CHAPTER 7

SCHIZOPHRENIA IN CHILDHOOD

Israel Kolvin T. P. Berney Joel Yoeli

DESCRIPTION OF THE DISORDER AND CLINICAL PRESENTATION

Introduction

The traditional concept of "schizophrenia," previously employed to encompass any psychotic process that was neither manic nor depressive, led to two thrusts in childhood classification: The first was the now discredited concept of a unitary psychosis (Fish, 1977); the second was the extensive eponymous labeling of childhood syndromes regarded as psychotic (Kolvin, 1971). However, as Anthony (1958) pointed out, the latter could be regrouped to give three main syndromic groups, broadly distinguished by their age of onset. This was the springboard for the next advance, which differentiated on empirical grounds between those psychoses specific to childhood and those with adult-type phenomenology (Kolvin, 1971). These syndromic groups appear to have a bimodal age of onset (Kolvin, Ounsted, Richardson, & Garside, 1971; Makita, 1966) and include the following:

• Infantile autism (Kanner, 1943) or psychosis (Kolvin, 1971): with an onset in the early years

of life. Subsequent work suggests an affiliation to a wider range of developmental disturbances, including Asperger's syndrome of autistic psychopathy (Asperger, 1944; Wing, 1981). More recent studies have suggested that these latter conditions may contribute to an autistic spectrum disorder of early childhood.

 Disintegrative psychosis: those conditions with an onset after infancy but still within the preschool years and that are characterized by widespread functional disintegration.

• Late onset psychosis or childhood schizophrenia: those conditions with an onset during the school years and characterized by primitive or more complex symptoms resembling those described in adult schizophrenia (Eggers, 1978; Green et al., 1984; Kolvin, 1971).

Later work highlighted those personality or other conditions purportedly at the margins of the psychosis and in due course has also given rise to the concept of childhood schizophrenia spectrum dis-

Some authors have described exceptions to this main pattern, and hence one leading research

group recommends that the diagnosis be based on the clinical picture rather than on the age of onset (Wing & Attwood, 1987).

Spectrum Disorders Versus Multiple Categories

In adult schizophrenia some authorities have favored a classification into multiple, complex, almost discrete subcategories, whereas other have moved toward agglomeration into the schizophrenia spectrum disorder. Can this recently fashionable concept of spectrum disorder be applied to childhood schizophrenia?

In the autistic psychoses there has been a similar dichotomy with the "splitters" in the United States (American Psychiatric Association, 1980) proposing a new class of disorders, the pervasive developmental disorders (PDD). In the United Kingdom a similar move has led Wing and her colleagues to propose their three-way split. In addition, and in contrast, there has been the delineation of the autistic spectrum disorder.

The Developmental Perspective

In this chapter we focus on the age of onset as a guide to classification, with the proviso that the diagnosis be based on the presence of the relevant clinical phenomena. This approach allows a focus on a developmental perspective that is widely endorsed in the standard schemata (International Classification of Diseases-9 [World Health Organization, 1979] and DSM-III [APA, 1980]) on both sides of the Atlantic.

This broad developmental pattern of onset is consistent with a diagnosis based on clinical phenomenology and coincides with the main categories of ICD-9 (WHO, 1979). With the advent of DSM-III (APA, 1980), it was broadly accepted because of the developmental typology suggested by Anthony (1958) and Kolvin (1971). However, it replaced the multiplicity of eponymous categories with an equal multiplicity of subcategories of disorder that are characterized by a pervasive lack of responsiveness to others, as well as by a gross and sustained impairment of social relationships. These categories are split both according to age of onset (before or after 30 months) and to whether or not there is a full syndrome or a residual state. Finally, there is a rag bag of other disorders that are difficult to classify elsewhere and that are lumped together as atypical pervasive developmental disorder. Inevitably the clinical description

of these groups overlaps, and the previous lack of clarity at the margins of the main psychotic disorders of childhood is replaced by subcategories with inadequate empirical foundations (Volkmar, 1987).

Subclassification of Autistiform Disorders

A newer clinical approach to the subclassification of preschool autistiform conditions that appears to have better empirical foundations than the DSM-III categories is provided by Wing and Gould (1979) and Wing and Attwood (1987), who describe three main groups of children—the aloof, the passive, and the active-but-odd.

The aloof are the most socially impaired, particularly in relation to their peers, and become agitated when in close proximity to others. They may approach others to obtain gratification of their basic needs, but more usually display a lack of overt affection. Both verbal and nonverbal communication may be seriously impaired. Speech is not employed as a means of social interaction. Further, even if speech has been present from infancy, there is little engagement in two-way communication in the form of cooing, babbling, or facial expression, nor are there the signs of early nonverbal communication, including social gesturing in later infancy. Associated features are poor eye contact and poverty of facial expression. In the main, children in this group have little symbolic or pretend play, or evidence of inner imagination (Wing & Gould, 1978, 1979). They are particularly prone to stereotypes, but, although these diminish with age, they tend to be replaced by more complex repetitive behaviors (Wing & Attwood, 1987). Broadly speaking, those in the aloof group have much in common with the classic picture of autism depicted in ICD-9.

The autistic features of the passive group are less marked than are those in the aloof group. The main distinction is that, although such childrer do not make spontaneous approaches to others except to meet their needs, they will accept the approaches of others without protest. Their abil ity to imitate and copy is also less impaired (At twood, 1984), as is their speech and verbal communication. They are the best behaved and the most easily managed; the more intelligent macope fairly well educationally, even surviving i ordinary schools.

The active-but-odd are described as makin "spontaneous approaches to others, but in pect liarly naive and one-sided fashion" (Wing & At

wood, 1987). Many of the characteristics described by the authors would tend to lead such children to be categorized as cases of Asperger's syndrome, and would include an indulgence of their circumscribed interests by talking at other people, together with literalness, repetitiveness, and longwindedness. One important feature is that, when older, they tend to replace their repetitive routines with more abstract but restricted interests that are "pursued so relentlessly, to the exclusion of virtually everything else, and with little grasp of the meaning or applicability to everyday life of the knowledge acquired" (Wing & Attwood, 1987).

Wisely, Wing, and Attwood (1987) do not see these as rigidly defined groups but rather as a range of abnormalities found in the spectrum of autistic disorders with the main changes over time being from the aloof into one of the other two groups. However, although the aloof group is diagnosable at an earlier age, the main characteristics of the passive and active-but-odd subgroups may not be appreciated until school age.

Disintegrative Psychosis

The onset occurs after several years of normal development and is characterized by a profound regression of general and social behavior, social interactions, and play, together with a deterioration in speech, language, and cognition. In due course, stereotypes and mannerisms may appear, giving rise to a picture reminiscent of that seen in autism. In some cases there is an evident organic basis such as encephalitis; in others there is an associated organic cortical degenerative disorder (Creak, 1963; Corbett, Harris, Taylor, & Trimble, 1977), but evidence of brain disease is not always clear-cut (Evans-Jones & Rosenbloom, 1978). In some the neurodegenerative basis becomes evident only in the course of time (Rivinus, Jamison, & Graham, 1975; Corbett et al., 1977). Thus, while there is usually the presumption of organic brain disease, in some the condition has an unknown etiology that invalidates the suggestion that it should be always classified as an organic dementia (Rutter, 1985). Rutter (1985) considers that these disorders will prove to be heterogeneous with some representing atypical forms of autism and others associated with acquired organic brain disorders. Although Rutter sees it as desirable to classify this group separately from autism, other authorities are less convinced of this (Wing & Gould, 1979).

In some there is a rapid downhill course, and in those few neurodegenerative disorders, this may progress to death. In the majority of others, after a number of months, this pattern may stabilize, leaving an overactive child with severe mental handicap, without speech, and often an autistic behavioral picture that tends to be consistent on follow-up 11 to 16 years later. Occasionally, seizures subsequently develop (Hill & Rosenbloom, 1986).

Descriptive Account of Childhood Schizophrenia

Previous work has emphasized that adult-type psychoses (mainly childhood schizophrenia) have their onset in the school years (Kolvin, Ounsted, Richardson, & Garside, 1971), being very rare in the early years and becoming less so as the child moves toward and into adolescence. This constitutes the basis of the bimodal pattern of onset already alluded to (Kolvin, Ounsted, Richardson, & Garside, 1971; Makita, 1966).

There are good reasons for suggesting that pervasive developmental disorders (or the autistic spectrum) can be distinguished from adult-type schizophrenia on phenomenological, demographic, and genetic grounds, although, inevitably, there are exceptions to this general rule (Kolvin, 1971; Petty, Ornitz, Michelman, & Zimmerman, 1984; Wing & Attwood, 1987).

Kolvin and his colleagues (Kolvin, 1971; Kolvin, Garside, & Kidd, 1971; Kolvin, Humphrey, & McNay, 1971; Kolvin, Ounsted, Humphrey, & McNay, 1971; Kolvin, Ounsted, Richardson, & Garside, 1971; Kolvin, Ounsted, & Roth, 1971) have highlighted the differences in diagnostic criteria and other features of early childhood psychoses (autism) and later-onset psychoses (childhood 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 and by demonstrating that schizophrenia can be diagnosed in prepubertal children using DSM-III criteria. Other reviews point out that there is a need for a more careful examination of the symptomatology thought to be diagnostic of childhood schizophrenia together with an exploitation of possible subdivisions on symptomatological grounds and also etiology, course, and prognosis of the proposed subtypes (Beitchman, 1985).

There are few satisfactory descriptions of the

presentation and course of childhood schizophrenia. Only four studies both provide the symptom frequency and use a definition of childhood schizophrenia that would qualify for a DSM-III diagnosis (Eggers, 1978; Green et al., 1984; Kolvin, Ounsted, Humphrey, & McNay, 1971; Kydd & Werry, 1982). Together these describe 129 children whose ages range from 6 to 15 years. A number of other research reports are undermined by the lack of clear distinction between autism and schizophrenia.

Two studies (Green et al., 1984; Kolvin, Ounsted, Humphrey, & McNay, 1971) have described the type of onset as acute (12% to 20%), insidious with an acute exacerbation (21% to 33%), and insidious (45% to 66%).

Delusions occur in just over one-half of cases (Green et al., 1984; Kolvin, Ounsted, Richardson, & Garside, 1971). In the younger child they are less frequent, and there is difficulty in distinguishing fantasy from reality. Formal thought disorder, usually a disturbance of association and thought blocking, is present in over one-half of the cases. A variant, present in about one fifth, is alienation of thought, including thought deprivation, insertion, and broadcasting (Kolvin, Ounsted, Humphrey, & McNay, 1971).

Auditory hallucinations occur in 80% of older children (Eggers, 1978; Green et al., 1984; Kolvin, Ounsted, Humphrey, & McNay, 1971). Visual hallucinations, present in about half the cases, can occur with other forms of disturbance and under stress (Egdell & Kolvin, 1972; Garralda, 1984a) without predicting subsequent psychosis (Garralda, 1984b). Somatic hallucinations occur in about one third of schizophrenic children (Kolvin, Ounsted, Humphrey, & McNay, 1971) and may affect several sensory systems simultaneously (Eggers, 1978).

The most common form of affective disturbance is a constriction or blunting that is experienced by about 60% of cases; next in frequency is a sense of affective incongruity and perplexity. Depression is common in the prodrome (Kolvin, Ounsted, Humphrey, & McNay, 1971), and in nearly 10% of cases, children with depression subsequently develop schizophrenia. Motor symptoms are common, particularly as a jerky incoordination of movement, as well as mannerisms and facial grimaces (Kolvin, Ounsted, Humphrey, & McNay, 1971).

In his 16-year follow-up of prepubertal schizophrenias, Eggers (1978) found that about 46% of such children developed an affective picture. He, too, indicated that there were developmental dif-

ferences. In those who developed their symptoms before the age of 10 years, the picture was an unobtrusive one with sudden, unexpected, and strange personality and behavioral changes. They included loss of contact with reality, the narrowing of interests, an indifference to the usual activities and pursuits, together with evidence of disturbed mobility, negativism, and speech disturbance. Some of these were previously affectionate children who suddenly became unkind, cold, and stubborn. Some became rather disinhibited, and some made unmotivated suicidal attempts. As the children approached 10 years of age, delusions and hallucinations became more prominent. The delusions often appeared as a rash of diffuse fears. In the prepuber and cases, delusions became more persistent, and religious and depressive themes emerged for the first time. At this age there was a greater systematization of delusional ideas, and thus, with increasing age, childhood and prepubertal psychosis began to resemble those of the adult (Eggers, 1978).

The next step therefore consists of the establishment of agreed-upon diagnostic criteria and the application of these with consistency in a disorder that manifests with protean symptomatology. Traditionally, diagnostic criteria have reflected various combinations of Schneider's (1959) approach, where there is an emphasis on the more positive delusions and hallucinations, with that of Bleuler (1950) where the emphasis is on the more negative symptoms of withdrawal, loosening of association, and blunted 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. Thus, 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 and his coworkers (Kolvin, 1971; Kolvin, Garside, & Kidd, 1971; Kolvin, Humphrey, & McNay, 1971; Kolvin, Ounsted, Humphrey, & McNay, 1971; Kolvin, Ounsted, Richardson, & Garside, 1971; Kolvin, Ounsted, & Roth, 1971), there was an attempt to sharpen and limit the concept and definition of schizophrenia in school-aged children and diagnosis was achieved by using the rank criteria (Schneider, 1959).

"Borderlands" and Spectrum Disorders

The Autistic Borderlands

There is confusion regarding the classification of Asperger's syndrome, as well as of that small proportion of cases of autisticlike conditions that fall into Wing and Goulds' "passive subgroup." The latter group consists of individuals who, when adults, may present with a social impairment that resembles the social withdrawal of those schizophrenic children whose symptomatology is mainly of the negative variety (Wing & Attwood, 1987). This is less likely to be the case with those schizophrenia-type disorders that present with positive symptomatology. Wing and Attwood (1987) point out that a correct diagnosis usually can only be made from a developmental history taken from an informant who has known the psychotic person over the whole life span.

The Schizophrenia Borderland

Despite the suggestion, as a result of recent research, that these so-called first-rank symptoms are rather weak predictors (Brockington, Kendell, & Leff, 1978), other authorities have advocated widening these diagnostic criteria of Schneider (and hence the concept of schizophrenia in adulthood) to allow the inclusion of a spectrum of schizophrenic conditions (Roth & 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 from 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. Unfortunately, however, 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.

Revival of the Concept of a Unitary Psychosis

Of considerable practical importance is the question of whether there is a small subgroup of

autics who develop schizophrenia. Although Kolvin and his colleagues (Kolvin, 1971; Kolvin, Garside, & Kidd, 1971; Kolvin, Humphrey, & Mc-Nay, 1971; Kolvin, Ounsted, Humphrey, & McNay, 1971; Kolvin, Ounsted, Richardson, & Garside, 1971; Kolvin, Ounsted, & Roth, 1971) 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 do 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.

Multiaxial Approaches

Such a behavioral classification of syndromes demands a complementary multiaxial classification (Wing & Attwood, 1987) with axes to accommodate gross etiology, on the one hand, and cognitive ability, on the other. For instance, a diversity of gross etiologies is reported in one third to one half of cases (Kolvin, Ounsted, & Roth, 1971; Wing & Gould, 1979; Rutter, 1972). Intelligence is also important, with up to 80% of autistic cases having quotients of <70 when measured on standardized tests (Kolvin, Humphrey, & McNay, 1971; Rutter & Lockyer, 1967; Wing & Gould, 1979).

EPIDEMIOLOGY

Autism

The prevalence of typical autism based on epidemiological research is approximately 4 per 10,000 (Lotter, 1967). About one half of these have a more nuclear autism that approximates to that described by Kanner with the remainder being less typical (Wing & Gould, 1979). Such epidemiological rates, even when the psychosis is narrowly defined, are far greater than the so-called administrative rates (the number in the community known to the local services). Hence the use of epidemiological rates in service planning carries the risk of overprovision. Wing and Gould (1979), from their English base, argue for a wider concept of childhood psychosis according to the quality of social contact and on these cri-

teria 10.6 per 10,000 were aloof, 5.7 were passive, and 4.9 active-but-odd, with an overall rate of 21.2 for children under 14 years of age. Within this figure 4.9 per 10,000 were childhood autists. How many of the residual psychoses were really Asperger's syndrome? Some might consider the active-but-odd as such. Bryson, Clark, and Smith (1988) from their Canadian base, also using a broader definition of autism (Denckla, 1986), calculate a prevalence of 10 per 10,000. An association with mental retardation is reported in between 66 and 75% of children with autism (Kolvin, Humphrey, & McNay, 1971; Rutter & Lockyer, 1967). Boys outnumber girls by up to 4 to 1 (Kolvin, Ounsted, Richardson, & Garside, 1971; Lotter, 1967; Wing, 1981). The Goteborg study (Gillberg, 1984) suggests that this relationship may be nonlinear, achieving a maximum of over 5 to 1 for the middle band of the mildly mentally retarded, and decreasing to 2 to 1 both above and below this ability band.

The earlier epidemiologically based reported association with higher social class (Lotter, 1967) has had some support (Steinhausen, Gobel, Breinlinger, & Wohleben, 1983), but most major surveys have not supported this distinction (Gillberg & Schaumann, 1981; Wing, 1981). On balance it is likely that the reported association was the consequence of referral bias.

Disintegrative Psychosis

Again, clinical research suggests this to be a rare disorder (Kolvin, 1971; Makita, 1966). There have been no specific studies, but the Camberwell study of early childhood psychoses allows an estimate of prevalence (Wing & Gould, 1979). Ninety-four percent of the psychotics with an autistic picture were reported to be abnormal before the age of 3 years and the rest between 3 and 5 years (Wing, 1982).

Schizoid Personality Disorder and Asperger's Syndrome

Knowledge about the epidemiology of these two disorders is rather limited. Wolff and Chick (1980) estimated that schizoid personality disorder was present in over 3% of their child psychiatric clinic population. The wider significance of this is not clear as it needs to be reinterpreted in terms of the selectiveness of referral to their clinic; such a perspective can be achieved only by epidemiological research. What about Asperger's syndrome? The difficulties in defining and distin-

guishing mild autism, Asperger's syndrome, and schizoid personality have hampered the accurate estimation of prevalence. Gillberg and Gillberg (1988) suggest that Asperger's syndrome is more common than autism, is not associated with mental retardation, and has a prevalence of about 10 per 10,000. This is double the estimate of the active-but-odd subgroup derived from the Wing and Gould (1979) research.

Childhood Schizophrenia

Prevalence is age dependent, being rare in the prepubertal population. Thus, whereas the adult incidence is about 1%, fewer than 200 cases of childhood schizophrenia have been described in the world literature. Kolvin, Ounsted, Richardson, and Garside (1971), in their wide clinical search, report a rate of three quarters that of autism, which gives a rough estimate of 3 per 10,000 children. This is similar to that reported by Kramer (1978). Other estimates suggest a rate some 50 times less before the age of 15 years than after (Beitchman, 1985), which 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. Provided that autistic contamination is rigorously avoided, the sex ratios of adult and childhood schizophrenia reveal a remarkable similarity (Babigan, 1980; Kramer, 1978). Most studies give evidence of a male preponderance but within these the sex ratio varies widely and this is especially true of those not based on rigorous diagnostic criteria, resulting in a contamination of the index groups by autism.

NATURAL HISTORY

Autism

The clinical features vary with the stage of development. The more florid symptoms peak in the preschool years but then usually lessen during middle and later childhood; for instance, in early infancy a commonly reported characteristic is a failure to cuddle, babbling may be absent, and there may be a failure to engage in preverbal two-way social interactions with the mother. In the toddler, prominent features consist of such characteristics as profound social withdrawal and avoidance of gaze, together with language abnormalities and repetitive behaviors. Subsequently, many children continue to have difficulties in in-

terpersonal relationships, particularly outside the home; such difficulties may arise with those adults who are not in regular contact with the child and who are unaccustomed to interpreting his or her communications or making allowance for his or her behaviors. Older autistic children may show an inability to appreciate the nuances of social relationships and have a lack of appreciation of other people's feelings. Often, the brighter autistic preadolescent and adolescent will show a desire for friendships, but the lack of social skills and empathy make this difficult. In a minority, especially in the less able autistic adolescent, there is pubertal deterioration comprising a worsening of symptomatology, a loss of developmental skills, and a change to a more gross physical appearance. Further, aggressive behavior may reemerge in some (Gillberg & Schaumann, 1981). In others, although many symptoms almost disappear, it is not uncommon for obsessional and ritualistic symptoms not only to persist, but also to expand. Finally, adulthood may bring improvement (Gillberg, 1984). Nevertheless, some of these processes can continue through to adulthood, resulting in an individual who, if the past history were to be ignored, might be thought to have Asperger's syndrome.

Although about one quarter of autistic children do moderately well, only 10% do sufficiently well to survive in an unsheltered work situation (Rutter, Greenfield, & Lockyer, 1967; Kanner, Rodriguez, & Ashenden, 1972). Three elements contribute to the limited work potential: poor cognitive development; adverse temperamental features such as inertia, inactivity, and poor concentration; and poor social skills (Lotter, 1974).

The outlook depends on the testable intelligence in the preschool years, the most able tending to show the greatest improvement, particularly on verbal skills (DeMyer et al., 1973). Closely related is the development of useful language in childhood. Other indicators are the severity of the symptomatology, the evidence of organic brain dysfunction, and the speed with which the more florid symptoms fade (Kolvin, 1985), the potential for substantial improvement usually being evident by 7 years of age (Rutter, 1967).

Childhood Schizophrenia

Any conclusion about the course of childhood schizophrenia depends on the narrowness of the diagnostic criteria; inclusion of other disorders clouds the picture. In the literature, autism is a common contaminant (Cantor et al., 1982; Howells & Guirguis, 1984), and so too may be the adolescent affective disorders that, particularly in their early episodes, can be mistaken for schizophrenia (Hassanyeh & Davison, 1980; Himmelhoch & 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 & 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, discordant premorbid personality traits (insecure, inhibited, and shy), and low intelligence (although this is not always the case—Kydd & Werry, 1982), but the presence of affective symptomatology may indicate a better prognosis (Eggers, 1978).

There is no evidence that a range of other factors make any contribution to the prognosis, although such evidence has been sought. These factors include a high family loading of schizophrenic illness or other neuropsychiatric disorders (Eggers, 1978), the duration of episodes, the presence of precipitating triggers, a family history, and family socioeconomic status. It is surprising that factors such as a disturbed family atmosphere, the type of symptoms the child displayed, and the frequency of the psychotic episodes, have no bearing on the prognosis either (Eggers, 1978). Although most of those cases in which onset is before the age of 10 years have a poor outcome, a substantial minority of the prepubertal cases showed a favorable outcome (Eggers, 1978). Finally, in between 20% and 40% of cases the condition can be expected to remit completely, especially if the onset is after 10 years of age; this supports the notion of the prognostic significance of the age of onset. It is also helpful to note that the majority of the children achieve some form of remission; although this was complete in only 20%, a relatively good social adjustment was achieved by a further 30% (Eggers, 1978). However, a significant postpsychotic personality change was found in 26 of the 57 (46%) cases followed by Eggers (1978).

IMPAIRMENT AND COMPLICATIONS

Autism

In autism, impairments are widespread, with some relating to the more general features of autism that have already been adumbrated and

others being more specific to an associated pathological entity. In this section there is a brief focus on the more general impairments outlined by Wing (1982). First and foremost, there are impairments of social interaction, with more recent research emphasizing difficulties in discriminating socioeconomic cues (Hobson, 1986) together with an impaired ability to appreciate how others think, which is based on a theory of mind (Baron-Cohen, 1989). Second, there are delays and deviances of language and communication, which include limitations in the understanding and use of gesture and also deviance in relation to eye-to-eye contact. Third, there are impairments of imaginative activities, as reflected in poor or even lack of symbolic play. These often go hand in hand with poor ability at imitation. There is often an overlay of socially bizarre and inappropriate behavior. Commonly, there are anomalies of motor control and variable anomalies of attention.

Autism is usually accompanied by intellectual impairment (Kolvin, Humphrey, & McNay, 1971; Rutter & Lockyer, 1967) and organic brain dysfunction (Kolvin, Ounsted, & Roth, 1971). As the basis of the organic pathology is so diverse, it has been postulated that there must be a common final pathway to dysfunction (Damasio & Maurer, 1978).

Epilepsy is a frequent complication, developing in about one quarter of autistic children (Deykin & MacMahon, 1979; Gillberg & Steffenberg, 1987; Rutter, 1970); the occurrence of seizures is linked to the intensity of autistic symptomatology, and about two thirds of the fits have an onset in adolescence (Deykin & MacMahon, 1979). In comparison with a normal population, these fits are substantially more frequent and of later onset—the peak being at 11 to 14 years—but it is not clear whether the association is with mental handicap or with autism.

Childhood Schizophrenia

Whereas some workers suggest a modest shift to the left in the distribution of IQ scores (Kolvin, Humphrey, & McNay, 1971), others do not support this (Eggers, 1978; Green et al., 1984). However, all studies report a deterioration from a previous level of general functioning, and, broadly, this has been true irrespective of the type of onset. The frequency of delusions and hallucinations has been described above.

The biological basis of childhood schizophrenia has not been the subject of such extensive research as has been carried out at the level of brain biochemistry, or of that at the level of the modern imaging techniques in adult schizophrenia (Crow, 1987).

Reports of organic impairment have been conflicting and often confused by the inclusion of autistic children. What is available are reports of persistent soft neurological signs, particularly hypotonia (Fish, 1984), poor fine motor coordination, and visuomotor deficits (Marcus, Hans, Lewow, Wilkinson, & Burack, 1985). These and others have been reported in a variety of studies of high-risk children (Nuechterlein, 1986). One interesting report suggested that biological impairment causes a relative inability to distinguish visual signals from background noise, but this is shared with other high-risk children (Asarnow & Mac-Crimmon, 1978; Nuechterlein, 1985). A related finding is that schizophrenic children fail in tasks concerning information processing (Asarnow, Tanguay, Bott, & Freeman, 1987). There is evidence to support the view that much of perceptual and attentional development in children consists of a transition toward more active control of information acquisition. This is achieved, in part, through more efficient deployment of attentional capacity (Asarnow, Sherman, & Strandberg, 1986). These last authors go on to assert that it is precisely these controlled attentional processes, which usually develop in middle childhood, that are impaired in schizophrenic children, while more automatic modes of attending remain relatively intact. To date, in contrast to the condition in adults, a search for other evidence of neurological impairment has yielded little. Nevertheless, a current popular view is that actual or presumed cerebral damage in early life contributes to the emergence of adult schizophrenia (Murray & 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 facts. Although not as common as in autism, presumptive brain damage/dysfunction as reflected in the complications of pregnancy and birth (PBC), 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, Ounsted, & Roth, 1971).

DIFFERENTIAL DIAGNOSIS

Autism

Autism has been characterized by a variety of bizarre behaviors and anomalies of development,

the latter being the basis of the inclusion of autism in the pervasive developmental disorder category with emphasis on the impairment of social relationships, an obsessional desire for sameness, an early onset and associated secondary features relating to communication, cognitive ability, and motor behavior (Kanner, 1943). Other criteria have proved broadly similar in terms of age of onset, self-isolating patterns of social behavior, catastrophic reactions, and repetitive behaviors (Kolvin, 1971). Rutter (1974) has pointed out that, as each of these main symptoms can occur in the absence of others, none can be regarded as specific.

A crucial issue is the differentiation of autism from other disorders of development with which there appear to be shared symptoms. Rutter and Schopler (1988) assert that the main feature distinguishing autism from these other disorders is the deviance in development rather than the delay. This pattern of deviance of social usage of language, of inner language, and socioemotional reciprocity between the child and others distinguishes autism not only from schizophrenic childhood disorders but also from other developmental disorders, particularly those associated with mental retardation. In those other disorders in which there is not this characteristic pattern of deviance but, rather, so-called "secondary autistic features," as may occur, for example, in profound deafness, the child may succeed in communicating through other modalities.

However, it is not merely these features that distinguish autism from childhood schizophrenia. A steady course is more typical of autism, while marked remissions and relapses frequently occur in childhood schizophrenia; marked intellectual retardation is a common feature of autism but less so with childhood schizophrenia; better visuospatial skills and poorer language skills are characteristic of autism but not of schizophrenia; there is a marked male preponderance in autism but equality of the sexes in schizophrenia; there is a higher frequency of adverse perinatal factors and organic factors in autism than in childhood schizophrenia (Kolvin, Ounsted, & Roth, 1971); there is a high genetic loading in the parents of schizophrenic children as well as in adults, but a very low loading in the parents of autists (Kolvin, Ounsted, Richardson, & Garside, 1971; Rutter, 1972). There are similar distinctions between infantile psychosis and adult-type schizophrenia, and for these reasons Rutter (1972) sees no reason for making any distinction between childhoodonset schizophrenia and adult-type schizophrenia.

There is also the issue of the relationship (and differences) between autism and specific receptive developmental language disorders (dysphasia). Although there are a number of overlapping features, such as social withdrawal and repetitive behaviors, sound empirical research has shown that children in the latter group differ from autistic children. Bartak, Rutter, and Cox (1975, 1977) have demonstrated that autism is associated with a language deficit that is more extensive in that it spans several different language modalities, including an impairment of the understanding of written language and also severe comprehension deficits. Furthermore, this language impairment involves deviance as reflected by echolalia, pronominal reversal, and metaphorical language in addition to linguistic delay.

Whereas profoundly mentally handicapped children may have some features of autism, notably they may have two-way social interactions that are appropriate for their developmental age. Thus, while their social interactions may be moderately impaired (Wing & Gould, 1979), they tend not to show the same pattern of deviance that is found in core autistic conditions.

Another question concerns extremely depriving environmental circumstances. It would be difficult to imagine how any of such deprivational experiences could give rise to the complex and specific patterns of impairments in such diverse areas as cognitive, perceptual, motor, and autonomic functioning (Wing, 1976). Rutter and Schopler (1988) point out that neither the indiscriminate friendliness of some institutionally reared children nor the insecurities of abused children (Mrazek & Mrazek, 1985; Rutter, 1981) constituted features reminiscent of autism. However, in some rare cases where children have been exposed to extreme deprivation and abuse, the picture can begin to resemble autism although the pattern of deviance is different (Skuse, 1984). Nevertheless, even here one must question how much of the picture is due to environmental adversity and how much due to constitutional influences (Rutter & Schopler, 1988).

Childhood Schizophrenia

As with the adult condition, childhood schizophrenia has to be differentiated from organic syndromes, affective disorders, and also personality disorders. Common organic conditions are the drug-induced states, such as amphetamine psychosis and temporal lobe epilepsy. Affective states are distinguished on the basis of the persistence of the dysphoria and the relationship of the hallucinations or delusions to the prevailing mood (Gelder, Gath, & Mayou, 1983). A common problem is that in the early stages the schizophrenia may be insidious and can be preceded by quite serious depression (Nissen, 1971). In all the foregoing a careful history is essential, as is an examination of the mental state together with a physical examination to exclude disorders with an organic basis.

The Borderlands of Childhood Psychoses

It has been postulated that the phenomenon of the schizophrenia spectrum may occur in adolescence. Differential diagnosis of these borderland phenomena from core childhood schizophrenia can be difficult and often can be achieved only on the basis of historical accounts of the insidious changes and monitoring over time for evidence of first-rank symptoms.

Asperger's Syndrome and Schizoid Personality Disorder in Childhood

The essential features of Asperger's syndrome are a gross lack of skills in social diplomacy, associated with a degree of naivety leading to an impairment of social relationships (Asperger, 1944). Additional features are male preponderance, diagnosis after the first two years of life, the unusual use of spoken language-particularly pedantic aprosodic speech - the use of stereotyped phrases, and also abnormalities in nonverbal 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 man's land" of classification between autism, on the one hand, and schizophrenia, on the other (Kay & 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 & Barlow, 1979; Wolff & Chick, 1980), on the other. These have been reviewed recently by Kay and Kolvin (1987).

Wolff and Barlow (1979) attempt to highlight

similarities between the adult-derived concept of schizoid personality disorder and Asperger's syndrome. They compare childhood schizoid personality with autistic and normal control children using relatively small samples matched for age, sex, and nonverbal intelligence. Reanalysis of their verbal and performance 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 significant difference between the schizoid personality disorder group and controls (Kay & Kolvin, 1987).

How do these disorders relate to each other? Wing (1981) considered schizoid personality disorder a vague and ill-defined concept, presupposing a relationship to schizophrenia. Asperger's syndrome shares many features with childhood autism, and (although there has been insufficient research into etiological links) it can be viewed as a mild variant of autism on the basis of symptomatology alone (Wing, 1981). The deviance, patterning, and severity of features are insufficient for it to be considered as psychosis but rather as a personality variant. 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, and both should be classified as personality disorders of childhood, probably with each being a mild variant of the other (Kay & Kolvin, 1987). However, from his examination of adult data, Tantam (1988) does not seem to support this position and sees these two conditions as distinct. The answer may be that schizoid personality disorder is a heterogeneous condition that includes cases from both of the two proposed spectra relating to autism and childhood schizophrenia.

Other Borderline States

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 that are relatively independent. For instance, clinicians in the United States use the term "borderline" in two different ways. The first is used to describe a patient group with a disorder assumed to be genetically related to a spectrum of disorders including schizophrenia; this has been labeled "schizotypal personality" to reflect the

concept of "borderline schizophrenia." Family and genetic data are suggestive of a link with schizophrenia (Kendler, 1984). Second, 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 these concepts to children and adolescents whose ego functioning and personalities are not fully developed (Kay & Kolvin, 1987). Steinberg (1983) has drawn a composite picture of the borderline personality from the American literature. It is characterized by complaints of misbehavior-particularly antisocial behavior, 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 behavioral and affective symptoms could equally well be encompassed within the well-known category of mixed conduct-neurotic disorder (Kay & Kolvin, 1987). Despite considerable continuity over time Stone (1984), in his review of the borderline syndromes, suggests that it is difficult to apply the concept meaningfully in adolescence.

CONTINUITY AND DISCONTINUITY WITH ADULT PRESENTATION

Autism

Although there is a lessening of the more bizarre and florid features, the impairments persist to result in an adult with an impoverished imagination and empathy (Bemporad, 1979; Rutter, 1970). Howells and Guirguis (1984) have suggested that this state is indistinguishable from chronic schizophrenia or schizotypal personality disorder (APA, 1980). There are reports of autistic children who subsequently develop schizophrenic syndromes (Petty et al., 1984; Watkins, Asarnow, & Tanguay, 1988; Nunn, Lask, & Cohen, 1986). However, this remains controversial as other research workers, such as Wing and Attwood (1987), report that they have not seen such a case and assert that there is nothing to be gained from locating these in the same category.

Asperger's Syndrome

A ten-year follow-up of 22 childhood cases of schizoid personality disorder found that the personality characteristics persisted into adulthood; in addition one of the patients became floridly schizophrenic and another was hospitalized with symptoms of simple schizophrenia (Wolff & Chick, 1980). In contrast, of Asperger's 200 cases, only 1 developed schizophrenia.

Childhood Schizophrenia

There have been few studies of the premorbid picture in childhood schizophrenia, but research into adult schizophrenia provides some clues. One strategy consists of the follow-up of children with a psychiatric disorder. From this emerges the picture of an emotionally unstable child with interpersonal difficulties leading to rejection by his or her peers and to isolation. Underlying these, and making the child particularly vulnerable, are a catalog of neurodevlopmental deficits, including poor motor functioning and visuospatial and verbal impairments, together with attention deficits (Asarnow & Goldstein, 1986). This pattern, present in about one-half of schizophrenics, although unusual does not depict the schizoid personality that might be expected from the concept of the schizophrenia spectrum (Parnas, Schulsinger, Schulsinger, Mednick, & Teasdale, 1982). However, Asarnow and Ben-Meir (1988) have shown childhood schizotypal personality disorder and schizophrenia to be similar (in contrast to depressive conditions) with regard to poor premorbid adjustment and poorer intelligence. Finally, Parnas and Schulsinger (1986) describe a continuity of formal thought disorder from childhood to adulthood, which implies that schizophrenia does not have a clear-cut onset but may develop by the gradual accretion of symptoms.

There are insufficient data to show that the very variable long-term outcome has any distinctive features or differs from that of adultonset schizophrenia. Follow-up between 9 and 40 years later found 6 of the 57 cases described by Eggers (1978) to be in malignant defect states such as a catatonia or hebephrenia. Delusions, usually unchanged, persisted in about half of the whole population. The personality was unaffected in only 11 of the 57 patients, although, in a further 8, the changes were so slight as to be almost unnoticeable.

SUMMARY

This paper is concerned with a developmental perspective in relation to the classification and diagnosis of the psychoses and allied disorders of childhood and adolescence. It provides evidence of wide differences in psychopathology, epidemiology, natural history, impairments, and complications of autism and childhood schizophrenia and their associated spectrum disorders. The overwhelming implication is that there is little to be gained by attempts to unify these diverse clinical disorders, some of which are evidently psychotic and others merely reflecting forms of personality variations.

REFERENCES

- Anthony, E. J. (1958). An experimental approach to the psychopathology of childhood autism. British Journal of Medical Psychology, 21, 211-225.
- American Psychiatric Association (1980). Diagnostic and statistical manual of mental disorders (3rd ed.). Washington, DC: Author.
- Asarnow, J. R., & Goldstein, M. J. (1986). Schizophrenia during adolescence and early adulthood: A developmental perspective on risk research. Clinical Psychology Review, 6, 211-235.
- Asarnow, J. R., & Ben-Meir, S. (1988). Children with schizophrenia spectrum and depressive disorders: A comparative study of premorbid adjustment, onset pattern and severity of impairment. *Journal of Child Psychology and Psychiatry*, 29, 825-838.
- Asarnow, R. F., & MacCrimmon, D. J. (1978). Residual performance deficit in chronically remitted schizophrenics: A marker of schizophrenia? *Journal of Abnormal Psychology*, 87, 267-275.
- Asarnow, R. F., Sherman, T., & Strandburg, R. (1986). The search for the psychobiological substrate of childhood onset schizophrenia. Journal of the American Academy of Child Psychiatry, 26(5), 601-614.
- Asarnow, R. F., Tanguay, P. E., Bott, L., & Freeman, B. J. (1987). Patterns of intellectual functioning in non-retarded autistic and schizophrenic children. *Journal of Child Psychology and Psychiatry*, 28, 273-280.
- Asperger, H. (1944). Die "autistischen psychopathen" im kindesalter [The autistic psycho-

- pathology in childhood]. Archiv fur Psychiatrie und Nervenkrankheiten, 117, 76-136.
- Attwood, A. (1984). The gestures of autistic children. Unpublished PhD thesis, University of London.
- Babigan, H. M. (1980). Schizophrenia: Epidemiology. In H. I. Kaplan, A. M. Freedman, & B. J. Sadock (Eds.), Comprehensive textbook of psychiatry. Baltimore, MD: Williams & Wilkins.
- Baron-Cohen, S. (1989). The autistic child's theory of mind: A case of specific developmental delay. *Journal of Child Psychology and Psychiatry*, 30, 285-297.
- Bartak, L., Rutter, M., & Cox, A. (1975). A comparative study of infantile autism and specific developmental receptive language disorders: I. The children. *British Journal of Psychiatry*, 126, 127-145.
- Bartak, L., Rutter, M., & Cox, A. (1977). A comparative study of infantile autism and specific developmental receptive language disorders:
 III. Discriminant function analysis. *Journal of Autism and Childhood Schizophrenia*, 6, 297–302.
- Beitchman, J. H. (1985). Childhood schizophrenia. Psychiatric Clinics of North America, 8, 793-814.
- Bemporad, J. R. (1979). Adult recollections of a formerly autistic child. *Journal of Autism and Developmental Disorders*, 9, 179-198.
- Bleuler, E. (1950). Dementia praecox or the group of schizophrenias (J. Zinkin, trans.). New York: International University Press.
- Brockington, I. (1983). Schizophrenia: Fact and fiction. Inaugural Lecture. Birmingham, England: University of Birmingham.
- Brockington, I., Kendell, R. E., & Leff, J. P. (1978). Definitions of schizophrenia: Concordance and predictions of outcome. Psychological Medicine, 8, 387.
- Bryson, S. E., Clark, B. S., & Smith, I. M. (1988). First report of a Canadian epidemiological study of autistic syndromes. *Journal of Child Psychology and Psychiatry*, 29, 433-446.
- Cantor, S., Evans, J., Pearce, J., & Pezzot-Pearce, J. (1982). Childhood schizophrenia: Present but not accounted for. *American Journal of Psychiatry*, 139, 758-762.
- Corbett, J., Harris, R., Taylor, E., & Trimble, M. (1977). Progressive disintegrative psychosis in childhood. *Journal of Child Psychology and Psychiatry*, 18, 211-219.

- Creak, M. (1963). Childhood psychosis: A review of 100 cases. *British Journal of Psychiatry*, 109, 84-89.
- Crow, T. J. (1987). Neurochemistry of schizophrenia in the living brain. *Transmission*, 1(2), 4-9.
- Damasio, A. R., & Maurer, R. G. (1978). A neurological model for childhood autism. Archives of Neurology, 35, 777-786.
- DeMyer, M. K., Barton, S., DeMyer, W. E., Norton, J. A., Allen, J., & Steele, R. (1973). Prognosis in autism: A follow-up study. Journal of Autism and Childhood Schizophrenia, 3, 199-246.
- Denckla, M. B. (1986). Editorial: New diagnostic criteria for autism and related behavioral disorder—guidelines for research protocols. Journal of the American Academy of Child Psychiatry, 25, 221-224.
- Deykin, E. Y., & MacMahon, B. (1979). The incidence of seizures among children with autistic symptoms. *American Journal of Psychiatry*, 136, 1310-1312.
- Egdell, H. G., & Kolvin, I. (1972). Childhood hallucinations. *Journal of Child Psychology and Psychiatry*, 13, 279-287.
- Eggers, C. (1978). Course and prognosis of child-hood schizophrenia. *Journal of Autism and Childhood Schizophrenia*, 8, 21-36.
- Evans-Jones, L. G., & Rosenbloom, L. (1978). Disintegrative psychosis in childhood. Developmental Medicine and Child Neurology, 20, 462-470.
- Fish, B. (1977). Neurobiologic antecedents of schizophrenia in children. *Archives of General Psychiatry*, 34, 1297–1313.
- Fish, B. (1984). Characteristics and sequelae of the neurointegrative disorder in infants at risk for schizophrenia: 1952-1982. In N. Watt, E. J. Anthony, L. C. Wynne, & J. Rolf (Eds.), Children at risk for schizophrenia: A longitudinal perspective. New York: Cambridge University Press.
- Garralda, M. E. (1984a). Hallucinations in children with conduct and emotional disorders: I. Study of the children. Psychological Medicine, 14, 589-596.
- Garralda, M. E. (1984b). Hallucinations in children with conduct and emotional disorders: II. A follow-up study. Psychological Medicine, 14, 597-604.
- Gelder, M., Gath, D., & Mayou, R. (1983). Oxford textbook of psychiatry. Oxford: Oxford University Press.

- Gillberg, C. (1984). Infantile autism and other childhood psychoses in a Swedish region: Epidemiological aspects. Journal of Child Psychology and Psychiatry, 25, 35-43.
- Gillberg, C., & Gillberg, I. (1988, June). The epidemiology of Asperger syndrome. Paper presented at the First Asperger Syndrome Symposium, London.
- Gillberg, C., & Schaumann, H. (1981). Infantile autism and puberty. Journal of Autism and Developmental Disorders, 11, 365-371.
- Gillberg, C., & Steffenberg, S. (1987). Outcome and prognostic factors in infantile autism and similar conditions: A population-based study of 46 cases followed through puberty. *Journal of Autism and Developmental Disorders*, 17, 273-287.
- Green, W. H., Campbell, M., Hardesty, A. S., Grega, D. M., Padron-Gayol, M., Shell, J., & Erlenmeyer-Kimling, L. (1984). A comparison of schizophrenia and autistic children. *Journal of the American Academy of Child Psychiatry*, 23, 399-409.
- Hassanyeh, F., & Davison, K. (1980). Bipolar affective psychosis with onset before age 16 years: Report of 10 cases. *British Journal of Psychiatry*, 37, 530-539.
- Hill, A. E., & Rosenbloom, L. (1986). Disintegrative psychosis of childhood: Teenage follow-up. Developmental Medicine and Child Neurology, 28, 34-40.
- Himmelhoch, J. M., & Garfinkel, M. E. (1986). Sources of lithium resistance in mixed mania. Psychopharmacological Bulletin, 22, 613-620.
- Hobson, R. P. (1986). The autistic child's appraisal of expressions of emotion: An experimental investigation. *Journal of Child Psychology and Psychiatry*, 27, 321-342.
- Howells, J. G., & Guirguis, W. R. (1984). Child-hood schizophrenia 20 years later. Archives of General Psychiatry, 41, 123-127.
- Kanner, L. (1943). Autistic disturbances of affective contact. Nervous Child, 2, 217-250.
- Kanner, L., Rodriguez, A., & Ashenden, B. (1972). How far can autistic children go in matters of social adaptation? *Journal of Autism and Childhood Schizophrenia*, 2, 9-33.
- Kay, P., & Kolvin, I. (1987). Childhood psychoses and their borderlands. *British Medical Bulletin*, 43, 570-586.
- Kendler, K. S. (1984). A family history study of schizophrenia-related personality disorders. American Journal of Psychiatry, 141, 424-427.

 Kolvin, I. (1971). Studies in childhood psychoses:
 I. Diagnostic criteria and classification. British Journal of Psychiatry, 118, 381-384.

Kolvin, I. (1985). Childhood autism. In M. Craft, J. Bicknell, & S. Hollin (Eds.), Mental handicap (13th ed.) (pp. 147-161). London: Balliere Tindall.

Kolvin, I., Garside, R., & Kidd, J. S. H. (1971). Studies in childhood psychoses: IV. Parental personality and attitude and childhood psychoses. *British Journal of Psychiatry*, 118, 403-406.

Kolvin, I., Humphrey, M., & McNay, A. (1971). Studies in childhood psychoses: VI. Cognitive factors in childhood psychoses. *British Journal* of Psychiatry, 118, 415-419.

Kolvin, I., Ounsted, C., Humphrey, M., & Mc-Nay, A. (1971). Studies in childhood psychoses: II. The phenomenology of childhood psychoses. British Journal of Psychiatry, 118, 385-395.

Kolvin, I., Ounsted, C., Richardson, L. M., & Garside, R. F. (1971). Studies in childhood psychoses: III. The family and social background in childhood psychoses. *British Journal of Psychiatry*, 118, 396-402.

Kolvin, I., Ounsted, C., & Roth, M. (1971). Studies in childhood psychoses: V. Cerebral dysfunction and childhood psychosis. British Journal of Psychiatry, 118, 407-414.

Kramer, M. (1978). Population changes and schizophrenia, 1970-1985. In L. C. Wynne, R. L. Cromwell, & S. Matthysse (Eds.), The nature of schizophrenia: New approaches to research and treatment. New York: John Wiley & Sons.

Kydd, R. R., & Werry, J. S. (1982). Schizophrenia in children under 16 years. *Journal of Autism and Developmental Disorders*, 12, 343-358.

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

Lotter, V. (1974). Social adjustment and placement of autistic children in Middlesex: A follow-up study. Journal of Autism and Childhood Schizophrenia, 4, 11-32.

Makita, K. (1966). The age of onset of childhood schizophrenia. Folia Psychiatrica Neurologica

Japonica, 20, 111-121.

Marcus, J. Hans, S. Lewow, E., Wilkinson, L., & Burack, C. (1985). Neurological findings in the offspring of schizophrenics: Childhood assessment and 5-year follow-up. Schizophrenia Bulletin, 11, 85-100. Masterson, J. C. (1956). Prognosis in adolescent disorders: Schizophrenia. *Journal of Nervous and Mental Disorder*, 124, 219-232.

Mrazek, D., & Mrazek, P. (1985). Child maltreatment. In M. Rutter & L. Hersov (Eds.), Child and adolescent psychiatry: Modern approaches (2nd ed.) (pp. 545-566). Oxford: Blackwell Scientific.

Murray, R. M., & Lewis, S. W. (1987). Is schizophrenia a neurodevelopmental disorder? British Medical Journal, 295, 681-682.

Nuechterlein, K. H. (1985). Converging evidence for vigilance deficit as a vulnerability indicator for schizophrenic disorders. In M. Alpert (Ed.), Controversies in schizophrenia: Changes and constancies (pp. 175-198). New York: Brunner/Mazel.

Nuechterlein, K. H. (1986). Childhood precursors of adult schizophrenia. *Journal of Child Psychology and Psychiatry*, 27, 133-144.

Nunn, K. P., Lask, B., & Cohen, M. (1986). Viruses, neurodevelopmental disorder and childhood psychosis. Journal of Child Psychology and Psychiatry, 27, 55-64.

Parnas, J., & Schulsinger, H. (1986). Continuity of formal thought disorder from childhood to adulthood in a high-risk sample. Acta Psychiatrica Scandinavica, 74, 246-251.

Parnas, J., Schulsinger, F., Schulsinger, H., Mednick, S. A., & Teasdale, T. W. (1982). Behavioural precursors of schizophrenia spectrum. A prospective study. Archives of General Psychiatry, 39, 658-664.

Petty, L. K., Ornitz, E. M., Michelman, J. D., & Zimmerman, E. G. (1984). Autistic children who become schizophrenic. Archives of General Psychiatry, 41, 29-135.

Rivinus, T. M., Jamison, D. L., & Graham, P. J. (1975). Childhood organic neurological disease presenting as a psychiatric disorder. *Archives of Disease in Childhood*, 50, 115-119.

Roth, M., & McClelland, H. (1979). Problems of diagnosis and treatment in the borderlands of schizophrenia. In J. M. Simister (Ed.), Neuroleptics and schizophrenics (pp. 63-81). Luton, England: Lundbeck House.

Rutter, M. (1967). Psychotic disorders in early childhood. In A. J. Coppen & A. Walk (Eds.), Recent developments in schizophrenia (pp. 133-151). Ashford, England, RMPA.

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

Rutter, M. (1972). Childhood schizophrenia re-

- considered. Journal of Autism and Childhood Schizophrenia, 2, 315-317.
- Rutter, M. (1974). The development of infantile autism. *Psychological Medicine*, 4, 147-163.
- Rutter, M. (1981). Maternal deprivation reassessed (2nd ed.). Harmondsworth, England: Penguin.
- Rutter, M. (1985). Infantile autism and other pervasive disorders. In M. Rutter & L. Hersov (Eds.), Child and adolescent psychiatry: Modern approaches (2nd ed.) (pp. 545-566). Oxford: Blackwell Scientific.
- Rutter, M., Greenfield, D., & Lockyer, L. (1967). A five to fifteen year follow-up study of infantile psychosis: II. Social and behavioural outcome. *British Journal of Psychiatry*, 113, 1183-1199.
- Rutter, M., & Lockyer, L. (1967). A five to fifteen year follow-up study of infantile psychosis: I. Description of the sample. *British Journal of Psychiatry*, 113, 1169-1182.
- Rutter, M., & Schopler, E. (1988). Autism and pervasive developmental disorders. In M. Rutter, T.
 A. Hussain, & I. S. Lann (Eds.), Assessment and diagnosis in child psychopathology (pp. 408-434). New York: Guilford Press.
- Schneider, K. (1959). Clinical psychopathology (M. W. Hamilton, trans.). London: Grune & Stratton.
- Skuse, D. (1984). Extreme deprivation in early childhood: I. Diverse outcomes for three siblings from an extraordinary family. *Journal of Child Psychology and Psychiatry*, 26, 523-541.
- Spitzer, R. L., & Endicott, J. (1979). Justification for separating schizotypal and borderline personality disorders. Schizophrenia Bulletin, 5, 95-104.
- Stern, A. (1938). Psychoanalytic investigation and therapy in the borderline group of neuroses. *Psychoanalytic Quarterly*, 7, 467-489.
- Steinberg, D. (1983). Schizoid personality and the borderline state. In D. Steinberg (Ed.), The clinical psychiatry of adolescence: Clinical work from a social and developmental perspective. Chichester, England: John Wiley & Sons.
- Steinhausen, H.-C., Gobel, D., Breinlinger, M., & Wohleben, B. (1983, October). A community survey of infantile autism. Paper presented

- at the 30th annual meeting of the American Academy of Child Psychiatry, San Francisco.
- Stone, M. H. (1984). Borderline syndromes: A consideration of sub-types and an overview, direction for research. *Psychiatric Clinics*, 4, 23.
- Tantam, D. (1988). Lifelong eccentricity and social isolation: II. Asperger's syndrome or schizoid personality disorder. British Journal of Psychiatry, 153, 783-791.
- Volkmar, F. R. (1987). Diagnostic issues in the pervasive developmental disorders. *Journal of Child Psychology and Psychiatry*, 28, 365-370.
- Watkins, J. M., Asarnow, R. F., & Tanguay, P. E. (1988). Symptom development in childhood onset schizophrenia. *Journal of Child Psy*chology and Psychiatry, 29, 865-878.
- Wing, L. (1981). Aspergers's syndrome: A clinical account. *Psychological Medicine*, 11, 115-129.
- Wing, L. (1982). Development of concepts, classification and relationship to mental retardation. In L. Wing & J. K. Wing (Eds.), *Handbook of psychiatry: Vol. 3. Psychoses of uncertain aetiology* (pp. 185-190). Cambridge: Cambridge University Press.
- Wing, L., & Attwood, A. (1987). Syndromes of autism and atypical development. In D. J. Cohen & A. M. Donellan (Eds.), *Handbook of autism and pervasive developmental disorders* (pp. 3-19). New York: John Wiley & Sons.
- Wing, L., & Gould, J. (1978). Systematic recording of behaviours and skills of retarded and psychotic children. *Journal of Autism and Childhood Schizophrenia*, 8, 79-97.
- Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders*, 9, 11-30.
- Wolff, S., & Barlow, A. (1979). Schizoid personality in childhood: A comparative study of schizoid, autistic, and normal children. *Journal of Child Psychology and Psychiatry*, 20, 29-46.
- Wolff, S., & Chick, J. (1980). Schizoid personality in childhood: A controlled follow-up study. *Psychological Medicine*, 10, 85-100.
- World Health Organization. (1979). International classification of diseases, injuries, and causes of death (9th ed.). Geneva: Author.