

Childhood Schizophrenia: Present But Not Accounted For

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The subjects of this study were 19 children and 11 adolescents who had been psychotic since childhood and who satisfied DSM-III criteria for schizophrenia except for the stipulation that a "deterioration from a previous level of functioning" must have occurred. Seven subjects had had documented signs of psychosis before the age of 30 months. The presence of thought disorder precluded giving these 7 subjects the diagnosis of early infantile autism. The authors argue that only symptoms and signs, not age at onset, can define a disorder. They also emphasize that in children and adolescents, developmental issues influence the clinical presentation.

Psychiatry has a long-established need to deny that an illness as serious as schizophrenia can occur in childhood (1). Thus, the diagnosis "childhood schizophrenia" was deleted from *DSM-III* despite the fact that children with schizophreniform thought and behavior have repeatedly been described and are acknowledged to resemble adults diagnosed as schizophrenic (1-7).

The modern controversy over the relationship of childhood psychosis to adult forms of schizophrenia began with the publication of Kanner's classic paper describing 11 "autistic" children (8). Kanner, who initially believed that these children had the earliest form of schizophrenia, later changed his opinion (9). In 1964 a British working party on childhood psychosis (10) proposed a definition of childhood schizophrenia so broad as to ensure that all psychotic children would fall within that group. Perhaps as a reaction to this, and certainly due to the findings of Kolvin and associates (4) that children with early-onset psychosis differed from those with later onset, the movement to discard the diagnostic term "childhood schizophrenia" gained momentum (11).

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There remained, however, children who "fell through the cracks." Thus, although the majority of early-onset psychoses may not mature into a schizophrenia-like disorder, there have always been some children whose illness began before they were 30 months old who developed an adult-type schizophrenia as they matured (5, 12). If we insist that symptoms which occur at age 2 are part of a different disease process than the same symptoms when they occur at age 12, we are conceptualizing a disease unlike any other. Although it has long been recognized that chronic illnesses with onset in childhood may differ in severity and in clinical expression from those with later onset, it is symptoms and signs that have defined a disorder, not age of onset (an 18-month-old juvenile diabetic has the same disease as a 9-year-old juvenile diabetic).

DSM-III has placed thought disorder (illogicality, loosening of association, and incoherence) in children within the group of schizophrenias. However, since one of the *inclusion* criteria for a diagnosis of schizophrenia is "deterioration from a previous level of functioning," the small number of children with schizophrenia-like illness whose development has been disordered since infancy must be excluded. Thought disorder has, however, been made an *exclusion* criterion for early infantile autism, thus prohibiting the diagnosis of autism for children with schizophreniform disorder. Therefore these children are not accounted for.

METHOD

The 30 subjects in our study (19 children and 11 adolescents) had all been given a diagnosis of psychosis by physicians who were independent of the research team. Except for the stipulation that there must have been a deterioration from a previous level of functioning, all of the study subjects met *DSM-III* criteria for schizophrenia. None of the subjects had received any psychotropic medication for at least 3 months before being assessed.

A symptom checklist and a physical characteristic scale were used in assessing the study subjects. The derivation of these two scales from work with a pilot group of schizophrenic children has been described elsewhere (7). The physical characteristic scale was

TABLE 1. Characteristics of the Study Population

Characteristic	Children (N=19) ^a			Adolescents (N=11) ^b		
	Mean	SD	Range	Mean	SD	Range
Birth weight (kg) ^c	3.43	0.57	—	3.43	0.78	—
Full scale IQ ^d	84.15	18.73	—	83.11	18.50	—
Age at which symptoms were first noted (years)	4.16 ^e	1.54	2.0–8.0	7.54	3.89	2–12
Age at which psychosis was diagnosed (years)	4.82 ^e	2.39	2.0–11.5	8.50	4.21	2–14
Duration of illness (years)	3.45	2.40	0.5–8.5	8.75	4.74	3–15

^a Male, 17; female, 2.

^b Male, 5; female, 6.

^c Of the 18 children for whom data were available, 3 were delivered by Caesarean section; of the 8 adolescents for whom data were available, 1 was delivered by Caesarian section.

^d Based on data for 13 children and 9 adolescents.

^e $p < .05$ (two-sample t test, $df=28$).

modified to facilitate its use with adolescents. Four characteristics were deleted from the scale without changing its statistical significance (i.e., the scale continued to distinguish schizophrenic children from normal and other developmentally disabled children). The 30 subjects in the present study included the 8 children from the pilot study (7) and 22 new subjects.

RESULTS

The Study Population

The children in this study had first shown symptoms and been given a diagnosis of psychosis at a significantly younger age than the adolescents (see table 1). There were, however, subjects in both groups in whom symptoms of psychosis were definitely documented before the age of 30 months (5 children and 2 adolescents). Only one of the 30 subjects (male) had received a diagnosis of psychosis postpubertally. Even in this subject, symptoms of psychosis had been documented before the onset of puberty (at age 12).

The earlier age of presentation of symptoms in the children was reflected in the higher incidence of speech delay as a presenting complaint (manifested by all but 2 children who were seen before age 5; see table 2). A history of incoherence (jargon), echolalia, and a prolonged interval of speech consisting of three- or four-word phrases was common to both age groups. In fact, only 3 of 19 children (16%) and 3 of 10 adolescents (33%) had histories of *normal* language development.

The diagnosis of psychosis was made by mental health workers for 4 of the children (21%) and 8 of the adolescents (73%). In all other cases, the diagnosis of psychosis was first made by pediatricians ("atypical autistic" was a favorite diagnosis because the children related relatively well). The girls showed symptoms later than the boys (mean age \pm SD = 6.88 ± 2.59 for girls, 4.02 ± 2.58 for boys). Three of the girls had been called slow learners by their schools before the onset of psychosis.

A history of significant birth complications was reported in only 1 of the 30 subjects. The delivery of

TABLE 2. Symptoms Manifested by the Study Subjects

Symptom	Children (N=19)		Adolescents (N=11)	
	N	%	N	%
Speech delay	13 ^a	68	3	27
Temper tantrums, negativism	6	32	2	18
Perseveration	6	32	2	18
Withdrawal, preoccupation	5	26	2	18
Incoherence	4	21	0	—
Hyperactivity	3	16	3	27
Thought disorder	2	11	2	18
Phobias	2	11	3	27
Hallucinations, delusions	1	5	4 ^a	36
Slow learner	0	—	2	18

^a $p < .05$ (by chi-square, 2×2 table, $df=1$).

this child had been induced at 34 weeks' gestation (birth weight, 2.07 kg) due to preeclampsia in his mother. Although he did spend 2 weeks in an incubator, his perinatal course was uneventful. Minor birth complications were noted in 7 other children and 2 other adolescents. These included minimal jaundice, prolonged labor, no documented weight gain during the last month of gestation, mild hypertension in the mother, and one child with the umbilical cord around his neck (without documented hypoxia). Two children were reported to have had choking episodes (with cyanosis) during the first week of life. Whenever documentation was available, the subjects of this study were regarded as normal, at least until 2 years of age, by the physicians who attended them. Birth, development, and family data were incomplete for 4 of the adolescents (1 adopted and 3 in permanent foster care).

All of the study subjects had been described as having no focal neurological abnormalities by examining neurologists. None of the subjects had a history of neurological trauma or disease. Four of the children and 1 adolescent manifested increased slow wave activity on the EEG. In 3 of the children the abnormal activity was reported to be in the left temporo-occipital region. No subject was described as having more than a grade 2 (of 5) abnormality on the EEG. Two children and 1 adolescent had had normal CT scans.

Family Data

All of the children lived with their nuclear families. Only 2 of the adolescents were still living with their own families; the other 9 lived in group homes for disturbed adolescents. The majority of the parents of both the children and the adolescents were members of social class IV or V. A high incidence of neuropsychiatric disorders was reported among the close relatives of both age groups (see table 3).

Symptom Checklist and Other Symptoms

Significant differences were observed between the pre- and postpubertal subjects on characteristics of language (see table 4). Severe language disturbances such as neologisms, echolalia, or clanging were rarely noted in the adolescents (although there was documented evidence that more than half of these subjects had manifested echolalia and clanging in childhood). Symptoms such as perplexity and autism (here defined as a preoccupation with internal stimuli) occurred with greater frequency in the adolescent group, as did ambivalence, paranoia, and hallucinations.

The delusions manifested in the adolescent group were all bodily delusions, whereas all but one delusion in the prepubertal group were related to identification (one child had a "Mr. Control," another believed he was the "poison ivy exterminator," and a third identified himself with various television characters). Visual hallucinations were more common in this study group than were auditory hallucinations. In all cases in which auditory hallucinations did occur, visual hallucinations were present as well. Visual hallucinations consisted of lights, perceptual distortions, and micropsia; auditory hallucinations consisted of incoherent voices or musical sounds. Only 1 subject, an adolescent, reported hearing accusatory voices.

Physical Characteristic Scale and Signs of Neuromuscular Dysfunction

The 30 subjects in this study all met the diagnostic criteria for "hypotonic schizophrenia" (7); that is, in addition to fulfilling *DSM-III* criteria for schizophrenia, these subjects all demonstrated signs of neuromuscular dysfunction. The only significant differences between the children and the adolescents in physical signs observed were in muscle power and in the mean number scores and mean weighted scores on the physical characteristic scale. (A weighted score [7] was assigned to each physical sign and each symptom based on the proportion of subjects in the pilot group of 8 who manifested that sign or symptom, e.g., 8/8 received a weighted score of 5, 7/8=4, 6/8=3, 5/8=2, 4/8=1.) Otherwise, the two groups were remarkably similar (see table 5).

All signs of neuromuscular dysfunction except lordosis were noted with similar frequency in the two groups of subjects. Since the subjects were receiving

TABLE 3. Family Histories of the Subjects

Measure	Children (N=19)		Adolescents (N=10) ^a	
	N	%	N	%
Parents belong to social class IV or V				
Mothers	13 ^b	87	6 ^c	67
Fathers	8 ^d	57	6 ^c	67
Incidence of neuropsychiatric disorders in first- and second-degree relatives				
Schizophrenia				
With hospitalization	3	16	2	20
Without hospitalization	2	11	1	10
Total	5	26	3	30
Acute psychotic episode				
With hospitalization	5	26	1	10
Without hospitalization	1	5	1	10
Total	6	32	2	20
Suicide	2	11	1	10
Epilepsy	2	11	1	10
Mental retardation	4	21	3	30
Speech delay	2	11	0	

^a Data were not available for 1 adolescent.

^b Based on data for the mothers of 15 of the children.

^c Based on data for the mothers and fathers of 9 of the adolescents.

^d Based on data for the fathers of 14 of the children.

TABLE 4. Percent of the Subjects Having Symptoms Shown on the Symptom Checklist or Otherwise Observed

Symptom	Children (N=19)	Adolescents (N=11)
From symptom checklist ^a		
Constricted affect	100	100
Perseveration	100	100
Good eye contact	100	100
Inappropriate affect	89	64
High anxiety	84	100
Fragmentation of thought	84	73
Hyperacusis	68	45
Monotonous voice	68	82
Loose associations	79	73
Neologisms	42 ^b	0
Echolalia	58 ^c	18
Illogicality	79	82
Mannerisms	63	55
Grimace	63	82
Perplexity	68	100 ^c
Autism	53	91 ^c
Clanging	47	27
Incoherence	58	55
Otherwise observed		
Ambivalence	47	91 ^b
Hallucinations	11	55 ^b
Delusions	16	18
Paranoia	21	73 ^d
Poverty of speech	74	45
Poverty of content of speech	84	73

^aMean (\pm SD) number scores: for children, 13.11 \pm 2.38; for adolescents, 12.64 \pm 2.29. Mean weighted scores: for children, 41.95 \pm 6.10; for adolescents, 40.55 \pm 4.99.

^bp<.02 (Fisher's exact test, 2 \times 2 table, df=1).

^cp<.05 (Fisher's exact test, 2 \times 2 table, df=1).

^dp<.01 (Fisher's exact test, 2 \times 2 table, df=1).

no psychotropic medication, "no arm swing" could not be considered as a side effect. The gait of both

TABLE 5. Percent of the Subjects Displaying Characteristics Shown on the Physical Characteristic Scale and Additional Signs of Neuromuscular Dysfunction

Characteristic	Children (N=19)	Adolescents (N=11)
From physical characteristic scale*		
Hypotonia	95	91
Brachycephaly	84	64
Long hands	58	45
Decreased muscle power	79 ^b	45
Decreased muscle mass	63	36
Hyporeflexia	68	55
Blue eyes	63	45
Increased outer canthal distance (greater than the 75th percentile)	58	55
Hypertelorism (greater than the 97th percentile)	21	18
Soft skin	68	55
Head size on higher percentile than height	68	73
Head circumference greater than that for the 75th percentile	53	45
Prominent nasal bridge	47	45
Deep-set eyes	47	55
Short fingers	42	27
Lax elbows	53	36
Lax metacarpal/phalangeal joints and wrists	58	36
Additional signs of neuromuscular dysfunction		
Lordosis	79 ^b	45
Strabismus	21	27
Articulation defect	53	45
Flat feet	74	45
Decreased flexor tone	42	27
No arm swing	74	64
Abnormal gait	53	45

* Mean (±SE) number scores: for children, 10.21±2.15; for adolescents, 8.09±2.51; two-sample t test, df=28, p<.05. Mean weighted scores: for children, 26.47±4.72; for adolescents, 20.45±6.90; two-sample t test, df=28, p<.05.

^b p<.10 (trend); Fisher's exact test, 2×2 table, df=1.

groups of subjects tended to be broad based and awkward. Among the adolescents, lunging from place to place was not unusual.

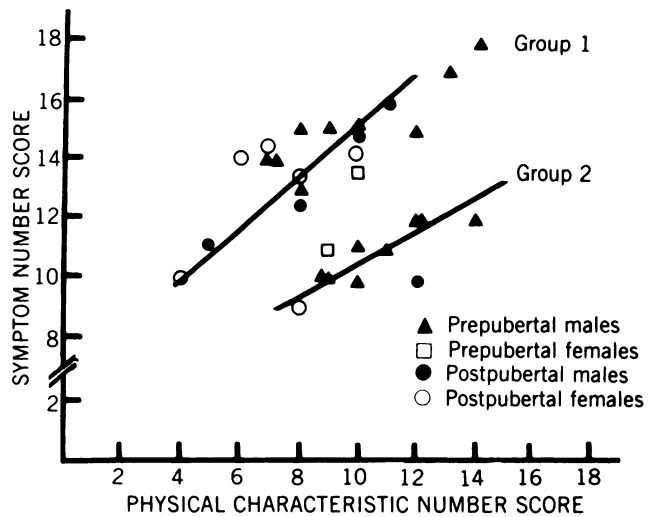
Symptom Scale and Physical Characteristic Scale

A significant positive correlation was noted between the symptom checklist scores and the physical characteristic scale scores for these subjects regardless of whether the number score was used or the weighted score was used (see figures 1 and 2). Two groups emerged. In group 2 the physical characteristic score was high relative to the symptom score. This group, however, contained only two adolescents, raising the possibility that group 2 children mature into group 1 adolescents.

DISCUSSION

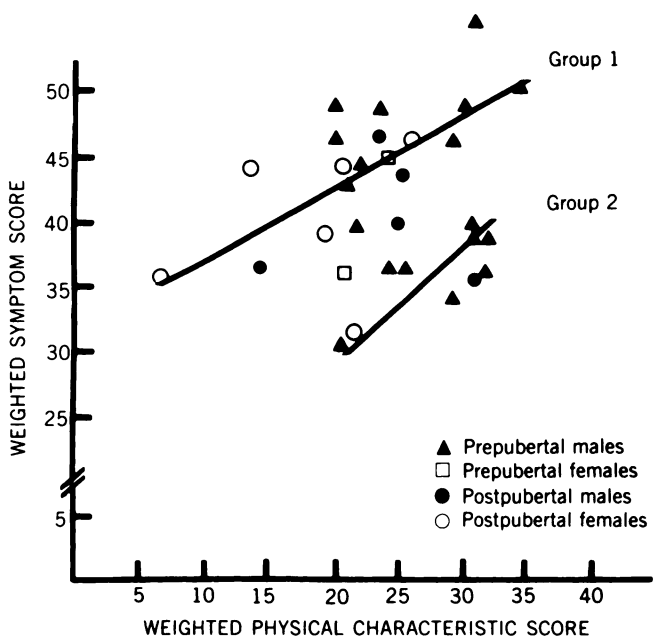
We have described the symptoms of 30 children and adolescents who had manifested psychotic symptoms since infancy or childhood. These subjects resembled

FIGURE 1. Correlation Between the Symptom Number Score and the Physical Characteristic Number Score*



*Two groups were identified: group 1, r=.874, p<.001; group 2, r=.747, p<.01.

FIGURE 2. Correlation Between the Weighted Symptom Score and the Weighted Physical Characteristic Score*



*Two groups were identified: group 1, r=.787, p<.001; group 2, r=.617, p<.05.

adult schizophrenic patients in every way except in sex ratio and in the way in which their illness manifested itself. The preponderance of boys noted here has been consistently reported for childhood-onset schizophrenia (3, 5, 6, 13). The clinical presentation of these subjects very likely reflected developmental issues. Thus, subjects who were seen before age 4 showed speech delay, and those who were seen between ages 3

and 7 displayed temper tantrums, hyperactivity, negativism, etc.

The need to consider developmental issues is further suggested by the observed differences between the pre- and postpubertal subjects. Thus, symptoms such as perplexity, autism, ambivalence, and paranoia were noted with greater frequency in adolescent subjects, who were also in fact under considerable pressure to make vocational choices and involve themselves in the community. The response of these subjects to such pressures was an increase in ambivalence, paranoid ideation, social withdrawal (and autism), and observable perplexity. Similarly, the only differences in physical signs that emerged between the pre- and postpubertal subjects were in parameters such as muscle power—parameters known to be influenced by the increase in androgens that attends puberty.

The rarity of symptoms such as echolalia and neologisms in the adolescent population and of symptoms such as hallucinations in the child population is more difficult to explain. One of the authors (S.C.) has been following up a number of schizophrenic children for the past 4 years and has observed a decrease in echolalia, clanging, and neologisms as the children mature. In addition, hallucinations were observed to begin at age 9½ in a child previously free of that symptom. Such changes in symptoms have been suggested as characteristic of childhood schizophrenia (14). As schizophrenic children mature, they increasingly resemble the adult schizophrenic population (15).

The significant positive correlation between the symptom and the physical characteristic scales (also reported with the pilot group [7]) continues to support the hypothesis that the neuromuscular dysfunction and the cognitive dysfunction share a common pathogenesis (16). So far, we have not seen thought-disordered children who meet *DSM-III* criteria for schizophrenia but who fail to manifest signs of neuromuscular dysfunction. We have seen the converse—children who manifest the physical signs but who are not psychotic. Such children have an assortment of learning, language, and behavioral disturbances. In some there is a strong family history of neuropsychiatric disorder. It seems probable that such children are manifesting the behavioral and neuromuscular disabilities that are believed to signal vulnerability to schizophrenia (17, 18). They must be carefully followed.

Thought-disordered children are not rare (2, 5, 6). Such children are seen in pediatric and child development clinics and are not infrequently institutionalized. In fact, the most likely explanation for the higher mean age of manifestation of symptoms in the adolescent group described here is that adolescents who had had an earlier onset of illness had already been institutionalized and were not available for study. Even in the group of adolescents studied here, most of the nuclear

families had broken down and the subjects were in group homes.

The literature strongly supports the hypothesis that early-onset schizophrenia is at the severest end of the schizophrenic spectrum (13, 15, 17, 19). The symptoms of the children described here, as well as the family histories of mental illness, strongly suggest that childhood-onset schizophrenia belongs within the schizophrenic spectrum. Energy and attention should be diverted away from controversy over diagnostic nomenclature and toward research into the etiology of this chronic and severe disorder. Childhood schizophrenia is present—we must account for it.

REFERENCES

1. De Sanctis S: Sopra alcune varietà della demenza precoce. *Rivista Sperimentale di Freniatria e Medicina Legale delle Alienazioni Mentale*. 1906, pp 141–165
2. Potter HW: Schizophrenia in children. *Am J Psychiatry* 89:1253–1270, 1933
3. Bender L: Childhood schizophrenia: clinical study of one hundred schizophrenic children. *Am J Orthopsychiatry* 17:40–56, 1947
4. Kolvin I, Ounsted C, Humphrey M, et al: Studies in the childhood psychoses, II: the phenomenology of childhood psychoses. *Br J Psychiatry* 118:385–395, 1971
5. Fish B: Neurobiologic antecedents of schizophrenia in children. *Arch Gen Psychiatry* 34:1297–1313, 1977
6. Wolff S, Barlow A: Schizoid personality in childhood: a comparative study of schizoid, autistic and normal children. *J Child Psychol Psychiatry* 20:29–46, 1979
7. Cantor S, Pearce J, Pezzot-Pearce T, et al: The group of hypotonic schizophrenics: a pilot study. *Schizophr Bull* 7(1):1–11, 1981
8. Kanner L: Autistic disturbances of affective contact. *Nervous Child* 2:217–250, 1943
9. Kanner L: Follow-up study of eleven autistic children originally reported in 1943, in *Childhood Psychosis: Initial Studies and New Insights*. Edited by Kanner L. New York, John Wiley & Sons, 1973
10. Creak M: Schizophrenic syndrome in childhood: further progress report of a working party (April 1964). *Dev Med Child Neurol* 6:530–535, 1964
11. Rutter M: Childhood schizophrenia reconsidered. *Journal of Autism and Childhood Schizophrenia* 2:315–337, 1972
12. Fish B: The recognition of infantile psychosis, in *Modern Perspectives in the Psychiatry of Infancy*. Edited by Howells JG. New York, International Universities Press, 1979
13. Belmont I, Birch H, Klein D, et al: Perceptual evidence of CNS dysfunction in schizophrenia. *Arch Gen Psychiatry* 10:395–408, 1964
14. Bender L: The life course of schizophrenic children. *Recent Advances in Biological Psychiatry* 2:165–172, 1970
15. Bennett S, Klein HR: Childhood schizophrenia: 30 years later. *Am J Psychiatry* 122:1121–1124, 1966
16. Cantor S, Trevenen C, Postuma R, et al: Is childhood schizophrenia a cholinergic disease? I: muscle morphology. *Arch Gen Psychiatry* 37:658–667, 1980
17. Offord DR, Cross LA: Behavioral antecedents of adult schizophrenia: a review. *Arch Gen Psychiatry* 21:267–283, 1969
18. Hanson DR, Gottesman II, Meehl PE: Genetic theories and the validation of psychiatric diagnoses: implications for the study of children of schizophrenics. *J Abnorm Psychol* 86:575–588, 1977
19. Yarden PE, DiScipio WJ: Abnormal movements and prognosis in schizophrenia. *Am J Psychiatry* 128:317–323, 1971