A Comment on Strauss' and Carpenter's Definitions of "What Is Schizophrenia"

by Arthur Rifkin

Abstract

Any disorder can be defined as the result of a myriad of influences, but it serves little purpose to include the entire causal network as the definition—assuming that we know the causes. It is better to limit the definition to the salient features shared by all or almost all persons with the disorder. Since we know so little about the etiology of schizophrenia, or its pathophysiology, it is best to use clinical features and some gross description of course, as is described in DSM-III, as the definition most likely to be useful and least likely to contain wrong information.

I would like to comment on the definitions of schizophrenia offered by Strauss (1983) and Carpenter (1983). Assuming that we all accept the premise that by "definition" we expect some utilitarian concept rather than an unchanging "given" (such as a puppy is a young dog), then the definitions presented should be judged by their utility; i.e., do they go beyond our data, or not far enough?

Aside from adoption studies, which appear to show some degree of genetic etiology (Kety et al. 1971), we know little about the etiology of schizophrenia. Predictors usually are of the nature of showing that the earlier the illness arises, and the more premorbid difficulties the future patient has, the worse his illness will be. I realize that this statement is an oversimplification, but my point is to suggest that we distinguish patho-plastic features from more cardinal ones. There are few disorders, mental or physical, that are not affected by environmental events and characteristics of the person. Should they be included in the definition of these disorders? One reason to include them is to emphasize to caretakers that people—not organs—become ill, and we must strive to have as wide vision as possible of what affects our patients.

But the disadvantages of this broad definition are compelling to me. When the true definition of a disorder appears, i.e., when the etiology and pathogenesis have been reasonably established as in pneumococcal pneumonia, it would seem silly to define the disorder by the myriad influences which determine which person develops the clinical features and their severity. Age, living conditions, socioeconomic status, nutrition, concomitant disorders, and probably many other factors are involved in how pneumococcal pneumonia is manifested; but wouldn't it muddy the waters to try to include these in answering the question, "What is pneumococcal pneumonia?"

If we do not know what are the crucial features of a disorder, it is tempting to take the approach of Strauss (1983) and Carpenter (1983), and present schizophrenia as the end result of a multitude of variables that cover all bases. The disadvantage of this approach is its vagueness—its substituting the language of systems theory, or other models (diathesis/stress, capabilities/supports) for clear knowledge. The fear is that a more narrow definition will choose the wrong corner, reduce the disorder to some feature incorrectly, and be insensitive to modifying variables.

My preference is to take my ignorance neat rather than hide behind fuzzy generalizations. The plain fact is that we just don't know what schizophrenia is. This shouldn't...
keep us from searching for etiologies and modifying factors and using whatever treatments are shown to work. The advantage of this approach is reduction of the tendency to assume facts exist because the assumption makes sense in our broad definition. If we say that schizophrenia is the result of the full panoply of what constitutes being human (stressors, personality, environment, biology, etc.) and is different from normality by its position along a spectrum, it becomes a huge blob without clear handles onto which the researcher and therapist can grasp. There seems to be much disparity between what we really know about schizophrenia and the huge vistas of these definitions.

Choosing the best treatment suffers from broad definitions. We know so little about what works. Drugs clearly help, but not well enough. The data, however, behind any other treatment become rather thin. But if our definition includes a multitude of features, especially psychosocial ones, it is a short step (but not a logically necessary one) to the assumption that psychosocial factors are an important part of treatment, without bothering to demonstrate this vigorously as has been done with drug treatment.

I suspect that the drive for broad vague definitions partially comes from our justifiable concern that patients with schizophrenia often are not treated humanely. Perhaps if we include psychosocial features as part of the definition, that might somehow compel society to improve the disgraceful psychosocial conditions in which too many patients live.

There is no reason to tie humane treatment to poorly proven theories of psychosocial etiology or pathogenesis. In fact, it may weaken the case. Being decent to sick people shouldn’t require experimental proof. But if some variety of psychosocial approach is deemed treatment, it must prove itself in the scientific arena. If a compassionate awareness of the suffering of these patients and an attempt to behave like caring persons and alleviate the social and mental anguish is considered part of standard medical care that is due to all sick people, then perhaps citizens will not deny these benefits through their governmental bodies with the excuse that it constitutes unvalidated treatment.

References

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The Authors' Reply
Dr. Rifkin (1984) raises two important points: (1) To what extent can one help to define a disorder by focusing on social functioning and environmental impacts? (2) To what extent does this only add "fuzz" to crisp thinking, diagnosis, and research?

I think no one knows the answer to these questions definitively, although some people have believed they did. The trend over the past several years has been toward the crisp—a trend that has been led and pushed by the important advances in descriptive psychiatry. To some extent, this trend has also received its impetus from the pendulum swing of the field away from the descriptively and diagnostically vague practices of the recent past.

But how crisp is crisp? Has the pendulum swung too far toward focusing on what is most striking and most measurable? Has the attraction to symptoms as diagnostic criteria pulled us away from an even higher priority of identifying pathological processes as the core of diagnosis? Shagass, for example, has noted with irony the relative lack of merit in painstakingly describing types of cough as the ultimate basis for defining pulmonary illness. So much, at least, for one approach to nonfuzzy criteria suggested by Dr. Rifkin's example of pneumococcal pneumonia.

In the historic back-and-forth swing of science between theorizing and counting, have we gone too far in the counting direction? I believe we have—or at least some have. It seems to me a very tenable hypothesis in mental disorders that the processes defining those disorders can be identified by further attention to the relationships of the disorder to environmental characteristics and to social functioning. These relationships and types of functioning have been shown, after all, to be associated with key factors, such as prognosis, in major psychiatric disorders. As Klein and others have suggested, diagnostic groups and basic processes of disorders can be distinguished by their relationships to pharmacologic treatments and