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An Introduction to Severe Closed Head Injury and Issues in its Rehabilitation

Jason Cool
Carroll College, Helena, MT

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An Introduction to Severe Closed Head Injury and Issues in its Rehabilitation

Jason Cool

Carroll College
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Abstract

This paper introduces the possible consequences of a closed head injury and the prospects for rehabilitation. It is divided into four sections. The first section explains the neurological consequences of acceleration/deceleration injuries. The second section deals with the clinical consequences. The third section introduces rehabilitation theories. And, the final section offers suggestions for developing a treatment program. It was concluded that further research must be conducted before rehabilitation programs will be able to effectively reintegrate head injury survivors into society.
An Introduction to Severe Closed Head Injury and Issues in its Rehabilitation

One in every 500 Americans suffers from some form of traumatic brain injury (Tollman, 1988), and the victims are far from random. People between the ages of 15 and 24 are the most vulnerable (Tate, 1987b). This fact makes severe head injury even more tragic, when one considers that it leaves people with permanent mental, and sometimes physical, deficits without affecting their longevity (ibid.).

The management of closed head injury is a relatively new issue in our society. Recent advances in emergency evacuation procedures and new neurosurgery techniques have saved many of those who otherwise would have died (Levin, 1990). Such new technology has left us with more survivors, but has left us unable to answer the tough question of how to deal with the survivors.

Advocacy organizations formed by family members, like the National Head Injury Foundation, demand a higher quality of life for their injured relatives. Organizations that must bear the cost of long-term care also eagerly await better rehabilitation options (Levin, 1990). Health care providers are frustrated too. They feel compelled to reintegrate survivors into society at the highest possible level of functioning (Ruff et al., 1989). Unfortunately, today's "highest possible level" is far from preinjury status.
This paper consists of four sections. The first section will describe the physical damage to the brain that occurs in closed head injuries. The second section will describe the possible clinical consequences of those injuries, and the third section will explain the current theories that guide rehabilitation. The final section will suggest guidelines for developing a treatment program.

The Injury

There are two major types of traumatic brain injury. The far less common kind occurs as the result of a penetrating wound. These injuries are usually caused by bullets or knives, but can be caused by anything capable of penetrating the brain (Kampen and Grafman, 1989). The more common type of brain injury is caused by rapidly accelerating or decelerating the head. This paper will focus on the latter type. This section will describe the brain damage that occurs in acceleration/deceleration type accidents.

Primary Injury

Primary injury refers to the damage that occurs immediately upon impact in a crash. As the head stikes a relatively immoveable object (e.g. steering wheel or concrete), the skull, which is light in comparison to the brain, comes to a quick hault. The brain, however, keeps
moving and crashes into the skull. This creates positive pressure at the point of impact and negative pressure opposite the point of impact.

Intuitively, we would expect the positive pressure to cause the most damage. However, studies have shown that as the neurons near the point of impact are compressed, they simply move closer together until the pressure subsides (Pang, 1989). Far more damage occurs in the regions opposite the impact (contrecoupe injuries). Here, the delicate neuronal connections are stretched beyond their elastic capacity. This causes wide-spread neuronal tearing and damage to the tiny blood vessels that vascularize those neurons (ibid.). If the impact is forceful enough to create extreme negative pressure, the phenomenon of cavitation may result. Cavitation, the "boiling" of cerebral spinal fluid, occurs whenever the pressure inside the skull becomes lower than the fluid's vapor pressure. During cavitation, gas bubbles violently form and then collapse, causing extensive tears to the region opposite the impact (Pang, 1989).

While the pressure created in an acceleration/ deceleration accident contributes to contrecoupe injuries, shearing is responsible for the majority of the damage incurred in the rest of the brain (Adams et al., 1982). Shearing occurs whenever two adjacent portions of the brain move in opposite directions. In acceleration/ deceleration accidents, the bulk of the brain moves toward the point of impact. During this movement, several pieces of the cortex
get caught on the many bony ridges on the inside of the skull. This pushes the surface of those brain areas in the opposite direction. As a result, neuronal tearing occurs on, and below, the brain's outer surface (Pang, 1989).

Both shearing and negative pressure account for surface injuries, but closed head injury also results in damage deep within the brain. Such damage may be caused by a phenomenon called ellipsoidal deformation. The skull, viewed from the top, is naturally in the form of an ellipse. When the forehead or the back of the head hits a stable object and comes to an abrupt stop, the long axis of the skull compresses. The short axis expands. In effect, the head's natural elliptical shape becomes more circular. The damage to the inner brain occurs as a combination of shearing and negative pressure created in the center of the brain as the lateral portions move outward to accommodate this new circular shape (Pang, 1989).

**Secondary Injury**

Secondary injury takes place in the hours or days following primary injury. After the initial insult to the brain, a variety of pathological events occur. Alone, each leads to further brain injury. Together, these events can cause death.

One of the most obvious sources of secondary injury comes from damage to the brain's circulatory system. Blood clots inside or around the brain, called cerebral hematomas,
occur from one to three days after initial injury. Wherever these hematomas form, they restrict blood flow to the surrounding neurons, causing them to die. Hematomas also take up part of the limited space inside the head. As a result, they contribute to increased intracranial pressure (Pang, 1989).

A less obvious cause of secondary injury is called cerebral edema. Edema is nonspecific response to brain trauma that increases the brain's water content. Cytotoxic edema occurs when metabolic toxins enter neurons through damaged cell membranes. These toxins cripple the cell's sodium-potassium pump, allowing sodium to seep into the cell. Water passively follows the sodium in an attempt to maintain the concentration gradient, and eventually engorges and kills the cell. Vasogenic edema, by contrast, is characterized by a breakdown in the blood-brain barrier and increased capillary permeability that eventually leads to water retention in the space between neurons. Both types increase intracranial pressure (Pang, 1989).

Cerebral swelling, edema, and hematomas all contribute not only to each other, but more importantly, to increased pressure within the skull. Increased pressure has two serious consequences. First, it forces cerebral arteries and arterioles to constrict or collapse. This cuts the blood supply to a great deal of the brain, and thereby cuts the oxygen and nutrient supply, causing cell death. Second, it forces the brain to shift outward into every available space
in the skull. The result is usually herniation through the base of the skull. Herniation and restricted blood flow combine to kill massive amounts of brain tissue, particularly in the brain stem and the hypothalamus (Pang, 1989).

**Damage Summary**

The medical advances that allow many closed head injury patients to survive deal exclusively in minimizing secondary injury (Pang, 1989). Nothing can be done about primary injury, except prevention. In the days and weeks that follow an accident, the first indicators of damage will emerge. The length and depth of the post-injury coma correlates fairly well with the severity of the injury (Uzzell et al., 1987). Another indicator is the the length and extent of post-traumatic amnesia (Gronwall, 1989).

Once a patient becomes both medically stabilized and conscious, physicians can assess the overall physical damage by employing computerized axial tomography ("CAT scans") (Prigatano, 1987). Closed head injury can cause a seemingly infinite number of injury combinations. Injuries are both localized in some areas and diffuse throughout the entire brain (Eames et al., 1989).

While each patient exhibits a unique profile, there are typical patterns found in many patients. For example, the frontal and temporal poles of the brain, the ventral surfaces of the frontal lobes, and the structures near the lateral fissures typically suffer the most damage (Tollman, 1988).
These injuries are easily explained by the brain's shearing on the bony structures on the lateral and ventral surfaces of the skull and by the pressures exerted at the skull's front and back. Because the frontal lobe is responsible for so many cognitive functions, the consequences of such injuries are profound (Prigatano and Altman, 1990).

In addition to surface injuries, deeper injuries from ellipsoidal deformation shear the neurons of the limbic system and its connections to and from the frontal lobe (Tate, 1987b). As a result, most closed head injury victims suffer at least some loss in motivation and ability to control their emotions.

The Clinical Picture

The first step in dealing with survivors involves formulating an accurate clinical assessment. To do this, clinicians use a variety of tests (Mattson and Levin, 1990). Unfortunately, few of them were designed specifically for those suffering closed head injury. Newer tests like the Executive Functioning Route-finding Task (Sohlberg and Mateer, 1989) are an exception. Consequently, an accurate diagnosis of a patient's impairments depends heavily on the skill and experience of the clinician. This section introduces the most common deficits that clinicians find when assessing closed head injury survivors.
Sensory-Motor/Perceptual Deficits

Motor deficits after a car or motorcycle accident are usually minor or nonexistent, and usually respond well to physical therapy (Tate, 1987b). Some sensory deficits are more common. For example, the shifting of the brain during a collision often severs the neural connections between the olfactory bulb and olfactory tissue, eliminating the sense of smell. If the impact of the crash is great enough to destroy significant portions of the occipital lobe, vision will also be impaired. The most common vision problem in head injured patients is loss of depth perception. Patients with such a loss perceive only two dimensions (Brooks, 1989). Additionally, due to auditory cortex damage (temporal lobe), some patients find it difficult to distinguish similar sounds (ibid.).

Communication Deficits

While gross motor control presents little problem, speech motor control is frequently impaired. The motor cortex controlling speech is located very near the lateral fissure. Damage to this area results in apraxia of speech. This involves the inability to fully control and coordinate movements of the mouth, tongue, and throat (Marshall, 1989). If the brainstem and cranial nerves that control speech suffer injuries, dysarthria may result (ibid.). Dysarthria is characterized by reduced speech intelligibility.

Head injured patients also have trouble comprehending
and using language (aphasia). Patients with left dorsolateral lesion of the frontal lobe have difficulty initiating speech and often exhibit perseveration: they repeat the same phrase or word continually. Patients with right frontal injuries tend to speak using stereotyped phrases and have difficulty putting ideas into words (Mattson and Levin, 1990).

**Learning and Memory Deficits**

Deficits in memory represent the most common subjective complaint after closed head injury. Over half of all patients with severe injury have at least some form of memory loss (Uzzell et al., 1987). Such impairments range from slight forgetfulness to severe amnesia (Brooks, 1989). Typically, patients suffer from a decreased short-term memory and have difficulty committing new information into long-term memory. In other words, many patients can no longer learn from experience (Miller, 1991).

In addition to problems with memory, many patients have a reduction in attention span and concentration. Therefore, they are easily distracted, often lose interest in the middle of simple tasks, and become confused if they are asked to do more than one thing at a time (Kay and Silver, 1989). Concentration deficits also reduce the speed of information processing, further hindering the ability to learn (Brooks, 1989).
Executive Deficits

Like learning and memory problems, executive impairments can gravely effect a patient's quality of life. The executive system includes all of the mental functions necessary for formulating goals, planning a means to achieve them, and carrying them out efficiently (Lezak, 1982). Shearing damage to both the limbic system and the orbital portion of the frontal lobe, often results in numerous executive problems. It is common for such patients to lack self awareness, awareness of deficits, and the ability to set goals or plan activities (Ylvisaker and Szekeres, 1989).

Many of them cannot inhibit their own behavior. Some cannot even make use of environmental cues about the appropriateness of their actions. Consequently, many patient's interactions and communications with others are socially incorrect. When, asked to evaluate their own behavior, many patients respond in strictly global terms. They either say that their behavior is always good, or that it is always bad, instead of evaluating the specific action in question (Ylvisaker and Szekeres, 1989).

Patients with executive problems may also fail to explore more than one solution when solving simple problems. They may also fail to evaluate relevant information regarding the appropriateness of their suggested solution (Foxx et al., 1988). Executive impairment erodes the patient's ability to utilize abstract thinking, basic reasoning techniques, and logic. These problems can appear to be subtle in comparison
to sensory and communication deficits, but they are terribly serious (Finlayson et al., 1987). All forms of cognitive rehabilitation rely heavily on executive functioning. Thus, impairment presents a formidable challenge to both clinicians and theorists.

Socioemotional Consequences

Patients with severe closed head injuries display a vast array of emotional changes. These include apathy, anxiety, silliness, irritability, inflexibility, disinhibition, immaturity, and depression (Miller, 1991; Tate, 1987b). The extent and quality of these changes varies greatly and depends on both reactive and organic factors. Depression and anxiety are often due to the patient's normal emotional reaction to his/her new disabilities (Miller, 1991). Drive disorders (apathy) and control disorders (disinhibition) are usually due to damage to the amygdala and uncus (Lezak, 1989).

Head injured patients may also suffer from profound changes in personality. Like emotional changes, personality changes are caused by a combination of organic and reactive factors. Patients may become paranoid, sexually impulsive, more angry and aggressive, more emotionally dependent, and much more egocentric (Miller, 1991). If frontal lobe damage is severe and bilateral, patients may appear lazy or aloof (Prigatano, 1986b). Specific patterns of frontal lobe damage seem to influence some of these changes. Preinjury
personality, however, can have even more of an influence. This is because many of the patient's new personality characteristics are due to decreased inhibition (Mattson and Levin, 1990).

With such drastic personal changes to the patient, come drastic interpersonal changes. Patients who unsuccessfully attempt to hide and compensate for their deficits often find themselves misunderstood and rejected (Carberry and Burd, 1985). Other patients suffer rejection simply because of their personality changes and the unrealistic expectations of others. This kind of rejection is widespread and is probably felt most when the patient is with his/her family. Immediate family members often feel trapped, abandoned by friends and extended family, guilty for their feelings of resentment toward the patient, and thoroughly frustrated (Miller, 1991).

**Current Rehabilitation Approaches**

The complex and devastating array of disabilities after severe closed head injury makes effective rehabilitation an immense challenge. Current theories and techniques of intervention lack widespread support and have significant weaknesses (Prigatano, 1986b). Nevertheless, rehabilitation attempts are made. At its best, rehabilitation allows some patients to return to work and live independently (Prigatano, 1984). For others, it improves social interactions
(Prigatano, 1986b). And for patients with more serious injuries, rehabilitation may foster emotional adjustment or eliminate the need for 24-hour care. Goals of rehabilitation are numerous and depend greatly on the quality of the patient's problems and remaining strengths.

**Behavioral Techniques**

Behavioral techniques are particularly useful to occupational therapists in retraining activities of daily living (dressing, bathing, etc.) (Giles and Clark-Wilson, 1988). Operant conditioning techniques that incorporate positive reinforcement (token economy) and overlearning have two advantages. First, they increase the incentive to complete targeted tasks. Thus, they often succeed with patients having drive disorders when other techniques fail (*ibid.*). Second, behavioral techniques inherently provide a structure by which patients with control disorders are better able to manage their actions (Tate, 1987a).

The behavioral approach alone cannot do much for the patient beyond teaching self care skills. Nevertheless, the value of positive reinforcement cannot be overemphasized. R.L. Tate (1987b) explains:

"...the severe head injured person is in a continual punishment situation and nothing he or anyone can do will turn off the punishment (i.e. be as he was prior to the injury). ...a common response of an organism that is being punished is escape or avoidance
behavior, which is likely to occur in situations such as therapy...positively reinforcing appropriate behaviors is not only an effective method by which to obtain behavior change, but also that given the circumstances it is more humane and readily accepted by the patient and family".

Compensation and Substitution

The compensation and substitution approaches assume that disabilities suffered from head trauma are permanent. In the case of substitution, a remaining cognitive asset is used to replace one lost to injury. This is analogous to teaching a blind person to read braille: using tactile information, to substitute for the ability to see. Substitution allows a patient to deal with the same problems as before the injury with new methods (Prigatano, 1986a). An example of substitution in the head injury rehabilitation setting would be the use of extensive notes to substitute for memory impairments.

The compensation approach attempts to teach patients to minimize or circumvent problems created by cognitive deficits (Prigatano, 1986a). For example, when using compensation instead of substitution, patients may be taught to compensate for memory loss by telling people, "I may not remember this later, so please remind me". In this way, patients can sometimes minimize the consequences of their impairment.
Restoration

The restoration approach attempts to restore or enhance impaired cognitive abilities through direct retraining or remedial techniques (Sohlberg and Mateer, 1989). It assumes that the brain can somewhat reorganize its own functions in response to injury. This reorganization will occur if the patient continually makes demands upon the impaired ability. The hope for spontaneous recovery was introduced by Luria in 1948 (cited in Prigatano, 1986b). He suggested a combination of proper teaching techniques and psychopharmacological techniques to disinhibit neosynaptogenesis. According to his hypothesis, the disinhibited brain will reassign lost functions to undamaged areas. For example, someone with left hemisphere damage might transfer those functions to the right hemisphere. The question of whether neosynaptogenesis may be a physiologic basis for recovery is currently being studied. While no clear answers have emerged, the implications are of enormous importance (Volpe and McDowell, 1990).

The Microcomputer

The use of the microcomputer in the rehabilitation of head injury obviously does not represent a theory. Rather, it can act as part of the therapy team by taking over many tasks previously done by therapists. In this role it has several advantages. The microcomputer can be particularly useful in assessment, since most tests and test batteries are easily adapted. The computer reduces administration
variation, experimenter bias, and errors in scoring (Davidson et al., 1987).

The microcomputer can also be programmed to administer rehabilitation tasks. These tasks can easily be tailored to individual needs and ability levels (Wood and Fussey, 1987). In addition to saving therapist's time, computers are much better able to precisely record and analyze performance data (Gianutsos, 1980). Most importantly, the majority of patients enjoy working with the computer; sometimes even more than with their therapists (Wood and Fussey, 1987).

Recent studies utilizing the microcomputer show promising results. One study found that computer therapy can reduce general confusion and help improve problem solving skills, new learning, and mental flexibility (Finlayson et al., 1987). Another study found that computer therapy resulted in improved verbal comprehension and improved memory skills (Ruff et al., 1989). While no studies appear to claim that computer therapy yields better results than more conventional methods, the cost effectiveness, record keeping ability, and flexibility of computers make them appear to be a worthwhile therapeutic tool.

When considering the microcomputer for use in a rehabilitation program, one must keep its limitations in mind. First, not all patients are suitable for computer therapy. To be suitable, patients must be able to discriminate between figure and ground, must be able to follow verbal or written instructions, and must not have
excessive visual or dexterity impairments (Stoneman, 1985). Second, very few programs are currently on the market. This means that a great deal of time will be required for designing and writing applicable programs (ibid.). Finally, the computer cannot replace the intuition, warmth, or empathy of a human therapist.

**Developing a Treatment Program**

Each of the current approaches have both limitations and strengths. Therefore, when developing a treatment program, it seems pragmatic to meld the best features of each. With this in mind, consider the following.

**General Program Guidelines**

After a comprehensive assessment, the first goal of a good rehabilitation program should be to include the patient's family members in the process. Brain injury disrupts the lives of all who are in close contact with the patient. Therefore, therapists should make a point of going beyond just answering family members' questions. They should immediately let the families know both what they can expect (the patient's specific clinical problems), and how they can best aid the patient (Miller, 1991). Therapists should carefully explain that there are limits to the progress that the patient can make. They should treat the concerns of
family members empathetically and, in some cases, offer the option of support groups or family psychotherapy (*ibid.*). If these and other considerations are not made, counterproductive family/staff conflict can result (Shaw and McMahon, 1990). This conflict can cause even the best of programs to fail (Tate, 1987b).

If a treatment program is going to succeed it must also be both flexible and holistic. All patients have a unique family situation. They all have a different profile of deficits and remaining strengths. And, they all have unique personalities. Therefore, the effectiveness of any treatment program is largely dependent on its ability to uniquely adapt itself to each patient (Tate, 1987a). Patient variety also requires a diverse rehabilitation team. Occupational, physical, speech, psychiatric, and psychological (cognitive and emotional) therapy may all be required for any given patient (Ruff and Nieman, 1990). Patient problems tend to be inter-related. Thus, it should not be surprising that experiments using a modular approach (treating a single deficit without treating others) have no long-term practical impact (Prigatano, 1987).

Even with a flexible and holistic program in place, careful selection of subjects is important. For example, one study found that patients who suffer more than eleven weeks of post traumatic amnesia are not likely to benefit from rehabilitation (Prigatano, 1987). There are also indications that certain injury patterns found in CAT scans shortly after
injury are good predictors of a patient's chances of responding to therapy (Uzzell et al., 1987). In addition to the injury, the patient's age, education level, premorbid psychiatric profile, family situation, and socioeconomic status all affect his/her chances for recovery (Kay and Silver, 1989). Without subjects who have a reasonable chance of benefiting from rehabilitation, no program can expect positive results.

The intensive holistic approach seems to offer the most hope, but it can create a problem. That problem is time/money. Many families can not afford the expense of a comprehensive in-patient treatment program for very long. Therefore the goals and methods employed in rehabilitation need to correspond not only to patient needs, but also to economic realities (Prigatano, 1987). Fortunately, minimizing time may not imply sacrificing effectiveness. One eight-week program showed treatment effects similar to those of a more complex six-month program (Ruff et al., 1989).

**Guidelines for Executive Impairments**

Executive impairments must be a key concern of any good rehabilitation program. The ability to become aware of one's deficits, to think logically, and to become motivated have always been assumed by current theories. Unfortunately, executive impairment takes those basic abilities away.

Many patients with executive impairments are hindered in forming even simple plans of action by their apparent lack of
motivation. In the past, amotivation was thought to result from emotional impairment or reaction. However, recent experiments indicate that it is probably caused by confusion (Prigatano, 1986a). This being the case, it is important to provide patients with enough structure in rehabilitation to keep them focused. Closed head injury patients suffer from more confusion than is typically suspected. Consequently, reducing it needs to be a major goal of rehabilitation (Prigatano, 1986b).

Because damage to executive functions often includes a lack of self-awareness, many patients grossly underestimate, or deny, that they have cognitive deficits. Several studies point to the fact that acknowledgement of deficits yields a better chance for recovery than does denial (Lam et al., 1988). Denial (agnosia) may be caused by psychological reaction or by organic factors. Damage to the heteromodal (association) cortex that lies just outside the structures of the limbic system has been implicated as one possible organic basis for deficit denial (Prigatano and Altman, 1990).

A good rehabilitation program should attempt to differentiate between organic based and reaction based agnosia. If denial is thought to be caused by patient reaction, the program should take time to address the problem as early as possible. After all, reaction based agnosia can usually be significantly reduced. If, on the other hand, agnosia is due to brain damage, it must be accepted as an obstacle to improvement that will make all stages of
rehabilitation more difficult. One tool therapists can use to evaluate extent and effects of agnosia throughout therapy is called the Brain Injury Rehabilitation Scale (Farmer and Frank, 1988).

Gains in cognitive functioning achieved in rehabilitation are of little value if they do not help the patient after rehabilitation. For this reason, the extent of skill carry over and problem solving generalization should be a primary concern. Early therapy programs usually failed to provide any lasting benefits (Prigatano, 1986b). More recently, researchers have discovered more effective methods. For example, Webb (1991) found that cues in the form of concrete symbols minimized the effects of learning impairments and poor abstract thinking. The result was increased carry over (Webb, 1991). Foxx et al. (1988) found that patients are much more likely to generalize problem solving skills when rehabilitation closely simulates real-life situations.

Problems with Measuring Treatment Effects

For treatment programs to improve, it is essential that clinicians make accurate records of each patient's assessment results, treatment received, and cognitive or emotional improvements. Scientific advancement demands it. Unfortunately, the scientific study of closed head injury is often difficult if not impossible for several reasons. First, current formal assessment techniques are unable to
present an accurate and comprehensive picture of patients' deficits (Sohlberg and Mateer, 1989). Furthermore, even if patients' deficits were clear, they are still complex and variable. Closed head injury has very few instances of isolated or "pure" deficits (Mattson and Levin, 1990). This makes the comparison of either matched or randomized subject groups difficult. Matching is almost impossible to do well without an enormous number of subjects to choose from, and randomization is unlikely to produce equivalent groups. The prospect of either randomization or matching is further complicated by ethical issues (Levin, 1990). The creation of no treatment control groups would likely violate many patients' right to the best possible treatment.

**Suggested Study: Computer Aided Problem Solving Module**

The scientific advancement of treatment programs will continue to be an important challenge. Given that the executive impairments are among the most devastating to the patient, one might consider designing a study that addresses one of them. For example, one might adapt the problem solving module developed by Foxx (See Foxx et al., 1989) to the microcomputer.

The problem solving module created by Foxx et al. (1989) used a nonrandom design that measured pretreatment/posttreatment performance. It successfully addressed generalization and carryover problems by using an overlearning approach and creating realistic problems. And,
it incorporated cuing to provide structure (*ibid.*).

This module could be easily adapted to the computer. Such an adaptation would not only save therapists' time, but would also provide more accurate records of patients' responses and progress. It could incorporate visual symbols (See Webb, 1991), instead of verbal questions, for cues. And, it could incorporate a positive reinforcement system for participation.

**Conclusion**

Severe closed head injury presents us with a terrible problem. It devastates the lives of both the victim and his/her family. It forces a significant financial burden on families and on health care providers. And worst of all, it is not very responsive to current rehabilitation approaches. Researchers remain far from reaching any solutions. Even a basic understanding how the brain responds, or can respond, to injury remains to be discovered. And unfortunately, it is difficult to employ traditional empirical methods when studying severe closed head injury. Nevertheless, the quest for answers must continue. Until they are found, the most effective method for addressing the problem will be to focus on prevention. Prevention techniques are simple. Wearing a seat belt, or a helmet, and driving below the speed limit dramatically reduce risk.
References


