Schizophrenia in children and adolescents

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The late William Ll. Parry-Jones

Introduction

For over 100 years, the onset of schizophrenia during adolescence has been accepted, but its onset in childhood, the question of its equivalence with the adult disorder, and the possibility of childhood antecedents of the adult form have been controversial clinical and research issues. Until 20 to 30 years ago, child psychiatrists were reluctant to acknowledge or diagnose psychoses in children and adolescents, and various euphemistic terms tended to be used. Even at present, there may be a disinclination to do so because of fears about the potentially adverse consequences of diagnostic labeling. Many factors have contributed to this, principally infrequency of presentation and uncertainty about diagnosis and classification, since formerly, the term schizophrenia has been used to refer to a remarkable range of severe, chronic disorders arising in children, with little or no differentiation. Furthermore, there has been a lack of research-based therapeutic guidelines. Recent years, however, have witnessed a rebirth of interest in schizophrenia in children, especially in identifying continuities and discontinuities with the condition presenting in adolescents and adults. In this context, the general aim of this chapter is to review the literature relating to schizophrenia in children and adolescents up to the 1970s, setting in historical perspective some of the issues presented and discussed in subsequent chapters, particularly in relation to diagnosis and classification.

Historiography of schizophrenia

Historical consideration of the concept of schizophrenia in children has to be located in the wider context of historical research on the functional psychoses as a whole, particularly schizophrenia (Howells, 1991; Berrios & Porter, 1995, pp. 261–430). The historiography of a subject of such complexity is fraught with methodological difficulties, including wide variation in the content and quality

of sources, especially manuscript material; uncertainty and imprecision about which symptoms or morbid processes constitute schizophrenia, even at the present day; and all the hazards associated with retrospective diagnosis and interpretation of both theoretical constructs and clinical practice.

Limited historical research has been undertaken on the history of schizophrenia in children using primary printed or manuscript sources. However, the historical background has been the subject of a number of reviews, including those by Bradley (1942), Lurie and Lurie (1950), Eisenberg (1957), Goldfarb (1970), Rutter (1972), Fish and Ritvo (1979), Cantor (1988), Bender (1991), Werry (1992) and Remschmidt et al. (1994). An annotated bibliography by Goldfarb and Dorsen (1956) reviewed the relevant literature up to 1954, and further coverage to 1969 was provided by Tilton et al. (1966) and Bryson and Hintgen (1971).

Before 1900: pre-nineteenth century

There has been considerable controversy whether schizophrenia has always existed or if it is of relatively recent origin (Klaf & Hamilton, 1961; Hare, 1988). The search for evidence of schizophrenia in the past is complicated by frequent terminological and nosological changes and overlaps. For example, since it was not viewed as a discrete entity until the end of the nineteenth century, it might have been perceived as a form of delirium, mania, melancholia, dementia, imbecility or idiocy. In general, the historical evidence lends support for its enduring nature, albeit associated with changing manifestations (Jeste et al., 1985; Howells, 1991). Up to the medieval period, the evidence is highly equivocal, but subsequently, to the end of the nineteenth century, the case for the existence of schizophrenia strengthens. In the absence of precisely defined diagnostic entities, it is difficult to reconstruct a picture of psychosis in juveniles or to estimate its prevalence in pre-nineteenth century accounts of insane children and young people. Instead, the focus has to be on common denominators, such as evidence of a qualitative difference in mental state and behavior in the absence of overt organic brain disease, such as odd, bizarre, incongruous, unintelligible thinking and behavior, and the presence of delusions and hallucinations. A diverse range of literature requires scrutiny including, for example, accounts of alleged demonic possession in juveniles (e.g., Baddeley, 1622), since seventeenth century perceptions of madness were intertwined with beliefs in witchcraft. Childhood disorders featured only sporadically in eighteenth-century lunacy texts (e.g., Perfect, 1791).

Nineteenth century

During the first half of the century, increasing publication of unusual cases and references to young lunatics indicated mounting medical interest. Haslam's detailed account, in 1809, of a disorder occurring in young persons associated with ''hopeless and degrading change'' is widely quoted as an early, if not the first, description of schizophrenia (Haslam, 1809, p. 64). The prevailing view was that madness rarely occurred before puberty, although a small number of cases of insane children were described (e.g., Cox, 1804; Rush, 1812; Burrows, 1828; Morison, 1828). Esquirol (1845), whose account of mania strongly suggests schizophrenia, described cases of mania in children, one child being reported as having hallucinations of taste and vision, but no link was established with a progressive dementing process. This emerged when Morel (1860) drew attention, ahead of Kraepelin, to premature dementia (*démence précose*) in a 14-year-old boy.

From the 1860s onwards, childhood insanity featured regularly in psychiatric publications, with references to conditions comparable to psychosis. It was accepted that all forms of mental disease that occurred in adults could present in children (e.g., Crichton-Browne, 1860; Maudsley, 1867). The relationship between the occurrence and form of insanity and the developmental stage was recognized. For example, Griesinger (1867) observed that monomania was ''uncommonly rare'' in children, because ''no persistent ego is as yet formed in which there could occur a lasting radical change.'' It was noted also that hallucinations and ''fixed delirious ideas'' were rarer than in adults (Griesinger, 1867, p. 143). However, mental diseases were regarded as more frequent after the age of 16 (Griesinger, 1867, p. 145). Similarly, Ireland described the changing manifestations of insanity in children according to age and development (Ireland, 1898, pp. 271–302). These could include hallucinations, especially in older children, but again, ''fixed ideas and delusions'' were not common, ''even when derangement is very decided, there is a want of persistence in the mental delusions of children.'' Down (1887) reported that cases had come to his attention with ''well-marked delusions of suspicion'' (Down, 1887, p. 92) and another example was provided by the ten cases of childhood psychoses described by Schöntal (1892).

Towards the end of the century, monographs on childhood insanity appeared, including innovative works by Emminghaus (1887) in Germany and Moreau de Tours (1888) and Manheimer (1899) in France. In addition, adolescent disorders became a new focus of interest, with puberty recognized increasingly as a physiological cause of mental disturbance. ''Developmental,'' ''pubescent'' or ''adolescent'' insanity was described frequently (e.g., Clouston,

1892). Adolescence was perceived as an important period for the emergence of ''ancestral influences'' and atavisms and for its predisposition to dementia praecox and manic-depressive insanity.

In addition to Morel's description of *démence précose*, other psychotic syndromes were delineated in the late-nineteenth century. Hebephrenia was reported by Hecker (1871), and Kahlbaum (1874) described catatonia in adolescents, culminating in terminal deterioration. In his seminal work, publicized in 1898, Kraepelin (1919) grouped together such syndromes within the diagnostic entity of dementia praecox, which in his view, was based on organic brain pathology and progressed inevitably towards mental deterioration. The subtypes were catatonic, hebephrenic, paranoid and simple. He made brief reference to the possibility of early onset during adolescence. Dementia praecox could commence in childhood and, in at least 3.5% of his 1054 cases, onset was before the age of 10 years, with another 2.7% of cases beginning between 10 and 15 years.

Twentieth century

The literature on psychoses in the early twentieth century was influenced strongly by Kraepelinian ideas, including the concept of dementia praecox as a discrete disease entity. These were reflected in the growing consideration of psychosis in children. Increasingly, individual case reports of juvenile psychosis, especially those occurring in the prepubertal period appeared, and the diagnosis tended to become inclusive, comprising many types of pubertal and adolescent insanity. It was accepted that all known psychoses could present during this period, the only form occurring exclusively at this time being hebephrenia. The latter was characterized by ''a change of superficial emotional conditions, beginning with mental depression, followed by odd, fantastic delusions, eccentric, silly behavior, and intense motor activity, and resulting often in a rapid or gradual passage into chronic dementia or into the condition of catatonia'' (Noyes, 1901).

In 1906, de Sanctis described *dementia praecocissimia*, in cases occurring in very early life, presenting with mannerisms, negativism and catatonic symptoms, which he thought resembled demmentia praecox (de Sanctis, 1906, 1925). Later, it was shown to comprise a collection of heterogeneous diseases, including the sequelae of encephalitis. In 1908, Heller described cases of a condition that he termed *dementia infantilis* (previously used by Weygandt), in which, like dementia praecocissima, there was rapid and profound speech disturbance, extreme restlessness and dementia, beginning in the third or fourth years, in previously normal children (Heller, 1908; Hulse, 1954). Other

authors (e.g., Zappert, 1921) described similar conditions (Lay, 1938), but differentiated from schizophrenia, and by the mid-1930s, Kanner felt able to assert that Heller's syndrome was ''an illness *sui generis* and should not be identified with schizophrenia'' (Kanner, 1935, p. 493). Nevertheless, the concept of a disintegrative psychosis continued to feature in the differential diagnosis of childhood schizophrenia.

Introduction of the term ''schizophrenia''

In his 1911 monograph, Bleuler (1950) introduced the term schizophrenia for dementia praecox. Although he was thinking of ''the group of schizophrenias,'' in practice, the two disorders came to be viewed as synonymous. There was, however, a widening of Kraepelin's diagnostic construct, making schizophrenia progressively less readily definable. Bleuler estimated that 0.5 to 1% of schizophrenia cases had onset before the age of 10, 4% beginning before 15 years. He stated that ''schizophrenia is not a puberty psychosis in the strict sense of the word, although in the majority of patients the sickness becomes manifest soon after puberty.'' Further, he noted that ''we know of no differences between the infantile and other forms of the disease. If we observe patients during childhood, they present the same symptoms as those seen in adults. We did note, however, that the analyses of such youthful patients are more difficult. In contrast to adults, children are not less clear in their desires and wishes, but the content is less clearly defined. The difficulty may also be due to our inadequate experience with the technique of handling youthful psychotics. The prognosis of those cases in which the onset of the illness occurs before puberty does not appear to be poorer for the next few years'' (Bleuler, 1950, pp. 240–1). Unlike Kraepelin, Bleuler did not believe that schizophrenia led inevitably to deterioration and emphasized intrapsychic and psychosocial aspects, encouraging a more positive and optimistic therapeutic attitude. In this respect, his views resembled those of Meyer (1906), who first raised the issue of the patient's previous personality and proposed a unique psychobiological basis for disorder, whereby schizophrenia could be regarded as the consequence of defective life accommodation. Such approaches were found more applicable to children than the more restrictive, pessimistic Kraepelinian views.

Increasing recognition of schizophrenia in childhood: the 1930s and 1940s

Up to the 1930s, dementia praecox or schizophrenia was described and diagnosed in small numbers of children using the same criteria as applied to adult patients (see Lay, 1938 for early twentieth century reflection, e.g., to the contributions of Vogt, Aubry, Voigt, and Weber). Initially, the literature on

childhood psychoses was limited compared with that about psychoses in adolescents and adults (e.g., Lutz, 1937; Bellak, 1948) and many psychiatrists disputed the existence of childhood schizophrenia. Subsequently, this concept became increasingly accepted, with wider and more encompassing criteria, and attention shifted from the study of a disease entity to the personal and developmental characteristics and family environment of psychotic children. This encouraged the polarization of the concepts of child and adult schizophrenia, with emphasis on the differences in causation, diagnosis and treatment, particularly on the developmental aspects. In this context, concurrent developments in child and adolescent psychiatry were relevant. During the late 1920s and 1930s, a recognizably separate discipline of child psychiatry emerged, accompanied by the rapid widening of the number and range of disturbed children presenting to child guidance clinics and to the early hospital-based outpatient departments (Parry-Jones, 1993). Further, new psychological and psychiatric methods of investigation came into use for the objective study of the child. The developing speciality directed attention away from the impersonal disease model, organ pathology, heredity, syndromal description and physical treatment, towards psychosocial and psychodynamic theories. In the process, it distanced itself from asylum psychiatry and from the most severely disturbed subjects, particularly adolescents with psychotic disorders.

Interest in schizophrenia in children and its diverse connotations expanded rapidly (e.g., Kasanin & Kaufman, 1929) and from the 1930s, there was a striking increase in related literature (Goldfarb & Dorsen, 1956), including sections in child psychiatry textbooks (e.g., Homberger, 1926; Kanner, 1935). The crucial questions were whether childhood schizophrenia was the same as adult dementia praecox, and what constituted the adult outcome of the childhood disorder. In a widely reported paper, Potter (1933) described six cases of childhood schizophrenia, which, typically, could occur before pubescence, including paranoid delusions, bizarre fantasies, auditory hallucinations and thought disorder. His diagnostic criteria were broad and he recognized the importance of developmental stage, since children ''do not possess the facility to fully verbalise their feelings, nor are they capable of complicated abstractions,'' their ''delusional formations'' are relatively simple and ''symbolisation is particularly naive.'' In the first edition of his influential textbook, Kanner (1935, pp. 484–507), discussed the major psychoses or ''parergastic reaction forms'' (characterized by odd, archaic types of behavior), using Meyerian concepts, to distinguish them from ''thymergastic reaction forms'' or affective disorders. He included three case illustrations, in which he emphasized antecedent factors, and observed that ''schizophrenic difficulties did not come upon

the patients out of a clear sky as a result of some cellular destruction or abscessed teeth or endocrine disorder or whatnot'' (Kanner, 1935, p. 500). Even though there was little to be done once ''dilapidation'' had set in, Kanner supported the notion of prophylaxis with children displaying ''daydreaming preoccupations, seclusive trends, oversensitiveness, and peculiar behavior'' (Kanner, 1935, pp. 501–2). Individual case reports and discussions proliferated, especially those relating to early onset psychosis (e.g., Lutz, 1937) and one of the first comprehensive reviews was published by Bradley (1942), in which he emphasized the primary significance of seclusiveness, bizarre behavior and regression. Based on work at the New York State Psychiatric Institute, from 1930 to 1937, Despert (1938) defined schizophrenia as a ''disease process in which the loss of affective contact with reality is coincident with or determined by the appearance of autistic thinking and accompanied by specific phenomena of regression and dissociation.'' Attention was given to onset history in clarifying life course and treatment response. The therapeutic task was to establish affective contact and to break into the child's autistic world. Although the ideas of Kraepelin and Bleuler continued to be influential, the Freudian psychoanalytic system (Freud, 1924) and Meyerian views were having an increasing effect. In the 1920s, for example, Klein started to use psychoanalytic treatment with young schizophrenic children, and later developed her techniques in conjunction with A. Freud.

Difficulties in the differentiation of childhood schizophrenia from mental deficiency and from deafness with mutism began to be reported. It was recognized that schizophrenia could be diagnosed as mental deficiency, and that schizophrenic children could be sent erroneously to institutions for the mentally deficient. Alternatively, schizophrenia could be superimposed on underlying feeble-mindedness to create the condition known as *pfropfhebephrenia* (Kasanin & Kaufman, 1929) or *pfropfschizophrenia* (Bromberg, 1934; Bergman et al., 1951), and Earl (1934) described a condition in low-grade mental defectives as ''primative catatonic psychosis of idiocy,'' characterized by signs of deterioration, catatonia and emotional dissociation. Ambiguities persisted and O'Gorman (1954) for example, considered schizophrenia as a possible cause, rather than a sequel, of mental deficiency.

The greatest problems in differential diagnosis arose, however, following Kanner's introduction of the term *early infantile autism* (Kanner, 1943), to classify certain conditions regarded as psychoses that occurred as early as in the first 2 years of life, characterized by extreme aloneness, impaired communication, obsessive insistence on sameness and fascination for objects. This was followed by application of the term to a wider group of disturbed children and

confusion about its relationship to schizophrenia. For example, Kanner (1949) noted that ''the basic nature of its manifestations is so intimately related to the basic nature of childhood schizophrenia as to be indistinguishable from it, especially from cases with insidious onset.'' Although there were claims that autistic children became schizophrenic, findings were controversial and uncorroborated (Bender, 1953) and, finally, Kanner changed his view (Kanner, 1971). The description of ''hyperkinetic disease'' in children in the early 1930s, and its classification as a form of childhood psychosis, further complicated the diagnostic picture (Lay, 1938). In general, psychoses caused by organic brain disease, whether due to trauma, neoplasms, infection, toxic agents, metabolic aberrations or degenerative diseases, were categorized separately. Isolated accounts of mania and hypomania, similar to the adult picture appeared more frequently among adolescents than prepubertal children (Parry-Jones, 1995). With regard to the manifestations of psychosis in children, there was particular controversy about the frequency and form of hallucinations and delusions (Lurie & Lurie, 1950).

Towards a unitary view of childhood psychosis: 1950s to 1970s

During the 1950s and 1960s, American and European concepts of schizophrenia diverged increasingly. In the USA, it had broadened and become much less precisely defined, while in Europe, the somatic Kraepelinian model tended to be retained and popularized by Schneider's publication of the first- and second-rank symptoms of schizophrenia, as defined in German psychiatry (Schneider, 1939). During the 1970s, the introduction of research-based diagnostic criteria was to change the situation, attaining increasing consensus (e.g., Feighner et al., 1972).

The amorphous nature of the symptoms of schizophrenia spawned many studies in the 1950s and 1960s, which reviewed and revised the diagnostic criteria of childhood psychoses, drawing on highly diversified clinical descriptions and causative theories. By this stage, an essentially unitary view of chidhood psychosis had emerged. As well as having specific meanings, the labels of childhood psychosis, or childhood schizophrenia, were employed to cover the whole range of psychotic conditions of childhood, as well as many different types of disturbance which might incorporate mental deficiency, severe emotional and behavioral disorders, and the effects of severe deprivation. Harms (1952) asserted that, ''If anyone were to take the trouble to summarize the descriptions of childhood schizophrenia by various authors in the past fifteen years, they would find every symptom ever occurring in abnormal psychology.'' This amalgamation was to be reflected formally in

DSM-II (American Psychiatric Association, 1968) and ICD-8 (World Health Organization, 1967), which used a single generic category of ''childhood schizophrenia.'' Many authors, however, were critical of the lack of clarity in diagnostic criteria (e.g., Harms, 1952) and were concerned especially about the differential diagnosis between disorders with, and without, evidence of brain damage. Kestenberg (1952) discussed differentiation from a specific type of severe neurosis, which she termed *pseudo-schizophrenia*. Other groups of workers continued to dispute whether schizophrenia occurred at all in children and Rank (1949), for example, preferred to apply the term ''atypical'' to children displaying ''arrested emotional development'' and ''fragmented scattered personality.'' Although there was greater acceptance of the occurrence of schizophrenia in adolescents, there was uncertainty about its nature, its relationship to adolescent maturation and ''whether or not an individual who becomes schizophrenic in adolescence is manifesting a disease that had been lurking within the personality since childhood, or earlier'' (Neubauer & Steinert, 1952).

Long-term work, commencing in the 1940s, of Bender and colleagues at Bellevue Hospital, New York, who observed more than 100 preadolescent children suffering from schizophrenia, was especially influential. It emphasized positive diagnostic criteria, rather than diagnosis by exclusion, in that ''the child must not be mentally defective, must not be post-encephalitic, the disturbance must not be understandable in mechanistic terms like a deeply inhibited or discouraged neurosis, and the child must not be a psychopathic personality'' (Bender, 1941). Bender (1958) viewed childhood schizophrenia as a ''total psychobiologic disorder'' and distinguished between the early onset ''pseudo-defective group,'' resembling infantile autistic children with onset in the first 2 years, the ''pseudo-neurotic,'' aged between 3 and 5 years, who displayed anxiety, and the ''pseudo-psychopathic or antisocial,'' aged 10 or 11 years. The same child, therefore, might pass through all these reactions, thereby blurring the definition between autism and schizophrenia. Her theories centered around the concept that schizophrenia was the consequence of a developmental lag of the biological processes from which behavior developed by maturation at an embryological level, characterized by embryonic plasticity, resulting in anxiety and neurotic defence mechanisms. Precipitation could be by physiological crisis in the perinatal period, leading to brain damage or personality deterioration, and the pattern of psychosis would be determined by psychological and environmental factors (Bender, 1966, 1991). Regarding outcome, she concluded that most schizophrenic children continued to be diagnosed as schizophrenic in adolescence and adulthood (Bender, 1953).

The psychoanalytic perspective was well established, based on the view that schizophrenia was determined psychodynamically, like a neurosis. In this context, Mahler, a child analyst of the ego psychology school, played an important part in developing the concept of symbiotic psychosis in children who were deeply dependent on an overanxious mother, becoming disorganized and regressed at the prospect of psychological separation (Mahler, 1952). This condition was distinguished from autistic psychosis, which arose if the infant failed to grow beyond the earlier, normal autistic phase. Similarly, Szurek (1956) and his colleagues were strong protagonists of the psychogenic basis of psychotic disorder, viewing it as the consequence of emotional conflict. With regard to adolescence, the general psychoanalytic view of adolescent ''storm and stress'' leading to a variety of clinical pictures (Freud, A., 1958), even to ''normal psychosis,'' created a heritage of diagnostic and therapeutic uncertainty for clinicians.

In the early 1960s, deliberations by a British Working Party, led by Creak (1961, 1964), proposed ''nine points'' as diagnostic criteria for the ''schizophrenic syndrome in childhood,'' without specifying age of onset. These comprised: (1) ''Gross and sustained impairment of emotional relationships with people;'' (2) ''Apparent unawareness of his own personal identity to a degree inappropriate to his age;'' (3) ''Pathological preoccupation with particular objects or certain characteristics of them, without regard to their accepted functions;'' (4) ''Sustained resistance to change in the environment and a striving to maintain or restore sameness;'' (5) ''Abnormal perceptual experience (in the absence of discernible organic abnormality) implied by excessive, diminished or unpredictable response to sensory stimuli;'' (6) ''Acute, excessive, and seemingly illogical anxiety;'' (7) ''Speech may have been lost, or never acquired, or may have failed to develop beyond a level appropriate to an earlier stage;'' (8) ''Distortion in motility patterns;'' (9) ''A background of serious retardation in which islets of normal, near normal or exceptional intellectual function or skill may appear.'' According to Goldfarb (1970, pp. 780–1), a review of 52 published reports of schizophrenia indicated that all behavioral symptoms were comprised in the nine points. The emergent model, therefore, based on the nine points, was that differing pathological processes might all result in a similar clinical picture.

Resolution of terminological and diagnostic chaos

By the early 1970s, dilution of the concept of childhood schizophrenia had resulted in a chaotic diagnostic situation and the term was misused widely. According to Rutter (1972), childhood schizophrenia had been used ''as a

generic term to include an astonishingly heterogeneous mixture of disorders with little in common other than their severity, chronicity and occurrence in childhood . . . A host of different syndromes have been included . . . infantile autism, the atypical child, symbiotic psychosis, dementia praecosissima, dementia infantilis, schizophrenic syndrome of childhood, pseudo-psychopathic schizophrenia, and latent schizophrenia to name but a few.'' To this list, organic psychosis and borderline psychosis (Ekstein & Wallerstein, 1957) might have been added. It was in this context that studies by Anthony (1958), Rutter, Greenfield and Lockyer (1967) and Kolvin et al. (1971) made a major contribution to a changed approach towards the diagnosis and classification of psychotic syndromes in children and adolescents. It was demonstrated that symptoms, including delusions, hallucinations and thought disorder, similar to those in adults, occurred in children. This enabled a clear distinction to be drawn between early-onset autism of the Kanner type, adult-form schizophrenia with late-childhood onset, and other psychoses with no clear relation to schizophrenia. The child and adult forms could be regarded as qualitatively similar and continuous, while allowing for developmental variation. A wide range of psychological tests had been used from the 1940s (Mehr, 1952; Goldfarb, 1970, p. 781). Later, in conjunction with the new diagnostic and nosological developments, there were major advances in the assessment techniques in schizophrenia, using interview schedules, rating scales, and measures of thought disorder.

The revised, differentiated view and subclassification of childhood psychosis was incorporated in ICD-9 (World Health Organization, 1978) and DSM-III (American Psychiatric Association, 1980), which advocated the application of the same diagnostic criteria as for adult-type disorders, with some allowance for different manifestations. Nevertheless, some ambiguity remained in ICD-9. For example, there continued to be a category for ''psychoses with origin specific to childhood,'' including early infantile autism, disintegrative psychosis and other atypical and unspecified conditions, such as ''schizophrenic syndrome of childhood NOS.'' The same general principles were incorporated in ICD-10 (World Health Organization, 1992) and DSM-IV (American Psychiatric Association, 1994), using symptoms derived, essentially, from the original work of Kraepelin, Bleuler and Schneider.

Psychotic juveniles in asylums

Throughout the nineteenth century, there is conclusive evidence that children and young people were admitted alongside adult patients to private and public

asylums (Parry-Jones, 1993). This practice continued into the twentieth century, until separate facilities were provided for juveniles, predominantly after the Second World War. The clearest profile of presenting clinical problems is derivable from asylum records, but medical labeling and precise diagnostic statements were rare, permitting only speculative retrospective diagnosis.

Very few studies of juveniles in asylums have been undertaken. In one such investigation of a series of patients aged up to 16, admitted to asylums in Oxfordshire, England, from 1846 to 1866 (Parry-Jones, 1990), only two children were hallucinated and, of four with delusional ideas, two were paranoid. Some excited states suggested mania, and one girl aged 16 was restless, talked incessantly and unconnectedly and uttered profanities. Acutely disturbed, noisy and destructive behavior was characteristic, as well as in other contemporaneous case reports. In a major study of 1069 juvenile admissions to Bethlem Royal Hospital, England, from 1815 to 1899, Wilkins (1987, 1993) investigated patterns of hallucinations and delusions and their possible relevance to the incidence of schizophrenia.

Premorbid characteristics and borderline disorder

From the 1920s, there was growing interest in the premorbid characteristics of childhood schizophrenia and the childhood antecedents of adult-onset schizophrenia. A few authors described children displaying symptoms resembling those characteristic of the early stages of schizophrenia. Childers (1931) applied the term ''schizoid'' to problem children on the basis of ''(i) the nature and extent of the child's social incapacity; (ii) his habitual reaction to the situations and requirements of reality by withdrawal rather than by attack or conforming; (iii) the nature, extent, and purpose of his phantasies; (iv) the occurrence in a given child of such definite mental symptoms as are usually observed in adult schizophrenics.'' Speculation has continued about the nature and inter-relationship of a group of poorly defined conditions, including schizoid personality type and schizotypical borderline configuration. Wolff and Chick (1980) used the term schizoid personality of childhood to refer to children with distinctive personality characteristics, but differentiation from Asperger's Syndrome has remained controversial (Asperger, 1944).

Epidemiology

In the nineteenth century, madness in children, in a form comparable to psychosis, was regarded as rare, although its occurrence increased steadily after

puberty. During this period, overall admissions to asylums increased rapidly and Hare (1983) has put forward the interesting theory that this may have been due to cases of dementia praecox, possibly produced by a ''slow epidemic'' of viral origin. Subsequently, until recent years (e.g., Remschmidt et al., 1994), limited epidemiological data continued to be available, complicated by the lack of a uniform system of diagnostic classification and variation in population sampling. No comprehensive reviews on the historical aspects of the epidemiology of childhood psychosis and schizophrenia have been published, although Lay (1938) summarized briefly a number of studies. In the early twentieth century, schizophrenia in children was regarded as extremely uncommon, but by the 1930s this view began to be revised (Potter, 1933; Despert, 1938; Bradley, 1942). Three representative studies are referred to briefly. At the Boston Psychopathic Hospital, during the period 1923–5, there were 160 children under 16 years among the 6000 admissions and only 65 cases were diagnosed as psychotic (Kasanin & Kaufman, 1929). A survey of 1000 randomly selected problem children in a child guidance home by Lurie et al. (1936) showed that 1.3% were diagnosed as dementia praecox. A study by Tizard (1966), using the ''nine points'' of the British Working Party, indicated a prevalence of psychosis of 4 per 10 000. Higher ratios of boys to girls have been reported in the literature on childhood psychosis, but, in general, sex ratios have varied with the populations studied.

Theories of causation

Multiple factors and morbid processes have been implicated in the causation of dementia praecox, schizophrenia and childhood psychosis, over the last century, and of analogous abnormal states in previous centuries. In general, disagreement has centered around the question whether the disorder was determined by an inherent biological defect or by psychological factors. Although consistently viewed as conditions characterized by psychological disturbance, causation has been attributed frequently to primary vulnerability generated by anatomical, biochemical and endocrine factors, toxins and infections, autointoxication, brain damage or disease, and to generic aberrations. With regard to the latter, Canavan and Clark (1923) followed up the children of dementia praecox patients and found five out of 381 with dementia praecox. Several studies, from the 1950s onwards, suggested that schizophrenia in children was associated with a high level of familial aggregation, e.g., a study by Kallman & Roth, (1956) of 52 sets of twins and 50 singletons. These confirmed the views of many other researchers, including some psychoanalysts, that there

was a hereditary or constitutional basis. Bender (1958), for example, concluded that the primary cause, namely, a form of encephalopathy-related maturational lag at the embryonic level, was genetically determined, and emphasized the significance of ''soft neurological signs'' (Bender, 1947). From the 1960s, increasing research attention was being given to the role of cognitive development, perception, speech, linguistic processes, neurobiological correlates, including biochemical and EEG studies, and the effects of pregnancy and birth complications.

Numerous hypotheses have implicated the psychosocial environment and interpersonal experiences. In particular, attention was paid to the parent–child relationship and the home atmosphere. In the 1930s, Potter (1933) listed various psychological factors, especially a dominant, overprotective mother, an unassertive father, and dependence on mother. Similar conclusions were reached by many other authors (e.g., Kasanin et al., 1934; Kanner, 1943; Rank, 1949) and the ''pernicious'' role of maternal overprotection–rejection became popular as an alleged precipitant of both early infantile autism and schizophrenia in children. The concept of the rejecting ''schizophrenogenic mother'' was introduced (Fromm-Reichmann, 1948) and the ''parental perplexity'' hypothesis was developed by Meyers and Goldfarb (1961). Parental attitudes, family characteristics and the family environment attracted increasing interest, as precipitating factors (e.g., Lidz & Lidz, 1949). These theories were not without their detractors, especially because of the variance in clinical observations and Rutter (1965) challenged the attribution of childhood psychosis to abnormal parental attitudes. Such views survived, however, for example, in the parental communication theories of Singer and Wynne (1963).

From an early stage, psychodynamic interpretations of the causation of schizophrenia were introduced, theories being dependent on various schools of thought (Stone, 1991). Brill (1926), one of the foremost early analytic theorists, concluded confidently that ''the nucleus of all these psychoses just as of the neuroses is a psycho-sexual maladjustment in childhood.'' The important contributions of Mahler (1952) and Szurek (1956) have been referred to previously. Szurek's theoretical position, in which psychotic symptoms were viewed as the consequence of self-destructive postnatal conflict, and its resolution, provided a particularly constructive basis for psychotherapy.

Treatment and outcome

Despite the extensive literature on schizophrenia in children and adolescents, there was relatively little discussion of treatment and outcome until the 1950s.

Treatment approaches have been remarkably diverse, often associated with evanescent, idiosyncratic etiological hypotheses and, generally, lacking evaluative research and controlled studies.

Pharmacological treatments of all descriptions have been utilized to alleviate symptoms and improve the psychotic child's accessibility, each generation producing its innovations, e.g., ephedrine, caffeine, tri-iodothyronine, sodium amytal and LSD-25, culminating in modern neuroleptics. Similarly, physical treatments have followed fashionable theories, so that, for example, shock therapy using insulin, metrazol or electric shock (Cottington, 1941; Bender, 1947) and prefrontal leukotomy (Freeman & Watts, 1950) have been utilized for juveniles with schizophrenia. Despite the disproportionately high mortality rates associated with leukotomy in children, Angus (1949) considered the risk "legitimate in view of the long hospitalization and unfavorable outlook of cases selected as these are on the basis of a hopeless prognosis from other methods of treatment."

From the early twentieth century, various forms of individual psychotherapy were practiced, with children with schizophrenia. Escalona (1948) categorized these methods as both "expressive" and "suppressive." Despite enduring criticism that psychotherapeutic approaches were not possible because of "the lack of an essential emotional rapport" (Potter, 1933), some authors claimed it was the most successful treatment method (Lourie et al., 1943). Specific psychoanalytic treatment was advocated by many authors, e.g., Klein (1949). During the 1960s, the role of behavior modification to improve language and social behavior was explored actively (Leff, 1968). An important aspect of management was work with parents, concerned principally to develop a positive and realistic attitude towards the child's illness (e.g., Kaufman et al., 1957).

In general, it has been recognized consistently that children with schizophrenia required a broad, multidimensional approach, including institutional treatment, to promote socialization and rehabilitation, thus creating the setting for milieu therapy. Bender's views, in the mid-1950s (Bender, 1958), based on her "maturational lag" theory, illustrate the range of treatment goals and therapeutic programs thought to be required. These were "(i) to stimulate maturation and patterning in all of the lagging and embryonically plastic biological and psychological processes; (ii) to relieve anxiety; (iii) to protect, correct or help the formation of adequate defense mechanisms; (iv) to place a high value on the time factor in children by promoting maturation at the earliest and most favorable period in order to avoid or shorten isolation experiences ...; (v) to help the child learn to tolerate and live with his

schizophrenic illness, and similarly to help the parents, the schools and the community.'' To achieve these goals, the lifelong treatment program required ''milieu or environmental therapy,'' ''specific psychotherapy,'' ''specific remedial procedures . . . such as remedial tutoring . . .,'' ''physiological therapies, especially electric convulsive treatment,'' ''pharmacological therapies with antihistamines, amphetamines, mephenesins, anticonvulsants, growth vitamins, etc.,'' and ''organizations and discussion groups for parents.'' Despite recognition of their often long-term institutional needs, special inpatient facilities for juveniles with psychoses did not develop, to any significant extent, until the 1940s and 1950s, and children and adolescents shared the same regime as adults.

A very variable historical picture emerges of the prospects of recovery and actual outcome, complicated, inevitably, by widely different diagnostic criteria, variation in the selection procedures in the different treatment settings, the length of follow-up, and the number and age of the subjects (Eggers, 1978). In general, while stopping short of therapeutic nihilism, most reports have indicated a uniformly poor prognosis (e.g., Bradley, 1942). At Bellevue Hospital, New York, however, Cottington (1941) felt that there was scope for progress using socialization, psychotherapy and shock therapy. The most satisfactory responses were reported following psychodynamic treatments (e.g., Klein, 1949; Kaufman et al., 1963). However, in a follow-up of 100 children diagnosed as suffering from schizophrenia, from 1935 to 1952, Bender (1970) showed that the disorder ''is an early onset of a life course of schizophrenia of every possible type,'' although the criteria used in adulthood have been questioned. Carter (1942) provided a brief, useful review of the prognostic factors in adolescent psychoses.

Conclusions

The historical study of schizophrenia highlights the remarkable degree of fluidity that has characterized its definition and diagnostic criteria and the special problems in relation to the existence and features of the disorder in children. The findings incorporated in this chapter broadly endorse the key historical trends identified by Goldfarb (1970, pp. 776–7), which may be summarized as follows: (i) ''Profound alterations in biological development, either in the form of regressions or of arrests, are noted by all observers;'' (ii) ''All workers refer to the very global and total integrative failure demonstrated by schizophrenic children. The total personality is disordered;'' (iii) ''Observers frequently refer to the highly variable and changing nature of the symptomatic

expressions of schizophrenic children;'' (iv) ''All observers note a serious disturbance of emotional organization;'' (v) ''A major advance in rationalizing the disorders subsumed by the diagnosis of childhood schizophrenia, or any of the other labels for childhood psychosis, is represented in the concept of ego aberration;'' (vi) ''If there has been a ''break through'' in the study of etiology, it consists of the implementation of the concept of a multiplicity of factors, centred in the child and in the environment, to explain the adaptive accommodation of the child, which is then classified as psychosis.''

Since the 1970s, nosological anomalies and confusion in relation to the disorders subsumed in the category of childhood psychosis have been largely resolved, with the application of the same core criteria for schizophrenia across all developmental periods and the recognition of very similar clinical features and comparable responses to pharmacological treatment. A universally accepted definition of schizophrenia, however, has remained elusive and evidence of causation is fragmentary. In historical terms, the present stage simply sets the scene for further fundamental clinical and research questions concerning the origins of schizophrenia, the significance of early onset, developmental variation in the expression of the disorder, the status of atypical symptomatic presentations that lie outside the current narrow ICD-10 and DSM-IV criteria, and the significance of comorbidity. Schizophrenia at all ages continues to be a perplexing and challenging disorder, in both clinical and research terms. Consequently, historical research has more relevance than its purely antiquarian interest, since it both elucidates the condition and establishes future research directions by highlighting enduring ambiguities. Finally, the historical dimension provides a corrective warning to each generation about the potential fallibility of received wisdom.

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