

WHIPLASH / NECK PAIN

Mild Traumatic Brain Injuries After Motor Vehicle Accidents

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Brain injuries were quite common in our earlier civilization. The Edwin Smith Surgical Papyrus, a copy of a manuscript dating back 5,000 years, is the oldest recorded medical document. It shows us that the ancient Egyptians were well aware of the myriad of disturbances brought about by injury to the brain. The ancient Greeks also understood that intellect and reasoning had their seat within the brain. Physicians of the Hippocratic school some 2,400 years ago discovered that incising a wound on one side of the brain caused, "a spasm in the opposite side of the body." Galen wrote that, "a loss of

memory for words," might follow head injury.¹

Motor vehicle trauma is probably the single most important agent in both fatal and mild brain injuries.

From 40 percent² to 60 percent³ of all mild brain injuries are caused by motor vehicle accidents

(MVAs) with the most common diagnosis given being concussion.² Many of these MVA-related injuries are the result of blunt head injury (a.k.a. soft head injury) which describes contact with some object but without penetration of the skull. An example would be striking the steering wheel or door post. These injuries probably constitute those at the more severe end of this continuum. On the near end of the spectrum we will find the less severe injuries which are primarily the result of acceleration and deceleration of the brain in a noncentroidal manner (i.e., angular acceleration).

These injuries affect a very extensive subgroup of patients. From within this subgroup, the spectrum of injury ranges from subclinical concussion (i.e., without a loss of consciousness or LOC) or posttraumatic amnesia (PTA), to a fully developed postconcussion syndrome (PSC). Posttraumatic headaches (PTHA) are very common residuals and may last anywhere from six months to several

years.⁴ Mild traumatic brain injury (MTBI) presents a risk of PTHA of about 40-60 percent⁵ and many of these injuries are too subtle to be caught in the snares of most of the diagnostic tests available

today. Barnat⁶ has shown that whiplash injuries are the second most common cause of PTHA in this country.

Barnat⁶ described the frequency of various complaints associated with PTHA (Table 1), and it becomes clear that he was actually describing a condition which is more appropriately described as postconcussion syndrome (PCS). Symptoms generally attributed to this latter condition are listed in Table 2.

Table 1 Common Complaints in PTHA Patients (after Barnat)⁶

1. Headache	82.9%
2. Irritability	66.7%
3. Insomnia	63.2%
4. Anxiety	58.1%
5. Memory problems	57.3%
6. Other pain	56.4%
7. Concentration problems	52.1%
8. Depression	52.1%
9. Dizziness	41.1%
10. Confusion	41.1%
11. No control of emotions	36.8%
12. Loss of libido	35.0%
13. Tinnitus	29.1%
14. Can't carry out plans	29.1%
15. Can't plan	28.4%
16. Flashbacks	28.2%
17. Don't enjoy sex	26.5%
18. Nightmares	26.5%
19. Arithmetic problems	17.9%

Table 2
The Postconcussion Syndrome
1. Lightheadedness
2. Vertigo/dizziness
3. Neck pain
4. Headache
5. Photophobia
6. Phonophobia
7. Tinnitus
8. Impaired memory
9. Easy distractibility
10. Impaired comprehension
11. Forgetfulness
12. Impaired logical thought
13. Difficulty with new or abstract concepts
14. Insomnia
15. Easily fatigued
16. Apathy

17. Outbursts of anger
18. Mood swings
19. Depression
20. Loss of libido
21. Personality change

Both conditions are produced by the same trauma and represent merely different points along a continuum of severity. Where patients have only headaches, I make the PTHA diagnosis in spite of the fact that it is not necessarily in keeping with the criteria established by the International Headache

Society.⁷ When more of the other symptoms of PCS are present, I make that diagnosis.

Radanov et al.,⁸ have described two clinically distinct clinical conditions arising out of soft tissue injuries to the neck. Most were the result of whiplash. In the first syndrome, which the authors termed the cervicoencephalic syndrome, patients complained of headache, fatigue, dizziness, poor concentration, disturbed accommodation, and impaired adaptation to light intensity. The authors found evidence of cognitive impairment in the area of divided attention and speed of information processing. In the second syndrome, which they termed the lower cervical spine syndrome, patients complained primarily of neck and upper extremity pain. Impaired divided attention was not seen in this group. Interestingly, loss of consciousness did not correlate with the divided attention test results.

More recently, Radanov et al.,⁹ have studied the incidence of posttraumatic headaches following CAD injury and have further evaluated the cognitive deficits seen following some CAD injuries. While they found that a history of headaches before the injury significantly increased the likelihood of PTHA (by a factor of more than two), this occurred only with findings indicative of significant injury to the cervical

spine.⁹ They studied a random sample of 98 "common whiplash" patients, and evaluated them for evidence of cognitive impairment. At six months, 67 patients had recovered fully while 31 were still symptomatic. It was noted that the symptomatic group, who were older at baseline, provided a greater variety of symptoms, greater neck pain intensity, and greater cognitive impairment. Neuropsychological function had returned to normal at six months in all. The symptomatic group

displayed delayed recovery in complex attentional functioning. Bohnen et al.,¹⁰ have also reported persisting cognitive deficits in CAD patients, primarily in the area of information processing and

memory function. These patients also had central otoneurological abnormalities. In another study,¹¹ they compared mild brain injured patients who had not developed PCS and found that the PCS group performed more poorly on tests of divided and selected attention.

Research continues to validate the concept of PCS following CAD trauma. Clinicians should be on the alert for subtle signs of this disorder in patients who are at risk and should not be hesitant to make the diagnosis when appropriate.

Reference list available upon request. Call (619) 423-9860.

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