

Physiatrie Management of Mild Traumatic Brain Injury

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Abstract

Mild traumatic brain injury (MTBI) is a common condition, afflicting as many as 1.5 million Americans yearly. Most individuals sustain MTBI as a result of motor vehicle collisions, but it may also occur as a result of falls, physical assault or sporting accidents. Problems related to MTBI include various pain syndromes, cognitive impairments, disorders of affect, cranial nerve dysfunction, and vertigo, arising from injury to the brain, head, or cervical spine. Symptoms are usually transient, although a small percentage of afflicted individuals develop long-lasting problems, often preventing them from leading productive lives. Recognition of these problems as arising from MTBI is difficult due to the frequent lack of abnormal findings on diagnostic tests and failure to identify a history of head trauma. The American Congress of Rehabilitation Medicine has defined MTBI, an important first step in identifying individuals who need treatment. Diagnosis is usually made by directed questions regarding trauma history and careful procurement and interpretation of appropriate tests. Once a diagnosis is made, proper care can be prescribed in order to lead patients toward more productive lives.

Key Words: Traumatic brain injury, rehabilitation.

Epidemiology

TRAUMATIC BRAIN INJURY (TBI) is a serious health concern, accounting for 500,000 hospitalizations per year in the United States, the vast majority of which are referred to as mild or minor injuries (1). These figures, however, underestimate the true incidence of mild TBI (MTBI), as there are as many as 1.5 million occurrences of transient loss of consciousness in the United States yearly that do not result in hospitalization (2). Many more individuals sustain mild head injuries as a result of sporting or recreational accidents, with at least 250,000 concussions occurring yearly in high-school football alone (3). The extent to which mild brain injuries represent a severe health concern is underscored when one considers

that for every head injury fatality, there are 27 individuals injured but not hospitalized. This alone accounts for an incidence of 1.5 million cases of mild TBI per year (4). Many individuals never seek medical treatment, contributing to the lack of accurate epidemiological data (5, 6). Mild TBIs are most commonly due to motor vehicle collisions, although a large percentage arise from sporting or other recreational activities, with still fewer occurring as a result of assault. Symptoms of mild TBI are usually transient, often lasting only a few seconds, although many individuals experience persistent symptoms, including pain, dizziness, hearing loss, olfactory and gustatory loss, visual disturbances, behavioral disorders, and impaired cognition. Accurate diagnosis and treatment require recognition of these sequelae as arising from mild TBI.

Terminology

“Mild traumatic brain injury” is a term that causes confusion for health care providers. Numerous terms have been used in the past, but

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they lack a cohesive definition or a set of criteria that effectively distinguishes minor brain injuries from more severe trauma. "Concussion" is the most commonly used term to describe these injuries, but definitions vary widely in both specificity and severity. Mild head trauma has also been referred to both as "trivial head injury" (7) and "minimal head injury" (8), suggesting that no adverse sequelae result. Some researchers have attributed the symptoms of mild head trauma to post-traumatic stress disorder (PTSD), which is a specific psychiatric disorder that must meet DSM 4 criteria and is not synonymous with MTBI. Others have used either accident or compensation neurosis to describe individuals sustaining minor injuries (9, 10). These terms denigrate the legitimate and often long-lasting symptoms of those truly afflicted and should be used cautiously, if at all, by health care providers.

Despite the varied definitions, "concussion" remains the most frequently utilized term to describe mild brain injuries. As part of an effort to better define the term as well as to improve treatment, the Quality Standards Subcommittee of the American Academy of Neurology established a definition of "concussion" which also describes its severity. "Concussion" is defined as a traumatically induced alteration in mental states, which may or may not result in unconsciousness (11). The two defining components of concussion are confusion and amnesia, which may occur immediately after the blow to the head (12) or several minutes later (13). Confusion is manifested as distractibility, inability to maintain coherent thoughts or carry out goal-directed movement, or a disturbance of vigilance (14). Concussion severity is based on duration of symptoms following the injury as well as the presence or absence of unconsciousness. The details are provided in Table 1. This grading system has been utilized to determine whether athletes sustaining a head trauma should seek additional medical treatment prior to considering resumption of competition (11).

The Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine has established "mild traumatic brain injury (MTBI)" as the preferred term for concussions. Specific criteria were established to distinguish MTBI from more severe forms of TBI (Table 2). While this definition is similar to the one provided by the American Academy of Neurology, it better differentiates mild from more severe head trauma. A grade 3 concussion is no longer considered mild when unconsciousness persists beyond 30 min-

TABLE 1
Concussion

Definition	A traumatically induced alteration in mental state that may or may not result in unconsciousness.
Grade 1 Concussion	<ol style="list-style-type: none"> 1. Transient confusion 2. No loss of consciousness 3. Concussion symptoms or mental status abnormalities on examination resolve in less than 15 minutes.
Grade 2 Concussion	<ol style="list-style-type: none"> 1. Transient confusion 2. No loss of consciousness 3. Concussion symptoms or mental status abnormalities on examination last more than 15 minutes.
Grade 3 Concussion	<ol style="list-style-type: none"> 1. Any loss of consciousness, either brief (seconds) or prolonged (minutes). <ol style="list-style-type: none"> (A) mild brain injury (B) moderate brain injury (C) severe brain injury

TABLE 2
Mild Traumatic Brain Injury

Definition	A traumatically induced physiological disruption of brain function, as manifested by at least one of the following:
	<ol style="list-style-type: none"> 1. any period of loss of consciousness; 2. any loss of memory of events immediately before or after the accident; 3. any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused); and 4. focal neurological deficit(s) that may or may not be transient.

but where the severity of the injury does not exceed the following:

1. loss of consciousness of 30 minutes;
2. after 30 minutes, a Glasgow Coma Score of 13-15; and
3. posttraumatic amnesia not greater than 24 hours.

This definition includes:

1. the head being struck;
2. the head striking an object; and
3. the brain undergoing an acceleration-deceleration movement without external trauma to the head.

This definition excludes stroke, anoxia, tumor, encephalitis, etc.

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utes, the Glasgow Coma Score (GCS) falls below 13, or if post-traumatic amnesia persists beyond 24 hours. The definition includes those individuals sustaining either a direct blow to the head or

an acceleration-deceleration injury to the brain. It excludes those individuals meeting the criteria due to stroke, anoxia, tumor, infection or seizures. While this definition has the most specific criteria thus far, situations in which MTBI arise are rarely medical emergencies, so patients often do not seek acute medical treatment. Hence, medical documentation of the criteria is often lacking, so it is appropriate to consider the constellation of symptoms described by patients in making the diagnosis of MTBI.

Pathophysiology

The pathophysiology of MTBI differs from that of more severe cases in both the degree and location of the lesions, but not in the nature of the injury. Animal and human studies have documented similar neuropathological lesions in the brains of individuals sustaining mild brain trauma as those with more severe injuries. Advances in neuroimaging, specifically magnetic resonance imaging (MRI), have allowed investigators to view structural lesions not previously viewed with older radiographic techniques (15, 16). Furthermore, neurobehavioral disturbances have been correlated with specific lesions in the frontal and temporal lobes on MRI. These disturbances resolve as the imaged lesions disappear (17).

Pathological lesions following TBI may be classified as either focal or diffuse. Focal lesions include lacerations, contusions, focal ischemic or hypoxic injuries, and basal ganglia hemorrhages. They often occur as a direct result of blunt-instrument trauma to the head, resulting in skull fractures, intracerebral hemorrhages such as subdural hematomas, dural or cerebral lacerations, and contusions. Contusions, caused by the brain moving within the cranial vault and striking the interior portion of the skull, are most often located in the inferior frontal and anterior temporal lobes, accounting for disturbances in both executive function and affect. Focal hypoxic lesions and basal ganglia hemorrhages cause local abnormalities, but usually arise in the setting of more diffuse injuries arising from acceleration-deceleration injuries. The hippocampus is extremely sensitive to hypoxemia, accounting for the frequency of memory impairments following TBI.

Much has been published regarding the process of traumatically induced neuronal injury caused by the release of excitatory amino acids. Numerous animal models have demonstrated massive increases in extracellular levels of glutamate, aspartate, and acetylcholine immediately following TBI. This initiates a process leading to increased intraneural calcium and the development of highly reactive oxygen radicals, causing lipid peroxidation of the cell membrane and ultimate neuronal demise. This process has been experimentally demonstrated in both mild and severe cortical injuries (18) and is likely a cause of functional impairment regardless of injury severity.

Diffuse axonal injury (DAI) was first described in the early 1980s as a process causing diffuse neuronal injury. It arises from shearing forces occurring in acceleration-deceleration injuries (19, 20), causing damage to white matter tracts. Originally, DAI was described as occurring in only the most severe cases of TBI, accounting for poor outcomes such as coma and vegetative state. However, DAI has also been found in MTBI, both in animal models (21, 22) as well as in postmortem examinations of individuals succumbing to other, more serious injuries (19, 23, 24). It has become evident that a spectrum of DAI exists, in which neuronal damage occurs due to axonal stretch, but that neuronal injury differs in both the amount and location, based on severity of the insult. Accordingly, four stages of DAI have been described, ranging from mild transient alteration of axonal function, to immediate axotomy resulting in coma (25). Stage I DAI describes a process of transient ionic fluxes occurring primarily at the node of Ranvier when the axon is subjected to no more than a 5% stretch of its resting length. Generation and propagation of action potentials are halted, although lasting only several minutes. Stage II DAI occurs with a 5-10% stretch, resulting in ionic fluxes described for Stage I, but which reach a metastable state. Osmotic forces cause localized swelling and possibly mild alteration in axoplasmic transport. The disturbances are transient and axon structure returns to baseline without ultimate axotomy. Stage III DAI occurs when the axon sustains a 10-20% stretch. Normal ionic equilibrium can no longer be restored, resulting in cytoskeletal injury and severe impairments in axonal transport. Severe localized swelling follows, impairing structural integrity of the cell membrane, both of which cause axotomy 24-72 hours after the initial impact (26). Stage IV DAI occurs with a greater than 20% stretch, resulting in primary axotomy. Location of DAI in part depends on the severity of injury; it occurs primarily in subcortical white matter areas in MTBI and in the corpus callosum and brainstem in severe TBI.

Diagnosis

Appropriate diagnosis of MTBI is challenging. Those afflicted with the sequelae are often misdiagnosed as malingers, symptom exaggerators or neurotics, or those suspected of being in search of secondary gain. The failure to make a proper diagnosis is due to many reasons, including the frequent paucity of focal neurological abnormalities, failure of both the clinician and the patient to attribute signs and symptoms to a brain injury, and lack of both medical documentation at the time of the injury and objective abnormal findings on diagnostic studies. This difficulty in diagnosis has led many clinicians to doubt the existence of MTBI as a clinical entity, fostering a belief that those complaining of symptoms are malingering. Furthermore, many symptoms following MTBI are not directly a result of brain injury, but are due to whiplash, which often accompanies brain trauma.

Despite these challenges, it is appropriate to consider the symptoms reported by a patient, the situation in which the injury occurred, and physical examination findings, as well as results of diagnostic tests when making a diagnosis of MTBI. Symptoms most often reported by individuals with MTBI include headaches, memory disturbances, dizziness, and weakness, frequently occurring in combination (27). Clinicians should be cautious before solely accepting self-reported symptoms when making a diagnosis, since patients without head injury are remarkably astute in accurately describing these symptoms (28). An accurate diagnosis must include a reasonable etiology of the injury as well as satisfy the definition set by the American Congress of Rehabilitation Medicine described earlier. One should not diagnose MTBI if a period of altered mental state did not exist at some time surrounding the injury. Routine diagnostic tests are often unrevealing. Computerized tomography (CT) is frequently normal, in part because scanning usually occurs after the acute phase, since most cases of MTBI are not medical emergencies. Magnetic resonance imaging is more sensitive than CT in detecting subtle lesions than CT, including white matter lesions arising from DAI (29–31). MRI is the preferred neuroimaging modality in non-emergent situations, although here as well, results are often unrevealing even when MTBI has occurred.

Additional tests may help support the diagnosis. Positron emission tomography (PET) may provide evidence of cerebral dysfunction in the absence of detected structural lesions on CT or

MRI. PET findings are not by themselves universally accepted as accurate in detecting cerebral lesions following trauma, although they do have added significance when findings are correlated with abnormalities on neuropsychological examination or other imaging tests. Single photon emission computerized tomography (SPECT) is more sensitive in detecting cerebral injury following MTBI than standard CT (32, 33). Electroencephalography is most often normal following MTBI, although abnormalities in the absence of preexisting neurological disease provide supportive evidence for diagnosis. Evoked potentials, posturography, and formal assessments of olfactory and gustatory function may lend additional supportive evidence in certain cases.

Neuropsychological testing is often the only reliable measure supporting the diagnosis. Deficits in executive function, attention, concentration, processing speed, and memory are often found on testing in individuals who have persistent complaints of cognitive dysfunction. The fact that neuropsychological test results are often the only positive findings is potentially problematic, as poor test performance may be intentional on the part of the individual examined. Careful interpretation of test results as well as of test behaviors is critical in order to ensure that a proper assessment has been made.

Although abnormal focal neurological findings may be lacking, they may also be overlooked by an unsuspecting clinician. Findings suggestive of any diagnosis may be missed if they are not specifically sought out on history and physical examination. This is particularly true with MTBI. A full neurological evaluation is imperative, including assessment of all cranial nerves. Olfactory disturbance, for instance, has been found in up to 40% of all TBI cases in one study (34), yet is neither inquired about nor tested for in most examinations. In general, however, as is often the case in medicine, careful attention to history, physical examination, and appropriate procurement and interpretation of diagnostic procedures will lead the clinician to the proper diagnosis.

MTBI-Related Conditions

Cognitive Impairments

Cognitive impairments following MTBI are usually transient. Areas of impairment are generally similar to those in more severe injuries, and include problems with attention, concentration, processing speed, executive function and memory. Complaints are more common immediately

following the injury (35–38), but in the vast majority of cases the impairments spontaneously resolve, allowing the return to normal daily functioning. When impairments are persistent, they may be subtle, becoming apparent only during times of increased cognitive demand (39). Repetitive MTBI results in more persistent cognitive impairments (40), which is an important consideration when allowing an injured athlete to return to competition.

The array and severity of cognitive impairments reported in the literature may cause some confusion regarding the exact nature and extent of the problem. This is in due part to the considerable variability in patient selection and to the design of the various studies. A recent meta-analysis of studies examining this issue revealed that patients with MTBI demonstrate subtle impairments, most commonly in attention, but suggested that clinicians would be more accurate in not diagnosing a brain injury on the basis of neuropsychological test results than in diagnosing a brain injury (41). The studies selected for this review included only those in which patients were not preselected on the basis of symptoms, and therefore its finding is consistent with the fact that most cognitive impairments are transient.

Individuals with persistent cognitive complaints should be referred for neuropsychological testing in order to identify specific areas of cognitive weaknesses as well as to determine areas of preserved strength. Careful interpretation of data is essential and should be compared with expected pre-morbid capability, which is based in part on the educational and vocational history of the individuals afflicted. Goals of cognitive remediation are to develop strategies using preserved strengths in order to compensate for areas of weaknesses. Successful remediation requires patient and family education on both the test results and compensatory strategies developed.

Disorders of Special Senses

Visual, auditory, olfactory, and gustatory disturbances occur in varying degrees following MTBI. Visual impairments manifest as oculomotor and accommodative dysfunction as well as visual field loss. Individuals sustaining MTBI experience difficulty with refraction, near point convergence and stereo acuity (42). Optometric evaluation and treatment should be sought for individuals with visual complaints, as visual rehabilitation has been shown to objectively improve function following MTBI (43).

Olfactory dysfunction occurs in up to 40% of all cases of TBI (34). The three pathophysiological mechanisms of olfactory injury in head injury include damage to the nasal passages, shear injury of the olfactory nerve, and cortical contusions. If recovery of olfaction does not occur within the first 12 months following injury, prognosis for this sensory modality is poor (44). Patients must be asked about olfactory loss, as they often will not offer it as a complaint, even when present. When testing olfactory function, it is necessary to avoid noxious stimuli that may inadvertently stimulate the trigeminal nerve, possibly resulting in a false negative response. True gustatory loss is rare, and when present, is most often due to olfactory injury.

Whiplash-Related Injuries

Many sequelae of MTBI are not directly related to cerebral injury, but rather to concurrent trauma to extracranial structures. Whiplash frequently accompanies head trauma, particularly in motor vehicle collisions, but also in cases of blunt-instrument trauma and recreational or sporting accidents. Many complaints following MTBI are due to pathological lesions in peripheral nerves, muscles, and vascular structures in the head and neck. Whiplash, which results from forceful cervical flexion-extension, with or without a torsional component, may result in clinical situations that manifest as neck or head pain, reduced cervical range of motion, autonomic dysfunction, and visual disturbances. Although not directly related to brain trauma, whiplash-related injury is included in the discussion of MTBI due to the frequency with which it is a co-morbid condition.

Headaches

Headaches are the most frequent acute and chronic somatic complaint following MTBI (27). Although headaches in the acute phase may be a result of subdural or subarachnoid hemorrhage, the vast majority of post-traumatic headaches are due to head and neck injuries. They are due most often to musculoskeletal injuries, followed by nerve entrapment or injury, vascular disorders, and less frequently to autonomic dyscontrol (45). Accurate diagnosis of post-traumatic headaches relies heavily on history and physical exam finding, and little on diagnostic tests. Accurate assessment requires an understanding of the various etiologies of headache, in order to recognize both the common and the unusual causes.

In 1942, Simons and Wolff (45) proposed a classification system for post-traumatic headaches

that is still useful today. In this scheme, headaches are classified by the affected anatomical structure and include those arising from musculoskeletal trauma, nerve entrapment, vascular injury, or dysfunction in autonomic control. This scheme aids the clinician in systematically identifying the etiology of headaches by exploring common as well as obscure causes of cephalalgia. Despite the infrequency of cerebrally mediated causes of headache, pain associated with focal neurological findings, nausea, or vomiting should prompt an urgent work-up, including appropriate neuroimaging techniques.

Musculoskeletal Induced Headaches. The vast majority of post-traumatic headaches arise from injuries to muscles in the neck, shoulders and head, and have been reported to be present to some degree in nearly all cases in one series (45). "Myofascial pain syndrome" describes a condition characterized by muscle pathology in which tenderness is palpated in a muscle over a taut band of fibers. The pain radiates to a specific but remote body region. This tender area is known as a trigger point (46, 47). Individuals with myofascial pain describe a sensation of steady pressure that may be either mild or severe. It may be exacerbated by physical exertion, forward bending, orValsalva. Associated symptoms may include photophobia, dizziness, and a sense of unsteadiness.

Myofascial pain syndrome most often arises in head trauma in the setting of whiplash. Flexion-extension injuries of the cervical spine produce stretch injuries to muscles, which may ultimately result in the development of trigger points. The muscles most susceptible to injury include the trapezius, sternocleidomastoid, splenius capitis, splenius cervicis, temporalis, levator scapula and the scalenes. The mainstay of treatment is injecting the trigger point with a local anesthetic. Spraying the skin overlying the muscle with a topical cooling agent, followed by stretching of the involved muscle, ischemic pressure over the trigger point, and various thermal modalities are all effective in treating myofascial pain (47). Work habits and posture need to be observed and abnormalities corrected. Nonsteroidal anti-inflammatory agents and tricyclic antidepressants may provide additional relief. Tricyclic antidepressants are particularly helpful with coexisting sleep disturbances. Individuals afflicted need to be instructed in a home exercise program, including stretching and limbering maneuvers, in order to maintain the relief obtained with the foregoing measures.

Neurogenic Headaches. Headaches arising from entrapped or injured peripheral nerves com-

prise the second most common form of post-traumatic headaches (45). Pain arises from damage to a peripheral nerve either by entrapment in a scar formed at the site of impact or laceration, or in a nerve injured by excessive stretch occurring during whiplash. Pain is exacerbated by pressure over the nerve and has an electric or burning quality. The greater occipital nerve, which is the continuation of the second cervical root, is particularly vulnerable to stretch during whiplash. When injured, it causes pain over the posterior portion of the scalp. The greater occipital nerve may also be compressed as it pierces the semispinalis capitis or trapezius, which may themselves be injured in whiplash injuries. Treatment measures are similar to those used for other neurogenically mediated pain syndromes and include membrane stabilizers and various antidepressant drugs. Anesthetic infiltration in neuroma formation, as well as counterstimulation techniques, provides relief in many cases. However, attention must be directed to the possibility of coexisting myofascial pain, as this is present in nearly all cases of post-traumatic headaches, and successful treatment will not occur if it is overlooked.

Vascular Headaches. Migraine headaches are thought to arise from vascular dilatation and are described as dull and aching, often associated with nausea, vomiting, and anorexia. These headaches are rarely a direct result of trauma. Rather, they may be exacerbated following trauma in those previously afflicted (48). Recent evidence suggests that migraines following TBI may be due in part to alterations in electrolyte balance, release of excitatory amino acids, impaired glucose metabolism, and abnormalities in catecholaminergic and opioid function (49). Treatment of acute attacks is aimed at decreasing vasodilatation, while prophylactic treatment is provided by such drugs as beta-blockers, tricyclic antidepressants, and calcium channel blockers.

Dizziness

Dizziness is reported as the third most common somatic complaint following MTBI (27). Symptomatically, it ranges from a nondisabling perception of lightheadedness to incapacitating vertigo. Causes of impaired balance are numerous, due to the many central and peripheral systems involved in maintaining a normal upright position. Following TBI, dizziness is most often due to injury to the ear, although attention must be directed toward other causes, including peripheral neuropathies, complications from whiplash, musculoskeletal injuries, and vascular disorders. Accurate diagnosis requires a thorough under-

standing of normal vestibular function, as well as the possible etiologies of vestibular impairment following TBI. Most causes of post-traumatic dizziness can be determined through a detailed history and physical examination, although specialized tests, including electronystagmography, posturography, caloric irrigation, audiology and central nervous system imaging may be helpful in delineation of the precise etiology. Associated symptoms must be sought out, especially hearing loss and tinnitus, as individuals may not mention them unless specifically questioned.

Benign Positional Vertigo. Benign positional vertigo (BPV) is the most common neuro-otological disorder following closed head injury (50). It arises when calcium carbonate is dislodged from the macula of the utricle and reattaches to the cupula of the posterior semicircular canal. Diagnosis is usually made at the bedside by maneuvering the patient in provocative positions and assessing for nystagmus and vertiginous symptoms. BPV is usually self-limiting, and reassurance is often all that is needed. Persistent complaints should be treated with vestibular exercises under the guidance of a trained physical therapist. Pharmacological agents, including anticholinergic agents, benzodiazepines, and membrane stabilizers, may be helpful, although vestibular exercises alone are often more effective (51). As pharmacological agents mentioned may cause sedation and impair cognition, they should be used with caution in individuals who have sustained a TBI. Surgery may be considered as a last option in patients refractory to other treatments; it is successful in the vast majority of cases (52).

Temporal Bone Fracture. Dizziness associated with hearing loss and tinnitus occurs less commonly than BPV. Temporal bone fractures may result in both hearing loss and profound vertigo, which may be incapacitating. Suspected cases of temporal bone fractures must be assessed by a temporal bone CT scan, as they are easily missed on standard head CT scans and plain radiographs. Dramatic improvements may be observed with low dose benzodiazepine, although caution is urged in cases of both impaired arousal and cognition.

Hydrops and Fistulas. Endolymphatic hydrops and post-traumatic perilymphatic fistula (PTPF) are uncommon causes of vertigo. PTPF may be associated with mild nausea and headache, with hearing loss occurring as a fluctuating late complication. Hearing loss is a major component of endolymphatic hydrops. Treatment of both disorders involves discouragement of rapid changes in head position, and limited bed

rest. Surgery is sometime indicated for endolymphatic hydrops, but rarely for PTPF.

Cervicogenic Dizziness. Cervicogenic dizziness is a controversial disorder in which dizziness occurs in the absence of vestibular injury. It is postulated to arise from injury to either the anterior or the posterior chain of cervical sympathetic nerves following whiplash (53) or compromise of vertebral-basilar blood flow (54). Injury to the anterior cervical sympathetics occurring in cervical hyperextension injuries may result in Bernard-Horner Syndrome. This causes dizziness with associated ipsilateral miosis, ptosis, and anhydrosis. Similarly, hyperflexion injuries may damage posterior cervical sympathetics. Known as Barre-Lieou Syndrome, posterior cervical sympathetic dysfunction is a controversial disorder and is considered rare even by those who support its existence. Theoretically, symptoms develop due to irritation of the posterior cervical sympathetics, which converge to form the vertebral nerve as well as the sympathetic plexus which surrounds the vertebral artery. Irritation may result in increased vascular tone, causing symptoms of vertebral-basilar insufficiency (55), which has been demonstrated in an animal model (56). Associated symptoms include tinnitus, occipital headache, impaired vision, and dysphonia (50). Therapeutic interventions are geared toward decreasing cervical dystonia and increasing cervical range of motion.

Conclusion

MTBI is a condition that occurs with alarming frequency, yet is often overlooked and misdiagnosed. Although the majority of individuals afflicted will experience transient symptoms, a significant minority will have persistent and sometimes disabling problems, preventing them from returning to previous lifestyles. Health care providers must be diligent in detecting the clinical signs associated with MTBI in order to properly diagnose and treat afflicted individuals. Careful attention to history and physical examination findings, as well as appropriate accrual and interpretation of diagnostic tests will ultimately lead the clinician to the proper diagnosis and treatment.

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