DISCUSSION—Joseph Zubin

Dr. Eisenberg has presented a clear picture of the current scene in the diagnoses of psychotic disorders in children. From his clinical acumen and his scholarly approach, we can see just how far this field has developed. He is not one to feel satisfied with the current situation, but is realistic enough to indicate that when we have no knowledge of etiology we must fall back on clinical description of behavior. I have only a few comments to make on his presentation, and these deal with the questions of manic-depressive psychoses in childhood and of the continuity hypothesis, which is one of the themes of this symposium. After dealing with these two items I would like to turn to the question of what can be done "while waiting for the doctor"—i.e., what can be done while waiting for the etiology to be discovered.

Regarding manic-depressive psychoses in children, it might be pointed out that the detection of such conditions in childhood presents some difficulties. When an adult gets so depressed that he cannot manage his affairs or cannot continue with his daily work, the detection of his condition is much easier than in a child whose needs for keeping up with daily schedules are not as demanding
and whose inability to go to school may be regarded as an indisposition of unknown cause. Similarly, the hyperactivity of the adult in the manic phase is readily detectable, but in the case of a child such hyperactivity may masquerade under a different label. Since these phases are self-limiting in most cases, their occurrence in childhood may be far more prevalent than our statistics indicate.

Regarding the continuity hypothesis, this question is often raised in controversies contrasting the so-called medical model with the behavioral model. While it is true that the medical model (discontinuity hypothesis) has attained greater success even in the mental disorders (general paresis, pellagra with psychosis, PKU, galactosemia) it is also true that 75 years of research in schizophrenia have not yet provided an acceptable medical model for that illness, nor do we have one for neurosis, manic-depressive psychosis, or any of the other mental disorders. The genetic studies in these disorders and in Huntington’s chorea lead us to hope that eventually the medical model will be found, but thus far success is still out of reach. While waiting for the medical model to appear, we must resort to behavioral description.

What, then, is this medical model? In fact, what is meant by “a scientific model”?

When one examines a well-articulated science like physics, with its parsimonious models and aesthetic appeal, and compares it with unarticulated sciences like economics, psychology, or psychopathology, one wonders why sciences like the latter three have lagged so far behind. It is the purpose of this discussion to examine the
role of models in scientific progress in the light of the developmental lag which characterizes some of them.

Let us begin by pointing out that scientific models are not guides for conducting experiments. They are ways of looking at an intellectual domain. Some models remain hidden in the mind of the investigator, while others are made public by means of expressed definitions and assumptions. These constitute the structure of the model, from which hypotheses can be elicited to test the tenability of the original definitions and assumptions. Most often, the actual formal structure is not necessary to further testing and experimentation, but it does become so in communicating results. Without the formal model from which the hypotheses stem and into which the results are incorporated, it is impossible to communicate to others why the experiment was undertaken and what its results signify. Consequently, the formal model is often an afterthought—after the results of the data have interacted with the prior assumptions and definitions that constituted the covert model before it was formalized. There are two types of models, those that are susceptible to testing and those that are not. The testable models consist of definitions and assumptions that permit the emergence of hypotheses that may be proved through observation and experimentation. The untestable models deal with concepts that are intangible; they do not lead to experimental verification.

The earliest models for explaining observed phenomena date back to magic and witchcraft. A good example of a primitive model is the explanation for good and poor
fishing expeditions. The good fishing expedition is one in which the water sprites turn the spears toward the fish and permit a good haul. The poor fishing expedition is occasioned by the spitefulness of the water sprites who turn the spear away from their quarry. The turning of spears under water may have been man's earliest recognition of refraction phenomena. Ages passed before the principle of refraction became clear, as a phenomenon of light being bent by the medium of the water, but primitive man was able to explain his good or bad fortune on a much more direct basis.

Another model which primitive man created emerged from his propensity to play. As soon as leisure became possible, games of chance arose. There are indications that gambling goes back deep into man's past. One statistician, F. N. David,\(^1\) refers to this problem in the following words:

> When man first started to play games of chance is a time problem we shall never clearly resolve. We may place on record that it is a commonplace thing for archaeologists to find a preponderance of astragali\(^2\) among the bones of animals dug up on prehistoric sites. One archaeologist stated that he had found up to seven times as many as any other bone, another put the figure at 500 (sic!), while yet a third, refusing to be drawn to a figure, stated that they were many. This fact has perhaps little significance. The astragalus has little marrow in it and was possibly not worth cracking for the sake of its contents as were the long bones: it is knobby and

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2. The astragalus is a small bone in the ankle, immediately under the talus or heelbone.
presents no flat curves for drawing as does the shoulder blade for example. All we may do is to place on record that round about 40,000 years ago there were large numbers of the astragali of sheep, goats, and deer lying about. The astragali of animals with hooves are different from those with feet such as man, dog and cat. In the case of the dog the astragalus is developed on one side to allow for the support of the bones of the feet. The astragalus of the hooved animal is almost symmetrical about a longitudinal axis and it is a pleasant toy to play with. In France and Greece children still play games with them in the streets, and it is possible to buy pieces of metal fashioned into an idealized shape but still recognizable as astragali.

Sometime between prehistoric man of four hundred centuries ago and the beginning of the third millennium before Christ, Homo sapiens invented games and, among these games, games of chance. We know from paints, terra-cotta groups, etc., that the astragalus was used in Greece like the ancient quoit, but there is no doubt from paintings on tombs in Egypt and excavated material that the use of the astragalus in games where it is desired to move counters is well established by the time of the First Dynasty. In one painting, a nobleman, shown playing a game in his after-life, delicately poises an astragalus on his finger tip, a board with “men” in front of him. A typical game of c. 1800 B.C. is that of “Hounds and Jackals.” The game seems similar to our present-day “Snakes and Ladders”; the hounds and jackals were moved according to some rule by throwing the astragali found with the game and shown in the figure. Variants of this game were undoubtedly played from the time of the First Dynasty (c. 3500 B.C.). (pp. 1-2)

3. From the name knucklebone, we might infer that among the early games were those in which the astragali were balanced on the bones of the knuckles and then tossed and caught again.
Whether or not devils and benign spirits were in control of the dice depended on the stage of development of man. It is interesting to note that the concept of contingency may have developed much later than we suspect. It is a more abstract notion, introduced when research men ceased trying to explain unique events on a causal scientific basis. The unique event—why it happened to me—was first explicable only in terms of witchcraft, and later in terms of some theological system. Science still cannot cope fully with the question—“Why, of all the three billion people on earth, did this happen to me?”

Once the model for games of chance was developed, it became possible to modify chance expectancy by means of certain strategic moves, such as weighting one face of a die. From experience with strategy, games such as chess developed. Here boundary conditions, rules, and constraints of various kinds made the game more difficult but, at the same time, increased the pay-off.

As leisure increased and man became more observant of natural phenomena, he began to build models of his universe as, for example, the development of geometry. Primitive Egypt was at the mercy of the Nile whose floods erased all boundaries. As the need for surveying personally owned land became greater, the corners of the fields and their connecting boundaries became the “points” and “lines” of Euclidean geometry, and theorems such as “the square of the hypotenuse” were developed.

As man began to observe his environment more closely, he began to focus on certain specific—because consequential—puzzles. With the help of strategic and chance models he was able to solve some of them. Thus
he adduced the ground rules for constructing modern scientific models.4

What are the scientific models that are most applicable to the field of mental disorders in childhood? Asking the question in this way, the dichotomy of medical vs. nonmedical disappears, and the question becomes transformed: What are the definitions and assumptions that have proved useful in the past in providing testable hypotheses; i.e., what are the useful scientific models? The useful dimensions on which our models are based are the following: (1) organic-functional, (2) hereditary-environmental, (3) multiple vs. unitary causation, (4) disease vs. reaction-pattern, (5) exogenous-endogenous, (6) acute-chronic.

The changes in behavior accompanying mental disorders have been attributed to a variety of etiological factors, and around each of these a suitable scientific model can be erected. In reviewing this field of etiology, the following scientific models seem to dominate: (1) social-cultural, (2) developmental, (3) conditioning or learning, (4) genetic, (5) internal-environment, and (6) neurophysiological or brain function. Until recently, the first three models—social-cultural, developmental, and learning were the most prominent. In recent years, genetics, internal-environment, and brain-function models have become more popular.

Which of these models is likely to be most useful in uncovering the sources of childhood psychoses and in

hastening their prevention or containment? It is a difficult question to answer. Perhaps all six models must be pursued simultaneously in the form of the supermodel of epidemiology which attempts to weight the contribution of each model to the total forces leading to a given illness.

But in order to make any progress in any of these directions we must achieve more behaviorally oriented objective descriptions of patient behavior. For this purpose, interviewing methods, behavior inventories, play techniques and other methods that will yield objective description must be devised. Armed with such descriptive measures, analysis of etiology can proceed rapidly. We may discover that because of the rather limited number of ways in which the human organism can respond, as contrasted with the multiplicity of agents which can produce disorders, many individuals who behave similarly belong to different disorder categories from the point of view of causation.