The paralyzed patient's overlooked mild or moderate traumatic brain injury

William Singer and J. Anderson Harp

THE TOLL OF TRAUMATIC head injury and traumatic spinal cord injury in the United States is enormous. It is estimated that total cases of traumatic head (or brain) injury exceed 1 million at an annual cost of $25 billion.[1] The traumatic spinal cord injury populace is estimated as in excess of 250,000 cases, costing well over $2 billion annually.[1]

Oftentimes, the rush to examine and diagnose one seemingly catastrophic injury causes one to overlook another potentially significant injury. One such example is the patient with significant craniocerebral injury who is at risk for a concurrent injury to the cervical spine. It has been estimated that as many as 20% of patients entering a hospital with injury to the brain have a concurrent injury to the cervical spine.[2] This has significant treatment concerns, as O'Malley et al estimate that as many as 3% of spinal cord injuries occur after the patient has commenced treatment within the emergency medical system.[2] As has been observed, the most common reason for such postadmission injury has been the medical team's failure to suspect and investigate for a spine fracture.[3] Nevertheless, the consequences resulting from such omissions may be quite severe.

Likewise, it is recognized that during the course of treating life-endangering injuries, less obvious injuries are sometimes overlooked.[4] One such injury that may be less life-endangering yet severe is the mild to moderate traumatic brain injury (TBI).

It is clear that one who receives a blow significant enough to fracture the cervical spine is likely also to have suffered a skull fracture or closed-head injury.[5] In a study of 371 patients who entered a computerized traumatic registry on admission to a surgical intensive care unit (SICU), Pal et al concluded that 41% of cervical spine fractures had an associated skull fracture or closed-

William Singer, MD, Cambridge Hospital, Cambridge, Massachusetts, and Harvard Medical School, Boston, Massachusetts.

J. Anderson Harp, JD, Taylor, Harp & Callier, Attorneys. Columbus, Georgia.

Top Spinal Cord Inj Rehabil 1995;1(1):71–76

head injury, and 19% of the patients with lumbar spine fractures had an associated skull fracture or closed-head injury.[6] What is unclear is how Pal et al defined the closed-head injury. It is likely that the mild to moderate TBI is not reflected in this study, thereby making the likelihood of such events even more significant.

Discussion

TBI is the resulting organic damage caused by a traumatic insult to the brain, resulting in physical, intellectual, and/or emotional deficits, with the potential for significant changes in academic, social, and vocational function. Mild TBI, which has been historically referred to by the clinicians as the so-called postconcussional syndrome, consists of Glasgow Coma Scale scores of 12 to 13[6] with damages also being quite possible at scores of 14 and 15. Moderate TBI exists with Glasgow Coma Scale scores of 9 to 11.[7] Mild, however, is a misnomer in that the effect of the injury to the brain may result in altered consciousness, impaired perception, decreased language skills, loss of executive skills, inappropriate social behavior, and memory impairment. The dilemma is that the injury that can cause such significant disorder may not be detectable by skull radiograph, computed tomography (CT) scanning, or magnetic resonance imaging (MRI). Reliance on such devices leads to significant underdiagnosis.[8] In a study of 838 patients with severe head injuries, Lobato et al noted 211 patients whose CT did not show focal mass lesions after they suffered an apparently nonsevere head injury but who subsequently deteriorated into coma.[8] Clearly, the patient with mild TBI suffers organic damage on the microscopic level, including diffuse anoxal and excitotoxicity neuronal injuries, which the commonly used laboratory diagnostic tools do not always identify. Cognitive deficits resulting from these undiagnosed TBIs may be of particular importance to the adjustment of spinal cord injury (SCI) patients as they attempt to acquire the skills necessary for self-reliance and reentry into school, the workplace, or society in general.

As is often seen in TBI without SCI, neurodiagnostic tests may reveal no abnormalities.

The catastrophic motor deficit resulting from the spinal cord injury may make the diagnosis of motor deficits of cerebral damage origin difficult or impossible. During the hospitalization, superficial mental status examinations or even a standardized "mini mental status examination" may not be sensitive to the cognitive and memory disorders, as well as the disorders of attention and concentration, that are the hallmarks of mild to moderate TBI. In addition, affective changes may be attributed to a reaction to the spinal-cord-induced paralysis and loss of function but may actually be the result of a mild to moderate TBI. As is often seen in TBI without SCI, neurodiagnostic tests may reveal no abnormalities. Abnormalities detected by a neuropsychological assessment may be the only indication of a brain injury accompanying SCI. Thus, a neuropsychological evaluation is essential in the thorough assessment of patients with SCI.

The diagnosis of mild to moderate TBI may require the supplementing of the inter-

disciplinary health care team with neurologists, neuropsychiatrists, and neuropsychologists who are attuned to the subtleties of this injury. The clinical neuropsychologist can significantly aid in evaluating and treating brain damage on this level. This may be of particular importance to the paralyzed patient.

In recovering from the injury, the patient must cope with the adjustment to his or her modified lifestyle. The problems span the areas of pain, adjustment to injury, health complaints, finances, transportation, sexuality, recreation, accessibility, and many other areas.[9] Of note, in a study of 358 persons postinjury, Gerhart recorded that at the 2-year contact, 58% suffered problems related to the financial aspects presumably in conjunction with their work environment.[9] A mild TBI would have significant effect on the patient's ability to cope with his or her work environment, and the patient's recovery would be greatly aided by appropriate diagnosis and treatment.

The Role of Neurolaw

The legal community also has followed the medical community's direction of specialization. Nowhere is this more salient than in the area of traumatic brain and spinal cord injury. *Neurolaw* is the field of jurisprudence designed to meet the challenges presented by TBI/SCI litigation. The term *neurolaw* was coined by attorney J. Sherrod Taylor and first appeared in the legal literature in *The Neurolaw Letter*.[10] Subsequently, this term apperared in Taylor's "Proving Long-term Soft Tissue Damage" in *Insurance Settlements Journal*.[11] *Neurolaw* as a term first appeared in the health care literature in Taylor and colleagues' "Neuropsychologists and Neurolawyers" in *Neuropsychology*.[12] Neurolawyers are those attorneys who, through interest, education, and training, have developed special expertise in representing clients with traumatic brain and spinal cord injury. In representing the TBI client, the neurolawyer has the responsibility of proving, by admissible testimony, the existence of the mild to moderate TBI. In an SCI case, this can be quite important, as the defense of the claim may generally be that the motor handicap alone is not sufficient to prevent the injured individual to return to a near-normal life, complicated by only minor motor limitations.

In the present state of law in the United States, the debate centers on the relevance and admissibility of neuropsychological evidence.[10] This is compounded by the mild TBI case in that the traditional radiographs, CT scans, and MRIs are normal and, as a result, the initial treating physician is oftentimes of the opinion that no brain injury exits. Nevertheless, the patient reports difficulty in concentrating, forgetfulness, loss of executive skills, decreased language skills, and other problems. Even if the clinical neuropsychologist's examination concludes the existence of a TBI, the admissibility of this opinion may be contested on the basis of the psychological tools that were utilized.

The clinical neuropsychologist's opinion is generally admissible and relevant as the expressed opinion of an "expert." The law permits opinion to be given by one who by virtue of skills, experience, training, or education, possesses knowledge outside that of the layperson.[10] The trend in most jurisdictions is to permit the admission of such expert testimony particularly if a convincing foun-

dation of testimony exists supporting its reliability.[13]

In regard to both the medical and legal interests of the patient, it is essential that the interdisciplinary medical team not underdiagnose TBIs in spinal cord injury patients. Thus, the treatment team should include members skilled in the diagnosis and treatment of the subtle cognitive deficits resulting from TBI, especially a neuropsychologist. The following case reports highlight the significance of this point.

Case Reports

Patient/client 1

This 22-year-old man suffered C5-C6 tetraplegia as a result of a motorcycle/motor vehicle collision. He was thrown a significant distance from the point of impact and was unconscious until arrival at the emergency department. CT scans and MRIs revealed no gross structural brain damage. Acute-care recovery was uneventful. Prior to the injury, he was a high-school graduate who had done well academically. One year postaccident, he noted headaches, irritability, difficulty in concentrating on simple tasks, and an altered sleep pattern. He attempted further education; however, his mathematics and spatial skills were at a much lower level than expected. His adjustment to society has been quite complicated. No neuropsychological testing was ordered nor was a TBI considered.

Patient/client 2

A 51-year-old man received a fracture of C5 resulting in tetraplegia when the scaffolding at a construction site collapsed, striking him in the back of the head and neck. He recalls being unconscious for only a brief period of time. Skull radiographs and CT scan testing of the brain were interpreted as normal. During postacute care recovery, his wife noted significant personality change. He was angry, sullen, and quick to strike out in response to minor provocation. He could not concentrate on simple mathematics tasks. His sleep pattern was erratic, and he struggled with the selection of words. His chief treating physician did not request any neuropsychological evaluation.

Patient/client 3

A 17-year-old male high-school junior, who had been the unrestrained passenger in an automobile, was ejected when a collision occurred. He was unconscious at the accident scene. Although he awakened in the ambulance, his first postaccident memories began on his third hospital day. He sustained a C4-C5 quadriparesis. A CT scan of the head on the day of his injury was normal. During his hospital course, he did not require treatment for obvious brain injury. While hospitalized, he complained of headaches, a disturbance of sleep, and dizziness when his position was changed. Although previously described as being an independent individual with a pleasant and relaxed personality, he became sullen and irritable during hospitalization and remained so after his return home. This was initially diagnosed as a depressive reaction to his motor handicap. He was felt not to have suffered a brain injury.

On return to school, he had difficulty accommodating to the classroom routine. He displayed short-term attention and concentration difficulty. While previously a strong student, his ability now to learn new informa-

tion was extremely impaired. When neuropsychological testing was performed, abnormalities of attention/concentration, memory, and executive functions were found consistent with the pattern often found in patients who suffer TBI. In reassessing his affect, his depression was now felt to be a combination of reaction to his acquired handicaps and an endogenous depression from his TBI.

Patient/client 4

A 40-year-old successful trial attorney was thrown down an embankment when he was struck by a car while riding his bicycle. He was unconscious at the scene, becoming gradually responsive by 30 minutes following the accident and was fully alert by 3 hours. Initial evaluation in the emergency department revealed a C4-C5 fracture subluxation. CT scan myelography revealed impingement of the cord at the C4 and C5 levels. A CT scan of the head was normal. He was found to have a motor handicap from C5 down. During the hospitalization, his examination gradually improved, and by discharge 3 months later, he had only minor residual weakness. In addition to the diagnosis of spinal cord injury, he was also diagnosed as having a mild concussion of no clinical significance due to his brief loss of consciousness. However, on return to work, he was unable to prepare his case work effectively, displayed poor organizational skills, and could not concentrate well in the courtroom. He became frustrated and depressed. Nine months following his accident, he had a neuropsychological evaluation that revealed significant deficits in executive functions, organizational skills, short-term memory, and auditory attention skills. He entered a cognitive rehabilitation program and has made significant improvement but continues to work a limited schedule and is not able to deal with complex cases as he had before.

• • •

The SCI patient is at high risk for underdiagnosis of other subtle injuries. Clearly, a trauma sufficient to fracture a vertebra or sever a spinal cord is capable of causing injury to the brain. However, SCI patients, despite normal skull radiographs, CT scans, and MRIs, are at high risk for having their erratic behavior interpreted as merely the psychological reaction to their injury. Therefore, the burden is on the interdisciplinary medical team to ensure that all injuries are diagnosed and treated.

REFERENCES

1. *Brain Facts: A Primer on the Brain and Nervous System.* Society for Neuroscience; August 1991.
2. O'Malley KF, Ross SE. The incidence of injury to the cervical spine in patients with craniocerebral injury. *J Trauma.* 1988;28:1,476–1,479.
3. MacDonald RL, Schwartz ML, Mirich D, Sharkey PW, Nelson WR. Diagnosis of cervical spine injury in motor vehicle crash victims: how many x-rays are enough? *J Trauma.* 1990;30:392–397.
4. Enderson BL, Reath DB, Meadors J, Dallas W, DeBoo JM, Maull KI. The tertiary trauma survey: a prospective study of missed injury. *J Trauma.* 1990;30:666–670.
5. Pal JM, Mulder DS, Brown RA, Fleiszer DM. Assessing multiple trauma: is the cervical spine enough? *J Trauma.* 1988;28:1,282–1,284.

6. Roberts AC. *Litigating Head Trauma Cases.* March 1991:8–1, 8–5.
7. *The Neurolaw Letter.* 1991;1(2).
8. Lobato RD, Rivas JJ, Gomez PA, et al. Head-injured patients who talk and deteriorate into coma. *J Neurosurg.* 1991;75:256–261.
9. Gerhart KA. Spinal cord injury outcomes in a population-based sample. *J Trauma.* 1991; 31:1,529–1,535.
10. *The Neurolaw Letter.* 1991;1(4).
11. Taylor JS. Proving long-term soft tissue damage. *Insurance Settlements Journal.* Winter 1991;3(1):21–26.
12. Taylor JS, Harp JA, Elliott T. Neuropsychologists and neurolawyers. *Neuropsychology.* 1991.
13. *Salem v United States Lines Company,* 370 US 31, 82 S Ct 1119.