THE UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

AN ANALYTIC VIEW OF THE ETIOLOGICAL THEORIES OF AUTISM

A DISSERTATION

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

BY

JO ELLEN BEARD

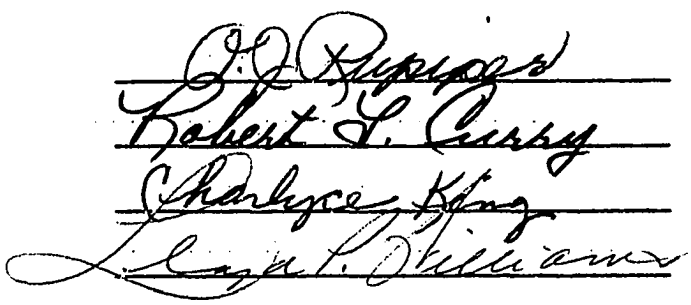
Norman, Oklahoma

1976

AN ANALYTIC VIEW OF THE ETIOLOGICAL

THEORIES OF AUTISM

APPROVED BY



DISSERTATION COMMITTEE

ACKNOWLEDGMENTS

I wish to express my appreciation to several individuals who were instrumental in the completion of my doctoral program. Special gratitude is extended to Dr. Omer J. Rupiper, Chairman of the advisory committee, who provided continuous support and guidance throughout my tenure as a graduate student. I would also like to acknowledge Dr. Robert Curry, Dr. Charlyce King, and Dr. Lloyd Williams for their aid and concern during the completion of this study.

I wish to thank my family for their continued encouragement and confidence throughout my graduate career. Appreciation is also extended to Mrs. Mary Savage for her aid in the preparation of this manuscript, and to Jeff Moorman for his patience and enthusiastic support.

TABLE OF CONTENTS

Chapter Page

AN ANALYTIC VIEW OF THE ETIOLOGICAL THEORIES OF AUTISM

CHAPTER I

INTRODUCTION

Early Infantile Autism had been studied extensively for more than 30 years since Leo Kanner first delineated the disturbance as a distinct clinical entity in 1943. Experts in the fields of medicine, education, and psychology contributed their efforts in providing volumes of empirical, clinical, and observational information about this childhood syndrome. Yet, by the 1970's, despite the hundreds of research attempts and the continually mounting number of medical reports, the lack of consensus on the etiology of autism left many questions unanswered.

With the present confusion as to the causes, diagnosis, prognosis, and treatment of this atypical personality development, the experts devoted to helping these children had little basis or hope for their attempts. Due to the number of divergent theories in this field and the lack of empirical concensus on the etiology of autism, attempts to unify and analyze the available information must be made.

Background of the Problem

The diagnosis of early infantile autism had been applied to hundreds of children, many of whom would never be brought out of their world of affective withdrawal. Although the incidence of autism was estimated at only 2 per 10,000 school children (Treffert, 1970) to approximately 4.5 per 10,000 school children (Lotter, 1966), the behaviors peculiar to this syndrome usually rendered the child unable to function normally in an educational or occupational setting. To intensify the prevalence of this syndrome, Kozloff (1973) indicated that autism was more common than blindness and that it occurred almost as frequently as deafness in children.

According to Kanner (1944), the parents referred the children primarily under the assumption that they were severely feebleminded or with the question of auditory impairment. When expressing the extreme autistic aloneness of their children, parents described them as always having been "self-sufficient," "like in a shell," "happiest when left alone," "acting as if people weren't there," or "giving the impression of silent wisdom" (p. 211). He further reported that the "common denominator in all these patients is their disability to relate themselves in the ordinary way to people and situations from the beginning of life" (p. 211).

Ornitz and Ritvo (1976) stated that because often the earlier and more subtle symptoms either had not been noticed or accepted by the parents, the most frequent initial

referral was made on the basis of a delay in speech development. They further reported that the majority of the children diagnosed as autistic were most commonly evaluated when the child was two to three years old.

The studies of prognosis were much in agreement, indicating that only approximately one-third of the autistics were able to achieve adequate adjustment to school and community (Kanner & Eisenberg, 1955; Kanner, 1971). One-sixth obtained regular paid employment and one-sixth made a fair social adjustment. Fifty per cent of the children employed speech, but continued to indicate a persisting language disability. It is interesting that, although the social relationships in many of the children were reported as improved, they rarely made close friends and none of them chose to marry. Rutter (1966) further reported that ordinarily by the age of six or seven, if noticeable improvement was to be made, it would be apparent by that age. When attempting to predict the prognosis for an autistic child, Rutter stated that:

> Four factors were found to be related to outcome: the initial IQ, the degree of language impairment, the total symptom score (reflecting severity of disorder), and the experience of schooling. Apart from language, no single symptom predicted outcome. In particular, social abnormalities did not. To summarize the findings on prognosis: When the I.Q. was below 50 the outlook was very poor more or less regardless of anything else; if the I.Q. was above

> 50 but there was a severe language disability the prognosis was for fair adjustment only; if there was normal non-verbal intelligence and only moderate language impairment the outlook was good, especially if the child had adequate schooling (1974, p. 149).

However, even with the best prognosis and treatment, it was shown that approximately 75% of the autistics functioned on a mentally retarded level throughout their lives (Halvelkova, 1968; Rutter, 1970). For an autistic child who had not acquired the use of language for communication purposes by the age of five, hope for future gains was very dim and it was assumed that the child would probably need institutionalized care (Eisenberg, 1956; Eisenberg & Kanner, 1956; Rutter, 1967). Despite the philosophical, etiological, or behavioral techniques employed, no specific treatment or therapy proved to alter the natural history of the disease (Ward, 1970). Rutter and Bartak (1971) stated that very few autistic children ever recovered completely.

Statement of the Problem

All too often psychologists and pediatricians were unaware of or inadequately educated about the etiology and diagnosis of infantile autism. As late as 1973, MacCulloch and Sambrooks (1973) reported that all of the members of those fields did not even believe in the syndrome of autism. The response by the pediatrician or psychologist was usually

a wait-and-see attitude; thus, the child was given no help until he developed more bizarre, entrenched behaviors. It was evident in the literature that in any childhood psychosis or disorder, including autism, the child had a far better chance of amelioration if he began treatment early than a child who became rigid and immobile in his perspective of the world. Therefore, a need to educate and inform those who might come in contact with autistic children was revealed in the literature.

As none of the treatment attempts appeared to alter the course of the syndrome, a need for etiologically related treatment approaches was present. Efforts to unify and analyze the existing, as well as the current, ongoing literature needed to be made. Advanced educational, clinical, and medical techniques were continuously presenting new data, and the implications of these research findings must be revealed in and reflected by the integrative attempts of researchers.

The primary focus of this study was to investigate the etiological theories of autism as they were presented in the literature and to analyze each theory as it related to the available empirical data. Independent, comprehensive, continuing efforts were needed to review the existing etiological theories of autism and to report the empirical data supporting or failing to support the theories. The problem of this study was, therefore, "What were the existing etiological theories of early infantile autism and what were the available empirical studies that related to these theories?"

Statement of the Purposes

Many physicians and psychologists previously considered early infantile autism a futile area of study, and due to no available effective treatment methods, the majority of autistics remained functionally autistic throughout their lives. Those who offered etiological theories in the literature appeared to present primarily those studies which supported their own views of the etiology of autism. With this in mind, the purpose of this study was to compile a concise, yet understandable, presentation of the postulated causes of autism, and to objectively review the empirical and observational correlates related to these theories.

Related Literature

Historically, the studies and literature relating to the childhood psychoses were introduced laggardly. Kanner (1971) reported that throughout the first 45 volumes of the American Journal of Insanity, from 1844-1889, not one of the articles pertained to children. And it was not until 1911 that Eugen Bleuler first termed autism to mean the extreme withdrawal into an inner life from the external environment. Although Bleuler wrote prolifically of the group of schizophrenias, he made very little mention of children.

In 1943 Kanner clinically identified a group of eleven children at the Children's Psychiatric Clinic at the Johns Hopkins Hospital as having "autistic disturbances of affective contact" (p. 217). The first child he saw was in

October of 1938. His name was Donald and he was five years old. From the age of two-and-a-half he could recite the names of all the presidents and vice-presidents yet he was unable to respond to or communicate with those around him in any meaningful way (Kanner, 1943). Because of the indications that this condition was present in early infancy, Kanner adopted the term "early infantile autism" (p. 211) in 1944. Kanner's recognition and definition of this childhood syndrome remained an inspiration and a hallmark in child psychiatry.

The authorities in the field were much in agreement as to the clinical features of autism and listed the behaviors suggestive of the disorder. A widely used diagnostic scale was developed by Clancy, Dugdale, and Rendle-Short in 1969. When seven of the following 14 recognized symptoms were present within the first three years of life a diagnosis of infantile autism was considered. In decreasing order of frequency the symptoms were as follows:

1) Great difficulty in mixing and playing with other children.
2) Acts as if deaf. No reaction to speech or noise.
3) Strong resistance to any learning--either new behavior or new skills.
4) Lack of fear about realistic dangers, e.g., may play with fire, climb dangerous heights, run into busy road, or into the sea.

5) Resists change in routine. Change in the smallest thing may result in acute, excessive, or seemingly illogical anxiety--e.g., child rejects new or all but a few foods.

6) Prefers to indicate needs by gestures. Speech may or may not be present.

7) Laughing and giggling for no apparent reason.

8) Not cuddly as a baby. Either holds himself still or clings limply.

9) Marked physical overactivity. Child may wake and play for hours in the night and yet be full of energy the next day.

10) No eye contact. Persistent tendency to look past or turn away from people, especially when spoken to.

11) Unusual attachment to a particular object or objects. Easily preoccupied with details or special features of this object and has no regard for its real use.

12) Spins objects, especially round ones. Can be totally absorbed in this activity and distressed if interrupted.

13) Repetitive and sustained odd play, e.g., flicking pieces of string, rattling stones in a tin can, tearing paper.

14) Standoffish manner. Communicates very little with other people. Treats them as objects rather

than people. (Clancy, Dugdale, & Rendle-Short, 1969, pp. 435-436).

A need to isolate the behaviors that were universal to and specifically characteristic of autism was present. Three such symptoms were noted in studies presented by Rutter (1966) and Rutter and Lockyer (1967). They were: 1) a profound and general failure to develop social relationships; 2) language retardation with impaired comprehension, echolalia, and pronomial reversal; and 3) ritualistic or compulsive phenomena. Both of these diagnostic criteria lists were supportive of Kanner's original list of symptoms as proposed in 1943.

Four out of five autistic children indicated no period of normal development, whereas 20% appeared to develop normally for a period of time and then regress (Rutter, 1972). Despite the pattern of the development, for a diagnosis of autism the symptoms were present in early infancy or at least by the age of 30 months (Rutter, 1971). A U-shaped relationship between the incidence of autism and ordinal birth position was shown by Wing (1966) and Rutter (1967) which indicated an increased risk to the first, fifth, and subsequent births. There appeared to be a high male to female ratio with autism being three or four times more common in males (Rutter, 1972). Creak and Ini (1960) estimated the male/female ratio at 3.6:1 while Lotter (1966) suggested a more conservative incidence of 3.1/2.8:1.

The parents of autistics were thought to be above average in intelligence and social class by many of the pioneer researchers (Creak, 1960; Kanner, 1954; Lotter, 1967). However, the more recent studies indicated that there were no significant differences in the verbal intelligence of the parents when they were matched for socioeconomic status with parents of normal children (Allen, DeMyer, & Norton, 1971; Levine & Olson, 1968). These findings were confirmed by Ornitz and Ritvo (1976) in their clinical experience and unpublished data. The results indicating superior social class were not confirmed when autistics were compared to non-autistic psychotics, non-autistic patients, and matched normals respectively (McDermott, Harrison, Schrager, 1967; Ritvo, Cantwell, Johnson, Clements, Benbrook, Slagle, Kelly, Ritz, 1971; Allen, DeMeyer, Norton, 1971).

Definition of Terms

For the purposes of this investigation, important terms were defined as follows:

Autism. Autism was derived from the Greek word "aphtotisis" which meant "withdrawn into oneself" (Savage, 1968-69, p. 77). The World Health Organization Glossary defined autism in the following manner:

> Includes a syndrome present from birth or beginning almost invariably in the first 30 months of life. Responses to auditory and visual stimuli are abnormal and there are usually severe difficulties in the understanding of spoken language.

Speech is delayed in developing; if it does develop, it is characterized by echolalia, the reversal of pronouns, immature grammatical structure and inability to use abstract terms. There is generally an impairment in the social use of both verbal and gestural language. Problems in social relationships are most severe before the age of five years and include an impairment in the development of eye-to-eye gaze, social attachments, and cooperative play. Ritualistic behavior is usual and may include abnormal routines, resistance to change, attachment to odd objects and stereotyped patterns of play. The capacity for abstract or symbolic thought and for imaginative play is diminished. Intelligence ranges from severely subnormal to normal or above. Performance is usually better on tasks involving rote memory or visuo-spatial skills than on those requiring symbolic or linguistic skills (World Health Organization Glossary, 1974, p. 54).

Etiology. The study of the cause of autism.

Theory. The New American Medical Dictionary and Health Manual defined a theory as a credible scientific idea; a supposition backed up by many scientific facts (Rothenberg, 1975, p. 336).

Questions to be Answered

This investigation was concerned with an attempt to answer the following questions:

1) What existing theories postulated parental or environmental pathology as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

2) What existing theories postulated a type of schizophrenia as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

3) What existing theories postulated mental subnormality as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

4) What existing theories postulated perceptual disorders as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

5) What existing theories postulated developmental or maturational disorders as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

6) What existing theories postulated language disorders as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

7) What existing theories postulated abnormal levels of physiological arousal as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

8) What existing theories postulated neurological or cognitive dysfunction as etiologic of early infantile

autism and what empirical data supported or failed to support these hypotheses?

9) What existing theories postulated genetic, metabolic, or bio-chemical factors as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

Procedure

The procedures associated with the collection of the data were those involving the analysis of available and accessible published materials located in libraries and affiliated agencies. Educational as well as psychological and medical libraries were searched during the collection of this data.

Limitations

This study was concerned with literature relating to children who were classified as having early infantile autism, and a conscientious effort was made to delete studies relating predominantly to children who would fall into other diagnostic classifications of childhood psychoses. An attempt was made to integrate the different fields of endeavor related to the study of autism; therefore, literature from the fields of education, psychology, and medicine was incorporated. It was not possible for this study to review all of the theoretical and empirical data that was available; thus, only those citations considered pertinent to and explicable of the etiology of autism were included.

CHAPTER II

THEORIES OF AUTISM

The literature was replete with theories postulating the cause of autism, and the disperse nature of the theories brought about a need to review and analyze them in several general classifications. Parental or environmental pathology, a type of schizophrenia, mental subnormality, perceptual disorders, language disorders, abnormal levels of physiological arousal, neurological or cognitive dysfunction, and genetic, metabolic, or bio-chemical factors that related to the etiology of autism were presented.

Parental or Environmental Pathology as Etiologic

Early infantile autism as an environmentally determined disorder ordinarily proposed that the children were able to feel the emotional coldness of the parents or, usually more specifically, the mother. They postulated that the children retired from the rejection and coldness and retreated into a world of fantasy and inanimate objects.

Kanner (1943) described the parents of autistic children as cold, emotionally rigid, compulsive people who appeared to have little capacity for warm interpersonal

relationships. They appeared more concerned with intellectual pursuits rather than interpersonal activities. They were often skillful in intellectual, scientific, or artistic abilities, but indicated little interest in people. He suggested that the disturbance was a result of the "refrigeration of the parents" which resulted in the "emotional privation" of the constitutionally vulnerable child. Kanner described these disturbances as "inborn", but he indicted the rejecting parents as the precipitating factor. Likewise, Rank (1949) also suggested that the emotional deprivation was caused by the rejecting parents.

Mahler (1952) described the etiology as a result of the lack of a love object relationship between the infant and his mother. Due to this traumatic relationship with the mother figure, the child exhibited no sign of affective awareness of other human beings because he could not incorporate the mother as an emotionally safe representative of the external world. He, therefore, withdrew from all contact because of the painfulness encountered with each experience. Mahler divided the social development of the child into five stages and postulated that the fundamental breakdown of the mother-child relationship in the first stage was responsible. In this symbiotic stage, the infant was not aware that he was separate from his mother nor that their emotions and reactions were independent. During this stage, she felt that the infant gained all of his life experience through his mother, and she used the lack of anticipatory reaction

displayed by the infant as documentation for her premise.

In the late 1960's the literature revealed several other proponents of parental pathology. Bettleheim (1967) postulated that the child rejected the world because of the cold, aloof, unresponsive behavior of his parents. The child attempted to make contact with the world but the response was so frightening that his only recourse was to seek comfort in an inner environment. Bettleheim believed that the child withdrew into his autistic world out of the fear that his parents wanted to destroy him. He went on to say that the success or failure of the early mother-child relationship determined the success or failure of all of the child's subsequent social relationships. Moreover, Bettleheim did not consider the attitude of the mother the critical factor, but rather the child's spontaneous reaction to it. Bettleheim wrote that the overpossessive mother, in anticipating the child's every need, denied the child the opportunity to explore, differentiate, experiment with cause and effect, communicate affect, and cause response. Thus the child gave up, withdrew his interest from the world and in that manner became autistic.

Ferster (1966, 1967) attempted to explain the etiology of autism in terms of operant conditioning. He suggested that the parents extinguished the child's attempts at relating simply by not reinforcing his attempts. In a similar manner, the self-stimulating behaviors were reinforced when the child experienced pleasure from his repetitive, nonsocial behaviors.

Eisenberg (1957, 1972) ascribed to the etiology proposed by Kanner and he implicated the emotional coldness and obsessive qualities of the parents as contributing to the psychogenic disorder. He contended that there was a biologically inborn defect in the child, but that the parents contributed in producing the resulting autistic behavior. He, however, divided infantile autism into two categories: those with etiologies of the type Kanner proposed, and a second group (the symptomatic group) with etiologies resulting from neurological damage.

Franknoi (1967) suggested that a disordered relationship between the mother and child was responsible for the condition, and she presented a study in support of her hypothesis. Her study suggested that the mothers had a significantly strong identification with the autistic condition in their children. She defined autism as

> a state of frozen balance between aggressive and libidinal drives with severely deficient--but not altogether absent--capacity for object cathexis, in contrast to anaclitic depression which is an objectless state in which aggression, by far outweighing libido, is turned toward the self, thus producing marasmus and even death. The autistic defense, on the other hand, is understood as insuring homeostasis and its rigid, largely impenetrable solidity as protection against any interference that might produce an

intolerable intensification of aggressive drives (Franknoi, 1968, p. 18).

A Type of Schizophrenia as Etiologic

Several authors considered infantile autism to be an early manifestation of adulthood schizophrenia. Bleuler (1911) pointed out that schizophrenics appeared to live in a world of their own, and that they kept their contact with the outside world to a strict minimum. He noted that, in a like manner, autism was also a retreat from reality.

At the inception of the study of autism, many theorists considered it merely as a form of schizophrenia because the severely disturbed and atypical relationships with other people were a prominent feature of both conditions. Attempts at the diagnosis, nosology, and treatment of autism brought about the suggestions to align the condition to schizophrenia which, at that time, was far better clinically understood. This was previously one of the most widely subscribed to philosophies on the etiology of autism (Rutter, 1968).

O'Gorman (1967) felt that autism and schizophrenia were the same disease, but that they had different variables and that they were at different stages of development. He cited as support for his hypothesis that it was difficult to clinically distinguish between the adult deteriorated autistic and the deteriorated schizophrenic who had been diagnosed only since adolescence.

Cohen reported that autistic children responded to different types of medication in a manner like schizophrenic

adults. Thus, when stimulants were administered the conditions were worsened in both the autistic children as well as the adult psychotics. He further noted that many of the same drugs also yielded positive results in the behaviors of both groups. Particularly with haloperidol and activating phenothiazines, decreases in sterotype behavior were noted as well as increased attention, social relatedness, and language profusion (Cohen, 1975).

Mental Subnormality as Etiologic

Subnormal intellectual functioning as well as the potential for exceptionally high intellectual functioning were both presented as possible causative factors in autism, although these theories were in the minority. It was not until the late 1950's that researchers suggested that standard psychological and physiological techniques could be employed in evaluating autistic children (Anthony, 1958; O'Connor & Hermelin, 1964, 1965) at which time progress in this area became more rapid.

As approximately 75% of autistics had been shown to function on a mentally retarded level throughout their lives (Halvelkova, 1968; Rutter, 1970) several theorists suggested that autism was simply a form of mental subnormality. Van Krevelen (1952) agreed with this position as he felt that autism was oligophrenia with concomitant emotional defects. Although many of the theories of mental subnormality incorporated environmental effects in their explanations of

autism, the mental subnormality was considered the dominant effect on the child.

Perceptual Disorder as Etiologic

Wing, et al. (1971) theorized that the autistic child was overwhelmed with a multiplicity of impairments in which the degree of severity varied. They believed that the impairments effected auditory perception, visual perception, comprehension and use of speech, comprehension and use of gestures, and certain specific aspects of physical development.

Goldfarb (1961, 1964) identified two groups of autistic children with different etiologies. He suggested a variety of possible etiologies which ranged from environmental determinants to organic involvement. However, he specifically noted that in the "organic" group of autistic schizophrenics, there appeared to be a high incidence of disturbances in perception and motility. In studying the receptor preferences of these children (Goldfarb, 1956) he noted a significant preference and usage of near receptors (tactile sensations) over far receptors (vision and audition).

Stroh and Buick (1964) hypothesized that the behaviors peculiar to autism resulted from different facets of an underlying perceptual disturbance. They further stated that "normal perceptual development proceeds in an orderly hierarchical manner and that failure of such orderly progression may lead to psychotic perceptual maldevelopment" (p. 291). They saw the psychotic behavior as a result of

the child's attempts to integrate or make meaningful the impinging stimuli or as an attempt to adapt to his environment in spite of the underlying perceptual limitations.

Perceptual abnormalities were also noted by Anthony (1958) as etiologic. In his analysis the distorted perception presented an inaccurate view of reality to the child and thus made object relations and contact with reality quite difficult to establish. He applied this perceptual distortion to the cases he termed "secondary" autism.

It was suggested that everyday stimuli might be recepted by the autistic child in a manner that could be overwhelming to him (Bergman & Escalona, 1949). This implicated a deficiency in primary autonomous ego functions that resulted from an inadequate stimulus barrier. This condition yielded unusual sensitivities and perceptual distortions in the child. Zaslow (1967), however, felt that inadequate sensorimotor, tactile, and kinesthetic handling in infancy left insufficient opportunities for the infant to establish basic trust through the mobilization and externalization of affect.

Kirk (1968) reported a learning problem in the form of a perceptual deficit for which the environment failed to compensate. He saw a circular relationship between the child's underlying perceptual handicaps which caused learning problems and the parental inabilities to appropriately compensate for and respond to the condition.

Developmental or Maturational Disorder as Etiologic

It was postulated in 1906 by Sherrington that lower organisms appeared to indicate near receptor dominance (touch, taste, and smell) and that higher organisms showed a preference for distance receptor dominance (vision and hearing). It was further shown by several authors (Schachtel, 1959; Zaporozhets, 1961) that there was a progressive development shift from the autocentric mode of perception (near receptors) to the allocentric mode of perception (distance receptors) in the normal development of an infant. As these psychotic children showed the preference for near receptors, a type which were ordinarily employed by younger children, a maturational or developmental disorder was proposed as etiologic. Thus, the behaviors noted were seen as normal behaviors for children at a younger age, but were inappropriate at later stages of development.

One of the strongest proponents of a developmental disorder as causative was Bender (1952). A maturational lag at the embryonic level was hypothesized, and this was thought to be responsible for all of the manifestations of the syndrome. Bender felt that diffuse encephalopathy was responsible for the maturational lag; however, other reasons such as environmental or physiological factors were postulated by other authors as inducing the developmental disorder. Bender's etiologic considerations were based on subjects whose diagnoses were not strictly infantile autism, thus,

her conclusions should be applied to several classifications of childhood psychoses.

Language Disorder as Etiologic

One of the most apparent and consistent disabilities in autism was the pervasive speech disorder. Several authors postulated that a language disorder in the form of impairing the comprehension of sounds was the basic abnormality in autism.

Rutter (1968, 1972) concluded that the symbolic, conceptual, or comprehensional use of language was the central disorder in autism and that this defect was sufficient to explain all of the behavioral manifestations of the syndrome. It was further explained that (Rutter & Bartak, 1971) the speech and language deficiency was almost assuredly a manifestation of autism and that one of the first symptoms noted was frequently the lack of response to sounds. The prognosis for further development in autism was determined first by the I.Q. level and secondly by the child's level of language skills (Rutter, 1967). He further stated that (Rutter, 1972) although there was organic brain disease or damage in some cases, it was not assuredly the cause of the cognitive defect in all of the cases. In conclusion, Rutter and Bartak (1971) suggested that "a central defect in the processing of symbolic and sequence information is likely to prove the basic defect, but the evidence is not yet available to decide conclusively between the different types of

cognitive or sensory disorders which have been postulated for autism (p. 27)."

The speech and language structure of autistics was reviewed by Savage (1968-69), and she noted that mutism was the most prominent speech disorder and echolalia was second. Pronomial reversal was also common and it was felt by some authorities that this was an indication of the child's lack of awareness of his personal identity. Some of the younger children were noticed to invent their own idiosyncratic language, and there was a considerable frequency of non-communicating and inappropriate speech. She reported that many of the children who did speak displayed articulation abnormalities, and a high percentage evidenced comprehension deficits.

Abnormal Physiological Arousal as Etiologic

An organism's ability to receive and be affected by environmental stimulation was in part determined by the level of physiological or cortical arousal. Thus, as the behavior of the autistics was shown to be quite stable, despite the remedial techniques employed, it was hypothesized that an inappropriate level of cortical arousal which did not maintain a state of normal alertness was responsible for the failure of the organism to respond to stimuli. The level of abnormal cortical arousal was suggested as chronically high (Hutt & Hutt, 1965), chronically low (Rimland, 1964), as well as levels of arousal that fluctuated between being either too high or too low (Ornitz & Ritvo, 1968).

Hutt and Hutt (1965) saw autism as an abnormality of physiological arousal because of their studies correlating stereotypes of autistic children with the desynchronized electroencephalography (EEG) activity. Their findings showed an increase of stereotypies in a more complex situation and in unfamiliar circumstances that correlated with desynchronization of the EEG. They postulated that the autistic child was in a continued state of high arousal and that the stereotypies were displacement mechanisms used to reduce and regulate the high arousal level.

Rimland (1964) proposed that "the secret of the veil of autism" was a disorder in the arousal system of the reticular formation and the lymphic system. This implied that the child's ability to relate new stimuli to previous experience was handicapped and, therefore, the child did not respond to his parents, as he failed to associate them with any previous enjoyable experiences. This postulated low level of physiological arousal did not allow the child to think in terms of concepts, symbols, analogies, or abstractions which left his perception of the world scattered and fragmented.

> Ornitz and Ritvo (1968) wrote that:
>
> Heightened awareness, hyperirritability, and obliviousness to external stimulation all may occur in the same child. All modalities of sensation may be involved. While auditory changes are most often noted, unusual perceptual

> aberrations may be seen in the visual, tactile, gustatory, olfactory, proprioceptive, and vestibular senses (p. 81).

They noted inadequate modulation of homeostatic regulation in the autistics sensory input which resulted in either too much or too little afferent stimulation. The children vacillated between hyperreactivity (hypersensivity) and hyporeactivity (hyposensitivity) to stimuli, which suggested underlying perceptual inconstancy which involved all of the sensory modalities. This inability to effectively modulate sensory input placed tremendous strains on the child's perception and handicapped his ability to respond to more than one stimulus modality at a time. The child was often overselective and responded to only one component of a stimulus complex that might include visual, auditory, and olfactory components (Ornitz & Ritvo, 1968).

Neurological or Cognitive Dysfunction as Etiological

Theories suggesting neurological damage ranged from specific to generalized statements, many of them viewing the organic dysfunction as interactive with other environmental or physiological factors. Rimland (1964) was quite specific in his designation of the nature and location of the cognitive impairment. He postulated that autistic children indicated an inborn capacity for high intelligence and that prenatal or noxious effects resulted in greater damage to a rapidly developing, highly differentiated

central nervous system. He suggested that damage to the reticular formation occurred as a result of hyperoxia, medically administered oxygen, or simply atmospheric oxygen due to the unusual vulnerability of the neuro-vasculature. The reticular formation extended from the upper end of the spinal cord into the hypothalmus through the central portion and sides of the thalmus and it was credited by French (1960) as the master control mechanism of the central nervous system. It was an area of diffuse neurons containing the 5th through the 12th cranial nerves which controlled the overall degree of CNS activity, including arousal and a portion of the mind's ability to attend to stimuli. Rimland (1968) suggested that this reticular damage impaired the child's ability to focus attention and ignore distractions.

Des Lauriers and Carlson (1969) cited an imbalance between the ascending reticular activating system (Arousal I) and the limbic reward system (Arousal II) which suppressed Arousal II. Thus, associations between novel and previously experienced stimuli could not be made due to the chronically high level of cortical arousal. Schopler (1965) also suggested the presence of inadequate reticular arousal mechanisms due to a constitutional deficiency as well as understimulating mothering.

Ruttenburg (1971) suggested an interactive theory and listed primary autism as a result of innate or congenital organic brain damage and secondary autism as a result of environmental stress. The autistic behaviors were considered

to be a developmental arrest at the preoral stage of psychosexual development in response to the perceived noxious or overwhelming mothering. The severity of the symptoms was determined by the degree of environmental stress or the organic damage.

Ward (1970) indicated three possible vulnerabilities in the autistic child: 1) an organically based, abnormally high stimulus barrier, 2) an organically based hypersensitivity to external stimuli, or 3) a nonstimulating, nonpatterned home environment. He also postulated the existence of an "organic" and a "psychogenic" subgroup division of autism.

Ornitz and Ritvo (1968) proposed that the autistic child's perceptual inconstancy was suggestive of a primary or secondary pathophysiologic organic brain dysfunction. In a later publication (Ornitz & Ritvo, 1976) they further implicated an organic basis of autism stating that "its symptoms are expressive of an underlying neuropathophysiological process that affects developmental rate; modulation of perception; language, cognitive, and intellectual development; and the ability to relate" (p. 609).

DeMyer, Barton, DeMyer, Norton, Allen, and Steele (1973) also supported a biologically based etiology of autism suggesting a static lesion occurring either during gestation or infancy. They listed possible reasons for the damage as a viral disease during or after gestation, birth trauma, malnutrition, oxygen lack, or generally any of the other known causes of brain damage.

MacCulloch and Williams (1971) stressed that more etiologic investigations should be directed toward the motor stereotypies which could indicate an abnormality along the afferent proprioceptive pathways from the limbs, trunk, or vestibular organs. MacCulloch and Sambrooks (1973) suggested neuronal damage, or a functional equivalent, at the head of the nucleus of the tractus solitarious which was functionally and spatially interconnected to the reticular formation. They contended that damage of this nature could disturb the perceptual processes and the level of cortical arousal as well as cause damage to the tenth nerve nucleus which could result in forms of under arousal such as immobility and failure to explore. They felt that simply the birth process or oxygenation during the last few weeks before birth could account for the damage incurred.

Several of the theorists suggested the probability of brain damage in at least a portion of the autistics, although they did not suggest neurological impairment as being globally etiologic. Bender (1952) proposed "diffuse encephalopathy" and later (1966, 1967) noted an embryonic maturational lag, especially in those functions innervated by the central nervous system. Hingtgen and Bryson (1971) felt that the present reserve of empirical data was congruent in many areas with postulated organic defects. They noted that the intellectual, perceptual, and language dysfunctions, as well as the maladaptive behaviors strongly

suggested the possibility of many forms of organic damage. Rutter (1967), in like manner, noted data which suggested the probability that some kind of organic brain lesion could account for at least a portion of the cases of autism.

Genetic, Metabolic, or Bio-Chemical Factors as Etiologic

Kanner (1943) wrote of the "inborn" nature of autism which suggested some form of constitutional vulnerability or malfunction as being partially causative of the disorder. Then, as early as 1953, Bakwin and Bakwin speculated that organic problems of a metabolic or neurochemical nature would later be discovered as etiologic of infantile autism.

Genetic and chromosomal aberrations were considered as etiologic of autism partially as a result of the suggested concordance of autism in monozygotic twins. Cytogenic studies indicated the presence of a long Y (male) chromosome in several of the autistic children (Judd & Mandell, 1968). Therefore, because there was a noted prevalence of males diagnosed as autistic, a need for chromosomal studies was proposed. Rimland (1965) suggested that autism might be genetically determined and Rutter and Bartak (1971) also considered the possibility of an autistic genotype. Treffert (1970) felt that autistic behaviors were the external manifestations of mutants of especially intelligent progenitors.

Efforts were made to link autism with a variety of biochemical and metabolic defects. Of primary concern in these studies were the neuronal communication of inhibition and excitation, the chemical basis of memory, and the

neurophysiological models of learning and adaptation. The chemicals of particular interest were those that appeared to influence the neuronal passage across the synaptic cleft in the adjacent nerve cells. Basic messages of this sort were controlled through biogenic amine neurotransmitters as well as through other hormonal neurotransmitters such as norepinephrine, dopamine, and serotonin. Cortisol, insulin, thyroxine, and growth hormone were also noted to modulate the activity of the brain. Specific chemical imbalances were suggested in the form of extremely high dopamine turnover rates and, relative to this, reduced serotonin turnover (Cohen, 1975).

Several authors attempted to align autism to schizophrenia by employing various types of chemical analyses. Bufotenin was found to be excreted in the urine of some autistics, a chemical similarity they shared with schizophrenics (Himwick, Jenkins, Fujimori, Narasimhachari, & Ebersole, 1972). Other suggested imbalances included indolamine metabolism, a decrease in urinary excretion of 5-hydroxy-indoleacetic acid in response to a tryptophan load (Sutton & Read, 1958), elevated blood serotonin levels (Schain & Freedman, 1961), and a tryptophan metabolism defect (Helley & Roberts, 1965). Magnesium imbalances were also investigated (Gittelman & Cleeman, 1969).

Because of the known relation of thyroid hormones to behavior and development, blood thyroxine levels were proposed to be deviant. It was suggested that, when thyroid

metabolism was reduced, there was a compensatory elevation in the synthesis of biogenic amines. Some autistic children were shown to demonstrate marked thyroxine lability, and swings from hypothyroidism to hyperthyroidism were noted within a span of only a few days (Cohen, 1975).

The lack of appropriate early affective development was considered by Despert (1971) to be significant in suggesting inadequate levels of a hormonal-type substance in autistics. He noted the lack of sexual drive characteristic of autistics in later life as support for his hypothesis.

In summary, Kanner's observation of early infantile autism as a uniquely observable clinical syndrome stimulated a variety of theoretical and observational postulations. Initially autism was felt to be a psychogenic disorder that resulted from the coldness and aloofness of the mother. In many of the theories the variable of a constitutionally vulnerable child was added. Quite commonly theorists noted the disturbed social relationships and the retreat from reality and attempted to classify autism as a type of schizophrenia. Yet, still other behavioral characteristics of autism prompted various etiologic considerations. The high incidence of subnormal intellectual functioning led to the philosophy that autism was merely a form of mental retardation and the language and receptive abnormalities were suggestive of possible perceptual disorders as etiologic. As some of the behaviors of autistic children were considered to be appropriate for children at younger ages but

inappropriate at later ages, a maturational or developmental disorder was suggested as causative. Language disorders were felt to be etiologic by several of the researchers, as the language disabilities were a prominent feature of the syndrome. Abnormal levels of physiological arousal as well as neurological or cognitive dysfunction were hypothesized as possible explanations of the disorder as a result of the abnormal EEG readings. And, finally, due to the high incidence of physiological abnormalities in the autistic children, genetic, metabolic, or bio-chemical factors were also put forth as possible etiologic considerations.

CHAPTER III

ANALYSIS OF THE THEORIES

In the analysis of the etiological theories of autism, the scientific, empirical studies were considered to be the most valuable means by which to support or refute the theories of causation. However, the behavioral, developmental, and clinical implications of the syndrome remained important. Not only did they provide observable correlates with which to relate the pragmatics of the theories, but they were the basis from which many of the empirical studies were formulated.

Empirical Evidence Related to Parental or Environmental Pathology as Etiologic

Several of the earlier studies revealed results which supported parental or environmental factors as etiologic. Lotter's (1967) epidemiological study of autistics indicated a higher proportion of parents who appeared to be above average in intelligence and of superior socioeconomic status. Franknoi and Ruttenberg (1968) also supported the presence of parental pathology as they reported, "We usually find evidence that the infant's unresponsiveness emerged in a much

less than optimal emotional climate" (p. 721). They noted that previously there had been no known cases of autism in the Japanese or Negro population, presumably because of the commonly warm, spontaneous child rearing practices in both of those cultures. However, they noted that more recently, in modern industrialized Japan as well as in the American middle class Negro population, increasing numbers of autistic children had been reported.

Many of the more recent studies did not support parental or environmental pathology as a precipitating factor in the development of autism. Rutter (1967) found no stereotype personality factors in the parents of autistics, and Wolff and Morris (1971) also failed to find any uniquely identifiable parental personality style. Furthermore, their personality profiles more often reflected normality rather than deviancy, and they did not support the presence of personality traits such as aloofness, obsessive-compulsiveness, perfectionisticness, detachment, or a lack of emotion. In a like manner, DeMyer, Pontius, Norton, Barton, Allen, and Steele (1972) failed to support either the parental or the parental-biological theories of causation. Ritvo, et al. (1971) found no significant differences in the parents' mean age at the time of the patient's birth, educational or occupational level, income, social class indices, or distribution.

It was pointed out by Chapman (1960) early in the literature that autism characteristically occurred in only

one child in a family. Thus, if the deviant behavior was reflective of the lack of appropriate parental affect and handling, then it should follow that the siblings would also display autistic traits or, at least, other types of emotional difficulties. However, Hingtgen and Bryson (1971) reported that, "there appears to be little or no evidence of parental psychopathology. On the contrary, they demonstrate low incidences of familial psychosis, broken homes, and sibling pathology, compared to control groups" (p. 37).

Ornitz (1973) also failed to support the theories of parental disturbances, but he went one step further in his explanation. He, rather, emphasized the emotional stress created in a family whose child was exhibiting such bizarre and unresponsive behaviors. It would seem appropriate that the lack of responsiveness in the child would cause frustration and depression in the family. Furthermore, the guilt caused by these assumptions of parental pathology only enhanced the feelings of frustration and blame in the families of autistics.

It would appear, assuming the gross behavioral abnormalities in autistics were caused by the parents, that psychotherapy could bring about some form of amelioration in the disturbances. However, this was not the case, as the outcome of the disorder was not altered by any form or duration of psychotherapeutic techniques (Rimland, 1964, Rutter, Greenfield, & Lockyer, 1967). And, finally, it would be difficult for a theory of parental or environmental

causation to account for many of the concomitant aspects of autism, such as the intellectual, bio-chemical, language, neurobiological, or perceptual deficits that had been conclusively identified in these children. It was noted, however, that optimal environmental factors could, in any affective disorder, help to modify the severity of the secondary handicaps (Rutter, 1974).

Empirical Evidence Related to a Type of Schizophrenia as Etiologic

The theory that autism was merely a variation of an early manifestation of schizophrenia was widely professionally accepted as late as approximately 1965. However, through the use of more abundant clinical and empirical evidence, autism was shown to be a distinct clinical entity rather than merely a variant of schizophrenia.

The age of onset was dissimilar, with autism beginning before the age of 30 months, and schizophrenia showing at least a period of normal development followed by a retreat into fantasy (Rutter, 1971). Statistics indicated that four out of five autistics never indicated any period of normal development (Rutter, 1972), whereas, the onset of childhood schizophrenia was noted as from three years to adolescence with the onset of adult schizophrenia occurring during adolescence or later (Ornitz & Ritvo, 1976). Also, autism was noted as predominantly a male syndrome while schizophrenia occurred about equally in males and females (Rutter, 1972). Also schizophrenia showed familial histories while autism did not.

Autism was extremely rare in the parents and siblings of autistic children, except for the previously mentioned concordance for monozygous twins, while schizophrenia occurred in approximately 10% of the parents of schizophrenia adolescents or adults (Rutter, 1972).

Schizophrenics notably displayed vivid and distorted fantasy lives, delusions, and halucinations, while autistics ordinarily cast a narrow focus of attention on stereotype physical activity (Ward, 1970; Eisenberg & Kanner, 1956). Kanner (1943) recognized from the beginning that autistics showed a persevering developmental arrest or fixation while schizophrenics indicated periods of normal development as well as periods of remission and regression.

Mental subnormality was noted in approximately 75% of the autistics (Halvelkova, 1968; Rutter, 1970), while mental subnormality was less frequently associated with schizophrenia. Other constitutional variants were noted by Goodwin, et al. (1971) in the form of a surprisingly significant number of biological abnormalities in autistics that were not found in schizophrenics. Rutter and Bartak (1971) summarized their views of the theory that autism was a type of schizophrenia by stating that:

> For many years autism was regarded as a particularly early manifestation of schizophrenia. It is now reasonably certain that this view is wrong. Autism and schizophrenia differ in terms of sex distribution, social background, family history

of schizophrenia, intellectual level, cognitive pattern, presence of delusions and hallucinations, and course of disorder (p. 24).

Empirical Evidence Related to Mental Subnormality as Etiologic

Although retardation and autism did coexist in three-fourths of the children, there remained one-fourth of the autistics who demonstrated stable intelligence test scores above the retarded range (Halvelkova, 1968; Rutter, 1970). Savage (1968-69) noted that as well as higher testable I.Q.'s, many more of the autistics did not display the vacant stare that was characteristic of the subnormal child. Further, subnormal children typically displayed flaccid muscle tone and poor coordination, but many of the autistics were graceful and skilled in their movements.

Lockyer and Rutter (1970) found that the intelligence profiles of the autistics varied somewhat from the profiles ordinarily shown by endogenous subnormals. Autistics indicated islets of intelligence, most of them with predictably high scores in certain consistent areas. Also, many retardates, although socially immature, did not display the gross deviancies in behavior that were so characteristic of the autistics.

Empirical Evidence Related to Perceptual Disorders as Etiologic

It was established that autistic children demonstrated

a multiplicity of perceptual disorders in various forms and in different modalities. Hermelin and O'Connor provided many clinical and experimental studies of autistic children and they concluded that psychotic (including autistic) children indicated more deficient visual discrimination than did severely subnormal children with commensurate performance intellectual functioning. They further suggested (Hermelin & O'Connor, 1964; Hermelin, 1965) that psychotic children failed to develop a sensory hierarchy. This was supported and further synthesized by Frith and Hermelin (1969) when they studied the performance of normal, subnormal, and autistic children in two perceptual-motor tasks. Under some conditions they were allowed to employ visual information, and under some conditions they were not. They concluded that autistic children made relatively less efficient use of the visual cues and relatively more of motor feedback than the children in the other groups. They noted this to be particularly the case with the developmentally more backward children.

Stimulus overselectivity was observed by Koegel and Wilhelm (1973), indicating that autistics responded to only a limited portion of a stimulus complex. The autistics primarily responded to only one modality (generally auditory or visual), while the normals responded to all three stimulus modalities (auditory, visual, and tactile cues). This stimulus overselectivity could account for many of the behaviors of the autistics. For example, selectively responding to

only one cue would deny the child the acquisition of appropriate affect through the use of the classical conditioning model. Another inference that the authors made was that the autistic children might have learned to recognize a person or a situation by only one of the many present cues. Therefore, if the child had learned to recognize his father by the presence of his glasses, it was understandable that the child became very upset if the glasses were removed.

Stroh and Buick (1964) attempted to explain the process of the way in which perceptual dysfunctions might lead to a disorder such as autism. They felt that, although the disturbances may not have been due to any inherent limitations of the perceptual apparatus, in the autistic child there was a partial or complete absence of external cues received which were necessary for normal perceptual functioning. Perceptual development was seen as a progression starting with movement, and with the integration of other modalities, a hierarchical development emerged which was characterized by the dominance of the distal sensory modalities. However, with autistics, this was not the case, as Goldfarb (1961, 1964) noted a significant usage of proximal receptors (smell, touch, and taste) over the distal receptors (hearing and vision).

Empirical Evidence Related to a Developmental or Maturational Disorder as Etiologic

The hierarchical development of humans was shown by many authors to progress in an orderly, specific manner. Each stage of development was dependent on the successful

mastery and completion of the previous stage or stages. The autistic children were shown to have many maturational or developmental lags or failures, one of the most serious being their preference for using proximal over distal receptors with which to explore their environment (Schachtel, 1959; Zaporozhets, 1961; Goldfarb, 1961, 1964). If a failure to mature properly at any stage of development did not allow later development to take place at all, or to occur properly, the subsequent abnormal development of the autistic child could be easily understood. If the autistics predominantly employed the use of tasting, touching, and smelling, while normals explored their environment primarily with the use of vision and hearing, the two groups would most certainly receive a vastly different comprehension of their environment. However, to explain the gross behavioral abnormalities of the autistics by stating only that they have a maturational or developmental lag seemed quite insufficient. Even Bender's (1952) suggestion of "diffuse encephalopathy" at the embryonic stage of development did not adequately explain the nature or cause of the failure of the development.

Empirical Evidence Related to a Language Disorder as Etiologic

Rutter (1972) stated his views on the language development of the autistic child when he wrote, "let these be summarized by stating that the evidence suggests that autism develops on the basis of a central disorder of cognition which involves the impairment of both the comprehension of

language and defects in the utilization of language or conceptual skills in thinking (p. 329)." Language abnormalities were, indeed, noted as both pervasive and severe in a large percentage of the autistics. Approximately one-third of the autistics developed no speech or language at all and they remained essentially mute throughout their lives (Chapman, 1960). Echolalia and perseveration in the use of language was also quite common in autistic children, ordinarily being associated with the reversal of pronouns (Chapman, 1960; Kanner, 1943; Wing, 1966a). When speech did develop, it was characteristically atonal and arrhythmic, with a lack of displayed inflection or emotion (Goldfarb, 1961). Many of the children also displayed other articulation disorders, which ranged from dyslalia (impaired power of speech) to articulatory apraxia (an inability to coordinate one's muscles and movements; usually due to a brain condition) (Savage, 1968-69).

Rutter, the most avid proponent of language disorders as the etiologic explanation of autism, presented a convincing discourse on the importance of language development in the autistic child. He noted that, other than the level of intelligence, language was the most important prognostic factor in autism (Rutter, 1968). It was shown that, even on tests which required the use of language, but did not require them to speak, autistics did poorly (Lockyer & Rutter, 1970; Rutter, 1968). Although many authorities felt that the child could speak but elected not to because of the social withdrawal

(Savage, 1968-69), Rutter contended that the language and speech abnormalities were primary and the withdrawal was secondary. Rutter further explained that inconsistency, both in response to sounds and in the production of speech, was characteristic of the aphasic child or a child with a developmental language disorder. O'Gorman (1967) also noted an inconsistent response or a failure of a response to sounds in autistic children. This response selectivity appeared at times to be toward certain people or certain types of stimuli only.

Tutter (1968) contended that the affective disorders in autistics could result from their inability to comprehend speech and gestures. This comprehension deficit would not allow them to understand the nuances and subtleties of abstract concepts, humor, or the expression of emotions. He further explained that all degrees of social withdrawal could be noted in people with a developmental language disorder involving a failure of the comprehension of sounds. He would explain the muteness exhibited by many autistics on this basis, also, because the comprehension of language must precede the speaking of the language.

Empirical Evidence Related to Abnormal Physiological Arousal as Etiologic

Autistics were postulated to have abnormal levels of cortical arousal in the form of chronically high arousal (Hutt & Hutt, 1965), chronically low arousal (Rimland, 1964), or alternating levels of arousal that were either too high or

too low (Ornitz & Ritvo, 1968). The levels of cortical arousal were primarily deduced from the children's behavior or from medically obtained EEG readings. The literature was inconsistent and contradictory in the presentation of the empirical results as well as the inferences from these results that related to the levels of physiological arousal.

Hutt and Hutt (1965) proposed that a dysfunction in the reticular formation resulted in a chronically high state of physiological arousal. They noted that when environmental stimulation increased, the stereotypies increased and the EEG became more desynchronised. They also cited the higher than usual dosage of medications or sedatives required by autistics as well as their high threshold for pain and auditory stimuli as further support for a chronically high level of arousal.

Ornitz and Ritvo (1968), however, cited their evidence for alternating levels of arousal by noting that if the stereotypies, agitation, excitation, and fearfulness indicated high arousal states, then the posturing, immobility, and unresponsiveness suggested low arousal states. Ornitz, Ritvo, Panman, Lee, Carr, and Walter (1968) showed that autistics under five years of age indicated significantly larger responses when amplitudes during eye-movement bursts were compared to ocular quiescent phases of Rapid Eye Movement (REM) sleep.

Hermelin and O'Connor (1968) found that the percentage of alpha rhythm in autistics, mongols, and normals was not significantly different for light stimulation. They did,

however, note a significantly higher state of arousal for autistics under the conditions of continuous noise. Kolvin, Ounsted, Humphrey, et al. (1971) and Hutt and Hutt, et al. (1965) noted the presence of unusually low voltage EEGs in autistics that were suggestive of hyperarousal. However, these studies were not confirmed by the results of Creak and Pampiglione (1969) and Hermelin and O'Connor (1968).

Empirical Evidence Related to Neurological or Cognitive Dysfunction as Etiologic

The early literature failed to support a neurological or cognitive dysfunction as etiologic, because the evidence could not be medically demonstrated in all of the autistics. However, further research indicated that even the most modern encephalography (EEG) or brain scan techniques could not prove with certainty that there was no neurological damage present. A positive EEG or brain scan indicated with certainty the presence of neurological impairment, but a negative reading meant only that there probably was no damage present. This uncertainty left quite a margin of doubt.

The EEG's of autistic children often revealed a non-specific abnormality with some cerebral dysrhythmia. Gubbay, Lobascher, and Kingerlee (1970) reported that over 50% of their autistic subjects indicated unequivocal neurological and electroencephalographic evidence of organic brain disease. The research showing the highest incidence of neurological dysfunction suggested that 50% to 80% of the autistic subjects indicated abnormal EEGs with focal or diffused spike,

slow wave, or paroxysmal spike and wave patterns (Hinton, 1963; Creak & Pampiglione, 1969; Ornitz, White, DeMyer, & DeMyer, 1964). In a more recent, well controlled study with complete EEG and neurological evaluations, a cumulative index was recorded on each child for a period of six-and-a-half years. The results indicated that 73.4% of the autistic children fell more than two standard deviations from the normal range, with 60% indicating grossly abnormal EEGs (DeMyer, et al., 1973). In further support of the high incidence of neurological involvement, Gittelman and Birch (1967) noted CNS pathology in 80% of their subjects. Several other EEG and neurological evaluations reported a lower incidence of abnormality (Ritvo, Ornitz, & Walter, 1970; Kolvin, Ounsted & Humphrey, 1971). Rutter and Lockyer (1967) suggested that 28% of their sample of psychotic children indicated probable brain damage and that another 25% indicated possible evidence of damage. Hutt, Hutt, Lee, and Ounsted (1964) reported that EEG readings on their autistic children at rest indicated a predominance of low voltage, fast, desynchronised waves. They further noted that a decrease in environmental stimulation produced a decrease in stereotypies and an increase in synchronisation of the EEG readings. Hutt, Hutt, Lee and Ounsted (1965) further reported the lack of dominant alpha rhythm in the EEGs of autistics.

The incidence of seizures increased as the children grew older, and some of the children who earlier had normal EEGs and neurological examinations subsequently developed

seizures. Between the ages of 11 and 19, 25% of a longitudinally studied group of autistic children first developed seizures (Rutter, et al., 1967; Rutter, Bartak, & Newman, 1971). Only a small minority of the autistic children with normal intelligence developed seizures, but a high percentage of the severely subnormal autistics began having seizures in adolescence (Rutter, 1974). In general, the severely mentally retarded autistics displayed overt neurological disorders more often than the autistics with higher intellectual functioning levels (Goldfarb, 1961; Rutter, 1970; Rutter, et al., 1971).

In 1964, Seller and Gold injected the cerebrospinal fluid of schizophrenic children into mice, and they noted that seizures occurred more quickly than when the mice were injected with control fluid (Seller and Gold, 1974). The later evidence (Gold, 1967) did not support the indications of more rapid seizure times; however, it did support the presence of epileptogenic factors in the serum of psychotic children.

As neurological impairment was not always medically demonstrable, "soft" neurological signs were examined. In approximately 40% to 75% of the cases, soft neurological signs were noted in the form of clumsiness, poor coordination, poor muscle tone, hypotonia, generalized hyperreflexia, hyperactive knee jerks, drooling, hyperkinesis, ankle clonus, and strabismis (Goldfarb, 1961; Hinton, 1963). Goodwin, Cowen, and Goodwin (1971) demonstrated the presence of other soft signs,

such as abnormal EEG seizures, symmetrical paroxysmal movements, delayed and uncertain laterality, stereotype gestures, impaired hearing and speech defects.

Twin studies reported almost a 100% rate of concordance for autism in monozygotic twins; however, the probability of dizygotic twins was higher for discordance for autism (Ornitz, 1976; Cohen, 1975). This further suggested the probability of a constitutional rather than an environmental cause of autism.

Empirical evidence supportive of an organic lesion in the dorsal brain stem, perhaps at the head of the nucleus of the tractus solitarious, was presented by Ornitz and Ritvo (1968) and Ritvo, Ornitz, Eviatar, Markham, Brown, and Mason (1969). Decreased postrotatory nystagmus was noted in the autistics when they were tested with their eyes open, and commensurate results were obtained despite varying factors in the children, such as mutism, echolalia, normal speech or thought disorders. The shorter durations of nystagmus were noted only with the presence of the multiple stimuli of rotation plus visual stimuli. This area of the dorsal brain stem was expressed as a juxtaposition for auditory, vestibular, and somatic afferent pathways. A lesion in this area was expected to affect the heart rate modulation, and MacCulloch and Sambrooks (1973) found that while the mean heart rate of the autistic and control groups was not significantly different, the autistics showed greater heart rate variability. When Bonvallet and Allen (1963)

induced damage to the nucleus of the tractus solitarious of a cat, this area's far-reaching regulatory function on the reticular formation was demonstrated. These results suggested that the cat's reticular formation was underdamped which caused the cat to over-respond to extroceptive and introceptive stimuli.

An increased incidence of prenatal and perinatal complications was noted in several studies of psychotic children (Pollack & Woerner, 1966; Whittam, Simon, & Mitler, 1966). This was noted to be especially the case in male infants (Taft & Goldfarb, 1964). The highest incidence of perinatal abnormality was reported by MacCulloch and Williams (1971), who indicated that the autistic group showed twice the number of complications as the two control groups combined. This suggested that evidence of possible perinatal complications was consistent with the theory of neurological involvement as etiologic of autism.

The evidence in the literature that failed to support a neurological or cognitive dysfunction as etiologic of autism primarily expressed the doubt that adequate information was available to make a judgment.

It was widely accepted that autism and neurological impairment frequently coexisted in the same child; however, several authors presented the issue that many children who had been diagnosed as brain damaged did not develop autism. It was further noted that functional brain damage could not be medically demonstrated in at least half of the autistic

children (Rutter, 1968). Rutter (1969) went on to say that the term brain damage was too generalized to lend adequate understanding to the developmental aspects of the syndrome.

Although it was hypothesized that autistic behaviors resulted from ongoing brain damage, it must also be remembered that the autistic conditions in a child could result in the appearance of brain damage. Rutter (1969) emphasized that profound environmental deprivation could cause organic dysfunction in the child which, on the surface, could simulate brain damage.

Kanner and Lesser (1958) contended that autism showed no clinical resemblance to the course of any recognized organic condition. They also noted that autistic children were not characteristically physically ill as were children with degenerative organic diseases.

Empirical Evidence Related to Genetic, Metabolic, or Bio-Chemical Factors as Etiologic

The observed concordance of autism in homozygous twins led researchers to more closely examine possible congenital or physiological causes of autism. Only one case of homozygous twins in the literature indicated discordance for autism with all of the other cases that adequately established homozygocity being concordant for the syndrome. However, this one established discordant pair showed perinatal trauma and anoxia in the child with the autistic behaviors, and he did not appear to be a classic case of autism (Ornitz, Ritvo, & Walter, 1965).

Very few well-controlled chromosome studies of autistics were noted in the literature. Book, Nichtern, and Gruenberg (1963) studied a group of psychotic children, some of whom were autistic, and they noted no significant consistent chromosomal abnormalities in the children. Judd and Mandell (1968) initially noted a long Y chromosome in three of their autistic subjects, and they investigated this further because of the noted higher prevalence of autism in males. The normal father of two of the autistics also demonstrated long Y chromosomes, and because the father was in good physical and emotional health, the long Y chromosome appeared simply to be an inherited trait not associated with autism. The authors concluded that, although the twin studies suggested possible hereditary factors in autism, they could not be demonstrated with current cytogenetic methodology.

Studies of the families of autistics indicated a very low incidence of major mental illnesses in the parents and siblings (Creak & Ani, 1960; Kanner & Lesser, 1958; Rutter, 1965), which did not support a genetic basis for autism. However, as no cases of children that had been born to autistics were known, it was difficult to refute this hypothesis merely because of the low rate of autism in parents and siblings.

Biogenic amines, which were known mood regulators, were studied in relation to autism as well as to other forms of psychosis. Fluctuations in the levels of metabolites of biogenic amines found in the urine and cerebrospinal fluid suggested that depression was associated with decreased

availability of biogenic amines and that mania was associated with increased levels. However, through experimentation with rats, it was shown that the fluctuations in the biogenic amine metabolism could follow behavioral or environmental changes. Therefore, the altered biogenic amine levels could be either causative of autism or merely a result of the abnormal affect and behaviors (Cohen, 1975).

Metabolic imbalances were noted in several forms in autistic children and the degree of the deviancy of the imbalances appeared to correlate with the severity of the autistic behaviors. Bufotenin was found in the urine of autistic children but was not found in the urine of control group children by several researchers (Himwich, et al., 1972; Narasimhachari & Himwich, 1973). Also, deviations in the pituitary gland functions were noted in the form of reduced secretions of gonadotropin, corticotropin, and thyrotropin (Brambilla, Viana, & Rossotti, 1969). Other metabolism disturbances were noted in a high proportion of hospitalized autistic children showing high concentrations of lead in the body. Conclusions were not drawn, however, as to whether the abnormal concentrations of lead were due to ingestion or to a lead metabolism defect. Autistic children were also shown to have impaired glucose tolerance as well as impaired insulin tolerance, which was suggestive of an abnormality in the pituitary supra-renal system in some of the children. Magnesium imbalances which were thought by some to produce abnormal behavior, were found by Gittelman and Cleeman (1969)

in normal levels in psychotic children. However, a tryptophan metabolism defect was found by Heeley and Roberts (1965) in autistics who were deviant from an early age but this condition was not noted in children who regressed into autism at a later age. Cohen (1975) noted a small group of autistic children who displayed extremely high dopamine turnover rates and, both absolutely, and relative to dopamine, reduced serotonin turnover. These children reportedly had the highest levels of stereotypic behavior. Further support for a metabolic cause for autism was the evidence of depressed growth and skeletal development in autistic, and also generally in psychotic, children (Sutton, 1964; Simon & Gillies, 1964).

Thyroxine lability was studied, and it was noted that the children with the highest cerebrospinal fluid concentrations of biogenic amine metabolites were generally those with the lowest levels of serum thyroxine. Hypothyroidism was associated with lethargy and depression, and hyperthyroidism was ordinarily associated with irritability and hyperresponsivity. In other words, the thyroid hormone notably helped in the fine tuning of the nervous system by sensitizing postsynaptic membranes to the effect of biogenic amines. Thus, quite an effect could be expected in the form of vast, fluctuations in consciousness and adaptation in cases of recognized thyroxine instability (Cohen, 1975).

Goodwin, et al. (1971) designed a series of investigations and medical diagnoses of autistic children because of

the gastrointestinal disorders observed in the children. They concluded that there was a fundamental neurobiological dysfunction in autistic children with a possible correlation to malabsorption and food sensitivities. As the most significant result of their experiments, they noted a transcephalic direct current (TCDC) abnormality known as "DC bursts" (p. 60) in autistic children. This systematic instability would relate to orienting reality structure, and various higher intellectual functions.

Drug studies with autistic children indicated that they ordinarily required much higher doses of medications than did normal children (Cohen, 1975; Hingtgen and Bryson, 1971). Stimulants reportedly exacerbated the disorder while haloperidol and the activating phenothiazines were notably beneficial. Haloperidol was listed as a potent inhibitor of dopamine activity. These drugs usually resulted in a decrease in stereotypic behavior, an increase in attention, and a better regulation of activity. In optimum cases, the social responsiveness improved, the child was possibly more interested in the use of language, and modulation of over-arousal, easily evoked agitation, and the insistence on the perseveration of sameness occurred (Cohen, 1975). Hingtgen and Bryson (1971) concluded, however, that drug studies, although not very encouraging to date, provided further information suggesting a possible etiological role for neurobiological factors related to autism.

CHAPTER IV

SUMMARY, CONCLUSIONS AND IMPLICATIONS

Summary

Early Infantile Autism was delineated as a distinct clinical entity which was present from birth or before the age of 30 months. It appeared to be present in approximately 2 per 10,000 school children to approximately 4.5 per 10,000 school children, and the prognosis for recovery was dim. No known treatment program significantly altered the natural history of the syndrome, and approximately two-thirds to three-fourths of the children remained functionally mentally retarded throughout their lives.

The behaviors that were noted as universal to and specifically characteristic of autism were listed as a profound and general failure to develop social relationships, language retardation with impaired comprehension, echolalia, and pronomial reversal, and ritualistic or compulsive phenomena. The bizarre and maladaptive behaviors manifest in this syndrome rendered the autistic child unable to function in a normal environment. In most cases milieu or custodial placement, therefore, became necessary.

The literature was replete with theories postulating the cause of autism and the disperse nature of the theories brought about a need to review and analyze them in several general classifications. Parental or environmental pathology, a type of schizophrenia, mental subnormality, perceptual disorders, language disorders, abnormal levels of physiological arousal, neurological or cognitive dysfunction, and genetic, metabolic, or bio-chemical factors were clustered as proposed etiologic agents.

Conclusions

This study was designed to investigate the etiological theories of autism as they were presented in the literature and to analyze each theory as it related to the available empirical data. As there had not been a professional consensus of opinion on the etiology of autism, a need for independent, comprehensive, continuing efforts to analyze the related data was present. The procedures associated with the collection of the data involved the analysis of available and accessible published materials located in educational, psychological, and medical libraries and agencies.

The data were presented and analyzed in an attempt to answer the following questions:

1) What existing theories postulated parental or environmental pathology as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

The theories of parental or environmental pathology postulated that the children were able to feel the emotional coldness of the parents (or particularly the mother) and they retired from the rejection and coldness, thus,withdrawing into a world of fantasy and inanimate objects. Although environmental factors were noted as having an effect on the secondary handicaps of autism, these theories did not adequately account for the intellectual, bio-chemical, language, neurobiological, or perceptual deficits in these children.

2) What existing theories postulated a type of schizophrenia as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

Prior to the late 1960's one of the most widely accepted philosophies on the etiology of autism was that it was an early manifestation of adulthood schizophrenia. This hypothesis was not supported by the literature as it was shown that autism and schizophrenia differed in terms of sex distribution, social background, the familial history of schizophrenia, level of intelligence, and the presence of delusions and hallucinations.

3) What existing theories postulated mental subnormality as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

As 75% of the autistics were shown to function on a mentally retarded level throughout their lives, it was

suggested that autism was simply a form of mental subnormality. The literature failed to support these hypotheses, however, because approximately 25% of the autistics functioned intellectually above the retarded range, with intelligence profiles differing significantly from the profiles of the retardates. Also, there were many retarded children who, although socially immature, did not develop autistic behaviors.

4) What existing theories postulated perceptual disorders as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

Autistic children were shown to have a multiplicity of perceptual disorders in the auditory, verbal, visual, and affective areas. It was felt that the autistic received a distorted, inaccurate view of reality and this made it impossible for him to relate to others in a normal way. The literature supported the presence of perceptual aberrations in the autistics and indicated that further study in this area was warranted.

5) What existing theories postulated developmental or maturational disorders as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

The literature suggested that the development of autistics did not progress in the proper hierarchical manner as was shown to be the case in normal development. However, a maturational failure did little to explain the gross behavioral abnormalities of the autistics and it failed to

adequately identify the cause of the maturational failure.

6) What existing theories postulated language disorders as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

Language abnormalities were demonstrated in a large percentage of children in the form of muteness, echolalia, the reversal of pronouns, and the comprehension of speech and language. It was shown that apart from the level of intelligence, language was the single most important prognostic factor in autism. Therefore, further study appeared to be needed in this area.

7) What existing theories postulated abnormal levels of physiological arousal as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

Levels of cortical arousal that were chronically high, chronically low, and levels that fluctuated between too high and too low, were all postulated as being etiologic of autism. The studies in this area were both inconclusive and contradictory and further well controlled empirical attempts were needed to support or fail to support these theories.

8) What existing theories postulated neurological or cognitive dysfunction as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

Positive EEGs were noted by different authors in the range of 28% to 80% of the autistics. It was further noted

that approximately 25% of the children with no known brain damage developed seizures between the ages of 11 and 19. Soft neurological signs were noted in a high percentage of the autistics as well as an increased incidence of prenatal and perinatal complications. The empirical evidence in the literature strongly supported the hypotheses of neurological or cognitive impairment as etiologic in at least a portion of the autistics.

9) What existing theories postulated genetic, metabolic, or bio-chemical factors as etiologic of early infantile autism and what empirical data supported or failed to support these hypotheses?

Chromosomal, metabolic, and bio-chemical theories of autism became more prominent as medical science began noting constitutional and chemical abnormalities in their autistic subjects. Although all chromosome studies proved to be negative, the metabolic and bio-chemical variants noted in the autistic children supported a significant need for further empirical and medical research in this area.

In this investigation, empirical and observational data was presented supporting or failing to support each cluster of theories. The available literature demonstrated a lack of support for the hypotheses of parental or environmental pathology, a type of schizophrenia, mental subnormality and genetic abnormalities as plausible etiologic considerations. Moderate support was given to the propositions of perceptual disorders, language disorders, and abnormal levels

of physiological arousal as etiologic agents. However, the majority of the recent clinical and empirical information appeared to implicate the theories of neurological or cognitive dysfunction, and metabolic, or bio-chemical abnormalities as more hopeful explanations for the syndrome of autism.

In the final analysis, further progress in this field must be dependent on the application of more extensive and sophisticated evaluative procedures for determining physiological development in children. Comparisons of the physiological data and characteristics of the normals and the autistics must be analyzed in order to properly isolate the deviant characteristics of the autistics' physiological make-up.

Implications for Further Research

The lack of professional consensus on the etiology of autism and the inadequate and unfruitful therapeutic methodology revealed a need for etiologically-based treatment programs. Further research designed to study the autistics' perceptual disorders, language development, and levels of physiological arousal appeared to be warranted. More importantly, attempts to examine the neurological and cognitive aspects of the disorder as well as the metabolic and biochemical aspects were clearly needed. The importance of the early recognition and diagnosis of autism indicated a need for greater awareness of this syndrome in parents, educators, psychologists, and doctors. Through the use of

content analysis, the interrelationships and compatibilities of the seemingly diverse etiological theories could be examined. Thus, the confirmation or refutation of the etiological concepts could be presented in co-existing, rather than mutually exclusive, formulations. In conclusion, the mounting evidence of physiological or constitutional abnormalities recently discovered in the autistic children, exposed the probability that medical technology must be employed to further the advancement in this field. Behavioral techniques, psychoanalysis, and educational rehabilitative programs all proved to be ineffective in the amelioration of the syndrome. Therefore, the medical profession appeared to provide the only viable means with which to further explore the complexities of the etiology of autism.

REFERENCES

Allen, J., DeMyer, M.K., Norton, J.A. Intellectuality in parents of psychotic, subnormal and normal children. Journal of Autism and Childhood Schizophrenia, 1971, 1, 311-326.

Anthony, E.J. An experimental approach to the psychopathology of childhood: Autism. British Journal of Medical Psychology, 1958, 31, 211-225.

Bakwin, H., & Bakwin, R.M. Clinical management of behavior disorders in children. Philadelphia: W.B. Saunders Company, 1953.

Bender, L. The concept of plasticity in childhood schizophrenia. In P.H. Hoch & J. Zubin (Eds.), Psychopathology of Schizophrenia. New York: Grune & Stratton, Inc., 1966.

Bender, L. Childhood schizophrenia: A review. Journal of Hillside Hospital, 1967, 16, 10-22.

Bender, L. & Freedman, A.M. A study of the first three years in the maturation of schizophrenic children. Quarterly Journal of Childhood Behavior, 1952, 4, 245-272.

Bergman, P., & Escalona, S.K. Unusual sensitivities in very young children. The Psychoanalytic Study of the Child. New York: International Universities Press, 1949.

Bettelheim, B. The empty fortress. New York: Free Press, 1967.

Bleuler, E. Dementia praecox or the group of schizophrenias. New York: International Universities Press, 1950.

Book, J.A., Nichtern, S., & Gruenberg, E. Cytogenetical investigations in childhood schizophrenia, Acta Psychiatrica of Scandinavia, 1963, 39, 309-323.

Brambilla, F., Viani, F., & Rossotti, U. Endocrine aspects of child psychoses. Diseases of the Nervous System, 1969, 30, 627-632.

Chapman, A.H. Early infantile autism. AMA Journal of Diseases of Children, 1960, 99, 97-100.

Clancy, H., Dugdale, A., & Rendle-Short, J. Diagnosis of infantile autism. Developmental Medicine and Child Neurology, 1969, 11, 432-442.

Cohen, Donald J. Childhood autism and atypical development. Taboroff Memorial Lecture, 1975. University of Utah School of Medicine, 1975.

Creak, M., & Ini, S. Families of psychotic children. Journal of Child Psychology and Psychiatry, 1960, 1, 156-175.

Creak, M., & Pampiglione, G. Clinical and EEG studies on a group of 35 psychotic children. Developmental Medicine and Child Neurology, 1969, 11, 218-227.

DeMyer, M.K., Barton, S., MeMyer, W.E., Norton, J.A., Allen, J., & Steele, R. Prognosis in autism: A followup study. Journal of Autism and Childhood Schizophrenia, 1973, 3 (3), 199-246.

DesLauriers, A.M., & Carlson, C.F. Your child is asleep: early infantile autism. Homewood, Ill.: The Dorsey Press, 1969.

Eisenberg, L. Autistic children in adolescence. American Journal of Psychiatry, 1956, 112, 607-612.

Eisenberg, L., & Kanner, L. Early infantile autism: 1943-1955. American Journal of Orthopsychiatry, 1956, 26, 556-566.

Eisenberg, L. The course of childhood schizophrenia. AMA Archives of Neurology and Psychiatry, 1957, 78, 69-83.

Eisenberg, L. The classification of childhood psychosis reconsidered. Journal of Autism and Childhood Schizophrenia, 1972, 2 (4), 338-342.

Ferster, C.B. The repertoire of the autistic child in relation to principles of reinforcement. In L. Goilschalk & A.H. Auerbach (Eds.), Methods of research in psychotherapy, New York: Appleton-Century-Crofts, 1966.

Ferster, C. Remarks made at workshop: Multidisciplinary approach to the study and treatment of infantile autism II. Annual meeting, American Orthopsychiatric Association, Washington, 1967.

Franknoi, J. Major defense configurations and identity patterns in mothers of a group of autistic children. Presented at workshop: Multidisciplinary Approach to the Study and Treatment of Infantile Autism II. Annual meeting, American Orthopsychiatric Association, Washington, 1967.

Franknoi, J. Theoretical implications of therapeutic results achieved with a group of autistic children. Presented to the Seventh International Congress on Mental Health, London, 1968.

Franknoi, J., and Ruttenberg, B.A. Formulation of the dynamic economic factors underlying infantile autism, Journal of the American Academy of Child Psychiatry, 1971, 10 (4), 713-738.

French, J.D. The reticular formation. In J. Field, H.W. Magoun, & V.E. Hall (Eds.), Handbook of physiology, Washington, D.C.: American Physiological Society, 1960.

Frith, U., & Hermelin, B. The role of visual and motor cues for normal, subnormal, and autistic children. Journal of Child Psychology and Psychiatry, 1969, 10, 153-163 (printed in Great Britain).

Gittelman, M., & Birch, H.G. Childhood schizophrenia: Intellect, neurologic status, perinatal risk, prognosis, and family pathology. Archives of General Psychiatry, 1967, 17, 16-25.

Gittelman, M., & Cleeman, J. Serum magnesium level in psychotic and normal children. Behavioral Neuropsychiatry, 1969, 1, 51-52.

Goldfarb, W. Receptor preferences in schizophrenic children. Archives of Neurology and Psychiatry, 1956, 76, 643-652.

Goldfarb, W. Childhood schizophrenia. Cambridge, Mass.: Harvard University Press, 1961.

Goldfarb, W. An investigation of childhood schizophrenia. Archives of General Psychiatry, 1964, 11, 620-634.

Goodwin, M.S., Cowen, M.A., & Goodwin, T.C. Malabsorption and cerebral dysfunction: A multivariate and comparative study of autistic children. Journal of Autism and Childhood Schizophrenia, 1971, 1 (1), 48-62.

Gubbay, S.S., Lobascher, M., & Kingerlee, P. A neurological appraisal of autistic children: Results of a Western Australian survey. Developmental Medicine and Child Neurology, 1970, 12, 422-429.

Havelkova, M. Follow-up study of 71 children diagnosed as psychotic in preschool age. American Journal of Orthopsychiatry, 1968, 38, 846-857.

Heeley, A.F., & Roberts, G.E. Tryptophan metabolism in psychotic children. Developmental Medicine and Child Neurology, 1965, 7, 46-49.

Hermelin, B., & O'Connor, N. Effects of sensory input and sensory dominance on severely disturbed, autistic children and on subnormal controls. British Journal of Psychology, 1964, 55, 201-206.

Hermelin, B., & O'Connor, N. Measures of the occipital alpha rhythm in normal, subnormal, and autistic children. British Journal of Psychiatry, 1968, 144, 603-610.

Himwich, H., Jenkins, R., Fugimorli, M., Narasimhachari, N., & Ebersole, M. A biochemical study of early infantile autism. Journal of Autism and Childhood Schizophrenia, 1972, 2 (2), 114-126.

Hinton, G.G. Childhood psychosis or mental retardation: A diagnostic dilemma - II. Pediatric and neurological aspects. Canadian Medical Association Journal, 1963, 89, 1020-1024.

Hingtgen, J.N., & Bryson, C.Q. Recent developments in the study of early childhood psychoses: Infantile autism, childhood schizophrenia, and related disorders. Rockville, Md.: National Institute of Mental Health (DHEW Publication No. HSM 71-9062), 1971.

Hutt, C., Hutt, S.J., Lee, D., & Ounsted, C. Arousal and childhood autism. Nature, London, 1964, 204, 908-909.

Hutt, S.J., Hutt, C., Lee, D., & Ounsted, C. A behavioral and electroencephalographic study of autistic children. Journal of Psychiatric Research, 1965, 3, 181-97.

Judd, L., & Mandell, A. Chromosome studies in early infantile autism. Archives of General Psychiatry, 1968, 18, 450-457.

Kanner, L. Autistic disturbances of affective contact. Nervous Child, 1943, 2, 217-250.

Kanner, L. Early infantile autism. The Journal of Pediatrics, 1944, 25, 211-217.

Kanner, L. To what extent is early infantile autism determined by constitutional inadequacies? Research Publication of the Association for Nervous and Mental Diseases, 1954, 33, 378-385.

Kanner, L. Childhood psychosis: A historical overview. Journal of Autism and Childhood Schizophrenia, 1971, 1, (1), 14-19.

Kanner, L. Follow-up study of eleven autistic children originally reported in 1943. Journal of Autism and Childhood Schizophrenia, 1971, 1, 112-145.

Kanner, L.& Eisenberg, L. Notes on the follow-up studies of autistic children. In P.H. Hoch, & J. Zubin (Eds.) Psychopathology of Childhood. New York: Grune & Straton Inc., 1955.

Kanner, L. & Lesser, L. Early infantile autism. Pediatric Clinics of North America, 1958, 3, 711-730.

Kirk, R.V. Perceptual defect and role handicap: Missing links in explaining aetiology of schizophrenia. British Journal of Psychiatry, 1968, 114, 1509-1521.

Koegel, R.L., & Wilhelm, H. Selective responding to the components of multiple visual cues by autistic children. Journal of Experimental Child Psychology, 1973, 15, 442-453.

Kolvin, I., Ounsted, C. & Humphrey, M. Six studies in the childhood psychoses. British Journal of Psychiatry, 1971, 118, 381-419.

Kozloff, Martin A. Reaching the autistic child: A parent training program. Champaign, Ill.: Research Press, 1973.

Levine, M., & Olson, R.P. Intelligence of parents of autistic children. Journal of Abnormal Psychology, 1968, 73, 215-17.

Lockyer, L., & Rutter, M. A five-to-fifteen-year followup study of infantile psychosis. 4. Patterns of cognitive ability. British Journal of Social and Clinical Psychology, 1970, 152-163.

Lotter, V. Epidemiology of autistic conditions in young children. I. Prevalence. Social Psychiatry, 1966, 1, 124-137.

Lotter, V. Epidemiology of autistic conditions in young children. II. Some characteristics of the parents and children. Social Psychiatry, 1967, 1, 163-173.

MacCulloch, M.J. & Sambrooks, J.E. Concepts of autism: A review. Australian Pediatric Journal, 1973, 9, 237-245.

McDermott, J.R., Harrison, S.I., Schroger, J. Social class and mental illness in children--the question of childhood psychosis. American Journal of Orthopsychiatry, 1967, 37, 548-557.

Mahler, M.S. On child psychosis and schizophrenia: Autistic and symbiotic infantile psychoses. Psychoanalytic Study of the Child, New York: International University Press, 1952, 1, 286-305.

Narasimhachari, N., & Hemwich, H. GC-MS identification of bufotenin in urine samples from patients with schizophrenia or infantile autism. Life Sciences, 1973, 12 (II), 475-478.

O'Connor, N. & Hermelin, B. Measures of distance and motility in psychotic children and severely subnormal controls. British Journal of Social and Clinical Psychology, 1964, 3, 29-33.

O'Connor, N. & Hermelin, B. Sensory dominance in autistic imbecile children and controls. Archives of General Psychiatry, 1965, 12, 99-103.

O'Connor, N. & Hermelin, B. Visual analogies of verbal operations. Language and Speech, 1965, 8, 197-207.

O'Gorman, G. The nature of childhood autism. London: Butterworth, 1967.

Ornitz, E.M., Ritvo, E.R., & Walter, R.D. Dreaming sleep in autistic and schizophrenic children. American Journal of Psychiatry, 1965, 122, 419-424.

Ornitz, E.M., & Ritvo, E.R. Perceptual inconstancy in early infantile autism. Archives of General Psychiatry, 1968, 18, 76-98.

Ornitz, E.M., Ritvo, E.R., Panman, L.M., Lee, V.H., Carr, E.M., & Walter, R.D. The auditory evoked response in normal and autistic children during sleep. Electroencephalography and Clinical Neurophysiology, 1968, 25, 221-230.

Ornitz, E.M. & Ritvo, E.R. The syndrome of autism: A critical review. American Journal of Psychiatry, 1976, 133 (6), 609-621.

Pollack, M., & Woerner, M.G. Pre- and peri-natal complications and "childhood schizophrenia": A comparison of five controlled studies. Journal of Child Psychology and Psychiatry, 1966, 7, 235-242.

Rank, B. Adaptation of the psychoanalytic techniques for the treatment of young children with atypical development. American Journal of Orthopsychiatry, 1949, 19, 130-139.

Rimland, B. Infantile autism. New York: Appleton-Century-Crofts, 1964.

Rimland, B. On the objective diagnosis of infantile autism. Acta Paedopsychiatrica, 1968, 35, 146-161.

Ritvo, E.R., Ornitz, E.M., Eviator, A., Markham, C.H., Brown, M.B., & Mason, A. Decreased postrotatory nystagmus in early infantile autism. Neurology, 1969, 19, 653-658.

Ritvo, E.R., Ornitz, E.M., & Walter, R.D. Correlation of psychiatric diagnoses and EEG findings: A double-blind study of 184 hospitalized children. American Journal of Psychiatry, 1970, 126, 988-996.

Ritvo, E.R., Cantwell, D., Johnson, E., Clements, M., Benbrook, F., Slagle, S., Kelly, P., & Ritz, M. Social class factors in autism. Journal of Autism and Childhood Schizophrenia, 1971, 1, (3), 297-310.

Rothenberg, R.E., MD, FACS. The New American Medical Dictionary and Health Manual (Third Revised Edition) Signet, Signet Classics, Mentor, Plume, and Meridian Books, 1975.

Ruttenburg, B.A. A psychoanalytic understanding of infantile autism and its treatment. In D.W. Churchill, G.D. Alpern, & M. DeMyer (Eds.) Infantile Autism: Proceedings of the Indiana University Colloquium, 1968. Springfield, Ill.: C.C. Thomas, Publisher, 1971.

Rutter, M. The influence of organic and emotional factors on the origins, nature, and outcome of childhood psychosis. Developmental Medicine and Child Neurology, 1965, 7, 518-528.

Rutter, M. Prognosis: Psychotic children in adolescence and early adult life. In J.K. Wing (Ed.) Early childhood autism, clinical, educational, and social aspects. London: Pergamon Press, Inc., 1966.

Rutter, M. Organic brain damage, hyperkinesis, and mental retardation. Working paper for the WHO Third Seminar on Psychiatric Diagnosis, Classification, and Statistics. La Psychiatrie de l'Enfant, (in press), 1967.

Rutter, M. Psychotic disorders in early childhood. In A. Copper & A. Walk (Eds.) Recent developments in schizophrenia: A symposium. London: Royal Medico-Psychological Association, 1967.

Rutter, M. Concepts of autism: A review of research. Journal of Child Psychology and Psychiatry, Great Britain: Pergamon Press, 1968, 9, 1-25.

Rutter, M. Autistic children: Infancy to adulthood. Seminars in Psychiatry, 1970, 2, 435-450.

Rutter, M. The description and classification of infantile autism. In D.W. Churchill, G.D. Alpern, & M. DeMyer (Eds.) Infantile Autism: Proceedings of the Indiana University Colloquium, Springfield, Ill.: C.C. Thomas, 1971.

Rutter, M. Childhood schizophrenia reconsidered. Journal of Autism and Childhood Schizophrenia, 1972, 2 (4), 315-337.

Rutter, M. The development of infantile autism. Psychological Medicine, 1974, 4, 147-163.

Rutter, M., & Bartak, L. Causes of infantile autism: Some considerations from recent research. Journal of Autism and Childhood Schizophrenia, 1971, 1 (1), 20-32.

Rutter, M., Bartak, L., & Newman, S. Autism--A central disorder of cognition and language. In Infantile autism: concepts, characteristics, and treatment. London: Churchill, 1971.

Rutter, M., Greenfeld, D., Lockyer, L. A five-to-fifteen-year follow-up study of infantile psychosis-II. Social and behavioral outcome. British Journal of Psychiatry, 1967, 113, 1183-1200.

Savage, Valerie A. Childhood autism: A review of the literature with particular reference to the speech and language structure of the autistic child. British Journal of Disorders of Communication, 1968-69, 3-4, 75-88.

Schachtel, E. Metamorphosis. New York: Basic Books, 1959.

Schain, R.J., & Freedman, D.X. Studies on 5-hydroxyindole metabolism in autistic and other mentally retarded children. Journal of Pediatrics, 1961, 58, 315-320.

Schopler, E. Early infantile autism and receptor processes. Archives of General Psychiatry, 1965, 13, 327-335.

Seller, M.J., & Gold, S. Seizures in psychotic children. The Lancet, 1964, 1, 1325.

Sherrington, C.S. The integrative action of the nervous system. London: Cambridge University Press, 1906.

Stroh, G., & Buick, D. Perceptual development and childhood psychosis. British Journal of Medical Psychology, 1964, 37, 291-299.

Sutton, H.E., Read, J.H. Abnormal amino acid metabolism in a case suggesting autism. American Journal of Diseases of Children, 1958, 96, 23-28.

Taft, L.T., & Goldfarb, W. Prenatal and perinatal factors in childhood schizophrenia. Developmental Medicine and Child Neurology, 1964, 6, 32-46.

Treffert, D.A. Epidemiology of infantile autism. Archives of General Psychiatry, 1970, 22, 431-438.

Van Krevelen, D.A. Early infantile autism. Acta Paedo-psychiatrica, 1952, 19, 91-97.

Ward, A.J. Early infantile autism: Diagnosis, etiology, and treatment. Psychological Bulletin, 1970, 73, 350-362.

White, P.T., DeMyer, W., & DeMyer, M. EEG abnormalities in early childhood schizophrenia: A double-blind study of psychiatrically disturbed and normal children during promazine sedation. American Journal of Psychiatry, 1964, 120, 950-958.

Whittam, H., Simon, G.B., & Mittler, P.J. The early development of psychotic children and their sibs. Developmental Medicine and Child Neurology, 1966, 8, 552-560.

Wing, J.K. Diagnosis, epidemiology, aetiology in early childhood autism. London: Pergamon Press, 1966.

Wing, J.K. (Ed.) Early childhood autism: Clinical, education, and social aspects. Oxford: Pergamon Press, 1966.

Wolff, W.M., & Morris, L.A. Intellectual and personality characteristics of parents of autistic children. Journal of Abnormal Psychology, 1971, 77 (2), 155-161.

World Health Organization Glossary of Mental Disorders and Guide to Their Classification. Geneva, 1974.

Zaporozhets, A.V. The origin and development of the conscious control of movements in man. In H. O'Connor (Ed.) Recent soviet psychology. New York: Liveright, 1961.