SHERRETS, STEVEN DENNIS

BEHAVIOR DISORDERS IN ADOLESCENTS: NEUROPSYCHOLOGICAL AND BEHAVIORAL CORRELATES

The University of Nebraska - Lincoln PH.D. 1980

BEHAVIOR DISORDERS IN ADOLESCENTS: NEUROPSYCHOLOGICAL AND BEHAVIORAL CORRELATES

by

Steven D. Sherrets

A DISSERTATION

Presented to the Faculty of

The Graduate College in the University of Nebraska

In Partial Fulfillment of Requirements

For the Degree of Doctor of Philosophy

Major: Educational Psychology and Measurements

Under the Supervision of Professor Roger Bruning

August, 1980

TITLE

Behavior Disorders in Adolescents: Neuropsychological and Behavioral Correlates

BY

Steven D. Sherrets

APPROVED	DATE
Frank J. Menolascino	8/5/80
Roger A. Bruning	8/5/80
Kenneth K. Berry	8/5/80

SUPERVISORY COMMITTEE

GRADUATE COLLEGE UNIVERSITY OF NEBRASKA

GRAD 52002 300 6-80(IT)

Acknowledgements

Numerous people deserve recognition for their support and help in accomplishing the completion of this dissertation. First of all I should like to thank my children Laura, Chad, and Noelle for their patience, understanding and support while I was otherwise preoccupied. It seems that when deadlines press, one is overly inclined to ask family members to give up time they are entitled to.

My most sincere thanks goes out to Shari Anderson who has read and typed the many drafts as well as always having been a source of support, to my committee members; Dr. Frank Menolascino, Dr. Kenneth Berry, and Dr. Robert Brown who made this as "painless" a task as is possible, and to Dr. Roger Bruning, my chairman, who spent time he really didn't have, reading and discussing the dissertation to assure my timely completion. Without his support and encouragement I may never have finished.

Appreciation is extended to the subjects who were more than cooperative, and to the staff and administration of the two institutions who made the investigation possible. The cooperation and support from all of these people was unbelievable.

Finally I should like to thank the colleagues, friends and staff at the Nebraska Psychiatric Institute

who tolerated my "grumpiness" during stressful periods, my absences from work which they made possible and because they agreed to stop asking if I was done yet.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF TABLES (Cont.)

LIST OF TABLES (Cont.)

LIST OF FIGURES

Chapter I

INTRODUCTION AND PURPOSE OF THE STUDY

Behavior disorders may be present in as many as 30% (Glidewell & Swallow, 1968; Wood & Zabel, 1978) or more of school-aged children. Despite the high frequency of occurrence, however, serious research into either etiologies or treatment of this group of children did not occur with any frequency until the end of World War II (Rie, 1971). Even then the primary concern was for individuals labeled as juvenile delinquents. In fact, there was no specific medical or psychological diagnosis even available for this group until 1968 (Diagnostic and Statistical Manual, American Psychological Association, 1968).

Historically, the concern over most problems of childhood has developed as the concept of childhood itself developed. As research and theories on problems of childhood emerged, they have mirrored the prevailing social and political attitudes toward children (Rie, 1971). Frequently, in fact, research and opinion have been closely aligned and at times indistinguishable from one another. Nowhere has this been more true than in the area of childhood behavior disorders. Experimental explanations of behavior disorders have been dominant for many year, both in guiding research and popular opinion. Because of the overwhelming evidence that social/environmental factors were frequent companions of behavior disorders and the prevailing attitude that

we would have a greater chance of successfully intervening with such factors, evidence to support the alternative explanations offered by the biological theories was not aggressively pursued. Biology was viewed rather narrowly and it was felt that to accept a biological theory one had to reject the role of learning and motivation (Merton & Nisbet, 1961).

Nash (1978) has struck a serious and hopefully fatal blow however, at the separatist view that the behavior of humans is due to either nature or nurture. For the first time we seem now to be able to explore the interaction of these two factors and to disregard the criticism that research or theorizing with one variable necessarily means we are rejecting the other. Consistent with this view are the conclusions of Rutter (1977), who in reviewing the available literature including his own research, reports that brain damaged individuals may be more susceptible to the development of behavior problems. Other authors have reported numerous findings that are suggestive of an interaction between environmental conditions and the presence of brain dysfunction. One may cause the other or each may interact to exacerbate the effects taken singly or together (Clark, 1970; Cravioto & Delicardie, 1970; Lewis & Balla, 1976; Pasamanick, 1956, 1961; Stott, 1962; Stott & Latchford, 1976).

As one can see from the date of the references the majority of the authors taking an interactive approach are from the last decade and a half. In 1937 Julian Huxley called for research of an interactive nature such as this because of what he saw was the then current trend toward such intensive specialization that each branch of science was reduced to a condition of meaninglessness. It has taken over 40 years for us to realize that behavior disorders must have multiple determinants, many of which interact with each other.

Despite the historic reluctance to initiate biological research in the area of behavior disorders or accept results indicating that biological factors play an important role, some research has still gone on. Werner and Strauss (1940) were among the first to present evidence that brain functioning may help to explain behavior disorders, although other authors had previously voiced the opinion that such a relationship existed (Mills, 1890). It was Strauss who first offered the concept of minimal brain damage (MBD), a disorder in which both neurological dysfunction and behavior problems are thought to be present. Minimal brain dysfunction is a condition which suggests the presence of abnormal brain functioning, but for which the evidence is not conclusive enough to be definitive. From this initial seed other studies have sprouted.

Descriptive studies found some form of brain dysfunction to be present in as high as 59 percent of behaviorally disordered children (Hanvick, Nelson, Hanson, Anderson, Dressler, & Zarling, 1961). Although the incidence of neurological dysfunction varies with the type of diagnosis, it generally occurs four to eight times more frequently in behaviorally disturbed populations than with matched controls (Hertzig & Birch, 1966).

Children with a history of known illness or injury of the brain are over-represented in behavior disordered populations (Pasamanick, 1961) although these account for relatively few actual cases (Rutter, 1977). While controversial and still accounting for only a minority of the children, but representing a larger number of cases, is the hypothesized relationship between seizure disorders and behavior problems. Psychomotor epileptic symptoms have been found to occur over twelve times more frequently in behavior disordered populations (Lewis & Balla, 1976). Overall, the majority of studies have simply found subtle indications of brain dysfunction or neurological delays with little if any localizing signs, or consistencies in the symptoms to clearly indicate that a particular type of neurological dysfunction is present.

There has, however, been a greater degree of consistency in studies indicating an immature, more slowly developing nervous system among children with behavior

disorders. This delay hypothesis is most clearly demonstrated in the results from electroencephalography studies. EEG abnormalities, primarily slow activity which exceeds age expectancy, are common among children with behavior disorders (Anderson, 1963; Beshai, 1971; Forsmann & Frey, 1953; Green, 1961; Gross & Wilson, 1974; Hill, 1952; Kelloway, Crawley, & Maulsby, 1965; Kennard, 1960; Klinkerfuss, Lange, & Weinberg, 1965; Laufer, Denhoff & Solomons, 1957). Stamm (1978) reports that the majority of findings are of diffuse, nonspecific patterns indicating a neurological immaturity rather than any specific syndrome. Ellingson (1954) concludes that no specific EEG abnormalities have been conclusively found to be related to any specific symptoms in children with behavior disorders.

Such a delay hypothesis would be relevant for explaining both the over-representation of males in behavior disordered populations (Werry & Quay, 1971) in that their nervous system develops more slowly, (Nash, 1979) and why so many appear to "grow out of it" (Reckless, 1967; Coleman, 1964).

While many studies have consistently concluded that the incidence of some type of neurological dysfunction is higher in behavior disturbed populations, much more controversy has arisen when neurological dysfunction is purported to be a causal agent rather than simply a correlate. Many authors believe that brain

dysfunction plays an etiological role (Anderson, 1963; Clements, 1966; Daly & Matthews, 1974; Gibbs & Gibbs, 1950; McCord & McCord, 1956), whereas others criticize this view and suggest that at best it is simply an unexplained correlate which is generally present, and only in subtle forms at that (Montagu, 1976; Pond, 1961; Werry, 1979).

Several factors account for the lack of any definitive conclusions regarding the possible causal role of brain dysfunction in this behavior. In general, these problems can be considered under the general topics of methodology, measurement, subject descriptions, and available theory.

Much of the research investigating brain dysfunction in behaviorally disturbed children has simply compared these children with normal controls and found nonfocal, nonlocalizing "soft" signs of dysfunction (e.g., Berman, 1972; Hertzig, 1969; Kennard, 1960; Larsen, 1964; Pollack, 1969; Wikler, Dixon & Parker, 1970). These cross-sectional studies do not allow one to investigate the developmental changes in either brain dysfunction or their behavioral problems.

Few studies have attempted to compare the severity or specific type of neurological dysfunction to the degree or type of behavioral problem. The amount of error variance present in both the measurement of the neurological disorder as well as the behavioral problems

appears to be the major drawback to this type of research. Werry (1979) points out that ". . . the diagnosis of brain dysfunction in the majority of children with behavior disorders is no more than an enlightened guess".

Multiple measurement problems have been a major obstacle to pursuing specific aspects of this area. Unfortunately, relying upon "soft signs" of neurological dysfunction all too often results in "soft" research. It is no wonder that loud cries from critics such as Pond (1969) abound at the suggestion that there is a connection between brain dysfunction and behavior disorders when the presence of the former is said to be indicated by a condition called "minimal brain damage" in which half of the children who are so diagnosed are "neurologically intact" (Wender, 1971). Wender (1971) indicates that the usual conclusive signs of neurological abnormality are frequently absent.

Neuropsychological tests have proven to be more valid than the clinical assessments of neurological dysfunction (Reitan & Davison, 1974) but even here the problems with measurement have proved frustrating. Much of the research with this population relied upon single tests (e.g., the Bender-Gestalt Test; Bender, 1938) or single tasks (e.g., Finger Tapping, Andrew, 1977) thought to be sensitive to brain dysfunction. Of greater promise

have been the neuropsychological batteries such as the Halstead-Reitan (Reitan & Davison, 1974). Despite its greater validity as a measure of neurological dysfunction it has never, in its complete form, been used with this population (Tramontana, Sherrets, & Golden, in press). Also, one problem is that performance on the battery has been shown to be influenced by variables which are characteristic of psychiatric patients but who do not appear to have brain dysfunction (Golden, 1978).

Recently, a new neuropsychological battery has been developed which promises to provide greater discrimination between brain damaged individuals, psychiatric populations without brain damage, and normal samples. This test is called the Luria-Nebraska Neuropsychological Battery (Purisch, Golden, & Hammeke, 1978). This battery requires far less time to administer than the Halstead-Reitan and has thus far proven to be generally more valid as a discriminator of brain dysfunction, as well as better able to indicate the specific location of the dysfunction than any previous single task, test or battery of tests (Golden, 1980).

The development of this scale represents a significant advancement of the available theory of brain functioning. Without knowledge of what area of the brain is involved in a particular behavior, little hope can be offered in understanding the potential causative or

correlative role of brain dysfunction. Even less optimistic would be the belief that some form of intervention or rehabilitation is possible. Efforts to achieve localization of function have historically been overly simplistic or lacking in validity.

Early theories of localization were simplistic and not much more than "higher phrenology" (Montagu, 1976). Lashley (1929; 1937) argued that localization was not possible beyond the basic sensory and motor skills. Both Montagu and Lashley correctly observed that early localization theorists had a simplistic view of the brain which could not be supported by the available evidence.

An English neurologist, J. Hughlings Jackson, demonstrated that one could produce accurate statements of localized brain behavior relationships without sacrificing the complexity and interrelatedness posed by Lashley's equipotential theory. It was Jackson who suggested that complex behavior and mental functions were not unitary abilities but made up of simpler basic skills (Luria, 1966). Krech (1962) sums this up well by concluding that: 1) no learning process or function is entirely dependent on any one area of the cortex and, 2) that each area within the brain plays an unequal role in different functions.

A.R. Luria (1860 - 1973) adapted the work of Jackson and developed a theory capable of explaining both the

localist and equipotentialist points of view. Luria developed a detailed qualitative system of theory and clinical observation in devising a measure of brain function and localization of brain-behavior relationships. These procedures are reflected in standardized form with the Luria-Nebraska Neurological Battery.

Luria's theory predicts several areas of the cortex which could be involved in the development of behavior problems. Table 2 presents an overview of some of the behaviors from various areas of the brain which are frequently related to behavior problems. The frontal lobes are the last area of the brain to develop (Burn, 1960) and yet they are extremely important in providing the mechanism for regulating and controlling behavior. It is here that the strongest relationships may exist with behavior disorders (Pontius, 1974; 1976). Since the frontal lobes are so slow to develop a deficit within this area would be consistent with the developmental delay theory.

Deficits in the temporal region may help to explain the presence of hallucinations (Mullan & Penfield, 1959), distortions of time and space (Williams, 1968), and aggressiveness (Falconer & Serafetinides, 1963; James, 1960).

Dysfunction with the parietal-occipital area may help to explain some of the reported educational diffi-

culties behavior disordered children often have (Sherrets, 1976). This is particularly so with tasks requiring spatial and language abilities (Benson & Wier, 1972; Kinsbourne & Warrington, 1964; Luria & Tsvetkova, 1964).

No single study has, to date, ever attempted to use a test battery that is sensitive to brain function in each of these cortical areas in order to differentially test their importance. With the availability of the Luria-Nebraska this now appears possible. Only one major obstacle remains, that of more complete subject and behavior descriptions. Many studies have only offered means for age and IQ and have not provided any additional objective descriptions of behavior. Most investigations of individual differences with behavior disordered children have produced inconclusive and inconsistent results because they have proceeded with comparisons of subject populations with matched controls as if the former was a homogenous group (Peterson, Quay, & Cameron, 1969). Clearly one cannot find the existence of individual differences without thorough objective descriptions of the individuals to begin with. As Reinert (1980) points out, definitions of behavior disorders are fraught with problems, not the least of which is the lack of an objective taxonomy. Commonly used classifications are based upon narrative descriptions that are not operationalized, have low validity and reliability, and do not differen-

tiate as to etiology, prognosis, or treatment (Edelbrock, 1979).

At about the same time as the Luria-Nebraska was being developed a scale for the empirical classification of behavior disorders was being constructed. Achenbach and Edelbrock (1979; 1978) developed an objective rating scale which provides descriptions of both narrow and more general patterns of behavior. This scale was developed by factor analyzing a diverse array of behavior problems as checked by parents or parent surrogates with over 1,000 cases. Each of the scales represents a factor with Internalizing and Externalizing as second order factors. The availability of this scale provides the objective description of behavior to address the last major criticism of previous investigations.

It would appear that the majority of prior cirticisms can now be addressed in a definitive investigation regarding the role of brain dysfunction in behavior disorders. For the first time empiricism and theory can proceed hand in hand.

An attempt will be made to operationalize definitions, blend neuropsychological theory and measurement, and differentiate problem behaviors to be able to not only indicate the rate of brain dysfunction but attempt to achieve localization of specific brain dysfunction based upon differential symptomatology in adolescents twelve to sixteen years of age.