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DISTINGUISHING and CLASSIFYING the INDIVIDUAL SCHIZOPHRENIC CHILD

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Table of Contents

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Childhood schizophrenia covers a wide range of the most extreme forms of behavior disorder in children in the period between birth and pubescence. In psychiatric discussion, it is sometimes known by other names as well, for example, "childhood psychosis." All these names refer to a very wide variety of disorders, so that it is usually uncertain what specific range of deviations is covered in the use of these terms. However, most workers with particular interest in this broad range of childhood deviancy have doubts about the usefulness of applying in mechanical and uncritical fashion to the condition of childhood schizophrenia the principles and facts that pertain to the symptoms, processes, and etiology of adult schizophrenia.

Whatever may be the relationship between childhood and adult reactions, it is still wise to study and illuminate schizophrenic reactions in childhood apart from those noted in adulthood. Our most urgent requirements are sensitivity to individual variations among schizophrenic children in their patterns of adaptation and life course and precise sub-classification in order to accomplish more homogeneous sub-clusters of schizophrenic children.

Even though it is often not clear just which specific aspects of the broad category of childhood schizophrenia may be under consideration in a given study or report, there is general agreement regarding the broad diagnosis of

childhood schizophrenia. There is an increasingly clearer understanding of its relationship to other childhood disorders, based on sharper delineations of the contributing factors and the phenomenological expressions. Thus, therapeutic skill has grown and key issues have emerged more precisely.

It is accordingly the purpose of this chapter to present a summary of the historical diagnostic and etiological issues connected with the study of childhood schizophrenia in a manner that might be of practical use in developing an approach to the management and observation of those children who are found in this category of behavioral disorder. This brief presentation will be utilized to cast light on some important questions which urgently need to be answered at this time. What is the link between childhood schizophrenia and adult schizophrenia? How homogeneous are children diagnosed as schizophrenic? For clarifying the study of the growth and development of schizophrenic children, how can schizophrenic children be sub-classified so as to achieve homogenous sub-clusters of children?

Detailed reviews of childhood schizophrenia and annotated bibliographies are available, and these will not be replicated. Rather, an effort will be made to draw out of the vast, oftimes confusing, literature essential conviction and information that can be applied in a simple and direct fashion in the treatment and study of childhood schizophrenia. In this distillation of thought and practice, we have been influenced by pragmatic experience in the

treatment and investigation of psychotic conditions in childhood.

A review of the history of the construct embodied in the phrase "the schizophrenic syndrome of childhood" will serve to expose some major contemporary issues in the study and treatment of the behavioral impairments to which it refers. In the historical review we will first consider the relationship of childhood schizophrenia to adult schizophrenia. A historical perspective will serve to emphasize the key significance of maturation and its disturbance in the manifestations of childhood schizophrenia. Then the presentation of criteria for diagnosis will stress the very broad range of children who are filtered out by present diagnostic methods. Similarly, the discussion of etiology that follows will evaluate the merits of multi-causal versus uni-causal hypotheses as a basis for elaborating programs of treatment and research. Based on an awareness of the diversity of children termed schizophrenic and the complex kind of interplay between social, intrapsychic, and biological factors, a practical basis for sub-classification will be suggested. The transactional point of view of the deviations of schizophrenic children is used in deciding how to sub-classify the children in meaningful and homogeneous sub-clusters. It is also the basis for an individualized approach to their care and corrective treatment.

Historical Sources of the Construct "Childhood Schizophrenia"

Currently, there is a strong disposition in the field of psychiatry to link childhood and adult schizophrenia. The similarity in names has reinforced this trend; but this is hardly a relevant or logical basis for such association, since similar-names could have been supplied to different conditions. A historical perspective is more useful for casting light on the impetus to connect the two classes of disorder, although historic association in the evolution of thought regarding the two psychiatric entities is also not evidence in itself that they represent a single class of disorder manifested at different times in life.

Contemporary concepts of childhood psychosis generally followed in the wake of developments in the delineation of adult psychosis. Even the names assigned the very severe behavioral disturbances in childhood were borrowed from those assigned to adult disturbances. For example, diagnostic labels, such as “dementia infantilis” and “dementia praecocissima” followed Kraepelin’s definition of dementia praecox in adolescence and adult years. In the same way, the diagnostic phrase “childhood schizophrenia” followed Bleuler’s definitions of the adolescent and adult schizophrenia.

In their fundamental contributions to adult psychiatry, both Kraepelin and Bleuler referred to on occasional, albeit infrequent, onset of the psychiatric disorders in childhood years. Kraepelin, for example, reported that 3.5 percent of his patients with dementia praecox had been under ten

years of age at time of onset; Bleuler similarly reported that 5 percent of his schizophrenic patients had been under ten years of age at onset. It is difficult to interpret what bearing these data have on the question of the link between adult and childhood schizophrenias. Both writers had little experience with young children, and they did not offer descriptions of predisposing traits and circumstances in childhood. Nor is it clear what modifications they made in applying criteria for adult diagnosis to children. Kraepelin's descriptions of simple, hebephrenic, paranoid, and catatonic reactions in dementia praecox are still considered pertinent in the categorization of adult schizophrenic reactions. However, generally speaking, these four syndromes have not been regarded as applicable to childhood reactions. Bleuler's precise definition of the symptoms of schizophrenia were more applicable to children. During the 1930's and 1940's child psychiatrists became intrigued by the likelihood that typical schizophrenic reactions could appear long before adolescence. On the other hand, they recognized that symptomatic expression of psychosis in childhood was influenced by the child's cognitive immaturity. Potter, for example, explained the infrequency and simplicity of the delusional reactions in psychotic children by their limitations in language and their concreteness of thought. Even Bleuler's symptoms had to be modified if they were to be used in the categorization of aberrant behaviors in children.

Progress in the definition and understanding of the psychoses of childhood thus began with the elaboration of symptomatic criteria uniquely

suited to the forms and boundaries of childhood expression. Even more crucially, a great impetus to the evolution of understanding and diagnosis of childhood psychoses was the recognition that the definable entities of childhood psychosis all represented patterns of disorganization or impairment in behavioral maturation. Thus, psychiatrists were impelled first to search for early onset histories of schizophrenia. They soon discovered, however, that the criteria used in the diagnosis of schizophrenic reactions in adolescent or later years had to be modified to include forms of behavior that are uniquely evident in early childhood. In this extrapolation, they made the rather large assumption of equivalence in psychopathological and dynamic significance of the different forms of deviant expression in childhood and later years. Finally, they discarded their efforts to find childhood equivalents of adult schizophrenic symptoms. Instead they tended to stress maturational deficit as the primary signs of psychosis. When they took this necessary diagnostic step it became evident that the presumed link between childhood schizophrenia and adult schizophrenia was tenuous indeed. It became clear that though historically there had been expressed a historic relationship between the two classes of impairment which had led to an equivalence in names, it did not follow that there was an equivalence in psychological significance and certainly not in etiology.

This historic trend toward differentiation of the two conditions should become evident if one compares Bleuler's criteria for the diagnosis of adult

schizophrenia with more recent criteria for diagnosis of childhood psychosis. Bleuler described primary and secondary symptoms of schizophrenia. Primary symptoms included (1) autism, that is, the unusual predominance of inner fantasy life over reality, (2) fragmentation and lack of continuity of association, (3) affective disharmony and disturbance, and (4) extreme emotional ambivalence. Secondary symptoms included (1) hallucinations, (2) delusions, (3) illusions, and (4) motor aberrations, such as catatonia.

In one of the earlier applications of Bleuler's criteria in the diagnosis of childhood schizophrenia, Potter proposed the following criteria: (1) a generalized retraction of interests from the environment; (2) unrealistic thinking, feeling, acting; (3) disturbances of thought, for example, blocking, condensation, symbolization, perseveration, incoherence, and diminution; (4) defect in emotional rapport; (5) diminution, rigidity, and distortion of affect; (6) behavioral alteration, exaggerated increase or decrease in motility, or bizarre, perseverative, stereotyped behavior. The absence of Bleuler's secondary symptoms, such as hallucinations and delusions, in Potter's criteria for diagnosis of childhood schizophrenia is noteworthy, inasmuch as Potter felt this reflected the immaturity of the children. Potter's criteria are, perhaps, more related to Bleuler's primary symptoms, although even these adult and child criteria do not precisely replicate each other. Potter's symptoms of childhood schizophrenia represent an early recognition that, quite unlike the approach to adult schizophrenia, diagnosis of childhood schizophrenia is

based on the observation of developmental deviation in crucial, purposeful functions.

Bradley and Bowen reflected the developing empiricism and the recognition that adult criteria were not suitable for diagnosis of childhood schizophrenia. They asked themselves what traits do in fact characterize schizophrenic children. They described observable, objective symptoms in a group of children under therapeutic observation, including four children with actual schizophrenic psychosis and ten children with evidences of schizoid personalities. (Implicitly, none of the children replicated precisely all the attributes of Bleuler's adult schizophrenia and certainly not of Kraepelin's dementia praecox.) Eight characteristics differentiated these 14 children from 124 other children admitted to residential treatment. In order of frequency these traits were (1) seclusiveness, (2) instability when seclusiveness was disturbed, (3) daydreaming, (4) bizarre behavior, (5) diminution in number of personal interests, (6) regressive nature of personal interests, (7) sensitivity to comment and criticism, (8) physical inactivity. Bradley was impressed, however, with the primary symptomatic significance of seclusiveness, bizarre behavior, and regression. These three primary or key symptoms have little obvious link to Bleuler's primary symptoms of adult schizophrenia, which still represent the current key criteria of adolescent and adult schizophrenia. Bradley recommended that the diagnosis of childhood schizophrenia be founded chiefly on aberrations in the child's psychological

growth.

Beginning in the 1950's, the emphasis on dysmaturation as the key to diagnosis of childhood schizophrenia was represented in the work of two major observers in the field, Lauretta Bender and Leo Kanner.

Although convinced from the beginning that childhood schizophrenia was related etiologically to adult schizophrenia and that both were genetically determined, Bender's many important descriptions of childhood schizophrenia have emphasized the central significance of disturbances in growth. The symptoms she recommended for diagnosis had little obvious link to the adult manifestations of schizophrenia. Indeed at one point, she noted that childhood schizophrenia could assume forms that subsumed every variety of childhood disorder. Thus she described a pseudo-defective type, a pseudo-neurotic type, a psychosomatic type, a pseudo-psychopathic type, a type with frank psychotic expression, and a latent type. Suffice it to say that she proposed that the central core of schizophrenia, which is expressed in many forms, is "a total psychological disorder in the regulation of maturation of all the basic behavior functions seen clinically in childhood. Thus it is a maturation lag with embryonic features as characterized by primitive (embryonic) plasticity in all patterned behavior in the autonomic or vegetative, perceptual, motor, intellectual, emotional and social areas." It is difficult to appreciate sufficiently the historic significance of Bender's

exquisite clinical descriptions which anticipated virtually all later descriptive and phenomenological investigation of schizophrenic children and which focused the attention of all subsequent observers on disturbances in biological and psychological maturation as a key to diagnosis. Again, however, we should like to stress that, although convinced herself of the unity of childhood and adult schizophrenia, Bender's criteria for diagnosis of childhood schizophrenia had no prima facie connection with Bleuler's primary and secondary symptoms of adult schizophrenia.

The second major observer was Kanner who, during 1942, published his classical report on early infantile autism. This report is still the best description of the autistic syndrome. Kanner's criteria for the diagnosis of infantile autism were as follows:

1. Aloneness, extreme in degree and evident in earliest infancy. The babies do not respond with normal anticipatory gestures as the adults reach to pick them up and do not adapt to the bodies of those who hold them.

2. Impaired communication. Speech and language are not used for the purposes of communication. Often the children are entirely mute or, if speech is present, it is echolalic and does not convey meaning. Pronominal reversals and literalness are frequent; and affirmation is expressed by repetition rather than the use of the word "yes."

3. Obsessive insistence on the maintenance of sameness, with great anxiety in new and unfamiliar situations, and with repetitive ritualistic preoccupation.

4. Fascination for objects, in contrast to disinterest in people.

Kanner and his closest colleague, Eisenberg, differentiated infantile autism from schizophrenia on the basis of onset history, course, and familial background. Thus, they held that, in contrast to schizophrenia, autism starts extremely early in infancy and the relatives of autistic children presumably do not show abnormally high incidence of schizophrenia. They also differentiated autism from mental deficiency, since the autistic child presumably shows segmental areas of normal or even superior intellectual capacity. Eisenberg recommended that the expression "infantile autism" be restricted in use to children with psychotic onset during the first year of life and schizophrenia to children with psychotic onset at eight years or older. Bender included autistic aberrations in her concept of multiple manifestations of a total psychological and maturational failure termed "schizophrenia." Others supported Kanner's separation of the early autistic reaction from childhood schizophrenia. In contrast to Kanner and Rutter, some workers observed an unusually large ratio of schizophrenic parents in the families of children with early psychotic onset. Certainly Rimland's recommendations for differentiating the autistic children from schizophrenic children on the basis of signs in the former such as physical beauty, normal

electroencephalograms, excellence in motor capacity, uniformly high intelligence and education of the parents, and the “idiot savant” character of intellectual organization are not supported by systematic clinical or experimental observation; indeed, these characteristics are often missing in children with early onset. It is safe to assume that apart from age of onset, the sharp differentiation of infantile autism from other psychotic reactions in early childhood is still uncertain.

As in Bender’s work, Kanner emphasized developmental impairment in a broad array of psychological functions. His diagnostic criteria also had little apparent relationship to the criteria of adult psychosis. All-important observers after Bender and Kanner have similarly called attention to the primary importance of maturational disorder in childhood as a key to the understanding and diagnosis of schizophrenic children.

Of special interest among these observers are those who, while recognizing predisposing factors, emphasized psychodynamic forces impeding the psychological growth of psychotic children. Of particular significance is the work of Mahler. In a series of papers Mahler and her colleagues developed a psychodynamic approach derived from classical psychoanalysis. Mahler proposed that normal children move through three states of self-differentiation. (1) During the autistic phase between birth and three months, the normal infant is presumably aware of inner stimuli only

and does not perceive objects outside his body. (2) During the symbiotic phase of development, beginning at about three months, the child is incipiently aware of an external object capable of satisfying his needs but does not sharply differentiate his mother from his image of self. (3) Then at twelve to eighteen months, during the separation-individuation phase, the baby begins more sharply to differentiate himself from the non-self. Mahler postulated that children suffering from the autistic psychosis have not developed beyond the normal autistic phase. In the symbiotic psychosis, Mahler postulated that the children have not been able to accommodate to the challenge of separation and individuation. In a state of panic, these children may regress to the autistic state, in which a clear personal identity is totally lacking.

Other workers, too, have represented a psychodynamic bias. Earlier than most, Despert took a psychotherapeutic position and described the difficulties of schizophrenic children in attaining normal emotional relatedness to reality. For many years, Szurek (Boatman and Szurek) related his therapeutic management of schizophrenic children to a psychodynamic hypothesis. He proposed that the disorders of schizophrenia result from emotional conflict and their miscarried resolution. He thus attempted to alleviate the disorders by intensive psychotherapy of the children and their parents. Rank and Putnam also emphasized psychodynamic and developmental features of the disorder.

Apart from their contributions to the issue of etiology, which we shall be considering later in this report, Despert, Mahler, Szurek, Rank, Putnam, and many others like them represent a group of psychiatric observers who have been impelled by therapeutic, rehabilitative objectives. With this orientation, they have been primarily concerned with the psychic growth of individual children over time. This is a developmental approach to childhood psychosis, in which schizophrenic children are appraised in terms of ontogenetic history and in the context of normal child development.

To recapitulate, psychiatrists first looked for expressions of adult psychosis in childhood. They then found that criteria for diagnosis of adult psychosis needed extensive modification before they could be applied to children. Soon, also, psychiatrists began to develop criteria that were uniquely suited to the delineation of extreme behavioral aberrations in childhood and that were quite unlike those employed for adult diagnosis. Theoretical or factual links between the criteria used in childhood and those employed in adulthood have not been well elaborated. These links thus remain ambiguous.

What hard facts, however, bear on the question? Certainly, virtually by definition, few individuals who manifest first signs of schizophrenia in adolescence or adult life will have shown manifestations of childhood schizophrenia during their early childhood. Follow-up studies of

schizophrenic children do show more suggestive overlap between diagnosed schizophrenia of children and schizophrenia of adolescent or later years. The most frequently reported study, that of Bender and Grugett, stated that 87 percent of a group of children who had been diagnosed schizophrenic during childhood were diagnosed as schizophrenic during adolescence or adult life. The very high incidence of adult schizophrenia when schizophrenic children reach adulthood has been confirmed in virtually all follow-up studies.

In Bennett and Klein's follow up of fourteen schizophrenic children thirty years after the diagnosis of childhood schizophrenia, nine were in hospitals and only one was maintaining himself outside. (Two were dead, and two could not be located.) Of particular interest was their observation that the nine hospitalized cases could not be differentiated from other chronically deteriorated adult schizophrenic patients in the same hospitals. How accurate is this observation, which at best is a qualitative one? It is undoubtedly difficult to distinguish the markedly deteriorated adult patient from the very regressed schizophrenic child who has not succeeded in maturing in adaptive function as he has grown older. Most follow-up studies are after all retrospective. There is obvious advantage in observing the schizophrenic child in a prospective fashion as he matures; for then it frequently becomes evident that the highly impaired or primitive schizophrenic child changes very little as he grows older and that his personality in adult years is quite like that in his childhood. Since he has not deteriorated in his adult years, he

has not “become” an adult schizophrenic. He is still in a sense a childhood schizophrenic in personality organization; and his early personality is merely residing in an older body.

A genetic study, such as that of Kallmann and Roth would seem to support the notion that childhood schizophrenia and adult schizophrenia are both processes related to the same gene-specific deficiency state. However, this study focused on children who had grown normally before onset of the psychotic disorder. Thus the average age of onset was 8.8 years. In this sense, the study has little bearing on the processes involved in the very early childhood psychoses.

In addition to differences in their diagnostic criteria, childhood schizophrenia and adult schizophrenia each subsume a wide diversity of disorders. Among schizophrenic children, the range in personality and adaptive capacity is so great that the diagnosis of childhood schizophrenia ordinarily has virtually no bearing on the treatment plans formulated for each individual child. Some of the children are largely devoid of adaptive skills, including intelligence, language, and social capacity. Others are extremely bright and verbal but laden with complex psychological defenses. In view of the heterogeneity of schizophrenic children and the broad diversity of adult schizophrenic reactions as well, it would seem wise to explore the problems of childhood schizophrenia apart from any links to adult schizophrenia. In

addition, empirical experience in the follow up of schizophrenic children has demonstrated that while a large proportion are ultimately classified as schizophrenic in late adolescence or adulthood, others are reasonably classified in other categories of adult pathology, for example, mental deficiency, organic brain syndrome, and a variety of other classes of disorder. A primary need is the longitudinal observation of schizophrenic children for detailed study of variations in life course to be noted among individual schizophrenic children. Since all of the schizophrenic children to be studied as individuals are, nevertheless, members of the same gross diagnostic class, a standard set of diagnostic criteria is required to select such children for purposes of treatment and study.

Diagnosis of Childhood Schizophrenia

As a general background for discussion of the diagnosis of childhood schizophrenia, Eisenberg's approach to a wider classification of childhood psychoses recommends itself. As in adulthood, psychoses of childhood refer to deep functional impairments relative to normal children of equal age including gross disorders in personality, regressive defenses, bizarre and socially unacceptable behavior, and markedly deficient testing of reality. In this larger group of psychoses, Eisenberg first differentiated disorders caused by or associated with impairment of brain tissue and with demonstrable pathology of brain tissue from psychotic disorders in which structural

changes in the brain have not as yet been demonstrated.

The psychoses with unequivocally demonstrable brain tissue pathology include the toxic psychoses (for example, atropine poisoning), metabolic psychoses (for example, pellagra), degenerative psychoses (for example, Schilder's disease), and infectious psychoses (for example, paresis), disrhythmic psychoses (for example, psychomotor seizures), traumatic psychoses, and neoplastic psychoses. These are often easily diagnosed and warrant immediate and appropriate treatment.

The remaining psychoses are those in which demonstrable and unequivocal brain tissue changes have not as yet been demonstrated. This group of functional disorders has never been adequately subdivided. It includes children previously described under a wide assortment of labels including infantile autism, childhood schizophrenia, atypical child syndrome, childhood psychosis, psychosis on top of mental deficit, and rare reactions termed "folie à deux." As previously reported, Eisenberg himself recommended restricting the infantile autistic reactions to disorders with onset during the first year and schizophrenia to disorders with onset after eight years of age. The latter disorders presumably satisfy the criteria for diagnosis of schizophrenia in adulthood. Eisenberg also implied that the categories of infantile autism and schizophrenia encompass the bulk of the functional psychotic disorders. In view of the obvious overlapping among the

children included in each of the above subgroups, there would seem to be wisdom in disregarding the many descriptive labels, in continuing to deal with the functional disorders as a large heterogeneous group termed "schizophrenic syndromes of childhood," and in seeking bases for subdividing the larger group along a series of empirically determined dimensions. Onset history, for example, is an example of a useful kind of parameter for such subclassification. Other dimensions for such subdivision will be recommended.

It is necessary first to have a set of diagnostic criteria for the diagnosis of the broad category of childhood schizophrenia that most workers could agree on. During 1961, after extensive discussion, a group of British workers agreed on the following criteria:

1. Gross and sustained impairment of emotional relationships with people. This includes the more usual aloofness and the empty clinging (so-called symbiosis); also abnormal behavior towards other people as persons, such as using them impersonally. Difficulty in mixing and playing with other children is often outstanding and long lasting.

2. Apparent unawareness of his own personal identity to a degree inappropriate to his age. This may be seen in abnormal behavior towards himself, such as posturing or exploration and scrutiny of parts of his body. Repeated self-directed aggression, sometimes resulting in actual damage, may be another aspect of his lack of integration (see also point 5) as is also the confusion of personal pronouns (see point 7).

3. Pathological preoccupation with particular objects or certain characteristics of them without regard to their accepted function.

4. Sustained resistance to change in the environment and a striving to maintain or restore sameness. In some instances behavior appears to aim at producing a state of perceptual monotony.

5. Abnormal perceptual experience (in the absence of discernible organic abnormality) is implied by excessive, diminished, or unpredictable response to sensory stimuli—for example, visual and auditory avoidance (see also points 2 and 4), insensitivity to pain and temperature.

6. Acute, excessive and seemingly illogical anxiety is a frequent phenomenon. This tends to be precipitated by change, whether in material environment or in routine, as well as by temporary interruption of a symbiotic attachment to persons or things (compare points 3 and 4, and also 1 and 2). (Apparently commonplace phenomena or objects seem to become invested with terrifying qualities. On the other hand, an appropriate sense of fear in the face of real danger may be lacking.)

7. Speech may have been lost or never acquired, or may have failed to develop beyond a level appropriate to an earlier stage. There may be confusion of personal pronouns (see point 2), echolalia, or other mannerisms of use and diction. Though words or phrases may be uttered, they may convey no sense of ordinary communication.

8. Distortion in motility patterns—for example, (a) excess as in hyperkinesis, (b) immobility as in catatonia, (c) bizarre postures, or ritualistic mannerisms, such as rocking and spinning (themselves or objects.)

9. A background of serious retardation in which islets of normal, near normal, or exceptional function or skill may appear.

Since the criteria were reported by the British working group in 1961, they have been widely applied to diagnosis of schizophrenic children. In one treatment center,[1] a review of the symptoms of all children discharged over a ten-year period with the diagnosis of childhood schizophrenia demonstrated that all the symptoms of the children were encompassed in the nine signs listed above. All these children evidenced at least five of the nine signs. In addition, all the children manifested impairment in human relationships, defects in personal identity, excessive anxiety provoked by change, and disturbance in speech and communication. As further support for the practical utility of the nine signs, a review of a large number of reports, which included descriptions of symptoms of childhood schizophrenia, showed that all the symptoms described by the many authors were encompassed by the nine points.

It can be stated unequivocally that the children who are filtered out by these clear though broad criteria are highly heterogeneous in behavioral attributes and capacities, in psychosocial and social class characteristics of

the family, and in neurological manifestations. Beyond the mere diagnosis of childhood schizophrenia, there is an obvious need to subdivide the children into homogeneous subclasses. In this regard, the dominant disposition of psychiatric observers is still to propose systems of sub-classification in which adult schizophrenia remains a central referent. In other words, these observers tend to ask only if childhood psychosis is or is not an extension of adult schizophrenia; and, by implication, they have no reason to sub-classify. Thus some workers would tend to term the reactions of all the children "schizophrenic" (for example, Bender). Others distinguish infantile autism from schizophrenia. The latter restrict infantile autism to children with onset of their behavioral disorders in infancy, that is, birth to approximately two years. Eisenberg and Rutter restricted the diagnosis of schizophrenia even further to children with onset of their symptoms after eight years of age. Presumably, these children are examples of schizophrenic reaction that does not differ from the schizophrenic reactions of later years. Children with histories of onset between two and eight years are considered most often to be cases of primary organic psychosis other than autism or schizophrenia.

Perhaps the most explicit and most precise differentiation of early childhood psychosis and schizophrenia is that of Rutter. Beyond the obvious differences in age of onset, Rutter believed childhood psychosis and schizophrenia to be independent, non-overlapping conditions. Thus, he pointed out that unlike schizophrenia, which rarely appears before

pubescence, childhood psychosis is frequently associated with mental sub-normality and cerebral dysfunction and is only rarely associated with the secondary symptoms of adult schizophrenia, such as hallucinations, delusions, and paranoid ideation. In addition, familial history of adult schizophrenia is presumably rare in childhood psychosis. However, even these findings of difference have not been established in a totally unequivocal fashion. Most systematic studies have confirmed the frequent association of early childhood psychosis with mental sub-normality, and by implication, the weaker association between low intelligence and adult schizophrenia. However, a significant proportion of children with early childhood psychosis have very superior intelligence. Sizable changes in IQ over a three-year period have been noted in a high percentage of psychotic children in residential treatment with a consequent tendency to augment the number with high IQs, though the children with lowest IQs tend not to change. Similarly, although a very high proportion gives signs of cerebral dysfunction, a sizable (albeit smaller) proportion does not present these neurological signs. To complicate matters, it would now appear that a large percentage of patients with onset of schizophrenia in adolescence also give evidence of cerebral dysfunction. In Pollin's very important studies of adult twins discordant for schizophrenia, the schizophrenic twin was more likely to manifest soft evidence of neurological dysfunctions. It is true, too, that children with very early onset of psychosis rarely present symptoms such as hallucinations, delusions, and

elaborate paranoid reactions. (However, we have been more impressed than Rutter by the frequent paranoid expressions, perhaps restricted to the brighter, more verbal children.) Finally, some workers have diagnosed schizophrenia in the parents, especially the mothers, with much greater frequency than noted by Kanner and Rutter.

Other data differentiating autistic from schizophrenic children have been described by a number of authors. For example, the superior educational and vocational status of parents of autistic children has been noted. Yet more recent studies have demonstrated families of children with early infantile psychosis come from all social classes. Recently, with increased referral of children by poverty agencies, the ratio of cases of early infantile psychosis among low social class families in therapeutic installations has been increased. Obviously, the artifact of sampling will influence the distribution of families in regard to social class position.

Among diagnosed schizophrenic children, there are wide variations in personality organization, symptomatology, clinical course, patterns and level of intellectual organization, and contributing circumstances. They range from near total absence of affective and social response, language, and cognitive capacity to high levels of ideational response and affectivity. In some, affective meagerness and social withdrawal are the dominant behavior. In others, the children show complex protective mechanisms, including phobic, obsessional,

paranoid and depressive reactions, and at times, delusional responses. Some grow up quite unchanged in clinical manifestation. Others improve dramatically and attain normal levels of educational and social response. The contributions to the child's symptoms of factors, such as familial or neurological deviance, vary from child to child. While Rutter has stressed the failures in language in the children he has studied, as shown, for example, in superiority of performance capacity over verbal capacity as measured in standard tests, such intellectual patterning and segmental failure in verbal response have not been confirmed in other samplings of schizophrenic children (for example, see Goldfarb and Goldfarb).

Differences among observers of schizophrenic children undoubtedly reflect the heterogeneity of the children and the artifact of sampling. No single description of a necessarily limited sample of children suffering from early childhood psychosis can be generalized to the entire population of psychotic children. Therefore, there is no simple answer to the question of whether schizophrenia is one condition encompassing the bulk of childhood as well as adult psychoses or, on the other hand, whether the adult and childhood psychoses are totally disparate and non-overlapping.

Fortunately, the absence of unequivocal evidence as to the presence or absence of association between childhood schizophrenia and adult schizophrenia is not a serious hindrance to the creative evolution of

treatment methods and of programs of investigation. After all, the diagnosis of a treatment plan for an individual schizophrenic child should not be linked crucially to the fact that he has been classified as schizophrenic. Rather, the treatment plan should be determined by a careful evaluation of each child's unique pattern of ego organization, his specific adaptive strengths and weaknesses, and the life experiences to which he has been exposed. In research, too, the most meaningful information will reflect the highly individual interplay between constitutional and environmental factors.

Sub-classification of Schizophrenic Children

The primary necessity is to have more information regarding the life course of individual schizophrenic children and to gather such knowledge through the use of precise baselines for description and appraisal of change. By grouping the children on the basis of common patterns of change in the appropriate factors, the clusters of children that emerge provide homogeneous groupings which otherwise remain hidden behind nonspecific categories such as schizophrenia and autism.

As will be stressed, a more profitable basis for sub-classification of schizophrenic children, therefore, is one that takes into account the broad range in individual adaptive capacity of the children and in factors, both internal and external, that influence their psychological growth. In such sub-

classification, also, the association between the psychotic child's individual growth, the adaptive capacities of the child, his neurological integrity, and the level of psychosocial functioning of his family should be noted.

For deciding which adaptive attributes might be included in systematic sub-classification, investigations of purposeful functions in schizophrenic children are already helpful. The levels of behavior that have been reported include sensation, perception, conceptualization, and psychomotor response. The functions represented are involved in the child's efforts to orient himself and contact reality, to make meaningful generalizations about reality, to test these generalizations, and to manipulate them in the service of adaptation and survival.

Frequently in clinical study of schizophrenic children, the possibility of sensory loss or marked elevation in sensory thresholds has had to be considered. Pseudo-deafness, for example, has been noted frequently. In contrast, observations of hypersensitive reactions have stimulated the hypothesis of diminished sensory thresholds. Actual studies of sensory thresholds, however, have demonstrated normal thresholds for vision (A-O charts at twenty feet), for hearing (pure tone audiometry), and for touch (Von Frey Test). Though they have demonstrated normal auditory thresholds to pure tone stimulation, schizophrenic children showed more elevated thresholds for speech than for pure tones. While further studies are needed,

this discrepancy would support the conclusion that the phenomenon of not hearing may reflect altered attention to and awareness of human speech rather than impaired sensory acuity.

In contrast to the evidence that the schizophrenic child's apparatus for receiving sensory impression is intact, all investigations have demonstrated inferiority, relative to normal children, in perceptual discrimination. Numerous studies have also confirmed deficits in abstract and conceptual behavior. Included in these studies of conceptual failure are the many investigations that have demonstrated the strong trend to low intellectual response and impairments in communication. Psychomotor behavior is found to be equally impaired.

Inferences regarding the defects in perceptual discrimination, in the ordering of perceptual information for the attainment of meaning, and in the execution of adaptive acts refer to studies using summary statistics based on groups of children. However, there is a very broad range of capacity in all the purposeful functions among individual schizophrenic children, which often remains unnoticed in the group summary statistics. For example, the children range from extreme intellectual deficiency, so severe that mental testing is not feasible, to very superior intellectual functioning (see for example, Goldfarb and Goldfarb.) Some of the children are totally devoid of language and are extremely restricted in educability and capacity for self-care. Others

are superior in intellectual and educational competence.

In addition, the children differ greatly among themselves in course of development. A large proportion of the children do remain chronically impaired. Others attain fairly normal levels of capacity for schooling and community living. In most follow-up studies, the ratio of children who attain such moderately normal levels range from a quarter to a third of the children. Recent studies of changes in specific adaptive functions show comparable variations. Individual curves of growth in reading in response to schooling while in residential treatment vary from a reflection of complete uneducability to a reflection of advanced educational response. Similarly, in recent studies of change in the Wechsler Full IQ of schizophrenic children while in a therapeutic residence, some of the children showed no change and others showed dramatic changes that seem to be linked to comparable shifts in clinical status.

In anticipation of a proposal for sub-classifying schizophrenic children, it should be noted at this point that the general level of adaptive capacity of schizophrenic children at the start of therapeutic observation is associated with their later and ultimate progress. Clinical follow-up studies, for example, have confirmed that children who have very low intelligence quotients and who are devoid of verbal speech at five to six years of age show uniformly poor clinical progress. Systematic observation of longitudinal change in

specific perceptual, conceptual, and psychomotor functions confirm the fact that children with lowest intellectual functioning (Wechsler Full IQs below 45) remain quite unchanged, whereas children of greater initial capacity show significant improvements.

Although schizophrenic children manifest failures at all levels of behavior, including receptor, integrative, and executive levels, a number of observers have emphasized particularly the deficiencies in perceptual-afferent response. Clinical and experimental observation has confirmed abnormality in the hierarchy of receptor organization and in intersensory integration. Thus, schizophrenic children often avoid focused visual and auditory attention to objects in their environment; touching, tasting, and smelling are substituted. The apparent auditory and visual imperception may well be a later and defensive reaction to the discomfort of initial hyperacusis. Such primary failure in the afferent organization of behavior apparently precludes discriminative response, separation of figure from ground, anticipatory response, and monitored learning more generally. It may be presumed that the failure to achieve a pattern of auditory commitment, for example, is a factor in the impaired speech of schizophrenic children or that the schizophrenic child's deficits in discrimination of difference in shape and directional orientation are exaggerated by visual inattention.

While stressing the deficiencies of schizophrenic children in the

reception and organization of sensory input, it must be repeated that failures have been noted in all aspects of adaptive functioning, that is, in central organization or conceptualization of perceptual information and in the motor and executive levels of behavior. In addition, a serious consequence of the failures in adaptive response is a drastic absence of sharp inner awareness of the self in action as differentiated from the non-self. Beyond this, schizophrenic children suffer intense anxiety as an outcome of their inability to achieve feelings of familiarity, permanence, and predictability.

How can a theory of etiology help in sub- classifying the children? The precise cause of the above noted adaptive failures in childhood schizophrenia has not been established. Three kinds of etiological hypothesis have been formulated. One variety of hypothesis emphasizes primary and intrinsic deficiencies in the schizophrenic child. Another variety of hypothesis proposes that the psychosocial environment is the primary causal agent leading to the development of the schizophrenic syndrome. The third view of etiology proposes that all schizophrenic reactions reflect the influence of both intrinsic and extrinsic factors. The latter position is favored in the present report for its value in diagnosis, sub-classification, formulating individual treatment plans, and designing research. This point of view assumes that primary atypism and deviant psychosocial influence are dimensional in character, that is, they vary in observable degree among individual schizophrenic children. The symptoms and character attributes embodied in

the schizophrenic reaction reflect the interaction of the child's potentialities for adaptive response and the expectations and reinforcements of his outer world.

There are two general bodies of evidence in support of a primary atypism. One body of evidence supports the etiological significance of inheritance and refers chiefly to familial concordance for psychosis. The other body of evidence refers to dysfunction and trauma of the central nervous system.

Bender and her coworkers argued most cogently that childhood schizophrenia and adult schizophrenia are both caused by the same genotype. Her evidence is twofold, that is, the observation that the large majority of schizophrenic children who had been under her care ultimately developed symptoms of adult schizophrenia and the further observation that an unusual proportion of the children's mothers (43 percent) and fathers (40 percent) were mentally ill. Other studies of prevalence of schizophrenia in the families of schizophrenic children have tended to support the high incidence of parental schizophrenia, but to a lesser degree. Most significantly, there are wide statistical variations in the studies reported. In one sample of parents of early school-age schizophrenic children at the Ittleson Center, 28 percent of the mothers and 13 percent of the fathers were classified as schizophrenic. In Kallmann and Roth's study of fifty-two twins and fifty singletons, the parental

schizophrenia rate was 9 percent. On the other hand, Kanner noted that only one of one hundred parents showed major mental illness. Nor is it possible to explain the range of frequencies by the fact that Bender had fewer cases of infantile autism -in her sample than Kanner, since Bender's sampling undoubtedly included a large percentage of cases of infantile autism by Kanner's criteria. Certainly, although the Kanner and Ittleson frequencies are quite different, a large percentage of the Ittleson Center population were cases of very early infantile psychosis.

Methodologically, there can be no doubt that Kallmann and Roth's study of twin concordance in preadolescent schizophrenia was the most mature methodologically for studying the hereditary factor and freest of ordinary contaminants. In their study, dizygotic and monozygotic twins differed in concordance rates for preadolescent schizophrenia (17.1 percent and 70.6 percent) and for adult schizophrenia (14.7 percent and 85.8 percent). In this study, as in Kallmann's study of twin concordance rates for adult schizophrenia, the differences between one-egg and two-egg twins were significant. The reporters concluded that preadolescent schizophrenia was determined by the same gene-specific deficiency state as adult schizophrenia. However, it must be emphasized again that the mean age of onset of Kallmann's group, about 8.8 years, was much older than that of children with early psychosis. In addition, Kallmann and Roth excluded mentally deficient children, who represent the bulk of children in most studies of children with

early childhood psychosis. The Kallmann and Roth results obviously are not applicable to children ordinarily included in investigations of early childhood schizophrenia.

Beyond these restrictions on the Kallmann and Roth findings for explaining childhood schizophrenia, more recent twin concordance studies (e.g. Kringlen) in adult schizophrenia have tended to show smaller differences in concordance rates between one-egg and two- egg twins. In addition, as Birch and Hertzig have argued, concordance rates in twins may reflect the greater risk of reproductive complications in the development of twins than in the development of singletons and also greater risk in the development of monozygotic twins than in dizygotic twins. This is of key importance since, as will be seen, the evidence of damage to the central nervous system in many children suffering from early childhood schizophrenia is very strong. The part played by a genetic factor in childhood schizophrenia still needs to be studied. Such study will have to include careful control of nervous and psychosocial factors.

Evidence for central nervous system impairment in a proportion of diagnosed schizophrenic children, however, is quite convincingly derived from many sources. Many studies have demonstrated a higher incidence of prenatal and perinatal complications. Developmental deviations supporting the inference of dysfunction in the central nervous system have been noted

by many observers. In addition to deviations in neurological history, a proportion of schizophrenic children tend to give observable, though soft, evidence of neurological dysfunction in physical examination, including deviations in gait, posture, balance, motor coordination, muscle tone, and integration of multiple simultaneous stimuli. In one clinical sample, neurological examination diagnosed neurological dysfunction in 65 percent of a group of schizophrenic children. Severe restrictions in level and pattern of perceptual, perceptuomotor, and cognitive response as measured in formal tests have been interpreted as evidence of neurological dysfunction. Perhaps of more direct significance, encephalographic studies have shown more frequent electroencephalogram abnormalities in schizophrenic children than in normals, and high incidence of convulsions has been reported.

In summary, therefore, prenatal and perinatal history, developmental trends, neurological histories, neurological examination, and systematic and controlled studies of neurological functions all offer strong evidence of a primary atypism in a high proportion of schizophrenic children. Historically, the disorders in integration of the central nervous system generally occur in the reproductive phase of development in early infancy. They express themselves early in infancy in disorders of sensorimotor integration and later in childhood in more complex cognitive and social failure.

The second general class of theories regarding the etiology of childhood

schizophrenia refers to theories of environmental and psychosocial causation. The elucidation of psychogenic and environmental factors has come in large measure from the therapeutic case study and is represented in the descriptions of observers with strong and primary motivation to heal. The therapeutic and case approaches to investigation are highly vulnerable to such errors as insufficient clinical documentation, vagueness of definition, and bias. Nevertheless, they are still our most valid tools for observing individual children in living situations and in process of growth, and for grasping the private, very subjective meanings and experiences of schizophrenic children. The high incidence of mental illness in the parents of schizophrenic children has been noted. While this can be interpreted as evidence for a specific genotype, it is also likely that schizophrenic parents represent an environmental challenge to the children as well. In support of this thesis, a systematic study of the psychosocial functioning of the families of schizophrenic children revealed that the families with one or two schizophrenic parents were less adequate in psychosocial functioning than those in which neither parent was schizophrenic. Finally, direct observations of family functioning and patterns of communication have tended to support the hypothesis that the families and parents of schizophrenic children deviate from normal.

However, it has always been clear that not all the schizophrenic children show evidence of neurological dysfunctioning and not all the parents and

families are functionally aberrant. A significant proportion of the children also offer fairly clear evidence of dysfunction of the central nervous system and, in addition, have families that are unequivocally and extremely aberrant. A multi-causal theory of etiology inclusive enough to explain all the known evidence would seem to be the most effective way of rationalizing the manifestations of childhood schizophrenia.

A multi-causal theory of etiology facilitates a transactional approach to the comprehension of schizophrenic children as individuals. In this approach the disordered adaptation of each schizophrenic child is presumed to reflect the interplay of intrinsic deficits in the child and of deviation in psychosocial organization of the family. It is also presumed that each of these classes of aberration varies dimension- ally from none to marked and that the relative contributions of each class of disorder to the functional impairments of the child vary from child to child. In some children, primary atypism of the child is the dominant causal factor; in others, the deviant family climate is the dominant causal factor. The former would be illustrated in the very seriously brain-damaged child in a normal family, and the latter would be illustrated in the neurologically and somatically intact child in a highly deviant family. Often too, one may note a neurologically impaired child who has been reared in a functionally deviant family. In a recent qualitative, psychiatric appraisal of neurological and familial contributions to the ego aberrations of forty schizophrenic children under intensive therapeutic care and very detailed

observation (for purposes of longitudinal study), the children were distributed as in Table 5-1. By clinical judgment, therefore, while the majority of the schizophrenic children appraised showed evidence of cerebral dysfunction, psychosocial and familial influences contributed in a primary or crucial way to the adaptive failures of the children in almost 73 percent of the cases. We are proposing that sub-classification include an assay of neurological and psychosocial influences.

TABLE 5–1.

Psychosocial factors dominant; no evidence of neurological dysfunction	30.0%
Evidence of neurological dysfunction	70.0%
Neurological dysfunction dominant; no evidence of psychosocial factors	27.5%
Psychosocial factors more dominant than neurological dysfunction	20.0%
Both psychosocial factors and neurological dysfunction significant	22.5%

In conclusion, we are now prepared to recommend a system of sub-classification. It has demonstrated its usefulness for attaining meaningful subgroupings of schizophrenic children. In other words, the subgroupings differ from each other in average adaptive capacity and etiological influence. In addition we have already been able to demonstrate that the subgroupings show significant differences in growth patterns.

Level of Intellectual Functioning

Tests of intelligence are viewed as tests of overall adaptive functioning and the IQ is seen as a measure of clinical status and functional capacity. The

value of the IQ as a predictor of clinical improvement has been demonstrated by a number of follow-up studies. Children who cannot speak and have the most extremely inferior IQs (for example, below fifty) show no significant clinical progress. In a recent study, children with intellectual functioning so inferior at admission as to be unmeasurable in the WISC tend not to show significant improvement in IQ during three years of residential treatment, In contrast, children at higher, measurable levels of intellectual response often do show significant improvement in WISC IQ. While absence of language by the age of five to six years has also been regarded as an important indication of bad prognosis, Rutter has demonstrated, however, that such language failure is of key predictive significance if linked to low intellectual functioning.

Age of Onset and Age of Admission to Treatment

Age of onset has been emphasized by many observers as a factor of major import in defining the diagnosis and life course of schizophrenic children. Quality of onset is in itself related to age of onset. Presumptively insidious onset, for example, is more likely to be associated with very early onset, and acute onset implies later onset. In experience with schizophrenic children of early elementary school age in treatment at the Ittleson Center, virtually all the children demonstrated developmental aberrations and symptoms from the earliest months of life, including the small percentage (about 13 percent) who also showed clear historic evidence of acute

reactions. On the other hand, age of admission may be defined in an objective and reliable fashion. A gross relationship between the constructs age of onset and age of admission to treatment may be presumed. It does emerge that age of admission to treatment does differentiate among schizophrenic children in terms of level of integrative and adaptive response and life course, even where the range of admission age is fairly narrow. This has been noted, for example, in a comparison of early school-age schizophrenic children admitted to residential treatment at eight years of age or older and those admitted below eight years of age. The children admitted at eight years or older showed higher levels of IQ than those admitted to treatment at younger ages. While both groups improved significantly in WISC Full IQ over three years of residential treatment, the younger children improved to a greater degree in IQ. Even so, the children admitted at the older ages maintained their cognitive superiority at each year of treatment over those children admitted at ages below eight years.

Sex

All investigations of childhood schizophrenia have confirmed the greater proportion of boys than of girls in those who are diagnosed as schizophrenic. The boy to girl ratio varies among sub-clusters of schizophrenic children grouped by a variety of other independent variables. For example, the proportion of boys is considerably higher among

schizophrenic children with evidence of neurological dysfunction than among those without evidence of neurological dysfunction, where the proportion of boys and girls are about equal. If we take into account the overlapping between sex and other variables and the evidence that differences between boys and girls in longitudinal change reflect, at least in part, the influence of these overlapping variables and of sampling as well, there is still great validity in including gender in a system of sub-classification inasmuch as the boys and girls seem to differ as groups. At the Ittleson Center, where attention has been paid to the issue of sex, group differences between schizophrenic boys and girls have been observed in psychodynamics, intelligence, level of ego organization, educability, and the influence of cerebral and psychosocial factors.

Level of Neurological Integration

Employing the judgment of qualified psychiatric neurologists and using neurological history and examination, it has been feasible to subdivide schizophrenic children with and without evidence of cerebral dysfunction. The neurological examination, of course, seeks hard evidence of neurological impairment, such as alteration in normal reflexes, abnormal reflexes, asymmetrical failures in sensory and motor response, and EEG abnormalities. However, in recent years, more emphasis has been placed on refined observation of impairments in gait, posture, balance, motor coordination and

control, muscle tone, and the integration of multiple or multimodal stimuli. When the schizophrenic children are grossly subdivided into those who give these evidences of cerebral impairment (organic) and those who do not (nonorganic), a number of empirical findings distinguish the two sub-clusters. For example, the nonorganic children are superior to the organic children in most adaptive functions, including perceptual, conceptual, and psychomotor response. The nonorganic and organic children also differ in regard to family patterns of interaction, psychiatric status of the parents, and in maternal communication. Direct observations of families have tended to confirm that families of nonorganic children are virtually always deviant in psychosocial functioning, while organic children have families which are more heterogeneous in regard to adequacy of functioning and which include average as well as deviant families. A higher proportion of the mothers of nonorganic children than of organic children are schizophrenic. The mothers of nonorganic children have poorer speech and are less clear in their communication. Finally the two groups of children differ in course of development, in response to day and residential treatment, and in changes in specific ego functions. The organic children include the most impaired and most unchanging children. On the other hand, while the organic children respond equally well today and residential treatment, nonorganic children appear to show more progress in residential care, that is, the most comprehensive form of environmental treatment. Neurological appraisal is a

cardinal step in the differentiation of intrinsic and extrinsic influences.

Social Class Position

Schizophrenic children come from families at every level of social class position. Increasingly, too, it has become evident that the social class position of their families is associated with differences among the schizophrenic children. For example, in a recent longitudinal investigation, the schizophrenic children at high, middle, and low social class position differed in mean IQ at admission to treatment and in amount of change in IQ between admission and third year of treatment. Thus, mean WISC Full IQs at admission and after three years of treatment were as shown in Table 5-2.

TABLE 5–2.

SOCIAL CLASS	HOLLINGSHEAD-REDLICH INDEX	MEAN WISC ADMISSION	FULL IQ THIRD YEAR
High	I, II	61.6	68.2
Middle	III	77.2	82.5
Low	IV, V	79.2	89.2

Conclusions

The reader has been asked to accompany me through a complex discussion of the ambiguities and inconsistencies in the construct of childhood schizophrenia. This discussion first stressed that the emergence of the category of childhood disorders termed “childhood schizophrenia” followed the prior evolution of the category of adult disorders termed “adult

schizophrenia" and the confusing consequences of this historic association were noted. Though there was some apparent overlap between the two classes of disorder, they were not completely identical in symptoms and life course. I concluded that it was still wise to study and treat childhood schizophrenia as a set of conditions apart from adult schizophrenia. In addition, the most relevant focus in the study of schizophrenic children was presumed to be on the disturbances in maturation of purposeful functions and on factors influencing these disorders in psychological growth.

Then I discussed the diagnosis of childhood schizophrenia to arrive at a common basis for the classification of schizophrenic children. I emphasized the empirical finding that in spite of careful diagnosis, schizophrenic children were highly diversified in many important abilities and attributes, interpersonal and family experience, social class, and neurological integrity. Paralleling this diversity, a multiplicity of factors would appear to be linked to the adaptive disorders of schizophrenic children. In some children, intrinsic factors were linked to the schizophrenic child's manifestations. Thus, a high percentage of the children gave strong evidence of deficits in neurological and cerebral integrity. In some children, deviations in family organization and functioning seemed to be associated with the schizophrenic child's behavior. In the latter connection, paralysis in parental functioning and unclear maternal communication have been noted.

In view of the heterogeneity of schizophrenic children and the apparent multiplicity of causative influences, there is little doubt that specific and precise therapeutic design to meet the needs of the individual schizophrenic child requires careful assay of his unique psychodynamic dispositions, functional capacities, and developmental experiences. In research and observation, too, it has seemed most profitable to seek a point of view that does not reject the seeming contradiction in observational data but rather rationalizes them. These inconsistencies are more apparent than real since such inconsistencies are inferred only if one begins with the assumption that schizophrenic children are homogeneous and that there is a single cause of childhood schizophrenia. Disparate findings begin to show a pattern if one assumes that schizophrenic children are highly diverse and that the causes are multiple. I have concluded that the key to the discovery of this pattern is the intensive developmental study of individual schizophrenic children. I have also proposed that the many levels of capacity, motivation, and experience need to be seen in dynamic interplay with one another as the child grows. For example, there is little value in merely labeling the social class position of the families of schizophrenic children as high (a currently favored conviction) or low. There is more profit, however, in defining the developmental implications of low or high social class experience for a specific schizophrenic child.

Finally, we have concluded that just as study of the growth of individual

schizophrenic children is essential to dispel current ambiguities, it is equally essential to characterize these children individually by certain pertinent dimensions. The purpose of such characterization of individual children is to achieve homogeneous sub-clusters of schizophrenic children that permit generalization from the data. The present discussion has offered one system for subdividing schizophrenic children in which the dimensions employed reflect empirical experience as well as theoretic considerations.

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Notes

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