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094

94

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PROSPECTS FOR RESEARCH ON SCHIZOPHRENIA

A Report based on an NRP Work Session

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II. CLINICAL ISSUES

Classical Symptoms of Schizophrenia: P. E. Meehl

Meehl gave a brief summary of schizophrenia as a clinical entity and the history of its nosology. Schizophrenia is the commonest of the functional psychoses; and because of its tendency to develop early in life and its chronicity, it accounts for a sizeable fraction of all hospital beds. Prior to the development of the phenothiazines and the growing emphasis on keeping psychiatric patients out of the hospital to avoid the "hospitalization syndrome," patients with schizophrenia occupied about half of all hospital beds—not just mental hospital beds, but all hospital beds—on any given day in the United States. The major classic features of the full-blown clinical entity include a characteristic thought disorder or disturbance of associations, a peculiar inappropriateness of affectivity, marked withdrawal from normal social interactions, a lack of contact with and interest in external reality, and, as a result, varying degrees of incompetence to function in social, economic, and sexual life. In addition to these "core defining symptoms," many schizophrenic patients show what Bleuler called "accessory symptoms" either continuously or intermittently, prominent among these being persecutory, grandiose, or somatic delusions, hallucinations, and a vague cluster of correlated phenomena known as catatonic symptoms; e.g., posturing, waxy flexibility, excitement or stupor, stereotyped movements, automatisms, negativism, mutism (Bleuler, 1911).

Early Investigators

The great nosological synthesis was made at the turn of the century by the German psychiatrist Kraepelin who gathered together under one diagnostic heading several psychiatric entities previously considered separately—the French "démence précoce," Kahlbaum's "Katatonia," Hecker's "Hebephrenia," and an entity called "dementia paranoides"—into a syndrome he called "dementia praecox" (Kraepelin, 1896,* 1918, and 1967). He called it "dementia" because these patients show (at least phenomenologically) an impairment of cognitive function, although it is usually quite different from that of an organic dementia, and "praecox" because of its tendency to appear in the late teens or early

* Kraepelin's "diagnostic synthesis" first appeared in 1896, but the translation of the concept appeared considerably later in Volume 3 of the 8th edition of his *Lehrbuch der Psychiatrie.*

adult life. He emphasized its malignant prognosis, in contrast to the other nosological group that he identified about the same time, namely, the manic-depressive psychosis, which, even without specific treatment, tends to have a favorable outlook and furthermore shows essential clinical normality, except, perhaps, for certain personality deviations before, after, and between attacks.

In 1911, the Swiss psychiatrist Eugen Bleuler published a classic work on *Dementia Praecox or the Group of Schizophrenias.* He focused attention upon the peculiar disorders of association and affectivity and considerably enlarged the aaaaThe next major nosological contribution, according to Meehl, was the paper published in 1949 by Hoch and Polatin entitled "Pseudoneurotic forms of Schizophrenia." According to this view, there are patients with schizophrenia who are likely to be misdiagnosed, and therefore mistreated clinically, as psychoneurotics. These patients present complaints of pan-anxiety, depression, a deficiency of pleasure (Rado's "anhedonia" (Rado, 1956)), fluctuating multiple somatization, psychophysiological reactions, chaotic sexuality, and (pathognomonic when present) short-term psychiatric explosions lasting from a few minutes to hours or days, which Hoch and Polatin christened "micropsychotic episodes." It is important to note that while such a "pseudoneurotic" patient is actually undergoing a micropsychotic episode, he is clinically schizo–phrenic by Bleuler's criteria.

Schizotypy and Schizophrenia

Some American clinicians strive to adhere closely to Bleuler's delineation of the clinical syndrome "schizophrenia," and therefore make use of additional terminology (some of which is in the standard *Diagnostic and Statistical Manual of Mental Disorders,* 1968) when referring to patients (or non-patient personality types) who they believe are disposed to schizophrenia even if they never develop the clinical disorder. Thus, some people speak of "schizoid disease," "schizoid personality," and "schizotype."

Meehl views clinical schizophrenia as a decompensation of the schizotypal personality and believes that for every diagnosed schizophrenic patient there are probably three or four times as many compensated and semicompensated schizotypes in the population. He compared schizotypy to compensated cardiac or kidney disease, or to a patient who has the diabetic genotype (and, perhaps, an abnormal glucose

tolerance curve) but does not present the symptoms of clinical diabetes, or to individuals with the genotype for gout (detectable by elevated uric acid titer), of whom only 10% have clinical gout symptoms.

There was disagreement about the diagnostic term ''acute schizophrenic reaction" applied by American psychiatrists to individuals who, without premorbid personality deviations, develop, quite abruptly, an acute psychosis that has many of the characteristics of schizophrenia. Ploog argued that schizophrenia not uncommonly begins in this way. Kety indicated, however, that adoption studies reveal that the term is often inappropriately applied to acute psychoses that are not related to schizophrenia.

Meehl proposed that, since the presence of genetic factors in schizophrenia is beyond question, the important research questions now are as follows:

1. How much of the determination is genetic?

2. What is the mode of inheritance? Because of the frequent occurrence of an unaffected individual in family histories, a major gene theory may also require the influence of modifier genes.

3. What is the inherited defect or cluster of dispositions? As Bleuler pointed out in 1911, one cannot inherit the *content* of schizophrenic behavior (e.g., you cannot inherit a delusion about Jesuits, you have to *learn* about Jesuits). Meehl argued that even the formal features, e.g., the preferred defense mechanisms, and the mere *existence* of the mechanisms of defense (projection, denial, displacement, symbolization, and the like) are products of social learning. He suggested that what is inherited is one or more higher-order dispositions involving parameters of nervous system function, rather remote in the causal chain from the clinical symptomatology.

4. What kind of a theory would explain the copresence of thought disorder, marked interpersonal aversiveness, ambivalence, and anhedonia?

Although much "clinical lore" does not stand up to careful scrutiny, the same is true of most experiments in the behavioral sciences, particularly those involving human subjects. It is not sensible to let weak experiments countervail universal clinical experiences. Meehl suggested that genes that predispose to fearfulness in social situations are among the polygenic influences contributing to the risk for

schizophrenia. Better statistics should be developed for taxonomic analysis. It is unlikely that definitive empirical tests can be made of various genetic models until there are taxonomic methods to classify borderline patients and compensated "normals" as schizotypal or not. Clinicians should try new psychometric tests, aimed at detecting the compensated or semicompensated schizotype. There are at present, for example, no sufficiently sensitive means for measuring hedonic capacity, mild depression, and subclinical cognitive slippage.

It is reasonable to believe, Meehl argued, that there is some "common core" behind the manifestations of schizophrenia. This accords with the fact that a single schizophrenic patient, if followed over a long enough period of time, may present the "textbook syndrome" for one subtype at one time and an equally classic picture of another subtype at another.

Meehl believes it is impractical to devote much research to studying relationships that are inherently ambiguous in causality; e.g., to show that the parents of a schizophrenic teenager do not interact with him in the same way that they interact with his nonschizophrenic siblings. Statistical studies of the incidence of schizophrenic subtypes in relation to demographic variables like social class or section of the city suffer from the same inherent ambiguity.