CLINICAL ELECTROENCEPHALOGRAPHY ©1991 VOL. 22 NO. 4

Neurodiagnostic Testing in Patients with Closed Head Injury

Peter G. Bernad

Key Words

Closed Head Injury, Types I & II
Concussion
Electroencephalography
Electronystagmography
Evoked Potentials
Neuropsychological Testing
Postconcussion Syndrome

Head injury is a major aspect of health care and a considerable drain on overall resources in the United States and other industrialized nations. In 1968, there were approximately three million motor vehicle accidents.[1] Head injury was the most frequent lethal injury in such accidents. Up-to-date figures for the 1980s indicate that more than ten million Americans sustain head injury annually, the majority of whom suffer "closed head injury" (CHI). [CHI is divided into mild, moderate and severe. Post-concussion syndrome (PCS) frequently follows a mild type of CHI.[2]] Available figures indicate that out of all the head injuries in 1980, there were 160,000 deaths. Approximately 66 percent of the total number were caused by accidents involving motor vehicles.[2] The majority of the more recent studies have focused on moderate and severe head injuries. In the last few years nationally, advocacy organizations have arisen to help families and survivors as well as to teach professionals regarding head injury.

Most of the concern, research, financial help, and emphasis has been toward the profoundly head injured and severely damaged individual. Mild CHI has received far less attention, and virtually little or no experimental studies have been published on this particular subject. Recently a Sc.D. dissertation[3] dealt with the treatment issues of patients with CHI and PCS and explored the possibility of the effects of progressive relaxation on reduction of related symptoms following mild CHI. The purpose of this article is to review mild CHI. With the typical history and normal "neurologic examination," the patients still experience significant behavioral problems, emotional problems, and cognitive difficulties and are unable to pursue their vocational interests or avocation. Total financial costs for evaluation, treatment and rehabilitation are estimated in the billions of dollars. These injuries often occur to those who are young with a potentially long life span with long-term disability. Indeed, accidents are the leading cause of death for persons ages 1 through 44 years, whether black or white, male or female. Society loses a significant portion of the young to premature death from head injuries associated with motor vehicle accidents and accidents of other kinds.

In the experience of this author over the last 10 years, head injuries, secondary to motor vehicle accidents, are by far the most common, accounting for approximately 90 percent of 1,000 patients seen with CHI. These individuals had not only mild CHI but also associated cervical muscle spasm and frequently diffuse spasm of the lumbosacral spine, thoracic spine, as well as the cervical spine and experienced acceleration-deceleration injury (whiplash-type injury). Approximately 7% of the 1,000 patients have had falls, either from a window, bridge, building, ledge or ladder, and 3% have occurred secondary to sport injuries and falls from bicycles, or interpersonal trauma, such as fighting or being hit by a rock or a ball, for example, a golf ball. Head injuries are the number one health concern that causes the most useful time lost or more functional time lost from work and from enjoyment of life and participation in society. The reason for this is readily apparent because accidents and, specifically, mild closed head injuries, strike those

Requests for reprints shoulld be addressed to Peter G. Bernad, M.D., M.P.H., F.A.C.P., Neurentox International, 2112 F Street NW, Suite 303, Washington, D.C. 20037

who are young, generally healthy, and frequently employed.

Recently, major newspapers have raised public concern by referring to mild CHI and PCS as "a silent epidemic of the 1980s." (*Wall Street Journal,* Nov. 14, 1984). PCS has become a topic of increasing concern to neurologists, neurosurgeons, and neuropsychologists.[4] Indeed, the first national conference on mild CHI was held only in 1987.[5] Most physicians and lay people have no formal knowledge concerning mild CHI. Physicians do not receive adequate medical training regarding minor head injury and regarding PCS; nonetheless, this topic is a significant public health concern. Patients frequently go from doctor to doctor without diagnosis. These same patients are inappropriately treated without any scientific rationale and are frequently discharged to the care of general practitioners, orthopaedic surgeons and psychiatrists. They are frequently thought to have "litigation neurosis" or "depression and neurotic tendencies."[6] The medical community is only now waking up to this major burden of head-injured individuals who survive motor vehicle accidents and other forms of trauma and continue to have significant syptoms with minimal-to-no findings on general neurologic exam or diagnostic testing. However, it is important to emphasize that various other diagnoses, such as fictitious illness, hysteria, malingering, somatiform disorder and personality disorder, exist and are commingled in this population of patients with mild to moderate CHI and PCS. The expert neurologist needs to know how to separate and tease out the real patients from those who have some form of secondary gain, financial, emotional or social.

The typical patient that this author has seen over the last 10 years with mild CHI and with subsequent PCS is usually a young male, who was involved in a motor vehicle accident in which he was the driver. Occasionally, the patient was a passenger and sometimes the patient was a pedestrian. The trauma itself may involve a "fall," a "blow to the head," and in a majority of cases, as mentioned, the head strikes a stationary object, such as a windshield or a mirror or a side window in the motor vehicle accident. The average patient usually experiences, along with the head injury, neck and back injuries as well.[7,8] The neck injury has been referred to in the past as whiplash injury.[9,10] Mild CHI with persistent symptoms may occur after a severe neck injury without the head actually striking any surface. The symptoms are worse when there is a rotational component to the head injury in addition to a to-and-fro movement of the head.

Loss of consciousness is not necessary, but there is always an alteration of consciousness. If one asks the patient carefully in obtaining a history, the patient will usually report a brief loss of consciousness or an alteration of consciousness, or describes being "dazed" or "seeing stars." Sometimes the patient is confused or agitated for some period of time after a closed head injury. The author has found that patients are often alone in a car, for example, where there are no witnesses who can attest to loss of consciousness. The patient frequently has a posttraumatic amnesia and cannot recall exactly the events immediately after the head injury, and sometimes may have antegrade amnesia as well. The usual situation is one in which if loss of consciousness occurs, it commonly lasts less than an hour. The patient may or may not be admitted to a local hospital. If admitted, it is often for less than 48 hours. On the Glasgow Coma Scale (GCS), the score is usually between 13 and 15. The Glasgow Coma Scale is useful as a predictor of coma and also for prognosis and for categorizing the patient in terms of the severity of the closed head injury.[11] The majority, and most severe, of the symptoms occurs immediately after the trauma and tends to improve over a period of days, weeks and months.

It is useful to consider the type of injury that persons have when they have a closed head injury. In this author's office experience, closed head injury may be divided into diffuse and focal types. The diffuse head injury is by far the most common. The patient with diffuse CHI often has had a mild or limited period of concussion with loss of consciousness. In this particular condition, usually the physical exam and, specifically, the neurologic exam are normal. Frequently, routine laboratory studies, including computerized tomographic scanning of the brain, are normal; nevertheless, ample documentation in the literature from animal experimentation has shown that even minor blows to the head will result in shear strain and tearing of axons diffusely.[12] This is usually widely scattered in the cortex and results from acceleration/deceleration injury to the brain.

With the injury, there is stretching and tearing of the axons. This represents an organic disruption of the brain, which can most often only be documented microscopically. Recently, use of newer techniques of brain imaging, such as magnetic resonance imaging, have raised the question of possible identification of subtle lesions in the cortex,[13] for example, unidentified bright objects (UBOs). Frequently, the microscopic stretching and the tearing that result from mild CHI are secondary to mechanical forces that are transmitted to the brain following trauma to the head.[14] This is best explained by using the concept of the brain as a relatively soft material with a consistency of jelly, in a bowl frame that is actually the skull with various areas of ridges and hard projections. When the head is hit suddenly or is shaken, there are mechanical forces that are transmitted to the brain.

Recent evidence also supports the concept of regeneration via axonal sprouting.[15] It has been the experience of this author that more symptoms are identified and the patient is less functional when there is not only a direct force but a rotational force, which places a torque on the brain stem. The most remarkable findings tend to be in the tearing of the axons. One of the explanations of the loss of consciousness is the interruption of fibers in the reticular activating system in the brain stem.[16] The stretched fibers may be permanently damaged but occasionally are merely impaired as, for example, in abnormalities having to do with axoplasmic flow or merely biochemical changes that are potentially only functional and undergo some repair.

The other type of abnormality is focal CHI in which one particular part of the brain is traumatized. This sometimes is identifiable on computerized tomographic scanning or magnetic resonance imaging. The two major areas where focal CHI is seen in highest frequency include the frontotemporal area lesions and coup/countercoup lesions. Focal lesions occur in the frontotemporal areas because of uneven ridge-like surfaces in the frontal and basal portions inside the skull. Both the undersurfaces of the frontal lobe as well as the areas of the temporal lobes are most vulnerable in the skull because of their proximity to rough edges. From the experience of this author, there is frequently a combination of diffuse CHI with focal CHI. The coup/countercoup occurs when there is a sharp blow to the head, that dents the skull inward bruising the brain immediately proximal to the skull, then sends a moving brain to hit the side of the skull that is immediately opposite where additional bruises occur. This kind of injury is also associated with diffuse CHI. There is additional evidence showing that there are small petechial hemorrhages that occur in the brain stem after a mild head injury in animals.[14]

Long recognized and established neurologic syndromes, such as seizure disorders and post-traumatic epilepsy, may arise following even a relatively mild CHI.[17–20] In childhood, a specific EEG pattern may be seen after mild CHI.[21,22]

The EEG lesser known aspects of CHI will be emphasized in this communication. This paper defines mild CHI with PCS as injury that is sustained without causing either a skull fracture, or clinically demonstrable laceration to the brain, or hemorrhage that is identifiable on computerized tomographic scan of the brain or magnetic resonance imaging. In this condition, the patients seemingly recover after a short period of unconsciousness or after an altered state of consciousness. Part of this definition, as mentioned earlier, includes either no hospital stay in a majority of patients or a limited hospital stay of less than 48 hours with a Glasgow Coma Scale between 13-15. Also, these patients have some element of posttraumatic amnesia, which may be antegrade or retrograde, but most often is antegrade or posttraumatic amnesia.

Mild CHI is increasing in the most developed nations, such as the United States, and even in those countries that have been considered less developed but have an increasing number of motor vehicles. A recent example is China. Newly-industrialized nations are experiencing the same epidemic of mild CHI as the United States.

Many of these patients have persistent symptoms beyond the expected recovery period. Twenty-five to 38 percent of the patients develop significant emotional disabilities which further limit their recovery from PCS.[23] The expected recovery period is usually approximately 1 month but may be as long as 5 months. The symptoms include headaches; 100 percent of the patients studied have had symptoms of headaches. Cervical, thoracic and lumbar pain, dizziness, tinnitus, a sense of imbalance, occasional loss of libido, impotence, and abnormal menstrual periods also occur. Some of these patients have loss of smell, taste, visual symptoms or numbness in the arms and legs.

Patients have also reported photophobia and phonophobia. Memory problems have been described mostly in terms of short-term memory but occasionally involve intermediate memory loss, and periods of amnesia. Some of the remarkable symptoms occur in terms of changes in cognition, mentation, behavior, attention and concentration. Patients with diffuse mild CHI frequently cannot process information as quickly as those who have not had a head injury. These patients seem to react less quickly, especially when faced with a choice. They have a reduced capacity to process large amounts of information, almost as if they are overloading their integrative system for information processing. These same patients have difficulty with attention span and shifting attention among tasks, and in carrying out complex functioning that requires multiple, simultaneous decisions and choices. As is often noted, these patients do well until they attempt to get back to work, especially back to highly complex jobs. Problems within their thinking processes in terms of flexibility and difficulty with shifting from one task to another, as well as with learning new routines, are often noted. The patients with frontotemporal focal lesions have primary deficits in learning and memory, planning and organization, and have problems with emotional control. Injury to the frontal lobe is associated with an abnormality of "executive functioning," which is a process by which the patients can plan and organize and adjust their thinking and behavior. Some patients are described as having actual changes in personality, such as having a "short fuse," being highly irritable, and "losing their cool" or "blowing up."

These symptoms are part of the syndrome that has been termed PCS. However, when the sypmtoms continue beyond the expected period of recovery, then a new and separate designation of PCS may be considered. This should be done for not only heuristic purposes but also for prognostication. For this reason and for clarity, PCS should be divided into two categories.[24] This author wishes to use the terminology of Type I PCS to refer to a benign condition of limited duration with patients having symptomatology for up to a few months, as part of the mild CHI experience. The prognosis is excellent, the patient returns to good functioning, and the ideal situation is noted in which there is no permanent, observable impairment. These patients may be termed completely recovered. Unfortunately, there are other patients, so-called Type II PCS, which represents those patients who have persistent symptoms past a 5 month period. These patients need proper diagnoses, best done by a neurologist, to exclude potentially more serious intracranial processes. This entity is "PCS associated with mild CHI" that occurred prior to or triggered the disease. Many of these patients, unfortunately, never recover their premorbid state and frequently do not go back to being completely functional. They simply do not have complete recovery. Many patients fall in between. Some patients notice minor problems with memory and problem solving but are able to do some work and to adapt to the changing situation. In Type II PCS, patients have very significant psychological problems, and there are neuropsychologic changes, which are documentable on testing. These patients often lose their jobs or terminate voluntarily. They have persistent symptoms of headache, dizziness, ringing in the ears, numbness, tingling, paresthesias, and go on to have multiple subjective symptoms with minimal-to-no neurological findings. These same people have often a persistent low level of social and vocational achievement and are the "walking wounded" of civilized life because they do not appear to have objective evidence of injury. Posttraumatic stress disorders may develop. Because evidence of neuropathology is unclear, that is, from neurodiagnostic testing, they frequently incur psychiatric diagnoses. Only after a detailed history and neurodiagnostic assessment with recognition by an experienced neurologist will the true diagnosis come to light.

A multitude of health care providers often need to be involved in treating patients with Type II PCS; e.g., a team that would include a neurologist, psychiatrist, neuropsychologist, vocational rehabilitation counselors, social workers, physical therapist, and other specialists. The legal establishment and insurance companies frequently become involved, and on occasion, they may even be helpful. It is to be emphasized that many factors play a role in the recovery process for these patients, including psychological, social and financial factors. One of the key features of the Type II PCS is that the patients seem to have a maladaptation to stress as it relates to the injury, are unable to cope appropriately and suffer consequences. They also seem to lack spontaneity and a sense of

humor and have long-term cognitive impairment. These patients develop significant emotional, interpersonal and behavioral problems. There are certainly elevated anxiety levels because of loss of confidence since they begin to doubt themselves. There is also a negative feedback loop in which their symptoms are not validated and are not recognized to be part of a head injury process. These patients experience increased stress at a time when they have decreased ability to cope with the stress, including the stress of everyday existence, working and interacting with family and friends. Treatment modalities should include a broad-based approach. Medication should be used judiciously. Physical therapy helps the soft tissues of the cerival region. Progressive relaxation, biofeedback techniques, are useful to alleviate stress related symptoms.

There is now substantial evidence from animal and human research to conclude that patients with PCS, both Type I and Type II, have varying degrees of cortical dysfunction that involves intellectual skills, memory, speed and rate of information processing, judgment, attention, and concentration. It is to be emphasized that from 25 to 38% of these patients develop significant emotional disabilities[23] that further limit their recovery from PCS, especially Type II.

There are subjective phases of "concussion," of seemingly minimal symptomatology (dazed reaction to symptoms where the patients indicate that they have "seen stars"). There is a natural continuum from slight alteration of awareness to actual loss of consciousness. Frequently, a loss of consciousness is not remembered by the patients because of post-traumatic amnesia.

Impairment of cognitive and intellectual functioning has been well-demonstrated and documented following CHI and PCS; intellectual functioning evaluated hours after concussion has been found to be abnormal, especially in measures of speed of thought and excessive fatigability of the patients.[23,24]

Children have been studied in CHI and PCS, and found to have significant impairment of visual, spatial, somatosensory, and language functioning.[25] Paced auditory serial addition test (PASAT), sensitive to the rate of information processing, has been found to be abnormal in these children.[26] Results have shown significant reduction in the capacity to process information rapidly in both children and adults.[27] The deficits in information storage and retrieval are exacerbated by subsequent injuries. There is a cumulative process that occurs when more than one closed head injury has occurred. After a head injury, for example, in a motor vehicle accident, should the victim then suffer a second head injury, soon thereafter there will be an additive component, a compounding effect or aggravation of a preexisting condition. The patient never seems to "return to the baseline state." There is always a scar that is left, even though current techniques in neurodiagnostic testing may fail to demonstrate this fact. A recent publication documented the importance of objective neurophysiologic evaluation of patients with mild CHI and PCS.[28]

What about studies and research on closed head injury? Previous research has documented the usefulness of dividing injuries to the head and neck into two categories. In one particular study, an assessment was made of 1500 patients who underwent neurodiagnostic evaluation after head and neck injury.[29] They were divided into two categories. The first group of patients had prevalent cerebral symptoms, consisting of headaches, memory problems, changes in cognition with little sensory organ symptoms. These patients were frequently referred to psychiatrists. Usually, neuropsychologic testing was recommended. The other category of patients fell into a group best described as "neck and sensory organ damaged individuals." These patients had more of a hyperextension-flexion injury of the neck of whiplash injury, and complained of dizziness, disorientation in space, visual changes and numbness. Neurodiagnostic testing was recommended for those patients. This type of injury frequently resulted in what the authors have described as "cervical vertigo." The neurophysiologic testing that proved useful was the electronystagmogram.[30] In the author's laboratory, a similar study has documented the usefulness of electronystagmography.[31] Other testing involved full audiologic evaluation and evoked responses. Sixty-five percent of the referred patients had one or other abnormalities, including "latent nystagmus," not seen at bedside because of visual fixation, abnormal caloric responses with canal paresis and directional preponderance as well as asymmetric responses. When taken together, over 90 percent of the referred patients had some abnormalities on the electronystagmograph. This

compares favorably to other studies recently published. It is useful to separate patients into categories since it allows more predictive diagnostic testing as well as indicating prognosis. This author feels that the Type I CHI with PCS is most like the "cervical vertigo" patient described in the literature. The Type II CHI with PCS is most like the one with prevalent cerebral symptoms.

What is the etiology of these symptoms and what is specifically the pathophysiology of the neurophysiologic changes? Multiple theories can be postulated including:

A. A possible disruption secondary to trauma or input from muscles and joints on one side of the neck. In the abnormalities in joints, such as the atlanto-axial joint, damage may also occur and may result in some of these symptoms.
B. Cervical sympathetic ganglia disruption or sympathetic chain disturbance. The abnormality in the sympathetic chain disturbance is also known as a Barre-Lieou syndrome.
C. Vertebral artery spasm with variable presentation because of deviations in congenital vascular distributions. It is conceivable that the vertebral artery may go into spasm for a variable length of time and usually is reversible.
D. Petechial hemorrhages in critical brain stem cortex: there is some animal experimentation documentation.
E. Neurotransmitter biochemical alterations and changes in blood-brain barrier.
F. Primary vestibular-labyrinthine damage or "concussion" of the primary balance mechanism labyrinth.

All of these and other possibilities may exist in combination. Further research will be necessary to identify which of these theoretical possibilities play the major roles. As part of the entire theory, the possibility of functional changes in axons, for example, alteration of axonal flow either rapid or slow that is reversible, may also be playing a role.

Experimental evidence of sudden deceleration injuries with subsequent brain stem petechial hemorrhages in animals has been well documented. Functional changes in the locus ceruleus with elevation of norepinephrine, and stimulation of the raphe nucleus with subsequent increased serotonin have also been documented.

Biochemical, self-limited changes in the brain and brain stem and the cortex may also be playing a role. Experiments using a rotation chair, with the head fixed in a headholding clamp and the patient very slowly moving at 1 degree/second rotation in the chair for 45 degrees in the dark so that fixation was eliminated, produced eye movements with C2 root stimulation, suggesting cervical root input.

Epidemiologic studies have shown that there are certain characteristics such as age, education, socioeconomic status and sex of the patient that seem to play major roles in terms of recovery.[32] One recent study showed that in 3 months' follow-up after mild CHI, 79% of the patients complained of persistent physical symptoms. Fifty-nine percent demonstrated problems with tension, concentration, memory and judgment, and 34% of these were unemployed. Impairment of functioning after months has been found by other researchers.[33,34] These patients have not been able to resume a normal level of functioning and continue to suffer some persistent symptoms, including headache, pain, dizziness, memory deficits, altered behavior, and other cognitive changes, presenting a challenge to the clinician and the physician taking care of them. They need help. They do not need to have their complaints minimized. The majority of patients with mild CHI and PCS improve over a period of months without sequelae.

Electroencephalography (EEG) and neurophysiologic evaluation, including evoked potentials, are helping to assess the significance of PCS, either Type I or Type II.[35–40] Although it is important to correlate PCS symptoms with EEG and EP abnormalities, it is not possible from a normal tracing to disprove the diagnosis of mild CHI with PCS. Focal EEG abnormalities in patients with CHI and PCS strongly suggest the presence of "brain damage" and probably a focal brain involvement or brain injury with mild CHI. More specifically, if a generalized abnormality is present 3 months or more after head injury, the chances are 6:1 that the abnormality antedated the head injury.[35] In other words, if an EEG abnormality or clinical seizure occurs soon after the closed head injury, the chances are greater that the EEG changes and the clinical seizure are related to the closed head injury. If the EEG shows a paroxysmal abnormality more than 3 months after head injury, the chances are 13:1 that the

patient has some type of epileptic or paroxysmal condition that antedated the CHI.[35] If the patient has clinical seizures and a focal paroxysmal abnormality more than 3 months after a head injury, then the chances are considered 3:1 that these seizures are the result of the trauma.[35] Other researchers have concluded that if an EEG is normal immediately after the closed head injury and continues to show a normal tracing, an organic lesion is less likely but still possible. Long-lasting EEG abnormalities with slow abatement are characteristics of major cerebral injuries. Marked changes in a previously normal EEG after a CHI suggest posttraumatic complication, such as subdural hematoma, abscess formation, or some other disease or injury of the central nervous system, which needs further neurodiagnostic and possible neurosurgical intervention.

EEGs should be recorded as early as possible after an acute head injury or mild CHI to identify abnormalities. In a classic paper, the recording of EEGs of numerous shipyard workers was taken within minutes of their accident.[41] Abnormalities were demonstrated. These EEG abnormalities disappeared completely in the follow-up studies. In another study, the EEG findings following mild CHI depended mainly on the elapsed time between the injury and the first recording. The sooner the EEG was done, the more likely the abnormalities were seen.

Other neurophysiologic studies should also be considered, including brain stem auditory evoked responses[42–46] and cognitive evoked responses.[47,48] Newer techniques, such as spectral analysis following CHI and quantitative computerized electroencephalogram (QEEG), promise greater sensitivity in identifying organic lesions and documenting subtle focal changes or diffuse lesions.

Neurophysiologic techniques that assess labyrinthine functioning (electronystagmography, as mentioned earlier) have been found to be very useful and should be an integral part of the neurophysiologic evaluation in patients with mild CHI and persistent PCS.[31]

Occasionally, computerized tomographic scanning as well as magnetic resonance imaging may also be helpful in evaluating CHI and PCS.[49] Positron Emission Tomography (PET) and Single Photon Emission Computed Tomography (SPEC) scanning that measure function and biochemical changes may prove useful certainly as research tools and hopefully, also clinically.

SUMMARY

This brief review summarizes the current understanding of mild closed head injury with postconcussion syndrome. It is to be emphasized that objective evaluation be performed because it is critical to the assessment of the patient. Increased awareness of patients with symptoms in association with heightened sensitivity and caring will go a long way in helping the millions of patients yearly with mild to moderate head injury. Only with objective scientific data will the understanding of the causes of the varied symptomatology of the postconcussion syndrome be thoroughly understood.

REFERENCES

1. Caveness W. Epidemiologic studies of head injury. Trauma 1977; 27:61-66.
2. Langfitt TW, Gennarelli TA. Can the outcome from head injury be improved? J Neurosurg 1982; 56: 19-25.
3. Bernad M. Boston University, UMI Dissertation Abstract International, 1988.
4. Mark VH, Ervin FR. Violence and the Brain. Harper & Row: N.Y. 1970.
5. Mild Head Injury Conference, Galveston, Texas, March 20-21, 1987.
6. Miller H. Accident neurosis. Br Med J 1981; 1:919-925.
7. Anderson FH, Lehrick JR. L'hermitte sign following head injury. Arch Neurol 1973; 29: 437-438.
8. Wilmot CB, Cope N, Hall KM, Acker M. Occult head injury: its incidence in spinal cord injury. Arch Phys Med Rehabil 1985; 66:227-231.
9. Carrol C, McAfee PD, Riley LH, Jr. Objective findings for diagnosis of "whiplash." J Musculoskeletal Med 1986;3:57.
10. Conlin FD. Whiplash revisited. Contemp Ortho 1983; 6:79.
11. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: practical scale, Lancet 1974; II:81-84.
12. Jane J, Stewart O, Gennarelli TA. Axonal degeneration induced by experimental non-invasive minor head injury. J Neurosurg 1985; 62:96-100.
13. Goswasser Z, Reider-Groswasser I, Scooker N, Machtey Y. Magnetic resonance imaging in head injured patients with normal late computed tomography scan. Surg Neurol 1987; 27:331-337.
14. Gennarelli TA, Thibault LE, Adams JH. Diffuse axonal injury and traumatic coma in the primate. Ann Neurol 1982; 12:564-574.

15. Povlishock JT, Becker DP, Cherry CL, et al. Axonal changes in minor head injury. J Neuropathol Exp Neurol 1983; 42:225-242.
16. Adams JH, Mitchell DF, Graham DI, Doyle D. Diffuse brain damage of immediate impact type: its relationship to primary brainstem damage in head injury. Brain 1971; 100:489-502.
17. Hauser WA, Anderson VE, Loewenson RB, et al. Seizure recurrence after a first unprovoked seizure. N Eng J Med 1982; 347:522-528.
18. Jerrett B, Van de Sande J. EEG prediction of post-traumatic epilepsy. Epilepsia 1975; 16: 251-256.
19. Jerrett B. Epilepsy after head injury and craniotomy. In: Godwin - Austin RB, Espir MLE (eds). Driving and Epilepsy. Royal Society of Medicine International Congress and Symposium Series. No 60. London: Academic Press 1983; 49-51.
20. Hughes JR. EEG in Clinical Practice. Butterworths: Boston; 1982.
21. Gibbs FA, Gibbs EL. Electroencephalographic study of head injury in childhood. Clin Electroencephalogr 1987; 18:10-11.
22. Gibbs EL, Gibbs Erich L, Gibbs FA. Electroencephalographic findings among children with head injuries. Clin Electroencephalogr 1982; 13:160-177.
23. Barth JT, Macciocchi SN, Jiordani B, et al. Neuropsychological sequelae of minor head injury. Neurosurgery 1983; 13:529-533.
24. Bernad PG. Closed head injury, Types I and II with post concussion syndrome. Clin Electroencephalogr 1988; 19:174-175.
25. Boll TJ. Behavioral sequelae of head injury. In: Cooper PR (ed). Head Injury. Baltimore: William & Wilkins; 1982.
26. Bruce DA, Raphaely RC, Goldberg AI, et al. Pathophysiology, treatment and outcome following severe head injury in children. Child Brain 1979; 5:174-191.
27. Gronwall D. Paced auditory serial addition task: a measure of recovery from concussion. Percept Mot Skills 1977; 44:367-373.
28. Frankowski RF, Anneger JF, Whitman S. The descriptive epidemiology of head trauma in the United States. Becker DP, Povlishock (eds). Central Nervous System Trauma Status Report. Washington, D.C.: National Institute of Neurological and Communicative Disorder and Stroke; 1985.
29. Toglia JV. Acute flexion-extension injury of the neck. Neurology 1976; 26:808-814.
30. Toglia JV. Dizziness after whiplash injury of the neck and closed head injury: electronystagmographic correlations. In: Walter AE, Caveness WF, Critchley M. (eds). Delayed Effects of Head Injury. Springfield, Illinois: Charles C Thomas Pub; 1969:72-83.
31. Bernad PG, Jylkka MM. Closed head injury with post concussion syndrome. Clin Electroencephalogr 1988; 19:46-47.
32. Rimel RW, Giordani B, Barth JT. Disability caused by minor head injury. Neurosurgery 1981; 9: 221-229.
33. Russell WR. Recovery after minor head injury. Lancet 1974; 2:1315.
34. Rutherford WH, Merrett JD, McDonald JR. Symptoms at one year following concussion from sequelae of concussion caused by minor head injuries. Injury 1979; 10:225-230.
35. Hughes JR, Wilson WP. EEG and Evoked Potentials in Psychiatry and Behavioral Neurology, Butterworths: Boston; 1983.
36. Gibbs FA, Weigner WR, Gibbs EL. Electroencephalogram in post-traumatic epilepsy. Am J Psychiatry 1944; 100:738-749.
37. Courjon J. Handbook of Electroencephalography and Clinical Neurophysiology. Vol XIVB, Traumatic Disorders. Amsterdam: Elsevier; 1972.
38. Jung R. Neurophysiologische Untersuchungsmethoden. In: Bergmann G, Frey W, Schivigkh G. (eds) Handbuch der Inner Medizin Vol. I. Berlin: Springer; 1953:1206-1420.
39. Bental E, Sharf B. Electroencephalographic changes following head injuries. Electroencephalogr Clin Neurophysiol 1968; 24:192 (abstract).
40. Jacome DE, Risko M. EEG features in post-traumatic syndrome. Clin Electroencephalogr 1984; 15:214-222.
41. Dow RS, Ulett G, Raaf J. Electroencephalographic studies immediately following head injuries. Am J Psychiatry 1945; 101:174-183.
42. Noseworthy JH, Miller J, Murray TJ, Regan DD. Auditory brainstem responses in postconcussion syndrome. Arch Neurol 1981; 38:275-278.
43. Schoenhuber R, Gentilini M, Scarano M, Bartolotti P. Longitudinal study of auditory brain stem responses in patients with minor head injuries. Arch Neurol 1987; 44:1181-1182.
44. Ahmed I. Brainstem auditory evoked potentials in dizziness. Clin Electroencephalogr 1984; 15: 110-115.
45. Rowe MJ, Carlson C. Brainstem auditory evoked potentials in post concussion dizziness. Arch Neurol 1980; 37:679-683.
46. Drake ME Jr. Brainstem auditory evoked potentials in whiplash injuries. Clin Ev Pot 1987; 5:26-28.
47. Drake ME Jr, John K. Long-latency auditory event-related potentials in post concussion syndrome. Clin Ev Pot 1987; 5:19-21.
48. Drake ME Jr, John K. Long-latency auditory event-related potentials in post concussion syndrome. Neurology 1986; 36:163 (suppl 1).
49. Snow RB, Zimmerman RD, Gandy SE, Deck MDF. Comparison of magnetic resonance imaging and computer tomography in the evaluation of head injury. Neurosurgery 1986; 18:45-52.