

Childhood Schizophrenia Reconsidered¹

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Childhood schizophrenia has tended to be used as a generic term to include an astonishingly heterogeneous mixture of disorders with little in common other than their severity, chronicity, and occurrence in childhood. To add to the difficulty, the term has been employed in widely divergent ways by different psychiatrists. Some make the diagnosis very frequently, others do so quite rarely. A host of different syndromes have been included in the general category of "childhood schizophrenia"—infantile autism, the atypical child, symbiotic psychosis, dementia praecocissima, dementia infantilis, schizophrenic syndrome of childhood, pseudo-psychopathic schizophrenia, and latent schizophrenia to name but a few. In addition, a collection of eponyms have been attached to different conditions—Kanner, Mahler, and Heller, for example, all have syndromes named after them. The diagnostic situation can only be described as chaotic. Clinicians from different centers use the same term to mean different conditions and different terms to mean the same condition. We must conclude that the term "childhood schizophrenia" has outlived its usefulness. It served a purpose—a very important purpose—in drawing people's attention to the fact that psychoses can occur in childhood. But it has ceased to have any scientific meaning or communicative value and it is high time that the term "childhood schizophrenia" was politely and respectfully, but firmly, put into its proper place in the section on "the history of psychiatry." It is a disservice to child psychiatry to imply that today no differentiation is possible among the severe disorders of childhood.

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DRAWBACKS OF AN ETIOLOGICAL CLASSIFICATION

If the term is rejected, what should be put in its place? Here, it is necessary to pause for a moment to consider concepts and purposes. Medical men, in such circumstances, tend to turn to an etiological classification. There is a widespread belief, in my view mistaken, that a classification based on causes is always the best. The most thoughtful and well-considered discussion of this approach to the classification of child psychoses has been presented by Eisenberg (1966). He argued convincingly that progress was being retarded by grouping together disparate conditions under one heading. More precise diagnosis and logical classification were required. He suggested that childhood psychoses should be split into two main subdivisions—those caused by impairment of brain tissue function, and those where structural changes in the brain have *not* been demonstrated. The organic psychoses were then further subdivided according to cause so that there were groups of “toxic” psychoses, “metabolic” psychoses, “infectious” psychoses, and so forth. Eisenberg’s work on classification was a major improvement on what had gone before, but there are serious disadvantages in having etiology as the main principle of classification.

The drawbacks are several, but four require special mention: (1) Reliance on the inadequacies of current methods of investigation; (2) lack of differentiation; (3) implication of one cause-one condition; and (4) unreliability of such a scheme. Eisenberg (1966) was at pains to emphasize that the assignment of psychoses into those with and those without tissue pathology was necessarily tentative in view of the limitations of knowledge. If future studies showed that a disorder now classified as functional was in fact due to some type of tissue pathology, that disorder would change its place in the classification. This would be perfectly satisfactory if the development of knowledge was such that a disorder could clearly be placed in one or another subdivision, but that is not the case. The inadequacies of our tools for neurological investigation are such that there is a considerable chance element in this type of classification. Because of maturational changes, neurological signs may be present at one age but not at another—yet the underlying condition remains the same. Furthermore, there is considerable controversy concerning the conclusions to be drawn from so-called “soft” neurological signs, from nonspecific EEG abnormalities, and from a history of perinatal complications (Rutter, Graham, & Yule, 1970). The effects of these problems were shown in the Maudsley follow-up study of children with infantile psychoses (Rutter, Greenfeld, & Lockyer, 1967). Many children who showed no evidence of neurological abnormality when first seen developed epileptic fits during adolescence. Should their disorders be classed as “organic” or “functional”?

The second drawback concerns the lack of differentiation between clinical psychiatric syndromes. Organic brain pathology may lead to transient confusional states carrying a good prognosis, to dementia with its progressive deterioration leading to death or to a variety of other psychotic states. Thus, metabolic psychoses would group together a brief episode of disorientation, confusion, and hallucinations due to hypoglycaemia; a more prolonged psychosis with depression and delirium due to pellagra and the inevitably fatal dementia of amaurotic familial idiocy. All metabolic causes, it is true, but the psychiatric syndromes take a different form, have a different prognosis, and require a different treatment. It is apparent that an etiological classification may involve such a serious loss of information that it is of little use to the researcher, to the administrator, or to the practicing clinician.

The third objection to a classification by causes is the implication that psychiatric disorders can be attributed to one main cause, and the accompanying assumption that organic causes take precedence over psychosocial or other influences. Undoubtedly, psychiatry would be both an easier and a tidier subject (although probably a less interesting one) if this implication were true, but it is not. This may be illustrated by reference to the Isle of Wight studies of child psychiatric disorders associated with neurological conditions (Rutter et al., 1970). These investigations clearly showed that organic brain dysfunction greatly increased the risk of psychiatric disorder. In fact, psychiatric disorder was about *five* times as common in children with cerebral palsy or epilepsy, as in children in the general population without neurological abnormality. There can be no doubt from these findings that organic factors may play a major part in the genesis of some child psychiatric disorders. Even so, the association was found to be indirect in most cases. The psychiatric disorders in the neuro-epileptic children were most varied. Furthermore, anxiety states, conduct disorders and the like predominated over the traditional "organic" types of disorders. Moreover, within the total group of children with cerebral palsy and epilepsy, the development of psychiatric disorder was influenced by "nonorganic" factors such as family discord and neurosis in the parents, as well as by brain pathology. It is vital to give full weight to the importance of organic brain dysfunction in the causation of child psychiatric disorder, but equally it is essential that this not serve as an excuse to avoid a full assessment of possible adverse influences in the home, in the school, and in the community.

It should not be thought that those espousing an etiological approach are unaware of these considerations. To the contrary, explicit reference to them has been made in discussions of classification (Eisenberg, 1966). Nevertheless, the effect of these three drawbacks on the use of such a classification has often been

underestimated. Of course, it could well be argued that these objections are theoretical and therefore that they should be disregarded as long as the system worked in practice.

Unfortunately, it does not work in practice, as seen by the *American Psychiatric Association Diagnostic Manual* and the *International Classification of Diseases* (ICD), both of which subdivide psychiatric disorders into those with and those without organic brain pathology. Over the last eight years, the World Health Organization (WHO) has conducted a series of international seminars on psychiatric diagnosis and classification. These have included case history and videotape exercises to assess the reliability of classification and to examine the defects in the present international scheme. The third annual seminar was devoted to child psychiatric disorders, and this showed the difficulties in the existing scheme when the exact cause was uncertain. This is best illustrated by giving one of the cases used in the seminar—that of a mentally retarded girl with a psychotic disorder who had had several epileptic fits (Rutter, Lebovici, Eisenberg, Sneznevskij, Sadoun, Brooke, & Lin, 1969). About a third of the psychiatrists diagnosed psychosis, a third mental retardation, and a third chronic brain condition. The psychiatrists agreed that the girl showed all three but they disagreed on which should take precedence in classification. The same difficulties arose in the fifth seminar, held in Washington, devoted to the subject of mental retardation (Tarjan, Tizard, Rutter, Begab, Brooke, De La Cruz, Lin, Montenegro, Strotzka, & Sartorius, 1972). The fourth digit coding of mental retardation classifies cases according to presumed etiology. Fine in principle, but in practice it turned out to be hopelessly unreliable.

If a strictly etiological classification is to be abandoned, what should take its place? Should etiology be disregarded? Certainly not! Etiology is a central feature in any worthwhile diagnostic formulation. The only issue is how it should be catered for in classification. What is needed is a classification scheme which works in practice, which provides clinically useful information, and which has a sound factual basis.

A MULTI-AXIAL APPROACH TO CLASSIFICATION

The WHO seminars suggested that a multi-axial approach to classification would meet these needs and would be preferable to the existing schemes (Rutter et al., 1969; Tarjan et al., 1972). A multi-axial classification is actually no more than a logical development of a multi-category scheme such as the ICD or the APA classification. What it does is simply separate out the different dimensions of diagnosis onto different axes and then provide straightforward rules to ensure that these are used in a systematic and standard manner. Thus, the multi-axial

scheme suggested had four axes: The first specified the clinical psychiatric syndrome; the second, the intellectual level; the third, any associated or etiological biological factors; and the fourth, any associated or etiological psychosocial factors. This scheme was put to the test and was tried out in the ordinary clinical setting by some two dozen child psychiatrists (Rutter, Shaffer, & Shepherd, 1972). The result was a clear preference for a multi-axial approach. It should be noted that the advantages of this system were most obvious in cases where psychiatric disorder was associated with either organic pathology or intellectual retardation. As this is a common occurrence with child psychosis, a multi-axial approach is of special importance in this group of disorders. Its use is again best illustrated by an actual case. Let us take once more the mentally retarded epileptic girl with psychosis. The first axis would record the type of psychosis, the second would note the mild intellectual retardation, the third would specify the occurrence of epilepsy, and the fourth axis would delineate any psychosocial influences. In this scheme it would be unnecessary to decide whether the psychosis led to the mental retardation or whether it was the other way round (a point on which clinicians frequently disagree), and equally it would be unnecessary to decide whether the epilepsy was the cause of the psychosis or merely a secondary contributory factor. In short, the scheme allows unambiguous and meaningful communication between clinicians of different theoretical persuasions—and that is what classification is all about.

If this view of classification is accepted, careful consideration must be given to what sort of subdivision will be provided on each axis. In this paper, only the first, that describing the clinical psychiatric syndrome, will be discussed. The classification of intellectual level, biological factors, and psychosocial influences is equally important, but as these should be classified in the same way regardless of whether the child has a neurotic or psychotic disorder, they will not be considered here. The subdivisions of intellectual level follow from the WHO seminar recommendations on the classification of mental retardation (Tarjan et al., 1972) and the classification of biological factors should be based on the existing ICD classification of physical disorders. The classification of psychosocial influences poses more problems (Rutter et al., 1972), as shown, for example, by the difficulties with the concept of "maternal deprivation" (Rutter, 1972c). The production for a viable scheme for classifying psychosocial influences remains an urgent task for the future.

SCHIZOPHRENIC PSYCHOSIS IN CHILDHOOD

As the title of this paper is "Childhood Schizophrenia Reconsidered," it is appropriate to begin by considering if schizophrenia as we know it in adults can

occur in children. Even in adults, there is some controversy over the limits of schizophrenia and it is known that there are national differences in the use of the term (Cooper, Kendell, Gurland, Sharpe, Copeland, & Simon, 1972). For present purposes the WHO draft glossary definition will be used. This describes schizophrenia as

a group of psychoses in which there is a fundamental disturbance of personality, characteristic distortion of thinking, often a sense of being controlled by alien forces, delusions which may be bizarre, disturbed perception, abnormal affect out of keeping with the real situation and autism.

The description goes on to note that hallucinations, especially of hearing, are common; perplexity may be present; thinking becomes vague, elliptical and obscure; and everyday objects and situations come to possess a special, usually sinister, meaning.

Disorders of this kind have certainly been described in children, and more commonly in adolescence. The most systematic study is that undertaken by Kolvin and his colleagues in Britain (Kolvin, 1971). They investigated 33 cases in which a schizophrenic psychosis began before the age of 15 years. The onset most often began after the age of 11 years at a time when the youngsters were approaching or entering the period of puberty, but there were 13 children in whom the disorder began between the age of 7 and 11 years. There was just one case in which the disorder began before 7 years of age.

In the great majority of cases the psychosis developed against the background of preexisting abnormalities of personality or behavior and in most instances the onset was insidious. The symptomatology was very similar to that seen in adults with schizophrenia. A disorder of thought association was present in three fifths of the children, the same proportion showed thought blocking, and a similar percentage exhibited delusions (most often of persecution). Over 80% of the children had auditory hallucinations and nearly half of these had bodily or visual hallucinations as well. However, no child had bodily or visual hallucinations without auditory ones (Kolvin, Ounsted, Humphrey, & McNay, 1971). Disturbances of mood were usual, and especially in the early stages, perplexity was characteristic. Nevertheless, blunting of affect was present in nearly two thirds of the cases. Mannerisms and grimacing were common and ambitendency was observed in three out of five children.

Quite apart from the symptomatology, the associations with family and other factors in this group of children with schizophrenia were also like those seen in adults. One in ten of the parents were themselves schizophrenic, a figure which

closely approximates that found in adult schizophrenics. There was some tendency for the children to come from a working class background and in a minority of cases the psychosis developed in children with preexisting temporal lobe epilepsy. Most individuals were within the normal range of intelligence but there was a downward shift so that the mean IQ was 86. Obvious psychological precipitating factors were present in nearly half the cases.

Kolvin found only three differences of note between the child schizophrenics and the usual picture of schizophrenia in adults. First, boys outnumbered girls by a ratio of 2½ to 1. This is in keeping with other findings in adolescents with schizophrenia but in adults the sex ratio is usually near equal. Second, there was some delay in speech development (usually transient and mild in degree) in nearly half the children, and thirdly, between a quarter and a third showed evidence (usually circumstantial) of organic cerebral dysfunction. While both these features have been found in adult schizophrenics, the proportion found in Kolvin's series is above that generally present in adults. However, it remains uncertain whether the difference is a real one in that most of the cases were collected from a clinic run by a psychiatrist with a special interest in epilepsy and organic problems.

Altogether, then, it is reasonable to conclude that schizophrenia as we know it in adults can begin in childhood. It does so most often during the pre-adolescent or adolescent period but the onset may be as early as 7 years and, rarely, even before that. Cases of schizophrenia in pre-pubescent children are, nevertheless, quite uncommon. It took Kolvin many years to collect a series of 33. However, when schizophrenia presents as a psychosis in childhood it does so in a way which is basically comparable to that in adults. The disorder seems the same and there is no reason to give the condition a special name. The diagnostic term "schizophrenia" is adequate and there is no need to add the adjective "childhood" any more than one might add the adjective adolescent, young adult, or middle-aged.

What is the nature of this schizophrenic condition? As the condition appears to be similar in children to that in adults, only the main considerations will be briefly mentioned.

For a long time there has been controversy over the genetic basis of schizophrenia because of uncertainties over the sampling and methodology of twin studies. However, the situation has been greatly clarified by the Scandinavian studies of adoptees and no longer can there be any doubt that there is an important and substantial genetic component in the causation of schizophrenia (Rosenthal & Kety, 1968). The concordance rate for monozygotic twins is well above that for dizygotic twins and among adoptees the prevalence

of schizophrenia in the biological (but not the adoptive) parents is markedly increased. The findings demand a genetic explanation.

Even so, it is equally necessary to note that the mode of inheritance and what is inherited remain unknown. Is it a predisposition in terms of personality traits which is inherited or is it a direct vulnerability to a disease which is handed on genetically? We do not know. Also, it is important to observe that the concordance rate in monozygotic twins (although above that in dizygotic twins) is relatively low, especially in the milder cases of schizophrenia. This means that, whatever the genetic components, there must also be some influential nongenetic factors which play a role in causation.

Mednick and Schulsinger's (1972) studies of the children of schizophrenics offer clues as to some of the possible elements in this nature-nurture interaction. They have compared, within a group of children of schizophrenic mothers, those children who broke down with psychiatric disorder and those who did not break down; both groups were also compared with a control group. The sick children were differentiated by their psychophysiological characteristics: In response to a stress (such as a loud noise), they showed an unusually rapid, marked, and transient galvanic skin response which failed to habituate. The development of disorder, however, was further increased if in addition to these psychophysiological characteristics the children were of low birth weight and had had stressful separation experiences. The findings require replication but they offer a nice model of the way in which constitutional factors, perinatal damage, and unpleasant life experiences may interact to produce psychiatric disorder in the offspring of schizophrenics.

Apart from these perinatal and psychophysiological factors, a variety of biological influences have been implicated at various times as possible causes of schizophrenia. For some years, biochemical studies have promised to be on the verge of discovering some metabolic anomaly specific to schizophrenia but so far no such metabolic abnormality has been unequivocally demonstrated (Kety, 1972). Perhaps the one association that has stood the test of time is that between temporal lobe epilepsy and schizophrenia. It seems clear that schizophrenia is particularly likely to develop in individuals with temporal lobe epilepsy (Slater, Beard, & Glithero, 1963; Davison & Bagley, 1969). The psychosis is probably secondary to the brain lesion rather than the fits, and it is usual for schizophrenia to be evident only some dozen years later. However, this length of delay is not invariable as shown by Kolvin's finding of temporal lobe epilepsy in some adolescent schizophrenics. Of course, this finding applies to only a small minority of schizophrenics and it is very uncertain whether it has much relevance to the bulk of cases.

Finally, there is the question of the extent to which psychological and social influences play a part in the genesis of schizophrenia. Many claims have been made that schizophrenia is due to disturbed parent-child relationships. The theories on this issue are multiple and, to some extent, contradictory. Most rely heavily on poorly controlled anecdotal evidence, but there are a few well planned systematic investigations. Mishler and Waxler (1968) studied the families of adolescent schizophrenics in an experimental problem-solving situation. The pattern of verbal interaction was measured in a way which allowed the sequence of conversation to be studied. They found that the level of patterning—that is to say, the predictability or rigidity of responses—was somewhat greater in the families of schizophrenics, although the differences from normals were fairly small. However, this difference applied only to the parents' interaction with the sick child, not to their interaction with the healthy children in the family. This observation immediately raises the question as to whether the pattern of family interactions found, measured *after* the fact of the child's illness, is a *response* to the illness and not in any sense a causal agent (Waxler & Mishler, 1970). There is no answer to that question as yet.

Singer and Wynne (1965) have used a different approach to study family communication patterns, having developed a method of scoring the transcripts of Rorschach tests of parents. They have claimed marked differences between the findings for parents of schizophrenics and parents of neurotics or normals (Wynne, 1968). However, a carefully controlled replication of their work by Hirsch and Leff (1971) has failed to confirm their results, and it seems likely that selective referral biases or diagnostic differences explain the earlier findings, although further studies are required to resolve the issue.

Whether or not there are specific patterns of parent-child interaction which precede and cause the onset of schizophrenia now seems somewhat dubious, although the matter is far from settled. On the other hand, there is good evidence that psychosocial factors do play a part in the precipitation of schizophrenia, and in its course once the disorder has started. Brown and Birley have shown convincingly that the onset of florid symptom is often preceded during the previous three weeks by a significant change in the patient's social environment (Brown & Birley, 1968; Birley & Brown, 1970; Brown, 1972). The time relationships are such as to suggest that the environmental stress has precipitated the psychosis, but not caused psychosis in an individual who would not have had it otherwise. However, it is not only in the precipitation of schizophrenia that family factors may play a part. Brown, Birley, and Wing (1972) have also shown that overinvolvement with a relative who expresses a lot of critical feeling is strongly associated with symptomatic relapse during the nine months after the patient's discharge from hospital.

In summary, it would appear that schizophrenia develops as the result of a complex pathogenic process which probably involves an interaction between certain genetically determined characteristics (possibly involving the individual's psychophysiology), certain psychosocial precipitants, and, in some cases, damage to the brain caused during the period of development. The nature of this process is ill-understood in adults and almost totally unexplored in children.

PRECURSORS IN CHILDHOOD OF SCHIZOPHRENIA IN ADULTS

So far, the discussion has been concerned with schizophrenia in childhood when it takes the same psychotic form as that seen in adult life. But this is too narrow a view. It has often been observed that schizophrenia in which florid symptoms do not appear until late adolescence or early adult life has often been preceded by oddities of personality or behavior which have been evident during childhood. It could well be that schizophrenia occurs in childhood but does so in a form which differs from that in adult life. This is certainly possible in that it is well known that there are several disorders (e.g., the hyperkinetic syndrome) in which symptoms change with age (Rutter, 1972a). Accordingly, it might be possible to identify a syndrome, which differs in symptomatology from adult-type schizophrenia, but which nevertheless can be reliably identified as the childhood form of the adult condition.

Evidence on this question has been increasing over the last 10 or 15 years by virtue of follow-up studies of normal populations and child psychiatric patients, by retrospective studies, by investigations of monozygotic twins discordant for schizophrenia, and by anterospective longitudinal studies (Offord & Cross, 1969; Rutter, 1972a). It is well demonstrated that individuals who develop overt schizophrenia in adult life have frequently shown abnormalities of a nonpsychotic type in childhood. The associations between low birth weight, psychophysiological characteristics, and schizophrenia have already been discussed, but there are also behavioral and cognitive precursors of schizophrenia.

It seems that schizophrenia is more likely to develop in individuals of somewhat below average intelligence, with poor academic achievement at school, who were somewhat socially isolated, who showed oddities of personality, and who had impaired relationships with their peers. The children of schizophrenic mothers appear to have poor motor development (Fish, 1971; Mednick & Schulsinger, 1972) and it may be that this also is a presage of schizophrenia. But this requires confirmation. In personality traits, children who later develop schizophrenia most commonly have shown a mixture of introverted and extroverted characteristics. When they have exhibited overt psychiatric disorder

in childhood, it has not followed any distinctive pattern; there may have been neurotic or antisocial problems but perhaps most commonly the disorders showed features of both. The traditional view that shy withdrawn children are most likely to become schizophrenic has not been confirmed. On the contrary, they seem to have a rather good prognosis.

These findings are important and clearly indicate that more attention needs to be paid to the childhood characteristics which portend the later development of schizophrenia. Offord and Cross (1969) have argued that the results are sufficiently striking that schizophrenic patients should be divided into two main groups, those who have difficulty in childhood before the onset of the overt schizophrenic illness and those who do not. A comparison of these two groups should prove rewarding. So far, little is known about how they differ, There is some suggestion that abnormalities in childhood are more characteristic of schizophrenia in males. Kolvin's finding that schizophrenic psychoses beginning in childhood are commoner in boys is in keeping with this suggestion but further investigations are called for.

In the meantime, it must also be concluded that there is nothing particularly distinctive about the child psychiatric disorders which precede the onset of schizophrenia. It would be a great advance to identify a syndrome which could be diagnosed as "pre-schizophrenia" but so far this is not possible. All that is sure is that among the mixture of children attending child psychiatric clinics some will have disorders which are a forerunner of schizophrenia in adult life. Unfortunately, we do not know how to tell which children will have such disorders.

DISINTEGRATIVE PSYCHOSIS

If we now return to overt psychoses and move down to a lower age period, the next group of psychoses is that of the disintegrative or regressive psychoses. Heller (1930) provided the first description under the heading of dementia infantilis. In this condition, development appears normal, or near-normal, on all counts up to the age of three or four years at which time there is profound regression and behavioral disintegration. Often there is a premonitory period of vague illness and the child then becomes restive, irritable, anxious, and overactive. Over the course of a few months there is an impoverishment and then a loss of speech and language. Comprehension of language deteriorates and intelligence often declines although an intelligent facial expression is usually retained. There is a loss of social skills, impairment of interpersonal relationships, a general loss of interest in objects, and the development of stereotypies and mannerisms (Rutter, 1972b).

This is the characteristic clinical picture but there is considerable individual variation in both the form and the development of the disorder. It is unlikely that this is a single condition, but owing to its rarity there has been little systematic study of this group of disorders. Makita (1966) and Kolvin (1971) both found that psychoses beginning at 3 to 5 years of age were the rarest of child psychoses. The condition may follow overt brain damage—such as measles encephalitis—but also it may occur in the absence of any known organic brain disease or damage. Usually there are no abnormalities on formal neurological examination, but postmortem studies have generally shown cortical degeneration, sometimes of a lipid type. It is evident that many cases are due to widespread organic disease of the brain but there are cases in which this does not seem to be so. Psychogenic varieties have been suggested but evidence on this proposition is lacking. The prognosis is poor, with the children often remaining without speech and severely handicapped in their intellectual development. A few improve considerably and it remains uncertain whether they constitute the same or a different disorder.

INFANTILE AUTISM

Moving again to an even earlier period of development, we must now turn to infantile autism—the “psychosis” most characteristic of childhood. This syndrome was first delineated by Leo Kanner in a paper which has rightly become a classic (Kanner, 1943). His original 11 cases all showed abnormalities from infancy, but it has become apparent since Kanner’s first report that the disorder may begin at any time up to about 30 months of age—but not after then (Rutter, 1971a). Most series show that some 4 out of every 5 autistic children never had a period of normal development, but in some 20% the disorder appears to involve a regression following an apparently normal maturation up to that time. Whether this minority group differs in etiology is not known but in symptomatology and outcome they are closely similar to the cases with the more usual picture of deviant development from the beginning.

Kanner’s precise description applied to a fairly circumscribed disorder, but unfortunately the term “infantile autism” has been applied by some people to a wider and wider group of disorders. Accordingly, it is necessary, before any further discussion of the syndrome, to make clear that this paper uses the more narrow use of the term. It may be appropriate once more to make reference to the WHO draft glossary description which notes that

Responses to auditory and visual stimuli are abnormal and there are usually severe problems in the understanding of spoken language.

Speech is delayed and, if it develops, is characterized by echolalia, 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 5 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.

In short, infantile autism is a disorder present from early infancy in which the three cardinal features are a failure of social development (of a specific type), a deviant and delayed language development, and various ritualistic activities (Rutter, 1971a). It is evident from this description that the term "autism" has rather misleading implications (Koupernik, 1971; Eisenberg, 1971). Autism means a withdrawal into fantasy but this is not what happens in the syndrome of autism. The schizophrenic may retreat from reality into fantasy, but the autistic child does not retreat, rather he fails to develop social relationships—a crucial distinction. Furthermore, so far as one can tell, the young autistic child has a deficiency of fantasy rather than an excess. It is hard to suggest what better alternative there was to Kanner's choice of term in 1943, but it is essential to recognize that the word autism as used for the syndrome of "infantile autism" means something quite different than the word used for the symptom in schizophrenia.

Does this mean, then, that autism is a different disorder from schizophrenia? It does. Originally, infantile autism was tentatively conceived as possibly the earliest manifestation of schizophrenia. This now seems most unlikely (Kolvin, 1971; Rutter, 1968).

As is evident from what has been said already, the symptomatology of the two conditions are quite different. One involves a failure of development, the other a loss of reality sense after development is well established. Delusions and hallucinations are characteristic of schizophrenia but are quite rare in infantile autism. This could be the result of symptom modification due to the stage of maturation but it should be noted that delusions and hallucinations are still rare even after autistic children reach adulthood (Rutter, 1970). The course of the disorder in the two conditions also differs. Marked remissions and relapses are

well recognized in schizophrenia but are decidedly uncommon in autism where a relatively steady course is much more usual. There are also striking intellectual differences between the disorders. Mental retardation is a common accompaniment of autism but it is much less frequently associated with schizophrenia. Furthermore, the characteristic pattern of IQ sub-test scores (high on visuo-spatial tasks and low on language skills) found with infantile autism is not a feature of schizophrenia. The sex distribution in autism differs sharply from that in schizophrenia. Schizophrenia in adults occurs with about the same frequency in men and women whereas autism is 3 or 4 times as common in boys as in girls. This could be a developmental difference in that schizophrenia in later childhood also shows a male preponderance. Organic factors also differentiate the two disorders. Perinatal complications are commoner in autism and epilepsy is much commoner. Moreover, the pattern is different even when organic factors are present. When schizophrenics are epileptic, the epilepsy is usually of the temporal lobe type. This is not so in autism.

Some of these differences might be explicable as consequences of a different age of onset and hence as due to the modifying influence of ontogenetic factors. However, there are three differences which are difficult to account for in this way. First, the social class of the parents of autistic children is most unlike that of the parents of schizophrenics. A high proportion of the parents of autistic children are of above-average intelligence and of superior socioeconomic status. This is not an artefact of referral policies to clinics, as the findings have been confirmed in a total population epidemiological study by Lotter (1967). In contrast, the social background of schizophrenics is the same as that of the general population. Secondly, the age of onset of psychoses in childhood follows a markedly bipolar distribution, as shown by both Kolvin (1971) in England and Makita (1966) in Japan. There are peaks under 3 years and over 11 years with a very decided trough in between. This strongly suggests a discontinuity between autism and schizophrenia. Thirdly, the family history of schizophrenia in the two conditions is quite different. All the major studies of infantile autism have shown that schizophrenia is rare in the parents and sibs of autistic children, whereas schizophrenia occurs in about 10% of the parents of schizophrenic adolescents or adults.

All these findings point to autism and schizophrenia being different disorders. What evidence is there that they are similar and why was autism once regarded as a variety of schizophrenia? Probably the idea arose because at the time autism was first described child psychiatrists were much involved with the issue of how schizophrenia was manifest in childhood. Rather than regard autism as entirely different, it was reasonable at first to suggest that it might constitute a very early

variety of schizophrenia. However, as evidence has accumulated it has become apparent that the two conditions have little in common apart from severity, chronicity, and an impairment (albeit of a different type) of social relationships. Almost the only positive evidence is Bender's (1953, 1971) claim that autistic children when grown up behave like schizophrenics. However, the group she followed was quite heterogenous (only a few children would be called autistic by Kanner's criteria), her use of the term schizophrenia is far wider than the WHO definition followed here, and her findings have not been confirmed by the systematic follow-up studies of better defined groups of autistic children. The inescapable conclusion is that, so far as one can judge from the available evidence, autism has nothing to do with schizophrenia.

If autism is not schizophrenia, what is it? Many theories have been put forward. These have been discussed at some length previously (Rutter, 1968; Rutter & Bartak, 1971) and the arguments will not be repeated in this paper. Rather, 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.

There is both clinical (Rutter, Bartak, & Newman, 1971) and experimental evidence (Hermelin & O'Connor, 1970; Hermelin, 1971; Frith, 1971) in favor of this proposition. Thus, retardation of speech and language is an almost invariable manifestation in infantile autism, and a lack of response to sounds is frequently the first symptom to be noted. Apart from the level of IQ, language is also the most important prognostic factor. The pattern of cognitive abilities in autism shows that the children are deficient in language skills even on tests that do not require speech. This means that the failure to speak results from a basic impairment in language function, not just from an absence of motivation to speak. Not only is the autistic child retarded in language, but also his *pattern* of linguistic abilities is significantly different from that exhibited by either normal or mentally retarded children. The autistic child is particularly poor in his transfer of information from one sensory modality to another, in his understanding of the meaning of the spoken word, and in his use of gesture. The deaf child or the child with a developmental language disorder will use gesture and mime to compensate for his inability to speak. The autistic child does not do that. There are other differences between the language impairment in autism and that in other disorders. Pronominal reversal and echolalia, which probably reflect defects in comprehension, are much more common in autism than in other disorders involving speech delay. Autistic children differ from deaf, "aphasic" and other children in having both a more severe and a more

widespread language handicap. It will be appreciated that this refers to a global *language* impairment and not a speech defect. Failure to speak is but one manifestation of a much wider problem. The impairment includes the *understanding* of language as well as its production, it includes language modalities (such as gesture) other than speech, it extends to the way in which language is used for communication as well as a knowledge of syntax and semantics, and it involves symbolic thought as well as language production.

The experimental studies of Hermelin, O'Connor, and Frith have carried the issue further. They have shown that autistic children are particularly unresponsive to verbal stimuli and that they make little use of concepts in memorizing. The verbal recall of autistic children is relatively independent of the meaning of what they hear. More recent experiments suggest that autistic children are not only impaired in their appreciation of the grammatically structured aspects of language but also lack the ability to associate words semantically. This defect may not be confined to auditory material. The findings suggest a central defect in the processing of any sort of coded, meaningful, or temporally patterned stimuli. Such a defect is likely to severely impair the infant's interaction with the environment and it is suggested that these specific cognitive defects are the cause of the social and behavioral abnormalities in most cases of autism. The view has profound implications for treatment, as has been argued previously (Rutter & Sussenwein, 1971).

The view that autism is based on some form of central cognitive disorder now has widespread (but not universal) acceptance in view of the very substantial weight of evidence. However, the type and limits of the cognitive disorder are more in dispute. It has been argued here that the disorder primarily involves language but others have suggested that sensori-motor integration or perception are equally important. The difficulty in resolving this issue is that autistic children commonly have multiple handicaps. Thus, the majority are mentally retarded as well as autistic. Accordingly, it is quite true that autistic children may have perceptual, sensory, and other handicaps as well as language impairment. But that is not the issue. Rather, the question is what cognitive defect is both *necessary* and *sufficient* for the development of autism? In other words, can autism develop on the basis of a global language impairment alone or must there also be perceptual and sensori-motor anomalies? My colleagues and I have attempted to answer this question by comparing a group of autistic children of normal intelligence (that is, with no other handicaps so far as we know) with a group of children with a developmental receptive language disorder (Rutter et al., 1971; Bartak, Rutter, & Cox, 1972). The findings show that autism *can* and does develop in children with a severe and global language

impairment but of normal ability on visuo-spatial tasks and without any overt perceptual handicap. In view of the fact that the reverse has not been demonstrated, it is reasonable to tentatively conclude that the basic defect in autism must involve symbolic language functions but it need not involve any other handicaps. Of course, the finding must be replicated in other studies but the evidence so far is reasonably clear-cut.

What the cause of this cognitive defect is remains unknown. Undoubtedly, sometimes there is organic brain disease or damage, as evidenced by the frequent development of epileptic fits during adolescence and the few cases with identifiable conditions such as toxoplasmosis or congenital lues. Whether in other cases there could be a developmental or a genetic disorder remains a possibility still to be tested (Rutter & Bartak, 1971). What is likely, however, in view of the very variable outcome and the very variable neurological findings is that biologically speaking the underlying pathology will probably prove to be quite heterogeneous.

The next issue is whether this cognitive defect is sufficient to cause autism or whether an interaction with some other variable (such as an environmental influence) is necessary. There have been numerous suggestions that the parents of autistic children show various specific abnormalities of behavior and that there are identifiable defects in parent-child interaction. This issue has been reviewed previously (Rutter, 1971b; Cox, Rutter, Newman, & Bartak, 1972) with the conclusion that there is no evidence of any such abnormalities in family function or family relationships, as judged by studies using a variety of research techniques. Not all the possibilities have been systematically tested but the findings so far are negative. The only exception is the consistent observation that the parents include a disproportionate number of professional and other middle-class individuals. No adequate explanation of this finding is available but it does not necessarily point to a psychogenic process.

Of course, the argument that autism is not primarily due to psychogenic factors does not mean that environmental influences are unimportant in the development of autistic children. On the contrary, they are as susceptible as are other children to family and other influences and it is necessary to use these in treatment (Rutter & Sussenwein, 1971). The argument is simply that psychogenic factors probably play no *essential* role in the causation of the basic disorder; but they are likely to be important in their effect on the course of the disorder as they may be in any disorder, organic or functional.

OTHER PSYCHOTIC DISORDERS IN CHILDHOOD

The descriptions given so far imply a rather sharp demarcation between the various psychotic disorders in childhood. This is correct insofar as it reflects

basic differences between the three main types of disorder discussed—schizophrenia, disintegrative psychosis, and infantile autism. However, it is essential to add there are children, a substantial minority, who do not fit easily into any of these categories, and it is necessary to have another category for “other psychoses of childhood.” Inevitably, these are rather a mixed bag and as further knowledge accumulates it should be possible to categorize them more precisely.

First, there are a number of cases in which disorders otherwise similar to infantile autism develop after the age of 30 months. Whether they constitute simply examples of autism of unusually late onset or whether they represent a different type of disorder remains uncertain and for the moment they are best classified separately in this “other” category. Secondly, there are rare cases in which severe disorders, apparently involving a loss of reality sense, but in which there is not the profound regression characteristic of “disintegrative psychosis,” develop at an age of about 3 or 4 years. They, too, are best included here. Thirdly, there is a large number of nonspecific disorders of psychotic intensity which usually arise in children with severe intellectual retardation and/or organic brain disease. Frequently they have some, but not all, of the essential features of infantile autism. Again, in order to retain the relatively homogeneous character of the three groups, they are also best placed in the “other” category.

CONCLUSIONS

In this review of the mixture of disorders sometimes included under the portmanteau term “childhood schizophrenia” limitations of space have forced a concentration on symptomatology and etiology. As one would expect, there are also differences in prognosis and outcome and it should have been obvious that there are also very important implications for treatment. In infantile autism the main goal of treatment is aiding the development of social and language skills in a child whose cognitive handicaps have impaired development. In schizophrenia, the aim is to restore equilibrium in an individual who has temporarily lost it after development is well established. In disintegrative psychosis, the problem is to restore function and to aid the recommencement of development in individuals who have suffered a severe regression in development, often following organic brain disease. Each of these situations requires rather different approaches.

Equally, however, it should not be thought that there is one treatment for one syndrome. We are dealing with individuals suffering from disorders, not with disembodied disease states. As indicated in the introduction, the discussion has been restricted to the first axis of a multi-axial classification—the axis dealing

with clinical psychiatric syndrome. But that is not enough in itself. Treatment and prognosis also depend on what is on the other axes. Thus, prognosis in infantile autism is heavily influenced by the child's intellectual level (Rutter, 1970) and that would be noted on the second axis in the WHO scheme. With all the different disorders mentioned, the intellectual level may vary from the very low to normal or even above normal and this is an essential part of diagnosis. Because the IQ is so variable even within one clinical syndrome, it is important to classify it separately.

The same issue applies to biological factors. It should be apparent from what has been stated already that there is no one-to-one association between the psychiatric syndrome and physical disease. Neurological disorders may be associated with autism, with schizophrenia, or with disintegrative psychosis. The nature of that disorder, whether it be a treatable metabolic disorder or an incurable degenerative disease, will again greatly influence prognosis and treatment. Because there is an inconstant association between the physical disease and the psychiatric syndrome, this too required separate classification. On the WHO scheme it constitutes the third axis.

Parallel with this, it is equally important to note associated or etiological psychosocial influences which will help determine the particular therapeutic approach used as well as having an effect on prognosis. In all the conditions discussed, the environmental situation will influence the children's development and in some instances it will have played a part in precipitating the onset of this disorder. On the WHO scheme this constitutes the fourth axis.

Both now and in the introduction there has been some discussion of a multi-axial approach to classification because it is thought that the traditional medical model of one diagnostic term for each disease is inappropriate for the needs of psychiatry. Such a model implies a unitary cause theory of disease and that is quite unsuitable in psychiatry where multi-factorial etiology is more usual.

Because the situation is complex, some clinicians have wanted to abandon diagnosis but that is a counsel of despair which suggests more chaos than is in fact the case. As Eisenberg (1966) has cogently argued, differential diagnosis is no mere academic exercise, it is the very stuff of medicine. It could be added that it is also the very stuff of psychology and of child development. These subjects may not deal with disease states but it is equally necessary to have classification to serve as efficient tools of communication with which to describe the phenomena and the concepts with which we deal.

It is with that in mind that an attempt has been made to replace the unsatisfactory diffuse and meaningless term "childhood schizophrenia" with a

more satisfactory means of classification. It has been suggested that this can only be accomplished by some form of multi-axial system of classification which can take account of intellectual level, biological influences, and psychosocial factors as well as clinical psychiatric syndromes. For this purpose, the proposals emanating from the World Health Organization seminars have been used as a basis. It has been argued that, so far as clinical syndromes are concerned, the term "childhood schizophrenia" includes at least three main disorders differing in age of onset: Autism, schizophrenia, and disintegrative psychosis, which constitute disparate conditions requiring separate classification. In addition, there is an ill-defined group of disorders, not so far clinically identifiable, which are the nonpsychotic precursors of schizophrenia. Lastly, there is a mixed group of disorders, less well understood, which do not fit readily into the above scheme. Like any classification, this only provides a crude summary of existing knowledge but if it has any value it will provoke further research to fill in the gaps and show where it is mistaken so that a better scheme may come to take its place.

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