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DIAGNOSIS, PHYSIOLOGY, PATHOLOGY AND REHABILITATION OF TRAUMATIC BRAIN INJURIES

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Accumulating clinical and experimental studies continue to elucidate and futher define the significance of intra- and extra-cranial factors which determine outcome of traumatic brain injury. These factors include severity of injury, age of the patient, presence or absence of premorbid brain insults, and associated pathophysiological events such as anoxia, respiratory arrest, hemorrage, edema, contrecoup and Wallerian degeneration. Following resolution of acute temporary symptoms, delayed complications include seizures, neurotic and psychotic disorders, earlier onset of stroke, earlier senescence, increased suicide risk, reduced life expectancy, progressive intellectual deterioration and development of symptoms comprising the post-traumatic syndrome. In spite of these diverse initial and later pathological sequelae, the reserve capacities of the brain for establishment of compensatory mechanisms can provide bases for a remarkable degree of spontaneous cerebral reorganization and recovery. The accumulating findings in patients with traumatic brain injuries reflect principles and factors underlying the organization, disorganization and reorganization of human brain function.

Key words: Head Injury, Whiplash, Post-Traumatic Syndrome, Cerebral Reorganization, Malingering, Cognitive Rehabilitation.

Current concepts of head injury reflect accumulating studies and observations whose origins can be traced back over 5000 years. The earliest known documentation of a systematic approach to diagnosis, prognosis and treatment of head injury is contained in the Edwin Smith Surgical Papyrus, thought to have been authored by the architect/physician Imhotep approximately 3000 years B.C. (Breasted, 1930). Head injuries suffered in war and accidents have long provided the clinical material which has spawned earlier and more recent theories of brain function. The advent of rail travel in 1825 in England and 1852 in Germany led to increasing numbers of head injuries due to inevitable accidents. Although the motor car was introduced in 1886, by the turn of the century auto related mishaps only accounted for a minority of injuries. For example, in 1904, the London Ministry of Interior Statistics revealed that of 24,375 accidents with 215 people killed, 90.8% were caused by horse drawn vehicles vs. 7.4% by autos. Even as late as 1932, horse drawn vehicles dominated the German freight industry (Neimann, 1995). In the same year only 6 of Russell's 200 Scottish head injury cases were the result of auto accidents (Russell, 1932). The first auto related fatality occurred in the United States in 1899, but this small beginning has grown to become a terrible stream of death and disablement, with the millionth U.S. road death being recorded by 1951 (Whitlock, 1971). Currently, it is estimated that there are 7 to 10 million new cases of head injury each year in the United States, with 50% or more the result of traf-

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fic accidents (Hartlage and Rattan, 1992). Motor vehicle accidents represent the major cause of traumatic brain insults in the United States, with approximately 9000 individuals per day suffering significant brain injury (Jennett, 1990b, Hartlage and Rattan, 1992). Furthermore, the numbers of individuals annually killed in traffic accidents in the United States exceeds the numbers of U.S. servicemen killed during the the entire Viet Nam Conflict (Rimel, Jane and Bond, 1990).

In the 1960s, recogition of the unfortunate side effects of automobile travel led Mercedes Benz to incorporate Bayrényi's concepts of passive safety such as energy impact zones and high surface area steering wheels (Niemann, 1995). However, many manufacturers found that when safety features were offered to customers as options, they were not purchased. In the United States, industry-wide auto safety standards were established by the Highway Safety Act of 1966 (Baker, 1971). Without government mandate of seat belts, front passenger head restraints, side impact protection, air bags and the like, mortality rates and severity of survivor's injuries would be even more staggering. Whitlock (1971) reported world-wide correlations between intrasocial aggression and traffic deaths, which prompted him to suggest that road injuries represent a form of social violence. While this position may seem extreme, we are nevertheless collectively willing to accept over 66,000 deaths per year and 3 to 5 million head injuries as part of the economic and human costs of convenient transportation. These human costs are apart from the enormous expense of medical treatments, rehabilitation and time lost from productive employment.

MECHANISMS OF BRAIN DAMAGE IN HEAD INJURY

The human brain has the consistency of Jell-O. It is bathed in cerebrospinal fluid (CSF) and is protected by the cranium, a term derived from the greek Kranion, meaning natural helmet. Further protection is provided by the 5mm thick scalp. Gurdjian (1973) demonstrated that presence of the energy absorbing scalp increases the force required to cause skull fracture ten fold. CSF has the protective effect of damping transmission of force to the brain during head injury (Pudenz and Shelden, 1949). Nevertheless, it has long been known that despite the remarkable mechanisms which protect the brain, it remains exquisitely sensitive and vulnerable to injury.

Until recently, the specific mechanisms of head injury which result in loss of consciousness and damage to the brain have been poorly understood. Courville (1953) credits Berengario da Carpi in 1518 for being the first to suggest a mechanism for concussion and thus establish the syndrome on a solid basis. Da Carpi noted petechial hemorrhages in the brain and postulated that concussion (or cerebrum commotium) was the result of the soft brain being thrust against the solid skull. In the next century, Paré (1649) called attention to the violent shaking of the brain mass following head injury. Exactly one hundred years later, Le Dran (1749) emphasized the importance of head movement in genesis of brain injury, a finding confirmed over 150 years later in experimental studies by Tillman in 1899 (Courville, 1953).

In this century, British studies, particularly those carried out at Oxford University and later at the University of Glasgow, have been prominent in the development of current concepts of traumatic brain injury. In 1941 Denny-Brown and Russell employed a swinging pendulum to deliver varying degrees of impact force to fixed and unfixed heads of live animals. They confirmed previous reports of the importance of head movement after impact in the genesis of brain damage. Yet their explanations of the mechanism for concussion re-

sion reflected persistence of the concept of commotium cerebrum. Two years later, an Oxford physicist named Holbourn applied Newtonian Laws of motion in studies modeling head injury with gelatin filled containers. He concluded that brain damage is caused by shearing stresses resulting from rotational acceleration. He called attention to the vulnerability of anterior temporal lobes due to the "grip" on the brain at this point exerted by the sphenoid bone. While he noted that impact injury could cause local damage, he claimed that rotational forces were responsible for the widespread diffuse damage which resulted in concussion. Holbourn illustrated the importance of head rotation by using the example of a flask filled with water and cotton fibers. If accelerated linearly, there is no movement of the fibers relative to the flask. However when the flask is rotated, there is obvious relative movement due to the inertia of the water and fibers.

Holbourn's findings were confirmed and elaborated by Pudenz and Shelden (1946) who fitted lucite calvaria to macaque monkeys and recorded the movements of the brain with high speed photography during experimentally produced head injuries. They clearly demonstrated the gliding movements predicted by Holbourn as the brain lagged behind the movement of the skull. They also confirmed that there was little brain movement after impact when the head was fixed. By contrast Gurdjian and Webster (1943) dropped weights from varying heights on to the fixed heads of dogs. They also used various devices (i.e. pendulum, spring and hammer) to strike unfixed heads. They disputed claims that the non-fixed head suffers more trauma than the fixed head.

In 1966 Ommaya, an Oxford trained neurosurgeon, reported the first of a series of studies focusing on differentiating the relative importance of impact vs. rotational forces as an approach to elucidating the mechanisms underlying genesis of brain damage due to head injury. He cautioned against direct extrapolation of findings in experimental studies with animals to human populations, because animals with small brains can tolerate much higher "g" forces than those with larger brains. For example, the mouse brain can experience 100 to 1000 times more "g" force than the human brain before suffering concussion. In 1971 Ommaya and Hirsch developed a precise apparatus for delivering either linear or rotational forces to the heads of primates. Their findings using living animals confirmed the importance of rotational forces predicted by Holbourn's physical experiments using gelatin filled containers. However, they did not confirm Holbourn's conclusion that rotational force alone was responsible for concussion. Instead, they determined that in order to produce concussion by rotational force alone, the brain had to be accelerated to twice the velocity that was produced in concussion by direct blow. They therefore hypothesized that 50% of the damage to the brain can be attributed to head rotation, and the remaining 50% to impact phenomena.

In an attempt to reconcile previous conflicting findings, Ommaya and Hirsch suggested that rotational forces were not found to be significant in Gurdjian's experiments because the cranio-spinal arrangements of dogs do not render them as vulnerable to rotational forces as much as the more upright postures which characterize primates. Moreover, Denny-Brown and Russell (1941) noted that the crushing effect caused by dropping weights on top of the fixed head is of a different nature than the mechanims underlying brain injury in clinical cases of head injury. Consistent with this Russell and Schiller (1949) pointed out that while crushing injuries of the skull are often fatal, survivors show either an absence or only slight degree of concussion.

In 1974, Ommaya and Gennarelli extended Holbourn's findings by pointing out that rotational forces decrease in magnitude from the surface to the center of the rotating brain

mass. They correlated loss of consciousness in their animals with injury to the rostral brain stem, which is centrally located in the rotating head. The principle of decreasing rotational forces predicts that a rotational injury sufficient to disrupt function of the brain stem will have even more severe pathological impact on neural structures the nearer they are to the surface of the brain. It is also possible that less severe rotational forces may cause damage to surface structures without significantly disrupting midbrain structures. Thus, according to the principle of decreasing rotational force, it is possible to suffer brain injury without loss of consciousness, but not loss of consciousness without brain injury.

These findings were subsequently confirmed by MRI studies documenting progression in severity of brain damage from midbrain to cortical structures after head injury (Eisenberg and Levin, 1989). The importance of midbrain structures in loss of consciousness has been supported in animal studies demonstrating axonal degeneration in the brain stem following "minor" head injury with brief loss of consciousness (Jane, Steward and Gennarelli, 1985). Some have suggested that an inhibitory cholinergic system within the rostral pons contributes to transitory unconsciousness in head injury (Hayes, Lyeth and Jenkins, 1989).

The role of shearing forces in destruction of neurons had been earlier documented at Oxford by Strich in 1961. She described white matter degeneration in 20 post-mortem examinations which she attributed to axonal stretching and shearing at the time of the accident. However, Oppenheimer (1968) subsequently demonstrated the importance of time as a variable in axonal shearing in post-mortem examinations of 59 head injured patients. He showed that axonal shearing with characteristic axoplasmic retraction balls was only seen 48 hours post injury. Later, Povlishock and Coburn (1989) confirmed delay of retraction ball appearance in head injury using a cat model. They described the sequence of events over a 12 hour period leading to axonal bifurcation. The first effect of head injury is impairment in anterograde axoplasmic transport. This is followed by accumulation of organelles and axoplasm with focal lobulation. Finally, 12 hours after injury, the swollen segment separates. This is followed by further Wallerian degeneration and focal deafferentiation.

CONTRECOUP

The mechanism of contre-coup injury has long been the subject of controversy. Fallopius first described damage to the brain opposite the site of impact in the 16th century, but the importance of this observation was not widely recognized until the 1760s when The Paris Academy of Surgery offered prizes to encourage papers on the subject (Pudenz and Shelden, 1949). In 1767 Morgagni claimed that contrecoup occurs because the brain when "shaken against a hard body, and driven back again thereby suffers two motions, diametrically opposite, in one moment of time" (Courville, 1953). This same mechanism explaining contrecoup was cited by Gurdjian almost two centuries later (1958). By contrast, Holbourn, Russell and others have attributed contrecoup injury to rotational forces tearing brain structures opposite the point of impact.

ACCELERATION, MASS AND VELOCITY

Experimental models of head injury based on physical laws of motion have clarified the roles of acceleration, mass and velocity in determining the amount of force exerted on the

brain. Russell (1932) pointed out that acceleration and deceleration of the head have identical pathological effects on the brain. He cited the simple yet effective example of an individual falling from a window to illustrate how differences in rates of deceleration can affect outcome. If the person falls onto a brick surface, the damage is great because of the suddenness of the change in velocity when the head hits the non-resilient bricks. In contrast, someone falling with the same velocity from the same height to soft grass suffers less damage to the brain because the rate of deceleration is less rapid.

In addition to acceleration and deceleration, mass and velocity are also important interacting factors determining the amount of force exerted on brain structures. High mass or velocity alone do not necessarily result in brain injury (Gurdjian and Webster, 1958). This can be illustrated by extreme examples of objects with great mass but little velocity (such as a glacier) and objects with little mass but high velocity (such as an electron). However, the less extreme masses and velocities which characterize cars and trucks on our public highways are more than sufficient to produce injury to the brain. But consider the example of person sitting in their car at a stop light who is suddenly hit from behind by a vehicle traveling 35 miles per hour. The potential for injury is much greater if the stationary car is hit by a large truck than by a subcompact car. This is because the truck, with its much greater mass, transmits much more energy to the stationary passenger than would a small car at the same velocity. Furthermore, the kinetic energy that a vehicle possesses increases at a rate proportional to the square of the velocity (Spivak, 1971). Therefore, a car travelling 50 miles per hour impacts with four times the energy of a car travelling at 25 miles per hour.

Even when the weights of vehicles and their rates of speed at impact are known, it is difficult to retrospectively assess the forces transmitted directly or indirectly to the head. As demonstrated by Russell's example, split second differences in rates of acceleration and deceleration which are difficult to measure outside of a physics laboratory can contribute to wide variations in degree of brain injury. One person may walk away from a serious car accident with no apparent sequelae even though the person sitting next to them was killed or permanently disabled. In addition to limitations in our capacity to determine the amount of energy transmitted to the brain, clinical assessment of head injury is complicated by effects of numerous other intra and extra-cerebral factors.

ASSOCIATED FACTORS CONTRIBUTING TO BRAIN DAMAGE

In 1941, Denny-Brown and Russell described transitory or permanent cessation of respiration immediately following head injury, calling attention to the role of anoxia in the pathophysiology of head injury. In comparison to other tissues, the vertebrate brain has much higher rates of energy consumption. While it constitutes only 2% of the body mass, the human brain consumes 20% of the total oxygen (Miller, Pentland and Berrol, 1990). Furthermore, the brain has a limited storage capacity compared to other tissues, and is therefore highly susceptible to interruptions in oxygen and glucose supply. In humans, tightening a neck cuff produces unconsciousness in 6 to 7 seconds and slow wave EEG activity in 10 seconds (Lutz and Nilsson, 1994). Patients who experience 5 minutes of anoxia or 15 minutes of substantial hypoxia sustain permanent brain damage (Walton, 1994).

Lutz and Nilsson (1994) pointed out that because brain reserves of oxygen and energy are trivial compared to rates of consumption, anoxia quickly leads to a series of systems failures in the brain which ultimately result in neuronal death. During the first 40 seconds

of anoxia, there is a precipitous fall in ATP, the energy which drives the intracellular pumps and maintains critical ion gradients. Failure of the ion pumps and loss of ion gradients leads to an explosive rise in excitatory neurotranmitters, which become excitotoxins. Ca^2 floods into the neurons, causing a number of dysfunctional events, some of which may lead to delayed neuronal death even after resolution of anoxia. In the later stages of anoxia, dissolution of neuronal integrity is hastened by edema and free radical formation (Miller, Pentland and Berrol, 1990). Associated injury to the body resulting in airway restriction and/or extensive blood loss can also cause anoxic brain damage. Acute emergence of seizures is not uncommon after head injury and can contribute to brain damage by increasing the brain's metabolic demands to levels which exceed available supply of oxygen and glucose. The pathological effects of seizures may be magnified at the time of head injury because oxygen and glucose supply may be already compromised as a result of respiratory or circulatory problems. Regardless of the apparent severity of head injury, there is always the risk of further brain damage as the result of intracranial bleeding. In such cases the patient may be lucid immediately after the accident but then lapse into a life-threatening coma.

Hemorrhage can lead to brain damage by causing vasogenic edema. Hemorrhagic blood can also enter the ventricular system and obstruct cerebro-spinal fluid absorption sites in the arachnoid villi. The result is development of hydrocephalus, which in turn causes further brain damage (Miller, Pentland and Berrol, 1990). Direct contact of blood with neural elements disrupts active transport mechanisms and triggers excessive acetylcholine release, leading to neuronal death. Immediate application of anticholinergic substances can greatly reduce the amount of neuronal destruction which normally would occur as the result of excitotoxicity (Hayes, Lyeth and Jenkins, 1989; Osterholm, Mathews, Irvin and Calesnick, 1995; Zasler, 1992).

Horn and Garland (1990) pointed out that brain injured patients are commonly the victims of multiple trauma and therefore may sustain damage to virtually any organ system extrinsic to the CNS. Conversely, damage to the brain alone can have widespread effects on multiple organ systems. Varying rates of participation and interaction of these and other pathological mechanisms in the process of neuronal destruction can produce a wide range of diverse initial and later outcomes following head injury. The unpredictability of the effects of the sum total of these mechanisms has prompted attempts to develop objective measures of severity of head injury which accurately reflect outcome.

MEASURES OF SEVERITY OF HEAD INJURY

Conventional clinical practice dictates diagnosis of concussion when head injury produces an alteration of consciousness without objective evidence of brain damage. If a CT or MRI study reveals an area of infarction or evidence of hemorrhage, the injury is then classified as a contusion or laceration. However, Jennett (1976) declared "Nothing has done more to confuse this field than continuing to classify cases according to the supposed occurrence of concussion, contusion and laceration of the brain." In 1928, Symonds suggested that duration of time until return to full consciousness might represent a good index of severity of head injury (Jennett, 1976). This concept was developed by Russell (1932), who demonstrated that duration of post-traumatic amnesia (PTA), or time until return of the patients capacity to record current events, is a useful predictor of outcome after head injury. In studies of over 1500 military cases with closed head injury Russell and Smith (1961) demon-

strated that patients with longer durations of PTA consistently showed poorer outcomes. Presence of PTA indicates that function of brain mechanisms underlying memory storage and retrieval has been disrupted either temporarily, or in more severe cases, permanently. It is important to point out that during the period of PTA, patients can carry out purposeful activity. Later, they often have isolated islands of memory for events which occured prior to resolution of PTA.

Some have criticized PTA as an index of severity of head injury, questioning the patient's ability to accurately recall when they regained the capacity to continuously record current events (Reitan and Wolfson, 1993). However, Jennett (1990b) and others have repeatedly pointed out that accuracy of PTA determination is not important beyond the ability to recall whether the duration was minutes, hours, days or weeks. The reliability of clinical estimates of PTA was demonstrated by Fortuny, Briggs, Newcombe, Ratcliff and Thomas (1980). Based on a study of 336 head injury patients, they found that objective estimates of PTA based on daily standardized testing matched well with clinical assessments of PTA by experienced neurosurgeons. The authors concluded that PTA was a measurable entity upon which independent observers can agree.

PTA less than one hour is considered to indicate a mild head injury, while PTA over one day is considered severe. Yet PTA of even a few minutes duration indicates that the patient has sustained brain damage (Jennett, 1976). Jennett also noted that duration of PTA is typically 4 times as long as coma. Henry Miller (1972) vigorously opposed validation of psychological complaints of head injured patients, but he nevertheless concluded that duration of post-traumatic amnesia is rarely faked or exaggerated.

PTA as an index of severity of head injury is of more limited value in cases with *focal* damage, for example due to depressed fracture or hematoma. Furthermore, it cannot be assessed until the patient has regained the capacity to record current events. As a result of these limitations, Teasdale and Jennett developed the Glasgow Coma Scale (GCS) in 1974 to permit immediate estimation of severity of injury in comatose and semi-conscious patients (Bond, 1990). The scale is based on the patient's eye opening, motor and verbal responses. PTA and the GCS permit assessment of severity regardless of the state of awareness of the patient after head injury. Jennett (1990a) concluded that the GCS is the best indicator of diffuse damage in the unconscious patient, and PTA is the best indicator in the conscious patient. The Rancho Los Amigos scale differentiates 8 levels of functioning ranging from "no response" to "purposeful and appropriate". The Rancho scale is typically used to track general level of function, improvements over time and for placement of head injured patients. However actual details of the patients capacities cannot be accurately deduced from the Rancho level (Lezak, 1995).

OTHER FACTORS DETERMINING OUTCOME: AGE AND PREMORBID CONDITION OF THE BRAIN

Symonds pointed out that it is not just the kind of head injury that matters, but the kind of head that is injured (Rosenthal and Bond, 1990). Subsequent studies have validated Symond's observation, and clearly demonstrated the importance of age and premorbid status of the brain as factors determining outcome of head injury. In 1932 Russell reported increasing fatality rates after head injury with increasing age. Two years later, his follow-up studies showed that older patients developed more frequent and severe symptoms than

younger patients. This led him to conclude that age is the most important single factor for estimating prospects of recovery after head injury. Russell's findings have been repeatedly confirmed in numerous subsequent follow-up studies of head injury (Denny-Brown, 1945; Barth, Macciochi, Giordani, Rimel, Jane and Boll, 1983; Binder, 1986; Dikmen and Reitan, 1977, Miller, Pentland and Berrol, 1990b).

By contrast, Levin, Benton and Grossman (1982) questioned the idea that "immaturity confers an advantage in withstanding the effects of diffuse cerebral insult". They instead claimed that the young brain "may be more susceptible to the effects of diffuse damage than the adult brain" (pages 190, 192). Fletcher, Levin and Landry (1984) cited poor outcomes in infants with respiratory distress, intraventricular hemorrhage and hydrocephalus as evidence that the younger brain is more vulnerable to diffuse injury and shows more limited recovery than the older brain. Since the studies they cited did not directly compare older and younger patients with similar degrees of brain insult, a more accurate conclusion for their review would be that infants with different types of early brain damage can show severe and persisting deficits and that infancy does not necessarily guarantee full recovery from early brain injury. Bruce (1995) pointed out that comparisons of effects of head injury in children and adults are invidious. Even comparisons of younger and older children are qualified by changes in anatomy, physiology, chemistry, and degree of myelination at different stages of development. Moreover, the skull of the younger patient shows decreasing malleability with increasing bone thickness and closing of the stutures and fontanelle.

Reports of poorer outcomes in younger than older head injured patients could partly reflect sampling bias and unwarranted assumptions about PTA and GCS scores. For example, older patients who do not survive severe head injuries are obviously excluded from study samples, while inclusion of younger patients who are more apt to survive similarly severe injuries bias findings toward the conclusion that younger patients have poorer outcomes. Reports of greater deficits and more limited recovery in older than younger head injury patients suggest that older patients will also show longer PTA and lower GCS scores than younger patients with similarly severe head injuries. Consistent with this, Russell and Smith (1961) reported poorer outcomes in older than younger adults despite similar duration of PTA. Thus, older patients may suffer less severe head injuries than younger patients yet show poorer outcomes even when duration of PTA is held constant. Therefore, the tacit assumption that duration of PTA and CGS scores reflect comparable extent and degree of brain injury or predict comparable outcomes across all age groups is clearly unwarranted.

Despite selected contradictory reports, numerous experimental and clinical studies have provided additional support for the conclusion that older patients survive less often and recover to a lesser extent than younger patients. For example, Margaret Kennard noted that younger animals tolerate and recover more completely from cerebral ablations than older animals (Finger and Almli, 1988). In support of her findings, Kennard quoted Vulpian's 1866 conclusions in experimental studies of animal hemispherectomies: "When one attempts an experiment of this sort on mammals; it is necessary whenever possible to make use of very young animals; for on the one hand, they support the operation well, and on the other, the functional relations between the different parts of the encephalon are not yet as narrowly circumscribed as they later become; to such effect that ablation from the brain has less influence on the action of the parts than in the adult animal" (Kennard and Fulton, 1942, p 595). Kennard's report of the lesser effect of ablations in the younger than older animal is now known as the Kennard Effect.

The development of hemispherectomy for adult onset malignant glioma and later for treatment of intractable seizures in infantile hemiplegics provides the most striking con-

firmation of Vulpian's and Kennard's findings regarding the importance of age in determining outcome. Smith (1983b) reported that adults showed persisting aphasia with no gross impairment of non-verbal visual spatial cognitive functions following left hemispherectomy and a reciprocal syndrome following right hemispherectomy. By contrast hemispherectomy for lateralized epileptogenic lesions in infantile hemiplegics showed no systematic differences in development between verbal and non-verbal capacities in long term studies of 36 children with left and 28 with right hemispherectomy. In studies of smaller samples of children, Dennis and Kohn (1975) and others have reported that special language testing revealed subtle defects and limited development of syntax and language following left hemispherectomy vs. more limited development of non-verbal visual spatial cognitive functions following right hemispherectomy. However, Leleux and Lebrun (1981) and Bishop (1983, 1988), who failed to replicate Dennis' findings, emphasized that the conclusions of Dennis and her colleagues are based on the unwarranted tacit assumption that after hemispherectomy, the remaining right or left hemisphere is intact and undamaged.

In addition to age, premorbid condition of the brain is also a critical factor determining outcome of head injury. Numerous studies have reported that patients with pre-existing head injuries show more marked and persisting deficits after a second head injury than would be expected based on the severity of the injury (Binder, 1986; Miller, Pentland and Berrol, 1990). This is because the initial head injury resulted in diminution of the brain's reserve capacity for reorganization and recovery of function. Head injured patients with other types of pre-morbid brain insults such as stroke, hydrocephalus, long term alcohol abuse, etc. will also show more severe and persisting impairments than those whose reserve capacities are intact. Furthermore, cases with mild head injury who show poorer than expected outcomes may include apparently normal individuals with unsuspected pre-, peri- and/or early post-natal brain insults.

DELAYED COMPLICATIONS OF TRAUMATIC BRAIN INJURY

Attention of emergency room and acute care health practitioners is typically focused on treating the emergent and often life threatening initial effects of head injury. However numerous studies have emphasized the importance of diverse serious delayed complications which may not develop until weeks, months or years after the injury. One of the most serious and debilitating delayed complication is the emergence of post-traumatic seizures. Gurdjian (1958) estimated that 4 to 5% of patients with closed and 15 to 22% with open head injury will develop seizures. In contrast, Jennett (1990a) reported a more conservative estimate for patients with closed head injury (1 to 2%), but cited higher rates of epilepsy after depressed fracture (up to 60%). Jennett also cited various factors which predict development of later epilepsy including age less than 5 years, presence of hematoma requiring evacuation, depressed fracture, epilepsy in the first week and duration of PTA over 24 hours. He noted that 1/4 of cases may appear after four years, but some first develop seizures as many as 40 years post trauma. Lishman (1987) and Rosenthal and Bond (1990) noted that head injured patients not infrequently show severe psychiatric problems including bipolar disorder, schizophrenia and depression. Other longer term studies of head injury patients report earlier onset of stroke (Anttinen, 1960), accelerated senescence (Walker, 1972) and shortened life-span (Hillbom, 1960; Walker, Leuchs, Gruter and Caveness, 1971). In contrast to the typical pattern of varying degrees of gradual improvement after head injury

some may show early improvements followed by progressive irreversible mental declines, even after mild head injury (Snoek, Minderhoud and Wilmink, 1984). Brooks (1990) reported that patients with previous head injuries showed greater deterioration in cognitive function than controls when oxygen levels are reduced. This supports Gronwall's (1989) caution that even with normal scores on standardized testing, the head injured patient may demonstrate impairment in the face of physiological, cognitive or social stress. Increased risk for suicide is another serious complication of head injury. Long term outcome studies of head injured patients have shown that suicide accounts for one out of every seven deaths (Lishman, 1987). Furthermore, suicide risk increases with time and peaks 15 to 20 years after the injury (Rosenthal and Bond, 1990).

THE POST TRAUMATIC SYNDROME

Commenting on a presentation by Dikmen and Reitan (1976) focusing on varying degrees of improvements in neuropsychological test performances after head injury, Weinstein pointed out that test results do not predict which patients will suffer from sexual problems, work difficulties, impairment of social skills, insomnia, concentration problems, headache and dizziness. Weinstein emphasized that neuropsychological batteries only touch tangentially on the wide spectrum of disturbances that collectively constitute the post-traumatic syndrome. Russell (1932, 1961) noted that unlike other outcomes of head injury, symptoms comprising the post-traumatic syndrome do not seem to be related to severity of head injury. In fact, Jennett (1990b) and Denny-Brown (1945) pointed out that post-traumatic sequelae are seen more frequently in patients with minor than severe head injury. Gronwall and Wrightson (1974) suggested that decreased information processing rate leads to headache, fatigability and irritability as patients must exert more mental effort and yet perform tasks less effectively than before the accident. Rutherford, Merrett and McDonald (1979) reported persistence of post traumatic symptoms after minor head injury for over a year in patients who were not involved in litigation. Rutherford subsequently reported that patients may still be complaining of symptoms as many as 16 years after minor head injury. In contrast to earlier reports which suggested no organic deficit in mild head injury, Benton (1989) and Wrightson (1989) described findings of later studies which confirmed the validity of the post-traumatic syndrome and its emergence regardless of age of the patient or claims of compensation.

Despite establishment of the validity of the post-traumatic syndrome and its prevalence in cases of mild head head injury, patients with apparently "minor" blows to the head are often sent home from emergency rooms with instructions that they will get better. Yet, left to their own devices, many do not. The remarkable sensitivity and complexity of the human brain permits us to experience an enormous range of emotions, sensory impressions, thoughts and creative impulses. Even an apparently minor blow to this sensitive organ (with little or no PTA) can result in very real and significant symptoms which may seriously impair the patient's ability to work productively or relate normally to others. Although they may present a normal social facade, patients with this "invisible malady" experience a wide range of disturbances which can persist even after test findings have returned to normal levels (Dikmen, Temkin and Armsden, 1989). Furthermore, patients report that sequelae comprising the post-traumatic syndrome can be more disturbing and debilitating than persisting cognitive losses (Smith, 1983b; Jennett and Teasale, 1981).

Therefore a thorough neuropsychological assessment of a patient with head injury should always include a careful evaluation of the emergence, nature, severity and frequency of symptoms comprising the post-traumatic syndrome.

COMMON SEQUELAE OF TRAUMATIC BRAIN INJURY

1. Headaches
2. Sleep Difficulties (falling asleep, waking in the night, nightmares, lack of dreaming).
3. Libido Changes (most often hyposexuality, but sometimes hypersexuality).
4. Unusual sensations (pressure, tingling, heat cold, numbness, pilocarpia, weakness, etc.)
5. Dizziness
6. Irritability
7. Fatigability
8. Distractibility
9. Social Isolation
10. Personality Change
11. Hearing difficulties (including tinnitus and phonophobia).
12. Visual Problems (including photophobia).
13. Anosmia and Ageusia
14. Weight loss or gain
15. Reduced tolerance to Alcohol
16. Unusual Mental Processes: Suicidal Ideation, Delusions, Hallucinations
17. Mood Swings

The wide range of cognitive deficits and post-traumatic sequelae reflect direct pathological disruption of brain mechanisms. However, the psychological impact of the head injury is another critical yet often overlooked issue in assessment and treatment of the patient.

THE PSYCHOLOGICAL IMPACT OF HEAD INJURY

Benton (1979), Jennett and Teasdale (1981) and Dikmen, Temkin, Armsden (1989) have pointed out that emotional difficulties, personality changes and neurotic reactions may be the most prominent, disabling and sometimes the only consequences of head injury. Bond (1990) cited Luria and Goldstein's shared view that changes to personality are often a more serious barrier to effective rehabilitation and functional recovery than cognitive or intellectual changes. Consistent with this, Denny-Brown (1945) reported that mental symptoms related to anxiety were more important than improvements on standardized cognitive measures in predicting long term disability. The impact of brain damage on personality was strikingly demonstrated in the remarkable 1848 case of Phineas Gage. As the result of an accident at work, he had a steel tamping rod driven up through his left cheek and out the top of his skull. Despite development of incranial infection, he survived, albeit with a marked personality change. Where before he was responsible, hardworking and well-liked, he subsequently became obstinate, capricious and childlike (Steegmann, 1962).

The relationship between brain injury and resultant changes in personality and behavior is central to Kurt Goldstein's organismic approach to the study of human behavior. According to Goldstein's (1952) view, the most basic drive underlying the behavior of an

organism is the struggle to realize its own nature and express its full potential. A prerequisite for such growth is the ability to cope effectively with basic environmental demands. Brain injured patients with reduced functional capacities still try to respond in the best way they can to discharge the tension brought on by environmental demands. An apparently minor demand which the patient cannot discharge effectively can result in a pathological reaction which appears grossly out of proportion to the demand. This is Goldstein's Catastrophic Reaction. This principle of behavior is reflected on a smaller scale in non-clinical populations in a friend's unexpectedly harsh response to our apparently neutral query. The outburst which is out of proportion to our query is ignited by underlying frustration about an unrelated problem. The clinician must be aware of and sensitive to the head injured patient's limitations and the potential that the assessment procedure holds to uncover compromised capacities and threaten self-esteem. The brain injured patient may of course suffer so much damage as to appear oblivious to functional loss. In this case Goldstein explains that the patient does not show emotionality because he has lost the capacity to grasp the situation in a way that would evoke emotion. Goldstein noted that in order to reduce the number of potentially threatening stimuli, brain injured patients often stay alone or may prefer to only be with people they know well. They may also choose to be in familiar surroundings. Like many non-brain injured people, they may preoccupy themselves with activities that are within their reduced limited capacities and avoid other activities with which they cannot cope.

The patient's reaction to cognitive losses, physical limitations, and symptoms comprising the post-traumatic syndrome can vary widely. Some may find themselves suddenly involved with the criminal justice system as a result of impaired judgement, increased impulsivity, marked irritability and inability to delay gratification. Others may attempt to self-medicate with drugs and alcohol. Many patients, especially young adults are prone to denial after head injury and are deeply threatened by the possibility that they may have actually suffered irreversible damage to their brains. In addition to denial, patients may employ other defense mechanisms of repression, rationalization and projection in attempting to come to terms with the sequelae of the injury. Others are aware of emerging problems but lack insight regarding their causal relationship to the head injury. The sometimes strange, indescribable physical sensations and emotional fluctuations often lead patients to fear that they are "losing their minds". Rosenthal and Bond (1990) reported that during the initial denial stages, patients are often irritable. With passage of time and increasing insight, depression and anxiety set in as they begin to realize that the rest of their life will be difficult and a struggle. Patients whose self-concept is firmly based on their roles as care-givers or wage-earners experience feelings of loss and lack of self-worth when they can no longer discharge these functions. Commenting on this phenonmenon Parker (1970) wrote: "Many patients with an accident neurosis remind me of those successul people who have attained their goal and stop work to enjoy life, but find they cannot. It is important for their state of mind to keep on working. If retirement or an accident prevents them, they go to pieces. When I appreciated this fact, so many of the perplexing features of accident neurosis fitted into place" (p. 365). Post-traumatic impairment of sexual function represents another dimension of loss which can have a devastating impact on the patient's self-concept and feelings of self-worth (Griffith, Cole and Cole, 1990). In Goldstein's terms, loss or impairment of capacity to function effectively strikes at the heart of our most basic drive for self-expression, fullfillment and self-realization. Therefore supportive therapy focusing on helping the patient to address and work through these issues is a critical and indispensable part of the rehabilitation process.

MILD HEAD INJURY

Of the 7 to 10 million people per year who suffer head injury in the United States, only 500,000 patients are hospitalized. Therefore, the majority of cases fall into the category of "mild" head injury. That is, head injury followed by either very brief or no apparent PTA. In the 4th and 5th century B.C., Hippocrates held that no head injury is trifling (Gurdjian, 1958). However in the 1960's the prevailing notion was that effects of concussion were completely reversible (Gronwall, 1989). In 1968, Oppenheimer reported pathological findings in 59 post-mortem cases, including 2 with mild head injury who died of pneumonia and fat embolism. The findings revealed "permanent damage in the form of microscopic destructive lesions" in the two patients who suffered what were considered to be "trivial head injuries".

Despite accumulating evidence to the contrary, many clinicians still cling to the view that loss of consciousness is the sine qua non of brain damage. They may cite lack of objective CT or EEG findings in such patients, or studies which have reported no conclusive evidence of cognitive impairment after mild head injury (Schoenhuber and Gentilini, 1989; Gentilini, Nichelli, Schoenhuber, Bortolotti, Tonelli, Falasca and Merli, 1985). However, absence of evidence is not evidence of absence. Furthermore, numerous other reports have documented organically based emergence and persistence of cognitive deficits, attentional problems, psychiatric sequelae and symptoms comprising the post traumatic syndrome after mild or minor head injury (Barth, Macciocchi, Giordani, Rimel, Jane and Boll, 1983; Benton, 1989; Binder, 1986; Gentilini, Nichelli and Schoenhuber, 1989; Leininger, Gramling, Farrell, Kreutzer and Peck, 1990; Merskey and Woodforde, 1972; Merskey, 1984; Miller and Jones, 1990; Ruff, Levin, Mattis, High, Marshall, Eisenberg and Tabaddor, 1989).

SPORT RELATED HEAD INJURY

Barth, Alves, Ryan, Macchiocchi, Rimel, Jane and Nelson (1989) documented neuropsychological deficits and symptoms comprising the post-traumatic syndrome in football players tested within 24 hours of mild head trauma (loss of consciousness less than 2 minutes duration). Follow-up testing suggested the problems resolved rapidly, within 5 to 10 days of onset. Binder (1986) concluded that the numerous reports indicating fewer sequelae in sports related injuries vs. those seen in auto accidents reflect greater mass and velocities involved in motor vehicle accidents. Although sequelae of sports related head injury may be generally less marked than those suffered in motor vehicle accidents, Gurdjian (1958) pointed out that boxers who sustain repeated blows to the head over many years can develop what Martland described in 1928 as the punch drunk syndrome. Sequelae include slurred speech, unsteady gait and Parkinsonian symptoms (Russell, 1932).

WHIPLASH INJURY

The term whiplash was introduced by Crowe in 1928 to describe injury to the neck in rear-end automobile accidents (Bernstad, Baerum, Lochen, Mögstad and Sjaastad, 1975). The injury consists of rapid hyperextension and/or hyperflexion of the neck without direct impact to the skull. The degree of hyperflexion of the neck and head forward and sideways is

restricted by the chest and shoulders. However during hyperextension of the head and neck backwards, extension may reach an angle of 140 degrees, far beyond the physiologically normal range of 45 degrees (Macnab, 1974; Toglia, 1976). Frankel (1959) and Gurdjian (1958) pointed out that arthritis increases the chance of nerve damage in whiplash. Onset of the pain can be delayed several days after the accident and may be progressive. Keith (1986) emphasized that the 2nd cervical nerve is vulnerable in whiplash, and though this syndrome is rarely diagnosed, it can account for persisting symptoms such as headache, neck tenderness and diminished sensation in the occipital region of the head.

Ommaya and Hirsch (1971) found that the chimpanzee is capable of withstanding much higher rotational forces than other primates because of its exceedingly strong neck muscles. Thus it is not surprising that in the studies of whiplash injury reviewed here, there is a predominance of females over males in the clinical samples by a ratio of more than 3 to 2. The greater female vulnerability to whiplash has been attributed to generally weaker neck muscles (Spitzer, Skovron, Salmi, Cassidy, Duranceau, Suissa and Zeiss, 1995). Weaker neck muscles not only subject women to greater hyperextension and hyperflexion, but also to greater rotational forces.

According to Fleming (1995) whiplash injury can cause muscle contusions, torn ligaments, bruising of the esophagus, intervertebral disc damage, fracture dislocations of the spine, carotid and vertebral artery injuries, as well as cerebral contusions and subdural hematoma. In view of earlier studies citing the importance of rotational forces in the genesis of brain injury, it is not surprising that the reported sequelae of whiplash include headache, amnestic symptoms, cognitive deficits, symptoms comprising the post traumatic syndrome, neck, shoulder and back pain, and spasms of the neck muscles (Bernstad et al, 1976; Ellertson, Sigurjónsson and Thorsteinsson, 1978; Hofstad, 1985; Kischka, Ettlin, Heim and Schmid, 1991; Nielsen, 1959; Seletz, 1958). Consistent with the anatomical vulnerability of the head and neck to hyperextension, Gay and Abbott (1953) pointed out that most of their 50 whiplash cases were the result of being struck from behind. Their patients reported numerous post-traumatic symptoms and remained handicapped for long periods of time considering the apparently "mild" character of the accident. Yarnell (1988) compared the findings of severe symptoms in whiplash despite objective confirmation of organic damage to similar reports of persisting and sometimes severe deficits after mild head injury. Gotten (1956) focused on the importance of monetary gain in recovery of 100 patients with whiplash. However he also noted that 12% did not show recovery after compensation and almost half who did recover had residual complaints five years post injury. Macnab (1974) reviewed outcomes of 266 patients with whiplash and reported that 45% continued to show symptoms despite resolution of their compensation claims.

Radanov, Di Stefano, Schnidrig and Ballinari (1991) studied 78 consecutive whiplash patients who were not involved in litigation and who did not show subtantial neurological findings. They concluded that premorbid psychosocial factors, negative affectivity and personality traits were not predictive of outcome. As might be expected if the symptoms were organically based, intensity of neck pain, degree of cognitive impairment and age were significantly related to outcome. However, in a follow up study, some of the same authors emphasized the apparent lack of major cognitive impairment after whiplash injury. Persistence of "cognitive disequilibrium" in some cases was noted, and attributed partly to medication effects (Radanov, Di Stefano, Schnidrig, Sturzenegger and Augustiny, 1993).

Consistent with numerous studies documenting the importance of rotational forces in the genesis of brain injury, Kischka et al (1991) attributed CNS damage in whiplash to high

acceleration forces (20 to 50g) acting intracranially. Interestingly, Macnab (1974) described recognition of whiplash injuries resulting from catapult assisted takeoffs on aircraft carriers. This problem was corrected by extending the pilot's seat to support the head.

Some have reported a lack of abnormal neuropsychological, EEG, CT or other findings in whiplash (Jacome, 1987). However Toglia (1976) reported that despite normal neurological examinations, 51% of 309 patients with whiplash injuries showed abnormal findings on vestibular testing. Torres and Shapiro, 1961 reported a high incidence of EEG abnormalities in patients with whiplash. Frequently the abnormalities increased with time and were present years later. Ommaya, Faas aned Yarnell (1968) demonstrated experimentally that whiplash injury without significant impact to the head can produce cerebral concussion, gross hemorrhages and contusions of the brain surface as a result of the rotational displacement. Ommaya and Yarnell (1969) later presented two cases with subdural hematoma after whiplash injury. Ettlin, Kischka, Reichmann, Radii, Heim, Wengen and Benson (1992) reported that despite normal CT or MRI, 10 of 18 unselected whiplash patients showed otoneurological abnormalities and overall lower performances on neuropsychological tests than matched controls.

In contrast to these numerous clinical and experimental findings, a recent, apparently exhaustive literature review and clinical study financed by the Societe d'Assurance Automobile du Quebec concluded that whiplash associated disorders are typically benign, resolve spontaneously in weeks or days and require little treatment (Spitzer et al, 1995). It is interesting to note that patients in the severe category were not included in their analysis. Ommaya's work was not cited or addressed because it did not fall within the arbitrarily determined 1980 to 1993 time period for literature search. Finally, despite numerous studies reporting cerebral involvement after whiplash injury, the word "brain" did not appear once in the article. The findings of this study of course were "warmly embraced" by the insurance company that funded the study in a brief note at the end of the article. The conclusions of this study illustrate that we have yet to bridge the gulf which arose in the last century between those who look upon patients with persisting cognitive and mental problems after mild head injury and whiplash as malingerers and those with a more disinterested view. In addition to apparent researcher bias, the conflicting findings in studies of whiplash may reflect methodological errors, unwarranted assumptions, difficulties assessing severity of whiplash, and failures to differentiate populations according to sex and type of injury (rear-end vs. frontal or side impact).

MALINGERING AND COMPENSATION NEUROSIS

The rapid rise in auto and work related accidents that directly resulted from industrialization almost immediately led to a polarization of thought regarding the complaints of patients with head injury and especially whiplash. Defense attorneys and industrial medical practitioners argued that the complaints were motivated by greed and were an attempt to profit from an "invisible" malady (Courville, 1953). In 1879 Rigler introduced the term "compensation neurosis" in reference to increases in reported disability claims following enactment of compensation laws in 1871 in Germany (Resnick, 1988). Compensation neurosis became a diagnostic category for post-traumatic symptoms which were considered to be bogus complaints of malingerers. In 1882 Erichson attributed severe symptoms in patients involved in railway accidents to "concussion of the spine". Consistent with current

concepts of whiplash injury, he noted that injuries were more serious if the patient's back was facing the rear of the train in a rear-end collision or facing the front of the train in frontal impacts (Trimble, 1981). In response to the widespread influence of Erichson's work, Page attributed symptoms after train injury to psychological factors. The debate has continued to this day over whether these problems reflect objectively verifiable organic causes or functional causes, due to disorder in actions of the organs of the mind. Hughlings Jackson however considered functional disorders to be due to minute pathological changes in the nervous system (Trimble, 1981).

Miller has been one of the more recent vocal critics of patients seeking compensation for psychological problems following industrial accidents. In 1961 he presented findings indicating that all but 2 of 50 patients he diagnosed with "accident neurosis" still complained of disabling nervous symptoms after settlement of their claims. He cited this as evidence that such symptoms can arise independently of "any physical injury of any kind". Later (1972) Miller observed that cases of accident neurosis are notable for absence of objective pathological findings and that symptoms were more common after "trivial" than severe head injuries. More recently, Mersky (1984) pointed out that most psychiatrists were skeptical of Miller's reports. Pankratz (1988) concluded that Miller's position was an over-reaction which obscured the reality of the post-traumatic syndrome. Resnick (1988) and Ruff (1993) pointed out that many studies failed to replicate Miller's Findings. Rutherford (1989) cited his own findings as failing to support Miller's claims and concluded that organic factors are responsible for producing symptoms after mild head injury. Others, including Benton (1989) and Binder (1989) have observed that post-traumatic symptoms are consistently found in head injured patients not involved in compensation. Courville (1953) pointed out that symptoms comprising the post-traumatic syndrome were described in the 16th century, much before the enactment of 19th and 20th century compensation laws. Parker's (1970, 1977) Australasian studies of "neurotic symptoms" in accident litigants suggests that emergence of psychological symptoms is multiply determined and occurs in situations where litigation is not an issue. He concluded that assignment of patients into malingering and non-malingering categories is an oversimplification of a complex problem.

CLINICAL ASSESSMENTS OF MALINGERING

Recent attempts to develop objective measures sensitive to malingering behaviors have focused on applications of forced choice and recognition memory tests. Brandt (1988) identified methodological issues which limit the extent to which the findings of such studies can be applied in clinical settings. First, there is no way of identifying which patients are malingering unless they confess or are observed in performances they claimed they were unable to do. Also, studies using normal subjects who are instructed to malinger are qualified because they lack the same motivation of real malingerers to malinger successfully. Furthermore, one cannot rule out the possibility that individuals who malinger are also brain injured. Brandt concluded that "At the present time, there are no scientifically valid and reliable clinical techniques for determining with certainty whether an individual is feigning amnesia (pp 81)."

Despite Brandt's cautious observations, Griffenstein, Baker and Gola (1994) presented findings which suggested they had validated a scale sensitive to malingered amnesia. However, the patients in this study consisted of subjects that the authors had previously

made determinations regarding presence or absence of malingering in reports to referring insurance companies and attorneys. These subjects were again assigned, "blindly" this time, to malingering and non-malingering groups and compared on a series of tests proported to be sensitive to malingering. Superficially, the findings seem to have confirmed the validity of selected tests in identification of malingering. However, the ultimate validity of the findings hinges not on the degree of agreement between malingering test scores and pre-determined status as malingerers or non-malingerers. Rather, the validity of their findings depends on the accurancy of the author's initial "blind" ratings of cases and assignment into malingering and non-malingering groups. As Brandt pointed out, there is no way of assessing how accurate these determinations were, aside from cases which were surreptitiously observed functioning normally and those that later confessed to malingering. Furthermore, without citing evidence in support of their conclusion, the authors dismissed Brandt's caution that findings indicative of malingering do not automatically rule out presence of brain damage.

Direct observation of patient behavior can often provide valuable clinical data which supersedes the importance of objective test scores in clinical assessments of head injured patients. However, many studies focus solely on test scores in attempts to determine presence or absence of malingering. Furthermore, many neuropsychologists use a technician to administer their entire test battery. They often only meet briefly, if at all with the patient. In the Jacksonian tradition, Smith (1994), Lezak (1995), Ruff, Wylie and Tennant (1993) and others have emphasized the critical importance of data which can only be obtained when the neuropsychologist directly observes the patient's behavior during testing. Those who use the "technician model" reap the benefits of testing a greater number of patients. However, this economy is achieved at the cost of direct access to critically important behaviors such as relating to the examiner, responses to success and failure, fatigability, capacity to focus and sustain attention, fluctuations in performance level, mood, affect, and other dimensions of human behavior which are not tapped by formal objective testing. Instead, practitioners of the "technician model" may rely on MMPI scores to assess emotional state, personality style and tendency to malinger. However patients with head injury typically show clinically significant elevations on MMPI scales. For this and other reasons, applications of the MMPI in assessments of head injured patients have been increasingly criticized in the literature (Lenninger and Kreutzer, 1991; Lezak, 1995; Ruff, Wylie and Tennant, 1993). An experienced neuropsychologist who has spent four to six hours with a patient obviously has more accurate and valid data upon which to draw conclusions regarding possible malingering, as well as host of other clinical issues, than does the practitioner who has only spent 10 to 15 minutes in casual conversation before turning the patient over to a technician. The experienced clinician also includes whenever possible, reports from family members, friends, co-workers and teachers in developing an appreciation of the patient's premorbid capacities, as well as the impact of the accident on those capacities.

In some cases, objective neuroradiological, neurological and neuropsychological test findings are equivocal or within normal limits. The inadequately trained, unscrupulous or biased clinician may ignore the possibility that average test scores can reflect declines from higher premorbid capacities. They may also fail to consider the limitations and potential lack of sensitivity of various assessment procedures and standardized tests to the wide range of diverse potential effects of head injury. They may wrongly conclude that there is no evidence of brain injury and attribute the patient's subjective complaints to compensation

neurosis or pre-existing personality disorder. The importance of assessing malingering behavior, especially in forensic settings or when litigation is involved has led to increasing attempts to develop approaches for detecting attempts to feign abnormal test performances. However, Ruff et al, 1993) observed that in view of the current state of the art, no clinician should be convinced that they are capable of identifying patients who malinger either based on test data or clinical impressions.

OUTCOME

Outcome of head injury reflects diverse interacting intra- and extra-cranial factors, some of which are difficult to accurately assess. Levin, Grossman, Rose and Teasdale (1979) reported "good" recoveries in adult patients with "relatively brief" durations of coma. However they pointed out that coma duration varied widely among outcome groups. Only 22% of patients with GCS scores of 8 or less returned to full time employment within a year of their injury. Levin, Ewing-Cobbs and Fletcher (1989) reviewed previous outcome studies of mild head injury in children, and found few reports of post-concussive symptoms, and lack of persisting memory deficit. However they cautioned that two year follow-up studies in children may be insufficient to demonstrate long-term sequelae. Miller and Jones (1990) reported that some degree of permanent disability is to be anticipated in patients with PTA of 1 to 7 days (severe injury). With PTA over 7 days, full return of neuropsychological function is the exception. They noted that 1/3 or more of patients with minor head injury suffer prolonged post-traumatic sequelae, and many have demonstrable neuropsychological deficits.

REHABILITATION

Following resolution of the acute symptoms of head injury, the next option for the insured patient with persisting deficits is residential or outpatient rehabilitation. Multidisciplinary rehabilitation programs may include physical therapy, speech therapy, occupational therapy, cognitive remediation, computerized exercises, vocational therapy, supportive psychotherapy, and in some cases, psychiatric treatment with psychotropic medications. Despite the best intentions of family, insurance companies and rehabilitation agencies, Ben-Yishay and Prigatano (1990) pointed to a lack of evidence that currently available methods of cognitive rehabilitation measurably improve cognitive and psychosocial function. They concluded that "There is a need for holistic approaches that exceed in scope and kind the highly circumscribed interventions which are usually subsumed under the term cognitive remediation". Jennett (1990a) has also emphasized the need for objective studies of new approaches to cognitive rehabilitation. Insurance companies have the most to gain financially by innovative approaches which could expedite return of the head injured patient to productive employment, or at least maximize the independence of the long term care patient. However, they are generally reluctant to fund promising new approaches, citing lack of objective studies or claiming the approaches are not accepted medical practice.

In contrast to the lack of demonstrable effects of standard rehabilitation procedures, studies have repeatedly confirmed the remarkable capacity of the human nervous system for spontaneous reorganization, even after drastic reductions in neuroanatomic economy (Pia,

1985). While recovery is most striking in the initial stages of recovery, Klonoff, Low and Clark (1977) noted that continuing reorganization of function in head injured children was still measurable five years post injury. Studies of hemispherectomy have shown continuing spontaneous reorganization with improvements on standardized objective measures of higher cognitive and lower sensory and motor functions over twenty years post operation (Smith and Sugar, 1975).

Since the brain's own capacity to reorganize appears to be the primary factor promoting recovery from head injury, reports of slower healing of bone fractures in smokers, delayed healing of flesh wounds under conditions of psychological stress, and other studies demonstrating the importance of "mind-body" effects may have useful implications for rehabilitation of head injury patients. Rehabilitation programs which include stress reduction through meditation practice, healthier dietary habits, positive lifestyle changes including cessation of smoking and limitation of alcohol and drug intake may accelerate recovery by reducing negative influences which attenuate the brain's reorganizational capacities while at the same time providing therapeutic interventions which directly enhance cerebral reorganization. There is ample evidence that manipulation of enviromental influences can enhance the rate and degree of cerebral reorganization (Piasetsky, 1982). Consistent with this, Ulrich (1984) reported faster recovery, fewer pain killing medications, and fewer complaints after cholecystectomy when hospitalized patients had a pleasant natural view instead of windows facing a building wall. Thus, in addition to positive influences of stress reduction techniques and lifestyle changes in promoting recovery, cerebral reorganization may also be expedited by therapeutic influences of direct care staff and enviromental and design features of the treatment facility. Continuing research *into* mind-body relationships may provide objective bases for development of more holistic rehabilitation techniques which synergistically enhance brain's inherent capacity to reorganize, compensate and recover from brain injury.

CONTRIBUTIONS OF NEUROPSYCHOLOGY IN ASSESSMENTS OF HEAD INJURY

MRI, CT scans and neurological examinations are considered central to medical assessments of the status of the brain after head injury. However patients with severe brain injury may have normal CT scans. For example Jennett (1979) reported that in a sample of patients with severe diffuse damage and coma of at least 6 hours, between 1/4 and 1/3 of the CT scans were normal, including scans of patients who subsequently died. Lezak (1995) noted that MRI is more sensitive than CT to pathological effects of head injury. However MRI studies in the acute stage do not predict outcomes as well as those obtained 5 months or more after trauma. This may reflect the pathological effects of delayed neuronal degeneration which is only apparent on later scans. Power spectral analysis of EEG have reported promising findings in diagnosis of mild head injury, but as of yet have not been widely applied in routine assessments (Thatcher, Walker, Gerson, and Geisler 1989).

Despite the limitations of CT scans and MRI studies, these techniques can localize focal brain lesions such as hematoma with far more accuracy than is possible with neuropsychological testing. However, CT and MRI cannot tell anything about what the patient can or cannot do, or how his or her life has been altered after a blow to the head. For example, I examined a 25 year old man whose CT scan revealed extreme hydrocephalus. 95 percent

of the cranial cavity was filled with cerebrospinal fluid and there was only an orange peel thickness of brain adjacent to the inner wall of the skull. Despite this drastic reduction in cerebral economy documented by CT scan, he obtained a VIQ of 140, graduated from college with an honors degree in mathematics, and works as an accountant. In contrast to the limitations of current neurodiagnostic procedures, a detailed interview and history coupled with a carefully selected battery of standardized objective tests administered by an experienced neuropsychologist can provide a multi-dimensional view of how the head injured patient's brain is functioning, including the nature and degree of current deficits, psychological impact of the injury, as well as potential for recovery.

As yet we do not have an adequate model which explains the complex processes and secrets of the brain. However, there are well-established neuropsychological principles underlying organization, disorganization and reorganization of brain function. Accumulating studies have also identified discrete factors that determine initial and later effects of brain damage. These factors include age, education, premorbid capacities, time elapsed since accident, type of test administered, nature of the underlying pathological process, momentum, or rapidity of destruction, and premorbid condition of the brain (Smith, 1979, 1983a). Neuropsychologists who have been trained to apply these principles and factors in interpretations of objective neuropsychological test findings are uniquely qualified to assess the head injured patients' deficits and estimate potential for recovery as well as the risk for emergence of delayed complications.

CLINICAL AND HEURISTIC CONSIDERATIONS

There have been numerous attempts to develop models which explain brain behavior relationships, ranging from ventricular localization, the phrenology of Gall and Spurzheim, the Anatomic Duality/ Functional Unity theory of Flourens, and Cortical localization pioneered by Broca in 1865, and extended by Wernicke in 1874. However, Jackson, von Monakow, Ferrier, Goldstein, Sherrington, Reise, Smith and others have advocated an approach which eschews model building or cerebral cartography (Berker, Berker and Smith, 1986). Their approach instead emphasizes development of principles of brain function and identification of factors determining outcome of brain damage based on careful clinical observations. For example Jackson's law of destroying lesions states that functions are more impaired as they serve in voluntary, efferent expressive capacities and less impaired as they serve in automatic, receptive afferent functions (Jackson, 1915). Careful observations of differences between temporary symptoms seen only in acute stages of brain injury vs. permanent symptoms seen in both acute and chronic stages led von Monakow to develop the concept of diaschisis. He explained the temporary symptoms as resulting from disruption of otherwise healthy tissue by transitory pathological radiating effects of damaged neural tissue. This principle was subsequently validated following development of neuroradiological techniques which demonstrated temporary attenuation of blood flow and metabolism at sites distant from focal lesions (Berker and Smith, 1988). Such principles are valid regardless of which model of the brain is in vogue. If a principle does not apply in a particular case, then another known or as yet unknown principle must be supervening. While current neurology texts continue to reproduce maps localizing specific functions to various cortical regions, others have pointed out that such schemes are inadequate to explain the infinite spectrum of potentialities and complexity of the human brain. Jackson suc-

cinctly critiqued the concept of cortical localization of function by pointing out that to localize a lesion that destroys a function and to localize a function are two different things (Berker and Smith, 1988).

Previously identified factors determining outcome of brain damage are clearly relevant to understanding the effects of head injury. The critical importance of age is apparent in studies reporting that older patients are less likely to survive and show more severe and persisting deficits than younger patients. These findings suggest that older patients also experience longer duration of PTA and obtain lower GCS scores than younger patients with comparable head injuries. In addition, reports of poorer outcomes in older than younger patients despite identical duration of PTA suggests that the older brain, by virtue of its reduced neuroanatomic economy and more limited reserve capacity, is also more vulnerable to the pathological effects of secondary degeneration and other later pathologial sequelae of head injury.

Current studies have clearly demonstrated the validity and reliability of PTA and the GCS as estimates of severity of head injury and their superiority over "meaningless" concepts of concussion and contusion. Yet our understanding of the diverse and complex interacting mechanisms of brain damnage in head injury remains incomplete. Their further elucidation may permit more accurate predictions of initial and later effects of head injury.

Premorbid condition of the brain is a major factor contributing to the wide variations in outcome after head injury. For example, patients with mild head injury may show severe persisting deficits as the result of a previous head injury or covert pre-, peri- or post-natal brain insult. By contrast those with a normal complement of reserve capacity may show better recovery even after more extensive brain trauma. The effects of diaschisis in early and perhaps later stages of recovery, have yet to be further defined. The importance of sex is apparent in studies of whiplash. The effect of momentum of the lesion, or rapidity of destruction of brain tissue has previously been demonstrated in comparisons of stroke vs. slow growing tumors. The factor of momentum directly influences outcome in head trauma as a function of the Newtonian properties of velocity and mass.

These known and other as yet unidentified factors, are the underlying interacting principles which determine the nature and extent of disorganization and reorganization of the brain after head injury. They will continue to outlast the historical parade of models of brain function which have come in and out of scientific fashion. We have yet to develop a model which clearly and accurately reflects the workings of the brain, including how sensory inputs of sight sound smell and touch interface with human awareness, or how neural mechanisms mediate the infinite spectrum of intellectual, emotional, creative and other uniquely human capacities. Some psychologists even dispute the existence of consciousness. In the absence of a definitive model of normal brain function, one should therefore not be surprised by current limitations in our capacity to conceptualize and remediate the pathological manifestations of head injury.

The accumulating scientific studies focusing on elucidating principles of human brain function are a reflection of our own ongoing search for meaning. This, in Goldstein's view, is the organism's pursuit of self-realization. At some point a definitive text documenting the mechanisms and processes which constitute human brain function may become available. However, simply being able to read such a book would not totally satisfy our curiosity, nor would it be the fulfillment of the hopes of those throughout history who have advanced the study of the brain. In Gestalt terms, knowledge of the brain (the figure) also depends on knowledge of our universe (the ground). This sobering prospect per-

haps has limited the scope of scientific pursuit and has encouraged focus on aspects of the universe that are amenable to scientific inquiry. However a satisfying understanding of the human brain remains inextricably linked to the mysteries of our origins, the phenomenon of consciousness, and the realization of our individual nature in terms of the unfolding universe.

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