Childhood-Onset Schizophrenia: A Followup Study

by Joan Rosenbaum Asarnow, Martha C. Tompsoon, and Michael J. Goldstein

Abstract

This article is an overview of our studies of childhood-onset schizophrenia. Data are presented demonstrating that (1) the majority of the sample showed continuing schizophrenia as they progressed through adolescence; (2) there was considerable variability in outcome, defined by global adjustment scores, with 56 percent of the sample showing improvement in functioning during a 2- to 7-year followup period and the other 44 percent showing minimal improvement or a deteriorating course; (3) schizophrenia in childhood could be diagnosed by the same criteria used for adults and was associated with severe dysfunction; and (4) some intrafamilial attributes found to be associated with schizophrenia in adults were also associated with schizophrenia in children, but there were some differences in the family environmental correlates of childhood- and later-onset schizophrenia. These data are consistent with the hypothesis that childhood- and later-onset schizophrenia represent the same illness or illnesses. Additional research is needed, however, to clarify the etiologic and clinical significance of the atypical early onset in childhood cases.


One of the most consistent observations about schizophrenic disorders is that most cases of schizophrenia have their onset during late adolescence or early adulthood (Bleuler 1911/1950; Kraepelin 1919/1921; Weinberger 1987; Riecher et al. 1991; Remschmidt 1993). Despite its relative rarity during childhood, schizophrenia has been described in children throughout at least the past century. Kraepelin (1919/1921), for example, estimated that the illness began before age 10 in at least 3.5 percent of cases, with another 2.7 percent developing between ages 10 and 15. Similarly, Bleuler (1911/1950) and Lutz (1937) estimated that 0.5 to 1 percent of schizophrenic cases had onsets before age 10 and 4 percent began before age 15. The fact that childhood onset of schizophrenia is atypical raises several questions with promise for elucidating the etiologic pathways to schizophrenia. Most notably, what accounts for this atypical early onset?

One hypothesis that has received some support in the literature is that childhood-onset schizophrenia represents a particularly severe and chronic form of the illness and that the very early childhood onset reflects a stronger biological disposition to the illness (Fish 1977). This hypothesis is supported by family studies that have suggested (1) similar genetic predispositions in adult- and childhood-onset schizophrenia when the same diagnostic criteria were employed across age groups and (2) a higher level of penetrance in childhood-onset cases. Indeed, Rosenthal (1970) concluded that the collective evidence supported the “biological unity” of preadolescent and adult schizophrenia and suggested that “preadolescent schizophrenia is a more virulent form which has virtually complete...
penetrance.” Perhaps the strongest evidence for this conclusion was derived from Kallman and Roth’s (1956) classic report of very high concordance rates for schizophrenia among monozygotic twins of schizophrenia probands with onset before age 15 (uncorrected rate = 88.2%), as compared with those for dizygotic twins (22.9%). Kallman and Roth (1956) also reported a twofold increase in the aggregation of schizophrenia among first-degree relatives of patients with childhood-onset schizophrenia. But these early findings require confirmation by more rigorous experimental procedures such as blind assessment of relatives (which minimizes the risk of biased diagnoses arising from knowledge of the proband’s diagnostic status), control groups, structured diagnostic assessments, and operational diagnostic criteria.

Additional support for the hypothesis that childhood-onset schizophrenia is a particularly severe variant of the illness is provided by followup data indicating that a large proportion of child and adolescent schizophrenia patients present with severe schizophrenia in later life (Eggers 1978, 1989; Werry et al. 1991, 1994, this issue; for review of earlier literature, see Fish 1977; Fish and Ritvo 1978). Similarly, data from recent studies of the performance of children with schizophrenia on neuropsychological, cognitive, and neuroimaging measures suggest that childhood-onset schizophrenia is both similar to later-onset schizophrenia and associated with an underlying central nervous system dysfunction (for reviews, see R.F. Asarnow et al. 1991, 1994, this issue; Gordon et al. 1994, this issue; Strandburg et al. 1994, this issue). Because there have been few family, followup, and neurobiological studies of children with schizophrenia (as defined in adults), however, these data must be viewed cautiously.

Alternative hypotheses about childhood-onset schizophrenia are that (1) childhood- and later-onset schizophrenia represent different illnesses; (2) the atypical early onset of childhood cases is associated with potentiating factors, such as severe psychosocial or biological stressors; and (3) childhood onset has no particular etiologic significance, so childhood-onset cases represent cases at the early tail of the age-at-onset distribution and early- and later-onset schizophrenia represent the same illness or illnesses with similar levels of clinical and etiologic heterogeneity.

Whichever hypothesis proves correct, childhood-onset subjects may be a particularly useful group for analysis because their young age should lead to fewer potential confounding factors, such as neuroleptic treatment, chronic institutionalization, and years of dysfunction. If childhood-onset schizophrenia proves to be a more severe variant of the illness with a stronger biological component, however, studies of childhood-onset cases are likely to be particularly informative for isolating etiologic factors and pathways.

This article aims to clarify these hypotheses by presenting results from our research program focusing on childhood-onset schizophrenia. First, by describing the clinical presentation of our sample, we examine whether schizophrenia can be diagnosed in children by the same criteria used for adults and whether childhood-onset patients show particularly severe dysfunction. Second, we present some preliminary followup data on clinical course and outcome, thus clarifying whether schizophrenia with childhood onset shows continuity with schizophrenia in later life. Third, we examine the family environmental contexts in which these children and their disorders progress to see whether the intrafamilial attributes found to be associated with adult-onset schizophrenia are also associated with childhood-onset schizophrenia. Because this article is only an overview of our findings, the reader is frequently referred to other articles for more detailed descriptions of methods and findings.

Clinical Presentation and Description of the Sample

Diagnostic Issues. In 1980, when we began our studies of children with schizophrenia, the diagnosis of schizophrenia in children was still controversial. Nevertheless, we adopted the DSM-III (American Psychiatric Association 1980) convention of using the same criteria to diagnose schizophrenia in children and adults. These criteria require (1) an active psychotic phase with delusions, auditory hallucinations, and/or thought disorder; (2) deterioration from a previous level of functioning; and (3) continuous signs of illness for at least 6 months.

In DSM-III the same criteria were employed to define schizophrenia across different ages. DSM-II (American Psychiatric Association 1968), by contrast, included the category “Schizophrenia, Childhood Type” to refer to a broader group of children that included those presenting with adult-type schizophrenia, infantile autism, childhood-onset pervasive developmental disorder, and other psychotic conditions. These other groups of children would not have
met the DSM-III definition of schizophrenia, which uses criteria established with adults.

Although future work will ultimately clarify the advantages and disadvantages of various diagnostic systems, DSM-III-R (American Psychiatric Association 1987), DSM-IV (American Psychiatric Association 1994), and ICD-10 (World Health Organization 1992) continue the policy of using the same criteria for defining schizophrenia in children and in adults. Using the same criteria across age groups should facilitate comparisons of child and adult cases as well as analyses of continuities between childhood and adulthood. However, there may also be developmental differences in the expression of the illness. The use of the same criteria across different ages may mask developmental trends and restrict the number of cases of schizophrenia that are identified during childhood.

**Description of the Sample at Project Intake.** This article focuses on a sample of 21 children with schizophrenia enrolled in our followup study. Children were recruited from consecutive admissions for inpatient psychiatric care at the University of California, Los Angeles (UCLA), and affiliated hospitals. Other criteria for inclusion in the sample were age between 7 and 14 years, full-scale IQ above 70, and no complicating medical condition. The sample contained 15 boys and 6 girls. Children came from a range of socioeconomic levels (mean Hollingshead socioeconomic status = 40.38, range 11–66). The sample included 17 whites, 3 African Americans, and 1 Asian and was roughly similar to the overall inpatient sample in demographic variables (Asarnow and Carlson 1985).

Diagnostic information was derived from semistructured diagnostic interviews using the Schedule for Affective Disorders and Schizophrenia for School-Age Children—Epidemiological Version (K-SADS-E; Orvaschel and Puig-Antich 1987) conducted with the child; K-SADS-E interviews with parents, and all other information available on the child, including observations of the child's clinical status during hospitalization and the results of other interviews and evaluations conducted during hospitalization. Two experienced clinicians had to agree on the diagnosis for a child to be included in this sample. All children met criteria for a current episode of schizophrenia. Estimates of interrater agreement revealed a high level of reliability on diagnostic judgments (kappa = 0.82–0.91, p < 0.001).

Major symptoms identified in the sample are shown in table 1; these children clearly met DSM-III criteria for schizophrenia. Nineteen of the 21 (90%) presented with clear hallucinations, 1 presented with possible hallucinations and definite delusions, and the remaining child presented with possible hallucinations and definite thought disorder. Hallucinations were most frequently auditory (76% definite, 95% definite + possible), although 48 percent of the sample presented with visual hallucinations, and 14 percent presented with other hallucinations (tactile or olfactory). Delusions were also frequent, with 81 percent of the sample presenting with clear delusions and another 14 percent with possible delusions. Thought disorder was observed in 48 percent of the sample, and all the children presented with a clear deterioration in functioning.

Consistent with the high risk of suicide and suicide attempts among adults with schizophrenia, suicidal behavior was a frequent symptom in these children. Suicide attempts were observed in 38 percent of the sample, with an additional 38 percent presenting with suicidal ideation but no attempts. Thus, 76 percent of these children with schizophrenia showed evidence of suicidality. Because suicidal behavior is frequently a reason for psychiatric hospitalization, rates of suicidality may be higher in our inpatient sample than in other samples.

The sample was restricted to cases with childhood-onset schizophrenia, with the age at onset ranging from 6 years to 11 years 3 months. The data on age at onset, however, should be viewed as estimates and interpreted with caution for two reasons. First, although most of the children were seen at a point of deterioration leading to the index hospitalization, most had been suffering from schizophrenic symptoms before their hospitalization. Consequently, age at onset often had to be determined on the basis of retrospective reporting supplemented by earlier school and clinical records. Second, the generally chronic or insidious development of the disorder made it difficult to identify a precise point of onset. In this context, it is interesting to recall Kraepelin's
### Table 1. Clinical characteristics of schizophrenic sample

<table>
<thead>
<tr>
<th>Case/sex</th>
<th>Age at onset (years, months)</th>
<th>Thought disorder</th>
<th>Hallucinations</th>
<th>Delusions</th>
<th>Suicide</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Auditory</td>
<td>Visual</td>
<td>Other</td>
</tr>
<tr>
<td>1 M</td>
<td>8, 4</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
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<tr>
<td>2 F</td>
<td>10, 0</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 F</td>
<td>11, 0</td>
<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>4 M</td>
<td>6, 0</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>5 M</td>
<td>7, 11</td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>6 M</td>
<td>9, 11</td>
<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>7 M</td>
<td>9, 4</td>
<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>8 M</td>
<td>10, 2</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>9 F</td>
<td>9, 7</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>10 F</td>
<td>9, 0</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>11 M</td>
<td>7, 5</td>
<td>X</td>
<td></td>
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<td></td>
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<tr>
<td>12 M</td>
<td>6, 0</td>
<td>X</td>
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<td></td>
<td></td>
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<tr>
<td>13 M</td>
<td>6, 8</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
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<td>14 M</td>
<td>11, 3</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>15 M</td>
<td>10, 9</td>
<td>X</td>
<td></td>
<td></td>
<td>Poss.</td>
</tr>
<tr>
<td>16 F</td>
<td>10, 1</td>
<td></td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17 M</td>
<td>10, 6</td>
<td>Poss.</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>18 M</td>
<td>9, 3</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>19 M</td>
<td>7, 0</td>
<td>Poss.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 F</td>
<td>9, 0</td>
<td>Poss.</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>21 M</td>
<td>6, 0</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
</tbody>
</table>

Note.—M = male; F = female; X = present; Poss. = possible; Blank = not present. Deterioration was present in all cases.

(1919/1921) early discussion of the development of dementia praecox, in which he noted "that the determination of the point of time at which the disease began is often very uncertain and arbitrary owing to the development being so frequently insidious" (p. 225).

The question of the severity of dysfunction seen in children with schizophrenia can be clarified by examining data on severity of impairment, chronicity/onset, and premorbid functioning. Using procedures described earlier (Asarnow and Ben-Meir 1988; J.R. Asarnow et al. 1994), ratings were made on the basis of reviews of the child's complete medical records, cumulative school records, and records from past evaluations and treatments. Severity of impairment at the time of hospitalization was rated on the Children's Global Assessment Scale (CGAS; Shaffer et al. 1983), which ranges from 1 to 100 and includes behavioral examples that serve as anchor points. Scores below 40 reflect major impairment; scores above 70 reflect normal adjustment. Interrater reliability on CGAS scores was assessed by intraclass correlation coefficients and ranged from 0.79 to 0.99 ($p < 0.011$) across three raters.

Premorbid adjustment was assessed by two measures. First, CGAS ratings were made for the period reflecting the child's highest level of functioning. Second, we employed the Premorbid Adjustment Scale (PAS; Cannon-Spoo et al. 1982) modified to include one item from Gittelman-Klein and Klein's (1969) Associality Scale.
Ratings focused on the period during which the child had shown the highest level of psychosocial adaptation and included assessments of the child's functioning in the following areas: sociability and withdrawal, the number and quality of peer relationships, scholastic performance, social and behavioral adaptation to school, and interests. Ratings for each item were made on a scale of 0 (ideal adjustment) to 6 (severe impairment). Scores above 3 indicated some impairment, and scores below 3 indicated relatively good premorbid adjustment. To minimize bias and avoid inferential impressions, items were rated only if the record contained explicit statements regarding the child's status. An overall PAS score was generated for each child by summing the scores for each item and dividing this sum by the highest possible score (6 \times \text{number of items rated}). Interrater reliability was excellent, as indicated by intraclass correlation coefficients which ranged from 0.75 to 1.00 \((p < 0.05)\) across the three raters.

Table 2 presents these data on onset patterns, severity of impairment, and premorbid adjustment for our sample of children with schizophrenia as well as a comparison group of children with major depressive disorder (MDD). Children with schizophrenia were more likely to have insidious rather than acute onset of disorder; only one presented with an acute onset. Although acute onsets were more common among children with MDD, the difference in onset patterns between diagnostic groups was only marginally significant (Fisher's exact test, one-tailed \(p < 0.07\)). Both groups of children showed severe impairment at the time of hospitalization, as indexed by mean CGAS scores below 40. However, children with schizophrenia were significantly more impaired than those with MDD \((t = 3.42, df = 47, p < 0.002)\). These differences were even more striking when the premorbid period was examined. Whereas the mean premorbid CGAS score for children with MDD was within the normal range (> 70), the mean CGAS score for those with schizophrenia was significantly lower \((t = 5.07, df = 47, p < 0.0001)\). There was, however, considerable variability in the premorbid CGAS scores of children with schizophrenia.

### Table 2. Onset pattern, severity of impairment, and premorbid adjustment for children with schizophrenia and children with major depression

<table>
<thead>
<tr>
<th></th>
<th>Schizophrenia ((n = 21))</th>
<th>Major depression ((n = 28))</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Onset pattern</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number with acute onset</td>
<td>1 (5%)</td>
<td>7 (25%)</td>
</tr>
<tr>
<td>Number with insidious onset</td>
<td>20 (95%)</td>
<td>21 (75%)</td>
</tr>
<tr>
<td><strong>Severity, global adjustment</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current CGAS</td>
<td>(30.43 \pm 7.12)</td>
<td>(39.14 \pm 9.91^1)</td>
</tr>
<tr>
<td>Range</td>
<td>15–42</td>
<td>15–55</td>
</tr>
<tr>
<td><strong>Premorbid, global adjustment</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Highest CGAS</td>
<td>(59.81 \pm 9.55)</td>
<td>(72.82 \pm 8.34^1)</td>
</tr>
<tr>
<td>Range</td>
<td>45–75</td>
<td>48–90</td>
</tr>
<tr>
<td><strong>Premorbid Adjustment Scale</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sociability/withdrawal</td>
<td>(2.95 \pm 1.56)</td>
<td>(2.23 \pm 1.61)</td>
</tr>
<tr>
<td>Range</td>
<td>1–6</td>
<td>0–5</td>
</tr>
<tr>
<td>Peer relationships</td>
<td>(4.10 \pm 1.45)</td>
<td>(2.50 \pm 1.50^1)</td>
</tr>
<tr>
<td>Range</td>
<td>2–6</td>
<td>0–5</td>
</tr>
<tr>
<td>Scholastic performance</td>
<td>(3.95 \pm 1.20)</td>
<td>(2.39 \pm 1.50^1)</td>
</tr>
<tr>
<td>Range</td>
<td>1–6</td>
<td>0–5</td>
</tr>
<tr>
<td>School adaptation</td>
<td>(3.76 \pm 1.18)</td>
<td>(1.89 \pm 1.22^1)</td>
</tr>
<tr>
<td>Range</td>
<td>1–6</td>
<td>0–5</td>
</tr>
<tr>
<td>Interests</td>
<td>(3.36 \pm 0.84)</td>
<td>(2.11 \pm 1.42^1)</td>
</tr>
<tr>
<td>Range</td>
<td>1–4</td>
<td>0–6</td>
</tr>
<tr>
<td><strong>Overall</strong></td>
<td>(0.52 \pm 0.24)</td>
<td>(0.36 \pm 0.21^1)</td>
</tr>
<tr>
<td>Range</td>
<td>0.06–0.87</td>
<td>0.04–0.80</td>
</tr>
</tbody>
</table>

Note.—CGAS = Children's Global Assessment Scale (Shaffer et al. 1983); SD = standard deviation; Premorbid Adjustment Scale (Cannon-Spoor et al. 1982).

\(^1\) Difference between children with schizophrenia and children with major depression is significant \((p < 0.05)\).
Clinical Course and Outcome

Procedures. Initial followup assessments were conducted between 1 and 5 years after the initial assessment. Followup information was obtained for 18 of the 19 children eligible for followup (those who had been out of the hospital for at least 1 year). The one child lost to followup had moved and could not be located. Eight children who were entered into the study early participated in a second followup assessment, resulting in an age at final followup of between 10 years 2 months and 19 years 10 months (table 3). Thus, followup information is based on data collected at either one or two followup assessments. Fifteen of the 18 children (83%) were over age 12 at the final followup, and the followup period was 3 years or more for 15 of the 18.

Children’s diagnoses, symptoms, and general functioning during the followup period were assessed with the K-SADS-E and the Social Adjustment Inventory for Children and Adolescents (SAICA; John et al. 1987). These measures were administered both individually with the child and with one parent or caretaker, if the child had been removed from the home and had minimal contact with the parents. A supplement designed to assess personality syndromes viewed as falling within the schizophrenia spectrum was also administered to children and parents or caretakers. This supplement was based on the Structured Clinical Interview for DSM-II Disorders (Spitzer and Williams 1986) as well as other clinical interviews designed to assess schizotypal symptoms in childhood (Fish 1981; Russell et al. 1989). Because of the controversy regarding the status of personality disorders in childhood, we have called this interview the Semi-Structured Kiddie Interview for Personality Syndromes (K-SKIPS; Asarnow and Talovic 1986). To be given a personality syndrome/disorder diagnosis, a child had to have met the criteria for at least 1 year. Clinical and school records were requested and available information reviewed. Diagnoses were derived independently by two diagnosticians, one of whom was always blind to the initial diagnosis. The reliability of diagnoses was excellent (kappa = 0.92, p < 0.0001).

In two cases direct interviews with the child were not available. However, in both cases the parent interview information was supplemented by school and clinical records. The child who committed suicide (case 3) had been interviewed directly at age 16 years 6 months, and extensive clinical records were reviewed in conjunction with the parent interview completed after her death.

Results. Table 3 lists the following information for each child: gender, age at intake, diagnoses at intake, followup age (or ages, if a second followup was completed), additional premorbid diagnoses, medication during followup, and diagnoses at each year of the followup interval. In considering these data, a few cautionary comments are needed. First, these children were receiving treatment in their communities, and outcome may have been affected by the type, quality, timing, and amount of treatment. All but one of the children (94%) were treated with antipsychotic medications. In four cases, antipsychotics were supplemented or replaced with other medications (lithium, carbamazepine, antidepressants, methylphenidate). All 18 children received some form of psychosocial intervention, including individual and family therapy, special school programs, and day treatment. Finally, although it is beyond the scope of this report to examine predictors...
<table>
<thead>
<tr>
<th>Case/sex</th>
<th>Followup diagnoses</th>
<th>Followup age (years, months)</th>
<th>Additional premorbid diagnoses</th>
<th>Medication during followup</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 M</td>
<td>None</td>
<td>9, 0</td>
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<td>2 F</td>
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<td>12, 2</td>
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<td>9 F</td>
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<td>10 F</td>
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<td>16, 8</td>
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<td>11 M</td>
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<td>12 M</td>
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<tr>
<td>17 M</td>
<td>None</td>
<td>16, 8</td>
<td>None</td>
<td>Antipsychotic</td>
</tr>
</tbody>
</table>

Note: SZ = Schizophrenia, ATYP = Atypical, AFF = Attention Deficit Hyperactivity Disorder, ADD = Oppositional Defiant Disorder, COND = Conduct Disorder, ANX = Anxiety Disorder, SPD = Separation Anxiety Disorder, DD = Developmental Delay, MDD = Major Depressive Disorder, PD = Personality Disorder, SUI = Suicide.
of outcome, the data and case descriptions below highlight a number of potential prognostic factors.

Comorbidity. Another point that merits attention is that hierarchical rules were generally suspended in making diagnoses. Thus, if a child met criteria for a diagnosis, that diagnosis was given regardless of whether the child met criteria for another more severe diagnosis. For example, children with schizophrenia who met DSM-III criteria for attention deficit disorder (ADD) were given that diagnosis. Because ADD by DSM-III criteria requires an onset before age 6 and schizophrenia typically has a later onset, 8 of the children (44%) were given a diagnosis of ADD before the onset of their schizophrenia. Although these children clearly met criteria for ADD on the basis of interview and collateral information, it is unclear how these symptoms should be viewed in the context of the more severe schizophrenic disorder. For instance, did these early attentional symptoms represent the prodrome or precursor to the full-blown schizophrenic syndrome? Or are both illnesses present in some cases?

Similarly, significant depressive symptoms were present in 10 cases (56%). Seven children (39%) met criteria for dysthymic disorder before the onset of schizophrenia. Eight (44%) met criteria for major depression in addition to schizophrenia. Because the depressive symptoms were relatively brief in comparison with the schizophrenia, these children were given intake codiagnoses of atypical affective disorder. Five of the eight children with atypical depression at intake had also displayed premorbid depressive symptoms severe enough and long enough to meet criteria for dysthymic disorder.

Continuity of schizophrenia symptoms. One question that can be addressed with the information presented in table 3 is the degree of continuity versus discontinuity in diagnostic presentation. Simply, do children receiving a diagnosis of schizophrenia at intake continue to meet diagnostic criteria during each year of the followup?

Most of the children (14 of 18, or 78%) continued to meet criteria for schizophrenia 1 year after discharge from the hospital. Two more male children were diagnosed as schizoaffective (cases 4 and 17), so 16 of 18 (89%) met criteria for schizophrenia or schizoaffective disorder 1 year after discharge. These boys (cases 4 and 17) had originally presented with significant affective symptoms, as evident from intake codiagnoses of atypical affective disorder, which became more pronounced during the year following hospitalization. One other female child (case 16) showed no evidence of psychosis or DSM-III-defined residual schizophrenia but continued to show symptoms of sufficient severity and duration to meet criteria for conduct disorder, dysthymic disorder, and ADD. This child had been receiving extensive treatment, including antipsychotic medication. A fourth male child (case 12) presented with remission of his schizophrenic symptoms but was receiving a combination of antipsychotic and antidepressant medications. At index hospitalization, this boy had met criteria for both conduct disorder and dysthymic disorder (coded as atypical affective disorder at admission). By the 1-year followup point, the conduct symptoms had remitted, but the depressive symptoms continued, resulting in a diagnosis of continu-
described by his mother as show-
eexample, one boy (case 18) was
direct interview been available. For
it is possible that additional symp-
toms might have emerged had a
direct interview been available. For
example, one boy (case 18) was
described by his mother as show-
ing a remission of psychotic symp-
toms and no residual schizo-
phrenic symptoms at the 2-year
followup. However, he continued
to meet criteria for conduct disor-
der and ADD, was rehospitalized
because of suicidality, was receiv-
ing antipsychotic medications, and
was not available for direct inter-
view, raising questions about
whether there had truly been con-
tral or remission of schizophrenic
symptoms.

The other three cases showing
changes in diagnosis were more
convincing. One male (case 1) who
had continued to present with
schizophrenia at year 1 showed a
remission of schizophrenic symp-
toms by year 2. This remission
was sustained over the course of
the 7-year followup. At age 17,
this boy showed no detectable
signs of relapse and was not tak-
ing antipsychotic medication. Al-
though a direct interview was not
available with this boy, there were
several objective indicators of re-
cover (e.g., he had held a respon-
sible summer job and was going
to college). The second male (case
4) presented as schizoaffective by
year 1 and continued to present
with severe symptoms for another
3 years. However, substantial im-
provement was evident by about
age 16 and confirmed across re-
ports from both the subject and
his mother. At the final followup,
when this boy was age 19 years
10 months, there had been a full
remission of psychotic and affect-
e symptoms, and he was no
longer taking antipsychotic medi-
cation. The third male (case 11)
showed continuing schizophrenia
between the ages of 7 years and
11 years 6 months. However, be-
tween 11 years 6 months and 12
years 11 months, this boy showed
only some mild symptoms of par-
anoid personality disorder (e.g.,
easily slighted and quick to coun-
terattack, questions loyalty of oth-
ers, expects to be tricked) but not
enough to meet criteria for para-
noid personality disorder. This boy
was not taking antipsychotic medi-
cation at the final assessment, and
direct interviews were available
with the boy and his mother.

Thus, by the final assessment
between 2 and 7 years after the
index hospitalization, remission of
schizophrenic or schizoaffective
syndromes was observed in 6 of
the 18 cases (33%). Continuing
schizophrenia (not counting schi-
zoaffective disorder) was observed
in 11 cases (61%). Note, however,
that only one male (case 1)
showed a remission of schizophre-
nia or schizoaffective disorder that
persisted beyond 2 years, and only
four children were free of any di-
agnosis at the final followup.

Global adjustment and psychosocial
functioning. Overall level
of global adjustment and psychoso-
cial functioning was also examined
in this sample. Children were clas-
sified into the following four cate-
gories based on their CGAS scores
at the end of their individual fol-
lowup intervals: (1) deteriorating
cases, with CGAS scores that were
lower at the end of followup than
at initial entry into the project and
fell below 40 at final followup; (2)
minimal improvement cases, with
final CGAS scores between 30 and
50; (3) moderate improvement cases,
with final CGAS scores between
50 and 60; and (4) good outcome
cases, whose final CGAS scores
were 60 or above. Figures 1–4
show the CGAS scores for children
each of these groups through-
out the followup intervals.

These figures highlight the vari-
ability in functioning shown by
children in our sample. Eight of
the 18 (44%) showed relatively se-
vere impairment throughout the
followup period. Three children
(17%) showed a deteriorating
course and persistent impairment
over a 5-year followup period (fig-
ure 1); another five (28%) showed
minimal improvement (figure 2).
The other 10 children (56%) showed
more substantial improve-
ment. Five (28%) received CGAS
scores between 50 and 60 at their
last followups and were classified
as showing moderate improve-
ment (figure 3); another five (28%) were
classified as showing good out-
comes because their CGAS scores
were 60 or above at the final fol-
lowup (figure 4). Four of these
five children also showed remis-
sion of schizophrenic symptoms.

Case descriptions. The data
described above are further clari-
tied by the case descriptions pre-
sented below. Three cases are
selected for review, all of whom
were followed into late adoles-
cence or early adulthood.2 Example
1 describes a deteriorating
course. Example 2 shows how

Pseudonyms are used and other
changes have been made in the case
histories to protect confidentiality.
Figure 1. Children's Global Assessment Scale (CGAS) scores, cases with deteriorating course ($n = 3$)

Example 3 demonstrates a good outcome case.

Example 1: Sue, deteriorating course (case 3).

Sue began to have difficulties upon entering school, exhibiting pathologic shyness around strangers, sporadic elective mutism, an absence of social skills, and occasional enuresis. At that time she began to be seen in individual psychotherapy twice weekly. The next year she was placed in a special education class. At age 8, Sue began to exhibit magical thinking, including the belief that she could read other's thoughts. She was frequently oppositional, and family tensions increased.

In the fourth and fifth grades, Sue was placed in a regular classroom, where she showed increased academic difficulties, failing to complete assignments and not paying attention in class. She had few friends. Her hygiene began to worsen, as she refused to remove her clothing to bathe, and her teachers frequently commented on her odor. She continued to display magical thinking and delusions of reference. Following a bout of pneumonia, Sue became obsessed with germs.

In February of her eighth grade year, Sue became increasingly depressed, developed delusions in which she felt that the devil was trying to make her do bad things, and experienced persecutory delusions about her teachers at school. With Sue's deterioration, conflict at home increased. After talking about killing both herself and her parents, Sue made her first suicide attempt, running out in the street in front of a moving car.

Following this suicidal episode, Sue was admitted for a 2-month inpatient psychiatric evaluation. During this hospitalization, Sue's bizarre behavior continued.
Figure 3. Children's Global Assessment Scale (CGAS) scores, cases with moderate improvement ($n = 5$)

Figure 4. Children's Global Assessment Scale (CGAS) scores, cases with good outcome ($n = 5$)

Example 2: Nick, coexisting conduct problems (case 5).

Nick began exhibiting attentional and conduct problems upon entering the first grade, following the death of his twin brother in a car accident. A few months later Nick was kidnapped by his father, who kept him for 5 months. Upon returning to his mother, Nick showed severe separation anxiety, which lasted about 4 months. At school Nick exhibited hyperactivity, brief attention span, impulsivity, and aggressiveness toward class-
mated. He was frequently involved in fights on the playground and soon became violent toward both peers and teachers, maintaining no friendships. By age 8 these problems had further escalated. Nick lied frequently, ran away from home, and killed a kitten during a rage. He stole a neighbor’s bicycle and was apprehended by police and placed on probation. This incident led to Nick’s first psychiatric hospitalization, at age 8 years 3 months.

During the inpatient evaluation it became evident that Nick was struggling with several psychotic symptoms, including hearing a voice that told him to jump off a cliff, ideas of reference, and paranoia. He began to believe that others were trying to poison his food. His speech became pressured, his affect flat, his hygiene poor, and his mannerisms bizarre.

Throughout hospitalization Nick was difficult to control and usually required one-on-one supervision. Consequently, he was placed in a long-term psychiatric facility, where he continued to exhibit antisocial behavior, complained of hearing voices, and threatened suicide. For the next 8 years Nick resided in numerous long-term placements, including group homes, where his behavior could not be managed and consequently he stayed only briefly. His placements were punctuated by brief stays with his mother; during these periods at home he was frequently violent, ran away, set fires, refused school, and used substances, including alcohol and marijuana.

At age 13 Nick became depressed for approximately 1 week and was briefly hospitalized after two suicidal gestures—once slashing his wrist and once taking an overdose of chlorpromazine and lithium. Over the next several years he experienced two additional periods of depression, always lasting about 1 week and resulting in suicidal behavior followed by a brief hospitalization.

By the time Nick reached 14 years of age he had joined a street gang and had been arrested and convicted of a series of crimes, including vandalism, robberies, and shootings. Nick claimed little remorse for these activities, instead describing them proudly and in great detail.

At age 16 Nick was interviewed at juvenile hall. He had been placed there temporarily because his violent outbursts and uncontrollable behavior could not be contained in a treatment camp for youth offenders. Neither psychosocial nor pharmacologic interventions had altered Nick’s very poor prognosis. He was tried on several psychotropic medications over the years, including neuroleptics, lithium, carbamazepine, antidepressants, and stimulants.

While some of these agents appeared to exert a calming influence for brief periods, Nick’s problems have continued largely unabated since his first psychiatric hospitalization.

Example 3: Bill, good outcome (case 1).

Bill’s difficulties began during infancy. He was described as a colicky baby who was in constant motion and prone to head banging. During early childhood he required constant supervision because of his high activity level, unpredictable behavior, and tendencies toward destructive behavior such as hurting family pets and lighting fires. Because of delayed visual-motor functioning, Bill was placed in a school for children with learning disabilities at 6 years of age.

Bill’s behavior became increasingly bizarre. He began to defecate and urinate in odd places, would scratch and hit himself, and threw himself against the walls crying. He became preoccupied with germs, death, and sex, and would panic if separated from his mother. At roughly 8 years of age, Bill’s language became illogical and difficult to follow and tended to drift to morbid themes. At home and at school Bill acted as if he was hallucinating. During one episode he claimed that blood was oozing from the walls and floors and frantically attempted to tear them apart. He began playing with knives, talked of killing himself, and jumped off a high roof. Bill showed increasing signs of depression, spent his time lying on the sofa, and talked about harming himself.

Concerns about suicidal behavior and deteriorating behavior led to Bill’s psychiatric hospitalization. A trial of haloperidol was initiated and his behavior stabilized with the combination of medication and the structured inpatient treatment program. After roughly 2 months of inpatient care, Bill returned home, where he received outpatient therapy, continued on haloperidol, and attended a school program with a highly structured behavioral program and considerable individual attention. His special education classroom contained eight students, and classroom work was supplemented with daily individual tutoring.

Until age 13, Bill continued to be described as highly anxious and as sometimes disorganized, out of touch with reality, bizarre, and silly. He showed persistent problems with attention, unpredictable mood changes, impulsivity, and daydreaming. These difficulties were most apparent during unstructured times, and the structure of the behavioral program appeared to help him control his behavior. Despite his difficulties, Bill was described as likable, popular with classmates and teachers, intelligent, and curious.

Bill showed gradual but steady improvement. At age 15 he was taken off haloperidol with no adverse effects. He transferred to his neighborhood high school, received A’s and B’s in his classes, and was described as a boy who “liked to study and apply himself.” He developed a group of friends.
and became active in sports and school activities. At the final interview, when Bill was 17 years old, he was described as a popular high school senior, editor of the school newspaper, and a member of the soccer team. The summer before his senior year, he had held a responsible job working in his father’s business. There were no signs of schizophrenia or other psychiatric disorder, and Bill was preparing for college the following year.

Summary. In summary, these case examples and followup data underscore the distress and dysfunction observed among children with schizophrenia. Over the course of a 2- to 7-year followup period, 61 percent of our sample showed continuing schizophrenia as they progressed through adolescence, demonstrating that childhood-onset schizophrenia is frequently continuous with schizophrenia in adolescence. Roughly 67 percent of the sample showed continuing schizophrenia or schizoaffective disorder throughout the followup period. As young adults, the most serious outcomes included suicide, long-term hospitalization, and criminal behavior; but outcome was not uniformly poor. Four children (22%) showed substantial recovery (improved CGAS scores) and remission of schizophrenic symptoms, underscoring the fact that some children who suffer from schizophrenia will recover and function well. It is important to recall, however, that children in our sample were receiving extensive psychosocial and pharmacologic treatment, and outcome might have differed had treatment differed.

Although there are few studies of outcome for childhood-onset schizophrenia, our data are consistent with the more optimistic reports of outcome. Notably, Eggers (1989) reported remission in 27 percent of children with onset of schizophrenia before age 14 at followup evaluations conducted 6 to 40 years later (mean = 16 years), with another 24 percent showing “slight defect.” “Severe defect” was observed in 49 percent of the sample. While our remission rate was somewhat higher (33% for schizophrenia or schizoaffective disorder), our followup interval was shorter, and we also found that 44 percent of our sample presented with minimal improvement or deterioration. Additionally, only four children (22%) showed remission of schizophrenia symptoms in the absence of antipsychotic medication.

A more pessimistic view of outcome is provided by Werry et al. (1991), who report remission (defined as an absence of subsequent schizophrenic episodes) in only 3 percent of their sample over a 1- to 16-year followup interval (mean followup interval of roughly 5 years). Ninety percent of the sample showed either chronic schizophrenic or two or more schizophrenic episodes, and 13 percent of the sample had died. All those who died were described as placing themselves in the lethal situation, although it was unclear whether these were clear suicides or delusion-driven accidents. Earlier work with pre-DSM-III samples has generally suggested that prognosis is poor for childhood-onset schizophrenia (for review, see Fish 1977).

Future work clarifying predictors of outcome for children with schizophrenia may help to clarify these cross-sample differences. In both the Werry and colleagues (Werry and McClellan 1992) and Eggers (1989) samples, abnormal premorbid adjustment predicted poor outcome. However, other prognostic variables may be identified in future work. Likely prognostic variables include the quantity and quality of psychosocial and pharmacologic treatment, comorbid presentation, and level of family stress versus support.

The Family Context of Childhood-Onset Schizophrenia

In our laboratory we have conducted a series of studies focusing on psychosocial stress in childhood-onset schizophrenia. Because these studies have emphasized family and environmental variables, they are useful for describing the family environments of children with schizophrenia. We have also included children with schizotypal personality disorder (SPD). SPD is controversial in childhood, but links between schizophrenia and SPD are suggested by research indicating elevated rates of SPD among the biological relatives of persons with schizophrenia (for review, see Kendler 1988), as well as by high-risk research suggesting that schizotypal symptoms such as social isolation and signs of thought disturbance may be early precursors of schizophrenia (for review, see Asarnow 1988). By including children with SPD in our studies, we aimed to examine the broader schizophrenia spectrum and to have an opportunity to examine predictors of the onset of schizophrenia among a group of children hypothesized to be at risk for full-blown schizophrenic disorders.

Two major questions have been addressed. First, are the intrafamilial attributes found to be as-
associated with the onset and course of adult schizophrenia also observed in families of children with schizophrenia spectrum disorders? Second, are there specific family environmental attributes that are associated with childhood-onset schizophrenia spectrum disorders and are less common in families of children with other psychiatric disorders and families of children with no evidence of psychiatric disorder? Family environmental variables emphasized in our work to date and described below fall into two major categories: (1) communication patterns and (2) affective patterns.

Before considering these data, it is important to underscore two major issues. First, because we are examining the family environments of children who already present with schizophrenia or SPD, it is not possible to determine whether the family variables identified reflect responses to the disturbed child or characteristics of the parent or family that antedated or may be relatively independent of the child’s behavior. Recognizing that both parents and children make crucial contributions to the quality of family interaction, we have examined both parent and child behaviors. Second, observed parent behavior may reflect subclinical psychopathology or genetic markers of vulnerability to schizophrenia. Although the data reported here do not directly address the question of associations between the examined family variables and psychiatric disorder in parents, this is an important issue requiring further exploration.

Family Communication Patterns.
The quality of communication was assessed by a measure of communication deviance (CD; Singer and Wynne 1965), as well as measures of direct parent and child interactive behaviors. As defined by Singer and Wynne (1965), CD refers to a confusing, unclear communication style that leads to a disruption in the focus of attention and is typically assessed on projective tests such as the Thematic Apperception Test (TAT; Jones 1977) or Rorschach test. Thus, CD is conceptualized as an interpersonal manifestation of thought and attentional disturbance at an individual level. Higher levels of CD have been found in parents of adult schizophrenia patients than in parents of patients with other disorders or in parents of normal controls. Moreover, Goldstein (1987) reported that CD in parents of disturbed adolescents who did not have schizophrenia was associated with an increased risk for schizophrenia spectrum disorders in young adulthood.

As in studies of parents of adult patients, rates of CD (by the TAT CD measure [Jones 1977], which is obtained in the child’s absence) were higher among parents of children with schizophrenia or SPD than among parents of children with depressive disorders (Asarnow et al. 1988). Interestingly, children with schizophrenia or SPD from high- as compared to low-CD families showed the most severe impairments (lowest CGAS scores) and the poorest attentional functioning as indexed by the distractibility factor of the Wechsler Intelligence Scale for Children–Revised (Wechsler 1974). Although CD was also found among some parents of depressed children, CD was not associated with differences in the severity of impairment or attentional functioning in depressed children, suggesting that the CD construct may be of particular significance for schizophrenia spectrum disorders.

Tomson et al. (1990) developed a coding system, similar to the TAT CD index, which could be employed to assess communication patterns during direct parent-child interactions. The interaction task they employed was a slightly modified version of the Family Consensus Rorschach Task in which families were asked to play a game in which they looked at an inkblot together and tried to agree about what the inkblot resembled. The coding system developed by Tomson et al. was called the Disordered Communication Coding Scheme and included codes drawn from the CD measure (Doane and Singer 1977) and the Thought Disorder Index (Arboleda and Holzman 1985). Three types of communication problems were assessed: (1) difficulty maintaining attention to task, or “attentional drift”; (2) thought disorder; and (3) lack of clarity and problems committing to ideas. Contrary to findings with the TAT CD index, no significant differences were found between parents of children with schizophrenia spectrum disorders and parents of children with major depression on the Disordered Communication Coding Scheme. In contrast, this coding scheme was highly sensitive to the communication problems of children with schizophrenia spectrum disorders. When compared with children with major depression, children with schizophrenia or SPD exhibited significantly higher levels of thought disorder and attentional drift. No significant differences were found between children with schizophrenia and those with SPD. These data thus demonstrate that the attentional and thought disturbances
shown by children with schizophrenia or SPD are manifested in their social interactions and therefore are likely to disrupt social functioning and development. (For reviews of other literature on thought disorder and attentional problems in children with schizophrenia, see R.F. Asarnow et al. 1994, this issue; Caplan 1994, this issue.)

Although our finding that parents of children with schizophrenia or SPD did not show communication problems like those detected with the TAT CD measure when interacting directly with their children was contrary to prediction, these data may highlight the strong effects of children's behavior on their parents. It would not be surprising, for example, if when confronted with their child's attentional and thought disturbance during the interaction, parents increased their efforts to communicate clearly. Alternatively, the possibility that the TAT CD measure was detecting subtler communication problems than those detected with the interactional measure cannot be ruled out. Future work is needed to clarify these issues.

**Affective Patterns.** The other major construct that we have examined is the affective quality of parent-child interactions. We have employed both measures of direct parent-child interaction and the expressed emotion (EE) measure, which is obtained in the child's absence. The EE measure assesses critical and emotionally overinvolved attitudes of the parent toward the child. Numerous studies have shown that EE is a strong predictor of outcome among adults with schizophrenia, and Goldstein (1987) reported that measures of EE obtained during adolescence were associated with an increased risk of schizophrenia spectrum disorders in young adulthood.

In contrast to results with adult schizophrenia, using the Five Minute Speech Sample (Magaña et al. 1986) EE measure, rates of EE among parents of children with schizophrenia spectrum disorders were relatively low (23% vs. 44% in the most comparable adult sample) and did not differ significantly from rates of EE in a normal comparison group (J.R. Asarnow et al. 1994). When parents of children with schizophrenia spectrum disorders were rated as high-EE, this score was based on the presence of critical rather than emotionally overinvolved attitudes. Interestingly, high EE was significantly more common among parents of depressed children than among those of children with schizophrenia spectrum disorders or normal controls, a finding that is consistent with other reports indicating high rates of EE among parents of children with other nonschizophrenic disorders (Hibbs et al. 1991; Stubbe et al. 1993).

Parents of children with schizophrenia spectrum disorders were, however, more likely to express harsh criticism toward the child than were parents of normal controls or parents of depressed children on an interaction task in which parents and children attempted to resolve an affectively charged family problem (Hamilton 1991). This again underscores the likely effect of children's behavior on the behavior of their parents. Interestingly, during this interaction task the affective behavior of children with schizophrenia spectrum disorders did not differ from that of normal controls. However, depressed children were more likely to use guilt induction and somewhat more likely to use harsh criticism than children in the other groups. This suggests that the more positive behavior of parents of depressed children during the interaction may reflect an effort to calm their difficult children. Alternatively, because children with schizophrenia spectrum disorders did not show elevated levels of negative affective behavior during the interaction, their parents may have felt freer to respond critically during the interaction, perhaps in response to their children's difficulties communicating and attending to tasks.

Some support for this interpretation is provided by results of sequential analyses examining patterns of mother-child interaction in an early subset of the sample (Cook et al. 1990). These analyses indicated that mothers of children with schizophrenia spectrum disorders were significantly more likely than mothers of depressed children to respond to their children's negative verbal behavior with a reciprocal negative response. This increased tendency to reciprocate child negativity is consistent with Hamilton's (1991) report of more frequent harsh criticism among parents of children with schizophrenia spectrum disorders, as well as our speculation that parents of children with schizophrenia spectrum disorders felt freer to respond critically to negative behavior because their children showed a relatively low rate of negative behavior. On the other hand, the tendency for parents of depressed children to respond more positively after coping with a child's negative behavior may have reflected their efforts to avoid escalating conflict with guilt-inducing and critical children. Note, however, that the increased
tendency to reciprocate child negativity among mothers of children with schizophrenia spectrum disorders was restricted to instances when the mother had initially been positive or neutral and the child had responded negatively. Thus, like parents of depressed children, parents of children with schizophrenia spectrum disorders appeared to respond to the possibility of escalating negative sequences by responding more positively in sequences in which two negative responses had already occurred.

In summary, results of studies of the family environments of children with schizophrenia spectrum disorders indicate that these children are likely to show difficulties with attention and thought disturbance during direct family interactions. In turn, their parents are likely to make harsh critical comments, perhaps in response to frustrations resulting from the child's tendency to drift off task or offer disorganized and peculiar ideas. These data underscore the stress and distress experienced by the families of children with schizophrenia spectrum disorders. Future work is needed to clarify the effect of family stress on the course of childhood-onset schizophrenia, as well as to clarify associations between genetic and environmental family variables.

Conclusion

The research summarized in this report highlights the following observations:

1. Schizophrenia in children can be diagnosed reliably by the same criteria used for adults.

2. Schizophrenia in children is most frequently insidious rather than acute in onset and is associated with relatively severe levels of current and premorbid impairment.

3. Despite extensive pharmacologic and psychosocial treatment, the majority of the children with schizophrenia (61% of our sample) continued to present with schizophrenia as they progressed through adolescence, with 67 percent of the sample meeting criteria for schizophrenia or schizoaffective disorder at the end of a 2- to 7-year followup. At 1 year after discharge, 78 percent of the sample continued to meet criteria for schizophrenia, and 89 percent met criteria for schizophrenia or schizoaffective disorder.

4. There was substantial variability in outcome patterns when global adjustment was examined. In our sample of child psychiatric inpatients with *DSM-III* diagnoses of schizophrenia, 56 percent showed substantial improvement in functioning during a 2- to 7-year followup period. The other 44 percent showed either minimal improvement or deterioration.

5. Some intrafamilial attributes found to be associated with schizophrenia in adults are also associated with schizophrenia in children. Most notably, CD was a frequent characteristic of parents of children with schizophrenia spectrum disorders. Other variables, such as parent EE, were less common among parents of children with schizophrenia when compared with the most comparable data for adult schizophrenic samples. These data underscore the fact that we will likely find differences as well as similarities in the features and correlates of childhood- and later-onset schizophrenia. Some differences may be associated with the fact that the onset of disorder occurs during a different developmental stage and family coping styles may vary with the child's age and developmental level. However, differences in the syndromes might also be associated with different etiologic, potentiating, or protective factors.

Collectively, these data are not consistent with the hypothesis that childhood- and later-onset schizophrenia represent distinct illnesses. Rather, our observation of continuities between schizophrenia in childhood, adolescence, and in some instances early adulthood, as well as similarities in clinical features and associated characteristics (Asarnow, in press), suggest that the atypical early onset is associated with some other factor or factors.

The hypothesis that childhood-onset schizophrenia represents a particularly severe biologically based variant of the illness receives some support from our data indicating a severe and relatively chronic form of the illness in 61 percent of the sample. However, it is also possible that the earlier onset of the disorder resulted in greater disruption in development, which may lead to greater impairment in some cases. Additional research is needed to clarify this issue (for more extensive discussion of this issue, see R.F. Asarnow et al. 1994, this issue).

An alternative hypothesis—that the atypical childhood onset of the disorder is associated with the presence of potentiating factors, such as severe psychosocial or biological stressors—also requires additional evaluation. While our data do point to early stress in the lives of these children, a limited set of stressors has been examined to date, and the etiologic signifi-
cance of these stressors remains unclear. For example, while parent CD and harsh criticism may lead to increased stress for the child, these parent behaviors may reflect reactions to the child’s disturbance, a shared genetic liability to schizophrenia, or some other factor.

Finally, it is possible that childhood onset has no particular etiologic significance and that childhood-onset cases represent cases at the early tail of the age-at-onset distribution. If this proves correct, studies of childhood cases will still be crucial for clarifying the significance of early age at onset for the course of illness and treatment response.

In conclusion, our findings to date underscore the many research questions that require resolution. The stories of the children and families in our sample highlight the strength required to cope with the illness, as well as the suffering experienced by those who struggle to overcome schizophrenic symptoms. A clear priority in the years ahead is to develop and test treatment, rehabilitative, and preventive approaches to childhood-onset schizophrenia.

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