Tonge/Burrows/Werry (eds.) Handbook of Studies on Child Psychiatry

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Childhood schizophrenia

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Introduction

The classification and diagnosis of adult schizophrenia has been, and remains, a complex matter with changing views as to whether the concept should be defined more widely or narrowly, and whether few or multiple categories are subsumed within the wider syndrome. For a long time the most prevalent view was that there was a unitary psychosis extending from infancy to adulthood with manifestations being age-related. There were also questions about personality variants of this over-arching psychosis. These views and concepts had fundamental implications for the classification and diagnosis of psychosis in children and adolescents. In this chapter we allude to these issues but give more attention to modern topical themes.

The practice of gathering various subtypes of child psychoses under the appellation of childhood schizophrenia gave rise to obfuscation and impairment of knowledge. A substantial leap was achieved by the distinction between childhood psychoses based on age of onset, first by Anthony (1958) and then by Kolvin (1971a). The latter work highlighted the differences in diagnostic criteria and other features of early-onset and later-onset psychoses, but hinted that there were many similarities between the diagnostic features of adolescent forms of later-onset childhood psychoses (technically adolescent schizophrenia) and adult schizophrenia. Green et al. (1984) have taken this one stage further by replicating the main distinctions between autism and adolescent psychosis highlighted by Kolvin and his colleagues (Kolvin 1971a,b; Kolvin et al. 1971a,b,c) by demonstrating that schizophrenia can be diagnosed in pre-pubertal children using DSM criteria. Recent reviews correctly point out that there is a need for a more careful exploration of the symptomatology thought to be diagnostic in adolescence and adulthood, and further divisions of adolescent psychoses may be necessary (Beitchman 1985), with more careful investigations into the aetiology, course and prognosis of the various subtypes. Unfortunately, the all-embracing term of childhood psychosis has started to envelop unusual personalities on the margins of these psychoses and this trend needs to be resisted unless there is strong proof of an intimate link.

Of greater practical importance is the question of whether there is a small subgroup of autists who develop schizophrenia. Although Kolvin et al. (1971a,b,c) demonstrated that autism and adolescent schizophrenia were discontinuous, in one case in that series a clear-cut diagnostic distinction could not be achieved. More recently, Petty et al. (1984) have argued that a small subgroup of autistic children develop schizophrenia. However, in this latter research all the autistic diagnoses were made retrospectively and this still leaves a question mark in relation to a common subgroup. The considerable rarity of such a subgroup should prevent any attempts to resurrect the discredited single-psychosis theory.

Diagnostic criteria

A central issue in the diagnosis of schizophrenia is how to establish agreed criteria and also how to apply these with consistency, especially in terms of severity, in relation to a disorder which manifests with protean symptomatology. Traditionally, diagnostic criteria have reflected various combinations of Schneider's (1959) approach (with an emphasis on the more positive delusions and hallucinations) and of Bleuler's (1950) (with the emphasis on the more negative symptoms of withdrawal, loosening of association and blunting of affect).

Diagnostic agreement has been improved, but disagreement not totally resolved by the tighter definitions available in DSM-III and ICD-9 (Brockington 1983). Such complexity is compounded in early childhood by developmental issues: for example, the very real problems of detecting relevant phenomena and distinguishing primitive variations of thinking, language, and emotional development from pathology. Even in the early school years there remains the allied question of how to weight immature variations of diagnostic criteria. In the original research of Kolvin et al. (1971a,b,c) there was an attempt to sharpen and limit the concept and definition of schizophrenia in school-age children and diagnosis was achieved by using the then credible Schneider first-rank criteria (Schneider 1959). Whereas, on the one hand, recent research suggests that these so-called first-rank symptoms are rather weak predictors (Brockington et al. 1978) other authorities have advocated widening these diagnostic criteria of Schneider, and hence the concept of schizophrenia, to allow the inclusion of a spectrum of schizophrenic conditions (Roth and McClelland 1979). These advocates admit that stricter definitions may give rise to greater cross-cultural agreement on diagnosis; however, broader definitions allow the heterogeneity of schizophrenia to express itself. Roth and McClelland (1979) have attempted to classify the components of a spectrum of schizophrenic disorder by delineating a gradation or ranking of disorders ranging fom nuclear schizophrenia through paraphrenic or paranoid, then cycloid or episodic, then schizoaffective, then psychogenic, then toxic, and finally schizophreniform psychosis. So far this attractive concept has not been widely studied in adolescence but an interest in examining these ideas and putting them into practice is beginning to emerge. For example, in his 16-year follow-up of pre-pubertal schizophrenias, Eggers (1978) found that about one-quarter of such children developed a typical schizoaffective picture.

The borderlands of childhood psychoses

In adolescence, schizophrenic spectra phenomena have been postulated. In this section an account is given of two of these.

The essential features of this syndrome comprise a gross lack of skills in social diplomacy, associated with a degree of naivety leading to an impairment of social relationships. The above features described by Asperger (1944) are supplemented by the following: male preponderance; diagnosis after the first 2 years of life; the unusual use of spoken language, particularly pedantic, aprosodic speech; the use of stereotyped phrases; and also abnormalities in non-verbal communication. Additionally, there is a lack of affective contact, stereotyped movement, an obsessional attachment to certain toys and surroundings, and a preoccupation with rotatory movement. Finally, Asperger regarded children displaying this syndrome as having special abilities in the areas of logic and abstraction. Such characteristics suggest a location in the 'no mans land' of classification between autism on the one hand and schizophrenia on the other (Kay and Kolvin 1987).

A crucial question is whether there is a relationship between autism and the adult-derived concept of schizoid personality disorder. Different positions are exemplified by Wing (1981) on the one hand and Wolff and her colleagues (Wolff and Barlow 1979; Wolff and Chick 1980) on the other. These have been reviewed by Kay and Kolvin (1987).

Wolff and Barlow (1979) attempt to highlight similarities between the adult-derived concept of schizoid personality disorder and autism. They compare childhood schizoid personality with autistic and normal control children using relatively small samples matched for age, sex and non-verbal intelligence. Re-analysis of their verbal IQ data reveals that there were no significant differences between the schizoid personality and the autistic group, nor between the autistic and control group. There was, however, a substantial difference between the schizoid personality disorder group and controls (Kay and Kolvin 1987).

On the Illinois Test of Psycholinguistic Abilities' sub-test of visual association both the autistic and schizoid groups had significantly lower scores than the control group. The schizoid children also followed the trend towards the autistic group on tests of visual perception and closure. On auditory recall both autistic and schizoid groups were significantly less helped to recall word strings by the introduction of meaning and syntactic structure than were controls. Thus a pattern of similarity emerged between the autistic and schizoid personality groups. Finally, Wolff and Barlow (1979) developed a measure of 'flattening of affect' and applied it to the three groups but there were no significant differences. Despite the above 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 seem to support this position (Kay and Kolvin 1987).

How do these disorders relate to each other? Wing (1981) considered schizoid personality disorder a vague and ill-defined concept, the origins of which lie in a supposed relationship to schizophrenia. The above data suggest that, given the similarity of so many of the features of schizoid personality disorder with autism, the attempted distinction is an inappropriate one. Furthermore, a separation of schizoid personality disorder and Asperger's syndrome does not make sense either on theoretical grounds, or in terms of clinical patterns. It can be argued that 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. Although there has been insufficient research into the aetiological 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 (Wing 1981); however, the pattern and severity of features are insufficient for it to be considered a psychosis. The evidence suggests that it is best classified as a personality disorder of childhood.

Initially this elusive concept was used to identify patients who were too unstable for deep psychotherapy but were not obviously psychotic (Stern 1938). Spitzer and Endicott (1979) argue that the borderline concept is not unitary and is best regarded as possessing at least two major dimensions which are relatively independent. For instance, clinicians in the U.S.A. use the term 'borderline' in two different ways. The first is used to describe a patient group assumed to be genetically related to a spectrum of disorders including schizophrenia; this has been labelled 'schizotypal personality' to reflect the concept of 'borderline schizophrenia'. Family and genetic data are suggestive of a link with schizophrenia (Kendler 1984). Secondly, the term is used to describe a patient group with a 'constellation of relatively enduring personality features of instability and vulnerability': this has been identified as an 'unstable personality'.

There are greater difficulties in applying the above concepts to children and adolescents whose ego functioning and personalities are not fully developed (Kay and Kolvin 1987). Steinberg (1983) has drawn a composite picture of the borderline personality from the American literature. It is characterized by complaints of misbehaviour – particularly antisocial behaviour, drug abuse, running away and promiscuity; there may also be educational difficulties. Poor impulse control and suicidal threats are also described. Some of these children are said to be depressed and withdrawn and to show a sense of futility or enraged hostility. The overall impression is that of a child with a poorly integrated sense of self and in whom transient psychotic states may occur, although 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 category of mixed conduct-neurotic disorder (Kay and Kolvin 1987). Despite considerable continuity over time (Aarkrog 1981), Stone (1984), in his review of the borderline syndromes, suggests that it is difficult to apply the concept meaningfully in adolescence.

Phenomenology

DSM-III provides a guide to the clinical criteria necessary to diagnose schizophrenia but the stipulation that there should be at least 6 months of overall disturbance, even though this may include the prodrome, seems unduly long. Furthermore, in the light of the classic concept of dementia praecox, it seems unacceptable that the notion of a progressive 'simple schizophrenia' should be excluded.

Only four studies provide the symptom frequency and use a definition of childhood schizophrenia which would qualify for the DSM-III diagnosis (Kolvin et al. 1971a; Eggers 1978; Kydd and Werry 1982; Green et al. 1984). Between them they describe 129 children whose ages range from 6 to 15 years. Although Bettes and Walker (1987) suggest that the presence of positive symptoms may be a function of age and intelligence, their conclusions must be limited by their ability to distinguish autism and schizophrenia.

Delusions occur in just over one-half of the subjects of British and American studies (Kolvin et al. 1971b; Green et al. 1984). A German study (Eggers 1978) suggests that these are age-related. In the younger child they are less frequent and there are difficulties in distinguishing fantasy from reality. By early adolescence they have become very common, and are more persistent and precisely formulated.

Formal thought disorder usually takes the form of disturbed association and thought blocking: it is present in over one-half of cases. Alienation of thought (which includes thought

deprivation, insertion and broadcasting) is a variation of this and is present in a substantial minority – 20% (Kolvin et al. 1971a).

Auditory hallucinations in a clear state of consciousness occur in about 80% of older children (Kolvin et al. 1971a; Eggers 1978; Green et al. 1984). About half the children also have visual hallucinations. These are not necessarily pathognomonic of schizophrenia as they may sometimes occur with other forms of disturbance and under stress (Egdell and Kolvin 1972; Garralda 1984a); nor do they predict subsequent psychosis (Garralda 1984b). Somatic hallucinations occur in 36% of psychotic children (Kolvin 1971a) and may affect several sensory systems simultaneously (Eggers 1978).

Affective change can take a variety of forms, of which the commonest are constriction or blunting of affect (in about 60%) closely followed by incongruity of affect. Perplexity is common; episodes of rage or giggling less so (Kolvin et al. 1971a). Clear depression has not been frequently reported but this may be because of the lack of systematic enquiry; when present, it is likely to be represented by moodiness or dejection. It occurs reasonably frequently in the prodrome, possibly as a consequence of a sense of inexplicable change (Kolvin et al. 1971a). Nissen (1981) reports that in just under 10% of cases of childhood depression, the depression proved to be a prodrome of schizophrenia. Finally, motor symptoms occur commonly (such as a jerkiness of movement) and particularly mannerisms and facial grimaces.

In the family context, the parents have a greater communication deviance than do the parents of depressed children. The extent to which this is present is proportional to the child's poor attentional function and overall degree of impairment (Asarnow et al. 1988)

The above view highlights the symptomatic similarity between childhood and adult schizophrenia. Inevitably, some symptoms are influenced by age being more frequent, rich and complex after the age of 10 years and this is particularly true of hallucinations and delusions (Eggers 1978).

Links between adolescent and adult psychoses

What are the grounds for considering late childhood, adolescent and adult schizophrenia to be expressions of the same diagnostic entity? Given that there is broadly similar phenomenology to that found in adulthood (Kolvin et al. 1971a; Rutter 1972), other evidence of aetiological similarities between adult and childhood schizophrenia is necessary to support the hypothesis of similar diagnostic entities (Kay and Kolvin 1987). What, then, is the nature of this evidence?

First, there is epidemiological evidence. Childhood schizophrenia is even rarer than autism (Kolvin et al. 1971a) with an estimate of approximately 3/10,000 school children. This is similar to the rate reported by Kramer (1978). Other estimates suggest that it occurs some 50 times less before the age of 15 years than after it (Beitchman 1985). This is consistent with a stress diathesis model by which few individuals within the population at risk would have a sufficient combination of biological loading and environmental stress to give rise to an early manifestation. Further, a comparison of the sex ratios of adult and childhood schizophrenia reveals a remarkable similarity, provided that autistic contamination is rigorously avoided (Kramer 1978; Babigian 1980).

Second, pre-morbid characteristics do not differentiate these groups. Such patterns of pre-morbid behaviour which do emerge probably are shared by both adult and childhood presentation (Goldstein 1980; Nuechterlein 1986) with an emphasis on shyness, diffidence or inhibition, and sensitivity (Kolvin et al. 1971a, Eggers 1978).

Family personality problems have not been studied commonly or systematically, but there are suggestions from the research of Kolvin et al. (1971b) of excesses of personality oddities in the parents of schizophrenics, consisting of introversion and suspiciousness which are reminiscent of those described in adult schizophrenia.

Third, do pregnancy and birth complications (PBCs) differentiate between the two groups (McNeil and Kaij 1978; Jacobsen and Kinney 1980)? Unfortunately, the data relating to PBCs and subsequent development of schizophrenia in childhood are limited. The studies by Kolvin and his colleagues (1971c) revealed a 12% incidence of PBCs in the schizophrenic group, but Green et al. (1984) reported no increase in PBCs compared with a group of conduct-disordered children. Unfortunately, a range of other studies have been compromised by the inclusion of autists within their schizophrenic group (Rutt and Offord 1971; Torrey et al. 1975).

Fourth, routine EEG studies of adolescent schizophrenia have failed to show any consistent differences from controls (Kolvin et al. 1971c; Beitchman 1985). Computer-analysed EEGs (CEEG) have allowed certain characteristic features to emerge which appear to correlate with the presence of psychopathology (Itil 1979) but again such studies are compromised by a lack of homogeneity of the sample and insufficient data.

Fifth, what is the biochemical evidence for a relationship? Biochemical hypotheses receive their main support from studies of clinical effects. Nevertheless, one is struck by the variety and proliferation of speculative biochemical mechanisms, suggesting failure to confirm experimentally the pharmacologically derived dopaminergic theory of adult schizophrenia (Post et al. 1975; Crowley et al. 1978). This lack of consistent support for any biochemical theory of adult schizophrenia, except for the central pharmacologically derived dopamine model, makes it extremely difficult to extrapolate to a comparison with childhood schizophrenia. In addition, the findings in the limited biochemical research undertaken into childhood schizophrenia are often contradictory.

Sixth, the evidence for a genetic link is more compelling. Kolvin and colleagues (1971b) on the basis of their own data concluded that there was a genetic connection between childhood and adult schizophrenia. Hanson and Gottesman (1976) have calculated that the parents of childhood schizophrenics have twice the 4% figure that Kessler (1980) calculated when reviewing the risk of parental schizophrenia in adult schizophrenics. Such a heightened risk of schizophrenia in the parents of childhood schizophrenics is consistent with a polygenic theory of aetiology. Accordingly, those individuals with a higher genetic loading will present earlier and with more intractable psychoses, and will be expected to have a higher prevalence of schizophrenia in first-degree relatives.

Behavioural precursors of adolescent schizophrenia

How do children destined to develop schizophrenia differ from their peers or siblings? More is known about adult than adolescent schizophrenia and most of the following relates to adult schizophrenia.

Commonly, identification of precursors has been by prospective studies of high-risk populations defined as children born to schizophrenic parents. The reasons why such prospective studies have not fulfilled their potential has been reviewed by Rutter (1984) and Lewine et al. (1981).

First, only a minority of schizophrenics have schizophrenic parents and hence any differences are unlikely to be representative of the schizophrenic population as a whole (Shur 1982).

Second, the cohort that is at risk has to be followed for many years. Third, the genetic risks are not focused on schizophrenia alone as a number of the parental partners will be mentally abnormal (Mednick et al. 1978). Thus high-risk research may be addressing itself to rather atypical sub-groups of schizophrenics (Rutter 1984). One shortcoming can be dealt with by appropriate sample selection, for instance by controlling for factors associated with the risk of schizophrenia (compared to a general risk of psychiatric disturbance) by utilizing control groups of children whose parents have other forms of psychiatric disturbance. What are the main findings?

School reports do not give support to the notion of a schizoid personality as reflected by shyness and withdrawal which appears to contradict the assumption that the schizoid personality is a precursor of schizophrenia spectrum disorder (Parnas et al. 1982). Nevertheless, as indicated below, at school the children who subsequently developed schizophrenia did display interpersonal difficulties and poor affective control. Other important dysfunctions have been sought and highlighted: first, impaired neurodevelopmental functioning; second, attentional deficits; third, evidence of cognitive dysfunctioning; and, finally, poor interpersonal relationships. Poor motor functioning and cognitive deficits have been reported by Hanson and Gottesman (1976) and Erlenmeyer-Kimling et al. (1984a,b). Further, Asarnow et al. (1977) have demonstrated similar cognitive deficits in the foster children of schizophrenics.

Unfortunately there remain serious questions of specificity, in that many anomalies or deficiencies reported in high-risk children are also manifested by children of parents with non-schizophrenic psychiatric disorders. Nevertheless, it is important to emphasise that social ineptitude and poor child-teacher relationships may well be mediating factors in children with attentional deficits. Asarnow and MacCrimmon (1978) at a speculative level support a link between earlier attention deficits, later social ineptitude and subsequent adult schizophrenia.

The argument runs thus: that these two major vulnerability factors, neurointegrative deficits and social skills deficits, interact and result in increasingly severe deficits, so that a child who may initially have had attentional deficits may be stressed by increasing peer rejection and social isolation (Asarnow and Goldstein 1986).

Retrospective designs have been employed to examine the childhood archives of individuals diagnosed as schizophrenic in adult life. One key study is of school records of schizophrenics and a number of control groups (Watt 1974) which again reports that schizophrenics show a greater degree of social ineptitude. Although there are certain sex differences, the overall picture was of one or other type of social isolation, with boys showing more in the way of aggressive social disturbance and girls more in the way of passiveness and social isolation. Nevertheless, such features were present only in about one-half of those youths who subsequently became schizophrenic.

Another strategy is to follow up children who previously had psychiatric disorder (Rutter 1984). Rutter points out that, in the Robins study (Robins 1966) compared with other clinic children pre-schizophrenics showed antisocial behaviour which expressed itself within the family, but with an absence of socialized delinquent behaviour. It has been reported that pre-schizophrenic socialization problems were more concerned with an active rejection by the peer group, with the peer group perceiving the pre-schizophrenic child as in some way unpredictable, unusual or odd (Roff 1963; Zeitlin 1986).

Finally, recent longitudinal research suggests a continuity of formal thought disorder from childhood to adulthood in a high-risk sample (Parnas and Schulsinger 1986). This implies that certain schizophrenic symptoms may develop by gradual accretion and that schizophrenia is not necessarily a sudden, unexpected disease.

Aetiological factors

In this section we address ourselves to aetiological factors in childhood schizophrenia. In this respect we are attracted by Asarnow and Goldstein's model (Asarnow and Goldstein 1986). Considered first are vulnerability factors assumed to be present in people 'at risk' for schizophrenia – these include biological, constitutional, and psychological. Considered second, are stressful environmental stimuli, particularly undesirable life events.

Brain damage/cerebral dysfunction

A current popular view is that actual or presumed cerebral damage in early life contributes to the emergence of adult schizophrenia (Murray and Lewis 1987). What evidence is there for this in childhood schizophrenia?

Unfortunately, many studies do not distinguish autism from schizophrenia, and therefore it is difficult to disentangle the facts. Although not as common as in autism, presumptive brain damage/dysfunction as reflected by PBCs, neurological soft signs, or specifically abnormal EEGs, are still more common than might be expected. For instance, temporal lobe discharges were reported in 12% of adolescent schizophrenics (Kolvin et al. 1971c).

Genetics

There is strong evidence of a genetic basis for adult schizophrenia. The residual debates concern its nature and mode of transmission. Here again the distinction of schizophrenia from autism is essential: whereas the incidence of psychosis in the families of autistic children does not differ greatly from that of the general population, the rate of parental schizophrenia ranges from 10 to 13% (Kallman and Roth 1956; Kolvin et al. 1971b) although the actual figure depends on how the disorder is defined. Much lower rates are reported if case selection is limited to those with core schizophrenia (Abrams and Taylor 1983); they are substantially higher if the criteria are those of the schizophrenia spectrum disorder (Baron et al. 1983). This suggests a much less specific genetic predisposition which then interacts with other factors, such as the complications of pregnancy and birth (PBCs), to result in the florid schizophrenic state (Murray and Lewis 1987).

Psychological aspects

Neuropsychological assessment of schizophrenic children points to a central deficit, similar to that reported in adults, in the child's ability to search for specific cues. This impairment appears specific to the process of early attention and does not involve short-term/iconic memory (Asarnow et al. 1986).

Electrophysiological research both in adults (Roth et al. 1980) and in children (Strandburg et al. 1984; Erwin et al. 1986) provides support for the hypothesis that there is a deficit of the later-developing tertiary association areas of the cortex (Asarnow et al. 1986). This would be consistent with the later onset of the disorder in a child who has been predisposed to it from conception.

Environmental precursors

There is substantial evidence that deficits in family communication, as reflected by critical or hostile interactions within the family, are associated with a range of poor outcomes that include schizophrenia spectrum disorders (Doane et al. 1981). However, such patterns are not specific to schizophrenia but, once schizophrenia is established, critical over-involvement within the family is associated with a relatively poor outcome (Leff and Vaughn 1981).

Asarnow and Goldstein (1986) speculate that vulnerability factors in individuals contribute to that individual's characteristic threshold for coping poorly with stress. They indicate that the factors are both constitutional and environmental. The stressful environmental factors include pre-natal and birth complications and deviant intrafamilial processes.

Management

Psychopharmacological agents are widely used in childhood schizophrenia but little is known of the long-term effect of medication on the developing organism. Although pubertal development appears to be unaffected, there are some hints of a destabilization of the hypothalamic-pituitary axis, affecting testosterone and prolactin levels as well as gonadotropin responses (Apter et al. 1983). For these reasons it is wise to avoid the long-term use of high-dosage neuroleptics wherever possible.

Further, diagnostic confusion has muddied a number of attempts to generate a systematic approach to management. This has been compounded by the paucity of rigorous controlled studies of homogeneous populations. One is therefore reliant on the clinical guides in the literature with much of the available information being anecdotal or derived from uncontrolled studies utilizing a wide range of neuroleptics. Neuroleptic medication is the central component of treatment but, as in adult schizophrenia, it is insufficient on its own and must be supplemented by an individual tailored programme of rehabilitation into the family and the community.

Clinical experience suggests that the more florid symptoms often respond rapidly to neuroleptics (Kaplan and Kolvin 1985) but Campbell (1985), while supporting this, emphasises that their sedative nature may impede scholastic functioning. Similarly, the advantage of using less-sedative drugs, such as the butyrophenones, is offset by the frequent need for an anti-parkinsonian agent (Kydd and Werry 1982) which can bring drowsiness in its own right. The literature is replete with evidence of enthusiasm for high-dosage regimens which bring in their wake the full range of side-effects listed in the adult literature.

Similarly, the literature suggests that depth psychotherapy is contraindicated. A consistent bridge with reality can be built by supportive counselling designed to help the patient to live and cope with their symptoms and problems, including social relationships (Cantor and Kestenbaum 1986).

Awareness of the importance of family relationships, with a particular emphasis on how emotions are experienced and expressed within the family, has helped the understanding of the patient's ability to survive comfortably at home. The clinical experience of those working with adolescents gives credence to the validity and utility of these concepts of family communication, especially in the prevention of relapse and in rehabilitation (Tanguay and Cantor 1986).

Finally, in contrast to adult schizophrenia, where the emphasis is on occupational rehabilitation, it is essential that educational resources are available to the child and adolescent. Because

of the inevitable variation in age and functional cognitive ability, as well as in the form and severity of the psychosis, any educational programme must be individually tailored.

Outcome

Any conclusion as to the course of childhood schizophrenia depends on the specificity of the diagnosis: inclusion of other disorders clouds the picture of the outcome. Although autism is suspected to be a common contaminant (Cantor et al. 1982; Howells and Guirguis 1984), so may be the adolescent affective disorders which, particularly in their early episodes, can be mistaken for schizophrenia (Hassanyeh and Davison 1980) or schizoaffective disorder (Himmelhoch and Garfinkel 1986).

Most of the reliable accounts of the course of schizophrenia concern adolescents (Masterson 1956); few deal with a childhood onset (Eggers 1978; Kydd and Werry 1982). However, the themes that emerge are consistent: a younger age of onset is associated with a poor prognosis; other, possibly associated, indicators of a poor outlook are an insidious onset, a simple or hebephrenic subtype, a poor level of pre-morbid adaptation and low intelligence; the presence of affective symptomatology may indicate a better prognosis.

There is no evidence that a range of other factors make any contribution to the prognosis although such evidence has been sought: examples include the frequency and duration of episodes, the presence of precipitating triggers, the family history, and the socioeconomic status. Finally, between 20 and 40% of cases can be expected to remit completely, particularly where the onset is after 10 years of age. Knowledge of this possible outcome, more optimistic than is generally appreciated, is helpful in the face of community and family pessimism.

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