in the brain tissue have been reported after experimental minor TBI.[9] Approximately 6–8% of patients with mild TBI will have specific injuries detectable on CT.[10-12] These injuries include subarachnoid hemorrhage, subdural hematomas, epidural hematomas, cerebral contusions, intraparenchymal hemorrhage, and edema and petechial hemorrhage consistent with axonal injury.

Table 1: Glasgow Coma Scale Scoring

Component		Score
Best Eye Opening	Spontaneous	4
	To verbal stimuli	3
	To painful stimuli	2
	No eye opening	1
Best Verbal Response	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible	2
	No verbal response	1
Best Motor Response	Obeys commands	6
	Localizes pain	5
	Withdraws to pain	4
	Flexion to pain	3
	Extension to pain	2
	No motor response	1
	Total	__/15

History

From a practical standpoint, minor TBI is a clinical diagnosis.[13] The American Congress of Rehabilitation Medicine defines mild TBI based on the following inclusion criteria: 1) any period of loss of consciousness of less than 30 minutes and a Glasgow Coma Scale (GCS) score of 13–15 after this period of unconsciousness (*see Table 1*); 2) any loss of memory of the event immediately before or after the accident, with post-traumatic amnesia spanning no more than 24 hours; 3) any alteration in mental state at the time of the accident, such as feeling dazed, disoriented, or confused.[14]

By the time most patients with minor TBI reach the ED, their symptoms are resolving or have completely resolved.[13] In the ED, the diagnosis of mild TBI relies on the history surrounding the injury, such as any alteration in mental state at the time of the event or subsequent to the event. This includes the layman's account of "getting your bell rung," "seeing stars," or being dazed and confused because of the injury. The presence of amnesia further supports the diagnosis and may be associated with injury that is more significant.

The mechanism of injury is a key component of the history and provides information regarding associated injuries. Mechanisms of injury that are associated with an increased risk of intracranial injuries include pedestrians being struck by a motor vehicle, falls from a height greater than 3 feet, or falls from more than five stairs.[11] The presence of loss of consciousness has been shown to increase the likelihood of intracranial injury, but its absence is only useful as a negative predictor if there are no associated symptoms.[12] Signs and symptoms such as seizure, GCS score less than 14, repeated vomiting, and focal neurological deficit have a significantly high positive likelihood of intracranial injury and should be gleaned from the patient.[15,16] Drug and alcohol use is associated with intracranial injury in patients with TBI and is an important factor in the history to help determine management, but it does not have a clear role as an independent indicator of outcome.[17,18] In sports, past history

Table 2: Signs and Symptoms Associated with Mild Traumatic Brain Injury

Observed Signs	Physical Symptoms	Cognitive Symptoms	Emotional Symptoms
Appears dazed or stunned	Headache or "pressure" in head	Difficulty thinking clearly	Irritable
Is confused about events	Nausea or vomiting	Difficulty concentrating	Depressed
Repeats questions	Balance problems or dizziness	Difficulty remembering	Anxious
Answers to questions slowly	Fatigue or feeling tired	Feeling sluggish, hazy, foggy, or groggy	Emotional lability
Cannot remember events prior to the traumatic event	Blurry or double vision	Orientation problems	Loss of initiative
Cannot remember events after the traumatic event	Sensitivity to light	Slow reaction time	Problems at work, home, or school
Loss of consciousness	Sensitivity to noise	Calculation difficulties and problems with executive functioning	
Shows behavior or personality changes	Numbness or tingling		
	Insomnia		
	Seizures		

of concussions, time since last concussion, and severity and duration of past concussion symptoms are all important factors and can assist in predicting outcomes after mild TBI.[19]

Many patients with mild TBI experience initial objective neuropsychological difficulties involving memory, attention, and executive functioning.[20] The most common complaint after minor TBI is headache. A complicating factor is that many of the signs and symptoms of concussion are non-specific and overlap those of other conditions. (*See Table 2.*) Clinical symptoms may begin immediately after the injury or be delayed for days to weeks. The lack of signs and symptoms at the time of evaluation does not exclude mild TBI if the history is consistent with the diagnosis.

Physical Examination

The physical findings of isolated mild TBI are often normal, but it is important to document a full neurologic examination and look for clues to the severity of the mechanism of injury and any neurologic impairment. Focal neurologic findings are rare and suggest potential significant intracranial pathology. Abnormal pupillary reflexes indicate underlying pathology and should be monitored serially with neurologic examinations.[21]

Although it is insensitive in mild TBI, it is important to document the GCS score in the patient's chart. Deficits in the motor component have the strongest correlation with poor outcome in patients with TBI.[21,22] Assess the patient for signs of global impairment, such as confusion and amnesia. An examination of gait is important to identify possible balance impairments. In addition, coordination can be assessed using finger-nose-finger testing, heel-to-shin testing, and rapid alternating movements.

The most common findings in mild TBI are subtle impairments in cognitive function. A prospective study conducted on 1,262 subjects revealed that intracranial injury on the CT scan does not predict cognitive deficits.[23] Additionally, cognitive tests have not been shown to predict abnormalities on head CT.[24] However, several prospective studies have demonstrated that memory tests can be used to predict post-concussive syndrome (PCS).[25-27] Assessing cognitive function in the ED is challenging. A patient with mild TBI can be assessed for cognitive deficits in the ED by testing short-term memory (using 3-item recall) and concentration by asking the patient to count serial sevens backward from 100, spelling the word "world" backward, or reciting the months of the year backward. Emergency physicians should be aware that no isolated test can exclude or confirm cognitive deficits. The neuropsychological examination administered by a trained neuropsychologist is the gold standard for evaluation, but it is time-consuming and not practical in the ED setting.[19,28,29]

Radiographic Indications

An emergency physician faces the challenge of identifying patients with acute intracranial injury from a spectrum of injuries that includes isolated cranial fractures, subarachnoid hemorrhage, subdural and epidural hematomas, and contusions; the emergency physician also has to identify those who can be safely

Table 3: ACEP Clinical Policy, New Orleans, and Canadian CT Clinical Decision Rules

ACEP Clinical Policy on CT Scan Criteria for Mild TBI	New Orleans Criteria*	Canadian CT Head Rule**
Headache	Headache	Failure to reach a GCS of 15 within 2 hours of injury
Vomiting	Vomiting	Suspected open skull fracture
Age greater than 60 years	Age greater than 60 years	Signs of basal skull fracture
Drug or alcohol intoxication	Intoxication	Vomiting more than once
Deficits in short-term memory	Deficits in short-term memory	Age greater than 64 years
Physical evidence of trauma above the clavicle	Physical evidence of trauma above the clavicle	Amnesia before impact of greater than 30 minutes
Post-traumatic seizures	Seizure	Dangerous mechanism of injury***
GCS score less than 15		
Focal neurological deficit		
Coagulopathy		

GCS = Glasgow Coma Scale
ACEP = American College of Emergency Physicians
*Patients must have a GCS of 15
**Patients may have an initial GCS of 13-15
***A dangerous mechanism was defined as pedestrian struck by a motor vehicle, occupant ejected from a motor vehicle, and fall from a height of greater than 3 feet or 5 stairs.

sent home. Up to 15% of mild TBI patients with a GCS score of 14 or 15 will have an acute intracranial injury on non-contrast CT; 1% will have a lesion requiring neurosurgical intervention.[30-36]

In 2008, the American College of Emergency Physicians (ACEP) published its clinical policy on CT scan criteria for mild TBI. The level A recommendation is for a non-contrast head CT in head trauma patients with loss of consciousness or post-traumatic amnesia only if one or more of the following is present: headache; vomiting; age greater than 60 years; drug or alcohol intoxication; short-term memory loss; physical evidence of trauma above the clavicle; post-traumatic seizure; GCS score less than 15; focal neurologic deficit; coagulopathy. A level B recommendation is for a non-contrast head CT in head trauma patients with no loss of consciousness or post-traumatic amnesia if one or more of the following are present: focal neurologic deficit; vomiting; severe headache; age 65 years or older; physical finding of basilar skull fracture; GCS score less than 15; coagulopathy; a dangerous mechanism of injury such as ejection from a motor vehicle, a pedestrian struck by a motor vehicle, or a fall from a height of more than 3 feet or five stairs.[37] However, it is important to note that these are recommendations for when to obtain a CT. They are not guidelines for when it is safe to forego a CT.

Haydel et al[10] prospectively enrolled 1,429 patients with a GCS score of 15 in the ED and a history of loss of consciousness or amnesia after the traumatic event. The study had two phases: an initial phase in which predictors from intracranial injury were identified (n = 520) and a validation phase to validate the predictors (n = 909). The study reported that 93 (6.5%) patients had an intracranial lesion and that six (0.4%) required neurosurgical intervention. The New Orleans Criteria derived from the study consisted of the following seven criteria: headache; vomiting; age greater than 60 years; intoxication; deficit in short-term memory; physical evidence of trauma above the clavicle; seizure. Absence of all seven findings had 100% negative predictive value (95% confidence interval [CI], 99–100%).

Steill et al[11] evaluated 3,121 patients prospectively, 2,489 of whom had a GCS score of 15. Patients attended a follow-up interview at 14 days to assess outcome. The primary outcome measure was the need for neurosurgical intervention, and the secondary outcome was a clinically important brain injury, defined by expert consensus. Clinically unimportant lesions were defined as solitary contusions less than 5 mm in diameter, smear subdurals less than 4 mm thick, isolated pneumocephalus, and closed depressed skull fractures not through the inner table. The Canadian CT Head Rule derived from the study consisted of five criteria that were considered high-risk features: failure to reach a GCS score of 15 within 2 hours of injury; suspected open skull fracture; signs of basal skull fracture; vomiting more than once; and age greater than 64 years. In addition, there were two criteria that were considered medium-risk features: amnesia of events before impact greater than 30 minutes and a dangerous mechanism of injury such as a pedestrian struck by a motor vehicle, occupant ejected from a motor vehicle, or fall from a height greater than 3 feet or five stairs. (*See Table 3.*)

Multiple studies have compared

the performance of the New Orleans Criteria and the Canadian CT Head Rule.[38-41] Smits et al[39] applied the two decision rules at four university hospitals in the Netherlands to 3,181 consecutive adult patients with a GCS score of 13 or 14 or a GCS score of 15 and one risk factor, as identified by the decision rules. The New Orleans Criteria had 100% sensitivity for identifying a neurosurgical lesion (95% CI, 34.2–100%) and 5.3% specificity (95% CI, 2.5–8.3%). In addition, the New Orleans Criteria had 98.3% sensitivity for identifying an intracranial lesion (95% CI, 94–99.5%) and 5.6% specificity (95% CI, 2.7–8.8%). The Canadian CT Head Rule had 100% sensitivity for identifying a neurosurgical lesion (95% CI, 64.6–100%) and 37.2% specificity (95% CI, 34.1–40.4%). Furthermore, the Canadian CT Head Rule had 83.4% sensitivity (95% CI, 77.7–87.9%) and 39.4% specificity (95% CI, 36–42.8%) for identifying an intracranial lesion. The study validated the high sensitivity of both rules for identifying lesions requiring neurosurgical intervention. It also demonstrated the superiority of the New Orleans Criteria over the Canadian CT Head Rule for identifying acute intracranial lesions. However, the higher sensitivity of the New Orleans Criteria came at the expense of a significantly lower specificity.

Papa et al[40] consecutively enrolled 431 patients at a United States level I trauma center who presented with mild head injury, who experienced loss of consciousness, disorientation, or amnesia, and had a GCS score of 13–15. Both the New Orleans Criteria and the Canadian CT Head Rule had 100% sensitivity for detecting traumatic intracranial lesions (95% CI, 82–100%), but the Canadian CT Head Rule was more specific, with a specificity of 36.3% (95% CI, 31–42%) vs. the specificity of 10.2% (95% CI, 7–14%) of the New Orleans Criteria. The Canadian CT Head Rule and New Orleans Criteria both had 100% sensitivity for detecting clinically important brain lesions (95% CI, 68–100%), but specificity was 35% (95% CI, 30–41%) for the Canadian CT Head Rule and 9.9% (95% CI, 7–14%) for the New Orleans Criteria. When the rules were compared for predicting the need for neurosurgical intervention, their sensitivity was equivalent at 100% (95% CI, 31–100%), but the Canadian CT Head Rule had higher specificity at 80.7% (95% CI, 76–85%) vs. the 9.6% (95% CI, 7–14%) specificity of the New Orleans Criteria. Again, the New Orleans Criteria had equivalently high sensitivities for detecting a traumatic intracranial lesion on CT, clinically important brain injury, and neurosurgical intervention, but the Canadian CT Head Rule was more specific.

Although both the New Orleans Criteria and the Canadian CT Head Rule have been validated, it is very important to ensure that they are applied within the limits of their inclusion criteria, and the clinician should understand the above sensitivity and specificity both for neurosurgical lesions and for intracranial injury. These rules are valid when applied to patients who have experienced loss of consciousness or amnesia and who are not on anticoagulants.

A combination of these rules provides a basis for identifying patients at risk for underlying intracranial injury and for determining the need for head CT. Decision rules can guide clinical practice, but each patient must be assessed individually, and none of these rules address short- or long-term non-neurosurgical sequelae.[28]

Laboratory Evaluation

Currently, no reliable tests can confirm the diagnosis of concussion. A number of serum biomarkers such as S100B, neuron-specific enolase, myelin basic protein, cleaved tau, and creatine kinase isoenzyme BB have all been investigated in minor TBI. At this time, only S100B predicts abnormal CT findings in minor TBI. However, S100B appears to lack central nervous system specificity and is often elevated in multisystem trauma patients with no head injury.[9] The development of serum markers is evolving and may play a new role in diagnosis and management in the future.[42-55]

Patients with known or suspected coagulation disorders, liver disease, or those taking anticoagulants should have coagulation studies performed as soon as possible.[56]

Management

There are no pharmacologic treatments for mild TBI.[28] The management decisions faced by the emergency physician are the same as those addressed when evaluating all patients with mild TBI, such as the extent of the workup to initiate in the ED and whether the patient can be discharged safely. The main objectives are to identify patients who have intracranial lesions requiring neurosurgical intervention, identify patients whose condition might deteriorate, and guide their return to normal activities.

Anticoagulants and Anti-platelet Agents with Mild TBI. The use of anticoagulation or anti-platelet agents is common for the treatment of various conditions, ranging from warfarin for atrial fibrillation to clopidogrel for cardiac stents. The prevalence of immediate traumatic intracranial hemorrhage in patients with pre-injury warfarin use ranges from 0% to 65%,[56-65] and that in patients with pre-injury clopidogrel use, prevalence ranges from 36% to 71%.[58,59,66] The risk of intracranial injury increases with increased international normalization ratios (INR). An INR of 2.4 or more increases the risk of immediate intracranial injury.[56,60,67] Regrettably, no specific INR can be used to exclude the risk of intracranial injury in patients with a sub-therapeutic INR.[68] Current guidelines recommend that patients with head trauma and pre-injury warfarin use undergo routine cranial CT scanning.[37,69-71] A recent retrospective study by Moore et al[2] found that failing to perform a CT in patients presenting with a GCS score of 15 and no history of loss of consciousness would have led to a

missed injury rate of 66% (43 of 65 patients).

A prospective, observational, multicenter study evaluated the incidence and prevalence of immediate and delayed traumatic intracranial hemorrhage in patients with blunt trauma who were receiving clopidogrel and warfarin. Seventy of the 1,000 patients had immediate traumatic intracranial hemorrhage on CT in the ED. The prevalence of immediate traumatic intracranial hemorrhage was higher in patients receiving clopidogrel (33/276; 12%; 95% CI, 8.4–16.4%) than in those receiving warfarin (37/724; 5.1%; 95% CI, 3.6–7%). Delayed traumatic intracranial hemorrhage was identified in four of 687 (0.6%; 95% CI, 0.2–1.5%) patients receiving warfarin and none of 243 (0%; 95% CI, 0–1.5%) patients receiving clopidogrel.[72] The prevalence of immediate traumatic intracranial hemorrhage in well-appearing patients is a matter of great concern. In the immediate traumatic intracranial hemorrhage study, more than 60% of both the warfarin and clopidogrel cohorts had normal mental status and a GCS score of 15. In addition, a significant proportion of patients experienced no loss of consciousness and exhibited no physical evidence of trauma above the clavicle. Thus, the authors recommended routine urgent CT imaging in head-injured patients with previous warfarin or clopidogrel use, even in well-appearing patients without a history of loss of consciousness or amnesia.

The concern for delayed intracranial hemorrhage in patients with warfarin use is based on several case reports and case series.[73-75] This has led to published guidelines recommending routine admission for all head-injured patients receiving warfarin despite a normal CT scan result.[76] Nevertheless, the results of the prospective, multicenter study by Nishijima et al indicate that delayed traumatic intracranial hemorrhage occurs infrequently in less than 1% of both the warfarin and clopidogrel populations. According to that study, patients receiving warfarin or clopidogrel who have a normal cranial CT scan result and no other indications for admission may be discharged home with explicit discharge instructions for close follow-up.[72]

Peck et al[77] retrospectively reviewed 500 patients taking prescription anti-platelet and anticoagulant agents. They found a 1% incidence of delayed intracranial hemorrhage following a repeat CT of the patients. Of four patients identified in the study with delayed bleeding, none had focal findings on examination and none required any intervention. These findings suggest that clinically significant delayed intracranial hemorrhage is rare.

Overall, patients taking anticoagulation agents with suspected head injury should undergo an initial head CT and an observation period for signs and symptoms of neurologic deterioration. If the initial head CT is negative, and the neurologic examination is normal or unchanged, these patients can be safely discharged without a second head CT and with close outpatient follow-up. This approach should be sufficiently sensitive to detect significant intracranial hemorrhage while avoiding unnecessary CTs. Patients who are on anti-platelet therapy or who are anticoagulated with a normal CT and continued symptoms of TBI or a supratherapeutic INR should be admitted for 24 hours.[67,78]

Emergency physicians should have a low threshold for factor replacement or reversal agents in patients taking anticoagulants or anti-platelet agents.[61,79] Patients on warfarin with intracranial injury on CT should undergo rapid reversal using fresh frozen plasma or prothrombin complex concentrates.[80] It should be noted that the standard dose of 10-20 mg/kg of fresh frozen plasma may far exceed the initial two units that are ordered by most clinicians.[81] Prothrombin complex concentrates have replaced fresh frozen plasma at many institutions due the considerable volume of fresh frozen plasma required to lower the INR. Yasaka et al[82] found that a dose of 500 IU of prothrombin complex concentrates was optimal for a rapid reversal for an INR less than 5.0, but that higher doses might be needed for higher INRs. 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. Recombinant factor VIIa (rFVIIa) may also be administered to anticoagulated patients who require urgent craniotomy. Administering rFVIIa to reverse anticoagulation is an off-label use, and further research is needed to better define its role in patients with traumatic intracranial bleeding.[83,84] In the case of anti-platelet agents, platelet transfusions in patients taking aspirin or clopidogrel have not been shown to influence outcomes after TBI.[85] Reversal of newer anticoagulants is more difficult, as they do not respond to vitamin K or fresh frozen plasma.

Post-concussive Syndrome

The symptoms of almost all patients with minor TBI will undergo rapid and complete resolution,[86] and there is currently no good evidence that uncomplicated minor TBI leads to long-term sequelae.[87] However, post-concussive syndrome affects a subset of patients that report persistent symptoms for prolonged periods after injury. The incidence of post-concussive syndrome is reported to be 10–25%, but these estimates are based on inpatient studies and are likely to be an overestimation of the incidence in mild TBI patients.[9,87,88] In EDs, there has been little focus on the potential development of post-concussive syndrome.[89] The most common delayed or persistent post-concussive complaints are headache, sensory sensitivity, memory or concentration difficulties, irritability, sleep disturbances, and depression. Studies of concussed athletes who undergo pre-concussion testing show that the cognitive domain most frequently involved in post-concussive syndrome is memory.[9] Dizziness that occurs early after trauma is associated with prolonged post-concussive syndrome.[11]

Resuming Sports Activities

It is imperative to emphasize the need for competent follow-up and clearance before resuming normal activities, especially contact sports.[90] Repeated concussions can lead to long-term cognitive deficits and structural damage to the brain.[20,91] Ensure that patients with mild TBI avoid any unnecessary risk of a "second impact" during the symptomatic period, which can result in devastating brain edema. Rapid onset of cerebral edema and death can occur when a second concussion occurs prior to recovery from the first, particularly in athletes returning prematurely to sports.[91] Decisions on return to play should be managed very carefully by health care providers trained specifically in concussion management. In view of the concerns for second impact syndrome, a graded activity program should be instituted until athletes can resume full activities.[20,92-94] Recent return to play guidelines no longer advocate return to play the same day of the traumatic event.[4] The assessment for return to play is not a decision made by the emergency physician, but is managed by primary health care providers and specialists with consideration of the severity of the injury, past injuries, and the possibility of future sports-related injuries.[95]

Admission Criteria

While most patients with mild TBI are safe to discharge, a brief inpatient observation period of 12–24 hours is advisable if any doubt exists regarding the safety of the discharged patient with minor TBI.[13] Other criteria for admission are mass lesions associated with head trauma requiring intensive monitoring or surgery, intracranial bleeding, and patients with ongoing symptoms that include repetitive questioning, anterograde amnesia, or disorientation.[96] Patients who are on anti-platelet therapy or who are anticoagulated with a normal CT and continued symptoms of TBI or a supratherapeutic INR should be observed for 24 hours.[67,78]

Discharge Criteria

Patients with isolated mild TBI who have a normal head CT scan and are at minimal risk for developing an intracranial lesion may be safely discharged from the ED.[37] A prospective study of 1,170 mild TBI patients with a GCS score of 15 and who underwent CT and 24-h admission found that no patient with a negative CT scan result later deteriorated.[97] A retrospective study of 2,252 trauma center admissions who had a head CT scan found that no patient with an initial negative head CT scan required subsequent neurosurgical intervention.[98] A comprehensive literature review involving 2,187 abstracts and 410 articles indicated a total mild TBI population of 62,000 subjects who presented with a GCS score of 15. Only three (0.005%) cases of an adverse outcome in the first 48 hours were reported in the literature. Eight additional patients with *possible* early adverse outcomes were also observed. The conclusion based on the literature review was that in patients meeting guidelines for non-contrast head CT, the evidence supports the CT strategy as a safe way to triage mild TBI patients for admission versus discharge.[99]

Patients with minor head trauma not meeting criteria for head CT and normal neurologic findings may be discharged home with a friend or family member and with head injury discharge instructions.[96]

Discharge Instructions

Several studies have documented poor understanding of discharge instructions in patients with mild TBI.[100,101] The decision to discharge a mild TBI patient from the ED must be coupled with appropriate discharge instructions. It is imperative to discharge the patient to the care of a responsible individual and to provide instructions to both the patient and the individual. If CT is not clinically indicated, the patient should receive careful instructions about delayed symptoms and circumstances for return to the ED. It is also very important to provide instructions regarding physical activity and primary care follow-up.

Bazarian et al[100] evaluated abstracted records from the National Hospital Ambulatory Medical Care Survey for 306 patients with isolated mild TBI. Of these 306 patients, 9% were discharged without any recommendations for follow-up, and 28% had only "return to the ED as needed" as a follow-up recommendation. Another study reviewed mild TBI discharge instruction sheets from 15 institutions for the presence of six factors that were deemed significant for post-discharge patient monitoring based on their literature review: signs and symptoms of declining GCS score; amnesia; headache; vomiting; neurologic deficit; seizure. Of the 15 institutions, the discharge instructions of only one institution contained all six factors. They also found that most of the discharge instructions were at a high-grade reading level.[101] Discharge instructions approved by ACEP and Centers for Disease Control and Prevention (CDC) are available on the CDC website at http://www.cdc.gov/concussion/pdf/TBI_Patient_Instructions-a.pdf.[102]

Summary

The ACEP clinical policy on CT scan criteria for mild TBI provides clearly defined guidelines for patients who should undergo non-contrast CT scanning. Although both the New Orleans Criteria and the Canadian CT Head Rule are validated decision rules, it is very important to ensure that they are applied within the limits of their inclusion criteria, and the clinician should be cognizant of their sensitivity and specificity both for neurosurgical lesions and for intracranial injury. It is imperative to emphasize the need for competent follow-up and clearance before resuming normal activities, especially contact sports. Repeated concussions can lead to long-term cognitive deficits and structural damage to the brain. The assessment for return to play is not a decision made by the emergency physician, but is managed by

primary providers and specialists with consideration of the severity of the injury, past injuries, and the possibility of future sports-related injuries.[95] The decision to discharge a mild TBI patient from the ED must be coupled with appropriate discharge instructions, and the patient should be discharged to the care of a responsible individual.

References

1. Faul M XL, Wald MM, Coronado VG. Traumatic brain injury in the United States: Emergency department visits, hospitalizations and deaths 2002–2006. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. Vol March 2010: CDC; 2010: http://www.cdc.gov/traumaticbraininjury/pdf/blue_book.pdf. Accessed 3/15/2013.
2. Moore MM, Pasquale MD, Badellino M. Impact of age and anticoagulation: Need for neurosurgical intervention in trauma patients with mild traumatic brain injury. *J Trauma Acute Care Surg* 2012;73(1):126-130.
3. Langlois JA, Rutland-Brown W, Wald MM. The epidemiology and impact of traumatic brain injury: A brief overview. *J Head Trauma Rehabil* 2006;21(5):375-378.
4. Herring SA, Cantu RC, Guskiewicz KM, et al. Concussion (mild traumatic brain injury) and the team physician: A consensus statement — 2011 update. *Med Sci Sports Exerc* 2011;43(12):2412-2422.
5. Hovda DA. Oxidative need and oxidative capacity following traumatic brain injury. *Crit Care Med* 2007;35(2):663-664.
6. Lifshitz J, Sullivan PG, Hovda DA, et al. Mitochondrial damage and dysfunction in traumatic brain injury. *Mitochondrion* 2004;4(5-6):705-713.
7. Cimatti M. Assessment of metabolic cerebral damage using proton magnetic resonance spectroscopy in mild traumatic brain injury. *J Neurosurg Sci* 2006;50(4):83-88.
8. Biasca N, Maxwell WL. Minor traumatic brain injury in sports: A review in order to prevent neurological sequelae. P*rog Brain Res* 2007;161:263-291.
9. Bazarian JJ, Blyth B, Cimpello L. Bench to bedside: Evidence for brain injury after concussion — looking beyond the computed tomography scan. *Acad Emerg Med* 2006;13(2):199-214.
10. Haydel MJ, Preston CA, Mills TJ, et al. Indications for computed tomography in patients with minor head injury. *N Engl J Med* 2000;343(2):100-105.
11. Stiell IG, Wells GA, Vandemheen K, et al. The Canadian CT Head Rule for patients with minor head injury. *Lancet* 2001;357(9266):1391-1396.
12. Mower WR, Hoffman JR, Herbert M, et al. Developing a decision instrument to guide computed tomographic imaging of blunt head injury patients. *J Trauma* 2005;59(4):954-959.
13. Marx JA, Hockberger RS, Walls RM, et al. *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 7th ed. Philadelphia: Mosby/Elsevier; 2010.
14. Kay T, Harrington DE, Adams, R, et al. Definition of mild traumatic brain injury. *J Head Trauma Rehabil* 1993;8(3):86-87.
15. Pandor A, Goodacre S, Harnan S, et al. Diagnostic management strategies for adults and children with minor head injury: A systematic review and an economic evaluation. *Health Technol Assess* 2011;15(27):1-202.
16. Bainbridge J, Khirwadkar H, Hourihan MD. Vomiting — is this a good indication for CT head scans in patients with minor head injury? *Br J Radiol* 2012;85(1010):183-186.
17. Godbout BJ, Lee J, Newman DH, et al. Yield of head CT in the alcohol-intoxicated patient in the emergency department. *Emerg Radiol* 2011;18(5):381-384.
18. Jacobs B, Beems T, Stulemeijer M, et al. Outcome prediction in mild traumatic brain injury: Age and clinical variables are stronger predictors than CT abnormalities. J *Neurotrauma* 2010;27(4):655-668.
19. McCrory P, Meeuwisse W, Johnston K, et al. Consensus Statement on Concussion in Sport: The 3rd International Conference on Concussion in Sport held in Zurich, November 2008. *Br J Sports Med* 2009;43 Suppl 1:i76-90.
20. Iverson GL, Gaetz M, Lovell MR, et al. Cumulative effects of concussion in amateur athletes. *Brain Inj* 2004;18(5):433-443.
21. Hoffmann M, Lefering R, Rueger JM, et al. Pupil evaluation in addition to Glasgow Coma Scale components in prediction of traumatic brain injury and mortality. *Br J Surg* 2012;99 Suppl 1:122-130.
22. Compagnone C, d'Avella D, Servadei F, et al. Patients with moderate head injury: A prospective multicenter study of 315 patients. *Neurosurgery* 2009;64(4):690-696; discussion 696-697.
23. Lannsjo M, Backheden M, Johansson U, et al. Does head CT scan pathology predict outcome after mild traumatic brain injury? *Eur J Neurol* 2013;20(1):124-129.
24. Stein SC, Spettell C, Young G, et al. Limitations of neurological assessment in mild head injury. *Brain Inj* 1993;7(5):425-430.
25. Faux S, Sheedy J, Delaney R, et al. Emergency department prediction of post-concussive syndrome following mild traumatic brain injury — an international cross-validation study. *Brain Inj* 2011;25(1):14-22.
26. Bazarian J, Hartman M, Delahunta E. Minor head injury: Predicting follow-up after discharge from the emergency department. *Brain Inj* 2000;14(3):285-294.
27. Sheedy J, Harvey E, Faux S, et al. Emergency department assessment of mild traumatic brain injury and the prediction of postconcussive symptoms: A 3-month prospective study. *J Head Trauma Rehabil* 2009;24(5):333-343.
28. Tintinalli JE, Stapczynski JS. *Tintinalli's Emergency Medicine: A Comprehensive Study Guide*, 7th ed. New York: McGraw-Hill; 2011.
29. Register-Mihalik JK, Guskiewicz KM, Mihalik JP, et al. Reliable change, sensitivity, and specificity of a multidimensional concussion assessment battery: Implications for caution in clinical practice. *J Head Trauma Rehabil* Jun 9 2012.
30. Stein SC, Ross SE. The value of computed tomographic scans in patients with low-risk head injuries. *Neurosurgery* 1990;26(4):638-640.
31. Jennett B, Teasdale G. Aspects of coma after severe head injury. *Lancet* 1977;1(8017):878-881.
32. Harad FT, Kerstein MD. Inadequacy of bedside clinical indicators in identifying significant intracranial injury in trauma patients. *J Trauma* 1992;32(3):359-361; discussion 361-353.
33. Miller EC, Holmes JF, Derlet RW. Utilizing clinical factors to reduce head CT scan ordering for minor head trauma patients. *J Emerg Med* 1997;15(4):453-457.
34. Borczuk P. Predictors of intracranial injury in patients with mild head trauma. *Ann Emerg Med* 1995;25(6):731-736.
35. Jeret JS, Mandell M, Anziska B, et al. Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. *Neurosurgery* 1993;32(1):9-15; discussion 15-16.
36. Nagurney JT, Borczuk P, Thomas SH. Elder patients with closed head trauma: A comparison with nonelder patients. *Acad Emerg Med* 1998;5(7):678-684.
37. Jagoda AS, Bazarian JJ, Bruns JJ, Jr, et al. Clinical policy: Neuroimaging and decisionmaking in adult mild traumatic brain injury in the acute setting. *Ann Emerg Med* 2008;52(6):714-748.
38. Stiell IG, Clement CM, Rowe BH, et al. Comparison of the Canadian CT Head Rule and the New Orleans Criteria in

patients with minor head injury. *JAMA* 2005;294(12):1511-1518.

39. Smits M, Dippel DW, de Haan GG, et al. External validation of the Canadian CT Head Rule and the New Orleans Criteria for CT scanning in patients with minor head injury. *JAMA* 2005;294(12): 1519-1525.
40. Papa L, Stiell IG, Clement CM, et al. Performance of the Canadian CT Head Rule and the New Orleans Criteria for predicting any traumatic intracranial injury on computed tomography in a United States Level I trauma center. *Acad Emerg Med* 2012;19(1):2-10.
41. Ro YS, Shin SD, Holmes JF, et al. Comparison of clinical performance of cranial computed tomography rules in patients with minor head injury: A multicenter prospective study. *Acad Emerg Med* 2011;18(6):597-604.
42. Townend W, Dibble C, Abid K, et al. Rapid elimination of protein S-100B from serum after minor head trauma. *J Neurotrauma* 2006;23(2):149-155.
43. Bazarian JJ, Beck C, Blyth B, et al. Impact of creatine kinase correction on the predictive value of S-100B after mild traumatic brain injury. *Restor Neurol Neurosci* 2006;24(3):163-172.
44. Biberthaler P, Linsenmeier U, Pfeifer KJ, et al. Serum S-100B concentration provides additional information fot the indication of computed tomography in patients after minor head injury: A prospective multicenter study. *Shock* 2006;25(5):446-453.
45. Biberthaler P, Mussack T, Wiedemann E, et al. Rapid identification of high-risk patients after minor head trauma (MHT) by assessment of S-100B: Ascertainment of a cut-off level. *Eur J Med Res* 2002;7(4):164-170.
46. Biberthaler P, Mussack T, Wiedemann E, et al. Elevated serum levels of S-100B reflect the extent of brain injury in alcohol intoxicated patients after mild head trauma. *Shock* 2001;16(2):97-101.
47. Ingebrigtsen T, Romner B, Marup-Jensen S, et al. The clinical value of serum S-100 protein measurements in minor head injury: A Scandinavian multicentre study. *Brain Inj* 2000;14(12):1047-1055.
48. Muller K, Townend W, Biasca N, et al. S100B serum level predicts computed tomography findings after minor head injury. *J Trauma* 2007;62(6):1452-1456.
49. Mussack T, Biberthaler P, Kanz KG, et al. Immediate S-100B and neuron-specific enolase plasma measurements for rapid evaluation of primary brain damage in alcohol-intoxicated, minor head-injured patients. *Shock* 2002;18(5):395-400.
50. Poli-de-Figueiredo LF, Biberthaler P, Simao Filho C, et al. Measurement of S-100B for risk classification of victims sustaining minor head injury — first pilot study in Brazil. *Clinics* (Sao Paulo) 2006;61(1):41-46.
51. Anderson RE, Hansson LO, Nilsson O, et al. High serum S100B levels for trauma patients without head injuries. *Neurosurgery* 2001;48(6):1255-1258; discussion 1258-1260.
52. Levitt MA, Cook LA, Simon BC, et al. Biochemical markers of cerebral injury in patients with minor head trauma and ethanol intoxication. *Acad Emerg Med* 1995;2(8):675-680.
53. Defazio MV, Rammo RA, Robles JR, et al. The potential utility of blood-derived biochemical markers as indicators of early clinical trends following severe traumatic brain injury. *World Neurosurg* Jan. 8, 2013.
54. Muller B, Evangelopoulos DS, Bias K, et al. Can S-100B serum protein help to save cranial CT resources in a peripheral trauma centre? A study and consensus paper. *Emerg Med J* 2011;28(11): 938-940.
55. Unden J, Romner B. Can low serum levels of S100B predict normal CT findings after minor head injury in adults?: An evidence-based review and meta-analysis. *J Head Trauma Rehabil* 2010;25(4): 228-240.
56. Claudia C, Claudia R, Agostino O, et al. Minor head injury in warfarinized patients: Indicators of risk for intracranial hemorrhage. *J Trauma* 2011;70(4): 906-909.
57. Li J, Brown J, Levine M. Mild head injury, anticoagulants, and risk of intracranial injury. *Lancet* 2001;357(9258): 771-772.
58. Brewer ES, Reznikov B, Liberman RF, et al. Incidence and predictors of intracranial hemorrhage after minor head trauma in patients taking anticoagulant and antiplatelet medication. *J Trauma* 2011;70(1):E1-E5.
59. Pieracci FM, Eachempati SR, Shou J, et al. Degree of anticoagulation, but not warfarin use itself, predicts adverse outcomes after traumatic brain injury in elderly trauma patients. *J Trauma* 2007;63(3):525-530.
60. Franko J, Kish KJ, O'Connell BG, et al. Advanced age and preinjury warfarin anticoagulation increase the risk of mortality after head trauma. *J Trauma* 2006;61(1):107-110.
61. Ivascu FA, Howells GA, Junn FS, et al. Rapid warfarin reversal in anticoagulated patients with traumatic intracranial hemorrhage reduces hemorrhage progression and mortality. *J Trauma* 2005;59(5):1131-1137; discussion 1137-1139.
62. Gittleman AM, Ortiz AO, Keating DP, et al. Indications for CT in patients receiving anticoagulation after head trauma. *AJNR Am J Neuroradiol* 2005;26(3):603-606.
63. Mina AA, Bair HA, Howells GA, et al. Complications of preinjury warfarin use in the trauma patient. *J Trauma* 2003;54(5):842-847.
64. Garra G, Nashed AH, Capobianco L. Minor head trauma in anticoagulated patients. *Acad Emerg Med* 1999;6(2):121-124.
65. Major J, Reed MJ. A retrospective review of patients with head injury with coexistent anticoagulant and antiplatelet use admitted from a UK emergency department. *Emerg Med J* 2009;26(12): 871-876.
66. Jones K, Sharp C, Mangram AJ, et al. The effects of preinjury clopidogrel use on older trauma patients with head injuries. *Am J Surg* 2006;192(6):743-745.
67. Menditto VG, Lucci M, Polonara S, et al. Management of minor head injury in patients receiving oral anticoagulant therapy: A prospective study of a 24-hour observation protocol. *Ann Emerg Med* 2012;59(6):451-455.
68. Rendell S, Batchelor JS. An analysis of predictive markers for intracranial haemorrhage in warfarinised head injury patients. *Emerg Med J* 2013;30(1):28-31.
69. Servadei F, Teasdale G, Merry G. Defining acute mild head injury in adults: A proposal based on prognostic factors, diagnosis, and management. *J Neurotrauma* 2001;18(7):657-664.
70. National Collaborative Centre for Acute Care and National Institute for Health and Clinical Excellence. Head injury: triage, assessment, investigation, and early management of head injury in infants, children, and adults. 2007; http://www.nice.org.uk/nicemedia/live/11836/36257/36257.pdf. Accessed 3/19/2013, 2013.
71. Fortuna GR, Mueller EW, James LE, et al. The impact of preinjury antiplatelet and anticoagulant pharmacotherapy on outcomes in elderly patients with hemorrhagic brain injury. *Surgery* 2008;144(4):598-603; discussion 603-595.
72. Nishijima DK, Offerman SR, Ballard DW, et al. Immediate and delayed traumatic intracranial hemorrhage in patients with head trauma and preinjury warfarin or clopidogrel use. *Ann Emerg Med* 2012;59(6):460-468 e461-e467.
73. Cohen DB, Rinker C, Wilberger JE. Traumatic brain injury in anticoagulated patients. *J Trauma* 2006;60(3):553-557.
74. Itshayek E, Rosenthal G, Fraifeld S, et al. Delayed posttraumatic acute subdural hematoma in elderly patients on anticoagulation. *Neurosurgery* 2006;58(5):E851-E856; discussion E851-E856.
75. Reynolds FD, Dietz PA, Higgins D, et al. Time to deterioration of the elderly, anticoagulated, minor head injury patient who presents without evidence of neurologic abnormality. *J Trauma* 2003;54(3):492-496.
76. Vos PE, Battistin L, Birbamer G, et al. EFNS guideline on mild traumatic brain injury: Report of an EFNS task force. *Eur J Neurol* 2002;9(3):207-219.
77. Peck KA, Sise CB, Shackford SR, et al. Delayed intracranial hemorrhage after blunt trauma: Are patients on preinjury anticoagulants and prescription antiplatelet agents at risk? *J Trauma* 2011;71(6):1600-1604.

78. Li J. Admit all anticoagulated head-injured patients? A million dollars versus your dime. You make the call. *Ann Emerg Med* 2012;59(6):457-459.

79. Brown CV, Sowery L, Curry E, et al. Recombinant factor VIIa to correct coagulopathy in patients with traumatic brain injury presenting to outlying facilities before transfer to the regional trauma center. *Am Surg* 2012;78(1):57-60.

80. Goodnough LT, Shander A. How I treat warfarin-associated coagulopathy in patients with intracerebral hemorrhage. *Blood* 2011;117(23):6091-6099.

81. Ansell J, Hirsh J, Hylek E, et al. Pharmacology and management of the vitamin K antagonists: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest* 2008;133(6 Suppl):160S-198S.

82. Yasaka M, Sakata T, Naritomi H, et al. Optimal dose of prothrombin complex concentrate for acute reversal of oral anticoagulation. *Thromb Res* 2005;115(6):455-459.

83. Bartal C, Freedman J, Bowman K, et al. Coagulopathic patients with traumatic intracranial bleeding: Defining the role of recombinant factor VIIa. *J Trauma* 2007;63(4):725-732.

84. Stein DM, Dutton RP, Kramer ME, et al. Reversal of coagulopathy in critically ill patients with traumatic brain injury: Recombinant factor VIIa is more cost-effective than plasma. *J Trauma* 2009;66(1):63-72; discussion 73-65.

85. Downey DM, Monson B, Butler KL, et al. Does platelet administration affect mortality in elderly head-injured patients taking antiplatelet medications? *Am Surg* 2009;75(11):1100-1103.

86. Carroll LJ, Cassidy JD, Peloso PM, et al. Prognosis for mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *J Rehabil Med* 2004(43 Suppl):84-105.

87. Rees PM. Contemporary issues in mild traumatic brain injury. *Arch Phys Med Rehabil* 2003;84(12):1885-1894.

88. Chamelian L, Feinstein A. Outcome after mild to moderate traumatic brain injury: The role of dizziness. *Arch Phys Med Rehabil* 2004;85(10):1662-1666.

89. Reuben A, Sampson P, Harris AR, et al. Postconcussion syndrome (PCS) in the emergency department: Predicting and pre-empting persistent symptoms following a mild traumatic brain injury. *Emerg Med J* Mar. 6, 2013.

90. Advanced trauma life support for doctors ATLS: Manuals for coordinators and faculty. 8th ed. Chicago, IL: American College of Surgeons; 2008.

91. Lovell M. The neurophysiology and assessment of sports-related head injuries. *Neurol Clin* 2008;26(1):45-62; viii.

92. Collins MW, Lovell MR, Iverson GL, et al. Cumulative effects of concussion in high school athletes. *Neurosurgery* 2002;51(5):1175-1179; discussion 1180-1171.

93. Guskiewicz KM, Bruce SL, Cantu RC, et al. Research based recommendations on management of sport related concussion: Summary of the National Athletic Trainers' Association position statement. *Br J Sports Med* 2006;40(1):6-10.

94. Covassin T, Elbin R, 3rd, Stiller-Ostrowski JL. Current sport-related concussion teaching and clinical practices of sports medicine professionals. *J Athl Train* 2009;44(4):400-404.

95. Cantu RC, Register-Mihalik JK. Considerations for return-to-play and retirement decisions after concussion. *PM R* 2011;3(10 Suppl 2):S440-S444.

96. Schaider J, Barkin RM, Rosen P. *Rosen & Barkin's 5-minute Emergency Medicine Consult*, 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2011.

97. Nagy KK, Joseph KT, Krosner SM, et al. The utility of head computed tomography after minimal head injury. *J Trauma* 1999;46(2):268-270.

98. Dunham CM, Coates S, Cooper C. Compelling evidence for discretionary brain computed tomographic imaging in those patients with mild cognitive impairment after blunt trauma. *J Trauma* 1996;41(4):679-686.

99. af Geijerstam JL, Britton M. Mild head injury: Reliability of early computed tomographic findings in triage for admission. *Emerg Med J* 2005;22(2):103-107.

100. Bazarian JJ, McClung J, Cheng YT, et al. Emergency department management of mild traumatic brain injury in the USA. *Emerg Med J* 2005;22(7):473-477.

101. Fung M, Willer B, Moreland D, et al. A proposal for an evidenced-based emergency department discharge form for mild traumatic brain injury. *Brain Inj* 2006;20(9):889-894.

102. CDC. What to expect after a concussion. 2008. http://www.cdc.gov/concussion/pdf/TBI_Patient_Instructions-a.pdf. Accessed 3/11/2013.

CME Questions

1. Which of the following patients has an increased risk of intracranial injury?
   A. a 21-year-old male patient struck by a motor vehicle while crossing the street
   B. a 30-year-old female who fell from an elevation of one foot
   C. a 19-year-old male patient who fell down three stairs
   D. a 39-year-old male patient who was involved in a low-speed motor vehicle accident and did not strike his head

2. Deficits in which component of the GCS score have the strongest correlation with poor outcome in patients with TBI?
   A. eye opening
   B. motor response
   C. verbal response

Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

1. Read and study the activity, using the provided references for further research.

2. Log on to **www.cmecity.com** to take a post-test; tests can be taken after each issue or collectively at the end of the semester. *First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice, or renewal notice.*

3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.

4. After successfully completing the last test of the semester, your browser will be automatically directed to the activity evaluation form, which you will submit online.

5. **Once the completed evaluation is received, a credit letter will be e-mailed to you instantly.** You will no longer have to wait to receive your credit letter.

D. None of the components of the GCS score correlate with poor outcome in patients with TBI.

3. Based on the American College of Emergency Physicians clinical policy level A recommendations on CT scan criteria, which of the following patients should undergo non-contrast head CT following mild TBI?
   A. a 40-year-old female with a GCS score of 15
   B. a 32-year-old male patient with a normal neurologic examination and no focal neurologic deficits
   C. a 61-year-old male with headache and vomiting in the ED
   D. a 23-year-old female who remembers the events surrounding her injury and has no short-term memory loss

4. Which of the following patients may an emergency physician return to play the same day as sustaining a concussion?
   A. an 18-year-old female field hockey player who sustained a concussion when her fellow player accidentally struck her in the forehead with her field hockey stick; negative head CT scan result and minimal risk for developing an intracranial lesion
   B. a 21-year-old male college wide receiver who sustained a concussion without the loss of consciousness when he was tackled; negative head CT scan result, minimal risk for developing an intracranial lesion
   C. a 20-year-old female soccer player who sustained a concussion when she was kicked in the head during a soccer game; negative head CT scan result and minimal risk for developing an intracranial lesion
   D. Recent return to play guidelines no longer advocate return to play the same day of the traumatic event. The assessment for return to play is not a decision made by the emergency physician, but managed by primary health care providers and specialists with consideration of the severity of the injury, past injuries, and the possibility of future sports-related injuries.

5. A 66-year-old male presents after falling and hitting his head on the floor. He is on warfarin for atrial fibrillation. Which of the following statements is true?
   A. If his INR is normal, he can be discharged without imaging.
   B. He should have a head CT.
   C. He should have a CT and, if normal, have another CT 48 hours later.
   D. He should receive vitamin K and fresh frozen plasma to prevent bleeding.

6. A 16-year-old football player has a head injury while playing. He is initially dazed but "shakes it off." He presents the next day with dizziness, headache, and difficulty concentrating. CT is normal. Which statement is *false*?
   A. He has post-concussive syndrome.
   B. Since he did not lose consciousness, he cannot have a concussion.
   C. He should not resume sports at this time.
   D. His symptoms may last 30 days.

7. Your 16-year-old son is hit in the head during a basketball game and is briefly dazed and confused. After 15 minutes, he is fine. Which is the best course of action?
   A. He can return to play now that he is asymptomatic.
   B. He can return to play once the coach checks him out.
   C. He should see his primary care or sports physician before returning to play.
   D. He cannot return to play until next year.

8. Which of the following is true regarding a second concussion in a brief period of time?
   A. A second concussion can cause permanent cognitive damage.
   B. A second concussion can cause delayed bleeding.
   C. A second concussion carries no greater risk than the first.
   D. A second concussion is more likely to lead to post-concussive syndrome.

9. All of the following are indications for admission *except*:
   A. intracranial bleed on CT
   B. a patient on anticoagulants who is symptomatic
   C. persistent headache
   D. abnormally elevated INR

10. A 20-year-old athlete is brought in by the coach after a head injury. He appeared to be fine after the hit and he denies loss of consciousness. He denies headache and wants to get back into the game. He is not on any medications. On examination, he has a third nerve palsy on his right. Can he be discharged without a CT scan?
    A. yes
    B. no

Emergency Medicine Reports™ (ISSN 0746-2506) is published biweekly by AHC Media, a division of Thompson Media Group LLC, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Senior Vice President/Group Publisher: Donald R. Johnston

Executive Editor: Shelly Morrow Mark

Managing Editor: Leslie Hamlin

GST Registration No.: R128870672

Periodicals Postage Paid at Atlanta, GA 30304 and at additional mailing offices.

POSTMASTER: Send address changes to Emergency Medicine Reports, P.O. Box 105109, Atlanta, GA 30348.

Back issues: $31. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Multiple copy prices: One to nine additional copies, $359 each; 10 to 20 additional copies, $319 each.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail:
customerservice@ahcmedia.com

Editorial E-Mail:
shelly.mark@ahcmedia.com

World Wide Web page:
http://www.ahcmedia.com

Subscription Prices

1 year *with* 60 ACEP/65 AMA/39 AAFP Category 1/Prescribed credits: $544

1 year *without* credit: $399
Add $17.95 for shipping & handling

Resident's rate $199

Discounts are available for group subscriptions, multiple copies, site-licenses or electronic distribution. For pricing information, call Tria Kreutzer at 404-262-5482.

All prices U.S. only.
U.S. possessions and Canada, add $30 plus applicable GST. Other international orders, add $30.

Accreditation

AHC Media is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

AHC Media designates this enduring material for a maximum of *65 AMA PRA Category 1 Credits™*. Each issue has been designated for a maximum of 2.50 *AMA PRA Category 1 Credits™*. Physicians should claim only credit commensurate with the extent of their participation in the activity.

Approved by the American College of Emergency Physicians for a maximum of 65.00 hour(s) of ACEP Category I credit.

This Enduring Material activity, *Emergency Medicine Reports*, has been reviewed and is acceptable for up to 39 Prescribed credit(s) by the American Academy of Family Physicians. AAFP accreditation begins January 1, 2013. Term of approval is for one year from this date with the option of yearly renewal. Each issue is approved for 1.50 Prescribed credits. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Please forward your comments on the quality of this activity to cmecomment@aafp.org.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

This CME activity is intended for emergency and family physicians. It is in effect for 24 months from the date of the publication.

AHC Media

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Minor Trauma Management: Part I

Glasgow Coma Scale Scoring

Component		Score
Best Eye Opening	Spontaneous	4
	To verbal stimuli	3
	To painful stimuli	2
	No eye opening	1
Best Verbal Response	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible	2
	No verbal response	1
Best Motor Response	Obeys commands	6
	Localizes pain	5
	Withdraws to pain	4
	Flexion to pain	3
	Extension to pain	2
	No motor response	1
	Total	__/15

Signs and Symptoms Associated with Mild Traumatic Brain Injury

Observed Signs	Physical Symptoms	Cognitive Symptoms	Emotional Symptoms
Appears dazed or stunned	Headache or "pressure" in head	Difficulty thinking clearly	Irritable
Is confused about events	Nausea or vomiting	Difficulty concentrating	Depressed
Repeats questions	Balance problems or dizziness	Difficulty remembering	Anxious
Answers to questions slowly	Fatigue or feeling tired	Feeling sluggish, hazy, foggy, or groggy	Emotional lability
Cannot remember events prior to the traumatic event	Blurry or double vision	Orientation problems	Loss of initiative
Cannot remember events after the traumatic event	Sensitivity to light	Slow reaction time	Problems at work, home, or school
Loss of consciousness	Sensitivity to noise	Calculation difficulties and problems with executive functioning	
Shows behavior or personality changes	Numbness or tingling		
	Insomnia		
	Seizures		

ACEP Clinical Policy, New Orleans, and Canadian CT Clinical Decision Rules

ACEP Clinical Policy on CT Scan Criteria for Mild TBI	New Orleans Criteria*	Canadian CT Head Rule**
Headache	Headache	Failure to reach a GCS of 15 within 2 hours of injury
Vomiting	Vomiting	Suspected open skull fracture
Age greater than 60 years	Age greater than 60 years	Signs of basal skull fracture
Drug or alcohol intoxication	Intoxication	Vomiting more than once
Deficits in short-term memory	Deficits in short-term memory	Age greater than 64 years
Physical evidence of trauma above the clavicle	Physical evidence of trauma above the clavicle	Amnesia before impact of greater than 30 minutes
Post-traumatic seizures	Seizure	Dangerous mechanism of injury***
GCS score less than 15		
Focal neurological deficit		
Coagulopathy		

GCS = Glasgow Coma Scale
ACEP = American College of Emergency Physicians
*Patients must have a GCS of 15
**Patients may have an initial GCS of 13-15
***A dangerous mechanism was defined as pedestrian struck by a motor vehicle, occupant ejected from a motor vehicle, and fall from a height of greater than 3 feet or 5 stairs.

Supplement to *Emergency Medicine Reports*, May 19, 2013: "Minor Trauma Management: Part I."
Authors: **Justin L. Weppner, DO,** Lieutenant, Medical Corps, USN, Naval Medical Center Portsmouth, Portsmouth, VA; and **James A. McCombs, DO**, Lieutenant, Medical Corps, USN, Naval Medical Center Portsmouth, Portsmouth, VA.