FAKING NEUROPSYCHOLOGICAL TEST DATA: A FURTHER EXAMINATION OF THE LURIA-NEBRASKA NEUROPSYCHOLOGICAL BATTERY

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CHAPTER I

INTRODUCTION

Closed head trauma is a serious yet relatively misunderstood injury to the brain. Though concussions and contusions occur typically without laceration to the head, both usually cause severe jarring of the brain substance within the skull. Consequently, there is likely to be diffuse damage to the neural tissue not easily detected by merely viewing the injured individual. Those who survive a closed head injury may show little or no impairment of higher cortical functions with minor injuries or a gross deterioration of intellect and personality with more severe damage (Walsh, 1978). However, all closed head injuries, even in cases with associated depressed skull fractures, hemiplegia, aphasia, or other clearly focal signs, result in diffuse and bilateral cerebral damage (Smith, 1981). A few of the possible complications of that damage include brain edema, intracranial pressure, subdural and extradural hematomas, and systemic complications, i.e., hypotension, anemia, and hypoxia (Levin, Benton, & Grossman, 1982).

Because of increased technological sophistication the likelihood of surviving a closed head injury has increased. Studies in both England and the United States have indicated that admission to hospitals for head injury have continued to increase over the past 20 years, but the number of resulting deaths has remained constant (Levin et al., 1982). About one-half of all closed head trauma in adults and adolescents in the

United States is caused by traffic accidents (Annegers, Grabow, Kurland, & Laws, 1980; Kalsbeek, McLaurin, Harris, & Miller, 1980). Age appears to be related to injury with young adults and late adolescents usually injured as occupants of motor vehicles while children and elderly people are usually involved in falls and pedestrian-car accidents (Levin et al., 1982). It has also been found that young adult males, typically 15 to 30 years of age, predominate as victims of closed head injury (Annegers et al., 1980). Those who survive and recover to some extent from closed head trauma will often seek legal action and/or will file insurance claims to receive monetary compensation for their injuries.

In many instances involving litigation or insurance claims, neurological evidence of brain damage can be provided (i.e., X-rays, PET or CAT scans, or surgical procedures) leaving no doubt that the individual should be compensated. However, in situations where weaknesses linger without a clear physical basis, a means to document change which would go beyond the capabilities of the neurologist or neurosurgeon has been needed. Even when there is clear evidence of damage, its effect on the person's ability to function needs to be evaluated. As Luria (cited by Christensen, 1975) noted, the clinical examination of the neurologist concentrating on the disturbances in sensibility, movement, muscle tone, and reflexes reflects the pathology of a very small portion of the cortex and the nervous system, the primary projection areas. But lesions outside those areas, in the secondary and tertiary zones which make up a large part of the cerebral cortex, are unavailable to the technique of the classical neurologist. If a diagnosis is necessary, the neurologist must examine the patient's behavior; he/she must use psychological methods of investigation. With neuropsychological testing, residual effects of closed

head injury which could not be detected by EEG, PET, or CAT scan, for example, and could not be treated by surgery or chemotherapy, can be better identified and evaluated.

Based on these evaluations, psychologists are often required to testify in and outside of court as to the competency of the individual. Without hard neurological evidence, accuracy of diagnosis based on neuropsychological testing increases in importance. When one considers compensation as an incentive for the patient to malinger (appear worse than he/she actually may be), it is necessary for the psychologist to base his/her conclusions on the strongest objective evidence that can be gathered. To facilitate the diagnostic process, some investigators have standardized neuropsychological test batteries. However, little research has been done concerning the benefits these instruments provide in the detection of malingering.

The purpose of the present study is to broaden this research area and explore the possibility of differentiating malingerers from closed head injury patients with the Luria-Nebraska Neuropsychological Test Battery (LNNB; Golden, Hammeke, & Purisch, 1978).

Golden et al. (1978), in developing the Luria-Nebraska Neuropsychological Battery, have attempted to follow Luria's (1973, 1980) philosophy of examining each functional/dysfunctional system closely. Items in a given area of the battery are administered, each item having a very subtle variation to tap the additional skills of the patient. The pattern of deficits of the patient allows for the interpretation of the nature of the patient's difficulty.

The battery is composed of 269 items and can be administered in two to three hours. The items are divided into 11 sections which include:

motor skills, rhythmic and pitch skills, tactile skills, expressive language skills, receptive language skills, reading, writing, arithmetic, memory, visual-spatial skills, and intellectual processes. Based on the scores from each section of the battery, a patient profile can be derived, and the pattern of strengths and weaknesses can be examined. If the profiles are characteristic of specific brain-injured groups (e.g., lateralized or diffuse damage, alcoholics, epileptics), then malingerers might also produce a distinctive profile.

The validity and reliability studies (Golden et al., 1978; Golden, Moses, Fishburne, Engum, Lewis, Wisniewski, Conley, Berg, & Graber, 1981; Golden, Moses, Graber, & Berg, 1981; Lewis, Golden, Moses, Osmon, Purisch, & Hammeke, 1979; Purisch et al., 1978) conducted by the Golden research group to date provide support not only for Luria's theory (Osmon, 1980) but for the standardized battery itself. The research also supports the concept of profiles characteristic of particular kinds of damage (e.g., Purisch et al., 1978; Chmielski & Golden, 1980; Golden, 1979; De Obaldia, Leber, & Parsons, 1981; Osmon, Golden, Purisch, Hammeke, & Blume, 1979). However, many of those outside the Golden research group would take issue with the battery's success (Crosson & Warren, 1982; Delis & Kaplan, 1982; Spiers, 1982, 1981).

Studies have yet to emphasize performance of closed head injury patients on the LNNB; however, it is likely that a diffuse pattern of involvement including, in particular, deficits in the areas of memory, intellectual processes, and language, would be indicated with the amount of impairment varying with the severity of the injury. The present study will attempt to provide some further insights into the use of pattern analysis with the LNNB by comparing patterns of scores produced by head

trauma patients, normal individuals, and malingerers. If performance profiles of brain-damaged groups are as representative as the Golden research team contends, then malingerers may also produce a characteristic profile. Provided the patterns are distinctive, it should be possible to differentiate not only between normals and brain-damaged patients, but also between malingerers and brain-damaged patients.

CHAPTER II

LITERATURE REVIEW

Although much research has been undertaken, there is a lack of comparability, in general, across the area of neuropsychology. Consequently, it is difficult to observe trends among studies and draw conclusions. Few experimenters have utilized the same assessment tools; in fact, the number of different tests used in neuropsychological research is frequently equal to the number of different studies under consideration. While tests for specific deficits, such as aphasia, memory, and visuospatial problems, are abundant, there are few formalized neuropsychological test batteries (Lezak, 1983) because many neuropsychologists still prefer to assemble their own test batteries to meet the unique needs of each of their patients (Lezak, 1983; Luria, 1980; Smith, 1975).

Testing for specific weaknesses may be an effective method for the experienced clinician; however, it does not provide much guidance in further clarifying brain/behavior relationships. Thus, most research with non-standardized, uniquely suited assessment devices may serve only to increase the confusion in the area. Additionally, when called upon to testify, recommend, or diagnose residual deficits from possible brain damage, the clinician, without a standardized battery, must rely on his/her intuition. The clinician may be accurate, but intuition typically does not provide much support for an insurance claim or legal action.

Ironically, the most recent development of a standardized neuropsychological test battery was based on the work and philosophy of a Russian neuropsychologist who felt clinical judgment and intuition were the best ways to evaluate and test a brain-injured patient.

Indeed, the qualitative evaluation of localizing and identifying brain dysfunction within each patient served as the foundation of Luria's (1973, 1980) neuropsychological technique. He developed sets of procedures often based on intuition for the assessment of each individual. But it was not this intuition that made Luria's work so valuable; it was his ability to "explain complex brain processes in terms of a simple and comprehensive model that could account for the mass of seemingly unrelated data that have been collected in neuropsychology" (Golden, Hammeke, Purisch, Berg, Moses, Newlin, Wilkening, & Puente, 1982, p. 1).

Luria's (1973, 1980) theory of brain function is based on his notion of "functional systems." He conceptualized the brain as a mass of specific areas working together within a particular functional system, each complimenting one another's function in the production of human behavior. Each brain area in the functional system was necessary but not sufficient to produce the behavior. Thus, when one brain area in a system was affected, the entire system also would be affected. The only loss which could be observed would be that of the entire functional system; the damage was evident, but exactly what brain area was damaged was not.

Luria's neuropsychological investigation was predicated on the identification of the damaged area through "syndrome analysis" (Luria, 1973, 1980). Luria wrote (cited by Christensen, 1975, p. 17) "each of the (brain) areas makes a highly specific contribution to ensure the operation of the functional system." If a specific area is damaged, it would

be expected that every functional system cooperating with that area would be affected as well. Luria (Christensen, 1975, p. 17) saw the aim of neuropsychology, in the diagnosis of focal lesions, as the analysis of "the disturbances of the higher cortical processes or the specific traits of a person's psychic activity."

Luria (1980) emphasized that to identify the specific deficits of the patient, it was not necessarily the patient's ability to perform the task which was important, but the way in which he went about solving it. In this regard, he administered items in a number of different ways in order to flesh out the exact nature of the deficit. This "syndrome analysis" would identify a unique pattern of deficits and enable interpretation of the exact nature of the patient's difficulty.

Luria was able to thoroughly explore his patients' problem areas, but his methods gained little recognition in the United States. He stressed the importance of flexibility in testing a patient and would tailor the tests in his neuropsychological investigation to the individual demands of each patient's difficulty. Although this technique was effective for Luria, it has made it difficult for others to use the test and derive any meaning from the results. There was little information as to how to interpret any results because Luria used no quantification of his measures and, finally, there had been little investigation into the reliability and validity of Luria's procedures. As Reitan (1976) pointed out, there has been no evidence, except Luria's word, that his methods have any validity at all.

Based on Luria's (1973, 1980) theory of brain function and his test administration techniques, the Luria-Nebraska Neuropsychological Battery (LNNB) was designed as a standardized and comprehensive tool for assessing

brain injury which would overcome the limitations of Luria's procedure but would not lose its qualitative aspects (Golden, Hammeke, & Purisch, 1978). The LNNB, being a theoretically supported test, has the potential for great utility. Osmon (1980) has emphasized that with each of the basic parts of a complex function identified, the differing qualities of the function can be evaluated individually leading to the specification of the dysfunctional part of the system in accordance with the way in which the brain theoretically performs the behavior.

Standardization of Luria's neuropsychological investigation began shortly after the work of Christensen (1975) became available. She published materials useful for the administration of the test battery which she obtained partially through her work with Luria and partially through her own experience. Christensen had a specific terminology with general instructions and cards which provided the actual foundation for the LNNB.

Using Christensen's work as a guide and keeping with the spirit of Luria's concept of syndrome analysis, Golden et al. (1978) developed items for the standardized battery. The initial study (Golden et al., 1978) was an attempt to establish validity and scoring reliability for the original 285 LNNB items. Fifty medical controls and fifty neurological patients were administered the test battery. Nearly 90 percent of the items differentiated between brain injured and normal patients and, in all cases, the neurological patients performed more poorly than normal patients.

Golden and his colleagues tried several different systems of scoring the items and emphasized the need for a summary scoring system which was developed later (Purisch, Golden, & Hammeke, 1978). The final method

of scoring is similar to that of Luria's ratings (Golden et al., 1980). Most of the items are scored as either 0, 1, or 2; "0" represents normal performance (no dysfunction), "1" is characteristic of a mid-range performance seen in both normals and brain-injured individuals (a weak degree of dysfunction), and "2" is suggestive of the performance of a brain-injured person (a strong amount of dysfunction). Scoring criteria include accuracy, frequency and adequacy of response, number of errors, time for performance, trials until correct performance, and number of items completed (Purisch et al., 1978). The scoring method varies based on the nature of the item and the quality being assessed.

This initial project also served as a check on the reliability of the scoring system (Golden et al., 1978). Pairs of independent examiners rated the performance of five patients on all items and achieved interrater agreement ranging from 92 to 98 percent. However, as the authors later pointed out, the percentages were likely to be inflated due to the dichotomous nature of almost one-third of the test items (scored as either "0" or "2"). In patients with chronic, unchanging organic conditions, Golden, Berg, and Graber (1982) found test-retest reliability over the 14 LNNB scales to range from 0.77 to 0.96 with an average of 0.88 and a test-retest interval average of 167 days. However, the reliability of measuring specific types of functions within scales has not been included in any studies to date (Crosson & Warren, 1982).

In addition to the 11 basic scales, 3 more scales were developed based on the already existing items (Purisch et al., 1978). The 32 items considered as the most effective indicators of brain damage when scored as "2" became the Pathognomonic Scale (Purisch et al., 1978). The other two scales are the sum of all items which require right- or left-handed

motor or tactile function and were labeled Right and Left Hemisphere Scales, respectively (Purisch et al., 1978).

Following the initial development of the standardized battery, Golden and his associates sought to demonstrate the ability of the battery to differentiate psychiatric patients from those with brain damage, to develop scales to measure laterality and localization of injury, and finally, to examine the effects of age, education, medication, chronicity, intelligence, and severity of injury on performance (Golden et al., 1978).

Purisch et al. (1978) found that the 14 summary measures when used as a pattern of performance could distinguish brain damaged from schizophrenic patients with 88 percent diagnostic accuracy. More recently, the LNNB has been shown to successfully differentiate among schizophrenics with and without brain damage (Puente, Heidelberg-Sanders, & Lund, 1982) and with and without ventricular enlargement (Golden, MacInnes, Ariel, Ruedrich, Chu, Coffman, Graber, & Bloch, 1982).

In the pursuit of developing validity for the capacity of the test battery to localize affected brain area(s), the results of the first study of localization suggested that each area of each hemisphere can be distinguished by specific patterns of scores (Lewis, Golden, Moses, Osmon, Purisch, & Hammeke, 1979). Twenty-four right-hemisphere and 36 left-hemisphere patients were divided into eight groups (left frontal, left sensory-motor, left parietal-occipital, left temporal, right frontal, right sensory-motor, right parietal-occipital, or right temporal) depending on hemisphere involved and location within that hemisphere as determined by neurological evidence, i.e., CAT scan, surgical results, and/or angiogram. It was found that the 14 summary scores when used together produced profiles which supported clinical patterns and heretofore had

been identified only theoretically. For example, right frontal patients performed in such a way as to produce deficits theoretically considered to be characteristic of right frontal patients. Overall comparisons among the eight groups and a normal control group resulted in 88 of 112 possible comparisons being significant. A far smaller number of significant differences were produced when all possible comparisons of the eight groups were examined without the inclusion of the normal control group in the analysis. Of the 392 comparisons, 79 were significant. The authors attribute this result to the small sample size of each group. Lewis et al. (1979, p. 1010) suggest that the profiles appear to show "configural discriminations between these groups."

Attempts to develop specific scales to diagnose lateralization of injury have also been made. McKay and Golden (1979a, 1979b) created left hemisphere (L*), right hemisphere (R*), and eight localization scales. They found the empirically derived hemisphere scales could lateralize the lesion accurately (by choosing the higher of the two scales) 87 percent of the time and the eight localization scales could correctly classify specific areas (again by choosing the highest of the scales) 88 percent of the time (McKay & Golden, 1979b).

The authors were quick to point out problems with the scales (the scales were based on small samples and numerous *t* tests were performed) and that cross-validation was necessary to lend support to their findings (McKay & Golden, 1979a, 1979b). Since then a cross-validation was undertaken which suggests that the eight localization scales tend to be better for lateralizing injury than the actual hemisphere scales (Golden et al., 1981). The localization scales accurately lateralized the injury for 82 of the 87 subjects while the empirically derived lateralization

scales correctly classified only 68 of the 87. Additionally, lesioned areas were correctly localized in 65 of the 87 cases and the affected quadrant was correctly identified in 73 of the 87. Extensive work on individual item interpretation has recently been completed (Golden et al., 1982). McKay and Golden (1981) have also derived numerous additional scales based on factor analysis of the items.

In normal individuals, scores of the LNNB will vary significantly with both age and education (Golden et a., 1980). To control for the performance differences which could be produced merely by differences in age and education, Golden et al. (1980) developed a regression equation which created a corrected baseline of performance for each individual.

Golden et al. (1980), in examiming the performance of 60 brain damaged patients with differing educational levels, found a score cutoff of more than 10 points above baseline accurately identified 86.6 percent of the brain damaged patients and 90 percent of the control subjects. This cutoff of 10 points above baseline has been termed the critical level which is generally used in clinical practice, and any LNNB score above it is considered abnormal. If two or more scales, with the exception of Arithmetic, Writing, Left Hemisphere, and Right Hemisphere, are above the critical level, it is likely the patient is brain damaged (Golden et al., 1980). In a sample of 120 subjects with confirmed brain injuries (diagnosis by medical tests, EEG, CAT scan or angiogram), 109 were correctly identified using the above mentioned criterion.

The LNNB is, of course, not without its critics (Lezak, 1983; Crosson & Warren, 1982; Delis & Kaplan, 1983, 1982; Spiers, 1982, 1981). Even Christensen (1975, p. 26) noted that "static standardized techniques are entirely discouraged." Crosson and Warren (1982), in a critique of the

use of the LNNB for the assessment of aphasia, cited the limited utility and validity for examining functional deficits, because the LNNB was designed without consideration for the construct validity of the scales. Scaled scores have little meaning for quantifying specific deficits because of the degree of heterogeneity within each scale (Delis & Kaplan, 1983). Crosson and Warren (1982) also pointed out that even the nonlanguage scales contain a strong verbal component. However, this could be characterized as consistent with Luria's intense interest in language and its role in the regulation of behavior (Shelly & Goldstein, 1982).

More recently, investigators outside the Golden research group have found neither the 14 summary scales nor the localization and lateralization scales to be particularly effective for determining laterality of brain damage. Sears, Hirt, and Hall (1984) tested 40 male VA patients who were assigned to one of four groups based on neurological evidence (i.e., computerized tomography scan, blood-flow studies, brain scan, and neurosurgery reports). Brain-damaged patients were classified as having unilateral left or right hemisphere lesions or bilateral diffuse brain damage. The fourth group was a medical control group without neurological involvement. They found that, for most scales, differences in mean group performances were between brain-damaged and non-brain-damaged subjects. The researchers concluded that their results supported the conclusion of Delis and Kaplan (1983) that the LNNB is limited in the identification of specific deficits because of the methods used in construction of the battery.

Golden has gone to great lengths to defend the heterogeneity of the scales by emphasizing individual itme interpretation (Golden et al., 1982). The LNNB could be considered a series of 269 independent tasks,

each with its own meaning (Golden, Ariel, Moses, Wilkening, McKay, & MacInnes, 1982) which is seen as in concert with Luria's concept of functional systems. Neither the scales nor the items should be seen as "pure" (measuring only a single skill or ability) because of Luria's emphasis on the analysis of how a patient performs an item or pattern of items; it is the qualitative nature of the performance which is important (Golden et al., 1982). For example, a brain-injured individual may miss a particular item on the Expressive Speech Scale not because he/she cannot verbalize the sound but perhaps because he/she cannot remember the sound long enough to reproduce it. This qualitative difference must not be overlooked when interpreting the patient's performance.

Spiers (1982, 1981) has been especially critical of the Golden research team on a number of issues. In particular, he has criticized the lack of control of subject characteristics, age, and onset of disorder. Spiers also noted that level of education was dealt with after the fact by analysis of variance and covariance, and there was a general disregard of subject handedness, and drug, alcohol, developmental, or psychiatric histories. This controversy is far from over and the criticism seems legitimate; however, the LNNB has been shown (Golden, Kane, Sweet, Moses, Cardellino, Templeton, Vicente, & Graber, 1981; Kane, Sweet, Golden, Parsons, & Moses, 1981) to be at least as effective in diagnosing brain damage as the more widely used Halstead-Reitan Test Battery (Halstead, 1947; Reitan, 1955).

Golden et al. (1981) and Kane et al. (1981) have compared the Halstead-Reitan to the LNNB. In a subject pool of 108 patients (30 schizophrenics, 48 brain-damaged, and 30 normal controls) tested in California, Oklahoma, and Nebraska, Golden et al. (1981) compared the

major 14 measures of the Halstead-Reitan with the 14 summary scores of the LNNB. The researchers, using a discriminant analysis, found both batteries to be equally effective in identifying brain damage (hit ratio over 85%). There was also a significant relationship between the LNNB scale scores and the Halstead-Reitan measures.

In comparing the diagnostic accuracies of the LNNB and the Halstead-Reitan battery in a mixed psychiatric and brain-damaged sample, Kane et al. (1981) had experienced raters classify the subjects in two separate comparisons. In the first comparison of 45 subjects, the Halstead-Reitan rater correctly identified 35 subjects while the LNNB rater accurately classified 37 of the 45. The second comparison using 36 subjects found the Halstead-Reitan rater to be correct 29 out of 36 times and the LNNB rater to be accurate in 28 instances.

Research examining the relationship between LNNB patterns and specific neurological disorders has just begun in the last several years (e.g., Chmielski & Golden, 1980; Golden, 1979; De Obaldia, Leber, & Parsons, 1981). To date, no work has been done specifically in the area of assessing the residual deficits of closed head injury with the LNNB, but investigators have compared the performance of those individuals suffering from right or left hemisphere damage with performances of those sustaining diffuse or bilateral damage (Osmon, Golden, Purisch, Hammeke, & Blume, 1979). Patients with injuries to either the right or left hemisphere or with diffuse damage were assigned to their respective groups based on definitive neurological evidence (i.e., CAT scan, surgery, EEG, angiogram, pneumoencephalogram). Using discriminant analysis of the 14 summary scale scores, 59 of the 60 patients were correctly identified. Overall, left hemisphere lesioned patients performed worst while those

with right hemisphere lesions performed best. The patients with diffuse damage, including those with head trauma, performed in between the two extremes.

It is apparent, at least from the research completed so far, that the LNNB is a valid and reliable instrument for detecting the presence of brain injury (Golden et al., 1978, 1981, 1982; Puente et al., 1982; Purisch et al., 1978). It has also been found useful for the specific localization and lateralization of lesions and in separating focal from diffuse injuries (Golden et al., 1981; Lewis et al., 1979; McKay & Golden, 1979a, 1979b). Some research with the LNNB suggests its utility with neurological diseases, such as alcoholism, epilepsy, and multiple sclerosis (e.g., Chmielski & Golden, 1980; Golden, 1979; De Obaldia et al., 1981). However, it is likely that no published research to date has singled out closed head injury patients for study because head trauma rarely produces focal lesions and would likely not be of interest in studies attempting to localize injury.

In an attempt to broaden research in the area, the present investigation will examine the performance of closed head injury patients on the LNNB. Due to the diffuse involvement of neural tissue, the patterns of performance would probably show a wide range of deficits. Although the profile of a closed head injury patient may not be as striking as, for example, that of a patient with a focal lesion to the left hemisphere, closed head trauma introduces an interesting variable. Because it is typically caused by an accident (Annegers et al., 1980; Kalsbeek et al., 1980), there is the possibility of malingering.

People who are seeking compensation for their injuries from insurance companies or through litigation may attempt to look more severely

disturbed than they actually are to insure monetary gain. This is far from a recent development; as early as 1888, deficits resulting from railway injuries were questioned based on the significance of the desire for compensation (Strauss & Savitsky, 1934). A question which arises at this point and, indeed, the focus of the present study is, could the LNNB differentiate between brain-damaged individuals and malingerers?

Personality research has often emphasized the susceptibility of its measures to faking and, therefore, provides some guidance for examining the possibility of malingering on a neuropsychological test. Holden and Jackson (1981) investigated the relative usefulness of subtle versus obvious scales with persons who have been instructed to "fake good" or "fake bad" on the Personality Research Form (PRF), Form E, and have been informed as to the exact nature of the test. They found faking effects for all scales and that with the "correct" response set, it could be possible to distort self-presentation. The investigators found they were able to detect faking using the Desirability scale of the PRF. It was also apparent that scale subtlety was not a determining factor of the subject's ability to fake; it was not immune to distortion.

In another study of test susceptibility to faking, Redmore (1976) found that on a sentence completion test of ego development, subjects were able to decrease their ego level test scores but were not able to successfully increase them. She noted that only with intensive study of ego development could a person actually raise test scores.

By far, most of the research performed in the area of response sets (faking) has been with the Minnesota Multiphasic Personality Inventory (MMPI; Dahlstrom, Welsh, & Dahlstrom, 1975). In fact, the MMPI was designed as a structured personality test which would detect faked responses

because, as Meehl and Hathaway (1980) pointed out, susceptibility to "faking" or "lying" is one of the greatest problems with the structured test.

Through their work with the MMPI, Meehl and Hathaway (1980) developed the L, F, and K scales which, theoretically, should correct for and allow detection of faking. This development provided many research opportunities. If the scales could aid in the detection of faking, then accuracy in the diagnosis of the presence/absence of a psychiatric disorder could increase.

Although a large portion of the MMPI response set literature has been devoted to "faking good" or socially desirable responding (e.g., Wales & Seeman, 1972, 1968; Wiggins, 1966; Walker, 1962), it is the research concerning malingering or "faking bad" which is more pertinent to the present study.

Differing outcomes have been reported by investigators studying the detection of malingering. Gough (1946) has suggested that malingered profiles may be detected by identifying odd patterns of T-scores, but Hunt, Carp, Cass, Winder, and Kantor (1948) found this to be of little use in diagnosis. Meehl and Hathaway (1980) reported that psychology students instructed to "fake bad" will obtain F scores greater than 15 (most normals score between 2 and 4). Gough (1946) found that subjects skilled in psychology and psychiatry (3 psychiatrists, 3 clinical psychologists, 3 psychiatric social workers, 2 personnel consultants) could not successfully simulate either a neurotic or psychotic profile and that the F and K scales allowed for this detection. Hunt (1948) found the F score to increase in court-martialed prisoners instructed to "fake bad" and the K score to decrease when instructed to "fake good." F scores were found to be helpful in identifying negative malingering and a

combined L and K score was found to be useful for revealing positive malingering by Cofer, Chance, and Judson (1949). Anthony (1971) found his subjects (40 United States Air Force male clients) to be more sophisticated in their malingering. The mean exaggerated profile was lower in elevation and had less of a positive skew than the saw-toothed pattern produced by normal, psychologically skilled subjects attempting to malinger. Branca and Podolnick (1961) found that subjects who took the MMPI under hypnotic suggestion of anxiety produced valid profiles, whereas those same subjects told to fake anxiety could not produce valid profiles.

Though the results gathered suggest different uses of the validity scales in identifying faked profiles, it seems clear that malingering can be detected by the MMPI regardless of how psychologically sophisticated the malingerer may be. If this is the case with personality measurement, then how likely would it be for a person to be identified as faking a brain injury? Very little work has been done in this area for a number of reasons, but it is likely due to the individualization of most neuropsychological assessment techniques. The experienced clinician can strongly attest to his/her ability to detect malingering, but without standardized tests and their accompanying normative data with which to compare patients' results, how strongly can those judgments be supported? For this reason, the present investigation has selected the LNNB for study. It is a standardized tool with a great deal of data supporting its reliability and validity for discriminating brain-damaged from non-brain-damaged individuals, but no work has examined its ability to detect faking. In fact, there has been almost no research conducted with regard to faking on neuropsychological test data.

In introducing their own study, Heaton, Smith, Lehman, and Vogt (1978, p. 893) wrote, "no research has shown whether malingerers' neuropsychological test scores can be distinguished from those of nonmalingering brain damaged patients." Heaton et al. (1978) compared the performances of volunteer malingerers with non-litigating head-injured patients on the MMPI, the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955), and the Halstead-Reitan battery (Halstead, 1947; Reitan, 1955). The volunteer malingerers were instructed to pretend they had suffered head injuries in accidents caused by other individuals. They were also told to consider themselves involved in legal action which would determine their financial compensation and to appear as severely disabled as possible without obviously faking. Technicians "blind" to the experiment administered the test batteries and 10 neuropsychologists provided independent "blind" ratings as to whether they thought the protocol was malingered. The authors pointed out that making a determination of faking based on one or two poor test scores would be very difficult for a clinician, but

> a consideration of the pattern of strengths and deficits shown on more comprehensive testing may make such discrimination possible; that is, to fool the clinician on the test battery, the malingerer must show a pattern of results that is similar to patterns of scores earned by head injury patients with real deficits (Heaton et al., 1978, p. 894).

They found overall impairment of the two groups to be equal, but patterns of strengths and weaknesses were different. The malingerers did especially poorly on motor and sensory tests but did relatively well on several of the cognitive tests most sensitive to brain dysfunction. Additionally, the malingerers' profiles on the MMPI showed severe disturbance. The likelihood of expert raters to correctly classify malingerers and nonmalingerers based only on test results as opposed to additional useful

information (i.e., "details of patients' injuries, findings of neurological clinical and laboratory tests, how the recovery periods had gone, how well the patients had done in life prior to their injuries, behavioral observations made by the neuropsychological technicians and even some test data," Heaton et al., 1978, p. 899) ranged from chance-level to about 20 percent above chance. Discriminant function analysis correctly classified 100 percent of the subjects based on the neuropsychological test results and 94 percent based on the MMPI results.

The investigators then used the discriminant functions with a larger sample (84) of head injury patients not included in the formula development but who had been clinically evaluated. Half of those patients were known to be involved in litigation, either civil or criminal, and had reason to appear more seriously disturbed and/or gave clinical evidence of doing so. The other half of the patients were not involved in any court action and were considered to have put forth a reasonable amount of effort during testing. Of the 42 patients involved in legal action, 27 were classified as malingerers using one or both formulas. Only 11 of the 42 patients who had no reasons to exaggerate were identified as malingerers by one or both formulas. Those subjects obviously faking and patients involved in legal action were significantly more likely to be classified as malingerers by the formulas.

They concluded that based on the statistical analysis, group differences in patterns of neuropsychological test data and MMPI scores are sufficiently reliable to allow for prediction of group membership (Heaton et al., 1978). The authors emphasized the intrinsic susceptibility of neuropsychological tests to faking along with the need for further research into patterns of performance characteristic of malingerers. The

authors suggested this goal would more likely be achieved through the use of standardized and comprehensive neuropsychological testing rather than through nonstandardized, briefer clinical examinations. That the clinicians were not very successful in separating malingerers from nonmalingerers lends support to the utility of the standardized test battery. A standardized instrument can be statistically analyzed and characteristic patterns of performance can be identified. Had more work been completed in this area, perhaps, the clinicians would have been more accurate in their predictions.

Statement of the Problem

The diagnosis of residual deficits from closed head injury has taken on more practical importance in recent years due to the increased chance for survival from head trauma. In addition to the neurological and/or neurosurgical procedures necessary for diagnosis, there is often a need for neuropsychological testing in instances where it is not obvious how an individual's life will be affected by the injury. These instances could include patients who have required little or no hospitalization and who have recovered sufficiently to resume normal activities but who have not recovered to a level of premorbid functioning. With testing, deficits can be identified and evaluated as to the type of impact they could have on the patient's future.

While the patient and his/her family are obviously interested in the final recovery, there are often other parties involved. Frequently, there is legal action necessary because of the high probability that the injury was due to an accident (Annegers et al., 1980; Kalsbeek et al., 1980). There is also a more likely possibility of involvement of an

insurance company because the patient will probably desire compensation for his/her injury and will file a claim. A valid question then arises when a neuropsychologist is asked to evaluate the patient's deficits. Is the assessment an accurate evaluation or is the patient attempting to appear more severely disabled in order to gain as much compensation as possible? If the patient is "faking bad" or malingering, how likely is it that the neuropsychologist would be aware of it?

Detection of malingering on psychological tests, such as the Minnesota Multiphasic Personality Inventory (MMPI), has been shown to be possible by examining the validity profile (e.g., Branca & Podolnick, 1961; Cofer et al., 1949; Hunt, 1948; Meehl & Hathaway, 1980) and the pattern of scores from the clinical scales produced by the malingerer (e.g., Anthony, 1971; Gough, 1947, 1946). However, little research has been undertaken with respect to faking neuropsychological test data.

Heaton et al. (1978), in the only published research to date, explored the likelihood of detecting believable deficits on the Halstead-Reitan Test Battery, the Wechsler Adult Intelligence Scale, and the MMPI. Using "blind" ratings, 10 neuropsychologists were able to correctly identify malingerers from nonmalingerers 50 to 70 percent of the time. However, the discriminant function analysis correctly classified 100 percent of the patients in both groups based on the neuropsychological test results and 94 percent based on the MMPI results. With a larger sample, 64.3 percent of the patients in both groups were correctly identified using the discriminant functions derived in the initial stages of the investigation.

These tests were chosen for the Heaton et al. (1978) study because of their psychometric properties; they have normative data and have been

shown to be valid and reliable. The Luria-Nebraska Neuropsychological Test Battery (Golden, Purisch, & Hammeke, 1978) has similar qualities. In direct comparison with the Halstead-Reitan Battery, it has been found to be equally effective in differentiating brain damaged from normals and brain damaged from psychiatric patients (Golden et al., 1981; Kane et al., 1981; Shelly & Goldstein, 1982). The LNNB has also been shown to have validity and reliability for detecting the presence of brain damage (Golden et al., 1978, 1981, 1982; Puente et al., 1982; Purisch et al., 1978). Initial research supports its ability to discriminate between lateralized and diffuse injury (Osmon et al., 1979), and the work completed in the development of scales for localization and lateralization of damage seems promising (Golden et al., 1981; Lewis et al., 1979; McKay & Golden, 1979a, 1979b). As the LNNB is a relatively new test battery, many have been critical of its development and subsequent use (Crosson & Warren, 1982; Delis & Kaplan, 1983, 1982; Spiers, 1982, 1981). A recent independent cross-validation of the LNNB found the battery to be effective in discriminating brain-damaged from normal control subjects, but it was relatively ineffective in determining laterality of brain damage (Sears, Hirt, & Hall, 1984).

As few attempts have been made to examine the probability of detecting faked deficits on neuropsychological tests, the present study examined the likelihood of detecting a malingered head injury profile on the LNNB. Specifically, the present study compared the profiles of subjects attempting to fake closed head injury with two samples of closed head injury patients, one younger and one older. The head injury sample was initially divided into two age groups for a number of reasons: a typical head trauma victim tends to be 15 to 30 years of age and the younger

patients would be more comparable in age and education to the available university undergraduate population. Also, an older head trauma victim may react differently to injury (i.e., he/she may be more traumatized by it) and may be chronic. Additionally, differences between groups may be produced because of age, not presence or absence of brain injury; therefore, the split into older and younger groups should allow for detection of real differences rather than artifacts. Finally, the malingering subjects may exaggerate deficits such that they would appear more similar to the older head-injured subjects than the younger head-injured subjects. The performance of the malingerers was compared to that of the closed head injury patient group and to the normal, nonmalingering group. Profiles of normal nonmalingering subjects were compared to profiles of both groups of head injury patients and to the malingered profiles. Finally, the two closed head injury patient samples (younger and older) were compared to one another.

CHAPTER III

METHODOLOGY

Subjects

All patient data were provided by Charles Golden of the Nebraska Psychiatric Institute from a large, diverse population (over 1200) of brain-damaged, psychiatric, and normal, non-brain-damaged individuals. These patients were from various areas of the United States and had been administered the Luria-Nebraska Neuropsychological Test Battery (LNNB). This group included individuals suffering from cerebral vascular accidents, metabolic and toxic diseases, neoplasms, closed and open head injuries, and various psychiatric disorders. Only patients who had sustained closed head injuries without laceration (a total of 32) were used in the present investigation. The college-age head-injury sample (17 to 26 years of age) was composed of 10 males and 3 females, with a mean age of 21.77 years (SD = 3.05) and a mean of 11.85 years (SD = 1.34 of education. The older head-injury sample (27 to 65 years of age) was composed of 15 males and 4 females, with a mean age of 41.37 years (SD = 11.26) and a mean of 11.89 years (SD = 3.41) of education.

The non-brain-damaged subjects were 24 males and 6 female students enrolled in an undergraduate psychology course at a large southwestern university. Subjects received extra credit points for participating in

the experiment. At the time of recruiting, the subjects were told of the general nature of the study and were assured of confidentiality.

Subjects were randomly assigned to one of two conditions, normal or faking, to achieve a ratio of males to females comparable to the closed head injury patient sample (approximately 4 to 1, males to females). The normal (control) group consisted of 11 males and 4 females, with a mean age of 19 years ($\underline{SD} = 1.36$) and a mean of 12.33 years ($\underline{SD} = 0.52$) of education. The faking (experimental) group was composed of 13 males and 2 females with a mean age of 20.13 years ($\underline{SD} = 2.26$) and a mean of 12.87 years ($\underline{SD} = 1.06$) of education.

Materials and Procedure

The Luria-Nebraska Neuropsychological Test Battery (LNNB) was administered to each non-brain-damaged subject individually by the experimenter. The LNNB contains 269 items scored as either 0, 1, or 2 and required approximately two to two-and-one-half hours to complete. The items are divided into 11 basic scales and 3 clinical scales which include the following: motor, rhythm, tactile, visual, receptive and expressive speech, writing, reading, arithmetic, memory functions, intellectual processes, right hemisphere, left hemisphere, and pathognomonic scales.

A research assistant helped with the instructional period of the experiment so that the experimenter did not know beforehand which subjects were taking the test normally and which were malingering. Before entering the experimental room, subjects were required to read and sign an informed consent form (Appendix B) and were screened for a possible history of brain injury using the following three questions:

1. Have you ever been unconscious due to a blow to the head?

2. Have you ever undergone brain surgery?

3. Have you ever had meningitis or encephalitis?

If a particular subject answered "yes" to any of the three questions, he/she was excluded from the study but still received the extra credit points for keeping the appointment. The research assistant then instructed each individual to read the information sheet for the appropriate condition.

Subjects assigned to the normal or control condition received the following information:

> You will be taking the Luria-Nebraska Neuropsychological Test Battery. It will require approximately two to two-and-one-half hours to complete. The test, which measures disabilities that result from brain injuries, will cover a variety of sensory, motor, and cognitive functions. Please respond to all items to the best of your ability.

Subjects assigned to the malingering condition received the following information:

> You will be taking the Luria-Nebraska Neuropsychological Test Battery. It will require approximately two to two-and-one-half hours to complete. The test, which measures disabilities that result from brain injuries, will cover a variety of sensory, motor, and cognitive functions. Pretend you have suffered head injuries in an accident caused by another person or persons. Assume you are involved in litigation to determine how much financial compensation you will obtain from the people responsible for the accident and/or from the insurance companies involved. Imagine that your everyday functioning in and outside of school has been much worse since your accident, that your potential earning power has been substantially reduced, and that you deserve all the money that the courts will allow you. The results of this test will help determine how large your settlement will be, so fake the most severe disability that you can without making it obvious to the examiner that you are faking.

The experimental room contained all testing materials for the LNNB and a testing table with two chairs on opposite sides. The research assistant took the subject to the testing room where the experimenter

conducted the testing session. The experimenter instructed the subject to be seated, insured his/her comfort, and then asked each subject to "attempt to perform exactly as your information sheet suggested." Test administration then began. At the completion of the test battery, the experiment was explained to each subject. All subjects were requested not to discuss the experiment with anyone.

CHAPTER IV

RESULTS

All statistical analyses were performed using the Statistical Analysis System (SAS; SAS Institute, 1982); specifically, PROC GLM, PROC ANOVA, and PROC MEANS were used. Multivariate analyses of variance were based on the Hotelling-Lawley Trace statistic (SAS Institute, 1982).

The mean profiles for the young and old brain-damaged groups were compared using a multivariate analysis of variance of the dependent variables (scale *t*-scores) and were not found to be significantly different, Hotelling T^2, $F(24,7)$, $p < 0.63$. This result did not support the assumption that the older and younger brain-damaged patients would perform differently. Consequently, the two groups were combined and a single factor (group) multivariate analysis of variance of the dependent variables (scale T-scores) was performed comparing the larger brain-damaged group with the normal and faking groups. Significant differences were found between the three groups, $F(48,70) = 3.14$, $p < 0.001$.

Using Tukey's studentized range (HSD) test, the differences between the performances of the brain-damaged, normal, and faking groups were further examined. Univariate analyses of variance were not performed prior to the post hoc comparisons because as Keselman and Murray (1974, p. 609) have pointed out, "experimentwise power does not change when Tukey tests are used without first having a significant analysis of variance."

The results were mixed in comparing the faking group and the brain-damaged group with 14 of the 24 scale t-score performances differeing (Table 1, Appendix A). As can be seen in Figures 1 and 2, the faking group and the brain-damaged group performed differently on the Receptive Speech, Expressive Speech, Writing, Arithmetic, Memory, Pathognomonic, and R* Scales and on all of the localization scales. With one exception, the faking group did not perform as poorly as the brain-damaged group. Inspection of the mean profile for the brain-damaged group showed nearly all scale t-scores as well above a t-score of 60; this was not the case with the mean profile of the fakers. All scale t-scores for the normal group were far below a t-score of 60 and were significantly different from those of the brain-damaged group. In all instances, the normal group performed better (lower t-scores) than the brain-damaged group (Table II, Appendix A).

The normal and faking groups performed differently on 11 of the 24 scales (Table III, Appendix A). In particular, differences in performance of the two groups were seen on the Motor, Rhythm, Tactile, Memory, Left and Right Hemisphere, Left and Right Sensory-Motor, Left Temporal, L* and R* Scales. However, even in the cases where the normals and the fakers did not differ significantly, the fakers had poorer (higher) scores than the normals. Mean profiles for each group are presented in Figures 1 and 2. The L* and R* Scales are not included in the figures as they have not been cross-validated or properly standardized and are experimental at this time. Mean t-score values for the L* and R* Scales are listed in Tables I, II, and III (Appendix A).

An important consideration at this point concerns the possibility that the differences in performance were produced due to the age and/or educational differences between the three groups. Because any particular

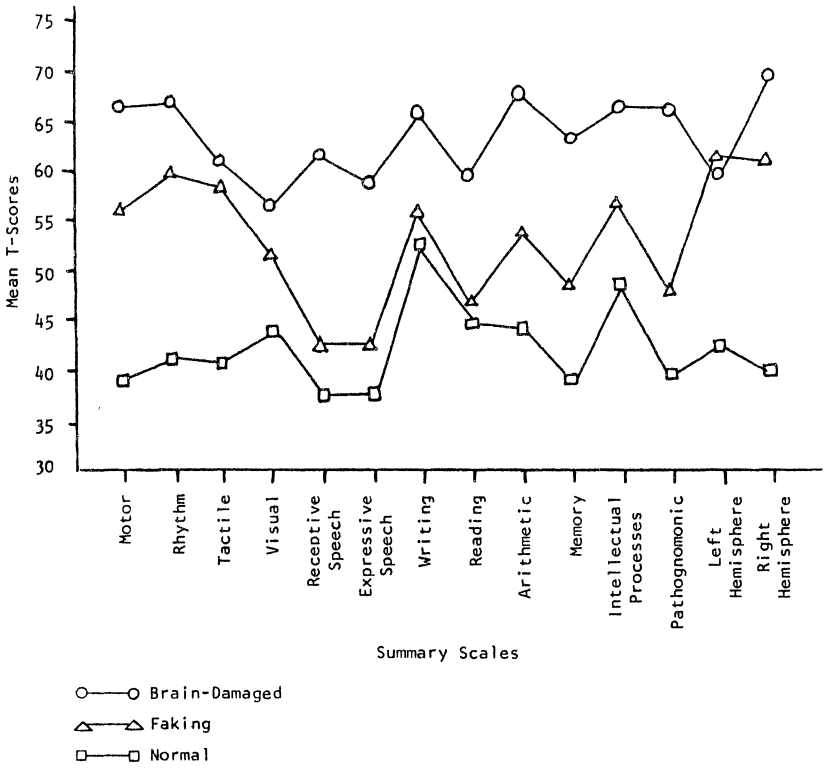

Figure 1. Group Mean Performance on Summary Scales

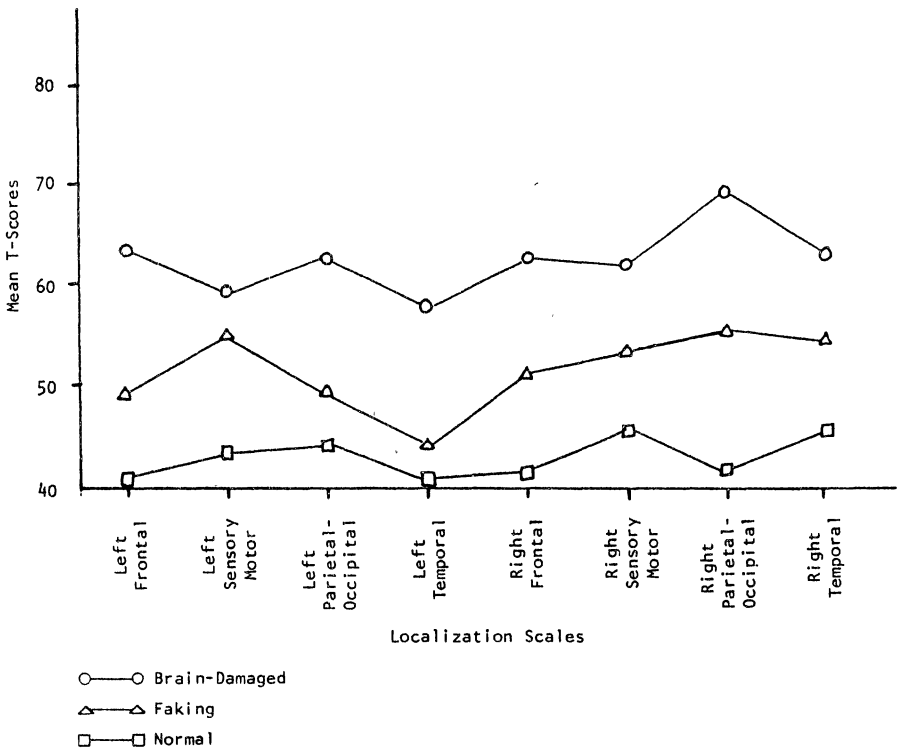

Figure 2. Group Mean Performance on Localization Scales

performance can be affected by age and education, the same baseline of performance cannot be used for every individual. Therefore, a critical level, or adjusted baseline, must be calculated for each subject using the following regression equation (Golden, Hammeke, & Purisch, 1980):

$$\text{Critical Level} = 68.8 + (\text{Age} * 0.412) + (\text{Education} * 1.47)$$

Critical levels of the three groups were then examined using a single factor (group) analysis of variance and were found to differ significantly, $F(2,59) = 4.85$, $p < 0.01$. The mean critical levels for the brain-damaged, faking, and normal groups were 58.78, 55.23, and 56.31, respectively. This being the case, the scale t-scores were then reanalyzed as deviations from critical level. A single factor (group) multivariate analysis of variance of the dependent variables (deviations from critical level) showed significant differences between the groups, $F(48, 70) = 3.00$, $p < 0.0001$. Using Tukey's studentized range (HSD) test, the analysis of the deviations from critical level revealed fewer significant differences between the brain-damaged and faking groups than the analysis of the scale t-scores for the same groups.

The analysis of the deviations from critical level showed the two groups to differ on only 10 of the 24 scales, suggesting that some of the differences produced by comparing the scale t-scores could have been reflecting age and education differences of the groups (Table IV, Appendix A). As shown in Figures 3 and 4, the faking and brain-damaged groups differed on the Receptive Speech, Expressive Speech, Reading, Memory, Pathognomonic, Left and Right Frontal, Left Parietal-Occipital, Left Temporal, and R* Scales.

The analysis of the deviations from critical level revealed that the normal and faking groups differed on 12 of the 24 scales (Table V,

Appendix A). Specifically, the two groups performed differently on the Motor, Rhythm, Tactile, Memory, Left and Right Hemisphere, Left Sensory-Motor, Left and Right Temporal, Right Parietal-Occipital, L*, and R* Scales. Without exception, the normal group had significantly different and lower scores than the brain-damaged group (Table VI, Appendix A). Figures 3 and 4 show the mean profiles using the deviations from critical level measure for the three groups. Again, the L* and R* Scales are not included in the figures but the values for the three groups are available in Tables IV, V, and VI (Appendix A).

Since the experimenter was "blind" to the type of instructions received by each subject, there was an opportunity for subjective identification of subjects performing normally and those faking. Based only on the experience with the subject during the testing, the experimenter was able to correctly identify those faking from those taking the test normally in 23 of the 30 situations (77% accuracy). Four normal subjects were thought to be faking while three faking subjects were identified as normal.

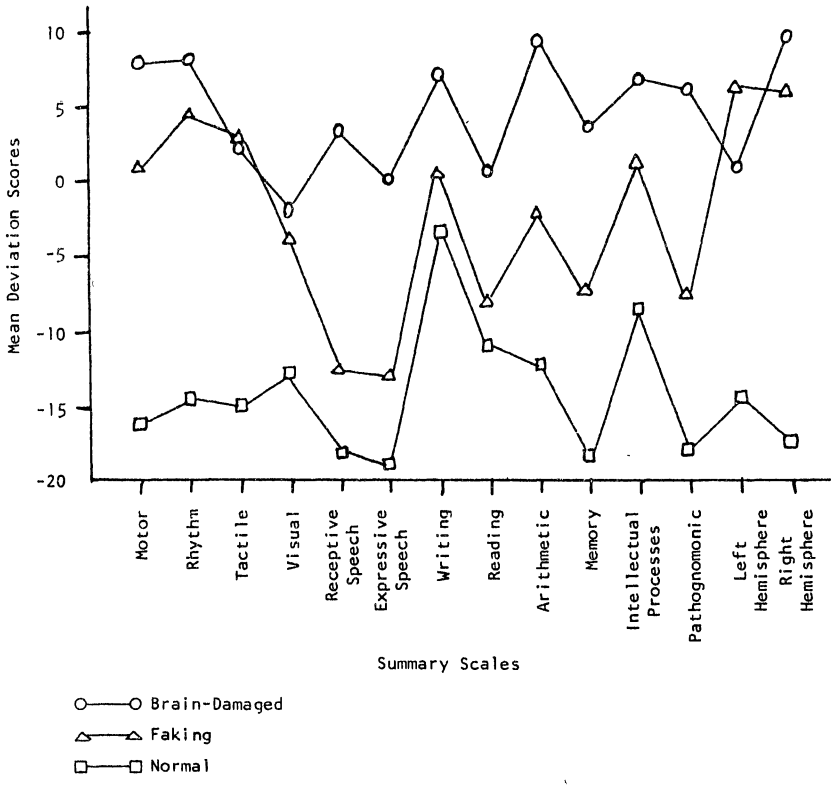

Figure 3. Group Mean Deviations From Critical Level on Summary Scales

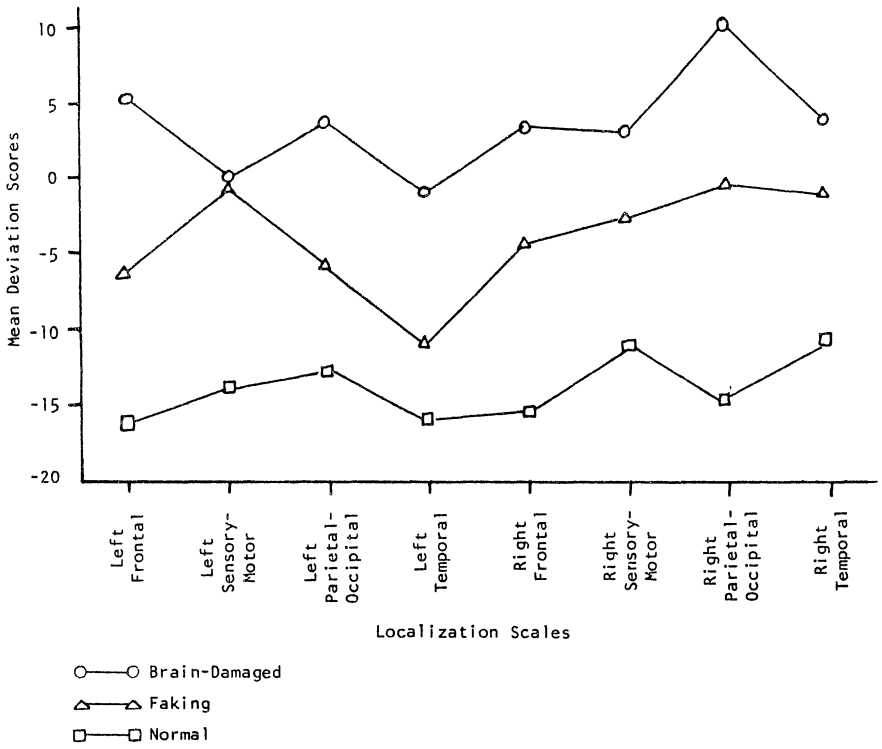

Figure 4. Group Mean Deviations From Critical Level on Localization Scales

CHAPTER V

DISCUSSION

The incentive for individuals to "fake bad" or malinger in order to gain greater monetary compensation for head injuries presents an interesting dilemma for those people making diagnoses or determining whether to grant compensation. When the current number of insurance claims and legal actions is considered, the question of true or "faked" disabilities becomes more of a problem. Hard neurological evidence of lingering damage is often absent or inconclusive and the reliability of the subjective evidence, if any, is called into further doubt. Even when damage is certain, the effects on a person's performance are hard to determine. The present study examined the value of a standardized neuropsychological test battery for assisting in the differentiation of malingerers from brain-damaged patients. In this instance, the Luria-Nebraska Neuropsychological Battery (LNNB; Golden, Hammeke, & Purisch, 1980) was used because of the many claims of its utility in the general diagnosis of brain injury and its ability to designate specific areas of cortical involvement.

The results of the present study provide overall support for the LNNB's validity and ability to discriminate between malingerers and brain-damaged individuals. Individuals with closed head injury theoretically should produce performance profiles with numerous deficits (many scales over the critical level) characteristic of diffuse cortical involvement.

This was indeed the case. The pattern of deficits shown by this particular brain-damaged group was certainly not striking. Almost all of the scales were above the critical level with no particular deficits apparent; the mean t-scores for the brain-damaged group ranged from a t-score of 57 to a t-score of 70.

It was found that malingerers do perform differently from brain-damaged patients on many of the scales from the battery. The localization scales, however, seem to be superior to the summary scales for discriminating the characteristic pattern of performance of the two groups. The malingerers performed markedly better, in general, than the closed head injury group. Most deficits were seen in the sensory-motor and right hemispheric areas with few deficits appearing on any of the other scales. Though the localization scales seemed better, in general, than the summary scales for differentiating the malingerers from the brain-damaged group, the Expressive Speech and Receptive Speech Scales when compared with the other summary scales showed a relatively clear pattern of faking. The elevations of those two scales were very low while the other summary scales had much higher elevations. This type of profile would not likely be produced by an individual with a closed head injury. The head-injured patient would be more likely to show similar levels of problems across all scales rather than a greatly superior performance on scales thought to measure verbal abilities. Generally, the malingerers produced fewer and less severe deficits than the brain-damaged patients.

An examination of the summary score profiles produced by the malingerers and the closed head injury patients supports the notion that malingerers perform in a characteristically different way from brain-injured individuals. However, with a reanalysis of the profile scores

converted to deviations from critical level (which corrects for the differences in age and education created by combining the brain-damaged groups), the differences were clearer and suggested a distinctive malingered pattern of performance.

The malingerers performed similarly to the closed head injury sample on tasks which tapped into generally lower cortical functioning and/or right hemispheric functions. The malingerers did a relatively good job of faking on the sensory-motor and rhythmic/musical tasks, for example. The exception to this faking pattern produced by the malingerers was the similar performance of both groups on the Intellectual Processes, Writing, and Arithmetic Scales. Golden et al. (1980) have pointed out the significant effects of age and education on the Writing and Arithmetic Scales. Neither scale is included when the number of scales above critical level is considered in determining the likelihood of the presence of brain injury. These scales then are not considered good indicators of brain damage. These results would suggest they may also be inappropriate for differentiating real brain damage from malingering because of the effect of age and education on performance.

The similarity in performance of the two groups on the Intellectual Processes Scale is somewhat more difficult to explain as it strays from the supposition that malingerers cannot fake well on tasks involving higher cortical functions. However, as was pointed out earlier, the summary scales are heterogeneous and this contrasting result may be reflective of that heterogeneity. The Intellectual Processes items are likely to tap into many areas, not just that of higher cortical processes.

The malingerers, generally, performed differently from the closed-head injury patients on tasks which involve language and memory functions.

Normal individuals appear relatively unable to fake deficits characteristic of damage to the left hemisphere and other higher cortical areas.

One of the most striking differences between the two groups was that of the Pathognomonic Scale scores. This scale consists of 32 measures (items from the LNNB summary scales) which are considered to be the most effective indicators of brain damage when scored as "2". The malingerers rarely achieved less than normal performance on any of those items. This lends further support to this scale's ability to assist in the diagnosis of the presence of brain injury. With two minor exceptions, the malingerers did not perform as poorly on any of the scales as the actual patients.

The localization scales provide still more support for the assumption that malingerers are unable to fake left hemispheric and higher cortical deficits. Malingerers performed differently and, in all cases, not as poorly as the closed head injury patients on the left and right frontal, left parietal-occipital, and left temporal scales. These results again suggest that the scale items which require higher level functions are better at discriminating fakers from brain-damaged individuals. However, higher cortical functions are not necessarily limited to any specific area of the brain. One could argue that the right temporal and parietal lobes also involve higher cortical functions; therefore, the above conclusion should be considered tentative.

The left and right sensory-motor, right parietal occipital, and right temporal scales did not differentiate the malingerers from the brain-damaged patients. This suggests that sensory-motor and general right hemispheric functions are more susceptible to faking.

The results produced by the analysis of the lateralization scales are inconsistent with those of the localization and summary scales mentioned earlier. Malingerers performed similarly to the brain-damaged group on the L* but differently on the R* scale. This would seem confusing but Golden et al. (1981) have emphasized the relative inferiority of these empirically derived scales to the localization scales for lateralizing injuries. In the present study, they also appear to be less useful. Different results were found recently in a study independent of the Golden research group (Sears, Hirt, & Hall, 1984). These investigators found the R* and L* Scales to be superior to the localization and summary scales for lateralizing injuries.

Overall support was provided for the general ability of the LNNB to differentiate normal individuals from those with brain damage. None of the scale t-scores of the normal group approached its critical level. This suggests the LNNB is a valid instrument as it successfully differentiated normals from brain-damaged patients in every instance.

The older and younger brain-damaged patients originally expected to produce different profiles did not differ in performance on any scale. Older patients are typically considered to have difficulties of a more chronic nature and generally be more severely affected by the trauma of the injury than younger patients. This assumption led to the initial division of the brain-damaged patients into an older and younger group. However, at least in this particular sample, this assumption was unwarranted. Both groups showed a wide range of similar deficits. Older patients did not appear to be more severely affected than younger patients. This being the case, the two groups were combined to form one brain-damaged group with which to compare the normal and malingering groups.

Though the results are promising, the present study was not without its problems and therefore one must be circumspect in drawing conclusions. The malingerers were not brain-damaged and were not involved in an insurance claim or court action. It may be that given additional incentives, i.e., monetary gain instead of extra credit points, the malingerers might have done a better job of faking. Additionally, the malingerers were not given any information as to how closed head injury patients behave or what kind of problems they might have. If more specific instructions had been provided, the malingerers again might have been more proficient at faking. However, the fakers were successful in elevating their scores on all scales. Though many of their performances were not significantly different from those of the normal, nonmalingering subjects, their scores were consistently higher. With sophistication (i.e., more detailed instructions), the fakers could conceivably be quite good at faking brain damage on a neuropsychological test.

Though the experimenter was "blind" to the type of instructions each subject received, it was assumed that since the subject was a university undergraduate, he/she was not brain-injured. Consequently, some experimenter bias may be present in the results. Finally, the brain-damaged patients were a heterogeneous group even though all had suffered closed head injuries. They came from various parts of the United States, were tested by different technicians, and were likely to be quite different from the undergraduate sample. Again, these differences may color the results. Had the brain-damaged patients been a more homogeneous group from the immediate geographical area and more like the undergraduates, different results could have been obtained.

The similarity of these results and those of the Heaton et al. (1978) study examining the Halstead-Reitan battery's value for detecting fakers is promising. Those investigators also found a tendency for malingerers to perform poorly on motor and sensory tasks but to do relatively well on cognitive tasks. This would appear to provide more support for the comparability of the LNNB to the more widely used Halstead-Reitan battery. Additional confidence may also be extended to the analysis of performance patterns and with that, the utility of standardized neuropsychological batteries.

There are a number of hypotheses which could be proffered in an attempt to explain these results. Because of the exploratory nature of the present study, any far-reaching or broad conclusions would be inappropriate but several interesting possibilities can be considered. One such possibility is generated by considering the past history of the subjects. It is unlikely that many of them had had experience with the behavior of brain-damaged individuals. Secondly, the subjects were not given specific information as to exactly what to do in the testing situation in order to accurately fake their performances. They were merely told to perform in the way in which a brain-damaged person might perform. These subjects probably had little idea of how to behave because of their lack of experience with brain-injured people. They may have found it easier to perform poorly on sensory-motor tasks rather than on intellectual tasks or, perhaps, did not know they should perform poorly on language and cognitive tasks. The assumption might be made that deficits in a sensory-motor modality are more obvious and/or easier to fake than those in the cognitive modality.

In this particular instance, it seemed especially difficult for the faking subjects to perform poorly (fake) on the more verbally oriented items. Their scores on the Expressive Speech and Receptive Speech Scales were quite low and did not differ significantly from the scores of the normal, nonfaking subjects. The depressed elevations (good performance) on those two summary scales when compared with the higher elevations on the other 11 summary scales seemed to be quite characteristic of the faking pattern. Not only could this be helpful in identifying a person who might be faking, but it provides some support for the continued use of the somewhat controversial summary scales of the LNNB.

It could also be concluded that the faking subjects were not attempting to fake. Closer examination of their scale scores shows a consistently poorer (higher scores) performance than that of the normal, nonfaking subjects across all scales. It is suggested then that because the faking subjects were not given specific instructions as to how to perform, they did not know what to do in order to successfully fake on verbal items. When the malingering subjects missed verbal items, it was typically in an obvious manner rather than what might be expected in the performance of a head-injured patient. For example, one subject when asked to name a particular item, identified a pencil as a barbell. A brain-damaged person would be more likely to respond that the same item was a pen or might only be able to identify its use (i.e., you write with it) and not be able to name it.

Because the subjects attempting to fake apparently did not recognize the subtleties of the residual deficits of a closed head injury, they did not know what to do and either responded automatically with the correct response or answered with an absurd and/or obvious response.

During the debriefing of each subject, many of the fakers reported an inability to prevent a correct verbal response.

The present study was an exploration of the utility of a standardized neuropsychological test battery in detecting malingerers. Considering the results obtained here and in the Heaton et al. (1978) study, it may be possible to identify malingerers by examining their patterns of performance. A closed head injury patient would be expected to exhibit a large number of deficits whereas a malingerer might be expected to show a limited number of difficulties (i.e., poor performance on the sensory-motor tasks with near normal performance on cognitive tasks). The LNNB appears to be a valid diagnostic tool useful in discerning these patterns. Malingerers do produce a characteristic profile on the LNNB different from that of a closed head injury patient. The malingered profiles resemble more focal than diffuse damage.

In referring to personality measures, Meehl and Hathaway (1980) have pointed out the inherent susceptibility of structured tests to "faking" or "lying." This susceptibility also exists in structured neuropsychological tests. The results of the present study are promising in that it appears possible to identify those who are faking, explain why the particular pattern of faking might occur, and thus reduce this susceptibility. Without a theoretically supported test such as the LNNB to assist in the integration and clarification of brain-behavior relationships, fewer and less reasonable explanations could be offered for the relatively consistent results of the present study.

It would seem thatthe criticism leveled at the LNNB concerning the intrascale heterogeneity of the summary scales (e.g., Crosson & Warren, 1982; Delis & Kaplan, 1983) is warranted as conflicting results are

difficult to explain without examination of the individual items. This type of examination, though often helpful, is likely to be not only time-consuming and tedious but it departs from the concept of pattern analysis (e.g., Lewis et al., 1979) and standardization of the LNNB (Golden, Hammeke, & Purisch, 1980). Without question, additional research is needed before firm conclusions can be offered. But a pattern may be emerging which eventually may give the clinician evidence more objective than intuition with which to base his/her diagnoses.

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APPENDIX A

TABLES

TABLE I

MEAN T-SCORES AND STANDARD DEVIATIONS FOR BRAIN-DAMAGED AND FAKING GROUPS

Variable	Group			
	Brain-Damaged (n = 32)		Faking (n = 15)	
	Mean	SD	Mean	SD
Motor	66.66	19.80	56.37	13.81
Rhythm	66.75	16.59	59.76	20.64
Tactile	60.82	19.06	57.94	11.85
Visual	56.58	12.12	51.50	10.57
Receptive Speech	62.01	18.37	42.51	8.96**
Expressive Speech	58.58	16.80	42.14	7.60**
Reading	59.45	11.56	46.87	7.57
Writing	65.89	13.80	55.74	7.91**
Arithmetic	67.61	22.43	52.90	11.81**
Memory	62.75	9.62	47.92	11.39**
Intellectual Processes	65.76	16.73	56.85	10.64
Pathognomonic	65.32	17.54	47.58	8.77**
Left Hemisphere	59.63	15.80	61.08	18.88
Right Hemisphere	68.25	31.17	60.98	12.40
Left Frontal	63.83	15.05	49.17	7.86**
Left Sensory-Motor	59.18	12.89	55.08	10.05**
Left Parietal Occipital	62.60	14.88	49.55	7.93**
Left Temporal	57.92	12.24	43.78	7.11**
Right Frontal	62.42	11.54	50.83	8.27**
Right Sensory-Motor	62.25	13.25	52.93	7.32**
Right Parietal Occipital	69.48	19.59	55.22	10.33**
Right Temporal	62.85	11.19	54.00	8.08**
L*	60.87	13.44	52.81	9.33
R*	61.67	15.11	48.00	7.12**

**$p < 0.05$.

TABLE II

MEAN T-SCORES AND STANDARD DEVIATIONS FOR BRAIN-DAMAGED AND NORMAL GROUPS

Variable	Group			
	Brain-Damaged (n = 32)		Normal (n = 15)	
	Mean	SD	Mean	SD
Motor	66.66	19.80	39.34	5.18**
Rhythm	66.75	16.59	41.58	11.86**
Tactile	60.82	19.06	41.08	5.50**
Visual	56.58	12.12	43.35	6.02**
Receptive Speech	62.01	18.37	37.53	3.39**
Expressive Speech	58.58	16.80	37.23	5.28**
Reading	59.45	11.56	44.57	5.50**
Writing	65.89	13.80	52.35	6.72**
Arithmetic	67.61	22.43	43.87	5.18**
Memory	62.75	9.62	38.07	4.97**
Intellectual Processes	65.76	16.73	47.85	6.59**
Pathognomonic	65.32	17.54	38.21	4.69**
Left Hemisphere	59.63	15.80	41.73	5.32**
Right Hemisphere	68.25	31.17	38.86	4.37**
Left Frontal	63.83	15.05	40.67	3.75**
Left Sensory-Motor	59.18	12.89	43.08	6.19**
Left Parietal Occipital	62.60	14.88	43.45	5.47**
Left Temporal	57.92	12.24	40.56	4.53**
Right Frontal	62.42	11.54	41.17	5.16**
Right Sensory-Motor	62.25	13.25	45.73	6.50**
Right Parietal Occipital	69.48	19.59	41.78	5.58**
Right Temporal	62.85	11.19	46.00	4.28**
L*	60.87	13.44	39.63	3.70**
R*	61.67	15.11	36.81	5.09**

**$p < 0.05$.

TABLE III

MEAN T-SCORES AND STANDARD DEVIATIONS FOR NORMAL AND FAKING GROUPS

Variable	Group			
	Normal (n = 15)		Faking (n = 15)	
	Mean	SD	Mean	SD
Motor	39.34	5.18	56.37	13.81**
Rhythm	41.48	11.86	59.76	20.64**
Tactile	41.08	5.50	57.94	11.85**
Visual	43.35	6.02	51.50	10.57
Receptive Speech	37.53	3.39	42.51	8.96
Expressive Speech	37.23	5.28	42.14	7.60
Reading	44.57	5.50	46.87	7.57
Writing	52.35	6.72	55.74	7.91
Arithmetic	43.87	5.18	52.90	11.81
Memory	38.07	4.97	47.92	11.39**
Intellectual Processes	47.85	6.59	56.85	10.64
Pathognomonic	38.21	4.69	47.58	8.77
Left Hemisphere	41.73	5.32	61.08	18.88**
Right Hemisphere	38.86	4.37	60.98	12.40**
Left Frontal	40.67	3.75	49.17	7.86
Left Sensory-Motor	43.08	6.19	55.08	10.05**
Left Parietal Occipital	43.45	5.47	49.55	7.93
Left Temporal	40.56	4.53	43.78	7.11
Right Frontal	41.17	5.16	50.83	8.27**
Right Sensory-Motor	45.73	6.50	52.93	7.32
Right Parietal Occipital	41.78	5.58	55.22	10.33**
Right Temporal	46.00	4.28	54.00	8.08
L*	39.63	3.70	52.81	9.33**
R*	36.81	5.09	48.00	7.12**

**$p < 0.05$.

TABLE IV

MEANS AND STANDARD DEVIATIONS FOR DEVIATIONS FROM CRITICAL LEVEL FOR BRAIN-DAMAGED AND FAKING GROUPS

Variable	Group			
	Brain-Damaged (n = 32)		Faking (n = 15)	
	Mean	SD	Mean	SD
Motor	7.88	19.58	1.13	13.66
Rhythm	7.97	16.49	4.52	19.59
Tactile	2.04	19.50	2.70	11.56
Visual	-2.20	12.77	-3.74	10.00
Receptive Speech	3.23	17.66	-12.73	8.17**
Expressive Speech	-0.20	15.91	-13.09	7.12**
Reading	0.67	11.45	-8.36	7.41**
Writing	7.11	13.88	0.50	7.33
Arithmetic	8.83	22.02	-2.34	11.37
Memory	3.97	9.47	-7.32	11.23**
Intellectual Processes	6.98	16.44	1.61	10.31
Pathognomonic	6.54	16.97	-7.66	7.95**
Left Hemisphere	0.85	15.48	5.84	18.99
Right Hemisphere	9.47	31.67	5.74	12.07
Left Frontal	5.05	14.69	-6.07	7.39**
Left Sensory-Motor	0.40	13.08	-0.15	9.57
Left Parietal Occipital	3.82	14.59	-5.68	7.65**
Left Temporal	-0.86	11.19	-11.46	6.61**
Right Frontal	3.64	10.99	-4.40	7.89**
Right Sensory-Motor	3.47	13.05	-2.30	6.90
Right Parietal Occipital	10.70	20.00	-0.01	9.61
Right Temporal	4.07	11.24	-1.24	7.39
L*	2.09	13.27	-2.42	8.86
R*	2.87	15.05	-7.23	6.63**

**$p < 0.05$.

TABLE V

MEANS AND STANDARD DEVIATIONS FOR DEVIATIONS FROM CRITICAL LEVEL FOR NORMAL AND FAKING GROUPS

Variable	Group			
	Normal (n = 15)		Faking (n = 15)	
	Mean	SD	Mean	SD
Motor	-16.97	5.24	1.13	13.66**
Rhythm	-14.74	11.64	4.52	19.59**
Tactile	-15.23	5.37	2.70	11.56**
Visual	-12.96	5.97	-3.74	10.00
Receptive Speech	-18.78	3.69	-12.73	8.17
Expressive Speech	-19.08	5.48	-13.09	7.12
Reading	-11.74	5.56	-8.36	7.41
Writing	-3.96	6.92	0.50	7.33
Arithmetic	-12.44	5.37	-2.34	11.37
Memory	-18.24	4.83	-7.32	11.23**
Intellectual Processes	-8.46	6.51	1.61	10.31
Pathognomonic	-18.11	4.82	-7.66	7.95
Left Hemisphere	-14.58	5.03	5.84	18.99**
Right Hemisphere	-17.45	4.46	5.74	12.07**
Left Frontal	-15.65	3.92	-6.07	7.39
Left Sensory-Motor	-13.23	6.09	-0.15	9.57**
Left Parietal Occipital	-12.87	5.46	-5.68	7.65
Left Temporal	-15.76	4.49	-11.46	6.61
Right Frontal	-15.15	5.37	-4.40	7.89**
Right Sensory-Motor	-10.58	6.55	-2.30	6.90
Right Parietal Occipital	-14.54	5.63	-0.01	9.61**
Right Temporal	-10.31	4.24	-1.24	7.39**
L*	-16.68	3.80	-2.42	8.86**
R*	-19.50	5.32	-7.23	6.63**

**$p < 0.05$.

TABLE VI

MEANS AND STANDARD DEVIATIONS FOR DEVIATIONS FROM CRITICAL LEVEL FOR BRAIN-DAMAGED AND NORMAL GROUPS

Variable	Group			
	Brain-Damaged (n = 32)		Normal (n = 15)	
	Mean	SD	Mean	SD
Motor	7.88	19.58	-16.97	5.24**
Rhythm	7.97	16.49	-14.74	11.64**
Tactile	2.04	19.50	-15.23	5.37**
Visual	-2.20	12.77	-12.96	5.97**
Receptive Speech	3.23	17.66	-18.78	3.69
Expressive Speech	-0.20	15.91	-19.08	5.48**
Reading	0.67	11.45	-11.74	5.56**
Writing	7.11	13.88	-3.96	6.92**
Arithmetic	8.83	22.02	-12.44	5.37**
Memory	3.97	9.47	-18.24	4.83**
Intellectual Processes	6.98	16.44	-8.46	6.51**
Pathognomonic	6.54	16.96	-18.11	4.82**
Left Hemisphere	0.85	15.48	-14.58	5.03**
Right Hemisphere	9.47	31.67	-17.45	4.46**
Left Frontal	5.05	14.69	-15.65	3.92**
Left Sensory-Motor	0.40	13.08	-13.23	6.09**
Left Parietal Occipital	3.82	14.59	-12.87	5.46**
Left Temporal	-0.86	11.19	-15.76	4.49**
Right Frontal	3.64	10.99	-15.15	5.37**
Right Sensory-Motor	3.47	13.05	-10.58	6.55**
Right Parietal Occipital	10.70	20.00	-14.54	5.63**
Right Temporal	4.07	11.24	-10.31	4.24**
L*	2.09	13.27	-16.68	3.80**
R*	2.87	15.05	-19.50	5.32**

**$p < 0.05$.

APPENDIX B

CONSENT FORM

Oklahoma State University
Department of Psychology
Stillwater, Oklahoma

Date _____

Name _____

I hereby voluntarily authorize Candace Conley (Researcher), Oklahoma State University, and such assistants that may be designated to perform the following study: "Testing With the Luria-Nebraska Neuropsychological Battery."

I understand that if I have a history of brain injury, I will not be allowed to participate in this study but I will receive the extra credit.

I further understand that strict confidentiality will be observed of all data collected under the guidelines established by the Department of Psychology, Oklahoma State University. Complete anonymity (no names will be used) will be preserved and data will be released only to qualified professionals for scientific or training purposes.

I further understand and agree that the data and information related to and resulting from the study may be used for publication in scientific journals but that my name shall not be used in association with these publications without my specific written permission.

I understand that if, at any point, I wish to withdraw from the experiment, I may do so without risk of losing the extra credit.

By signing this consent form, I have not waived any of my legal rights or released this institution from liability for negligence. Should any problems arise during this study, I may take them to the Chairman, Research Committee: Dr. Robert Schlottmann, Fourth Floor, North Murray Hall, O.S.U., Phone: 624-6027.

I have read and understood this form.

Signature of Participant _____

VITA 2

Candace Conley

Candidate for the Degree of

Doctor of Philosophy

Thesis: FAKING NEUROPSYCHOLOGICAL TEST DATA: A FURTHER EXAMINATION OF THE LURIA-NEBRASKA NEUROPSYCHOLOGICAL BATTERY

Major Field: Psychology

Biographical:

Personal Data: Born in Tulsa, Oklahoma, November 10, 1956, the daughter of Marjorie Evelyn Bailey and Robert Eillis Conley.

Education: Graduated from Holland Hall School, Tulsa, Oklahoma, in May, 1974; received the Bachelor of Arts degree in Mass Media News from the University of Tulsa, Tulsa, Oklahoma, in 1979; in 1980, enrolled in the doctoral program at Oklahoma State University, Stillwater, Oklahoma; completed requirements for the Master of Science degree at Oklahoma State University in July, 1982; completed requirements for the Doctor of Philosophy Degree at Oklahoma State University in July, 1985.

Professional Experience: Undergraduate statistics laboratory assistant, Department of Psychology, University of Tulsa, 1979; graduate teaching assistant, Department of Psychology, Oklahoma State University, 1980-1982; administrative assistant, Psychological Services Center, Oklahoma State University, 1983; clinical practicum student, Central State Hospital, Norman, Oklahoma, 1983-1984; psychology intern, Mid-Missouri Psychology Internship Consortium, Columbia, Missouri, 1984-1985.