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SUSPECTED EARLY MINIMAL BRAIN DAMAGE AND SEVERE PSYCHOPATHOLOGY IN ADOLESCENCE.

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A GROUP OF ADOLESCENT AND YOUNG ADULT HOSPITALIZED PSYCHIATRIC PATIENTS (10 MALES AND TWO FEMALES) PREVIOUSLY DIAGNOSED AS HAVING SCHIZOPHRENIC OR PERSONALITY DISORDERS WERE REDIAGNOSED AS HAVING CHRONIC BRAIN SYNDROME. DEVELOPMENTAL DEVIANCY, BEHAVIOR DISORDERS STARTING IN CHILDHOOD, AND PSYCHOLOGICAL TEST PERFORMANCES WERE COMPATIBLE WITH AN EARLY MINIMAL BRAIN DAMAGE SYNDROME. A SHORT REVIEW OF THE LITERATURE REGARDING EARLY MINIMAL BRAIN DAMAGE AND ADOLESCENT PSYCHIATRIC SYNDROMES IS PRESENTED, AS IS A CASE STUDY OF ONE OF THE SUBJECTS INVOLVED IN THE STUDY. THIS PAPER WAS PRESENTED AT THE AMERICAN ORTHOPSYCHIATRIC ASSOCIATION CONVENTION, WASHINGTON, D.C., MARCH, 1967. (SK)

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SUSPECTED EARLY MINIMAL BRAIN DAMAGE AND SEVERE PSYCHOPATHOLOGY IN ADOLESCENCE[1]

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Abstract

A group of adolescent and young adult hospitalized psychiatric patients who had been diagnosed previously as schizophrenic or personality disorders were rediagnosed as chronic brain syndrome although none had specific cerebral disease. Developmental deviancy, behavior disorders starting in childhood, and psychological test performance were compatible with an "early minimal brain damage syndrome."

The role of minimal (diffuse) brain dysfunction is recognized as an etiological factor in a large percentage of children with severe behavior disorder.[1,13] In one survey of 150 children attending a clinic, the psychiatric diagnosis of brain damage was 59 per cent.[4] The investigators state "the incidence of brain damage among children with behavior problems might be much higher than has often been suggested in the past (p. 374). The incidence of brain damage varies with the type of behavior disorder reaching close to one hundred per cent in those diagnosed as "childhood schizophrenia."[9,14,15,16] However, the relation of early (childhood) evidence of minimal brain dysfunction to the development of "functional" psychiatric disorders of adolescence and adulthood (e.g.: schizophrenia, personality trait disturbance) is obscure.

Recently, several investigators[6,8,10] have demonstrated, through well designed studies, that neurological signs indicative of central nervous systems (CNS) damage, are significantly higher in hospitalized adolescent psychiatric patients than in normals. Hertzig and Birch[6] found neurologic dysfunction four to eight times more frequently on clinical examination in the psychiatric patients. Larsen[10] states "The significantly higher incidence of physical and neurological deficits among these adolescent patients suggests that hospital psychiatric diagnoses may incompletely reflect the comprehensive diagnostic picture. The small percentage (9% of boys, 4% of girls) of these patients with the diagnostic label of acute or chronic brain syndrome may also be an understatement" (p. 64). In a

previous study[17] of adolescent and young adult schizophrenic patients, we rated 26 per cent as having suggestive evidence of CNS abnormality.

The present study details developmental history, psychological, behavioral and EEG examinations in patients aged 15 to 25 who have been admitted to Hillside Hospital because of severe psychiatric disturbances. All of these patients had been diagnosed as having "functional" psychiatric disorders prior to their present hospital admission. Psychiatric treatment was started in childhood or adolescence. None of the cases suffered recent brain trauma nor was any unsuspected cerebral disease detected. The diagnosis of chronic brain syndrome therefore reflects the hospital staff's belief that the functional diagnosis previously designated for these patients was symptomatic rather than etiologic and that the diagnosis of chronic brain syndrome was more compatible with history and current findings.

Subjects

623 patients (242 males and 381 females) aged 15 to 25 were discharged from Hillside Hospital in a four year period (1962-1965). Of these, 14 were diagnosed as having chronic brain syndrome; two had unequivocal evidence of a cerebral disease and they were excluded from the study. In all of the remaining 12 cases there was evidence suggestive of minimal brain damage in childhood and severe psychopathology in adolescence.

Sex

The sex, years of education, family socioeconomic status (SES), age of first treatment, hospitalization and current hospitalization are shown in Table 1. Ten of the 12 patients were male. This 5:1 ratio is significantly different from the male-female ratio of 2:3 for the total hospital sample of patients under 26 years during the four years surveyed. (Chi square 8.37, $p < .01$)

SES

The SES measure used is the Hollingshead two-factor score based on a weighted average of the occupation and education of the head of the family. The scores ranged from 1 to 5 with a median of class 3. This is not different from the total hospital sample for that age group.

Education

Years of completed schooling range from seventh grade to one year of college with a median of eleven years. This is one year less than the total hospital sample for that age. The fewer years of education cannot be accounted for by SES or previous hospitalization but reflect social and intellectual inadequacy associated with psychiatric illness. This point will be elucidated later on.

Age of First Psychiatric Treatment and Hospitalization

There is a bimodal distribution of ages of first treatment with half of the patients treated in childhood and the other half in adolescence. Five of the six subjects treated in

childhood had learning defects, the sixth had delayed speech development and poor coordination (Case 5, Table 4). One patient was hospitalized in childhood (for a three month period, Case 10), nine in adolescence and two in yound adulthood. In all cases but one, either the Hillside Hospital admission was the patient's first or else the preceding hospitalization took place in the same year.

Previous Diagnoses (Table 2)

As shown in Table 2 all of the patients received several different psychiatric diagnoses prior to their Hillside Hospital admission. Nine patients had at some time been diagnosed schizophrenic or schizoid personality, eight as personality disorder or psychoneuroses. The majority had received both diagnoses. Five patients had been diagnosed as mentally retarded or brain damaged, and it is of interest that all five received these diagnoses in childhood.

All patients except Case 6 were initially given a primary or secondary diagnosis of chronic brain syndrome by the Hillside Hospital staff. Clearly such a diagnosis must have been based in part on the patient's long psychiatric history. All of the psychiatric disorders must be considered as chronic since these patients were all deviant in childhood.

Current Neurological and Psychological Test Findings

Neurological and EEG Findings (Table 3): The neurologist's report indicated evidence of CNS dysfunction in cases 4, 5 and 6. None of these patients had evidence of cranial nerve or gross sensory impairment. Four of the EEG tracings were abnormal,

three others showed the 14 & 6 cps positive spiking pattern. The latter is considered by some electroencephalographers to be indicative of CNS dysfunction.[3,5,7] Gibbs[3] states "Fourteen and six per second positive spikes indicate a specific form of epilepsy, arising in part of the brain that is not highly convulsive but which is peculiarly susceptible to dysrhythmia" (p. 293). The high frequency of the 14 & 6 positive spike pattern in behaviorally and neurologically normal subjects[12] makes it difficult to accept this pattern as an unequivocal sign of CNS dysfunction. Only case 4 had <u>both</u> neurological and EEG examinations indicating CNS dysfunction.

<u>Psychological Findings</u>: All patients but one were seen by the hospital psychologist and received the standard clinical battery of psychological tests: Wechsler Adult Intelligence Scale (WAIS), Rorschach, drawings of the human figure, Bender-Gestalt and, occasionally, other tests. The psychologist's report indicated evidence of CNS dysfunction in all 12 cases (in case 6 the evidence was suggestive). The base rate for the psychologists diagnosing CNS dysfunction was not surveyed but it would certainly not exceed ten per cent of the hospital population. Perhaps a major factor in the psychologist's conclusion was the patient's poor cognitive performance, particularly his poor perceptual-motor performance on the WAIS. The Performance Scale IQ's were below average for all of the subjects. In eight cases it was in the defective or borderline defective range.

The ten males had the following mean IQ's: Full Scale 89.0, Verbal 97.2, Performance 79.7. A survey of consecutive Hillside Hospital admissions in 1964 revealed that for 74 male subjects in the same age group, the mean WAIS IQ's were: Full Scale 111.9, Verbal 117.8 and Performance 102.5. Differences between these two groups are significant at the .01 level.

Almost all of the twleve patients had one or more previous psychological examinations. Examination of the previous reports indicates a high degree of stability of the IQ scores on repeated testings.

Paranatal, Developmental, Neurological and Behavioral History (Table 4)

Paranatal: Based on parental reports and hospital records seven of the cases had histories of prenatal and/or gestational difficulties. There was a wide range of severity with some of the cases having unequivocal complications, while in others the difficulties are more difficult to evaluate. The paranatal complications were associated with deviant temperamental and activity patterns (e.g., crankiness, hypoactivity).

Developmental Deviancy: All of the patients had abnormal childhood histories characterized by difficulties in socializing with peers and occasionally difficulty with adults. While the behavioral patterns varied, the patients were typically socially inappropriate, extremely infantile or irritable and bossy. They lacked friends and were frequently scapegoated by schoolmates and neighborhood children. Neurotic symptoms, conduct disorders, faulty habit patterns were common.

Neurological, "Soft Signs" and Cognitive Disorders: Some impairment in perceptual, perceptual-motor, coordination or speech development (either singly or in combination) was noted in 11 of the 12 cases. Ten of the patients had learning and concentration difficulties that were reflected in poor academic achievement. Hyperkinesis and impulsivity were common and many of the patients could be classified as having shown the hyperkinetic-impulse disorder syndrome. These behavioral difficulties were still present in adolescence in many of the cases and were noted as the presenting symptoms associated with hospital admission, (e.g., poor concentration, impulsivity, poor judgment).

Posthospital Adjustment

Although we did not carry out a systematic posthospital follow-up on these patients, the hospital does offer an after-care service and the patients were traced through that. There was no contact in two cases. Not one of the ten patients for whom there was follow-up information was doing well. Seven patients have been rehospitalized in city or state psychiatric hospitals. Three other patients held jobs temporarily but have not been able to function independently.

The poor prognosis must be evaluated with awareness of the considerable effort expended by the hospital (the average hospitalization is close to one year) and other agencies in rehabilitation, work-training, sheltered workshop programs and after-care psychiatric treatment. The outcome for this group is considerably poorer than that found in our follow-up[11] of

a large sample of Hillside Hospital patients discharged in 1960 and 1961. However, it does not differ from the results[17] obtained for the subgroup of patients in that sample who were classified as having "suggestive early minimal brain damage."

Comment

The twelve cases presented can best be conceptualized as defective children with deviant temperament and cognitive development, both factors reflecting a diffuse cerebral dysfunction, most likely of paranatal origin. The psychiatric symptoms that required treatment in childhood and hospitalization in adolescence are manifestations of a life-long difficulty which becomes exacerbated with the sexual, social, educational and occupational challenges of impending adulthood. The transition from childhood dependency to adulthood independence is extremely difficult for these inadequate patients. They meet stresses in almost all areas of life and the satisfactions are few and far between. The schema outlined is compatible with the temperamental-environmental interaction theory proposed as an explanation for certain behavioral disorders of childhood[21] and the "reciprocal-escalation" theory outlined by Rosenthal[20] as having etiologic importance for adult schizophrenia. The latter theory implies a "gradual ongoing interaction between the environment and inherited factors, each reciprocally intensifying the action of the other, building up acceleratively to the eruptive state observed in acute schizophrenic reaction" (p. 579). The schema I have outlined differs from that of Rosenthal's in that the biological impairment need not be due to an inherited factor but could be related to pre or paranatal abnormalities.

Early Minimal Brain Damage and Adolescent Psychiatric Syndromes

Although the literature indicates an abnormally high incidence of CNS dysfunction among hospitalized adolescent psychiatric patients, there is little data on the relationship between CNS dysfunction and specific developmental deviancies and psychiatric syndromes.

Previous studies of hospitalized adolescent psychiatric patients have found that neurological signs and psychological test evidence of CNS dysfunction were most frequent in those patients diagnosed as schizophrenia.[6,8]

In a comparison of monozygotic twins discordant for schizophrenia, Pollin et al.[18] found a significant difference in the presence of minimal neurological signs. Neurological "soft signs" were evident on examination in eight of the 11 patients, but in only one of the twin controls. No specific neurological syndrome or disease was detected. In addition to the neurological signs, the patients were significantly lower in birth weight and more often were described as having had perinatal difficulties such as cyanosis or respiratory difficulty. Pollin et al.[18] relate the suggestive CNS dysfunction to developmental deviancy as follows: "The index's generalized higher level of physiological instability plus a diffuse CNS malfunction if it exists may lead to significant differences between him and his larger co-twin with regard to his behavior, his subjective experience of himself and the world and the response to him of those who care for him. He appears to have greater difficulty in achieving

control of his body and later in manipulating his physical environment. He may be subject to a higher level of distractibility and therefore have greater problems in developing and maintaining focal attention. Objectively, he is likely to be less competent in observing and dealing with his environment; subjectively, he will have a diminished sense of personal competence and effectiveness" (p. 507).

The diagnosis of adolescent patients as personality trait disturbance or schizophrenia is supposedly descriptive (and therefore etiologically nonspecific) nevertheless, etiology is implied as either psychogenic (familial) or genetic, depending on the observer's orientation. Insufficient attention is paid to the etiologic role of early minimal brain damage. Essen-Moller[2] suggests a double classification, syndromic and etiologic, as a means of clarifying the diagnostic picture. He points out that neglecting the question of etiology often produces a diagnosis which is a meaningless compromise between divergent schools.

The ongoing effort to delineate those patients who manifest "schizophrenia-like" behavior as a sequela to diffuse CNS dysfunction, such as epilepsy or encephalitis, from the larger body of patients with functional psychiatric diagnoses is of great interest. This hypothesis was first postulated by Rosanoff et al.[19] in 1934. "We submit, as a part of our theory of so-called schizophrenic psychoses, that a large proportion of such psychoses originate in a cerebral trauma at birth or in child-

hood; that such cases are more prevalent in the male than in the female sex, and in young subjects than in those over thirty years of age; that the type of injury is often asymptomatic, or almost so, at the time of its occurrence..." (p. 284). Recently Slater and Beard[22] have shown that "schizophrenia-like psychoses" may develop 12 to 15 years after the onset of seizures. Furthermore, there was a very high correlation between the age of onset of epilepsy and psychosis. They also found that the duration of epilepsy plays a curcial role in determining the psychosis.

Just as Slater and Beard[22] argue that these patients with epilepsy should not be diagnosed as schizophrenia, it would be helpful if the type of adolescent and adult psychiatric patients described in this study, with childhood onset and suggestive evidence of CNS dysfunction, were separated from those whose illnesses may very well be due to genetic or other causes. The percentage of schizophrenic patients with suggestive evidence of early minimal brain damage is not insignificant. The magnitude of this group is not reflected in the small number of cases reported in this study. In a previous study of Hillside Hospital patients, 26% of schizophrenic patients were rated as having suggestive early minimal brain damage.[17] As reported earlier this group of patients had a significantly worse post-hospital course than the other schizophrenic patients.

The history of paranatal abnormalities, learning disabilities, psychological test evidence suggestive of CNS dysfunction, and neurological "soft signs" found among patients with a history

of severe behavior disorder in childhood, should militate against a primary descriptive diagnosis (e.g., schizophrenia, personality trait disturbance), and the adoption of a more pertinent, etiologically meaningful diagnosis such as "early minimal brain damage syndrome" or "chronic brain syndrome." This point is illustrated by a detailed report of a patient in this series (case 4) who had been diagnosed as schizophrenia prior to hospital admission.

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Case Study

Arthur B. - (Case 4)

Identification: Arthur is an 18 year old white single boy, born in New York City where he lived until his family moved to the suburbs when he was 14 years old. His father is a 58 year old physician who carries on a successful general practice. His mother, age 50, holds a master's degree and was employed as a social worker until the patient's birth. Arthur's only sibling is his sister, a 16 year old high school student. There is no history of psychiatric illness in any close relation.

Three months prior to his hospital admission, Arthur dropped out of high school, where he was beginning his senior year. He attended local public schools, having difficulty throughout; he repeated the third grade and was placed in slow classes in Junior High School.

Present Illness: Arthur has had difficulties, and been receiving some form of treatment from early childhood. However, the events which led to his current hospitalization began about one year prior to it, following an incident in which he was forced into performing perverse sexual acts by a neighborhood boy. Following this episode Arthur became increasingly fearful and suspicious; this progressed to the point where he refused to go to school, and remained in the house with the doors and windows locked. His parents made an unsuccessful attempt to get him to attend a private high school for disturbed boys. He became preoccupied with thoughts about death, about his body and sex. He told his father that his arms were too long, and that he wanted to go to the hospital to have them shortened. He felt that people could see through his pants. Arthur was fearful about coming into the hospital; he acquiesced when his parents promised him a boat as a bribe.

Developmental History: Arthur was the product of a planned, uneventful first pregnancy. Mrs. B. went into labor three weeks earlier than expected. It was a precipitous labor and Arthur was born an hour and a half after it began, immediately after their arrival at the hospital. Birth weight was five pounds. Arthur was placed in an incubator for twelve hours but was discharged from the hospital in one week. He had severe diarrhea for the first month and his weight declined to 4 lbs. 8 oz. He was a cranky, difficult baby. His motor development was slow and his speech development delayed. Bowel training was not accomplished until age four. Nightly bed wetting continued until age 16. From the age of two, Arthur had frequent respiratory illnesses, multiple allergies and asthma.

From the time he began to speak, a severe speech defect was noted and described as resembling cleft palate speech. In addition, Arthur had a peculiar waddling gait and severe difficulty with fine motor coordination. Arthur was described as hyperactive during the preschool period and his mother reports having restricted his activity a great deal. He had considerable difficulty adjusting to nursery school at age 4½.

During his elementary school years, he was described as a demanding, irritable, unhappy, shy child, who was teased by other children and unsuccessful in establishing friendships. Severe sleep problems and fear of the dark persisted throughout his childhood and adolescence.

He had difficulty with school work from the start. His mother reports having spent hours every night attempting to help him with school work; these sessions were continued despite extreme resistance and temper tantrums on the part of Arthur.

Adolescence brought little change in his behavior patterns except for a decline in peer contacts, increasing fearfulness and the appearance of obsessive symptomatology.

Previous Illness: At the age of four, Arthur was taken to a neurologist who reported no neurological pathology and recommended treatment at a speech clinic. He received individual and group therapy at a college-affiliated speech and hearing center for three years. Their report describes him as having infantile articulation, nasality and unintelligible speech resembling cleft palate speech. In addition they noted a short attention span, behavior problems and described him as uncooperative, remote and asocial. A Merrill Palmer score in above normal range was reported. The clinic staff regarded his speech problem as stemming from "emotional factors" but after three years of treatment they reported little progress with the speech defect, despite "significant growth in socialization."

When Arthur was seven, a leading psychoanalyst was consulted who described him as a child with a borderline schizophrenic character disorder, suffering from over protection by his mother and distance from his father. Psychotherapy was recommended.

During the same year Arthur was seen for diagnostic testing and consultation by a leading child psychologist, who described him as a "visual deviant with too much hyperopria for his age", poor coordination of eye muscles and poor control of speech musculature. His intellectual functioning was felt to reflect "marked restriction rather than straight

retardation." The recommendation was for placement in first grade (he was then entering third) in a special school. It was felt that he was "potentially normal" and that he would achieve with "proper school experiences."

Dr. and Mrs. B. rejected the special school considering it a school for retardates and placed Arthur in psychotherapy for two years with a well known psychologist who specializes in treating brain-damaged children. This psychologist's diagnosis was that Arthur's disorder was "neurological basis and emotional disturbance." He viewed Arthur as "a shy, timid, withdrawn boy who was pushed by well-meaning but eager parents and a brighter sister."

Between the ages of 8 and 11, Arthur was tested five times with I.Q. scores reported to range from 88 to 117. At age 11 he was seen in consultation by a leading child psychiatrist whose diagnosis was of a neurotic behavior disorder in a child with organic impairment of coordination and speech. It was felt that the organic factor did not affect his intelligence and that the behavioral symptoms were a secondary defense, rather than a direct organic manifestation. The recommendation was for continued psychotherapy.

At age 12, another psychiatrist treated Arthur for one year, after which he concluded that little progress had been made and that tutoring was more likely to prove beneficial. He regarded Arthur as a boy of limited intelligence and a schizoid personality "who has proved a major disappointment to his demanding parents." An electroencephalogram taken at this age was within normal limits.

Psychological testing at age 12 revealed a full scale W.I.S.C. - I.Q. of 88 (Verbal 97, Performance 79). The psychologist's opinion was of "severe emotional disturbance in a preadolescent boy...characterized by a pronounced obsessive-compulsive rigidity, pervasive self-doubting and disturbances in thinking which point to a schizophrenic process."

Testing by the same examiner two years later produced a W.I.S.C. full scale I.Q. of 82 (Verbal 103, Performance 64) and the recommendation to consider a residential treatment school.

For two years prior to his hospitalization, Arthur was seen by yet another psychiatrist in joint psychotherapy with his parents. Thorazine and Miltown medication was also employed. The diagnosis of this physician was schizophrenic reaction with paranoid trends in an adolescent with low average intelligence. The deterioration of behavior described under present illness caused this psychiatrist to refer Arthur for hospitalization at Hillside.

Family Structure:

Sister: Arthur's sister is described by the parents as a normal, happy, outgoing child who has done exceptionally well in school, has many friends and participates in a variety of activities. She has won awards for athletics and photography. She was interviewed and tested by the research staff, who agreed with the parent's view of her personality. Her I.Q. on the W.A.I.S. was 121 (Verbal 123, Performance 116).

Mother: Mrs. B. impressed both the clinical and research staff as being an energetic, domineering woman, who had very high aspirations for her children. She herself had excelled in academic work and she appears to place extremely high value on this form of accomplishment in others. It was readily apparent when she discussed Arthur that she never gave up the idea that he was really a bright child whose difficulties were entirely "emotional", (hence to her, reversible). She blamed herself for having been the cause of his problems. His first psychological test score at age four and the opinion of the first psychoanalyst consulted (who remained a continuing consultant to the family) remained salient for her despite the many contradictory opinions encountered afterward.

Father: Dr. B. is a quiet man who has devoted himself largely to his medical practice, although he appears to have been very attentive to Arthur throughout. He was more able to accept the idea of neurological impairment in his son. In fact, he believed it to be the result of the precipitous delivery and he blamed himself for having left the house despite his wife's complaint of pains and for not having delivered her at home when he returned, instead of rushing her to the hospital.

Course in Hospital: The diagnosis on admission was schizophrenia, chronic undifferentiated, but on the basis of psychological test and EEG reports of CNS abnormality and the equivocal neurological examination as well as history, it was changed to chronic brain syndrome.

Initially Arthur was a severe management problem; thorazine medication proved unsuccessful and was terminated after he had a drug-related seizure. Marked improvement followed a course of twenty ECT, supportive psychotherapy and encouragement of participation in social activities. Work with the parents centered on relieving the pressure of their ~~quiet~~ guilt feelings and helping them to lower their level of expectation in terms of further academic education for Arthur. Before discharge he began attending a hospital-supervised settlement house and a vocational training school for mildly defective individuals. Discharge plans included continuation of these

activities and continued private psychiatric care and medication. Arthur was discharged as clinically improved after 327 days of hospitalization.

Follow-up: Following discharge, Arthur attended a Mental Health Association Workshop and engaged in Hillside Hospital supervised social activities at a settlement house. However, despite these excellent after care facilities, in addition to continued psychotherapy and drug treatment, his condition deteriorated. Nine months after discharge from Hillside Hospital he was admitted to a state hospital. At that time there was an exacerbation of his previous symptom pattern consisting of fear of neighborhood boys and homicidal and suicidal threats.

Table 1. Patient Characteristics

Case #	Patient	Sex	Family S.E.S.	Education Years	First Treatment Age	First Hospital Age	Hillside Admission Age
1	S.P.	M	3	10	5	16	16
2	J.G.	M	5	10	15	16	16
3	L.M.	M	3	11	14	17	17
4	A.B.	M	1	11	4	18	18
5	H.B.	M	3	13	6	18	18
6	D.I.	M	3	11	15	19	19
7	W.P.	M	5	11	17	19	20
8	H.S.	M	3	12	16	22	22
9	M.K.	M	4	9	7	22	22
10	J.H.	M	N.R.*	10	7	7	25
11	B.B.	F	3	7	9	17	17
12	M.L.	F	2	11	16	18	18

* Not ratable

Table 2. Previous Diagnoses*

Case #	Patient Age	Diagnosis
1	5	Functional Dyslalia
	6-11	Psychoneurosis anxiety & depression
	16	Psychoneurosis, obsessive-compulsive, phobic & depressed
	H.H.**	(1) Schizoid personality with depressive features (2) Chronic brain syndrome, etiology unknown
2	15	Schizoid personality
	16	(1) Schizophrenia (2) Mental retardation
	H.H.	Chronic brain syndrome
3	14	Suspected brain damage
	17	Schizophrenic reaction, paranoid type
	17	Schizophrenic reaction with grandiose & paranoid features
	H.H.	Organic brain syndrome with a schizophrenic reaction
4	4	Unintelligible speech, probably stemming from emotional factors
	7	Borderline schizophrenic character disorder
	7	"Marked (intellectual) restriction rather than straight retardation"
	8-10	Emotional disturbance with a neurological basis
	11	Neurotic behavior disorder with organic impairment of coordination and speech
	12	Limited intelligence and schizoid personality
	12	Severe emotional disturbance, possible schizophrenic process
	14	Infantile character structure, possibly schizophrenia, unclassified
	15	Schizophrenic reaction with paranoid trends
	H.H.	(1) Schizophrenia, chronic undifferentiated (2) Chronic brain syndrome
5	6	Diffuse organic involvement
	8	Physical and emotional retardation, possibly on constitutional basis, with neurotic trends
	10	Neurotic behavior disorder
	11	Neurotic character disorder, passive-aggressive type
	14	Severe personality disorder with diffuse organic involvement
	16	Severe personality disorder with schizoid trends and diffuse organic involvement
	H.H.	Chronic brain syndrome, unknown cause, in a person with schizoid personality

Table 2. Previous Diagnoses*

Case #	Patient Age	Diagnosis
6	15	Depression
	17	Passive-dependent individual
	H.H.	Antisocial reaction
7	17	Adolescent adjustment reaction in a schizoid boy
	18	Borderline schizophrenic reaction
	19	Schizophrenia, chronic undifferentiated
	H.H.	Chronic brain syndrome, with sociopathic character disorder
8	16	Passive-dependent personality with depression and suicidal ideation
	22	Schizophrenia, chronic undifferentiated
	22	Schizophrenia
	H.H.	Chronic brain syndrome, etiology unknown
9	7	Borderline intelligence and emotional problems
	10	Schizophrenia, chronic
	10-15	Severe character neurosis, schizoid
	15-21	Schizophrenia
	21	Inadequate, passive dependent boy
	H.H.	Chronic brain syndrome, unknown cause, with psychosis
10	25	Schizophrenic reaction, schizo-affective type
	H.H.	Chronic brain syndrome, etiology undetermined
11	9	Possible mental deficiency
	15	Anxiety hysteria with phobic features
	H.H.	Chronic brain syndrome in an hysterical personality with psychopathic features
12	16	Passive-aggressive personality, aggressive type
	17	Passive-aggressive personality
	17	"Possible organic factors"
	H.H.	Chronic brain syndrome with borderline retardation

* Diagnoses obtained from reports of previous consultation and treatment centers, including clinics and private psychiatrists.

** Hillside Hospital initial diagnosis.

Table 3. Current Neurological and Psychological Test Findings

Case #	Neurological Exam. Findings	EEG Findings	W.A.I.S. Full IQ	W.A.I.S. Verbal IQ	W.A.I.S. Performance IQ	Psychologist Report of CNS Dysfunction
1	Negative	Borderline Abnormal: Right-sided slowing	91	100	81	Definite evidence
2	Negative	Normal	73	74	75	Definite evidence
3	Negative	14 & 6 positive spiking	85	93	77	Definite evidence
4	Equivocal: Right-left confusion suggests organicity	Abnormal: diffuse slowing and spiking accentuated on right side	83	98	67	Definite evidence
5	Suggestive of minimal organic cerebral dysfunction: Awkward jerky movements including eye movement; disarthria	Normal	(121	146	92)*	Definite evidence
6	Probable congenital right cerebral pathol: Left Babinski; Left biceps & triceps reflexes greater than right	14 & 6 positive spiking	105	115	90	Suggestive evidence
7	Negative	Abnormal: Right posterior slowing	92	89	97	Definite evidence
8	Negative	Borderline:diffuse slowing	90	104	72	Definite evidence
9	Negative	Normal	67	81	52	Definite evidence
10	Negative	14 & 6 positive spiking	88	86	93	Definite evidence
11	Not done	Not done	85	91	79	Definite evidence
12	Negative	Normal	81	89	73	Definite evidence

* Wechsler-Bellevue scores on testing three years prior to hospitalization

Table 4. Paranatal, Developmental, Neurological and Behavioral History

Case #	Paranatal and Infant Difficulties	Childhood Deviance	History of Physical - Neurological - Behavioral "Soft Signs"
1	History of two prior abortions. Staining first trimester. Asthma, adrenalin taken weekly throughout pregnancy.	Very few friends. Very little peer-interaction. Friction with parents and siblings. Inattentive, demanding. Fearful of new situations. Low frustration tolerance.	Disarthric speech. Mild hyperkinesis. Poor coordination. Concentration difficulty. Learning defect.
2	None	Poor peer relations. Over dependence. Dawdling, daydreaming. Eye blink and mannerisms "nervous child."	Strabismus. Poor coordination. Inability to follow instrustions. Distractible. Learning defect.
3	Seven-month gestation. Birth weight 3 lbs. 11 oz. Cyanotic. Born with right side hydrocele and right inguinal hernia. Incubator two months.	Moody, infantile, demanding, bizarre behavior, enuresis, poor peer relations.	Retrolental fibroplasia. Learning defect.
4	Eight month gestation. Precipitate delivery. Birth weight 5 lbs. Incubator 12 hours. Neonatal: Severe diarrhea with weight loss first month.	Temper tantrums, enuresis to age 16, few friends, teased by other children, shy, sleeping problems, irritable, unhappy, overdependent.	Slow motor development. Delayed speech. Severe speech defect. Poor coordination. Peculiar gait. Hyperkinesis. Learning defect.
5	Mother had fibroid tumor during pregnancy. Forceps delivery. Maternal postnatal hemorrhage. Neonatal: very cranky, sedation prescribed.	No friends, teased by other children.	Delayed motor and speech development. Speech defect. Poor coordination.

Table 4. Paranatal, Developmental, Neurological and Behavioral History

Case #	Paranatal and Infant Difficulties	Childhood Deviance	History of Physical - Neurological - Behavioral "Soft Signs"
6	History of prior abortion. Difficulty conceiving. Eight month gestation. Birth weight 4 lb. 10 oz. Did not breathe spontaneously. Neonatal: hospitalized one month; too weak to nurse; very cranky, sedation prescribed.	Few friends. Enuresis. Stealing, gambling, truancy.	Impulsivity, Poor coordination.
7	Unwed mother, no prenatal care, inadequate nutrition; home delivery with mid-wife assisting, dry, breech delivery.	Seclusive child, avoided peer-interaction, accident prone, discipline problems, truant.	Impulsivity. Learning defect.
8	First four months of pregnancy mother in concentration camp: severe physical and emotional stress. Neonatal: hypoactive infant.	Withdrawn, irritable, unhappy child, many fears, temper tantrums, feeding and sleep problems, poor judgement, inappropriate laughing, repetitive speech.	Slow speech development. Concentration difficulty. Hyperkinesis.
9	None	No friends, scape-goated by children, disruptive in school, demanding, overdependent, shy.	Strabismus. Slow speech development. Concentration defect. Impulsivity. Learning defect.
10	None	Periods of depression and withdrawal. Discipline problem in school.	Congenital bilateral cataracts, left exotropia. Strabismus. Concentration and memory defect. Learning defect.
11	None	Feeding problem, disruptive behavior in school, temper tantrums.	Learning defect
12	None	Poor peer relations, teased by children, overdependent, low frustration tolerance.	Slow speech development. Speech defect. Concentration defect. Learning defect.