Schizophrenic Syndrome Related to Minimal Brain Dysfunction: A Possible Neurologic Subgroup

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with the collaboration of Edward Charles

Abstract

One adult subgroup of the schizophrenic syndrome seems to share minimal brain dysfunction (MBD) as Anlage, mostly but not exclusively on a familial basis and occurring in varying degrees. Pathogenically, MBD usually involves problems of spatial orientation, the development of language, and a low external and internal stimulus barrier. These cortical problems can lead to problems of establishing self-boundaries and coordination, to feelings of perplexity, difficulties with the written and spoken word and abstract conceptualization, poor impulse control and defenses, overload by external stimuli, and resulting disorganization. These phenomena in turn cause secondary emotional problems, especially during the school years. In addition to neuropsychological and soft neurological signs and symptoms, this group is diagnostically characterized by a poor response to phenothiazines alone. Therapeutically, energizers, together with diphenylhydantoin, at times in combination with lithium, are often useful. Psychotherapy must include education concerning MBD and must deal with the primary and secondary cognitive and emotional aspects of MBD as well as other problems. Prevention of overload and genetic counseling may be indicated.

I first stated my belief that schizophrenia was not likely to be a single disease, but rather a syndrome consisting of different disorders over 30 years ago (Bellak 1947, 1948). I have also long held that research in schizophrenia will make progress only if an attempt is made to isolate subgroups likely to share some common pathogenesis (Bellak 1949, 1975; Bellak and Loeb 1969). The tendency to look for one overall factor relevant for all schizophrenics seems to me to account for the lack of conclusive findings so far, a viewpoint that is slowly gaining adherents. Both research and clinical experience increasingly lead me to believe that one subgroup of the schizophrenic syndrome is most likely predicated upon some neurological deficit. If this hypothesis proves correct, I propose a diagnostic label for this subgroup: Schizophrenic syndrome related to minimal brain dysfunction (Bellak 1976). In my experience, the treatment of this subgroup needs to be differentiated from that of others suffering from the schizophrenic syndrome.

In my earlier publications, I remarked upon the fact that a significant number of schizophrenics with early onset reported an episode of high fever in their childhood. I suggested that diffuse encephalitic changes resulting from high fever or other organic brain disease might account for diffuse and slight brain damage, which in turn might be related to diffuse and slight impairment of ego functions (Bellak 1955). More recently, research on ego functions in schizophrenics delineated five subgroups by factor analysis.
(Bellak, Hurvich, and Gediman 1973). One of these, representing about one fifth of the schizophrenic sample, was characterized by having a single, distinctly different ego function: low stimulus barrier. This made me wonder whether they might not share some “soft” neurological disorder like minimal brain dysfunction (MBD). Subsequent clinical experience with adults seemed to confirm that MBD was significantly related to the schizophrenic syndrome (and other psychiatric disorders) in some patients.

The establishment of MBD as a well-defined concept in children may be arbitrarily dated from a 1966 Conference on MBD at the National Institutes of Health (Clement 1966). Until then, the syndrome had been referred to by various terms that, in fact, designated only some of the symptoms and syndromes now subsumed under the definition—such conditions as dyslexia and some of the other specific learning difficulties. Wender (1971) has provided the most systematic description of MBD in children.

Why has MBD not been associated with adults? In some cases, adults learn to compensate for their difficulties. They mask them; when they are not in school, no one may hold them to account. Or they are unaware that some problems of adolescence or young adulthood, such as constantly getting lost while driving, are related to difficulties with spatial orientation—a sign of their minimal neurological dysfunction. Emotional outbursts (including assaultive behavior), restlessness, the inability to hold jobs, and many forms of acting out are typical manifestations of the lack of adequate regulation and control of drives and impulses (Borland and Heckman 1976). There is now evidence that in many of those diagnosed as having MBD in childhood, the symptoms persist into adolescent and adult life.

Cantwell (1972) hypothesized that hyperactive children grew into hyperactive adults. To test the theory, he conducted a systematic psychiatric examination of the parents of 50 hyperactive children and the parents of 50 matched controls. The parents of the hyperactive children had an increased incidence of alcoholism, sociopathy, and hysteria, and 10 percent of these parents Cantwell judged to have been hyperactive children themselves.

Borland and Heckman’s (1976) study supports Cantwell’s findings. These authors suggest that many emotional problems result from the persistence of symptoms of hyperactivity into adult life; they believe that most social and psychiatric consequences of the disorder relate to its presence in both childhood and adulthood.

In the study of Borland and Heckman, hyperactive children and their brothers were followed up 25 years after the initial contact. MBD symptoms had been considerably reduced in the patients. Nevertheless, half of the probands had psychiatric problems that affected their socioeconomic status, despite normal intelligence and education. They frequently changed jobs; to them, work was a means of avoiding feelings of restlessness.

A number of longitudinal and adoption studies suggest that MBD persists into adult life, where its existence is camouflaged by the application of a variety of diagnostic labels. Wood et al. (1976) identified 15 putative MBD adults on the basis of current MBD-like complaints and self-descriptions of MBD characteristics in childhood. Eleven of the 15 were then given a double-blind trial of methylphenidate hydrochloride, and eight of them showed a significant response to it. Other stimulants or tricyclic antidepressants were also tried, and 8 of the 15 showed a good response, 2 a moderately favorable response. Wood et al. concluded that MBD does persist into adult life, with the signs and symptoms lessening or disappearing in the twenties and thirties rather than in the teens.

Mann and Greenspan (1976) have offered suggestions for the identification and treatment of adult brain dysfunction. Their 20 patients each had the following characteristics: a history of early learning disorder with short attention span, diffuse severe symptoms in adulthood with prominent elements of anxiety and depression, dramatic improvements with the administration of imipramine, and a mental status characterized by rapid speech and many shifts of subjects (but without overt indicators of psychotic thinking).

According to Hartocollis (1968), evidence of soft neurological signs is often overlooked in adolescent and young adult psychiatric patients. Reporting on such a group, whose difficulties were not obviously organic but who had responded poorly to treatment, he noticed that there was often an unevenness in psychological test performance—for example, a discrepancy between verbal and performance IQ scores. He found that these patients had a history of frustration, failure to perform up to parental and school expectations, poor results from psychiatric treatment, and evidence of organicity. But the evidence had been overlooked or ignored. Hartocollis notes that such persons can learn to function with much less frustration once these neurological signs are recognized and a properly structured environment is provided.
Schizophrenia and MBD

Several authors have referred to the relationship between MBD and schizophrenia, and some have described the interaction of MBD in schizophrenics. Tucker, Campion, and Silberfarb (1975) found a strong but not exclusive relationship between neurological impairment and thought disorder, as well as between neurological impairment and schizophrenia. Rochford et al. (1970) found that patients with affective disorders did not have neurological abnormalities, while those diagnosed as schizophrenic or having personality disorders and neuroses did. Furthermore, schizophrenics had a greater percentage of impairments. Moreover, patients with neurological abnormalities exhibited various kinds of poor social judgment and behavior, as well as such symptoms as blocking and distractibility. These results “support the hypothesis that diffuse CNS dysfunction adversely affects the individual’s personality development . . .” (Rochford et al. 1970, p. 118).

Quitkin, Rifkin, and Klein (1976) suggest the existence of a subgroup of schizophrenics characterized by soft neurological signs. They found that patients diagnosed as schizophrenic with premorbid asociality, as well as those with emotionally unstable character disorders, exhibited neurological impairments not found in the rest of the patients. Therefore, the authors consider central nervous system impairment a criterion for syndrome validity.

Huey et al. (1978) have also recently reported on a case of a patient diagnosed as chronic paranoid schizophrenia based on clinical and research criteria. The patient had a history of childhood MBD with current adult manifestations and poor response to antipsychotic drugs. Significant improvement was found with the use of methylphenidate.

There is suggestive evidence that persons with childhood MBD may be predisposed to schizophrenia as adolescents and adults. Handford (1975) hypothesizes that individuals who experience brain hypoxia perinatally, or immediately after birth will be at risk for MBD in childhood and for schizophrenia as adults, possibly because of damage to the dopaminergic pathways.

Fish et al. (1965) have analyzed early developmental profiles to identify infants vulnerable to schizophrenia. In one pilot study at a baby clinic, 16 infants from families with a high incidence of social and psychiatric disorders were examined periodically from the age of 1 month, and predictions of schizophrenia were made on the basis of uneven neurological development. The children were given psychological examinations at 9 to 10 years of age, and those originally diagnosed as vulnerable to schizophrenia had significantly higher incidence of disorder than the others. In another study, by Fish and Higin (1972), the visual-motor development of 10 infants whose mothers were schizophrenic was measured from birth to 2 years of age. Psychological evaluation after 10 years revealed a relationship between poor neurological integration in childhood and later emotional impairment. Both Handford and Fish favor early therapeutic intervention for children at risk and their families.

Neurological findings in identical twins discordant for schizophrenia were discussed by Mosher, Pollin, and Stabenau (1971). In this study, some very suggestive evidence was found for a relationship between soft neurological signs and schizophrenia. Huessy’s (1974, 1976) findings in adult schizophrenics, whom he considers hyperkinetic, further support this hypothesis.

Powerful though inadvertent support comes indirectly from a study reported by Rosenthal et al. (cited in Mosher and Feinsilver 1973) at the National Institute of Mental Health, in collaboration with a Jerusalem-based group, in which 50 children of schizophrenic parents and 50 controls were examined neurologically in great detail. According to Mosher and Feinsilver’s (1973) summary of this work, each group was divided at the median to form subgroups composed of high and low scorers. Comparison of the two high-scoring subgroups revealed that the index group had significantly higher scores than the controls. No significant differences were found between the two low-scoring subgroups. However, when the subjects were divided into those above and those below age 11, the younger index group had higher neuropathology scores than the older group. These findings suggested that certain abnormal neuro-pathological traits detectable at younger ages may disappear, decrease, or be masked as puberty approaches. The authors concluded that these outward signs of an apparently inherited predisposition to schizophrenia tend to disappear at puberty. Since the general opinion is that maturation at least decreases the manifestations of MBD, it seems likely that we are dealing with the role of MBD in the offspring of some schizophrenic parents. It is my unsupported theory that these particular “schizophrenic” parents also suffered from MBD.

Clinical Characteristics

At this point my clinical hunch is supported by the study of more than 50 patients and their extended families, whom I came to know well in
individual, intensive, prolonged psychotherapy. A more comprehensive and systematic investigation is underway.

Typically, I will have a referral of a schizophrenic who had been seen by several psychiatrists and tried on all available neuroleptic drugs with little success or with actual worsening. Characteristically, such patients then reveal themselves to be either left-handed and/or to have a history of learning difficulties, difficulties with numerical concepts and complex words, mirror writing, reversal of syllables when speaking, or more specifically dyslexia in some form.

Problems of impulse control are often an outstanding feature of their clinical picture—escaping with a stolen car from a hospital, outbursts of anger, or merely irritability. When referred for neuropsychologic testing, such patients frequently show evidence of soft neurological signs; poor performance on the Block Test of the Wechsler Adult Intelligence Scale and other performance tests; poor spatial perception and difficulties in sequencing, such as not being able to name the months of the year despite an IQ of 145. On neurological examination, there is usually evidence of synkinesis, overflow dominances generally, variable problems in coordination, and mixed thought disorder, and to have done so for 6 months or more. Others, such as the 20-year-old girl discussed on p. 484 (who had been diagnosed as schizophrenic in eight hospitals) do not satisfy DSM-III criteria. Even in those who do fulfill DSM-III criteria for schizophrenia, however, the characteristic features of MBD dominate the clinical picture:

- Lack or frequent loss of impulse control, often of sociopathic nature.
- General tension and restlessness.
- Lack of response to phenothiazines.
- Response to amphetamine-like drugs.
- Delusions and hallucinations, if present, usually not prominent in the clinical picture; patients often hospitalized for violent acts, sometimes, but not always, in relation to ideas of reference.

DSM-III may help prevent the inappropriate diagnosis of schizophrenia in some instances. However, it will not be more useful clinically than DSM-II with patients who meet DSM-III criteria for schizophrenia but also suffer from MBD. In such patients, symptoms of MBD are frequently the outstanding clinical feature, are of primary therapeutic and prognostic importance, and possibly are of pathogenic and even etiologic significance. Although DSM-III will undoubtedly improve the repeat reliability of the diagnosis of schizophrenia, it is unlikely to contribute much to the validity of the diagnosis, or to the planning of treatment and the formulation of a prognosis.

In most instances, I was able to see three generations in consultation: that is, grandparents, parents, and adolescent or college-aged children. In some cases, my patient was in the middle generation, and I saw aged parents and adolescent children coincidentally—often because they also had some problems or contributed to the patient’s difficulties. In some cases, the primary patient was not schizophrenic, but even in adult life showed residual aspects of MBD with varying psychopathology. Characteristics of this subgroup of patients appear in tables 1 and 2.

Of course, in all instances, the initial impression of MBD on the basis of history needs to be confirmed (or rejected) by neuropsychologic testing and a neurological examination for soft signs.

Case Histories

A 40-year-old woman was referred to me by her previous psychiatrist, who had considered her schizophrenic. My own diagnostic impression was of a woman with an inability to express herself in logical sequence under pressure. She also suffered from migraine headaches, became easily disorganized when she had more than one task to perform, had difficulty in geographical orientation, and had a history of dyslexia. I found no reason to diagnose her as schizophrenic, but rather as suffering from a personality disorder complicated by MBD. Both her parents were left-handed, and her father was especially given to violent temper outbursts. One sister, however, suffered from grand mal seizures, was diagnosed as schizophrenic, and had committed suicide in a psychiatric institution. The patient’s husband had difficulties in spatial orientation and some symptoms of dyslexia. All four of their children suffered from symptoms of MBD diagnosed independently by several psychiatrists, psychologists, neurologists, and specialists in learning difficulties.

1To be sure, I am focusing in this article on a group of patients suffering from MBD who were referred to me because they had been therapeutic failures; these patients had usually been previously diagnosed by more than one psychiatrist, in more than one institution, as “schizophrenic.”

Especially when examined in the light of the criteria formulated in DSM-III, only some of these patients can be considered schizophrenic: that is, to have shown delusions or hallucinations or a delusion, or to have done so for 6 months or more. Others, such as the 20-year-old girl discussed on p. 484 (who had been diagnosed as schizophrenic in eight hospitals) do not satisfy DSM-III criteria. Even in those who do fulfill DSM-III criteria for schizophrenia, however, the characteristic features of MBD dominate the clinical picture:

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Table 1. Personal and family history data relevant to MBD

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
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<tr>
<td>Difficult delivery</td>
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<tr>
<td>Slow development</td>
<td></td>
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<tr>
<td>Unexplained high febrile episodes</td>
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<td>Head trauma</td>
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<td>Head-banging, knee-elbow rocking</td>
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<tr>
<td>Convulsions</td>
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<tr>
<td>Hyperkinesis</td>
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<tr>
<td>Poor coordination: Gross and small</td>
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<tr>
<td>Difficulties with writing, reading</td>
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<tr>
<td>Left-handedness</td>
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<tr>
<td>Mirror writing</td>
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<td>Letter reversals</td>
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<td>Speech difficulties</td>
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<tr>
<td>Low stimulus barrier</td>
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<tr>
<td>Disturbance by bright lights</td>
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<td>Disturbance by loud noises</td>
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<tr>
<td>Visual disturbances</td>
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<td>Severe headaches</td>
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<td>Fainting</td>
<td></td>
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<tr>
<td>Impulsivity, sociopathy</td>
<td></td>
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<tr>
<td>Migraine</td>
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<tr>
<td>Epilepsy</td>
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</table>

This is in the presence of high intelligence on the part of the children as well as the parents. The 13-year-old son, for instance, suffered from problems in motor coordination and impulse control, as well as defects in auditory sequencing. He could not enumerate the months of the year, had problems of right-left discrimination and spatial orientation, and suffered from impulsivity and emotional problems in part secondary to these disorders.

In a second instance, my patient was a middle-aged woman presenting with ordinary neurorotic difficulties. However, she frequently had difficulty finding the right word or pronouncing it correctly, in the presence of superior intelligence. She had poor impulse control and typically got lost when driving to a new place. Her parents and grandparents had symptoms of dyslexia; her father was left-handed. Her husband suffered from petit mal seizures. Of four children, one, whom I also saw, had been independently diagnosed as schizophrenic, one girl had a severe behavior disorder, one adolescent son suffered from petit mal and occasional grand mal seizures. The younger daughter and son had been diagnosed as suffering from severe learning difficulties.

In a third instance, a man in his twenties was referred to me in consultation after several hospitalizations for what were considered to be schizophrenic episodes, but are probably best considered as a schizoaffective disorder. Among his presenting symptoms were being a troublemaker on the ward, engaging in pranks, and showing irritability and grandiose delusions. He turned out to be left-handed, and to have a history of letter reversals and mirror writing. He was unusually sensitive to alcohol and responded very poorly to phenothiazines. The patient's father was left-handed; his mother was schizophrenic with a variety of symptoms of dyslexia; the paternal grandfather, who was left-handed, was a manic-depressive. There were various symptoms of mental illness and MBD in the mother's family, and she had a psychotic sister. The patient responded well to a combination of Dilantin, Ritalin, and Tofranil.

A prototypical case in which MBD played a crucial role was that of a 20-year-old girl, diagnosed as schizophrenic, who was referred to me for consultation. She had been in eight hospitals during the previous 2 years and was being considered for treatment with dialysis as a last resort. As a matter of fact, her father had arranged for an arteriovenous shunt in preparation for it. Since the waiting time for the treatment was over a year, the father took the girl to another psychiatrist, who was also doing dialysis, but who had treated 30 cases of patients with MBD and psychosis and had taken an interest in two of my articles on MBD (Bellak 1976, 1977). He had the patient tested neuropsychologically and found that she suffered primarily from MBD with some psychotic phenomena that might or might not be considered schizophrenic. He referred the girl to me for further treatment.

I have seen a number of manics who suffered from MBD and responded well to Ritalin and Dilantin.
Aside from the data of the neuropsychological testing, the patient's history was fairly typical for MBD in adults. She had an early history of learning difficulties in the presence of above-average intelligence. She moved somewhat awkwardly and was very self-conscious. Part of her self-consciousness stemmed from her awareness of the symptoms of MBD during childhood. A history of violent outbursts had precipitated her series of hospitalizations during the previous 2 years. These involved a serious automobile accident, a suicide attempt with drugs, assaulting other patients in one institution, and throwing furniture in another. She had reacted unfavorably to phenothiazines, and her only hallucinatory and delusional experiences occurred when she was on Thorazine. She then believed that she was the Virgin Mary. She had responded favorably to Ritalin and similar drugs while in treatment with the referring psychiatrist. Unfortunately, she was forced to leave the private hospital where she practiced after a violent outburst and her attempt to overturn a piano.

In the course of her 3-month hospitalization, while she was under my care, I noted that violence always occurred on the day preceding her menstruation. All of her previous instances of loss of impulse control had also occurred premenstrually. At no time did she show a classical thought disorder or, in my acquaintance with her, any hallucinations or delusions. She often exhibited pressure of speech, and at times would be so self-centered that she did not engage in any real exchange but would only talk about whatever obsessed her at the moment. She could be interrupted, however, if it was forcefully done, and she was then quite capable of responding rationally and was definitely not deluded. She avoided other patients and was considered schizophrenic by hospital staff. They described her behavior as withdrawn. Since she sometimes talked to herself, they also thought she was deluded.

Initially, the patient responded well to Ritalin (5 mg t.i.d.), but soon after a visit by her father, with whom she had strong emotional conflicts, she became especially tense, speaking of wanting to die and kill others. I prescribed amitriptyline (Elavil 75 mg H.S.) and, when this failed to control her tension and anger, I added lithium (300 mg t.i.d.) at first, which was subsequently increased to a higher dose (600 mg t.i.d.). Since the patient's menstrual periods were irregular, I added Dilantin (100 mg b.i.d.) to her daily drugs to avoid the chance of another violent outburst. In one such episode, she hit a roommate over the head with a portable radio in the middle of the night, after having been teased by her. The roommate subsequently required several stitches to close her head wound.

The family history was typical; the patient was left-handed, as was one sibling who had learning difficulties, at times of loss of impulse control had also occurred premenstrually. At no time did she show a classical thought disorder or, in my acquaintance with her, any hallucinations or delusions. She often exhibited pressure of speech, and at times would be so self-centered that she did not engage in any real exchange but would only talk about whatever obsessed her at the moment. She could be interrupted, however, if it was forcefully done, and she was then quite capable of responding rationally and was definitely not deluded. She avoided other patients and was considered schizophrenic by hospital staff. They described her behavior as withdrawn. Since she sometimes talked to herself, they also thought she was deluded.

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The family history was typical; the patient was left-handed, as was another member of the family. One sibling had learning difficulties, attentional deficits, and poor coordination on neuropsychological testing. This case is quite representative of a series of patients, usually diagnosed as schizophrenic, who have come to my attention over the past 10 years. Closer examination reveals a history of MBD in childhood and residues of MBD in adulthood. My preferred diagnosis for this patient is actually: Personality disorder (with transient psychosis) with MBD.

The Concept of MBD

The most sophisticated concept and model of minimal brain dysfunction is probably that formulated by Eugene Arnold (1976). Arnold uses a Venn diagram to show the overlap of MBD, behavior disorders, and learning disorders. A hydraulic par-fault model is used to illustrate how behavior and learning disorders "spill over" when the level of total pathology from any combination of dysfunctions becomes high enough. When the symptom level rises sufficiently, a diagnosis of MBD is made. Thus, the diagnosis implies a psychophysiological disorder, with a multidimensional spectrum of causes.

In understanding the nature of MBD, it may be useful to compare it with the aphasic syndrome. Aphasia, in its sensory and motor forms (and in many variations on the two), presents a similar problem in which the symptoms are apparently vague and ill-defined. Yet, aphasia is a well-established concept in medicine. As a matter of fact, in certain types of MBD we find what seems to be a continuum of aphasia.

Ego Function Assessment of MBD

MBD needs to be operationally defined in adults as well as in children, and it can be so defined by the systematic assessment of crucial ego functions.

This is a technique of assessment I have previously described (Bellak, Hurvich, and Gediman 1973) for schizophrenia (another slippery concept!). In a way analogous to Arnold's concept of MBD, I described schizophrenia as a multifactorial psychobiological syndrome of different causes and pathogeneses, sharing as a final common pathway severe but variable disturbances of the functions of the ego. I have therefore found it useful to study schizophre-
nics in terms of their ego-function disturbances.

The functions can be graphed as in figure 1, which plots not only the patient’s current ego functioning but characteristic optimal and minimal functioning as well. In plotting such curves, one can see at a glance the amount of discrepancy that exists between ego functioning during periods of minimal and maximal stress, as well as the specific ego functions that exhibit the greatest discrepancy (Bellak 1979).

**Pathogenesis**

Kirk (1968) has proposed a theory of the etiology of schizophrenia based on perceptual deficits. According to her model, the child with unrecognized neurological impairment not only has a handicap of his own, but also creates a “role handicap” for his parents by not fulfilling their expectations or responding appropriately to their interactions. This can lead to a vicious cycle, particularly if the mother is herself schizophrenic and therefore offers the child even less consistency than might otherwise exist.

My own observations, presented below, are similar to Kirk’s regarding the pathogenesis of psychosis in the presence of MBD.

• Soft neurologic signs, if they include the acquisition of language and conceptual thinking, vitally interfere with adaptation to the world.

Figure 1. Ego-function profile of a schizophrenic patient with coexistent MBD before and after treatment with imipramine¹

![Diagram](https://example.com/diagram)

is confronted with the schizophrenic syndrome (or other psychoses). A thorough examination should include a careful family history for MBD; a personal history noting early life experiences with regard to school development; an attempt to ascertain whether there existed a low-stimulus barrier in infancy or any febrile disorder or developmental lag; a neurological examination for soft signs; an EEG (which is usually not helpful in most MBD disorders); neuropsychological testing (which often is helpful); a study of self-boundaries; and a careful evaluation especially of impulse control, stimulus barriers, and synthetic function, preferably on our 7- or 13-point scale of the ego function profile. A diagnostic label, "Schizophrenic syndrome related to MBD" or "Personality disorder with MBD" or "Schizoffective disorder with MBD," seems warranted.

**Therapeutic Implications**

Therapeutically, there are very definite implications if this hypothesis is substantiated. Such patients do not respond well to phenothiazines. On the contrary, they often react adversely to them, with panic, restlessness, and dissociation. Monroe (1970) discusses this point in relation to schizophrenics in whom he finds some cerebral dysfunctions. On the other hand, diphenhydantoin sodium (Dilantin) and diazepam (Valium) often are very effective. I have found methylphenidate (Ritalin) useful. Imipramine hydrochloride (Tofranil) combined with Valium is often very effective, as is lithium. Huey's (1974) experience in the treatment of adult schizophrenics whom he considers hyperkinetic coincides with mine.

If one explains to patients the nature of their disorder and symptoms, they usually respond with marked relief, whether they are psychotic or not. In a few schizophrenics, I found analytic exploration contraindicated because their synthetic function was impaired, probably due to the MBD; it led to increased problems with impulse control. Aside from strictly cathartic interpretations of aggression, help with reality testing, judgment, and an increasing of defenses is indicated. Often a change of life style and work to one with less sensory and social overload is advisable.

When an analytic approach is possible, special attention to disturbances of body image and self-image is important. The patient has usually been at least preconsciously aware of functioning "differently" and needs to understand this retrospectively and in the present. Feelings of stupidity due to manual or athletic inadequacy need to be worked through. Awkwardness, such as bumping into people and things, often is a special problem.

Feeling "different" to the point of catastrophic anxiety and feelings of being "crazy" and "stupid" result especially from the linguistic and conceptual problems of some patients with MBD. A person of normal or superior intelligence may not be able to tell time, at least intermittently. Unable to think of "stirrup," he may call it a "hop-up thing," and altogether present a word salad, which is not the result of a schizophrenic thought disorder, but rather the result of an aphasia-like difficulty. Decreasing anxiety, understanding of the symptom, and avoiding work under pressure, especially in people still in school, are helpful. Particularly for those still in school, including college, remedial training in reading, writing, concept formation, and eye-hand coordination may be indicated.

Preventive management is important in patients with MBD. They should be helped to avoid overload by any kind of stimuli or burdens or tasks, because of the difficulties they encounter with synthetic functioning or, as the vernacular would have it, in "getting it together." Since they frequently have a low-stimulus barrier and poor impulse control, they also need some counseling about conducting their lives with these liabilities, including neuropsychological counseling.

The prognosis will vary with the degree of MBD involvement as well as experiential sociopsychological variables interacting with it.

**Prevention of Consequences of MBD**

Whether the disorder is familial, congenital, or due to later brain damage, I agree with Handford that early recognition and special attention to MBD liabilities is essential for prevention. Such children should be protected against overload; they should not be overstimulated by cares, tasks, interactions, or noise. One should ameliorate even such stimuli as teething pain and attempt to curtail high-febrile episodes.

Children with MBD may often require special eye-hand coordination training and need help with reading, perceptual difficulties, and coordination problems. They should be placed in special classes, if necessary, and be helped to avoid the embarrassment of doing badly under time pressure, in order to avoid unnecessary traumatization and possible severe psychiatric disorders, such as the schizophrenic syndrome. Genetic counseling of parents with MBD, with regard to these precautions, is worth considering.
Conclusions

My long-stated hypothesis that the schizophrenic syndrome consists of many etiological and pathogenic subgroups is finding increasing support. One such subgroup may be characterized by soft neurological signs, probably justifiably subsumed under the label minimal brain dysfunction (MBD). While MBD may be due to prenatal, perinatal, and postnatal factors, I am especially impressed with the frequency of familial occurrence of MBD—in different degrees neurologically and with different degrees of psychiatric disturbance, including absence of major problems. Interaction of MBD and experiential factors probably plays a role.

Etiologically the question is raised whether MBD is in fact most often a genetic disorder, which would account for the genetic data increasing reported for a percentage of schizophrenics—schizophrenia then being the epiphenomenon. A reexamination of the incidence of familial MBD, as well as of the presence of MBD in patients diagnosed as schizophrenic in high-risk groups and patients identified in genetic studies, may help clarify this question.

The pathogenesis of schizophrenia as a function of MBD can be understood in terms of the severe affliction of the body image and the self-image, due to problems in spatial cognition. Linguistic and conceptual problems further complicate maturation, as seen in dyslexias. Impulse control is impaired cortically, especially since the low stimulus barrier leads to easy overloading and "overflow" not only strictly neurologically, but also emotionally. The low stimulus barrier also interferes with the organization of thought and language.

The diagnostic differentiation of this subgroup can be accomplished by a careful personal history and family history concerning MBD, and with neurological and neuropsychological testing. Such individuals are also likely to show more impulse disorders and often schizoaffective features. They frequently have a low stimulus barrier—noise on the ward will often make them violent and disorganized. Some people referred as schizophrenic may merely suffer from a personality disorder with transient nonschizophrenic psychoses and MBD. (MBD, in fact, may coexist and color any other psychiatric state.)

Therapeutically, such patients often come to attention because they do not respond to phenothiazines, or respond adversely. They usually react well to methylphenidate (Ritalin), imipramine hydrochloride (Tofranil), or diphenylhydantoin (Dilantin), at times in combination with lithium.

Psychotherapy has to deal with the specific effects of MBD on the self-image, including the secondary emotional ones of feeling stupid, awkward, and crazy.

Prognosis depends in part on the severity of the MBD.

Prevention in terms of avoidance of overloading and early cognitive training is important. Genetic counseling, especially of parents with MBD on both sides, is also worth considering.

The above findings are so far tentative and clinical, but important enough to warrant attention. Systematic research is still necessary.

Even if my hunch should eventually prove correct, I am sure it will hold true for only a small percentage of schizophrenics. I hope such a finding will encourage further search for subgroups of different etiology and pathogenesis.

References


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