What Is Schizophrenia?

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One of the main questions related to schizophrenia is, naturally enough, what is it? Such a question may seem obvious, naive, impossible, or any combination of these. And certainly it is a bit demanding to expect that anyone could say what schizophrenia is in 1,000 words. On the other hand, we felt that it was worth the effort. We hope that presenting these brief discussions on "what is schizophrenia" by persons who have worked extensively in the field will allow the reader to note areas of overlap and disagreement as well as variations in emphasis. Although no one may yet be able to provide the definitive answer, at least this collection of informed opinions may help clarify the major questions. The essays by Malcolm B. Bowers, Jr., and John Wing are the fifth in this series. Further collections of these statements will be presented in subsequent issues. Readers' responses and comments are cordially invited.—J.S.S., M.B.B., and S.J.K.

This deceptively simple question has motivated my interest in psychosis research for nearly 20 years, an interest which was initially stimulated and is currently sustained by clinical experience. This experience and the research which grew out of it have led me to a viewpoint which emphasizes the multicomponent nature of these disorders. The clinical research was based upon two kinds of studies. The first was an analysis of the early onset experience in psychosis and a comparison of this experience with that produced by hallucinogenic drugs. This work led to the conclusion that some but not all aspects of psychotic disorders could in fact be produced by hallucinogenic drugs (Bowers 1974). The second avenue of research involved the use of cerebrospinal fluid monoamine metabolites as a link between animal and human neuro-psychopharmacology. When I considered the likely clinical correlates of these neurochemical measures, it became apparent that psychiatric diagnosis as such was too derivative a concept in this regard. That is, variations in many behavioral subsystems such as motor activity, arousal, and general motivational level which seemed, in animal research, to be sustained by monoamine systems lay imbedded and to some degree hidden in the various diagnostic typologies (Bowers 1978).

Direct clinical work has strengthened further my interest in a component approach to psychotic disorders (Bowers 1977). I regularly examine psychotic individuals whose clinical evaluation requires an assessment of such factors as developmental history, hallucinogenic drug use, onset pattern, symptoms, neuroleptic drug response, self-perception of disorder, treatment compliance, residual symptoms, persistence with aftercare, and long-term experience with medication. There may be specific illnesses imbedded in the multiplicity of patterns I see, but more often I have difficulty identifying obligatory linkages between these problem areas. Nevertheless the important components emerge time and again, and require attention in the course of clinical care.

I believe this view is consonant with the conclusions of a number of other investigators (some of whom have expressed their views in this series) including Crow, Carpenter, Goldstein, Strauss, Wing, and Zubin. In my estimation, the common thread running through their work is the idea that several subdiagnostic behavioral systems comprise the essential components which are most relevant to treatment and outcome in
the psychotic disorders. Let me suggest what I think these components are.

The first component consists of the various prepsychotic deficits which characterize in differential fashion individuals who develop psychotic disorders. These personal and interpersonal liabilities have long been noted and were perhaps initially systematically assessed by Phillips (1953). The recent vulnerability research suggests the possibility that early cognitive dysfunction contributes to the development of these clinical precursors of psychosis (Marcus et al. 1981; Parnas et al. 1982). In addition, current neuroradiological evidence (Weinberger et al. 1982) and neuropsychological test findings (Golden et al. 1980) indicate that subtle but important equipmental deficits may lie at the root of the cognitive and interpersonal trait abnormalities. Indeed the eye tracking abnormalities, carefully researched by Holzman and his colleagues, may be a physiological marker linked to these deficits (Holzman 1983).

Thus it appears that in some patients the precursors, if not the actual development of some aspects of negative symptoms, precede the onset of actual psychosis. It also seems that these premorbid trait deficits may be one heritable component in the psychotic disorders (Kendler, Gruenberg, and Strauss 1973). It is interesting to speculate whether a single neurochemical abnormality is involved in the production of the wide spectrum of symptoms which may be encountered including perceptions of reference, hallucinations, delusions, and abnormal excited states. The fact that essentially all of these symptoms may be encountered in the so-called manic psychoses, for which the genetic evidence is strong, suggests that this component too many be heritable (Carpenter, Strauss, and Muleh 1973). This makes it possible to speculate whether a single neurochemical abnormality is responsible for both the trait and state components of the psychotic disorders, and a very useful discussion of this possibility with respect to dopamine has occurred (Crow 1980; Mackay 1980).

The third component, the residual or accrued deficits, is undoubtedly a complex one. As indicated above, some deficit symptoms probably exist to varying degrees before the onset of psychosis and are carried along as the nidus of negative symptom formation which may increase in the course of illness. In addition, residual psychotic symptoms, medication side effects, and the demoralization resulting from persistent social and vocational failure add to the total sum of negative symptoms over time. Whether or not some psychotic disorders bear a component of true progressive dementia is as yet unclear, but this possibility has not been excluded in the disorders which run a particularly malignant course.

What then is schizophrenia? By definition, it is a psychotic disorder with severe premorbid trait deficits and a progressively downhill course characterized by varying degrees of persistent psychotic symptoms, impoverished social and vocational attainment, and a progressive waning of emotional breadth and initiative. Is it a single disorder from birth to grave? It may be, but in the real world of clinical practice an assessment of the components discussed above allows the clinician an opportunity for in-depth and comprehensive evaluation and usually identifies a number of ways to enhance treatment.

References


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There is a strong hierarchical component in psychiatric diagnostic practice. If “schizophrenic” symptoms occur during the development of a dementing process, or as the aura to a temporal lobe fit, or in close relationship to amphetamine intoxication, most psychiatrists will assume they are “released” (to use Jacksonian terminology) by the primary organic process and that this latter will be the chief determinant of treatment, management, and outcome. If such pathologies are absent, schizophrenic symptoms will usually be given precedence over other types. In an earlier essay in this series, Seymour S. Kety (1982) pointed out that Eugen Bleuler adopted this position. It is not universally accepted but has the merit of being empirically testable.

The symptoms generally regarded as necessary for a diagnosis of schizophrenia can be divided into two broad groups: the first designated “florid,” “positive,” or “productive”; the second, “negative,” “defect,” or “deficit.” The positive group includes the experiences described most clearly by Kurt Schneider (1959) as “symptoms of the first rank” and the hallucinations and explanatory delusions based upon them. Experiences based on depression or elation are excluded. Other noncongruent delusions and hallucinations are frequently present as well. When they occur in the absence of first rank symptoms, clinicians often regard them as partial evidence for a schizophrenic disorder (World Health Organization 1973) although, for research purposes, nothing is lost by labeling them under various paranoid syndromes.

The group of negative symptoms includes flatness of affect, slowness, underactivity, poverty or incoherence of speech (and probably, therefore, of thought), and difficulty in using nonverbal means of communication. These symptoms may precede, accompany, or follow an acute florid episode. The florid symptoms have little value for prognosis (except to suggest the likely content of any future florid episode), but the severity and duration of negative symptoms are strongly associated with social outcome (World Health Organization 1979). On the whole, deterioration (deficit increasing with time) does not occur after the first few years unless brought about artificially, as will be discussed later.

There is a substantial literature (antedating the introduction of the phenothiazines) on the occurrence of movement disorders in schizophrenia (for review, see Ban 1982).

Kraepelin (1919) regarded both types of symptoms as necessary for a diagnosis of dementia praecox, thus ensuring that the diagnosis was associated with a generally poor outcome. Eugen Bleuler (1950) extended the limits of Kraepelin’s concept, not only in his descriptions of simple and latent subgroups (he was convinced that the latter was the most frequent form) but also at the margins of all the subgroups. Since then there has been a proliferation of other definitions.

The attempt, in DSM-III (American Psychiatric Association 1980), to introduce some precision into the classifying rules for differential diagnosis is courageous and timely, but the disadvantages must be taken with the advantages. The criteria for schizophrenia require many specific cutoff points (for example, on age and length of episode), and many decisions on the types and combinations of symptoms
that should or should not be included. These will not be agreed upon by all clinicians. But for public health and statistical purposes the increase in comparability is invaluable. For more intensive research, greater flexibility is necessary, and it is useful to be able to consider the individual syndromes separately (including the various groups of positive and negative symptoms of "schizophrenia"), and to treat continuous variables as such. Systems such as PSE-ID-CATEGO have made a start in providing a technology for investigation but, as the authors point out in the instruction manual, the end-classifications should not be regarded as clinical diagnoses (Wing, Cooper, and Sartorius 1974; Wing and Sturt 1978). The Diagnostic Interview Schedule provides another approach (Robins et al. 1981).

Eventually, what is required is a broad data base to which can be applied algorithms for several different classifications.

The title of this series of articles would attract less controversy if genetic, biochemical, and pathological studies provided more specific clues to classification. The evidence is highly suggestive in general, but, so far, little practical help can be offered to improve differential diagnosis, subclassification, prevention, treatment, management, or prognosis. Recent studies of environmental influences do, however, provide some guidance.

During the 1930s, admission to hospital for the first time with a diagnosis of schizophrenia led to long-term residence in two-thirds of cases. Now fewer than 10 percent stay as long as a year. Follow-up studies suggest that lasting social recovery occurs in 20-25 percent of cases, while a similar proportion suffer chronic disablement, but the experience of most afflicted people is fluctuating deficit with occasional episodes of florid symptoms (cf. Bleuler 1978; World Health Organization 1979).

Positive symptoms tend to be precipitated by threatening life-events (Brown and Birley 1968), too vigorous attempts at rehabilitation (Goldberg et al. 1977), and emotional intrusiveness on the part of some relatives or friends (Brown, Birley, and Wing 1972; Vaughn and Leff 1976). An afflicted individual has various strategies available to cope with these stressful situations. Medication is one, since it not only helps to suppress positive symptoms but is often protective, at least in the short run. It does not seem to be so necessary when environmental circumstances are favorable. Intervention, based on these principles, helps to prevent relapse (Leff et al. 1982).

Negative symptoms tend to be made worse by an environment that fosters underactivity and social withdrawal. Medication may also make negative symptoms worse. Corrective action has a rapid effect but only to a basic level of disability characteristic of each individual (Wing and Freudenberg 1961; Wing and Brown 1970). Since social withdrawal is also a means of coping with environmental intrusiveness, the point of balance between too much and too little stimulation is difficult to achieve. Afflicted individuals have to walk a tightrope with different kinds of danger on each side.

There is another, less specific, group of factors involved. The course of any chronically disabling or frequently relapsing condition is affected by the self-attitudes of the person concerned, which also depend on personal resources and the social support available. The gradual increase of dependency in "institutionalism" (Wing and Brown 1970), which has its counterpart in noninstitutional settings as well, is an obvious example. People with communication difficulties are particularly at risk if they are not able to explain their problems and thus obtain help based on realistic understanding.

The interaction of the three types of factors considered above—the two key groups of symptoms, the environmental characteristics that make each better or worse, and the personal reactions of the afflicted individual—leads to the unique pattern of problems seen in any particular individual. Not only should counseling, and the allocation of services, be firmly grounded in the knowledge currently available, but it must extend to the people who form the afflicted person's social group. The family is the obvious source of support or stress, but many people are not living in a family setting and their friends and potential supporters are just as much in need of knowledgeable advice and help (Wing 1977). Formation of groups like the National Schizophrenia Fellowship in the United Kingdom and the Alliance for the Mentally Ill in the United States demonstrates this need and suggests one way of helping to meet it.

Even if we cannot yet validate a diagnosis, or diagnoses, of schizophrenia, there is much we can do (but are not doing) to meet the needs of those afflicted.

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