Schizophrenia: Fact or Fiction

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Paper delivered before the Second Annual Symposium of the Joint Committee on Schizophrenia of the New York State District Branches of the American Psychiatric Association's New Strategies Series, Schizophrenia: Fact or Fiction, Grand Ballroom, Hotel Roosevelt, New York, N.Y., November 17, 1973, 2:00-4:30 P.M.

This morning's symposium is a tough act to follow and it would require the talents of a Renaissance man to cover the panorama portrayed by my colleagues. I am not a Renaissance man, I am only a biometrician. Perhaps you are wondering what a biometrician is doing here and what his function really is.

The chief characteristic of the biometrician is his interest in measurement. He believes with Lord Kelvin that until you can measure you do not understand, and with Thorndike that whatever exists, exists in some amount and therefore ought to be measurable. In the field of psychopathology the biometrician serves as the common denominator for all the sciences that have entered the arena. Without the biometrician there would be no way of connecting the results obtained in neurochemistry, biochemistry, psychophysics, psychiatry, psychology and social science. Although all these fields are quite disparate in their content and their methods, measurement cuts across all of them and correlations of these measures often throw great light on the findings. Even in the purely clinical area the biometrician serves an important function.

Here it is the function of the biometrician to take the still amorphous, intuitive, subjective concepts of the clinician out of his private compound and drag them wriggling and struggling, willy nilly
across the threshold of objectivity and make them public. Those clinical concepts that survive this exposure to public scrutiny become part of the corpus of certitude of the rest of science. Those that do not, either disappear or remain in the clinical arena for yet another confrontation with biometrics. In this way our Biometrics Research Unit has already succeeded in measuring such clinically useful concepts as flatness of affect, comprehensibility of speech, friendship patterns, social distance and interconnectedness and a number of other variables including diagnostic evaluation via computers that had been unavailable to measurement previously. In this manner we stand between the humanistic and scientific spheres, measuring the measurable but appreciating the still unmeasurable. Whether there are clinical concepts and phenomena which will never yield to the onslaughts of measurement is debatable, but that we have enough imponderables to satisfy our craving for measurement for the rest of our lifetime is quite certain.

It is true that measurement, classification, and categorization are not the only ways of attaining knowledge. Understanding of nature comes in a multitude of forms, and man’s creativity in satisfying his curiosity can not be fettered to one method. However, the only way now available to establish the scientific value of understanding nature and communicating effectively to others is through the accepted scientific methods of building scientific models, developing hypotheses from them and then subjecting these hypotheses to experimental scrutiny.

It is with this in mind that I attempt to discuss the variety of issues emerging from this symposium. If this prospect doesn’t turn you on, I am sorry, but you have lost only one-seventh of your fees.
I. Introduction

The role of a discussant for a panel such as the one we heard this morning is quite a difficult one, as I already pointed out. There would seem to be at least four options open to me. I could (1) summarize, (2) deftly synthesize, (3) critically analyze, or (4) just deliver another paper of my own.

But I have chosen still another option, that of fence-sitting and observing and commenting. The advantage of fence-sitting is that you can keep a cooler head up there, but the disadvantage is that you are an easy target for brickbats from the contending sides. From my point of vantage, I noted that almost the entire spectrum from descriptive existentialism to the etiological medical model was covered. How can one term cover such a wide expanse? This chameleon quality of schizophrenia is perhaps the essence of our problem.

I had suggested the title for today's topic, Schizophrenia: Fact or Fiction, and chose it because it is sufficiently ambiguous to permit several contrasted meanings and in this way help clarify the issue that is before us. Some scientists, such as the extreme objective behaviorists claim that they deal only with facts and have no use for the fictions we call theory, while others, such as the extreme subjective existentialists, live only by man's subjective, intuitive fictions and have no use for the facts gathered by the behaviorists. Both of them capture important aspects of man's behavior in both health and illness.

There is, however, another meaning to the word fiction which indicates that the object or fact referred to has no existence, in the sense of our absent colleague Laing, whom many of you came to hear, that there is no schizophrenic disorder; it is merely an unusual style of life.
But science, be it physical or social, lives on fictions as well as on facts. [The fact of today often becomes the fiction of tomorrow as was the case with the flatness of the earth, while the fiction of today becomes the fact of tomorrow, as was the case with the atom and the gene.] By scientific fact we usually mean some ostensive object or relation, something you can point to, while by scientific fiction we mean a scientific model which consists of definitions and assumptions patterned into a parsimonious structure from which certain testable hypotheses can emanate. We live by these fictions scientifically because without them we have no way of organizing the facts that we observe.

All classification depends upon the fiction that a group of many objects, though they differ in many other respects, nevertheless belong together because they have some elements in common. These categories are pure man-made fictions which are useful in daily existence. While of a fact we can ask whether it is true, of a fiction we can only ask if it is useful. Thus, it is purposeless to inquire whether schizophrenia exists, but we may ask whether it is useful to regard it as a category for classifying human beings. In the history of psychopathology, psychology, and psychiatry, we have often seen certain fictions brought into question. Thus, Knight Dunlap in the '20s wondered whether the term "emotion" had not outlived its usefulness and was ready to show it the door, but on second thought, realized that it would only return through the back door. Similarly, "consciousness," "personality," and "will" have been excommunicated by various groups, only to be readmitted eventually.

Thus, the question is not whether schizophrenia exists, but whether it is a useful concept to deal with.
There are certain types of categories and of models that might be regarded as poor, or low in the hierarchy of usefulness, and, as in the case with Gresham's law, poor categories or models tend to drive out good ones, e.g., invalid projective techniques have driven out more objective personality tests. Undefined categories belong in the poor group and untestable models also belong there. As an example of a category that lost its usefulness we might take the term Dementia Praecox which has finally been dropped and absorbed into schizophrenia. Another example is the classification of animals by location -- land, sea, air, which Darwin rendered useless for scientific purposes.

It is not necessary that a category be defined rigorously in a closed-ended way, like a mathematical concept. If this were necessary, such widely useful concepts as species would go by the board. Julian Huxley has pointed out that "...there is no single criterion of species. Morphological difference; failure to interbreed; infertility of offspring; ecological, geographical, or genetical distinctness -- all those must be taken into account, but none of them singly is decisive. Failure to interbreed or to produce fertile offspring is the nearest approach to a positive criterion. It is, however, meaningless in apogamous forms, and as a negative criterion it is not applicable, many obviously distinct species, especially of plants, yielding fertile offspring, often with free Mendelian recombination on crossing. A combination of criteria is needed, together with some sort of flair. With the aid of these, it is remarkable how the variety of organic life falls apart into biologically discontinuous groups. In the great majority of cases species can be readily delimited, and appear as natural entities, not merely convenient fictions of the human intellect. Whenever intensive analysis has been applied, it on the whole confirms the judgments of classical taxonomy."
If biologists cannot define species rigorously, we need not be shamefaced if we cannot rigorously define schizophrenia.

Despite the difficulties involved in defining schizophrenia we must attempt it in order to be able to communicate. First, we must distinguish between the presence and absence of a disorder in general. We might regard a disorder as a progressive condition which, unless adequately dealt with, will result in either premature death, severe loss of efficiency and severe reduction of happiness (or increase in excessive pain) or some or all of these. A disorder is to be distinguished from a defect which is a stationary condition that will result in either severe loss of efficiency or in reduction of happiness or both but will not necessarily shorten life. It is easy enough to accept premature death as a criterion of a disorder albeit not a very useful one since it can be of use only retrospectively now, though it may be possible in the future to utilize this criterion by means of suitable prognostic indicators. When it comes to the criteria based on efficiency and on happiness, we are at a loss how to apply them since these criteria have no readily available baseline. However, if we accept, as Gurland does, an additional proviso, we may make some progress. Gurland suggests that we might define a disorder as a condition for which society has either provided some treatment already or is searching for an adequate treatment to remedy it. Thus, a disorder is an undesirable state from which relief is to be sought. Whether this definition is to be locally applied in each subculture or whether a universal classification is to be developed remains an open question. If a given condition is universally recognized as a disorder, the recognition of this state offers no problem. If only a small group
of individuals or some highly isolated culture regards a given condition as a disorder, it would probably not be generally accepted as a disorder unless sufficient evidence for its general acceptance were available. In between these two extremes we have a continuum of growing consensus as to whether a given condition is to be regarded as a disorder, and the higher the consensus across cultures, the greater the probability of its acceptance.

As far as schizophrenia is concerned, it is clear that it satisfies three of our criteria for being regarded as a disorder. First, it interferes severely with efficient living, it severely reduces happiness, and wherever it is recognized some treatment for it is sought. It might be argued that were it not for therapeutic intervention, shortening of life, too, would also take place due to the inability of the patient to adjust to living requirements in society. However, these criteria are not very operational since they apply equally to a vast spectrum of both physical as well as mental disorders. For operational purposes glossaries have been developed which specify the characteristic behaviors that must be present for a given condition of a patient to qualify as schizophrenia. There are several such glossaries available now and it is hoped that we will eventually accept a universal glossary such as the one proposed by the World Health Organization that will serve the purposes of better communication across different nations and cultures. [GIVE EXAMPLE OF GLOSSARY USED BY THE U.S.-U.K. STUDY.] However, even the availability of glossaries can not insure an operational definition. To attain this desideratum we have to specify more clearly the way in which the information regarding a given person should be collected, and the way the data obtained is to be integrated. Systematic structured
interviews with predesigned item choices for recording have given an added reliability to the information collected and computer programs based on decision tree procedures or utilizing Bayesian approaches can provide better uniform integration of the data for diagnostic purposes. But these diagnoses, despite their high reliability and reproducibility, are no better than the clinician who developed the decision tree. Only continued follow-up studies relating the diagnosis to outcome of specific treatment, to duration of illness, to eventual outcome etc. can provide us with the validity criteria for the diagnosis even though we have the highest degree of reliability.

So much for the methods of establishing the usefulness of the fiction involved in the category of schizophrenia. How does one establish the usefulness of the fiction of an etiological model for schizophrenia? One of the fundamental requirements for its usefulness is the testability of the hypotheses that emanate from it. Unless we can provide situations in which its tenability can be tested and either negated or not disconfirmed, the model is rather useless for scientific purposes.

As an example of an untestable model we might take the psychoanalytic model, which despite its wide use and extreme usefulness defies thus far the testing of its tenability.

My own earliest attempts at testing some of the hypotheses emanating from the Freudian model dealt with the Oedipus situation. A group of us as graduate students tried to develop a test which would examine the relative preference of children for parents of the opposite sex and we finally developed a test consisting of a considerable number of items in which a preference for the parent of the same sex or opposite sex
could be expressed. Among the items were such items as "Suppose you were out in a canoe with both of them and could only save one, whom would you save?" The results indicated that Freud was right, namely, that up to about age 6 there was a definite preference for the parent of the opposite sex and at age 6 apparently the latency period set in and everyone went for mother. After sending all this material, together with statistical tables, to Freud we waited for a long time before receiving the following answer: "Ganz Amerikanisch, but I do not quite see what you can prove with your statistics." Taken aback by this refusal to accept verification I inquired of some of my psychoanalytic friends as to what might have been the difficulty only to be told that, "Suppose you came to the Pope with proof that God exists, what do you suppose he'd say?" Years later, however, when that part of Freud's library which he had left behind him in Vienna was brought to the Psychiatric Institute I was able to ferret out from his early reading what might have been the reason for his refusal to accept statistical proof. Among the volumes I found were volumes written by Ernest Mach who seemed to have been one of the early idols of Freud. Apparently the early Freud was influenced greatly by men like Mach, Helmholtz and Wundt and his experience at the Physiological Institute at Vienna under the aegis of Ernst Brücke led him to develop the famous "Project for a Scientific Psychology." The underlying basis of this project was the belief that only physical-chemical forces are active within the organism and appeal to extraneous forces were unnecessary to explain behavior. The neurons were the units which carried these physical-
chemical forces. Freud, however, modified this belief when he found it impossible to explain psychopathology especially that which emerged from hysteria and resorted to a postulation of new forces beyond the physical-chemical inherent in matter, forces which could be of equal stature in producing the attraction and repulsion tendencies in man parallel to the attraction and repulsion forces in inanimate matter. This eventually resulted in his anatomy of personality, i.e., the forces of the Id, ego and superego in relation to conscious and unconscious behavior. Despite his basic fundamental adherence to the belief in causality, his basic approach to it was that of the early Mach and his followers who believed that there was always a prime mover and that every event had a direct cause and therefore he had no use for contingency. In this way it becomes plausible why statistical treatment of data based upon contingency notions would be foreign to Freud since, genius that he was, he could not transcend the models of his youth.

I would like at this point to refer to the usefulness of the category of schizophrenia, which has been brought into question, and then go on to indicate the need for the scientific models that have been developed for its etiology and for its treatment.

What evidence do we have that the concept of schizophrenia is useful?

It has been claimed for some time that schizophrenia does not recognize cultural boundaries and is evenly distributed in all cultures—in other words, that schizophrenia is not merely a product of Western culture, but that it exists universally in all cultures. The recent WHO International Pilot Study of Schizophrenia has provided direct evidence
for the existence of a group of patients in all the nine Field Research Centers, who have the common classic characteristics that place them in a schizophrenia category. The nine centers were located in Aarhus, Denmark; Agra, India; Cali, Colombia; Ibadan, Nigeria; London, U.K.; Moscow, USSR; Taipei, Taian; Washington, U.S.A.; and Prague, Czechoslovakia—a sufficiently wide spectrum to warrant making cross-cultural inferences.

A concordant group of patients was found who were originally diagnosed as schizophrenics but who were found also to cohere independently of their clinical diagnosis on the basis of a computer analysis—Catego—and on the basis of a statistical clustering technique. Of the total number of approximately 1,000 patients examined, 306 were found to belong to this concordant category. The following psychopathological characteristics are noted: 97% of patients have lack of insight, 74% have auditory hallucinations, 70% verbal hallucinations, 70% ideas of reference, 67% delusions of reference, 66% suspiciousness, 66% flatness of affect, 65% voices speaking to the patient, 64% delusional mood, 64% delusions of persecution, 64% inadequate description, 52% thought alienation, and 50% thoughts spoken aloud.

Comparison of the psychopathology of concordant schizophrenics and discrepant schizophrenics (patients with the clinical diagnosis of schizophrenia, but not in the concordant group), indicates that they differ markedly with regard to hallucinations, delusions, flatness of affect, and depressive symptomatology. The concordant schizophrenics score much higher on delusions, hallucinations, and flatness of affect, while the discrepant schizophrenics score higher on depressive symptomatology.
When the psychopathological characteristics of the concordant group are compared to those of the group of psychotically depressed patients, it is found that the concordant group of schizophrenics shows even less similarity to the psychotically depressed patients than does the group of all schizophrenics.

There are concordant schizophrenics in every one of the Field Research Centers. When the average percentage scores (on Groups of Units of Analysis for concordant groups of individual Centers) are compared Center by Center, using an analysis of variance, there are no significant differences between any pair of Centers. Thus it can be concluded that it is possible to identify a concordant group of schizophrenic patients that has a distinctive pattern of symptoms, that this pattern is consistent across Centers, and that there are patients belonging to this group in every Center in the study.

Another confirmation of the existence of a core group of schizophrenics comes from the U.S.-U.K. study which compared the diagnoses of the mental disorders in Metropolitan New York with Metropolitan London. Though there were differences in the frequency of schizophrenic versus affective disorders in the local diagnoses, the project's diagnostic team did not find such differences and furthermore established the existence of similar patterns of psychopathology on both sides of the ocean. Even in the relatively unreliable local diagnoses, there was a hard core of schizophrenics recognizable in both metropolitan areas.
Last but not least, the genetic evidence for the existence of a strain of schizophrenia is unmistakable. While the hypothesis of the inheritance of a schizophrenic spectrum rather than of a specific schizophrenic disorder is somewhat baffling, it nevertheless speaks for the usefulness of the concept of schizophrenia, if not for the existence of the condition itself.

Thus far we have dealt with the classification of hospitalized mental patients, that is, those who come for help. How well do our diagnostic schemes hold up when we examine a sample of the general population? In other words, how well can we discriminate the well from the ill in those who do not come for help?

If we are to be fair to ourselves and to our colleagues as well as to the public who puts its trust in us, we have to admit that though we can classify individuals who come for help into such categories as schizophrenia, affective disorders, neuroses, etc., and in many cases find these labels useful for the selection of treatment and for prognosis, we are not as successful in determining whether a given person who has not come for help belongs in one of these categories. In other words, we are far more successful in differential diagnosis than in screening the ill from the well in the general population. As a matter of fact, mere insistence on the need for help without sufficient psychopathology will often bring about admission to a mental hospital as Rosenhan has recently demonstrated. But even if we were more successful in descriptive psychopathology, we could not rest, since descriptive diagnosis never cured anyone. We must look for causes if we hope to advance.
A second admission we must make is that as far as etiology or cause is concerned, we are abysmally ignorant. We have had some successes in unravelling the causes of such disorders as general paresis, pellagra with psychosis, and some of the mentally retarded states, but even these victories have been pyrrhic, since the moment the cause of a disorder becomes known, it is lost to psychopathology. Other disciplines take over our hard-won victories and we are left with the disorders of unknown origin.

What does one do when faced with abysmal ignorance? All we can do is resort to fiction and imagine "as if" causes. But these fictions are not the end of our search, they are only the beginning. The scientific fictions we invent -- the scientific models we construct -- give rise to certain expectances -- to certain hypotheses, if you will -- and these can be tested for their tenability. Thus, the fictions do not remain fixed dogmas but are flexible structures which bend to the weight of the data produced in testing their tenability. This is the way Newton's model gave way to Einstein's, and this is the way Einstein's model may eventually give way to his successor. This is also the way Henry Cotton's hypothesis of focal infection as a cause of mental disorders was tested at the Psychiatric Institute in the '20s and found wanting -- the first controlled experiment in psychopathology, and this is the way the spirochete of syphilis was found to be the cause of general paresis. The requirements of a good model are, therefore, that it be testable by means of objective observations, and for this reason, good models die young and from their ashes rise, phoenix-like, their successors. Only the untestable models remain monolithically fixed in their absoluteness.
What are the models that have been proposed in addition to the popular medical model as opposed to the psychogenic or non-medical model? In order to eliminate these terms which have become perjorative I have suggested the following types of models which grow out of the medical-non-medical antithesis.

There are on the one hand, the models representing the field theory approach -- and on the other hand, those which represent the molecular approach. The field theory approach begins with the ecological model which simply stated assumes that man's development in health as well as illness depends on the ecological niche he occupies as measured by the physical -- social-cultural-economic-educational parameters of the niche. At the opposite pole is the genetic model which assumes that health and illness are predicated on the genetic equipment man is born with. The middle ground between these two extreme positions is held by the developmental and learning theory models, which lean towards field theory, and the internal environment and neurophysiological models, which lean towards the molecular. To be more specific, while the ecological model places its bets on the ecological niche, the developmental model postulates that the transitional phases between the stages of man's development -- from fertilized ovum, to fetus to neonate, childhood, adolescence, adulthood, middle age and the senium -- constitute the danger points at which development may go awry. The learning theory model, simply stated, is that man learns to become sick according to the same behavioral laws which govern his normal development. The internal environment model
stipulates that the roots of man's illness are to be sought in his metabolism, body fluids, and body chemistry in general. The neurophysiological model concentrates on the functioning of man's nervous system and its capacity to take in and process information. At this symposium, though it is difficult to pigeonhole, Dr. Warren Dunham has presented the ecological approach, Dr. Litz the developmental family approach, Dr. Shakow mostly the information processing or neurophysiological approach, Dr. Sarnoff Mednick the learning theory and genetic approach, Dr. Cancro the biomedical approach including the genetic-internal environment models and last but not least, Dr. Mosher has presented the descriptive existential approach which lies beyond the testable scientific models I have dealt with so far, since in his own words it is neither explanatory nor causal.

The evidence produced by these various models for causation of mental disorders would require an additional symposium and I have tried to summarize it in several papers elsewhere. Here, let us turn our attention to the evidence provided by my fellow symposiasts.

Dr. Mosher has given us an interesting thumb-nail sketch of the history of the development of phenomenology and existentialism and went on to trace some of the history of insanity, dementia and madness, Dementia Praecox and its evolution into schizophrenia. He then went on to develop the basic concerns of phenomenology. The essential process in phenomenology according to the psychopathologists who adopt the phenomenological point of view (Jaspers, for example) is that the inner world of the patient is uncovered to the phenomenologist. One wonders how this can be objectified or brought to the public scrutiny of others. Essentially, the phenomenological approach consists of the interaction of two private worlds, that of the phenomenologist entering and interacting with the private world of the patient. For this reason, it is beyond the pale of the usual scientific method, though
science can make use of the hunches and insights of the phenomenologist for further investigation by the scientific method. In fact, without the rich hunch world of the phenomenologist, the work of the scientist would be very attenuated.

Let us first look at Dr. Mosher's descriptive existentialism. In our description of various approaches to psychopathology we must separate the academic from the cristic. The academic approach, represented by psychoanalysis, existentialism and transactionalism consists essentially of a complete conceptual framework which is based on logical development of underlying assumptions that create beautiful aesthetic metapsychologies which depend for their acceptance more on logical consistency than on verifiable hypotheses. These logically consistent edifices, despite their plausibility, have not built in possibilities for their negation through experiment. If both love and hate, for example, are possible outcome of a given relationship, it is difficult to conceive of experimental verification of the outcome. In a sense, the academic approach is pre-experimental and we are still waiting for the genius who can provide us with testable hypotheses emanating from the psychoanalytic, existential and transactional frameworks. Thus, despite the plausibility and persuasiveness of Dr. Mosher's approach and its poetic quality we are in the same boat as Wagner von Jauregg's clinic was when he returned from Stockholm with the Nobel Prize for his discovery of fever therapy for general paresis. He found his team under Paul Schilder's guidance bending over a psychoanalytic interpretation of a case. He interrupted them saying: "Maybe you too can win the Nobel Prize. If not for science, maybe for literature!"

The description of Soteria and its function is a most intriguing
answer to the problems of some schizophrenics. One wonders, however, whether there is a way of transferring the modified behavior developed in Soteria to actual life outside. I have recently heard that the Stanford University Hospital has set up a fully furnished house on its grounds to serve as a temporary residence for patients and their families who must learn to cope with a long term handicap or disability. While staying in the house, family members are trained to perform nursing and physical therapy tasks. Is this a suitable paradigm for schizophrenic families who are so sorely in need of guidance in dealing with their vulnerable relative when he returns home? Perhaps living in Soteria with fellow patients or clients is not sufficient preparation for living outside. Living with family or friends under the protective roof of Soteria may enable the important others in the patient's life to learn how to deal with him when he returns home. The problem of those who choose to live alone can be similarly handled by approximating the conditions under which the loner will have to live in society.

The essential humane quality and appeal of Dr. Kosher's plan should not be taken as prima facie evidence of its efficacy.

In viewing the scene in the United States from the turn of the century on with regard to the movements for improving the lot of the disadvantaged, whether they be a minority group, or poor, or physically or mentally ill, or whatever, it struck me that there is a good deal of similarity between the concept of do-gooding and that of earning of 'mitzvahs' or brownie points in heaven. This essentially is an institution motivated by the needs of the giver rather than of the receiver, and how
many old ladies have been dragged by Boy Scouts across streets despite themselves, and how many unnecessary or even harmful interventions in family life have been perpetrated by the do-gooder, will never be known. This concept of reward for charity and alms-giving, which may have been transmitted from Judaism to other religions or may have sprung up on its own throughout the world, was probably a very efficient system for helping the disadvantaged as long as the locale in which this charity was given consisted of small towns or shtetels. Under such conditions an evaluation of the outcome of the do-gooding could easily be established and if the recipient turned out to be a drunk or unworthy of the charity, the situation could soon be corrected. Even in the shtetels there arose a group of schnorrers (itinerant free-loaders) who took advantage of the need on the part of the donor for racking up mitzvahs in heaven and would become sufficiently full of 'chutzpah' to scold the giver if the amount was insufficient. Schnorrers knew very well that they were rewarding the donor by offering him an opportunity to earn a mitzvah. There even developed a system of giving in secret so that the recipient would not have to feel the shame of having to receive charity, though in Japan, beggars are uniformed and licensed.

The wholesale transfer of these concepts to the urban situation has brought about a condition which was indeed far from satisfactory. The Lady-Bountifuls of the turn of the century who left food baskets for the poor, thinking that in that way they would discharge their duty towards society and earn brownie-points in heaven, rarely realized that they often did more harm than good to the recipient's families. And
the story of the evaluation of effectiveness of social work in this country is, of course, one of the sad stories in the history of welfare. Similarly, child guidance clinics, psychiatric therapies, the mushrooming of confrontation therapy, psychodellic movements, the Kingsley Halls, the Laingian approach, Mosher's Soteria approach, cannot be faulted for their intentions, but without built-in evaluation methods they would all turn out to be examples of how good will may go wrong. Unless we develop scientific models for explaining the nature of the intervention and developing methods for testing the hypotheses emanating from such models, all we will be doing is racking up mitzvahs in heaven, but no good may come of it. It is often surprising how hard-bitten businessmen who would never countenance one-sided ledgers showing only the credits in their own business, will look at the intentions of the movements they support and never worry about their accomplishments.

Dr. Lidz's elegant classification of families into the schismatic and the skewed marks an advance in the taxonomy of family types. One can only wonder at the careful painstaking methods he has developed. Without controls however, it is difficult to fathom whether Dr. Lidz is not in reality providing us with a taxonomy of so-called normal family life. Just as Freud, before him, thought he was dealing with neurotics, actually seemed to have dealt with psychotics but in the last analysis did the most good for normals by liberating them from their hangups, so Lidz may also be doing the most good for normals by providing a taxonomy of normal families. Whether his analysis will prove of value to schizophrenics and their families is a hopeful but not yet realized ambition.
Lidz's concept of decentering in which the child frees himself from parental dominance in favor of peer group relationships lends itself to examination experimentally. As a matter of fact, one of our former students, Dr. Dolores Kreisman, under our guidance studied the development of adolescent friendship patterns during the pre-schizophrenic period and contrasted this with development in normals. The outcome indicated that though by and large there was no great difference in the number of friends, intensity of friendship etc. there was one remarkable difference. The measure of intimacy differentiated the two groups significantly, the normals having the higher scores.

Regarding the presence of thought disorder in Dr. Lidz's families, it has often been a puzzle to me why so much stress is laid on thought disorder in the schizophrenic when the evidence for its presence is not overwhelming when it is tested experimentally. Though the comprehensibility of schizophrenic speech is not as high as that of normal speech, the laws that govern speech especially those based on association tests, are not violated by the schizophrenic. The association of the schizophrenic are different from those of the normal because the experience and world of the schizophrenic is different but not necessarily disordered. In sorting test behavior the schizophrenic is more influenced by the immediate surround by the immediately preceding event than is the normal according to Kurt Salzinger. Alfred North Whitehead has pointed out that we have two types of abstractions in thinking -- abstraction from actuality and abstraction from possibility. Abstraction from actuality refers to the immediate perception of attributes of objects such as color and shape. Abstraction from possibility refers to classification of objects or attributes according to such relations as use, composition, complementarity of colors etc., the
basis for which depends upon past memories. In abstraction from actuality the schizophrenic seems to be poor by normal performance standards probably because the variety of possible attributes available to him for selection has a different order of priority or salience for the schizophrenic, or, because of distraction produced by the multiplicity of possibilities. The immediate attribute to which he responds may not be salient to most normals. However, when it comes to abstraction from possibility, well preserved, undeteriorated schizophrenics do as well or even better than normals though the basis of their sorting, logical as it may be, is rarely utilized by normals. Based upon these considerations, it is difficult to infer thinking disorder in the schizophrenic, though thinking with premises rarely conceived by normals is often seen.

A good example of apparent thought disorder leading to meaningless speech which on closer examination ceases to be meaningless is given by Laing in his analysis of Kraepelin's case which the latter exemplified as showing thought disorder.

The literature on overinclusive thinking has recently been reviewed by Robert Payne. Unfortunately as he progressed in his work he found that though overinclusive thinking was found in schizophrenics, it was also found in other patients and in normals and probably is independent of psychopathology, relating more to cognitive style or personality.

With regard to self- and non-self, it is clear that self reference is one of the fundamental bases for sorting ability. In order to sort adequately one must possess the capacity for perceiving differences, similarities, equalities and identities. William Stern, one of Piaget's forerunners had pointed out that the development of these judgments of
difference, similarity, equality and identity date back to the early experience of the child with mother or important others. The feeling of difference develops when mother disappears from the scene even momentarily and the emotion it evokes is one of disruption. The feeling of similarity is also self-referred in so far as two objects are regarded as similar if they serve the self similarly -- sticks and strings are similar if they both perform the same function. Gradually, the child divests himself of the self reference in his judgments of similarity, difference etc. and thus deconcretizes his performance. Zubin and Thompson (1941) have postulated that in schizophrenia, the patient returns to self-referred judgments in his behavior and things are regarded as equivalent only if they have the same significance for him, and as different, if they have different significance for him.

One of the principles prominent in developmental theory is the principle of critical periods. There may be certain events that are crucial for the occurrence of certain types of developments if such events occur at the critical juncture. If these events come too early, they are ineffective and if they come too late, the particular developmental progress is irretrievably lost. Thus, separation of neonate from mother may not be as crucial at age 6 days as at age 6 months. Similarly being orphaned at age 5 is more crucial than at age 15. Left hemispheric lesions affect speech less if they occur in childhood than in adulthood. Pubertal maturation at an early age may produce more social deviancy than normal pubertal development. Thus precocious maturity in high school students has been found to lead more frequently to a variety of deviant behavior
ranging from emotional disturbance, juvenile delinquency, illegitimate pregnancy and criminality.

Dr. Sarnoff Mednick's genetic-learning model is one of the most persuasive, especially since he looks for the interaction of genetic and learning effects. [LEAVE REST OF PAGE BLANK.]
As far as learning theory is concerned, several hypotheses have been proposed for the etiology of schizophrenia based on learning principles. Among these are (1) superstitious behavior, (2) effects of intermittent reinforcement, (3) conditioned reinforcement and (4) immediacy hypothesis.

1. Superstitious behavior

This type of behavior occurs in situations where responses are "conditioned by reinforcers that are actually occurring at random" (Herrnstein, 1966). Since temporal contiguity between response and reinforcer is all that is needed to establish conditioning, the subject can not tell between a reinforcer that the experimenter delivers and a reinforcer that happens to be contiguous to his response by sheer chance. Thus it is quite clear why animals as well as humans will learn to do things for which there is no systematic "real reinforcement contingency." It is not possible that in a society as complex as ours that of all the recurring stimuli some might inadvertently and unnoticeably acquire control over some people's behavior? Is this the basis for ritualistic behavior? compulsive behavior? -- behavior which seems to have no contingent stimuli as reinforcer. Why are some people more prone to superstitiously developed behavior? Is this a genetically based propensity or is it dependent upon the ecological niche -- social deprivation? Is it possible that delusions are built up in this fashion? The beauty of this approach is that relatively simple organism like the pigeon can develop superstitious behavior and this makes the animal literature serve as a paradigm in examining the phenomena.
2. Intermittent reinforcement

The fact that even aperiodic non-systematic and non-continuous reinforcements will nevertheless prove to condition behavior is a very important factor in the utilization of learning theory in explaining abnormal behavior.

3. Conditioned reinforcement

-- Events associated with primary reinforcers can themselves become reinforcers.

4. Antecedent stimulus

-- Imediacy hypothesis [LEAVE REST OF PAGE BLANK.]
Dr. Dunham's discussion of social causation vs. social selection leads one to look for more definitive evidence for either hypothesis. Social causation has been suggested by H.B.M. Murphy in his examination of data on admission rates for schizophrenia in a variety of ethnic groups composed of both Catholic and non-Catholic subgroups. Across all of the ethnic groups he studied, the Catholic subgroups invariably had the higher admission rates for schizophrenia in males. He tries a variety of possible explanations, among which are the abstemious asexual model for Catholic youth provided by the priest and the nun. Another possibility is that the males in Catholic families do not receive adequate preparation for competing in the outside world. It is possible, however, that the Catholic subgroups may utilize hospital facilities more than the non-Catholic and this may explain the observed difference.

A rather striking example of social causation, or social labelling is afforded by Ackerknecht in his reference to reports that among some South American Indians a very disfiguring spirochaetal infection of the skin, known as pinto, is so widespread that almost all men suffer from it. The few who escape infection are regarded as ill and are precluded from marriage. If we were to liken the stressful pressure of the Puritan ethic for getting jobs and making a living as the infectious agent in our culture, we might emerge with the possibility that the small percentage of our population who are not infected by the Puritan ethic are sick and should be pre-empted from marrying. This may in fact be the basis for labelling some individuals as schizophrenic!

One way of determining whether social causation or social selection is at the basis of schizophrenia has been suggested by the Dohrenwends. They point out that if we compare old Americans and newly arrived immigrants for rates of schizophrenia, we should expect the following contrasting results. If social causation is the basis of schizophrenia, the
newly arrived immigrants who have climbed into the upper social classes after undergoing great stress should have the higher rates when compared with the well-situated upper class Americans who were born into their social class. On the other hand, if social selection is the cause of schizophrenia, it would follow that the newly arrive immigrants in the lower social class would have lower rates than the corresponding old Americans. The old Americans have lived here long enough to have climbed out of the lower classes but for some reason failed to do so, hence they must be kept back by some selective factor such as genetics; the newly arrived immigrants have not yet had time to rise out of the lower class and will consist of a mixture of the unfit who belong in the lower socio-economic stratum as well as of the fit who have not yet had time to rise, thus diluting and lowering the rate of schizophrenia. The Dohrenwends are now engaged in testing these two contrasting hypotheses.

Dr. Cancro has reviewed the biomedical approach to schizophrenia in a very comprehensive way and has given the details for the development of the genetic approach beginning with family studies which found schizophrenia to be a familial disease, progressing to twin studies and finally to studies of adoptees. It is interesting to note that if one investigates the families of schizophrenics and tries to develop a systematic approach to classification it becomes quite clear that some of the diagnostic procedures tend to show greater heritability than others. For example, following the system of classification due to Erik Essen-Möller it was found that the proportion of individuals affected by schizophrenia in the relatives of schizophrenic probands
seems to be higher than the proportion provided by any of the other diagnostic systems especially the American system with its rather loose criterion of schizophrenia and on the other hand the European system especially the British in which there is a stricter criterion. Apparently the middle-of-the-road criterion seems to give the best evidence for a genetic amlage and if one were to assume the truth of the genetic hypothesis it would follow that Essen-Müller's system seems to be the best method for classification.

One of the striking findings of the adoptees studies points to the possibility that far from being a definitive disorder, schizophrenia covers a wide spectrum of disorders from the very mild to the very severe and this raises the question of what is the best method for regarding the schizophrenic process, should it be a typological approach in which definite subcategories are delineated and regarded as based upon different etiologies, or should it be a dimensional approach in which there is a quantitative continuum between all the various types of schizophrenia even though they may require different types of treatment in conformity with their severity.

This conflict between the typological and dimensional approach may turn out to be a pseudo-problem. If we take as our paradigm the way the pomologists separate good from bad apples it becomes clear that typology and dimensionality are two aspects of the same problem. Originally the "apple knockers" would tap the apple with their knuckles to see whether it had water-core or whether it was sufficiently ripe. Today the profession of apple-knockers is extinct and instead the apples are conveyed on conveyor-belts under two sets of two monochromatic lights and the relative degree of absorption of the two monochromatic lights by the apple is indicated on a dial which separates off the apples that
are mature and free of water-core from those which are immature and have a considerable amount of water-core. Thus the early typological division into good and bad apples has been converted into a continuum which is utilized automatically for separation of the good from the bad. However, a little consideration of what has transpired here would lead one to ask the question "What produces the water-core and the immaturity?"

Perhaps it is a genetic element in the apple seed and if that were the case we are again returned to a typological rather than a dimensional approach by adopting the typology of the presence or absence of good and bad genes. However, if we go one step further and inquire "What is it that the good and bad genes do?" it becomes clear that the secretion of amino-acids is again a continuous dimension and that we are thrown back again on continuity rather than on discontinuous typology. Thus, the answer to the question whether we should accept the typological or dimensional approach really depends upon the state of the art at the moment and is not a question that has a definitive, final answer at this time.

There is one problem arising from the general tendency of recent genetic findings to discover spectra of schizophrenic - like behavior rather than core schizophrenia in offspring of schizophrenics and their relatives. Since the genotypic schizophrenic may vary in the expression of his schizophrenia from normality through the schizoidal spectrum to core schizophrenia it is possible that many of the members of our control group may be carrying genotypic but unexpressed schizophrenia. This may nullify the comparison of schizophrenic and normal controls of the normal controls containing an unspecified proportion of unexpressed
genotypes of schizophrenia. Thus many of our negative findings in the biochemical and neurophysiological areas may be suspect. Negative behavioral findings may suffer from the same difficulty. How to purify our control group by eliminating unexpressed schizophrenic genotypes is one of the problems facing genetic investigations today.

Dr. David Shakow has given us a double-barreled presentation. On the one hand he has presented a sensitive assessment of the nature of schizophrenia and on the other, the hard data regarding how schizophrenics differ from normals in their reaction time and other levels of response. In this sense, Dr. Shakow belongs to the descriptive approach and also to the neurophysiological approach to schizophrenia.

This neurophysiological approach consists of at least three separate trends: (1) experiments that attempt to relate the performance of the schizophrenic to certain personality characteristics in the cognitive area; (2) the physiologically related experiments dealing with such questions as temporal integration and auditory thresholds etc. and (3) the approach which to some extent encompasses the first two, namely, that of regarding the central nervous system as the information processing system for input and directing of output. Many of the experiments which have been done in this field and many of those which Dr. Shakow has pointed to seem to belong to the group in which schizophrenics tend to do much worse than normals with respect to their performance. Since schizophrenics do not have a high level of motivation and interest in performing laboratory tasks it is small
wonder that they tend to be found to be less capable than normals.

For this reason our laboratory has attempted to find tasks in which
schizophrenics seem to excel over normals because in that way we can
be certain that motivational deficits will not be the basis for the
explanation. We have actually succeeded in finding several such tech-
niques in which the schizophrenic excels. [LEAVE REST OF PAGE BLANK.]
How can our diagnoses and etiology be brought to bear on treatment?

With regard to the descriptive existential approach, for which both diagnoses and etiology are superfluous, treatment consists in dislabeling and distreatment -- permitting the individual to have his own life and do his own thing with proper reinforcement -- and in so far as he can do so without interfering with others, and not injuring himself; if he does not seek help, society might leave well enough alone. I have had chronic schizophrenics who were hospitalized for long periods and who had spent some periods of time on escape status, or on release status, wandering about the county, tell me that they can't understand why they are not permitted to wander. It's usually the family or some social agency that opposes their release, but perhaps these innocents, despite the hazards they might create might be better off wandering, especially since our treatments seem to be of so little help.

Etiology like perception has both a distal and approximate aspect and with regard to these two aspects of etiology psychoanalysis and behavior modification tend to occupy two polar points. In psychoanalysis the search is for the original causes that may have produced the deviation, namely, the distal aspects of etiology; in behavior modification we look for the proximate sources, those factors which maintain the current deviation in behavior and pay little or no attention to the distal etiology. It may be possible to demonstrate eventually that even though the proximate causes or contingencies are the most important as behavior modification theory would dictate, nevertheless if we examine the distal etiology we may discover
that some specific kinds of treatments are more amenable to certain types of distal etiology than they are to other types.

Another point to bear in mind is that the mere diagnostic label sometimes dictates the kind of therapy that would ensue. The label dementia praecox with all its frightening implications from Kraepelinian days certainly would have led psychiatrists of that period to dismiss the case as incurable and not try to deal with it in a more helpful way.

Certain implications from our models would lead one to engage in the development of behavioral prosthetics for schizophrenia, for example, just as near-sightedness can be alleviated in individuals by the use of glasses and perhaps we can improve the lot of the aged by slowing down any input so that they can assimilate it better, so it may be possible that in the case of schizophrenics certain prosthetic applications could mitigate their progress. For example, if we accept the immediacy hypothesis we might perhaps develop tape recorders that would allow the particular message to be played back in its entirety so that the attention of the schizophrenic would be called not only to the immediate aspect of the message but also to the long range aspects of the message and in that way overcome the immediacy aspects of his behavior. Or, perhaps to speed up the message would tend to bring the fullness of the communication in better perspective. At all events, this would open up an avenue for investigation with regard to the development of prosthetics to mitigate the problem of communication in schizophrenia. (In the above tape recording device, one should refer to the use of the metronome for stutterers by Brady.)
Another possibility is to take the slower reaction time expectations of the schizophrenic into consideration and deal with him in a way which would compensate for the slowness.

With regard to token economy this is a system for converting immediate responses required for earning the token into later responses that the token would eventually buy, so that a chain connecting immediate response and reinforcement to the long chain response of reinforcement is established.

With regard to neurophysiological model it may be possible that if we accept Venables' notion that there is a change in the arousal level of the schizophrenic, it may be advisable to train him to lower his arousal level by means of biofeedback.

It would take too long to indicate which treatments are most suitable for each of the presumed etiological models. But it is clear that the biologically based causes are not necessarily to be treated by biological intervention alone and the same holds true, mutatis mutandis for the psychogenic etiologies. But it is well to have some model for the therapeutic endeavor itself.

In searching for a model for therapeutic intervention, I harked back to the literature on prognosis. This literature is rather vast and varied; I had surveyed it in the early '50s in connection with our prognostic study. The one outstanding effect of this survey was to make us realize that in most cases, long before the disorder struck, the future patient had a personality, a style of life, a degree of adjustment. This premorbid personality, usually overlooked, was an important determiner
of the change in behavior which the onset of a disorder produced. This is the reason why no two TB patients, even if they develop the same lesion, behave in the same way or have the same outcome. While the tubercle bacillus may be the focus of the disorder, the response of the patient to the bacillus, the degree of resistance he exhibits, whether he is an optimist or a pessimist, the degree of emotional involvement and the entire context of his condition all determine the immediate response to the disorder as well as the eventual outcome. This is also the reason why no two schizophrenics look alike. In their case, not only are we ignorant of the focus of the disorder, but its interaction with the premorbid personality and the ecological niche so color the behavior, that similarities between two schizophrenic patients' behavior may be far less than the differences they exhibit. But premorbid personality and the ecological niche not only determine the immediate response to the disorder, they also largely determine eventual outcome.

If I were to summarize the results of our prognostic review in one phrase, it would be that the best predictor of outcome is the premorbid personality. In trying to fathom why the premorbid personality plays such an important role in determining outcome, I proposed the following model for therapeutic intervention. I am now leaving my perch on the fence and entering the arena fully realizing that I am now, if not before, vulnerable to counter attack.

Let us assume that the schizophrenic is a vulnerable personality who, when subjected to sufficient stress, will be catapulted into an episode. Let us further assume that all episodes are time-limited and that when the episode ends, the patient returns to his premorbid level or close to it. If he had a good premorbid level to begin with, he returns to his
premorbid status in society and is regarded as recovered or at least improved. If his premorbid level was poor, what has he got to return to? He could hardly cope before he was catapulted into the episode and he still cannot cope when the episode is ended. As a matter of fact, it is difficult to determine in his case when the episode ends. This is why the premorbid personality plays such an important role in outcome. We might go a step further, somewhat beyond our data, and assume that all schizophrenics recover from their episodes. What we call chronic schizophrenia represents a category of individuals who are no longer suffering an episode, but who appear to be still sick because the premorbid personality to which they have returned is not sufficiently well developed to enable them to cope. Furthermore, the hospital stay often teaches them the patient role so well that they develop what Ernest Cruenberg has dubbed the SBD -- social breakdown syndrome.

The number of such patients is not inconsiderable. Before 1930, before the advent of the shock therapies and the more recent chemotherapies, about one-third of the schizophrenic admissions got out and stayed out, about one-third oscillated back and forth in the hospital, and the remaining third stayed permanently in the hospital. Currently we have continued to release the first third; the middle third, with the help of drugs and other interventions also stays out; the last third has become the oscillating group because of the poor premorbid status. It is the last group that needs the special intervention I am discussing here. One might go still another step further and say, don't bother treating the good premorbrids -- they'll get well anyhow or will get well quicker with suitable intervention. Concentrate on the poor premorbrids and utilize whatever therapeutic talent you have to rehabilitate them, using psychotherapy, behavior modification, chemotherapy, or what not. Of course, a moral issue might arise here.
What right have we to remould the personality of the poor premorbid so that he will better fit our social-cultural mold? I suppose our only justification is based on the fact that he came for help. If he does not want to improve his premorbid personality, does not want to adjust to society, well, then he can join Laing's cohorts and remain either unreconstructed or do his thing the best way he knows how, or find an outlet for his latent style with the help of Laingean therapy. Thus, the thrust of intervention is removed from cure to remoulding personality -- a new venture worthy of our attention.

To summarize my discussion, our symposium has pointed out that schizophrenia is far from being a myth, though it may be regarded as a useful fiction, and that there are a variety of approaches to uncovering the causes of schizophrenia and sufficient evidence for the usefulness of the concept. It no longer needs to fight for its scientific existence. The variety of approaches presented this morning has both an advantage and a disadvantage. The advantage inheres in the fact that by pushing each model to its limits, we can uncover more and more of the roots of the disorder. The disadvantage lies in the fact that the interaction between the models is probably of greater importance in understanding the underlying causes. Thus, genetics alone is necessary but not sufficient, even as noxious ecological niches are necessary to elicit the schizophrenic enlagement but not sufficient in themselves to bring it about. The same holds true of all the other models. Perhaps the best paradigm to view the problem is the technique of analysis of variance. In this technique we first examine the main effects and then, having exhausted this source of variation we examine the various orders of interaction. This is the most fruitful view to take.

When viewed from this angle, the multidisciplinary contributions to the etiology of schizophrenia exemplified in this symposium are a guarantee
that we are leaving no options closed to the discovery of the sources of schizophrenia and to its treatment and hopefully also to its prevention. Only the combined efforts of all the disciplines will eventually bring down the obstacles to the solution of our common problem.