File ① Ext: brain damage
② general: head injury

CEM: 85-010

Cognitive and Behavioral Consequences of Closed Head Injury

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Cognitive and behavioral consequences associated with traumatic brain injuries have been observed since the dawn of humankind. As early as 3000 BC, the Egyptians recorded the effects of a left temporal depressed skull fracture on motor speech. The Greeks, physicians of the Hippocratic school, Renaissance surgeons, and others also described the sequelae of head injury. Not until the early 20th century, however, with the advent of the Industrial Revolution, mechanized warfare, and motorized transportation, did head injury become a worldwide epidemic.

Today, traumatic head injuries from all causes exceed 1,000,000 new cases annually.[1] Incidence and prevalence are estimated as 200/100,000 and 400/100,000,[2] respectively. The most frequently injured are males between 15 and 24 years of age. The annual incidence for this group is 600/100,000.[3] Many of these injuries are associated with alcohol and automobiles.[4] Unfortunately, 70% of all trauma cases resulting in coma occur in motor vehicle accidents.[5] Thus, the ranks of head-injured persons with significant intellectual and behavioral deficits grow yearly; many are young, with normal or almost normal life expectancies. As Cope[2] noted, "the disabilities suffered by patients, particularly those with the most severe injuries . . . will continue to persist with their immense economic, social, and personal costs for 40 to 50 years after injury."

Closed head injury has become the most common serious neurologic disorder in the United States. Concomitantly, there has been an increasing demand for head injury rehabilitation services for several reasons:

1. Mortality rates associated with severe craniocerebral trauma have decreased dramatically with the advent of improved emergency transport and neurosurgical interventions that prevent or reduce secondary complications (such as brain damage resulting from hypoxia) associated with central nervous system insult.[6]
2. Recent research suggests that even mild injuries are associated with cognitive and behavioral sequelae. Individuals who were at one time prematurely dismissed from medical and rehabilitative care are now seeking treatment for symptoms previously considered solely in the province of psychiatry.
3. Relatives have begun to organize support groups and political lobbying activities to decry inappropriate treatment modalities, the dearth of rehabilitative services, and the di-

lemma posed by a technologically advanced society that saves lives but then offers few guidelines for living with multiple enduring intellectual, social, and physical handicaps. The head injured and their families can no longer be ignored.

A CONTINUUM OF DISABILITY

Head injury is optimally conceptualized as a continuum that ranges from very mild (no or brief loss of consciousness) to extremely severe (prolonged coma). The generic term "head injury" should be modified by mild, moderate, or severe to discourage the impression that craniocerebral trauma is a homogeneous entity. Efforts to establish a methodology for classification have been crude, but researchers have often relied on such measures as the Glasgow Coma Scale[7] or the Disability Rating Scale.[8] The relative advantages and disadvantages of these two measures have been discussed elsewhere.[9] Rimel et al[10] used Glasgow scores obtained approximately 6 hours after hospital admission of 8 or less, 9 to 12, and 13 or more to designate severe, moderate, and mild injuries, respectively. Other indices for establishing severity of injury include depth and duration of coma,[3] length of post-traumatic amnesia,[11–13] presence and type or absence of hematoma, and loss or preservation of brainstem reflexes (such as pupillary reaction to light).[14,15]

Even though this head injury spectrum exists, it is also clear that the pathophysiology of traumatic injuries dictates predictable cognitive, behavioral, and social sequelae; these will be the focus of this article.

BEHAVIORAL CONSEQUENCES OF MODERATE TO SEVERE HEAD TRAUMA

Behavioral problems associated with closed head injury are common and represent major obstacles to rehabilitation efforts designed to help patients resume productive domestic or community roles.[16] Furthermore, behavioral problems are the most frequent source for the family's perception of head injury as a persisting burden.[17]

During the acute recovery phase after trauma, the moderately to severely injured patient may manifest a range of disordered behaviors, including but not limited to restlessness, agitation, combativeness, emotional lability, confusion, hallucinations and other disturbed perceptions, disorientation, depression, paranoid ideation, hypomania, and confabulation.[6,18] Although these derangements are disturbing, families often associate them with the early recovery phase and anticipate their resolution. Nevertheless, although the character of patients' behavioral problems may change with time, there is a growing corpus of research that suggests that persisting psychiatric morbidity may often be the rule, rather than the exception, after moderate to severe trauma.[14,17,19] Furthermore, these behavioral problems are ultimately more disturbing, burdensome, and unacceptable to family members than are physical stigmas.[19–25]

Researchers have reported a range of persisting personality alterations among the head injured, including disorders of behavior, mood, and thought. These disturbances reflect a complex blend of the site of brain damage, premorbid character,[16,19] and the patient's growing insight into the significance of residual disabilities.

A most dramatic example of these changes is provided by Jarvie:[26]

> When the acute phase of the injury is over it may be found that the patient's character has been permanently altered. He may now be restless and impulsive, excessively talkative and emotionally blunted. He may be boastful and the content of his talk may be indiscreet or frankly obscene. Increased sexual activity, with associated decreased control, may lead to antisocial acts. His personality is less inhibited and the emotional blunting may make the patient unaware of the effects of his behaviour on others.

Table 1 lists some common, persisting behavioral problems associated with moderate to severe head injury. A large subset of these behaviors, including impaired judgment, euphoria or silliness, marked apathy, disinhibition, childishness, blunting of emotional responsiveness, egocentricity, and aggressivity, are components of the frontal lobe syndrome.[19]

There is general agreement that damage to the frontal lobes leads to readily recognizable changes in emotions and behavior.[27] Lishman[28] examined soldiers with focal frontal wounds and found that the most severe effects were produced by bilateral injuries to the basolateral or convex-lateral frontal cortex. The extent of disturbance after frontal in-

Table 1. Behavioral Problems Associated with Moderate to Severe Head Trauma

Irritability	Hypersexuality
Impulsivity	Hyposexuality
Egocentricity	Dependency
Emotional lability	Silliness or euphoria
Impaired judgment	Aggressivity
Impatience	Apathy
Tension or anxiety	Childishness
Depression	Disinhibition

jury, according to Lishman,[21] may range from a mild coarsening of personality to gross and severely disabling behaviors. The quantity of impairment is a function of the extent and location of damage. Most data suggest that lesions of the convex-lateral surfaces are associated with aspontaneity, hypokinesis, and a generally pseudodepressed presentation,[29] whereas injuries to the orbitofrontal undersurfaces are associated with uninhibited, aggressive, euphoric, and sexually indiscreet behavior.[30] Lishman[31] suggested that frontal syndromes may arise independently of premorbid personality. This assertion has been reiterated by Jennett and Teasdale.[14]

Behaviors that constitute the frontal syndrome are frequently described by relatives as burdensome and distressing. Thomsen[25] interviewed families and found their most frequent complaints centered on their loved one's irritability, hot temper, restlessness, aspontaneity, stubbornness, emotional lability, and regression. Similarly, Brooks[17] found that 1 year after injury, patients with personality changes were characterized by family members as more excitable, irritable, and anergic. Weddell et al[32] interviewed patients and families 2 years after injury. Patients were described by loved ones as either more irritable, more affectionate, or more disinhibited than premorbidly. Luria[33] noted that of all head injuries, those associated with significant frontal lobe involvement have the poorest prognosis for both return to work and successful readjustment to family and environment. Even after 5 years, families experiencing the greatest subjective burden typically cite behavioral problems, including quick temper, irritability, and apathy, as most troublesome.[17]

Depression is commonly associated with head injury.[34] It is an overdetermined symptom with multiple causality. With returning awareness of the limitations imposed by their deficits, many patients become discouraged, demoralized, or even hopeless. As Rosenthal[16] noted, the head injured "are less able to perform physically, more dependent socially, and often feel a sense of impotence in trying to reconstruct their lives. Social relationships are less rewarding and often diminish, vocational prospects are dim, and life becomes less interesting."

This form of reactive depression, however, should not be confused with either endogenous affective changes or the pseudodepressed syndrome associated with injury to the frontal convexities. Recent research suggests that depression among the traumatically brain injured varies as a function of the proximity of damage to the frontal pole.[35] This may reflect disruption of norepinephrine projections from the locus ceruleus to other cortical areas.

COGNITIVE IMPAIRMENTS ASSOCIATED WITH MODERATE TO SEVERE HEAD INJURY

Intellectual impairments associated with moderate to severe head trauma are shown in Table 2. These deficits encompass a range of processes, including memory, central language functions, performance intelligence, information processing speed, and planning or organizing skills. A review of Table 2 suggests that impairments can be classified as arising from either focal (such as dysphasia) or diffuse (such as slowed information processing) lesions.[36] Clearly, the pathophysiology of trauma, coupled with the cytoarchitecture of the brain, renders specific regions especially susceptible to injury, and hence dysfunction.[37] As a consequence, although patients may present with variable cognitive disorders, there are overlapping features shared by almost all.[13]

The most consistent clinical residual problem faced by the head-injured survivor is disordered verbal and nonverbal learning. This disturbance may persist well beyond the cessation of post-traumatic amnesia. Faulty memory is often the most disabling cognitive disability after severe head trauma.[38] Brooks[39] found that head-injured patients had poorer short-term verbal and nonverbal recall than a control group and, after a 30-minute interval, remembered proportionately less of what they did learn. Using the Wechsler Memory Scale, Brooks[40] examined 82 severely injured patients within 2 years of injury. Although digit span forward was almost normal, digits backward, immediate and delayed recall of brief prose passages, and paired associated learning were all defective. Others have demonstrated similar disturbances of new learning abilities among moderately to severely injured patients.[10] Research suggests that impaired new learning remains a major problem for months, years, and even decades after injury.[13,41] These findings are not surprising, since damage to the temporal lobes and other neocortical structures is common after closed head injury.[14]

Table 2. Cognitive Impairments Associated with Moderate to Severe Head Trauma

Memory deficits
Decreased abstraction
Slowed information processing
Poor concentration
Deficits in processing and sequencing information
Slowed reaction time
Dysarthria
Anomia
Impaired auditory comprehension
Decreased verbal fluency
General intellectual deficits
Planning or organizational problems

Another cognitive sequela of trauma has been revealed by standardized intelligence testing. As a rule, even with nonlateralized injuries, patients' performance IQ scores show greater impairment than verbal IQ scores.[42–45] This may reflect the overlearned, more resilient nature of verbal information as well as the reliance of performance IQ tests on more complex responses, motivation and attention, and motor speed.[44] Research suggests that verbal-performance discrepancies may diminish over time.[45,46] Several studies, in fact, indicate that IQ scores among the moderately injured may not differ significantly from normal within 1 to 3 years after injury.[3,44] These data could lead to the faulty inference that head-injured patients eventually experience full recompensation of cognitive abilities. Such a conclusion would overlook the wealth of more focal test data that demonstrate persisting cognitive deficits, especially in such areas as memory, language skills, and organizational abilities.

Despite the indication from intelligence data that verbal functions may be relatively spared after closed head injury, more detailed language evaluations do not support this conclusion. Subclinical language disturbances, including impoverished verbal fluency, anomia, decreased word finding, and impaired auditory comprehension of complex commands, have been noted.[3,36] Nonaphasic disorders of speech (such as dysarthria) have also been described.

Groher[47] evaluated 14 severely injured patients 4 months after injury and detected marked anomia and receptive language problems, although all patients could converse well in routine exchanges. Defects in writing included problems with spelling, syntax, and sentence construction. Similarly, Hagen[48] noted receptive-integrative-expressive language dysfunction associated with trauma.

Sarno[49,50] administered a standardized aphasia test to 69 postcoma patients at 48 weeks and again after 1 year. At both assessments, all patients had linguistic impairment, which was not necessarily apparent clinically, but was detected by testing. Speech and language problems included three major categories: dysphasia, dysarthria accompanied by linguistic deficits, and "subclinical" dysphasic deficits. The last category denoted defective language processing as detected by scores on the NeuroSensory Center Comprehensive Examination for Aphasia, although conversational speech was without obvious impairment. Sarno[49] suggested that linguistic or cognitive functions are sensitive to severe head injury, a fact that must be acknowledged in patient management.

Other common cognitive deficits that have been associated with moderate to severe trauma include slowed information processing and difficulties in spontaneously planning and organizing intellectual strategies. As Adamovich et al[36] and others[51] suggest, the former problem reflects more widespread cortical-subcortical damage, whereas the latter is associated with more focal frontal lobe impairments.

Slowed information processing has been convincingly demonstrated by various investigators.[52–54] This slowing is found especially in the domain of decision making, response selection, and mental transformations.[51]

There are numerous reports underscoring frontal lobe involvement in planning.[30,55,56] Luria[33] believed the anterior frontal areas regulate, organize, and coordinate neural activity. Although formal studies of such deficits in the traumatically head injured are limited, there are ample clinical data that suggest that moderately to severely injured patients have significant problems in planning, organizing, and executing activities that require deliberate thinking.[54,57]

MINOR HEAD INJURY

Severe head injuries are greatly outnumbered by those incurred after minor trauma. Estimates of the annual incidence of minor craniocerebral injuries range from 400,000 to several million.[58] In part, this discrepancy reflects how minor head injury is conceptualized. Definitions vary from loss of consciousness requiring hospitalization to lacerations of the scalp and face that do not affect the brain.

A clinical mythology, reviewed by Trimble,[59] has burgeoned during the 19th and 20th centuries regarding the genuineness of cognitive, psychologic, and social symptoms that may arise after minor trauma. The cause of symptoms has been attributed to both organic and psychogenic underpinnings. The latter viewpoint minimizes the contribution of neurologic damage while highlighting neurotic phenomena tied to secondary gain, pending litigation, or post-traumatic stress. Although the argument for psychogenic determinants of symptoms may have merit,[60] recent histologic, neuropsychologic, and neurophysiologic data support Symonds'[61] assertion: "It is questionable whether the effects of concussion, however slight, are ever completely reversible."

Rimel et al[58] defined minor trauma as brief loss of consciousness (20 minutes or less), a Glasgow Coma score of 13 or higher, and a brief hospitalization (2 days or less). Although efforts to define minor injury are now under way, it is evident that

past failures to use systematic criteria for classifying such injuries have contributed to discrepancies among published reports and differing clinical and forensic opinions regarding these patients.[62,63]

Typically, individuals with postconcussional damage develop cognitive and psychologic complaints within days or weeks after the injury.[64] These patients present with a constellation of symptoms, as shown in Table 3. This symptom constellation is remarkably consistent from patient to patient, although the degree and duration of complaints vary greatly, as does the extent to which they prove disabling. Evidence suggesting that postconcussional symptoms reflect cortical-subcortical dysfunction has been derived from postmortem studies,[65] animal research,[66] investigations of information processing speed,[67] cerebral blood flow,[68] and evoked potentials.[69] As Trimble[59] noted: "changes of brain function do occur after head injury, even in the absence of loss of consciousness or clearly defined neurologic deficits and this is frequently accompanied by neuronal damage and cell loss in selective areas of the brain."

Rutherford et al[70] found that 51% of 145 concussed patients had at least one symptom 6 weeks after injury, and two thirds of those had more than one. Those patients who complained of headache and diplopia within 24 hours of injury were more likely to have symptoms at 6 weeks. Barth et al[71] examined 71 patients 3 months after minor head injury, using the Halstead-Reitan neuropsychologic test battery. A significant percentage had cognitive deficits (such as memory problems) that appeared to be unrelated to the length of unconsciousness or post-traumatic amnesia. These patients had problems returning to work. Rimel et al[58] examined 424 patients 3 months after injury. These researchers reported that 79% complained of persistent headaches, 59% described problems with memory, and 34% of patients gainfully employed before injury could not work. Among 69 selected patients, the neurologic examination was completely normal, but neuropsychologic tests demonstrated problems with attention, concentration, and memory in almost the entire sample. Rimel et al[58] also reported that emotional stress generated by chronic symptoms contributed to patients' long-term disability. It is interesting that litigation had an insignificant role in this morbidity because only 6 of 424 patients were involved in legal proceedings 3 months after injury. This study replicates that of Merskey and Woodforde,[72] who compared 10 cases of mild head injury for whom no compensation claim was made and 17 similar cases in which compensation had already been decided. Both groups exhibited a similar range of problems associated with the postconcussional syndrome; both displayed improvements over time, but in many cases the symptoms persisted for an appreciable period after the court case had been settled.

Table 3. Symptoms Associated with Minor Head Trauma

Headache	Anxiety
Dizziness	Insomnia
Fatigue	Hyperacusis
Reduced concentration	Photophobia
Memory deficit	Depression
Irritability	Slowed information processing

Gronwall and Wrightson[67] examined patients who sustained post-traumatic amnesia for less than 24 hours. Using the Paced Auditory Serial Addition Test (PASAT), a measure of information processing speed, they found slowing in many patients. Among those who complained of postconcussive symptoms and an inability to carry out normal work, all demonstrated reduced information processing abilities. PASAT scores improved as postconcussive symptoms receded. More recently, MacFlynn et al[73] examined 45 patients with minor trauma with a four-choice reaction time test at 1 day and 6 weeks after injury. In contrast to matched controls, the concussion cases displayed significantly poorer performance on four measures at both assessments. No differences, however, were detected between groups at 6 months.

The question of organic versus neurotic causation of postconcussional symptoms can be reconsidered by reviewing the typical events experienced by most patients with minor head injury. Initially, trauma patients are informed of their good fortune to have escaped serious brain damage, that their injury is inconsequential, and that no treatment or follow-up is indicated. This fosters the expectation among patients and their families that there will be no enduring sequelae. The patient then resumes the responsibilities he had before he was injured. If symptoms are not yet evident, the cognitive demands associated with work or school may encourage their expression. Vocational or academic efficiency may be significantly compromised by slowed information processing, forgetfulness, fluctuating concentration, or headache. The patient may react with puzzlement and alarm to these changes, since he was reassured that recovery would be immediate and complete. The patient may then try even harder to overcome the symptoms, and his efforts, if unsuccessful, create further frustration, anxiety, and depression. These psychologic forces may eventually incapacitate the patient even though the organic effects may have largely disappeared.

CONCLUSION

Current knowledge of long-term cognitive and behavioral effects after head injury is extensive. Outcome is affected by such variables as age, availability of rehabilitation services, severity of initial injury, and premorbid personality.[74] Residual cognitive and behavioral deficits, as underscored in this article, substantially interfere with the patient's ability to maintain gainful employment or adequate psychosocial adjustment. Prigatano et al[75] estimated that only one third of patients with severe closed head injury return to work after "traditional" rehabilitation. Thomsen[76] examined 40 severely injured patients 15 years after trauma. They had not participated in intensive rehabilitation. She found memory defects, dysarthria, and permanently disruptive emotional and personality changes in almost all patients. The poorest outcomes were associated with brainstem involvement or severe anterior lesions. Van Zomeren and Vandenburg[13] followed 50 patients with severe head injuries; after 2 years, 84% still reported forgetfulness, cognitive slowing, poor concentration, and behavioral problems, such as irritability, loss of initiative, and depressed mood. These patients did not participate in active rehabilitation. Levin et al[15] followed 27 patients who had a Glasgow Score of 8 or less at the time of hospital admission. At 1 year follow-up, they found that 17 patients had significant residual problems with memory, language, and personal social adjustment.

Several studies suggest that immediate interdisciplinary rehabilitation may assist patients and their families to realize cognitive, behavioral, psychosocial, and physical gains they might otherwise not achieve. Prigatano et al[75] compared 18 head-injured patients who participated in an active rehabilitation program with 17 untreated controls. Treated patients showed modest gains in neuropsychologic functioning, but significant improvements in emotional status and interpersonal skills; however, the mean interval after injury was almost 2 years at the time rehabilitation was initiated. More to the point, Cope and Hall[81] suggested that early intervention increases the probability for successful outcomes. The patients treated by Prigatano et al might have shown even greater improvements with earlier rehabilitation.

Emerging clearly from the existing literature is the need for comprehensive rehabilitative services for head-injured adults and their families. Although the behavioral and cognitive consequences associated with traumatic brain injury are now recognized, remediation approaches are poorly understood. Head-injured patients must be offered rehabilitation programs designed to improve neuropsychologic impairments,[77] treat behavioral deficits,[78,79] and educate families.[80] Through careful outcome studies, the impact of these programs can be evaluated. Systematic efforts to educate medical personnel about the untoward cognitive and behavioral consequences of both mild and moderate to severe craniocerebral trauma are also long overdue and require immediate attention.

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Vol. 5, No. 3, Sept., 1985
Thieme Publishing Co., New York