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APPLICATION OF LATENT STRUCTURE ANALYSIS TO THE REITAN-INDIANA APHASIA SCREENING TEST

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APPLICATION OF LATENT STRUCTURE ANALYSIS TO THE REITAN-INDIANA APHASIA SCREENING TEST

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As members of the Final Examination Committee, we certify that we have read the dissertation prepared by Janet Steele Van De Voorde

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TABLE OF CONTENTS

LIST OF TABLES

ABSTRACT

The Reitan-Indiana Aphasia Screening Test was examined using a latent structure analysis which involved the assumptions that a latent variable X (brain function), could explain the relationships among the manifest variables A, B, C, D (categories of responses to the Aphasia Test). It was further assumed that within the latent variable a four-class model would be the preferred model. And, it was assumed that persons clinically assigned to the latent classes would be similarly assigned by their test responses.

Latent class models were used to test item reliability as well as to determine whether or not the four-class model afforded a better fit to the data. Of 19 item pairs tested, all proved to be highly reliable with reliability coefficients of .83 to .85. Fourteen of the 19 showed that the model of equivalence in item pairs afforded an excellent fit to the data. Reliable item pairs were then selected such that variables A and B represented left cerebral hemisphere functions and items C and D represented right cerebral hemisphere functions.

The model was represented by mathematical equations which express the probability of a given response pattern in a contingency table in terms of the joint probabilities of each latent class and the response pattern. Chi-square values were obtained for the model of independence, with 11 degrees of freedom; for the four-class model with eight degrees of freedom; for the three-class model with 10 degrees of

freedom; and for two two-class models with 12 degrees of freedom each. Hierarchical subtractions were made which resulted in a preferred model, leaving a Chi-square value to be referred to the Chi-square distribution to determine whether or not the latent class model offered an improvement of fit to the data. Twelve comparisons were made using different item combinations. All 12 showed the four-class model offered a significant improvement over the model of independence and either of two two-class models tested. Eleven of the twelve comparisons showed that a four-class model provided an adequate fit to the data. In seven of those instances the four-class model was accepted as the preferred model. In the remaining four instances the heirarchical subtractions showed that a four-class model did not offer significant improvement over the three-class model, so the three-class model was accepted as the preferred model due to the criterion of parsimony.

The study showed that there are four categories of brain function which can be identified by means of a behavioral test. It also gave the exact coefficient of agreement between assignment of individuals into those categories by clinical and test classification.

It was concluded that latent structure analysis is an effective technique to describe brain-behavior relationships. It was further suggested that consideration be given to the use of latent trait statistics to continue to refine the Aphasia Test without compromising the extensive results already achieved.

CHAPTER 1

INTRODUCTION

Language, thought, and speech are of such paramount importance to the human organism that they have been the object of study for many hundreds of years. Philosophers, linguists, neuroanatomists, neurologists, speech pathologists, and many subgroups of psychologists have pursued an interest in the nature, structure, anatomy, development, and impairment of the functions of language and speech. One of the most directly involved of the psychologies in the study of brain damage and subsequent language impairment is the recent new discipline of clinical neuropsychology (Benson, 1979). With the initial work of Ward C. Halstead, and the subsequent major research efforts of Ralph M. Reitan, the study of brain damage and impaired language functions has provided important areas of investigation.

Early studies of language and the relationship of the brain as the organ of intellect date back to the fifth century, B.C., but the first formal mention of aphasia and its relationship to brain disorder dates back to 1861 with the findings of Paul Broca (Benson). Since that time when Broca demonstrated that a man with frontal-temporal brain damage suffered a loss of speech, a new interest in the role of the brain in language and speech evolved and continues to grow.

The nature and classification of aphasia, brain localization of the aphasias, and the assessment of aphasia have been addressed throughout the literature. Increased sophistication of apparatus and

techniques for study have led to new methods of assessment which have resulted in even greater clarity and understanding of aphasic disorders.

The early lateralization findings of Broca (1861), Wernicke (1874), and Dejerine (1891), led to decades of validation studies which located the aphasias to the left cerebral hemisphere in the great majority of cases. Among the more recent validational studies are the computerized tomography studies (Damasio & Damasio, 1979a, 1980, 1981; Keretz, Harlock, & Coates, 1979; Mazzochio & Vignolo, 1979; and, Naesar & Hayward, 1978), and the cerebral blood flow studies (Halsey, Blauenstein, Wilson & Wills, 1980; Ingvor & Schwartz, 1974; Meyer, Sakai, Yamaguchi, Yamamoto, & Shaw, 1980; Risberg, 1980; Risberg, Halsey, Wills, & Wilson, 1975; and, Wood, 1980). These same studies locate spatial relations to the right cerebral hemisphere.

Assessment of the aphasias has seen a change from the clinical examinations of Broca, Wernicke, and Jackson (1915), to more formal testing batteries (Head, 1926; Schuell, 1955, 1966; Wepman, 1951; Wissenberg & McBride, 1935). Assessment batteries in use today range from short screening tests which identify the presence or absence of aphasia (Aphasia Language Performance Scale-ALPS, Reitan-Indiana Aphasia Screening Test, and the Sklar Aphasia Scale--SAS), to comprehensive examinations of aphasia (Appraisal of Language Disturbance--ALD, the Boston Diagnostic Aphasia Examination--BDAE, and the Minnesota Test for Differential Diagnosis--MTDDA) (Spreen & Risser, 1981).

One of the most widely used screening batteries for aphasia is the Reitan-Indiana Aphasia Screening Test and the Reitan-Kløve Sensory

Perceptual Examination. The sensitivity of the tests to the organic integrity of the cerebral hemispheres has been demonstrated by Heimburger and Reitan (1961), who correlated aphasia symptoms with left cerebral lesions and constructional apraxias and spatial relations with right cerebral lesions. Wheeler & Reitan (1962), using a set of rules, were able to predict with greater than 78% accuracy lateralization of left cerebral lesions, right cerebral lesions, and bilateral diffuse lesions.

Reitan (unpublished), in a recent study, looked at the differential frequency of occurence of the aphasia symptoms. He postulated that language functions which were learned early in life and were well established were well rooted in brain function and would be more resistant than language functions of more recent origin. He found that the more basic biological functions such as sensory imperceptions in visual, auditory, and tactile modalities, and naming body parts occured with less frequency than did higher order, more complex functions such as constructional dyspraxia, dyscalculia, and central dysarthria. Both receptive and expressive functions were correlated with one another and were found to be about equally related to one another.

The correlation of expressive and receptive functions brings up one of the problems Reitan has encountered in attempts to submit his well-validated tests to statistical analyses. He correctly pointed out the limitations of the statistical technique of correlation when attempting to compare the differential functions of the left and right cerebral hemispheres. Namely, left cerebral functions correlated positively with one another, as did right cerebral functions, but consistently negative correlations occurred when the two sides of the brain were compared with

one another. Statistics have not been able, in many cases, to reflect adequately in meaningful ways the complex workings of the brain.

Wheeler, Burke, & Reitan (1963), applied discriminant functions to 24 behavioral indices and made comparisons between 61 non-brain damaged controls and 79 subjects showing brain damage. Using a single weighted score for each subject, predictions were made comparing controls to all categories of brain damage, with 90.7% success; controls versus left damage, 93%; controls versus right damage, 92.4%; controls versus diffuse damage, 98.8%; and right versus left damage, 92.9%. Several other indices were examined for percentages of correct prediction but the discriminant function was superior in all comparisons.

While such procedures have been useful in making gross discriminations between brain damage and non-brain damage, Reitan has continued to emphasize the complexity of brain function and the need to consider a number of methods of inference in the study of brain impairment. Much of the statistical analyses have attempted to treat the data as a continuum. There has been no methodology which would allow an analysis of qualitatively different response patterns associated with qualitatively different brain lesions. Reitan has identified distinct categories of brain function associated with specific kinds of localization of brain lesions. His existing research suggests a more elaborate categorical approach to the study of brain damage which has not yet been investigated.

The purpose of the present study is to apply a categorical approach to the data analysis of the Reitan-Indiana Aphasia Screening Test as that test relates to brain function, using a technique originally

described by Lazarsfeld (1950a, 1950b), and later expanded by Goodman (1974a) and Bergan (in Press).

Lazarsfeld offered a technique which would permit the consideration of latent, or unobserved, variables by means of latent structure analysis. Goodman later developed latent class models to be used with categorical data to hypothesize causal relationships among categories of latent variables. It is only with recent advances in parameter estimation, and particularly with the use of computer programs that latent class models are being applied in a wide variety of educational research programs (Bergan). No one has yet attempted to apply this effective tool as a means of studying brain damage and describing this kind of categorical data in a new, interesting, and meaningful way.

Latent structure analysis and latent class models will be explained in detail in a later section of this paper, but essentially what is proposed is that this method allows a more elaborate and sophisticated statistical procedure which more accurately reflects the data supported by existing research. While much previous research proposed a two-class model of brain function, namely, normal and brain damaged, it is proposed that a latent class model which suggests four latent classes representing the unobserved variable of brain function is a more adequate representation of the data. These four latent classes are defined as follows: 1) Normal controls, 2) Patients with left cerebral hemisphere damage, 3) Patients with right cerebral hemisphere damage, and 4) Patients with bilateral diffuse damage. The items on the Aphasia Test represent categories of manifest variables, the response

to which may represent an aphasic disorder either receptive or expressive in nature.

Using this four-class model, independent probabilities can be set to predict dysnomia, dyspraxia, dyscalculia, etc., depending on the latent class. Several hypotheses can be formulated based on research which has demonstrated the lateralization of brain function.

1. The first hypothesis is that there is a latent polytomous variable X (brain function) that can explain the relationships among the manifest variables A, B, C, D (categories of response to the Aphasia Test).

2. The second hypothesis is that within the latent variable X, a four-class model is the preferred model to explain the relationship.

3. The third hypothesis is that persons clinically assigned to each of the latent classes will be similarly defined by their test assignment.

If the above hypotheses are true, the following will also be true.

4. Given that an individual is in latent class, Normal, the probability is that he will pass every item and not fail any items, since the Aphasia Test is a sign test, and an error constitutes a pathognomonic sign.

5. Given that an individual is in latent class Left cerebral hemisphere damage, there is a high probability that he will fail those items which reflect left hemisphere function. Conversely, there is a high probability that he will pass those items which reflect right hemisphere function.

6. Given that an individual is in latent class Right cerebral hemisphere damage, there is a high probability that he will fail those items which reflect right hemisphere function. And, there is a high probability that he will pass those items which reflect left hemisphere function.

7. Given that an individual is in latent class Bilateral diffuse, there is a high probability that he will fail those items which reflect both left and right cerebral hemisphere function.

CHAPTER 2

LITERATURE REVIEW

Although the formal study of aphasia as we know it today is only slightly over a century old, the origins of aphasic disorder can be dated back to the earliest medical writings in the eighteenth century, B.C. (Benton, 1981). The relationship of the brain as the organ of intellect dates back to the fifth century, B.C. with Hippocrates of Groton. Herophilus, in the third century, B.C., studied the structure of the brain and regarded the middle ventricle as the seat of cognition and the posterior ventricle as the seat of memory. Galen, one hundred years later, decided it was the substance of the brain itself which accounted for intellect, but it waited for Vesalius 1800 years later to validate that fact (Heilman & Valenstein, 1979). Since then the fact of the brain as the organ of behavior has been accepted, and within the eighteenth century, A.D., the clinical descriptions of aphasia were reported by a number of physicians. The basic nature of aphasia as a neurological entity remained obscure (Benton).

At the end of the eighteenth century, A.D., Franz Joseph Gall proposed that various human faculties were localized in different centers of the brain, and that intellectual qualities were to be found in various parts of the two cerebral hemispheres. Unfortunately for him, he also laid the foundation for the study of phrenology, which led to his eventual disrepute, and obscured the importance of his contribution to modern neuropsychology (Heilman & Valenstein). By the early nineteenth

century there had evolved two clearly distinct beliefs about the brain's function (Benson, 1979). Some investigators followed the lead of Gall and felt strongly that there was localization of brain function, that specific functions were subsumed by specific areas of the brain. Others believed equally strongly that mental function required the entire brain working as a unit and was a reflection of total brain volume (Benson).

Localizationists

The first formal mention of the relation between brain disorder and language loss occurred in 1861 with the now-famous presentation of Paul Broca to an anthropological society in Paris. He produced a case which demonstrated that a man who had suffered frontal-temporal brain damage subsequently showed a loss of speech (Benson). On the basis of this case, he postulated that the posterior-inferior part of the frontal lobe was responsible for speech production. His later extensive research bore this out and led to his wide acceptance as having shown the posterior part of the third frontal convolution of the left hemisphere as the center for articulated speech. It remains today as Broca's aphasia.

In 1869, Bastian defined additional areas for localizing auditory and verbal word centers and centers for the tongue and hand and used diagrams to demonstrate his theories.

Wernicke agreed with Broca that the posterior part of the third frontal convolution was the center for spoken language, but he added the notion of sensory, or receiving, zones of the brain (Wepman, 1951). The sensory aphasia, he thought, was due to the pathology of the left temporal

lobe, while a third aphasia, conduction aphasia, was related to a break in connections between the sensory and motor centers.

While the scope of the present paper does not allow for a full review of all those important contributors to localization theories of aphasia, the following must be noted because of their influence. Lichtheim, in 1885, expanded on Broca and Wernicke and introduced seven types of aphasia; Charcot, in 1889 developed the idea of the writing center; and, Henschen, in 1922, outdid them all by reviewing 1337 cases of aphasia in the published literature, which all leant credence to his structural theories, and from which he described thirty different specific locations in the brain which subserved particular functions (Wepman).

In 1959 Penfield and Roberts published a book which was the culmination of ten years of study of brain dominance, aphasia, and other speech disturbances. Studies of several hundreds of patients with brain damage helped to provide an account of the neurophysiology of language and to locate its mechanisms. Electrical stimulation of the temporal regions elicited activation of earlier memories and experiences, leading to the temporal lobes being described as the interpretive cortex.

Holists

One of the earliest proponents of a more universal approach to aphasia and language function was John Hughlings Jackson, who, in 1884, proposed that aphasia was a dynamic, psychological process which involved the entire brain. He felt that aphasia was due to an inability to propositionalize. The ability to propositionalize was considered to be a two-stage process; the first was the arousal of the mind with the idea, the

second was the fitting of the words, spoken or unspoken, to that idea (Wepman). Perhaps his most meaningful contribution was his introduction of the concept that, "the evolution and dissolution of the nervous system shows a hierarchy of functional levels, with successively higher levels developing later and breaking down sooner in the process of dissolution". The higher level was the ability to propositionalize, the lower level was more automatic and emotional. Not only was this significant for describing what happened to people who suffered brain damage, but it was the basis for Freud's later notion of regression.

Freud followed keenly the ideas of Jackson, and in 1891, published a monograph which criticized the works of the localizationists. Freud felt that although there were problems with the localization theories, they were not entirely incompatible with a functional approach. He described three types of aphasia, and was the first to coin the term, agnosia. He felt that the apparatus of speech was a continuous cortical area in the left hemisphere extending from the terminations of the acoustic and optic nerves and the origins of the motor tracts for the muscles serving articulation. From the psychological point of view he recognized the word as a complex of impressions and images which, through its sensory, or auditory, component was connected with the complex of object associations. With that, he defined verbal aphasia as a disturbance within the word complex, assymbolic aphasia as a separation of the latter from the object associations, and agnostic aphasia as a purely functional disorder of the speech apparatus (Freud, 1953).

As with the localization theories, the holistic theories multiplied, and their influence was felt over the early part of the twentieth

century. Head (1926) gave direction to the psychological approach, and while acknowledging that pathological damage to certain neuroanatomical lesions resulted in certain types of aphasic symptomatology, recognized that acts of greater symbolic formulation required a greater degree of abstraction. He developed an extensive test battery to assess aphasic disorders and included non-verbal as well as verbal material, insisting that such performance measures were valid indicators of the fundamental cognitive capacity that was impaired in aphasia. Wiesenburg and McBride (1935) held that language dysfunction in aphasia was not intimately associated with losses in basic intellectual functions arising from brain damage. Studies by Reitan (1953) agreed with that notion, also held by Jackson.

Goldstein (1942) worked with soldiers in Germany following World War I who had sustained cerebral injuries. He concluded that the problem of aphasia could only be fully understood by the concepts of Gestalt psychology; that for every excitation in a certain place there was a dynamic process which was characterized by the influence of the total nervous system and even the whole organism. Halstead (1939) was able to demonstrate that the individual after brain damage showed an inability to categorize, to shift, and to group things conceptually (Wepman).

Modernists

In the second half of the twentieth century there has been a continued interest in brain-behavior relationships. The work of the classical neurologists has been replicated, and Broca's aphasia, Wernicke's aphasia, and conduction aphasia are all recognized today. In addition, anatomical studies permitted new mapping procedures to determine connections, and

advances in neurochemistry and neuropharmacology led to investigations of behavioral-chemical relationships. It is accepted that all behavior is mediated by physical processes, that complex behavior depends on physical processes of the central nervous system, and that brain lesions disrupt certain aspects of behavior. It is likely that specific functions are indeed subserved by certain areas of the brain, but that more complex abstract functions require the greater portion of the cerebral cortex (Reitan).

Hemispheric Lateralization of Language

The great majority of humans show left cerebral dominance for language functions. This is true for almost every person who is right handed and for most persons who are left handed. Correlation of language syndromes and gross anatomical findings have been shown consistent since the nineteenth century, even though there is considerable variation in the development of intellectual functions between individuals.

As mentioned earlier, Broca observed among his patients that those who lost speech following brain damage had pathology involving the left cerebral hemisphere, while comparable pathology in the right cerebral hemisphere was rarely associated with language disturbances. The incidence of left handed persons who show right cerebral dominance for language remains undefined. Wepman reported in 1951 that even though Luria proposed that they were likely to show bilaterality of language function based on the fact that they showed better recovery rates, Luria's own study of 394 brain-injured patients showed not one case of right brain damage with aphasia.

In the Penfield and Roberts' study of 1959, sodium amytal was injected into the carotid artery while the patient counted in order to determine which hemisphere subserved speech. If the non-dominant hemisphere was involved, the patient stopped counting, then recommenced within thirty seconds. If the dominant hemisphere was involved, the patient remained silent for a minute or two, and then was confused and had trouble naming and reading. Study of 522 patients showed no difference in the frequency of aphasia after operation on the left hemisphere between right and left handed people.

It is important to keep in mind that unilateral damage provides useful information, but may be misleading. A dominant left hemisphere may have significant pathology without aphasia, depending on a number of factors including size, location, and extent of the lesion.

Current research studies

Cerebral dominance as described by Brown and Jaffe (1973) is a continuous process which evolves throughout life and accounts for age-dependent forms of aphasia. They considered the right cerebral hemisphere dominant in infancy for the type of visual and auditory communications relevant to the prelinguistic child. They noted that 88% of newborns posture themselves to the right, meaning that the left eye and ear are the major receptor organs. In dichotic listening studies they found a significant increase in the right ear for verbal material in children between the ages of five and six.

Gott (1973) found that excision of the dominant left cerebral hemisphere for malignancy in adults resulted in severe expressive aphasia

except for expletives, a few stock words, and singing. Removal of the right cerebral hemisphere produced defects is visuospatial and visual memory.

Mazzochi and Vignolo (1979) looked at localization of lesions in aphasia patients as measured by clinical CT scans in stroke patients. Ninety right handed patients were given standardized language batteries and a CT scan. Presence and type of aphasia were correlated with location and extent of CT scan lesion. The findings were compatible with the traditional views about the localization of lesions in aphasia. The authors stressed the importance of the fact that both aphasia and underlying lesion evolve with time. The distinction between acute lesions which are recent, and longstanding lesions must always be made since stabilized lesions show improvement over time.

Underwood and Paulsen (1981) described a 57 year old man who was congenitally deaf, who suffered a CVA with resulting right hemiplegia and aphasia, suggesting that the fundamental linguistic processes are the same for the congenitally deaf and normal hearing population.

Obler, Goodglass, and Benson (1978) analyzed the relationship of age and clinical types of aphasia in 167 right handed men who suffered CVA's. Patients with unequivocal diagnoses such as Broca's, Wernicke's, or conduction aphasia were studied and it was found that the median age for Broca's group (51) was lower than that for the total group (56), while the median age for Wernicke's group was significantly higher (63). Incidence of Wernicke's aphasia increased steadily with age while evidence of other types peaked in the sixth decade and diminished with increasing age.

A number of recent studies have looked at blood flow patterns using the ^{133}Xe-inhalation technique with interesting results. Ingvor and Schwartz (1974) looked at 10 patients with normal neurological findings and studied cerebral blood flow patterns which tended to show that production of spoken language activates regions in the dominant left hemisphere which included not only the upper, the anterior and posterior speech cortex, but also substantial parts of the middle and Rolandic region. This pattern differed from the one recorded during abstract thinking and problem-solving in which frontal and post central associational areas were more activated.

Risberg (1980) demonstrated the ability of rCBF technique for differential diagnosis of dementia, for the evaluation of treatment by drugs, ECT, or psychosurgery, as well as for following cases of toxic influence. In auditory hallucinations there was increased bulk in temporal regions; in visual hallucinations there was increased bulk in occipital regions. Global dysfunction in the cases of organic brain disease and toxic influence was shown. In amphetamine abuse with accompanying feelings of paranoia there was a doubling of the frontal flow levels.

Consistent and statistically significant hemispheric asymmetries were demonstrated by Risberg, Halsey, Wills and Wilson (1975b). A verbal analogy test gave larger flow increases on the left side, particularly over Wernicke's areas, while a non-verbal perceptual test gave a right superiority in frontal and parietal regions in normals.

Maximillan, Prokovnik, Risberg, and Hahansson (1978) demonstrated the involvement of the left parietal region during visually presented

word-pair learning and recall. There was greater frontal involvement during learning as opposed to recall. A study by Risberg in 1973 showed the activation pattern changed drastically when parallel forms of Raven's matrices were given on subsequent days. There was high frontal lobe increase on day 1 and habituation of the frontal lobes on day 2 with stability of the posterior areas. He noted that this seemed to indicate an involvement of the frontal regions in processes related to habituation to new situations and tasks, and to general organization and control of mental processes as postulated earlier by Halstead and others.

Halsey, Blauenstein, Wilson, and Wills (1980) showed that verbal activation in normals engaged in banal verbal activity showed symmetrical patterns of flow. Unilateral lesions depress general intellectual function and sometimes motor activity in a much more diffuse and bilateral fashion than could occur if each region of the brain operated in isolation without important connections to other regions within the same hemisphere and to homologous regions in the opposite hemisphere via the corpus collosum.

Meyer, Sakai, Yamaguchi, Yamamoto, and Shaw (1980) found a relative increase in the frontal and precentral areas of both hemispheres of normal subjects related to the intention of performing a task and in certain individuals to apprehension related to such anticipation. Two types of cerebral activation processes appeared to operate characterized by distinctive patterns of rCBF change. There are localized increases in rCBF in appropriate regions of cortical representation related to specific aspects of the mental task such as in Broca's and Wernicke's areas during speech and counting, and there is generalized

activation of brain function necessary for conceptualization and performance of the task. When the test was applied to patients with cerebral disorders of speech and mentation there were three types of abnormal responses: 1) A diffuse failure of rCBF response seen in all patients with dementia; 2) A regional failure of response as was seen, for example, in patients with good recovery from motor aphasia with failure for regional blood flow to Broca's area during speech activation, and 3) excessive regional or generalized response seen in patients with focal or generalized epileptic seizures.

Location and Clinical Signs of the Aphasias

While there are many classification schemes, some of which break down the aphasias into as many as 11 separate categories, only the three most widely used will be described here.

Broca's aphasia, which is also known as expressive, non-fluent, or motor aphasia, while currently well established, nevertheless has produced many of the controversies because the patient first described by Broca turned out not to have Broca's aphasia, and the degree and involvement of Broca's area and the surrounding frontal operculum produce very different degrees of aphasia (Mohr, et al., 1978). Broca's aphasia, when seen clinically, involves relatively intact comprehension, but difficulty in expressive speech, repetition, naming, reading or writing. In addition, the patient almost always presents with a right sided motor defect which is clearly neurological in nature (Damasio, 1981).

Wernicke's aphasia, which is also known as sensory, receptive, or fluent aphasia, is associated with lesions in the left superior temporal gyrus, and may extend into the second temporal gyrus and into the nearby parietal region. Clinical manifestations include fluent speech, poor comprehension, verbal paraphasia, difficulty with repetition, naming, reading, and writing. In contrast to patients with Broca's aphasia who speak haltingly, with few words in a telescopic manner, these patients ramble on with many words saying nothing, and sound confused, and are much more likely to be diagnosed as mental patients. They rarely have the well defined right sided motor deficit seen in Broca's aphasia.

Conduction aphasia is associated with left perisylvian lesions involving the primary auditory cortex, the insula, its subcortical white matter, and the supramarginal gyrus. Clinically, these patients present with fluent speech, good comprehension, literal paraphasia, and difficulty with repetition, naming, reading and writing (Damasio).

Definitions

Acalculia. Acalculia is the inability to calculate or perform arithmetical calculations because of brain damage.

Agnosia. Agnosia refers to not knowing. It is the inability to identify familiar objects, one's fingers, sounds, or visual symbols caused by brain damage.

Agraphia. Agraphia is the inability to produce written language caused by brain damage.

Alexia. Alexia is the inability to comprehend the written language as a result of brain damage.

Aphasia. Aphasia is the loss or impairment of language as a result of brain damage.

Apraxia. Apraxia is the inability to execute learned or voluntary movements caused by brain damage.

Classification of Aphasia

Although there are numerous classification schemes described throughout the literature which use various naming systems, multiple syndromes, simple dichotomies, or vague, all-inclusive terms such as central language process (Brown, 1968), for purposes of this paper the most useful classification scheme will be that described by Heimburger and Reitan (1962).

The two major categories include the agnosias and the apraxias. The agnosias include defects in the sensory avenue of language function (reception and perception), while the apraxias involve motor defects. Agnosia is present when the person cannot comprehend the functional and symbolic significance of a stimulus even though the necessary sensory organs are intact and functioning. Apraxia is present when the person cannot produce sounds, write, calculate or carry out specific acts. These will be dealt with later on in more detail.

Methods of assessment

Brain behavior relationships have been studied in many ways: Black box approaches, brain ablation studies, brain stimulation paradigms, neurochemical manipulations, electrophysiological studies and introspection. Most of those have relied on animal experimentation for obvious reasons. Human experimentation has relied on the study

of normals with electroencephalographic correlates of hemispheric asymmetries, case reports of individuals, observation, electrode stimulation studies, blood flow studies, computerized tomography, carotid injection, and surgical ablation.

Clinical testing for aphasia includes the observation of spontaneous verbalizations of the patient with attention being paid to the degree of fluency or non-fluency. Non-fluency is evidenced by a decrease in output of language, increased effort required to produce a word, poorly articulated words which are difficult to understand, decreased phrase length, non-melodic speech and deletion of syntactic structures such as prepositions, articles and adverbs. Fluency would be the reverse of this, with increased output, normal articulation with little effort, normal length and phrasing, but with circumlocution and empty speech, and paraphasia.

In addition to spontaneous speech, attention has been paid to repetition and comprehension of spoken language, word-finding problems, reading and writing.

The clinical approach can be useful in the hands of trained and experienced examiners who can obtain valuable information from a given individual in a short period of time. However, the results of clinical evaluation are subject to theoretical biases and may be misleading or detrimental to the patient if inaccurately performed. The need for more exact standardized testing is obvious.

Formal tests of aphasia

Following the example of Head in 1926, Wiesenburg and McBride in 1935, produced a formal standardized battery of tests which led to the development of even more test batteries to study aphasia in greater detail. Today the tests which are most widely used to study aphasia in detail to determine diagnosis and prescriptive programs for remediation are the Minnesota Test for the Differential Diagnosis of Aphasia (Schuell, 1957), the Boston Diagnostic Examination (Goodglass & Kaplan, 1972), and the Sklar Aphasia Scale (1966). These are extensive batteries which purport to classify the various forms of aphasia, are time-consuming, and require clinical interpretation. Each test has proved useful in some phase of evaluation and no single test has been shown to be superior tc all others. The choice of tests varies among clinics, depending on the focus of diagnosis and rehabilitation.

One of the most widely used short screening tests for aphasia in use today is the modified version of the Halstead-Wepman Aphasia Screening Test, known today as the Reitan-Indiana Aphasia Screening Test, and it is that test, along with the Reitan-Kløve Sensory Perceptual Examination, that is the focus of this paper. In order to do it justice, an introduction to the Halstead-Reitan Neuropsychological Test Battery, and the role of the aphasia examination in the greater context of the total battery will be made.

Halstead Reitan neuropsychological test battery for adults

In 1935 at the University of Chicago, Ward C. Halstead developed the first full-time experimental laboratory to study the psychological

effects of brain impairment. In addition to careful naturalistic observation, which included the study of persons with brain damage in all aspects of personal and interpersonal activities, he developed a battery of neuropsychological tests which permitted valid inferences regarding many aspects of impaired brain functions. With his recognition of the complexity of the brain as the organ of adaptive behavior, provision was made for a broad enough knowledge base as a result of the test battery, to permit the emergence of principles on which diagnostic inferences could be made (Reitan, undated).

The test batteries were designed to meet three criteria:

1. The range of behavioral functions to be evaluated were to be broad enough to describe accurately the effects of cerebral lesions. Thus, higher level functions such as logical analysis, reasoning ability, abstraction, concept-formation, and language abilities, as well as lower level functions such as sensory-perceptual and motor tasks, and psychomotor problem-solving tasks were to be included.

2. Only tests which had previously been validated with respect to the effects of cerebral lesions were to be included.

3. The following set of principles of inference could be integrated for use in interpretation of results obtained from individual subjects.

   a) Level of performance. This refers to the most often used method of inference in psychological testing, which compares individual performance to normative data, such as the Weschler Intelligence Test.

   b) Differential score approach. This refers to the evaluation of patterns of response or relationships among the various

tests for individual subjects. This allows for comparison of intraindividual ability patterns as a result of impaired brain function.

c) Right-left comparisons. Because of the contralaterality of brain function due to the anatomy of the brain itself, direct comparisons can be made of the functional efficiency of the two sides of the body for an individual subject. If he shows sensory or motor deficiencies on the right side of his body, the left cerebral hemisphere may be implicated as impaired, while the reverse is true if the left side of his body were to show deficiencies.

d) Pathognomonic sign approach. The sign approach has long been used in medicine and psychology and refers to identification of pathology or abnormality. It says essentially that a particular kind of response is in and of itself indicative of pathology.

A review of these four methods of inference and the ability to use them in a complementary and integrated way reflects a fusion of the experimental and clinical approaches to the study of brain-behavior relationships, and is the foundation of the neuropsychological test batteries.

Although the original tests were developed by Halstead, and showed promise of assessment of brain impairment, a number of additional tests was developed by Ralph M. Reitan, which included adding the Weschler-Bellevue Scale for a more complete assessment of general intelligence, the finger-tapping tests for both hands, grip strength measures

for both hands, and in conjunction with Kløve, the sensory perceptual tests which also allowed for direct comparisons between both sides of the body. Some of the original Halstead tests which contributed to a summary score known as the Impairment Index, were subsequently dropped from the battery because they did not meet the stringent requirements for clear discrimination between brain-damaged individuals and normals. Today seven of the original 10 tests comprise the Impairment Index. In addition, a number of brain-sensitive tests have been added to the battery for assessment of deficits.

The tests which currently comprise the Halstead-Reitan neuropsychological Test Battery for Adults include the following measures.

1. The Halstead Category Test is a complex concept-formation test which requires the ability to note similarities and differences, to formulate hypotheses, and with a reinforcement feedback system of bell and buzzer, to be flexible enough in thinking to change the hypothesis if necessary in order to solve the problem. While normal subjects do not have much trouble with the test, brain-injured persons show an inability to shift, categorize, and group things conceptually.

2. The Tactual Performance Test is a modification of the Seguin-Goddard form board and is a complex kinesthetic-motor task which contributes three separate scores to the summary index. These include total time, a memory component and a localization score. In addition, this test permits direct right-left comparisons of the two sides of the body.

3. The Seashore Rhythm Test requires ability to discriminate rhythmic pairs, alertness, and concentration to detail.

4. The Speech-sounds Perception Test requires the ability to discriminate between consonant sounds, to perceive them on a written form, and to underline the sound that is heard.

5. The Finger Oscillation Test is a measure of finger-tapping speed and permits direct comparisons between the left and right sides of the body, and also contributes to the summary index.

In addition to the tests described above which are the Halstead Tests, the following are part of the present battery.

6. The Trail Making Test, forms A and B, require recognition of the symbolic significance of numbers and letters, ability to scan a page quickly, to anticipate the next symbol in sequence, and to alternate between numbers and letters in a time frame.

7. The Sensory Perceptual Examination includes measures of bilateral comparisons of visual, auditory, and tactile modalities.

8. The modification of the Halstead-Wepman Aphasia Screening Test provides the measures of the agnosias and apraxias to be further defined.

Two other major tests which are included in the battery are the Weschler Adult Intelligence Scale and the Minnesota Multiphasic Personality Inventory, which are well-known and need not be discussed further.

Several of the tests in the battery are sensitive to the general effects of brain damage, while others are sensitive to differential effects of the two cerebral hemispheres. As indicated earlier, it has been known for many years that the left cerebral hemisphere plays a major role in speech and language communications and the right cerebral hemisphere is important for visuospatial configurations. Effects of lateralized lesions show up on the Verbal and Performance subtests on

the Weschler Tests, the Verbal being associated with the left cerebral hemisphere and the Performance with the right (Reitan, 1955).

In the early 1960's a number of studies were done regarding the aphasia examination and all showed clearly lateralizing functions in language and non-language disorders (Doerhing & Reitan 1961; Heimburger & Reitan, 1961; and Wheeler & Reitan, 1962). These studies showed that when left cerebral lesions were found, language disorders were frequently seen, while when right cerebral lesions were found, visuospatial and manipulation difficulties were often present. One additional finding which validated earlier studies was the fact that acute destructive lesions in either hemisphere had a more devastating effect in relation to specific losses than did chronic stabilized damage.

Kløve (1959), Reed (1967) and Reitan (1959, 1964), have reported validation studies of the comparison of sensory-perceptual and motor functions of the two sides of the body, which are differentially organized contralaterally.

Aphasia Screening Test and the Sensory Perceptual Examination

The Sensory Perceptual Examination offers the following measures:

1. Sensory Imperception
   a) Tactile: Unilateral and bilateral double simultaneous stimulation of the left, right, and both hands; unilateral and bilateral double simultaneous stimulation of the right hand and left face; and unilateral and bilateral double simultaneous stimulation of the left hand and right face.
   b) Auditory: Unilateral and bilateral double simultaneous stimulation of the left, right, and both ears.

c) Visual: Unilateral and bilateral double simultaneous stimulation of the right, left, and both eyes on three planes.

2. Finger agnosia

The person is asked to identify which finger is being touched, relying on tactile cues only.

3. Fingertip number writing

The person is asked to identify numbers written on the fingertips, with his eyes shut.

The aphasia examination consists of 32 items which measure various language and related abilities. The items on the aphasia examination give measures of ability to copy geometric designs, to name familiar shapes and objects, to spell, calculate, read, repeat spoken words, to perform previously learned tasks, to comprehend the meaning of words, to write, to identify body parts, and to differentiate the right from the left sides of the body.

The test does not provide a continuous distribution of scores on individual items, but is organized in such a way that performance is examined in the terms of the particular sensory modality through which the stimuli are perceived. In addition it allows for a determination of the expressive or receptive nature of the difficulty. It is a sign test in which normals are expected to make no errors, and any error may be significant for brain damage. Parametric analysis is thus not appropriate, and one needs to consider non-parametric methods of analysis.

The classification of the agnosias and apraxias as they relate specifically to the aphasia examination and first described by Heimburger and Reitan are:

Agnosia: Visual form agnosia - inability to recognize geometric shapes and other objects.

Visual number agnosia - inability to recognize numbers through the visual modality.

Visual letter agnosia - inability to recognize individual letters of the alphabet.

Visual word agnosia - inability to recognize symbolic significance of words through vision (also known as alexia).

Auditory verbal agnosia - inability to comprehend the spoken word.

Auditory number agnosia - inability to recognize numbers through the auditory modality.

Tactile form agnosia - inability to perceive the form of an object held in the hand (also known as astereognosia).

Body agnosia - inability to identify body parts.

Finger agnosia - inability to identify which finger is being touched.

Right-left disorientation - confusion of left and right sides of the body.

Agraphagnosia - inability to know numbers written on the fingertips.

Apraxias: Naming apraxia - inability to name (anomia, amnesic aphasia).

Spelling apraxia - inability to spell.

Writing apraxia - inability to reproduce the written word (agraphia).

Word order apraxia - confusion or substitution of one word for another; inability to speak syntactically or grammatically (paraphasia).

Calculation apraxia - inability to calculate (acalculia).

Construction apraxia - inability to reproduce geometric designs.

Ideo-kinetic apraxia - inability to carry out a simple act.

Enunciatory apraxia - inability to reproduce a spoken word (dysarthria).

Manipulatory apraxia - inability to manipulate objects.

In 1961, Wheeler and Reitan predicted the presence and lateralization of brain damage for patients with left, right, and bilateral diffuse lesions and controls based on their responses to the aphasia screening test. They were able to classify patients on the basis of positive signs in 26 categories according to three hypotheses. When the rule was 1) one or more signs indicates brain damage, the conditional probability of correct classification was 78%. When the rule was 2) predict left damage when the number of signs in a set of left indicator variables is greater than or equal to one and there are no signs in a set of right indicators, correct classification was 80%.

When the rule was 3) predict right damage when the number of signs in the right set of indicators is greater than or equal to one, and the number of signs in the left set is less than or equal to one, the correct classification was 85%. Finally, when the rule was 4) if the number of signs in the right indicator set is greater than or equal to one and the number of signs in the left indicator set is equal to or greater than two, predict diffuse damage, the conditional probability was 84%.

Latent Structure Analysis

The present paper presents an analysis of the aforementioned aphasia and sensory perceptual examinations using a highly sophisticated nonparametric statistic introduced originally by Lazarsfeld in 1950, expanded by Goodman in 1974, and applied extensively in educational research programs recently by Bergan. Bergan has emphasized the importance of latent structure analysis as an effective tool to describe specific response patterns and processes in the sequence of behaviors which may be explained by latent, or unobservable, variables. While direct application has been made to explain the structure of knowledge in terms of individual problem-solving tasks in learning, there has to date been no attempt to apply this technique to the study of brain damage until now.

The application of latent structure analysis to categorical data involves the development of latent class models (Goodman, 1978). The use of latent class models in educational research has been well-detailed by John R. Bergan in his chapter in Review of Research in Education (in press). It is only recently with the advent of computer programming and increased sophistication of parameter estimation that

latent class models are being realized for their wide-ranging application and usefulness in describing the association between those manifest variables which can be observed, and those latent variables which cannot be observed.

A latent class model as it relates to the Reitan-Indiana Aphasia Screening Test and the Reitan-Kløve Sensory Perceptual Examination can be conceptualized as follows: The aphasia and sensory perceptual examination consist of a number of items (manifest variables) which represent measures of brain function (a latent variable). In the past one might have considered performance on the test items to represent measures which could be explained in terms of only two latent classes, normal or brain damaged. In that case a two-class model would be proposed and tested by assessing the degree of correspondence between the observed responses to the items and responses which would be expected under the assumption that the two-class model were true. From what we know about brain function from existing research, however, it is clear that a two-class model would not fit the data very well. For example, if one were to select two items from the test, both representing naming items, and one were to predict that latent class normals would pass the items and latent class brain-damaged would fail the items, one can readily see that the observed responses would often fail to meet the prediction in all those cases where brain damage principally involved the right hemisphere.

The research studies referred to earlier suggests that a more accurate representation of the data could be made by hypothesizing a a four-class model instead. This model has one latent variable, brain function, consisting of four latent classes. Those four classes are:

1) latent class Normal, 2) latent class Left cerebral damage, 3) latent class Right cerebral damage, and 4) latent class Bilateral diffuse damage. Within each latent class there are two possible responses to each category of items on the tests; one can pass the item, or one can fail the item. Reitan, in earlier publications made judgments regarding the qualitative aspects of responses to each of the items, and determined whether the response categories represented an aphasic disorder, either expressive or receptive in nature. The present study examines those same data using the latent class models to be described.

A latent class model is a mathematical representation of the relationship between latent classes and observations expected under the assumption that the model is true. A given model is tested by assessing the extent to which response patterns expected correspond to those which are actually observed. In setting model specifications which express the model in mathematical terms, the basic assumption is that the association between or among a set of items can be explained by one or more latent variables, each composed of a set of latent classes.

Goodman (1974a) proposed a general unrestricted latent class model used to analyze contingency tables of dichotomous or polytomous variables in which he expressed cell probabilities in terms of a latent variable composed of a number of latent classes. For instance, assume there are four manifest variables, which represent the aphasia items, A, B, C, & D, which can be explained by latent variable X made up of T latent classes. Goodman's general unrestricted model expresses the cell probabilities, where cell ijkl is expressed as i = 1 to I, j = 1 to J, k = 1 to K, and l = 1 to L, as follows:

$$\pi_{ijkl} = \sum_{t=1}^{t} \pi^{ABCDX}_{ijklt},$$

where π_{ijkl} is the probability for cell ijkl, and π^{ABCDX}_{ijklt} is the joint probability of latent class t and pattern ijkl. The joint probability is given by:

$$\pi^{ABCDX}_{ijklt} = \pi^{X}_{t} \pi^{\bar{A}X}_{it} \pi^{\bar{B}X}_{jt} \pi^{\bar{C}X}_{kt} \pi^{\bar{D}X}_{lt}$$

where π^{X}_{t} is the probability of the latent class t, $\pi^{\bar{A}X}_{it}$ it is the conditional probability that item A will be responded to at level i given latent class t, and $\pi^{\bar{B}X}_{jt}$ $\pi^{\bar{C}X}_{kt}$ $\pi^{\bar{D}X}_{lt}$ are similarly defined. This general model, when T = 1, is directly equivalent to the test of independence of the Chi-square statistic.

In addition to the unrestricted model where T = 1, one can increase the number of latent classes and also impose restrictions on the model in two ways. One can place limitations of the conditional response probabilities associated with latent classes or one can impose restrictions on the latent class probabilities.

An important aspect of latent class models which involve unobservable variables has been the determination of estimating model parameters which are the true population values of arbitrary constants included in the mathematical expressions of the model, e.g., π^{X}_{t} and $\pi^{\bar{A}X}_{it}$ (Bergan). Cliffort Clogg (Note 1) constructed a computer program to carry out an iterative process initially developed by Goodman. With appropriate start values the procedure converges to a maximum liklihood solution. The procedure is appropriate for generating parameter estimates for unrestricted latent class models, and with modification, also for restricted latent class models.

In addition to the assumption that the association among a set of items can be explained by the latent variable(s), a further assumption is made that within any given latent class, the probability of a particular response on any one item is independent of any given response on any other item in the set being considered for investigation. Lazarsfeld (Lazarsfeld & Henry, 1968) called this the axiom of local independence, local indicating the fact that independence prevails only within latent classes. By imposing restrictions on models built under this assumption, a wide range of model variations can be developed. In the situation where T is greater than 2, the assumption of local independence holds, and the multiplicative relation among the variables exists within the latent classes. The hypothesis of independence is tested by the χ^2 statistic. If the hypothesis is supported by the data, the association among the manifest variables is said to be accounted for by the latent variable. If the model with four latent classes provides an acceptable fit for the data, the latent variable is assumed to explain the association.

Restricted latent class models. Variations may be produced, not only by adding to the latent classes, but also by imposing restrictions on the general model. For instance, if one were to look at a two-class model of Normal and Brain-damaged, one might want to assume that within the latent class, Normal, or within the latent class, Brain-damaged, the probability of passing or failing an item should be equal across items. This could be expressed as follows:

$$\pi\, \bar{A}X_{11} = \pi\, \bar{B}X_{11} = \pi\, \bar{C}X_{11} = \pi\, \bar{D}X_{11}$$

$$\pi\, \bar{A}X_{22} = \pi\, \bar{B}X_{22} = \pi\, \bar{C}X_{22} = \pi\, DX_{22}$$

where the 1's represent correct responses and the t = 1 represent latent variable X is the Normal class; the 2's represent incorrect responses and t = 2 is the Brain-damaged class.

One can also restrict one or more conditional response probabilities to a particular value. For instance, one might assume that normals should always pass items, which is a reasonable assumption in the aphasia test, since it is a sign test, and errors constitute pathognomonic signs. In that case, one could set the restrictions as follows:

$$\pi\, \bar{A}X_{11} = \pi\, \bar{B}X_{11} = \pi\, \bar{C}X_{11} = \pi\, \bar{D}X_{11} = 1,$$

where a response at level 1 indicates a passing response and latent class t = 1 is the Normal class. If one also wanted to assume that Brain-Damaged should always fail items, that would be expressed as:

$$\pi\, \bar{A}X_{22} = \pi\, \bar{B}X_{22} = \pi\, \bar{C}X_{22} = \pi\, \bar{D}X_{22} = 1,$$

where a response at level 2 indicates a failing response and latent class t = 2 represents the Brain-damaged class.

Once the model has been specified, determination needs to be made whether or not the parameters can be uniquely identified, and the parameters need to be estimated by the interative procedure mentioned earlier. Clogg's computer program printouts indicate whether or not the model being considered is identified, and specifies the proper degrees of freedom associated with the model.

Imposition of restrictions on latent class models changes the degrees of freedom associated with the particular model. When all the model parameters are identified, the imposition of restrictions results in added degrees of freedom. In the simplest case, the two-class model

described earlier, there were six degrees of freedom in the unrestricted model. When conditional probabilities for the responses for the first class in the model were restricted to be equal, only one conditional probability needs to be estimated within the first latent class; the others are determined by the restriction. Thus, rather than six degrees of freedom, the restricted model has nine degrees of freedom.

To assess the degree of fit of the observed to the expected cell frequencies, the Pearson Chi-square, X^2, or the liklihood-ratio L^2 is used. The Clogg computer program uses both. The advantage to this is that when there is a sufficiently large sample, the two are identical, providing that the model under investigation is true. If they are not identical, sample size should be increased.

A model is regarded as an acceptable fit for the data when $p > .05$ (Goodman). In addition the criteria of goodness of fit and parsimony must be met. The parsimony criterion dictates that the smallest number of estimated parameters needed to afford the most adequate available fit is indicated. From this it should be clear that a saturated model, or one with no degrees of freedom although it fits the data exactly, would not be accepted since it would not meet the criterion of parsimony. Use of the L^2 statistic, since it is log-linear and thus additive in nature, permits hierarchical comparisons of models. Two models are heirarchical when both are identifiable and one contains all the estimated parameters of the other, plus one more, and thus has different degrees of freedom. The liklihood ratio is used because it permits exact partitioning of the data, which makes it possible to subtract one Chi-square from another to get a resultant Chi-square. The effect of

this is to allow one to select a preferred model from a group of models, all of which might be considered to accurately represent the data.

While a two-class model was presented as the simplest case possible to review the use of latent structure analysis, it must be remembered that the present study proposes a four-class model as a more adequate representation of current research findings. With the information presented it should be clearer how the present model should be an improvement and offer a model of best fit.

For example, suppose one looks at four items or categories on the Aphasia Test, two of which represent left cerebral hemisphere function, and two of which represent right cerebral hemisphere function.

A and B = Naming items

C and D = Constructional Dyspraxia

where A and B represent left hemisphere functions and C and D represent right hemisphere functions.

Each of those manifest variables has two possible response patterns; 1 = correct response, and 2 = incorrect response. The latent variable, brain function, has four latent classes:

X_1 - Normal

X_2 = Left cerebral damage

X_3 = Right cerebral damage

X_4 = Bilateral diffuse damage

The model is represented mathematically as follows:

$$\bar{A}X_{11} = \bar{B}X_{11} = \bar{C}X_{11} = \bar{D}X_{11}$$

$$\bar{A}X_{22} = \bar{B}X_{22} = \bar{C}X_{12} = \bar{D}X_{12}$$

$$\bar{A}X_{13} = \bar{B}X_{13} = \bar{C}X_{23} = \bar{D}X_{23}$$

$$\bar{A}X_{24} = \bar{E}X_{24} = \bar{C}X_{24} = \bar{D}X_{24}$$

which states that the items are equivalent, and that Normals will pass those items representing both left and right cerebral hemisphere functions; that those with left cerebral damage will fail those items representing left hemisphere functions and pass those items representing right cerebral hemisphere functions; that those with right cerebral damage will fail those items representing right cerebral hemisphere functions and pass those items representing left cerebral hemisphere functions; and, that those with bilateral diffuse damage will fail those items representing both left and right cerebral hemisphere functions.

Model of Latent Agreement. Latent agreement measures can indicate the extent to which the classification of persons into one or another of the four classes by means of clinical classification as a result of neurological examination corresponds to the classification of those same persons into one or another of the classes according to their responses on the Aphasia Test and offers an important validity check. Bergan (in Press), described the use of restricted latent class models involving polytomies, using quasi-independence models to express reliability.

In order to do this, one constructs a 4 X 4 contingency table cross-classifying the classifications. The four diagonal cells reflect agreement between the clinical and test response classifications, and

the remaining cells reflect disagreement. Agreement is represented mathematically as follows:

$$\pi_{ij} = \sum_{t=1}^{t} \pi \, ABX_{ijt} ,$$

where the possible levels of i, j, and t are each 1, 2, 3 and 4. The first four latent classes represent agreement, and are restricted as follows:

$$\pi \, \bar{A}X_{11} = \pi \, \bar{B}X_{11} = 1,$$
$$\pi \, \bar{A}X_{22} = \pi \, \bar{B}X_{22} = 1,$$
$$\pi \, \bar{A}X_{33} = \pi \, \bar{B}X_{33} = 1,$$
$$\pi \, AX_{44} = \pi \, BX_{44} = 1.$$

The fifth latent class, t, represents disagreement and is unrestricted. It is this class which assesses the unreliability of classification into the various groups.

By putting structural zeroes in the diagonal cells which one does by making the expected cell frequencies equal to 1, one can use the L^2 statistic to test the hypothesis of quasi-independence with the diagonal cells removed from consideration. Goodman has shown that the probabilities for categories pertaining to cells with structural zeroes and the probability for the set of cells not containing structural zeroes sum to 1. Because of that he showed that the estimated proportion of responses for the sum of all of the cells containing structural zeroes is 1 minus the probability for the category representing cells without structural zeroes. This proportion serves as an index of agreement, which expresses the magnitude of agreement in a directly interpretable and easily understood way (Bergan, in Press).

In summary, the application of latent structure analysis permits the specification and testing of all kinds of hypotheses which then suggest further models closely related to theories, and offers procedures for possible theoretical interpretations shaped by the theoretical models one advances.

Latent class models in this study are used to assess such fundamental questions as the role of the integrity of the brain and its relationship to associated language and related disorders.

CHAPTER 3

METHOD

Subjects

The subjects for this study are those of the original Wheeler and Reitan study of 1962. A total of 262 subjects were administered the aphasia screening test, which yielded 26 scores per subject. After administration and scoring by highly trained administrators, the test protocols were examined by Reitan who had no information about the subjects other than their names. He classified the test responses into 26 categories, each of which represented a specific type of behavioral impairment. Thus, for any subject, his score could vary from 0 to 26 positive signs for brain damage. Of these 26 categories only 19 were used in the analysis due to frequency of occurrence.

Of these subjects given the test, criterion groups consisted of 47 subjects with left cerebral hemisphere damage, 57 with right hemisphere damage, 54 with bilateral diffuse damage, and 104 who showed no evidence of brain damage. The diagnoses for brain damage were based upon thorough neurological examinations, detailed medical history, electroencephalography, and in some cases, cerebral angiography, pneumography, results of surgery, or autopsy findings.

Of the 47 subjects with left cerebral damage, 12 suffered from cerbrovascular accident, 15 had intrinsic tumor, three had extrinsic tumor, five had open head injury, four had closed head injury, three

had cerebral abcess, two had infantile hemiplegia, two had degenerative brain disease, and one had arteriovenous malformation. Of those with right cerebral damage, 12 had cerebrovascular accident, 15 had intrinsic tumor, six had extrinsic tumor, five had open head injury, eight had closed head injury, four had cerebral abcess, four had arteriovenous malformation, two had degenerative brain disease, and one had infantile hemiplegia. Of those with bilateral diffuse damage, 15 had diffuse cerebrovascular disease, 10 had multiple sclerosis, nine had bilateral intrinsic tumor, nine had degenerative brain disease, five had closed head injury, two each had epilepsy and encephalitis, and one each had dementia paralytica and diffuse subdural hygroma. Forty subjects without brain damage were normal controls, 25 were diagnosed neurotic, 24 paraplegia, 12 non-CNS surgical conditions, and three non-CNS medical conditions.

Age and years of education were considered in the investigation. The diffuse group was significantly older than all other groups, and the group without brain damage was significantly younger than all other groups. The groups with lateralized lesion did not differ in chronological age. The group without brain damage had significantly more education but the brain damaged groups showed only chance differences among themselves. The test requires only sixth grade skills.

Tests Given

The Reitan-Indiana Aphasia Screening Test and the Reitan-Kløve Sensory Perceptual Examination were given to the 262 subjects described

earlier. These tests yielded 26 separate categories of brain impairment, 19 of which were used for the final analysis.

The left indicators are 1) dyscalculia, 2) central dysarthria, 3) dysnomia, 4) dysgraphia, 5) spelling dyspraxia, 6) dyslexia, 7) right-left disorientation, 8) right finger dysgnosia, 9) right dystereognosis, 10) visual letter dysgnosia, 11) right tactile imperception, 12) auditory verbal dysgnosia, and 13) visual number dysgnosia. The right indicators are 14) construction dyspraxia, 15) left dystereognosis, 16) left finger dysgnosia, 17) left tactile imperception, 18) left visual imperception, and 19) left auditory imperception.

Statistical Analysis

Latent Structure Analysis -- Latent Class Models

1. The General Unrestricted Model

$$\pi_{ijkl} = \sum_{t=1}^{T} \pi_{ABCDX_{ijklt}}$$

where $ijkl$ = probability for cell ijkl, and $ABCDX_{ijklt}$ = joint probability of latent class t and pattern ijkl.

The joint probability is given by:

$$\pi_{ABCDX_{ijklt}} = \pi_{X_t} \pi_{\bar{A}X_{it}} \pi_{\bar{B}X_{jt}} \pi_{\bar{C}X_{kt}} \pi_{\bar{D}X_{lt}}$$

where π_{X_t} = probability of latent class t, and $\pi_{\bar{A}X_t}$ = conditional probability that item A will be responded to at Level i, given latent class t, and $\bar{B}X_{jt}$, $\bar{C}X_{kt}$, and $\bar{D}X_{lt}$ are similarly defined.

2. Restricted Latent Class Model -- Equivalence

The present study proposed a four-class model to test the hypothesis that four classes of brain function could best explain the data. The four class model is defined as follows:

$\bar{A}X_{11} = \bar{B}X_{11} = \bar{C}X_{11} = \bar{D}X_{11}$ = Latent Class Normal

$\bar{A}X_{22} = \bar{B}X_{22} = \bar{C}X_{12} = \bar{D}X_{12}$ = Latent class Left lesion

$\bar{A}X_{13} = \bar{B}X_{13} = \bar{C}X_{23} = \bar{D}X_{23}$ = Latent Class Right lesion

$\bar{A}X_{24} = \bar{B}X_{24} = \bar{C}X_{24} = \bar{D}X_{24}$ = Latent Class Bilateral diffuse

It was tested to determine whether the expected categories of brain function could, in fact, be used to explain the data.

Additional models were tested to determine whether a more parsimonious model would afford a model of better fit. Two two-class models are defined as follows:

$\bar{A}X_{11} = \bar{B}X_{11} = \bar{C}X_{11} = \bar{D}X_{11}$ = Latent Class Normal

$\bar{A}X_{22} = \bar{B}X_{22} = \bar{C}X_{12} = \bar{D}X_{12}$ = Latent Class Left lesion

This model says there are two latent classes, Normals and those with left lesions, which are sufficient to explain the data.

$\bar{A}X_{11} = \bar{B}X_{11} = \bar{C}X_{11} = \bar{D}X_{11}$ = Latent Class Normal

$\bar{A}X_{13} = \bar{B}X_{13} = \bar{C}X_{23} = \bar{D}X_{23}$ = Latent Class Right lesion

This model says there are two latent classes, Normals and those with right lesions, which are sufficient to explain the data.

A three class model which says there are three latent classes; Normals, those with left lesions, and those with right lesions which can be used to explain the data. This model is defined as follows:

$\bar{A}X_{11} = \bar{B}X_{11} = \bar{C}X_{11} = \bar{D}X_{11}$ = Latent Class Normal

$\bar{A}X_{22} = \bar{B}X_{22} = \bar{C}X_{12} = \bar{D}X_{12}$ = Latent Class Left lesion

$\bar{A}X_{13} = \bar{B}X_{13} = \bar{C}X_{23} = \bar{D}X_{23}$ = Latent Class Right lesion

In addition to these latent class models, a latent class model of agreement was tested to determine the degree of agreement between the classification of subjects by test classification and by neurological diagnosis. The model of agreement is defined as follows, with the agreement categories set equal to 1 and the disagreement categories left free to vary. This model is defined as follows:

$\bar{A}X_{11} = \bar{B}X_{11} = 1$

$\bar{A}X_{22} = \bar{B}X_{22} = 1$

$\bar{A}X_{33} = \bar{B}X_{33} = 1$

$\bar{A}X_{44} = \bar{B}X_{44} = 1$

$\bar{A}X_{15} = \bar{B}X_{25}$

The restrictions for the first four latent classes assert perfect agreement, and the fifth latent class represents disagreement and indicates that the types of disagreement are independent of one another.

CHAPTER 4

RESULTS

Latent-class models were used in the analysis of the data in an attempt to explain the associations in a contingency table in terms of latent classes described earlier. Prior to testing the model of four latent classes which represented different categories of brain damage or non-brain damage, a review of the files of the 262 subjects identified by Wheeler and Reitan as having specific aphasic disorders, was made in order to examine some of the individual test items which contributed to each of the categories. No attempt was made to include each of the items in each of the categories. Instead, only a few categories were selected. The purpose was to verify that there are four categories of brain damage, and that they can be discriminated by means of a behavioral test. No personal judgments were made in determining whether or not an individual patient suffered a specific disorder. For example, if the patient had been classified as having Dysnomia in the earlier study, an inspection was made to determine which of the naming items were missed by the patient which contributed to that classification. This resulted in a test of thirty manifest variables which are described in Table 1.

A model of latent agreement was used to assess the reliability of items assumed to be homogeneous. Pairs of items which represented reading, spelling, computation, etc., were examined using a model described by Bergan which includes three latent classes. The three

Table 1

Thirty manifest variables selected from the Reitan-Indiana Screening Test.[a]

Aphasia Category	Item Number	Item Task
Dysnomia	1	Name Square
	2	Name Cross
	3	Name Triangle
	4	Name Baby
	5	Name Fork
Dysgraphia	6	Write Clock, Square
	7	Write He shouted the warning.
Right-Left Confusion	8	Place left hand to right ear.
Spelling Dyspraxia	9	Spell Square
	10	Spell Cross
	11	Spell Triangle
Visual Letter and	12	Read 7 SIX 2
Number Dyspraxia	13	Read MGW
Dyslexia	14	Read See the black dog.
	15	Read He is a friendly animal, a famous winner of dog shows.
Dyscalculia	16	Compute 85 - 27 =
	17	Compute 17 x 3 =
Central Dysarthria	18	Repeat Triangle
	19	Repeat Massachusetts
	20	Repeat Methodist Episcopal

Table 1 (Continued)

Aphasia Category	Item Number	Item Task
Right TVA Imperception	21	Double Simultaneous Stimulation in Tactile, Visual, and Auditory Senses.
Right Finger Dysgnosia	22	Identify finger being touched while eyes are closed.
Right Dysstereognosis	23	Identify shapes by feel only.
Construction Dyspraxia	24	Copy Square
	25	Copy Cross
	26	Copy Triangle
	27	Draw Key
Left TVA Imperception	28	Double Simultaneous Stimulation in Tactile, Visual, and Auditory Senses
Left Finger Dysgnosia	29	Identify finger being touched while eyes are closed.
Left Dystereognosia	30	Identify Shapes by feel only.

a. Items 1-23 represent Left Cerebral Function and Items 24-30 represent Right Cerebral Function.

latent classes reflect possible types of agreement or disagreement between the responses to each of the two items. The first latent class mastery, and the second latent class, non-mastery, reflect agreement classes, and the third latent class, which reflects unreliable responding, represents the disagreement class. Assumptions made under this model are expressed mathematically and reflect restrictions on conditional response probabilities as follows:

$$\bar{A}X_{11} = \bar{B}X_{11} = 1$$

$$\bar{A}X_{22} = \bar{B}X_{22} = 1$$

$$\bar{A}X_{13} = \bar{B}X_{23} = .5$$

The restrictions for the first two latent classes assert perfect agreement, and the third latent class, which represents disagreement indicates that the two types of disagreement are equiprobable. In terms of the 2 X 2 contingency table, the agreement cells are represented by response patterns 11 and 22, and the disagreement cells are represented by response patterns 12 and 21.

This model was used to test nineteen different item pairs. The items reflected aphasia and sensory-perceptual disorders of dysnomia, dysgraphia, right-left confusion, spelling dyspraxia, visual and letter dysgnosia, dyslexia, dyscalculia, central dysarthria, right tactile, visual, and auditory imperception, right finger dysgnosia, right dysstereognosis, construction dyspraxia, left tactile, visual and auditory imperception, left finger dysgnosia, and left dysstereognosis. The assessment of items reliability by means of this model offered two important measures; the probability of both agreement and disagreement, and the degree of correspondence between observed and expected cell

frequencies reflected by the likelihood ratio Chi-squared statistic which determined the extent to which the model fits the data. Table 2 reflects the item reliabilities, the Chi-square values, and the significance levels for each of the item pairs tested. The cutoff level is .05, which, with one degree of freedom, is 3.84. Thus, in reviewing the table it should be kept in mind that anything less than 3.84 is congruent with the assumption that the items are homogeneous and that the model of equivalence gives an adequate fit for the data. Anything over 3.84 reflects inconsistent responding, the items are not equivalent, and the model does not offer a good fit. Given disagreement, it is to be interpreted that one item is more difficult than the other. Disagreement does not represent random responding.

Of the nineteen item pairs tested, all proved to be highly reliable with reliability coefficients of .83 to .86. Fourteen of the nineteen showed that the model afforded an excellent fit to the data. Five of the item pairs, although highly reliable, did not offer a good fit. If one looks at some of the categories of items the idea of best fit or equivalence becomes more clear. For example, compare the items in spelling and central dysarthria. The spelling items compared were 9) Spell Square, 10) Spell Cross, and 11) Spell Triangle. When 9 and 11 are compared, the X^2 value of .403 suggests they are equivalent items and thus are a good fit. When 9 and 10 are compared, the X^2 value of 3.98 reflects that, while they are not equal, they are not entirely dissimilar. In contrast, consider the items in central dysarthria which compare 18) Repeat Triangle, 19) Repeat Massachusetts, and 20) Repeat Methodist Episcopal. The comparison of 19 and 20 results in a X^2 of

Table 2

Item Reliability Analyses for Paired Items Representing Equivalent Function.

Task	Items	X^2_L	P	Probability of Agreement
Naming	1,5	.182	<.80	.84
	1,4	3.680	>.05	.83
	2,3	.053	<.90	.85
Spelling	9,10	3.980	<.05	.86
	9,11	.048	<.90	.86
Writing	6,7	.403	<.50	.85
Visual Letter Dysgnosia	12,13	1.699	<.20	.84
Reading	14,15	1.046	<.30	.84
Computing	16,17	.000	1.00	.86
Repeating	18,19	46.600	<.01	.85
	19,20	.501	<.50	.86
Right TVA Imperception Dysgnosia	21,22	.535	<.50	.84
Reading, Right TVA Imperception	14,23	3.050	<.10	.85
Construction Dyspraxia	24,25	67.600	<.01	.85
	24,26	.287	<.20	.85
	25,26	64.900	<.01	.86
Left TVA Imperception; Left Finger Dysgnosia	28,29	3.992	<.05	.85
Left TVA Imperception; Dystereognosis	28,30	2.490	<.10	.85
Left Dystereognosis, TVA Imperception	29,30	.866	<.50	.86

.501 which reflects that the items are of equal difficulty. When 18 and 19 are compared, the resulting X^2 for these items is 46.6, which reflects that they differ considerably in level of difficulty. Similarly, copying tasks which involve the reproduction of a square and a triangle are homogeneous items, but copying a square and a cross, or copying a cross and a triangle are not.

While it is outside the scope of this paper, it should be pointed out that, given the above findings, for those item pairs which are not homogeneous, the latent class models offer an excellent opportunity for suggesting and testing other hypotheses such as ordered relations or hierarchical ordering. These hypotheses could be tested using latent class models other than the one described here. Table 3 provides contingency tables which permit direct comparisons between the expected and observed cell frequencies for the various response patterns in each of the item pairs.

Once the item reliabilities were obtained, items which were both highly reliable and afforded good fits to the data were selected to test the hypotheses proposed. In each case, four manifest variables were selected in such a way that variables A and B represented left cerebral hemisphere functions, and variables C and D represented right cerebral hemisphere functions. A latent class model was used in an attempt to explain the associations in the contingency table in terms of the four latent classes described earlier. The model was represented by mathematical equations that express the probability of a response pattern in a contingency table in terms of the joint probabilities of each latent

Table 3

Observed and Expected Cell Frequencies for Item Pairs.

Items	Response Pattern	Observed	Expected
1,5	11	236	236.00
	12	10	11.00
	21	12	11.00
	22	4	4.00
1,4	11	243	243.00
	12	13	9.00
	21	5	9.00
	22	1	1.00
2,3	11	215	215.00
	12	9	9.50
	21	10	9.50
	22	28	28.00
6,7	11	213	213.00
	12	6	5.00
	21	4	5.00
	22	39	39.00
25,26	11	182	181.99
	12	53	27.01
	21	1	27.01
	22	26	25.99
24,25	11	182	181.99
	12	1	28.01
	21	55	28.01

Table 3 (Continued)

Item	Response Pattern	Observed	Expected
24,25 (Cont.)	22	24	23.99
29,30	11	202	202.00
	12	17	14.50
	21	12	14.50
	22	31	31.00
28,30	11	207	207.00
	12	12	16.50
	21	21	16.50
	22	22	22.00
24,26	11	230	230.00
	12	6	7.00
	21	8	7.00
	22	18	18.00
14,23	11	222	222.00
	12	18	13.50
	21	9	13.50
	22	13	13.00
21,22	11	215	215.00
	12	13	15.00
	21	17	15.00
	22	17	17.00
19,20	11	191	191.00
	12	14	16.00
	21	18	16.00

Table 3 (Continued)

Item	Response Pattern	Observed	Expected
19,20 (Cont.)	22	39	39.00
16,17	11	200	200.00
	12	7	7.00
	21	7	7.00
	22	48	48.00
14,15	11	228	228.00
	12	1	2.00
	21	3	2.00
	22	30	30.00
12,13	11	239	239.00
	12	10	7.50
	21	5	7.50
	22	8	8.00
9,11	11	210	210.00
	12	11	10.50
	21	10	10.50
	22	31	31.00
9,10	11	214	214.00
	12	15	10.50
	21	6	10.50
	22	27	27.00
28,29	11	199	199.00
	12	15	21.50
	21	28	28.00

Table 3 (Continued)

Item	Response Pattern	Observed	Expected
28,29 (Cont.)	22	20	20.00
18,19	11	206	206.00
	12	3	25.00
	21	47	25.00
	22	6	6.00

class and the response pattern. The joint probabilities were computed by an iterative procedure.

Restrictions were placed on the data by setting the expected frequencies to the observed frequencies in the four cells of interest in the contingency table. Response pattern 1111 represents latent class 1, or Normals, who are expected to pass all of the items. Response pattern 2211 represents latent class 2, or those with left cerebral lesions, who are expected to fail the first two items and pass the second two. Response pattern 1122 represents latent class 3, or those with right cerebral lesions, who are expected to pass the first two items and fail the second two. Response pattern 2222 represents latent class 4, or those with bilateral diffuse cerebral lesions, who are expected to fail both sets of items.

Twelve sets of item comparisons were made. In every case the four-class model improved significantly over the model of independence and in eleven of the twelve cases it also afforded an excellent fit to the data. Four hierarchical models were tested for each of the twelve comparisons to determine whether the four-class model could be considered the model of best fit even when it clearly provided an adequate fit for the data. These comparisons are detailed in Table 4.

The first model tested was that of independence. This model is represented by hypothesis H_0 and is not hierarchical to the other models tested, since restrictions were placed on each of these four models. In each comparison made, the Chi-square values for the model of independence were well beyond the .001 level of significance. This is interpreted to mean that the model of independence is not a model

Table 4

Chi Square Values for Models Tested.

Model	Hypothesis	X^2	df	p	Preferred Model
Items 1,5,29,30					
Independence	H_0	96.530	11	<.001	
Two class (1111,2211)	H_1	15.413	12	<.000 n.s.*	
Two class (1111,1122)	H_2	15.413	12	>.100 n.s.	
Three class	H_3	8.373	10	>.500 n.s.	H_3
Four Class	H_4	8.372	8	>.200 n.s.	
Items 2,3,24,26					
Independence	H_0	181.630	11	<.001	
Two class (1111,2211)	H_1	100.140	12	<.001	
Two class (1111,1122)	H_2	91.619	12	<.001	
Three class	H_3	12.384	10	<.100 n.s.	
Four class	H_4	8.022	8	>.300 n.s.	H_4
Items 6,7,24,26					
Independence	H_0	246.660	11	<.001	
Two class (1111,2211)	H_1	113.479	12	<.001	
Two class (1111,1122)	H_2	113.479	12	<.001	
Three class	H_3	30.476	10	<.010	
Four class	H_4	6.628	8	>.800 n.s.	H_4

*n.s. = not significant.

Table 4 (Continued)

Model	Hypothesis	X^2			
Items 19,20,28,30					
Independence	H_0	151.660	11	<.001	
Two class (1111,2211)	H_1	76.316	12	<.001	
Two class (1111,1122)	H_2	140.773	12	<.001	
Three class	H_3	12.440	10	>.200	n.s.*
Four class	H_4	9.096	8	>.200	n.s. H_4
Items 9,11,28,30					
Independence	H_0	156.280	11	<.001	
Two class (1111,2211)	H_1	88.452	12	<.001	
Two class (1111,1122)	H_2	129.746	12	<.001	
Three class	H_3	11.150	10	>.300	n.s. H_3
Four class	H_4	10.360	8	>.200	n.s.
Items 21,22,28,30					
Independence	H_0	103.660	11	<.001	
Two class (1111,2211)	H_1	53.141	12	<.001	
Two class (1111,1122)	H_2	53.141	12	<.001	
Three class	H_3	13.149	10	>.100	n.s. H_3
Four class	H_4	10.278	8	>.200	n.s.

*n.s. = not significant.

Table 4 (Continued)

Model	Hypothesis	X^2	df	p	Preferred Model
Items 16,17,29,30					
Independence	H_0	266.990	11	<.001	
Two class (1111,2211)	H_1	161.943	12	<.001	
Two class (1111,1122)	H_2	237.554	12	<.001	
Three class	H_3	17.780	10	>.050 n.s.	H_3
Four class	H_4	14.416	8	>.050 n.s.	
Items 21,22,24,26					
Independence	H_0	126.020	11	<.001	
Two class (1111,2211)	H_1	67.311	12	<.001	
Two class (1111,1122)	H_2	83.354	12	<.001	
Three class	H_3	18.137	10	>.050 n.s.	
Four class	H_4	11.037	8	>.200 n.s.	H_4
Items 14,23,24,26					
Independence	H_0	117.990	11	<.001	
Two class (1111,2211)	H_1	94.668	12	<.001	
Two class (1111,1122)	H_2	67.388	12	<.001	
Three class	H_3	20.544	10	>.020	
Four class	H_4	10.455	8	>.020 n.s.	H_4

Table 4 (Continued)

Model	Hypothesis	X^2	df	p	Preferred Model
Items 9,11,24,26					
Independence	H_0	188.360	11	<.001	
Two class (1111,2211)	H_1	98.948	12	<.001	
Two class (1111,1122)	H_2	166.712	12	<.001	
Three class	H_3	25.491	10	<.020	
Four class	H_4	11.386	8	>.050 n.s.	H_4
Items 12,13,28,30					
Independence	H_0	85.910	11	<.001	
Two class (1111,2211)	H_1	41.063	12	<.010	
Two class (1111,1122)	H_2	41.063	12	<.010	
Three class	H_3	22.701	10	<.020	
Four class	H_4	21.660	8	<.010	
Items 16,17,24,26					
Independence	H_0	259.230	11	<.001	
Two class (1111,2211)	H_1	112.268	12	<.001	
Two class (1111,1122)	H_2	112.268	12	<.001	
Three class	H_3	26.450	10	<.010	
Four class	H_4	13.226	8	>.050 n.s.	H_4

of good fit, and could not be used to explain the associations in the contingency table.

The second model tested was a two-class model which included the response patterns 1111 and 2211. This model says there are two latent classes, Normals and those with left cerebral lesions which can explain the association. This model is represented by hypothesis H_1. In all instances this model improved significantly over the model of independence, but, except for one instance, did not provide an adequate fit. In the one instance it afforded an adequate fit, it turned out not to be the preferred model.

The third model tested was another two-class model which included response patterns 1111 and 1122. This model is represented by hypothesis H_2, which says there are two latent classes, Normals and those with right cerebral lesions. This model has the same number of degrees of freedom as the previous model and is thus not hierarchical to H_1. This model also showed significant improvement over the model of independence but offered an extremely poor fit to the data, except for the same instance mentioned previously. Again, however, this two class model was not accepted as the preferred model.

The fourth model tested was a three-class model which included response patterns 1111, 2211, and 1122. Hypothesis H_3 represents this model which says there are three latent classes to explain the data, Normals, those with left and those with right cerebral lesions. This model has all the features of the previous one, plus one additional constraint and differing degrees of freedom. It is thus hierarchical in nature and nested in an additive way to permit an exact partitioning

of the larger Chi-square into subsets. When subtractions are made from H_1 and again from H_2, the resultant Chi-square reflects that this model showed significant improvement over either of those models, and in seven instances afforded a good fit to the data. In order to determine whether or not it offered a best fet and could be accepted as the preferred model, it was compared directly to the four-class model.

The four-class model, it will be remembered, included response patterns 1111, 2211, 1122, and 2222, which represented the four latent classes of Normals, those with left and right cerebral lesions and those with bilateral diffuse lesions. As did the three-class model, this model improved significantly over the model of independence and both of the two-class models. In eleven of the twelve cases, it afforded an adequate fit to explain the data. When it was compared directly to the three-class model, in seven instances it offered a significant improvement over the three-class model and was accepted as the preferred model. In the other four instances, while both the three and four-class models could be used to explain the data, the three class model was accepted as the preferred model because it was the more parsimonious, having fewer constraints and degrees of freedom.

In addition to the Chi-square values obtained, attention was paid to the standardized residuals in each of the cells of interest. These are shown in Table 5. The magnitude of these residuals reflects the degree of association and is an important consideration. Standardized residuals have the property of being normal in large samples with mean 0 and standard deviation of 1. In the analysis of the contingency table, cells which do not fit the data are easily spotted and patterns discerned can suggest models of better fit. In those three-class

Table 5

Standardized Residuals for Cells 1111, 2211, 1122 and 2222.

Items 1,5,29,30

Response Pattern	Cell	4 Class	3 Class	2 Class	2 Class
1111	1	- .13	- .13	.05	.05
2211	4	.00	.00	3.50	- .01
1122	13	- .01	- .01	- .01	3.50
2222	16	- .11	- .11	- .11	- .11

Items 2,3,24,26

Response Pattern	Cell	4 Class	3 Class	2 Class	2 Class
1111	1	- .04	- .03	.10	.01
2211	4	- .06	.00	.42	5.32
1122	13	- .11	.00	15.02	.56
2222	16	- .08	2.54	1.45	- .87

Items 6,7,24,26

Response Pattern	Cell	4 Class	3 Class	2 Class	2 Class
1111	1	- .03	.02	.83	.83
2211	4	- .03	.54	.65	17.46
1122	13	- .00	.00	17.47	.65
2222	16	.00	7.33	8.18	8.18

Table 5 (Continued)

Items 9, 11, 28, 30

Response Pattern	Cell	4 Class	3 Class	2 Class	2 Class
1111	1	- .10	- .10	.26	.74
2211	4	- .04	- .02	.11	14.21
1122	13	- .15	- .11	10.27	.09
2222	16	- .04	+ .92	2.73	1.87

Items 19, 20, 28, 30

Response Pattern	Cell	4 Class	3 Class	2 Class	2 Class
1111	1	.01	.04	.30	.63
2211	4	.00	.30	.25	12.71
1122	13	- .00	.07	10.38	.00
2222	16	.07	1.63	3.06	4.35

Items 21,22,28,30

Response Pattern	Cell	4 Class	3 Class	2 Class	2 Class
1111	1	.00	- .04	.10	.10
2211	4	.02	.27	5.18	4.28
1122	13	.00	.01	4.28	5.18
2222	16	.00	1.80	- .09	- .09

Table 5 (Continued)

Response Pattern	Cell	4 Class	3 Class	2 Class	2 Class
Items 21, 22, 24, 26					
1111	1	- .05	- .05	.04	.04
2211	4	- .05	.14	4.55	4.55
1122	13	- .02	.03	6.43	6.43
2222	16	- .03	3.03	.01	.01
Items 9, 11, 24, 26					
1111	1	- .12	.00	.96	1.65
2211	4	.02	.66	.84	18.83
1122	13	.02	.03	14.14	1.35
2222	16	.00	4.23	5.86	4.85
Items 14, 23, 24, 26					
1111	1	- .07	- .07	.84	.53
2211	4	- .00	.32	.48	10.43
1122	13	- .00	- .00	14.08	.43
2222	16	.00	3.54	4.84	6.88

Table 5 (Continued

Response Pattern	Cell	4 Class	3 Class	3 Class	2 Class
Items 12, 13, 28, 30					
1111	1	- .04	- .05	.31	.31
2211	4	.00	.37	6.41	6.41
1122	13	.18	- .00	.18	.18
2222	16	2.98	3.69	4.29	4.29
Items 16, 17, 24, 26					
1111	1	.04	- .00	.98	.98
2211	4	.04	.02	.87	.87
1122	13	.31	.97	15.36	15.36
2222	16	.00	4.25	7.66	7.66
Items 16, 17, 29, 30					
1111	1	- .01	- .00	1.95	3.42
2211	4	- .05	- .01	.55	16.07
1122	13	- .10	- .02	15.30	.41
2222	16	- .08	1.96	3.43	4.47

models which were the preferred models, the standardized residuals in cell 2222 suggested that this cell should not be entirely dismissed. This will be discussed later in the paper.

A comparison was made between the coding of response patterns made by the test itself and the diagnosis made by clinical neurological examination. Results showed excellent agreement in each of those cases where consistent responding was made to the two left and the two right hemisphere functions. When inconsistent responses occurred, however, there was disagreement. The preferred code in the test was likely to be Normal whenever a passing (1) response occurred in any three out of four items, regardless of the hemisphere represented by a failing (2) response. In those instances, Reitan was more likely to have classified the patient as having a left, or right or bilateral lesion depending on the hemisphere represented by the particular item, and depending on the quality of the response. For example, examine the results of items 9, 11, 28, and 30 (Table 7, p.80) which compares the spelling of square and triangle to left tactile visual or auditory imperceptions and left dystereognosis. The preferred code for cell 2 (2111), according to the statistical probability was X_1 Normal, with .8477 probability. The actual code showed that one normal subject misspelled square, while six with left cerebral damage misspelled square, and two with bilateral damage misspelled square and got the other three items right.

In that same example, an examination of cell 1112, which represents an error in identifying geometric shapes by touch, the statistical probability for Normal was .7819. In the actual code, of the 18 people who were in that response category, 13 had right cerebral damage, and

five had bilateral diffuse damage. While this represented disagreement, it is to be interpreted that the items are not measuring the same thing and a model other than the one of equivalence would offer a better fit.

Tables 6 and 7 show the exact probability that a particular response pattern will be coded as one of the four latent classes, on one of the three latent classes, depending on the preferred model. The assignment of individuals with respect to the latent variables was made using the following mathematical formula:

$$\pi\ ABCD\bar{X}_{ijklt} = \pi\ ABCDX_{ijklt} \ /\ \pi\ ijkl$$

This says, given that an individual is at level (i, j, k, l) on the joint variable (A, B, C, D), $\pi\ ABCD\bar{X}_{ijklt}$ denotes the conditional probability that he will be at level t on variable X. These conditional probabilities were only determined in each case for the model of best fit, i.e., seven four-class models and four three-class models. Assignment to a latent level with respect to the latent variable X, was made by selecting the estimated modal latent level t corresponding to the observed level (i, j, k, l). For example, if an individual's response pattern was 1111 on the joint variable ABCD, then he would be assigned to latent class 1 with respect to latent variable X, since the estimated $ABCD\bar{X}_{1111t}$ (for t = 1, 2, 3, 4) indicated that the corresponding estimated modal level was obtained when t = 1. Similarly, it was determined that the model latent level t corresponding to an observed response pattern of 2211 was latent class two, the modal latent level for observed response pattern 1122 was latent class three, and the modal latent level for observed response pattern 2222 was latent class four. Modal latent levels are given for

Table 6

Conditional Probability $\pi^{ABCD\bar{X}}_{ijklt}$ that an individual will be in latent class t with respect to latent variable X (for t = 1, 2, 3, 4) given that his response pattern is (i, j, k, l) on manifest variables (A, B, C, D).

Items 19,20,28,30

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for latent class t			
			t = 1	t = 2	t = 3	t = 4
1111	151	150.90	.9977	.0006	.0005	.0012
2111	11	12.21	.8250	.1416	.0031	.0033
1211	12	12.21	.8250	.1416	.0031	.0033
2211	32	31.98	.0210	.9582	.0001	.0207
1121	6	10.50	.9610	.0005	.0011	.0374
2121	1	1.44	.4675	.0671	.0003	.4615
1221	2	1.44	.4675	.0671	.0003	.4615
2221	4	2.87	.0156	.5999	.0051	.3794
1112	16	10.50	.9610	.0005	.0011	.0374
2112	1	1.44	.4675	.0671	.0003	.4615
1212	2	1.44	.4675	.0671	.0003	.4615
2212	2	2.87	.0156	.5999	.0051	.3794
1122	18	18.00	.0374	.0000	.9246	.0380
2122	0	1.16	.0391	.0048	.0010	.9485
1222	2	1.16	.0391	.0048	.0076	.9485
2222	2	1.90	.0016	.0510	.0075	.9399

Table 6 (Continued)

Items 6,7,24,26

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for latent class t			
			t = 1	t = 2	t = 3	t = 4
1111	187	187.44	.9997	.0000	.0000	.0000
2111	6	5.69	.7932	.1993	.0005	.0002
1211	4	5.69	.7932	.1993	.0005	.0002
2211	32	32.18	.0033	.9961	.0000	.0001
1121	6	4.51	1.0000	.0000	.0000	.0000
2121	0	.15	*			
1221	0	.15				
2221	0	1.18				
1112	5	4.51	1.0000	.0000	.0000	.0000
2112	0	.15				
1212	0	.15				
2212	3	1.18	.0000	.9983	.0000	.0165
1122	14	14.00	.0000	.0000	.9924	.0010
2122	0	.01				
1222	0	.01				
2222	5	5.00	.0002	.0005	.0004	.9895

*Whenever Observed Frequency equals 0, no calculations were made.

Table 6 (Continued)

Items 2,3,24,26

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for latent class t			
			t = 1	t = 2	t =3	t = 4
1111	191	191.51	.9991	.0003	.0002	.0000
2111	7	6.22	.8099	.2038	.0000	.0000
1211	8	6.22	.8099	.2038	.0000	.0000
2211	23	23.27	.0055	.9934	.0000	.0000
1121	6	6.12	.8099	.0000	.2002	.0000
2121	0	.32				
1221	0	.32				
2221	0	1.48				
1112	5	6.12	.8099	.0000	.2002	.0000
2112	0	.32				
1212	0	.32				
2212	3	1.48	.0023	.8518	.0059	.1400
1122	13	13.40	.0096	.0000	.9884	.0018
2122	2	1.38	.0074	.0026	.8286	.1598
1222	2	1.38	.0074	.0026	.8286	.1598
2222	2	2.11	.0000	.0318	.0472	.9132

Table 6 (Continued)

Items 9, 11, 24, 26

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for latent class t			
			t = 1	t = 2	t = 3	t = 4
1111	185	183.38	.9999	.0000	.0000	.0000
2111	7	7.71	.9147	.0093	.0000	.0000
1211	10	7.71	.9147	.0093	.0000	.0000
2211	27	26.90	.0000	1.0000	.0000	.0000
1121	5	7.48	.9437	.0001	.0571	.0000
2121	1	.30	.9392	.0563	.0462	.0000
1221	0	.30				
2221	0	.67				
1112	5	7.48	.9437	.0001	.0571	.0000
2112	2	.30	.9392	.0565	.0462	.0000
1212	0	.30				
2212	1	.67	.0159	1.0000	.0000	.0000
1122	14	13.92	.0159	.0000	.9808	.0000
2122	1	.44	.0287	.0000	.9572	.0000
1222	0	.44				
2222	4	4.00	.0000	.0040	.0033	.9869

Table 6 (Continued)

Items 14, 23, 24, 26

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for latent class t			
			t = 1	t = 2	t = 3	t = 4
1111	192	192.96	.9648	.0334	.0858	.0099
2111	17	9.29	.9982	.0021	.0000	.0000
1211	9	9.29	.9982	.0021	.0000	.0000
2211	11	11.00	.0017	.9568	.0000	.0000
1121	6	9.29	.9982	.0000	.0031	.0000
2121	0	.45				
1221	0	.45				
2221	0	.02				
1112	7	9.29	.9982	.0000	.0031	.0000
2112	1	.45	1.0000	.0000	.0000	.0000
1212	0	.45				
2212	0	.02				
1122	17	17.00	.0263	.0000	.9738	.0000
2122	0	.02				
1222	0	.02				
2222	2	2.00	.0000	.0000	.0000	1.0000

Table 6 (Continued

Items 16, 17, 24, 26

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for Latent Class t = 1	t = 2	t = 3	t = 4
1111	177	176.45	1.0000	.0000	.0000	.0000
2111	6	5.04	.7589	.2396	.0000	.0000
1211	4	5.04	.7589	.2396	.0000	.0000
2211	42	41.77	.0020	.9980	.0000	.0000
1121	5	5.21	.7285	.0000	.2659	.0000
2121	0	.28				
1221	1	.28	.2869	.0728	.5681	.0034
2221	0	1.23				
1112	4	5.21	.7285	.0000	.2659	.0000
2112	0	.28				
1212	2	.28	.2869	.0728	.5681	.0034
2212	2	1.23	.0000	.9786	.0156	.0893
1122	13	11.91	.0069	.0000	.9963	.0241
2122	1	1.40	.0013	.0007	1.0000	.0000
1222	0	1.40				
2222	5	5.00	.0000	.0251	.1079	.9581

Table 6 (Continued)

Items 21, 22, 24, 26

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for Latent Class t			
			t = 1	t = 2	t = 3	t = 4
1111	190	190.64	.9992	.0005	.0000	.0000
2111	12	9.00	.8782	.1206	.0002	.0000
1211	14	9.00	.8782	.1206	.0002	.0000
2211	13	13.18	.0250	.9736	.0000	.0000
1121	4	8.63	.9183	.0000	.0828	.0000
2121	0	.49				
1221	1	.49	.6608	.1848	.0804	.0000
2221	1	1.39	.0095	.8018	.0016	.2053
1112	7	8.63	.9183	.0000	.0828	.0000
2112	0	.49				
1212	0	.49				
2212	1	1.39	.0098	.8018	.0016	.2053
1122	13	13.08	.0251	.0001	.9711	.0002
2122	1	1.02	.0137	.0053	.7166	.2864
1222	2	1.02	.0137	.0053	.7166	.2864
2222	3	3.06	.0002	.0293	.0127	.9307

Table 7

Conditional Probability $\pi^{ABCD\bar{X}}_{ijklt}$ that an individual will be in latent class t with respect to latent variable X (for t = 1, 2, 3) given that his response pattern is (i,j,k,l) on manifest variables (A, B, C, D).

Items 21,22,28,30

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for latent class t		
			t = 1	t = 2	t = 3
1111	173	173.46	.9935	.0069	.0002
2111	8	12.50	.7579	.2425	.0001
1211	15	12.50	.7579	.2425	.0001
2211	10	9.17	.0001	.9428	.0000
1121	7	10.64	.8904	.0035	.0751
2121	2	1.62	.3205	.6542	.0241
1221	1	1.62	.3205	.6542	.0241
2221	3	3.06	.0741	.9973	.0006
1112	17	10.64	.8904	.0035	.0751
2112	2	1.62	.3205	.6542	.0241
1212	0	1.62	.3205	.6542	.0241
2212	2	3.06	.0741	.9973	.0006
1122	17	16.97	.0307	.0077	.9610
2122	1	1.20	.2730	.3555	.7626
1222	1	1.20	.2730	.3555	.7626
2222	3	1.10	.0200	.0023	.4981

Table 7 (Continued)

Items 1,5,29,30

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for latent class t		
			t = 1	t = 2	t = 3
1111	178	179.79	.9997	.0020	.0000
2111	8	10.78	1.0000	.0000	.0000
1211	12	10.78	1.0000	.0000	.0000
2211	4	4.00	.1625	.8421	.0000
1121	16	11.34	.4527	.0021	.0296
2121	1	.66	.9877	.0572	.0164
1221	0	.66			
2221	0	.04			
1112	12	11.34	.9527	.0021	.0296
2112	0	.66			
1212	0	.66			
2212	0	.04			
1122	30	30.04	.0215	.1117	.9787
2122	1	.60	.0389	.1439	.5655
1222	0	.60			
2222	0	.01			

Table 7 (Continued)

Items 9,11,28,30

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for latent class t		
			t = 1	t = 2	t = 3
1111	163	164.22	.9975	.0005	.0012
2111	9	9.99	.8477	.1507	.0045
1211	7	9.99	.8477	.1507	.0045
2211	27	27.11	.0160	.9833	.0002
1121	11	10.79	.7819	.0412	.2172
2121	0	.84			
1221	0	.84			
2221	2	1.57	.0142	.9550	.0030
1112	18	10.79	.7819	.0412	.2172
2112	0	.84			
1212	1	.84	.5187	.1059	.3857
2212	2	1.57	.0142	.9550	.0030
1122	17	17.44	.0249	.0000	.9743
2122	2	2.37	.0051	.0020	.9944
1222	2	2.37	.0051	.0020	.9944
2222	1	.41	.0028	.2016	.7714

Table 7 (Continued)

Items 16, 17, 29, 30

Variable ABCD	Observed Frequency	Expected Frequency	Estimated Conditional Probability for Latent Class t		
			t = 1	t = 2	t = 3
1111	159	159.06	.9934	.0004	.0004
2111	2	5.64	.3115	.6161	.0210
1211	5	5.64	.3115	.6161	.0210
2211	36	36.05	.0007	.9900	.0003
1121	11	6.06	.3385	.0010	.6574
2121	0	1.05			
1221	0	1.05			
2221	6	3.59	.0001	.9668	.0328
1112	6	6.06	.3385	.0010	.6574
2112	1	1.05	.0253	.3191	.6531
1212	1	1.05	.0253	.3191	.6531
2212	4	3.59	.0001	.9668	.0328
1122	23	23.10	.0011	.0001	.9983
2122	4	4.01	.0001	.0016	.9926
1222	1	4.01	.0001	.0016	.9926
2222	3	1.02	.0000	.3359	.6885

each of the item sets used for comparison in which either a three-class or a four-class model was accepted as the model of best fit.

Tables 8 and 9 represent the models of agreement discussed earlier in the paper and present the cross-classification of individuals by clinical classification and test assignment. Table 8 gives the results of the cross-classifications in 4 X 4 contingency tables. Cells 11, 22, 33, and 44 represent agreement and all other cells represent disagreement. Individual response patterns were determined from the raw data. If, for example, an individual was found to be in latent class 1 on both clinical and test assignment, he was assigned to cell 11. If he were found to be in latent class 2 on both, he was assigned to cell 22, and so on. If, however, the modal response by test assignment was latent class 1, but his actual clinical diagnosis was latent class 2, he was assigned to cell 21, which represents disagreement.

Table 9 gives the same kind of information for the three-class models which were the preferred models. In this case the cross-classifications are represented by a 3 X 3 contingency table. Again, cells 11, 22, and 33 represent agreement and the remaining cells represent disagreement. Table 8 was determined by using a five-latent class model, and Table 9 was determined by using a four-latent class model. The coefficient of agreement was obtained by adding the probabilities of the agreement classes.

It can be seen from the tables, that in instances of disagreement, there tended to be classification by test assignment into latent class 1 in those instances when only one of an item pair was missed, regardless of whether it represented left or right cerebral function.

Table 8

Validity check for clinical and test classifications: A measure of agreement in the four-class models.

I. Repeat Massachusetts; Repeat Methodist Episcopal; Left TVA Imperception; Left Dystereognosis.

Test Classification

Clinical Classification	1	2	3	4	
1	104	0	0	0	104
2	21	26	0	0	47
3	43	0	14	0	57
4	34	13	5	2	54
	202	39	19	2	262

Reliability Coefficient = .534

II. Write Clock, Square; Write He shouted the warning; Copy Square; Copy Triangle.

Clinical Classification	1	2	3	4	
1	104	0	0	0	104
2	30	17	0	0	47
3	45	0	12	0	57
4	35	11	3	5	54
	214	28	15	5	262

Reliability Coeffficient = .505

III. Name Cross; Name Triangle; Copy Square; Copy Triangle.

Clinical Classification	1	2	3	4	
1	104	0	0	0	104
2	28	19	0	0	47
3	42	0	15	0	57
4	40	7	5	2	54
	214	26	20	2	262

Reliability Coefficient = .516

Table 8 (Continued)

IV. Spell Square; Spell Triangle; Copy Square; Copy Triangle.

Test Classification

Clinical Classification	1	2	3	4	
1	104	0	0	0	104
2	23	24	0	0	47
3	47	0	10	0	57
4	41	4	5	4	54
	215	28	15	4	262

Reliability Coefficient = .525

V. Read See the black dog; Right Dysstereognosis; Copy Square; Copy Triangle.

Clinical Classification	1	2	3	4	
1	104	0	0	0	104
2	36	11	0	0	47
3	40	0	17	0	57
4	52	0	0	2	54
	232	11	17	2	262

Reliability Coefficient = .500

VI. Compute 85-27; Compute 17X3; Copy Square; Copy Triangle.

Clinical Classification	1	2	3	4	
1	104	0	0	0	104
2	18	29	0	0	47
3	45	1	11	0	57
4	29	14	6	5	54
	196	44	17	5	262

Reliability Coefficient = .540

Table 8 (Continued)

VII. Right TVA Impercption; Right Dysstereognosis; Copy Square; Copy Triangle.

		1	2	3	4	
	1	104	0	0	0	104
Clinical	2	42	5	0	0	47
Classification	3	48	0	9	0	57
	4	34	10	7	3	54
		228	15	16	3	262

Reliability Coefficient = .430

Table 9

Validity check for clinical and test classifications. A measure of agreement in the three-class models.

I. Right TVA Imperception; Right Finger Dysgnosia; Left TVA Imperception; Left Dysstereognosis.

		Test classification 1	2	3		
Clinical Classification	1	104	0	0	104	Reliability
	2	40	7	0	47	Coefficient = .572
	3	45	0	12	57	
		189	7	12	208	

II. Name Square, Name Fork; Left Finger Dysgnosia; Left Dystereognosis.

		1	2	3		
Clinical Classification	1	104	0	0	104	Reliability
	2	42	5	0	47	Coefficient = .611
	3	35	0	22	57	
		181	5	22	208	

III. Spell Square, Spell Triangle; Left TVA Imperception; Left Dysstereognosis.

		1	2	3		
Clinical Classification	1	104	0	0	104	Reliability
	2	25	22	0	47	Coefficient = .645
	3	45	0	12	57	
		174	22	12	208	

Table 9 (Continued)

IV. Complete 85-27; Compute 17X3; Left Finger Dysgnosia; Left Dysstereognosis.

		1	2	3	
Clinical	1	104	0	0	104
Classification	2	17	30	0	47
	3	32	2	23	57
		153	32	23	208

Reliability Coefficient = .750

This is a function of the differences in item difficulty levels in those cases. It is also due to the fact that the model selected for testing allowed for less than complete adherence to the notion that any error constitutes a sign of brain damage. In no case were individuals with left lesions assigned to latent class three, and vice versa. Also, in no case were Normals assigned to any category of brain impairment. In the case of latent class four, there were so few individuals in that response category that interpretation is uncertain. They are distributed throughout the other latent classes, with most falling in latent class one.

CHAPTER 5

DISCUSSION

The present study used a latent structure analysis to explain the association between various categories of brain damage and responses to a behavioral test, the Reitan-Indiana Aphasia Screening Test. It showed that a two-class model of brain damage was not a sufficient model to explain the response patterns in a contingency table. It showed that a strong case can be made that the Aphasia Test does discriminate very well between normals, and individuals with left, and with right cerebral hemisphere lesions, and in many cases, between those three groups and individuals with bilateral or diffuse damage.

The observed and expected frequencies in response pattern 2222, were so small (n=0-5), that interpretation is uncertain. One cannot tell if the results are due to error, to an insufficient number of individuals in the bilateral-diffuse category, or whether they are due to the fact that those individuals have generalized brain inpairment and the Aphasia Test is sensitive to specific deficits. The results suggested that the test discriminates between the first three groups very well, and not so well with the fourth group. It would be important in further research to examine a much larger number of persons with bilateral diffuse damage to clarify this. In fact, this group should probably be subdivided into subjects with bilateral focal lesions and subjects with generalized, non-focal lesions.

No attempt was made to test all items on the Aphasia Test. Nor was it expected that any items tested here as equivalent were previously purported to be so. In fact, anyone who is familiar with the test is aware that items within the various receptive and expressive aphasia categories are not of equal difficulty. The selection of items was made because equivalent items are necessary to maximize the sensitivity of the model tested. Before any major conclusions can be drawn, a number of hypothetical models would have to be tested in a systematic fashion which would account for all items involved in the test. The value of latent structure lies in hypothesis testing. The rejection of one hypothesis can generate new models to be tested.

The study showed that items which were paired showed a high degree of reliability and suggested that a model of equivalence did offer a good fit to the data. In some cases, while it offered a good fit, it was found that it should not be considered the model of best fit, particularly in those cases when a given item pair were not of equal difficulty level. In those cases a different model would have to be tested to explain the data more adequately.

Cross-classification was made by clinical diagnosis and modal response by test assignment, using a latent model of agreement. These reliability coefficients ranged from .43 to .75. In those cases where a three-class model was the one preferred, the reliability coefficient ranged from .57 to .75. In those cases where the four-class model was the preferred one, the reliability coefficients ranged from .43 to .65, indicating that latent class 4 showed the greatest variance.

The lower coefficients also represent the fact that item pairs were not equivalent in level of difficulty, and the fact that the model tested permitted individuals who made errors to be considered as belonging to latent class 1. Future studies should set the probability of being in cell 1111, given latent class Normal, equal to 1. This model would assert that an individual could only be included in latent class 1 if all the items were passed. Then any error would signify brain damage and the modal assignment would be made to latent classes 2, 3, or 4. This would have the effect of assigning individuals to those latent classes when any one of an item pair was missed, and might offer a more satisfactory model which would reflect the fact that a person could have a lesion in one hemisphere and not necessarily miss every item which reflects the function of that hemisphere.

Future research in the assessment of brain-behavior relationships should include the application of latent trait models. Latent trait statistics would permit the linkage of the existing pool of items in this test, for instance, to new items which would have a common scale. Items could be set up in such a way to identify those that may be so simple that normal individuals would always get them right, and anyone who made an error would have to be considered brain-damaged. Those items could be tested using a model suggested above which would set the probability of cell 1, with response pattern 1111 equal to 1. Other items on the test may be of sufficient difficulty level that even a few normal individuals would be expected to miss them a certain percentage of the time, and a model could be tested that would predict that probability level. In this way items could be tested, those with high discrimination retained,

and new items from the existing pool could be tested until a maximal level of efficiency was reached in discriminating between the various categories of brain damage. Such an approach is entirely possible, could contribute to a refinement of the test and would add to the results already reported by Reitan and others.

It is hoped that the present study has contributed in some small way to encourage the use of latent structure and latent trait analysis to the investigation of such important areas of research as the study of brain-behavior relationships, for it offers a unique and meaningful statistic to explain the degree of association between various categories of brain damage and the differential response patterns on a behavioral test such as the Reitan-Indiana Aphasia Screening Test. Definitive findings would offer enormous contributions to this essential field of study.

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