

Shaffer, D., Stokman, C., O'Connor, P.A., Shafer, S., Barmack, J.E., Hess, S., Spalten, D., & Schonfeld, I.S. (1986). Early soft signs and later psychopathology. In L. Erlenmeyer-Kimling (Ed.). *Life-span research on the prediction of psychopathology* (pp. 31-48). Hillsdale, NJ: Erlbaum.

# 2

## Early Soft Neurological Signs and Later Psychopathology

David Shaffer  
Cornelius S. Stokman  
Patricia A. O'Connor  
Stephen Shafer  
Joseph E. Barmack  
Suzanne Hess  
D. Spalten  
Irvin S. Schonfeld

Total population studies, as well as studies of children selected for neurological rather than psychiatric disorders, have shown that children with unequivocal brain damage or epilepsy have considerably higher rates of psychiatric, learning, and cognitive problems than children who are neurologically intact (Rutter, 1977; Rutter, Graham, & Yule, 1970; Shaffer, 1977; Shaffer, McNamara, & Pincus, 1974). However, the relationship between lesser neurological disturbance (i.e., minimal brain dysfunction, or MBD) and psychiatric disorder remains a matter of controversy.

The concept of an MBD syndrome has generally included behavioral deviancy, most commonly taking the form of hyperactivity or inattention, learning difficulties and cognitive abnormalities, and a characteristic response to stimulant treatment. These have been linked to various indicators of neurological impairment, most frequently a history of perinatal morbidity and the presence, on examination, of neurological signs or EEG abnormalities that fall short of being indicative of a classical neurological disease or defect state.

The controversy centers around two issues: first, whether or not the psychological correlates are specific in their relationship to neurological dysfunction, i.e., can they be regarded as pathognomonic, either alone or as a syndrome (see Shaffer, 1980); and second, whether the components are found together with any frequency in the clinical population.

It seems clear that the psychological components of the MBD syndrome cannot be regarded as pathognomonic. Hyperactivity, as it is most commonly observed, is a situation-specific behavior (i.e., occurs either at home or at school), an observation that is in itself inconsistent with a fixed, neurologically based phenomenon. Hyperactivity occurs similarly in both neurologically normal and abnormal children with a behavior disturbance (Shaffer et al., 1974). Both hyperactivity and inattention have biological and environmental correlates (Rapoport & Quinn, 1975; Sandburg, Rutter, & Taylor, 1979), although the relatively less prevalent cross-situational hyperactivity which is observed both at home and at school may be found more often in organically impaired children (Campbell, Endman, & Bernfield, 1977; Schachar, 1979).

Learning difficulties and deviant behavior are commonly associated (Rutter & Yule, 1973; Rutter et al., 1970), and although the presence of this association has been used by some to argue for an underlying brain or neurological abnormality as a causal factor for both, numerous alternative explanations are available. Reading disability could lead to psychiatric disorder (or vice versa), and the common antecedent of both could be some external influence (e.g., poor schooling) rather than brain damage. Further, there is no firm evidence that the association between learning disorder and psychiatric problems is greater in children who have neurological abnormalities. Indeed, the relationship may actually be weaker in neurologically impaired children when schooling is appropriate (Seidel, Chadwick, & Rutter, 1975). Finally, the notion of a characteristic response to stimulants seems unlikely in the light of the work by Rapoport and her colleagues (1978), who have found that improved cognitive performance and diminished motor activity can be produced by amphetamine in nonbrain-damaged and nondeviant children, i.e., that any favorable drug response seen in hyperactive children is a nonspecific phenomenon.

The issue of how often the components of the syndrome go together has been studied in both clinic patients (Routh & Roberts, 1972; Werry, Minde, Guzman, Weiss, Dogan, & Hoy, 1972) and unselected subjects (Nichols & Chen, 1981). These investigations show low correlations among measures of hyperactivity, learning disorder; and neurological abnormality, indicating that these are not commonly found in the same individual. The failure of MBD components to occur together is also suggested by some experiments. Rapoport in a study of the effects of caffeine (Rapoport, Buchsbaum, Weingartner, Zahn, Ludlow, Bartko, Mikkelsen, Langer, & Bunny, 1980) has observed that the stimulant may result in a simultaneous increase in concentration and activity in the same individual.

Taken together, these findings argue strongly against the existence of an MBD syndrome characterized by overactivity and inattention, cognitive difficulties, and some marker of neurological abnormality. Perhaps the notion of MBD as a syndrome is incorrect, and that it is better looked at as an etiological agent in which the forms of psychiatric disorder are varied and shaped by environment and temperament, or perhaps there is a different MBD syndrome with different

associated behaviors. If this is a disorder of brain function, the present study, may be a more useful approach to research in the field. It is held to be indicative of brain damage (or success) their relationship to an

In the present study, neurological signs of minor neurological dysfunction. Cognitive and psychiatric disturbances were elicited and could thus serve as a marker for a later psychiatric disorder and research.

The neurological signs included hyperactive type, motor abnormalities such as diadochokinesis and general clumsiness, in particular, astereognosis.

This group of signs is called "soft signs" and is reliably elicited (Rutter et al., 1970). It is not reliably identified neuropathology in origin rather than being inherited. Only a minority of children with MBD (Meidermayer, & Richardson, 1970) who have neurological disease have soft signs. This is assumed from cross-sectional studies (Rutter et al., 1978; Mikkelsen, Brown, Minde, Romine, & Dykman, 1974) than from longitudinal groups, rather than from longitudinal studies.

The relationship of soft signs to MBD is established in a number of studies (Rutter et al., 1973; Myklebust, 1973; Peters, Romine, & Minde, 1966; Stine, Saratsiotis, & Mosteller, 1970). Studies have examined the relationship of soft signs in younger children. Among these studies in male child psychiatric patients (Rutter et al., 1970; Wikler, Dixon, & Parker, 1970) soft signs are more common in those with MBD than in those who are not.

Soft signs have been shown to be associated with MBD and adults by Hertzog and Birch (1970), Bucker, and Harrow (1970), and others. They have reported higher rates of soft signs in psychiatric patients than among controls, but this is confounded by the inclusion of

associated behaviors. If this is so, then identifying a marker of neurological dysfunction and proceeding to seek its behavioral correlates, as has been done in the present study, may be a more appropriate approach to the question "can minor degrees of brain dysfunction influence behavior?" than the traditional approach to research in the field that has consisted of (1) identifying behaviors held to be indicative of brain damage and (2) attempting to establish (without success) their relationship to an index of neurological disorder.

In the present study, neurological soft signs have been chosen as the index of minor neurological dysfunction. There is evidence that they are related to cognitive and psychiatric disturbance, and they can be readily and inexpensively elicited and could thus serve as an accessible marker both for the prediction of later psychiatric disorder and research into the etiology of the dysfunction itself.

The neurological signs include involuntary movements of a choreic or athetoid type, motor abnormalities such as synkinesis (mirror movements), dysdiadochokinesis and general clumsiness, as well as sensory integrative abnormalities, in particular, astereognosis and dysgraphesthesia.

This group of signs is called "soft" not because they cannot be readily and reliably elicited (Rutter et al., 1970; Shaffer, 1978) but rather because they have no reliably identified neuropathological locus and are held to be "developmental" in origin rather than being indicative of any fixed disorder or abnormality. Only a minority of children with soft signs have abnormal EEG's (Capute, Meidermayer, & Richardson, 1968), and by no means all children with neurological disease have soft signs. Their "developmental" nature has been assumed from cross-sectional studies (e.g., Camp, Bialer, Sverd, & Winsberg, 1978; Mikkelsen, Brown, Minichiello, Millican, & Rapoport, 1982; Peters, Romine, & Dykman, 1974) that have found a lower prevalence in older age groups, rather than from longitudinal studies.

The relationship of soft signs to learning problems in young children has been established in a number of studies (Adams, Kocsis, & Estes, 1974; Boshes & Myklebust, 1973; Peters, Romine, & Dykman, 1975; Rutter, Graham, & Birch, 1966; Stine, Saratsiotis, & Mosser, 1975; Wolff & Hurwitz, 1973). Several studies have examined the relationship between neurological signs and behavior in younger children. Among these it has been found that signs are more prevalent in male child psychiatric patients than in normal controls (Peters et al., 1975; Wikler, Dixon, & Parker, 1970); and within a group of disturbed children, soft signs are more common in those who are impulsive, distractable, dependent, and sloppy than in those who are not (Paulsen, 1978; Paulsen & O'Donnell, 1979).

Soft signs have been shown to be related to psychiatric disorder in adolescents and adults by Hertzog and Birch (1966), Hertzog et al. (1968), Rochford, Detre, Bucker, and Harrow (1970), and Mosher, Pollin, and Stabanau (1971), all of whom reported higher rates of soft signs among hospitalized or diagnosed psychiatric patients than among controls; however, the results of those studies were confounded by the inclusion of subjects on medication, by the questionable

blindness of the raters, and/or by the inclusion of patients with frank neurological disease. A methodologically satisfactory investigation in adults has been carried out by Quitkin and his colleagues (1976). Two patient groups, schizophrenics with a history of childhood asociality and individuals with "emotionally unstable character disorders," had more soft signs and were more likely to have a history of perinatal morbidity than patients in other diagnostic categories. In all these studies the subjects studied belonged to clinically identified groups. As a result, relative risk for psychiatric problems accruing from soft signs could not be assessed, and the comprehensiveness and generalizability of the findings is likely to be limited. This is so because studies on inpatients (e.g., Hertzog & Birch, 1968, Mosher et al., 1971, Quitkin et al., 1976, and Rochford et al., 1970) may not reveal a relationship between soft sign and a type of psychiatric disorder that does not usually lead to hospital admission. Studies on specialized clinical or institutional populations such as children with hyperactivity or delinquents (Camp et al., 1978; Lewis, Shanok, Pincus, & Glaser, 1979; Lucas, Rodin, & Simson, 1965; McMahon & Greenberg, 1977; Paulsen, 1978; Paulsen & O'Donnell, 1979; Wikler et al., 1979) may not reveal a relationship with other psychiatric conditions, and referral bias in such studies may lead to unrepresentative findings. The few studies in nonreferred populations have been limited to the examination of a single neurological sign (Rutter et al., 1966; and Wolff & Hurwitz, 1966, studied the choreiform syndrome), did not examine behavioral or emotional variables (Adams et al., 1974), or examined only a limited set of behavioral variables so that a full psychiatric diagnosis could not be made (Wolff & Hurwitz, 1973).

The present study examined the relationship between minor neurological signs in childhood and both psychiatric and cognitive disorders in adolescence. Our sample was drawn from the Collaborative Perinatal Project, which has the advantage of contemporaneous documentation of early neurological and psychological measures and of having been initially unselected for either the dependent or independent variables of interest to us. Furthermore, as it is a longitudinal study, we can examine both the predictive strength of soft signs for *later* psychiatric and cognitive problems, and we can examine the issue of developmental changes in soft signs in a longitudinal sample.

The research described in this chapter sets out to examine the following specific issues: first, whether soft neurological signs, as measured at age 7, persist into adolescence, and second, whether a relationship exists between the presence of early signs and later psychiatric diagnosis (at age 17).

## METHODS

Subjects ( $N = 126$ ) were drawn from the Columbia Presbyterian Hospital chapter of the Collaborative Perinatal Project (CPP). The CPP, a prospective study coordinated by the National Institute of Neurological and Communicative Disor-

ders and Stroke (NINCDS), was a study of perinatal mortality, stillbirth, and infant mortality (Berendes, 1966; Stokman, and Shafer (in press)). The study was clearly independent of the current study, and to describe the sample would be generalizable to similar studies. The Columbia Presbyterian Hospital sample of approximately 55,000 mothers and their children were repeatedly tested or questioned for neurological, neurological, general health, and behaviorally recorded.

Data collection for the original study was from the mother to the participating medical center at the child's seventh year.

The Columbia Presbyterian sample was drawn from a perinatal clinic between January 1959 and December 1966. The adoption donors and women who were not adopted. The ratio of participants to the total population that the average sampling ratio was 1:100. The sample of registrants (Niswander & Gordon, 1969) was 2% of the time of the 7-year examination, 2% of the total population. The Columbia sample was small, and the other centers (generally 70–80%

### *Criteria for Inclusion for Current Study*

English-speaking male children born in the Columbia sample, who were in school at the time of follow-up, and who were available for and facilitating subject tracing system.

Experimental subjects were selected for neurological examination. A positive neurological signs, broadly grouped into motor, coordination, and sensorimotor (listing) qualified the subject for inclusion. The subject was then matched for sex, race, and neurological signs on the 7-year

### Psychiatric Evaluation

This report is confined to data obtained from the current study. A semistructured interview was conducted with the adolescent and to explore the range

ders and Stroke (NINCDS), was initiated for the purpose of investigating pregnancy wastage (Berendes, 1966) and is described in detail in O'Connor, Shaffer, Stokman, and Shafer (in press). The intent of the CPP was to select a sample clearly independent of the outcome variables of interest, i.e., pregnancy wastage, and to describe the sample sufficiently thoroughly so that findings would be generalizable to similar populations. Fourteen medical centers, including Columbia Presbyterian Hospital, participated in the CPP, yielding data on approximately 55,000 mothers and their offspring. Both mothers and children were repeatedly tested or questioned and data covering a wide range of psychological, neurological, general health, and demographic variables were systematically recorded.

Data collection for the original study began with the initial prenatal visit of the mother to the participating medical center and continued at most centers through the child's seventh year.

The Columbia Presbyterian sample consisted of women admitted to the prenatal clinic between January 1959 and April 1963, with the exception of declared adoption donors and women who had received no more than one prenatal examination. The ratio of participants to registrants was initially 1:6, later reduced so that the average sampling ratio was 1:4.4, with a total of 2235 prenatal clinic registrants (Niswander & Gordon, 1972), of which 2067 were live births. At the time of the 7-year examination, 2019 children had survived, and 83.5% of them attended for reexamination (Gates, 1973). Overall, the loss and attrition rate for the Columbia sample was small, and these rates compare favorably with loss at other centers (generally 70–80% examined, Niswander & Gordon, 1972).

*Criteria for Inclusion for Current Study.* This study is a follow-up of black, English-speaking male children born in 1962 and 1963. This cohort, the youngest in the Columbia sample, was chosen because the children would still be in school at the time of follow-up, allowing us to obtain teachers' ratings of behavior and facilitating subject tracing, which could be done through the school system.

Experimental subjects were selected on the basis of the findings at the 7-year neurological examination. A positive rating for any one of 18 specific neurological signs, broadly grouped within the constructs of involuntary movements, coordination, and sensory integration (see Table 2.1 for a complete listing) qualified the subject for inclusion ( $N = 63$ ). A similar number of controls was then matched for sex, race, closest birth date, and the absence of these neurological signs on the 7-year neurological examination.

### Psychiatric Evaluation

This report is confined to data obtained from the direct evaluation of the adolescent. A semistructured interview was used to assess the current status of the adolescent and to explore the range and adequacy of current social relationships.

TABLE 2.1  
Frequency of Neurological Soft Signs Rated Present  
at Age 7 Examination

<i>Involuntary Movements</i>	<i>N</i>	<i>Coordination</i>	<i>N</i>	<i>Sensory Integration</i>	<i>N</i>
Spontaneous tremor	1	Dysdiadochokinesis	35	Astereognosis	9
Tic	1	Awkwardness not otherwise classi- fied	12		
Mirror movements	13				

The interviewers, as well as the cognitive and neurological testers, were blind with respect to subject status. The presence and severity of somatic, anxiety, affective and psychotic symptoms, antisocial behavior, and delinquency were assessed, and detailed information was obtained about peer and sexual relationships, and about the quality and quantity of family relationships.

The interview includes portions of the Schedule for Affective Disease and Schizophrenia (SADS) (Spitzer & Endicott, 1977), which allowed us to determine whether Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978) for affective and psychotic disorders had been met. Sections of the interview developed by Rutter and Graham (1968) cover the adolescent's social relationships and antisocial behavior. Other sections dealing with assessment of sexual behavior and substance and alcohol use were adapted from the SADS (Spitzer & Endicott, 1977) and from forms developed by Kandel, Singer, and Kessler (1976). Ratings of the adolescent's social behavior during the interview have been adapted from the Mental Health Assessment Form (Kestenbaum & Bird, 1978) and include ratings of motor activity, interaction with the interviewer, and emotional responsiveness.

Glossary descriptions and rating scales with defined anchor points were incorporated into the body of the interview.

Overall functioning has been rated on the Global Assessment Scale (GAS) (Endicott, Spitzer, Fleiss, & Cohen, 1976). A psychiatric diagnosis was assigned for cases where the GAS rating was 70 or less. A GAS score ranging between 61 and 70 is defined on the scale as: some mild symptoms (e.g., depressive mood and mild insomnia or some difficulty in several areas of functioning, but generally functioning pretty well, has some meaningful interpersonal relationships and most untrained people would not consider him "sick").

*Assessment of Reliability.* Intrarater reliability and intrasubject or interrater reliability were assessed. Sessions were conducted at which the interviewers watched and rated videotapes of their respective interviews. All interviewers watched interviewer X with subject A to obtain ratings on the measure used. This procedure constituted the framework for the assessment of reliability (by providing intrasubject and intrarater measures) and furnished additional training.

The GAS rating is a single indicator of rate of agreement. continuum from 0 to 100 with differential evaluations within viously, is clearly defined. Th reliability are: Do raters agree rating that is given?; do raters (a atric diagnosis (GAS rating of 7

Nine subjects were rated by with a mean percentage agreee categorical placement); howev ratings may differ by only one categories. With the interview's ment increased to 59% for all so 88.5% for all scores within 10

The second question concern Nine subjects were scored by fo 79% rate of agreement on the p score of 70.

*Neurological Assessment.* fully elsewhere (Shafer, Stokm preparation) and was designed subjects in ways broadly simila significance of the constituents micity of a particular motor tas allow for a more exact determ sign and to obtain a greater ra

Our criteria for selecting an the sign had been present in a were present in more than three been described with sufficient our current protocol—one of wardness," did not fulfill this

Three early signs that fulfil present report are described in

*Astereognosis.* As a meas was required to identify, with a nickle, and a button. At age dime-sized washer, and a penn testing to ensure familiarity v presented once and a single pos one of the three items. At age

The GAS rating is a single summary measure that could be used as an indicator of rate of agreement. The rating scale consists of 10 categories on a continuum from 0 to 100 with a continuum nested in each category to permit differential evaluations within a category. Each category, as indicated previously, is clearly defined. Thus, the questions relevant for the purposes of reliability are: Do raters agree with the interviewer on the category of the GAS rating that is given?; do raters (and interviewer) agree on who warrants a psychiatric diagnosis (GAS rating of 70 or less)?

Nine subjects were rated by three to six raters, yielding a total of 41 ratings, with a mean percentage agreement of 38% (rater agreeing with interviewer's categorical placement); however, such a figure may be misleading, because ratings may differ by only one or two points and yet be placed in different categories. With the interview's rating as an index, the mean percentage agreement increased to 59% for all scores within five points of the index rating and to 88.5% for all scores within 10 points of the index rating.

The second question concerns the rate of agreement on the cutting point of 70. Nine subjects were scored by four to seven raters (including index rating), with a 79% rate of agreement on the placement of a subject above or below the GAS score of 70.

*Neurological Assessment.* The neurological assessment has been described fully elsewhere (Shafer, Stokman, Shaffer, Schonfeld, O'Connor, & Wolfe, in preparation) and was designed both to reevaluate the neurological status of the subjects in ways broadly similar to those used at age 7, and also to identify the significance of the constituents of the particular signs, e.g., speed, errors, rhythmicity of a particular motor task. Ratings in the current study were designed to allow for a more exact determination of the components of each neurological sign and to obtain a greater range of responses.

Our criteria for selecting an age-7 sign to examine persistence were: (1) that the sign had been present in a reasonable number of children—only four signs were present in more than three children (see Table 2.1); and (2) that the sign had been described with sufficient clarity to enable us to replicate the examination in our current protocol—one of the four frequently present at age 7, "awkwardness," did not fulfill this criterion.

Three early signs that fulfill these criteria and that are the subject of the present report are described in more detail.

*Astereognosis.* As a measure of sensory integration, at age 7, the subject was required to identify, with eyes closed and using only one hand, a bottle cap, a nickle, and a button. At age 17, the items were a quarter, a nickel, a penny, a dime-sized washer, and a penny-sized button. Subjects named each item before testing to ensure familiarity with the objects. At age 7, each item had been presented once and a single positive rating had been given for failure to name any one of the three items. At age 17, each item was presented twice and a positive



rating was given for each failure on 10 presentations. Scoring at 17 may be either dichotomous, one or more errors versus no errors, or continuous, as the sum of all items.

Dysgraphesthesia, an alternative measure of sensory integration, was assessed only at age 17. The subject was asked to identify (with eyes closed) a square, a circle, an "X," and the numeral "3," all 1 inch in height, each drawn with a stylus on the palm of the subject's hand. The figures were presented randomly, each one appearing twice. Subjects were asked to name the stimuli before testing, and dysgraphesthesia was considered present if the subject failed to name correctly one or more of the eight stimuli presented.

*Mirror Movements.* At age 7, the subject had been asked to perform rapid thumb-forefinger apposition, first with the right, then the left hand. A positive rating had been given if the opposite hand (first the left, then the right) showed similar involuntary movements. At age 17, the testing was expanded to successive rapid thumb-finger apposition for a period of 10 seconds. Finger movements in the opposite hand (order of hands determined by a coin toss) were scored as none, one to six movements, more than six movements within the 10-second period.

*Dysdiadochokinesis.* At age 7 and 17, subjects were asked, as a measure of coordination, to alternately pronate and supinate each hand as fast as possible, with the further instruction at 17 that the hand should be lifted up and rotated about the axis of the middle finger, starting with the right hand. Difficulty in executing the rapid alternating movements yielded a positive rating for this sign at age 7. At 17, both irregularity in rhythm and failure to alternate were assessed with scoring for each on a 3-point scale: regular, perceptibly irregular, and grossly irregular, for irregularity in rhythm, and no failure, one failure, and two or more failures, for failure to alternate.

For the analysis in this chapter, signs measured at 17, where possible, were rescored on a present/absent dichotomy to match the dichotomy in the age 7 scoring.

The order of items in the neurological examination was the same for all subjects with the exception that the presentation of certain rapid movement items was counterbalanced.

*Assessment of Reliability and Validity.* The assessment of the reliability of the neurological examination involved three groups other than the study population: (1) 10 adolescent inpatients at a nearby psychiatric center, (2) young adult research staff, and (3) 15 normal adolescents. The 10 inpatients were given parts of the examination twice in a 4-week interval. Simultaneous ratings were obtained by two or three of the neurological testers. Inter and intrarater com-

parisons were made. Rank correlations revealed a high degree of reliability for positive ratings (retest  $r_s = .77$ ) and retest  $r_s = .93$ , for rater 1:  $r_s = .92$  and rater 2:  $r_s = .77$  (analysis).

### Cognitive Assessment

Although a broad battery of cognitive tests (O'Connor et al, in press), this chapter's IQ was measured with the Wechsler Adult Intelligence Scale (WAIS).

### Study Procedures

*Tracing.* Of the 126 designees, 100 were traced through procedures utilizing information from newspaper address and phone number, date of birth, and attending at age 7, and any other identifying information. Parents' addresses and phone numbers were retrieved for subjects whose names were not found in the telephone directory.

Of the 123 identified subjects, 100 were located in the metropolitan area (to southeastern states), 23 were incarcerated, and one was deceased.

*Testing.* The order of testing was determined by the WAIS first, followed by the psychiatric interview. Each parent was contacted as close as possible to the day of the testing.

When subjects were seen out of state, the scheduling order was, of necessity, determined by travel schedules.

Source of

---

Located through phone  
 Located through early  
 Located through friends  
 Located by other means  
 Not located  
 Total

---

parisons were made. Rank correlation coefficients ( $r_s$ ) for total number of positive ratings revealed a high degree of agreement between raters for both test ( $r_s = .77$ ) and retest ( $r_s = .93$ ), as well as intrarater test-retest reliability (rater 1:  $r_s = .92$  and rater 2:  $r_s = .77$ ; rater 3 did not test enough subjects for the analysis).

### Cognitive Assessment

Although a broad battery of cognitive tests was given (described in detail in O'Connor et al, in press), this chapter reports only on IQ test results. Full-scale IQ was measured with the Wechsler Adult Intelligence Scale (Wechsler, 1955).

### Study Procedures

*Tracing.* Of the 126 designated subjects, 123 were found. The retrieval procedures utilized information from the original files, such as last-known address and phone number, date of birth of adolescent, name of school subject was attending at age 7, and any other potentially useful information, such as grandparents' addresses and phone numbers. The sources of information that led to the retrieval of subjects are shown in Table 2.2.

Of the 123 identified subjects, 14 had moved out of the New York metropolitan area (to southeastern states, Colorado, and California), four adolescents were incarcerated, and one was in a psychiatric institution.

*Testing.* The order of testing was partially counterbalanced. All subjects were given the WAIS first, followed by either the neurological assessment or the psychiatric interview. Each parent informant was interviewed on the same day or as close as possible to the day that his/her son or stepson was seen.

When subjects were seen out of town, whether in their homes or in prison, the scheduling order was, of necessity, more flexible, dependent on prison and/or travel schedules.

TABLE 2.2  
Source of Information for Retrieval

	<i>N</i>	(%)
Located through phone book	51	(41%)
Located through early school data	32	(25%)
Located through friends or relations	30	(24%)
Located by other means	10	( 8%)
Not located	3	( 2%)
Total	126	

RESULTS

Social and Family Characteristics

The two groups, early signs present (ESP) and early signs absent (ESA), were compared, at age 17, on the following social and family characteristics: anomalous family composition, mother's educational achievement, dissatisfaction with housing, and ever having been on welfare (Table 2.3). No significant differences among the groups were found.

Persistence of Signs

Of the three signs tested for persistence from age 7 to adolescence, the two motor signs, dysdiadochokinesis and mirror movements, showed significant stability (Table 2.4). More than half the ESP subjects manifested the abnormal sign in adolescence. Both dysgraphesthesia and failure to alternate hands (dysdiadochokinesis) occurred among adolescents who had comparable signs at 7 at more than twice the prevalence rates found among adolescents who were free of signs at age 7.

The finding of persistence did not apply to astereognosis where a high proportion of adolescents in both the ESP and ESA groups misidentified objects. The technique used to elicit astereognosis at 17 was more difficult than that used at age 7. Thus the finding that dysgraphesthesia, a broadly comparable index of sensory integration, differentiated between the two groups (see Table 2.4), suggests that the instability of astereognosis may have been artifactual.

TABLE 2.3  
Demographic Characteristics by Sign Group (in percents)

	Signs at 7			
	Present		Absent	
	%	of n	%	of n
<i>Family composition</i>				
Parents separated, divorced, never married, or widowed	58%	60	56%	55
<i>Education of Mother</i>				
Incomplete high school or less	26%	61	20%	55
<i>Housing</i>				
Moderate or severe dissatisfaction	32%	59	27%	55
<i>Welfare</i>				
Ever on welfare	49%	61	44%	55

Persistence of Signs

Age 17

Irregularity (positive rating)  
Failure to alternate (positive rating)

Age 17

Astereognosis (positive rating)  
Dysgraphesthesia

Age 17

Mirror movements  
None  
1-6 per 10 seconds  
7+ per 10 seconds

Early Signs and Later Psych

Thirty-seven adolescents were g  
obtained from the adolescent int  
the *Diagnostic and Statistical M*  
tion. To reduce the number of  
analysis, five categories were es  
tive/emotional, psychotic, misc  
subjects given a diagnosis did r  
more of the adolescents with

TABLE 2.4  
Persistence of Signs from Age 7 to Age 17 (in percents)

A) Dysdiadochokinesis				
Age 7				
Age 17	Sign		$\chi^2$	<i>p</i>
	Present (N = 32)	No Signs (N = 55)		
Irregularity (positive rating)	53%	39%	NS	—
Failure to alternate (positive rating)	55%	26%	7.22	.01

  

B) Astereognosis				
Age 7				
Age 17	Sign		$\chi^2$	<i>p</i>
	Present (N = 8)	No Signs (N = 55)		
Astereognosis (positive rating)	62%	54%	NS	—
Dysgraphesthesia	57%	27%	6.56	.01

  

C) Mirror Movement				
Age 7				
Age 17	Sign		$\chi^2$	<i>p</i>
	Present (N = 12)	No Signs (N = 55)		
Mirror movements				
None	—	20%		
1-6 per 10 seconds	33%	47%		
7 + per 10 seconds	67%	33%		
			5.09	.03

### Early Signs and Later Psychiatric Disturbance

Thirty-seven adolescents were given a psychiatric diagnosis on the basis of data obtained from the adolescent interview. The diagnostic criteria were taken from the *Diagnostic and Statistical Manual III* of the American Psychiatric Association. To reduce the number of diagnoses to a manageable set of categories for analysis, five categories were established a priori. These were antisocial, affective/emotional, psychotic, miscellaneous, and no disorder. The proportion of subjects given a diagnosis did not differ by sign group; however, significantly more of the adolescents with early signs fulfilled criteria for an affec-

TABLE 2.5  
Mean IQ by Psychiatric Diagnosis by Early Sign  
Status

	Sign at 7	
	Present (N)	Absent (N)
No diagnosis	88.86 (39)	95.45 (40)
Antisocial	92.00 (6)	89.75 (8)
Affective/emotional	86.25 (12)	98.30 (3)
Other diagnosis (Including 1 schizo.)	76.50 (4)	91.25 (4)
$\bar{x}$	87.8	94.5

tive/emotional disorder during the 3 months prior to the assessment ( $X^2 = 5.4$ ,  $df = 1, p < .05$ ).

A variety of studies have revealed a relationship between soft signs on one hand and intellectual deficits and learning difficulties on the other. One possibility for our finding of a high prevalence of affective/emotional disorders in the ESP children would be that they had experienced scholastic difficulties and that the disorders were a consequence of persistent failure in school. An analysis of mean IQ differences by sign group and by psychiatric diagnostic group was carried out (Table 2.5). A two-way analysis of variance for unequal cell size (Edwards, 1975) was performed to examine the effects of neurological status and psychiatric diagnosis group on IQ. The results show a significant main effect of neurological status, such that the ESP group had a significantly lower mean IQ than the ESA group ( $F = 6.21$ ,  $df = 1, 107$ ,  $p < .02$ ). Psychiatric diagnosis, as a main effect, and the interaction were not significant. It is noted that the ESP subjects diagnosed as having an affective/emotional disorder had a lower mean IQ than the ESA subjects within the same diagnostic category.

## DISCUSSION

### Persistence of Soft Signs

At age 17 two motor signs, mirror movements and dysdiadochokinesis, were found in more than half the subjects known to have had the respective signs at age 7. These rates were significantly higher than rates found within the group of subjects who were sign free at age 7.

The persistence of a third sign, astereognosis, was not supported. At age 17 a high proportion of all subjects performed poorly on this task, and no significant performance differences between subjects with and without signs at age 7 were found. The explanation for this is not clear, although it may have been a result of

inappropriate measurement. The was, in two respects, altered for similarity were chosen and a larg tion. These changes were designe felt that identification of the obje less of a perceptual challenge at to have been equally difficult for regardless of their neurological them. Inappropriate measurement finding of a relationship between suggesting that sensory integrati cussed earlier, persist through cl

A second finding was of a hig control group. There are at least is that there were differences in t that a significant proportion of ch had signs would have been regist age 17. It should be noted howe the Columbia chapter of the CPP likely that many false negative surement error at Time 2 has bee retest reliability of neurological s able ranges.

The second explanation of th measurement or recording techn descriptions given of the items sent/absent were not as detaile each of the signs were measure positive recording of a sign at different dimensions, although positive recording of a sign at that might not have qualified t examination.

A third possible explanation quired since age 7, and thus sign the impression of the authors th smoked marijuana and this may this sort will be examined in fi proached by examining whether tween signs and disorder for su since age 7 and for subjects w Different relationships within th signs are different.

inappropriate measurement. The task as it had originally been applied at age 7 was, in two respects, altered for use in the adolescent sample. Objects of greater similarity were chosen and a larger number of items was targeted for identification. These changes were designed to make the task more difficult because it was felt that identification of the objects from the age 7 examination would present less of a perceptual challenge at age 17. However, the reformulated task appears to have been equally difficult for both groups, and a high proportion of subjects, regardless of their neurological status, misidentified the objects presented to them. Inappropriate measurement as an explanation is further supported by the finding of a relationship between early astereognosis and later dysgraphesthesia, suggesting that sensory integration difficulty, as well as the motor signs discussed earlier, persist through childhood.

A second finding was of a high base rate of neurological signs at age 17 in the control group. There are at least three competing explanations for this. The first is that there were differences in threshold at Time 1 and Time 2. Thus, it may be that a significant proportion of children who at age 7 were recorded as not having had signs would have been registered as sign positive using the criteria adapted at age 17. It should be noted however that the prevalence of neurological signs in the Columbia chapter of the CPP was higher than at other centers, making it less likely that many false negative assignments were made. The problem of measurement error at Time 2 has been examined through a separate study of the test-retest reliability of neurological signs. Reliability across time was within acceptable ranges.

The second explanation of the high base rate in the controls could be that measurement or recording techniques at ages 7 and 17 are not comparable. The descriptions given of the items measured at 7 with a simple rating of present/absent were not as detailed as those at 17, where several components of each of the signs were measured separately. The result may have been that the positive recording of a sign at age 7 required the presence of a number of different dimensions, although these were not explicitly defined, whereas the positive recording of a sign at age 17 required the presence of a single dimension that might not have qualified the subject for a positive rating in the earlier examination.

A third possible explanation is that neurological dysfunction has been acquired since age 7, and thus signs were acquired *de novo* after the age of 7. It is the impression of the authors that a high proportion of the subjects frequently smoked marijuana and this may have influenced neurological states. Factors of this sort will be examined in further analyses. This problem will also be approached by examining whether there are different patterns of relationship between signs and disorder for subjects who have had neurological signs present since age 7 and for subjects who were noted only to have signs at age 17. Different relationships within these groups would suggest that the origins of the signs are different.

Regardless of the explanation for these phenomena, the implications are clear, i.e., soft signs persist well after most features of neurological development have been completed. They must be regarded as being potentially stable indications of a neurological difference. Whether the differences matter is of course a function of their correlates.

### Correlates of Early Signs—Later Psychiatric Disorder

A second main finding suggests that early signs do indeed matter. Significantly more of the adolescents who had had soft signs in early childhood had recently experienced a depressive episode. Several explanations suggest themselves for this finding.

The first is that despite statistical significance, we have a chance finding. This is always a possibility in a study with small numbers, and it makes some form of replication essential. The data upon which the present chapter is based are derived solely from the psychiatric interview with the adolescent and do not take into account parents' and teachers' reports. Although there is evidence that information about emotional state derived from a direct interview with the adolescents is likely to be more complete and accurate (Carlson & Cantwell, 1980) than that from parents, the available secondary sources will make it possible to test the consistency of the findings. However, it must be further noted that, given the limited congruence between adolescents' and parents' reports of psychiatric disorder in other studies (Gould, Wunsch-Hitzig, & Dohrenwend, 1980), a failure to obtain internal replication would be of only limited value.

A second and more complex explanation might be that, rather than indicating a relationship between an early neurological marker and later psychiatric disorder, the findings indicate continuity of affective psychiatric disorder; that is, there might be an interaction between either the recording or the manifestation of soft signs and psychiatric disorder that has led to systematic bias. It may be that common neurological phenomena, such as the ones studied here, are more likely to be rated present if they are accompanied by some other disturbance of behavior manifest during the examination. Thus seven-year-olds with both signs and disturbed behavior at examination might be reported as having signs at a higher rate and with more consistency than children who have signs but who show normal behavior during examination. Alternatively, it might be that neurological soft signs are latent in many children and simply more likely to emerge under conditions of stress. In this case, children with early disturbance would be more likely to show stress during physical examination and would therefore be more likely to be rated positive for neurological change.

There are a number of strategies open to examine the questions. These include an examination of the psychiatric correlates of *current* signs in both groups and an examination of the subsequent relationships of the ratings made of behavior during testing. These are pursued as our data analysis continues.

A third possibility might be that, as has been noted, i.e., that there is so much overlap between soft signs and to both signs and to a disturbance of neurological dysfunction and affective disorder, there is convincing evidence of a relationship. Bijur, Chadwick, & Rutter, submit that the emphasis in most of the research on dysfunction, the understanding of that the possibility of a broader

Finally, although the relationship between there is a suggestion that the depression means IQ's (see Table 2.5). An alternative interpretation. On the one hand, the extent of brain dysfunction is likely to be found in individuals with a psychiatric disorder and that the dysfunction. Children with low IQ have more school difficulties and Affective/emotional disorders were an instance but rather would be an example of the low-IQ group. Albeit with some because there is a suggestion that soft sign children is low relative to were correct, it would be expected sign status.

Regardless of the mechanisms that suggest the neurological examination potential value in the prediction of disorder. This alone is important

### ACKNOWLEDGMENTS

This research is being supported by Psychiatric Education Grant #MH

The authors wish to express their appreciation to the children who participated in the cognitive testing

Adams, R. M., Kocsis, J. J., & Estes, J. L. (1966). The structure of children and controls. *American Journal of Orthopsychiatry*, 36, 1-10.  
Berendes, H. W. (1966). The structure of

A third possibility might be that a true minimal brain dysfunction effect is being noted, i.e., that there is some ill-defined brain dysfunction that is leading to both signs and to a disturbance of affect. The relationship between neurological dysfunction and affective disorder has been reviewed extensively and there is convincing evidence of its importance (see Flor-Henry, 1979; Shaffer, Bijur, Chadwick, & Rutter, submitted for publication; Wexler, 1980). Although the emphasis in most of the research has been on effects of localized cortical dysfunction, the understanding of the anatomical basis for soft signs is so limited that the possibility of a broader relationship cannot be excluded.

Finally, although the relationship between diagnosis and IQ is not significant, there is a suggestion that the depressed and "other" soft signs groups have lower means IQ's (see Table 2.5). An observation of this sort lends itself to at least two interpretations. On the one hand, it might be that low IQ is simply a marker of the extent of brain dysfunction and that affective/emotional disorder is more likely to be found in individuals with more pervasive involvement. The trend could also be interpreted as indicating that soft signs are only indirectly related to psychiatric disorder and that the primary relationship is with the cognitive dysfunction. Children with low IQ scores, regardless of their soft sign status, may have more school difficulties and more difficulties with the stresses of life. Affective/emotional disorders would not be constitutionally determined in this instance but rather would be an expected consequence of the life experiences of the low-IQ group. Albeit with small numbers, the results here do not support this because there is a suggestion that the mean IQ of the affective/emotional disorder soft sign children is low relative to their sign-free counterparts. If this model were correct, it would be expected that the low IQ would apply regardless of soft sign status.

Regardless of the mechanisms that might explain the relationship, the findings suggest the neurological examination of the 7-year-olds yields information of potential value in the prediction of later adjustment difficulties and psychiatric disorder. This alone is important in its implications for preventive psychiatry.

#### ACKNOWLEDGMENTS

This research is being supported by NIMH Center Grant #MH 30906 and by NIMH Psychiatric Education Grant #MH 07715-17.

The authors wish to express their gratitude to Dr. Stephanie Portnoy, who substantially participated in the cognitive testing.

#### REFERENCES

- Adams, R. M., Kocsis, J. J., & Estes, R. E. (1974). Soft neurological signs in learning-disabled children and controls. *American Journal of Diseases of Children*, 128, 614-618.
- Berendes, H. W. (1966). The structure and scope of the Collaborative Project on cerebral palsy,



- mental retardation, and other neurological and sensory disorders of infancy and childhood. In S. S. Chipman, A. M. Lilienfeld, B. G. Greenberg, & J. F. Donnelly (Eds.), *Research methodology and needs in perinatal studies*. Springfield, IL: Charles C. Thomas.
- Boshes, B., & Myklebust, H. R. (1964). A neurological and behavioral study of children with learning disorders. *Neurology*, *14*, 7-13.
- Camp, J. A., Bialer, I., Sverd, J., & Winsberg, B. G. (1978). Clinical usefulness of the NIMH physical and neurological examination for soft signs. *Am J Psychiatry*, *135*(3), 362-364.
- Campbell, S. B., Endman, M., & Bernfeld, G. (1977). A 3-year follow-up of hyperactive children in elementary school. *Journal of Child Psychology and Psychiatry*, *18*, 239-249.
- Capute, A. J., Meidermayer, E. F., & Richardson, F. (1968). The electroencephalogram in children with minimal cerebral dysfunction. *Pediatrics*, *41*, 1104-1114.
- Carlson, G. A., & Cantwell, D. P. (1980). *Diagnosis of childhood depression—a comparison of the Weinberg and DSM III criteria*. Paper presented at the annual meeting of the American Psychiatric Association, San Francisco.
- Edwards, A. L. (1975). *Experimental design in psychological research*. New York: Holt, Rinehart, & Winston.
- Endicott, J. S., Spitzer, R. L., Fleiss, J., & Cohen, J. (1976). The Global Assessment Scale. *Archives of General Psychiatry*, *33*, 766-771.
- Flor-Henry, P. (1979). Neuropsychological and power spectral EEG investigation of the obsessive-compulsive syndrome. *Biological Psychiatry*, *14*(1), 119-130.
- Gates, M. J. (1973). *Final report: Collaborative Perinatal Study of National Institute of Neurological Diseases and Stroke*. New York: Columbia University College of Physicians and Surgeons.
- Gould, M. S., Wunsch-Hitzig, R., Dohrenwend, B. P. (1980) Formulation of hypotheses about the prevalence, treatment and prognostic significance of psychiatric disorders in children in the United States. In B. P. Dohrenwend, B. S. Dohrenwend, M. S. Gould, B. Link, R. Neugebauer, & R. Wunsch-Hitzig (Eds.), *Mental illness in the United States: Epidemiological estimates*. New York: Praeter Publications.
- Hertzog, M. A., & Birch, H. G. (1966). Neurologic organization in psychiatrically disturbed adolescent girls. *Archives of General Psychiatry*, *15*, 590-599.
- Hertzog, M. A., & Birch, H. G. (1968). Neurologic organization in psychiatrically disturbed adolescents. *Archives of General Psychiatry*, *66*, 43-53.
- Kandel, D., Singer, E., & Kessler, R. (1976). The epidemiology of drug use among New York State high school students: Distribution, trends and change in rates of use. *American Journal of Public Health*, *66*, 43-53.
- Kestenbaum, C. J., & Bird, H. R. (1978). A reliability study of the Mental Health Assessment Form for school age children. *Journal of the American Academy of Child Psychiatry*, *17*(2), 338-347.
- Lewis, D. O., Shanok, S. S., Pincus, J. H., & Glaser, G. H. (1979). Violent juvenile delinquents. *Journal of the American Academy of Child Psychiatry*, *18*, 307-319.
- Lucas, A. R., Rodin, E. A., & Simson, C. B. (1965). Neurological assessment of children with early school problems. *Develop Med Child Neurol*, *7*, 145-56.
- McMahon, S. A., & Greenberg, I. M. (1977). Serial neurologic examination of hyperactive children. *Pediatrics*, *59*(4), 584-587.
- Mikkelsen, E. J., Brown, G. L., Minichiello, M. D., Millican, F. K., & Rapoport, J. L. (1981)- Neurologic status in hyperactive, enuretic, encopretic, and normal boys. *J Am Acad Child Psychiatry*, *21*, 75-81.
- Mosher, L. R., Pollin, W., & Stabanau, J. R. (1971). Identical twins discordant for schizophrenia. *Archives of General Psychiatry*, *24*, 422-430.
- Nichols, P. L., & Chen, T. C. (1981). *Minimal brain dysfunction: A prospective study*. Hillsdale, NJ: Lawrence Erlbaum Associates.
- Nichols, P. L., Chen, T., & Pomeroy, among symptoms. Paper presented at a meeting, Washington, DC.
- Niswander, K. R., & Gordon, M. (1975). *Perinatal Study of the National Institute of Health*. No. (NIH), 73-379.
- O'Connor, P. A., Shaffer, D., Stokman, N. E., & Rapoport, J. L. (1977). *Longitudinal research in the Collaborative Perinatal Study of the National Institute of Mental Health*. No. (NIH), 77-379.
- Paulsen, K. (1978). Reflection-impulsivity in children with learning disabilities. *Journal of Child Psychology and Psychiatry*, *19*, 273-278.
- Paulsen, K., & O'Donnell, J. P. (1977). Reflection-impulsivity in children with learning disabilities: Relationship to activity level. *Journal of Child Psychology and Psychiatry*, *18*, 273-278.
- Peters, J. E., Romine, J. S., & Dykens, C. R. (1977). Learning disabilities in children with reading disabilities. *Journal of Child Psychology and Psychiatry*, *18*, 273-278.
- Quitkin, F., Rifkin, A., & Klein, D. (1977). A study of the relationship between character disorder. *Archives of General Psychiatry*, *34*, 119-124.
- Rapoport, J. L., Buchsbaum, M. S., & Kessler, E. J., Langer, D. H., & Bunney, W. E., Jr. (1977). Behavioral effects in normal and hyperactive children. *Journal of Child Psychology and Psychiatry*, *18*, 933-946.
- Rapoport, J. L., Buchsbaum, M. S., & Kessler, E. J. (1978). Dextroamphetamine: A study of its effects on hyperactive children. *Science*, *199*, 560-563.
- Rapoport, J., & Quinn, P. (1975). Mental retardation: A major biologic substrate. *Mental Health*, *4*, 29-44.
- Rochford, M. M., Detre, T., Buckner, J., & Rutter, M. (1977). Interventions in functional psychiatric disorders. *Journal of Child Psychology and Psychiatry*, *18*, 1-12.
- Routh, D. K., & Roberts, R. D. (1977). Evidence for a behavioral syndrome in hyperactive children. *Journal of Child Psychology and Psychiatry*, *18*, 1-12.
- Rutter, M., & Brown, G. W. (1966). Relationships in families containing a child with a psychiatric disorder. *Journal of Child Psychology and Psychiatry*, *7*, 1-12.
- Rutter, M., & Graham, P. (1968). The child: I. Interview with the child. *Journal of Child Psychology and Psychiatry*, *9*, 1-12.
- Rutter, M., Graham, P., & Birch, H. (1968). Reading disability and psychiatric disorder. *Journal of Child Psychology and Psychiatry*, *9*, 149-159.
- Rutter, M., Graham, P., & Yule, W. (1968). Spastics International Medical Publications. *The first review of special education*. London: Heinemann.
- Rutter, M. (1977). Brain damage syndrome. *Journal of Child Psychology and Psychiatry*, *18*, 1-22.
- Sandberg, S. T., Rutter, M., & Tannock, R. (1977). Attention deficit disorder. *Dev. Med. Child Neurol*, *19*, 1-12.
- Schachar, R., Rutter, M., & Smith, R. (1977). Hyperactive children: Implications for education. *Journal of Child Psychology and Psychiatry*, *18*, 1-12.

- Nichols, P. L., Chen, T., & Pomeroy, J. D. (1976). *Minimal brain dysfunction: The association among symptoms*. Paper presented at the annual meeting of the American Psychological Association, Washington, DC.
- Niswander, K. R., & Gordon, M. (1972). *The women and their pregnancies*. The Collaborative Perinatal Study of the National Institute of Neurological Diseases and Stroke, DHEW Publication No. (NIH), 73-379.
- O'Connor, P. A., Shaffer, D., Stokman, C., & Shafer, S. (in press). A neuropsychiatric follow-up of children in the Collaborative Perinatal Project population. In S. Mednick, & M. Harway (Eds.), *Longitudinal research in the United States* (tentative). Boston: Martinus Nijhoff.
- PANESS, MH-9-41, National Institute of Mental Health, Department of Health, Education, and Welfare, United States Government Printing Office, 490-127.
- Paulsen, K. (1978). Reflection-impulsivity and level of maturity. *J Psychol*, 99, 109-112.
- Paulsen, K., & O'Donnell, J. P. (1979). Construct validity of children's behavior problem dimensions: Relationship to activity level, impulsivity, and soft neurological signs. *J of Psychol*, 101, 273-278.
- Peters, J. E., Romine, J. S., & Dykman, R. A. (1975). A special neurological examination of children with learning disabilities. *Developmental Medicine and Child Neurology*, 175, 63-75.
- Quitkin, F., Rifkin, A., & Klein, D. F. (1976). Neurologic soft signs in schizophrenia and character disorder. *Archives of General Psychiatry*, 33, 845-853.
- Rapoport, J. L., Buchsbaum, M. S., Weingartner, J., Zahn, T. P., Ludlow, C., Bartko, J., Mikkelsen, E. J., Langer, D. H., & Bunney, W. E. Jr. (1980). Dextroamphetamine: Cognitive and behavioral effects in normal and hyperactive boys and normal adult males. *Archives of General Psychiatry*, 37, 933-946.
- Rapoport, J. L., Buchsbaum, M. S., Zahn, T. P., Weingartner, H., Ludlow, C., & Mikkelsen, E. J. (1978). Dextroamphetamine: Cognitive and behavioral effects in normal prepubertal boys. *Science*, 199, 560-563.
- Rapoport, J., & Quinn, P. (1975). Minor physical anomalies (stigmata) and early developmental deviation: A major biologic sub-group of "hyperactive children." *International Journal of Mental Health*, 4, 29-44.
- Rochford, M. M., Detre, T., Bucker, G. J., & Harrow, M. (1970). Neuropsychological impairments in functional psychiatric disease. *Archives of General Psychiatry*, 22, 114-119.
- Routh, D. K., & Roberts, R. D. (1972). Minimal brain dysfunction in children: Failure to find evidence for a behavioral syndrome. *Psychological Reports*, 31, 307-314.
- Rutter, M., & Brown, G. W. (1966). The reliability and validity of measures of family life and relationships in families containing a psychiatric patient. *Social Psychiatry*, 1, 28-53.
- Rutter, M., & Graham, P. (1968). The reliability and validity of the psychiatric assessment of the child: I. Interview with the child. *British Journal of Psychiatry*, 114, 563-579.
- Rutter, M., Graham, P., & Birch, H. G. (1966). Interrelations between the choreiform syndrome, reading disability and psychiatric disorder in children of 8-11 years. *Devel Med Child Neurol*, 8, 149-159.
- Rutter, M., Graham, P., & Yule, W. (1970). *A Neuropsychiatric study in childhood*. London: Spastics International Medical Publications.
- Rutter, M., & Yule, W. (1973). Specific reading retardation. In L. Mann, & D. Sabatino (Eds.), *The first review of special education*. Philadelphia: Buttonwood Farms.
- Rutter, M. (1977). Brain damage syndrome in childhood: Concepts and findings. *J Child Psychol Psychiat* 18, 1-22.
- Sandberg, S. T., Rutter, M., & Taylor, E. (1978). Hyperkinetic disorder in psychiatric clinic attenders. *Dev. Med. Child Neurol*. 20, 279-299.
- Schachar, R., Rutter, M., & Smith, A. (in press). Of situationally and pervasively hyperactive children: Implications for syndrome definition. *Journal of Child Psychology and Psychiatry*.

- Seidel, U. P., Chadwick, O. F., & Rutter, M. (1975). Psychological disturbance and physically disturbed children. *Developmental Medicine and Child Neurology*, 17, 563-573.
- Shafer, S. Q., Stokman, C. J., Shaffer, D., Schonfeld, I., O'Connor, P. A., & Wolfe, R. (in preparation). *Ten-year consistency of neurological test performance in boys without focal neurological deficit.*
- Shaffer, D. (1977). Brain injury. In M. Rutter, & L. Hersov (Eds.), *Child psychiatry: Modern approaches*. London: Blackwell Scientific Publications.
- Shaffer, D. (1978). "Soft" neurological signs and later psychiatric disorder—A review. *Journal of Child Psychology and Psychiatry*, 19, 63-65.
- Shaffer, D. (1980). An approach to the validation of clinical syndromes in childhood. In S. Salinger, J. Antrobus, & J. Glick (Eds.), *The ecosystem of the sick kid*. New York: Academic Press.
- Shaffer, D., Bijur, P., Chadwick, O. F. D., & Rutter, M. D. (Submitted for publication). *Localized cortical injury and psychiatric symptoms in childhood.*
- Shaffer, D., McNamara, N., & Pincus, J. (1974) Controlled observations on patterns of activity, attention, and impulsivity in brain-damaged and psychiatrically disturbed boys. *Psychological Medicine*, 4, 4-18.
- Spitzer, R. L., & Endicott, J. (1977). *Schedule of affective disorders and schizophrenia (SADS*, 3rd ed.). New York: Biometrics Research, New York State Psychiatric Institute.
- Spitzer, R. L., Endicott, J., & Robins, E. (1978). *Research diagnostic criteria for a selected group of functional disorders* (3rd ed.). New York: Biometrics Research, New York State Psychiatric Institute.
- Stine, O. C., Saratsiotis, J. M., & Mosser, R. S. (1975). Relationships between neurological findings and classroom behavior. *American Journal of Diseases of Children*, 129, 1036-1040.
- Wechsler, D. (1955). *Wechsler Adult Intelligence Scale*. New York: Psychological Corporation.
- Werry, J. S., Minde, K., Guzman, A., Weiss, G., Dogan, K., & Hoy, E. (1972). Studies on the hyperactive child: VII. Neurological status compared with neurotic and normal children. *American Journal of Orthopsychiatry*, 42, 441-450.
- Wexler, B. E. (1980). Cerebral laterality and psychiatry: A review of the literature. *American Journal of Psychiatry*, 137(3), 279-291.
- Wikler, A., Dixon, J. F., & Parker, J. B. Jr. (1970). Brain function in problem children and controls: Psychometric, neurological and electroencephalographic comparisons. *Amer J Psychiatry*, 127(5), 94-105.
- Wolff, P. H., & Hurwitz, J. (1966). The choreiform syndrome. *Dev Med Child Neurol*, 8, 160-165.
- Wolff, P., & Hurwitz, J. (1973). Functional implications of the mentally brain-damaged syndrome. *Seminars in Psychiatry*, 5, 105-115.

# 3

## Prospective Study

Elsie R. Broussard

Generally somebody has a pro population. This may be on the observations or observations ma promising ideas are developed—these ideas. Findings are share grams, sometimes modifying or small increments—bit by bit—

Attempts to identify factors t social disorder and developme studied risk factors have been th Krakow, 1983). Attention has offspring of teenage mothers, dencing symptoms of perinatal 1981). Risk factors have also in environments associated with p deficiency are often regarded Schulsinger, H., Mednick, S., handicapping factors are quite

The prospective longitudinal 1963 have focused on quite a di infants. These studies have dem tion of her newborn to the chil 4½ and 10/11 years of age. Thi analysis of the most recent pha at age 15 years.