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Managing mild TBI in adults

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INSHOT / ISTOCK

ABOUT 90% OF THE 2 MILLION annual reported brain injuries in the United States are diagnosed as mild traumatic brain injury (mTBI).[1] However, because most mTBI cases aren't reported to hospitals, the true incidence isn't known.[2]

The Brain Injury Association of America states that mTBI can be caused by direct blows to the head, gunshot wounds, violent shaking of the head, or force from a whiplash-type injury. Both closed and open head injuries can produce mTBI.[3] Although the terms *concussion* and *mTBI* are often used interchangeably, for clarity, this review will use the term mTBI exclusively.[3,4]

mTBI has been referred to as a "silent epidemic" because the problems experienced after injury are often unnoticed or untreated, yet they can have profound consequences to patients' long-term physical, mental, social, and occupational well-being.[1] This article discusses the pathophysiology and diagnosis of mTBI, nursing assessment, and patient education for patients with mTBI.

Mechanisms of TBI

A traumatic brain injury (TBI) is a brain injury resulting from direct blunt force trauma to the head or an acceleration/deceleration force, as from whiplash or a blast, that injures the brain with or without direct external head trauma.[5,6] As discussed in detail below, mTBI is described as a less severe type of TBI on initial

diagnosis, characterized generally by transient signs and symptoms that typically improve rapidly in 2 weeks and resolve fully within 90 days, although a small number of patients may continue to have symptoms for a year or more.[1,7]

Although the vast majority of patients with mTBI survive and recover, a subset will develop continued neurologic dysfunction and die from progression of their primary injury. Risk factors for death associated with mTBI include severity of primary injury, presence of extracranial injuries, and, importantly, use of anticoagulants.[6,8]

Some of the primary effects of mTBI are immediate and manifest within hours of the injury, although peak signs and symptoms may not occur from hours to days postinjury.[9] Cognitive impairments are common, particularly in the domains of visual-motor reaction time, information processing, memory, and attention.[10]

Although most patients who experience a single mTBI recover fully, others develop a lengthy postconcussion syndrome or other long-term complications that can be disabling.[10,11] For example, patients with a history of repetitive subconcussive head impacts can experience cumulative damage that can prolong their recovery or develop into a chronic condition. Consequently, nurses must inform patients that even a "mild" TBI must be taken seriously and educate them about the importance of following treatment recommendations to prevent further injury and ensure a complete recovery.

No two TBIs are alike

The physical properties of brain tissue within the cranial vault are critical determinants of how the brain moves and deforms during impact. These properties account for the variability and extent of injury among individuals and explain why no two TBIs are alike.[12-14]

Although the brain is one of the largest consumers of oxygen in the human body, it can't store oxygen, making it particularly sensitive to any reduction or interruption of oxygen delivery.[15] In addition, as brain size decreases with age, the subdural space enlarges. This may increase the risk of all TBI classifications in older adults.[16]

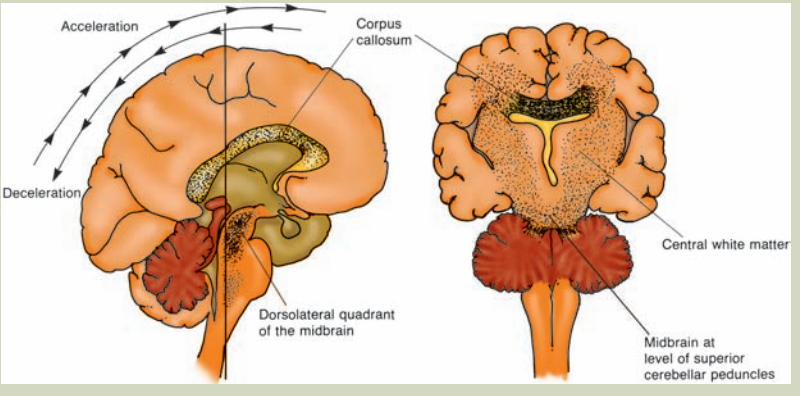
Combined forces of acceleration and deceleration from the force of a blow to the head can lead to a trauma-induced metabolic mismatch: an increased demand for substrates of metabolism including oxygen, simultaneously accompanied with decreased blood flow and oxygen delivery. The metabolic mismatch causes an energy deficit that results in variable signs and symptoms, most commonly headache, dizziness, sleep disturbance, cognitive problems, and emotional difficulties, but can also lead to loss of consciousness and posttraumatic amnesia.[14] The signs and symptoms associated with mTBI result from alteration of brain function rather than from gross structural damage.

Cerebral blood vessels are just as susceptible to the shear-strain biomechanics of head injury as are neurons. Each neuron depends on receiving a continuous source of glucose and oxygen; the smallest capillaries are large enough for just a single red blood cell to deliver oxygen and glucose. Vascular damage can lead to neural dysregulation by altering the hemodynamic response necessary for normal cellular function.[9]

As previously mentioned, a direct blow to the head isn't the only mechanism that can produce a TBI. Other causes include blast forces such as occur in battle or wars that cause the brain to accelerate, rotate, and decelerate. Different areas of the brain move at different speeds, resulting in contusions and shear strain to susceptible brain tissues.[13,14] Signs and symptoms reflect the severity and extent of contusions and shear strain.[14]

Once injured, brain tissue requires even more oxygen to survive and recover. The injury alters brain

Mechanical forces leading to DAI[1,6,40]



As the name suggests, diffuse brain injuries aren't localized to one area of the brain but are distributed throughout the brain. DAI is diagnosed by the appearance of axonal injury at the microscopic level in selected regions of the brain as seen on magnetic resonance imaging.[11,12] DAI is caused by the acceleration and deceleration of three kinds of mechanical forces that result in strain and shearing of the axons in the brain:

- Linear damage occurs as a result of forces that make the head move in an anterior-posterior direction, as when hitting the front or back of the head.
- Rotational acceleration occurs as a result of forces that make the head move sideways, such as in a punch to one side of the head or the force of a blast from an explosion.
- Impact deceleration occurs when the head forcefully decelerates, as when the head hits the ground.[9]

metabolism, leaving neurons highly susceptible to damage from hypoxia, free radicals, and mitochondrial dysfunction. This is the start of a pathologic process that can sometimes take years to resolve.[15]

TBIs of all degrees are highly variable. Two injuries that superficially appear very similar in terms of mechanism of injury and the distribution and severity of damage can be associated with very different outcomes. While the specific pattern of TBI varies, outcome is related to age at time of injury and initial injury severity: Younger patients with less severe injuries generally have the best recovery. More severe or repeated TBIs are associated with greater risk of long-term neurodegenerative disorders, such as Alzheimer disease, chronic traumatic encephalopathy (CTE), and Parkinson disease.[12]

Diffuse axonal injuries (DAI) are considered the most common diffuse brain injury seen in severe TBI. However, DAI can also trigger many of the behavioral consequences of mTBI.[17] (See *Mechanical forces leading to DAI*.)

Diagnosing mTBI: A question of severity

Following any suspected or known TBI, initial neurologic signs and symptoms are typically assessed based on Glasgow Coma Scale (GCS) scores, considered the gold standard in the assessment of initial TBI severity. GCS scoring performed within 24 hours of injury and serially helps clinicians determine the extent of TBI and monitor subsequent improvement or deterioration. (See *GCS scoring*.)

Patients are given a total GCS score ranging from 3 to 15 based on degree of impairment in three domains (eye opening, verbal response, and motor response), with lower scores indicating greater impairment. Scores from 13 to 15 represent mild brain injuries consistent with mTBI. Scores from 9 to 12 represent moderate injuries, and scores of 8 or less represent severe TBI.[15,18]

The American Congress of Rehabilitation Medicine was the first to establish diagnostic criteria for mTBI, describing it as a traumatic disruption of brain function manifested by at least one of the following:[5]

- loss of consciousness for any period
- loss of memory for events occurring immediately before or after the injury
- any alteration of mental state at the time of injury, such as disorientation or confusion
- focal neurologic deficits, which may or may not be transient.

To meet criteria for mTBI, most experts specify loss of consciousness (if present) lasting no more than 30 minutes, an initial GCS score of 13 to 15, and posttraumatic amnesia (and/or other transient neurologic signs and symptoms) persisting for no longer than 24 hours.[1,18]

mTBI can be classified as *complicated* (with radiographic evidence of intracranial injury) or *uncomplicated* (without radiographic evidence of injury). Such a distinction is important because it helps clinicians determine the need for further neuroimaging, hospital admission, and/or neurosurgical intervention.[15]

Based on current data, patients younger than age 55 with an admission GCS of 15 who aren't taking any type of anticoagulant medication comprise a low-risk group that could safely be considered for non-ICU admission with frequent neurochecks. Patients admitted with a GCS less than 15 and those older than age 55 are at risk for significant neurologic decline and should be treated in an ICU.[19]

mTBI is sometimes described as a diagnosis of exclusion because clinicians must systematically rule out other potential causes for signs and symptoms. Besides taking a careful history of the precipitating event, clinicians must document the course and nature of signs and symptoms and conduct appropriate objective testing to identify alternative explanations, such as a preexisting or coexisting medical or psychiatric disorder.[20]

GCS scoring

Eye opening	
Spontaneous	4
To verbal stimuli	3
To pain	2
No response	1
Verbal response	
Oriented	5
Confused but can answer questions	4
Inappropriate words	3
Incomprehensible speech	2
No response	1
Motor response	
Obeys commands	6
Purposeful movement to pain	5
Withdraws from pain	4
Flexion (decorticate posturing) to pain	3
Extension (decerebrate posturing) to pain	2
No response	1

Severe head injury—GCS score of 8 or less
Moderate head injury—GCS score of 9 to 12
Mild head injury—GCS score of 13 to 15

Source: Centers for Disease Control and Prevention. Glasgow Coma Scale. www.cdc.gov/masstrauma/resources/gcs.pdf.

Potential long-term consequences of mTBI

Although improvement in mTBI signs and symptoms typically occurs very rapidly—usually within 2 weeks, with full recovery expected by 90 days—some 10% to 15% of patients remain symptomatic for much longer.[18] Persistent consequences include the following.

- ***Postconcussion syndrome (PCS)***. PCS is defined as the persistence of symptoms such as poor concentration, dizziness, fatigue, headache, sleep disturbance, irritability, anxiety, and depressed mood, lasting beyond the typical 90-day recovery period.[1,7] Up to one-third of patients with mTBI experience PCS.[7] Mild aerobic exercise below the threshold of symptoms may speed recovery from PCS, even in those who didn't exercise before the injury.[14] The patients who may not recover within 3 months and experience PCS include those who sustained

a high-impact mechanism of injury, those with multiple concussions, those with underlying neurologic conditions, and those who don't recover with appropriate treatments.[14]

• ***CTE.*** Convincing evidence suggests that repeated mTBI may be associated with changes in mood, cognition, and motor coordination persisting for months to years and can develop into CTE.[7] This progressive neurodegenerative disorder is associated with long-term neuropsychiatric symptoms, behavioral changes, and cognitive deficits.[7,18,21] A study by Viano et al showed that on average in the typical sports-related concussion, the brain displaces between 4 and 8 mm, causing shear-strain to neurons and their associated axons.[9]

• ***Second-impact syndrome***. Patients may develop second-impact syndrome when they sustain a second head impact before symptoms from the first impact have resolved. Another injury during this period could be fatal.[18,22] Within minutes of a second impact, brain herniation can occur, followed by coma and death. Morbidity for second-impact syndrome is 100%; mortality is 50%.[23]

Patients with mTBI need to understand that the brain is highly vulnerable to new injuries during the recovery phase, termed the *window of vulnerability.* Even one additional sub-concussive head impact in this phase can lead to potentially lethal complications, including cerebral hyperemia due to impaired vascular autoregulation, cerebral edema, and increased intracranial pressure (ICP). Although the exact incidence of second-impact syndrome isn't known, clinicians and patients must recognize the risk and prevent it by allowing adequate recovery time following the initial mTBI.[23-25]

• ***Other health disorders***. Long-term health disorders associated with recurrent mTBIs include neurocognitive deficits (attention, memory, processing speed), posttraumatic stress disorder, psychosocial health problems such as binge drinking, major depression, impairment of social functioning and ability to work, and suicidal ideation. It may also lead to epilepsy, headache, and alterations in personality or behavior.[1,22]

To scan or not to scan

Brain computed tomography (CT) is the standard diagnostic tool for evaluating the intracranial condition of patients with any degree of acute TBI. The incidence of intracranial abnormalities on CT associated with mTBI ranges from 0.7% to 20%.[26,27] A clinician deciding whether to perform a brain CT must weigh the pros and cons on an individual basis: Excessive use of CT increases exposure to radiation, but overly conservative usage may risk missing life-threatening lesions.[28] Performing routine brain CTs on all patients with mTBI is resource-intensive, and results in unnecessary radiation exposure given the low percentage (an estimated 1%) of patients with mTBI who ultimately require neurosurgical intervention.[29]

Several sets of criteria to help clinicians make appropriate decisions about CT scanning in patients with mTBI have been developed; for example, the Canadian CT Head Rule (CCHR), the New Orleans Criteria (NOC), the CDC criteria, and the Nexus (National Emergency X-radiography Utilization Study) II criteria.

The CCHR specifies seven clinical items that support the decision to scan:

- GCS under 15 at 2 hours after injury
- suspected or known open or depressed skull fracture
- any sign of basal skull fracture
- vomiting 2 or more times
- age 65 or older
- retrograde amnesia for more than 30 minutes
- dangerous mechanism of injury such as motor vehicle collision, falls, or blast injuries resulting from explosions.[27]

Under the NOC, which is used for patients with minor head injury and a GCS score of 15, the following seven clinical items support a decision to scan: headache, vomiting, seizure, alcohol or drug intoxication, persistent anterograde amnesia, age over 60 years, or visible injury above the clavicle.[6,27] The presence of intoxication is defined clinically by slurred speech, presence of alcohol in blood or on breath, or nystagmus in the presence of other indicators of alcohol intoxication.[27]

Research has found that the overall performance of the CCHR is superior to the NOC in patients with mTBI.[27]

Under CDC criteria, a noncontrast head CT is indicated in patients with head trauma who experience a loss of consciousness or posttraumatic amnesia if one or more of the following is present: headache, vomiting, age over 60 years, drug or alcohol intoxication, deficits in short-term memory, physical evidence of trauma above the clavicle, posttraumatic seizure, GCS score under 15, focal neurologic deficit, or coagulopathy. These criteria are designated as generally accepted principles for patient management that reflect a high degree of clinical certainty, or Level A recommendation.[30]

Under Nexus II criteria, CT is indicated for patients with significant skull fracture who also have scalp hematoma, neurologic deficit, GCS of 14 or under, abnormal behavior, coagulopathy, or persistent vomiting.[6]

The use of brain imaging as a stand-alone marker for intracranial pathology in patients with closed head injuries would lead to unnecessary interventions and elevated cost for patients and hospitals. The cost of a noncontrast brain CT in an American ED varies from $391 to $2,015.[29] However, given the absence of specific diagnostic markers for mTBI, the demand for head CT is high. The supply of CT scanners, though widely available in the United States, can be limited in smaller, rural, and critical access hospitals. The availability of these scanners drops precipitously

worldwide in resource-limited countries and war zones.[29]

Unfortunately, no single biomarker for TBI has been identified, although multiple biomarkers have been proposed in the literature, ranging from markers of inflammation such as tumor necrosis factor alpha and interleukin-1 beta, to markers of astrocyte activation such as S-100 calcium binding B levels (S-100B). Given the complexity of the intracranial response to injury, it seems unlikely that any single biomarker would be sufficiently robust for use as a clinical diagnostic test to determine presence and extent of mTBI or any degree of TBI. However, a peripheral, adjunctive blood-based biomarker panel consisting of multiple markers may flag patients with structural brain lesions who are in greater need of timely, advanced management.[29] Under CDC guidelines, consideration can be given to not performing a CT in patients with mTBI who have no significant extracranial injuries and a serum S-100B level less than 0.1 micrograms per liter (mcg/L) measured within 4 hours of injury.[30]

Nursing assessment

On presentation and during the acute phase of any TBI, the nurse focuses on the mechanism of injury and existing health history as well as assessing airway patency and the patient's ability to protect the airway, breathing effort and effectiveness, and circulation. The head and neck must be stabilized if a cervical spine injury is known or suspected.

When taking the patient's history, the nurse should ask the patient to describe the event and details leading up to the event and immediately following it. The response can help determine the degree of amnesia, if present. Findings can be documented using a symptom checklist, such as the Standardized Assessment of Concussion (SAC). Besides amnesia and neurologic signs and symptoms, SAC grades arm and leg strength, sensation, and coordination. Sport Concussion Assessment Tools such as SCAT-3 have been developed to assess injured athletes on the field.[6,31-34]

The nurse must also perform a thorough physical assessment to help differentiate mTBI from injuries to other organ systems. Many of the signs and symptoms associated with mTBI are nonspecific—for example, headache, dizziness, and nausea and vomiting—and may occur without mTBI or other trauma, or may indicate an injury other than TBI.[14,34]

The nurse conducting a neurologic assessment of a patient with suspected mTBI should document level of consciousness, including alertness, short-term memory and recall, attention and concentration, and orientation to self, date, time, location, and situation.[6] Headache, dizziness, vision abnormalities, vomiting, and disequilibrium are key diagnostic clues.

Cranial nerve assessment including extraocular movements, limb strength and coordination, and pupillary reactivity should also be performed. The presence of any focal neurologic deficit will prompt further diagnostic testing, including brain imaging with noncontrast brain CT or, in some cases, multimodal neuroimaging such as functional MRI.[14]

Advanced neuroimaging techniques can display neural networks and connectivity that underlie behavior and cognition. Through brain mapping, these advanced neuroimaging techniques can identify abnormalities affecting functional connectivity, contributing to developing an individualized plan of care for each patient.[8,14,35] The decision of whether and when to scan and what type of diagnostic imaging to perform is based on extent of injuries, neurologic assessment findings, and the patient's overall clinical stability.

Plan of care and patient education

Teach patients and families that mTBI and concussion are terms that are used interchangeably, and that a concussion is a brain injury. Tell them that mTBI is defined as a self-limiting phenomenon and that recovery occurs within 3 months of the inciting event in most patients who follow all treatment recommendations.[14,18,36]

Following mTBI, both physical and cognitive rest for 3 to 5 days is indicated to promote recovery. Although challenging, cognitive rest includes avoiding reading, texting, playing video games, and using computers. Explain to patients involved in school or academic work that adjusting their daily schedule can help them return to a preinjury academic schedule and level of activity faster. Advise them to return gradually to work or school rather than attempting to immediately return to their preinjury level. If symptoms worsen with activity, tell them to decrease their activity to a level where symptoms are no longer present and wait several days before attempting to increase activity.[14]

Gradual progression from light physical activity such as walking or stationary bicycling to more vigorous aerobic activity followed by resistance activities will promote recovery. For athletes who sustain an mTBI, the National Collegiate Athletic Association has published an algorithm for a gradual return to sport-specific training following concussion.[14]

Warn patients about the danger of second-impact syndrome and tell them to avoid activities that might lead to any new head impact, especially during the recovery period. Make sure they understand that although helmets help protect against scalp injuries and skull fractures, they don't prevent brain injuries resulting from whiplash-type or rotational head motions.[22]

The following interventions apply to specific signs and symptoms commonly associated with mTBI.

• **Headache.** Posttraumatic headache is a common symptom of mTBI, so teach patients and families about headache management. Treating headache early with effective therapy prescribed by the healthcare provider

is the most important intervention for management of posttraumatic headache. Treatment can include analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs). However, warn patients against overusing NSAIDs and other over-the-counter (OTC) remedies: An estimated 80% of those who self-treat posttraumatic headache have incomplete relief and may develop overuse headache, also known as rebound headache.[14] Advise them to report severe or persistent headaches to the healthcare provider, who may prescribe an alternate analgesic or, in some cases, refer the patient to a pain clinic or headache specialist.[14]

• **Dizziness.** For patients complaining of dizziness, nurses should determine if it's associated with nausea or motion. Advise these patients to rest for 3 to 5 days and then gradually resume both physical and cognitive activity and to avoid activities that could result in additional head trauma during the recovery period. Reassure them that dizziness typically resolves spontaneously over time. Recovering athletes should be given strict and specific guidelines to minimize the risk of reinjury.[14]

• **Sleep disturbances**. Educate patients complaining of sleep disturbances about sleep hygiene practices such as avoiding stimulants and alcohol and restricting exposure to TV or any type of illuminated screens such as computer monitors or phones for at least 1 hour before bedtime because the light wavelengths from these screens can suppress melatonin.[14,37] Advise them to go to bed at the same time every night and to avoid napping during the day. Performing light exercise approved by the healthcare provider, such as walking or stationary bicycling before bedtime, can be beneficial.[14]

Teach patients about any medications such as melatonin that may be prescribed to manage sleep disturbances. Warn them that most OTC sleep aids contain an antihistamine (commonly diphenhydramine) and aren't recommended for people with TBI because they may cause disturbances in memory and new learning.[14,38]

• **Depression.** Also common following mTBI, depression may be related to the event that caused the injury, such as a motor vehicle crash or an assault, or an exacerbation of depressive illness present before the injury. The incidence of psychological distress, including depression, after mTBI has been reported as low as 4% to 5% and as high as 49% to 63%.[20] A combination of medications including selective serotonin reuptake inhibitors and referral to appropriate therapists can be beneficial for patients with depression.[14]

• **Cognitive problems such as forgetfulness, distractibility, loss of concentration, and mental fatigue.** Advise patients to avoid alcohol during recovery. Attention, memory, problem solving, and reaction times are all affected while someone is under the influence of alcohol. Studies have shown that for some individuals with PCS, driving should be avoided until symptoms resolve.[39] Although these problems should all improve with time and appropriate treatment, referral to a neuropsychologist or cognitive therapy program may be indicated if symptoms persist.[14]

• **Sexual dysfunction**. Besides specific physiologic problems, sexual dysfunction also encompasses behavioral issues, including impulsiveness and inappropriate behavior, global emotional sexual difficulties, changes in libido, and sexual frequency.[17] Because the injury and recovery period will likely disrupt the patient's personal and family life, a holistic and collaborative plan should be developed to address all concerns and issues.[14]

Looking to the future

mTBI is a multifaceted injury with secondary pathophysiologic and potentially long-lasting neurologic complications affecting the patient's behavior, functional abilities, and quality of life. Continued research into all degrees of TBI, including mTBI, in both civilian and military populations will yield new knowledge that could lead to improved diagnostic tools and therapies as well as better protective headgear, safer automobiles, and even safer sporting environments. Patients and families can benefit from referral to appropriate postdischarge therapists and programs to promote maximum recovery and improve long-term outcomes in this vulnerable population. ■

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INSTRUCTIONS

Managing mild TBI in adults

TEST INSTRUCTIONS

- To take the test online, go to our secure website at **www.nursingcenter.com/ce/nursing**. View instructions for taking the test online there.
- If you prefer to submit your test by mail, record your answers in the test answer section of the CE enrollment form on page 38. You may make copies of the form. Each question has only one correct answer. There is no minimum passing score required.
- Complete the registration information and course evaluation. Mail the completed form and registration fee of $17.95 to: **Lippincott Professional Development**, 74 Brick Blvd., Bldg. 4, Suite 206, Brick, NJ 08723. We will mail your certificate in 4 to 6 weeks. For faster service, include a fax number and we will fax your certificate within 2 business days of receiving your enrollment form.
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