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A Selective and Critical Review of Neuropsychological Deficits and the Frontal Lobes

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Presumptions about the functions of the frontal lobes, and the sensitivity and specificity of certain tests to measure frontal lobe functions, are having a substantial influence on both clinical and research conclusions. In this paper the authors examine the details of the studies that have contributed to these presumptions, and find that the evidence to support these conclusions is weak. A detailed evaluation of the evidence relating to the Wisconsin Card Sorting Test and the Thurstone Word Fluency Test is also presented. Finally, the development of the belief that frontal lobe functions can be specifically measured is reviewed. The authors of this paper conclude that the "bewildering array" of deficits attributed to frontal lesions still seems to prevail.

KEY WORDS: frontal lobe functions; Wisconsin Card Sorting Test; Thurstone Word Fluency Test; General vs. specific deficits.

INTRODUCTION

In the late 1930s and 1940s a great surge of interest in the frontal lobes appeared in the literature, only to wane in the 1950s (Hecaen and Albert, 1978; personal observations). A resurgence of interest began in the late 1970s and continues to the present time. Many factors contributed to this phasic attention to the frontal lobes, including (1) the large anatomical area of the brain occupied by the frontal lobes (Damasio, 1991), (2) attention captured by the explosion of interest in right-brain/left-brain differentiated functions (Gazzaniga *et al.*, 1963; Sperry *et al.*, 1969), and (3) the

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availability of the frontal lobes to be assigned to the more general functions of adaptive abilities not already claimed by more posterior areas of the brain (nature abhors a vacuum).

Even a brief perusal of the recent literature reveals the current emphasis being placed on the frontal lobes in neuropsychological functioning. A casual literature review identified 16 articles published within the last few years that reached conclusions about the frontal lobes. Eight articles reported empirical findings (Axelrod *et al.*, 1992; Boone *et al.*, 1990; Fiducia and O'Leary, 1990; Gnys and Willis, 1991; Grodzinsky and Diamond, 1992; Rybash and Colilla, 1994; Shute and Huertas, 1990; Welsh *et al.*, 1991); in one article the authors reported a meta-analysis of tests for diffuse brain damage, and felt it was useful to include a category of tests intimately related to the frontal lobes (Chouinard and Braun, 1993); and seven theoretical review articles were devoted to explicating frontal and prefrontal functions (Goldman-Rakic, 1993; Grafman *et al.*, 1993; Hart and Jacobs, 1993; Lezak, 1993; Schwartz *et al.*, 1993; Sohlberg *et al.*, 1993; Varney and Menefee, 1993).

The topics of the empirical articles included the following:

1. the deterioration of frontal lobe functions among healthy older subjects (two articles),
2. the maturation of frontal lobe functions in children (two articles),
3. establishing the test–retest reliability and construct validity of tests of executive functioning in young children,
4. investigating the relationship of a Piagetian formal operational reasoning process to measures of frontal lobe dysfunction,
5. determining the role of frontal lobe functions in boys with attention deficit–hyperactivity disorder,
6. the relationship of a special type of memory (source memory) to a particular dimension of frontal lobe functioning.

Except for the test–retest reliability study (in which the results were not fully supportive of the hypothesis), each of these investigations drew conclusions about the role of the frontal lobes in neuropsychological functioning. In fact, one study of normal aging effects (Axelrod *et al.*, 1992) "revealed the sensitivity of the MMSE (Mini-Mental State Examination) to changes in frontal lobe functioning" (p. 70), essentially because performances on the MMSE, like the performances on three "frontal lobe" tests, deteriorated across the age span of 50–90 years. In addition, these authors found a correlation of 0.49 between the MMSE and a composite score based on the three frontal lobe tests, and viewed this finding as "noteworthy because the MMSE was intended as a measure of gross cognitive status rather than of frontal lobe functioning" (p. 68).

Although each of the studies noted above drew conclusions about the frontal lobes (except for the study that did not fully support its hypothesis), none of the investigations used any subjects who had documented evidence of frontal lobe involvement. In addition, none of the subjects in any of the studies were examined neurologically for independent evidence of frontal lobe damage. What, then, was the basis for such conclusions? The presumption was that certain tests had previously been validated as frontal lobe tests, and any evidence of impairment on these tests was, *ipso facto*, an indication of frontal lobe impairment. (In most cases the tests used with children had to be adapted or even changed substantially from the adult versions, and they had never been validated by investigations of children with frontal lobe lesions. However, this lack of validation was rarely considered to be a detriment to drawing conclusions about frontal lobe functioning.)

Even more recent reports have suggested that the Category Test and Part B of the Trail Making Test are "frontal lobe" tests (Butters *et al.*, 1994; Farmer, 1994; Jarvis and Barth, 1994), a presumption also made in two of the investigations cited above (Grodzinsky and Diamond, 1992; Shute and Huertas, 1990). A rigorous test of this postulate, however, revealed that neither the Category Test nor Part B of the Trail Making Test was specifically sensitive to frontal damage (Reitan and Wolfson, in press).

The validity of this expanding body of knowledge about the frontal lobes obviously depends on evidence that the tests and procedures are valid as specific indicators of frontal, as opposed to nonfrontal, functioning. Presumptions about frontal lobe functions have clearly gotten out of hand, unless there is strong evidence that the so-called frontal lobe tests are *specifically* sensitive to frontal lobe damage.

The seven theoretical and/or review articles identified in our brief literature search describe the fundamental neuropsychological features of the frontal lobes. Hart and Jacobs (1993) described frontal lobe functions as those that (1) subserve selective attention; (2) provide continuity and coherence to behavior; (3) modulate behavior so that drives are satisfied within the constraints of the internal and external environments; and (4) act as an overseer, based on insight and self-awareness, to monitor, adjust, and evaluate behavior.

Goldman-Rakic (1993) referred to the literature as a basis for concluding that the frontal lobes subserve (1) attention; (2) synthetic reasoning and planning; (3) the ability to grasp the essence of a situation, utilize past experience, or regulate behavior through verbal input; (4) initiative, spontaneity, and verbal and constructional fluency; (5) spatial orientation; and (6) behavior restraint, social affect, and global features of personality.

Lezak (1993) offered a four-part definition of frontal lobe functioning: (1) volition, including capabilities for awareness of one's self and surround and motivational state; (2) planning, including abilities to conceptualize change (look ahead), be objective, conceive of alternatives and make choices, develop a plan conceptually, and sustain attention; (3) purposive action, including productivity and self-regulation; and (4) performance effectiveness, or quality control.

Varney and Menefee (1993) referred to Lezak's definition, and offered a list of descriptors that characterize the individual with frontal lobe symptoms: poor empathy, poor judgment, absentminded, indecisive, immature, poor insight, disorganized, impulsive and impolitic, poor planning, nonreinforcing, nonspontaneous, self-centered, perplexed, low motivation, rigid/inflexible, repeats mistakes, flat affect, disinhibited, risk seeking, and stimulus bound. It seems that the frontal lobes are, if nothing else, versatile!

Sohlberg *et al.* (1993) related their conceptual framework of frontal lobe functions with the theory proposed by Stuss (1991), and identified frontal lobe functions as (1) sensory and perceptual, or knowledge-based information; (2) executive control or the supervisory functions; and (3) self-reflectiveness or self-awareness (which is dependent on the prefrontal areas).

Schwartz *et al.* (1993) focused on the fact that some subjects with severe brain injury are unable to perform routine, simple activities of daily living. These authors cited Luria's concept of frontal apraxia in this context, but suggested that a more plausible account of frontal apraxia involves difficulties in "assembling or activating the components of the action plan or in sustaining that activation (or both)" (p. 61).

Grafman *et al.* (1993) proposed that prefrontal lobe injury or dysfunction causes problems in (1) temporal coding, (2) estimation (impairment in estimating lesser known facts as compared with first-order facts), (3) sustaining attention and maintaining focus, (4) accuracy in estimating extended time intervals, (5) forming and shifting concepts, (6) verbal functions that influence working memory, (7) anticipating an expected forthcoming motor or verbal response, (8) maintaining registered information over time, (9) developing second-order associative responses, (10) social conduct, and (11) mood (depression, anxiety, apathy, and disinterest). The authors postulated that these deficits result from damage to the prefrontal cortex, and can be subsumed under a concept of "the managerial knowledge unit."

Grafman *et al.* noted, however, that the deficits that "have long been associated with prefrontal lesions . . .may appear in patients with subcortical degenerative disorders like Parkinson's disease, cerebellar disease, progressive supranuclear palsy, and so forth because they are part of a neural

network that includes the frontal lobes" (p. 85). This statement obviously minimizes the specific significance of the frontal lobes, and places the function of the frontal lobes in the context of the extensive areas of connection between the frontal lobes and the rest of the brain. If the deficits described by these authors are attributed to impairment or interruptions within these neural networks, the task of delineating and specifying the frontal lobe component of the functions of these neural networks becomes even more imposing than identifying specific deficits caused by lesions contained entirely within the frontal lobes. One cannot permissively presume that certain deficits occur because the frontal lobes are a part of the neural network. Considering the extensive inter- and intracerebral connections within the hemispheres, a strict application of this notion would significantly diminish the entire concept of regional localization of function.

The theoretical/review articles cited above present an extensive range of subjective impressions of frontal lobe functions. In fact, the list of functions and deficits exceeds the entire range of behaviors included in some theories of brain–behavior relationships (see Reitan and Wolfson, 1992a, 1993), and one would have to presume that the other areas of the brain (the temporal, parietal, occipital, and subcortical structures) have at least some functions. One is reminded of *The Meaning of Intelligence*, a book in which Stoddard (1943) offered a broad definition of intelligence:

> Intelligence is the ability to undertake activities that are characterized by (1) difficulty, (2) complexity, (3) abstractness, (4) economy, (5) adaptiveness to a goal, (6) social value, (7) the emergence of originals and to maintain such activities under conditions that demand a concentration of energy and a resistance to emotional forces.

Interestingly, Stoddard's characteristics of intelligence seem to overlap extensively with the "frontal lobe" functions described previously.

As Halstead had noted, by 1947 psychological conceptualizations of intelligence had become quite diversified, and related to a number of criteria that were poorly defined and almost certainly overlapping, just as frontal lobe functions have become today. Halstead's comment that the essential requirement of abilities involved in intelligence was only that they reflect "a capacity of some kind which has a Gaussian distribution in the general population" (Halstead, 1947, p. 10) appears to apply equally to frontal lobe functions.

Neisser (1979) felt that there is no hope of offering a definition of intelligence (which is presumably more extensive than frontal lobe functions). He concluded that "the concept of intelligence *cannot* be explicitly defined, not only because of the nature of intelligence, but also because of the nature of concepts" (p. 179). Perhaps the same conclusion applies to frontal lobe functions.

Goldman-Rakic (1993) commented that such "a bewildering array of behavioral deficits have been attributed to frontal lobe injury that a common functional denominator would appear elusive" (p. 13). Considering the range and diversity of definitions and characterizations of frontal lobe functions, together with their apparent importance for competent human performance, Halstead (1947) perhaps summarized it best nearly 50 years ago when he commented that the frontal lobes were "the organs of civilization — the basis of man's despair and of his hope for the future" (p. 49). Alternatively, in our quest to identify the neuropsychological effects of frontal lobe lesions, we could perhaps follow Hebb's (1949) approach to the definition of intelligence: he ultimately decided that intelligence was an equivocal term that "each writer can define to suit himself, and there is no sense in arguing over terminology" (p. 277).

EMPIRICAL EVIDENCE

Wisconsin Card Sorting Test

The validity of the conclusions of empirical studies or the various theoretical/review publications obviously depends on the experimental studies on which they are based. Do the scientific research findings identify certain tests as indicators of deficits specific only to the frontal lobes? Exactly this presumption is made in the research literature, with a considerable number of tests presumed to be specific frontal lobe indicators (for example, the Wisconsin Card Sorting Test, the Thurstone Word Fluency Test, the Category Test, the Stroop Test, the Design Fluency Test, the Porteus Mazes Test, Part B of the Trail Making Test, subtests of the Wechsler Memory Scales, the Consonant Trigram Test, and experimental procedures involving verbal recency procedures and go–no go tasks).

The Wisconsin Card Sorting Test (WCST) and the Thurstone Word Fluency Test (TWFT) are among the instruments identified most frequently as frontal lobe tests. What is the evidence that these tests are specific indicators of frontal lobe functions?

Milner's studies (1963, 1964) are often cited as a basis for identifying frontal lobe functions and for using the WCST to assess frontal lobe damage and normal development of frontal lobe functioning. Milner studied patients who had undergone discrete surgical excisions for treatment of epilepsy, and reported that 18 patients with frontal excisions involving dorsolateral areas made significantly more mistakes on the WCST than did patients with lesions in other areas (mostly the temporal lobe).

It must be noted that all of Milner's subjects had epilepsy so severe that they had a refractory response to medication and met the criteria for surgical intervention. Such patients are not representative even of epilepsy generally, and certainly do not represent the full range of conditions that produce brain damage. For example, Matthews and Kløve (1967) found that patients who had complex partial seizures of unknown etiology performed at significantly better levels than patients who had major motor seizures of unknown etiology. Thus, Milner's findings may have limited generalizability to patients with other types of cerebral lesions.

Dodrill (1981) reviewed reports of patients with refractory seizures whose neuropsychological functions deteriorated over time. He noted that Harris (1972) and Wasterlain and Duffy (1976) demonstrated neuronal degeneration associated with repeated seizures in animals, and that seizures also have an inhibitory effect on brain protein synthesis, on brain growth, and eventually on behavioral development. Citing Arieff and Yacorzynski (1942), De Haas and Magnus (1958), and Lennox and Lennox (1960), Dodrill commented that "with these studies as a background, it is not surprising that several investigators do implicate a gradual deterioration of abilities" in patients who experience epileptic attacks repeatedly.

In addition to the effects on neuropsychological performances caused by continued seizures and the long-term use of medications, and the many social and emotional problems among epileptics "that are far in excess of the base rate of the population" (Dodrill, 1981, p. 380), the consequences of brain impairment among epileptics during the course of development must be considered. Gilroy and Meyer (1979) state that "about 90% of epileptic patients have histories of epilepsy beginning in childhood" (p. 359). Although many factors obviously determine an individual's neuropsychological competency — and the duration of epilepsy considered by itself is not a strong influence — Dodrill (1981) noted "that the earlier the age of onset and the longer the duration, the lower the mental abilities" of the epileptic patient (p. 371). Kløve and Matthews (1966) and Dikmen *et al.* (1975) have reported similar findings.

In contrast to the Kennard principle (see Finger and Wolf, 1988), cerebral disease or damage during the developmental years seems generally to have an adverse effect on adult neuropsychological functions in comparison with persons who have had the advantage of normal development of brain functions (Reitan and Wolfson, 1992b). Mahoney *et al.* (1983) documented the impairment associated with severe head trauma sustained by young children. Using the WCST, Strauss *et al.* (1993) also found deficits associated with complex partial epilepsy experienced early in life. Therefore, a number of variables may limit the generalizability of Milner's findings.

Immediately following Milner's (1964) presentation of her findings, Teuber (1964a) said in his discussion,

> Our cases of frontal gunshot wounds did show a deficit on the Wisconsin Card Sorting Test — at least as a group — although there were striking exceptions in individual cases . . .On the other hand, our posterior lobe patients also had a deficit which seemed somewhat more severe. (p. 333)

Thus, even at the time of the original report, Milner's results were controverted by Teuber's findings, with differences possibly relating to the type of lesion rather than the location.

In conclusion, there are many reasons why Milner's studies should be replicated in a study design that would relate to the effects of brain lesions more generally and control for developmental effects among the types of subjects she studied.

Drewe (1974) studied WCST results of patients who had types of lesions other than the epileptogenic foci studied by Milner, and found that patients with frontal lesions demonstrated significantly more perseverative errors. Robinson *et al.* (1980) pointed out that there was reason to believe Drewe's frontal patients may have had greater overall impairment than her nonfrontal groups, and raised a question about the specificity of the findings on the WCST.

Robinson *et al.* (1980) performed a study using 123 normal subjects and eight groups of subjects with lesions in the following locations: right frontal, 13; right frontal plus more extensive involvement, 11; left frontal, 10; left frontal plus, 12; right nonfrontal, 9; left nonfrontal, 14; and diffuse, 38. Except for the normal subjects, each group included subjects with brain lesions of various etiologies, but the lesions were not equivalent across etiologies. The normal group made fewer perseverative responses than the total brain-damaged sample. The frontal group performed more poorly than the nonfrontal groups, even when perseverative scores were adjusted for the Average Impairment Rating in an attempt to control for differences in overall impairment. However, the frontal groups were not more impaired than the group with diffuse involvement.

These investigators attempted to replicate clinical classifications based on Milner's (1963) results. Whereas none of Milner's frontal cases were able to achieve more than three categories on the WCST, 43% of the frontal subjects in the Robinson *et al.* study achieved four or more categories, and 39% of the nonfrontal cases achieved three or less. Using a clinically derived cutoff score, the lack of difference between the frontal and diffuse groups led to more than half of the diffuse cases being classified as having frontal lesions. Thus, even though frontal cases performed somewhat more poorly than nonfrontal cases, Robinson *et al.* concluded that the value of the WCST in localizing brain lesions appears to be questionable, and stated

that their findings "would not support the use of the WCST in attempting to discriminate focal frontal from diffuse lesions" (p. 613).

Anderson *et al.* (1991) also published a study of the WCST as a measure of frontal lobe damage with the following justification:

> Research to this point has suggested that patients with frontal lobe damage may perform worse on the WCST than do patients with focal nonfrontal damage (Drewe, 1974; Heaton, 1981; Milner, 1963). However, one study found that subjects with posterior lesions performed worse than subjects with anterior lesions (Teuber, Battersby, & Bender, 1951), and most studies have found considerable variability in WCST performances across subjects with frontal lobe damage. There have been conflicting findings with regard to the role of dorsolateral frontal vs. orbitomesial areas in WCST performance (Drewe, 1974; Milner, 1963). No study up to this time has made use of modern neuroradiological techniques to investigate the locus of damage in patients who failed the WCST, and many of the subject groups used in prior studies have been less than ideal for purposes of the lesion method. Some studies have combined subjects with different pathological processes, such as tumors and strokes, into single groups, a practice which may introduce considerable error variance (Anderson, Damasio, & Tranel, 1990). Some subjects have been studied in the acute epoch, when neuropsychological profiles are likely to be changing and considerable recovery may still take place. Given this situation, together with observations of patients with extensive frontal lobe damage who performed well on the test (Anderson, Damasio, Tranel, & Damasio, 1988; Eslinger & Damasio, 1985; Heck & Bryer, 1986), we undertook an investigation of the sensitivity and specificity on the WCST as an index of frontal lobe damage. (p. 910)

The presumption by Anderson *et al.* that studies combining subjects with different pathological processes, such as tumors and stroke, into single groups are "less than ideal for purposes of the lesion method" and "may introduce considerable error variance" must be tempered by a consideration of the purpose of the study. In fact, these investigators based their results on 91 patients with cerebrovascular accidents ($n = 71$) and tumors or seizures ($n = 20$), apparently including in the group patients with neoplasms as diverse as meningiomas and metastatic carcinomas. Forty-nine patients had frontal lesions, 24 had nonfrontal lesions (affecting the temporal, parietal, and occipital lobes as well as the thalamus and basal ganglia), and 18 patients, included in some analyses, had lesions of, but not limited to, the frontal lobes.

Various analyses of WCST data supported the following conclusions:

1. No significant differences were found between the frontal and nonfrontal groups. In fact, the scores were quite similar.
2. Optimal cutoff scores for frontal and nonfrontal patients correctly classified only 62% of the subjects.
3. Comparisons of left, right, and bilateral frontal damage failed to produce any significant differences.
4. No reliable differences were found among patients having different areas of damage within the frontal lobes.

5. The size of the lesion within the frontal lobes was not correlated with WCST scores.

The investigators concluded that their findings failed to support a consistent relationship between WCST scores and frontal lobe damage, and they inveighed against using WCST performances to compose frontal and nonfrontal groups for research purposes.

Grafman *et al.* (1990) compared the WCST results of 421 brain-damaged subjects with 48 controls matched for age, preinjury intelligence, education, and military service. In the brain-damaged group, 126 subjects had lesions involving only the frontal lobes and 183 subjects had nonfrontal lesions. The controls performed better than the brain-damaged groups, but "patients with lesions limited to the frontal lobes performed as a group no worse than patients with nonfrontal lesions" (p. 1120).

These authors cited Cronin-Golomb (1990) in pointing out that the WCST "continues to be used as a measure of frontal lobe functioning in psychiatric, focal lesion, and subcortical dementia patients" (p. 1120), and advised using caution in the "clinical research practice of attributing poor performance on the card-sorting test to frontal lesions" (p. 1121).

In another recent study, Van den Brock *et al.* (1993) used a modified version of the WCST and found no significant differences between patients with either frontal vs. nonfrontal lesions or left hemisphere vs. right hemisphere lesions. However, the task did have high specificity and good sensitivity in differentiating patients with brain lesions, regardless of the site, from control subjects.

Thurstone Word Fluency Test

The nature of the task involved in the TWFT serves as a basis for reviewing the literature about this test. The TWFT requires the subject to write (or say) as many words as possible that begin with a certain letter within a specified time period. Details of the procedure have varied among investigators concerning letters and time periods, but the subject's task has consistently been to report as many words as possible. There can be no doubt that this test requires the subject to express verbal material of a prescribed nature (beginning with a selected letter), and that the score depends on speed of performance (the number of words expressed within a given time period).

On one hand, this type of task is related to verbal intelligence, and this is exactly the purpose for which the Thurstones developed the test (Thurstone, 1938; Thurstone and Thurstone, 1949). A subject's level of intelligence is therefore very likely to influence the results, with more intel-

ligent subjects performing better than less intelligent subjects. Because of its significant correlation with intelligence, education would presumably be another factor affecting performance, at least among normal subjects. More important in a sample of brain-damaged subjects, however, would be the presence or absence of dysphasia and, more specifically, the presence or absence of dysnomia and an impairment of verbal fluency.

In their review of the formal dysphasia examination, Albert *et al.* (1981) identified six scales to cover the range of deficits shown by dysphasic subjects. Two of these scales related to Grammatical Form (which, according to the authors, correlates closely with verbal fluency in aphasia), and Word Finding, which represents an impaired ability "to find the desired word for production in spontaneous speech and is present in every type of dysphasia" (p. 7). Wheeler and Reitan (1962) reported word-finding deficits in 53% of a group of subjects with heterogeneous left cerebral lesions and in 0% of a similar group with right cerebral lesions. It appears that the TWFT can be adversely affected by dysphasia, and might be a specific test for certain dysphasic symptoms.

These observations suggest that an evaluation of the sensitivity of the TWFT, either comparing groups with left vs. right cerebral lesions or frontal vs. nonfrontal lesions, should consider the role of aphasia in producing deficits. The left hemisphere's dominance for language functions (including verbal fluency) is well known. It should also be recognized that expressive verbal tasks are likely to be more impaired by left frontal lesions than by left hemisphere lesions that are more posterior in location.

The TWFT appears to depend on expressive (as contrasted with receptive) verbal abilities inasmuch as it requires the production of words. Albert *et al.* (1981), in discussing differential location of left hemisphere lesions that cause expressive (nonfluent) aphasia and receptive (fluent) aphasia, comment that "non-fluency is usually, but not always, associated with anterior lesions; fluency with posterior lesions" (p. 9). Including dysphasic patients in a left hemisphere group should therefore produce the lowest TWFT scores in subjects with anterior lesions. Because of the nature and requirements of the TWFT, any study using this test with patients having left hemisphere lesions should describe the incidence and type of aphasia in the group and discuss how dysphasic deficits may have affected the results.

In attempting to delineate the effects of dysphasia on other abilities, Reitan (1960) studied the effects of dysphasia on a broad range of neuropsychological tests. Brain-damaged subjects were examined with the Reitan-Indiana Aphasia Screening Test (Reitan, 1985) in order to compose two groups: one with definite indications of dysphasia and one with no evidence of dysphasia. Because of the differential influence of various types

of lesions on neuropsychological measurements (Reitan, 1964; Hom and Reitan, 1982, 1984), the dysphasic and nondysphasic groups were matched not only for gender, chronological age, and years of education, but also for type of brain lesion. A non-brain-damaged comparison group, matched with the brain-damaged groups for gender, age, and education, was also composed. It was necessary to review the comprehensive neurological evaluations of approximately 1500 patients in order to compose matched triads across the three groups composed of 32 subjects each.

Each subject was given the Wechsler-Bellevue Scale (14 variables, including Verbal IQ, Performance IQ, and Full Scale IQ), nine tests devised by Halstead plus his Impairment Index (ten variables), and Part A and Part B of the Trail Making Test (two variables). Both brain-damaged groups consistently performed more poorly than the controls, except that on the Wechsler verbal tests the scores of the nondysphasic group approached the scores of the controls. The effects of dysphasia were shown by comparing the dysphasic and nondysphasic brain-damaged groups. These two groups had nearly identical mean scores on all measures *except* the tests that explicitly required expressive or receptive language functions. The dysphasic group performed more poorly than the nondysphasic group on each test that included language or language symbols. It was hypothesized that implicit verbalization during the task, especially on complex measures such as the Category Test and the Tactual Performance Test, might be impaired among the dysphasic subjects and lead to lower scores; however, on these measures the means for the two groups were nearly identical.

These findings strongly suggest that the incidence and possible effects of dysphasia should be identified when tests that require production or processing of verbal material are used in comparative assessment of brain-damaged groups.

In terms of controlled comparisons, the TWFT has been less thoroughly researched than the WCST. The first report suggesting that the TWFT might be a frontal lobe test came from Milner (1964), presented in about one and one-half pages and based on seven left frontal, seven left temporal, and four right frontal cases. All of Milner's subjects were drawn from a limited sample of patients who had focal cortical excisions for treatment of epilepsy. She found that her group of seven patients with left frontal lesions performed poorly on the TWFT, and her report of the performance of this group of seven patients is frequently cited in the literature as a basis for concluding that the TWFT is a frontal lobe test. Milner did not provide information about the incidence of dysphasia in her subjects, 14 (77%) of whom had left hemisphere lesions. She did state that lobectomies that spare Broca's area are not followed by any lasting dysphasia, and that scores on most verbal tests rapidly return to normal.

However, her patients were retested "about three weeks" after the first examination, with the surgery for intractable epilepsy performed during the interval. One could not be sure that residual dysphasia was not a factor influencing the test results. No information was given about the age and education of the subjects.

Benton (1968) studied three groups with frontal lesions (right, n = 8; left, n = 10; bilateral, n = 7), but made no frontal vs. nonfrontal comparisons. Although he did not give specific evidence of comparability of these groups regarding the types of lesions, he noted that the subjects mainly had intrinsic tumors, extrinsic tumors, and degenerative disease. The means for age and education were not significantly different. Six tests were administered: (1) FAS words to test verbal fluency; (2) assembling structures from a tray of assorted blocks; (3) learning a list of word pairs; (4) copying designs; (5) interpreting proverbs; and (6) citing the day of the week, the day of the month, the month, the year, and the time of day.

Benton made 18 predictions and reported that 15 predictions were confirmed. Using six tests and three groups permitted a total of 18 comparisons. Three of the 18 intergroup comparisons were significant at the .01 level, one was significant at the .025 level, four were significant at the .05 level, five were significant at the .10 level, and the remaining five comparisons were not significant. Only 8 of the 18 comparisons were significant at the .05 level or less, but Benton felt that "given such small groups, it is perhaps permissible to consider differences at the 0.10 probability level (i.e., approaching the conventionally accepted level of 0.05) as suggestive of a true difference" (p. 57). While one must recognize that a small n is a condition that validly limits scientific conclusions, even accepting as significant the five comparisons at the .10 level does not yield enough "significant" results to confirm 15 of 18 hypotheses. Benton obviously "confirmed" some hypotheses on the basis of differences attributable to chance.

The three groups were also assessed for frequency of defective performances on the six tests, with a defective performance being defined as a score "exceeded by 95–96 percent of control subjects" (p. 57). These control subjects were not described in the report; no information was given about their age or education distributions or any other characteristics. Nevertheless, the frequency of "defective" performances by the frontal lobe patients was used to determine which deficits were characteristic of each group. In fact, the test that required naming the day of the week, date, month, year, and time of day was described as a bilateral test "*par excellence*" because four of the seven bilateral patients (57%) performed defectively, whereas none of the 18 patients with unilateral frontal lesions performed defectively.

Finally, it must be noted that because no patients with nonfrontal lesions were used for comparisons, the specificity of the performances of patients in this study of frontal lobe deficits was entirely undetermined. Despite these problems, this study is frequently cited to support tests of verbal fluency as frontal lobe measures.

Using the TWFT, Ramier and Hécaen (1970) studied 76 subjects with unilateral lesions, the majority of which were tumors (53%). Lesion types included tumors, hematomas, traumatic injuries, angiomas, and cortical excisions. The groups were not equivalent for types of lesions; tumors were underrepresented in the right frontal group (33%) and overrepresented in the right nonfrontal group (71%). The lesions were in the following locations: left frontal, 17; right frontal, 12; left nonfrontal, 30; right nonfrontal, 17. Fifteen of the 47 patients with left cerebral lesions were identified as aphasic, but no information was given about the criteria used for determining aphasia. There appeared to be age and education differences in the groups with left vs. right cerebral lesions, but data were not reported for frontal vs. nonfrontal lesions. Differences on these variables were not tested statistically.

Ramier and Hécaen analyzed their results with relation to lateralization (left hemisphere vs. right hemisphere) and location of the lesion (frontal vs. nonfrontal). On the TWFT, the group of subjects with left hemisphere lesions (which included all of the aphasic subjects) performed more poorly than the subjects with right hemisphere lesions. Since the anterior portion of the language area in the left hemisphere has long been known to be principally involved in language expression, and the TWFT obviously is an expressive verbal task, it is not surprising that the left frontal group performed most poorly among the four groups. Although it is not clear from this report, there is also the possibility that frontal lesions without clinically obvious dysphasia produced deficits in verbal fluency.

It is apparent that, in addition to the location of the lesions, the intergroup differences could have been influenced by age, education, dysphasia, and type-of-lesion differences in the groups.

Perret (1974) examined 118 subjects using the TWFT and other measures. Of these subjects, 83% had brain tumors, but the report did not specify the types. The lesion locations were as follows: left frontal, 23; right frontal, 27; left temporal, 15; right temporal, 17; left posterior, 18; right posterior, 18. The author stated that most of the patients in the frontal groups had large tumors. Age and education intergroup differences were not statistically significant. Perret did not report any systematic attempts to evaluate dysphasic deficits, but did presume that left hemisphere lesions would cause impairment because of the nature and requirements of the test.

In Perret's study, the left hemisphere patients performed more poorly than the right hemisphere patients, and the left frontal group performed more poorly than any other group. Bilateral frontal involvement did not cause more impairment than a left frontal lesion. Perret therefore concluded that "the deficits are summed in the left frontal group" (p. 329).

This study provides no assurance that expressive verbal deficits, with or without frank aphasia, were not the basis for the results. The role of aphasia in limiting verbal productions has been known for many years (see Reitan, 1985), and as described previously, expressive verbal functions can be expected to be principally impaired with left anterior lesions.

Miceli *et al.* (1981) measured verbal fluency using a procedure identical to Benton's to evaluate patients with cerebral lesions. Patients who demonstrated dysphasia on their standard examination were excluded from the investigation. Of the 149 subjects used in this study, 70% had cerebral neoplasms, but the types of tumors were not identified. Left hemisphere lesions were in the following locations: frontal, 15; temporal, 7; parietal, 19; and occipital, 10. Right hemisphere lesions were as follows: frontal 22; temporal, 6; parietal, 15; and occipital, 11. Additional groups included subjects who had a lesion involving more than one lobe: left hemisphere, 13; right hemisphere, 31.

Miceli *et al.* presented means and standard deviations in tabular form, but the results of inferential statistical analyses were reported only in the text. Consequently, it was somewhat difficult to identify the significant findings on the verbal fluency test, but it appears that these investigators found that subjects with left hemisphere lesions performed somewhat more poorly than subjects with right hemisphere lesions ($p < .05$). No significant differences were found in comparisons of groups with lesions of individual lobes. Including patients with lesions involving more than one lobe did not change the results substantially, and the only significant finding (probability level not specified) represented a difference between the right frontal and right nonfrontal group. The results of this investigation differed from the other studies that showed deficits particularly in the left frontal group, apparently demonstrating the significance of screening out the dysphasic patients.

Pendleton *et al.* (1982) performed the most comprehensive study of the TWFT published to date. They evaluated 203 subjects with cerebral lesions (119 focal and 84 diffuse) and 134 normal paid volunteers. This type of control group does nothing to match groups for the general illness factor, anxiety, and other emotional stresses that may accompany hospitalization. Therefore, the influence that a serious illness may have on an individual's performance cannot be evaluated, and it is difficult to determine whether factors other than brain damage have had an adverse influence

on a subject's performance. Other researchers (e.g., Reitan, 1955b) have attempted to equate such factors by using control subjects who are ill and hospitalized but in whom a detailed neurological examination has ruled out past or present cerebral disease or damage.

In the Pendleton *et al.* study, the subjects with focal lesions were categorized according to lesion location: frontal, 47; frontal plus more extensive involvement, 33; nonfrontal, 39. The numbers were nearly equal when subdivided into left and right hemisphere lesions, left and right frontal lesions, left and right nonfrontal lesions, and total frontal and nonfrontal lesions. A diversity of types of lesions was included, providing the advantage of being able to generalize the results to a broad spectrum of neurological diagnoses. However, 35% of the patients with focal lesions had intrinsic tumors, and an additional 26% had either cerebral infarcts or hemorrhages.

These investigators used the procedure described by Milner (1964) in which subjects were asked to produce words beginning with S and C rather than the F, A, S words used in the Controlled Oral Word Association Test (Benton and Hamsher, 1976).

Unfortunately, in some instances comparisons of the various subgroups showed significant differences on age and education. The investigators performed an adjustment of TWFT scores according to age and education, a procedure whose effect is not fully known. In fact, after a careful evaluation of the use of analysis of covariance (ANCOVA) as a remedy for mismatch of groups on demographic variables, Adams *et al.* (1985) concluded that "ANCOVA should not be used in neuropsychological research to equate groups unequal on variables such as age and education or to exert statistical control whose objective is to eliminate consideration of the co-variate as an explanation for results" (p. 445).

The major findings of the Pendleton *et al.* study were as follows:

1. The TWFT results were significantly different at probability levels beyond .001 in comparisons of the normal subjects and any of the brain-damaged groups, and differentiated brain-damaged and control subjects with about 95% accuracy. This finding may be limited in its clinical significance, since it is rarely necessary to differentiate persons with brain lesions from persons who are healthy and have no complaints.
2. The TWFT showed more impairment with frontal than nonfrontal patients ($p < .05$), but because of overlap of the groups, the authors could derive no effective cutoff score to use in clinical application. It is even entirely possible that failure to control for dysphasia is the basis for this relatively weak finding.

3. While statistical data, as contrasted with clinical application, showed a mild difference between left and right frontal involvement, a comparison of the frontal subjects with the diffuse subjects (who had no focal lesions at all) showed no significant differences. The authors pointed out that the frontal groups would have to perform more poorly than the diffuse group for the TWFT to be considered an accurate indicator of frontal lobe lesions. However, there was no significant difference between these two groups on the TWFT, a finding similar to the results reported by Robinson *et al.* (1980) in their study of the WCST.

In summary, Pendleton *et al.* found some differences that exceeded the .05 level, but differences at this level imply a substantial degree of overlap of the groups being compared, and the TWFT, considered by itself, has limited significance in localizing frontal lobe dysfunction.

Overall Evaluation of the Wisconsin Card Sorting Test and Verbal Fluency Measures

A careful analysis of the literature that has given rise to using the WCST and the TWFT as frontal lobe indicators reveals that the evidence for such applications is tenuous. In addition, a recent comparison of groups with frontal and nonfrontal lesions, with types of lesions held comparable, yielded no significant differences in performance between the groups on the Category Test and Part B of the Trail Making Test (Reitan and Wolfson, in press).

Perhaps we should not consider these findings surprising. Several fairly recent reviews, published after the "validational" studies reviewed above (except for Anderson *et al.*, 1991), have concluded that there are no valid tests for identifying neuropsychological functions unique to the frontal lobes. Bigler (1988) stated that "clinical neuropsychology long has sought specific tests of frontal lobe damage but to date no such tests exist that are exclusively sensitive to frontal lobe function." He also noted that tests purported to evaluate frontal lobe function, such as the WCST and the Category Test, are not specific to frontal lobe damage.

Wang (1987) also reviewed neuropsychological functions supposedly related to the frontal lobes, and concluded that there are no tests specifically sensitive to frontal lobe damage or dysfunction.

Costa (1988) concluded that although many tests are sensitive to cerebral dysfunction, few of them are uniquely sensitive to particular cortical areas. Concerning frontal lobe dysfunction specifically, he wrote that "it is

easy to find tests that are sensitive to frontal-lobe dysfunction and very difficult to find tests that are specific for it!"

The many studies that reported significant deficits in groups of subjects who did not have frontal lesions should have forewarned us that the so-called frontal lobe tests were not specific to frontal lobe functions. For example, Parsons (1975) and Tarter and Parsons (1971) reported that long-term alcoholics showed various types of deficits on the WCST. Hermann *et al.* (1988) used the WCST to study a group of patients with complex partial seizures arising from either the left or right temporal lobe and a comparison group consisting largely of patients with primary generalized epilepsies. In apparent contrast to the "normal tendency" that Milner (1964) reported for patients with nonfrontal lesions, these investigators found that if the usual standards of frontal lobe involvement were applied, 57% of their subjects performed in a manner that would be considered suggestive of frontal lobe pathology.

The pervasive tendency among many psychologists to believe so strongly in certain tests as frontal lobe indicators has fostered a host of reports in the literature. As noted previously, recent publications have included studies that (1) draw specific conclusions about the deterioration of frontal lobe function with normal aging (without considering the possibility that there might have been just as great a decline on "nonfrontal" tests), (2) evaluate the frontal lobe influence on Piagetian concepts of cognitive development, (3) devise adaptation for children of frontal lobe tests (which have never been checked for validity despite the vast differences in child and adult neuropsychology [Reitan and Wolfson, 1992b; Rourke *et al.*, 1983]), and (4) assess the role of the frontal lobes in development of childhood abilities. Considering the findings reported in the original studies reviewed above, this use of tests as frontal lobe indicators almost seems to be a bizarre extension of the limited validity of these "frontal lobe" tests.

One could postulate that this pervasive use of frontal lobe tests occurs because "nature abhors a vacuum," and, as a result, there is a natural urge to attribute functions to the frontal lobes. In a well-known and often-cited reference, Teuber (1964b) referred to the "riddle of frontal lobe function," and everyone loves to solve a riddle. Or perhaps frontal lobe tests are gaining in popularity because of a cyclic variation over time — the last great surge of interest in the frontal lobes occurred in the 1930s and 1940s, and a new generation of psychologists has rediscovered the frontal lobes.

While any of these postulates may have some validity, the answer probably lies in the proliferation of articles and books that promulgate the role of the frontal lobes (e.g., Benton, 1968; Damasio, 1991; Heilman and

Valenstein, 1979; Levin *et al.*, 1991; Lezak, 1983; Luria, 1966; Milner, 1963, 1964, 1971; Stuss and Benson, 1984, 1986). With this barrage of publications, every definition and function of the frontal lobes cited above can find a friendly home. Though the end result of controlled investigations may be that the frontal lobes have no specific cognitive or intellectual functions, it is certainly true that they play an important role in the general higher level functions of the brain.

We noted many limitations of the studies reviewed above, including extremely small sample sizes, limited use of appropriate and necessary comparison groups, selectivity of lesion types when using a brain-lesion inferential model, permissive relaxation of probability levels and use of questionable statistical adjustments of raw scores, failure to describe and evaluate the effect of variables such as dysphasia, and emphasis on "statistical significance" even though the data did not support "clinical significance."

Many psychologists raise questions about the influence of the frontal lobes on the Halstead-Reitan Battery, and some of the questions specifically relate to the "frontal lobe syndrome." It is probably not surprising to hear questions of this type, particularly since Halstead (1947) felt his tests identified a much greater degree of deficit in patients with frontal lobe lesions than in patients with in nonfrontal lesions.

The results of Reitan's research on the Halstead-Reitan Battery, however, have not supported the hypothesis that there is more specific and severe impairment in persons with frontal lesions than in subjects with nonfrontal lesions (Reitan, 1964). In fact, Reitan's research indicated that the anterior frontal areas are related to the general indicators of neuropsychological impairment, whereas more posterior areas, in both the left and right cerebral hemispheres, subserve more specific functions depending upon which hemisphere is involved.

Nevertheless, there are some claims, particularly in the literature of behavioral neurology (but also in clinical neuropsychology), of specific deficits associated with frontal lobe impairment. How does this whole situation add up? Do frontal lobe lesions cause any deficits specific only to the frontal lobes? Do these deficits differ according to whether the left or right frontal lobe is damaged? What is the evidence to substantiate the claim that the frontal lobes are the seat of analytical reasoning and "executive functions"? At this point we will briefly review the history and background of studies of the frontal lobes in order to place the current status of the frontal lobe question in more proper perspective.

OVERVIEW OF THE HISTORY AND DEVELOPMENT OF CONCLUSIONS ABOUT FRONTAL LOBE FUNCTIONS

For many years there has been a great deal of interest in the frontal lobes. This is hardly surprising, if for no other reason than that the frontal lobes constitute such a large anatomical percentage of the cerebral hemispheres in humans. Second, although primary motor functions and coordination of motor functions are known to be controlled by the posterior frontal cortex, a large remaining (anterior) area of the frontal lobes seems to be involved in no specific behavioral functions, and is therefore a tempting target for placement of the elusive "highest" brain functions.

Since this is the case, and portions of the cerebral cortex posterior to the central sulcus are devoted to primary sensory reception areas (especially involving tactile, hearing, and visual functions), there has been a strong temptation among theorists to assign the higher level neuropsychological functions such as reasoning, planning, logical analysis, and executive functions (involving the final output of the brain with respect to organized and effective responses) to the anterior frontal areas.

Reitan and Wolfson (1992a) have reviewed the historical development of brain–behavior relationships, and because of the interest in the frontal lobes, many citations in this review were necessarily directed to the functions subserved by the frontal lobes. (In this paper we will not present a comprehensive review of frontal lobe functions, but the interested reader may refer to the introductory chapter of *Neuroanatomy and Neuropathology: A Clinical Guide for Neuropsychologists* by Reitan and Wolfson, 1992a, for a more complete discussion of this topic.) However, a brief review of the historical background is pertinent in attempting to provide answers to the above questions.

Fulton (1943) credited the French neurologist Flourens (1824) for conducting the first experiments involving the frontal lobes by performing ablations of the anterior frontal lobes in dogs. Most of the early studies, which did not localize any functions specific to the frontal lobes, supported a holistic theory of higher level aspects of brain functions.

Loeb (1902) found that ablations of the frontal cortex in dogs had few, if any, specific effects. He noted minimal impairment when he amputated only a single anterior frontal area, and found that both anterior frontal areas had to be ablated to produce some loss of alertness. Loeb argued that the frontal lobes had no special functions, even when bilateral frontal resection had been performed, and claimed that for a dog there was perhaps no operation as harmless as the removal of its frontal lobes.

Lashley's (1929) studies with rats provided no evidence of localization of specific functions in the cerebral cortex, as stated in his principle of

equipotentiality (any cortical area was equivalent to any other area). Nevertheless, he did find that maze-learning ability decreased as larger total areas of cortex were destroyed (the principle of mass action). However, considering the much more limited range of higher level behavioral capacity in lower animals than in humans, it might well be expected that experimental studies with lower animals would fail to demonstrate any obvious evidence of specific or regional localization of functions.

Broca (1861) attracted great attention from the scientific community by correlating impairment of verbal communication skills with damage to the posterior-inferior frontal area as well as adjacent cortex of the left temporal lobe. Broca, believing speech was dependent on the frontal lobe cortex, minimized the significance of the temporal lobe involvement. As a result, the base of the third frontal convolution has come to be known as Broca's area. Although this part of the cerebral cortex is still recognized as significant in the production of expressive speech, a considerably larger area, including a major portion of the left temporal lobe, has also been identified as a part of the language area (Penfield and Roberts, 1959).

Only a few years after Broca published his reports, Fritsch and Hitzig (1870) demonstrated that electrical stimulation of the posterior part of the frontal lobes in dogs induced motor movements on the contralateral side of the body. This discovery increased the impetus to pursue other locations of function in the brain.

Franz (1907) adopted standardized testing procedures to evaluate cats and monkeys before and after surgically imposing cerebral lesions. He concluded that the frontal lobes were particularly important for the solution of puzzle-box performances, and that unilateral lesions produced much less of an effect than bilateral lesions. Franz deserves credit for using standardized experimental tasks that met scientific standards of replicability. He concluded that deficits in the animals with surgical lesions would not have been noticeable using "simple observational methods," because such observations were not made under conditions that adequately and properly stressed the brain.

During the 1920s and 1930s, there was a continuous gradual evolution toward identifying the frontal lobes as the seat of intellectual functions. Gelb and Goldstein (1925) used a number of problem-solving tasks to evaluate human subjects, and concluded that patients with anterior frontal lesions were more impaired than subjects with lesions located elsewhere in the cerebral cortex.

In 1935 Ackerly described in detail a patient with bilateral anterior frontal atrophic lesions, and provided extensive information about the patient's emotional and intellectual deficits. Brickner (1936) published a book on the frontal lobes that cited detailed individual cases to substantiate his

conclusion that the highest levels of intellectual functioning were subserved by the anterior frontal cortex. Jefferson (1937a, 1937b) also described significant intellectual and behavioral deficits associated with lobectomies of both the left and right frontal areas in humans.

At about this same time two neurosurgeons, Moniz and Lima, introduced the surgical procedure of prefrontal lobotomy (prefrontal leukotomy; Moniz, 1936, 1937). This procedure involved sectioning the white matter pathways between the dorsomedial nucleus of the thalamus and the prefrontal cortex. Since the dorsomedial nucleus of the thalamus received projections from the hypothalamus, and the hypothalamus was believed very significantly involved in emotional responses, Moniz postulated that prefrontal lobotomy would limit the possibility of interference with intellectual functions served by the frontal cortex, functions that in some individuals might otherwise be diminished by strong affective and emotional arousal.

During the 1940s and early 1950s, prefrontal lobotomy was performed on thousands of persons who had principally psychiatric difficulties rather than problems for which the neurological bases were more clearly understood. Many clinical studies compared the pre- and postsurgical status of the patients, but standardized methods for evaluation of brain–behavior relationships in human beings were not generally used at this time.

Halstead *et al.* (1946) studied a series of patients before and after prefrontal lobotomy, and found variable neuropsychological test results. Although these investigators observed changes in their patients, it was not possible to predict precisely what the changes would be, and Halstead described the prefrontal lobotomy as a procedure in which "knowns" (the patient's preoperative condition) were traded for "unknowns" (the patient's condition after the surgery). By this time, the importance of the frontal lobes had been widely accepted by the scientific community. In 1947 Rylander, a Swedish psychiatrist, revealed his respect for the frontal lobes by describing prefrontal lobotomy as a procedure for "ablation of the soul."

It was in this scientific environment that Halstead developed standardized neuropsychological tests to evaluate the effects of brain lesions in human beings. In 1935, at the University of Chicago, he established the first full-time laboratory in the world for studying the effects of cerebral lesions on higher level brain functions. Fortunately, Halstead did not prejudice his selection of testing procedures; he developed standardized experiments that could be performed repeatedly in the laboratory. His experiments were initially based on his observations of routine, everyday tasks performed by persons with cerebral lesions that varied in location and type (Reitan, 1994).

Halstead must have felt a strong urge to identify and define the functions of the frontal lobes. In his studies involving the tests that he had

developed, he found that patients with frontal lesions performed much more poorly than patients with nonfrontal lesions. The subjects with frontal lesions also earned higher Impairment Indices. On the basis of these results, Halstead postulated a new concept of intelligence, called biological intelligence, that was dependent principally upon the frontal lobes. The final statement in his book, *Brain and Intelligence*, was that "the frontal lobes, long regarded as silent areas, are the portion of the brain most essential to biological intelligence. They are the organs of civilization — the basis of man's despair and of his hope for the future" (Halstead, 1947).

Halstead's work, together with the great interest in prefrontal lobotomy, may have been influential in bringing to a zenith scientific attention to the anterior frontal lobes. However, in the 1950s it became apparent that prefrontal lobotomies were not a cure for the emotional problems experienced by many psychiatric patients, and other investigators, such as Teuber (1964a), argued for the significance of impairment of nonfrontal as well as frontal cortex. The interest in the frontal lobes eventually subsided, though clinical neuropsychology had received a significant boost, and was on its way toward eventual recognition as a discipline in its own right.

It must be noted that behavioral neurologists gradually but persistently continued to investigate the relationship between the frontal lobes and behavior, and have had a considerable influence on the current conceptualization of the supposedly specific disorders associated with frontal lobe lesions. Lezak (1983) discussed frontal lobe disorders in some detail, devoting a section of her well-known book, *Neuropsychological Assessment*, to the topic. She provided a comprehensive analysis of frontal lobe functions that illustrates the difficulties implicit in this task.

Lezak began her review by noting that "the frontal lobes developed most recently to become the largest structures of the human brain. It was only natural for early students of brain function to conclude that the frontal lobes must therefore be the seat of the highest cognitive functions" (p. 78). While some early students may have reached this conclusion, even a brief review of the history of the development of understanding the frontal lobes tends to contest this statement, and indicates quite clearly that "early students of brain function" (as noted above and reviewed in Reitan and Wolfson, 1992a) often concluded that the frontal lobes had little, if any, significant function.

It is interesting that Lezak principally cited behavioral neurologists to support her statements about the function and importance of the frontal lobes. She noted that Hécaen and Albert (1978) wrote that "the frontal lobes regulate the 'active state' of the organism, control the essential elements of the subject's intentions, program complex forms of activity, and constantly monitor all aspects of activity." (p. 79). She indicated that Nauta

(1971), a neuroscientist trained as a neuroanatomist, characterized frontal lobe disorders as "the derangement of behavioral programming," (p. 79), and cited Luria (1966, 1973) and Nauta (1971) for her contention that the "cortex and underlying white matter of the frontal lobes is the site of interconnections and feedback loops between the major sensory and the major motor systems, linking and integrating all components of behavior at the highest level . . . . Thus, the frontal lobes are where already correlated incoming information from all sources — external and internal, conscious and unconscious, memory storage and visceral arousal centers — is integrated and enters ongoing activity." (p. 79).

Lezak cited Valenstein (1973) as differentiating between the behavioral effects of dorsolateral lesions and orbitomedial lesions. Dorsolateral lesions supposedly result in defects in the control, regulation, and integration of cognitive activities. Orbitomedial lesions, on the other hand, impair drive and affective integration centers in the diencephalon, and are therefore most apt to impair emotional and social adjustment. Girgis (1971) and Blumer and Benson (1975) have also suggested that these frontal lesions result in different personality and psychological consequences.

Lezak cited Luria (1970) as observing that Broca's area in the posterior-inferior portion of the left frontal lobe "receives information from both the posterior language area and the temporal lobe" and serves as "the final common path for the generation of speech impulses." A review of Luria's publication indicates that this statement represents his theoretical impression rather than a conclusion supported by any objective evidence.

Lezak reviewed Luria's impression that the right posterior-inferior frontal area reflects deficits of perceptual organization and planning (as represented by performances on the Block Design subtest of the Wechsler Scale). She further cited Shapiro *et al.* (1980, 1981) as indicating that impaired capacity to process musical elements such as pitch, rhythm, and phrasing tends to occur with right anterior lesions.

Reitan and Wolfson (1989) compared a group of subjects with left hemisphere lesions to a group with right hemisphere lesion and found that there were no differences between the groups on Seashore Rhythm Test performances. Conflicts of this type clearly identify the problems implicit in the area of behavioral neurology. Standardized experiments are usually not performed, and the examination of individual subjects provides the basis for most conclusions; the investigator observes the subject, and forms impressionistic conclusions about the individual's behavioral strengths and weaknesses. These circumstances provide a limited opportunity to employ one of the major hallmarks of the scientific method — namely, replication by other investigators. Nevertheless, in her review of frontal lobe disorders, Lezak principally cited behavioral neurologists who used these types of in-

vestigative methods (Hécaen and Albert, 1975; Luria, 1966; Nauta, 1971). As will be noted below, the methods of behavioral neurology are concerned principally with identifying specific deficits rather than the general, nonlocalized effects of cerebral lesions (Reitan and Wolfson, 1992a).

Lezak also categorized the types of behavioral difficulties associated with prefrontal damage. She noted that these problems may also occur with lesions involving other areas of the brain, but in such cases the patient's difficulties "are apt to be associated with specific intellectual, sensory, or motor disabilities." (pp. 80-81). This statement in its own right denies the specificity of deficits that might constitute a frontal lobe syndrome.

Nevertheless, Lezak next identified the "behavioral disturbances associated with frontal lobe damage [which] can be roughly classified in five "general groups," (p. 81), noting that the groups involve considerable overlap. First, Lezak noted that persons with frontal lobe damage have "problems of starting [which] appear in decreased spontaneity, decreased productivity, decreased rate at which behavior is emitted, or decreased or lost initiative." (p. 81). She referred to Goldstein (1944) to support this contention, but it must be noted that this particular publication by Goldstein concerns his evaluation of patients with traumatic brain injury in which neuropathological involvement has generally been recognized as diffuse (even though focal lesions may also be present). Thus, even though the frontal lobes as well as other cortical areas are often damaged in head injuries, this study by Goldstein is hardly the most appropriate for identifying specific frontal lobe deficits.

Lezak next referred to the findings of Luria (1966) and Milner (1964). It is well known that Luria's description of deficits was based principally on his personal observations, and as noted earlier, Milner studied patients with chronic, focal epileptic lesions, a group of subjects that provides a limited basis for generalization about the effects of brain damage.

Lezak's next point about frontal lobe damage concerned "difficulties in making mental or behavioral shifts, whether they are shifts in attention, changes in movement, or flexibility in attitude, coming under the heading of perseveration or rigidity." (p. 81). Although she identified this type of impairment as a frontal lobe deficit, she immediately controverted the specificity of this conclusion by indicating that these deficits also occur with lesions involving other areas of the brain. For example, Part B of the Trail Making Test, one of the tests frequently identified as a measurement of behavioral shifts in attention and flexibility, has clearly been demonstrated to be sensitive to impaired brain functions generally, regardless of lesion localization or lateralization (Reitan, 1955c, 1958). Specific comparisons of performances of groups with frontal and nonfrontal lesions yield no significant intergroup differences (Reitan and Wolfson, in press).

According to Lezak, the third specific deficit in patients with frontal lobe lesion relates to "problems in stopping — in breaking or modulating ongoing behavior — [and] show up in impulsivity, overreactivity, disinhibition, and difficulties in holding back a wrong or unwanted response, particularly when it may either have a strong association value or be part of an already ongoing response chain." (p. 81). This type of behavior has been reported in individuals with frontal lobe lesions, but no references were given to support this "specific" deficit. In fact, the behavior is not at all specific, is difficult to define in operational terms, and often is seen in patients with generalized brain impairment rather than only in persons with frontal lobe lesions.

The fourth point about frontal lobe damage concerns a deficient self-awareness. Lezak wrote that this deficit "results in an inability to perceive performance errors, to appreciate the impact one makes on others, or to size up a social situation appropriately." (p. 81). Again, no references are given to support this contention. It should be noted, however, that several investigators (Meier and French, 1966; Milner, 1954; Reitan, 1955a) have commented that impairment in the ability "to size up a social situation appropriately," as manifested by explicit results on instruments such as the Wechsler Picture Arrangement subtest and the McGill Picture Anomalies Test, is related to nonfrontal rather than frontal lesions.

Finally, Lezak noted that "a concrete attitude is also common among patients with frontal lobe damage." (p. 82). She referred to (1) Goldstein (1944, 1948), whose studies were based upon patients with traumatic brain injuries, and therefore probably do not represent lesions restricted to the frontal areas; (2) Landis (1952), whose studies were based upon chronic, deteriorated schizophrenics who underwent cortical topectomy, and are scarcely relevant for purposes of generalization; (3) Teuber (1964), who also studied head-injury cases and based his inferences about localization on the point of impact to the skull and the evidence of underlying cortical damage; (4) Lezak (1978), who described her impressionistic observations in an article entitled "*Living with a Characterologically Altered Brain-Injured Patient*"; and (5) Milner (1971), whose sample of patients with focal, long-standing epileptogenic lesions, subject to surgical excision, are limited in their representation of patients with brain damage in general.

Lezak noted a number of other references that also have problems regarding their nonspecificity for frontal as compared with nonfrontal involvement. These references characterize patients with frontal lesions as having an attitude that takes objects, experiences, and behavior "at their most obvious face value. The patient becomes incapable of planning and foresight or sustaining a goal-directed behavior." (p. 82). In general, frontal patients show evidence of "apathy, carelessness, poor or unreliable judge-

ment, poor adaptability to new situations, and blunted social sensibility." (p. 83). Anyone who has studied persons with cerebral lesions, frontal or nonfrontal, has seen these deficits demonstrated, regardless of the location of the lesion. In addition, such deficits occur frequently among persons with diffuse or generalized cerebral damage (such as Alzheimer's disease), even when there is no specific or isolated involvement of the frontal lobes.

It must be noted that Lezak (1983), in a final section concerning the effects of frontal lobe lesions, commented that "the uncertain relation between brain activity and human behavior obligates the clinician to exercise care in observation and caution in prediction, and to take nothing for granted when applying the principles of functional localization to diagnostic problems." (p. 84). This final paragraph seems to caution the reader concerning generalizations about function attributed to the frontal lobes as well as the "limitations of its applicability of the individual case." (pp. 83-84).

The above review of Lezak's five criteria, while presenting some of the current generalizations about the frontal lobes, also reveals the great difficulties inherent in postulating a theory of frontal lobe functions.

There are many other reviews of frontal lobe functions, several of them overlapping in their content. Jouandet and Gazzaniga (1979) cited a number of the references noted above in our review of validation studies in addition to reviewing the anatomy of the frontal lobes, connections with other ares of the brain, and surgery (especially prefrontal lobotomy) for psychiatric disorders. Citing Hebb (1939), Mettler (1949), and Teuber (1959), Jouandet and Gazzaniga contrasted the earlier tendencies to attribute "the most stupendous of cognitive capabilities" to the frontal cortex with a reaction contending that no specified functions could be attributed to the prefrontal areas — that there was no "indisputable evidence suggesting that there existed any cognitive processes subserved exclusively by the prefrontal regions" (p. 26). In their analysis of the studies of Milner, Benton, and others cited above as well as various additional publications, Jouandet and Gazzaniga concluded that "the frontal lobes have come to contribute in their later stages of phylogenetic evolution to the limitless analytic powers of the human psyche by superimposing on the three spatial dimensions a profound mastery of the fourth dimension of time" (p. 54).

In the same volume, Goodglass and Kaplan (1979b) reviewed the traditional functions attributed to the frontal lobes (such as lack of drive and spontaneous expression, socially inappropriate behavior, impairment of flexibility, and lack of ability to plan sequential aspects of behavior), and identified the Wisconsin Card Sorting Test, verbal fluency procedures, and the Stroop Test as examples of frontal lobe tests.

In an analytical review, Damasio (1985) integrated clinical and neuropsychological deficits of patients who have frontal lesions with anatomical considerations. The impact of this analysis, however, was diminished by a tendency to accept the rather weak statistical results on tests such as the WCST and the TWFT as invariant and specific characteristics of frontal lobe damage.

Stuss and Benson (1984) also reviewed neuropsychological studies of the frontal lobes, using headings of (1) motor functions; (2) sensory, perception, and construction functions; (3) attention; (4) syndromes of abnormal awareness; (5) flexibility-perseveration; (6) language, including various types and manifestations of aphasia; (7) memory; (8) cognitive functions; (9) frontal lobe personality; and (10) localization and hemispheric asymmetry of frontal lobe function. These authors described many changes or deficits supposedly resulting from frontal lobe lesions, but communicated a mixed message when they concluded that "current explanations of apparent frontal lobe malfunction remain limited and vague," and "it would appear that a few specific prefrontal functions can be extracted. These are broad and vague and certainly incomplete . . ." (p. 22). One must wonder about the authors' use of the term "specific functions," described in the next sentence as "broad and vague." Stuss and Benson provided a comprehensive review of the literature, couching their conclusions about frontal lobe damage in qualified terms (which do not necessarily identify the deficits as exclusively frontal), and concluded that "the neuropsychologic picture secondary to prefrontal pathology is a complex and confusing set of behaviors" (p. 23).

A recurring observation in theoretical and review papers on the frontal lobes, mentioned by Stuss and Benson, is that the cognitive deficits experienced by persons with frontal lesions are difficult to measure with psychological tests, and that improved or more appropriate tests will likely lead to further and clearer exposition of these subtle deficits (Hart and Jacobs, 1993; Levin *et al.*, 1991; Lezak, 1993; Varney and Menefee, 1993). The vagueness with which the effects of frontal lesions are described and the "bewildering array of behavioral deficits [that] has been attributed to frontal lobe injury" (Goldman-Rakic, 1993, p. 13), may also be factors contributing to the apparent overlap (to the point of essential identity?) with the effects of other cerebral lesions.

When one considers the many differences among patients with cerebral lesions in terms of location, severity, course of recovery or deterioration, etc., it is difficult to organize, with any degree of precision, selective, impressionistic, clinical descriptions of deficits of individual subjects. In contrast, readily identified (specific) deficits are often prominent in persons with nonfrontal lesions, a fact that may tend to promote a presumption of

understanding of these patients. Among frontal patients, however, the specific deficits are less prominent, even though the general deficits are pronounced. The critical need may therefore be to gain further insight and understanding of the general neuropsychological deficits, over and beyond specific deficits, that are common to persons with both frontal and nonfrontal lesions (Reitan and Wolfson, 1994). The field of behavioral neurology has focused principally on the study of specific deficits, whereas nearly all of the tests of generalized neuropsychological impairment have their roots in clinical neuropsychology (see Reitan and Wolfson, 1992a and 1993, for a more complete discussion of general and specific neuropsychological tests together with their differential origin and use in clinical neuropsychology and behavioral neurology).

Mountain and Snow (1993) presented a detailed analytical review of research publications and the potential for clinical applications of findings using the Wisconsin Card Sorting Test. Their review was comprehensive rather than selective, and included a number of studies not mentioned in this paper. These authors concluded, as we did, that the evidence is weak that patients with frontal lesions, when compared to patients with nonfrontal lesions, perform more poorly on the WCST, and that clinical findings do not support the use of the WCST as a frontal indicator. In addition, Mountain and Snow cautioned against using the WCST as a marker of frontal lobe functions for research purposes — apparently, as indicated in the introductory section of this paper, a tendency of striking prevalence.

The recent volume edited by Levin *et al.* (1991) provides a comprehensive synthesis of research on the frontal lobes, and reviews many areas and types of deficits. Many topics of interest are addressed, but are presented in a research rather than a clinical framework. For example, the most recent comprehensive studies comparing frontal and nonfrontal groups on the WCST (Robinson *et al.*, 1980) and the TWFT (Pendleton *et al.*, 1982) are not mentioned or referenced, even though they were published years earlier, and were omitted probably because the focus of this volume was directed toward experimental rather than clinical neuropsychology.

The numerous theoretical and review papers on the frontal lobes, as well as other secondary sources, appear to have had a significant influence on many psychologists. This type of influence was exemplified by Knopman *et al.* (1990), who reported on their examination of patients who had dementia without distinctive histological features. The principal point of interest of this study was that the subjects did not qualify histologically for a diagnosis of Alzheimer's disease. Nevertheless, they had memory loss and personality changes, in the context of an illness that lead to death, usually within two to seven years.

The patients in the Knopman *et al.* study had been examined with "mazes, trail making, and word fluency." (p. 253). The psychometric findings were interpreted as being "consistent with a 'frontal' lobe dementia" (p. 251), a conclusion reached on the basis of the fact that these tests measured "frontal based functions." (p. 253). Nevertheless, the authors characterized their patients as having rather generalized cerebral involvement, including "degeneration of the cerebral cortex, which was most severe in the frontal and parietal cortex, less severe in the temporal cortex, and generally absent in the occipital cortex." (p. 252). The authors concluded that the findings were consistent with a "frontal" lobe dementia, apparently because of the psychological examinations that had been performed and the attribution in the literature that the tests used to examine the patients reflect frontal damage.

It is apparent that the generalizations and conclusions communicated by prominent neuropsychologists tend to be accepted rather uncritically by psychologists as well as other professionals. This highlights the responsibility of researchers to be accurate in reporting their findings, particularly when making specific statements about the behavioral correlates of pathological conditions such as frontal lobe damage.

One of the tests used by Knopman *et al.* was the Trail Making Test, a measure with which Reitan has probably had more clinical and research experience than anyone. The fact is that the Trail Making Test is sensitive to generalized cerebral damage rather than damage specific to the frontal lobes (Reitan, 1958), but the assumptions of these investigators had been based not on the facts, but instead on an unsupported belief that the Trail Making Test, because of its requirements, must fit within the range of abilities described theoretically as relating to frontal lobe functions. A recent study confirms that Part B of the Trail Making Test is equally sensitive to frontal and nonfrontal cerebral lesions (Reitan and Wolfson, in press).

Finally, we will refer specifically to Halstead-Reitan Battery results obtained by patients with frontal and nonfrontal lesions. As noted above, Halstead felt that the ten tests that he developed, and which provided a basis for computing the Impairment Index, were much more adversely affected by frontal than nonfrontal damage. A considerable debate ensued after Halstead (1947) reported this conclusion, with Teuber in particular contending that nonfrontal lesions were at least equally as impairing as frontal lesions. In his book, *Brain and Intelligence*, Halstead (1947) published neurological summaries as well as the actual test scores of the patients used in his studies. Thus, it is possible to inspect his findings in detail with relation to results reported by many additional investigators.

Halstead's data provides the basis for a remarkable insight. His patients with frontal lobe lesions generally performed quite poorly, document-

ing the fact that frontal lobe damage is associated with significant cognitive impairment. His statistical comparisons also indicated that the patients with frontal lobe lesions performed more poorly than the subjects with nonfrontal lesions. It was therefore of interest to study the results for the individual subjects in the nonfrontal group. It was the results on his nonfrontal patients, rather than his frontal patients, that disagreed with later findings by other investigators. Halstead's nonfrontal patients performed extremely well on the tests compared to thousands of patients tested by Reitan and other investigators.

Thus, the unusual aspect of Halstead's findings is not the results of his frontal patients, but the performances of his nonfrontal patients. The frontal patients showed evidence of impairment, just as essentially every investigator had found. However, his nonfrontal patients usually performed considerably better than nonfrontal subjects studied by later investigators. In fact, Reitan's data indicate that, in an overall sense, patients with frontal lesions perform approximately equivalently to patients with nonfrontal lesions, and both groups demonstrate significant impairment compared with non-brain-damaged control groups (Reitan, 1964).

Frontal subjects differ from nonfrontal subjects in some basic respects. Frontal patients routinely demonstrate generalized neuropsychological impairment, but usually do not show any very specific higher level deficits. Nonfrontal patients, however, often demonstrate impairment on tests that involve auditory, tactile, and visual perception, and frequently have dysphasic disorders (particularly with left cerebral lesions) and visual-constructive deficits (particularly with right cerebral lesions). Therefore, patients with posterior lesions may exhibit indications of general impairment together with more specific and lateralizing findings, whereas patients with frontal lesions show indications of general impairment but often do not demonstrate specific deficits. As Reitan and Wolfson have emphasized repeatedly, a comprehensive neuropsychological assessment must include both general and specific indicators of neuropsychological deficits, not only to identify localized damage, but to also provide a thorough assessment of an individual's cognitive structure (Reitan, 1988; Reitan and Wolfson, 1986, 1988, 1993, 1994).

A historical review of frontal lobe studies suggests that the "riddle of frontal lobe functions" may be a result of methodology rather than content. Promising leads about specific frontal deficits essentially lost their specificity when more adequate and carefully controlled studies were performed. Persons with frontal lobe lesions may demonstrate generalized neuropsychological impairment without any specific deficits (aside from dysphasic symptoms). If this is true, the search for specific deficits, fueled by vague theoretical descriptions, could continue forever quite fruitlessly. The meth-

ods of behavioral neurology, with a focus on the individual person, are not well suited to identifying general neuropsychological impairments, and will likely lead, on a case-by-case basis, to a further elaboration of the "bewildering array" of deficits that have already been described.

In attempting to gain greater understanding of the function of the frontal lobes, it may be necessary to concentrate on general, rather than specific, neuropsychological tests (Reitan and Wolfson, 1994), and such general measures have been derived almost entirely from the field of clinical neuropsychology rather than behavioral neurology (Reitan and Wolfson, 1993). It appears that such an approach would also lead investigators to recognize that other cerebral areas share essentially all of the higher level cognitive functions of the frontal lobes.

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