The Relationship Between Anger Control Problems and Neuropsychological Deficits in Individuals Who Have Sustained a Head Injury.

Kathryn Lawson Kerr
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THE RELATIONSHIP BETWEEN ANGER CONTROL PROBLEMS AND NEUROPSYCHOLOGICAL DEFICITS IN INDIVIDUALS WHO HAVE SUSTAINED A HEAD INJURY

The Louisiana State University and Agricultural and Mechanical Col.  Ph.D.  1986

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The Relationship Between Anger Control Problems and Neuropsychological
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A Dissertation

Submitted to the Graduate Faculty of the Louisiana
State University and Agricultural
and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy
in
The Department of Psychology

by
Kathryn Lawson Kerr
B.A. Washington University, 1975
M.A. University of Minnesota, 1979
December, 1986
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The present study assessed whether neuropsychological tests could be used to discriminate between groups of CHI individuals with closed head injuries (CHI): those with anger control problems, and those without. The study also assessed whether these groups differed on tests which assess various aspects of neuropsychological functioning; intelligence, memory skills, language functioning, concept formation and set shifting skills, and psychomotor performance.

Forty two individuals with CHI were given neuropsychological tests after assignment to one of two groups: problematic anger (P) or nonproblematic anger (NP). Group assignments were made on the basis of information obtained during the course of: 1) a structured interview with the patient and family member; 2) reports from the patient's physician; 3) the patient's score on the Novaco Anger Inventory. Injury severity was estimated from information obtained from the interview and medical records.

The groups differed significantly on factors such as educational level (p < .05), injury severity (p < .05), sex ratio (p < .004), and FSIQ (p < .001). The P group (N = 22) was predominantly male, more severely injured, and of lower intelligence as compared to the NP group (N = 20). The groups did not differ on age, time since injury, handedness, or race.

These groups differed in memory skills (p < .002), and language functioning (p < .001), with the P group consistently performing at a lower level (MANOVA analysis). After education was used as a covariate (MANCOVA), the P group continued to show relative deficits on measures of memory (p < .015) and language functioning (p < .001).

Abstract

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Discriminant analyses indicated that neuropsychological tests discriminate between these two groups (overall classification rate = 79%). The P group could be discriminated from the NP group on the basis of test performance in the following areas: intelligence ($p < .0009$), memory skills ($p < .0009$), concept formation and set shifting skills ($p < .01$), and language functioning ($p < .0001$).

This study suggests that CHI individuals with anger control problems have sustained more severe injuries, are more likely to be male, and have greater neuropsychological deficits as compared to CHI individuals without anger control problems. Further research is needed to ascertain whether individuals with anger management problems are more likely to be of below average intelligence on a premorbid basis and if remediation of the pattern of neuropsychological deficits identified may decrease anger control problems.
I. Anger control problems in individuals with cerebral dysfunction.

Very few quantitative or qualitative studies have been done to assess anger control problems in individuals with cerebral dysfunction. This lack of systematic research is curious as it has been estimated that as many as 67% of individuals who have a history of head injury experience significant temper problems one year after the injury (McKinlay et al., 1981). In fact, this problem appears to get worse over time in contrast to progressive improvement in physical status (Fordyce et al., 1983). This frequently leaves the patient's family members in a dilemma, as they have watched the patient progressively recover from horrendous physical deficits only to discover that he has never been the same since the accident....his personality is just not what it used to be.

Along with anger control problems, victims of head trauma experience a wide variety of other problems. Delineating these deficits can be an enormously complex undertaking, as each individual has a different emotional, social, educational, and physical (etc.) history prior to their injury. Once the injury occurs, there is a tremendous cascade of interacting factors that affect each patient in a different way. It becomes easier to understand the affective problems these patients experience by looking at the other problems they and their families are faced with.

A. Physical problems

Over eight million individuals a year receive a head injury of some type in the United States, and of this group, approximately 1,600,000 have a serious injury (Webster and
Scott, 1983). Head injury is the most frequent type of trauma sustained in automobile accidents (53% of those injured). Of this group, at least 28% will have sustained a concussion (Kilhberg, 1982). Figures from the National Head Injury Foundation (1984) indicate that the prevalence of head injuries in the U.S. is 1,000,000–1,800,000, and that 50,000 people a year are permanently disabled as a result.

Estimates of morbidity and mortality associated with head injury vary widely. At the turn of the century, it was reported that mortality rates ranged from 50-90%. However, with the introduction of intubation and mechanical ventilation, the mortality rate decreased to 40-50%. The current mortality estimate has been reduced to 35%. Unfortunately, these figures do not take into consideration comparable populations; i.e., patients with homogeneous head injuries.

The vast majority of head injuries that occur have been characterized as minor, and up until the 1980's were not considered relevant to a proper discussion of head injury (Boll, 1982). Although these patients appear, for all practical purposes, to have made an excellent recovery, there are often subtle deficits that linger on. This has frequently been referred to as the Post Traumatic Syndrome, and is associated with headache, fatigue, dizziness, irritability, and difficulties with concentration and memory. These patients often are labelled as malingerers or neurotics, as their injury was mild and left them with no visible neurological sequelae. Few authors describe the characteristics
of a minor head injury in any kind of detail however. The Automotive Crash Injury Research Program developed a scale to define the terms minor, moderate (nondangerous), and dangerous head injury (Appendix A). They define a "minor" head injury as one which produces a concussion with no loss of consciousness.

It is now apparent that even mild concussions can produce neuronal damage which may not be evident upon gross examination. Gennarelli et al (1981), and Jane (1982) have produced diffuse brain damage in monkeys through the use of an acceleration technique. This method induces loss of consciousness for less than two minutes without a blow to the head. "Fink-Heimer stains revealed pronounced degenerative changes in axons and their terminal arborizations in the reticular and vestibular nuclei and dorsal regions of the medulla (Boll, 1982)." This, as Boll points out, raises an interesting question. How mild can a head injury be before significant damage has occurred? It has also become apparent that cumulative sub-concussive blows may produce damage equivalent to a single mild concussion (Windle et al., 1944). The severity and nature of the residual physical and neuropsychological deficits is also highly dependent upon the age of the patient. All current evidence suggests that the effects of a mild concussion in an older individual may be quite different than the deficits experienced by a young individual. With increasing age, the mortality and morbidity from head injury increase significantly, and the sequelae are generally more severe. Some clinicians have pointed out that older individuals are more at risk for problems after head injury, as they have
already experienced normal age related declines in both physical and intellectual functioning (Miller, J., 1983; Miller, E., 1979 and 1984).

Many of the individuals who sustain head trauma suffer other types of injuries and secondary sequelae that complicates their future recovery/rehabilitation (Appendix B). Associated pain problems are common, and some individuals may cope with this by abusing alcohol or other drugs, particularly if they were prone to these methods of coping prior to the accident (Rimel and Jane, 1983). Reduced mobility secondary to residual orthopedic or neurological problems may also complicate matters, and occasionally sensory problems such as impaired visual, auditory, kinesthetic or other combinations of these deficits lead to problems in recovery. Some of these individuals also have to cope with changes in their physical appearance such as scars, amputations, enucleated eyes, gait changes, etc. Residual seizure disorders of various types may occur in some individuals, particularly those who have sustained a moderate to severe head injury (Jennett, 1983), and may require pharmacological treatment. All of these physical changes make a significant impact on the patient and the people around them, and call for multiple adjustments. Physical deficits, however, are only one aspect of the total problem.

B. Site of lesions after post-traumatic head injury

Three types of injury (with various combinations of all three) may occur after head injury, dependent upon the nature of the accident (Miller, J., 1983). Damage may occur
at the point of impact (focal damage), or on the opposite side of impact (contrecoup injury). Contusions and lacerations may also occur. Bleeding or edema may produce further focal effects, and if serious enough, may produce diffuse effects as well due to increased intracranial pressure. Damage may also be produced secondary to abrupt acceleration or deceleration of the brain within the skull. When this occurs, damage often occurs to the frontal and temporal poles as they collide with the walls of the anterior and middle cranial fossae. Orbital-frontal, anterior temporal, and mesial temporal damage are more common with this type of accident, although occasionally occipital injury occurs. Rotational forces on the brain produce diffuse white matter damage due to the shearing of axons within their myelin sheath. On autopsy, brains subjected to rational injury may appear grossly normal, or there may be evidence of petechial hemorrhages in the white matter and/or ventricular dilatation (see Appendix E).

All of the injuries discussed so far (with the exception of edema) occur on an immediate basis. After the acute injury, more damage may occur (Miller, J., 1983) due to a variety of secondary insults (e.g., arterial hypotension, edema, hydrocephalus, etc.).

On the basis of clinical observations, Levin and Grossman (1978, p 413) stated that:

"Early compression of the ventricular system of oedematous brain is predictive of substantial behavioral disturbance and that persistent CT abnormalities such as ventricular enlargement without signs of
obstructive hydrocephalus are accompanied by prolonged psychiatric manifestations."

As one can imagine, it is virtually impossible to pinpoint where the damage is when discussing injury due to head trauma. It is more useful for outcome research purposes to describe the patient's functional condition at the time of admission to hospital using instruments such as the Glasgow Coma Scale (Appendix D) and the length of post-traumatic amnesia, rather than dwelling on the hypothesized location of the lesion. Unfortunately, the Glasgow Coma Scale (which is a standardized scale rating the severity of the patient's altered consciousness) is primarily used in major hospitals that follow research protocols. Post-traumatic amnesia (PTA) length is also a good measure of the severity of the injury, although accurate calculations of this time period are also done on an inconsistent basis. In addition, PTA length is best assessed while the patient is still in hospital by interviewing the patient on a daily basis.

C. Neuropsychological Deficits

The type of neuropsychological deficits experienced by each individual after head injury varies widely, as the brain damage may be focal, diffuse or mixed. It is now hypothesized that varying degrees of diffuse axonal injury occur at the time of the head trauma (Adams et al., 1982). As mentioned previously, other post traumatic complications may ensue (hypoxia, hematoma, edema, etc.) that contribute to the overall damage sustained. Deficits may be observed in various areas of performance, but are most
commonly seen in terms of declines in cognitive capabilities, memory functioning, language, concept formation/set shifting, and motor skills. For example, many authors (Roberts, 1979; Levin et al., 1979; Klove et al., 1972; and Mandelberg et al., 1975) have reported intellectual declines in patients who have sustained closed head injury (hereafter referred to as CHI), although selection criteria for these studies have varied widely and cast some doubts on general conclusions one may draw from this literature. Unfortunately, many studies have not excluded patients with a known history of substance abuse, and not all studies report when patients were assessed in relation to time since injury. However, declines in performance (from estimated premorbid intellectual level) have been reported on both an acute (Mandelberg et al., 1975) and delayed basis (Levin et al., 1979; Roberts, 1980). A number of studies also indicate that there is an association between indices of head injury severity and the presence, persistence and degree of cognitive impairment. Factors such as "duration of coma (Dye et al., 1981), level of coma (Williams et al., 1981), duration of posttraumatic amnesia (Mandelberg, 1976), and abnormal EEG/neurological status (Klove and Cleeland, 1972) have all been found to correlate positively with poorer neuropsychological recovery from head injury" (Drudge, p.259, 1984).

Cognitive deficits may persist even in individuals who have achieved good levels of recovery. Stuss et al (1985) found information processing deficits in individuals who had experienced a closed head injury and were deemed 'fully recovered' as compared to
normal control subjects (with subject-matching on age, sex, handedness, education, language, and full-scale IQ variables). CHI subjects performed significantly worse on Wechsler Memory Scale paired associated delayed (p < .0001); Wechsler memory Scale story recall delayed (p < .05); Wisconsin Card Sorting test - perseverative errors (p < .05), and a series of other measures. A discriminant function analysis significantly discriminated the two groups (classification rate = 85%).

Current evidence suggests that recovery occurs in verbal areas first with more gradual recovery in perceptual abilities over time as assessed by the WAIS (Mandelberg et al., 1975). However, further research with larger CHI patient populations is needed to substantiate this pattern. In addition, given the fact that CHI produces a variety of neuropathological changes, studies need to report in greater detail the characteristics of the populations assessed (ie, posttraumatic amnesia length, CT scan findings, neurological deficits, etc.). Patterns of deficit vary in relation to the nature of the sustained injury, and this is reflected by the wide range of WAIS-R subtest variations reported in the literature (Levin et al., 1982).

Memory deficits are also a very common sequela of CHI. Russel (1971) reported that 23% of a large sample of British soldiers (N = 892) had some degree of mnestic problem after sustaining nonpenetrating head injuries. The presence of residual memory problems was clearly related to the duration of posttraumatic amnesia; half of the patients with a
posttraumatic amnesia greater than seven days had memory problems.

Brooks (1975), also reported finding significant memory problems in head injured individuals as assessed by the Wechsler Memory Scale. Brooks found that the head injury sample assessed (N = 82) had significant problems on almost all subtests of this scale as compared to a group of patients with orthopedic problems (N = 34). Head injury patients were found to have difficulty with retention of both verbal and non-verbal information. Although memory deficits may lessen over time, some clinicians have found that impairments may persist for years (Smith, 1974) and in some instances, may never completely resolve.

Classic aphasic disturbances are rare after head injury (Geschwind, 1964), although they may occur after focal involvement of the left hemisphere (Levin, 1982). Severe trauma may impair expressive and receptive abilities for various reasons (Levin et al., 1976). The types of language deficits commonly seen however, include impoverished verbal associative fluency, word finding problems, anomia, and impaired comprehension of complex commands. Circumlocutions and verbal paraphasic errors are far more common than the presence of a true aphasic disorder, and it is thought that naming problems and word retrieval deficits may result from disproportionate injury to the temporal lobes (Groher, 1979; and Sarno, 1980). Persistent language problems appear to be closely associated with general cognitive impairment (Levin et al., 1982).
Individuals with CHI have been reported to have difficulty with tasks requiring alternation between sets (Klove et al., 1972; Rimel et al., 1981, 1982; Stuss et al., 1985) and with tasks involving concept formation (Dikmen and Reitan, 1976; Rimel et al., 1981, 1982; Stuss et al., 1985). These deficits have been noted even after minor head injury (defined as loss of consciousness less than 20 minutes: Rimel et al., 1981). It has been hypothesized that attentional deficits and memory problems contribute to the aforementioned deficits although this has not been adequately researched (Levin et al., 1982). Tests which are thought to assess these types of cognitive problems (Trail Making Test, the Categories Test and the Wisconsin Card Sort) have been shown to be sensitive to frontal and fronto-temporal dysfunction (Milner, 1964); a frequent finding in individuals with CHI. It is thought that there may be some relationship between performance on such tests and duration of coma (Klove et al., 1972) although this needs to be studied further.

Motoric impairments may occur after head injury due to CNS dysfunction or peripheral damage. The most common types of impairment seen after CNS damage include: spasticity, bradykinesia, ataxia, hemiparesis and tremors (Griffith, 1983). Any of these deficits may contribute to slowed time scores on commonly used tests of motoric functioning including finger tapping (Halstead, 1947; Rimel et al., 1981, 1982) and the Grooved Pegboard test (Lezak, 1983). Grip strength may also be reduced unilaterally or bilaterally, as assessed by hand held dynamometer readings (Lezak, 1983; Drudge,
The type of deficits observed depends upon the nature of the injury. Deficits may persist or clear over time, dependent upon the nature of the injury, and the age of the patient. Interestingly, identical injuries in young (versus older) individuals, may produce a strikingly different set of residual neuropsychological deficits due to differences in recovery of function over time and the age related plasticity of the CNS (Lezak, 1976; Rosenthal et al., 1983; Golden, 1978). Cortical/subcortical damage produced by head trauma may also lead to affective changes as a result of damage to the limbic system.

C. Personality changes after head injury

There is general agreement within the literature that personality changes may occur in many individuals who have sustained a head injury (Brooks and Aughton, 1979; Fordyce et al., 1983; Lishman, 1973; Newcombe, 1982; McKinlay et al., 1981; Ota, 1969; Prigatano, in press; Rosenthal, 1983; Lezak, 1978; Bond, 1975; and Oddy et al., 1978), and that these changes may have far more impact on the patient and their family than the physical disabilities (Lezak, 1978; Bond, 1975; and Oddy et al., 1978). How these personality changes are defined differs from one study to the next, and the populations examined are usually heterogeneous across studies. In addition, many studies vary on such important variables as mean age of the subject, the level of injury studied, and the source of the data (patient versus family) etc. The patient's friends and family members often report that their family members personality has changed in unexpectedly
negative ways. The type of psychological changes noted in these patients varies widely and are summarized by Rosenthal (1983). Rosenthal describes a diverse range of personality changes that may or may not be associated with a variety of neuropsychological deficits.

Few studies have systematically examined the types of psychological changes that occur in individuals with acquired cerebral dysfunction. A few clinicians (Brooks and McKinlay, 1983; Levin and Grossman, 1978; McKinlay et al.; Thomsen, 1974; Weinstein and Wells, 1981; Dikmen and Reitan, 1977; and Lezak, 1978) have done systematic studies with head injured patients to further delineate the type of psychological problems observed. Brooks and McKinlay have led the way in this area of research by asking close relatives of head injured patients to describe the personality changes they have observed in their injured relatives. They developed a bipolar adjective checklist to assess the patient's current as well as premorbid personality characteristics. Half to two thirds of the relatives surveyed reported significant personality changes in their relatives. Interestingly, these personality changes became more evident over time and were not entirely related to the severity of the injuries sustained at the time of the accident. The extensive changes noted by relatives included increasing dependence, irritability, cruelty, and social withdrawal (to identify a few). Patients who were initially described as unchanged by the accident, were described as being more irritable, lethargic and listless one year after the accident. The authors feel that relatives may be less tolerant of the
personality changes over time, and thus more likely to report problematic behavior; a reasonable hypothesis. They point out that patients who are viewed as a burden to their family are also more likely to receive negative personality ratings. Other changes noticed in this patient group in a previous study (McKinlay et al., 1981) included: word finding/fluency problems (44%), dysarthria (29%), receptive language problems (15%), and memory difficulties (69%). Unfortunately, the specific types of neuropsychological problems these patients had were not delineated.

Other clinicians have noted similar personality changes in brain injured patients. Lezak (p. 592, 1978) stated that such adjustment/personality problems may be conceptualized in:

"five broad and often overlapping categories: (1) an impaired capacity for social perceptiveness...(2) an impaired capacity for control and self-regulation ...(3) stimulus bound behavior...(4) specific emotional alteration ...(5) and a relative and sometimes quite complete inability to profit from experience."

The majority of research that has been done in this area has been focused on psychological changes that have occurred after moderate or severe head injury, although there is increasing evidence that these changes can occur after mild injury (Newcombe, 1982). In addition, some authors (Fordyce et al., 1983; McKinlay and Brooks, 1984) have tried to examine various hypotheses as to why the psychological changes develop. Fordyce, for example, found that patients with more chronic head trauma, tended to be
more anxious and depressed, more confused in their thinking, and more socially withdrawn than the acute patient group. These differences in emotional functioning appeared to be independent of level of neuropsychological impairment and length of coma. As pointed out earlier by McKinlay et al., (1981), patients have increased personality problems over the course of a year post-accident, and these changes may be related to how 'burdensome' the patient is perceived by the family. McKinlay and Brooks (1984) are the only authors who have attempted to find out if certain personality characteristics of the relatives are correlated with the degree of stress they experience in living with a head injured patient. Using a brief version of Eysenck's personality scale, they found that the neuroticism (rather than extroversion or psychoticism) score was related to the high stress experienced by relatives. In fact, neuroticism was highly correlated with reports of emotional/behavioral alterations in the patients, and was less correlated with the patients physical/cognitive changes. They conclude that although a relative's personality is related to the reports they give (about their injured family member), the extent of the influence of personality is not overwhelming.

Other authors have looked at the premorbid demographic characteristics of patients who have sustained head trauma in an attempt to examine Symond's hypothesis that "it is not only the kind of injury that matters but the kind of head" (Symonds, p. 108, 1937). The incidence of head injuries is highest among young single males (Jennett and
MacMillan, 1981) who come from lower social and economic groups, have a previous history of head injury (Rimel and Jane, 1983), and a higher incidence of premorbid "antisocial behavior than individuals from the same social background (Bond, 1983)." Many of the patients admitted with head injury to the emergency room are legally intoxicated at the time of admission as well (Rimel and Jane, 1983), which some authors have used as further support to argue that many of these patients have a premorbid history of risk-taking.

In reviewing the literature, there seem to be as many theories regarding why personality changes occur as there are number of affective changes observed. In addition to the theories already discussed, some authors have emphasized that there is a neurological basis for the affective changes due to limbic damage, brainstem/reticular activating system involvement, or possible effects secondary to the hemisphere involved. This will be discussed in more detail elsewhere.

Others have suggested that the psychological and physical changes resolve quickly after the patient's insurance claims have been settled, although this theory has lost some support in the last few years (McKinlay, 1981; Rimel et al., 1981). Many have mentioned that patients may be unable to adjust to their acquired neuropsychological deficits and that they may also have to deal with increasing numbers of injury related stressors. To conclude, few researchers have tried to develop a comprehensive theory/model that would
account for why affective changes occur, and fewer have tried to test their ideas.

D. Additional stressors

In addition to the problems discussed so far, it is likely that many of these patients also experience significant financial strain due to the cost of hospitalization, follow-up medical care, time lost at work, and in some cases, inability to return to their previous level of employment. One frequently cited study reported that 50% of their severely head injured patients returned to their previous level of employment four months after injury, with another 15% returning to part-time work within this time period (Oddy et al., 1978). However, this particular group of patients was quite young (80% under 25 years of age), and they were followed for only six months. Given the observations by Brooks and McKinlay that many of the psychological deficits first seem apparent at the 6-12 month post-injury period, it would be interesting to follow Oddy's patients for an additional 6 months to see if their job status was maintained.

For those patients who do not return to their previous level of employment, other stressors may begin to accumulate, such as the assumption of a job by a previously unemployed spouse. Bjorn-Hansen (1957) pointed out that marital role changes are very common in this population and can be very disruptive. Crawford (1983) substantiated this finding, noting that two out of eighteen couples within his study had separated, and another four of the remaining sixteen couples were on the verge of separation. Although
other relationships within the family may become strained (i.e., between the patient and their children), it appears that the relationship between injured children and their mothers is better than that between an injured individual and their spouse (Thomsen, 1974).

Perhaps the worst stressor of all for both the patient and the family is the lack of knowledge about the patient's future potential and problems. It is difficult, even under the best of circumstances, to predict how a particular patient will fare. Frequently, patients/families are given only vague information, and often they are unable to remember or process what they have been told (Thomsen, 1974). Thomsen (1974) was one of the few individuals to ever examine the level of information relatives were exposed to while their family member was hospitalized. Judging from his research, it is entirely possible that many relatives have little understanding of the patient's problems by the time the patient leaves the hospital. Even if patients are fortunate enough to be admitted to a rehabilitation program, it is not always clear what information their families receive concerning functional capacity after discharge. As can be imagined, this population may experience an intense number of stressors, although it is difficult to predict how it affects a specific individual and their family.

F. General methodological flaws in the existing literature pertaining to psychological changes
As Brooks and McKinlay (1984) point out, the research that has been done on head injury and affective changes is fraught with methodological problems. One needs to carefully examine how patients are selected for these types of studies and who provides the database. It has been shown that there is often a significant degree of disagreement between the patient and their relative concerning the presence of emotional/behavioral difficulties, with patients being more likely to deny problems, whereas patient/relative reports tend to be more congruent when asked about the patient's physical deficits.

Interestingly, this discrepancy was found not to be related to the patient's cognitive deficits (McKinlay and Brooks, 1984). In addition, very few studies have used a chronic illness group (without brain damage) as a control to partial out effects due to brain damage from strictly psychological reactions to illness. To this author's knowledge, there have not been any studies to examine why some patients with brain damage have anger control problems and some do not. Many authors have speculated about factors that contribute to affective changes in this population, but few have empirically examined so-called causative factors.

Fortunately, within the last few years, there have been more attempts to control for certain parameters that are known to affect study results in this area (i.e., subject age, mean length of post-traumatic amnesia, severity of injury, data information sources, etc.). Perhaps part of the reason more research has not been done in the area of anger
control problems following head injury is because studies addressing anger contol
management problems have been few and the ones that exist are methodologically
problematic.

II. The assessment of anger

Little has been written about the assessment of anger control problems that is based
on a strong theoretical foundation and supported by an empirical data base. As many
authors have suggested (Biaggio, 1980; Novaco, 1975; Rothenberg, 1971) this is
undoubtedly due to the lack of a concrete definition of anger. Many have assumed that
anger and aggression are one and the same, resulting in a great deal of literature on
aggression (particularly within the social psychology literature) and little on anger. For
the purpose of this discussion, anger will be defined as:

An emotional response to perceived provocation (generated either from
external or internal sources) that is mediated by the autonomic and central nervous
system. Furthermore, anger is associated with certain cognitions that are linked to
the individual's past experiential/learning history. These cognitions may increase
or decrease the individual's level of anger leading to either positive consequences
(conflict resolution), or negative consequences (aggression, violence, somatic
problems, interpersonal problems, etc.). Aggression and violence are inately
different from anger, as they imply physical destruction of some type, whereas
anger, although correlated with aggression/violence, does not necessarily lead to
physical harm.

Anger assessment has been based on numerous conceptual frameworks. These
frameworks generally fall within three categories: (1) assessment of faulty cognitions or
personality variables; (2) assessment of assertion skill deficits and, (3) assessment of neurological/physiologically based parameters. Each framework addresses a different facet of anger expression, and as a result, different assessment approaches have proliferated. Various sociological theories also exist, but will not be discussed in detail as they apply primarily to the study of aggression.

A. Assessment of faulty cognitions or personality variables

Both Ellis (Rational Emotive Therapy) and Meichenbaum (Self Instructional Training) have focused on assessment of cognitive-behavioral aspects of faulty anger control. The therapist's job, with either approach, consists of identification of maladaptive cognitions that lead to anger and its expression. Both of these approaches preach a philosophy of assessment with little in the way of concrete (standardized) assessment techniques offered to quantify anger control problems. As a result, there has been some empirical support for the use of these approaches (Lehman-Olson, 1974; Novaco, 1977), but unfortunately there are overwhelming methodological problems in the existing assessment literature. These problems have primarily revolved around unstandardized assessment approaches that have not been cross validated, small subject samples, and flawed data analysis techniques.

Novaco has embellished on these cognitive assessment approaches, and is one of the few individuals to develop a cognitive model that is supported by an empirical data base in the
treatment literature. His stress inoculation approach requires assessment on multiple levels including: (1) an assessment inventory (the Novaco Anger Inventory), (2) self monitoring of anger episodes, and (3) clinical (unstandardized and/or unspecified) assessment of assertiveness and problem solving skills. His assessment inventory is standardized and has been shown to have clinical utility.

Buss and Durkee (1957) also developed an inventory used to assess anger called the Buss-Durkee Hostility Guilt Inventory. This 75-item inventory assesses various facets of hostility, including assault, indirect hostility, irritability, negativism, resentment, suspicion, verbal expression of hostility, and guilt. This instrument has been shown to have adequate psychometric properties and a reasonable degree of clinical utility. Unfortunately, it has not been used with neurological populations, which is unfortunate because it is easy to administer and complete.

The Novaco Anger Inventory and the Buss-Durkee are the only two standardized self report inventories commonly used to specifically assess anger. Unfortunately, both of these instruments rely on the patient’s self report of problematic behavior. As a result, individuals with problematic behavior or those who are unconcerned about their expression of anger may look asymptomatic on these instruments. This could be a potential problem when assessing these patients and can only be avoided by interviewing people who know the patient well.
Three authors have examined various personality characteristics thought to be associated with anger control problems. Biaggio found that angry subjects (as assessed by the Novaco Anger Inventory) were less dependable, mature, conforming, and less capable of forming a good impression (as assessed by the California Psychological Inventory (CPI)). Megargee, Cook, and Mendelsohn (1967) took this one step further with their development of the Over Controlled Hostility Scale (taken from the Minnesota Multiphasic Personality Inventory (MMPI)). They found that individuals who guard against the expression of anger and generally appear passive, have the potential for acting out (sometimes rather violently) when faced with extreme provocation. Little has been done to pursue Biaggio's line of investigation, and unfortunately many patients with closed head injuries have difficulty completing a valid MMPI or CPI. This may explain why there have not been any studies, to this author's knowledge, utilizing the MMPI or the CPI to assess anger control problems after head injury. In addition, there has been little research assessing the personality characteristics of "angry" people who do not have neurological problems, making it difficult to do contrast studies between so called normal individuals and those with CNS dysfunction.

B. Assessment of assertion skill deficits

Those who lean toward operant rather than strictly cognitively based theories of
behavior have pushed for the assessment of assertion skill deficits and excesses as a primary factor in the maintenance of anger control problems (Fredericksen et al., 1977; Rahaim et al., 1980; Matson et al., 1978; Foy et al., 1975; Rice et al., 1980; Eisler et al., 1974; Rimm et al., 1974; and Turner et al., 1978). The predominant themes throughout this body of literature suggest that patients inappropriately express anger because: (1) they never developed the skills to handle confrontational situations (Rahaim et al., 1980), or (2) expressing anger inappropriately can be reinforcing for patients because it gets them what they want in the short run, and they do not anticipate the long term aversive consequences of such behavior (Fredericksen et al., 1977). Assessment approaches have used single-case design studies based primarily on videotaped role play assessments that are generally unstandardized and vary significantly in format from one study to the next. Dependent measures have consisted of (1) the appropriateness of the patient's response, and (2) aspects of nonverbal behavior (eye contact, gestures, facial expressions, etc.). Deficient or excessive behaviors are often defined on the basis of the clinician's opinion instead of a normative group data base. This type of assessment approach and research methodology (single case design) does have its advantages, as patients generally receive a great deal of attention and are given very specific feedback designed to increase appropriate aspects of performance and decrease inappropriate behavior. This type of tailor-made assessment also provides a data base and future
performance can be reassessed compared to this base.

Turner, Hersen and Bellack (1978) used this type of assessment approach to treat a 19-year-old individual with organic brain syndrome and mild mental retardation. They found that the patient improved significantly using this approach. Unfortunately, little information was provided on this patient's neuropsychological deficits and strengths. It is likely that many patients with CHI may not be appropriate subjects for videotaped social skills assessment/treatment due to perceptual problems and mnestic deficits. Neuropsychological assessment prior to using such techniques would provide this type of information and may predict success in such programs. This issue has not been addressed, however.

C. Assessment of physiological/neurological variables

The past few years have seen an increase in research designed to assess neurological functioning in specific clinical populations. The majority of these studies have involved individuals with episodic dyscontrol and/or antisocial personality disorder, and have pointed out a high incidence of neurological abnormalities. The primary abnormalities revealed suggest a high incidence of minimal brain dysfunction (41%), developmental or acquired deficits (94%), and complex partial seizures (30%) (Elliot, 1982). Others have confirmed these findings in divergent populations known to have episodic dyscontrol (Mark and Ervin, 1970; Thompson, 1953; Monroe, 1970; Bach-Y-Rita, et al., 1971;
and Andrulonis, 1980). Incidence figures reported in these studies need to be interpreted with caution, however, as referral criteria may have inflated correlations found between neurological abnormalities and occurrence of episodic dyscontrol.

Treatment efforts have attempted to target patients with dyscontrol problems believed to be due to neurological/physiological etiologies. Intervention in these cases is often geared towards surgical or pharmacological methods. In individuals with episodic dyscontrol/rage attacks, pharmacological treatment is often tried using anticonvulsants (phenytoin, carbamezapine, etc.), stimulants (amphetamines, methylphenidate, etc.), beta blockers (propanolol), lithium, and natural progesterone (Elliott, 1982), depending upon the suspected underlying physical etiology. As a last resort, surgical intervention has been tried to treat patients with extremely severe anger control problems. This form of treatment has had mixed success (Heath, 1980).

III. The neuroanatomical mediation of emotion (anger)

A. Overview

It is beyond the scope of this manuscript to try and provide a detailed description of the current state of knowledge pertaining to the neuropsychology of human emotions. However, it is appropriate at this point to discuss in general terms the neuroanatomical basis of emotion that is relevant to the discussion of anger control after head injury.

Papez came to the conclusion that the limbic structures (which in turn act on the hypothalamus) are involved in the production of emotional states. His conclusions were
based on the finding that patients suffering from rabies had significant emotional and behavioral problems during the course of the disease (anxiety, fear, explosive rage, etc.) and that at autopsy, there was significant hypothalamic damage. It is currently believed that Papez's model is at best an incomplete one, and that the interaction between the neocortex (particularly the orbital frontal cortex, amygdala, and portions of the anterior temporal cortex) and the limbic system/hypothalamus, may be the major anatomical determinant of emotion. The importance of the neocortical mediation of emotion was made clearer with the advent of psychosurgery during the 1940's and 1950's, although the actual role of the neocortex is still somewhat unclear. It has been hypothesized that the positive behavioral changes brought about by psychosurgery were due to interruption of the pathways between the frontal cortex and the limbic system (Livingston, 1969). After such surgery, many patients seem unconcerned about problems with chronic pain, obsessive thoughts, delusions, or daily activities/events that they were formerly concerned about (Valenstein, 1973). The areas thought to be most effective in bringing about these affective changes were the medial and orbital frontal cortex (Livingston). Cases have also been reported however, of individuals who have sustained trauma to these areas or had neoplastic disease, who had inexplicable fits of anger (Bailey, 1948).

In the last decade, there has been a distinct increase in the literature examining hemispheric differences in the mediation of emotion. It is well known that the two hemispheres have different neuropsychological capabilities in terms of their ability to integrate verbal versus nonverbal information, so it would not be unexpected to find that
hemispheric specialization is also involved in the perception, interpretation and expression of emotion. Studies based on normal humans (i.e. those without CNS dysfunction) tend to support this idea. Ley and Bryden (1979) in a review of research based on dichotic listening and visual half-field paradigms, concluded that the right hemisphere is significantly more adept at the accurate perception of emotional faces and oral intonations. Heilman et al., (1975) have found evidence that patients with right hemisphere disease are significantly impaired on tasks requiring comprehension of emotional faces and understanding emotional prosody. It is thought that they may also have difficulty with generating emotional facial expressions or affectively laden prosody (aprosodia). This lends credence to the longstanding clinical observation that patients with right hemisphere lesions appear to be emotionally bland (Heilman et al., 1983).

There is evidence that the left hemisphere is more adept in the comprehension of the content of speech, rather than the emotional tone (see Appendix C). This is not surprising considering the left hemisphere's specialization for language functions (Lezak, 1976).

Kolb and Whishaw (1980) have nicely summarized the findings of various researchers who have studied the perception of emotion (Appendix C). They have concluded that the right hemisphere may process nonverbal aspects of behavior (facial expressions and voice tone) rather than what is said, whereas the left hemisphere processes verbal content, rather than tone of auditory material (Kolb and Whishaw, 1980).
What about the production of emotional behavior however? Obviously this requires various motor responses (i.e. smiling, gestures, etc.) that are mediated by the CNS and various muscles. It also requires other abilities (besides the accurate perception and interpretation of emotional stimuli) that are not well defined. Several studies have examined production of affective behavior; e.g., Tucker et al., 1977, for example, found that right hemisphere lesions impair mimicry of emotional states. Ross (1981) provides supportive evidence for his theory that the right hemisphere mediates the expressive and receptive components of prosodic affect. He found that individuals with right hemisphere lesions had difficulty interpreting and expressing the affective component of speech and gestures. Other authors have found that left hemisphere impaired patients are more likely to have catastrophic reactions to stress, whereas right hemisphere impaired patients show indifference (Goldstein, 1939; Gainotti, 1972; and Hecaen et al., 1951). These studies suggest that there is also lateralization of functions involved in the production of emotional behavior. One must keep in mind when reviewing this literature base that typical head injuries produce both focal and diffuse damage, and many of the previously mentioned studies were based on either stroke or tumor cases. This has significant implications when trying to apply this literature to head injury patients; they generally do not have "clean" focal lesions. Theoretically then, these patients may present
with mixed symptoms of emotional misperception or dysfunctional expression of affective states.

B. Brain damage as a factor in faulty anger control

To state that there is a direct etiological relationship between anger control problems and brain damage would be somewhat misleading as there are many other intervening variables involved. However, evidence from the animal literature indicates that destructive lesions of the septum or stimulation of the amygdala can produce aggressive and sometime vicious behavior in fairly placid animals. Studies with humans have noted a relationship between focal temporal lobe EEG abnormalities and the disinhibition of aggression (Lishman, 1968) although this continues to elicit intense debate. Interictal aggressive behavior due to a temporal lobe focus could be considered a legal defense under the concept of diminished responsibility (Beresford, 1980) although there is little empirical evidence to support this. Few if any epileptics engage in purposeful aggressive behavior that would be attributable to cerebral dysfunction. Kretschmer (1949) points out that hypothalamic and basilar branch injuries have been noted to be associated with a variety of behavioral disturbances, including marked irritability. Irritability has also been associated with frontal lobe damage on occasion.

Interestingly, aggressive and violent behavior has been reported in a number of individuals who have recovered from meningitis or encephalitis and in a few cases,
treatment with extremely large doses of a beta blocker (propanalol) has significantly reduced these types of behavior problems (Yudofsky et al., 1981; Ratey et al., 1983; and Elliott, 1977). Pincus (1980) recently suggested that anticonvulsants may have some effect on the reduction of violent behavior in brain damaged individuals, but little has been done to follow up this suggestion. Pharmacological treatment of anger, when tried, has often been based on the maybe-it-will-work, maybe-it-won’t work principle, with little success in many instances.

To conclude, anger control problems are a significant manifestation in individuals with brain damage. As Rosenthal (1983) stated, behavioral alterations may be due to diffuse cerebral dysfunction, the patient's premorbid emotional characteristics, or the environment's response to the individual.

C. Neuropsychological assessment of violent individuals

To date, this author is not aware of any studies that have examined the relationship between poor anger control and performance on neuropsychological tests. However, there have been less than a dozen studies in the literature using neuropsychological tests to discriminate between violent and nonviolent groups of individuals. In general, these studies have been done on psychiatric populations or prison inmates; not neurologically impaired populations. Of the two studies that used 'organic' populations (Bryant et al., 1984 and Krynicki, 1978), one classified subjects as brain damaged purely on the basis of their neuropsychological test performance (Bryant et al., 1984) and the other did not specify how the diagnosis was made.
All of the studies reviewed (Bryant et al., 1984; Gudjonsson and Roberts, 1981; Krynicki, 1978; Lea, 1977; West, 1981; Blakenship, 1980; Spellacy, 1977 and 1978) claimed to have found significant neuropsychological test performance differences between violent and nonviolent subjects. However, the vast majority of these studies violate a variety of principles. For example, a quarter of the studies (Spellacy, 1977 and 1978) did not control for IQ differences between groups. It is presumed that this would increase the probability of groups looking significantly different on neuropsychological tests as performance on these tests is influenced by intellectual ability. This basic violation would not have been as serious if Spellacy had not initially hypothesized that violent individuals are not as bright, or if he had used IQ as a covariate when analyzing other variables (neuropsychological tests) used to predict group membership (violent versus nonviolent). Education can be used as a covariate in such cases when it is unclear whether IQ differences are due to premorbid intellectual differences or the sequelae of the injury.

In addition, many of these studies violate basic statistical principles of analysis. Four of the studies reviewed used stepwise discriminant analyses (West, 1981; Blakenship, 1980; and Spellacy, 1977 and 1978), with a variable to subject ratio ranging from 1:1 to 1:3. In one case, a series of univariate ANOVA's were run without any particular rationale for doing so, when a MANOVA would have been more appropriate (Spellacy, 1977). In other cases, multiple t tests were run on data rather than using a more appropriate method that would decrease the Type I error rate.
At a more basic level, some of the studies do not mention how they have screened these subjects for neurological problems (Spellacy, 1977 and 1978; Gudjonsson et al., 1981; West, 1981; Lea, 1977) or other medical/psychiatric problems.

Taking the above caveats into consideration, all of these studies seem to support the hypothesis that habitually violent individuals are more likely to have greater deficits on neuropsychological tests when compared to nonviolent individuals. This hypothesis fits nicely with Williams (1969) research indicating greater CNS dysfunction in individuals who are habitually aggressive. The patterns of neuropsychological deficits found have varied from study to study, which may simply be due to the sample sizes involved (range: \( N = 21-110 \); modal \( N = 40 \); number of studies surveyed = 8).

Given the methodological problems of the current available literature, it would seem premature to conclude anything about the role of neuropsychological deficits as a contributing factor to anger control problems in brain damaged individuals. In fact, there have not been any group studies that have tried to discriminate between subjects with poor anger control and nonproblematic (anger control) individuals on the basis of their performance on neuropsychological tests. If individuals who have anger control problems show more deficits on neuropsychological tests, one might suspect that their cognitive impairments contribute to their affective problems in some way. It may also be that cognitive deficits (if any), are simply associated with these affective problems and are not necessarily a causative factor. In any case, if one can discriminate between these two
subject groups on the basis of their neuropsychological test performance, then there could be important implications for future research and possibly for the treatment of these patients and their families.

D. The present study

The current study is designed to evaluate whether one can discriminate between two groups of individuals based on their performance on neuropsychological tests; those with anger control problems and those who do not have this type of dyscontrol. As discussed earlier, individuals with head injuries are known to have specific types of neuropsychological deficits. These deficits are most frequently seen in the areas of altered intellectual capabilities, difficulty with set shifting/concept formation, memory, language, and motor skills. It is not known, however, whether individuals who have anger control problems are more impaired in these areas than those who do not have this type of dyscontrol. To be specific, the following questions are of particular interest:

1. Intelligence
   A. Can one predict group membership simply on the basis of a significant IQ difference between the two groups?
   B. In addition, can one predict group membership as a function of disparate verbal-perceptual capabilities (i.e. the absolute value of the difference between Verbal IQ and Performance IQ)?

2. Concept/Set formation
   A. Can the two groups be discriminated between simply on the basis of their differing performance on tasks thought to assess concept formation and set shifting abilities?

3. Memory
   A. Can one discriminate between the two groups because they perform in
significantly different ways on memory tests?

4. Language
   A. Are word finding/language problems predictive of anger control problems?

5. Psychomotor performance
   A. Can performance on motor skills tests be used to predict group membership?

This study will address these questions and will attempt to support the author's hypotheses that individuals with anger control problems will have more neuropsychological deficits.
Method

A. Subjects

A total of forty two subjects who sustained a closed head injury were used for this study (22 subjects with anger control problems, and 20 subjects without this problem). The nature of the injury was verified after reviewing the patient's medical record. Subjects were identified and referred to the study through one of three routes: 1) they were referred by their local physician or other allied health care professional; 2) their name was drawn from a computerized list of all individuals who had sustained a head injury and been treated at the University of Mississippi Medical Center, and permission was obtained (from the patient's physician or the department head involved) to contact the patient; or 3) they were solicited from the outpatient clinic of the Mississippi Methodist Rehabilitation Center. Subjects selected for the study did not have a premorbid history of treatment for substance abuse, psychosis or other significant (uncontrolled) medical problems that cause CNS dysfunction. In addition, subjects selected for the study had not been diagnosed as mentally retarded prior to the development of their injury.

Both groups of subjects were between the ages of 18 and 65 and all were at least six months post-injury. This post injury time frame was selected as subjects have typically stabilized in terms of their medical disabilities. Also, if anger control problems are going to appear, they generally seem to do so by this point in time (McKinlay and Brooks,
1983).

All subjects agreed to let a family member be interviewed in order to verify information given by the patient. (see group assignment criteria).

**Group selection criteria**

**Problematic Anger Control group (P)**

Patients were assigned to this group if they met the two of the three selection criteria listed below:

(A) if the patient or their family member reported that a problem exists. (As assessed by information obtained from a structured interview administered to the patient and their relative).

(B) if the physician (health care professional) reported that in their judgement an anger control problem exists.

(C) if the patient had a Novaco Anger Inventory score one standard deviation above the normative mean established (ie scores greater than 284) by Novaco (1977).

**Nonproblematic Group (NP)**

Patients were assigned to the nonproblematic group if they did not meet the selection criteria for the problematic group.
Instrumentation

Various tests were used to assess aspects of performance in the areas previously outlined. Norms for these tests are listed in Appendix I. These tests are described (by area) as follows:

**Intellectual performance**

The Wechsler Adult Intelligence Scale –R (WAIS-R)

This test was used to assess general intelligence and to provide specific information about inter and intra-subject variability on subtests designed to measure verbal and perceptual performance. All subjects were given a pro-rated WAIS-R, consisting of the following subtests: vocabulary, similarities, comprehension, digit span, block design, picture arrangement and digit symbol. These subtests have been found to be sensitive to the effects of head injury, and have been frequently used in studies that assess recovery of function over time (Mandelberg and Brooks, 1975; Drudge et al., 1984; Mandelberg, 1975; Prigatano et al., 1984; and Long et al., 1983). The rationale for using a pro-rated WAIS-R is based on administrative time considerations. CHI subjects frequently have attentional problems, and an abbreviated WAIS-R closely approximates test results obtained with a full battery (Wechsler, 1981). The WAIS-R has been shown to have adequate reliability and validity in the assessment of intellectual performance (Wechsler; 1981).

**Concept Formation – Set Generation**
(A) **Wisconsin Card Sort Test**

The Wisconsin Card Sort Test (Berg and Milner, 1964) was designed to assess the ability to form abstract conceptual sets and shift between them based on a series of logical deductions. The patient is asked to sort 128 cards (one at a time) by matching them to one of four stimulus cards. Cards are sorted based on three target principles; the color of the target, the shape, or the number of stimuli that form the target. Once the patient has correctly guessed the sorting principle (which is determined by the examiner) by placing ten cards correctly, the examiner changes the sorting principle without telling the patient, and they must once again guess the principle involved (color, number or form). The test is discontinued when the patient makes six correct category runs or uses more than 64 response cards to complete a single category of the test, without meeting the criterion of ten consecutive correct responses. Patients with frontal lobe dysfunction are more likely to make perseverative errors and show an inability to form conceptual sets (Milner, 1964). Stuss et al. (1985) also found that individuals with CHI tended to make more perseverative errors than a matched control group, although there was a great deal of variability in performance among the CHI group.

(B) **The Trail Making Test**

The Trail Making Test was originally developed by the Army (1944) and is currently used to assess visuomotor tracking skill and the ability to shift between mental sets. The test consists of two "Trails", part A and B, that the patients must complete by either 1)
connecting numbers in sequence, or 2) alternating between numbers and letters in sequence. Time scores are generally used for analysis, although error scores are often used to examine qualitative aspects of performance on this test. Patients with cerebral dysfunction tend to do poorly on this test (Spreen and Benton, 1965). A number of authors (Drudge et al., 1984 and Stuss et al., 1985) have found that this test reveals deficits in individuals with CHI, and they have hypothesized that faulty performance is indicative of a divided attention deficit.

Memory

(A) Wechsler Memory Scale

The Wechsler Memory Scale, (Wechsler, 1945) is composed of seven subtests used to assess various aspects of orientation, verbal memory and non-verbal memory. It is one of the most widely used instruments to assess mnestic disorders and has had some additional nuances added to it to assess delayed recall (Russel, 1975). Russel asks the patient to recall material from the Logical Memory subtest and the Figural Memory Subtest after a half hour delay. Normative data are available for both the Wechsler Memory Scale (Wechsler, 1945; and Hulicka, 1966) and Russel's adapted form (Russel, 1975).

Brooks (1975), has found that the Wechsler Memory Scale is a clinically useful instrument with CHI patients. This population was noted to have impaired performance on specific subtests, including: orientation, logical memory, reversal of digits, paired associate learning, and visual reproduction. Smith (1974) and Stuss et al. (1985) also
found that individuals with CHI did not do well on this test.

(B) Rey Osterrieth Complex Figure Test

Rey (1941) developed this test to assess perceptual organization and visual memory. The subject is asked to copy a complex design. At intervals, they are given different colored pencils in order to assess their approach to the task over time. Upon completion, the materials are removed and the subject is given a blank piece of paper and asked to draw the design from memory (after a three minute delay). Both drawings (the copy and the recalled design) are then quantitatively scored. Normative data is available for both drawing conditions. Osterrieth (1944) and Bennett-Levy (1984) found that this test was particularly sensitive to individuals who had sustained traumatic head injuries (Osterreith, 1944).

Language

As mentioned earlier, individuals with traumatic head injury frequently do not present with typical aphasic disorders, but often present with word finding problems. The following tests are thought to be sensitive to word finding difficulties, and non-specific language problems.

(A) Thurstone's Word Fluency Test

This test was developed as one of Thurstone's Primary Mental Abilities Tests (1938;
and has been used to assess verbal fluency. The patient is asked to write as many 
words as they can (people’s names and contractions excluded) that begin with the letter S 
and to do so within five minutes. They are then asked to write as many words as they can 
in four minutes that start with the letter C. Patients with frontal lobe dysfunction, 
particularly left frontal damage, generate significantly fewer words within nine minutes 
than those without damage (Milner, 1964; 1967). Tests of verbal fluency have been used 
to successfully identify individuals with focal or diffuse cerebral dysfunction (Lezak, 
1983). This test is of particular interest as it requires the generation of classes of 
verbal information as well as intact writing/spelling skills. Individuals with CHI often 
complain of an inability to express themselves as articulately as they once did, although 
few authors have systematically examined this subjective complaint (Lezak, 1983; and 
Rosenthal et al. (Eds), 1983).

(B) The Boston Naming Test

The Boston Naming Test (Kaplan et al., 1983) is a sixty item set of pictures that 
must be named by the subject. The items range in complexity. It is one of the few tests 
available specifically designed to identify word finding difficulties; a common problem in 
individuals who have sustained a head injury (Levin et al., 1982).

(C) The Halstead-Weinform Aphasia Screening Test

This is a brief 32 item exam used to screen for quantitative as well as qualitative 
language deficits. This test is primarily given for qualitative reasons by experienced
examiners as it does not provide a way to analyze quantitative scores to pinpoint specific aphasic deficits. It is one of the few, widely administered, brief tests for the assessment of language disorders however, and it taps a wide variety of disorders seen in CHI individuals (apraxia, agnosia, anomia, and dysarthria (Lezak, 1983)). Many CHI individuals do not have a specific type of aphasia, but have a mosaic of nonspecific language problems (Rosenthal et al (Eds), 1983).

**Psychomotor Performance**

Strength, manual dexterity and motor speed are sometimes impaired after closed head injury and measures of these facets of motoric ability can provide information regarding localization of dysfunctional areas. Three easily administered tests will be used to measure these abilities.

(A) **Lafayette Grooved Pegboard**

The Lafayette Grooved Pegboard is designed to provide a measure of speed and manual dexterity (Trites, 1977) with either hand. The subject is asked to place 25 keys (pegs) into a pegboard using their dominant hand (trial 1) and then their nondominant hand (trial 2) as quickly as they can. Both trials are timed and the number of pegs dropped are also counted. This test requires fine motor skills which may deteriorate for a variety of reasons after CHI (i.e. due to apraxic deficits, limb ataxia, or other forms of fine/gross motor incoordination (Nelson, 1983)).

(B) **The Finger Tapping Test**
The subject is asked to use their index finger to tap a key attached to a device that measures the number of cumulative taps. Each hand must complete five (ten second) trials, with a short rest period between each trial. Dominant hand performance is assessed first. Additional trials are given if the patient demonstrates inconsistent performance (all trials must be within five taps of all other trials for each hand). This test has been found to be an excellent measure of fine motor control (Horton et al., 1984). CHI individuals have been found to have impaired scores as compared to noninjured control subjects (Drudge et al, 1984).

(Hand Dynamometer)

This test provides a measure of grip strength for each hand, and is manufactured by the Lafayette Instrument company (1984). The subject squeezes a spring loaded instrument that registers kilograms of pressure. Three trials are given for each hand. Scores are examined for differences in left-right grip strength. Limited normative data is available for this measure (Lafayette Instrument Co. 1984). Drudge et al., (1984) found that CHI subjects demonstrated significant bilateral grip strength impairments as compared to control subjects.

Procedure

Potential subjects for the study were identified as previously mentioned. They were
then either contacted by phone, or in person (if they attended a clinic appointment), and asked if they would like to participate in the study. If they were agreeable, they were given an appointment time, and asked to report for an all day session accompanied by a family member (or an individual who knew them well before and after the accident). All subjects signed an informed consent form prior to examination (see Appendix G). The patient was then given a structured interview. Upon completion of the interview, they were asked to complete the Novaco Anger Inventory while their relative was given the same structured interview. Subjects were then assigned to either the high anger group or the nonproblematic group (based on the criteria previously listed for group assignment). If they could not be clearly assigned to one group or the other, they were thanked and excused from further participation.

Qualified subjects were given a battery of neuropsychological tests (in the same order for all subjects) as follows:

1. Wechsler Memory Scale - form 1
2. Wechsler Adult Intelligence Scale - R (pro-rated)
3. Wechsler Memory Scale - thirty minute delayed recall of the logical memory, visual reproduction and paired associate sections.
4. Trail Making Test - Path A and B
5. The Dynamometer test
6. The Finger Tapping test
7. The Lafayette Grooved Pegboard test

Lunch Break

8. The Rey Osterreith Complex Figure Test (copy and immediate recall).
9. The Wisconsin Card Sort test
10. The Boston Naming test
11. The Aphasia Screening test
12. Thurstone's Word Fluency test

At the conclusion of testing, subjects and their relatives were thanked for their participation. They were then debriefed, and their questions (if any) were answered. In the majority of cases, patients reviewed their test results at a later date after the test findings (and report) had been reviewed by a licensed psychologist. A research report was then forwarded to the referring physician, or if the patient wanted a copy sent to a different individual, they were asked to sign a release before the information was forwarded.
Results

An initial multivariate analysis of variance (MANOVA) was conducted to determine if significant differences existed between the two groups in terms of age, years of education, number of months since injury, and full scale IQ. The MANOVA indicated that the two groups were significantly different \( F(4,37) = 3.53, p < .015, \) canonical correlation = .53. Subsequent one-way analyses of variance revealed that Full Scale IQ was significantly higher in the nonproblematic group (NP) when compared to the problematic group (P) \( F(1,40) = 11.65, p < .001 \) and educational levels were significantly higher in the NP group than the P group \( F(1,40) = 4.23, p < .05 \). Although differences existed in IQ and educational levels, groups were of similar age, and were tested at about the same time interval post injury. There were also significant sex ratio differences between these two groups \( \chi^2 \) with Yates correction \( (1, N=42) = 8.107, p < .004 \). Although there was an even male to female ratio in the NP group, the P group was predominately male. Means and standard deviations of the basic demographic data are provided in Table 1. It should be noted that both groups were impaired on a variety of neuropsychological tests as compared to the normative data bases used for these instruments (see Appendix I).

Education was used as a covariate in subsequent analyses. Full Scale IQ was not used as a covariate during subsequent analyses because of injury differences between the groups; the P group having more severe CHI injuries. Although it is possible that these groups may differ in performance on various neuropsychological instruments due to significant premorbid IQ differences, there is a great deal of supporting evidence in the literature indicating that individuals with more severe injuries will have lower IQ scores (Dye et
Table 1. Basic Demographics: Means and Standard Deviations

<table>
<thead>
<tr>
<th></th>
<th>No Problem group (N = 20)</th>
<th>Problem group (N = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Age (yrs.)</td>
<td>34.38</td>
<td>12.25</td>
</tr>
<tr>
<td>Education (yrs.)</td>
<td>14.70</td>
<td>2.62</td>
</tr>
<tr>
<td>Full Scale IQ</td>
<td>103.45</td>
<td>21.23</td>
</tr>
<tr>
<td>Months Since Injury</td>
<td>31.10</td>
<td>38.77</td>
</tr>
</tbody>
</table>

Other Characteristics

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex*</td>
<td>10 male - 10 female</td>
<td>20 male - 2 female</td>
</tr>
<tr>
<td>Handedness</td>
<td>20 right</td>
<td>21 right - 1 left</td>
</tr>
<tr>
<td>Race</td>
<td>16 white - 4 black</td>
<td>15 white - 6 black</td>
</tr>
</tbody>
</table>

Causes of Injury

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Motor Vehicle Accident</td>
<td>13</td>
<td>21</td>
</tr>
<tr>
<td>2. Fall</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>3. Sports Related</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>4. Assault</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>5. &quot;Unusual&quot;</td>
<td>0</td>
<td>7</td>
</tr>
</tbody>
</table>

* Chi-Square with Yates correction (1, N=42) = 8.107, p < .004
Therefore, use of Full Scale IQ as a covariate may be inappropriate as it would overlook injury differences that exist between these groups.

**Novaco Anger Inventory Scores**

There was no significant difference between the two groups on this instrument (NP group mean = 249, \(SD = 44.9\), and P group mean = 242, \(SD = 48.6\); student t test, \(p < .44\)). These mean scores are similar to the normative scores reported by Novaco (mean = 241, \(SD = 42.185\)). Subjects were asked to provide their own "behavioral anchors" for the likert scale used to rate how angry each item would make them. It became apparent that patients used a wide variety of anchors. For example, an item that would be rated as a 5 (very angry) by two patients, may be rated quite differently when the patient described what a "5 was for them"; one patient may report that he would be angry enough to assault someone, whereas another patient would report that they would just walk away when they were at a 5.

**Severity of Injury**

There were quantitative and qualitative indications that the groups differed in terms of severity of injury. Individuals in the problematic group had eight times as many multiple head injuries (P group, \(N = 8\); NP group, \(N = 1\)); indicating a significant difference between these two groups (Chi Square test with Yates correction: \(1, N=42\) = 4.39, \(p < \)).
Each subject was also rated using the Glasgow Outcome Categories (see Appendix F),
and assigned to one of the following four groups: good outcome, moderate disability, severe
disability, or persistent vegetative state. There was a tendency for individuals in the
problematic group to be more impaired as judged by this index. A comparison between the
two groups by outcome category revealed the following findings: good outcome (P group, N
= 4; NP group, N=13); moderate disability (P group, N = 11; NP group, N = 5); severe
disability (P group, N = 7; NP group, N = 2).

Information was also drawn from interviews done with each patient, and a rough
estimate of post traumatic amnesia (PTA) was obtained for each subject. Each subject was
assigned a mild, moderate, or severe PTA score based on the time criteria provided by
Jennett (1983). A mild PTA was defined as lasting anywhere from a few minutes to an
hour. A moderate PTA was defined as lasting between one and twenty four hours, and a
severe PTA was defined as anything beyond the length of one day. Once again, there was a
trend for the problematic group to have longer PTA's, with the following distribution of
scores between groups: mild PTA length (P group, N = 5; NP group, N = 10); moderate
PTA length (P group, N = 2; NP group, N = 1); severe PTA length (P group, N=15; NP
group, N = 9).

Patient's medical records were also reviewed in order to retrospectively ascertain the
severity of their injury using criteria established by Levin and Grossman (1978). These
criteria were selected because the information required was available on all but one patient. They classified level of injury using the following criteria: mild injury (conscious on admission and during hospitalization with no focal neurologic manifestations); moderate injury (comatose for less than 24 hours and may or may not have a neurologic deficit); severe injury (coma exceeding 24 hours, and focal neurologic manifestations may be present). A Student's t test indicated that the P group was more severely injured ($t(1,39) = -1.81, p < .05$, one tailed test).

Subjects were also asked how they sustained their head injuries, and an interesting pattern of differences was revealed between the two groups. Although motor vehicle accidents were the most frequent cause of injuries in both groups, the P group sustained more injuries due to "unusual causes". Examples of unusual causes included: being kicked by a cow; hit by a rock; hit with an iron bar by "accident", and being struck by a piece of wood that was being cut by a large table saw etc. (see Table 1).

Means and standard deviations for each test by class are reported in Table 2 for each group. Each data class was then subjected to a MANOVA. The MANOVA results (see Table 3) will be discussed by data class.

**MANOVAs by Class**

**Intelligence**
Table 2. Means and Standard Deviations for Each Test by Class

**Class: Intelligence**

<table>
<thead>
<tr>
<th>Group</th>
<th>FSIQ</th>
<th>PIQ</th>
<th>VIQ</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No problem</td>
<td>103.45</td>
<td>97.05</td>
<td>108.05</td>
<td>13.40</td>
</tr>
<tr>
<td>(SD)</td>
<td>21.23</td>
<td>18.51</td>
<td>22.59</td>
<td>11.44</td>
</tr>
<tr>
<td>Problem</td>
<td>82.91</td>
<td>81.45</td>
<td>85.32</td>
<td>10.95</td>
</tr>
<tr>
<td>(SD)</td>
<td>17.75</td>
<td>15.16</td>
<td>18.59</td>
<td>8.86</td>
</tr>
</tbody>
</table>

**Class: Memory**

<table>
<thead>
<tr>
<th>Group</th>
<th>LMI</th>
<th>LMD</th>
<th>PAI</th>
<th>PAID</th>
<th>REY</th>
</tr>
</thead>
<tbody>
<tr>
<td>No problem</td>
<td>19.20</td>
<td>15.25</td>
<td>9.45</td>
<td>8.95</td>
<td>37.25</td>
</tr>
<tr>
<td>(SD)</td>
<td>7.73</td>
<td>7.89</td>
<td>1.00</td>
<td>1.64</td>
<td>33.07</td>
</tr>
<tr>
<td>Problem</td>
<td>12.50</td>
<td>7.55</td>
<td>7.82</td>
<td>6.38</td>
<td>36.82</td>
</tr>
<tr>
<td>(SD)</td>
<td>7.24</td>
<td>6.91</td>
<td>2.50</td>
<td>2.89</td>
<td>35.37</td>
</tr>
</tbody>
</table>
Table 2 (continued)

Class: Concept Formation - Set Shifting

<table>
<thead>
<tr>
<th>Group</th>
<th>Wisconsin Card Sort</th>
<th>Trail Making Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td># Categories achieved</td>
<td>Perseverative Responses</td>
</tr>
<tr>
<td>No problem</td>
<td>4.15</td>
<td>18.95</td>
</tr>
<tr>
<td></td>
<td>(SD) 1.87</td>
<td>16.16</td>
</tr>
<tr>
<td>Problem</td>
<td>3.91</td>
<td>21.68</td>
</tr>
<tr>
<td></td>
<td>(SD) 2.24</td>
<td>11.18</td>
</tr>
</tbody>
</table>

Class: Language

<table>
<thead>
<tr>
<th>Group</th>
<th>Thurstone's WF</th>
<th>Boston Naming Test</th>
<th>Aphasia Screen Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>No problem</td>
<td>63.35</td>
<td>52.55</td>
<td>1.80</td>
</tr>
<tr>
<td></td>
<td>(SD) 25.87</td>
<td>9.36</td>
<td>3.78</td>
</tr>
<tr>
<td>Problem</td>
<td>28.91</td>
<td>43.64</td>
<td>4.64</td>
</tr>
<tr>
<td></td>
<td>(SD) 14.55</td>
<td>13.84</td>
<td>6.11</td>
</tr>
</tbody>
</table>
Table 2. (continued)

Class: Motor

<table>
<thead>
<tr>
<th>Group</th>
<th>PegD</th>
<th>PND</th>
<th>FTD</th>
<th>FND</th>
<th>DYNND</th>
<th>DND</th>
</tr>
</thead>
<tbody>
<tr>
<td>No problem</td>
<td>107.00</td>
<td>97.65</td>
<td>40.70</td>
<td>38.45</td>
<td>34.65</td>
<td>31.65</td>
</tr>
<tr>
<td>(SD)</td>
<td>70.69</td>
<td>50.81</td>
<td>12.79</td>
<td>5.91</td>
<td>17.05</td>
<td>12.33</td>
</tr>
<tr>
<td>Problem</td>
<td>110.67</td>
<td>122.29</td>
<td>39.82</td>
<td>35.50</td>
<td>40.05</td>
<td>34.73</td>
</tr>
<tr>
<td>(SD)</td>
<td>49.65</td>
<td>55.83</td>
<td>14.32</td>
<td>12.62</td>
<td>13.25</td>
<td>15.19</td>
</tr>
</tbody>
</table>
Table 3. Multivariate Analyses by Class

<table>
<thead>
<tr>
<th>Class Name</th>
<th>Wilk's Lambda Value</th>
<th>F</th>
<th>df</th>
<th>Significance of F</th>
<th>Canonical Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intelligence</td>
<td>.741</td>
<td>3.22</td>
<td>(4, 37)</td>
<td>.02</td>
<td>.508</td>
</tr>
<tr>
<td>Memory</td>
<td>.601</td>
<td>4.78</td>
<td>(5, 36)</td>
<td>.002</td>
<td>.632</td>
</tr>
<tr>
<td>Concept Form.</td>
<td>.803</td>
<td>2.26</td>
<td>(4, 37)</td>
<td>.08</td>
<td>.443</td>
</tr>
<tr>
<td>Language</td>
<td>.563</td>
<td>9.82</td>
<td>(3, 38)</td>
<td>.001</td>
<td>.661</td>
</tr>
<tr>
<td>Motor</td>
<td>.854</td>
<td>.99</td>
<td>(6, 35)</td>
<td>.446</td>
<td>.381</td>
</tr>
</tbody>
</table>
The intelligence class consisted of the following components: Wechsler Adult Intelligence Scale-R 1) Full Scale IQ; 2) Performance IQ; 3) Verbal IQ; 4) and the absolute value of the difference between an individual's performance IQ and verbal IQ.

A one-way fixed-effects MANOVA (see Table 3) using group (NP versus P) as the independent variable and the four WAIS-R scores as dependent variables yielded a significant differentiation between the two groups [F(4,37) = 3.21, p < .02, canonical correlation = .51; accounting for 26% of the variance]. Subsequent univariate F tests demonstrated significant differences between the two groups on Full Scale IQ [F(1,40) = 12.79, p < .001]; Performance IQ [F(1,40) = 8.99, p < .005]; and Verbal IQ [F(1,40) = 12.78, p < .001]. The problematic group performed at a much lower level on these measures. A one-way fixed effects MANCOVA (see Table 5) using level of education as the covariate yielded no significant differences between the group centroids created by VIQ, PIQ, FSIQ, and P [Approx. F(4,36) = 1.98, p < .119]. Means and standard deviations adjusted for education are presented in Table 4.

Memory

This class was composed of several subtests from the Wechsler Memory Scale (1. the combined summary score for the immediate recall of bits of information from both of the logical memory paragraphs (LMI); 2. the number of bits recalled from these paragraphs after a 30 minute delay (LMD); 3. the total number of easy and hard paired associates
Table 4. Means and Standard Deviations
for Each Test by Class (Adjusted for Education)

<table>
<thead>
<tr>
<th>Variable</th>
<th>No Problem group</th>
<th>Problem group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adjusted Mean</td>
<td>SD</td>
</tr>
<tr>
<td>FSIQ</td>
<td>99.81</td>
<td>23.78</td>
</tr>
<tr>
<td>PIQ</td>
<td>94.04</td>
<td>21.38</td>
</tr>
<tr>
<td>VIQ</td>
<td>104.29</td>
<td>25.92</td>
</tr>
<tr>
<td>LMI</td>
<td>17.90</td>
<td>9.59</td>
</tr>
<tr>
<td>LMD</td>
<td>14.15</td>
<td>9.91</td>
</tr>
<tr>
<td>PAI</td>
<td>9.27</td>
<td>2.79</td>
</tr>
<tr>
<td>PAID</td>
<td>8.71</td>
<td>3.37</td>
</tr>
<tr>
<td>WCS</td>
<td>3.97</td>
<td>3.02</td>
</tr>
<tr>
<td>WIS</td>
<td>20.44</td>
<td>1.94</td>
</tr>
<tr>
<td>TRAIL</td>
<td>50.55</td>
<td>82.03</td>
</tr>
<tr>
<td>TRA</td>
<td>108.36</td>
<td>154.93</td>
</tr>
<tr>
<td>TWF</td>
<td>60.63</td>
<td>28.71</td>
</tr>
<tr>
<td>BNT</td>
<td>57.18</td>
<td>16.85</td>
</tr>
<tr>
<td>AST</td>
<td>2.49</td>
<td>7.06</td>
</tr>
<tr>
<td>PEGD</td>
<td>81.63</td>
<td>131.22</td>
</tr>
<tr>
<td>PND</td>
<td>88.76</td>
<td>144.63</td>
</tr>
<tr>
<td>FTD</td>
<td>40.06</td>
<td>20.34</td>
</tr>
<tr>
<td>FND</td>
<td>38.04</td>
<td>14.96</td>
</tr>
<tr>
<td>DYND</td>
<td>35.34</td>
<td>22.68</td>
</tr>
<tr>
<td>DND</td>
<td>32.62</td>
<td>20.47</td>
</tr>
</tbody>
</table>
recalled on an immediate basis on the best trial of either (total possible = 10) (PAI); 4. PAI after a 30 minute delay (PAID);) and 5. the patient's recall score on the Rey-Osterreith Complex figure test.

A MANOVA on this class (see Table 3) indicated a significant difference between the two groups [F (5,36) = 4.78, p < .002, canonical correlation = .63; accounting for 40% of the variance]. Subsequent univariate analyses of variance indicated that there were significant differences between the two groups on logical memory immediate recall [F (1,40) = 8.42, p < .006], logical memory delayed recall [F (1,40) = 1.37, p < .002] paired associates immediate recall [F (1,40) = 7.43, p < .009] and paired associated delayed recall [F (1,40) = 12.86, p < .001]. Once again, the problematic group did not perform as well on instruments that assess memory.

A MANCOVA (see Table 5) was used again to covary educational differences between the two groups. The MANCOVA revealed that the groups differed on these measures even after the effect of education was removed [ approximate F (5,35) = 3.56, p < .01, canonical correlation = .58]. A univariate analysis of covariance indicated that the groups differed on specific tests even after educational differences were covaried [logical memory immediate recall F (1, 39) = 4.04, p < .05; logical memory delayed recall F (1,39) = 6.62, p < .01; paired associates immediate recall F (1, 39) = 4.46, p < .04; paired associated delayed recall F (1, 39) = 8.51, p < .006].
Table 5. Multivariate Analysis of Covariance
(Education as the Covariate): by Data Class

<table>
<thead>
<tr>
<th>Class Name</th>
<th>Wilk's Approximate Lambda Value</th>
<th>Wilk's Approximate F</th>
<th>Significance of F</th>
<th>Canonical Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intelligence</td>
<td>(4, 36) 0.820</td>
<td>1.98</td>
<td>0.119</td>
<td>0.424</td>
</tr>
<tr>
<td>Memory</td>
<td>(5, 35) 0.680</td>
<td>3.29</td>
<td>0.015</td>
<td>0.565</td>
</tr>
<tr>
<td>Concept Formation</td>
<td>(4, 36) 0.867</td>
<td>1.39</td>
<td>0.258</td>
<td>0.365</td>
</tr>
<tr>
<td>Language</td>
<td>(3, 37) 0.623</td>
<td>7.48</td>
<td>0.001</td>
<td>0.614</td>
</tr>
<tr>
<td>Motor</td>
<td>(6, 34) 0.841</td>
<td>1.07</td>
<td>0.399</td>
<td>0.399</td>
</tr>
</tbody>
</table>
Language

This class consisted of performance on three tests; the Boston Naming test (number of correct items), the Halstead Wepman Aphasia Screening test (number of errors), and Thurstone's Word Fluency test (total number of words generated). A one-way fixed effects MANOVA was completed (see Table 3) using the variables within this class and was found to be significant \( F(3, 38) = 9.81, p < .001 \), canonical correlation = .66; accounting for 44% of the variance. Subsequent one-way analyses of variance revealed significant differences between the two groups; the P group always demonstrating more impairments than the NP group on Thurstone's Word Fluency test \( F(1,40) = 28.97, p < .001 \), and the Boston Naming test \( F(1,40) = 5.86, p < .02 \). There was a tendency for the P group to make more errors on the Halstead Wepman Aphasia Screening test as well \( F(1,40) = 3.20, p < .08 \), although this finding was not statistically significant.

Education was again used as a covariate (see Table 5), and the groups differed on these variables after the differences due to education were removed \( F(3, 37) = 7.48, p < .001 \), canonical correlation = .61. A further analysis of covariance indicated that the two groups still differed significantly on Thurstone's Word Fluency test \( F(1, 39) = 21.81, p < .001 \), and there were not significant performance differences on the Boston Naming test \( F(1,39) = 2.93, p < .095 \).

Concept Formation

This class was composed of: 1) the number of categories achieved on the Wisconsin
Card Sort test, 2) the number of perseverative responses made on the Wisconsin Card Sort test, 3) the time score from Trail Making test A, and 4) the time score from Trail Making test B. There were no significant differences between the two groups as determined by a MANOVA \( F(4,37) = 2.26, p < .08, \) canonical correlation = .44.

**Motor Performance**

Variables within this class consisted of: 1) dominant and 2) nondominant hand time scores on the Grooved Pegboard test; 3) dominant and 4) nondominant tapping scores on the Finger Tapping test; and 5) dominant and 6) nondominant kilograms of grip strength pressure on the Hand Held Dynamometer test. A MANOVA computed on these variables was not significant \( F(6,35) = .99, p < .44, \) canonical correlation = .38.

**Sex Ratio Differences**

Due to the sex ratio differences between groups, a MANOVA was carried out to examine the differences between the males in the NP group versus the P group across the best dependent variables (Full Scale IQ, TWF, Trails B, PAID, and PND) chosen from each class. The two groups were significantly different \( F(5,24) = 6.62, p < .001, \) canonical correlation = .76]. Subsequent univariate F tests demonstrated significant differences between the two groups on Full Scale IQ \( F(1,28) = 12.8, p < .001 \); PAID \( F(1,28) = 7.16, p < .01 \); Trails B \( F(1,28) = 5.20, p < .03 \) and TWF \( F(1,28) = 36.72, p < .001 \).

**Discriminant Analyses**

A stepwise discriminant function analysis was performed on each class, and the following
variables were found to be the best predictors by class (see Table 6): Full Scale IQ \( [E (1,40) = 12.79, p < .0009, \text{canonical correlation} = .49] \); paired associates delayed, \( [E (1,40) = 12.86, p < .0009, \text{canonical correlation} = .49] \); Trails B \( [E (1,40) = 6.78, p < .01, \text{canonical correlation} = .38] \); and Thurstone's Word Fluency test \( [E (1,40) = 28.97, p < .0001, \text{canonical correlation} = .65] \) (see Table 6). Non-dominant performance on the Grooved Pegboard test approached significance \( [E (1,40) = 3.73, p < .06, \text{canonical correlation} = .29] \). The percent of cases correctly classified by group by these predictors was: Full Scale IQ (69% overall); paired associates delayed (71% overall); Trails B time score (67% overall); Thurstone's Word Fluency test (76% overall), and non-dominant Grooved Pegboard time (67% overall). The percent of individuals correctly identified by group by these predictors is outlined in Table 7.

The above (significant) variables were then put into a stepwise discriminant function, and it was found that the Thurstone's Word Fluency test could be used alone to correctly classify 76% of the cases \( [\text{Chi-Square} (1) = 21.57, p < .0001, \text{canonical correlation} = .65; \text{accounting for 42% of the variance}] \) (see Table 8). This resulted in correct classification of the P group 86% of the time, and 65% of the time in the NP group (see Table 9).

A direct discriminant function analysis was carried out using Full Scale IQ, paired associates delayed, Trails B time scores, Thurstone's Word Fluency, and nondominant performance on the grooved pegboard as predictor variables and group membership (P or NP) as the criterion variable revealed a significant discrimination between the two
Table 6. Results of a Stepwise Discriminant Analysis to predict Group Membership from Neuropsychological Performance by Class

<table>
<thead>
<tr>
<th>Class</th>
<th>Variable</th>
<th>Steps Needed</th>
<th>Wilk's Lambda</th>
<th>F</th>
<th>Significance Level</th>
<th>Canonical Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intelligence</td>
<td>FSIQ</td>
<td>1</td>
<td>.758</td>
<td>12.79</td>
<td>.0009</td>
<td>.492</td>
</tr>
<tr>
<td>Memory</td>
<td>PAID</td>
<td>3</td>
<td>.757</td>
<td>12.86</td>
<td>.0009</td>
<td>.493</td>
</tr>
<tr>
<td>Concept Formation</td>
<td>Trail B</td>
<td>1</td>
<td>.855</td>
<td>6.78</td>
<td>.01</td>
<td>.381</td>
</tr>
<tr>
<td>Language</td>
<td>TWF</td>
<td>1</td>
<td>.580</td>
<td>28.97</td>
<td>.0001</td>
<td>.648</td>
</tr>
<tr>
<td>Motor</td>
<td>PND</td>
<td>1</td>
<td>.915</td>
<td>3.73</td>
<td>.06</td>
<td>.292</td>
</tr>
</tbody>
</table>
Table 7. Classification of Subjects by Best Variable Obtained by Stepwise Discriminant Analysis by Class

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Predicted Group Membership</th>
<th>Overall Classification Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Full Scale IQ</strong></td>
<td></td>
<td><strong>69%</strong></td>
</tr>
<tr>
<td>Actual Group</td>
<td>NP group*</td>
<td>P Group**</td>
</tr>
<tr>
<td>NP</td>
<td>11 (55%)</td>
<td>9 (45%)</td>
</tr>
<tr>
<td>P</td>
<td>4 (18.2%)</td>
<td>18 (81.8%)</td>
</tr>
<tr>
<td><strong>Paired Associates</strong></td>
<td></td>
<td><strong>71%</strong></td>
</tr>
<tr>
<td><strong>Delayed</strong></td>
<td>NP</td>
<td>15 (75%)</td>
</tr>
<tr>
<td>NP</td>
<td>15 (75%)</td>
<td>5 (25%)</td>
</tr>
<tr>
<td>P</td>
<td>7 (31.8%)</td>
<td>15 (68.2%)</td>
</tr>
<tr>
<td><strong>Trails B</strong></td>
<td></td>
<td><strong>67%</strong></td>
</tr>
<tr>
<td>NP</td>
<td>16 (80%)</td>
<td>4 (20%)</td>
</tr>
<tr>
<td>P</td>
<td>10 (45.5%)</td>
<td>12 (54.5%)</td>
</tr>
<tr>
<td><strong>Thurstone's Word Fluency</strong></td>
<td></td>
<td><strong>76%</strong></td>
</tr>
<tr>
<td>NP</td>
<td>13 (65%)</td>
<td>7 (35%)</td>
</tr>
<tr>
<td>P</td>
<td>3 (13.6%)</td>
<td>19 (86.4%)</td>
</tr>
<tr>
<td><strong>Grooved Pegboard-Non-dominant hand</strong></td>
<td></td>
<td><strong>67%</strong></td>
</tr>
<tr>
<td>NP</td>
<td>14 (70%)</td>
<td>6 (30%)</td>
</tr>
<tr>
<td>P</td>
<td>8 (36.4%)</td>
<td>14 (63.6%)</td>
</tr>
</tbody>
</table>

* No problem Group
** Problematic (high anger) Group
Table 8. Results of the Stepwise Discriminant Analysis Utilizing the Best Predictor Variable from Each Class

<table>
<thead>
<tr>
<th>Variable Selected</th>
<th>Steps Needed</th>
<th>Wilk's Lambda</th>
<th>Minimum F</th>
<th>Significance level</th>
<th>Canonical Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thurstone's Word Fluency</td>
<td>1*</td>
<td>.58</td>
<td>28.97</td>
<td>.0001</td>
<td>.648</td>
</tr>
</tbody>
</table>

*No further analysis was needed as other variables did not contribute unique variance.

Table 9. Classification Results Obtained Using a Stepwise Discriminant Analysis Based on the Best Predictor from Each Class

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Predicted Group Membership</th>
<th>Overall Classification Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thurstone's Word Fluency Test</td>
<td></td>
<td>76%</td>
</tr>
<tr>
<td>Actual Group NP group</td>
<td>13 (65%)</td>
<td>7 (35%)</td>
</tr>
<tr>
<td>NP group</td>
<td>3 (13.6%)</td>
<td>19 (86.4%)</td>
</tr>
</tbody>
</table>
groups (Chi-Square (5) = 21.495, \( p < .0007 \)). The canonical correlation between the predictor variables and group membership was .66 which accounts for 44% of the variance. The classification rate by group was similar to that produced by the stepwise analysis (see Table 10).

A hierarchical discriminant analysis was also carried out (see Table 11), entering variables in a predetermined order (order of entry by "hypothesized clinical relevance": Full Scale IQ, paired associates delayed, Trails B, and Thurstone's Word Fluency), and produced an identical classification rate of 79% [Chi-Square (4) = 21.774, \( p < .0002 \), canonical correlation = .66, accounting for 44% of the variance]. Once again, the overall classification by group was the same as before (79%). Predicted group membership assignment is outlined in Table 12. It should be noted that all variables passed the tolerance test (level .001) and were retained in the discriminant function.
Table 10. Classification Results of the Direct Model
Discriminant Function Analysis

<table>
<thead>
<tr>
<th>Actual Group</th>
<th>N</th>
<th>Predicted Group Membership</th>
<th>Overall Rate of Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>NP group</td>
<td>P group</td>
</tr>
<tr>
<td>No Problem group</td>
<td>20</td>
<td>14 (70%)</td>
<td>6 (30%)</td>
</tr>
<tr>
<td>Problem group</td>
<td>22</td>
<td>3 (13.6%)</td>
<td>19 (86.4%)</td>
</tr>
</tbody>
</table>

Table 11. Results of the Hierarchical Discriminant Analysis:
Order of Variable Entry Predetermined

<table>
<thead>
<tr>
<th>Step Order</th>
<th>Variable Entered</th>
<th>Wilk's lambda</th>
<th>Equivalent F</th>
<th>Significance Level</th>
<th>Canonical Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1</td>
<td>FSIQ</td>
<td>.758</td>
<td>12.79</td>
<td>.0009</td>
<td></td>
</tr>
<tr>
<td>Step 2</td>
<td>PAID</td>
<td>.723</td>
<td>7.46</td>
<td>.0018</td>
<td></td>
</tr>
<tr>
<td>Step 3</td>
<td>Trails B</td>
<td>.715</td>
<td>5.04</td>
<td>.0049</td>
<td></td>
</tr>
<tr>
<td>Step 4</td>
<td>TWF</td>
<td>.564</td>
<td>7.16</td>
<td>.0002</td>
<td>.660</td>
</tr>
</tbody>
</table>
Table 12. Classification Results of the Hierarchical Discriminant Analysis

<table>
<thead>
<tr>
<th>Actual Group</th>
<th>N</th>
<th>Predicted Group Membership</th>
<th>Overall Rate of Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>No Problem Group</td>
<td>20</td>
<td>14 (70%)</td>
<td>6 (30%)</td>
</tr>
<tr>
<td>Problem Group</td>
<td>22</td>
<td>3 (13.6%)</td>
<td>19 (86.4%)</td>
</tr>
</tbody>
</table>
Discussion

The present findings support the hypothesis that neuropsychological tests can discriminate between groups of individuals with closed head injuries (CHI); those who have anger control problems (P), and those who do not (NP). Specific tests (WAIS-R Full Scale IQ, paired associates delayed recall from the Wechsler Memory Scale, Trail Making test path B time score, and Thurstone's Word Fluency test) seemed to be the best predictors of group membership. As a group, individuals with anger control problems were found to have more severe intellectual, memory, and language deficits than those individuals who do not have anger control problems. Even after statistically removing educational differences between these two groups, there were still significant performance differences between the two in terms of their memory and word finding skills. In fact, group membership could be predicted 76% of the time based on their performance on Thurstone's Word Fluency test alone.

Previous studies have found that individuals with CHI have significant deficits on tests that assess intellectual abilities (Mandelberg et al, 1975; Roberts, 1979, Levin et al., 1979; and Klove et al., 1972), mnestic functioning (Stuss et al., 1975; Brooks, 1975; Russel, 1971) set shifting (Rimel et al., 1981, 1982; Stuss et al., 1985) and word finding problems (Levin et al, 1976). The pattern of results obtained during the course of this study fits well with what is known about the variety of neuropsychological deficits that individuals with CHI display, and suggests that there maybe certain performance risk factor patterns that can be used to differentiate these groups. The direction of the findings
makes sense from a clinical standpoint; individuals who are more cognitively impaired were more likely to have anger control problems. Although severity of the injury sustained was evaluated on a retrospective basis, there seems to be a reasonable amount of evidence that individuals in the P group had more severe injuries as compared to the NP group. This finding also fits with what is currently known about individuals who are habitually aggressive; they may have greater CNS dysfunction than those who are not aggressive (Williams, 1969; Elliott, 1982; Mark and Ervin, 1970; and Andrulonis, 1980). Unfortunately the sample sizes used in this study did not permit the use of a hold-out sample to see if a discriminant function could be developed with part of the sample and then used to predict membership classification on the hold-out sample.

It would seem that motor performance, as assessed by the few instruments given, does not differentiate between these two groups or aid in predicting group membership. There may be many reasons why these tests did not have any utility in this study. Many of the patients in both groups had a history of peripheral injuries (broken arms, fingers etc.), which decreases the utility of these tests when they are used to look at motor performance mediated by the cerebral cortex. Peripheral injuries also add variance problems that are difficult to covary out given the sample sizes involved in this study. In addition, motor tests are sensitive to drug effects. Many of the patients in the study were taking anticonvulsants or other drugs, and practical considerations precluded doing a psychometric evaluation during a drug holiday period.

It also appears that the Novaco Anger Inventory was of little value from a clinical or
research standpoint during the course of this study. It did not reveal significant differences between these two groups, despite the fact that relatives and physicians could clearly identify subjects who had anger control problems. Relatives and physicians who knew subjects in the high anger group described them as more likely to: 1) destroy property; 2) have temper outbursts on a frequent basis; 3) assault someone; 4) be in need of treatment for anger control problems. Individuals in the nonproblematic group were described by their relatives in a much different manner. In all cases, relatives did not report that treatment for anger control problems was warranted. Some relatives were concerned about other personality changes they had noticed, such as depression and increased irritability, but they did not describe their relative as being assaultive or as someone who would have frequent temper outbursts, or destroy property.

As a result, the Novaco Anger Inventory could not be used to help classify subjects into groups because of the pattern that began to develop when the test was administered. Subjects who had clear anger control problems would have been misclassified by this test, as would subjects without anger control problems. As mentioned earlier, this test may have been of limited utility because it is not behaviorally anchored. High scorers in the nonproblematic group frequently described very mild behavioral anchors when asked to characterize themselves at the highest level of anger on the scale (5 = extreme anger). "I would walk away from someone if I was that mad." High anger subjects who received low scores anchored the items much differently; "I would beat someone to a pulp if I was that mad (level 5)." One high anger subject described his midpoint level (rating = 3) of anger
as: 'I suppose I would just slap the person.' Further research with larger samples using a behaviorally anchored instrument may demonstrate whether this instrument is of utility with an organic population.

This study addressed a number of issues that others have failed to do in the past. For example, other studies have primarily focused on inmate populations (violent versus nonviolent offenders) who had not been medically screened. No previous group design study could be found in which subjects: 1) were not inmates, and 2) were all diagnosed as having a specific type of physical insult. In addition, other studies have not addressed specific forms of cognitive impairment issues (ie, memory versus language problems, etc.). The majority of studies to date have employed unsound statistical analysis procedures as well, which have increased the probability of Type I errors (ie, multiple t test analysis, or inappropriate subject/variable ratios). As a result, all that can be said about previous studies is that: 1) habitually aggressive offenders may have more neurologic soft signs, and 2) aggressive offenders may not do as well on neuropsychological batteries such as the Luria-Nebraska, or the Halstead-Reitan. However, both of these conclusions are fraught with problems, as the first conclusion does not take into account base rate phenomena or experimenter bias, and the second suffers from Type I error problems. This study is distinctive in a number of ways. The population examined consisted of outpatients who had a clear etiology for their neurologic problems: closed head injury. As a result, the inclusion criteria were strict. In addition, few studies have tried to increase the validity of the assessments conducted by
doing such extensive family interviews to verify information provided by individuals who may be questionable historians. The study also sought to examine specific areas of cognitive impairment, rather than looking at a global impairment index. This type of approach seems to make more sense from both a clinical and a research standpoint. If specific deficits have been described in the literature, and if these deficits are associated with anger control problems, then why not design a study to see if specific cognitive deficits are predictive of group membership? In addition, very conservative statistical techniques were used in an attempt to control for Type I error problems.

Unfortunately, this design made research subjects difficult to recruit because of the number of hours involved, and the other stringent entrance criteria that had to be met. Approximately 650-700 medical records were reviewed to determine whether individuals met the criteria for the study (ie, no pre-accident history of treatment for drug/alcohol abuse, diagnosis of mental retardation, or other preexisting neurological disorders). Of this group, only 42 subjects met the criteria and agreed to participate. Twenty nine other subjects met the medical criteria for the study, and were contacted by mail (N=22), or by phone/in person. Of the 22 letters sent, 18 were returned with no forwarding address, and none of these individuals could be contacted by phone. Four individuals contacted by phone refused to participate because of ongoing litigation, and two individuals were unable to be evaluated because of "transportation problems" or other health reasons. Five individuals were tested but had to be excluded from the study because
of a history of treatment for drug/alcohol abuse. They initially reported that they had not undergone treatment for this problem, but medical records obtained from other facilities proved otherwise. The sample sizes obtained however, are comparable to studies that did not set such stringent entrance standards.

One of the issues raised by this study, which cannot be definitively answered, is whether there were premorbid intellectual differences between these two groups. An attempt was made to control for this problem during the data analysis by covarying out educational differences between these two groups. Covarying out intelligence was considered as an alternative. If IQ had been used as a covariate however, one would be assuming that this measure is a stable reflection of pre-morbid intellectual ability, which is not the case. IQ scores are effected by the level of severity of the injury. This became a salient consideration when it was found that there were qualitative/quantitative differences between these two groups with respect to severity of injury. There are ways to calculate premorbid intellectual levels based on demographic data (Goldstein et al, 1986) through the use of regression equations, but these methods have limited reliability for subjects who score one standard deviation above or below the mean. As a result, these techniques would have limited utility for this sample.

It is just as likely, if not more likely, that the high anger group may have had significantly more problems on neuropsychological tests because they had experienced
more severe head injuries. It is clear that this group had a significantly higher incidence of multiple injury, which as Boll (1982) points out, may have a cumulative negative effect on cognitive skills. In addition, there was a tendency for individuals in the anger group to do less well in terms of post-injury outcome (as assessed in a post hoc manner with the Glasgow Outcome Scale). Examination of medical records also provided some support for the qualitative finding that these subjects were more severely injured on admission (using Levin's et al. (1982) criteria). Qualitative evidence does support the contention that the anger control group is more cognitively impaired than the nonproblematic group due to a difference in severity of injury. This suggests that greater damage maybe associated with anger control problems, although additional studies are needed in order to directly test this hypothesis.

Further studies are needed that either control for premorbid intellectual ability, or severity of injury issues. It may be that the only feasible way that this type of study could be done, would be by looking at populations who have had psychometric examinations prior to injury (such as military personnel, or certain government employee populations). This would allow one to look at premorbid intellectual differences between groups. It maybe that individuals in the high anger group are more at risk for developing head injuries because they are not as bright, and therefore are not good problems solvers. They may leave themselves at risk for the development of a head injury because they are risk takers in situations which brighter individuals would avoid. Future research of this kind would have to be done by a multidisciplinary team. This would enable one to control
severity of injury issues as members of the team would be responsible for systematically rating all patients on admission with the Glasgow Coma Scale (or a similar instrument) in addition to carefully documenting neurologic deficits.

The conclusions of this study suggest directions for a series of studies, particularly with regard to assessment and treatment of the problematic population. Can these findings be replicated? Could one predict group membership with another sample using a discriminant function developed from this data set? Can you identify individuals (early in the hospitalization process) who will develop anger control problems simply on the basis of their neuropsychological deficit profile? If so, wouldn't these clients benefit from early intervention, focused on remediation of their specific neuropsychological deficits? Also, this study indicates that CHI men are more likely to have anger control problems than women. It is not clear whether this finding was due to a sampling bias (for whatever reason), or whether this is a characteristic of the greater population. Although the literature suggests that men are more likely than women to be arrested and convicted for participation in violent crimes (Heller et al., 1984), there is very little written in the literature regarding whether CHI men are more likely to have anger control problems than CHI women. As a result, future studies should address whether CHI men are more likely to experience this problem. It is interesting however, that P men are significantly more impaired on neuropsychological tests than NP men. This would seem to indicate that the groups differ quite significantly on neuropsychological tests despite the fact that gender may be a significant predictor of group membership. Another issue raised by this
study is whether cognitive rehabilitation will prevent the development of anger control problems. Also, if individuals with anger control problems have memory deficits and word finding problems, are they also more likely to have social skills problems that are not as amenable to conventional treatment procedures? Do we have to alter the way we carry out psychotherapeutic processes with these patients because of the specific neuropsychological deficits they have? All of these questions have yet to be answered, and all deal with enormously complex issues. Future studies may shed light on these areas, and lead to more efficient and compassionate treatment for very distressed individuals and their families.

In summary, this study indicates that CHI individuals with anger control problems are significantly different from CHI individuals who do not have temper problems. The P group was less educated, and demonstrated greater deficits on neuropsychological instruments that assess intelligence, memory and language skills. Even after statistically removing educational differences between the groups, the P group continued to demonstrate significantly more memory and language deficits. In addition, CHI individuals with anger control problems are more likely to be male, and are more likely to have experienced a severe CHI. Further work is needed to replicate these findings with a larger sample using a multidisciplinary team. This study does pose interesting questions that could be the focus of future basic or applied research studies.
References

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Appendix A

Classification of Head Injury Severity

Minor
a. contusions, abrasions, superficial lacerations
b. fractures, dislocation of nose
c. mild concussion with no loss of consciousness
d. teeth loosened, broken, or knocked out
e. whiplash (unqualified)

Moderate
a. "deep" or "disfiguring" lacerations
b. extensive laceration without dangerous hemorrhage
c. compound, comminuted fractures of nose
d. concussion with unconsciousness 5-30 minutes
e. skull fracture without concussion
f. loss of eye

Dangerous
a. laceration with dangerous hemorrhage
b. skull fracture with concussion as evidenced by loss of consciousness up to two hours
c. concussion as evidenced by loss of consciousness from 30 minutes up to two hours without reference to possible intracranial injury
d. depressed fractures of skull
e. evidence of critical intracranial damage

Appendix B

General Medical Complications

1. Infection
   a. pulmonary
   b. genitourinary
   c. infections of monitoring devices
   d. septicemia
2. Drug toxicities
   a. allergic reactions
   b. CNS depression
   c. movement disorders
   d. renal failure, other organ system toxicities
3. Gastrointestinal hemorrhage
4. Upper respiratory trauma, infection, obstruction from intubation
5. Embolic-pulmonary
   a. thrombophlebitis
   b. fat emboli
6. Endocrine-metabolic disorders
   a. electrolyte-fluid imbalances
   b. malnutrition
   c. overeating-obesity
   d. pituitary failure
   e. inappropriate antidiuretic hormone secretion
   f. Cushing syndrome due to steroid administration
7. Musculoskeletal disorders
   a. heteroptic bone formation
   b. osteoporosis
   c. disuse muscle atrophy
   d. contractures
   e. secondary injuries; falls, etc.
8. Peripheral neuropathies
   a. drug induced
   b. compression neuropathies
General Medical Complications
(continued)

9. Hematologic disorders
   a. anemias
   b. bleeding diatheses

10. Dermatologic disorders
    a. acne, drug induced
    b. decubitus ulcers
    c. dependent edema
    d. injuries: self-induced, restraints, etc.

11. Autonomic disturbances
    a. hypertension
    b. sweating disorders
    c. hyperventilation

12. Urinary tract disorders
    a. infection
    b. calculi
    c. postcatheter urethral stricture
    d. neuropathic bladder dysfunction

### Appendix C

**Neuropsychology of Affective Behavior**

Summary of experiments on perception of socially relevant stimuli

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
<th>Basic References</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Normal subjects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Dichotic nonverbal sounds</td>
<td>Left ear superiority</td>
<td>King and Kimura, 1972</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Carmon and Nachshon, 1973</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Haggard and Parkinson, 1971</td>
</tr>
<tr>
<td>2. Dichotic emotionally toned sentences</td>
<td>Left-hemisphere comprehends content;</td>
<td>Safer and Leventhal, 1977</td>
</tr>
<tr>
<td></td>
<td>Right comprehends emotional tone</td>
<td></td>
</tr>
<tr>
<td>3. Tachistoscopic faces</td>
<td>Right-hemisphere superiority for recognition of facial expression</td>
<td>Ley and Bryden, 1979</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Buchtel et al., 1978</td>
</tr>
<tr>
<td>4. Split-field moves</td>
<td>Elaboration of emotional tone by right hemisphere</td>
<td>Dimond et al., 1976; 1977</td>
</tr>
</tbody>
</table>

| **Neurologic patients**                   |                                             |                                |
| 1. Judgment of mood in others             | Right-hemisphere lesions impair comprehension | Hellman et al., 1975           |
| 2. Judgment of propositional affect       | Left-hemisphere lesions impair comprehension | Kolb, 1977                     |
| 3. Comprehension of verbal humor          | Left-hemisphere lesions impair comprehension | Kolb et al., 1980              |
| 4. Comprehension of non-verbal humor      | Right-hemisphere lesions impair comprehension | Gardner et al., 1975           |
| 5. Matching pictures of emotional facial expressions | Right-hemisphere lesions impair performance | Kolb et al., 1980              |
|                                           |                                             | Kolb, 1977                     |

New York, N.Y.: The Guilford Press.
# Appendix D

## Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Examiner's Test</th>
<th>Patient's Response</th>
<th>Assigned Score</th>
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<tbody>
<tr>
<td><strong>Eye Opening</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>Opens eyes on own</td>
<td>4</td>
</tr>
<tr>
<td>Speech</td>
<td>Opens eyes when asked in a loud voice</td>
<td>3</td>
</tr>
<tr>
<td>Pain</td>
<td>Opens eyes when pinched</td>
<td>2</td>
</tr>
<tr>
<td>Pain</td>
<td>Does not open eyes</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best Motor Response</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Commands</td>
<td>Follows simple commands</td>
<td>6</td>
</tr>
<tr>
<td>Pain</td>
<td>Pulls examiner's hand away when pinched</td>
<td>5</td>
</tr>
<tr>
<td>Pain</td>
<td>Pulls a part of body away when examiner pinches patient</td>
<td>4</td>
</tr>
<tr>
<td>Pain</td>
<td>Flexes body inappropriately to pain (decorticate posturing)</td>
<td>3</td>
</tr>
<tr>
<td>Pain</td>
<td>Body becomes rigid in an extended position when examiner pinches victim (decerebrate posturing)</td>
<td>2</td>
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<tr>
<td>Pain</td>
<td>Has no motor response to pinch</td>
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<tr>
<td><strong>Verbal Response (Talking)</strong></td>
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<td></td>
</tr>
<tr>
<td>Speech</td>
<td>Carries on a conversation correctly and tells examiner where he is, who he is, and the month and year.</td>
<td>5</td>
</tr>
<tr>
<td>Speech</td>
<td>Seems confused or disoriented</td>
<td>4</td>
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<tr>
<td>Speech</td>
<td>Talks so examiner can understand victim, but makes no sense</td>
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</tr>
<tr>
<td>Speech</td>
<td>Makes sounds that examiner can't understand</td>
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<tr>
<td>Speech</td>
<td>Makes no noise</td>
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</table>

Appendix E

Simulation Diagrams of Rotational Shear Strains Produced by Brain Trauma

Model 1: A blow on the occiput

Model 2: A sideways blow in the neighborhood of the upper jaw

Model 3: A blow above the ear.

Shading indicates the scale of maximum shear strain

### Appendix F

#### Data Sets by Area

<table>
<thead>
<tr>
<th>Area</th>
<th>Test Description</th>
<th>Reference Label</th>
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<tbody>
<tr>
<td><strong>Intelligence</strong></td>
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<tr>
<td>A. WAIS-r PIQ</td>
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<td>PIQ</td>
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<tr>
<td>B. WAIS-r VIQ</td>
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<td>C. WAIS-r FSIQ</td>
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<td>FSIQ</td>
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<td>D. WAIS-r [PIQ - VIQ]</td>
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<tr>
<td><strong>Memory</strong></td>
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<td></td>
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<tr>
<td>A. Wechsler Memory Scale</td>
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<td>WMS</td>
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<tr>
<td>1. Logical Memory Section - immediate recall</td>
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<td>LMI</td>
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<tr>
<td>2. 30 minute delayed recall</td>
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<td>LMD</td>
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<td>3. Paired Associate Recall - immediate recall</td>
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<td>PAI</td>
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<td>4. 30 minute delayed recall</td>
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<td>PAID</td>
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<td>B. Rey Osterreith Complex Figure - 3 minute delayed recall</td>
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<td>Rey</td>
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<td><strong>Concept Formation - Set Generation</strong></td>
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<td>A. Wisconsin Card Sort - Number of Categories Achieved</td>
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<tr>
<td>B. - Number of Perseverative Errors</td>
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<td>WCS</td>
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<td>C. Trail Making Test - Part A (time score)</td>
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<td>TRAIL</td>
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<tr>
<td>D. - Part B (time Score)</td>
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<td>TRA</td>
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<tr>
<td><strong>Language</strong></td>
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<td></td>
</tr>
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<td>A. Thurstone's Word Fluency Test - Total Number of words generated.</td>
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<td>TWF</td>
</tr>
<tr>
<td>B. Boston Naming Test - Number of pictures correctly identified</td>
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<td>BNT</td>
</tr>
<tr>
<td>C. Aphasia Screening Test - Number of errors</td>
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<td>AST</td>
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<tr>
<td><strong>Psychomotor Performance</strong></td>
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<td></td>
</tr>
<tr>
<td>A. Grooved Pegboard - Time: (dominant hand)</td>
<td></td>
<td>PEGD</td>
</tr>
<tr>
<td>B. - Time: (other hand)</td>
<td></td>
<td>PND</td>
</tr>
<tr>
<td>C. Finger Tapping - Time: (dominant hand)</td>
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<td>FTD</td>
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<tr>
<td>D. - Time: (other hand)</td>
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<td>FND</td>
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<tr>
<td>E. Dynamometer - Kilograms: (dominant hand)</td>
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<td>F. - Kilograms: (other hand)</td>
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<td>DND</td>
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Appendix G
Structured Intake Interview

General Identification Information

1. Age ____
2. Sex ____
3. Race: Caucasian ____
   Negro ____
   Occidental ____
   Indian ____
4. Family member present at interview: (circle one)
   Husband  Brother  In-law
   Father   Aunt    very close friend
   Wife     Uncle
   Mother   Cousin
   Sister   Grandparent
5. Years of education (patient): ______

Description of Injury

1. When did the accident happen? ________ (date)

Check one:
Less than 3 months ago
4-6 months ago
7-11 months ago
12-24 months ago
25-36 months ago
37-48 months ago
Longer than 4 years ago

2. What kind of accident did you have?

Check one:
Motor vehicle
Fall
Sports related
Assault
Other
3. How long did you stay in the hospital after the injury?

Check one:

- Not hospitalized
- Few days
- Week
- 2-3 weeks
- Month
- Months

4. How long were you unconscious?

Check one:

- No loss of consciousness
- Minutes
- Hour(s)
- Day
- Several days
- Week
- Longer than 1 week

5. Thinking back, can you remember:

Check one:

- The accident
- Events minutes before the accident
- Events hours before the accident
- Events a day before the accident
- Events days to weeks before the accident

6. After the accident, was the first thing that you can remember:

Check one:

- The accident
- Minutes after the accident
- Hours after the accident
- A day after the accident
- Days to weeks after the accident

Perceived Neuropsychological Changes Since the Injury

1. Since the accident, have you had more problems with your memory?

Never
2. Do you ever forget what people tell you if it is something important?

Never
Sometimes
Frequently

3. Do you ever forget to do things, or begin to do something and forget what it was you were going to do?

Never
Sometimes
Frequently

4. Do you ever get lost?

Never
Sometimes
Frequently

5. Have other people noticed (commented) that you have problems with your memory?

Never
Sometimes
Frequently

6. Do you find it hard to concentrate/pay attention?

Never
Sometimes
Frequently

7. Do you ever have difficulty understanding what others are saying to you?

Never
Sometimes
Frequently
8. Do you have trouble understanding what you read more than you used to?

Never
Sometimes
Frequently

9. Since the injury, do you have difficulty finding words you want to use or problems remembering the names of objects?

Never
Sometimes
Frequently

10. Do you find yourself doing things without thinking ahead or planning?

Never
Sometimes
Frequently

Social/Interpersonal Activity level

1. What do you do for fun? Patient spontaneously lists:

0 activities
1-2 activities
3-4 activities
5-6 activities
7 or more activities

2. Do you have any hobbies? Patient spontaneously lists:

0 hobbies
1-2 hobbies
3-4 hobbies
5-6 hobbies
7 or more hobbies

3. How often do you do any of these hobbies now?

Everyday
Every other day
Once a week
Once every two weeks
Once every month or less
4. Do you belong to any clubs/organizations/support groups? If so, how many?
0
1-2
3-4
5-6
7 or more

5. How often do you go to club/support group/organization meetings:
Every day
Every other day
Once a week
Once every two weeks
Once every month or less

6. Do you enjoy your hobbies/activities that you do for fun as much as you did before your injury?
Yes (no change)
No (not as much)

7. Do you socialize with your friends as much as you did before your injury?
Yes (has not noticed a change)
No (has noticed a change)

8. If you don’t socialize as much, why not?
Patient states: (check all applicable answers)
They do socialize as much – question irrelevant.
Does not know or “other” response.
Physical reasons given...i.e., decreased mobility, pain, appearance change.
Psychological reasons given...i.e., personality change, nerves, lack of interest.
Neuropsychological reasons ...i.e., can’t remember what people say, can’t think of words in conversation (etc.).
Financial reasons
Changes in Family Interactions Since the Injury

1. Since your injury, have you had an increase in problems getting along with your family?
   - No change
   - Some additional problems
   - Many more problems

2. Do you think anyone in your family believes that your personality has changed since the accident?
   - No
   - Not sure (not discussed)
   - Yes

3. Do you think they understand the problems you have been having since your injury?
   - Yes
   - Not sure (not discussed)
   - No

4. Do people in your family do too much for you? (i.e., do things for you that you feel capable of doing yourself?)
   - No
   - Not sure (possibly)
   - Yes

5. Do you need more help with things from your family? (i.e., do things for you that you cannot do yourself now?)
   - No
   - Not sure (possibly)
   - Yes

6. Are you having more problems with your spouse/significant other/parents now as compared to the time before your injury?
   - No
   - Not sure
   - Yes
Work/School

1. Are you employed/in school?
   Yes (full time)
   Yes (part time)
   No

2. Occupation/school level: __________________

3. What occupation/school grade were you in before the injury? __________________

4. If you are not employed now, are you planning on going back to work?
   Yes (already working)
   Yes (planning on going back)
   Not sure
   Tried to, but quit
   No

5. If you cannot go back to work right now, what problems are preventing you from working?
   Question irrelevant - patient working
   Memory problems
   Language problems
   Motor/Seizures/Other physical problems
   Attention/Concentration problems
   Psychological problems
   Not sure
   Other reason given

6. Did you go back to school or get into a vocational rehabilitation program after your injury?
   Yes
   Thought about it - looking into it
   No
Anger Problems

1. Do you lose your temper easily?

Never
Sometimes
Frequently

2. Do you lose your temper more now than before your injury?

No
Maybe
Yes

3. How often would you estimate you lose your temper nowadays?

More than 1 time per day
Once a day
Once every few weeks
Once a week
Once every 2 weeks
Once every month

4. At its worst, how bad has your temper been since the accident?

No one knew I was angry.
Others knew I was angry, but I didn’t argue.
I argued/screamed/swore.
I was so angry I had to leave the situation.
I threw or smashed things.
I hit someone.
I physically hurt someone (some animal).

5. Who do you lose your temper with?

Spouse  
Employer/teacher
Parents  
Coworker/classmate
Siblings  
Stranger
In-laws/other relatives  
Other
Friend
Neighbor
6. Can you control your temper?

Most of the time.
Some of the time.
None (little of the time).

7. Do you feel you need to learn how to control your temper better?

No – satisfied with control level
Not sure
Yes – not satisfied with current control level

8. In your everyday dealings with people, how often do you expect they will say or do something that will make you angry?

Rare – not often.
Sometimes
Frequently

9. Can you tell when you are going to lose your temper?

Yes – always.
Some of the time.
No – can’t usually tell.

10. Are other people afraid of you or avoid you because of your temper?

No
Sometimes
Yes

11. Do other people try to get you angry on purpose?

No
Sometimes
Yes

12. Should other people be concerned about the way you manage your anger, or are people making too much of this problem?

People are making too much of it...
There is some room for concern.
People have a right to be concerned.
13. How much do you like or dislike having to actually express anger or irritation towards other people?

I don't like to: if I can avoid expressing anger I will.
I don't mind too much; I express anger when I feel I should.
I don't mind at all; if I get angry, people will definitely know.

14. How do you know you are getting angry?

I pick up on physical cues (turn red, shake, get muscle tension, etc.)
I keep thinking angry thoughts.
Both of the above.
Don't know/other response.

15. What is the worst thing that has happened because you couldn't control your anger?
Appendix H

Consent Form

I hereby consent to participate in a study involving the assessment of anger control problems. I understand that I will be taking a number of paper and pencil tests, and that a member of my family (or a designated friend) will be interviewed.

I realize that this information will be held in strict confidence and will be used for research purposes. I further understand that I am free to withdraw from this study at any time.

The University of Mississippi Medical Center has no mechanism to provide compensation for subjects who may incur physical injuries as a result of participating in biomedical research. This means that while all investigators will do everything possible in providing careful medical care and safeguards in conducting this research, there is no way in which the institution can pay for the unlikely occurrence of injury resulting solely from the research itself. We will, of course, provide our best medical treatment to which you are entitled for the illness, if any, for which you consulted us whether or not you participate in this study and whether or not you decide to withdraw from the study.

I have read, understood, voluntarily signed, and have been given a copy of this informed consent statement this ____ day of 19____, at Jackson, Mississippi.

_________________________________________  _______________________________________
Participant's Signature                       Witness' Signature

Principal Investigator:
Kathryn Lawson Kerr, M.A.
Psychology Instructor
Department of Psychiatry and
Human Behavior
University of MS Med Center
2500 N. State Street
Jackson, MS 39216
Tele * 601-984-5804

Human Investigations Committee:
James Achord, M.D., Chairman
University of MS Med Center
Human Investigations Committee
2500 North State Street
Jackson, MS 39216
Tele * 601-984-4540
### Appendix I

**Normative Data for Neuropsychological Tests Administered**

#### Intelligence

<table>
<thead>
<tr>
<th>Test</th>
<th>Mean</th>
<th>SD</th>
<th>Significance</th>
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<tr>
<td>Wechsler Adult Intelligence Scale - R</td>
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<tr>
<td>1. Full Scale IQ</td>
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<tr>
<td>2. VIQ</td>
<td>100</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>3. PIQ</td>
<td>100</td>
<td>15</td>
<td></td>
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<td>4. [VIQ – PIQ]</td>
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#### Memory

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<tbody>
<tr>
<td>Wechsler Memory Scale</td>
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<tr>
<td>1. Logical Memory Immediate*</td>
<td>16</td>
<td>2.95</td>
<td>30-39</td>
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<tr>
<td>2. Logical Memory Delayed**</td>
<td>14</td>
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<tr>
<td>3. Paired Associates Immediate*</td>
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<td></td>
<td></td>
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<td>4. Paired Associates Delayed**</td>
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<td>5. Rey-Osterreith Complex Figure™ (percentile scores listed in table 2)</td>
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Memory Trial

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Normative Data is not presented, as scores were transformed for the purposes of this study. Further information is available upon request.

Appendix I
Normative Data (continued)

Set Shifting and Concept Formation

1. Wisconsin Card Sort test - categories achieved*  4-6
2. Trail Making Test** (for ages 20-39)

Percentiles .................................................................
  90  75  50  25  10

<table>
<thead>
<tr>
<th>Time (seconds)</th>
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<tbody>
<tr>
<td>Path A</td>
</tr>
<tr>
<td>Path B</td>
</tr>
</tbody>
</table>


Language

1. Thurstone's Word Fluency test - cutting score of 45●
2. Aphasia Screening Test - no quantitative norms used.

3. Boston Naming test (for ages 30-39)@  

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<thead>
<tr>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
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<tr>
<td>56.65</td>
<td>2.84</td>
<td>47-60</td>
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</table>


Appendix I
Normative Data (continued)

Psychomotor Performance

1. Grooved Pegboard test

Dominant: < 70" = normal; 70-79" = borderline; > 79" = impaired range
Nondominant: < 75" = normal; 75-84" = borderline; >84" = impaired range

2. Finger Tapping

Dominant: Mean = 55.87 ± 4.91 for males
Mean = 51.08 ± 4.35 for women

3. Dynamometer

Dominant: Mean = 48 SD = 9
Nondominant: Mean = 44 SD = 7


*Source: Paul Malloy, Ph.D.
Butler Hospital
345 Blackstone Road
Providence, Rhode Island 02906
Curriculum Vita

Kathryn Lawson Kerr

Personal Data:

Birthdate: November 16, 1954
Birthplace: St. Louis, Missouri
Social Security #: 469-66-9472
Marital Status: Married
Home Address: 2213 Cherry St
               Vicksburg, MS 39180
Office Address: University of Mississippi Medical Center
               Department of Psychiatry and Human Behavior
               Psychology Division
               2500 North State Street
               Jackson, Mississippi 39216-4505
               Phone: (601) 984-5804

Education:

B.A. (1975) Washington University
      St. Louis, Missouri
      Major: Psychology
      Major Professor: Martha Storandt, Ph.D.

M.A. (1979) University of Minnesota
      Minneapolis, Minnesota
      Major: Psychology
      Major Professor: Manfred Meier, Ph.D.

Ph.D. (1986) Louisiana State University
           Baton Rouge, Louisiana
           Major: Clinical Psychology (medical subspecialty)
           Minor: Behavioral Neurology
           Major Professor: Phillip Brantley, Ph.D.
           Minor Professor: Arthur Riopelle, Ph.D.
Internship:
University of Mississippi Medical Center
Division of Psychology
Department of Psychiatry and Human Behavior

Rotations:
Neuropsychology (6 months) Jeff Webster, Ph.D.
Forensic Psychology (3 months) William Johnson, Ph.D.
Behavioral Medicine (3 months) John Martin, Ph.D.

Current Position:
Psychology Instructor
University of Mississippi Medical Center
Division of Psychology
Department of Psychiatry and Human Behavior

Past Positions Held:

<table>
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<tr>
<th>Clinical Experience</th>
<th>Supervisor</th>
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<tr>
<td>Extern Supervisor</td>
<td>Phillip Brantley, Ph.D.</td>
<td>August, 1982-</td>
</tr>
<tr>
<td>General Consult Service</td>
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<td>August, 1983</td>
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<td>Psychology</td>
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<td>Earl K. Long Memorial Hospital</td>
<td></td>
<td></td>
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<tr>
<td>Baton Rouge, Louisiana</td>
<td></td>
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<tr>
<td>Extern Supervisor</td>
<td>Phillip Brantley, Ph.D.</td>
<td>May-</td>
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<tr>
<td>Pediatric psychology</td>
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<td>Earl K. Long Memorial Hospital</td>
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<td>Baton Rouge, Louisiana</td>
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<tr>
<td>Extern Supervisor</td>
<td>Phillip Brantley, Ph.D.</td>
<td>August, 1982</td>
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Psychology Extern
Behavioral Medicine Unit
Earl K. Long Memorial Hospital
Baton Rouge, Louisiana
Phillip Brentley, Ph.D.
August 1981 - May 1982

Psychology Extern
The Psychology Group and the Runnymede Clinic
Baton Rouge, Louisiana
Darlyne Nemeth, Ph.D.
August 1980 - May 1981

Private Practice
Francis Pirozzolo, Ph.D.
1979

Neuropsychology Clerk
University of Minnesota
Minneapolis, Minnesota
Manfred Meier
January, 1976 - May, 1979

Psychology Technician
Rochester State Hospital
Rochester, Minnesota
John Hawkinson, Ph.D.
1974 (summer)

Research Experience

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Psychology Department
Louisiana State University
Baton Rouge, Louisiana
Ralph Dreger, Ph.D.
August, 1980 - May, 1981

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University of Minnesota
Minneapolis, Minnesota
Manfred Meier, Ph.D.

Research Assistant
Department of Neurology
Hennepin County Medical Center
Minneapolis, Minnesota
Gail Risse, Ph.D.
September, 1977 - September, 1978
Joint Clinical/Research Positions Held

Health Science Specialist
Departments of Neurology and Geriatrics (GRECC)
Veterans Administration
Medical Center
Minneapolis, Minnesota
David Webster, M.D.
Francis Pirozzolo, Ph.D.
James Mortimer, Ph.D.

Accepted Appointment
Departments of Neurology and Geriatrics (GRECC)
Veterans Administration
Medical Center
Minneapolis, Minnesota
Francis Pirozzolo, Ph.D.
James Mortimer, Ph.D.

Teaching Experience
Instructor
Department of Psychiatry and Human Behavior
Psychology Division
University of MS Medical Center
Jackson, Mississippi
Ron Drabman, Ph.D.
Ellie Sturgis, Ph.D.
William Johnson, Ph.D.

Teaching Assistant
Allied Health Sciences Department
University of Minnesota Medical School
Minneapolis, Minnesota
Manfred Meier, Ph.D.
Workshops Presented

Neuropsychological Assessment Workshop
Presented with Jeffrey Webster, Ph.D.
Mississippi Psychological Association October, 1983

Psychological Presentations of Physical Illness
and Related Neuropsychological Deficits
Presented at the East Louisiana State Hospital
Jackson, Louisiana July, 1983

Poster Presentations:

Anger Control Problems in Individuals After Closed Head Injury:
the Need for Integrated Assessment.
Association for the Advancement of Behavior Therapy
Chicago, Illinois November, 1986

Anger Control Problems in Individuals with Cerebral
Dysfunction. A New Assessment Approach and Exploration
of a Clinical Model.
Kathryn Lawson Kerr, Virginia Goetsch, Jeffrey Knight, Glenda
Cottam and Jeffrey Webster
Association for the Advancement of Behavior Therapy
Philadelphia, Pennsylvania November, 1984

Neurolinguistic Features of Subangular Alexia
International Neuropsychological Society
Francis Pirazzolo, Kathryn Lawson Kerr, John Obrut, Gerald Morley, Jim Haxby, and Sandra Lundgren
San Francisco, California February, 1980

Invited Presentations:

The Role of the Neuropsychologist in Assessment and Rehabilitation.
The Addie McBride Center For the Blind: Tri-State Regional Workshop
Jackson, Mississippi (Videotape available) November, 1984
The Neuropsychologist's Role in the Assessment of Cerebrovascular Accidents
Stroke Management Update Conference
Veterans Administration Medical Center
Jackson, Mississippi  
September, 1984

Assessment of Suicidal Intent
Behavioral Medicine Conference
Earl K. Long Memorial Hospital
Baton Rouge, Louisiana  
July, 1982

The Role of Neuropsychologists in Hospital Settings.
Behavioral Medicine Conference
Earl K. Long Memorial Hospital
Baton Rouge, Louisiana  
November, 1981

Adjustment to Retirement
Civil Service Employment Program
Minneapolis, Minnesota  
June, 1980

The Porteus Maze Test: An Analysis of Quantitative and Qualitative Errors in Patients with Focal and Diffuse Cerebral Dysfunction
Behavioral Neurology Conference
Veterans Administration Medical Center
Minneapolis, Minnesota  
March, 1979

Visual-Spatial Disorders
Geriatrics and Neurology Service Conference
Veterans Administration Medical Center
Minneapolis, Minnesota  
January, 1979

Information Processing Deficits in Alcoholic Korsakoff Syndrome: A New Look at the Visual Modality. (presented by Dr. Risse)
Gail Risse, Silvia Strauman, and Kathryn Lawson Kerr
International Neuropsychological Society
New York, New York  
February, 1979
Professional Activities:

Journal of Behavioral Assessment: Student Editorial Board (past member)

Professional Memberships (student affiliations):

Southeastern Psychological Association
Association for the Advancement of Behavior Therapy
American Psychological Association
International Neuropsychological Society
National Head Injury Foundation

Publications:


Grant Award:


Dissertation Title:

The relationship between anger control problems and neuropsychological deficits in individuals who have sustained a head injury.

Research Interests:

Neuropsychology

- Neuropsychological deficits present after head trauma
- Psychological sequelae of neurological disorders
- Rehabilitation and recovery of function
- Cognitive and motoric changes in Parkinson's Disease
- Alexia
- Memory

Behavioral Medicine

- Illness Behavior
- Stress and Illness
- Psychological presentations of physical disorders
Behavioral approaches to neuropsychological rehabilitation
Lifestyle factors in cardiovascular functioning

References:

Phillip J. Brantley, Ph.D.
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and
Associate Professor of Medicine
Louisiana State University
Earl K. Long Memorial Hospital
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Jeffrey S. Webster
Associate Professor of Psychology
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V.A. Medical Center
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William F. Waters, Ph.D.
Director of Clinical Training
Department of Psychology
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Baton Rouge, Louisiana 70803
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Ellie Sturgis, Ph.D.
Director, Chemical Dependency Unit
Medical University of South Carolina
Department of Psychiatry and Behavioral Sciences
171 Ashley Avenue
Charleston, South Carolina 29425
Telephone *(803) 577-5011, ext. 260

Ron Drabman, Ph.D.
Director of Clinical Internship Training
University of MS Medical Center
Department of Psychiatry and Human Behavior
2500 North State Street
Jackson, MS 39216
Telephone *(601) 984-5855
DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Kathryn Lawson Kerr

Major Field: Psychology

Title of Dissertation: The Relationship Between Anger Control Problems and Neuropsychological Deficits in Individuals who have Sustained a Head Injury

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

Mary H. Kelley

Frank M. Szeshoom

Antony J. Piccolle

Patricia J. Waiznak

Date of Examination:

November 24, 1986