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NEUROPSYCHOLOGICAL AND SOCIAL MALADJUSTMENT PATTERNS IN LEARNING DISABLED CHILDREN

DISSERTATION

Presented in Partial Fulfillment of the Requirements for the Degree Doctor of Philosophy in the Graduate School of The Ohio State University

By

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* * * *

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1976

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Introduction

The literature on learning disabilities generally shows a lack of consensus on the definition of the problems and persons under investigation. Often the terms learning disability and minimal brain dysfunction are used synonymously. In addition, such children may also show hyperkinesis and/or behavioral disturbances. Bannatyne (1971) discusses the inter-relatedness of these descriptors, viewing learning disability as the most inclusive of these terms.

It is likely that studies of children with any one of those labels will include subjects who might accurately be classified under any of the other terms as well.

This investigation treats the term learning disabled as the centrally important descriptor. Children may be viewed as being learning disabled if school achievement is markedly lower than psychometric intelligence, or if one or two academic skills are deficient in comparison to most other skills (Lyklebust 1968). In addition, the child must not be mentally retarded. Reading disability or dyslexia is the most common specific disability, and as such overlaps the classes of learning disabled (LD) and minimal brain dysfunction (MBD) children.

A list of the classical behavioral symptoms associated with MBD will further describe these children. This represents a composite of descriptions (Strauss & Lehtinen, 1947, Strauss & Kephart, 1955, Cruikshank, Bentzen, Ratzberg, 1961, Stock, 1969, Wender, 1971): distractibility,
hyperactivity, disorganization of experience, stimulus binging, disinhibition of inappropriate motor acts, perseveration, figureground confusion, disturbed body image, impulsivity, aggressive, attention seeking, easily frustrated, cognitive rigidity or concretism, pseudo-neurotic, psychotic, psychopathic, and specific learning disabilities.

Frostig, Whittlesey, and LeSever (1961) estimated that 10 to 20% of all children have some of the characteristics of the brain injured child. Wender (1971) believes it is the most common disorder seen by child psychiatrists. Expanding upon the terms used in the above studies, Eisenberg (1965) stated that 10 to 50 per cent of the school age population has reading disability.

It is the hypothesis of the present author that ISD is one etiology of learning disabilities, and that ISD is not unitary in character nor is it the sole etiology. Many labels may be given to LD children. These labels may be based upon presumed etiologic agents or measured academic disabilities and will reflect the theoretical biases of the professional doing the assessment.

While the number of labels applied to the children may be confusing to experimenters, it is assumed that the terms are meaningful to schools and clinics, else they would not be in use. However, it would be foolhardy to assume that useful labels necessarily imply that such children are part of an homogenous group. By factor analyzing the data to be collected in this study, it is believed that sub-
groups of related symptoms and/or children will be identified. Basic dysfunctions found in these children could lead to a deeper understanding of the neuropsychological dynamics of learning disability and of the label as a sociological phenomenon.

The issue being addressed in this study is more a search for the right questions to be asked in the future than it is for the right answers presently. This investigation deals with symptoms of neuropsychological deficit and social maladjustment in children designated as learning disabled. It is hoped that meaningful patterns of symptoms will emerge from the data analysis, patterns useful in understanding the dynamic basis of learning disability. By discerning these clusters of symptoms, it may become possible to reduce the chaotic multitude of labels and to form more reasonable sub-groups on the basis of empirical evidence. At that point, it is likely that researchers and educators will be able to investigate etiologies and treatments for children with similar problems.

Specifically, this research attempts to delineate various neuropsychological mechanisms and emotional problems found in LD children. It is believed that several types of cerebral dysfunction underlying LD and independent emotional maladjustment problems will be identified.
Hypotheses

1. Learning Disabilities can be caused by a number of different neuropsychological dysfunctions, acting alone or in various combinations.

2. Emotional disturbance, as evidenced by BSAG results, may be present with or without neuropsychological deficit in LD children.

3. LD children may be grouped into a classification system on the basis of common problems among sub-groups.

Since the late thirties, when Werner and Strauss (1941) identified a group of perceptually disordered, learning disabled, hyperactive children whom they categorized as being "brain-injured", much attention has been focused on this group. While conventions in nomenclature and diagnosis have changed often, there remains a lack of underlying theory or of order in psychological and educational treatment. In the early literature, these children were viewed as a unitary group with common symptomatology (Strauss and Lehiten, 1947). This view persists today among educators and in research which deals with LD children in general terms (Dykman, Ackerman, Clements, and Peters, 1971).

Unfortunately, this approach fails to account for the wide range of symptoms and their possible causes. For example, Luria (1974) notes many possible avenues by which language learning is impaired in damage to separate brain systems. Bannatyne (1971) suggests that learning disabilities can have multiple causes which may act in isolation, in concert, or sequentially. Among these etiologies are:

Primary Emotional Dylexia due to inadequate parental sti-
ulation, Minimal Neurologic Dysfunction or (Minimal Brain Dysfunction), Genetic Dyslexia or reduced inherited capacity to learn language skills, Socio-cultural Deprivation, and Emotional-motivational disorders.

This study attempts to establish the heterogeneity of LD children empirically. The existence of specific types of cerebral dysfunctions can be identified with the measures to be used. In addition, social adjustment problems may be found to occur independently of, or concurrently with neuropsychological deficit. The inter-correlations between patterns of learning disability, neuropsychological dysfunction, and social maladjustment should lead to a fruitful system for classifying the bases of learning disability and for devising psycho-educational treatment.

Beyond simply identifying symptoms, the instruments to be used allow the clinician to analyze the mechanisms responsible for the disability. It is further intended that this analysis will make it possible to distinguish various possible foundations of LD from each other, for example, auditory versus visual analysis deficits. In this manner, emotional problems may be found in some children to be the only correlate of LD, while in others a specific type of emotional problem may always be associated with cerebral dysfunction. This sort of finding may help us to distinguish primary behavior disorder from that which is secondary to brain dysfunction.

The separation between research and clinical inference is indeed tenuous in this case. The factor analytic method
to be employed can only give an indeterminant solution. It remains the experimenter's task to name the clusters of related variables, integrate the findings into individual reports for the children, and ultimately to attempt a classification system which will bring some degree of order into the way in which LD children are treated educationally and in research.

At the base of the confusion in the literature would seem to be the original Straussian notion (Strauss and Lehiten, 1947) which held brain damage to be an homogeneous diagnostic category and the etiology of LD. However, Strauss lived in a wholistic world, heavily influenced by Kurt Goldstein. In that world, one might conceptualize the brain as working as a homogeneous whole, excluding the primary sensory and motor areas. It was meaningful, therefore, for Strauss and Goldstein (Goldstein 1942) to speak of qualitative changes in brain function after damage. This made the term 'brain damage' a meaningful, all-or-none diagnostic category for many psychologists.

Reviewers of psychological deficit in the Annual Review of Psychology (Reitan, 1962, Yates, 1965, Zimet and Fishman, 1970) have all commented upon this issue. Reitan (1962) believes that the notion of brain damage, without further qualifications, as being meaningful was attributable to the fact that research in the area was in its infancy. In addition, he feels that a greater proportion of children than adults sustain the kinds of damage which render the
ensuing dysfunctions assessable by impressionistic methods alone.

Much of the confusion in this area does in fact stem from Strauss' identification of brain-injured children by behavioral-impressionistic techniques. Zimet and Fishman (1970) report a dearth of experimental studies and an overabundance of clinical-impressionistic studies in the LBD literature. This is understandable when one considers the fact that readily identifiable brain lesions are usually not present in learning disable children. Instead, certain behaviors serve as the basis for much of the research; i.e., hyperkinesis, dyslexia, distractibility, impulsivity, perseveration (Stock, 1969).

In a brilliant criticism of the problem of brain damage in children, Birch (1964) states that the fact of brain damage is structural. Damage may or may not lead to cognitive, perceptual, or motoric sequelae depending upon age of onset, area damaged, and environmental factors. The concept of brain damage in children, on the other hand, designates a pattern of deficits, originally identified by Strauss as distinguishing exogenous from endogenous retardation. Since it is a concept, rather than a structural-anatomical finding, it could conceivable apply to children who have not suffered brain damage, and might not apply to others who have.

The literature review will show that Strauss' concept
of brain injury was discovered by psychological means, and that psychological assessment is still the best means for diagnosing this syndrome or syndromes. First, the lack of clear, medical indicators of minimal brain dysfunction will be demonstrated. Then, a review of psychological assessment of brain damage will be undertaken. Next, findings regarding developmental aspects of brain damage and neuropsychological characteristics of LD children will be explored.

After that section, we will examine current theory and research in learning disabilities. Special emphasis will be given to work dealing with emotional problems and social maladjustment in the subject population.

The reason for this emphasis is twofold. In a pilot study, the present author (Schachter, 1974) found a cluster of learning disability symptoms which occurred independently of neuropsychological deficit variables. This hinted at the possibility of children being labeled as LD on the basis of school maladjustment and subsequent poor academic achievement without cerebral dysfunction. Secondly, previous studies on learning disabled children using neuropsychological instruments have excluded children with emotional problems (Rourke, 1975; Doebring, 1983). While this is justified for experimental purposes, the present author believes that clinically it is useful to assess emotional disturbance and theoretically it is necessary for a full understanding of the range of problems encountered by this population.
CHAPTER 3
Review of the Literature

The reader will note that among the symptoms of KBD listed in the introduction no gross sensory or motoric disturbances are listed. This is in keeping with the denotation of a minimal dysfunction. However, since most theorists postulate some brain dysfunction to be present in KBD, medical practitioners are often given the task of trying to make a diagnosis on the basis of neurological symptoms. Most frequently, signs of KBD fall under the rubric of soft neurological signs. Unlike classical or hard signs, such as anesthesia, paralysis, or hyperreflexivity, soft signs do not localize lesions. Instead, they reflect upon the general state of integrative functioning of the central nervous system.

Among the soft signs are unsustained ankle clonus, general motor clumsiness, fine tremor of fingers, dysdiadochokinesia, directional confusion, strabismus, asymmetrical or hyperactive deep tendon reflexes, tics, intention tremors, inconsistent Babinski reflexes, and inability to stand or hop on one leg (Clemmons & Glasser, 1987). These findings may be inconsistently present, open to subjective appraisal, and rarely have prescriptive value (Werry, 1983).

Various studies have implicated different soft signs as discriminating KBD children from controls. Vukovich (1988) found that KBD Ss were unable to imitate hand or finger movements or to accurately reproduce vertical or horizontal tongue movements. Difficulty in complex postural
Tasks and presence of asymmetrical reflexes were also noted. Zyklebust, Boshes, Olson and Cole (1962) noted difficulties in producing tongue movements and presence of left-right graphesthesia. Dykman, Ackerman, Clements and Peters (1968) performed a factor analysis of neurological signs, finding 24 that did discriminate controls from MBDs on the basis of the following factors: fine and gross coordination in upper and lower limbs, face and tongue movements, right-left confusion, mixed laterality, and tendon reflex abnormality. High overlap between factors was reported.

Kenny, Clemmens, Hudson, Lentz, Cicci, (1971), reported that fine motor coordination was the best discriminator between control and MBD groups. Wikler, Dixon, and Parker (1970) found dysdiadochokinesis and inconsistent Babinski signs to be the most discriminating neurological criteria.

It must be noted that the presence of one or more soft signs is not pathognomonic of MBD. Wikler et al (1970) found that 20 of 24 control Ss had one or more such signs. Only group averages revealed clear differences. Kennard (1966) found that all Ss in the group of MBD adolescents with whom she had worked had one or more soft signs, but so did 70% of a control group. The group averages were 5 and 1 signs respectively. These findings illustrate both the equivocal nature of soft signs, as no one sign is always present in MBD, and the large percentage of false positives using purely neurological criteria. Soft signs would appear to be of little value for psychoeducational planning.

Less subjective means of assessing the neurological status of these children, such as electroencephalography,
have also produced equivocal findings. Hughes (1969) reports that the most common EEG findings in learning disabled children include long bursts of slow waves, usually coming from the posterior cerebrum, and positive spike discharges of 6 to 7 or 14 per second. These findings are supported by others (Benton & Bird, 1963, Muehl, Knolt and Benton, 1965).

These wave forms are also commonly found in normal children. In a study by Capute, Niedermeyer and Richardson, 1968) 106 Ss diagnosed as MBD were given EEGs. Positive spikes were found in only 8 of the records, while the authors estimated that across all children 20% might show this pattern. Hughes (1971) reported slow waves in 22% of a learning disabilities group and 13.3% of a control group. In the same study, positive spikes were found among 19.9 and 15.2% of the same respective groups. Hughes concluded that other EEG studies which find significant inter-group differences actually hide the fact that current EEG techniques do not provide accurate differentiation between individuals. Furthermore, the EEG does not contribute anything to the design of educational programs. Its main advantage would appear to be the detection of distinctly pathological conditions, such as epileptic or neoplastic foci.

Sohwalb (1967) states that the physician should consult a psychologist before making an MBD diagnosis. Historically it has been the psychologist's tools which have identified organic behavior patterns. MBD, learning dis-
abilities, and the other labels in use are clearly more behavioral than medical, both conceptually and functionally. As such, LBD is more readily, but not unequivocally identifiable through behavioral-functional instruments. Test construction, however, seems to be dependent upon the author's view of cerebral organization, especially among tests for brain damage. This in turn will determine which behaviors will be examined and how the child's functioning will be conceptualized. Since learning disability is believed by many, including the present author, to be the manifestation of some brain dysfunction in at least some proportion of children, and since the pioneering work of Strauss and Werner (1941) assumed brain injury to be a causative factor, it will be necessary to review the pertinent literature on psychological assessment of brain damage in children.

Methodological Difficulties

Methodological difficulties in this field have been reviewed by Hebb (1945). The lesioned area may have less influence on the patient's behavior than side effects of the lesion upon neighboring cerebral structures. It is often impossible to identify the original site of lesion, even if autopsy results are available. The present author notes that in LBD, where there is little evidence to presume gross morphological lesions, and where developmental level of the subject must also be taken into account, the difficulties are further amplified.

A major figure in the development of psychological
tests for brain damage was Kurt Goldstein. Goldstein believed that symptoms of brain damage produced unitary, qualitative changes in brain functioning. Goldstein (1942) notes the following bases for symptom formation: psychogenic symptoms arising from the individual's struggle to cope with impaired abilities, taking the form of avoidance of novel situations and rigid ordering of the environment; direct or negative symptoms resulting from loss of cortical tissue, paralysis and anesthesia being examples; and positive symptoms caused by the de-differentiation of lesioned areas from the gestalt activity of the brain as a whole. The latter are described as positive symptoms because they are reflected in new, emergent properties, or cognitive strategies. In particular, the brain damaged individual is seen as being rigid and concrete in his thinking, unable to generate rules, shift response sets, or in general assume abstract attitudes.

Goldstein envisioned a qualitative, dichotomous distinction between the functioning of the healthy versus damaged brain. This led to the designing of tests which differentiated concrete from abstract thinkers on a very gross level. Furthermore, there was no call for specifying variations in deficits as a function of site of lesion. Taking Gestalt theory as his stepping stone, Goldstein believed that during normal activity there is a background of on-going neural firing, and a figure of special activity
within the ground which represents stimulus being processed at the moment. Normally, figure and ground are distinct, both in the neural substrate and in the individual's subjective world. Pathology alters this figure-ground relationship, creating perceptual inadequacy. This view was heavily stressed and employed later on by Strauss.

Goldstein's own tests are mainly of the sorting type and are designed to detect concrete versus abstract reasoning. Descriptions of these tests are from Anastasi (1968) and Goldstein (1943). The Goldstein-Scheerer Cube Test is a modification of the Kohs Block Test. The patient must reproduce printed designs using colored blocks. Like the other Goldstein tests, norms are not used, and most of the work done with this test dealt with adult, war wounded Ss. Pathognomonic signs include perseveration, and inability to use clues provided by the examiner.

The Weigl-Goldstein-Scheerer Color-Form Sorting Test involves small shapes reproduced in several colors. The patient is instructed to sort these any way he wishes. After the first sort, he must re-sort the shapes in a different manner. Inability to do so is indicative of concretism and brain damage. Other tests, using similar inferential procedures and theoretical base included the Goldstein-Scheerer Stick Test and the Goldstein-Scheerer Object Sort. Validation studies of these tests in diagnosis of brain damage have produced meager results according to Anastasi (1968).
Dunn (1932) describes the work of Strauss and his collaborators as the extension of Goldsteinian theory into the area of child development. Strauss and Werner began their work in the late thirties, with the former continuing until the fifties. Like Goldstein, Strauss adopted a unitary dysfunction view of brain damage in describing a behavioral syndrome in children, the Brain-Injured child, which is the forerunner of the present LBD and LD classifications.

Strauss believed that these children had suffered some form of brain damage and that the perceptual-cognitive sequelae differed little from those found in adults, as described by Goldstein (Strauss & Werner, 1941). These include figure-ground confusion, perseveration, and concretism. Strauss also noted the presence of hyperactivity and other, now well known symptoms of the LBD child. Although Strauss does caution against making a diagnosis on the basis of behavioral evidence alone, his concepts are more firmly anchored by such data than by neurological findings.

Strauss did place emphasis on an important cause of childhood learning problems, brain damage. Although the concept of brain damage may be looked at far more critically than he thought necessary, many authors have accepted it as one cause of LD. Passamanick and Lillienfeld (1955) postulated a continuum of reproductive casualty extending from severe, fatal brain damage to minor neurologic dysfunction. Some learning disorders may be attributed to pre- and paranatal CNS insult.
Educators have placed emphasis upon devising programs for the brain-injured child. Owing to Straussian influences, tests which screen children solely on perceptual tasks have become popular diagnostics for WBD or brain damage in school age children. The tests developed by Strauss illustrate the extent to which theory determines test content and therefore analysis of deficit.

Strauss and Werner (1941) devised a formboard test in which children had to copy arrangements of marbles placed on a board with a confusing background design. Brain-injured Ss did more poorly than endogenous retardates on this task. This was viewed as evidence for figure-ground confusion. Werner and Strauss (1943), believing that imagery was a primary thought process, examined this function with the Picture Object Test. The child is shown a picture and given a collection of toys which he is told to group before the picture. It was found that brain-injured children tend to think animistically, expanding upon the theme of the picture in space and time.

Unfortunately, work did not expand into studies of other modalities under the Strauss group. One exception to this was a study by Werner and Bowers (1941) on sound perception. Brain-injured Ss were less able to vocally reproduce melodies they had heard than were endogenous retardates. Had work been continued along this line, the undue stress on visual perception problems and the notion
of brain damage as a unitary entity might have been abated.

The study of Strous and Werner (1942) uses one of the many visual perception and reproduction tests which have been employed to detect brain dysfunction. These tests work under the assumption that perceptuo-motor integration is a valid indicator of cerebral integrity (Zimet and Fishman, 1970). Probably the most widely used test of this type is the Bender Visual Motor Gestalt Test, commonly called the Bender-Gestalt.

In the Bender-Gestalt (BG), 8 geometric figures or designs are presented to the child one at a time for copying. Many scoring systems have been devised for various screening purposes such as intellectual level, emotional disturbance, and organicity. Some of these systems have been reviewed by Cresson (1964). These include the Pascal and Sutell (1951) and Clawson (1962) systems of scoring. Reliability and validity studies have generally produced unimpressive results (Cresson 1964).

Billingslea (1963) reviewed the work published on the BG and reported the most common error found among brain damaged children was rotation of the figure being copied. Kosher and Smith (1965) compared the usefulness of two objective scoring systems for identifying damage, the Hein and Peer-Quast methods. The two systems hold different signs to be pathognomonic. Neither method proved effective in screening individual Ss for brain damage, although group
differences between control and lesioned groups did appear. Both methods identified 70% or more of the control group as being brain damaged. Kelpin (1955) reported that rotation errors alone did not distinguish brain-injured from endogenous retarded Ss.

Elizabeth Koppitz (1954) undertook an extensive normative study to determine which BC errors are significant at various age levels. A list of signs of organic impairment was compiled by Koppitz, with special reference to differential significance of these signs by age level. Misshapen angles, rotation of figure, distortion, and a long list of other errors was established by Koppitz. Inspection of her data, however, reveals poor intra-subject discrimination of brain damage or of learning disability. Koppitz cautions against the use of strict cutting scores in assessing test results, recommending analysis of total and types of errors in any protocol.

Other visually oriented tests often used in screening for LBD include the Frosting Developmental Test of Visual Perception (Frostig, Lefever, and Whittlesey, 1951), Bentor Visual Retention Test, and Archimedes Spiral Afterimage. These tests all examine deficits in visual perception presumed to result from brain damage, and reflect the widespread stress on visual processing deficits in the LBD literature (Chalfant & Scheffelin, 1969).
Another group of tests which purport to detect brain damage employ the differential score approach to diagnosis. Comparisons are made between sub-tests presumed to be sensitive to insult and others presumed impervious to insult, (Reitan, 1962). This is a partial modification of holistic theories since it assumes that not all functions change after brain injury.

The Babcock-Levy Examination of Efficiency of Mental Functioning is noted as being the first of this kind (Reitan, 1962). The Babcock-Levy theory assumes that certain intellectual abilities decline more rapidly with age than others. Trauma to the brain is seen as being comparable to aging. Therefore, it is assumed that the same intellectual deterioration accompanies both phenomena (Rosenzweig, 1948). When aphasia is not present, the vocabulary sub-score is compared to tests of new learning to give insight into discrepancies between past potential for learning and present abilities. The Babcock-Levy has served as a prototype for other differential score approaches.

The most widely used tests of this kind are the Wechsler scales. Localization of brain damage was not the intention of Wechsler, merely gross screening for cerebral impairment. Wechsler (1944) proposed that tests of information, comprehension, and vocabulary showed little decline with aging or organic damage while many of the performance scale tests
showed marked decline under those conditions. Wechsler noted that new learning is most severely impaired. He also developed a Deterioration Index, computed by summing the scaled scores on tests which hold up with age and also on those which deteriorate. The sum of Hold tests is then divided by the sum of Don't Hold tests to derive the index. Anastasi (1968) reports low validity in tests of the Deterioration Index as a diagnostic tool.

Hopkins (1984) analyzed the efficacy of the WISC as a detector of organicity in MBD children. He found that Arithmetic and Digit Symbol scores were low in his brain-injured group, but that individual inter-test differences were of greater value clinically than Deterioration Index measures.

Leland (Undated mimeo) has summarized the clinical inferences which may be drawn from the Wechsler tests using a non-neuropsychological framework. The verbal tests are seen as being heavily influenced by cultural differences while the performance sub-tests are more disrupted by anxiety. The WISC and WAIS may be used to assess the individual's cognitive preoccupations, associations, and capacity for social insight.

Without shunning the importance of gestalt cerebral functioning, it appears that finer distinctions can be made in neuropsychological deficits than the previously mentioned
instruments would allow. By the late forties, an upswing in behavioral and neurosurgical evidence indicating at least moderate localization of function was coming to light. This of course opened the way for devising psychological tests differentially sensitive to specific locations of brain damage and for a much more detailed analysis of deficits resulting from such damage.

In 1947, Ward Halstead published Brain and Intelligence, a monograph which details the results of an intensive, large N study on the effects of brain damage in adults. Halstead, being disdainful of the concept of IQ, sought to redefine intelligence as a function of the brain, with separate, localizable factors. Halstead's sample of 237 was composed of pre-frontal lobectomy, pre- and post-lobotomy, closed head injury, and control Ss matched to experimental Ss by income and religion. Experimental Ss were included with or without emotional disturbances, as were controls.

Halstead employed a lengthy battery of 27 tests, and found that 13 reflected declines in functioning after brain damage or excision. Four factors emerged from Halstead's statistical analysis; power (attention), abstraction, central integration, and direction (using experience). In comparing frontal lobe patients with others, Halstead concluded that the frontal lobes were the seat of biological intelligence, with a gradient of localization extending.
posteriorly and inferiorly. This is both a departure from and a detente with holistic theorists. By finding four factors of intelligence, Halstead split with the unitary dysfunction school of thought. However, by reporting a gradient of localization of function, rather than strict specificity of function, there was room within Halstead’s findings to accommodate modified gestalt views.

Halstead did not himself propose the use of his test battery as a diagnostic tool, although he did suggest that the ten most discriminating tests in the battery could be used as an index of brain damage. His student, Reitan, has extended and modified the Halstead battery for use as a clinical instrument (Reitan, 1959b). This work was at first carried on with adults, in cooperation with the neurosurgical service of Indiana University, and later extended to children. The battery is now referred to as the Halstead-Reitan, and is used in modified forms at several universities and neuropsychiatric hospitals across North America.

Reitan’s research program has been extensive both in the theoretical and in the clinical vein. Reitan sought first to establish a firm theoretical base from which to proceed. He therefore tested Goldstein’s hypothesis that brain damage results in qualitatively different performance on psychological tests (Reitan, 1959). Comparing Wechsler-
Bellevue sub-test profiles in a group of heterogeneous brain damaged Ss and a control group, Reitan (1959b) hypothesized that qualitative changes in functioning, if they existed, should be reflected by the formation of different clusters of subjects in the two groups. This was not the case, and Reitan argued for conceptualizing changes after brain damage as quantitative, not qualitative in nature. Gerald Goldstein, Neuringer, and Olson (1958) also showed that quantitative deficits exist, using the Categories Test, one of Halstead's own devices.

Reitan began with the ten tests Halstead had found to be the best discriminators between control and brain damaged Ss, those which composed the Impairment Index. He then added other tests, and designed modifications and additional tests for children 9–15 years old and 5–8 years old. As the battery is described in the methods section, no attempt will be made to do so here. However, an appreciation of the validity of the battery and of the special techniques used in diagnosis with the battery is desirable at present.

Attacking the use of the Wechsler tests as only gross screening devices, Reitan and his students have performed an extensive research program to demonstrate that they can be used to lateralize the site of damage. Reed, Reitan, and Klive (1958) reported that the Categories Test was a better predictor of brain damage than the Wechsler-Bellevue,
but that the latter could more easily be used to lateralize site of lesion. Reed and Reitan (1963) reported that persons with right side motor deficits did more poorly than left side impaired Ss on the verbal sub-tests. The relationship was reversed for performance tests.

Reed and Fitzhugh (1966) showed that different degrees of severity of impairment created differing sub-test patterns on the Wechsler-Bellevue. Fitzhugh, Fitzhugh, and Reitan (1960) noted that there was less variance among sub-test scores in patients with chronic CNS disorders than among those with acute disease or trauma. They also noted that performance test scores declined with lesions to the non-dominant hemisphere while verbal scores were diminished in lesions to the dominant hemisphere.

These findings clearly attenuate Wechsler's contention that brain damage is analogous to aging, and lend further credence to theories of functional localization. Later work with the battery has centered around brain damage in children. Knights (1970) and Spreen and Gaddes (1968) have published normative developmental tables for children 5-15. These have been employed in the present study. In addition, profiles of epileptic, dyslexic, and MD children on the battery have been researched (Hinton & Knights, 1966, Mosley & Knights, 1968, Knights & Hinton, 1967).
Statistical studies of the efficiency of the battery have produced impressive results. Wheeler, Burke, and Reitan (1963) applied a 24 item discriminant function to test results and found that they could correctly identify between 90.7 and 98.8% of the subjects, depending upon nature and site of lesion. Reitan (1964) found that by using his own clinical insight he was able to exceed the predictive power of actuarial methods. Reitan (1968b) has summarized validation studies with individual tests in the battery. Using a best cut-off point approach for diagnosis of brain damage versus control Ss, the Categories Test yielded 13% false negatives, 12% false positives; the Impairment Index 4% false negatives, 14% false positives; the Localization score of the Tactual Performance Test 16% false negatives, 20% false positives. The Aphasia Screening Test was shown to correctly lateralize side of lesion on the basis of analysis of symptomology exhibited.

In general, profile analysis, comparisons of performances of the two sides of the body, and examination of pathognomonic signs allow the clinician to lateralize and localize brain damage through a functional analysis of the patient's neuropsychological characteristics. Furthermore, the Halstead-Reitan is the only instrument existing as an established, standardized battery designed to accomplish these ends. Of paramount importance in work with children
is the appraisal by Halstead and Rennick (1966) that culturally acquired skills involved are minimal. Reitan (1965) noted the lack of correlation between overall test scores and IQ. This should certainly put at ease objections to psychological testing in general on the grounds that it is discriminatory against minority group children. While the WISC is routinely administered with the battery, it is interpreted only in conjunction with the other instruments involved.

The present author believes that the neuropsychological approach of the Halstead group offers several clear advantages over other psychological tools for the assessment of brain damage. The battery as a whole has been shown to be a valid index of brain functioning in cases of MED (Knights & Hinton, 1967). The danger of cultural or emotional factors contaminating test results is minimal. The battery has provision to lateralize and localize brain damage, and to suggest the possible etiology (Reitan, 1959b). The test protocol represents an in-depth analysis of perceptual, cognitive, and motoric abilities and may be compared to developmental norms. Such detailed analysis may have great value in designing appropriate remediation programs.

There are certainly some shortcomings in the Reitan approach, but these are outweighed by its attributes.
Reitan's approach is essentially empirical, as is the research on the battery. This leads to an impoverishment of theoretical inquiries into neurodynamics. Presently, this author will be employing Lurian formulations of brain functioning to discuss the results of this study. In addition, there has been some question as to the validity of making clinical inferences about childhood brain dysfunction using instruments which are simplified, downward extensions of the adult Reitan battery. It is not clear that brain damaged children are behaviorally analogous to their adult counterparts (Teuber and Rudel, 1932). However, a research program conducted by Rourke (1975) indicates that Halstead-Reitan battery patterns of children and adults were quite similar. These issues are explored below.

Reitan (1966) states that his approach to diagnosis of brain dysfunction is essentially atheoretical. The present research, however, does have as its goal the intention of making some theoretical statements about the neurodynamics of LD. To bridge this gap, the theoretical stance of Luria (1974) will be called upon. These two positions are fundamentally congruent. Halstead's original work, upon which Reitan has based the neuropsychological test battery, favored a moderate localized function map of the brain (Halstead, 1947). A psychometric gradient of functions was derived from Halstead's data. This was presented in
such a manner as to indicate that certain areas subserved one function discretely. Surrounding cortical tissue was presented as subserving one or two functions, in a less specific manner. Halstead did not offer an integrated model of brain functioning, largely due to his strong psychometric bent, and to his interest in frontal lobe functioning. However, he did lay a bridge to Lurian theory which the present author will employ.

Luria (1974) has summarized decades of clinical experience in human neuropsychology in his book *The Working Brain*. While the treatise deals specifically with adult patients, other works elucidate Luria's views on mental development, particularly of language skills. Luria argues against the localization of behavioral functions within circumspect cortical areas on two grounds. First, functions must be broken down into component acts. By doing so, it is seen that even elementary cognitive functions require many separate aspects to be performed and integrated. Secondly, functions once seen as being discrete but now viewed as complex require the integrated activity of three systems of the brain.

Luria notes that the three systems are as follows: The unit for regulating waking and neuronal tone, located in the medial-basal aspects of the brain. The second system, for receiving, analysing, and storing information,
includes the posterior portion of the cerebral hemispheres. This system contains modality specific projection areas, intramodality association areas, and inter-modality zones. The third system subserves the general function of programming, regulating, and verifying action, and is located in the anterior portion of the cerebral hemispheres.

From a clinical point of view, Luria notes that any complex cognitive function depends upon the integrated activity of specific foci within each of the three systems of the brain. Disturbance at any point neurologically will interrupt the smooth, natural carrying out of the higher mental operation. However, what is most important for the present study, is the fact that dysfunction at a particular focus disturbs the function in a unique way, peculiar to the dropping out of that focus from a complex, hierarchical functional system. Luria calls for a dynamic localization of defects by closely examining the qualities of the disturbed function, and has developed an extensive schema for localizing lesions on the basis of scrutinized symptoms.

Unfortunately, Luria's assessment methods are neither quantifiable nor designed for use with children. Both these points ruled out use of his tools instead of only his theory. The Halstead-Reitan battery does examine the functioning of cerebral foci in a quantifiable manner and has
an extensive literature and developmental norms. While the tasks in Lurie's investigation differ from those in the Halstead battery in content, they are akin in their capacity to localize lesions and to analyze the particulate defects responsible for the disintegration of higher cognitive functions. Unfortunately, the Reitan tests do not allow for close analysis of the medio-basal brain system as does Luria's method. As such, this limits the range of neuropsychological inferences which can be made, but the two systems for the analysis of the effects of brain lesions are reconcilable.

Another problem mentioned above is the question of differential effects of early versus late-in-life brain injury. This issue is noteworthy because the Reitan tests for children are simplifications of those included in the adult's battery. This is more than a theoretical issue in that the inferential system used in the Reitan battery assumes that the simplified tests used for children tap the same skills and are subserved by the same cerebral foci as are the original, adult level tasks. If this assumption is false, then the findings become tenuous.

As standardized on adults, the battery aims to localize lesions. In simplifying and modifying the instruments, Reitan (1956) sought to achieve these same ends with children. However, Teuber and Rudel (1932) present strong documentation,
using the Aubert task, for the hypothesis that at least some deficits which in adulthood are signs of specific sites of lesions may be indicative of brain lesions, generally, in childhood. They advance the opinion that some early lesions may be minimally disabling in elementary functions but could, if acquired in maturity, be crippling. On the other hand, early insult could be very harmful to the development of higher, integrative functions such as reading. In adults, however, such deficits may be compensatable through the reconditioning or re-organization of already developed functional systems (Luria, Maydin, Tsvet Kova, and Vinarskaya, 1938).

These views are in fact supported by some findings on the Halstead battery. Halstead and Rennick (1936) found that WISC IQ scores could detect presence or absence of brain damage in children about as well as the battery as a whole. However, WISC sub-tests had no value in localizing areas lesioned as they inter-corrrelated quite highly. The Halstead measures themselves did not highly inter-correlate and could be used to localize sites of lesions and to analyze particular strengths and weaknesses. They attributed the WISC findings to the brain injured child's inability to integrate the various stimuli in learning situations and to attentional deficits as well. This argument is fairly consistent with Teuber and Rudel's, but also indicates the
feasibility of finding specific deficits in children, probably associated with particular types of brain damage.

Most notably, the Halestead and Rennie study supports the Teuber and Rudel hypothesis that certain tasks may be localizers of brain damage in adulthood, but only gross discriminators of presence versus absence of brain damage in childhood. The value of the WISC was found to be limited to gross assessment of brain damage in children. However, in using the WAIS with adults this instrument's sub-tests are quite useful in localizing site of lesions (Reitan, 1959b). The WISC is designed as a downward extension of the WAIS, and yet it does not retain the same neuropsychological patterns in children as it exhibits with adults. A further example of this comes from Mc Fic (1931) who found that children who had had hemispherectomies before one year of age did not show the pattern of impairments exhibited by adults after the same operation. Children showed more generalized cognitive deficits, but speech functions developed regardless of which hemisphere had been removed. Children also showed a full scale IQ ceiling effect which adults did not.

Graham and Berman (1931) reviewed the status of behavioral tests of brain damage in infants and preschoolers. Many problems for devising such tests were noted by these authors. Reliability over time is usually a useful thing
for a test to have. However, the greater plasticity of the child's nervous system makes it more desirable to have tests whose results reflect the recovery of function, and which would therefore have low reliability. The reader is left feeling that there is no completely acceptable criteria against which tests for brain damage in children may be compared. Graham and Berman note that the tests available in 1951 were often constructed to be put to use with MD children. However, these tests, such as the Bender Gestalt, were based upon criteria which were not adequate indices of MD and which were confounded by extraneous variables. Reitan (1966) used neurological criteria and adult neuropsychological symptoms to standardize his tests for brain damage in children. In addition, the tests in the Reitan children's battery only extended down to the five year old level. This averts the problem of standardizing the tests against criteria which also purport to define MD. In addition, excluding very young children from consideration abates the reliability problem mentioned above.

Recent evidence indicates that the children's battery is an adequate clinical and experimental tool for assessment of brain damage. Rourke (1975) conducted an extensive research project with these tests. He has found that when older children are put into high verbal-low performance
and high performance-low verbal IQ groups their neuropsychological profiles are consistent with adults in such groups. Likewise, Trail Making Test grouping with older children yields Halstead-Reitan battery profiles which are consistent with adult norms. It was Rourke's conclusion that patterns of performance were far more important than the levels of performance on individual tests and that these patterns were congruent to those exhibited by adults. The pattern approach is stressed in the present study through the use of factor analysis, and has already proven its merit in a pilot study by the present author (Schachter, 1974).

The trend in the literature seems fairly clear. WISC scores are not as useful for neuropsychological assessment with children as they are with adults. However, the individual elements of the Halstead-Reitan tests retain their expected patterns of deficit in older children as compared to adult norms. This trend makes the methodology of the present study more justifiable.

There remains the theoretical issue of differential effects of cerebral lesions as a function of age at the time of insult. It is apparent that on some tests children and adults with brain damage perform differently. Several theorists have sought to explain the developmental dimension of brain injury.

Hebb (1949) argues that early damage is more deleterious
than late damage. He believes that more intact brain cells are required to establish learning than are needed to retain what is learned. He cites studies to show that children with brain damage are more limited in IQ than are adults with analogous damage. This argument is flawed in its reliance upon Binet type, verbally loaded IQ tests.

Lenneberg (1967) advances the view that a critical periods concept is best suited to explaining the developmental parameter of cerebral insult. Hypothyroidism, rubella, and other conditions cause brain damage to the fetus but not to the adult. Hemiplegias acquired in adulthood are often associated with language disorders if the injury is to the left side of the brain. Infantile hemiplegias are not often associated with aphasic speech disorders according to Lenneberg. He argues that the plasticity of the child's brain allows for the reconstructuring of the language system, while in adults aphasia often accompanies right hemiplegia.

Luria (1974) espouses a view which is congruent to Lenneberg's in that he too feels that brain damage has different effects when acquired in childhood as compared to adulthood. However, Luria does not explore the issue of critical periods. He theorizes that the brain has
regions of diminishing modal specificity. The sensory projection areas are modality specific and are surrounded by association or secondary areas. The latter allow for attribution of meaning to the perceived stimuli within the single modality. Tertiary areas are found at the confluence of the association areas. These tertiary zones permit simultaneous synthesis of information across modalities. Wernicke's area, which is a prime focus for the completion of reading and writing skills is in the tertiary zone of the left hemisphere.

Luria hypothesizes that mental development proceeds from an organization of the primary and secondary areas, which are largely modality specific, to the organization of the tertiary zones. Once the tertiary zones are conditioned to synthesize information from separate modalities, higher mental functioning is possible in the areas of speech and regulation of behavior.

More concretely, in early childhood the primary and secondary zones are responsible for the development of the late evolving tertiary regions. Damage to the hierarchically lower areas in childhood will reduce the efficiency of the tertiary zones since disordered signals will be synthesized. If the brain develops without insult to the lower order cerebral zones, then the tertiary zones will
come to regulate behavior through the use of language and planning functions. Under this condition, the tertiary areas are at the head of a hierarchy of cerebral organization. They are said to control the functioning of the primary and secondary zones.

Citing Russian language literature and clinical data Luria devises the following scheme: Damage in childhood to the primary zones has an upward effect on the higher centers. This is due to the tertiary zones' dependence upon input from the lower zones in the hierarchy. The diminished functioning of the tertiary zones may go undiscovered until middle childhood when the child is expected to regulate his own behavior somewhat, and to read and write. Early damage to the primary and secondary zones therefore affects higher functions. Early damage to the tertiary cerebral areas may be compensatable.

Later life cerebral damage has different qualities. Insult to the primary zones has little or no effect upon higher mental functioning. The tertiary zones are seen as being in control at this point. Damage to the latter is likely to cause dysfunction in primary and secondary region skills as control of behavior is in a downward direction in Luria's cerebral zone hierarchy.
In summary, early childhood injury to primary and secondary regions exerts upward influence to the evolving tertiary zone, but in the reverse condition the tertiary zone does not affect the lower centers. In adulthood, insult to the lower zones does not affect the higher region, but in the reverse condition the tertiary area damage causes dysfunction to the lower centers.

This position allows an understanding of the IQ ceiling effects found in the McFie study and by Hebb. Since the IQ tests tap skills which require tertiary zone integrity, childhood damage to the lower zones will reduce IQ by compromising the development of the tertiary regions. Likewise, Lenneberg's data regarding the recoverability of language functions in childhood are accounted for by seeing the tertiary zones as being more plastic. Lenneberg's view that infantile hemiplegia is not associated with later aphasia is not supported by Luria, but there may be some question as to the aphasia assessments used to come to his findings. It is more likely that infantile right hemiplegia causes less severe language dysfunction than its adulthood counterpart. The greater plasticity of the child's brain and the possibility of reorganization of language processing or of development of vicarious functioning in other areas may be the mechanisms responsible (Rosner, 1970).
It is clear that a great deal of careful study into the developmental aspects of brain damage still needs to be done. Discovery of the parameters of vicarious functioning, the time course for recovery of function at different ages and for different functions, and the neuropsychological basis for reorganization of higher mental processes are important areas for future research. However, given these lacunae in our knowledge, the literature on the Reitan battery appears to justify a clinically based study on the neuropsychology of LD.

There is a relatively small literature on the neuropsychology of LD. Much of what there is comes from a desire for comprehensive clinical assessment of LD children. Reitan and Heineman (1968) published a clinical impressionistic report on LD children with and without emotional disturbance. Their case history presentations pointed to the importance of behavioral analysis of brain function in LD children and to the interaction of emotional and neuropsychological problems.

Later studies have by and large centered on neuropsychological aspects of MBD, LD, or specific reading disability. Most have excluded children with emotional problems for the sake of experimental control and have therefore minimized clinical usefulness. In other studies, only children in need of major psychiatric inter-
vention were excluded, while milder form of emotional disturbance remained unaccounted for. The relevant studies are summarized below.

The Halstead-Reitan Battery has been used with LBD children in a study by Knights and Hinton (1937). The most common deficits associated with LBD on the battery were signs of language deficiencies as revealed on the aphasia test. However, more detailed analysis of deficits and chief complaints revealed the following pattern: Children whose presenting complaint was reading disability most frequently did poorly on tests of spatial abilities. Where the chief problem was distractibility and motor control, children frequently did poorly on pegboard tasks (not included in the present study). The group with poor concentration as chief complaint tended to do most poorly on the aphasia test, while those referred as being hyperactive did the worst on the Trail Making Tests. Halstead and Rennick (1936) reported that deficits of LBD children on the battery showed much general scatter. The probable cause of this was hypothesized to be a deficit in attention.

Doehring studied patterns of impairments in children with specific reading disability, a sub-group of the population under investigation here. Many measures from the Reitan battery were included along with independent measures of aphasia, visual-motor, and visual-verbal perception.
The reading disabled Ss performed below the mean of their matched controls on all but five measures, all requiring somesthetic perception. Significantly more experimental Ss were judged by raters to have cerebral dysfunction on the basis of the test data than were controls. The dysfunction was limited to the left hemisphere. Only two factors emerged from an analysis of 76 variables: Reading—Spelling and Visual Perceptual Speed (Doehring, 1968). This study was limited to non-emotionally disturbed children with Performance Iqs higher than Verbal Iq. Neuropsychologically, this would have excluded children with right cerebral dysfunction by coincidence.

Rourke's research program on neuropsychological correlates of LD indicates that patterns of results on neuropsychological tests were better predictors of performance on academic skill variables than were simple level of performance scores. The data also indicated that cerebral dysfunction is present in many retarded readers and that sensory-motor integration problems were common among younger Ss while higher conceptual skills deficits were most common in the older Ss. Rourke argues that reading disability is a non-specific deficit associated with different patterns of ability impairments across the range of children.

The present author (Schachter, 1974) conducted a pilot project to examine patterns of neuropsychological
deficit in LD-MLD children. Nine factors relating to LD were obtained from an analysis of Halstead Reitan battery, WISC, WRAT, and hyperactivity questionnaire data. These factors included motoric deficit, spatial relations dysfunction, auditory analysis deficit, visuo-motor deficit, fronto-temporal lobe dysfunction, and a cluster of school failure variables without neuropsychological symptoms. Also emerging from the factor analysis were clusters which related to use of amphetamines, sex, and class placement.

The first four factors mentioned above are easily amenable to an explanation of LD using Lurian theory, i.e., learning to read is disrupted by dysfunction in any one of a number of specialized cerebral systems which must work in an integrated manner with each other for reading to develop. Reading is conceptualized as a multi-sensory process, requiring mediation within and between various cerebral areas. Dysfunction to one or more areas alters the entire process in a manner unique to the function of the damaged region(s). Lurian theory's implication for LD is explored in a subsequent section. But, the pilot project suggested the feasibility of using factor analysis with pathognomonic signs generated from knights' (1970) and Spreen and Gaddes' (1969) normative data. This was a two-fold departure from level of performance analyses in that the data was converted into dichotomous pathognomonic signs, much as clinicians do in practice, and then factor analyzed.
The factors made good sense neuropsychologically and in terms of independent signs of LD. However, only 20 Ss were employed in the study and the possibility of spurrious results cannot be ruled out. Also, the presence of a school failure without cerebral dysfunction factor has led to the inclusion of the BSAG in the present study. This will enable the researcher to examine particulars of classroom maladjustment.

The pilot project results were suggestive of the heterogeneity of neuropsychological deficits accounting for LD. In addition, factors were for the most part not totally independent of each other. This suggested that the neuropsychological deficits often acted in concert within a child. The impact of that study was most striking in suggesting the importance of patterns of deficit and of detailed symptom analysis, as put forth by Luria.

Ayres' (1965) factor analytic study of perceptual-motor dysfunction of LD children has implications for the neuropsychological analysis of LD. She too found separate factors in her data. These were labeled apraxia, perceptual dysfunction-form and position sense, tactile defensiveness, deficit in integration of the two sides of the body, and perceptual dysfunction-figure-ground confusion. Ayres' also found that control Ss shared only one factor in common with the LD children and that few of the
latter had deficits in only one factor.

The studies of both Schachter and Ayres indicate that that LD is a multi-deficit category and that careful analysis of cognitive functioning and sensory-perceptual functioning may be use in highlighting the particular deficits responsible for the more general learning disorder. In addition, simplistic nosological classification systems must be seen as being inadequate to understand the complex dynamics of learning disabilities as a consequence of these studies. Only wide ranging assessment procedures are up to the task of delimiting the dynamics of learning disorders. The complexity of this field is explored in the following section.

Just as in human neuropsychology, the LD literature has moved from consideration of one or two symptoms to more complex formulations. The learning disabilities literature has undergone several changes independent of theories of cerebral integration. As mentioned previously, Strauss and Lehtinen (1947) had seen the group as suffering from a unitary dysfunction. They also envisioned these children as being exogenous retardates. That is, suffering from an acquired rather than inherited disturbance. Strauss and Kephart (1955) then extended this concept to include children of average and higher IQs. The new concept made it possible to redefine the characteristics of these
children as learning disabilities, or discrepancies between ability and performance on academic tasks. Myklebust (1968) defines learning disability as a discrepancy between ability and performance caused by presumptive CNS damage.

Criticism of Straussian theory began as early as the fifties. Wortis (1955) criticized the concept as being stereotypic, and inadequate to describe the full range of behaviors found across brain damaged children. Strauss' notions regarding hyperactivity were also reviled in the literature (Gardner, Coomwell and Foshee, 1959). Strauss had assumed that hyperactivity was purely emotional response pattern which could be controlled by reducing the complexity of the child's environment. However, Gardner et al were able to demonstrate that stimulus-free environments increased, rather than decreased activity level.

Myklebust (1968) proposed that the term KBD should be reserved to denote the medical basis of learning disability. He stressed the ability versus performance comparison as the behavioral basis for diagnosis. Reduction in general bodily integrity (left-right confusion etc.), learning deficits, and minimal brain dysfunction are presumed to be shared characteristics of the LD children. However, Myklebust acknowledges the fact that each sensory system must be examined both independently and in relation
to other systems for a complete understanding of the perceptual-cognitive bases of learning disabilities.

Literally thousands of articles have appeared on LD and its correlates, substrates, or etiologies. The confusion in terminology and lack of consensus on the who, what, and whys of LD have led to a dearth of synthesis and integration of the findings. It would be beyond the scope of this text to attempt to review all of these studies. However, some of the work does put forth, either explicitly or implicitly, a theory about the etiology and dynamics of LD. Rourke (1976, in press) has divided these theories into two types, deficit and developmental lag. One might have also used the term defect and difference theories to describe this dichotomy.

According to Rourke, the deficit theories account for LD by postulating cerebral dysfunction or damage as causing learning problems. There is no necessary reason inherent in such positions to assume that the LD child will ever catch up to other children. However, such theories often account for compensatory education and training in neurodynamic formulations. Restructuring the cognitive processes, using more efficient sensory modalities to compensate for deficient ones, or developing individual skills which would normally not come into play in certain academic tasks are remedial implications of deficit theories.

The developmental lag or difference position assumes
that there is a delay in the development of certain skills, but that LD children do not suffer from any disease or damage entity. They are not viewed as being qualitatively different from normal learners, but as being delayed. They move through the same developmental sequence as non-LD children. Remediation is made via a speeding-up rationale and not through restructuring of the cognitive functions. The various theories will be discussed in detail later.

The present author assumed that there is a kind of sociological phenomenon underlying the labeling or diagnosing of a child as being learning disabled. Richardson (1964) pointed out that functions affected by brain damage in children were also affected by the social environment. Placement in a special class will likely affect the child for a variety of reasons including the effects of special materials and instruction, changes in self-concept, and alterations in classmate characteristics. Also, there may be alterations in teacher attitudes, parent attitudes, and non-classmate peer attitudes. Richardson believes that the chronological sequence in which dysfunctions come to the attention of school staff are based upon the ages at which the expectancies of society are firmly enough established to permit recognition of abnormality. Tomiyasu and Matsuda (1974) report similar findings in adaptive behavior development of the retarded.
Awareness by a professional person that a given child has a problem is almost the last step in diagnosing it. Since professionals vary, diagnostic criteria will also vary. Werry's factor analytic study of hyperactive children revealed ten basically independent factors (Werry, 1968). Each of these was derived from one source of information only, such as educator or psychologist or neurologist, etc. However, these children, also labeled MBD, were similarly treated and grouped into a single category by society in general.

Whether LD is viewed as a lag or a defect, it should always be kept in mind that this sociological process of labeling has somehow been accomplished through the school, clinic, or hospital. It has been the experience of the present author that the concurring opinions of several professionals are required to place a child in an LD program. These persons may hold implicit or explicit views of LD as being a defect or difference. But, the opinions have concordance in viewing the child as being somehow abnormal. This is in accord with Richardson's view of a failure to meet expectations of society. Such standards may be failed in one or more areas, such as activity level, learning rate, or unevenness development of different skills. As will be seen further on, defect and difference theories need not be antagonistic but may complement each other in explaining the complex symptomatology of LDs.
Defect theories have been far more numerous in the area of LD than have difference theories. Strauss' early work on the brain-injured child (Strauss and Lehtinen, 1947) is an example. The defect was seen as the perceptual-motor dysfunctions concomitant to brain-injury. Orton (1928) thought of reading disability as solely a visual defect, word-blindness. Hallgren (1950) found a genetic component to specific dyslexia. Benton (1964) argued for establishment of developmental aphasia as a syndrome. The presumptive etiology was early brain damage leading to high level cerebral-auditory imperception. Dykman, Ackerman, Clements, and Peters (1971) saw specific LD as being the educators', equivalent of the medical diagnosis of MBD. They presented psychophysiological evidence that MBD is an attentional deficit of a unitary character. This research was seriously deficient in confounding LD with hyperactivity. It used a Lurian model of hyperactivity.

The implications of Lurian theory in the area of LD are quite far ranging. Luris (1974) advances several means by which cerebral damage or dysfunction might interrupt higher cognitive functions. Some of these implications were gleaned from the early works of Luria by Chalfant and Scheffelin (1969). While a Lurian schema is used by the latter two authors, it is not a complete account of all of the Lurian implications. Therefore, the present author will make some additional commentary after review-
ing Chalfant and Scheffelin's system. In their system the following deficits are outlined:

Auditory processing anomalies are noted as one factor in learning disability. Perceptual problems in this area may leave the child unable to structure the auditory world, to combine or analyze speech sounds, or to discriminate between phonemes. Inability to process language receptively may then lead to psycholinguistic deficits.

Visual processing dysfunctions have received the most attention, in the literature according to these authors. They view problems in visual acuity and perception as lying on a continuum, rather than as being discrete. Motoric and symbolic processing problems are found in this area, as well as spatial confusion.

Chalfant and Scheffelin also list errors in haptic (cutaneous and kinesthetic) processing as a possible mechanism for learning disorders. Haptic sensation is an integral component in the development of spatial concepts and also is implicated in the control of muscles and joints. Errors in the articulation of language and in the production of writing may be due to this type of dysfunction.

Inter-sensory integration must also be accomplished by the child in the course of developing academic skills. Individual sense modalities are composed of separate processes, such as tuning and sharpening contrasts, scanning, and filtering signals. Intra-sensory integration must there-
fore be attained before inter-sensory integration is effective. The various modalities are then in turn integrated in such higher cognitive tasks as reading. Chalfant and Scheffelin report that all too little work has been done in areas other than visual processing.

The implications are even more complicated than indicated above. It will be recalled that Luria (1974) postulated the existence of three brain systems which must act in concert in order for higher cognitive functions to be performed. The problems outlined by Chalfant and Scheffelin are limited to the second cerebral system. This is the unit for receiving, analyzing and storing information.

Dysfunction or damage to the first functional system, responsible for waking and tone, in anatomical terms would be found in the medio-basal aspects of the cerebrum. Disturbances in consciousness and memory which cut across all sensory modalities would result. In addition, errors in orienting and habituating to stimuli in all sensory modalities would be classified under dysfunctions of the first cerebral system. Milder lesions of these zones may be in part responsible for the hyper- and hypokinesis found in some LD children.

The third functional system is comprised of the frontal and pre-frontal cortex and subserves the programming, verification and regulation of behavior. Dysfunction here leads to disinhibition of orienting reflexes and interferes
with orderly sequencing of plans of action. This may be reflected in distractibility and impulsivity, hallmarks of MED. Also, verification that an action has or has not been successfully carried out may be interrupted. The individual with such a defect may be viewed as being perseverative by observers. As mentioned earlier, the three functional units must act in concert for higher mental operations to be carried out successfully. These units should not be confused with the first and second signal systems.

The role of speech is given prominence in Luria's developmental theory (Luria, 1961). According to this view, language at first exerts control over the child's behavior by commands and labels provided by adults. Later on, it becomes internalized. At first, the child uses language only as an accompaniment to activity. Then it is used to regulate and help plan future action.

Left temporal lobe lesions in adults may interfere with understanding logico-grammatical relationships and with solving verbal or mathematic problems. This is due to difficulties in comprehension rather than in planning. In children, Luria (1961) places more emphasis on the role of speech, the second signal system, in regulating simple behavior patterns. The systems of the frontal lobe and of the left temporal lobe act in concert in the higher mental processes. The frontal lobes have greater sway over verifying and sequencing actions while temporal functions allow for categorization and logico-grammatical understanding.
Some suggestive evidence of the interconnectedness of these systems comes from the clinical use of the Reitan battery (Reitan, 1966). Trail Making Test A only has a sequence of numbers and is therefore basically non-linguistic, while Trails B has a sequence of numbers and letters. Lesions of the left frontal lobe are associated with Trails B deficits while lesions of the right frontal lobe more often show Trails A decrements. Therefore, the frontal lobe of the language bearing hemisphere is more related to processing linguistic stimuli in a sequencing task than is the other frontal lobe.

Luria (1961) has studied a sub-group of LD children whom he referred to as cerebro-aesthenics. This group appears to correspond to Western classifications of hyper- and hypo-active children. Luria asserts that the basic excitatory and inhibitory processes in the central nervous system are out of balance in these subjects. In the hyper-active group, the inhibitory process is weak and the child acts impulsively and prematurely. He fails to habituate orienting reflexes to unimportant stimuli in the environment and is easily exhausted due to over-reactivity. The hypo-active child has weakness of the excitatory process. He suffers from a decline in nervous tonus, is passive and under-reactive.

Luria (1961) reports studies in which teaching verbal labels for stimuli in a bulb pressing paradigm helped to alleviate the deficits of these two sub-groups. He feels
that speech helps to improve the functions of sensory analysis and motor regulation. This compensatory method requires relative intactness of the speech system. The child is taught to verbalize the appropriate command to each stimuli as it is presented, for example "push" or "stop."

This helps him to regulate his behavior in the experimental situation. Also it illustrates the point made earlier that defect theories often imply specific remedial procedures. Hyperactivity, hypoactivity, and distractibility may or may not be intimately related to all learning disabilities. The survey by Chalfant and Scheffelin (1969) would indicate that activity and attention disorders cannot be considered the heart of the problem in all children. The present study, while not specifically concerned with such problems will attempt to gain some understanding of the interaction between hyperactivity, MBD, and learning disability.

There are several additional implications of Lurian theory. Attention is not necessarily a unitary process as suggested by the Dykman et al (1971) study. Luria (1974) suggests that there are complex interactions between the three brain systems which determine orienting and maintenance of attention. The Dykman et al study selected Ss on the basis of demonstrated hyperactivity, but then sought to generalize findings to LD children as a group. This appears to be a serious logical and methodological flaw.

The present author finds it far more plausible to work within a broader Lurian framework when dealing with LD.
The variety of intra- and inter-sensory deficits, together with problems in maintaining nervous tone and in regulating and verifying action allows the clinician to account for the wide range of symptomatology exhibited by LD children. It should be noted that these problems may occur in isolation or multiply within a given child. In addition, time of onset of the cerebral dysfunction is important in respect to developmental parameters of loss and recovery of function after brain damage. Luria’s views on this topic have already been summarized. There are far fewer difference theories, possibly a reflection on the historical roots of the field. A summary of those theories is, however, necessary.

Ames (1938) advanced the view that LD may be due to pushing the child into learning situations for which he is not yet ready. The child responds as best he can, but differently from one who is ready to learn the skills being taught. The delayed child may acquire faulty learning styles which further cripple his cognitive growth. The main implication of this position is not to try to teach skills until reasonable probability of success is attained.

Satz’ group (Staz and Van Nostrand, 1873; Satz and Friel, 1973) ascribes LD to a developmental lag of the left hemisphere. These investigators believe that the delayed maturation of the language bearing hemisphere is not only responsible for the reading retardation found in many LD children, but also for the pattern of test results found in many studies of LD and which is interpreted as reflect-
ing brain damage by other researchers. Their own research predicts, and finds, that LD children have perceptual-motor dysfunction early in life and that they catch up to control Ss later on. The extra time spent in catching-up delays acquisition of higher cognitive skills. Older LD children are shown to be deficient in these higher functions, such as generalization and abstraction. The Satz data are weakened by ceiling effects in the perceptual-motor tests. The results are congruent with Luria's theory of developmental effects of brain damage as they are with a lag explanation. Damage to perceptual or motor systems in childhood would be predicted to insult later cognitive development by Luria.

Herbert Birch and his group (Birch, 1964; Birch and Lefford, 1964; Birch and Belmont, 1965) present data to support the theory that LD is due to delays in inter-sensory integration. Birch and Belmont (1965) demonstrated that reading readiness is highly correlated with the ability to integrate auditory and visual cues in the age range of kindergarten through the second grade. Later on, reading readiness is more highly correlated with IQ score. This finding seems to parallel those of the Satz group in that early deficits are sensory-motoric while later ones appear to be more conceptual. Once again, ceiling effects in sensory integration tasks leaves unanswered the question of whether or not the LD subjects do in fact catch up, and the pattern of results is amenable to Lurian explanation.

The defect and difference theories are by no means
mutually exclusive. Bannatyne (1971) proposes a multiple etiology theory of language and learning disabilities which includes defect, developmental lag, and emotional causes. These problems may be present singly or in a group. Furthermore, their action may be additive, as one follows another, or interactive, as when two or more act in unison.

Bannatyne discusses four etiologies of LD. The first is primary emotional communicative dyslexia. Insufficient or inaccurate verbal stimulation of the infant by the mother is seen as leading to a failure in the development of spoken language. Speech defects, maturational lag in language development and disinterest in auditory stimulation may be the results.

A second cause is minimal neurologic dysfunction, as Bannatyne terms MND. Bannatyne adopts a Lurian view of the mechanisms of such deficits. These include intra- and inter-sensory problems, attentional deficits, motoric dysfunction and conceptualizing disorders. Physical brain damage or genetic malformation is seen to underlie this etiology.

Thirdly, Bannatyne argues for the establishment of genetic dyslexia as an etiology. Children with this problem are conceptualized as falling at the low end of the normal distribution of language learning potential. These children are not retarded generally, only in linguistic processing. Decreased ability to analyze auditory signals is the key symptom in genetic dyslexia according to Bannatyne. He believes that fully one third of all LD cases
suffer from this specific form of disablement.

The fourth cause of LD is posited to be socio-cultural or educational deprivation. This differs from the first cause in that the problem lies with a sub-group of the general population and is not primarily emotional. The fifth and final cause postulated by Bannatyne is secondary emotional LD. Such children are not truly learning disabled, but may be acting out, or otherwise behaviorally disordered. The behavior problem interferes with school work and the child does not keep pace of the new learning being acquired by age mates.

Bannatyne calls for the use of factor analytic techniques to help in the classification of homogenous groups of LD children. His own landmark handbook stresses the interaction of etiological agents both in succession and in interaction. His treatment of emotional causes of LD stresses the dearth of systematic studies in this area.

Much of the work on emotional disturbance in LD or brain-injured children has impressionistic rather than quantitative. Bender (1959) reported that few LD children were without emotional problems, while Rabinovitch (1962) stated that a significant percentage of children referred to his clinic for behavioral and adjustment problems were found to have reading disability. Connoly (1971) speculated that emotional disturbance and learning disability were highly correlated, but admitted that there was little hard data to substantiate that widely held opinion. Connoly also
distinguished between primary emotional disturbances which impede school progress and secondary disturbances which are a reaction to the learning disability and/or ME. This is different from Bannatyne's categories of primary and secondary emotional causes.

Connoly remarked on the poverty of good instruments to assess social and emotional adjustment in children. This was a problem encountered by the present author as well. However, Connoly's psychodynamic formulations are noteworthy. He notes that early clinical work in LD tended to attribute blame to the child for being unmotivated or acting out. Connoly feels that far more frequently the emotional problems are secondary to LD and are contingent upon the chronicity and severity of the disability. The child's competence needs are unmet in school and he is frustrated, leading to unproductive behaviors. These behaviors further reduce his chances of success in school, forming a vicious cycle of failure. Social acceptance by peers and social maturity are low, while anxiety is high in LD children according to Connoly.

Rubin (1971) postulated that maximum emotional disturbance in LD comes from severe unevenness in development of cognitive skills coupled with inflexible environmental demands. Rubin found that a high percentage of children labeled emotionally disturbed were suffering from cognitive, perceptual, and motoric problems.

There were few quantitative studies of emotional dis-
turbance in LD children. Those that exist use either behavior ratings (Weiss, Minde, Werry, Nemeth, Douglas, 1971) or the Bristol Social Adjustment Guides (Stott, 1971). The Weiss et al. study compared hyperactive to control subjects. As expected, the hyperactive Ss were more behaviorally disordered.

Part of the problem is gathering quantitative data on emotional disturbance in childhood is a scarcity of applicable instruments. The present author could find no actuarial prediction instruments, like the MMPI, which were applicable to children. Use of behavioral observations was considered in this study but later rejected because of statistical considerations. Instead, it was decided to employ the Bristol Social Adjustment Guides (Stott and Sykes, 1966) for several reasons. It is a compromise between behavior ratings and observations (Chazan, 1971), assessing more global characteristics than observations, yet more tied to overt behavior than projective tests. The questions are tied to specific environmental situations, there being separate forms for the child at home or at school, and the data is broken down into a number of maladjustment dimensions. The 1966 version included anxiety and hostility categories, restlessness (hyperactivity), depression, withdrawal, and unconcern about adult values.

Scores are quantitative within dimensions, and a summary maladjustment score is also obtainable. The Bristol Social Adjustment Guides would appear to tap many of the
emotional characteristics of LD children, but in a way which is tied to behavior more than underlying personality dynamics.

The 1970 revision of the BSAG was used in the present study (Stott, 1970). This instrument was standardized on 2527 children from three different settings in Ontario, Canada. The guides are a questionnaire-type instrument composed of 116 short, descriptive phrases which are worked into paragraph form. The teacher reads the BSAG booklet and circles phrases which apply to the child.

A factor analysis of the items in the BSAG was carried out so as to place items into clusters or syndromes (Stott 1970). These syndromes are labeled as follows: Depression, Withdrawal, and Unforthcomingness are the core syndromes reflecting under-reactivity by the child. Depression is defined as lethargy and lack of interest; Withdrawal as voluntary rejection of social activity without loss of need for affection; and Unforthcomingness as shy behavior due to low self-concept (Stott, 1966).

The over-reactive syndromes are Hostility, Inconsequence, and Peer-maladaptiveness. Hostility is defined as a range of behaviors encompassing active rejection of others through open violence; Inconsequence as a failure to inhibit responses and impulses; peer-maladaptiveness as anti-social or hostile attitudes to other children. The inconsequence syndrome closely parallels definitions of hyperactivity and impulsivity in the American literature.
Cut-off scores for placing children in the maladjusted range are given for each syndrome, as are means and standard deviations for the standardization sample. The 1970 revision of the BSAG is far more empirically based than the earlier version and excludes several previously designated syndromes.

External validity of the BSAG was established by showing significant correlations between under-reactive and over-reactive summary scores and ill-health, motor impairment, and delinquency. Low SES children score higher on the over-reactive items, but there is no reported SES trend on under-reactivity. Reliability for the BSAG total score is .80, using the Winer formula. Coefficients for the core syndromes are as follows: Unforthcomingness, .67; Withdraw, .48; Depression, .54; Inconsequence, .71; Hostility, .68. The over-reactive syndromes generally have higher reliability than the under-reactive ones. Stott (1970) attributes this to the unobtrusiveness of the latter. As this instrument is situation specific and represents a hybrid of behavior ratings and observations (Chasan, 1970), the reported coefficients seem acceptable to this investigator.

In addition, there has been some research on characteristics of LD children on the guides (BSAG). Chasan (1964) reports that more than one third of the educationally subnormal children he tested scored in the maladjusted range of the summary score. Frost (1965) found that poor
readers, those with reading levels two years below mental age, were anxious about adult and age peer approval and affection. There was also a tendency toward depression in about half of Frost's sample. On the summary maladjustment score, 40% were maladjusted, 40% unsettled (a range just below the severe maladjustment range), and only 20% were in the adjusted end of the continuum.

In another study, Gregory (1965) examined BSAG results from children whose reading level was two years below chronological age. In general, the LD children showed symptoms of withdrawal, restlessness, anxiety concerning adults, and anxiety concerning children. More closely examined, the profiles showed that anxiety concerning children did not appear before age eight. It was hypothesized that this symptom complex is a product, over time, of the failure to learn to read.

Chazan (1968) tested children in regular school classes, special education classes, and in special schools for the developmentally disabled. Only 1.8 per cent of the first group were rated as having inconsequential behavior while 17 and 5.9 per cent of the other groups, respectively, were found to have inconsequential behaviors. Chazan found the concept of inconsequence to be a useful one, but feels it reflects less severe behavior than that which is described as hyperkinetic. Stott (1965) has postulated a neurological cause for inconsequence.
This review of the literature has examined the complexity of terminology in LD, the inadequacy of medical diagnosis and reliance on psychological assessment historically. Different tests and theories of brain function have been examined and theoretical rationale for the present study established through Luria. Developmental studies of brain function were explored. General theories of LD and classification schemes for LD children have been reviewed with special emphasis given to those which stress defects or developmental lags. Finally, the literature on social maladjustment in LD children was summarized.

It can be seen that there is a complex interaction between emotional, neuropsychological, and learning factors in LD cases. The present study will attempt to delineate the relationships between these classes of variables.
Subjects. Learning disabled children were identified by their enrollment in special programs for LD children in public and private schools. No specific behavioral characteristic was used to define LD other than the school having labeled the child as such. This was done so as to avoid confounding of the variables subsumed under the general category of learning disability (Graham and Berman, 1981).

Three school settings were involved in obtaining children. The South Western City School District outside of Columbus, Ohio contributed 20 children. St. Vincent’s Children’s Center, Columbus contributed 6, and Ashbourne School, Elkins Park, Pennsylvania 17. In all, 43 children were included, with Mean Age = 10.2 years and a range of 7 to 14 years of age. The sex ratio of the sample was in close agreement to that found in the general LD population, with 36 boys and 7 girls (Bannatyne, 1971). Children came from a wide range of SES levels, as assessed by school staff. The South Western City School District children came from working class homes in suburban Columbus. Children at St. Vincent’s Children’s Center came largely from lower class families living in center city. Ashbourne School children are not so easily categorized.
Those subjects ranged from low to high SES, as the school is a privately owned but publicly subsidized facility for LD children.

All 43 children were given the Halstead-Reitan Neuro-psychological Test Battery in offices at their school. The Wechsler Intelligence Scale for Children (WISC) and Wide Range Achievement Test (WRAT) were administered by an experimenter if the school did not have results for these tests as recent as one year old. On the basis of the results of the pilot study (Schachter, 1974), the chief investigator decided to omit the Categories Test and the Tactile Forms Test of the Halstead-Reitan battery as these instruments showed few significant loadings in the factor analytic solution.

The 17 children from Ashbourne School were the only subjects for whom Bristol Social Adjustment Guides could be obtained. Staff turnover and graduation of children from the other two LD programs, and the time lag between testing of those children and availability of the BSAG made it impossible to obtain accurate social adjustment data.

Treatment of children, including maintenance of confidentiality of results, was in accord with APA ethical principles. All testing was done with the permission of the proper school authorities and parents. Short clinical reports were submitted to the children's school in exchange
for the opportunity to employ the subjects in the study.

Procedure. As the WISC and WRAT are commonly used psychometric instruments, no attempt will be made to describe them here (Wechsler, 1949; Jastak and Jastak, 1965). The testing sessions were divided between administration of the WISC and WRAT and the Halstead-Reitan tests proper. The latter are unfamiliar to many psychologists, and so will be described in some detail. For many of these tests, no standardized wording of instructions has been devised. This is a deliberate strategy in Reitan's (1966) overall effort to reduce the effects of emotional factors and misinterpretation of instructions on the outcome of the tests. There are however, guidelines for instructing the subject as to what is expected of him. These will be included in the descriptions to follow.

The Tactual Performance Test examines the abilities to use tactual cues and to construct spatial concepts without the use of vision. A six piece modified Sequin-Goddard formboard was placed before the subject at a 70 degree angle to the tabletop. The board was placed long side horizontal for children 5 through 8 years old, and long side vertical for subjects 9 through 15. The child was blindfolded before the test was taken out of its case and after it was replaced, the blindfold was removed. Subjects were instructed to put the pieces in the holes and that there were six pieces in all. Three trials were given, the first required use of the preferred hand only, then
the other hand only, and finally both hands were used. The puzzle was then replaced and the children asked to draw a picture of the formboard, indicating shape and location of each form. Time for completion of each trial and total time elapsed were recorded along with the number of blocks correctly remembered by shape and by location.

A lateral dominance examination was administered to each subject. The child was asked to demonstrate 7 tasks with his hands and 2 with his legs. The experimenter recorded which side the child performed the demonstrations with, left or right. Included in the lateral dominance exam were two items involving aiming a gun and locking through a telescope and the Miles A-B-C Test of Ocular Dominance. In the latter, as in all of the above lateral dominance items, the subject was never apprised of the fact that E was examining sidedness. In the Miles Test, the child held a cardboard, V-shaped funnel up to his eye, sighted through it, and attempted to identify the picture on a small card which E was holding 10 feet away. Ten trials were given using 10 different pictures. Subjects choose one of three funnels for each trial, which necessitated a new motoric adaptation every time. Subjects pick up the V-scopes with either hand, as E simply recorded the eye that was being used on each trial. The V-scopes were so constructed as to obviate vision through the other eye. Additional items in the Lateral Dominance Exam include timing of subjects writing of his first name with each hand and measurement of strength of grip with each hand. The
latter measure was omitted from this study as Reitan (personal communication) has shown it to be the poorest predictor of behavioral change due to brain damage of 41 variables selected for study by him.

The Halstead finger Oscillation test was administered to assess motor speed. Procedures followed for this test were those proposed by Spreen and Gaddes (1966). The finger tapping devices are supplied through Dr. Reitan's Laboratory. Younger children, 5-8 years old used an electric counter mounted on a heavy wooden base, while older subjects used a manual counter mounted on a masonite base. In both instances, the child was given a 50 finger tap warm-up period and was then required to tap as fast as he could for six 10-second trials. The hand must remain in a fixed position, with the child using only her index finger to oscillate the lever of the finger tapping device. First a set of trials was given using the preferred hand, followed by the non-dominant hand trials.

The sensory-perceptual examination was a simplification, with standardized procedures, of some aspects of the classical neurological examination (Reitan, 1966). In the first phase of this series of procedures tactile imperception is scrutinized. The examiner taps gently on the child's right side, left side or both sides as the child indicated which side was tapped. The experimenter first determined the minimum amount of force needed to elicit a response, using a plastic stylus to deliver the stimuli.
With the child's eyes closed, the examiner randomly delivered tactile stimuli unilaterally and bilaterally to the following pairs of areas in this order: right and left hands, right hand and left cheek, left hand and right cheek. The subject was never informed that bilateral stimulation would be given, but merely to respond "left or right" to the tap. The order of presentation of stimuli in the tactile imperception test, as well as the auditory and visual imperception tests, described below was as follows: right, left, left, right, both, left, right, both, both, left, both, and right sides. In all of the sensory imperception tests, inability to perceive bilateral stimuli is pathognomonic of damage to the hemisphere contralateral to the side of the body which exhibits sensory suppression.

Errors of imperception to unilateral stimulation are equivocal in meaning. Errors of confusion, as in reporting that the opposite extremity had been touched, were not scored as imperceptions. By touching Ss' hand and face combinations, it was possible to control for left-right confusion by having subjects respond with the words "face" or "hand".

Auditory bilateral sensory suppression was examined as the examiner stood behind the subject and made a rasping sound with his fingertips at the child's ears. The experimenter first established the minimum audible sound for each child and instructed her to close her eyes. Stimuli were then delivered in the same order as for the tactile
imperception test. The same diagnostic rational applied as above.

Visual imperception was tested as well. The experimenter sat 10 feet in front of the subject and instructed him to stare at the tip of E's nose. The child then indicated which of the examiner's outstretched hands was wiggled. Examiners completed a full series of stimulations in the order given above.

The finger Agnosia Test required closing of the child's eyes as the examiner gently touched, in random order, first the digits of the dominant and then those of the non-dominant hand. Subject and experimenter work out a system whereby the child could identify fingers by name or number. This tested basic tactile perceptual ability and intactness of the contralateral sensory strip. Each finger was tapped four times during this test.

The Fingertip Number Writing and Fingertip X & O tests analyzed the patient's capacity to perceive symbols presented by physically tracing on the fingertips. For children aged 5 - 8, Xs and Os were traced on their finger tips using a stylus. The numbers 3, 4, 5, and 6 were the traced stimuli for older children. S closed his eyes as E traced symbols first onto the right hand and then the left, using a standardized format (Reitan, mimeographer instructions for Aphasia Screening and Sensory-Perceptual Exam, no date). For these tests as well as the other
tests of sensory perception, only errors were recorded.  
8 merely calls out the symbol which he perceived on each presentation. The symbol writing tests overlay considerably in the function examined with the Finger Agnosia Test, although the former presented 8 with a different discrimination.

The Reitan modifications of the Halstead-Wepman Aphasia Screening Exam were also administered. Younger and Older Children's versions were employed. The former was given to Ss below 9 years of age. These tests are available through Dr. Reitan's laboratory along with an instruction manual and will not therefore be described in item by item detail. Both versions of the instrument examine symptoms classically related to aphasia. Several items are used to examine any particular symptom. Diagnostic categories explored include anomia, dyslexia, dysgraphia, dyspraxia, dyscalculia, and agnosia. A spiral bound pad of stimulus cards is employed, containing figures to be drawn, pictures for identification, and reading materials.

The Rhythm Test requires sustained attention and the ability to perceive rhythmic sequences. It is a sub-test of the Seashore Tests of Musical Talent. (Seashore, Lewis and Saetwitz, 1956). The child was permitted to adjust the loudness of a cassette tape recorder used to deliver the sound so as to control for deficits in auditory acuity. The child is instructed to determine if both members of pairs of rhythmic beats are the same or different. It
appears to be a useful index of auditory analysis, free from language training. An error score is obtained.

The Trail Making Test consisted of two parts, A and B. Each part consists of a sample test on one side of a sheet of paper, and the actual test on the reverse side. In part A, the subject must connect circles numbered from 1 to 15 with a pencil line as quickly as possible. In part B, the subject was required to connect circles using alternating sequences of numbers and letters. Circles are numbered from 1 to 8 and lettered A to G. The child proceeds in ascending order, alternating between numbers and letters. This test examines visual scanning ability, the ability to identify and organize a sequence of operation, and symbol recognition. It is differentially sensitive to frontal lobe damage, (Reitan, 1959b).

The teachers of the 17 subjects from Ashbourne School were asked to fill out a copy of the BSAC for each child in their class who was part of the study. Teachers had not been informed of any details of the study. The BSAC takes about 15 minutes to complete (Stott, 1970) and contains statements which are circled when applicable to the child being considered. The guides contain many distractor items which are not scored in the clinical syndromes.

The chief investigator scored all test materials after data had been collected on all children from a given setting. Names were omitted from the data tabulation sheets until after they had been filled out. Testing
was conducted by the principle investigator and two assistants.
Results

Treatment of Data. In order to avoid the problem of comparing test scores which have different number scales, 43 variables derived from the WISC, WRAT, and Halstead-Reitan tests were converted to Z-scores. The WISC and WRAT manuals were used (Wechsler, 1949, Jastak and Jastak, 1965) respectively. Knights (1970) developmental norms were used for all of the Reitan tests except the Tactual Performance and Finger Oscillation Tests. The norms of Spreen and Gaddes (1969) were applied to those results.

In some instances, children below 9 years old were given tests which were downward extensions of those given to older subjects. Younger children were given the Progressive Figures Test in lieu of Trail Making B, and Fingertip X & O Writing rather than number writing. The younger children's version of the Aphasia Screening Examination was administered when applicable. This was coded using the system of Telegdy, Richardson, and Knights (1969), for both younger and older subjects. As the coding system is somewhat different at the two age levels, a manipulation of the data was performed so as to have comparable and complete data for all subjects. Spelling, right-left discrimination, and dysarthria were omitted as variables as they appeared in only one version of the Aphasia Examination. Auditory agnosia and body orientation were coded as equivalents for younger and older subjects as the items overlapped.
While the use of Z-scores eliminated scaling problems from the data, two other problems emerged. Most of the Halstead-Leitai tests were error scores. While the IQ scores were increasing, this indicated superior performance. But when error scores increased, this would indicate poor performance. Therefore, high error scores were coded as negative Z-scores, low ones as increments in the Z-score. This allows for the direction of the Z-score to uniformly indicate the same thing across all variables. The higher the score the more efficient the performance, whether originally measured in errors, time, or number of items correct.

On the timed tests, there was no upward limit to the time it might take a subject to complete the task. Theoretically, there would have been no downward limit for negative Z-scores. To avoid the spurrious correlations which would have resulted from the ceiling on positive Z-scores, the investigator placed a downward limit of -4 on negative scores. Some of the values eliminated by this method were as low as -16.

In all, 43 variables were originally included in the factor analysis and profile analysis. Table 1 lists all of these, but the reader will note that not all variables were included in every statistical analysis performed. This fact is noted in the relevant portions of this chapter.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean Z-score</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. VISC Verbal IQ</td>
<td>-0.8162</td>
<td>0.8034</td>
</tr>
<tr>
<td>2. Performance IQ</td>
<td>-0.3813</td>
<td>0.8449</td>
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<tr>
<td>3. Full Scale IQ</td>
<td>-0.6581</td>
<td>0.9477</td>
</tr>
<tr>
<td>4. Comprehension</td>
<td>-0.5255</td>
<td>1.0377</td>
</tr>
<tr>
<td>5. Similarities</td>
<td>-0.1697</td>
<td>0.9477</td>
</tr>
<tr>
<td>6. Arithmetic</td>
<td>-0.1023</td>
<td>0.9477</td>
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<tr>
<td>7. Digit Span</td>
<td>-0.7558</td>
<td>0.8446</td>
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<tr>
<td>8. Coding</td>
<td>-0.6116</td>
<td>0.8034</td>
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<tr>
<td>9. Picture Completion</td>
<td>-1.906</td>
<td>0.9477</td>
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<tr>
<td>10. Picture Arrangement</td>
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<td>11. Block Design</td>
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<td>12. Object Assembly</td>
<td>-1.372</td>
<td>1.026</td>
</tr>
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<td>13. Apraxia Screening</td>
<td>-6.023</td>
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<td>14. Dysgraphia</td>
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<td>15. Dyslexia</td>
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<td>16. Auditory Agnosia/Body Orient.</td>
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<td>17. Dyspraxia</td>
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<td>18. Dyscalculia</td>
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<td>19. Total Errors Aphasia Screening</td>
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<td>1.1478</td>
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<tr>
<td>20. Rhythm Test</td>
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<td>1.1218</td>
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<tr>
<td>21. WRAT Reading</td>
<td>-1.148</td>
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<td>22. WRAT Spelling</td>
<td>-1.420</td>
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<td>23. WRAT Arithmetic</td>
<td>-1.106</td>
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<td>24. Trail Making A time</td>
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<td>25. Trail Making A errors</td>
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<td>26. Trail Making B time</td>
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<td>27. Trail Making B errors</td>
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<td>28. Tactual Performance Test (TPT)</td>
<td>.2697</td>
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<tr>
<td>Dominant Hand time</td>
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<td>29. TPT Non-dominant time</td>
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<td>30. TPT Memory</td>
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<tr>
<td>31. TPT Location</td>
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<td>32. Finger Tapping Dominant</td>
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<td>1.431</td>
</tr>
<tr>
<td>33. Finger Tapping Non-dominant</td>
<td>-1.193</td>
<td>1.225</td>
</tr>
<tr>
<td>34. Tactual Impression Dominant</td>
<td>.1325</td>
<td>1.235</td>
</tr>
<tr>
<td>35. Tactual Impression Non-dominant</td>
<td>-1.279</td>
<td>1.568</td>
</tr>
<tr>
<td>36. Auditory Impression Dominant</td>
<td>-1.488</td>
<td>1.465</td>
</tr>
<tr>
<td>37. Auditory Impression Non-dominant</td>
<td>-1.093</td>
<td>1.060</td>
</tr>
<tr>
<td>38. Visual Impression Dominant</td>
<td>.3860</td>
<td>1.129</td>
</tr>
<tr>
<td>40. Finger Agnosia Dominant</td>
<td>-2.127</td>
<td>1.869</td>
</tr>
<tr>
<td>41. Finger Agnosia Non-dominant</td>
<td>-2.369</td>
<td>1.727</td>
</tr>
<tr>
<td>42. Finger Number Writing Dominant</td>
<td>-0.9744</td>
<td>1.1943</td>
</tr>
<tr>
<td>43. Finger Number Writing Non-dominant</td>
<td>-0.7534</td>
<td>1.812</td>
</tr>
</tbody>
</table>
**Factor Analysis.** A Wherry-Wherry 250 Factor Analysis was performed on the 43 variables comprising the WISC, WRAT, and Halstead-Reitan test results, converted to Z-scores. The first solution obtained was heavily influenced by the inclusion of the WISC sub-tests in the correlation matrix. The WISC sub-tests inter-correlated so highly as to obscure the factors related to neuropsychological deficit. Previous investigators (Halstead and Rennick, 1966, Rourke, 1975) have found WISC sub-tests to have less variability than the Halstead-Reitan tests do with children. The present author therefore omitted from the factor analysis the nine WISC sub-tests which had previously been included. This method allowed for examination of the patterns of neuropsychological tests and had the additional advantage of reducing the number of variables to 34. With 43 subjects, the reduced number of variables adds to the validity of the analysis.

The Wherry-Wherry 250 yields a varimax solution, using both the minimum residual and principal factors methods. This program also yields a hierarchical solution, which was found to be unreliable in this case. The distribution of residuals for the varimax solution was found to be well within acceptable limits. Most of the residuals were distributed close to zero, with few at the extremes.
Table 2. Distribution of Residuals

<table>
<thead>
<tr>
<th>Lower Limit</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>.149</td>
<td>0</td>
</tr>
<tr>
<td>.140</td>
<td>1</td>
</tr>
<tr>
<td>.131</td>
<td>0</td>
</tr>
<tr>
<td>.122</td>
<td>0</td>
</tr>
<tr>
<td>.113</td>
<td>3</td>
</tr>
<tr>
<td>.104</td>
<td>1</td>
</tr>
<tr>
<td>.095</td>
<td>3</td>
</tr>
<tr>
<td>.086</td>
<td>4</td>
</tr>
<tr>
<td>.077</td>
<td>6</td>
</tr>
<tr>
<td>.068</td>
<td>7</td>
</tr>
<tr>
<td>.059</td>
<td>15</td>
</tr>
<tr>
<td>.050</td>
<td>13</td>
</tr>
<tr>
<td>.041</td>
<td>21</td>
</tr>
<tr>
<td>.032</td>
<td>39</td>
</tr>
<tr>
<td>.023</td>
<td>30</td>
</tr>
<tr>
<td>.014</td>
<td>47</td>
</tr>
<tr>
<td>.005</td>
<td>62</td>
</tr>
<tr>
<td>-.004</td>
<td>62</td>
</tr>
<tr>
<td>-.013</td>
<td>46</td>
</tr>
<tr>
<td>-.022</td>
<td>52</td>
</tr>
<tr>
<td>-.031</td>
<td>43</td>
</tr>
<tr>
<td>-.040</td>
<td>38</td>
</tr>
<tr>
<td>-.049</td>
<td>23</td>
</tr>
<tr>
<td>-.058</td>
<td>15</td>
</tr>
<tr>
<td>-.067</td>
<td>12</td>
</tr>
<tr>
<td>-.076</td>
<td>4</td>
</tr>
<tr>
<td>-.085</td>
<td>6</td>
</tr>
<tr>
<td>-.094</td>
<td>6</td>
</tr>
<tr>
<td>-.103</td>
<td>0</td>
</tr>
<tr>
<td>-.112</td>
<td>2</td>
</tr>
<tr>
<td>-.121</td>
<td>0</td>
</tr>
<tr>
<td>-.130</td>
<td>0</td>
</tr>
<tr>
<td>-.139</td>
<td>0</td>
</tr>
<tr>
<td>-.148</td>
<td>0</td>
</tr>
<tr>
<td>-.157</td>
<td>0</td>
</tr>
</tbody>
</table>
A cut-off point for including a variable into any factor was set at .2140 by doubling the highest residual, .157. In practice, only two variables were included below .3200. This was done in accordance with the method of Wherry (personal communication). In all 10 factors were generated and 8 are believed to be meaningful. All 10 factors are listed with tables below, and justification is given for the choice of name applied to each.

Table 3. Factor 1: Auditory Analysis Deficit

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>12. WRAT Reading</td>
<td>0.7115</td>
</tr>
<tr>
<td>13. WRAT Spelling</td>
<td>0.6949</td>
</tr>
<tr>
<td>6. Dyslexia</td>
<td>0.4210</td>
</tr>
<tr>
<td>11. Rhythm Test</td>
<td>0.3988</td>
</tr>
<tr>
<td>32. Finger Agnosia Non-dominant</td>
<td>0.3851</td>
</tr>
<tr>
<td>28. Auditory Imperception Non-dom.</td>
<td>-0.3351</td>
</tr>
</tbody>
</table>

The very high loadings on such items as WRAT Reading and Spelling are accompanied with the presence of deficient scores on the Rhythm Test. The latter is a test of non-language auditory analysis. While the developmental parameters of auditory analysis skills are little known, Luria (1974) suggests a relationship between such skills and learning to read. The variables loading on this factor have both indications of LD and of cerebral dysfunction.
Table 4. Factor: Spatial Relations Deficit

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>21. TPT Memory</td>
<td>.8306</td>
</tr>
<tr>
<td>2. Performance IQ</td>
<td>.5628</td>
</tr>
<tr>
<td>22. WRAT Spelling</td>
<td>.5196</td>
</tr>
<tr>
<td>3. Full Scale IQ</td>
<td>.5152</td>
</tr>
<tr>
<td>32. Finger Agnosia Non-dominant</td>
<td>.3599</td>
</tr>
<tr>
<td>19. TPT Dominant</td>
<td>.3388</td>
</tr>
<tr>
<td>18. Trail Making B errors</td>
<td>-.3355</td>
</tr>
<tr>
<td>12. WRAT Reading</td>
<td>.3292</td>
</tr>
<tr>
<td>1. Verbal IQ</td>
<td>.3225</td>
</tr>
</tbody>
</table>

This factor is called aspatial relations cluster due to the high loadings of two variables from the Tactual Performance Test and the presence of Performance IQ. All of these variable are based on tasks requiring the use of spatial cues, some visual, some haptic.

Table 5. Factor 3: Motoric Speed Dysfunction

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>23. Finger Tapping Dominant</td>
<td>.8113</td>
</tr>
<tr>
<td>24. Finger Tapping Non-dominant</td>
<td>.7531</td>
</tr>
<tr>
<td>17. Trail Making B time</td>
<td>.4721</td>
</tr>
<tr>
<td>19. TPT Dominant</td>
<td>.4652</td>
</tr>
<tr>
<td>26. Tactile Imperception Dom.</td>
<td>.4240</td>
</tr>
<tr>
<td>20. TPT Non-dominant</td>
<td>.3939</td>
</tr>
</tbody>
</table>

This factor would also appear to reflect spatial confusion at first glance, due to loadings on the TPT variables. However, the extremely high loadings on dominant and non-dominant Finger Tapping strongly indicate that the poor TPT performance of children with this cluster is mainly the result of slow motoric speed. This is in accord with Reitan's (1959b) inferential system.
Table 6. Factor 4: Visual-spatial Deficit

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>29. Visual Imperception Dom.</td>
<td>.6361</td>
</tr>
<tr>
<td>26. Tactile Imperception Non-dom</td>
<td>.4950</td>
</tr>
<tr>
<td>15. Trail Making A time</td>
<td>.4812</td>
</tr>
<tr>
<td>20. TPT Non-dominant</td>
<td>.4557</td>
</tr>
<tr>
<td>5. Dysgraphia</td>
<td>-.3890</td>
</tr>
<tr>
<td>9. Dyscalculia</td>
<td>-.3445</td>
</tr>
<tr>
<td>3. Full Scale IQ</td>
<td>.3193</td>
</tr>
</tbody>
</table>

Visual and tactile imperception have the highest loadings on this factor. Also notable are the presence of Trail Making A time and TPT Non-dominant variables. Both of these require good orientation in space. This factor differs from the others in the presence of visual difficulties. It was therefore deemed appropriate to label this factor Visual-spatial Deficit in that visual as well as tactual perception problems are present.

Table 7. Factor 5: Tactile Sensory Deficit

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>8. Dyspraxia</td>
<td>.6608</td>
</tr>
<tr>
<td>18. Trail Making B errors</td>
<td>-.5010</td>
</tr>
<tr>
<td>25. Tactile Imperception Dom.</td>
<td>.4328</td>
</tr>
<tr>
<td>4. Anomia</td>
<td>.4037</td>
</tr>
<tr>
<td>17. Trail Making B time</td>
<td>-.3835</td>
</tr>
<tr>
<td>10. Aphasia Screening Total errors</td>
<td>.3519</td>
</tr>
<tr>
<td>30. Visual Imperception Dominant</td>
<td>-.3389</td>
</tr>
<tr>
<td>32. Finger Agnosia Non-dominant</td>
<td>.3180</td>
</tr>
</tbody>
</table>
The several negative loadings, indicative of good performance or pathology indicating variables allows the clinician to identify this cluster by both positive and negative symptoms. With dyspraxia, tactile imperception dominant, and finger agnosia non-dominant, this may be a cluster reflecting tactile sensory problems and damage to the sensory strips of the brain. Language deficits are represented prominently in this cluster, via the Aphasia Screening Test variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>33. Finger Number Writing Dom.</td>
<td>.8113</td>
</tr>
<tr>
<td>34. Finger Number Writing Non-dom.</td>
<td>.7319</td>
</tr>
<tr>
<td>14. WRAT Arithmetic</td>
<td>.6337</td>
</tr>
<tr>
<td>13. WRAT Spelling</td>
<td>.4915</td>
</tr>
<tr>
<td>10. Aphasia Screening Total errors</td>
<td>.4887</td>
</tr>
<tr>
<td>7. Auditory Agnosia</td>
<td>.4836</td>
</tr>
<tr>
<td>9. Dyscalculia</td>
<td>.4570</td>
</tr>
<tr>
<td>12. WRAT Reading</td>
<td>.4522</td>
</tr>
<tr>
<td>1. Verbal IQ</td>
<td>.4500</td>
</tr>
<tr>
<td>3. Full Scale IQ</td>
<td>.4159</td>
</tr>
</tbody>
</table>

The loadings on this factor are uniformly high. Higher level sensory perceptual skills are seen to be deficient and there are many signs of ID from the WRAT and the Aphasia Screening Test. This is labeled the Tactile Perceptual Deficit since more subtle discriminations in tactile somesthetic sense are required on the variables in this group than on those in Factor 5. The latter comes closer to being a sensory rather than a perceptual problem.
Table 5. Factor 7: Spatial Sequencing

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>28. Auditory Impression Non-dominant</td>
<td>-.5732</td>
</tr>
<tr>
<td>19. TPT Dominant</td>
<td>.5486</td>
</tr>
<tr>
<td>5. Dysgraphia</td>
<td>.4943</td>
</tr>
<tr>
<td>27. Auditory Impression Dominant</td>
<td>-.4845</td>
</tr>
<tr>
<td>16. Trail Making A errors</td>
<td>.4035</td>
</tr>
<tr>
<td>14. WRAT Arithmetic</td>
<td>.3989</td>
</tr>
</tbody>
</table>

The loadings here are relatively strong, as with Factor 5 there are positive and negative loadings. Again, this would allow for a dissociation of this cluster from others by the presence of positive symptoms. The TPT and Trail Making Test require the subject to sequentially deal with space. In the former this is through fitting in the pieces of a formboard, while in the latter through connecting numbered circles. The presence of dysgraphia would indicate an association between deficits in spatial sequencing and the inability to copy from writing. Factors 4 and 6 overlap this one.

Table 10. Factor 8: Left Pronto-temporal dysfunction, left hemisphere

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>6. Dyslexia</td>
<td>.7089</td>
</tr>
<tr>
<td>10. Aphasia Screening Total errors</td>
<td>.4938</td>
</tr>
<tr>
<td>18. Trail Making B errors</td>
<td>.4765</td>
</tr>
<tr>
<td>1. Verbal IQ</td>
<td>.4531</td>
</tr>
<tr>
<td>3. Full Scale IQ</td>
<td>.4235</td>
</tr>
<tr>
<td>32. Finger Agnosia Non-dominant</td>
<td>.3900</td>
</tr>
<tr>
<td>31. Finger Agnosia Dominant</td>
<td>.3831</td>
</tr>
<tr>
<td>5. Dysgraphia</td>
<td>.3549</td>
</tr>
<tr>
<td>14. WRAT Arithmetic</td>
<td>.3398</td>
</tr>
</tbody>
</table>
This appears to be a very strong factor, with many high loadings. The highest of these is dyslexia, and it is associated with variables 10 and 18. The first is indicative of left frontal lobe dysfunction and the second with left temporal lobe dysfunction (Reitan, 1959b). Also present are indicators of bilateral parietal dysfunction, variables 31 and 32. It is believed that this cluster reflects diffuse brain damage with maximal impairment to the left frontal and temporal areas, and lesser involvement of the rest of the left hemisphere.

Table 11. Factor 9: No meaning attributed

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>31. Finger Agnosia Dominant</td>
<td>.7089</td>
</tr>
<tr>
<td>11. Rhythm Test</td>
<td>.5971</td>
</tr>
<tr>
<td>4. Anosia</td>
<td>.5673</td>
</tr>
<tr>
<td>15. Trail Making A time</td>
<td>.3750</td>
</tr>
<tr>
<td>34. Finger Number Writing Dominant</td>
<td>.3712</td>
</tr>
<tr>
<td>2. Performance IQ</td>
<td>.3279</td>
</tr>
<tr>
<td>30. Visual Impression Non-dom.</td>
<td>-.3238</td>
</tr>
</tbody>
</table>

Table 12. Factor 10: No meaning attributed

<table>
<thead>
<tr>
<th>Variable</th>
<th>Loading</th>
</tr>
</thead>
<tbody>
<tr>
<td>25. Tactile Impression Dominant</td>
<td>-.4456</td>
</tr>
<tr>
<td>4. Anosia</td>
<td>-.4181</td>
</tr>
<tr>
<td>22. TPT Location</td>
<td>.3832</td>
</tr>
<tr>
<td>7. Auditory Agnosia</td>
<td>.3244</td>
</tr>
</tbody>
</table>

This investigator could find no explanation to account for these two factors. The occurrence of chance groupings is a common problem in factor analysis.
Profile Analysis. In order to find common profiles of deficits among the sample, and thereby derive sub-groups of ID children, the Wherry PROANS method was used. This yielded a regular inverse factor analysis, grouping subjects rather than variables. The solution was not acceptable, with subjects showing no sub-groups. Almost all of the factor loadings were zero. The residual distribution revealed most frequent occurrence of residuals at the extreme high and low ends.

Correlational analysis. A point biserial correlation was computed for 43 WISC, WRAT, and Halstead-Reitan battery variables and 5 variables derived from the BSAG. Using Stott's (1970) norms, it seemed that maladjustment could not be considered to be normally distributed nor a continuous measure. The five core syndromes of the BSAG were scored for moderate to severe maladjustment versus no significant maladjustment. This yielded a dichotomous variable, scored one or zero, for maladjustment versus no maladjustment, respectively. The original 43 variables were coded as Z-scores and the 5 BSAG variables as dichotomous categories.

Table 13. Results of Bristol Social Adjustment Guides

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Unforthcomingness</th>
<th>Withdrawl</th>
<th>Depression</th>
<th>Inconsequence</th>
<th>Hostility</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean:</td>
<td>2.11</td>
<td>0.88</td>
<td>1.05</td>
<td>5.5</td>
<td>6.55</td>
</tr>
<tr>
<td>Standard Deviation:</td>
<td>2.57</td>
<td>1.57</td>
<td>2.13</td>
<td>5.05</td>
<td>4.64</td>
</tr>
<tr>
<td>% Cases Maladjusted:</td>
<td>5%</td>
<td>29%</td>
<td>17%</td>
<td>41%</td>
<td>58%</td>
</tr>
</tbody>
</table>

Based on a sample of 17 children from Ashbourne School and using the norms and cutoff scores of Stott (1970).
It can be seen that this sample tended to show a higher incidence of the over-reactive syndromes, Inconsequence and Hostility, than of the under-reactive ones. Hostility was the most frequent syndrome to appear in the moderate to severe range of maladjustment rather than Inconsequence, which has more congruence to the term hyperactivity. This was contrary to the expectations of the chief investigator.

Table 14. Variables with Significant Point Biserial Correlations to ESAG Core Syndromes P<.05

<table>
<thead>
<tr>
<th>Variable</th>
<th>Syndrome a</th>
<th>W</th>
<th>D</th>
<th>Q</th>
<th>H</th>
</tr>
</thead>
<tbody>
<tr>
<td>4. Comprehension</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.5244</td>
</tr>
<tr>
<td>14. Dysgraphia</td>
<td></td>
<td></td>
<td>-.4905</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16. Aud. Agnosia</td>
<td></td>
<td></td>
<td></td>
<td>-.4993</td>
<td></td>
</tr>
<tr>
<td>16. Dyscalculia</td>
<td></td>
<td>-.5085</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23. WRAT Arithmetic</td>
<td></td>
<td>-.4898</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>26. Trails B errors</td>
<td></td>
<td>-.5044</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>35. Tactile Impercep.</td>
<td></td>
<td>-.5733</td>
<td>-.5633</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Dominant

Syndromes listed by first initial except Inconsequence=Q. No significant correlations found with Unforthcomingness.

Only 8 of 215 correlations in the matrix reached the .05 level of significance. This is only 3.7% of the total number of correlations in the matrix and is well within chance expectancy.
Discussion

Hypotheses 1 and 3

Hypothesis 1 postulates that different neuropsychological deficits acting alone or in concert may underlie LD. Closely related is hypothesis 3 which states that LD children may be combined into meaningful sub-groups on the basis of common symptoms. The factor and profile analyses are relevant to the above hypotheses. The present investigator does not, however, mean to convey the belief that these statistics comprise anything but an indeterminant solution. The analyses represent a test of the clustering of variables and of subjects, but do represent the kind of test for which there are measures of statistical significance.

The emergence of eight meaningful factors in the data are taken as an indication of the variability of neuropsychological profiles in the sample. The failure to derive sub-groups of children on the basis of profile analysis is also suggestive of their heterogeneity, although it does not prove it logically. The separate factors will be discussed below as they relate the complexity of symptom clusters. The gestalt of the factors will be examined following this.

Factor 1 was labeled an Auditory Analysis Deficit.
Luria (1974) and Chalfant and Scheffelin (1969) note the importance of auditory analysis in the development of reading. Specifically, it is necessary for the child to be able to decode the spoken language of others in order to develop an organized second signal system. Oral language preceded the use of written words, and in fact is seen as its prerequisite (Luria and La Yudovich, 1956). The Rhythm Test is the key variable in Factor 1 in terms of deriving a label. In adults, this test is an index of right temporal function, but its localizing significance is not so clear in children. While it is not possible to lateralize the cerebral hemisphere responsible for this cluster of deficits, it seems likely that it could be either left or right. Presumably, the speech centers in these children's brains have not as yet become fixed and what is of importance is the close association of auditory analysis deficit and LD.

The Motoric Speed Dysfunction cluster may be indicative of bilateral insult to the motor strips of the cerebral hemispheres and also some sensory loss. TPT and Trail Making B are timed tests and are associated with this factor. It is believed that the slowness of children with mild cerebral motor impairment may lead to delays in acquisition of certain other skills. Piagetian theory would predict slow cognitive development to ensue slow perceptual-motor development. Damage to sensory and motor areas in childhood would, in Luria's (1974) framework disrupt the development of secondary cerebral zones and delay academic development or leave it defective.
In line with this reasoning, the Tactile Sensory and Tactile Perceptual factors may likewise retard development and reflect cerebral impairment to the sensory strip and anterior parietal lobes.

The Spatial Relations, Visual Spatial, and Spatial Sequencing factors are indicative of deficits in the simultaneous synthesis of spatial cues and in the orderly sequencing of such cues for the completion of tasks. While the variables underlying these factors are varied, for the most part they indicate damage or dysfunction to the posterior and inferior parietal areas. These dysfunctions may be in part due to haptic processing errors, visual imperception, or planning difficulties. The processing of spatial information is thus associated with a wide range of modality specific variables as well as higher level, multi-modality variables. This complexity is as expected from Luria's (1974) account of the neurodynamics of the tertiary zone at the temporo-occipitoparietal confluence.

Factor 8, labeled the Fronto-temporal dysfunction is representative of diffuse cerebral which is maximal in the left frontal and temporal lobes. Those who work with LD children may see many of their characteristics as deriving from this cluster. Above all, the children cannot read, but they also show planning difficulties, writing problems, and other deficits.
The factor analytic solution, as a whole, is highly suggestive of the complexity of neuropsychological problems to be found across LD children. There have been noted defects in tactile, motor, auditory, visual, and higher level systems. The latter include the prefrontal and temporal-parietal-occipital zones. The present author can only reiterate Luria's (1974) argument for detailed symptom analysis as a logical extension of these findings. The fact of dyslexia or dysgraphia is not adequate for making remediation formulae. Clinicians and educators must examine the entirety of cognitive functions before making plans for remediation. It is clear to this writer that the professionals involved in assessing LD both clinically and in research must learn to take into account a host of variables. This is due to the many possible ways to interrupt functional systems of the brain.

The present study cannot definitively find that these factors are independent. Indeed, it seems more likely that the neuropsychological factors presented may be found acting in concert within individual children. The combinations of the deficits may be quite varied. As Bannatyne (1971) notes, time course and sequence may also vary. For example, child A may have had perinatal head trauma leading to a Motoric Speed dysfunction. Over the years, cell migration and cerebral development may be
disrupted by the original damage, leading to a Fronto-temporal dysfunction. Alternatively, child B may have suffered intrauterine insult leading to both deficit clusters simultaneously. The academic achievement, social adjustment, and severity of dysfunctions may differ in these two cases.

Questions regarding the sequence of development of deficits and their remediation over time must remain for future studies to answer. For the present, it can only be pointed out that the neuropsychological deficits underlying LD appear to be complex and highly variable between subjects. Hypothesis 1 is answered affirmatively by the factor analytic solution in the mind of this investigator.

Profile analysis did not produce factor or sub-groups of children as a basis for a classification system. The present author would be most skeptical of any nosological treatment of LD children. Coupled with the strong results of the factor analysis, it seems likely that different deficits can be grouped, but children cannot. Once again, this is interpreted as illustrating the need for detailed symptom analysis of LD. The third hypothesis is not substantiated by the results.

Hypothesis 2.

This hypothesis stated that it would be possible to find social maladjustment with or without neuropsychological
deficits in LD children. The results of the point biserial correlation as a whole are interpreted to show that social maladjustment and neuropsychological deficits are independent of one another. Only 8 of 215 correlation values reached the .05 level of significance. This is no more than one would expect by chance. In terms of the general relationship between neuropsychological and social maladjustment variables, the two classes appear to be orthogonal to each other. However, regression equation equations were not computed.

In clinical practice, psychologists often try to distinguish between primary and secondary emotional disturbances in relation to brain damage. The first is seen as pre-dating the brain damage or as being independent of it while the second is a reaction to the damage and reduced cognitive efficiency. In this case, the data does not allow for such a distinction to be made. This investigator feels that an acausal relationship exists maladjustment and neuropsychological deficit. Neither can be said to determine the other, but both will be found in individuals.

Apparentely, no particular form of social maladjustment is highly correlated with specific neuropsychological deficits. The presence of one does not predict the occurrence of the other in a significant number of cases. Emotional and social maladjustment following brain damage.
appears to be extremely variable in the sample under scrutiny. It is possible that social maladjustment may be, in some cases, the child's response to the environmental impact of his being labeled "Learning Disabled." That is, there may be complex chains of events leading to social maladjustment which are not adequately conceptualized by the terms primary and secondary emotional disturbance. The environment reacts to the child's deficits and the child in turn is affected by this reaction, and so changes his adjustment pattern. Many other equally convoluted examples might be provided to illustrate this concept of acausal relationships.

Richardson (1964) discussed the interaction between the social environment and the fact of brain damage. He argues that many of the functions affected by brain damage are also affected by the child's environment. The almost uniformly low mean scores on neuropsychological test and the high percentages of social maladjustment in LD children suggest that both contribute to the disability. Their contribution to each other is much less clear.
Conclusion

Neuropsychological and social maladjustment problems are frequent in the LD population. They appear to exhibit statistical independence when assessed by correlational techniques, however. An acausal relationship conception is postulated which sees the two categories of problems as being contributory to each other, but not in the sense of etiologies. Social maladjustment may make neuropsychological deficits more visible by bringing attention to the child generally. Neuropsychological dysfunction may dispose the child towards acting out upon his feelings of frustration or of being different. In turn, both maladjustment and neuropsychological defects lead to labeling, which in turn has consequence in the child's behavior. The neuropsychology of LD, while complex, still seems better understood that the parameters of social maladjustment as a result of this research. Clusters of deficits in brain functioning are seen as interacting within the child. This gives each child's learning disability a kind of neuropsychological uniqueness. Detailed analysis of the underlying mechanisms responsible for LD in each child are called for. Assessment, more than simple diagnosis should be the goal of concerned professionals. Instruments such as the Halstead-Reitan
battery which examine the totality of the sensorium and reveal modality specific and cross-modality deficits should be routinely used in the assessment process.

Luria's view (Luria, 1974) that damage to the modality specific regions can lead to disruption of the functioning of the tertiary zones is sharply illustrated by the factor analysis. Direct dysfunction of the tertiary zones is also a likely cause of LD. Future research is needed to answer questions regarding the developmental parameters in the emergence of cerebral dysfunction, and the time course of restoration of functions. It would also be valuable to get some idea of the extent of recovery or reorganization of the different functional systems of the brain in children. This study cannot shed light on Luria's (1974) contentions regarding the age related effects of brain damage to different cerebral regions.

All in all, this study has been a search for the right questions to ask in regard to LD. Some of these questions are given above. This author believes that LD research should employ multivariate strategies to assess the information processing or neuropsychology of these children. The complexity of their problems holds out the threat of experimental quandry and inability to control for all the variables in question. On the other hand, this same complexity holds the promise of leading to deeper understanding of human developmental neuropsychology.
and of the feedback between brain dysfunction and emotional-social maladjustment.
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