1

Research in
Clinical Diagnosis

JOSEPH ZUBIN

1. Introduction

By its very nature, the process of clinical diagnosis simulates a research process since it involves the testing of a variety of plausible hypotheses before a final decision is reached. Thus, in a sense, every clinical diagnosis is a research undertaking. Despite this, formal research into clinical diagnosis is of relatively recent vintage. It has two aspects: research into the decision-making process—leading to a diagnosis and research into the results of this process—the diagnosis itself. While the decision-making process itself is now being investigated experimentally, we shall limit ourselves to research in diagnosis itself with special reference to its improvement.

Clinical diagnosis in the mental disorders has a long past, reaching back nearly 34 centuries to the ancient Hindu scriptures (Caraka Samhita, 1949), but its history is rather brief, dating back only to Pinel and Esquirol at the beginning of the nineteenth century, who were among the first systematizers in psychopathology. Research in clinical diagnosis was hardly begun before the last quarter of the nineteenth century, after Kraepelin’s systematic nosology took hold.

A historical survey of the development of research in clinical diagnosis will have to be left to historians. Here we will be concerned primarily with the current status and future of research into clinical diagnosis.

Perhaps the most apt characterization of the continued search for improvement in diagnosis was made by Heinrich Kluver (personal communication), who wrote:

I guess something has to be done in re the diagnostic discrepancies, etc. you speak of. As things are, attempts are still made, as [A.] Hoche in Freiburg once expressed it, “To clear turbid solutions by pouring them continually from one glass to the other.”

2. Interest in Diagnosis

For a long time, clinical diagnosis was in the doldrums; because no effective therapies were available, it was a mere academic exercise, even when the diagnosis was
undisputed. The first breakthrough came with the discovery of the cause of general paresis, which enabled the clinician to appeal to an objective criterion, the Wassermann test, to validate the results of his clinical interviews, observations, and history of the patient. A similar breakthrough occurred subsequently when the cause of pellagra with psychosis was discovered. These advances are hardly remembered as achievements of psychopathology today because the disorders are rare and are no longer regarded as part of psychopathology. Similar notable discoveries in etiology were made in the field of mental retardation, and, although quite a number of inborn errors of metabolism and other genetic abnormalities have been discovered, the proportion of mentally retarded conditions for which the etiology is known is still very small. In the etiology of the mental disorders, especially in the functional disorders, little if any dramatic progress has been made, although the research efforts have been prodigious. We are still abysmally ignorant. Despite the conquest of general paresis, pellagra with psychosis, and several mental defect disorders, interest in diagnosis remained at a low ebb until the advent of the somatotherapies in the 1930s and 1940s. Since these therapies seemed to be most suitable for only certain types of patients, interest in diagnosis was rekindled, but choice of treatment, important as it is, is only one of the purposes of diagnosis. The ideal diagnostic nomenclature should maximize the communicative utility of the diagnostic label, optimize its prognostic potential and its explanatory powers in relation to etiology, and, of course, optimize choice of treatment. To attain this ideal is the goal of research in clinical diagnosis.

3. Defining Mental Health

The first set of problems that one faces in entering this field is the absence of defining boundaries between mental health and mental disorder and, within the vague boundaries of the mental disorders, the nebulous distinctions between the various disorders. The response to this chaotic condition has varied from Szasz's (1961) denial of the existence of mental disorders to the attempts of men like Leonhard (1936, 1959) to subdivide the disorders into minute subgroupings. Leonhard makes meticulous distinctions within diagnostic categories based on hypothetical brain dysfunctions. Szasz, on the other hand, claims that mental disorders are based on arbitrary distinctions between essentially normal but nonconforming individuals made by a psychiatric establishment bent on preserving its status and powers. Apparently, some clinicians and researchers can live with the fluid boundaries like fish in the ocean, while others, rather like groundhogs, require more specific boundaries.

Perhaps the most important concept requiring definition is mental health itself. No one would quarrel about the diagnosis of a severely chronic schizophrenic whose condition is of long duration, even though the etiology of this disorder would involve considerable controversy. But the vast majority of those seeking relief from mental disorder and those affected with it but who have not accepted the patient role do not fall into this extreme category. How can one arrive at a definition of mental health and mental disorder which though not ironclad can nevertheless be useful, or do we not need such definitions? Could we not hide behind the definition adopted by some practitioners that mental disorders are defined by the types of individuals whom mental health professionals serve? This, indeed, is the definition suggested by Linder (1965).

It is true that even in a more basic science like biology definitions of such funda-
mental terms as “species” remain open-ended (J. Huxley, 1940). Nevertheless, it is important to define the field of interest even though the boundaries remain vague. If we accept the notion that man is bombarded continually by environmental challenges emanating from the ecological niche which he occupies (physical, social, and cultural parameters of the niche) and that his health depends on his capacity to maintain his homeostasis in the face of these challenges, then “health” can be defined as the degree to which he maintains his dynamic balance and “illness” is its opposite.

4. Taxonomic Problems

The taxonomic problem of classifying the types of illnesses which can arise from the exogenous and endogenous challenges that face man’s adjustment is quite complicated. Simple description is not enough, since similar topographical descriptions can arise from a variety of causes. We need to dig deeper under the surface into the etiology of the disorders. Just as simple description of animals is not sufficient for a good taxonomy unless the developmental and evolutionary aspects of the animal’s structure and behavior are uncovered, so in the case of the mental disorders the sources of the maldevelopment must be understood for a complete classification. Unfortunately, we know very little about the etiology of most mental disorders, and, furthermore, once the etiology becomes known, the disorder is lost to psychopathology, as was the case with general paresis, pellagra with psychosis, and various types of mental retardation. But what can one do when facing the disorders of unknown origin? One solution is to limit oneself to description alone, but, as was pointed out previously, this is not enough. A better solution is to develop “as if” etiologies in the form of scientific models of etiology. This has been done elsewhere (Zubin, 1972), and the most useful models now dominating research are (1) ecological, (2) developmental, (3) learning, (4) genetic, (5) internal environmental, and (6) neurophysiological. Each of these models is so broad in scope that entire schools of psychopathology can pass through its portals without touching. Furthermore, none of them works in isolation. In order to provide for interaction between them, a super-model or second-order model has been suggested, viz. vulnerability (Zubin, 1976). This model stipulates that when a stressor of sufficient magnitude impinges on a person, a crisis develops which, depending on the degree of vulnerability of the person, either is dissipated so that the individual can return to his precrisis status or, if the person is sufficiently vulnerable, develops into an episode of disorder. It further postulates that the disorder is time limited and that when the stressful situation is overcome the individual will return to his preepisode status (Zubin, 1976). The causes of this vulnerability are to be sought in the six etiological models described earlier, and each of these models can provide markers to identify the vulnerable individuals. (Zubin & Spring, 1977).

Why is it so difficult to deal with diagnosis in psychopathology? Psychopathologists tend to look up to their colleagues in the field of the physical disorders as being more advanced in their nosology. The advantages that physical bases provide for classification are obvious. But how systematic is the classification of so-called physical (nonmental) disorders? To begin with, the first classification systems were merely based on individual symptoms or signs. The development of the concept of a syndrome as a concatenation of symptoms and signs is due to Sydenham in the seventeenth century.
Then, as science and medicine advanced, the classification of disorders followed the new discoveries. When anatomy yielded its secrets through dissection, morbid anatomy became the basis for classification; then followed cellular pathology in the wake of the microscope, and bacterial infection following the work of Koch and Pasteur; and thus the concept of disease entities each produced by a single etiological agent was born. Since then, electrophoresis, chromosome analysis, and electron microscopy have provided new criteria for classification based on biophysical structures, genes, and molecules. Ironically, the new techniques did not make a clean sweep of the status quo in nosology, but, like the English language, absorbed all the new invaders and produced a crazy quilt of classification embodying both the old and the new. Thus there are still diseases based on symptoms, syndromes of symptoms, morphological and physiological anomalies, morbid anatomy and physiology, bacterial or other exogenous agents, biochemical defects, genetic abnormalities, ultrastructural abnormalities, and so on and on. Any attempt at finding a unifying concept in current nosology is doomed to failure because of the congeries of methods used in classification. In the field of psychopathology, the nomenclature could be simpler since we deal primarily with symptoms and signs and their combination into syndromes. Had the various DSM's and their progenitors limited themselves to description alone, diagnosis might have indeed been simpler. However, various admixtures of description and presumed etiology tended to be the basis for diagnosis in the past. It is one of the advantages of the newly proposed DSM III that putative etiology is kept out of the diagnosis. However, here too the advances made in genetics, biochemistry, and biophysics are beginning to have their effect, although because we have not yet found the etiology of any of the mental disorders we must depend on behavioral syndromes. But the behavior of the patient is determined not only by his mental disorder but also by his underlying premorbid personality, and it is this mixture which he presents at the time of admission. If we could separate the behavior due to the mental disorder from the behavior due to his premorbid personality and to their interaction, we could recognize the focal disorder in isolation from its surround and probably find this factor characteristic of all similarly afflicted patients. What we perceive, however, is not the effect of the focal disorder alone, but the effect of the illness, which reflects the premorbid personality and the focal disorder and their interaction. This is why no two schizophrenics are alike—their focal disorder may be the same but their illness is different. The relation between premorbid personality and psychopathology is still moot and involves such thorny issues as the distinction between "trait" and "state."

Elsewhere (Zubin, 1965) I have examined the relationship between premorbid personality and psychopathology and have pointed out that logically they might be related in one of three ways: (1) As identical—as Freud might have viewed them. (2) As independent—as Kraepelin might have thought. Or (3) as interacting—as Adolf Meyer might have thought. But it might be valuable to adopt the assumption of independence or the null hypothesis and study premorbid personality as an independent variable or set of variables. In fact, studies in Lund, Sweden, have found that the premorbid personality does not relate to occurrence of mental disorder, but does color it once it appears. Consequently, we must begin a determined effort to study premorbid personality if we are to clarify diagnosis.

Turning now to the current scene, what has research in clinical diagnosis accomplished?
If we accept the dictum of the philosophy of scientific taxonomy following Alexander Wolf (1929) regarding the need for mutually exclusive and exhaustive categories based on the most important characteristics of the thing concerned (the disorder) and on the actual relations between them and accept the importance of having "natural" rather than "artificial" classification systems, then our progress in attaining our goals has been but little. We have, however, made some progress. In the first place, when the unreliability of the clinical interview method became apparent as a result of investigations of the interview after World War I, attempts were made to develop self-rating inventories and scales to be filled out by the patient and checklists and rating scales to be filled out by the interviewer. The self-reporting instruments have the advantage of eliminating interviewer bias and interpretation by putting the onus on the individual's response rather than on the response of the interviewer to the patient's behavior. That this strategy does not guarantee a better result is, of course, quite obvious. The self-reporting inventories have generally not been found to be so useful for the classification of inpatients, although they have been found quite useful in minor disorders, primarily in dealing with student populations and in surveys of the general population.

5. Diagnostic Techniques

It is interesting to note that in a recent study Dohrenwend and his colleagues found that the common denominator in such self-reports seems to be related more to Jerome Frank's (1973) demoralization factor than to psychopathology.

The checklists and rating scales used by interviewers were not provided with standardized methods for obtaining the information and therefore were not always comparable from patient to patient. To obtain a more systematic method for the collection of the information, the clinical interview itself was brought under control by providing a systematic structured format for it. The first step was to standardize the usual mental status examination (Mental Status Schedule, Spitzer, Burdock, & Hardesty, 1964). The questions to be asked of the patient were specified in a proper sequence and directions for coding the responses were laid down. The mental status schedule, which had been the psychiatrist's mainstay, was converted into this kind of systematic structured interview in our laboratory, yielding high reliability in scoring of items, as well as considerable validity. This technique underwent a series of modifications based on experience gained in the various studies conducted under the aegis of the Biometric Research Unit of the New York State Department of Mental Hygiene (Zubin, 1972). We worked with three types of interviews: (1) a nonprobing Structured Clinical Interview (SCI) by Burdock and Hardesty (1968), (2) a medium-probing Mental Status Schedule (MSS) by Spitzer et al. (1964) and the Psychiatric Status Schedule (PSS) by Spitzer, Endicott, Fleiss, and Cohen (1970), and (3) a deep-probing Present State Examination (PSE) by Wing, Birley, Cooper, Graham, and Isaacs (1967). These interviewing methods have demonstrated their value in two major studies—the US-UK Diagnostic Project (Cooper, Kendell, Gurland, Sharpe, Copeland, & Simon, 1972) and the WHO Pilot Study in Schizophrenia (World Health Organization, 1973). The US-UK project was initiated to determine why the national statistics show such disproportionate frequency of affective disorders in the United Kingdom and of schizophrenia in the United States. When the newly developed systematic structured interviews (Gurland, cited in Zubin, Salzinger,
Fleiss et al., 1975) were applied to samples of patients admitted to hospitals in the two countries, the cross-national differences turned out to reflect different diagnostic practices of psychiatrists rather than differing characteristics of patients in the two countries. Whereas the US-UK project limited itself to two cultures but investigated the entire panoply of mental disorders, the WHO study limited itself to one disorder but investigated its incidence, form, and course in nine different cultures. This investigation found specific syndromes of schizophrenia ubiquitously distributed in developing and in advanced cultures from Ibadan, Nigeria, to Washington, D.C.

Other advances in descriptive psychopathology have been (1) the use of computers that simulate the clinician’s decision processes and arrive at a diagnosis (Spitzer & Endicott, cited in Zubin et al., 1975; Wing, Cooper, & Sartorius, 1974), (2) the provision of mathematical methods for clustering individuals with shared psychopathological characteristics into more homogeneous subgroups (Fleiss & Zubin, 1969; Wing & Nixon, 1975), and (3) the development of behavior analytic descriptions of patient characteristics (Kanfer & Saslow, 1969; Salzinger, cited in Zubin et al., 1975). Rather than emphasizing the presence of symptoms per se, behavior analytic description relates deviant behavior to environmental contingencies that initiate and sustain it. Description is therefore focused on aspects of the individual and his environment that are of immediate relevance to behavior modification therapy.

A dispassionate view of where we stand in descriptive psychopathology today leads to the conclusion that we have made considerable progress and perhaps gone as far as we can go but that this is still not far enough. Through the use of systematic structured interviews and operational criteria for selecting a diagnosis a high degree of agreement on diagnosis can be attained. We can now objectively describe the characteristics of patients we deal with so that replication of basic research and treatment findings on similar patients is possible. Moreover, we can demonstrate that individuals suffering from such syndromes of psychopathology as schizophrenia can be found in parts of the world that differ widely in cultural and ecological conditions.

However, we cannot presuppose that reliable description of a syndrome implies valid understanding of its underlying cause. We need to do more than describe, since description never cured anyone, nor did it by itself reveal etiology. Although treatment can often not await a thorough knowledge of etiology, the discovery of an effective cure for any disorder is less likely to result from chance than from an understanding of factors that cause the disorder. As in the classification of organisms, in which taxonomy looks to common origins of descent, so in the classification of disorders we look for common causes producing the disorder. But how can progress be made when with but few exceptions we are abysmally ignorant of the causes of mental disorder and even more poignantly ignorant of the efficacy of the treatment? When faced with such ignorance, one can only contemplate possible or “as if” causes, formulate them into parsimonious scientific models, and proceed to test the hypotheses they generate (Zubin & Spring, 1977).

We have already indicated the types of scientific models which can help establish a more basic aspect of the etiology of the mental disorders.

What needs to be done further? We cannot be satisfied with reliability alone—we must also ask whether our judgments are valid. Here is where we flail our wings in a vacuum at present. How can we improve validity?
6. The Problem of Validity

Of the four types of validity—predictive, construct, concurrent, and content—we will deal only with the first two, predictive and construct validity, since concurrent validity is really a type of reliability and content validity refers to whether the measure covers adequately the entire area under investigation.

6.1. Predictive Validity

Outcome and course of the disorder provide criteria for the predictive validity of the diagnosis. Thus, if the diagnosis of affective disorder leads to a more rapid release than for schizophrenics, this can be taken as a vindication of the diagnosis since this is the outcome we would expect.

6.2. Construct Validity

What else do we expect patients in a given category to have in common besides similarity in outcome?

These expectations have been embodied in a set of scientific models for etiology and have been described earlier in two groups: field theory models, consisting of the ecological developmental and learning theory models, and molecular or biological models, consisting of genetic, internal environmental, and neurophysiological models.

Accepting the genetic model, we would expect that diagnostic system to be most valid which showed the highest transmission rate of schizophrenia in relation to consanguinity. As a matter of fact, a test of the American and British diagnostic systems as well as a Swedish one—Essen-Mölle's—was made in Gottesman and Shield's (1972) twin study, and the highest transmission rate was found in Essen-Mölle's system.

Accepting the ecological model, we would, for example, expect to find the highest rates of schizophrenia in individuals occupying the deprived, isolated, and oppressed ecological niches in our cities—and this indeed turns out to be the case.

Accepting the developmental model, we would, for example, expect the highest rates among those who according to Sullivan never had a chum, i.e., had deviant friendship patterns in their adolescence.

Accepting the learning theory model, we would expect that families with deviant rearing patterns would give rise to more psychopathology.

Accepting the internal environment model, we would expect to find biochemical indicators such as monoamine oxidase differentials in schizophrenics and their relatives.

Accepting the neurophysiological model, we would expect to find deviant patterns in the processing of information input reflecting deviation in attention and arousal. These hypotheses have been tested by such techniques as reaction time, sensory integration, pupillography, skin conductance, and evoked potentials.

Each of these scientific models would require some modification of our diagnostic procedures in order to provide data for the construct validity of the diagnosis. Thus the genetic model would require more careful interviewing methods of blood relatives to determine family incidence of mental disorder. The ecological model would require a more careful examination of the ecological niche the person occupied, stressing not
only the generally accepted parameters such as socioeconomic status and crowding but also the more subtle aspects of available opportunities for growth, privacy, etc. The developmental model would place more emphasis on premorbid history, and the learning theory model would require more careful surveys of family structure. The internal environmental model would require a careful survey of body chemistry and metabolism along the lines dictated by recent findings, while the neurophysiological model would require the application of the specific laboratory techniques found to be differential between patients and normals as well as between patient groups (Sutton, 1973).

7. Problems in Research in Diagnosis

In addition to the problems presented in the general overview described thus far, there are certain specific problems in research in diagnosis which require further investigation.

7.1. Determination of “Casedness”

Much of the research in diagnosis has dealt with individuals who come willingly or are brought for help and hence may be considered, with rare exceptions, as probably being mentally ill. The function of the examiner in such instances is to determine the particular type of disorder that is present. Rarely does he have to decide whether the individual is a “case” or not. In community surveys, the very opposite situation holds. The number of cases is relatively rare, but the decision whether a given individual represents a case or not must be made very frequently. We have made considerable progress in differential diagnosis in the studies of patients, but have made little if any progress in determining casedness. This is where more research is needed, especially longitudinal studies, in which preliminary indications of casedness can be followed up. More indirect approaches such as the findings of markers which do not indicate currently present psychopathology but characterize individuals who may later develop a disorder are needed (Zubin & Spring, 1977; Zubin & Gurland, 1977).

7.2. Dimensionality vs. Typology in the Distribution of Mental Disorders

If two categories of patients are to be differentiated, some discontinuity must exist which separates them into two distinct groups. Since there is no single characteristic which identifies a given disorder (excepting general paresis and a few other disorders for which a single cause has been found), we must look at a pattern or syndrome of characteristics or dimensions as a basis for classification. If we consider the distribution of groups of individuals along the dimensions of a given syndrome, we might consider the following possibilities. Limiting ourselves to three dimensions for the sake of clarity, although the argument could be extended to n dimensions, the population may be distributed normally or symmetrically over the three dimensions, so that there are no troughs or valleys of rarity in the three-dimensional surface. In that case, it would be difficult to find natural boundaries for separating the total population into subgroups. On the other hand, if troughs exist, so that clusters of individuals can be found between pairs of troughs, it would be easy to identify these clusters into subgroups. However, even if there are no natural cleavages in the dimensions,
there might be certain boundaries such as arise in hypertension which are recognized as separating different types of individuals, even if there are no points of rarity or cleavage in the three-dimensional space. Last, there may be certain qualitative characteristics that separate groups such as combinations of hallucinations and delusions. Which of these possibilities now characterize the distribution of the mentally ill is difficult to determine, but unless we can find boundaries between groups our definitions of disorder categories will remain nebulous. The problem becomes even more complicated because the boundaries may reflect the patient’s behavior, the behavior of the examiner in evaluating the patient’s behavior, or the norms imposed by society.

The advent of the drug era has brought a search for subtypes of the various diagnostic categories which have similar responses to specific drugs. This has opened up a demand for clustering technique which would identify the individuals belonging to the various homogeneous subtypes by means of their common profiles. The frequent failure to replicate such findings has resulted in the conclusion that blind empirical searches for such subtypes without any underlying testable hypotheses constituted a vain attempt at lifting oneself by his own bootstraps. Fleiss and Zubin (1969) have pointed out that we need better mathematical models for typology before wasting our time in uncharted courses.

Furthermore, the differences between the dimensional approach and the typological approach are often vitiated by the factor analytic approach used in determining the dimensions, since this approach makes assumptions which nullify typological assumptions. For example, typology thrives on nonlinear relationships between variables and on discontinuous nonnormal distributions, the very assumptions that factor analysis contradicts. Consequently, it is foolhardy to expect to find types when the assumptions they are based on are not compatible with the techniques used to find the types.

There is another point of view, however, which would indicate that the conflict between dimensionality and typology is ephemeral. It can be best illustrated by an incident during a prior conference on diagnosis (Katz, Cole, & Barton, 1967). The same question had been raised then, over 10 years ago, and the discussion had lasted until late in the evening. I couldn’t fall asleep that night, and, waking at 4 a.m., I turned on the TV only to discover that I had tuned into the Farmer’s Hour and was listening to a lecture on pomology—how to tell good from bad apples. Apparently, the earlier method was to have an apple knocker tap the apple with his knuckles to determine whether it had too much core and whether it was immature. Today, the reporter indicated, the method consists of conveying the apples on a conveyor belt under two sets of monochromatic lights. The amount of light absorbed is read off a dial which automatically determines the fate of the apple. Apparently, the typology of the apple knocker has been converted to a dimensional measure for classifying the apples. Upon further thought, I concluded that actually this dimensionality is probably the reflection of the genetic makeup of the apple seed—again a typological classification into good and bad genes. But genes accomplish their work by secreting certain biochemical substances (amino acids) in smaller or larger quantities. Again, the typological genetic classification has been altered to a continuous dimensionality. And so on. . . . Apparently the state of the art determines whether typology or dimensionality is to be preferred for classification, and there is no essential difference between the two.

Furthermore, mathematical techniques can probably be developed for converting a typological classification into a dimensional one and vice versa.

Currently, the battle between typology and dimensionality is seesawing with no
definitive superiority by either approach. Thus the dimensional approach which has been championed by factor analysts like Eysenck on psychometric grounds has received some support from an unexpected quarter in the form of the spectrum concept of schizophrenia promulgated by Kety, Rosenthal, Wender, and Schulsinger (1968). On the other hand, investigators of depression have found markers which support the typological approach in differentiating unipolar from bipolar depressions (Fieve & Dunner, 1974).

7.3. Operational Criteria

Because of the absence of objective criteria for classification based on either behavioral or psychophysiological markers, dependence must be placed on the phenomenological approach through interviewing. Nevertheless, although the phenomenological data are not easy to classify, certain combinations can be established as guides which will render the classification procedure more objective. To accomplish this end, specific definitions are required for the common terms used in psychopathology, such as “anxiety,” “depression,” and “depersonalization.” Specific criteria can be prescribed that must be present before a decision regarding the presence or absence of a given phenomenon can be accepted. Similarly, in codifying the individual symptoms into a syndrome, specific criteria can be prescribed before the syndrome is regarded as present. Kraepelin had originally focused on the course of the illness as a primary criterion in separating dementia praecox from manic-depressive psychosis, but, following Bleuler’s reformulation of the problem, more primacy was given to symptomatology. This second tradition has been further developed by Schneider (1959), Langfeldt (1939), the Biometrics Research Unit (Gurland, cited in Zubin et al., 1975; Spitzer & Endicott, cited in Zubin et al., 1975), the WHO Pilot Study of Schizophrenia (World Health Organization, 1973), and cognate groups. These groups have specified the psychopathological characteristics of schizophrenic behavior along the well-known dimensions of perceptual dysfunction, speech disorder, delusions, hallucinations, affective and cognitive dysfunction, and so on. However, these classic symptoms do not in themselves indicate a particular diagnosis, since they tend to be manifested by a diverse and heterogeneous group of patients. To reduce this heterogeneity and yet save the symptom or syndrome approach, Feighner, Robins, Guze, Woodruff, Winokur, and Munoz (1972) and subsequently Spitzer and Endicott (cited in Zubin et al., 1975) have proposed operational definitions of schizophrenia and the other mental disorders for the new Diagnostic and Statistical Manual (DSM III) of the American Psychiatric Association. Each disorder is operationally defined by explicit criteria. These criteria take the form of sets of characteristics, a specified number of which must be presented before a given diagnosis is warranted. Because the specific psychopathological features associated with each official DSM III diagnosis can now be explicaded, the reliability of diagnostic decisions can be enhanced (Spitzer & Endicott, cited in Zubin et al., 1975) and the heterogeneity that characterized the DSM III diagnostic categories can be greatly reduced (Zubin & Spring, 1977).

8. Conclusion

Research in clinical diagnosis in modern times was forced on psychopathology by the advent of the somatotherapies in the 1930s and 1940s, when it became obvious that the various somatotherapies did not affect all patients similarly. The greater need
for classification was even more accentuated by the drug therapies, and better diagnoses and assessment became a necessity. The ideal diagnosis should maximize the communication value of the diagnostic label, optimize its prognostic potential and its explanatory power, and provide a basis for the best therapeutic choice.

To attain this ideal goal, mere description was not enough. First, description leads to characterization of the illness rather than the disorder itself. By "illness" is meant the interaction between the focal disorder and the premorbid personality and the ecological niche the person occupies. That is why individuals who suffer from the same focal disorder do not always seem alike. Despite these difficulties, considerable success has been attained in providing systematic interviewing techniques and operational criteria for describing the disorders.

Second, we need to dig deeper than the surface description and deal with the etiology of the disorder. Since we do not have any knowledge of the etiology of most mental disorders, we have to resort to the stratagem of inventing scientific models of etiology and testing them against observation and in this way attempt to establish the validity of the diagnoses. Six scientific models have been described: (1) ecological, (2) developmental, (3) learning, (4) genetic, (5) internal environmental, and (6) neurophysiological. Each of these can provide hypotheses for testing predictive and construct validity. A second-order model which may prove to be a common denominator running through all the models is provided by the vulnerability model, which regards the patient as a vulnerable individual who when subjected to sufficient stress and strain will develop an episode. But this episode is time limited; only his vulnerability persists after the episode ends. The goal of preventive intervention is to prevent future episodes from developing in the vulnerable. To this end, two types of markers must be found: (1) those that identify the vulnerable individual and (2) those that identify the beginnings and ends of episodes. When these are discovered, the diagnosis of the mental disorders will become clearer and the tremendous affliction which they bring society will be reduced.

9. References


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