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For a number of reasons, prognosis in schizophrenia has proved controversial. Each of the papers in this issue of the Schizophrenia Bulletin addresses at least one of the areas of controversy. In so doing, each of the authors takes a rather different approach to the problem, and it is hoped that the sum of these differing viewpoints will provide readers with a fresh synthesis.

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A major reason for the difficulty surrounding prognosis in schizophrenia has been confusion over diagnosis. In general, the concept of prognosis depends upon the existence of discrete disease entities. Yet, as soon as he coined the term, Eugen Bleuler (1950) pointed out that we are dealing with a group of schizophrenias, and soon afterwards, Adolf Meyer (1951) suggested that schizophrenia may not even be a group of discrete disorders but merely a type of reaction to an adverse environment. Certainly, in the six decades that have passed since Bleuler and Meyer expressed these opinions, no investigator has provided an adequate etiological explanation for schizo-
phrenia, nor have we found an objective means of definitive diagnosis. As a result, investigators are at the mercy of different diagnostic definitions. For example, this issue discusses “narrow” and “broad” concepts of schizophrenia, the relatively objective Present State Examination (Wing, Cooper, and Sartorius 1974) that was incorporated into the International Pilot Study of Schizophrenia, the more operational criteria of Feighner et al. (1972) and Eugen Bleuler’s (1950) more impressionistic “Four A’s” (Autism, Affect, Association, and Ambivalence). Although each definition encompasses a group of schizophrenics who in about 15 to 25 percent of cases will enjoy full remissions, each definition includes a slightly different, if overlapping, population of patients. In this issue, Strauss and Carpenter and, to an even greater extent, Stephens point us toward a possible solution. A single population of “schizophrenics” should be diagnosed by a variety of different criteria, and the longitudinal usefulness of each method compared.

I believe that Stephens’s review of prognosis in this issue provides the most important, complete, and encompassing review that this field has seen. The explanation, I think, is that Stephens has not become fixed in the Scandinavian, the St. Louis, the Burgholzli school, etc.; rather, he has dispassionately contrasted literature from many different decades, many different countries, and many different diagnostic viewpoints. Equally important, he has applied different diagnostic and prognostic schemes to the same cohort of patients, thus providing a methodological solution to many fruitless psychiatric controversies. More often, in psychiatry, we need to look at the same patients from conflicting points of view.

The second reason that prognosis in schizophrenia has proved difficult is that, until now, investigators have not adequately defined clinical course. Often, outcome has been defined simply as the presence or absence of hospitalization discharge. Even in the long-term studies by Stephens, prognosis is confined to terms like “remission,” “improved,” “unimproved,” or “worse”—at a single point in time. Thus, Manfred Bleuler's (1972) vast monograph, summarizing a prospective study lasting a quarter of a century, helps remedy the defects in our conceptions of schizophrenia, which have arisen from cross-sectional followup.

Originally, schizophrenia was defined by Kraepelin (1919) as a progressive, nonremitting illness—a disease like multiple sclerosis with an inexorable, if intermittently, deteriorating prognosis. Even before Kraepelin, in the mid-19th century, Luther Bell, superintendent of the McLean Hospital, over a period of a few years went from being impressed at how many patients with “mania” recovered to being depressed at how many, over time, deteriorated. Luther Bell, Emil Kraepelin, and many of us who have followed in their footsteps have passed from optimism to pessimism; 5-year followups of schizophrenics often reveal chronic deficit states.

That this formulation is too gloomy for some schizophrenics is amply documented in Stephens’s review; however, Gerd Teschke’s summary of Manfred Bleuler’s (1972) lifelong study goes further. Bleuler contradicts the concept of continued “deterioration” in even poor-prognosis schizophrenia. Because translation of Bleuler’s extraordinary monograph into English has been delayed for several years, the full impact of its ideas has been unavailable to readers who lack fluent German. What Bleuler has done is to follow the same patients for a lifetime—and notice the word is follow, not follow up. One cannot assess the clinical course of a dynamic illness like schizophrenia at only two points in time. By diligently following his patients, Bleuler has been able to arrive at the novel conclusion that it is only for the first 5 years that Kraepelinian deterioration may occur. Then, if even modest rehabilitative efforts are sustained, a clinical plateau or even gradual improvement occurs in the most chronic cases. Although Bleuler’s study is unique, a number of senior clinical investigators of schizophrenia, whose own longevity has allowed them to follow the same schizophrenics for decades, anecdotally corroborate Bleuler’s findings.

A third reason for difficulty in prognosis has arisen from too narrow a definition of outcome. The paper by Strauss and Carpenter, like Teschke’s review of Manfred Bleuler’s work, produces a why-didn’t-I-think-of-that? response
in the reader. By dividing prognosis into subcategories of symptomatology, social relationship, work history, and hospitalization, Strauss and Carpenter underscore that these facets of outcome differ for different individuals and are correlated with different prognostic factors. Indeed, Strauss and Carpenter almost imply a return to the Meyerian point of view, when mental illness was seen as merely reflecting individual reaction patterns rather than discrete diagnoses.

The more Kraepelinian papers by Fowler and myself are foils for the paper by Strauss and Carpenter. Each paper attempts to elaborate a new tool for understanding the remitting schizophrenics. Fowler points out that even if we cannot diagnose schizophrenia with certainty, even if we cannot discover the specific etiology of schizophrenia, we do know that “schizophrenia” in relatives predisposes to schizophrenia in probands. Thus, his data support the idea of the Kraepelinian disease concept. He and Michael McCabe and their associates (McCabe et al. 1971) have demonstrated beyond a reasonable doubt that most schizophrenics with a family history positive for schizophrenia fail to recover, and that most schizophrenics who do recover have a positive family tree for affective psychosis. Fowler makes a potentially important forward step by suggesting that in ambiguous cases, rather than assuming that affective patients with some of Bleuler’s “Four A’s” are “really” schizophrenic, we should regard schizophrenics with symptoms of affective psychosis as likely to represent affective disorders.

Fowler goes on to say that “the family data suggest that mania, unipolar depression, and schizophrenia account for most cases of remitting schizophrenia.” Also, on the basis of genetic evidence, Fowler argues persuasively that reactive psychosis is distinct from remitting schizophrenia.

This point raises another controversial issue in the literature on prognosis, namely the trend to divide schizophrenias into process and reactive. The implication is that the etiology of process schizophrenia is biological and that of reactive schizophrenia situational. In my own work, instead of depending upon the longitudinal variable of family history to identify “real schizophrenics,” I have used refollowup of remitting and/or reactive cases. Just as family history allows one to make schizophrenia more tangible, so does allowing adequate time for relapse. Some of the most reactive or psychogenic schizophrenics, if followed long enough, often assume a picture consistent with the defects of process schizophrenia. Thus, the process/reactive dichotomy may better serve as an imaginary construct than as a way of classifying biological reality. In my work, as in that of Fowler and McCabe, it was heredity rather than clinical symptoms that predicted which remitting or acutely schizoaffective patients would eventually resemble chronic schizophrenics or bipolar depressions.

In integrating the different views in this issue, the reader may wish to reexamine a fundamental problem in our methodology for studying psychological disorders. Namely, does one wish to have reliable instruments or to reliably predict clinical course? Put differently, the current efforts to obtain cross-sectional rater reliability in schizophrenia may demand too high a price over the long term. While unquestionably the Present State Examination (Wing, Cooper, and Sartorius 1974) and the proposed DSM III (Spitzer, Endicott, and Robins 1975) allow for greater rater reliability in identifying schizophrenics at one point in time than the more fuzzy clinical concepts of Bleuler’s “Four A’s,” in the hands of a good clinician Bleuler’s concepts may better identify “schizophrenics”—i.e., patients who respond better to phenothiazines than to lithium and tricyclic antidepressants, patients who will go on to chronic social deficit states, and patients whose relatives manifest schizophrenic traits.

To make this point more explicit, let me offer an example. In the Fall 1970 Schizophrenia Bulletin Norman Garmezy reprinted his review, “Process and Reactive Schizophrenia: Some Conceptions and Issues.” In the same year, in Seminars in Psychiatry, Joseph Stephens published an earlier draft of his current paper, “Long Term Course and Prognosis in Schizophrenia.” Although each writer attempted a full review of the literature, their reviews had only three references in common; yet Garmezy’s review had 119 refer-
ences, and Stephens's review had 101 references. How could this be?

Certainly, it was not because there were several hundred excellent articles on the subject—there were not. Nor was it due to parochial scholarship on the part of Garmezy or Stephens. Both men were familiar with and recognized the significance of the majority of papers quoted by the other. Nor could the divergence between the bibliographies have been due to widely differing points of view between the authors. While both reviewers considered the issue in doubt, both gave serious credence to the possibility that the process reactive dichotomy in schizophrenia existed.

The difference was one of focus. Over 90 percent of the papers cited by Garmezy had been written by Ph.D.'s, and virtually all the papers cited by Stephens had been written by M.D.'s. Psychologists often make clinical distinctions at one point in time; and such measures as the Wechsler Adult Intelligence Scale, the Thematic Apperception Test, the psychophysiological experiment, etc., each try to determine the diagnosis and/or prognosis of the patient at a single point in time. In the psychological literature, little effort has been made to follow up differentiated patients; rather, the investigative effort has been to refine, validate, and explore the properties of the instrument.

In contrast, psychiatrists interested in the problem of prognosis—from the days of Kraepelin—have made little effort to differentiate patients prospectively, but they have waited until the patients' life courses proved to be quite different. Then, retrospectively, they have tried to reconstruct the differences. Thus, the review by Stephens suggested that the schizophrenics with a favorable life course would have in most cases scored favorably on the Phillips Scale. On the other hand, Garmezy suggests that schizophrenics with favorable scores on the Phillips Scale of Premorbid Adjustment ought to have a favorable life course.

In our haste to construct instruments that will reliably allow investigators to agree upon who is and who is not "schizophrenic," we must not lose track of alternative means of defining schizophrenia—especially family history and a long-term outcome reflecting characteristic chronic social deficit.

A final stumbling block to understanding prognosis in schizophrenia has been how to account for the differential effects of treatment. Do schizophrenics who are virtually untreated, or patients who have received the most modern and intensive modes of treatment, best reflect prognosis? James Beck's provocative review suggests that, as physicians, we do not always control our patients' prognoses. In America from 1880 to 1950 the harder we tried to treat schizophrenia, the more congested our psychiatric hospitals became, until finally we had the highest rate of hospitalized schizophrenics in the world. It became clear that the prognosis in schizophrenia had been better in early 19th century America, or in modern underdeveloped countries, than it was in most American mental hospitals.

Some of the evidence in James Beck's paper suggests that in our treatment of schizophrenia we may be in a situation analogous to that in which Semmelweiss found himself in the management of puerperal sepsis. We are as capable of making schizophrenia worse as we are of ameliorating it.

James Beck leaves us with much the same instructions as does Manfred Bleuler. In the last four decades, the prognosis of schizophrenia has changed in only one way. As we have learned not to overtreat schizophrenia, fewer patients have become chronic. If we supply chlorpromazine, not restraints, during the early phase of the illness; if we shield the patient from too-intrusive families or hospital treatment; if we offer hospitals that provide shelter and community rehabilitation rather than involuntary incarceration and phenothiazine-induced tardive dyskinesia; if we learn to use group membership to support and not to threaten; then, although we may not increase the cures, we can minimize social disability.

In the future, prognostic studies will do well to borrow a leaf from each of the writers in this issue. First, samples must be examined from more than one diagnostic vantage point and assessed by more than one scheme of prognostic prediction. Second, patients must be followed for decades,
not years. Third, we should study first admissions. Fourth, we must learn to quantify outcome measures and to divide them into appropriate subcategories. Fifth, we must learn how to make better use of family histories. Sixth, we should use prognostic predictors as clusters, not as black or white indicators of clinical course. And finally, at the present point in history, the treatment of schizophrenia is a little bit like the management of wound healing; we dress it, and God heals it. Instead of trying to prove that a given parochial method is the best for treating schizophrenia, we may do well to study prognosis over cross-cultural samples. One of the exciting findings from the International Pilot Study of Schizophrenia (IPSS) is reported in the last article in this issue. The IPSS has documented what many observers have anecdotally reported—namely, in underdeveloped countries the prognosis of schizophrenia appears superior to what it is in the most modern Western clinics.

At the same time, if we must be modest with regard to our own efforts to alter prognosis, there is a final element that, if impossible to measure, should never be forgotten. In understanding the prognosis of wound healing, we do not want to ignore the effect of the charismatic healer. At the turn of the century William Osler wrote to a friend of his who had been treating consumptives: "That is a fine record . . . I am afraid one element you have not made proper stress upon—your own personality. Confidence and faith count for so much in these cases" (Cushing 1925). John Rosen, Elvin Semrad, Otto Will, William Sargeant, Manfred Bleuler, Frieda Fromm Reichmann, R. D. Laing, whoever else comes to the reader’s mind, each have brought a special human element to bear on the natural history of schizophrenia that cannot be easily isolated or replicated, but of which we never wish to lose sight.

References


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