Postconcussional Disorder: Time to Acknowledge a Common Source of Neurobehavioral Morbidity

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It is estimated that 2 million persons in the United States suffer closed head injuries annually and that of these injuries, 500,000 are serious enough to require hospitalization. Although most head injuries resulting in unconsciousness are rated as "mild" (that is, when first assessed at the site of injury such persons achieve Glasgow Coma Scale[1] [GCS] scores of 13 or better), it is also now recognized that long-term, perhaps even permanent sequelae frequently complicate even such "mild" head injuries.[2] Even head injuries without loss of consciousness[3–5] or "whiplash" have been shown to produce persistent symptoms and cognitive impairment.[6]

Although the majority of individuals who sustain a mild closed head injury (CHI) see their posttraumatic symptoms resolve within 3 months,[3,5,7,8] several studies have shown that 10% to 20% suffer some symptoms up to 6 months or longer[9–11] and that some patients may have persistent symptoms for months to years.[3,12–16] For example, a recent study of consecutive patients admitted with mild CHI and followed for 1 year showed that 1.9% to 5.8% of these patients had persistent constellations of postconcussive symptoms.[17] Therefore, postconcussional disorders may represent one of the most common etiologies of neurobehavioral disorder, especially in younger persons.

Despite increasing recognition of the extent to which head injuries produce changes in thinking, feeling, and behavior, review of the current and proposed DSM taxonomy reveals no adequate location for the signs and symptoms of postconcussional disorder. This constellation does not fit properly into the categories of delirium, dementia, or amnestic disorder. Postconcussional disorder does have some features in common with "secondary disorders due to a nonpsychiatric medical condition" as proposed for DSM-IV—for example, with the proposals involving secondary mood disorder, secondary anxiety disorder, secondary sleep disorder, and secondary personality change. However, it would often be necessary to code all of these disorders (plus additional features) to encompass what is typically conceived as postconcussional disorder.

Given the scope of the problem that head injury presents to society, it seems sensible to propose a specific category, as has been done for other disorders where the phenomenology and natural course have become better defined (such as Alzheimer's disease).

There are serious consequences to the lack of a proper diagnostic category for postconcussional disorder. First, communication among clinicians is made difficult by lack of an adequate diagnostic characterization. Second, the lack of a precise diagnostic framework has perpetuated the myth that many patients, especially those with "milder" head injuries, exaggerate their complaints after the fact either because they are "hysterical" or because they are motivated malingerers seeking some sort of compensation. Although postconcussive symptoms, once called "accident neurosis,"[18] were originally thought to be predominantly related to secondary gain, it is now clear that the prevalence of malingering was overestimated.[12,14,15,19,20] Third, research on epidemiology, natural history, phenomenology, and treatment is hampered by the use of sometimes imprecise, incomplete, or contradictory schemes by different groups of authors. The overall result of these problems has been continued stigmatization of patients who are already suffering from a neurobehavioral disorder occasioned by head injury.

To improve diagnosis and communication, we propose that DSM-IV or its successors contain the specific diagnostic category "Postconcussional Disorder." To establish criteria for this diagnosis, we reviewed the literature on head injury, with emphasis on outcomes of mild and moderate closed head injury.

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METHODS

The literature search began with a review of several recent volumes on closed head injury edited by international authorities.[2,21-23] Studies cited in these reviews that appeared likely to address the issues were then examined in detail. Additionally, a MEDLINE search covering the period 1986–1993 identified recent articles on definition of concussion and symptom profiles over time. The Results section concentrates on those studies that provide substantial illumination of the main issues—definition, symptomatology, natural history—without excessive duplication.

RESULTS

Definition of Concussion

Despite varied approaches, most studies would agree that a concussion has occurred if, after an impact to the head, a person suffers 1) a period of unconsciousness and 2) a period of reduced alertness after emerging from unconsciousness.

Concussion is relatively easy to infer following more severe head injuries characterized by unconsciousness of several hours or more and posttraumatic confusion and amnesia lasting several days. A greater challenge has been to define sequelae of milder head injuries, typically considered to be those with unconsciousness lasting a few minutes to a few hours and GCS scores > 12 on presentation to a medical team. (The GCS is a 15-point clinical rating. The lowest score (3) indicates unresponsiveness; the highest score (15) indicates normal alertness).[1] From a public health standpoint the problem of mild head injury is of great importance because the majority of head injuries fit into this category (Table 1).

The diverse approaches to defining mild head injury are illustrated by the criteria proposed in several studies summarized in Table 2. Although there is a wide range of definitions, there appears to be a consensus emerging in favor of criteria along the lines developed by a multicenter investigation reported by Levin et al.[23] (see Table 2), which defines mild head injury (concussion) as existing when the period of unconsciousness does not exceed 30 minutes, the GCS on admission is 13–15 and never falls below 13 on continued observation, and both a clinical neurologic examination and a cranial CT scan (one assumes MRI could be substituted) are normal.

TABLE 1. Prevalence of mild head injury (MHI)

Authors/Year	Place	Dates of Data	Rate MHI per 100,000	Percentage of All Head Injuries
Annegers et al. 1980[24]	Olmsted, MN	1935–1974	149	60
Rimel et al. 1981[15]	North Central VA	1977–1979	Not given	49
Kraus et al. 1989[25]	San Diego, CA	1981	131	82
Whitman et al. 1984[26]	Chicago area	1979–1980	120–284	80

Note: The importance of "mild head injury" has been quantitated in several populations and epidemiologic studies. This table has been adapted from Kraus and Nourjah (in *Mild Head Injury*, H. Levin et al. (eds), 1989).[2]

Observations from sports injuries, where immediate examination of the athlete is often practicable, have led to a proposal to further subdivide mild CHI into three grades. Grade 1 refers to confusion without amnesia or loss of consciousness; Grade 2 refers to the presence of amnesia without loss of consciousness; and Grade 3 includes injuries with loss of consciousness.[36,37] Although such a grading system is potentially useful, it is difficult to apply in the more typical clinical situation where examination and history of injury are delayed.

Symptom Profiles After Closed Head Injury

The symptoms and complaints of those with postconcussive syndrome have been long known to physicians, with references to "traumatic neurosis" going back at least to H. Oppenheim in 1889. The longer term sequelae of severe head injuries include poor memory, lack of spontaneity, irritability, restlessness, fatigue, childishness, and emotional lability.[38,39] Common persistent mild CHI symptoms include headaches, dizziness, fatigue, concentration and memory impairment, irritability, and depression.[3,12] Headache is the most common symptom after mild CHI, and patients with headaches are more likely to have associated symptoms of dizziness, impaired concentration, depression, and anxiety.[40] A review of 12 studies by Bohnen et al.[41?] noted a prevalence of postconcussive symptoms ranging from 16% to 49% at 6 months and from 1% to 50% at 1 year.

Some symptoms may be more prominent after mild than after severe injury. For example, Alexander[42] prospectively examined 35 cases of closed head injury more than 6 months after injury and found that mild CHI cases had significantly greater prevalence of severe chronic headache and depression compared with severe CHI cases. Uomoto[43] found that 89% of mild CHI outpatients had chronic headaches compared with 18% of moderate to severe CHI patients at about 2 years after injury. Some authors have suggested that early postconcussive symptoms are mostly somatic ones, such as headache, dizziness, nausea, drowsiness, fatigue, and intolerance to environmental stimuli, whereas the later manifestations include more psychological symptoms, such as depression, anxiety, and irritability.[3,41,44]

Symptom Profiles Over Time

Symptoms once established tend to persist. For example, Alves[12] found that at 6 months after injury 30% reported headaches, 17% memory problems, 14% dizziness, and 8% weakness. These and other symptoms did not change in frequency at 1 year after the injury. There was substantial correlation between symptoms reported at 3 and 6 months and between 6 and 12 months.

This conclusion is supported by several studies that examined subjects at different times after injury. The results are surprisingly similar even though the time frames varied from one month to 3–5 years after concussion. The results from several illustrative studies are provided in Tables 3 and 4.

In summary, headache, dizziness, and concentration and memory problems appear to be the most common symptoms following mild CHI. Although in many patients such symptoms resolve and do not recur, in some patients they do indeed persist.

Cognitive Disturbance

Neuropsychological testing has attempted to define the cognitive sequelae of mild CHI more precisely. Data from such studies are important in that they provide relatively objective indicators of persisting postconcussive neurocognitive impairments. The presence of cognitive deficits following moderate and severe head injury is beyond dispute;[21,22] therefore, our review focused on evidence of persisting neuropsychological impairment following mild head injuries.

Most studies we reviewed indicated that neuropsychological deficits could indeed be demonstrated. Most commonly, impairments were noted on measures of choice reaction time[5,45] and speeded information processing (for example, on Paced Auditory Serial Addition Test),[27,46] as well as on memory and more general neuropsychological functions.[29,34] Ewing et al.[47] found deficits in divided and selective attention in patients symptomatic at 6 months compared with both asymptomatic and control subjects. Ewing et al. showed that under conditions of induced hypoxic stress, mild CHI patients performed worse than control subjects on neuropsychological tests, despite having no subjective complaints 1 to 3 years after injury. Williams et al.[32] found that degree of neuropsychological deficit was related to GCS score and presence of cerebral complications. Those with GCS scores of 9 to 12 had more neuropsychological deficits; also, those with GCS scores of 13 to 15 but with depressed skull fractures or brain lesions were more impaired.

TABLE 2. Examples of criteria for mild head injury

Study	Criteria
Gronwall & Wrightson 1974[27]	Head trauma with posttraumatic amnesia < 24 hours.
Minderhoud et al. 1980[28]	Unconscious < 30 minutes and posttraumatic amnesia.
Rimel et al. 1981[15]	Cranial trauma with loss of consciousness, 20 minutes, and admission. GCS ≥ 13; hospitalization < 48 hours.
Barth et al. 1983[29]	Period of unconsciousness not exceeding 20 minutes; GCS ≥ 13 at admission; hospitalization < 48 hours.
Edna et al. 1987[30]	One of the following: 1. Loss of consciousness following head trauma. 2. A skull fracture. 3. Development of an intracranial hematoma.
Jakobsen et al. 1987[31]	A period of complete amnesia no matter how short, but maximum of 24 hours, caused by a blow to the head.
Levin et al. 1987[23,a]	Loss of consciousness < 30 minutes; GCS ≥ 13 on hospitalization; no decrease of GCS to < 13; normal CT; normal neurological exam.
Williams et al. 1990[32]	Uncomplicated closed head injury: mild impairment of conciousness, GCS ≥ 13. Mild CHI: complicated by brain lesion or depressed skull fracture. Moderate CHI: GCS 9–12, unconciousness ≤ 20 minutes.
Leininger et al. 1990[33]	Confusion, disorientation or LOC < 20 minutes, GCS ≥ 13, no deterioration or neurosurgical intervention.
Bohnen et al. 1991[34]	PTA, 60 minutes, LOC, 15 minutes, GCS = 15, absence of focal neurological deficit.
Kay et al. 1993[35,b]	Any period of loss of consciousness. Any loss of memory for events immediately before or after the accident. Any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused). Focal neurologic deficits, which may or may not be transient, when the severity of the injury does not exceed the following: 1) Loss of consciousness of approximately 30 minutes or less; 2) after 30 minutes, an initial GCS ≥ 13; 3) posttraumatic amnesia ≤ 24 hours.
Middelboe et al. 1992[11]	Unconciousness 1 hr, PTA < 24 hours, no signs of contusion or suspicion of hemorrhage.

Note: PTA = posttraumatic amnesia; GCS = Glasgow Coma Scale; CHI = closed head injury; LOC = loss of consciousness.
[a]A recent three-center study using the criteria of Levin et al.[8] has gone far in creating a working consensus as to the definition of a concussion.
[b]Developed by the Mild Closed Head Injury Subcommittee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine.

TABLE 3. Postconcussional symptoms

Study/Follow-up/Symptoms	*n*	%
Jakobsen et al. 1987[31] (*N* = 55)		
At 1 month, at 3 months		
Headache		29, 15
Fatigue		29, 5
Impaired concentration		16, 9
Vertigo		11, 5
Irritability		9, 2
Rutherford et al. 1979[44] (*N* = 131)		
At 1 year		
Headache	11	8.4
Irritability	7	5.3
Anxiety	5	3.8
Depression	2	1.5
Insomnia	3	2.3
Fatigue	3	2.3
Loss of concentration	4	3.1
Loss of memory	5	3.8
Amnesia	2	1.5
Diplopia	1	0.8
Visual defect	3	2.3
Hearing defect	2	1.5
Dizziness	6	4.6
Epilepsy	0	0
Sensitivity to alcohol	0	0
Others	1	0.8
Middelboe et al. 1992[11] (*N* = 28)		
At 1 year		
Headache	9	32
Dizziness	7	25
Memory deficit	7	25
Concentration deficit	7	25
Fatigue	6	21
Irritability	6	21
Anxiety	5	18
Sleep disturbance	5	18
Sight disturbance	5	18
Alcohol intolerance	3	11
Gastrointestinal symptoms	3	11
Hearing disturbance	2	7
Disturbance in smell	1	3
Tactile disturbance	1	3
Edna et al. 1987[30] (*N* = 485)		
At 3–5 years		
Headache	113	23
Impaired memory	99	20
Dizziness	90	19
Fatigue	89	18
Irritability (noise and light)	88	18
Impaired concentration	68	14
Insomnia	65	13
Tinnitus	61	13
Hearing defect	51	11
Depression	45	9
Anxiety	37	8
Double vision	12	2

Studies of patients with mild CHI without loss of consciousness have also shown deficits. Hall[48] studied 22 patients an average of 1 year after injury; 16 (73%) had suffered a closed head injury without loss of consciousness. He reported significant serial-position effects suggesting memory deficits. Leininger et al.[33] studied mild CHI patients who reported continued symptoms at 1 month to 2 years following injury. Among symptomatic patients 1 to 22 months after injury, neuropsychological impairment was not significantly different in those who had suffered loss of consciousness with the head injury compared with those who had suffered only dazing and disorientation without loss of consciousness.

Although the results of the above studies represent what appear to be an emerging consensus that there are sequelae to mild CHI, some disagreements are noted. For example, Gentilini et al.[7] could not distinguish mildly head-injured patients from control subjects on a series of tests that assessed attention, memory, and intelligence. And Dikmen and Temkin[49] have cautioned that the effects of confounding factors such as alcohol/drug use, prior injuries, and other medical problems may be difficult to sort out from the effects of a mild head injury. Nevertheless, the preponderance of evidence favors the conclusion that concussed persons are at risk of developing cognitive disturbances.

Personality Test Changes

Several studies have used the Minnesota Multiphasic Personality Inventory (MMPI) in attempts to characterize emotional adjustment after head injury. Mild CHI patients examined 3 months after injury showed significant psychological distress on MMPI that was similar to that seen in a comparison group of patients with long-standing neurologic damage.[50] A minor head injury group scored substantially higher than a severe head injury group on MMPI scales 1, 3, and 7, suggesting problems with somatic preoccupation and concentration difficulties.[33] Bornstein et al.[19] found that 25% of head-injured patients had moderate emotional adjustment problems characterized by anxiety, withdrawal, depression, and concern over somatic function when tested an average of 2.5 years after injury. There was no clinically significant relationship between severity of injury and personality adjustment.

Effects of Mild Closed Head Injury on Life Functioning

Although cognitive deficits following mild CHI may be subtle, they may be sufficiently disabling that patients may have difficulty returning to or maintaining employment; patients may also experience disruption in social life and an increased incidence of depression and somatic

TABLE 4. Symptom patterns of adult patients (experiment patients)[a]

Description	Percentage of Patients With Symptoms							
	At Discharge[b]		At 3 Months[b]		At 6 Months[b]		At 12 Months[b]	
	$N = 587$	$N = 569$	$N = 587$	$N = 360$	$N = 587$	$N = 314$	$N = 587$	$N = 189$
No symptoms reported	31.5	32.5	22.8	37.2	30.2	56.4	19.2	59.8
Headaches	26.9	27.8	8.2	13.3	6.5	12.1	2.9	9.0
Memory deficit	4.3	4.4	1.2	1.9	1.7	3.2	1.4	4.2
Dizziness	2.6	2.6	2.2	3.6	0.8	1.6	0.3	1.1
Weakness	1.9	1.9	0.5	0.8	0.3	0.6	0.0	0.0
Numbness	0.8	0.9	1.2	1.9	0.8	1.6	0.0	0.0
Nausea	0.8	0.9	0.5	0.8	0.2	0.3	0.0	0.0
Diplopia	0.2	0.2	0.8	1.4	0.2	0.3	0.0	0.0
Tinnitus	0.0	0.0	0.5	0.8	0.5	1.0	0.5	1.6
Hearing deficit	0.2	0.2	0.5	0.8	0.7	1.3	0.3	1.1
Headaches and others[c]	7.2	7.4	4.3	6.9	2.2	4.1	1.0	3.2
Dizziness and others[c]	1.4	1.4	1.0	1.7	0.5	1.0	0.3	1.1
Memory and others[c]	0.5	0.5	1.2	1.9	0.7	1.3	0.5	1.6
Headaches, dizziness, and others[c]	9.0	9.3	5.6	9.2	2.4	4.5	1.5	4.8
Headaches, memory, and others[c]	6.1	6.3	3.6	5.8	2.4	4.5	1.5	4.8
Dizziness, memory, and others[c]	0.7	0.7	1.0	1.7	0.5	1.0	0.3	1.1
Headaches, memory, dizziness, and others[c]	1.9	1.9	4.4	7.2	2.0	3.8	1.9	5.8
Others[c]	0.7	0.7	1.7	2.8	0.8	1.6	0.3	1.1
Undetermined	0.3	0.4	0.0	0.0	0.0	0.0	0.0	0.0
Unknown	3.1	—	38.7	—	46.5	—	67.8	—

[a]Source: Alves et al;[17] p. 49. Reprinted with permission of the authors and Aspen Publishers, Inc.
[b]First column under each assessment time: distribution of symptom patterns including those "unknown" due to nonreturn for follow-up. Second column: adjusted percentages, excluding "unknown."
[c]"Others" refers to symptom patterns not involving headaches, dizziness, or memory.

illness.[51] In a consecutive series of young adult men, 68% of mild CHI patients still reported one or more symptoms after returning to work.[52] Rimel et al.[15] showed that only 34% of mild CHI victims who were employed at the time of injury had returned to work at 3 months. In another series, about 10% of mild CHI victims who were employed at the time of the injury were unemployed at 1 year after the injury.[9,30] This study also found that those who were unemployed at the time of follow-up had more frequent complaints of impaired concentration, anxiety, insomnia, and double vision. Individuals with mild CHI also had worse social outcomes at follow-up, and this correlated with posttraumatic symptoms.

Thus, although data remain fragmentary, there are indications that individuals with mild CHI have decreased social and occupational functioning at follow-up, and these decreases in functioning may be correlated with posttraumatic symptoms.

Posttraumatic Seizures

Early seizures (within 7 days of injury) occur in about 5% of patients with closed head injury and are particularly common in patients with prolonged posttraumatic amnesia (PTA), depressed skull fracture, or intracerebral hemorrhage.[53] Late posttraumatic seizures occur in about 12%, 2%, and 1% of severe, moderate, and mild adult CHI patients, respectively.[24] Factors predicting late seizures include seizures in the immediate postinjury period, the presence of intracranial hematoma, and depressed skull fracture.[53] In a study of 96 consecutive outpatients referred for neuropsychological testing after minor CHI, 17 reported partial complex seizure–like symptoms.[54] Only 4 of these patients had abnormal standard EEGs. Partial kindling of limbic structures may help to explain this phenomenon. Seventy-one percent of these symptomatic patients reported either dysphoric spells or suicidal ideation. Of the 10 who were treated, 7 had moderate or substantial improvement with carbamazepine or valproic acid alone, and two improved with the addition of fluoxetine for affective symptoms. The incidence of posttraumatic seizures in children less than 5 years old is significantly higher than in the adult population and does not usually lead to a long-term seizure disorder.[55]

Pathological Evidence of Mild Closed Head Injury

Animal models suggest that there may be distinct neuropathologic and neurophysiologic damage following mild CHI.[56,57] Monkeys experiencing minor head injuries (acceleration-deceleration nonimpact injuries) were found to have degenerating axons in the inferior colliculus, pons, and dorsolateral medulla 7 days after injury.[58] Povlishock and Becker[59] performed studies on a concussive model in cats, using sham controls. These studies demonstrated that although the mild concussive injury produced no focal parenchymal hemorrhages, contusions, or lacerations, there was distinct axonal change, beginning with intra-axonal peroxidase pooling and leading later to lobulation and then to cloudlike swellings, suggesting axonal separation from distal axonal segments.

In humans, Oppenheimer[60] published autopsy results of brain-injured patients. In 5 patients who suffered mild concussion and died of other causes, Oppenheimer found microglial clusters, evidence of "microscopic destructive foci" that he felt were inflicted on the brain by what were regarded as trivial injuries.

Brain Imaging and Electrophysiological Evidence of Mild Closed Head Injury

Several studies have documented magnetic resonance imaging (MRI) abnormalities in individuals suffering mild CHI.[61] These range from scattered punctate hyperintensities, suggesting axonal shearing, to more obvious cranial lesions such as contusions and hematomas. Some workers classify patients based on the presence or absence of significant lesions (contusions and hematomas) because mild CHI complicated by an intracranial lesion results in greater symptoms, neuropsychological deficits, and disability than mild CHI with no intracranial lesions.[32] Some have argued that any injury with a contusion or hematoma on brain imaging should not be classified as a mild head injury. Most of the focal lesions found on MRI in mild CHI patients are localized in the frontal and temporal regions, where their presence tends to correlate with neuropsychologic and behavioral abnormalities.[62,63] As the lesions resolve, so do the neurobehavioral abnormalities.

Although standard EEG findings do not usually correlate with postconcussive symptoms, one series of 280 children did show that 42.5% had abnormal EEGs following mild CHI.[64] History of loss of consciousness appeared to correlate with EEG abnormalities.[65]

Functional imaging studies are still uncommon. However, Humayun[66] detected decreased glucose metabolism using fluorodeoxyglucose positron-emission tomography in 3 cognitively impaired patients following mild CHI. Evoked potential abnormalities may follow head injury. For example, Montgomery[67] reported that one-half of 26 patients with mild CHI had abnormal brainstem auditory evoked potential (BAEP) immediately after injury, and 4 had abnormal BAEP that persisted for 6 months, suggesting persisting brainstem disturbance.

DIAGNOSTIC RECOMMENDATIONS

The work we reviewed, as well as the work on more severe head injuries, suggests that three clusters of symptoms commonly occur. There is a *somatic* cluster that can include headache, fatigue, dizziness (or vertigo), and visual or hearing impairments; a *cognitive* cluster that may include difficulties with concentration and memory; and an *affective* cluster that may involve irritability, anxiety, and depression—and, in the more severely injured, aspontaneity, lability, childishness, and social isolation.[38,39]

We recommend that DSM-IV or its successors adopt a diagnostic category titled "Postconcussional Disorder." Criteria for this disorder are presented in Table 5.

Criterion Set A seeks to establish that an injury sufficient to cause *commotio cerebri* has taken place. It might be argued that Criteria A.1. and A.2. should be more strict (for example, loss of consciousness for 30 minutes and PTA lasting 24 hours). In recommending the criteria as we do, we attempt to balance between momentary, fleeting loss of consciousness with short amnesia (that is, symptom complexes that might be difficult to diagnose reliably, risking Type I error), and missing significant

TABLE 5. Proposed criteria for postconcussional disorder

A. History of head injury that includes at least two of the following:
   1. Loss of consciousness for 5 minutes or more.
   2. Posttraumatic amnesia of 12 hours or more.
   3. Onset of seizures (posttraumatic epilepsy) within 6 months of head injury.

B. Current symptoms (these must be either new symptoms or substantially worsening preexisting symptoms) to include:
   1. At least the following two cognitive difficulties:
      a. Learning or memory (recall).
      b. Concentration.
   2. At least three of the following affective or vegetative symptoms:
      a. Easy fatiguability.
      b. Insomnia or sleep/wake cycle disturbances.
      c. Headache (substantially worse than before injury).
      d. Vertigo/dizziness.
      e. Irritability and/or aggression on little or no provocation.
      f. Anxiety, depression, or lability of affect.
      g. Personality change (examples: social or sexual inappropriateness, childlike behavior).
      h. Aspontaneity/apathy.

C. Symptoms are associated with a significant difficulty with maintaining premorbid occupational or academic performance or with a decline in social, occupational, or academic performance.

concussive events through excessively restrictive criteria (Type II error). We acknowledge that the criteria chosen (> 5 minutes loss of consciousness and > 12 hours posttraumatic amnesia) are arbitrary. However, we contend that these criteria, and/or posttraumatic epilepsy, represent good evidence of *commotio cerebri.*

Criterion Set B represents an attempt to look at the cognitive disturbances or affective and vegetative symptoms that are found to be frequent following head injury and that generally occur only following significant head injuries. In addition, we include the requirement that the cognitive difficulties or affective and vegetative symptoms must be new or must have significantly increased since the CHI.

Criterion C parallels similar criteria proposed for other cognitive disorders in DSM-IV, namely, that the symptoms must produce some social-occupational disability.

SUMMARY

Closed head injuries are common, affecting approximately 2 million people annually in the United States. The majority of these are "mild" in the sense of not being associated with prolonged unconsciousness, intracranial bleeding, skull fracture, or protracted periods of confusion. Yet a proportion of such "mild" injuries are accompanied by persisting cognitive, vegetative, and affective-behavioral sequelae, some of which affect day-to-day life.

We argue that there is sufficient research to indicate that postconcussional symptoms occur and that they tend to have a predictable configuration. It is necessary to recognize the existence of "Postconcussional Disorder" in our nosology in order to provide more prompt diagnosis and management and to facilitate scholarly communication and research regarding this important neurobehavioral disorder.

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