Adolescent schizophrenia

Chris Hollis

Schizophrenia is a devastating chronic disorder that typically presents in early adult life and impacts on a broad swathe of social and psychological functioning. It is not surprising that psychiatrists have tended to be circumspect about making this ominous diagnosis in children and adolescents. Genuine concerns about the validity of applying 'adult' psychotic diagnoses in this young age group, together with the lack of diagnosis-specific interventions, have suggested a cautious approach to diagnosis. Furthermore, the relative rarity of schizophrenia in this age group has meant that most psychiatrists have relatively little experience with 'atypical' early presentation of the disorder.

Clinicians have not been helped by the fact that the vast bulk of schizophrenia research has excluded children and younger adolescents with psychotic disorders. However, over the past decade research activity in child and adolescent schizophrenia has been sparked by several factors. First, awareness of the greater clinical severity of schizophrenia in childhood and adolescence and the possibility of greater aetiological liability have encouraged researchers to investigate genetic and neurobiolgical correlates in very early-onset cases (McKenna *et al*, 1994; Jacobsen & Rapoport, 1988). Second, the emergence of a 'neurodevelopmental' formulation of schizophrenia (Weinberger, 1987) and the perspective of developmental psychopathology (Hollis & Taylor, 1997) have focused more attention on early developmental processes and the premorbid childhood course of schizophrenia from "birth to onset" (Jones & Done, 1997). Finally, the arrival of the atypical antipsychotics has stimulated new interest in pharmacotherapy and the need for clinical trials to guide prescribing (Clark & Lewis, 1988).

Research in adolescent schizophrenia has needed to address the key question of continuities and discontinuities with adult schizophrenia. What is the clinical and aetiological significance of the atypically early onset of schizophrenia in childhood and adolescence? Does child and adolescent schizophrenia have the same causes and outcomes and does it respond to the same treatments as adult schizophrenia?

Terminology in this field is often confusing, with the terms 'childhood-onset', 'adolescent-onset' and 'early-onset' schizophrenia being used interchangeably without precise definitions. In this article, the shorthand term 'adolescent schizophrenia' will be used to refer to child and adolescent cases with an onset up to the age of 17. Although this age limit is arbitrary, it roughly corresponds with the upper age cut-off in most published studies of child and adolescent psychosis, and is also the lower age cut-off for most adult psychosis studies and clinical services. Here, the term 'adolescent schizophrenia' includes children below 10 years of age who are usually described as pre-adolescent. However, it is worth noting that the onset of schizophrenia before the age of 10 is exceedingly rare, and in fact the majority of cases referred to in the literature under the rubric of 'childhood-onset schizophrenia' have an onset of psychosis in early adolescence.

The goal of this article is to summarise what is currently known about adolescent schizophrenia and indicate the extent and limitations of the evidence base for clinical diagnosis, management and treatment. The article is essentially practical in orientation and is not meant to be a comprehensive review of recent research. Relevant review papers are highlighted in the list of references.

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Concepts of adolescent schizophrenia

Historical perspectives

Both Kraepelin and Bleuler believed that schizophrenia presents in a similar form, albeit more rarely, during childhood and adolescence. Kreapelin (1919) found that 3.5% of cases of dementia preacox began before the age of 10, with a further 2.7% arising between the ages of 10 and 15. Bleuler (1911) suggested that about 5% of cases of schizophrenia had their onset prior to age 15.

However, from the 1930s until the early 1970s the concept of childhood schizophrenia broadened to encompass autism and other developmental disorders that were seen as childhood manifestations of adult schizophrenia. This 'lumping together' of different disorders under the common rubric of childhood schizophrenia makes most research carried out during this period very difficult to interpret.

During the 1970s, the pendulum swung back to the view that schizophrenia in childhood and adolescence should be defined by unmodified adult diagnostic criteria. From ICD–9 (World Health Organization, 1978) and DSM–III (American Psychiatric Association, 1980) onwards, the same diagnostic criteria have been used for schizophrenia regardless of the age of onset.

Developmental issues in diagnosis

Although use of the same diagnostic criteria aids comparability across the age range, it does not exclude the possibility that schizophrenia may present rather differently in childhood and adolescence. Developmental variation in symptoms occurs in other neuropsychiatric disorders such as Wilson's disease and temporal lobe epilepsy, so it is not unreasonable to consider this possibility in schizophrenia. If schizophrenia did show symptom variation in children and adolescents, then the use of unmodified adult criteria could result in 'true' cases being missed (false negative diagnoses). The main argument used against the proposition of 'developmental variants' is the finding that the diagnosis of schizophrenia can be made reliably in children as young as seven using unmodified adult criteria (McKenna *et al*, 1994). However, this still does not really help to resolve the status of children and adolescents with 'partial syndromes' that share some diagnostic features, or prodromal symptoms, of schizophrenia but do not meet full DSM–IV (American Psychiatric Association, 1994) or ICD–10 (World Health Organization, 1992) criteria. For example, in the USA the term "multidimensionally impaired" (MDI) (Jacobsen & Rapoport, 1998) has been coined to describe children with multiple early impairments in cognitive and social functioning who develop transient psychotic symptoms in late childhood and early adolescence. A higher than expected rate of schizophrenia among first-degree relatives suggests that MDI children may lie on the schizophrenia continuum, but longer follow-up studies are needed to tell whether or not these cases progress to more typical schizophrenic presentations.

In pre-adolescent children, diagnostic issues also relate to the difficulty of distinguishing between psychotic symptoms and normal developmental variation. For example, delusions can be confused with normal childhood fantasies, and formal thought disorder may be impossible to distinguish from illogical thinking and loose associations seen in children with immature language development. Children may also find it hard to describe accurately the location of hallucinations. Hence, the limitations of normal cognitive development make it very difficult to identify psychotic symptoms reliably in children below the age of seven.

In summary, efforts to describe age-specific variation in schizophrenic symptoms are constrained by the lack of biological markers providing external validation of the disorder. At present, schizophrenia is defined only at a symptomatic level. Hence, careful longitudinal and family genetic studies will be needed to clarify the nosological status of those psychotic presentations in childhood and adolescence that lie outside adult-based diagnostic criteria for schizophrenia.

Assessment and diagnosis

Diagnostic stability

Although there is compelling evidence that schizophrenia can be diagnosed in children and adolescents from the age of seven using unmodified adult criteria, the predictive validity of these early diagnoses is largely unknown. One view is that adolescent schizophrenic and affective psychoses are poorly differentiated and show a high degree of diagnostic instability from adolescence into adult life (Zeitlin, 1986; Werry *et al*, 1991). However, this view has been challenged by a recent follow-up of 110 cases of adolescent-onset psychoses presenting as a consecutive series to the Maudsley Hospital from 1973 to 1991 (further details available from the author upon request). We will refer to this subsequently as the Maudsley study. The study used a 'catch-up'

longitudinal design in which DSM–III–R (American Psychiatric Association, 1987) diagnoses were retrospectively applied to adolescent first-episode case notes. Eighty-five per cent (93/110) of the original cohort were then reassessed on average 11 years after their first admission. The positive predictive value (PPV) was high for adolescent diagnoses of both schizophrenia (80%) and affective psychoses (83%). In contrast, the PPV for adolescent schizoaffective psychoses was only 33%. These findings suggest that a diagnosis of schizophrenia using standard diagnostic criteria is likely to be just as stable in adolescence as it is in adult life. However, the stability and diagnostic status of schizoaffective psychoses in childhood and adolescence is much less certain.

Clinical features of adolescent schizophrenia

The characteristic clinical features of adolescent schizophrenia are summarised in Box 1.

Premorbid functioning

Adolescent schizophrenia has been shown to be associated with poor premorbid functioning and early developmental delays (Alaghband-Rad *et al*, 1995; Hollis, 1995). Retrospective accounts of premorbid functioning are often plagued by potential recall bias, however, the consensus is that premorbid developmental impairments are more common in adolescent than in adult schizophrenia. In the Maudsley study, significant early delays were particularly common in the areas of language (20%), reading (30%) and bladder control (36%). Although just over 20% of cases of adolescent schizophrenia have significant early delays in either language or motor development, a similar pattern of developmental delays is reported in less than 10% of cases with adult schizophrenia (Jones *et al*, 1994).

In the Maudsley study, about one-third of cases of adolescent schizophrenia had significant difficulties in social development affecting the ability to make and keep friends. Similar, but less frequent, difficulties with premorbid sociability have been noted in representative population samples of adult schizophrenia (Malmberg *et al*, 1998). However, premorbid social and behavioural difficulties are not specific to schizophrenia. Premorbid deficits also occur in adolescent affective psychoses, at a lower rate than in schizophrenia, but more frequently than in non-psychotic psychiatric controls (Sigurdsson *et al*, 1999).

One interpretation of these findings is that one-third of cases of adolescent schizophrenia have abnormal premorbid development, with the rest developing normally. In fact, more careful analysis shows that there is no abnormal developmental subgroup – this is simply an artefact of using rather crude categorical measures of premorbid development. Continuous intelligence quotient (IQ) measures show that the whole distribution of IQ is shifted down relative to both adolescent affective psychoses and adult schizophrenia.

Finally, the Maudsley study found that premorbid developmental impairments show longitudinal continuity with negative symptoms and poor adult outcome. This suggests that premorbid social and developmental impairments may have the same underlying neurobiological substrate as negative symptoms.

Onset and symptoms

Adolescent schizophrenia frequently presents with an insidious as opposed to an acute onset. For this reason, early recognition of the disorder can be very difficult, as premorbid cognitive and social impairments gradually shade into prodromal symptoms before the onset of positive psychotic symptoms. In the Maudsley study, 65% of cases (33/51) with DSM–III–R adolescent schizophrenia had an insidious onset (over six months) compared with only 19% of cases (8/42) with adolescent affective psychoses. Non-specific behavioural changes – including social withdrawal, declining school performance, uncharacteristic and odd behaviour – began, on average, over a year before the onset of positive psychotic symptoms. In retrospect, it was often apparent that non-specific behavioural changes were frequently early negative symptoms, which in turn had their onset well before positive symptoms such as hallucinations and delusions.

Adolescent schizophrenia is characterised both by more prominent negative symptoms (e.g. flattened or inappropriate affect and bizarre, manneristic behaviour) and by relatively fewer well-formed systematised delusions and auditory hallucinations when compared with adult schizophrenia. Taking the DSM–III–R subtypes of schizophrenia, Beratis

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Box 1. Clinical features of adolescent schizophrenia

Poor premorbid functioning
Below-average IQ (mean IQ 85, 30% IQ<70)
Insidious onset
Strong family history of psychosis/ schizophrenia
Predominantly negative symptoms
Severe and unremitting course

et al (1994) found that the disorganised and undifferentiated subtypes were predominantly of adolescent-onset, whereas the paranoid subtype was most frequently first diagnosed in adult life. Although all subtypes can occur in adolescence, there is a relative predominance of the disorganised subtype, which in earlier systems of classification would have been described as hebephrenia.

Short-term course

Adolescent schizophrenia tends to run a chronic course with only a small minority of cases making a full symptomatic recovery from the first psychotic episode. In the Maudsley study, only 12% of cases of schizophrenia were in full remission at discharge compared with 50% of cases with affective psychoses. Taking all cases of adolescent psychoses together, those who were still showing psychotic symptoms after six months had only a 15% chance of subsequent full remission, while over half of all cases that made a full recovery had active psychotic symptoms for less than three months. Hence, if a full recovery did occur it was most likely to happen within the first three months of illness, but after six months of psychosis the prognosis for a full recovery was poor and changed very little thereafter. The clinical implication is that the early course over the first six months is the best predictor of remission and that longer observation beyond six months adds relatively little new information.

Differential diagnosis

The differential diagnosis of adolescent schizophrenia is summarised in Box 2.

Affective and 'atypical' psychoses

There is little, if any, evidence that atypical psychoses are relatively more common in adolescence than in adult life. The breakdown of DSM–III–R first-episode diagnoses in the Maudsley study was 55% (51/93) schizophrenia, 13% (12/93) schizoaffective psychoses, 25% (25/93) affective psychoses and 7% (7/93) atypical psychoses. This distribution of diagnoses is very similar to reports from adult first-episode psychosis cohorts (van Os *et al*, 1996).

Diagnostic confusion can occur with affective psychoses because of the increased prevalence of positive psychotic symptoms in adolescent affective psychoses. Diagnostic errors are most likely if clinicians apply a narrow Schneiderian concept of schizophrenia, with its emphasis on positive psychotic first-rank symptoms. In contrast, the presence of negative symptoms and an insidious onset are the best predictors of diagnostic continuity

of adolescent schizophrenia. Meanwhile, early (<6 months) and complete remission are the best predictors of maintaining a diagnosis of affective psychosis. The Maudsley study suggests that clinicians (at the Maudsley at least) were very cautious about applying the diagnosis of schizophrenia to adolescent patients. One-quarter of the cases given a case note DSM–III–R diagnosis of schizophrenia were originally assigned by Maudsley child psychiatrists to atypical or other unspecified psychoses, with the vast majority of these receiving a diagnosis of schizophrenia at adult follow-up.

Autistic spectrum and developmental language disorders

Kolvin (1971) in a landmark study clearly distinguished the symptoms and correlates of core autism with onset before the age of three, from adult-type schizophrenia beginning in late childhood and early adolescence. However, some children with atypical autism and Asperger's syndrome have social and cognitive impairments that overlap closely with the premorbid phenotype described in schizophrenia. Furthermore, these children on the so-called autistic spectrum can also develop psychotic symptoms in adolescence (Volkmar & Cohen, 1991). Similarly, an increased risk for psychosis has also been noted in developmental language disorders (Rutter & Mawhood, 1991). Although some children on the autistic spectrum show a clear progression into classic schizophrenia, others show a more episodic pattern of psychotic symptoms without the progressive decline in social functioning and negative symptoms characteristic of adolescent schizophrenia.

Organic and neurodegenerative psychoses

Schizophrenic symptoms, in particular positive symptoms, can be produced in adolescence by acute

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Box 2.   Differential diagnosis of adolescent schizophrenia

Developmental disorders: atypical autism, Asperger's syndrome, language disorders
Personality disorders: schizotypal personality disorder, borderline personality disorder
Functional psychoses: affective, schizoaffective, atypical psychoses
Organic psychoses: drug psychoses, frontal and temporal lobe epilepsy, neurodegenerative disorders (Wilson's disease), metachromatic leukodystophy

drug-induced psychoses, complex partial seizures and some very rare neurodegenerative disorders. DSM–III–R and DSM–IV definitions of schizophrenia specify six-month duration criteria (including prodromal and active symptoms), which helps to distinguish between schizophrenia and acute organic psychoses. Psychotic symptoms can occur in epilepsy, in particular temporal and frontal lobe partial seizures. Ambulatory electroencephalogram monitoring and telemetry may be required if the diagnosis remains in doubt. Rare neurodegenerative disorders that can mimic schizophrenia include Wilson's disease (hepato-lenticular degeneration) and metachromatic leukodystrophy. These disorders usually involve significant extrapyramidal or other motor abnormalities and a progressive loss of skills (dementia) that can aid the distinction from schizophrenia. Suspicion of a neurodegenerative disorder is one of the clearest indications for brain magnetic resonance imaging in adolescent psychoses. Adolescents with schizophrenia show relative grey matter reduction with white matter sparing. In contrast, characteristic frontal and occipital white matter destruction and demyelination is seen in metachromatic leukodystrophy. In Wilson's disease, hypodense areas occur in the basal ganglia, together with cortical atrophy and ventricular dilatation.

Associated features

Family psychiatric history

The increased clinical severity of adolescent schizophrenia is associated with a greater familial risk than the adult-onset form of the disorder. In the Maudsley study, a positive family history of schizophrenia among first-degree relatives was found in 20% of adolescent probands with schizophrenia. This is about double the rate reported in comparable studies of adult-onset schizophrenia. Interestingly, it appears that it is the presence of negative symptoms in the proband that predicts a family history of schizophrenia. This suggests that negative symptoms may represent the genetically transmitted phenotype in schizophrenia.

Gender ratio

Although a male predominance (2:1) is a consistent finding in incident samples of early adult-onset schizophrenia (Castle & Murray, 1991), the picture is far less clear in adolescent schizophrenia, with some studies reporting a similar male predominance and others finding no gender difference (Jacobsen & Rapoport, 1998). It is possible that differences between studies may be a result of referral biases, and at present good population-based epidemiological

studies of adolescent schizophrenia and psychoses are notably lacking.

Perinatal complications

Perinatal and obstetric complications have been implicated in the aetiology of adult schizophrenia, although it is unclear whether they are a consequence or a cause of neurodevelopmental problems. In the Maudsley study, perinatal complications were equally prevalent in schizophrenia and affective psychoses. Also, there was no difference in birth weight by diagnosis. Alaghband-Rad *et al* (1995) found no difference in the rate of obstetric complications between adolescent probands with schizophrenia and their well siblings.

Assessment measures

The recent growth of research in child and adolescent schizophrenia has spawned a number of assessment instruments for evaluating schizophrenic symptoms. Many of these are so-called 'kiddie' versions of adult instruments such as the K–SADS (Schedule for Affective Disorders and Schizophrenia for School-age Children; Ovraschel *et al*, 1980). For adolescents, most adult psychosis interviews and rating scales will suffice, as the reworking of these measures has largely been aimed at making them more acceptable to pre-adolescent children.

One area that remains poorly covered by assessment instruments is premorbid development. A shortened version of the Autism Diagnostic Interview (ADI; Lord, 1991) can be useful in providing a detailed picture of the course of premorbid social and communicative behaviour. It is likely that measures of premorbid functioning will grow in sophistication as the process of diagnosing schizophrenia broadens from a narrow cross-sectional assessment of psychopathology (epitomised by instruments such as the Present State Examination) to include a longitudinal life course perspective.

Course and outcome

Adolescent schizophrenia is associated with a severe and unrelenting clinical course. The Maudsley study found the outcome of adolescent schizophrenia to be worse than the outcome of adolescent affective psychoses and adult-onset schizophrenia, while adolescent affective psychoses fared worse than adult affective psychoses. These results suggest that outcome is affected independently by both age of onset and diagnosis. Interestingly,

neither gender nor the duration of illness was associated with outcome. If this latter finding is replicated, it suggests that symptoms and functioning may plateau after the first 2–3 years of illness without further progressive decline.

A striking finding was the extremely poor social outcome associated with adolescent schizophrenia. In adult life, about 50% had never established friendships or love relationships, and only 2% (1/51) had ever had a girl/boyfriend compared with 21% (9/42) with affective psychoses. Approximately one-third of those with adolescent schizophrenia were resident in a long-stay hospital or hostel accommodation. Only one subject (2%) was living independently of his or her parents. Fifty per cent had a continuous illness course without any episodes of remission.

Prediction of outcome

In the Maudsley study, poor adult outcome from first-episode adolescent psychosis was predicted independently by premorbid impairments and negative symptoms at onset. Positive symptoms and DSM–III–R diagnoses added nothing to prediction once these two factors had been taken into account. This finding highlights the importance of assessing negative symptoms at the onset of illness. It also suggests that premorbid impairment and negative symptoms lie at the core of a valid clinical concept of adolescent schizophrenia (see Box 3).

Neuropsychology

Adolescent schizophrenia is associated with a significant cognitive impairment. Several studies have found a mean IQ of between 80 and 85 (one standard deviation below the population mean), with about one-third of cases having an IQ below 70 (Jacobsen & Rapoport, 1998; Hollis, 1999). This represents a mean IQ score about 10 points lower than those reported in studies of adult schizophrenia.

These findings raise several important questions. First, are the cognitive deficits specific or generalised? That is, are some aspects of cognitive functioning affected more than others? Second, which deficits precede the onset of psychosis and could be causal, and which are consequences of psychosis? Third, is the pattern of deficits specific to schizophrenia or shared with other developmental disorders? Fourth, are cognitive impairments progressive or static after the onset of psychosis?

Current research (Asarnow *et al*, 1994*a*) suggests that adolescents with schizophrenia have particular

difficulties with cognitive tasks that make demands on short-term, working memory and selective and sustained attention and speed of processing. In contrast, well-established, 'over-learned' skills are maintained. There seems to be a relatively good correlation between deficits on frontal and executive neuropsychological tasks and negative symptoms. However, these deficits do not appear to be specific to schizophrenia, with the same pattern of deficits being reported in other neurodevelopmental disorders, in particular attention-deficit hyperactivity disorder (ADHD). So far, the issues of causal significance and the long-term course of cognitive deficits remain intriguing but unresolved. However, a good understanding of specific cognitive deficits in individual cases of adolescent schizophrenia can be particularly helpful in guiding education and rehabilitation. For example, efforts should be made to break tasks down into small, manageable parts and reduce demands on working memory and speed of processing.

Neurobiology

A range of studies in adolescent schizophrenia involving psychophysiological measures and structural and functional neuroimaging have confirmed a broadly similar pattern to findings reported in adult schizophrenia (Jacobsen & Rapoport, 1998). This supports the view that adolescent and adult schizophrenia share the same neurobiological substrate. Structural brain imaging (magnetic resonance imaging) in adolescent schizophrenia has shown a consistent pattern of decreased cerebral volume, relative white matter sparing and ventricular dilatation. Total cerebral volume is strongly correlated with negative symptoms (smaller brains are associated with more negative symptoms). Intriguingly, treatment with traditional antipsychotics appears to cause progressive enlargement of the basal ganglia, with these structures returning to their original size when

Box 3. Predictors of poor outcome in adolescent psychoses	
Poor premorbid functioning	Yes
Negative symptoms	Yes
Disorganisation	Yes
Duration of untreated psychosis	Yes
Male gender	No
Positive symptoms	No

patients are transferred to the atypical antipsychotic clozapine (Frazier *et al*, 1996). Longitudinal neuro-imaging is a particularly important area for future research as it may provide direct evidence of progressive brain changes after the onset of psychosis.

Treatment issues

General principles

The general principles of assessment and treatment are listed in Box 4. Early recognition and accurate diagnosis are essential for effective treatment of adolescent schizophrenia. Delays in diagnosis and treatment may result in a poorer long-term outcome. In the Maudsley study, there was a strong association between the duration of untreated psychosis (DUP) and a chronic illness course. This echoes the relationship between DUP and outcome in adult schizophrenia (Birchwood *et al*, 1997), which suggests that early interventions that reduce the DUP in adolescent schizophrenia may lead to better outcomes. Despite the obvious attraction of primary prevention in schizophrenia, our current understanding of the premorbid phenotype lacks specificity and has a very low predictive value. Similarly, the predictive value of prodromal states is too low to consider intervention unless it is likely to benefit to the whole population at-risk, most of whom will not develop schizophrenia.

Although antipsychotic drugs remain the cornerstone of treatment in adolescent schizophrenia, all young patients with schizophrenia require a multi-modal treatment package that includes pharmacotherapy, family and individual counselling and education about the illness, and assessment of social and educational needs (Clark & Lewis, 1998). It is important to note that although this multi-modal approach represents current best practice, few, if any, non-pharmacological interventions have been systematically evaluated in adolescent schizophrenia. Furthermore, it is probable that some elements of family intervention effective in adults may not work in children. Parents of children and adolescents with schizophrenia express lower levels of criticism and hostility than parents of adult-onset patients (Asarnow *et al*, 1994*b*), hence family interventions aiming to reduce high expressed emotion are likely to be misguided.

Antipsychotics

There are only a handful of clinical trials of traditional antipsychotics that relate specifically to children and adolescents. Hence, most evidence of their efficacy is extrapolated from adult studies. The studies that do exist suggest that traditional antipsychotics, such as haloperidol, significantly reduce positive psychotic symptoms in about 70% of cases (Spencer & Campbell, 1994). However, traditional antispsychotics have very little action against negative symptoms and carry the serious risk of extrapyramidal side-effects, both of which are common in adolescent schizophrenia. Hence, the new atypical antipsychotics that claim improved action against negative symptoms and a lower risk of extrapyramidal side-effects would seem to offer important advantages. So far, there is just one published placebo-controlled double-blind trial showing improved action of clozapine over haloperidol for positive and negative symptoms in 21 patients with adolescent schizophrenia (Kumra *et al*, 1996). The small risk of serious blood dyscrasias on clozapine is dealt with by routine heamatological monitoring. Other side-effects including hypersalivation, drowsiness and seizures are largely dose-dependent and may result in up to one-third of patients dropping out of treatment (Kumra *et al*, 1996). Because of the risk of dose-related side-effects, the dose should be increased more slowly than in adults, titrating benefits against adverse effects. If seizures are troublesome, sodium valproate can be added. At present, clozapine is licensed only for patients resistant to, or intolerant of, traditional drugs and can only be initiated in hospital in-patients. Other atypical antipsychotics, including resperidone,

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Box 4. Principles of assessment and treatment in adolescent schizophrenia

Obtain detailed developmental history and information from multiple informants, including parents and school
Carefully apply standard ICD–10 or DSM–IV diagnostic criteria
Remember that negative symptoms present early and have a strong prognostic value
First-rank symptoms are rare and unhelpful in diagnosis and prognosis
Do not delay making diagnosis if criteria are met
Educate and support parents – avoid all suggestions of blame
Carefully assess cognitive and social deficits and make early plans for special education
Consider atypical antipsychotics as a first-line treatment

Box 5. Summary points

Adolescent schizophrenia is a devastating illness with greater clinical severity and a poorer outcome than the adult-onset form of the disorder.

Unmodified adult-based diagnostic criteria for schizophrenia can be used and have good predictive validity – identifying a more severe course and outcome compared with adolescent affective psychoses.

Developmental variants of schizophrenia in adolescence could still be missed by current diagnostic criteria. The status of these phenotypic variants needs to be resolved by longitudinal and family genetic studies.

Adolescent schizophrenia is more familial than adult schizophrenia and is associated with more impaired premorbid development and lower IQ – suggesting that adolescent cases lie at the extreme end of an aetiological continuum for schizophrenia.

The clinical presentation of adolescent schizophrenia is characterised by insidious onset and predominant negative symptoms. These features, together with premorbid impairment, are the best predictors of a continuing diagnosis of schizophrenia and poor long-term outcome. In contrast, Scheiderian first-rank symptoms are unhelpful in distinguishing between adolescent schizophrenia and affective psychoses.

A similar pattern of neuropsychological and neurobiological abnormalities is found in both adolescent and adult schizophrenia. It remains unclear to what extent these occur before the onset of psychosis and whether they show progression after onset.

Adolescent schizophrenia is probably under-diagnosed and under-treated. There is a strong case for the more frequent and earlier use of atypical antipsychotics. A model of specialist early-psychosis centres should be considered – integrating the best of treatment approaches for adolescents and young adults with psychosis.

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olanzapine and quetiapine, may be used as first-line drugs in out-patients. However, evidence for the efficacy and safety of these drugs in adolescent schizophrenia is limited to case reports and extrapolation from trials in adults.

The uptake of atypical antipsychotics among British child and adolescent psychiatrists is still low. A recent survey in the Trent region found that only 10% of psychiatrists who had prescribed anti-psychotics for adolescent psychoses had used an atypical drug (Slaveska *et al*, 1998). The most common reason for not prescribing atypicals was lack of clinical experience with these new drugs, rather than concerns about side-effects or drug costs. Given the clinical severity of adolescent schizophrenia and the potential superiority of atypical antipsychotics, there is clearly a need for improved training of child psychiatrists in psychopharmacology and for randomised controlled trials of atypical antipsychotics in adolescent-onset patents.

Services in the future

Although many child and adolescent mental health services provide excellent treatment for adolescents with psychosis, the national picture is of patchy provision, with a significant number of young patients denied the most effective treatments. One possible solution is the development of specialist regional early-psychosis centres, using a similar model to the specialist treatment of cancer or paediatric intensive care. These centres could provide a regional focus for research, education and tertiary clinical services.

Outside the UK, services such as the EPPIC (Early Psychosis Prevention and Intervention Centre) Programme in Melbourne, Australia (McGorry, 1993) provide innovative models of intensive first-episode interventions for psychosis spanning adolescence and early adult life. In the UK, adolescent and adult services for psychosis are traditionally quite separate with little cross-fertilisation of ideas and skills. Both can learn from each other, and integrated models of service provision for early-onset psychosis that span traditional age boundaries (e.g. age 14–24) deserve National Health Service investment and evaluation against more traditional models of service delivery.

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*indicates articles of particular interest

Multiple choice questions

1. The diagnosis of schizophrenia:
   a can reliably be made in children below the age of seven
   b requires different diagnostic criteria in children and adolescents than in adults
   c in adolescence shows good predictive validity
   d may exclude developmental variants in childhood and adolescence
   e can be distinguished from affective psychoses by the presence of first-rank symptoms.

2. Premorbid impairments in schizophrenia:
   a are more severe in adolescent- than adult-onset cases
   b are an indication for early preventive use of antipsychotics
   c overlap with features of other developmental disorders
   d predict negative symptoms
   e may reflect underlying deficits in cognitive and social development.

3. The clinical course of adolescent schizophrenia:
   a is characteristically episodic with periods of full remission
   b shows increasing deterioration over time
   c shows that social functioning is well-maintained
   d is worse than that of adolescent affective psychoses
   e is most likely to end in recovery if active psychosis lasts less than three months.

4. In adolescent schizophrenia:
   a brain magnetic resonance imaging scan shows relative sparing of white matter
   b brain changes are similar to those seen in metachromatic leukodystrophy
   c family dysfunction is a common cause
   d atypical antipsychotics may offer significant treatment benefits
   e delaying the diagnosis carries greater risks than early intervention.

5. In the clinical assessment of adolescent schizophrenia:
   a a detailed developmental history is essential
   b a one-off mental state assessment is the most reliable guide to diagnosis
   c reports should always be obtained from school
   d brain magnetic resonance imaging findings are usually diagnostic
   e abnormal patterns of family interaction are causal factors in the illness.

MCQ answers

1		2		3		4		5	
a	F	a	T	a	F	a	T	a	T
b	F	b	F	b	F	b	F	b	F
c	T	c	T	c	F	c	F	c	T
d	T	d	T	d	T	d	T	d	F
e	F	e	T	e	T	e	T	e	F