Cognitive and Perceptual Differences Between Schizophrenics and Organics
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The question of how one distinguishes between psychological deficit of “functional” and “organic” etiologies is old and well known. Instead of having reached some definitive resolution, research on the problem has diminished in recent years, as have new theories and conceptualizations. In clinical psychology, the problem is primarily that of devising and validating procedures that discriminate between individuals with functional psychoses, particularly schizophrenia, and individuals with structural brain lesions. Indeed, early psychiatric conceptualizations of schizophrenia were couched in terms of dementia with no known organic cause. For Kraepelin (1919), the problem was one of distinguishing individuals with schizophrenia (or dementia praecox) from those with the more well-understood dementias.

It is the purpose of this article to review the literature regarding attempts to distinguish between cognitive and perceptual functioning in brain-damaged and schizophrenic individuals, and to offer certain substantive and methodological formulations and recommendations based on this review. The diminution of interest in differentiating schizophrenics and organics is unfortunate, since it remains a significant clinical and theoretical problem that has the potential of further resolution.

Theoretical Considerations

It is not the purpose of this review to attempt to summarize the massive amounts of literature concerned with schizophrenic thinking or with the thinking deficits of brain-damaged patients. The focus here is on the interface—the similarities and differences between the brain-damaged and schizophrenic patient. The problem, as most practicing clinicians know, is that despite much research effort, valid and reliable procedures for achieving this differential diagnosis are not currently available. The numerous theories of schizophrenic thinking, as well as the recent sophisticated research in clinical neuropsychology, have not provided the instrumentation needed to accurately distinguish schizophrenic from brain-damaged patients.

Traditionally, the problem has been treated as a dichotomy in which it is somehow important to determine whether the patient is “schizophrenic” or functionally psychotic on the one hand, or “organic” or structurally brain damaged on the other. This kind of thinking has become a bit old fashioned, and as will be seen, leads not only to oversimplifications but to blind alleys in regard to finding useful solutions to current diagnostic and modern etiological problems. Modern differential diagnostic research tends to be more oriented to process than categorization and is becoming more concerned with developing and understanding of specific perceptual and cognitive dysfunctions than in finding procedures that happen to provide group discriminations on a statistical basis. Thus, while a great deal of literature contrasting “schizophrenics” and “organics” is reviewed here, the implication should not be drawn that this approach is viewed as being particularly productive. It is simply that this approach was seen as productive at one time and thus gave rise to a large number of studies.

Similarly, terms like organicity and organic brain syndrome, while still a part of standard psychiatric nomenclature, are seen by many researchers and clinicians as being unduly vague and the product of a bygone age. While the term organicity implies structural damage to the central nervous system (CNS), the wide varieties of damage that can occur have rendered the term insufficiently specific to be really useful. When one considers the differential effects of lesions in various regions of the brain and in various sorts of neuropathological processes, the practical value of one global term like “organicity,” when used to describe the consequences of brain lesions, is open to serious question. In the area of differential diagnosis the view that “organicity” implies CNS disruption, while schizophrenia does not,
has turned out to be a naive one; there is ample evidence for CNS deficit in at least some schizophrenics. Although the question of differential diagnosis still looms large, it may be more productively formulated in terms of the various cognitive and perceptual processes associated with the disorders under study. In large measure, the unsettled state of knowledge in the area of differential diagnosis may be attributed to the naive and dichotomous thinking implied in the research designs of past decades.

Substantial progress might have been made with this problem had research been guided by unitary conceptualizations or models of the conditions under question. As is well known, there are numerous psychological models for schizophrenia as well as various conceptions of the psychological consequences of brain damage. When contrasted with each other, these models show varying patterns of overlap and conflict. To make the situation more difficult, much of the research on differential diagnosis involves no explicit model of brain damage or of schizophrenia, but rather consists of ad hoc comparisons of contrasted groups of patients with some procedure or set of procedures. Thus the research tends to have a somewhat chaotic nature, making it difficult to organize into bodies of positive and negative results. Nevertheless, an attempt will be made to organize the literature on differential diagnosis around theoretical models, with the understanding that the model may be implicit in the procedures used by the investigator and may not be explicitly stated.

* Cognitive Models

Historically, the cognitive models appear to be the first to contain some explanation of the difference between schizophrenic and brain-damaged patients. Kurt Goldstein was clearly the early leader in this area (Bolles and Goldstein 1938 and Goldstein 1939, 1944, and 1959). His formulation that the schizophrenic, like the brain-damaged patient, suffers primarily from an impairment of the abstract attitude gave rise to a major hypothesis concerning the thought disorder of schizophrenic patients. It was Goldstein's view that both the schizophrenic and the organic suffer from impairment of the abstract attitude, but that the schizophrenic makes different kinds of concretistic errors. Special tests, particularly sorting tasks (Goldstein and Scheerer 1941), were used to test this and related hypotheses, and a large amount of literature emerged over a time span of some 40 years (Chapman 1961, Chapman and Taylor 1957, Drasgow and Feldman 1957, Feldman and Drasgow 1951, Hanfmann 1939, Hanfmann and Kasanin 1942, Kasanin 1944, Kasanin and Hanfmann 1938a and 1938b, Rashkis 1947, Rashkis, Cushman, and Landis 1946, Tolor 1964, Vigotsky 1934, Wegrocki 1940, Zaslow 1950 and 1961, and Zubin and Thompson 1941). Cameron (1938a, 1938b, and 1944) also proposed a qualitative-cognitive theory related to his views about formal thought disorder in schizophrenia as a function of threat of social relations. The Cameron (Shimkunas 1972a and 1972b) and Goldstein (Wright 1975) views remain very much alive in the contemporary literature.

An important refinement of the Goldstein theory is to be found in the work of McGaughran and his group (Leventhal, McGaughran, and Moran 1959; and McGaughran and Moran 1956 and 1957). These investigators introduced the notion of "conceptual area" as an explanatory construct relevant to the reasoning of brain-damaged and schizophrenic individuals. Using an object-sorting task, they found differences between the sorting behaviors of paranoid schizophrenics and brain-damaged patients. The paranoid subjects characteristically sorted on an "open-private" basis while the brain-damaged subjects' sorting was "closed-public." In other words, the concepts developed by the paranoid subjects were overly generalized and obscure while those formed by the brain-damaged subjects were narrow but readily communicable to others. Although an attempt to replicate this work by Sturm (1964) failed, studies by Silverman and Silverman (1962) and Tutko and Spence (1962) tended to support the conceptual area scheme.
The concept of generalization has also played a major role in the cognitive model. Mednick (1955) attempted to explain both organic and schizophrenic deficits in terms of distortions in stimulus generalization gradients. There has also been a vast amount of research concerning a concept initially formulated by Cameron (1938a, 1938b, and 1944), the overinclusive thinking of the schizophrenic (Payne 1973). This issue has gone back and forth and many problems remain (Carson 1958, Garmezy 1951, Knopf and Fager 1959, Mednick and DeVito 1958, and Venables 1964), but the preponderance of evidence suggests that many acute schizophrenics tend to overgeneralize or overinclude on tasks requiring concept formation while chronic schizophrenics and organics undergeneralize.

Another branch of cognitive models focuses on the problem of language. While the language disorder of the schizophrenic has been studied in great detail, the language of the brain-damaged patient—with the exception of the restricted area of aphasia—has not been extensively studied. There are very few empirical studies of differences in the language of schizophrenics and organics. Chapman and Chapman (1965) found that schizophrenics regard words of similar meaning as synonymous more often than do brain-damaged subjects. In a followup study, Neuringer et al. (1972) asked brain-damaged, schizophrenic, and normal subjects if words which had both strong- and weak-meaning associates could be classed with the weak definition only, with the strong definition only, with both weak and strong definitions, or with neither of these. They found that brain-damaged subjects chose the “both strong and weak” alternative significantly less frequently than did schizophrenics and normals. Chapman (1960) reported that schizophrenics made significantly more literal than figurative misinterpretations of written statements, while brain-damaged subjects did the reverse.

It is unfortunate that such sophisticated approaches to schizophrenic language as those recently developed by Chapman (Chapman and Chapman 1965 and 1973 and Chapman, Chapman, and Miller 1964) and by S. Salzinger (1973) and K. Salzinger and Feldman (1973) have not been widely used in studies of differential diagnosis. Likewise, the recent applications of linguistic analysis to the study of aphasia (Goodglass and Kaplan 1972 and Goodglass et al. 1966) have not yet been employed to study differences in language between brain-damaged and schizophrenic individuals.

**Empirical-Psychometric Models**

Numerous investigators have attacked the differential diagnosis problem not with a theoretical formulation of cognitive and perceptual differences between brain-damaged and schizophrenic individuals, but with some particular procedure or set of procedures tried out on an empirical basis. A major subdivision of this model involves the use of tests in which speed or accuracy is the dependent variable. Thus there is a plethora of studies comparing organics with schizophrenics on the Bender-Gestalt test (Bowland and Deabler 1956, Brilliant and Gynther 1963, Freed 1969, Kramer and Fenwick 1966, Lacks et al. 1970, Reznikoff and Olin 1957, Robinson 1953, Rosenkrans and Schaffer 1969, Watson 1968, and Weinstein and Johnson 1964). Related tests, such as the Benton Visual Retention Test (Benton 1955 and 1963) and the Minnesota Percepto-Diagnostic Test (Fuller 1962 and 1969) have also been tried (Ascough et al. 1971, Fuller 1962 and 1969, L'Abate et al. 1962, Uyeno 1963, and Watson and Uecker 1966). The Trail Making Test (Reitan 1955 and 1958) has been widely used to compare brain-damaged and schizophrenic subjects (Boll 1974, Brown et al. 1958, Goldstein and Neuringer 1966, Korman and Blumberg 1963, Orgel and McDonald 1967, Smith and Boyce 1962, and Watson et al. 1969). Without going into detail, it can safely be said that the results of the speed and accuracy studies are equivocal and inconclusive.

A second branch of this model employs intelligence tests and other measures of cognitive and perceptual abilities, sometimes in an attempt to develop characteristic profiles for
different diagnostic groups. As most older clinicians will recall, attempts were made to develop such profiles using the Wechsler-Bellevue scales (Hewson 1949, Magaret 1942, Rabin 1941 and 1942, Rapaport, Gill, and Schafer 1945, and Wechsler 1941) and the Stanford-Binet scales (Altman and Shakow 1937, Babcock 1930, Davidson 1937, Harris and Shakow 1937 and 1938, Kendig and Richmond 1940, and Malamud and Palmer 1938). The generally negative or equivocally positive findings for intelligence test profile and qualitative index comparisons as discriminators of organics and schizophrenics are documented in studies by Bersoff (1970), DeWolfe (1971), McKeever and Gerstein (1958), Smith (1962), Spence (1963), Watson (1965a, 1965b, 1965c, and 1972), and Wentworth-Rohr and MacIntosh (1972).

Some investigators worked with quantitative multivariate methods such as factor and discriminant analysis (Greenhouse 1968 and Mcguire 1973). Burgess et al. (1970) used a multiple regression analysis to predict diagnosis from a battery of ability tests. Knehr (1962a and 1962b) used an ability test battery to compare acute schizophrenic, brain-damaged, and normal subjects. In analyzing his data, Knehr used both a multiple cutoff and a factor-analytic approach, concluding that factors containing relatively simple measures are best for distinguishing between brain-damaged and schizophrenic patients. Goldstein and Shelly (1972) used discriminant analysis in an attempt to distinguish between brain-damaged and primarily psychiatric patients based on a battery of ability tests. All of these multivariate studies reported positive results, but in the cases of the Burgess et al. (1970) and Goldstein and Shelly (1972) studies, the contrast made was between organics and psychiatric patients, and not specifically between organics and schizophrenics. The Knehr studies (1962a and 1962b) only contrasted acute schizophrenics with organics, a discrimination generally felt to be easier than is the case for chronic schizophrenics vs. organics. Berger et al. (1964) were unable to discriminate between their organic and chronic schizophrenic groups, thus illustrating this point. It is anticipated that with the increased use of high-speed digital computers, research of this type will be done more frequently in the future. In this regard, the use of stepwise multivariate procedures shows promise as a means of assessing the discriminating power of various measures (Dixon 1973).

The battery of neuropsychological tests originally devised by Halstead (1947) and expanded and extensively researched by Reitan (1966) and his various collaborators (Reitan and Davidson 1974) has been tried out on an empirical basis in various psychiatric settings. It was hoped that these instruments, although not developed in a psychiatric setting, might contribute to a refinement of differential diagnosis. The conclusions concerning the usefulness of this procedure are clouded by the fact that two groups who studied it in psychiatric settings reached differing conclusions.

A group of investigators at the St. Cloud VA Hospital (Watson et al. 1968a and 1968b) achieved completely negative results. The Halstead battery did not discriminate among the four groups studied (short-term hospitalized and long-term hospitalized organics and schizophrenics). These negative findings held for both statistical comparisons and clinical judgments made by expert interpreters of the battery. On the other hand, a group of investigators at the West Haven VA Hospital (Levine and Feirstein 1972) made essentially the same comparison with positive results. The differences between the St. Cloud and West Haven studies eventuated in a literature controversy in which it was implied that the findings of Watson et al. (1968a) may have been an artifact of chronicity. In order to clarify this matter, Watson (1974) reanalyzed his data by matching 34 pairs of organics and schizophrenics on chronicity of illness. Again, there were no significant differences. Levine (1974) replied that his reanalysis did not put the chronicity issue to rest because Watson's (1974) matched group data were apparently highly skewed, the schizophrenics selected for matching were not representative of the original total
sample with regard to length of hospitalization, and the criterion used by Watson for matching (no more than a 10-month difference) was not sufficiently precise.

There are a number of other studies that reflect the same sort of equivocality. Positive findings were achieved by DeWolfe et al. (1971), Fields and Fullerton (1975), Small et al. (1972), and Stack and Phillips (1970). In addition to the Watson group, negative findings were obtained by Klonoff, Fibiger, and Hutton (1970) and by Lacks et al. (1970). Watson (1971) attempted to replicate the DeWolfe et al. (1971) findings using their method of profile patterns analysis, but the replication failed. On the other hand, when Levine and Feirstein (1972) reanalyzed the Lacks et al. (1970) data by equating triads of subjects for chronicity, more significant group differences emerged than had appeared when chronicity was not controlled. While numerous post hoc explanations have been offered, definitive reasons for the apparently contradictory findings with the Halstead battery have not been established.

It might be suggested that clinical judgment should be emphasized in future differential diagnosis research since quantitative studies have not come up with anything definitive. Many clinicians feel that they know how to tell the difference between organics and schizophrenics and can do so effectively through combining their preferred diagnostic instruments with their clinical experience. For those who maintain this belief the following studies may be cited. Cohen (1955) obtained Wechsler-Bellevue profiles from 100 brain-damaged, psychoneurotic, and schizophrenic patients. These profiles were given to seven expert clinical psychologists who were asked to predict the diagnostic group from which each of the profiles came. They were unable to do so in any consistent manner. Hunt and Walker (1962) asked trained clinicians to review a series of Wechsler Adult Intelligence Scale (WAIS) Vocabulary and Comprehension subtest protocols and to classify them into groups of schizophrenics, neurotics, organics, mental retardates, and normal controls. While they exceeded chance level in their performance, so did advanced and sophomore psychology students. In a followup study, Hunt, Schwartz, and Walker (1965) found that the reliability of judges was good for ratings at the upper and lower extremes of pathology but less good in the middle of the range. Watson et al. (1968a) found that expert clinical neuropsychologists could not differentiate between organics and schizophrenics using the Halstead neuropsychology battery and the Wechsler scales. Thus, the evidence suggests that the answer does not lie in clinical judgment.

The empirical-psychometric and clinical judgment approaches appear to have run aground primarily because of a lack of consistent findings. As will be discussed, many studies reflecting these models also have major methodological difficulties. In any event, no test, test profile, or clinical scale has emerged that holds up as a reliable instrument for differential diagnosis. One might conclude that there seems little point in continuing to administer test after test with the hope of coming up with something useful, only to be faced with perpetual replication failures and inconclusive findings. The empirical approach, however, may have contributed one potentially important lead. It may be that many of the measures used in the past have been overly complex. The studies of Knehr (1962a and 1962b) cited above, as well as studies by Goldstein (1974), Goldstein and Halperin (1977), and Orzack and Kornetsky (1966), all suggest that simple measures differentiate better than complex ones.

Attentional-Motivational Models

There is a belief among many that psychological deficit in schizophrenia can often be attributed to such considerations as failure to attend to the task and lack of motivation, while the deficit of the brain-damaged individual is "real" and not simply a matter of unwillingness to attend or cooperate. A large body of research evidence has accumulated that strongly sug-
suggests that the performance of schizophrenics is highly susceptible to the influence of attentional and motivational variables. Earlier studies by Affleck (1954), Webb (1955), and Whiteman (1954) have been followed up by more recent and sophisticated studies such as those by Birch and Walker (1966), Price (1972), Shimkunas (1972a and 1972b), Stilson, Walsmith, and Penn (1971), and many others. Attentional deficits in schizophrenics have been documented in numerous studies including those of Chapman (1956a and 1956b), Chapman and McGhie (1962), Kopfstein and Neale (1972), Lawson, McGhie, and Chapman (1967), Levy and Maxwell (1956), McGhie (1970), McGhie, Chapman, and Lawson (1965), Silverman (1964), Spohn, Thetford, and Woodham (1970), and many others. Zubin and Sutton (1970) speak of 70 years of research with the reaction time experiment, a widely used means of studying attentional processes in schizophrenics. It would not be useful to continue reviewing this abundant literature at this point. Suffice it to say that there is ample evidence to suggest that the performance of the schizophrenic subject frequently does not reflect his actual capacity but is highly subject to the influences of distraction and motivational-affective factors. While brain-damaged individuals also have attentional difficulties and motivational lags, the extent to which they can alter their performance as a result of changes in these factors is generally felt to be far more limited than is the case for schizophrenics. However, there have been studies (e.g., Mayer and Coons 1960) in which it was found that the performance of brain-damaged patients could show such alterations. Thus while attention and motivation may have a particularly strong influence on the performance of schizophrenics, there is some evidence that such influences also obtain in the case of the brain-damaged. Indeed, the best tests for assessment of deficit in brain-damaged patients are those that are less susceptible to such influences and more directly associated with documentable neurological variables. It might be added that there is some evidence to the effect that motivating influences on the performance of simple tasks are no greater for schizophrenics than they are for nonschizophrenics (Benton, Jentsch, and Wahler 1960).

The attentional-motivational model would be quite attractive if it could in fact be shown that the apparent thinking and perceptual disorders of the schizophrenic can be unequivocally attributed to failures of concentration or lack of appropriate test-taking motivation, while deficits affecting the brain-damaged patient can be unequivocally attributed to some structural CNS defect. The fact that tests are taken by human beings, be they schizophrenic or brain-damaged, makes this goal exceedingly difficult to achieve. The attentional-motivational model has, however, served the valuable purpose of pointing out the need for seeking measures that are resistant to the influence of transient attentional and motivational states.

It may be useful at this point to suggest a distinction between two aspects of this problem. There are the well-documented defects in attention and motivation that characterize both brain-damaged and schizophrenic individuals. However, one must also bear in mind the methodological problem of the influence of attentional and motivational states on measures that are not intended to assess these variables. The idea that differences between organics and schizophrenics may exist in the areas of attention and motivation is not easily dismissed. Thus there is some objection to the use of measures in differential diagnosis that are subject to the influences of transient attentional-motivational states when it is not these states that are being evaluated.

Neuropsychological and Information Processing Models

Neuropsychological models may be defined as explanations in terms of levels of function in the central nervous system. The studies with the Halstead neuropsychological tests reviewed above do not involve a neuropsychological model in that these tests were used on an entirely empirical basis without consideration of the type or level of nervous activity assessed by the vari-
ous procedures in the groups studied. Typically, neuropsychological models contain a number of components including sensory input (coding of the input into meaningful stimuli), central processing (coding of the output in an organized way), and motor activity. There is often an interest in the relation between these behavioral components and physical structures in the nervous system. A neuropsychological theory might postulate that the schizophrenic patient has a defect at one or more of these levels while the brain-damaged patient's defects are associated with different levels. Such psychological activities as sensation, perception, attention, memory, problem solving, and motor execution are used to test the model. There is often the hope that the model will be ultimately fortified by the direct observation of some structural defect in the areas thought to mediate the defective level of function. While some success has been achieved in regard to brain dysfunction, in the case of schizophrenia such direct observation has not yet been achieved in any definitive sense.

The laborious process of building a neuropsychological model for distinguishing between brain damage and schizophrenia is far from accomplished. Various studies can be characterized in terms of the level of function at which the problem was attacked from the neuropsychological point of view, but there has been no unified systematic effort. Furthermore, many of the techniques commonly used are ambiguous. For example, difficulties in performing the Bender-Gestalt test may be attributed to misperception of the figures (input), an inability to coordinate perception with skilled movement (central processing), or a pure movement disorder (motor output).

Models based on information theory are in many respects similar to neuropsychological models. Ideally, the attempt would be made to seek differences in levels at which particular information processing components are performed by normals and schizophrenics. Instruments derived from the information processing theories of schizophrenia such as dichotic listening procedures (Hawks and Robinson 1971, Korboot and Daminani 1976, Payne, Hochberg, and Hawks 1970, and Spring and Zubin 1975), eye movement studies (Silverman 1964 and 1968), size perception studies (Price and Erikson 1966), and the like may prove to be quite useful. Tests that utilize the distinction made in signal detection theory between sensitivity and criterion may also be of value. The schizophrenic may have the same sensitivity or extent of information processing as the normal, but he may have a different criterion or willingness to report that he has processed the information. The situation may differ in the case of the organic.

The neuropsychological and information-processing models have the advantage of not assuming the level at which the deficit occurs, and so remain open to discovering whether the interference occurring in the brain-damaged individual is at the same level as it is for the schizophrenic. The cognitive and attentional-motivational models make the assumption that the difficulty is in central processing, but difficulties may also occur at the input or output levels in one or both of the groups under discussion. The task of building tests and designing experiments for discovering the location of the information-processing defect(s) in groups of brain-damaged and schizophrenic patients would appear to be a significant and fascinating challenge. This challenge has been expressed in a provocative comment by Dimond and Beaumont (1974):

Schizophrenia is a complex disorder, and requires amplification of our understanding on a range of levels. It has been clear that the description of the psychological deficit is a profitable level of analysis on which we should work. Further research centered around the operation of the dual hemisphere system and a model of the dysfunction of this system in terms of the phenomenon of disconnection could well lead to a broader understanding of the psychological problems with which the schizophrenic patient is faced. [p. 81]

These authors imply in a programmatic way that the cognitive and perceptual disorders of the schizophrenic may be characterized as a central processing defect in which the normal work-
ing relationship between the left and right cerebral hemisphere is somehow compromised. The empirical testing of this hypothesis remains a matter for the future.

It would appear that something can be learned from all of the models, but by virtue of their powerful methodologies and concepts, and perhaps of their deserving a fair chance, the information-processing and neuropsychological models appear to contain the best present hope of success. In terms of signal detection theory, schizophrenics and organics may differ in sensitivity or in criterion. Within the framework of neuropsychological inquiry, schizophrenia may be a form of disconnection syndrome with observable characteristics that differentiate it from other forms of neuropsychological deficit. The task involves the specific formulation of hypotheses and the construction and application of the appropriate tests.

Methodological Considerations

Three persistent methodological issues have haunted studies involving comparisons among diagnostic groups and have rendered many past investigations open to serious difficulties in interpretation. The first of them has to do with accuracy of diagnosis, the second with adequacy of sampling, and the third with how one interprets the subject's performance. Indeed, it is difficult to find differential diagnostic studies in the literature that cannot be faulted on the basis of one or more of these considerations.

Diagnostic Accuracy

The point at issue here involves the interpretability of diagnostic studies when the basis for the formation of the nosological groups being compared is unclear. It is probably fair to say that the basis for group selection in earlier studies was what physicians wrote into the medical records. If the art of psychiatric and neurological clinical diagnosis had been more advanced, this practice might not have produced substantive difficulties. However, there are two problem areas that bear directly on the present problem. First, there is a tendency among psychiatrists to overdiagnose schizophrenia and underdiagnose affective disorders (Gurland et al. 1974). Thus there is a strong possibility that schizophrenic samples formed on the basis of clinical diagnosis may contain unknown numbers of subjects who are in fact suffering from affective disorders. Second, the clinical neurological examination of the brain is a relatively primitive procedure and tends to underdiagnose brain damage (Filskov and Goldstein 1974; Klove 1974; Reitan 1966; and Russell, Neuringer, and Goldstein 1970). To aggravate the situation further, there is often no evidence to indicate that such examinations were even performed on the patients used in some of the earlier studies. This matter becomes particularly worrisome in the case of schizophrenic samples in differential diagnostic studies. Often, a "careful" chart review is substituted for the use of examinational procedures to rule out brain damage. Thus there may be an unknown proportion of patients with brain lesions in schizophrenic samples purported to contain only non-brain-damaged individuals.

More contemporary studies of schizophrenia sometimes employ explicit diagnostic criteria based on structured evaluations (Feighner et al. 1972 and Spitzer and Endicott 1975). Attempts are made to perform evaluations that are adequate for the purposes of the research rather than depending on "clinically useful" diagnostic examinations. In a corresponding manner, neurological evaluations are performed that sometimes use the advanced technology now available for the diagnosis of brain lesions. Such evaluations may include quantitative electroencephalographic techniques, cerebral angiography, radioisotope uptake studies, and computerized tomography. The important point is that the same procedures should be administered to all groups involved in the differential diagnostic comparison.

In the area of schizophrenia research, perhaps some of the best examples of studies of carefully diagnosed cases come from work done
at the New York State Psychiatric Institute (Kietzman, Sutton, and Zubin 1975, Zubin 1968, and Zubin et al. 1975). These investigators have developed and applied research diagnostic criteria that provide a set of definitions of the major psychiatric syndromes. These definitions are all based on objective data, usually obtained from direct examination of the patient with a structured psychiatric interview.

In the area of clinical neuropsychology, adequacy of diagnostic documentation has become a major consideration determining the acceptability of research findings (Reitan and Davison 1974). The problem lies in finding differential diagnostic studies in which it is clear that the documentation has met these more rigorous criteria from both neurological and psychiatric standpoints. Goldstein (1974) and Goldstein and Halperin (1977), for example, reported studies in which the subjects, all of whom were schizophrenic, were given careful neurological evaluations, but the establishment of the psychiatric diagnoses was accomplished by means of the usual clinical procedures. Barnes and Lucas (1974) and Matthews, Shaw, and Klove (1966) did comparisons of neurological and "pseudo-neurological" patients in which they obtained careful examinations for subjects from all groups involved in the study. While these studies provide good methodological examples, they do not involve specific comparisons of schizophrenic with brain-damaged patients. The study in which all subjects in both the schizophrenic and the brain-damaged groups receive structured interviews and psychiatric diagnoses based on objective criteria, as well as detailed neurological evaluations using some of the newer technologies for diagnosing brain dysfunction, apparently is still to be carried out.

**Appropriate Sampling**

The problem in the area of sampling relates mainly to those studies in which speed or accuracy are used as the dependent variables. When evaluating schizophrenics or brain-damaged patients it is possible to sample from a wide range of levels of deterioration in both groups. If groups are not somehow equated for level of deterioration, it is quite possible to obtain speed or accuracy differences between groups simply on the basis of any inequity that might exist. Thus differences noted may not reflect essential differences between brain-damaged and schizophrenic patients. The inconsistent nature of the research findings with speed and accuracy measures may be partially attributable to this phenomenon. "Significant" results appear when groups are not equated for level of deterioration, while "nonsignificant" results may sometimes emerge because the groups compared are equal with regard to deterioration level. One is tempted to suspect that for every case in which the "schizophrenic" group performed in a manner different from the "organic" group, one could have constituted groups in which such a difference would not be found. In effect, the formidable sampling problem in this area makes the findings of any single unreplicated study very tenuous.

This difficulty has highly negative implications for many of the studies of the 1940s and 1950s in which relatively unselected samples of schizophrenic and brain-damaged subjects were compared on the basis of their performance on measures such as the Bender-Gestalt or Trail Making tests. It is unfortunate that the bulk of these studies are so tainted by inadequate sampling and possible diagnostic error that their results, be they positive or negative, tend to be uninterpretable by present standards.

The sampling issue is further complicated by the tendency to draw research subjects from populations of institutionalized patients and frequently from long-term institutionalized patients. Zubin (1975b) has pointed out that in the case of schizophrenia, the long-term patient only represents the most difficult, recalcitrant case and does not represent the schizophrenic individual who, having responded well to initial treatment, never returns to the hospital. Neither does the long-term patient represent the largely unknown populations of schizophrenics in the community who never seek or receive
psychiatric treatment. Likewise, there are numerous individuals with brain lesions who do not reside in institutions. Thus our failures at discriminating may be to some extent an artifact of two considerations: a tendency to sample from the extremely pathological range of brain-damaged and schizophrenic individuals, and the confounding effects of institutionalization on cognitive and perceptual abilities. Needless to say, institutionalization implies the possibility of numerous other confounding conditions, particularly the long-term use of psychiatric medications and the often unknown effects of other somatic therapies.

Interpretation of Results

The difficulties mentioned above are augmented by recent formulations concerning the nature of the performance of schizophrenics on behavioral tests. Zubin (1975b) has described this transformation as a Kuhnian paradigmatic revolution (Kuhn 1970). Until recently, many investigators of schizophrenia made the implicit assumption that the schizophrenic adapted to a testing situation much like the normal person regarding such matters as attention to the task and motivation to perform optimally.

The “paradigmatic revolution” involves, in part, a movement away from this assumption and toward the rejection of many of the measures traditionally used in schizophrenia research. Such measures are felt to be ephemeral, easily modifiable from occasion to occasion, and thus nonrevelatory in regard to the discovery of characteristic traits of schizophrenia. For example, the widely used sorting tests do not clearly differentiate between deficits associated with stable characteristics and those associated with attentional-motivational states. Sutton (1972), Zubin (1975a and 1975b), and others have called for a fresh approach to studying schizophrenia by using measures that are not subject to attentional or motivational control (such as psychophysiological indices) or by using tests at which the schizophrenic does “better” than his control (thus minimizing the effects of attentional and motivational influences on performance). When the aim is to discriminate between brain-damaged and schizophrenic patients, a study that does not use procedures that incorporate one of the above two criteria will confront a serious measurement problem—particularly regarding data obtained from schizophrenic patients. Measures that are highly subject to transient motivational, affective, and attentional influences are hardly appropriate for the purposes of making a differential diagnosis.

The issue raised here may be more than methodological in that it makes the philosophic assumption that the most adequate diagnostic instruments are those that relate most directly to the essential nature of the condition to be detected, rather than to its epiphenomena. To quote Zubin and Sutton (1970), “In the biometric approach to the study of schizophrenia, an attempt is made to discover the organization and regularity characterizing the behavior of schizophrenics, excluding as far as possible uncontrolled variables such as motivation, attention, differential reinforcement history, etc.” (p. 261). Whether or not one agrees with this point, the lack of success in the past would suggest that increased attention should be paid to the characteristics of the measures used in diagnosis in general and perhaps differential diagnosis in particular. “Happening onto” a significant difference and reporting it is a practice that infrequently leads to findings of lasting value. “Measures” based on a patient’s reluctance to cooperate, misunderstanding of the instructions, or lack of attention to the task cannot serve as reliable diagnostic instruments. In these cases, the results obtained today may look nothing like those obtained tomorrow. Ideally, diagnostic tests should evaluate dispositional characteristics of the condition under study, as discovered and documented through basic research. The challenge of Zubin (1975b) and Sutton (1972), to discard the old methods and concentrate on procedures in which transient attitudinal or motivational states cannot be the major determinants of performance, must be answered in some way. As Sutton (1972) has pointed out, it is important to know whether a threshold difference
is in fact a threshold difference or a finding resulting from the schizophrenic subject’s failure to attend to the stimulus when it was presented; it is important to know if a slow reaction time is the result of psychomotor slowness or involvement in distracting ideational material at the onset of the stimulus. It is difficult to see how stable diagnostic procedures can ever be constructed out of tests that cannot resolve these ambiguities.

Substantive Considerations

Ideally, differential diagnosis research should keep pace with recent developments regarding the various conditions to be differentiated. In the case of differences between schizophrenics and brain-damaged individuals, many new developments are not always taken into account, thereby compromising the value of the research done. What is new and what is important are, of course, matters of opinion, but several developments can be mentioned that may have particularly important implications for differential diagnosis.

Neurological and Neuropsychological Deficit in Schizophrenics

Recent formulations have called into question the meaningfulness of the present topic. In its simplest form, the proposition is that it is difficult to discriminate between schizophrenics and brain-damaged patients because either all or some forms of schizophrenia are, in whole or in part, the results of brain damage. This view is supported by direct studies of neurological dysfunction in schizophrenics (Belmont et al. 1964, Brackbill 1956, Brackbill and Fine 1956, Hertzig and Birch 1966 and 1968, Houston and Bloom 1975, Klonoff, Fiebig, and Hutton 1970, Lilliston 1970, Mirsky 1969, Mosher, Pollin, and Stabenau 1971, Quitkin, Rifkin, and Klein 1976, and Rochford et al. 1970). It is also supported by the extensive longitudinal-developmental studies now commonly characterized as “vulnerability” or “high risk” research. One major group is led by Mednick (Mednick and Schuls-inger 1968) and another by Fish (Fish 1957, 1959, 1960, and 1971 and Fish et al. 1966). A third approach to the high risk or vulnerability problem has been proposed by Zubin (Zubin 1975b and Zubin and Spring 1977). These ambitious research programs all posit some kind of CNS defect in at least some kinds of schizophrenics. CNS damage or maldevelopment during gestation and early life is particularly important in the Mednick and Fish theories. Thus brain damage acquired during early life may be an etiological factor in schizophrenia, and does appear to increase the probability of becoming schizophrenic. In correspondence with these views, as indicated above, studies of schizophrenic adults have documented a relatively high incidence of neurological and neuropsychological defect.

These developments make the differences between schizophrenics and organics something of a moot point. Apparently, many individuals diagnosed as schizophrenic are also brain-damaged. While the syndrome of a schizophrenic condition in combination with structural brain damage has been recognized at least since the time of the elder Bleuler (1950), it was only thought to be a relatively rare condition associated with gross mental retardation. It was called propfischizophrenia, or schizophrenia “grafted” onto mental retardation. Now, the combination of schizophrenia and neurological dysfunction is viewed as a more common occurrence, and on the basis of longitudinal studies, the presence of neurological difficulties around the time of birth seems to be an important etiological factor. It appears that there must be some reformulation of the differential diagnostic question with which we began.

Typologies of Schizophrenia

Another development has centered around the search for an adequate typology of schizophrenia. While everyone would grant that there are different kinds of schizophrenics, no satisfactory system for classification has yet emerged. There has been a great deal of interest in the process-reactive distinction (Becker 1956, Garmezy 1968,
Herron 1962, Higgins 1969, Kantor and Herron 1966, and Kantor, Wallner, and Winder 1953) and the paranoid-nonparanoid distinction (Andrews 1972, Feigenberg 1964, Mirsky 1969, Wahl and Wishner 1972, and Zigler, Levine, and Zigler 1976). An additional distinction made by many is iatrogenic in nature—the distinction between acute and chronic schizophrenics. This distinction is often defined at least in part on the basis of length of institutionalization (Ricks and Berry 1970, and Tyrell, Struve, and Schwartz 1965). It is possible that individuals with certain characteristics tend to be institutionalized for lengthy periods or that the institutionalization itself produces these same characteristics. In any event, there appear to be systematic differences between patients who have been institutionalized for long or short periods, and length of institutionalization is a variable frequently considered in research studies (Berger et al. 1964, Feigenberg 1964, Korboot and Damiani 1976, Levine 1974, Levine and Feirstein 1972, Mednick 1958, Mirsky 1969, Venables 1964, Watson 1974, and Watson et al. 1968a).

**Typologies of Brain Damage**

In a corresponding manner, studies begun in the 1950s have shown systematic behavioral differences among different kinds of brain-damaged individuals. The pioneering work was done by Reitan and his collaborators (Reitan 1966 and Reitan and Davison 1974). Initially, this group was able to show systematic differences among patients with right hemisphere, left hemisphere, and diffuse brain lesions (Reitan 1955 and Klove 1959). They later showed that the age of the lesion (i.e., its acuteness or chronicity) has important differential effects (Fitzhugh, Fitzhugh, and Reitan 1961 and 1962). Russell, Neuringer, and Goldstein (1970) demonstrated significant differences among patients with lateralized and diffuse lesions as well as among patients with acute, static, and congenital lesions.

This progress in developing useful typologies within the general categories of schizophrenia and brain damage also calls for a redefinition of the original differential diagnostic question. It now becomes a matter of assessing differences among various types of schizophrenia and brain damage. In reality, current difficulties in differential diagnosis are not as acute for certain subtype comparisons as they are for others. For example, Boll (1974) readily discriminated between schizophrenics and subjects with lateralized brain damage using the Trail Making Test, while numerous other investigators using the same test on patients with mainly chronic diffuse brain damage were unable to make this kind of discrimination (Brown et al. 1958, Orgel and McDonald 1967, and Watson et al. 1969). Apparently, discriminating between the patient with chronic diffuse brain damage and the chronic schizophrenic patient has become the nub of the problem.

The parallel is striking between the diminution of specific symptoms of lateralization and localization as brain lesions become more static (Fitzhugh, Fitzhugh, and Reitan 1961 and 1962) and the lessening of florid psychotic symptoms as the schizophrenic patient becomes more chronic (Weiner 1966). Based on clinical folklore as well as on the common failure of research to find differences between chronic schizophrenics and chronically brain-damaged individuals, it would appear that the behavioral differences between brain damage and schizophrenia also diminish with increasing chronicity. This tendency appears to increase to the extent that members of both groups reside in common institutional settings. Since most of the research is done in such settings, the study of differences between diagnostic groups is almost necessarily confounded with a length of institutionalization variable.

**“Three-Way” Studies**

The newer formulations appear to have taught at least one lesson. In differential studies, it is no longer common to find comparisons between haphazardly selected samples of miscellaneous brain-damaged and schizophrenic patients. Subtypes are considered in one way or another. A

The three-way studies generally found that process or chronic schizophrenics were undistinguishable from the brain-damaged subjects while the reactive schizophrenics differed from both groups (Brackbill and Fine 1956, Lilliston 1970, Parsons and Klein 1970, and Tutko and Spence 1962). The temptation to speculate that process schizophrenics are really brain-damaged should be tempered, however, by a study by Davis, DeWolfe, and Gustafson (1972) in which process schizophrenics performed differently from brain-damaged subjects on several WAIS subtests, and by McDonough (1960) who found that normals, process schizophrenics, and reactive schizophrenics did not differ from each other on critical flicker frequency and spiral aftereffect, but they all differed significantly from a group of brain-damaged subjects. There is also the logical consideration that the lack of a difference in level of performance cannot be unequivocally interpreted as implying a common etiology.

In summary, while there have been numerous substantive advances concerning what is known about schizophrenia and brain dysfunction, the bulk of them are relevant within the framework of one or the other diagnosis and not to the interface between them. However, two major developments of relevance to differential diagnosis have emerged. First, the traditional distinction between organic and functional psychoses is not so clear-cut as it was once thought to be, and there appear to be many intertwinnings between the state of the central nervous system and the schizophrenic process. Second, there has been much progress in the direction of establishing useful typologies of schizophrenia and of brain damage. While distinctions presently made are not entirely satisfactory, they are sufficiently clear to indicate the serious limitations of studies that do not involve some consideration of subtypes.

Reformulating the Task

In consideration of the methodological and substantive criticisms offered above, one might wonder if there is any wheat to be separated from the chaff. A product in the form of a good set of procedures for discriminating between brain-damaged and schizophrenic individuals did not emerge. As stated at the outset, no fully acceptable procedure is currently available, and the review that followed may be viewed as an exegesis of why this is the case. On the other hand, it may be possible to extract a set of criteria for adequate procedures to be developed in the future. The stage may be set for a new beginning.

It may be useful to summarize the main points made:

1. Past differential diagnosis research has been greatly compromised by inadequate sampling procedures. There is no sure way of ascertaining that schizophrenic and organic groups are comparable, but care should be taken to equate groups on such variables as age, education, length of institutionalization, length of illness, and related matters. In general, the best way to deal with this issue is by making comparisons among well-defined subtypes of brain damage and schizophrenia and avoiding comparisons between unselected samples of miscellaneous "schizophrenics" and "organics." Multiple group designs, such as those used in "three-way" studies, are often useful.

2. Past differential diagnosis research has also been tainted by questionable diagnostic accuracy. On the schizophrenia side, it is recommended that samples be formed on the basis of structured, objective examinational procedures. On the brain-damaged side, diagnoses should be based on detailed neurological evaluations ad-
administered close to the time of the research testing. Greater attention should be paid to the diagnosis of neurological dysfunction in schizophrenics. How this should be done is not quite clear, but two suggestions can be offered. As indicated above, the incidence of early life brain damage is typically not recorded in the medical records of adult patients. Therefore, a chart review as the basis for group formation will almost inevitably lead to assignment of brain-damaged schizophrenics to the supposedly “non-brain-damaged” group. While greater certainty concerning formation of neurologically normal groups must await greater refinement of neurological diagnostic procedures, efforts should be made to use the best procedures currently available. At any rate, it would be wise to eschew the review of medical records as a means of ruling out brain damage in schizophrenic groups and instead develop research criteria for this kind of decision making. The second suggestion is to study the schizophrenic patient who is also brain-damaged rather than eliminate such individuals from investigations. Much might be learned from the inclusion of “brain-damaged schizophrenics” as a separate group in differential diagnosis research.

3. Tests selected for diagnostic research should ideally consist of measures of traits or stable properties of the conditions under study. In the case of schizophrenia vs. brain-damage studies, the major difficulty appears to be in obtaining measures that are not subject to the influence of transient attentional and motivational factors. In this regard, the methodological suggestions of Zubin (1975b) and Sutton (1972) for obtaining such measures should be heeded. For example, find tasks that the pathological group performs better than the normal group.

4. As a more general consideration, the entire matter of whether it is worthwhile to pursue differential diagnostic research in which diagnosis is the major independent variable is open to question. Might it not be more profitable to try to gain a more detailed understanding of the nature of the disorders under study? As knowledge of this type is acquired, differential diagnosis will become increasingly simple as a matter of course. For example, if Zubin (1975b) is correct that schizophrenia is actually a state of vulnerability marked by stress-provoked acute episodes and that this state can eventually be identified by specific markers not seen in other pathological conditions, then the presence of a history of episodic rather than stable or deteriorative illness in combination with the proposed markers will clearly identify the schizophrenic as such. In the area of diagnosis of brain damage, the quantum leap contributed by computerized tomography (Peterson and Kieffer 1976) has greatly increased our capability of directly observing even relatively subtle brain lesions. Perhaps in the future the establishment of definitive markers of schizophrenia in combination with advances in neuroradiology and neurophysiology will in large measure solve the diagnostic aspects of the problem under discussion in this review.

Potentially Useful Measures

The practicing clinician may be less concerned with the appropriate conceptual model or with the rigor of the methodology than with the actual tests used. The most commonly raised questions in the area of differential diagnosis focus on the choice of tests. In this regard, the more popular standardized psychological tests have not proved particularly useful. They tend to reflect one or more of the methodological problems outlined above, or research with them has failed to come up with anything definitive.

It may be of some value to offer suggestions concerning what procedures may work, in our opinion. The following should not be viewed as exhaustive in any sense, but as a sample of what looks promising. Holzman et al. (1974) reported that schizophrenics and their relatives show deviant eye tracking when following a pendulum. Because of the genetic implications of the findings, and the apparent refractoriness of this task to attentional-motivational influences, it would seem to be very worthwhile to run a comparison between schizophrenic and brain-dam-
aged patients. It should be noted, however, that while such a study may be productive, the whole problem of pathology of eye movement is a complex one (Yarbus 1967) and deviant movements are not only seen in schizophrenics but in neurological patients as well. Depending on the nature of the defect, the neurological disorder could involve the brain stem, the cerebellum, the vestibulo-cerebellar pathways, or various areas in the cerebral hemispheres (DeJong 1967). Thus the findings of Holzman et al. (1974) must be viewed with caution. Probably the most intriguing finding of the study is not the detection of deviant eye tracking in the schizophrenic subjects but its presence among the unaffected relatives.

The schizophrenic may have a kind of language disorder that differs from what is seen in brain-damaged patients. In essence, schizophrenic language may be different from aphasic language. Sophisticated linguistic techniques can be brought to bear here, such as those used by K. Salzinger (Salzinger and Feldman 1973), to evaluate the intelligibility of speech. He has used Cloze-type procedures on speech samples taken from schizophrenics in order to assess commonality and comprehensibility of speech. There may be important differences between brain-damaged and schizophrenic individuals in this area. Collins (1972) found that schizophrenics could make a finer discrimination between visual stimuli than normals, using reaction time to brief light pulses as his dependent measure. It seems quite likely that individuals with brain damage may not show this superior-to-normal performance.

Signal detection theory provides an intriguing and very testable hypothesis. The schizophrenic may differ from the brain-damaged patient in sensitivity or criterion. The actual procedure used is not crucial, but the flicker fusion experiment may serve as an example. A subject's fusion threshold may reflect his actual ability to discriminate the light flashes or it may vary with the degree of caution exercised in reporting a change. Sutton (1972) cites a study by Clark, Brown, and Rutschmann (1967) to illustrate the point that when schizophrenics are tested under conditions that do not permit the exercise of their typically cautious criteria, they may not differ from normals in threshold. The particular method used by Clark, Brown, and Rutschmann (1967) was a forced-choice technique. Thus if criterion can be held constant or factored out through the use of such methods, differences between schizophrenics and organics in sensitivity may emerge. The fact that brain-damaged individuals differ in sensitivity rather than in criterion has not been firmly established yet, and so we must again offer the precaution that only a hypothesis is being suggested and not an established finding. Indeed, Mayer and Coons (1960) reported findings that can be interpreted to mean that the failure of brain-damaged patients to perceive the spiral after-effect illusion can be viewed as a criterion problem. Under proper conditions of motivation, possibly inducing a criterion shift, they became more capable of perceiving the effect.

It is possible that the particular instruments used to make one subtype comparison may not be so appropriate for other comparisons. For example, sorting tests may distinguish between reactive schizophrenic and brain-damaged subjects (Tutko and Spence 1962) but critical flicker frequency might be a better test for discriminating between process schizophrenic and brain-damaged subjects (McDonough 1960) or between paretics and schizophrenics (Irvine 1954). Goldstein and Halperin (1977) found that a simple motor task was the best procedure in a battery of tests for discriminating between brain-damaged and non-brain-damaged schizophrenics, but a complex problem-solving test was the best task in the battery for discriminating between acute and chronic schizophrenics. This study used a stepwise multivariate analysis with the best test being defined in statistical terms as the one that by itself had the greatest discriminatory power.

Conclusions
In large part, this review has been a survey of the mistakes of the past, and individuals who have lived through the period covered may
raise some questions concerning the need to go through it once more. The major benefit we can see in doing so is that we are now better equipped to deal with the problem in a more sensible way. We have new conceptual and substantive tools with which to work. Perhaps the major conceptual “breakthrough” was a discovery of the nonproductivity of approaches directed toward binary classification into schizophrenic and brain-damaged groups. There appears to be a growing consensus that schizophrenia is based on some kind of CNS difference although its specific nature is far from understood. Thus there has been a shift in emphasis from diagnostic classification to studies of those underlying processes that produce the kinds of dysfunction seen in schizophrenic patients. Major experimentally based theoretical formulations have come to the fore, notably the “segmental set” theory of Shakow (1977), the immediacy hypothesis of K. Salzinger (1973), the arousal theory of Venables (1964), and the various “high risk” and vulnerability theories mentioned above. In the field of clinical neuropsychology there has been a corresponding attempt to understand and conceptualize disorders associated with structural brain lesions that goes beyond binary classification into “brain-damaged” and “non-brain-damaged” groups. Studies of interhemispheric differences (Dimond and Beaumont 1974 and Kinsbourne and Smith 1974) and systematic clinical investigations of patients with lesions of various types and in various locations (Luria 1973 and Reitan and Davison 1974) have radically altered clinical psychology’s traditional approaches to the assessment of brain-damaged patients.

It now seems clear that the solution to the differential diagnostic problem initially posed is not so much the development of some accurately discriminating procedure as it is a matter of reformulation. Perhaps we are making an oversimplification here, but our inability to solve the puzzle seems to have stemmed from our having asked the wrong question. A closer approximation to the “right” question must involve further investigation of the schizophrenic process as well as of the general and specific aspects of the behavioral consequences of structural brain lesions. Perhaps some important new developments will come from the relatively new field of clinical neuropsychology which has available to it a variety of tools for assessment of cognitive and perceptual skills (Lezak 1976) as well as a body of experiments involving the elucidation of relationships between various forms of brain dysfunction and behavior. Clinical neuropsychologists have already made preliminary attempts to apply their methods to the problem of psychiatric disorder (Dimond and Beaumont 1974, Flor-Henry 1969, 1974, and 1976, and Gruzelier and Venables 1973 and 1974). Perhaps oversimplifying again, these investigations utilize a neuropsychological model as described above in an attempt to discover underlying brain mechanisms that may be associated with the forms of dysfunction seen among schizophrenics.

The above considerations should not create the impression that the determination of whether a patient has a structural brain lesion or a schizophrenic condition is a trivial or inconsequential matter. Clinicians still commonly ask this question and often for good reason. It is important to know for treatment and planning purposes whether a patient has some deteriorative neurological illness, such as multiple sclerosis or Alzheimer’s disease, or a functional psychiatric condition. Diagnostic error in this area is still not uncommon, and while the new radiological techniques are quite promising, they have not eliminated the need for behavioral assessment. Furthermore, the peculiar sensitivity of patients with structural brain lesions to certain of the psychoactive drugs makes a correct differential diagnosis even more important than it was before the advent of these medications. The point we are attempting to make is that dichotomous thinking in terms of schizophrenia vs. organicity is not the best approach to the problem, and that it would be more productive to seek distinctions among a number of behavioral and neurological variables as they may pertain to patients we presently call brain-dam-
aged or functionally psychotic. For example, we might initially consider the problem of attentional deficits as they exist in various forms of brain damage and schizophrenia, and then go on to seek similarities and differences between these groups with regard to such deficits. From a diagnostic standpoint it is often ultimately necessary to return to a dichotomy. In order to treat the patient it must be determined that he has condition A and not B, which may resemble A in some respects but is in fact a different condition requiring different forms of treatment. However, the approach of distinguishing between conditions A and B on the basis of a sophisticated understanding of their underlying mechanisms would appear to be more productive than that of happening onto procedures that may on occasion yield statistically reliable numbers of correct binary classifications. It is hoped that the promise of more definitive knowledge based on the application of the recent substantive and methodological developments touched on here will rekindle research interest in this still significant clinical problem.

Summary

Distinguishing between brain-damaged and schizophrenic patients remains a significant clinical problem. However, substantive and methodological difficulties have led to discouragement and consequent loss of interest in this area of differential diagnosis. Several models for making this distinction were proposed: cognitive, empirical-psychometric, attentional-motivational, neuropsychological and information processing. In evaluating these models, three persistent methodological problems were identified: diagnostic inaccuracy, inadequate sampling, and difficulty in interpretation of the subject's performance. Various suggestions were made for resolving these difficulties through such means as equating samples for level of deterioration, utilizing modern, objective diagnostic techniques in the selection of samples, comparing subtypes of brain damage and schizophrenia, and using test procedures that have been shown to assess dispositional characteristics of the conditions under study, rather than their epiphenomena. It was suggested that recent substantive findings, mainly those involving neurological deficit in schizophrenia and detection of major behavioral differences among various subtypes of schizophrenia and brain damage, could be used in attempts to resolve the differential diagnostic problem under discussion. It was suggested that resolution of the methodological problems posed in combination with new substantive data concerning schizophrenic and brain-damaged patients may set the stage for a new start for investigating an old problem.

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