CLINICAL PRACTICE

Concussion

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This Journal *feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the authors' clinical recommendations.*

A 64-year-old woman slipped on an icy walk, falling forward and striking her forehead. She had a brief convulsion immediately after the fall, was unresponsive for less than 1 minute, and awakened with a severe generalized headache and nausea but no vomiting. In addition to being perplexed about the circumstances of the fall, she could not recall the previous few hours. She was awake and oriented and had no abnormalities on neurologic examination. Tenderness and a scalp contusion were apparent at the site of the impact, and there were abrasions on her right cheek. What is the expected course, and how should her case be managed?

THE CLINICAL PROBLEM

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"Concussion" refers to an immediate and transient loss of consciousness accompanied by a brief period of amnesia after a blow to the head. This event is so common, affecting about 50 people per 100,000 population in the United States yearly,[1] that almost all physicians are called on at some time to provide care at the scene or to treat the sequelae of concussion. The clinical status of the momentary sensation of being "starstruck," or dazed, after head injury without a brief period of loss of consciousness is uncertain, but it is generally considered the mildest form of concussion.[2] Young children have the highest rates of concussion. Sports and bicycle accidents account for the majority of cases among 5- to 14-year-olds, whereas falls and vehicular accidents are the most common causes of concussion in adults.

Considerable confusion persists among physicians and the public regarding concussion and the postconcussion syndrome. The extent of concussive amnesia roughly correlates with the duration of loss of consciousness and the severity of the head injury. There is both anterograde amnesia (the inability to retain new information) and retrograde amnesia, with the latter encompassing the moments before injury or, in rare cases, extending backward for several days or longer.[3] In exceptional cases, a very minor blow to the head causes a memory disturbance that lasts for several subsequent hours.[4] The period of anterograde memory loss tends to be briefer than the period of retrograde memory loss, and both improve over a period of hours or in less time.[5] Concussion does not cause a loss of autobiographical information, such as one's name and birth date; this type of memory loss is a symptom of hysteria or malingering. Patients with concussion-related amnesia do not confabulate, and in many ways the clinical state resembles transient global amnesia.

A single brief convulsion may occur immediately after an otherwise mundane concussion, often leading to the erroneous attribution of the problem to a seizure.[6,7] The mechanism is not known, but a brief convulsion neither presages epilepsy nor requires the administration of anticonvulsant medication. Concussion of a more serious grade may be followed by a confusional or delirious state or a period of sleep.

The brief loss of consciousness that characterizes concussion appears to be the result of rotational forces exerted at the junction of the upper midbrain and thalamus that cause transient disruption of the functioning of the reticular neurons that maintain alertness (Fig. 1). Other mechanisms, such as seizure or abrupt elevation of intracranial pressure, have been suggested but with limited supporting evidence. The cause of the accompanying amnesia, however, is not known.

STRATEGIES AND EVIDENCE

EVALUATION OF THE PATIENT WITH CONCUSSION

Those attending to the patient at the scene of the injury should keep the airway clear and be attentive to a possible accompanying neck injury. If the patient has any neck pain, cervical immobilization is required. It is prudent to insist that a patient with concussion be taken to an emergency department for evaluation, although many patients object.

Criteria for Cranial Imaging

The primary concern is that the force of the impact may have caused a subdural, epidural, or parenchymal brain hematoma, although less than 10% of patients have intracranial bleeding after a concussion, and less than 2% require neurosurgery.[8] Cranial computed tomography (CT) without contrast enhancement is adequate to detect important intracranial bleeding; magnetic resonance imaging is not necessary for this purpose.

Neurologic signs such as hemiparesis or poor arousal certainly necessitate CT, but predicting which person is likely to have abnormalities on imaging, and at the same time avoiding unnecessary scans, has proved difficult. Evidence that the injury was minor, including a normal neurologic examination, does not ensure the absence of an intracranial lesion. For example, 209 of 1538 patients with normal neurologic examinations in one series were found to have abnormalities on CT, with 58 requiring neurosurgery.[9] On the basis of such findings, some groups have advocated scanning all patients with concussion, an approach that would lead to a predominance of negative scans that did not need to be obtained. In the past, the presence of a skull fracture was considered a marker of intracranial injury,[10] but it has since been recognized that a simple fracture actually dissipates much of the energy of an impact and is not a strong indicator of intracranial bleeding. Certain fractures, such as those that are depressed or involve the base of the skull, have some predictive value, as discussed below.[11,12]

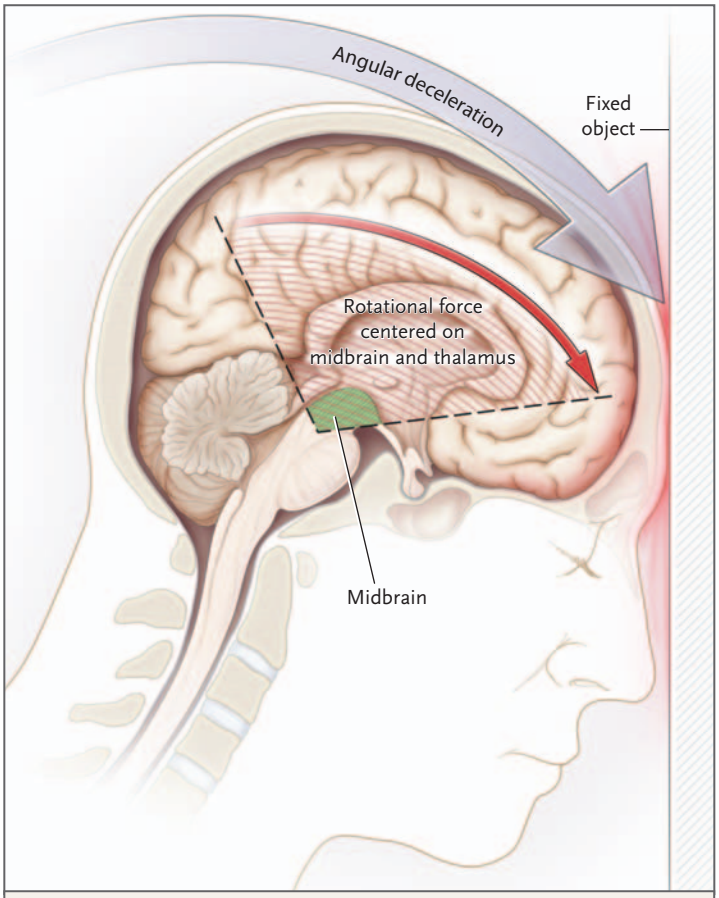

Figure 1. Mechanism of Concussion.

Biomechanical investigations dating back to the beginning of the 20th century suggest that concussion results from a rotational motion of the cerebral hemispheres in the anterior–posterior plane, around the fulcrum of the fixed-in-place upper brain stem. If the neck is restrained, concussion is difficult to produce. Concussions as portrayed in movies and cartoons, in which the back of the head is struck with a blunt object and no motion is transferred to the brain, are implausible. The modern view is that there is disruption of the electrophysiological and subcellular activities of the neurons of the reticular activating system that are situated in the midbrain and diencephalic region, where the maximal rotational forces are exerted. Alternative mechanisms for concussive loss of consciousness, such as self-limited cortical seizures or a sudden increase in intracranial pressure, have also been proposed, but with limited supporting evidence.

To refine the criteria for CT scanning, several clinical decision rules have been developed. Two of these rules — the New Orleans Criteria[13] and the Canadian CT Head Rule[14] — have been validated prospectively. Each contains seven criteria, although the only ones they have in common are older age and vomiting (albeit defined slightly

differently) (Table 1). In two large prospective studies of minor head injury,[15,16] the presence of any of the clinical features in these rules identified essentially all patients requiring immediate neurosurgical intervention. In one of these studies,[15] the Canadian rule had slightly lower sensitivity than the New Orleans rule for any "important" injury. However, both rules had low specificity, although it was slightly higher for the Canadian rule, whose use was thus projected to result in modest but greater reductions in CT than use of the New Orleans rule (Table 1). Since patients 15 years and younger were excluded from one of the original studies[14] and from both validation studies, the applicability of the rules to this age group is uncertain.

Table 1. The New Orleans and Canadian Clinical Decision Rules for CT after Concussion.*

New Orleans Criteria† — Glasgow Coma Scale score of 15
Headache
Vomiting
Age >60 yr
Drug or alcohol intoxication
Persistent anterograde amnesia (deficits in short-term memory)
Evidence of traumatic soft-tissue or bone injury above clavicles
Seizure
Canadian CT Head Rule‡ — Glasgow Coma Scale score of 13–15 for patients 16 years and older
High risk of neurosurgical intervention
Glasgow Coma Scale score <15 within 2 hr after injury
Suspected open or depressed skull fracture
Any sign of basal skull fracture§
Two or more episodes of vomiting
Age >65 yr
Moderate risk of brain injury detected by CT
Retrograde amnesia for ≥30 min
Dangerous mechanism¶

* The presence of at least one criterion from the New Orleans Criteria or the Canadian CT Head Rule is considered an indication for a cranial CT scan. A score on the Glasgow Coma Scale of 15 signifies a fully alert and oriented patient, spontaneously conversing and following commands. In the context of concussion, a score of 13 or 14 denotes disorientation or less than full alertness. The Canadian CT Head Rule is adapted from Stiell et al.[14] with the permission of the publisher.

† The sensitivity and specificity of the New Orleans Criteria are 99% and 5%, respectively, for detecting any lesion or clinically important CT abnormality; the sensitivity and specificity for detecting lesions requiring neurosurgery are 100% and 5%, respectively. Clinically important lesions not requiring immediate surgery include contusions; subarachnoid blood; small subdural, parenchymal, and intraventricular hematomas; and certain skull fractures. Data are from Haydel et al.,[13] Stiell et al.,[14] and Smits et al.[15]

‡ The sensitivity and specificity of the Canadian CT Head Rule criteria for detecting clinically important CT lesions are 87% and 39%, respectively; the sensitivity and specificity for detecting lesions requiring neurosurgery are 100% and 38%, respectively. Adapted from Stiell et al.,[14] Smits et al.,[15] and Stiell et al.[16]

§ Signs of basal skull fracture include hemotympanum, raccoon eyes, otorrhea or rhinorrhea, and Battle's sign (mastoid ecchymosis).

¶ A "dangerous mechanism" refers to a motor vehicle that strikes a pedestrian, ejection from a motor vehicle, or a fall from an elevation of 3 ft (about 1 m) or more or five or more stairs.

It is important to appreciate that no clinically based rule for obtaining a CT scan is likely to be universally accurate. In using these rules, the physician should consider the level of acceptable risk for overlooking any intracranial lesion as opposed to missing lesions that require monitoring or immediate neurosurgical intervention. With these limitations in mind, for patients who are 16 to 65 years old and have no postconcussive symptoms except mild headache, no external signs of injury or basilar skull fracture, and a normal neurologic examination, the frequency of intracranial clots that require neurosurgery is so low (<1%) that it is reasonable to forgo CT scanning. Imaging is routinely recommended for children younger than 16 years, for intoxicated patients in whom the manifestations of cerebral injury are easily obscured, for patients who cannot be dependably observed after discharge, and for patients who take anticoagulants or have other bleeding tendencies.

Observation after Concussion

The duration and setting of monitoring depend in part on the periods of unconsciousness and amnesia and the presence or absence of systemic injuries. Patients with a normal neurologic examination are generally observed for approximately 2 hours and safely discharged to the care of a responsible person.[16-18] It is helpful to provide a written instruction sheet with a list of symptoms that should prompt a return to the hospital, such as increasing headache, repeated vomiting, weakness, clumsiness, drowsiness, or fluid from the nose or ear that might represent a leak of cerebrospinal fluid. Headache and irritability are common for a day or more after concussion, particularly in children, and sometimes do not appear for several hours.[19] Whether it is necessary to awaken a patient at night to confirm that he or she can be aroused has not been established; if this is a concern, hospitalization is more reasonable. It is generally recommended that patients not resume normal activity until they are free of headache and dizziness, but there are no data to indicate that an earlier return may be harmful.

New drowsiness, hemiplegia, or aphasia after concussion is cause for concern about the possibility of a delayed subdural or epidural hematoma

and warrants examination and imaging studies. If these focal signs are not due to intracerebral bleeding, the possibility of a stroke from inevident carotid artery dissection should not be overlooked. When imaging studies of the brain and major cervical and cerebral vessels show no abnormalities, a migrainelike phenomenon is presumed to be responsible for focal neurologic features.[20]

Certain CT findings influence the duration of observation and the need for hospitalization. Small surface contusions of the brain or limited amounts of subarachnoid hemorrhage occur in approximately 5% of cases. These injuries usually do not give rise to neurologic problems other than headache, but they indicate that the impact was severe and call for more prolonged observation, generally overnight in the hospital. A fracture through the groove of the middle meningeal artery represents a special risk for epidural hematoma. Follow-up imaging is reasonable for all of these intracranial lesions, although its value and optimal timing have not been studied.

Table 2. *International Classification of Diseases, 10th Revision*, Criteria for Postconcussion Syndrome (Code 310-2).

Interval between head trauma with loss of consciousness and development of symptoms, ≤4 wk
Symptoms in at least three of the following categories:
Headache, dizziness, fatigue, noise intolerance
Irritability, depression, anxiety, emotional lability
Subjective concentration, memory, or intellectual difficulties without neuropsychological evidence of marked impairment
Insomnia
Reduced alcohol tolerance
Preoccupation with above symptoms and fear of brain damage, with hypochondriacal concern and adoption of sick role

POSTCONCUSSION SYNDROME (POSTTRAUMATIC NERVOUS INSTABILITY)

Postconcussion syndrome consists of a constellation of sometimes disabling symptoms, mainly headache, dizziness, and trouble concentrating, in the days and weeks following concussion (Table 2).[21,22] The frequency and natural history of the disorder are unclear. In case series, incidences of headache and dizziness have been as high as 90% at 1 month and approximately 25% at 1 year or more, and the incidence of memory difficulty has ranged from 4 to 59% at various times.[23] Once established for more than a few weeks, symptoms often persist for months and tend to resist treatment, although they eventually lessen. Unresolved issues of compensation and litigation have been associated with persistent symptoms.[24,25] Countries in which litigation after accidents is infrequent have extremely low rates of postconcussive disability,[26] and the problem is almost unknown in young children. Nonetheless, difficulty concentrating certainly occurs in the absence of these complicating features and can be demonstrated with neuropsychological tests, in some cases for months after concussion.

Anxiety and depression are reported by more than a third of patients with persistent postconcussive symptoms, but it has been difficult to establish whether these traits preceded the injury.[27,28] Postconcussive symptoms are more common among patients who express a preoccupation with brain damage or who have a marked intensification of symptoms after mental or physical effort.

Imbalance and disequilibrium reflect vestibular damage ("vestibular concussion").[29] Affected patients have vertigo or experience motion of the environment while walking or driving. The vestibular damage is evident in an abnormal vestibulo-ocular reflex, which is tested by rapidly turning the patient's head several degrees to one side while the patient focuses on a fixed point and observing the patient for a slippage of fixation.

Data from controlled trials are lacking to guide treatment of the postconcussion syndrome. However, reassurance and education about the effects of concussion in an early single encounter have been shown to reduce the incidence and duration of symptoms at 6 months.[30] Clinical experience suggests a benefit from the use of mild analgesics for headache, avoidance of narcotics, and the use of meclizine, promethazine (Phenergan), and vestibular exercises for dizziness, although these treatments have not been carefully studied in the postconcussion syndrome. Antidepressants are used in practice for patients with protracted symptoms of worry, sleeplessness, poor concentration, and daily headache, but data on the effectiveness of antidepressants in these cases are also limited; one small study has failed to demonstrate a benefit of antidepressants for headache after head injury.[31] In patients with a history of migraine, concussion may trigger prolonged headaches. Clinical experience supports using therapies for spontaneously occurring migraine (triptans, anticonvulsants, calcium-channel or beta-adrenergic blockers, or corticosteroids).[32]

CONCUSSION DURING ATHLETICS

Athletes who have had a concussion have a slightly higher incidence of subsequent concussions during the same playing season.[33] Extensive testing of several hundred amateur and professional athletes after a single concussion has demonstrated a return to baseline cognitive and motor performance within several weeks.[34,35] Several studies of collegiate rugby and soccer players have shown decreased scores on selected neuropsychological tests in proportion to the number of self-reported head injuries,[36] but other studies, including a recent one of Australian football players,[37] indicate no such association. The occurrence of cognitive decline after repeated concussion has been more clearly demonstrated in boxers who have sustained multiple knockouts. There has also been concern that a second impact within a short period could lead to catastrophic neurologic damage. This largely unfounded concern is based on rare and mostly disputed cases in which a second mild head injury in children caused massive cerebral edema.[38,39]

There are scant data to guide decisions about the timing of a return to sports after concussion.[40-42] Evaluation at the sidelines generally includes tests of mental function and coordination and provocation of symptoms such as headache, dizziness, or unsteadiness with exertion (Table 3). Recommendations are generally conservative, reflecting concern about an increased risk of a second concussion.

AREAS OF UNCERTAINTY

The cerebral lesions associated with severe traumatic brain injury certainly cause cognitive and personality changes, but the effects of concussion are less clear.[43] A recent concern has been the possibility of persistent cognitive decline as a result of single or repeated concussions, even from nonconcussive "heading" of the ball in soccer, termed "microtrauma" or "subconcussive" injury. The magnitude of the risk of microtrauma, in particular, if it exists, has been difficult to ascertain because few studies include measures of performance before the injury and control groups have been of uncertain comparability. Better-designed studies have not demonstrated a loss of intellectual function. The appropriate duration of abstinence from sports after concussion has not been determined. In addition, the causes and optimal management of postconcussive symptoms remain unclear.

GUIDELINES

The American Academy of Neurology,[44] the Canadian Academy of Sport Medicine,[45] and several

Table 3. Guidelines for the Management of Sport-Related Concussion.*

Symptoms	First Concussion	Second Concussion
Grade 1: no loss of consciousness, transient confusion, resolution of symptoms and mental abnormalities in <15 min†	Remove from play Examine at 5-min intervals May return to play if symptoms disappear and results of mental-function examination return to normal within 15 min	Allow return to play after 1 wk if there are no symptoms at rest or with exertion
Grade 2: as above, but with mental symptoms for >15 min	Remove from play and disallow play for rest of day Examine for signs of intracranial lesion at sidelines and obtain further examination by a trained person on same day Allow return to play after 1 wk if neurologic examination is normal	Allow return to play after 2-wk period of no symptoms at rest or with exertion Remove from play for season if imaging shows abnormality
Grade 3: any loss of consciousness	Perform thorough neurologic examination in hospital and obtain imaging studies when indicated Assess neurologic status daily until postconcussive symptoms resolve or stabilize Remove from play for 1 wk if loss of consciousness lasts seconds; for 2 wk if it lasts minutes; must be asymptomatic at rest and with exertion to return to play	Withhold from play until symptoms have been absent for at least 1 mo

* These guidelines reflect consensus opinion, are not evidence-based, and are under revision. Adapted from the American Academy of Neurology guidelines.[44]

† Testing includes orientation, repetition of digit strings, recall of word list at 0 and 5 minutes, recall of recent game events, recall of current events, pupillary symmetry, finger-to-nose and tandem-gait tests, Romberg's test, and provocative testing for symptoms with a 4-yd (3.5-m) sprint, five push-ups, five sit-ups, and five knee bends.

international symposiums[41] have developed recommendations for the evaluation and management of concussion in athletes. These guidelines reflect expert opinion in the absence of data, and there is no consensus on which set of guidelines or grading system is most appropriate. The recommendations of the American Academy of Neurology (Table 3) are under revision but do provide one approach to making sideline decisions.

SUMMARY AND RECOMMENDATIONS

The patient in the vignette had a concussion complicated by an impact-related seizure but had a normal neurologic examination. Because she was over the age of 60 and had facial and scalp bruises, as well as prolonged retrograde amnesia, it would be prudent to obtain a cranial CT scan, according the New Orleans and Canadian rules. With a normal examination and scan, she could be safely discharged to the care of a trusted person who was given written instructions to check on the patient several times over the next 24 hours and to return if drowsiness, vomiting, confusion, weakness, or increased headache occurs. There is no indication for an anticonvulsant, but non-narcotic analgesics may be given. The common sequelae of concussion should be reviewed with the patient, including the possibility that headache, dizziness, and mild difficulty concentrating may persist for days or weeks. A temporary leave from work or change to less taxing assignments may be appropriate, and if there is litigation, its prompt resolution should be encouraged. In the absence of controlled trials to guide postconcussion management, it is reasonable to treat persistent headaches and dizziness with medications and nonpharmacologic strategies commonly used for these symptoms. If impaired concentration persists for several weeks, neuropsychological testing should be considered to document and monitor the deficit.

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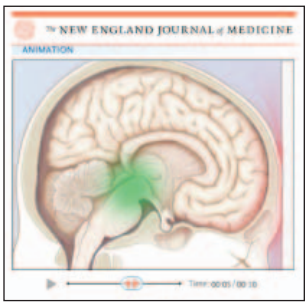

A video showing the head movement causing concussion is available with the full text of this article at www.nejm.org.

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