Review Article

Definition, Diagnosis, and Forensic Implications of Postconcussional Syndrome

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Injuries from blows to the head often are manifested only as subjective complaints. Postconcussional syndrome thus can be feigned for financial or psychological gain. The authors review the pathology of brain trauma, symptoms of postconcussional syndrome, and criteria for diagnosis. In addition to somatic deficits, psychological and cognitive problems are common. The likelihood and severity of postconcussional syndrome are greater for women. Malingering may be suspected in cases involving litigation, and tests to detect it are available. Treatment for postconcussional syndrome depends on the specific symptoms. Pharmacotherapies may be helpful, but care should be used in prescribing drugs that could produce deleterious CNS effects.

(Psychosomatics 2005; 46:195–202)

The fact that being hit in the head is detrimental to one's health has been known since the dawn of time. Where the controversy has raged in academic, clinical, and legal medicine over the years has been in the answers to the following questions: What is the force necessary to cause head trauma? How is it manifested? and What can one expect after such injury occurs? Additionally, as with other conditions evidenced largely by subjective complaints, are the effects of such injuries dramatized or feigned for primary (financial) or secondary (psychological) gain? In 1866, this controversy began in earnest when Erichsen published results about patients who developed persistent complaints after sustaining mild head trauma.[1] He postulated that these detriments were due to a trauma-induced "molecular disarrangement" of the spinal cord. Since most of these injuries resulted from work on the Prussian railroad, the condition was initially termed "railroad spine." In 1879, Rigler challenged this concept and proposed that these injuries were due to "compensation neurosis." He believed that the reason for the increase in long-term disabilities following "minor head trauma" was the initiation by the Prussian railroad of a policy for compensating in-

jured workers.[1,2] Charcot later weighed in with his belief that the long-term symptoms resulting from "mild head injury" were due to "hysteria and neurasthenia" (i.e., vague fatigue and confusion brought on by "psychological factors").[1,3] By 1934, the diagnostic entities of "railroad spine," "compensation neurosis," and "hysteria" gave way to the current diagnosis of "postconcussional syndrome."[1] Unfortunately, the change in nomenclature did little to resolve questions about the actual existence of this condition, its pathophysiology, the prognosis, and other psychological factors (i.e., primary and secondary gain).

POSTCONCUSSIONAL SYNDROME DEFINED

Postconcussional syndrome is generally defined as a condition arising after "head injury" that produces deficits in

three areas of CNS functioning: 1) somatic (neurological—usually headache, tendency to become fatigued), 2) psychological (affective change, lack of motivation, anxiety, or emotional lability), and 3) cognitive (impaired memory, attention, and concentration) (Table 1.[2,4,5]

Postconcussional syndrome is difficult to define medically, as many of its symptoms are subjective. Several different sets of diagnostic criteria exist, many defined by the specialty of the physician who examines the patient (neurology, psychiatry, pain management, etc.), the clinical setting in which the patient is seen (emergency room, hospital, forensic evaluation), and whether or not more rigorous research criteria are applied.[6] Studies suggest that postconcussional symptoms of some nature occur in 38% to 80% of people who experience "mild head trauma" (Table 2).[1,2,7–10]

The American Psychiatric Association's (APA's) current criteria for postconcussional disorder, which are under study at this time, require that there be an "acquired impairment in cognitive functioning, accompanied by specific neurobehavioral symptoms, that occurs as a consequence of a closed head injury of sufficient severity to produce a significant cerebral concussion" (Table 3).[11] APA notes that there is insufficient evidence to establish a definite threshold for the severity of closed head injury but suggests

that at least two of the following three items should be present: "1) a period of unconsciousness lasting more than 5 minutes, 2) a period of posttraumatic amnesia that lasts more than 12 hours after the closed head injury, or 3) a new onset of seizures (or marked worsening of a preexisting seizure disorder) that occurs within the first 6 months after the closed head injury." APA also requires that "cognitive deficits in either attention (concentration, shifting focus of attention, performing simultaneous cognitive tasks) or memory (learning or recalling information)" must be clearly documented. In addition to cognitive disturbances, three or more of the following symptoms must persist for at least 3 months following the closed head injury: easy fatigability; disordered sleep; headache; vertigo or dizziness; irritability or aggression on little or no provocation; depression, anxiety, or affective lability; lack of spontaneity or apathy; or a personality change (such as social or sexual inappropriateness). These criteria also require that any cognitive, somatic, or behavioral symptoms that develop after head trauma need to arise de novo or to represent a significant worsening (if those symptoms existed before the head trauma) and must be accompanied by a significant impairment in the patient's social or occupational functioning and represent a significant decline from the patient's previous level of functioning.

APA notes that essential in the differential diagnosis of postconcussional disorder are fictitious disorders, when patients have a need to assume a sick role, which relieves them from social responsibility, and frank malingering, in which there is a desire for compensation that leads to the production or prolongation of symptoms. Other diagnoses to be considered in the differential diagnosis include cognitive disorders, dementia, mild neurocognitive disorders, somatization disorder, posttraumatic stress disorder, and undifferentiated somatoform disorder.[11]

The good news about postconcussional syndrome is that a preponderance of the literature shows that the majority of people recover fully within 3 to 6 months.[1,4,6,15] Only 7–15% of all people who suffer from even severe cases have any symptoms 1 year after the injury and can thus be considered to suffer from persistent or long-term postconcussional syndrome.[1,16–19] Factors found to be predictive of developing persistent postconcussional syndrome are female gender, ongoing litigation, low socioeconomic status, age greater than 40 years, history of alcohol abuse, significant comorbid medical or psychiatric illness, prior head injury, documented low preinjury cognitive abilities, poor preinjury psychosocial functioning, personality disorder (antisocial, borderline, hysterical, dependent), and

TABLE 1. Complaints of Patients With Postconcussional Syndrome

Somatic	Psychological	Cognitive
Headache	Depression	Decreased concentration
Dizziness	Anxiety	Forgetfulness
Photophobia	Irritability	Difficulty in learning
Phonophobia	Apathy	Difficulty with reasoning
Tinnitus	Emotional lability	Difficulty in processing information
Blurring of vision		Impaired memory
Tendency to become fatigued		
Lost or altered sense of smell and taste		

TABLE 2. Classification of Head Injury[10]

Injury Classification	Score on Glasgow Coma Scale	Duration of Loss of Consciousness (hours)	Duration of Amnesia (hours)
Mild	13–15	<0.5	<1
Moderate	9–12	≤24	≤24
Severe	≤8	>24	>24

prior headaches or psychiatric illness.[1,4,8,16,18,20] Patients less likely to suffer from postconcussional syndrome are well-motivated, younger patients who experience no loss of consciousness. Individuals likely to recover in 6–12 weeks experience a brief loss of consciousness, posttraumatic amnesia lasting less than an hour, and a score of 15 on the Glasgow Coma Scale.[18] When patients have persistent, dramatic, or unusual complaints, other factors, such as personality disorder, psychosocial problems, or secondary gain, should be considered as causative.[1]

PATHOLOGY

The primary pathological injury seen in brain trauma is axonal sheering and tensile strain damage, most commonly due to rotational acceleration forces.[1,2,20–22] The extent of axonal injury correlates with the duration of posttraumatic amnesia and loss of consciousness.[23] Once neurons are damaged, inhibitory neurotransmitters, such as γ-aminobutyric acid, as well as excitatory neurotransmitters, such as acetylcholine, glutamate, and aspartate, are released. Such neurotransmitter release can produce further neuron damage (i.e., the cascade of injury).[1,10,20,24] Other post-injury changes that can lead to further diffuse neurological injury include excessive calcium influx into damaged neurons, release of cytokines, oxidative free radical damage, damage to cell wall receptors, inflammation, and changes in the acetylcholine, catecholamine, and serotonergic neurotransmitter systems.[10] Post-mortem studies of humans with chronic posttraumatic headache and primates with induced concussions have demonstrated diffuse axonal injury, microglial clusters, and evidence of small petechial

hemorrhages that did not cause focal neurological deficits.[2,20,25] Experimental traumatic head injuries induced in animals have shown that both neurons and axons recover, usually within a few months of the injury.[10,26]

Clinically significant concussions may produce no detectable findings on computed tomographic (CT) scanning or magnetic resonance imaging (MRI) because of the diffuse nature of the damage.[20,21,27] Some studies have found MRI to be a more sensitive indicator of CNS postconcussion damage than CT scanning. MRI is better able to detect abnormalities such as contusions, focal edema, and microscopic lesions (i.e., micropetechial hemorrhages), especially if the scan is performed a few days after the event.[28] Single-photon emission computed tomography (SPECT) studies have shown decreased or asymmetric regional blood flow for up to 3 years after a concussion, particularly in patients suffering from posttraumatic headaches, which suggests that long-term physiologic changes have occurred after the concussion.[29] Studies with positron emission tomography (PET) show a reduction in the metabolic rate of glucose utilization in individuals diagnosed with postconcussional syndrome.[2] PET scans, although expensive, may be useful to document injury in cases of possible malingering, but they are not currently specific or sensitive enough to provide a definitive diagnosis.[1] Electroencephalograms may, on occasion, show asynchronous slow-wave activity after mild head trauma, but they usually produce normal results and are not a good screening test for postconcussional syndrome.[2,20]

SOMATIC DEFICITS

Headaches are the most common complaint of patients who experience minor head trauma.[2,30] Increased headaches are

TABLE 3. Classification of Concussion

Source and Concussion Grade	Confusion	Amnesia	Loss of Consciousness
Cantu guidelines[12]			
I	Not applicable	Resolving in 30 minutes	No loss of consciousness
II	Not applicable	Lasting longer than 30 minutes but less than 24 hours	Resolving in 5 minutes
III	Not applicable	Lasting longer than 24 hours	Lasting longer than 5 minutes
Colorado Medical Society guidelines[13]			
I	+	−	−
II	+	+	−
III	+	+	+
American Academy of Neurology guidelines[14]			
I	Resolving in 15 minutes	Not applicable	No loss of consciousness
II	Lasting longer than 15 minutes	Not applicable	No loss of consciousness
III	Any level	Not applicable	Loss of consciousness for any period of time

reported by 30% to 90% of patients with postconcussional syndrome; 8% to 32% still report increased headaches 1 year after their head trauma.[1,16,31] Generally, the type of headache (e.g., tension, migraine) experienced by individuals with postconcussional syndrome is similar to the type of headache they experienced before the trauma.[2] Patients with postconcussional syndrome generally report that the headaches last longer and occur more frequently than those they experienced before the event. The International Headache Society's diagnostic criteria for posttraumatic headaches are divided into the categories of acute and chronic headaches. Acute headaches begin within 2 weeks of the trauma and resolve within 2 months. Chronic posttraumatic headaches begin within 2 weeks of the event and continue for more than 8 weeks.[23] Eighty-five percent of headaches associated with postconcussional syndrome are described as steady, aching, tension-type headaches.[1,23] These headaches are believed to be due to soft and hard tissue injuries, such as myofascial injuries, intervertebral disk damage, facet joint injury, temporomandibular joint injury, and muscle spasms of the superior trapezius and semispinalis capitis muscles in the suboccipital region.[1,2,30] Although less common, migraine headaches, with or without auras, have been reported to develop hours to weeks after concussion.[1] Migraines are common in adolescents who participate in sports that cause multiple minor head traumas, such as football, boxing, and hockey. This form of headache has been termed "footballer's migraine."[28] Cluster headaches rarely develop after mild head trauma.[2]

The second most common complaint in postconcussional syndrome is dizziness, which is reported in about 50% of the cases, with a 1-year prevalence of 19%–25%.[1,2,16,31] Age has been found to be a correlating risk factor. The older the individual, the greater the likelihood of developing dizziness, which can be central and/or peripheral in origin (i.e., labyrinthine concussion, benign positional vertigo, or brain stem injury).[1,32]

Postconcussional syndrome often involves disturbances of the senses. Blurred vision occurs in 14% of patients and is usually caused by an optical convergence disorder.[1] Ten percent of patients with postconcussional syndrome report increased sensitivity to light and noise; 5% experience damage to cranial nerve I and/or tears of fibers passing through the cribiform plate, causing decreased smell and taste.[1,18]

PSYCHIATRIC SYMPTOMS

Half of the patients who experience concussion report nonspecific psychological symptoms, such as personality change, irritability, anxiety, and/or depression.[1,33] Most experience these changes within the first 3 months following the injury and have normal CT scans.[1,33]

Associated anxiety disorders include generalized anxiety disorder, panic disorder, obsessive-compulsive disorder, and posttraumatic stress disorder and have been reported to occur in 11% to 70% of people who sustain traumatic brain injury.[10,34] The most commonly reported symptoms are free-floating anxiety, fearfulness, intense worry, generalized uneasiness, social withdrawal, interpersonal sensitivity, and dreams about anxiety.[10] Anxiety disorders have been reported following traumatic injuries to both the right and left hemispheres.[35]

Apathy is commonly reported in postconcussional syndrome. Apathy can be an isolated primary syndrome or may be secondary to depression.[36] Primary apathy has been defined as a lack of motivation with reduced emotional, cognitive, and/or behavioral drive that is not attributed to intellectual impairment, emotional distress, or a diminished level of consciousness.[37] Primary apathy has been reported in 10% of patients following a closed head injury, while secondary apathy occurs, usually temporarily, in up to 60% of patients with closed head injuries.[36] Neurological damage to the subcortical-frontal region, basal ganglia, and thalamus has been associated with the pathogenesis of primary apathy.[10,38,39]

Although extremely rare in case reports of postconcussional syndrome, psychosis has been reported following severe head injury. A schizophrenic-like psychosis has been reported in 0.7% to 9.8% of subjects following severe traumatic brain injury.[40,41] Risk factors for developing psychosis following head injury include greater severity of initial trauma, a history of temporal lobe epilepsy, preexisting congenital neurologic disorder, and head trauma during adolescence.[10,42] Treatment of this condition can be difficult since in animal models "typical" neuroleptics, such as haloperidol, are less effective than when used for other psychotic conditions. These agents may also contribute to the impairment of neuronal recovery after the trauma.[10] It is not clear whether "atypical" neuroleptics have similar effects on neuronal recovery. There have been studies that show that risperidone and clozapine are beneficial for the treatment of psychosis occurring after head injury.[10,43]

People with preexisting diagnoses of affective disorders (depression, bipolar disorder), generalized anxiety disorder, somatoform disorders, and personality disorders seem to suffer from or are at least diagnosed with postconcussional syndrome at a higher rate than those without such

preexisting mental disorders.[2,44–46] Many of the symptoms of affective disorders, such as mood change, lability of mood, diminished attention and concentration, sleep disturbances, and anxiety, are similar to symptoms seen with postconcussional syndrome. Their presence after an accident can lead to the misdiagnosis of postconcussional syndrome. The patient may report feeling worse, when in actuality the pre- and posttrauma levels of functioning are comparable. Such errors, termed "recall biases" or "the good old days" phenomenon, are introduced when patients are unable to accurately remember their preinjury level of functioning.[47] It is critical, particularly if litigation is involved, for physicians to obtain old neuropsychiatric test reports, academic records, and work performance appraisals and to talk with family and friends to accurately determine the patient's pre- and posttraumatic levels of functioning.

COGNITIVE DEFICITS

Cognition can be defined as the processes involved in the brain's acquisition and analysis of data and the management of information.[10] Classic cognitive functions are attention, memory, language, abstraction, executive function, and perceptual skills.[10,33] Deficits in cognition are defined by an inability to concentrate, an inability to process information, word-finding difficulties, and the inability to integrate thought processes. Patients with postconcussional syndrome evidence reductions in information-processing speed, attention, and reaction time, which can be documented by neuropsychological testing.[1,33] Indices shown to have high specificity and a positive predictive value for cognitive deficits from postconcussional syndrome are the Stroop Color Test and the 2&7 Processing Speed Test, which both assess mental processing speed.[48] The Continuous Performance Test of Attention is another test with high sensitivity for predicting a negative outcome for cognitive deficits following concussion.[48] In the emergency room, the Digit Span Forward examination and the Hopkins Verbal Learning A test were shown to predict development of postconcussional syndrome and, in certain populations, also predict the duration of symptoms.[8]

The most common cognitive deficit after head trauma is verbal and nonverbal memory impairment. Depending on the severity of the closed head trauma, the percentage of people suffering some form of memory impairment ranges from 20% to 79%.[1,49] It has been estimated that between 4% and 25% of people who have postconcussional syndrome will experience some memory deficits after 1

year.[31] One explanation for the decreased ability to form new memories is decreased concentration, which reduces the effectiveness of memory formation. Deficits of short-term memory (e.g., misplacing items, difficulty recalling conversations) are the type most commonly seen in postconcussional syndrome. O'Shanick and O'Shanick found that memory impairment is greater for "effortful" than for "incidental" memory after head trauma.[50] When individuals with memory deficits related to severe closed head injury undergo neuropsychological testing, impaired episodic or declarative memory is seen, while procedural memory remains unimpaired.[51]

Brain-injured patients also demonstrate impairment of sustained and divided attention, while selective attention is usually spared.[10,52] This is evident in patients' difficulty in concentrating, problems in focusing on one task, and easy distractibility. Cholinergic dysfunction leading to impaired sensory gating and the inability to "shut off" distracting stimuli has been proposed as the cause of this "attention deficit."[53]

Cognitive deficits are thought to result from cortical damage, particularly damage involving the anterotemporal and the orbitofrontal lobes, which occurs frequently because of the close proximity of these lobes to the bony protuberances of the skull.[10,54] These deficits persist for varying periods of time following impact, with most showing full recovery within 6 months.[1,2] Disturbances of memory, attention, language, and executive functions are the most persistent, with most clearing by 1 year after the concussion.[10]

GENDER

Being female is associated with an increased likelihood of developing postconcussional syndrome, and women generally have more severe symptoms and a longer time to recovery.[5,7] Farace and Alves published a meta-analysis of postconcussional syndrome, which showed that women have a worse outcome than men following traumatic brain injury. Women showed more impairment than men on 85% of the variables measured.[55] Numerous theories have been advanced to explain these observations. Some feel that women, because of their smaller size and lighter weight, experience more injurious rotational forces than men do.[5,55,56] Others propose that men's and women's brains are organized and affected by sex hormones in different ways, resulting in differing abilities to recover from head trauma.[55] Corrigan et al. believe that women suffer more postconcussional syndrome because they are more likely

to sustain injury during abuse or physical assault and that these assaults produce greater rotational forces and, consequently, greater injury.[7]

SOCIETAL INFLUENCES

Postconcussional syndrome seems to have a societal component.[57,58] Many researchers have found that the extent and duration of injuries in the United States, where compensation is obtainable, are greater than in countries such as Lithuania, where financial compensation is less likely to occur. In Lithuania, Mickeviciene et al. sent questionnaires to 200 individuals who had suffered a concussion involving a loss of consciousness and to a comparison population who had experienced no such head injury.[57] They found that 96% of the Lithuanian respondents' headaches had completely resolved within 1 month after the concussion and that there was no difference in headaches, dizziness, or cognitive dysfunction between the respondents who had received a concussion and those who had not. The concussion group did demonstrate a significant increase in depression, alcohol intolerance, and concern about brain damage.

MALINGERING

Several studies have found that patients who are involved in litigation demonstrate more prolonged and intense symptoms from postconcussional syndrome than do similar patients not involved in litigation.[59] Proposed explanations for these findings include the possibilities that 1) litigating patients have read more about their condition and are influenced by the "symptom knowledge," 2) these patients are coached by their lawyers to magnify the "right" symptoms, 3) their symptoms are exacerbated by the stress of litigation, 4) they are being either consciously or unconsciously influenced by secondary gain, or 5) they are "malingering for money" (i.e., overtly lying).

The question of whether a particular patient is malingering is often difficult to answer since almost all of the symptoms of postconcussional syndrome are subjective. Mittenberg et al. attempted to determine the frequency of malingering by reviewing 33,531 court cases. They determined that 30% of disability cases, 29% percent of personal injury cases, 19% of criminal cases, and 8% of medical malpractice cases involved probable malingering and/or extensive symptom exaggeration. When cases that involved mild head trauma were reviewed, the authors felt

that 35% met criteria for frank malingering or symptom exaggeration.[60]

Part of the problem with postconcussional syndrome is that much of the history is based on self-report, which, in general, has been shown to be unreliable. In a study by Greiffenstein et al. of the reliability of the histories of postconcussional syndrome that are used in litigation, it was found that individuals with postconcussional syndrome inflated their scholastic performance to a greater degree than nonlitigating comparison subjects.[61] What was surprising was that individuals with postconcussional syndrome did not rate higher when given personality tests that measure malingering. This inflation in academic performance was attributed to "recall biases," such as the aforementioned "good old days" bias, and a response shift bias due to the adversarial context of litigation.

Several psychiatric and neurological tests are available to detect individuals who are malingering. A meta-analysis of data by Rogers et al. found that the most sensitive measures on the MMPI-2 for discerning malingering were the F scale, the F-K index, and the O-S interval.[62] The Halstead-Reitan battery is reported to be 93.8% reliable in detecting people who are intentionally trying to fake cognitive sequelae of head trauma.[16,63] Other scales shown to be potentially beneficial in detecting malingering are the Dissimulation Scale, the Ego Strength Scale, and the Fake Bad Scale.[4,64]

TREATMENT

Treatment for postconcussional syndrome depends on the specific symptom constellation presented by each patient.[7] One of the most effective treatment approaches is to educate the patient and his or her family about postconcussional syndrome, telling them that most patients fully recover within 6 months.[7,9,16,19,33,65] Studies have shown that delayed diagnosis and a lack of education lead to worsening of the psychogenic components of the disease and prolong recovery time.[7,16,19,33] For persistent headaches, standard headache therapy ranging from nonsteroidal anti-inflammatory drugs to prophylactic migraine medications, such as fluoxetine and verapamil, have been helpful.[20] If necessary, physical therapy and transcutaneous electrical nerve stimulators (TENS units) may provide benefit for tension headaches related to muscle stiffness.[20] Patients with psychological symptoms benefit from supportive psychotherapy, education, and some pharmacotherapies, such as antidepressants or anxiolytic medication given for a limited time.[20] Selective serotonin reuptake inhibitors are the

antidepressants of choice in most cases and may relieve headache, anxiety, tension, and depression. Dopamine agonists, psychostimulants, amantadine, and cholinesterase inhibitors have been used for treatment of decreased attention or memory and deficits in cognitive functioning, but they produce only mild benefit for most head trauma patients.[10,66–69] Physicians need to be careful in prescribing medications that may have deleterious effects on the CNS, such as phenytoin, haloperidol, barbiturates, and benzodiazepines.[10,70,71] These medications may produce significant side effects, such as delayed neuronal recovery, paradoxical rage, and further memory impairment, which may be mistaken for worsening symptoms of the postconcussional syndrome and/or may prolong the patient's recovery.[10]

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