by John S. Strauss

Abstract

The concept of negative symptoms has become increasingly important since it was reintroduced into psychiatric thinking several years ago. As the possible significance of negative symptoms has become clarified, the complexity of this concept has also become apparent. In dealing with this complexity, major progress has been made in describing negative symptoms more reliably and in taking steps toward identifying possible biological correlates. But two other particularly important directions also need to be pursued. First, psychosodal factors need to be explored further both in terms of their etiological contributions to negative symptoms and in the way these symptoms influence psychosodal aspects of the evolution of disorder and recovery. Secondly, more attention must be focused on the potential that negative symptoms have for providing an understanding of the interface between biological, psychological, and social processes in psychiatric disorder. In these ways the study of negative symptoms may contribute major clues to the nature of psychopathology, its etiologies, and its course.

When we reintroduced Hughlings Jackson's (1884) concepts of positive and negative symptoms in our 1974 article (Strauss, Carpenter, and Bartko 1974) and considered them as reflecting different processes in schizophrenia, we did so because they seemed to fit the data that had recently become available. These data suggested the relative independence of the two types of symptoms cross-sectionally and in terms of their different etiologies and prognostic significance. Subsequently, as the concepts of positive and negative symptoms have become more widely used, their origins have also become clearer, reaching back beyond Hughlings Jackson (1884) whom we had cited to the pre-Jacksonian conceptualization of Reynolds (Berrios 1985). Although it has been suggested that Fish had also used these terms (Andreasen, this issue), the work cited (Fish 1962) does not reference Hughlings Jackson and only notes positive and negative types of formal thought disorder, a distinction that barely resembles Jackson's concepts.

When we began using the positive/negative symptom conceptualization, it was immediately apparent that the Jacksonian notions had many difficulties even on the surface as demonstrated by such problems as deciding where various symptom and sign manifestations of schizophrenia fit. For example, was catatonic hyperactivity a positive symptom (sign) and catatonic muteness a negative symptom (sign)? Such a distinction, although descriptively consistent, made little sense conceptually. In spite of these problems, and our suggestion of a third dimension, social relations functioning, the basic notion seemed to be valid. Subsequent research has tended to support this validity in terms of biological correlates and the potential of the distinction for clarifying the heterogeneity found in schizophrenia.

The reports in this issue of Schizophrenia Bulletin provide an excellent overview of the more recent progress that has been made in understanding negative symptoms. Viewed from the developmental concepts of Heinz Werner (1940), this progress reflects an impressive process of differ-

Reprint requests should be sent to Dr. J.S. Strauss at Dept. of Psychiatry, Yale University School of Medicine, 25 Park St., New Haven, CT 06519.
entiation. Clinically defined subtypes of negative symptoms, syndromes, and disorders are identified (Carpenter, Heinrichs, and Alphs; Sommers; Andreasen, this issue), factor subtypes are described (Gibbons et al., this issue), various etiologies, biological relationships, treatment response characteristics, definitions, and measurement approaches have been implicated and suggested (Sommers; Goldberg; Pogue-Geile and Harrow, this issue).

Now, the pieces are all spread out on the table, or at least many of them are. Can we proceed to the next step of development described by Werner, that of integration? No. Perhaps at some time a common underlying process will be identified to fit all these pieces into one structure. My impression, however, is that now that these pieces are on the table, they will never fit neatly together again. As with so much research, biological and psychosocial, a relatively simple finding as it is explored often becomes increasingly complex. When the value of chlorpromazine for treating schizophrenia was noted, that was the most impressive fact. But as understanding increased, numerous other factors began to appear—the many actions of the drug, the side effects, the patients for whom it did not work, dysfunctions for which it worked and didn't work, and the possibility of long-term structural and functional central nervous system changes. The more we learn, the more the diversity and complexity of processes involved seem to emerge.

But even if the separate threads untangled by the reports published in this issue never again reunite, even if they subdivide further, suggestions can be made about ways of pursuing these to optimize our understanding of negative symptoms and our abilities for treatment and prevention. I would like to make two such suggestions.

The first of these relates to an area of research particularly likely to be underemphasized. The biological research for understanding negative symptoms appears to be going well. The use of new brain-imaging techniques, the improvement of their reliability and the combined approaches to using biochemical, physiological, and structural investigations of the central nervous system are well under way and exceedingly impressive. Similarly, improvement in descriptive approaches to assessment and monitoring negative symptoms, including Andreasen's recommendation regarding more longitudinal research, is also well advanced. Or at least the directions for future progress in description are clear. The most severe lack, and that in particular need of increased attention, is the understanding of psychological and psychosocial factors in negative symptoms.

The resurgent interest in negative symptoms, in fact, carries with it implications for a return to learning more about some of the psychological processes described by pioneers in the field. These processes include such concepts as "will" and "energy," concepts that have problematic but fascinating roots in the history and understanding of schizophrenia.

For a while in recent years, it was hoped by many trying to study and understand schizophrenia that Kurt Schneider's (1959) first-rank symptoms, all positive symptoms, could be considered as the hallmarks and essential processes of the disorder. One of the advantages of such a direction, had it been valid, was that schizophrenia would have been based on easily ratable, highly reliable, and clear-cut symptoms and would not need to involve vague concepts. But when it was shown that Schneider's symptoms were not pathognomonic for schizophrenia (Carpenter, Strauss, and Muleh 1973) and increased attention once again began being paid to Bleuler's (1911/1950) views about underlying pathological processes, the role and potential importance of negative symptoms once again began to emerge. The apparent hope that things that weren't easily ratable could be ignored simply did not fit the data.

So now Gibbons et al. (this issue) discuss Bleuler's notion of "goal orientation." Because of its teleologic implications, "goal orientation" is a difficult concept to measure if one is to do it justice. But the pioneers Kraepelin and Bleuler described even more difficult concepts such as "energy" and "weakness of will" (Bleuler 1911/1950, p. 70), which perhaps we had all hoped to overlook in spite of the fact that they were seen as major issues in schizophrenia.

It seems highly unlikely that concepts like energy and goal orientation will or should disappear. We have much difficulty in measuring them, but instead of avoiding or denying their existence, we need to deal with them more directly. This can be accomplished through hypothesis development and exploratory research even if we are not yet ready to study these concepts with more highly structured and controlled approaches.

And, as we get to working more with these issues, we need to focus more on the descriptive-psychosocial interface that is such an important part of negative symptomatology. Carpenter, Heinrichs, and Alphs (this issue) begin to make this point when they describe the trauma experienced by the person with schizophrenia and the possible psychological reactions
to that experience. But it will be extremely important not to pass over this humane and important concern as though we understood it or knew how to treat it adequately. For example, as Frank (1974) and others have shown, demoralization is an important, indeed often a key factor, in many kinds of mental illness. Such factors may be crucial in many instances of negative symptoms and just as important to pursue as the biological components.

Currently, phenomena such as demoralization and sources of "low energy" or poor goal orientation in schizophrenia tend to be left to the few psychotherapists who still work with such patients, to rehabilitation systems, and to so-called "aftercare" clinics (that may often be "real" care centers). But more concerted attention is warranted. What are the sources of energy or goal-directedness in people with schizophrenia? What interferes with these phenomena—discouragement, social isolation, fear, or more basic psychological dysfunctions? How can problems in these areas be avoided? How can they be optimally treated?

These are not simple questions. Just as it is important to expend great effort in pursuing the highly technical and extremely complex issues involved in brain imaging in relation to negative symptoms, it is also important to pursue the psychological and psychosocial aspects of these symptoms. The possible roles of social supports, self-esteem, hope, coping mechanisms, and their aberrations are all crucial pieces for understanding negative symptoms and for their prevention and treatment.

The second major suggestion I would like to make is that the inquiry into negative symptoms can provide a fascinating and important pathway into understanding biological/psychosocial interactions.

Carpenter, Heinrichs, and Alphs; Andreasen; Cornblatt et al.; Pogue-Geile and Harrow; Goldberg; and others in this issue have set forth various hypotheses. The possible form of these hypotheses is abstracted in figure 1.

Figure 1. Model of biological/psychological interactions in negative symptoms

\[ P \rightarrow P' \]

\[ B \]

\[ P = \text{psychosocial phenomenon.} \]
\[ B = \text{biological phenomenon.} \]

Several causal possibilities exist singly or in combination. The psychological phenomenon, negative symptoms (\( P' \)), in some instances at least, is probably caused by a psychological phenomenon, \( P \) (for example, demoralization). Negative symptoms may also be caused by a biological phenomenon, \( B \) (such as cortical deterioration, enlarged ventricles, or frontal lobe pathology). It is also possible, of course, that biological phenomenon \( B \) causes both \( P \) and \( P' \), and there may be other possible \( P's \) and \( B's \) that cause certain subgroups of each other.

But it is also possible that psychological event \( P \) may cause biological events \( B \) and \( B' \), \( B'' \), etc. This could happen through a mediating factor such as when a psychological symptom leads to the ingestion of certain kinds of medications which have a secondary effect on structural or functional characteristics in the brain, or more directly as when an environmental life stress changes a whole range of endocrine levels.

So the possible processes related to negative symptoms become more complex. In attempts to understand these relationships, it may be most useful to pursue further the direction that Andreasen notes (this issue). Genetic, other biological, and many psychosocial factors may interact over time in important ways to generate negative symptoms. These interactions may occur over years in a progressive sequence interrupted or accelerated by various phenomena. Thus, for example, it is possible that when Kendler, Gruenberg, and Strauss (1984) or Watt (1978) describe children at risk for becoming schizophrenic as having problems in relating socially, that these phenomena are potential precursors to negative symptoms. Perhaps ultimately, to understand negative symptoms, we will have to chart these sequences over time. Such a process is extremely difficult to carry out prospectively before illness onset and might be accomplished most efficiently by studying persons who have become schizophrenic (e.g., Zubin and Spring 1977). This is especially likely, if as Andreasen (this issue) and others suggest, these symptoms often develop after the onset of manifest schizophrenia.

It does not seem possible now to provide an integration of the vast number of considerations and complexities that the focus on negative symptoms has generated. The various pathways for understanding these symptoms have become clearer, however, and with this clarity is the increasing possibility of improved prevention and control of these tragic and debilitating phenomena.

References

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The Author

John S. Strauss, M.D., is Professor of Psychiatry at Yale University and Director of the Center for Studies of Prolonged Psychiatric Disorder at the Connecticut Mental Health Center, New Haven, Connecticut.