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Childhood psychoses and their borderlands

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In this chapter we have reviewed evidence concerning the origins of the childhood psychoses. We have also re-examined evidence for the distinction between autism and schizophrenia in childhood and have looked at the place of Asperger's syndrome and borderline states within the classificatory system. In the latter two conditions, we have explored possible links with psychotic disorders of childhood.

This chapter examines the concept of functional psychosis in relation to childhood by reviewing evidence concerning the existence of underlying causes. It is confined to those psychotic disorders which are neither manic nor depressive. This leaves three broad syndromes consisting of infantile autism, which has an onset in infancy;[1–8] disintegrative psychoses, which have an onset in the pre-school years and late onset psychosis (childhood schizophrenia), which has an onset in the school years.[3]

The rare disintegrative psychoses are preceded by near normal development during the infancy period. Subsequently there is a progressive and rapid downhill course with deterioration in speech, language, cognition, behaviour and social relationships. A number of cases follow obvious organic illnesses, such as measles and encephalitis; but in those who have no obvious preceding illness, there are often post-mortem findings of organic cortical degeneration as seen in the lipodoses or leukodystrophies. Given the strong evidence of underlying organic aetiology, the disintegrative psychoses will be excluded from further consideration.

in the autistic group. This was on the basis of perinatal anomalies, electroencephalograph abnormalities and seizure disorder. Temporal lobe epilepsy was found in three of the schizophrenic group but in none of the autistic group.[7] Fifthly, as Rutter[19] pointed out, the course of the two conditions differs markedly, autism having a characteristically steady course, while childhood schizophrenia has a relapsing and remitting pattern. Finally, there is a marked male prevalence in autism compared to a near parity between the sexes in childhood schizophrenia. More recent research has provided comfirmatory evidence that autism and childhood schizophrenia are distinct diagnostic entities.[20] This study was of particular interest since DSM III[21] adult-derived criteria were used.

Are the psychoses of late childhood and adulthood manifestations of the same diagnostic entity?

Having differentiated infantile autism from childhood schizophrenia, what are the grounds for considering childhood and adult schizophrenia to be expressions of the same diagnostic entity? Given that we can convincingly diagnose schizophrenia in childhood using DSM III[20] and Schneiderian criteria,[4] we require good evidence of aetiological differences between adult and childhood schizophrenia in order to support a separate category for each within the classificatory system. What then is the nature of the evidence?

1. *The epidemiology of the conditions.* Kolvin (1971)[3] comments on the rarity of childhood schizophrenia. Beitschman (1985)[22] has calculated that the treated prevalence of schizophrenia under the age of 15 is some 50 times less than that between the ages of 15 and 54 years. However, this finding is not inconsistent with a stress diathesis model, which holds that very few individuals within the population at risk will have sufficient biological loading and environmental stress to produce an early presentation. Further, a comparison of the sex incidence of adult and childhood schizophrenia reveals a remarkable similarity (provided autistic contamination is rigorously excluded).[22-24]

2. *Can pre-morbid characteristics differentiate between the groups under consideration?* The answer must be emphatically in the negative. The pre-morbid characteristics of schizophrenia, whether presenting in childhood or during adult life, are of low specificity. Those patterns of pre-morbid behaviour which do

schizophrenic children suffered from schizophrenia, and concluded that there was a genetic connection between childhood and adult schizophrenia. Hanson & Gottesman[35] have reviewed the genetics of schizophrenia in childhood. They calculate that the parents of childhood schizophrenics have an 8 to 9% rate of adult schizophrenia. This is around twice the 4% figure Kessler[36] calculated when reviewing the risk of parental schizophrenia in adult schizophrenics. Such evidence of a heightened risk of schizophrenia in the parents of childhood schizophrenics, when compared to adult schizophrenics, is consistent with a polygenic theory of aetiology. Accordingly those individuals with a higher genetic loading will present earlier, and with more intractable diseases, and will be expected to have a higher prevalence of schizophrenia in first degree relatives.

We began this sub-section by emphasising that both adult and childhood onset schizophrenia could be diagnosed using both DSM III[21] and Schneiderian criteria, and that we would require strong evidence to support their separation into distinct categories within the classificatory system. On balance our review leads us to conclude that a hypothetical distinction between childhood and adult onset schizophrenia cannot be sustained.

BIOLOGICAL AND GENETIC FACTORS IN AUTISM

There is a great deal of evidence to suggest that autism may be the common behavioural expression of a wide diversity of organic-cum developmental influences.

1. Presumptive evidence of brain insult has been obtained from studies of perinatal complications, with particularly high rates being reported in clinical studies[7,37,38,84] but less so in population studies.[39,40]

2. In about half of the cases in the two major UK hospital series there is evidence of brain dysfunction.[7,41] Further, up to one autistic patient in three in the course of time develops epileptic fits.[7,41,42] EEG studies confirm this evidence with high rates of clear-cut abnormalities—i.e. spike, or spike and wave activity[7,43–45]—while an excess of abnormalities in evoked potentials during waking and sleep has also been shown.[46,47]

3. Certain organic conditions appear to have a close relationship with infantile autism; for instance, it occurs 100 times more frequently in children whose mothers have had Rubella than in the

caps of impairment of language perception, motor ability and behaviour described by Wing & Wing[58]—'A number of different brain functions could be affected by, for example, a single genetic or biochemical abnormality, or anatomical proximity could make different brain centres vulnerable to the same lesion.' On this basis, these workers assert that any condition which produces abnormality or delayed maturation of relevant brain areas could theoretically lead to the impairments of infantile autism.

7. Finally, the study of the biochemistry of infantile autism has revealed a number of interesting possibilities. Hyperserotonaemia has been implicated in a sub-group of rigorously diagnosed autistics, the raised level not being simply related to the degree of mental retardation[54] or to a deficiency in monoamineoxidase.[60] Investigation of maternal serotonin levels has revealed a correlation with levels in their autistic children.[59] Cohen & colleagues assert that they were able to identify a sub-group of autistic children and correlate CSF biochemical disturbances with behavioural and motor abnormalities. However, their ability to differentiate between broader diagnostic groups was less successful, there being widespread variation within each group. Gillberg & colleagues[61] studied the urinary excretion of peptides and protein-associated peptide complexes in infantile autistics, non-autistic psychotics, a group identified as having minimal brain dysfunction, a mental retardation group and a normal group of children. They found that 54% of the autistic children and 17% of the children with other psychoses showed a distinct pattern that was not seen in any of the other groups. Approximately half of the autistic group displayed a specific urinary chromatographic profile: the overall severity of the autistic symptoms in this group was greater than in the remainder of the autistic children. The authors were cautious in their interpretation of the results speculating on the possibility that the abnormal profiles were a result of high motor activity or possible dietary factors. However, they conclude that whatever the origin and mode of action of the identified peptides they almost certainly reflect an underlying biological abnormality. Nevertheless, in most studies there has been insufficient rigour in primary definition of sub-groups. There remain questions about assay procedures and control of dietary factors and motor activity. Many findings are non-specific and have not been replicated. Undoubtedly, organic brain disease can be implicated in autism. Overall, the research findings suggest that infantile psychosis is aetiologically heterogeneous. Each of the

tive, behavioural and language abnormalities seen in autistic children represents the final common expression of a variety of underlying organic and developmental influences.

THE BORDERLANDS OF CHILDHOOD PSYCHOSES

Asperger's syndrome and schizoid personality disorder in childhood

In both the European and American literature there has been an abiding interest in the syndrome described by Asperger.[67] This syndrome comprises gross lack of skills in social diplomacy, associated with a degree of naivity leading to impairment of social relationships.

Interest has centred in particular on the place of this syndrome within the classificatory system. It appears to lie in a classificatory 'no man's land' between autism on the one hand and schizophrenia on the other. If we are correct in our assertion that there is discontinuity between autism and schizophrenia, then is Asperger's syndrome a related personality disorder, or perhaps a mild psychotic variant of either condition? Furthermore, what is the relationship (if any) between Asperger's syndrome and the adult-derived concept of schizoid personality disorder?

The nature of the debate is exemplified by the work of Lorna Wing[68] on the one hand and by that of Wolff & colleagues[68–71] on the other. A starting point for our analysis will be the essential features of Asperger's description of the syndrome.[72–74] These include a striking male preponderance, diagnosis after the first 2 years of life, unusual use of spoken language including pronominal reversal, pedantic aprosodic speech, and use of stereotyped phrases. There are abnormalities in non-verbal communication characterised by lack of affective contact; stereotyped movement, obsessional attachment to certain toys or surroundings, and a preoccupation with rotatory movement is also described. Finally, Asperger regarded children displaying this syndrome as having special abilities in the areas of logic and abstraction.

From the above description it is easy to see why a number of workers have regarded Asperger's syndrome as a variant or milder form of childhood autism.[68,74,75]

Wolff & Barlow[70] on the other hand, have drawn attention to similarities between the adult-derived concept of schizoid personality disorder and Asperger's syndrome. These authors describe

significantly more than the normal controls. While the schizoid personality disorder children used fewer emotional constructs than the autistic children, this finding is diminished by the fact that the autistic group used emotional constructs slightly more often than the normal children!

The authors conclude that the schizoid personality disorder children lie 'intermediate in their functioning between autistic and normals'. They refer to them sharing: 'autistic children's stereotypy, their tendency to impose patterns, some of their linguistic handicaps and their lack of receptiveness for meaning'. Nevertheless, despite the weight of evidence in favour of an association between the autistic and schizoid groups the authors seek to explain this away by saying that the schizoid group 'were poorly motivated in tasks of cognition and memory'. The weight of evidence does not support this position. In a subsequent report, Wolff & Chick[69] highlight the clinical similarities between schizoid personality disorder and autism but these are dismissed—apparently on the basis that 'they did not exhibit the three cardinal features all beginning under 2.5 years'.

Wing[68] considered that schizoid personality disorder is a vague and ill defined concept, the roots of which lie in a supposed relationship to schizophrenia. We would agree that a distinction between autism and Asperger's syndrome has not been demonstrated; neither does a separation of the two conditions make sense on theoretical grounds, nor in terms of practical implications. Furthermore Asperger's syndrome shares so many features with childhood autism, not least its massive male predominance, that its biological link must be with that condition. We conclude that the term schizoid personality, given its implicit associations with schizophrenia, is inappropriate in the context of Asperger's syndrome.

Thus far there has been insufficient research into the closeness of links between Asperger's syndrome and autism. On the basis of symptomatology alone, Asperger's syndrome could be classified as a mild variant of autism,[68] but the pattern and severity of features are insufficient for it to be considered a psychosis. The evidence suggests that it is best classified with the personality disorders of childhood. Van Krevelen[76] has postulated that such a personality disorder could be turned into an autistic psychosis by earlier brain damage, but so far insufficient is known about the aetiology of Asperger's syndrome to confirm or refute this hypothesis.

Poor impulse control and suicidal threats are also described. Some of these children are said to be depressed and withdrawn and show a sense of futility or enraged hostility. The overall impression is of a child with a poorly integrated sense of self and in whom transient psychotic states may occur, though the abiding state is not one of psychosis. It is arguable that such a miscellany of behavioural and affective symptoms could equally well be encompassed within the well known mixed conduct-neurotic disorder category. Certainly Stone (1981) in his review[77] of the borderline syndromes suggests that it is difficult to apply the concept meaningfully in adolescents and that it is probably not a term that is justified in connection with children. Although borderline conditions are questionable as diagnostic entities, little attention has so far been given to the schizotypal variant.[79] In the adult literature, at least, family genetic data is suggestive of a link with schizophrenia.[82]

SUMMARY

First, autism and childhood schizophrenia are undoubtedly aetiologically distinct entities. In addition we find there are no good reasons for placing childhood schizophrenia in a separate category from adult schizophrenia. Second, from a review of the available evidence, we regard Asperger's syndrome as a condition which is allied to childhood autism. We agree with Wing that the schizoid personality concept has little utility, being particularly misleading because of its implied association with schizophrenia. It would be fair to say that we have even less confidence in the usefulness of the borderline concept in childhood.

Finally, what is the current status of the concept of functional psychosis as applied to 'those psychic changes for which no physical cause can be found, and where at present there is, in the somatic sphere, no real ground for supposing such causes exist'?[83] Having identified many biological factors in the psychoses of childhood, especially in autism, it seems that 'grounds for supposing such causes exist' have tended to increase with the sophistication of our investigative techniques, so that the term 'functional' has become correspondingly less useful with the passing years. Some may consider it retains currency in the borderland conditions. However, the evidence for a relationship between Asperger's syndrome and psychosis is meagre, and any relationship between the nebulous borderline states and schizophrenia must remain speculative.

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