

Mild Traumatic Brain Injury

Toward Understanding Manifestations and Treatment

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Mild traumatic brain injury is misleading as a diagnostic term, as it may include a spectrum of manifestations ranging from transient mild symptoms to ongoing disabling problems. It is a source of significant economic burden to society in terms of days lost from work and costs related to medical treatment. Symptomatic individuals will frequently present to primary care general medical practitioners days, weeks, or even months after the trauma. General medical practitioners are thus put in the position of being the “gatekeepers” of medical care for such patients and become responsible for appropriate determination and authorization of medical tests, specialized referrals, and treatment. The purpose of this article, which is based on a review of the literature to 1997, is to provide physicians with an understanding of mild traumatic brain injury, including manifestations and treatment, as it occurs commonly and can significantly impact the quality of life of those affected. *Arch Intern Med.* 1998;158:1617-1624

Mild traumatic brain injury (MTBI) is an epidemic in the United States. It is estimated that 8 million individuals suffer head injuries in the United States annually,¹ 400 000 to 500 000 of whom are hospitalized,² and of those, 80% meet criteria for MTBI.³ Many individuals with MTBI do not come to medical attention at the time of initial injury but instead present to their primary care physicians days, weeks, or even months later with complaints of persistent troubling symptoms. The term *mild traumatic brain injury* may be misleading, as it includes a spectrum of manifestations that can range from transient mild symptoms to ongoing disabling problems. In the worst cases, MTBI results in disability that can have an impact on social relationships, employment, and routine daily functions. The number of individuals at risk for sequelae following MTBI is somewhere between the thousands of those who are hospitalized and the millions who have “minor” head trauma every year but who do not initially seek medical attention or are discharged home from emergency

departments. It is estimated that more than 50% of the persons with MTBI will develop symptoms⁴ and that approximately 15% will develop persistent disabling problems.⁵ The clinical manifestations and treatment of MTBI will be reviewed in this article.

DEFINITION

Traumatic brain injury may occur with or without evidence of external trauma following violent contact forces or rapid acceleration/deceleration movements of the head. The usual causes include assaults, crashes, and accidents involving motor vehicles, bicycles, pedestrians, construction, and sports. Definitive signs must be present at the time of head trauma for a traumatic brain injury to be diagnosed. These signs include confusion, loss of consciousness, amnesia, and focal neurological deficits. Traumatic brain injury is classified at the time of injury by certain measures, including duration of loss of consciousness, duration of posttraumatic amnesia, and the Glasgow Coma Scale (GCS) score. Strict criteria allow for classification of brain injury as mild, moderate, or severe. Subdividing

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patients with a brain injury into these categories facilitates determination of appropriate medical treatment and prognosis for recovery. Mild traumatic brain injury was defined by the American Congress of Rehabilitation Medicine in 1993 as head trauma with loss of consciousness lasting less than 30 minutes, a GCS score of 13 or more, and posttraumatic amnesia lasting less than 24 hours.⁶ Brain concussion, cortical contusions, intracranial hemorrhage, and axonal shear injury may occur with both open and closed head trauma in MTBI.

DIAGNOSIS

The typical patient with MTBI suffers a brain concussion. The term *brain concussion* is often used in the literature as a synonym for MTBI and implies a transient disturbance of neuronal function secondary to mechanical forces. The severity of concussion is roughly proportional to the magnitude of the applied traumatic force.⁷ A concussion is manifested by a loss of consciousness lasting less than 30 minutes or a period of confusion usually lasting less than 1 hour, but not more than 24 hours. If loss of consciousness lasts longer than 30 minutes, or if the duration of posttraumatic amnesia exceeds 24 hours, then a diagnosis of brain concussion and MTBI no longer applies. Hallmarks of concussion are confusion and amnesia, often without a preceding loss of consciousness. Early symptoms may include headache, dizziness, nausea, vomiting, slurred speech, imbalance, and incoordination. Signs of confusion may include vacant stare, disorientation, delayed verbal or motor responses, and poor concentration or attention. Confusion and memory dysfunction may be immediate in presentation following trauma,⁸ or may evolve gradually over several minutes.⁹ Traditionally with uncomplicated brain concussion, there is no structural brain injury evident on diagnostic magnetic resonance imaging (MRI) or computed tomographic (CT) scans. However, it is important for clinicians to realize that brain concussions may be complicated by coexistent cortical contusions or development of intracranial hemorrhages. Also, limited structural axonal injury may be present but

not evident on CT or MRI scans.^{10,11} Occasionally, associated transient cortical neurological deficits, such as global amnesia or cortical blindness, can occur; they are thought to be secondary to vascular hyperreactivity and may be trauma-induced, migraine-equivalent phenomena.¹²⁻¹⁴ Cumulative neuropsychological deficits can result from multiple brain concussions over months or years.^{15,16} Repeated concussions that occur over a short period, as may occur during contact sports, can result in the "second impact syndrome."¹⁷ This term describes a concussion that occurs while an individual is still symptomatic from an earlier one, which results in loss of cerebrovascular autoregulation and progressive cerebral edema.^{18,19} Cerebrovascular congestion resulting from the second impact syndrome may be detectable on brain CT scans.¹⁸ Increasing awareness of the cumulative effects of repeated concussions, and the second impact syndrome, has resulted in the development of practice parameters regarding standardized assessment and management of concussion in sports.¹⁹⁻²¹

PATHOLOGIC FEATURES

Brain Contusion

Contusions are areas of focal cortical injury that result from direct external contact forces or from the brain being slapped against intracranial surfaces with acceleration/deceleration trauma. Commonly involved sites include the bases of the frontal and anterior temporal lobes. Parasagittal contusions may occur with whiplash injury²² and result from acceleration/deceleration of the head without direct impact.²³ Cortical contusions are associated with localized ischemia, edema, mass effect, and poorer outcome in MTBI.²⁴ Signs of a contusion vary with cortical location and may include focal weakness, numbness, incoordination, aphasia, and difficulties with memory and cognition.

Intracranial Hemorrhage

Epidural, subdural, subarachnoid, or intracerebral hemorrhages may complicate MTBI. Intracranial hemorrhages occur less often with MTBI

than with other types of brain injury. However, anticoagulant therapy or coagulopathies increase risk. Neurological deterioration in a patient previously classified with MTBI is highly suggestive of an evolving intracranial hematoma. It is estimated that in 20% to 50% of cases of epidural hemorrhage there is a "lucid interval" following a brief loss of consciousness or period of confusion prior to neurological deterioration.²³ Epidural hemorrhage may occur secondary to tearing of a middle meningeal artery, middle meningeal vein, or dural sinus and may be acute or subacute in presentation.

Subdural hemorrhage occurs when trauma results in tearing of bridging veins or dura and may be acute, subacute, or chronic in presentation. Chronic subdural hematomas can present clinically months or years after a seemingly trivial head injury and often occur in the elderly.²³ Manifestations of a chronic subdural hematoma may be mistaken for another disease process, including stroke or progressive dementia.²³ Abuse should be considered as a cause of subdural hematomas in the elderly,²⁵ toddlers, and infants.

Subarachnoid hemorrhage, which is more common with severe brain injury, results from bleeding into the cerebrospinal fluid from small vessels torn by forces generated by trauma. Subarachnoid hemorrhage can result in cerebral vasospasm and ischemia.²³

Axonal Injury

Axonal shear injury is the primary pathologic features of traumatic brain injury of all types.^{10,11} It is a consistent finding in mild,²⁶ moderate, and severe classifications, with the distribution and number of axons involved increasing with worsening severity of injury.^{11,26} Trauma-generated shear forces produce nonuniform strains and distortions within the brain that disrupt axons and small blood vessels, causing axonal injury roughly proportional to the direction and magnitude of the applied traumatic force.^{7,27} Great forces result in structural injury and permanent loss of axonal function, while limited forces yield potentially reversible physi-

ological injury. The extent of axonal injury is suggested by duration of loss of consciousness or of post-traumatic amnesia and the GCS score.⁵ Areas of axonal shear injury may be indicated on CT or MRI scans by petechial hemorrhages that result from concomitant disruption of small blood vessels.

EVALUATION

Many patients with head injury are initially evaluated in the emergency department after trauma, although some with MTBI will present to their primary care physicians with symptomatic complaints sometime later. Classification and prognostic history to be gathered on initial evaluation include duration of loss of consciousness or of amnesia and GCS score if available. On history, patients or families may report a period during which the patient was "out." This period will approximate the duration of posttraumatic amnesia. Hemodynamic instability documented at the scene or in the emergency department may indicate hypoxic brain injury and thereby change treatment and expected outcomes. Seizures at the scene or in the emergency department can be related to physiological or structural brain injury. The incidence of immediate post-traumatic seizures is greatest in the pediatric population,²⁸ but the overall risk for developing epilepsy is low after MTBI.²⁹ Seizures that occur within the first week after trauma, intracerebral hematoma, cortical contusions, or depressed skull fractures may increase the risk for posttraumatic epilepsy, making a course of prophylactic anticonvulsants prudent.²³ Seizures that occur after trauma may also represent a preexisting, poorly controlled seizure disorder or a previously undiagnosed seizure disorder. An uncontrolled seizure disorder may have been the cause of the accident that resulted in head trauma. Intoxication with alcohol or other drugs may artificially depress GCS score and cognition, confounding the true clinical picture immediately after trauma. The mechanism of injury is also important. More serious injury is expected with high-velocity trauma, such as motor vehicle crashes or falls from great heights.

Relevant medical history includes previous head trauma, psychi-

atric disorders, and substance abuse, all of which may negatively affect outcomes. Alcohol and drug abuse are important causes of head injury and require treatment if the results of toxicology screens are positive. Poorly controlled chronic medical problems can result in head trauma by causing confusion or syncope and can also contribute to ongoing symptoms. A review of current medications is essential. Medications that may cause sedation or confusion can result in trauma or confound the clinical picture afterward.

A complete review of systems should be obtained to establish and document symptoms that may impair safety or vocational performance, and these symptoms will need further evaluation and follow-up. Individuals with clerical responsibilities should be forewarned of possible difficulties in concentrating and attending to tasks. Patients working in jobs such as construction and commercial operation of motor vehicles or machinery should not return to work without a period of observation for symptoms that may interfere with safety.

Physical examination in the emergency department should include evaluation for cranial adnexal injury and thorough neurological examination and mental status testing. Adjunct studies should include a brain CT scan if there was a loss of consciousness³⁰ and if the GCS score is less than 15, or if there is confusion, amnesia, lethargy, or focal neurological signs.^{30,31} A brain CT scan is also recommended for patients with head injuries caused by falls during seizures, as intracranial hematoma is especially likely in this group.³² Radiography of the cervical spine is recommended if injury is suspected. Cervical spine injury is unlikely in alert, oriented patients without complaints of neck pain or other painful trauma.³³ Plain films of the thoracic, lumbar, and sacral spine regions should be obtained as indicated. A cervical spine CT or MRI scan may be performed if clinically indicated. Alcohol and blood toxicology screening should be performed. Computed tomography of the brain is an excellent modality for imaging neurosurgically significant lesions. Patients with normal findings on CT

scans rarely have late neurosurgical deterioration.³⁰ Abnormal findings on CT scans of the brain should be followed up as indicated to rule out clinically significant evolution of lesions. Intracranial bleeding, or evolving cerebral edema associated with cortical contusions or diffuse axonal injury, may account for progressive deterioration in neurological function from the time paramedics or physicians first evaluate a patient with head trauma. Patients whose CT scans reveal no abnormalities but who have clinical abnormalities on examination or significant symptomatic complaints may have abnormalities on brain MRI scans. Magnetic resonance imaging of the brain is more sensitive at showing small areas of contusion or shear injury than CT and may be most sensitive if performed shortly after trauma.^{34,35} Lesions on MRI scans may resolve by 3 months after MTBI.^{34,35} Though still controversial, single photon emission computed tomography of the brain may be more sensitive than both CT and MRI in detecting lesions in symptomatic patients with MTBI³⁶⁻³⁹ and may remain sensitive to such findings beyond 6 months after the trauma.³⁹

EMERGENCY DEPARTMENT ADMISSION CRITERIA

Patients with MTBI should be admitted for observation if in the emergency department there is confusion, lethargy, a GCS score of less than 15, abnormal findings on CT scans of the brain, focal neurological signs, or a confounded clinical picture owing to seizures or alcohol or drug intoxication.^{30,31,40} Blood alcohol concentrations greater than 0.20% may artificially depress cognition and the GCS score.⁴¹ Admission should be considered if there is absence of a responsible person at home to monitor for progression of symptoms or if there is suspicion of abuse so that proper consultations may be obtained.

COMPLICATIONS

Head trauma may also result in injuries of head and neck structures. Skull fractures may be linear or depressed. Linear fractures are often benign, but any skull fracture can be

associated with underlying pathologic findings, including brain contusions, dural tears, and vascular trauma.⁴⁰ Communication between the intracranial compartment and the air constitutes an open head injury and can result from linear fractures with overlying scalp lacerations, depressed fractures, frontal bone fractures, and fractures involving the base of the skull. Any of these fractures may complicate minor head injury.⁴⁰ Basilar skull fractures can result in intracranial infections and cerebrospinal fluid fistulas manifesting as otorrhea. Fractures of the frontal bone can penetrate paranasal sinuses and are suggested by pneumocephalus seen on brain CT or MRI scans. Frontal bone fractures may also result in cerebrospinal fluid fistulas heralded by rhinorrhea and may lead to intracranial infection. Neurosurgical consultation is advisable with all open head trauma.

Cervical spine injury, which is often associated with acceleration/deceleration trauma, may be screened for by history, examination, and imaging studies. Major vascular injury within the neck may involve extracranial or intracranial portions of the vertebral or carotid arteries. Signs of vascular trauma may include neck pain, unilateral headache or facial pain, and transient or fixed neurological deficits referable to specific vascular territories of the brain. Trauma is among the primary causes of carotid cavernous fistulas and the cavernous sinus syndrome.⁴² Neurological examination and imaging studies can confirm a diagnosis of carotid cavernous fistula. Eye injuries may occur, especially with frontal impact head trauma. Cranial nerves II, III, IV, and VI may be injured in association with orbital trauma. Cranial nerves VII and VIII can be injured in association with temporal bone fractures. Generally, cranial nerve symptoms improve as perineural edema resolves, but neurosurgical consultation is necessary if entrapment is suspected. Ocular motility disorders, including diplopia, may be related to cranial nerve dysfunction or secondary to disruption of central pathways. Vestibular injury is common in head trauma and will be discussed further.

POSTCONCUSSION SYNDROME

Somatic, affective, and cognitive symptoms may complicate the recovery period after MTBI. These symptoms are often brought to the attention of primary care internists or family practitioners days, weeks, or even months after the initial traumatic event. The most common symptoms are headache and dizziness. Other common symptoms include disordered sleep, neck pain, and emotional or cognitive disturbances. The postconcussion syndrome refers to the ongoing occurrence of several such symptoms that gradually taper in severity over time. Postconcussion symptoms may result from brain injury or from trauma involving head and neck structures.

Somatic Symptoms

Headache is the most common symptom of MTBI.⁴ Possible causes include posttraumatic migraine, tension headache, exacerbation of a preexisting headache disorder, analgesic abuse, and cervical radiculopathies. Posttraumatic migraine may occur more commonly in the pediatric population and in adults with a family history of migraine.⁴³ Onset of basilar artery migraine has been reported after whiplash injury,⁴⁴ manifested as intense headaches that may follow or accompany the onset of posterior cerebral circulation aura, such as visual impairment, ataxia, dysarthria, paresthesias, vertigo, drop attacks, and weakness.⁴⁵ Vertebral artery insufficiency secondary to compression has been demonstrated with angiography in some symptomatic patients.⁴⁶ Radiculopathy involving the second and third cervical nerve root has also been implicated as a cause of chronic headaches after trauma.⁴⁷ Inner ear disturbances, such as benign positional vertigo or perilymphatic fistulas, may cause headaches.⁴⁸ Frequent ingestion of analgesic medications may also result in chronic headaches.⁴⁹ Less common causes of headache after trauma may include pneumocephalus, cerebrospinal fluid fistulas, or chronic extra-axial collections, such as subdural hematomas. It is important for clinicians to identify the cause

of headaches, and then to treat accordingly. If not previously performed, brain imaging studies and neurological consultation should be considered. If analgesic rebound headache is suspected, analgesic therapy should be tapered, and the patient should be observed for improvement over a drug-free interval.⁴⁹ Posttraumatic migraine, a diagnosis of exclusion, may respond to pharmacological treatment.

Dizziness is estimated to occur in 40% to 60% of individuals after MTBI.⁴ It is usually peripheral rather than central in origin. Peripheral causes originate outside the brain, while central causes involve the brain. There are multiple possible causes. Most commonly, the cause is a labyrinthine concussion, or benign positional vertigo, thought to be due to dislodgement of calcium carbonate crystals from sensitive nerve endings at the macula of the utricle. A labyrinthine concussion is often manifested by vertigo, nystagmus, and nausea that occur after rapid changes in head position. Generally, labyrinthine concussion resolves with adaptation over a period of weeks. Vestibular suppressants, such as meclizine hydrochloride, may delay adaptation recovery after the first few days of symptoms.⁵⁰ A program of vestibular exercises may be prescribed to promote recovery by enhancing physiological habituation.⁵⁰ Surgery has been shown to be effective, but should be considered only as a last resort.⁵¹ Other less common causes of posttraumatic dizziness may include a perilymphatic fistula, a central nervous system abnormality, or dysfunction occurring at the cervical spinal cord. Traumatic rupture of the oval or round windows of the inner ear may result in development of a perilymphatic fistula and inappropriate stimulation of labyrinthine receptors. Symptoms may include vertigo, hearing loss, tinnitus, chronic nausea, and exertional headaches.⁴⁸ Individuals who have perilymphatic fistulas may also complain of worsening balance and dysequilibrium with sudden head turning, riding on escalators, walking through revolving doors, and moving in relation to crowds.⁴⁸ A perilymphatic fistula can heal spontaneously, and treatment measures may include a period of bed

rest, head elevation, and avoidance of straining.⁴⁸ A chronic perilymphatic fistula can be corrected surgically with “patching” of the defective oval or round window. This procedure may be indicated if symptoms are intense, include progressive hearing loss, and persist more than 6 weeks despite bed rest.⁴⁸ Brainstem contusion is a possible source of “central” posttraumatic dizziness, which would be evident on neuroimaging studies and neuro-otological testing. Symptoms of a central source may include nausea with nonpositional vertigo and imbalance. Nonvertiginous dizziness in the absence of true peripheral or central vestibular dysfunction subsequent to whiplash-type trauma may be of cervical spinal origin. Proposed mechanisms of cervicogenic dizziness include aberrant afferent input from positional proprioceptors in the cervical spine,⁵² overstimulation of cervical sympathetic nerves,⁵³ and compromise of vertebral artery blood flow.⁵⁴ Evaluation of the dizzy patient may include audiological testing, electronystagmography, a fistula test, and posturography.⁵⁵ Patients with ongoing posttraumatic dizziness should be referred for neuro-otolaryngology evaluations.

Neural or conductive hearing loss may occur in up to 20% of individuals after MTBI.⁵⁶ Causes of conductive loss include tympanic membrane rupture, disruption of the ossicles, and middle ear hematoma. Neural loss is often associated with temporal bone fractures or vestibular injury, and usually involves high-frequency hearing. Low-frequency hearing loss may indicate a perilymphatic fistula.⁴⁸ Conductive hearing loss related to a hemotympanum often resolves with resorption of the hematoma, while other forms of conductive loss may be corrected surgically. Tinnitus, which has been reported to develop after as many as 70% of all head injuries,⁵⁷ may be due to electrophysiological dysfunction in the cochlea, cranial nerve VIII, or the central nervous system.⁵⁸ A trial course of antidepressant medications, biofeedback, avoidance of stimulants such as caffeine, and proper sleep hygiene have been recommended treatments for tinnitus.⁵⁸ Audiological testing and otolaryngology consultation are indicated for hearing loss or tinnitus after MTBI.

Loss of olfaction and taste occurs in some individuals with MTBI and is often associated with occipital head trauma.⁵⁹ Consequences may include safety risks, poor appetite, depression, and effects on personal hygiene and vocation. Possible causes are shear injury involving the olfactory nerve at the cribriform plate, mechanical injury to nasal bones or sinuses, and anterior frontal or temporal lobe contusions.⁵⁹ Otolaryngology consultation is indicated, especially if mechanical injury to nasal bones or sinuses is suspected.

After an MTBI, there may be problems with onset and maintenance of sleep.⁶⁰ Chronic pain can play a role. Patients may respond to sleep hygiene measures or to short-term pharmacological interventions. Polysomnography is indicated in patients who are refractive to these interventions, and the findings can help diagnose specific sleep disorders that may then be treated accordingly.

Neck pain and stiffness may also complicate recovery. These complications often occur after motor vehicle crashes that result in hyperflexion/hyperextension of the neck. Horner syndrome may occur in severe cases owing to sympathetic pathway injury. Other associated pathologic findings may include torn muscles or ligaments, vertebral column injury, and radiculopathy. Headache may occur with injuries to cervical roots C2 and C3.⁴⁷ Carotid or vertebral artery injuries may occur. Myofascial damage can result in referred pain to the shoulders, face, and scalp. Evaluation of chronic neck pain should include a neurological examination and cervical spine imaging studies. Neurosurgical consultation should be obtained if cervical vascular injury, radiculopathy, or vertebral column injury is suspected. Treatment of uncomplicated “whiplash” injury includes soft-collar immobilization, ice, and nonsteroidal anti-inflammatory therapy during the first 2 to 3 days. Thereafter, heat, specialized exercises, and nonsteroidal anti-inflammatory drug therapy may facilitate improved range of motion. Analgesic injections of myofascial trigger points, massage, ultrasound treatments, and acupuncture may also be helpful.

Neuropsychological Symptoms

Emotional symptoms may include irritability, lability, anxiety, and depression. These symptoms may be multifactorial in origin. Premorbid personality and psychological state can influence the emotional response to being injured.²² Emotional dysfunction may stem from the subjective experience of somatic or cognitive problems. Organic brain injury involving the temporal lobes or portions of the frontal lobes can also play a role. The amygdala region of the temporal lobes and the orbitofrontal cortex have multiple limbic system connections and play important roles in modulating emotion and personality stability.²² Emotional symptoms that may develop after MTBI may be a source of significant disability and can contribute to the development of the persistent postconcussive syndrome discussed below. Neuropsychological evaluation and treatment are indicated.

Possible cognitive symptoms include difficulties with concentration, attention, memory, word finding, information processing, and executive functioning. These problems become more common with increasing severity of brain injury as indicated by duration of loss of consciousness or of posttraumatic amnesia. Those patients with unilateral or multifocal brain lesions on CT or MRI scans are more likely to have neuropsychological symptoms after trauma.^{24,35} Frontal parenchymal lesions have been related to impairments in executive function, and temporal lobe lesions seen on MRI scans have been associated with impairment of memory.²⁴ Cognitive impairment may compromise safety, vocation, and life skills. Neuropsychological evaluation and intervention are indicated. Nonorganic factors can play a role and are discussed further in the “Persistent Postconcussive Syndrome” section.

RECOVERY

Patients who are symptomatic after MTBI generally recover over a 3-month period,^{35,61,62} in terms of ability to perform well on neuropsychological measures and ability to return to work despite the possible persistence of minor

symptoms. It has been shown that focal parenchymal lesions on MRI scans of the brain resolve within 1 to 3 months and that these changes are paralleled by improvement in performances on neuropsychological tests and resumption of previous routine activities.^{24,35} The typical patient who has an MTBI with a brief loss of consciousness, a GCS score of 15 in the emergency department, and posttraumatic amnesia lasting less than 1 hour will usually recover in 6 to 12 weeks.^{61,62} Athletes and young persons may recover in a few days.⁶³ Longer recoveries may be expected in older patients and in persons with preexisting medical conditions, including psychiatric disorders, alcohol or drug dependency, and previous head trauma. Patients with prolonged loss of consciousness or posttraumatic amnesia may demonstrate symptoms that may never clear completely.⁶⁴⁻⁶⁶ Over 6 to 9 months, the patients who are still symptomatic will continue to recover.⁶⁷ Reportedly, by 1 year after the initial injury, 85% to 90% of patients will be able to resume prior responsibilities, although some may still be susceptible to various degrees of impairment when subjected to physiological or psychological stress,^{15,68} and those returning to demanding vocations may always be aware of limitations in performance.⁶⁹ The 10% to 15% of patients who are still symptomatic 1 year after injury may include those with the persistence of 1 troubling symptom, varying clusters of symptoms, or even worsening of their entire symptom complex. This condition has been termed *the persistent postconcussive syndrome*.^{5,70} Such individuals are at high risk for emotional and cognitive disabilities that may involve the inability to carry out ordinary daily activities and work responsibilities and to maintain important social relationships.

PERSISTENT POSTCONCUSSIVE SYNDROME

The persistent postconcussive syndrome may result from brain injury or may be related partially or entirely to chronic pain, anxiety, or depression.^{5,70-75} Interaction between these factors can result in cognitive and emotional disturbances that may have an impact on social relationships, em-

ployment, and daily function. Anxiety and depression are often present in persistently symptomatic patients with MTBI,^{22,70-73} and complaints of chronic pain, particularly headache, are also common in this group.^{22,70,74} Anxiety or depression may be (1) rooted in premorbid psychological and personality factors, (2) a response to the subjective experience of being injured, or (3) related to organic brain injury.^{22,70} Chronic anxiety can disrupt concentration and high-level cognition.^{22,66} Depression is also known to disrupt cognitive functions, including concentration, attention, memory, and executive function.⁷⁶ *Pseudodementia*⁷⁷ and *conversion pseudodementia*^{70,78,79} are terms that have been used to describe the cognitive impairment seen in persistently symptomatic patients with depression as well as MTBI.⁷⁹ Pain in MTBI may result from headaches, cranial adnexal injury, back injuries, peripheral nerve injuries, or other bodily trauma. The physiological and psychological causes and effects of chronic pain are quite complex. It is known that chronic pain requiring analgesic palliation can negatively impact sexual function, sleep hygiene, and mental alertness.²² Reduced capacity to perform complex cognitive tasks may be attributable to the direct distraction of pain or related to associated fatigue, sleep deprivation, depression, anxiety, poor motivation, or the effects of analgesics.²² The pattern of behavior resulting from chronic pain can have a negative impact on relationships with family, physicians, and employers.²² Subjective experience of pain, anxiety, or depression can yield dysphoric moods, anger, irritability, loss of self confidence, and other maladaptive psychological reactions to being injured.²² These reactions in turn play a role in the exacerbation of postconcussive symptoms.

MALINGERING

Patients who have MTBI with neuropsychological impairment and functional disability that are disproportionate to objective injury, especially in the presence of symptoms that are inconsistent with known effects of neurological lesions, are often construed as malingering.^{80,81} *Accident neurosis* is a diagnostic label that has been used to describe such patients,

particularly those involved in ongoing litigation.^{82,83} Depression may be present in up to one third of this chronically symptomatic patient population.⁵ Also, a portion of these patients may be placed in the role of a "disabled person" by families in which there is belief that organic brain injury is responsible for all symptoms.⁸³ Both cognitive and somatic symptoms typical of the postconcussive syndrome can occur in patients with many other neurological or psychological disorders, as well as in the general population.⁸⁴ A subpopulation of postconcussive patients may overattribute their symptoms to brain injury, while underestimating the frequency of the same symptoms occurring in the general population.⁸⁵ Malingered and exaggeration of symptom severity for purposes of litigation is thought to play a role in only a small portion of the persistently symptomatic MTBI group.^{65,66,86-88} A number of studies have failed to show any relationship between litigation and magnitude or duration of symptoms.⁷¹ In those studies that have suggested such a relationship, litigation was found to be unrelated to resumption of work activity.^{65,66,89} An assumption has been offered in some reports that the greater the subjective perception of the injury is, the more likely it is that the subject will seek compensation.^{22,87} Also, the resolution of litigation does not reliably put an end to disabling symptoms.^{83,90} Measures of symptom validity and motivational bias are worthwhile in symptomatic patients with chronic disability in whom there are discrepancies between subjective complaints and objective findings.^{80,81,88} Neuropsychological evaluation and symptom validity assessment tests, such as forced choice testing,⁹¹ should be used to screen for malingering or other nonorganic disorders in such patients.

CLINICAL MANAGEMENT SUMMARY

The determinants of postconcussive symptoms are multiple and complex. Treatment outcome is dependent on the appropriate diagnosis of factors responsible for the ongoing symptoms. Careful consideration must be given to somatic, neurologi-

cal, neuropsychological, emotional, motivational, and social factors that alone or together may contribute to a patient's disability. The subjective complaints offered by a patient provide clinically meaningful information and are strongly related to the nature and extent of disability and treatment needs.⁹² In those patients who present early, intervention should be directed at evaluation and treatment of somatic complaints, and there should be careful documentation of baseline neurological examination findings, including cognitive and emotional state. Patients and families should be educated at the first office visit regarding rationale for treatments and expectations regarding outcomes. Those patients experiencing significant cognitive or emotional difficulties and those who present later with reports of persistent somatic complaints or worsening of symptoms are at risk for the persistent postconcussive syndrome. Patients who have prominent cognitive or emotional symptoms that interfere with previous routine daily activities or interpersonal relationships should be referred to a skilled neuropsychologist for evaluation and treatment. Also, a referral to a psychiatrist experienced in treating such patients may be necessary. A course of anxiolytic or antidepressant medication may be indicated if prominent anxiety or depression is evident. Prominent somatic complaints, such as headaches or vertigo rooted in emotional dysfunction, may be refractive to traditional treatments but responsive to psychopharmacological interventions.^{74,93} Long-term use of narcotic, sedative, or anxiolytic medications should be avoided. Close family members should be incorporated into the long-term treatment plan, as they may indirectly be promoting the "sick role" and persistent symptoms. Those individuals suspected of having the persistent postconcussive syndrome may benefit from referral to specialized pragmatic multidisciplinary cognitive therapy programs that include components of psychotherapy, occupational/vocational interventions, and adaptive strategy training.^{22,92,94} Other important considerations include determination of a patient's ability to safely return to sports, work, or op-

eration of motor vehicles. Specific guidelines exist regarding the management of concussion in sports, and these should be carefully reviewed by physicians who are involved in the care of sports competitors of any age.^{19,20} Knowledge of a patient's functional capacity is essential in making decisions regarding capability for return to work or operation of motor vehicles. If there is doubt, functional capacity evaluations can be performed by trained specialists at most large rehabilitation centers. Patients who have areas of functional impairment should be referred for appropriate therapy, and temporary restrictions should be placed on potentially unsafe activities. In summary, the management of MTBI should be practical; it should always include patient and family education and, when necessary, somatic medical treatments, psychological-psychiatric therapies, and pragmatic occupational interventions. Regarding prevention of head injury, strategies include persuasion, laws (eg, seat belts and helmets), and automatic protection (eg, air bags). Persuasion to change unsafe behaviors is most difficult. National safety education campaigns that have been successful include Think First⁹⁵ and Safe Kids.

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Public educational materials are available from the National Head and Spinal Cord Injury Prevention Program (Think First Foundation, 22 S Washington St, Park Ridge, IL 60068) and the National Safe Kids Campaign (111 Michigan Ave NW, Washington, DC 20010-2970).

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