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# A Critical Analysis of the
# Minimal Brain Dysfunction Syndrome

*Overactive and distractible youngsters who underachieve in school often represent a large proportion of child clinical populations. The hypothesis that these children suffer from minor neurological impairments not demonstrable through conventional neurodiagnostic methods enjoys widespread influence. Proponents of this minimal brain dysfunction viewpoint typically admit that there is little direct evidence to support their position, yet they tend to minimize its weaknesses and dangers. Attention to the literature in this field reveals numerous pitfalls, which can be avoided by alternative cognitive–developmental hypotheses. These alternative interpretations do not ignore relevant brain–behavior relationships, but their tenets are more consistent with the available evidence, and they may ultimately lead to improved strategies of intervention.*

The term minimal brain dysfunction (MBD) refers to a cluster of problem behaviors in children that have been discussed in the clinical literature at least since the post–World War I era (Sarason & Doris, 1969, pp. 409–414). Interest in MBD has been spurred by a combination of important factors: (a) reports of high incidence in the school-age population (Paine, 1968), (b) the prospect of a brain–behavior linkage with far-reaching implications for psychopathology generally (Millon, 1969), and (c) the necessity for multidisciplinary interface in identification, evaluation, and treatment of the target population (Birch, 1964). Selection of an appropriate intervention strategy has also generated controversy, particularly over the utilization of psychoactive drugs (Freeman, 1972) and reinforcement principles (Winett & Winker, 1972).

Despite this comparatively high visibility, available topical reviews (Ross, 1976; Sarason & Doris, 1969; Zimet & Fishman, 1970) either do not detail the methodological and empirical complexities raised by MBD or do not fully consider alternative hypotheses. Therefore, this article will evaluate the MBD construct and show that its precepts are untenable. It is also argued that diagnostic fads like MBD are not benign enterprises, because they can risk inappropriate interventions and obscure longer-range, but potentially more effective, lines of inquiry. In short, the MBD thesis and its chief corollary—organic etiology—appear to be artifacts of the clinical–inferential methods by which they are known.

### Definition of the Syndrome

There are numerous, often contradictory, lists of descriptive characteristics of MBD children (Small, 1973). However, there is general agreement in the literature on certain basic points: Virtually all observers hold that the affected population consists of grade school children, roughly 5 to 12 years of age, with measured IQs that exceed 80 (Benton, 1962). Substantially greater disagreement emerges when specific behavioral manifestations are sought (Strother, 1973). Some commentators stress known

or presumed brain dysfunction per se (Strauss & Lehtinen, 1947); others emphasize motor performance, communication skills, or learning abilities (Clements, 1973).

For expository purposes, Wender's (1972) descriptive list will be accepted as definitive of the syndrome because of his exhaustive coverage of the empirical and theoretical literature. Based on this survey, Wender proposed 10 primary behavioral characteristics: (a) hyperactivity—constant, involuntary elevation of general activity level; (b) attention–concentration deficits—inability to persist in an activity for long periods of time; (c) impulsivity—inadequate inhibition or mediation of behavior by the higher thought processes; (d) disobedience—difficulty reported by parents or other authority figures in controlling the child's conduct by routine disciplinary methods; (e) reduced capacity for delaying gratification; (f) academic achievement below the child's measured intellectual ability; (g) generalized unhappiness and lack of self-esteem; (h) presence of troublesome behavior patterns, such as temper tantrums, destructiveness, and school truancy; (i) so-called "soft" neurological signs, including clumsiness, poor balance, impaired fine motor coordination, and deficient visual–motor organization; (j) perceptual and cognitive dysfunctions, such as visual and auditory discrimination problems and reduced capacity for abstract concept formation.

Because this set of descriptors is clearly literary rather than operational in character (Underwood, 1957), basic scientific questions about objectivity, circularity, and quantification remain unanswered. The proposed definition is probably overinclusive of the behavior disorders generally (Anthony, 1970). Furthermore, it employs concepts—such as attention and delay of reward—whose apparent correspondence with laboratory usage is doubtful and probably misleading (Ross, 1976). However, the degree of consensus among observers that the behavior described actually occurs is impressive, even though much of the evidence is purely anecdotal, and it is widely conceded that few, if any, individual children show all the attributed characteristics (Small, 1973).

### Arguments Favoring an MBD Hypothesis

#### PHYSICAL AND EPIDEMIOLOGICAL EVIDENCE

It has long been known that children with certain documented medical conditions suffer MBD-like symptoms as apparent sequelae. This is particularly the case with the

postencephalitic syndrome (Bender, 1949), but has also been reported in various prenatal and postnatal conditions (Knobloch & Pasaminick, 1959). A number of studies further indicate that clinical populations of children show a greater incidence of overactive, distractible behavior combined with a greater incidence of presumed brain injury or disease (e.g., Stewart, Pitts, Craig, & Dieruf, 1966).

The reasoning of MBD adherents is that these observations establish a brain–behavior relationship. Nevertheless, demographic research like that reviewed by Stewart et al. (e.g., Minde, Webb, & Sykes, 1968; Werry et al., 1972) has often produced contradictory results, and existing reports often do not control for IQ, even though a confounding is known to exist (Palkes & Stewart, 1972). Furthermore, physical and epidemiological evidence alone cannot readily account for the large age and sex differences in such MBD-like problems as reduced concentration, restlessness, and temper tantrums, which have been revealed by longitudinal studies like that by Schechtman (1970). Because overactive and distractible behavior is extremely commonplace in the normal population (Lapouse & Monk, 1958; Wolff, 1967), it is difficult to conceive of supposedly MBD-affected youngsters as a truly distinct group. In other words, it is easy to concede the prevalence data and the importance of constitutional or physiological factors without presumption of a unitary syndrome, whether in terms of causes, complaints, or cures.

Three major errors have attended most of the physical–epidemiological speculations on the MBD phenomenon. The first of these is overgeneralization from unquantified case studies. Proponents (Wender, 1971) and detractors (Zimet & Fishman, 1970) alike describe the clinical MBD literature as uncontrolled and impressionistic in the extreme, yielding data that frequently have no scientific merit whatsoever. A closely related second error is gross confusion of correlation with causation. The initial conceptual departure in this respect occurred when early findings on mental retardation and brain injury were extrapolated to children of average intelligence for whom there was no independent evidence of neurological disorder (Sarason & Doris, 1969). Presence of suspect behavior alone became sufficient to judge a child neurologically impaired. The third error concerns an antiquated view of the relationship between brain and behavior. Contemporary neuropsychological research (Reitan, 1958; Satz, 1966) has largely discredited the old assumption that brain pathology has fixed, unequivocal effects on behavior. Heterogenous behavior patterns have been obtained in several experiments with brain-involved children, in both essentially nonretarded (Ernhart, Graham, Eichman, Marshall, & Thurston, 1963) and retarded (Gallagher, 1957) samples.

RESPONSE TO STIMULANTS

A study by Bradley (1937) first showed that stimulant drugs can exert a paradoxical, calming effect on the behavior of a large percentage of excessively active children. This observation may be taken to imply that MBD has a physical origin, since it is so often

subject to change by somatic treatment, and that MBD is a relatively unitary dysfunction because only a certain class of substances generates behavior change.

Neither of these conclusions is justified. A detailed examination of the drug therapy literature by Connors (1971) revealed that the effects of stimulant drugs depend upon the specific therapeutic agent and the response measure employed. Through cluster analysis, Connors (1972) identified seven subgroups of children distinguishable by their differential response to treatment by certain drugs. The methodological and empirical adequacy of the drug therapy literature is also questionable. For example, several otherwise excellent clinical studies of amphetamine treatment in children (e.g., Satterfield, Cantwell, Saul, Lesser, & Podosin, 1973) have been plagued by a lack of appropriate control groups as well as failure to obtain blind ratings of improved behavior. Several of the experiments reviewed by Connors (1971) demonstrated that administration of placebo produced marked, stable changes in behavior, though not of the magnitude shown by the drug-treated groups. Thus, Connors (1972) referred to the conviction that there is a certain type of child who is uniquely responsive to stimulants as a myth that has not been validated empirically.

## SECONDARY MANIFESTATIONS

The assertion that the many behavioral and emotional problems described as MBD are actually secondary manifestations of an underlying condition constitutes a faulty application of the clinical disease model. In the absence of evidence that distinguishes a genuine "underlying" entity, this reasoning violates the principle of parsimony by invoking an unnecessary level of inference between responses and their putative causes. There is another difficulty with the secondary manifestation argument: If emotional and educational problems in general are regarded as evidence for MBD, then the MBD label apparently may be extended to include virtually every form of behavior pathology in children. Thus, MBD becomes synonymous with childhood psychopathology as a whole, dropping any pretense of uniqueness.

In summary, the case for a distinct MBD syndrome is exceedingly weak. Most of the supportive evidence is correlational and speculative, with circular propositions that defy empirical test. The "definitions" suggested are so overgeneralized that they practically invite an MBD label for every uncooperative schoolchild who underachieves. Perhaps this explains why the estimated incidence of MBD can run as high as 20% of the school-age population (Paine, 1968). Precisely what is to be gained from such an indiscriminate exercise in diagnostic futility is not clear.

## Arguments Refuting an MBD Hypothesis

### ABSENCE OF EVIDENCE FOR A UNITARY MBD DIMENSION

Many MBD adherents persist in espousing a unitary syndrome even though they emphasize behavioral or etiological heterogeneity (Strother, 1973; Wender, 1971).

The logic of this approach seems to be that the concordance of behavioral manifestations in MBD is greater than would be expected by chance. By this same argument, one could also postulate that all hospitalized psychiatric patients actually suffer from depression, because depressive signs and symptoms are known to predominate in this population.

The demonstration of a meaningful unidimensionality is crucial to the MBD thesis, but the sparse evidence bearing on this problem is negative. For example, Werry (1968b) reported a factor-analytic study of 67 variables obtained by intensive assessment of 103 cases of apparent MBD. A principal-components analysis yielded 10 factors, the largest of which accounted for but 16% of the variance. Moreover, the obtained factors tended to be specific to each source of information, hardly supporting a unitary interpretation. This study was replicated by Routh and Roberts (1972).

Factor-analytic and cluster-analytic studies of neuropsychological test batteries (Crinella, 1973; Crinella & Dreger, 1972) have also contradicted the existence of a unitary MBD dimension. Crinella (1973) reported cluster comparisons of children with known brain lesions, children manifesting at least two behavioral criteria of brain damage, and children with no demonstrable cerebral pathology. Only a subgroup of the MBD children overlapped the brain-damaged group in test performance.

CRITERIA OF NEUROPATHOLOGY

In a very real sense, the MBD hypothesis survives because the most logical means of sustaining or refuting it—independent neurodiagnostic evidence—is asserted to be unnecessary to establish the disorder. For a variety of reasons, psychologists are less mesmerized by neurodiagnostic methods than was once the case (Saunders, 1975), and there are legitimate reasons to suspect neurological dysfunction undetected by conventional techniques. However, a *total* lack of independent criteria inevitably leads to circularity, and independent evidence assumes even greater importance in the assessment of children, because findings are more often equivocal and more likely to be affected by the subject's developmental course (Teuber & Rudel, 1962).

An additional criterion difficulty is posed by the so-called "soft" signs of brain dysfunction. These signs are often highly unreliable in clinical assessment (Kaspar & Schulman, 1971), and some of the signs appear relatively often in presumably normal subjects (Werry et al., 1972). Ingram (1973) has roundly criticized the notion of soft signs, largely because their neurological significance is ambiguous: "The fact remains that the use of the term 'soft signs' and 'minimal brain damage' is diagnostic of soft thinking . . . . Paediatricians should describe what they find. Then they should evaluate their findings and state the inferences they draw from their evaluation of their findings" (p. 529).

Because Reitan (e.g., 1974a) has repeatedly demonstrated the sensitivity of certain neuropsychological assessment methods to brain lesions in human beings, it is worthwhile to consider this approach as well. In one study, Reitan and Boll (1973)

compared the performance of four groups of children on a battery of tests described in detail by Reitan (1974b). One of the four groups consisted of children with clearly demonstrable brain pathology, another consisted of children with no such evidence. The two remaining groups were composed of subjects who met MBD behavioral criteria but were initially referred for either academic deficiency or behavior problems. In general, statistical comparisons showed that the two MBD groups were more similar to the controls than the brain-injured subjects in terms of level of performance. Although performance by individual subjects did permit clinical judges to separate the two MBD groups from both the control and the brain-injured children, 36% of the controls were misclassified as mildly impaired.

Besides this issue of excessive false positives, neuropsychological assessment of children is complicated by the fact that several factors (e.g., lesion chronicity, age at evaluation) have a differentially greater influence on the neuropsychological test performance of children, as compared with adults (Boll, 1974). Therefore, it seems fair to conclude that the neuropsychological approach has promise in illuminating our understanding of the behavior disorders, but problems remain that preclude acceptance of such assessment as definitive. Initial comparison of MBD children with children with known brain injuries has revealed considerable between-groups behavioral differences.

BRAIN-BEHAVIOR RELATIONS

Natural science necessarily assumes a perfect correspondence between central nervous system activity and behavior (Ellis, 1969). In this light, the assertion that children with MBD-like symptoms suffer from brain pathology pales considerably; it is inevitable that the behavioral attributes of these children have some locus in the nervous system. The crux of the problem remains the *nature* of the disorder or disorders. In the absence of reliable evidence that distinguishes abnormal processes in the central nervous system, to say nothing of abnormal behavioral processes, it is superfluous to assert that it is the hyperactive or distractible child who is physically unique.

Efforts to disentangle the relevant brain-behavior correlates have not been terribly successful to date. As an example, the literature on activity level reviewed by Cromwell, Baumeister, and Hawkins (1963) implicated several structures—the hypothalamus, the reticular activating system, and the frontal lobes—in alterations of general activity level. Yet the specific anatomic areas of involvement remained obscure, as did the precise nature of the relationships between brain dysfunction and overt behavior. A more recent survey by Werry (1968a) led to essentially the same conclusions. Thus, despite an increasingly biological orientation among psychologists (Rimland, 1969) and an increasingly behavioral orientation in the neurosciences (Pincus & Tucker, 1974), it is debatable whether the physiological processes underlying MBD-like behavior are better understood, or whether there is simply a greater willingness to accept biological hypotheses in the contemporary zeitgeist.

## BASIC PRINCIPLES OF LEARNING

Advocates of the MBD position seem logically committed to the view that the basic principles of behavior are in some way different for brain-involved (as opposed to non-brain-involved) children. There is a substantial literature on the modification of MBD-like problems which refutes this position (Allen, Henke, Harris, Baer, & Reynolds, 1967; Doubros & Daniels, 1966; Rickard & Saunders, 1971). If psychologists take as their goal the prediction and control of behavior, the premature assumption of inherent defects in the organism may actually impede efforts to extend fundamental behavioral principles to the entire group.

## RESEARCH WITH INTACT GROUPS

Ellis (1969) noted that the two-group, correlational designs that long characterized research in mental retardation simply reaffirmed the validity of intelligence tests by showing that individuals inferior in one type of problem solving were also inferior in other types. The proliferation of circular, behavioral definitions of MBD forebodes a similar confusion in research on the behavior disorders generally. No matter how objective the selection criteria, research on intact groups cannot lead to causative conclusions from any obtained between-groups behavioral differences, because obtained differences in performance may logically be attributed to any variable correlated with the classification factor in the population. As an antidote, many investigators (e.g., Baumeister, 1967) have advocated the study of *interactions* of task or environmental variables with subject variables as a more appropriate research strategy.

## INTRACTABILITY OF SYMPTOMS

It is frequently tempting to assume that refractory behavioral problems result from organic pathology (Kernberg, 1969). The process–reactive and endogenous–exogenous distinctions in schizophrenia and depression, respectively, are analogous instances in which responsiveness to treatment has affected classification of abnormal behavior in terms of an organic–psychogenic dichotomy (Phillips & Draguns, 1971).

Obviously, the fact that certain types or forms of disturbance are relatively inaccessible does not necessarily imply anything about their etiology. The presumption of a physical basis for MBD or some subtype of MBD may ultimately prove to be correct, and the research cited earlier by Crinella (1973), Reitan and Boll (1973), and Connors (1972) tends to support the latter possibility. Nevertheless, it is difficult to refute the view that *all* clinical problems have biological, psychological, and social implications and that evaluation and remediation must be carried forward on each of these fronts simultaneously (Sarason & Doris, 1969).

## TREATMENT IMPLICATIONS

Since the appearance of Szasz's (1960) polemic on mental health mythology, it has become increasingly common for applied psychologists to examine their work in terms of actual effects, rather than stated goals alone. In this connection, Doris and Solnit (1963) reported that the presumption of central nervous system dysfunction in children is often accompanied by a sense of hopelessness on the part of remedial resources. Wender (1971) countered that an organic rationale for MBD may relieve unwarranted guilt or anxiety about the child's behavior and that the syndrome's response to stimulant drugs actually brightens the prognostic picture. In contrast with Wender, the approach advocated by Chess (1960) retains this hopeful element without causal attribution to abnormal brain function. This stance encourages a flexible posture and permits whatever intervention—behavioral, physical, or educational—may be required by the specific case.

## SUMMARY

The weaknesses of the MBD hypothesis thus extend to empirical, methodological, and clinical issues alike. Lacking suitable criteria and independent evidence, MBD enthusiasts have managed to extend a proposition that was originally meaningful (certain abnormal conditions of the brain have specifiable behavioral consequences) to a proposition loaded with excess conceptual baggage (maladaptive behavior in general is attributable to unspecifiable abnormal conditions of the brain). What remains is to demonstrate that other approaches, without special assumptions, can better account for the available data and lead to more constructive avenues for intervention.

## Alternative Conceptions

### DEVELOPMENTAL CONTINUUM

As Ross (1976) has shown, construing children's behavior in normative fashion can lead to a conception of MBD-like problems, either as an instance of the extreme case or as a deviation in some specific function, such as attention processes. Questions about the origin of these differences arise primarily when it can be shown that abnormal factors affect the incidence of the condition (Dingman & Tarjan, 1960) or when it can be demonstrated that the individual case shows characteristics known to be associated with specific causative agents. It is well established (Millon, 1969) that marked, stable individual differences can be observed, practically from birth, along several dimensions of behavior relevant to MBD (response intensity and persistence, distractibility, and mood patterns). Obviously, children do show individual differences of genetic or constitutional origin with implications for behavior later in life.

There is abundant evidence that this MBD-like behavior can be interpreted normatively and is affected by important psychological factors. Cross-sectional, factor-

analytic studies of "problem behavior" checklist data (e.g., Dielman, Cattell, & Lepper, 1971; Speer, 1971) typically yield a three-dimensional structure across samples that consist of conduct disorder (externalized, antisocial behavior), personality disorder (internalized, withdrawn, and anxious behavior), and inadequacy–immaturity (preoccupied, disinterested, and irresponsible behavior). Connors (1970) essentially replicated these three dimensions in a population of 316 psychiatric clinic patients and 365 normal children. Also, Connors found it was the severity of the behavior problems that distinguished the patient group from the normal group; subpopulations of neurotic versus hyperkinetic patients were found to differ primarily in factor scores on the basic dimensions, not in the dimensions themselves.

Longitudinal research (Battle & Lacey, 1972; Bayley, 1970) reveals that characteristics associated with MBD (activity level, impersistence, impaired self-image, distractibility) are in turn related to such variables as measured intelligence, parental expectations, and child-rearing patterns. These data tend to support the position that psychological factors affect the manifestation of the target behaviors. Under these circumstances, it seems more parsimonious to attack the maladaptive behavior within a framework of similarity, rather than differentness.

On a probabilistic basis, a normal continuum hypothesis can also incorporate diagnostic questions. For example, Kaspar, Millichap, Backus, Child, and Schulman (1971) found that heightened activity level and increased distractibility are simply more *likely* to occur when there is fairly strong presumptive evidence of brain pathology. Similarly, Satterfield et al. (1973) demonstrated that teacher ratings of improvement under methylphenidate were a direct function of the number of soft signs and combined electroencephalogram ratings; the greater the preponderance of evidence that brain abnormalities actually existed, the greater the likelihood of improvement under drugs. What this means is that under a normal continuum hypothesis, assessment information would be used to evaluate levels and patterns of performance for remedial purposes, rather than to establish a cause or to dictate a specific intervention strategy. All elements, including physical factors, could then be explored for their relative weight in the specific case.

CONCEPTUAL TEMPO

The research of Kagan and his associates on analytic and reflective predispositions in children (Kagan & Kogan, 1970; Kagan, Rosman, Day, Albert, & Phillips, 1964) represents a second potential alternative to the MBD hypothesis. Since impulsivity is mentioned so often by advocates of the MBD position as an important characteristic of the target child, Kagan's explorations of children's performance with tasks involving response uncertainty definitely seem relevant.

Two aspects of children's problem-solving behavior have been investigated by Kagan: the analytic–relational dimension and the impulsive–reflective dimension (Denney, 1972). An analytic style is one in which the child differentiates small details in complex

visual arrays and relates them to each other on the basis of some inferred quality. A relational style is one in which the child pairs stimuli on the basis of a functional relationship between them. The impulsive–reflective dimension, on the other hand, amounts to a latency measure. Fast solution times are typical of impulsive children; slow solution times characterize reflective children.

On the standard tasks employed by Kagan, accuracy of performance tends to increase, and response time tends to decrease, with the age of the child (Kagan & Kogan, 1970). Task performance bears a low positive relationship to verbal subtests of the Weschler Intelligence Scale for Children, and classification of the individual child as reflective or impulsive is relatively stable. The impulsive–reflective dimension also generalizes to a variety of other tasks involving response uncertainty, including tachistoscopic recognition, an informal interview situation, prose reading, and reproduction of Bender–Gestalt designs.

Of special relevance to MBD is Kagan's demonstration that analytic cognitive processing is related to task-irrelevant distractibility and gross motor behavior (Kagan et al., 1964, Study 8). Less analytic children were found to be higher in proportion of off-task behavior and to exhibit longer periods of gross motor activity in a playground situation. Perhaps equally important is the subsequent finding that cognitive processing can be altered by systematic reinforcement and by exposure to impulsive or reflective models (Denney, 1972; Kagan & Kogan, 1970, pp. 1312–1313). · Kagan (1966) has thus suggested that training excessively impulsive children to reflect longer over alternative solutions may be an effective alternative to traditional methods of instruction with these youngsters.

The implications of conceptual-tempo research for MBD are clear: Without special assumptions about the condition of the brain, stable differences can be observed among individuals in their manner of approach to many different tasks. Phenotypically, at least, the behavior of impulsive children does not differ remarkably from that of children traditionally labeled MBD (Juliano, 1974). These children are regarded as being at the extreme of a naturally occurring distribution, and variables known to have considerable influence have been successfully applied to impulsive problem solving.

## Recapitulation

The central question at issue in this critique is whether the available evidence justifies an MBD categorization as it is used by its proponents—a distinct behavioral entity that has identifiable precursors and requires a specific remedial approach. There is no quarrel with an organic perspective as such: Since psychology is a biological science, exploration of biophysical variables as determinants of problem behavior is an essential part of the scientific enterprise.

There are many unresolved difficulties with the MBD thesis. The proposed behavior pattern simply does not hang together in the supposedly affected population, and independent evidence of brain dysfunction is very difficult to come by. Adoption

of the MBD hypothesis encourages an arcane research strategy that can generate uninterpretable results. As far as intervention is concerned, the assumption that the behavior of MBD children is not lawful or different in principle from that of other children has chilling prospects. A cognitive-developmental perspective may lead in the long run to greater understanding of children's inattention and impulsivity. In the interim, direct methods of intervention have been found effective in alleviating many of the problem behaviors associated with MBD. With these considerations in hand, applied clinician and researcher alike are well advised to follow the dictates of Ingram (1973): describe thoroughly, infer cautiously, and treat conservatively.

## REFERENCES

Allen, K. E., Henke, L. B., Harris, F. R., Baer, D. M., & Reynolds, N. J. Control of hyperactivity by social reinforcement of attending behavior. *Journal of Educational Psychology,* 1967, *58,* 231–237.

Anthony, E. J. The behavior disorders of childhood. In P. Mussen (Ed.), *Carmichael's manual of child psychology* (3rd ed.). New York: Wiley, 1970.

Battle, E. S., & Lacey, B. A context for hyperactivity in children, over time. *Child Development,* 1972, *43,* 757–773.

Baumeister, A. A. Problems in comparative studies of mental retardates and normals. *American Journal of Mental Deficiency,* 1967, *71,* 869–875.

Bayley, N. Development of mental abilities. In P. Mussen (Ed.), *Carmichael's manual of child psychology* (3rd ed.). New York: Wiley, 1970.

Bender, L. Psychological problems of children with organic brain disease. *American Journal of Orthopsychiatry,* 1949, *19,* 404–415.

Benton, A. L. Behavioral indices of brain injury in school children. *Child Development,* 1962, *33,* 199–208.

Birch, H. G. (Ed.). *Brain damage in children.* Baltimore: Williams & Wilkins, 1964.

Boll, T. J. Behavioral correlates of cerebral damage in children aged 9 through 14. In R. M. Reitan & L. A. Davison (Eds.), *Clinical neuropsychology: Current status and applications.* Washington, D. C.: Winston, 1974.

Bradley, C. The behavior of children receiving benzedrine. *American Journal of Psychiatry,* 1937, *94,* 577–585.

Chess, S. Diagnosis and treatment of the hyperactive child. *New York State Journal of Medicine,* 1960, *60,* 2379–2385.

Clements, S. D. Minimal brain dysfunction in children. In S. G. Sapir & A. C. Nitzburg (Eds.), *Children with learning problems.* New York: Brunner/Mazel, 1973.

Connors, C. K. Symptom patterns in hyperkinetic, neurotic, and normal children. *Child Development,* 1970, *41,* 667–682.

Connors, C. K. Recent drug studies with hyperkinetic children. *Journal of Learning Disabilities,* 1971, *4,* 476–483.

Connors, C. K. Psychological effects of stimulant drugs in children with minimal brain dysfunction. *Pediatrics,* 1972, *49,* 702–708.

Crinella, F. M. Identification of brain dysfunction syndromes in children through profile

analysis: Patterns associated with so-called "minimal brain dysfunction." *Journal of Abnormal Psychology*, 1973, *82*, 33–45.

Crinella, F. M., & Dreger, R. M. Tentative identification of brain dysfunction syndromes in children through profile analysis. *Journal of Consulting and Clinical Psychology*, 1972, *38*, 251–260.

Cromwell, R. L., Baumeister, A. A., & Hawkins, W. F. Research in activity level. In N. Ellis (Ed.), *Handbook of mental deficiency.* New York: McGraw-Hill, 1963.

Denney, D. R. Modeling effects upon conceptual style and cognitive tempo. *Child Development*, 1972, *43*, 105–119.

Dielman, T. E., Cattell, R. B., & Lepper, C. Dimensions of problem behavior in the early grades. *Journal of Consulting and Clinical Psychology*, 1971, *37*, 243–249.

Dingman, H. F., & Tarjan, G. Mental retardation and the normal distribution curve. *American Journal of Mental Deficiency*, 1960, *64*, 991–994.

Doris, J., & Solnit, A. J. Treatment of children with brain damage and associated school problems. *Journal of the American Academy of Child Psychiatry*, 1963, *2*, 618–635.

Doubros, S. G., & Daniels, G. J. An experimental approach to the reduction of overactive behavior. *Behavior Research and Therapy*, 1966, *4*, 251–258.

Ellis, N. A behavioral research strategy in mental retardation: Defense and critique. *American Journal of Mental Deficiency*, 1969, *73*, 557–566.

Ernhart, C. B., Graham, F. K., Eichman, P. L., Marshall, J. M., & Thurston, D. Brain injury in the preschool child: Some developmental considerations: II. Comparison of brain injured and normal children. *Psychological Monographs*, 1963, *77*(11, Whole No. 574).

Freeman, R. D. The drug treatment of learning disorders: Continuing confusion. *Journal of Pediatrics*, 1972, *81*, 112–115.

Gallagher, J. J. A comparison of brain-injured and nonbrain-injured mentally retarded children on several psychological variables. *Monographs of the Society for Research in Child Development*, 1957, *22*(2, Serial No. 65).

Ingram, T. T. S. Soft signs. *Developmental Medicine and Child Neurology*, 1973, *15*, 527–530.

Juliano, D. B. Conceptual tempo, activity, and concept learning in hyperactive and normal children. *Journal of Abnormal Psychology*, 1974, *83*, 629–634.

Kagan, J. Reflection–impulsivity: The generality and dynamics of conceptual tempo. *Journal of Abnormal Psychology*, 1966, *71*, 17–24.

Kagan, J., & Kogan, N. Individual variation in cognitive processes. In P. Mussen (Ed.), *Carmichael's manual of child psychology* (3rd ed.). New York: Wiley, 1970.

Kagan, J., Rosman, B. L., Day, D., Albert, J., & Phillips, W. Information processing in the child: Significance of analytic and reflective attitudes. *Psychological Monographs*, 1964, *78*(1, Whole No. 578).

Kaspar, J. C., Millichap, J. G., Backus, R., Child, D., & Schulman, J. L. A study of the relationship between neurological evidence of brain damage in children and activity and distractibility. *Journal of Consulting and Clinical Psychology*, 1971, *36*, 329–337.

Kaspar, J. C., & Schulman, J. L. Organic mental disorders: Brain damages. In B. B. Wolman (Ed.), *Manual of child psychopathology.* New York: McGraw-Hill, 1971.

Kernberg, P. F. The problem of organicity in the child. *Journal of the American Academy of Child Psychiatry*, 1969, *8*, 517–541.

Knobloch, H., & Pasaminick, B.  Syndrome of minimal cerebral damage in infancy.  *Journal of the American Medical Association*, 1959, *170*, 1384–1387.

Lapouse, R., & Monk, M.  An epidemiologic study of behavior characteristics in children.  *American Journal of Public Health*, 1958, *48*, 1134–1144.

Millon, T.  *Modern psychopathology*.  Philadelphia:  Saunders, 1969.

Minde, K., Webb, G., & Sykes, D.  Studies on the hyperactive child:  VI.  Prenatal and paranatal factors associated with hyperactivity.  *Developmental Medicine and Child Neurology*, 1968, *10*, 355–363.

Paine, R. S.  Syndromes of "minimal cerebral damage."  *Pediatric Clinics of North America*, 1968, *15*, 779–801.

Palkes, H., & Stewart, M.  Intellectual ability and performance of hyperactive children.  *American Journal of Orthopsychiatry*, 1972, *42*, 35–39.

Phillips, L., & Draguns, J. G.  Classification of the behavior disorders.  In P. H. Mussen & M. R. Rosenzweig (Eds.), *Annual Review of Psychology* (Vol. 22).  Palo Alto, Calif:  Annual Reviews, 1971.

Pincus, J. H., & Tucker, G.  *Behavioral neurology*.  New York:  Oxford University Press, 1974.

Reitan, R. M.  Qualitative versus quantitative mental changes following brain damage.  *Journal of Psychology*, 1958, *8*, 271–276.

Reitan, R. M.  Methodological problems in clinical neuropsychology.  In R. M. Reitan & L. A. Davison (Eds.), *Clinical neuropsychology: Current status and applications*.  Washington, D.C.:  Winston, 1974.  (a)

Reitan, R. M.  Psychological effects of cerebral lesions in children of early school age.  In R. M. Reitan & L. A. Davison (Eds.), *Clinical neuropsychology:  Current status and applications*.  Washington, D.C.:  Winston, 1974.  (b)

Reitan, R. M., & Boll, T. J.  Neuropsychological correlates of minimal brain dysfunction.  *Annals of the New York Academy of Sciences*, 1973, *205*, 65–88.

Rickard, H. C., & Saunders, T. R.  Control of "clean-up" behavior in a summer camp.  *Behavior Therapy*, 1971, *2*, 340–344.

Rimland, B.  Psychogenesis versus biogenesis:  The issues and the evidence.  In S. C. Plog & R. B. Edgerton (Eds.), *Changing perspectives in mental illness*.  New York:  Holt, Rinehart & Winston, 1969.

Ross, A. O.  *Psychological aspects of learning disabilities and reading disorders*.  New York:  McGraw-Hill, 1976.

Routh, D. K., & Roberts, R. D.  Minimal brain dysfunction in children.  *Psychological Reports*, 1972, *31*, 307–314.

Sarason, S. B., & Doris, J.  *Psychological problems in mental deficiency* (3rd ed.).  New York:  Harper & Row, 1969.

Satterfield, J. H., Cantwell, D. P., Saul, R. E., Lesser, L. I., & Podosin, R. L.  Response to stimulant drug treatment in hyperactive children:  Prediction from EEG and neurological findings.  *Journal of Autism and Childhood Schizophrenia*, 1973, *3*, 36–48.

Satz, P.  Specific and nonspecific effects of brain lesions in man.  *Journal of Abnormal Psychology*, 1966, *71*, 65–70.

Saunders, T. R.  Toward a distinctive role for the psychologist in neurodiagnostic decision making.  *Professional Psychology*, 1975, *6*, 161–166.

Schechtman, A. Age patterns in children's psychiatric symptoms. *Child Development*, 1970, *41*, 683–693.

Small, L. *Neuropsychodiagnosis in psychotherapy*. New York: Brunner/Mazel, 1973.

Speer, D. C. Behavior Problem Checklist (Peterson-Quay): Base-line data from parents of child guidance and nonclinic children. *Journal of Consulting and Clinical Psychology*, 1971, *36*, 221–228.

Stewart, M. A., Pitts, F. N., Craig, A., & Dieruf, W. The hyperactive child syndrome. *American Journal of Orthopsychiatry*, 1966, *36*, 861–867.

Strauss, A. A., & Lehtinen, L. E. *Psychopathology and education of the brain-injured child*. New York: Grune & Stratton, 1947.

Strother, C. R. Minimal cerebral dysfunction: An historical overview. In S. G. Sapir & A. C. Nitzburg (Eds.), *Children with learning problems*. New York: Brunner/Mazel, 1973.

Szasz, T. S. The myth of mental illness. *American Psychologist*, 1960, *15*, 113–118.

Teuber, H. L., & Rudel, R. G. Behavior after cerebral lesions in children and adults. *Developmental Medicine and Child Neurology*, 1962, *4*, 3–20.

Underwood, B. J. *Psychological research*. New York: Appleton, 1957.

Wender, P. H. *Minimal brain dysfunction in children*. New York: Wiley, 1971.

Wender, P. H. The minimal brain dysfunction syndrome in children. *Journal of Nervous and Mental Disease*, 1972, *153*, 55–71.

Werry, J. S. Developmental hyperactivity. *Pediatric Clinics of North America*, 1968, *15*, 581–599. (a)

Werry J. S. Studies on the hyperactive child: IV. An empirical analysis of the minimal brain dysfunction syndrome. *Archives of General Psychiatry*, 1968, *19*, 9–16. (b)

Werry, J. S., et al. Studies on the hyperactive child: VII. Neurological status compared with neurotic and normal children. *American Journal of Orthopsychiatry*, 1972, *42*, 441–451.

Winett, R. A., & Winker, R. C. Current behavior modification in the classroom: Be still, be quiet, be docile. *Journal of Applied Behavior Analysis*, 1972, *5*, 599–604.

Wolff, S. Behavioral characteristics of primary school children referred to a psychiatric department. *British Journal of Psychiatry*, 1967, *113*, 885–893.

Zimet, C. N., & Fishman, D. B. Psychological deficit in schizophrenia and brain damage. In P. H. Mussen & M. R. Rosenzweig (Eds.), *Annual Review of Psychology* (Vol. 21). Palo Alto, Calif.: Annual Reviews, 1970.