Negative Symptoms: Are They Indigenous to Schizophrenia?

by Joseph Zubin

Abstract

The sudden popularity of positive and negative symptoms (PNS) has probably arisen as a result of the need for an instrument for clustering schizophrenic patients into subgroups that would respond similarly to the various treatments. The relation of PNS to the three major models for the etiology of schizophrenia (disease, socioenvironmental, and vulnerability) has been delineated. The question was raised regarding the indigenous nature of negative symptoms, their permanency, and whether they can be used to test the tenability of the three models. A strategy was suggested for determining whether the negative symptoms are permanent features of schizophrenia by providing techniques for attempting to eliminate them through therapeutic intervention. The need for developing operational criteria for the presence of each symptom was suggested to improve reliability of ratings, and construct validity designs were formulated for increasing their validity. The provision of a valid and reliable scale for negative symptoms and a scientific model for encompassing the various claims for negative symptoms is necessary before progress can be made.

The purpose of this review was to extract the fundamental model that presumably underlies the current research in negative (and positive) symptoms by analyzing the content of the seven articles in this issue of the Schizophrenia Bulletin. After searching for a common thread on which to base the model, I was forced to give up and instead undertook to relate the various findings to three basic models of the etiology of schizophrenia: (1) the disease model (Kraepelin-Crow), (2) the socioenvironmental model (Wing), and (3) the vulnerability model (Zubin). Since the symptoms are fundamental to schizophrenia in the eyes of many investigators, the attempt to establish an independent model for the symptoms may not be possible.

Positive and negative symptoms seem to have burst on the horizon like a recurring comet. The first intimation that there was a possibility of dividing symptoms into active and somewhat passive behavior arises in Kraepelin's and Bleuler's descriptions. While the terms "positive symptoms" and "negative symptoms" do not appear in their writings, their descriptions reflected these concepts. Thus, Kraepelin regarded what is today termed blunted affect and poverty of speech as permanent characteristics of dementia praecox, while Bleuler regarded inappropriate affect and poverty of content of speech as variable characteristics of schizophrenia, giving schizophrenic patients their well-known variability (Lewine and Sommers 1984). All of these characteristics fall well into the net of positive and negative symptoms (PNS) today. PNS, however, did not begin in schizophrenia, but date back to John Russell Reynolds (Berrios 1985) who first applied them to the classification of symptoms in epilepsy, anticipating John Hughlings Jackson by 18 years. However, Jackson was the first to apply the concepts of PNS to psychiatry and classified the symptoms according to "the influence of different local bodily states and of different external circumstances on the persons . . ." (Berrios 1985). Berrios (1985) points out that Jackson did not identify . . .
either the personal or social variables that might be involved, but this task, he indicates, was undertaken by such notables as Ribot, Freud, E. Bleuler, Janet, and de Clérambault (Berrios 1985). Whether Russell’s and Jackson’s views are truly the basis for PNS in schizophrenia, or merely served as metaphors for the current use of the concepts of PNS, is debatable. Eye has pointed out that de Clérambault reintroduced PNS in 1942 but veered away from the Jacksonian view by making positive symptoms independent of negative symptoms, more in keeping with the contemporary view (Berrios 1985). The recent rebirth of interest in PNS is difficult to trace and must be left to future historians. Thus, John Wing (personal communication, 1983) states that he first used the analogous terms of “florid” and “defect” symptoms in his Ph.D. dissertation (submitted November 1959) and in *Institutionalism and Schizophrenia* with George Brown in 1970, where the terms negative and florid symptoms appeared. These uses of the concepts of negative (defect) and positive (florid) symptoms seem to antedate their use by Fish (1962), Strauss, Carpenter, and Bartko (1974), and Crow (1980). Despite the early beginnings of PNS they lay fallow until the current spate of interest rose in the wake of the publication of Strauss, Carpenter, and Bartko (1974). When the 12 items of the flexible diagnostic system proposed by Carpenter, Strauss, and Bartko (1973) were clustered, the logically based clusters that resulted were (1) items unfavorable to a diagnosis of schizophrenia, (2) items dealing with delusions, (3) items dealing with disturbances in communication, and (4) items dealing with insight and affect (Zubin 1975). An examination of the individual items in each of these four clusters indicated that they correspond in some rough fashion to PNS. Thus, category (2) is definitely a cluster of positive symptoms, category (3) is definitely a cluster of negative symptoms, and category (4) is a mixture of both.

But why did the interest in PNS develop in the last two decades and with such tremendous acceleration in the 1980s? It is my impression that the need for them arose in response to the attempted fractionation of the schizophrenic molecule under the impact of the various drug and psychological therapies. It became apparent that some therapies produced no response in some patients, good response in others, and negative responses in still others. The assumption was made that these types of responses reflected different subcategories. There arose a need for scales to predict treatment responses, since the classic subcategories of schizophrenia did not serve, and PNS were brought in to fill the vacuum.

But the available descriptions of PNS in the literature were unsuitable for clinical application and new instruments had to be developed. The field was in great turmoil, reminiscent of the days before systematic interviews were introduced to lay the foundation for improving diagnosis. There was a general lack of operational definitions for the items used in the rating scales, lack of systematic ways of interviewing as a basis for the ratings, and in general, an often perplexing overlap between the labeling of the same items as either negative or positive. As one surveys the chaotic field, one comes suddenly upon Andreasen’s clinical scales, Lewine's psychometrically analyzed scales, and Crow’s bold splitting of schizophrenia into two disorders—type I and type II. The feeling one gets in viewing these three accomplishments is like coming suddenly upon Grand Central Station in a jungle. The question is not so much how useful or valid are they, as how did they get there in the first place, and where is all the traffic going?

The question before us is what can be done to bring order out of the chaos. To this end, some type of theoretical model has to be propounded stemming out of the consensus that may be discovered in analyzing the group of articles presented in this issue with the provision that it be spelled out rigorously but parsimoniously, and be testable. One virtue of testable models is that their half-life is no greater than that of romantic love, yielding their ardor to the next sequence that arises phoenix-like from the ashes of the old. This is likely to be the fate of any model proposed now.

In order to specify the model, we first need to define the concepts involved and then spell out their various parameters so that the model can be tested for its tenability through observation and experimentation.

It would have been desirable to list the various definitions given in the seven articles and let the reader draw his own conclusions. This, however, would only be redundant. I therefore took the next best brave step and distilled what I took to be the consensus into the following statement:

Positive symptoms are behaviors that schizophrenic patients engage in, but normals do not. Negative symptoms, when inverted to their opposites (e.g., “withdrawal” to “sociability”) are behaviors which normals engage in but schizophrenic patients do not, or only in diminished fashion.

The following findings were
reported by the various contributors to this issue:

- Positive symptoms generally respond to neuroleptic treatment while negative symptoms generally do not, though Goldberg (this issue) makes a case for the responsiveness of even negative symptoms to neuroleptics.
- Positive symptoms tend to be independent of intelligence while negative symptoms show an inverse correlation with intelligence, especially in patients who show structural brain impairment and have a poor premorbid personality.
- Negative symptoms seem to persist and thus become more apparent in long-enduring episodes or multiple episodes, while positive symptoms have shorter durations and seem generally to wax and wane with the episodes.
- Positive symptoms are aggravated by amphetamine injections, presumably because dopamine transmission is facilitated. The effect on negative symptoms is either absent or nonspectacular.
- Negative symptoms correlate with deviations in brain structure especially in patients with poor premorbid histories.
- There is general agreement that both negative and positive individual items are not specific to schizophrenia. Whether the combination of items into syndromes will show greater specificity remains to be seen.
- There is some agreement that positive symptoms are associated more with neurochemical than with structural anomalies in the brain.
- It is generally agreed that there is difficulty in distinguishing between negative symptoms and depressive symptoms and criteria for schizoid personality.
- There is general agreement that positive and negative symptoms are independent and that there may be four categories of schizophrenics with regard to the presence and absence of both types of symptoms: (1) both present, (2) both absent, (3) negative present but not positive, and (4) positive present but not negative. The majority of patients seem to fall in the first category.

In developing models for PNS, three approaches present themselves: (1) phenomenological (descriptive), (2) etiological, and (3) response to treatment.

Some of the proposed conjectures for the etiology of PNS are:

- They are the result of the impact of the psychosis, leading to reduction of normal behavior because the individual has been overwhelmed by the disruption produced by the psychosis.
- The effect of antipsychotic drugs, e.g., akinesia and sedation.
- The effect of institutionalization.
- Residual effect of the psychosis after the episode ends.
- Effect of depressive episodes accompanying schizophrenia.
- Reemergence of the premorbid negative personality traits at the termination of the acute episode.

Before proceeding to develop such models, I would need to relate them to the models that now exist for schizophrenia itself in order to determine their interrelationship (Zubin et al. 1985).

The descriptive approach to PNS is the one that has been the most popular and has given rise to the most progress. It has carved out that part of the phenomenology of schizophrenia which is associated generally with the acute phase into the positive manifold and that part of the phenomenology which is associated with postepisode and long-enduring symptoms (chronic or defect state) into the negative manifold. However, these two manifolds are not mutually exclusive, and the majority of patients exhibit both manifolds. Nevertheless, there are a sufficient number of clear-cut positive symptom cases and negative symptom cases to warrant a continuation of the attempt to refine the concepts, and perhaps find distinctive patterns cutting across the manifolds that characterize subgroups.

From the point of view of etiology of schizophrenia, each of the seven etiological models—ecological, developmental, learning, genetic, internal environment, neurophysiological, and neuroanatomical (Zubin et al. 1985)—can be implicated in the development of positive and negative behaviors. The ecological niche that the patient occupies—be it in the hospital, outpatient services, family, or community—can produce PNS in response to either understimulation (negative) or overstimulation (positive or negative).

The developmental model may provide the ontogeny of both types of symptoms under the impact of such factors as maternal interaction, communication deviance in the family, and peer group influence on intimacy during the preadolescent or adolescent development of friendship patterns.

The learning theory model may provide a consideration of the reinforcement necessary for the development of negative as well as positive behavior by delineating the contingencies in which the deviant behaviors tend to occur and get reinforced.

The genetic model may provide a consideration of the hereditary traits that may begin to be exhibited premorbidly or are programmed to appear with the progress of the disorder (as is the case with
Huntington's chorea), but may be independent of it.

The internal environment model may provide a consideration of the imbalance in the metabolic homoeostasis and internal secretions of neurotransmitters. This model constitutes the basis for Crow's (1980) model and also for the akinesia and sedation symptoms.

The neurophysiological model may provide a consideration of the deviations in information processing in cognition, perception, sensation, and motor behavior that may underlie PNS.

The neuropsychological model may provide a consideration of the organic basis (enlarged ventricles, tissue atrophy) which may be associated with the PNS.

As a superordinate and integrative model to these seven submodels, the vulnerability model has been proposed.

The vulnerability model proposes that a certain proportion of the general population is endowed with a level of vulnerability which, under suitable circumstances, will express itself in an episode of schizophrenic illness. Each of the seven etiological submodels offers suggestions about the possible origins of such vulnerability. There are, however, three somewhat novel elements in our formulation. First, we acknowledge numerous contributions to an individual's degree of vulnerability, ranging from his genetic inheritance to his acquired propensities. Second, we are concerned with measuring vulnerability. That is, we are attempting to capture empirically those characteristics of individuals that might predict the probability that a schizophrenic episode will develop. Third, we distinguish between a "vulnerability" to schizophrenia, which is regarded as a relatively permanent, enduring trait, and "episodes" of schizophrenia, which are viewed as waxing and waning states. In this connection, one important area of continuing work is the search for specific "markers" associated with either the patient's underlying vulnerability to illness or the occurrence of episodes of the disorder (Zubin and Steinhauser 1981; Zubin, in press).

The vulnerability hypothesis assumes that the concept of schizophrenia does not imply a chronic disorder so much as a permanent vulnerability to develop the disorder. Episodes are usually time limited, arising in the wake of life stress and abating when its aftereffects dissipate. When the episode terminates, the individual returns to his premorbid level of adjustment. Viewed in this way, schizophrenia becomes an episodic illness in the same sense as depression, epilepsy, or allergy.

A vulnerable individual is one with sufficient etiological loading, regardless of its sources, to have the potential for a schizophrenic episode. The possession of this potential does not, however, make an episode inevitable. Prior estimates (see Zubin and Steinhauser 1981) suggest that, perhaps, as many as three of every four individuals with an etiological loading for schizophrenia never experience an identified, manifest episode of the disorder—a fact, by the way, that needs to be kept in mind when undertaking the task of choosing nonschizophrenic control subjects for biological and genetic studies. The actual probability for an individual to experience an episode of illness at some point in his lifetime seems to be contingent upon at least five kinds of factors: (1) the level of etiological loading for the disorder, (2) the overall stressfulness of the immediate environment, (3) the individual's personal competence and learned coping skills (i.e., premorbid personality), (4) the type of social network he is related to, and (5) the type of ecological niche he occupies.

PNS present the vulnerability model with a dilemma. The positive symptoms present no problem, but the negative do. According to the vulnerability model, the negative symptoms are largely associated with the so-called chronic state which the vulnerability model regards as an artifact, and not as an indigenous factor in the disorder, while the positive symptoms are largely connected with the acute state or episode and disappear with the end of the episode.

How the vulnerability model deals with this dilemma will be presented after contrasting the vulnerability model with several competing models. There are at least two competing models. First is the Kraepelinian and Bleulerian disease model for schizophrenia, which regards negative symptoms as indigenous to the disorder, and permanent characteristics of the patient. Second is Wing's model, which is midway between the disease model and the vulnerability model.

The Kraepelinian disease model regards schizophrenia as a disease similar to diabetes or arteriosclerosis and regards the negative symptoms as indigenous factors in the disease process. Thus, both the disease and the negative symptoms are permanent characteristics of the patient. Crow's (1980) model is essentially a disease model and his concept of negative symptoms fits well into the disease model, with the assumption that they are permanent indigenous aspects of his type II patients.

He postulates two etiologically distinct schizophrenic subtypes in which negative symptoms are
associated with gross structural abnormality (cerebral atrophy) and positive symptoms with biochemical dysregulation (probably dopaminergic overactivity).

Mackay (1980) goes further to postulate that negative symptoms may reflect chronic dopaminergic underactivity, while positive symptoms emerge when there is a burst of dopaminergic overactivity; but he describes a clinical picture in which negative symptoms are chronically present and on which positive symptoms are periodically acutely superimposed. Thus, according to Crow, Mackay, and their followers, PNS form a permanent feature of schizophrenic behavior.

The second model to be discussed comes from the work of Wing and his associates (Wing and Brown 1970; Wing 1978a, 1978b, 1978c) at the Institute of Psychiatry in London. This model is firmly rooted in Anglo-European traditions of descriptive psychopathology and more recent British thought about the role of social factors in psychiatric disorders (e.g., Lewis 1953, 1958). Wing’s model is primarily concerned with socioenvironmental factors affecting the onset, phenomenology, course, and outcome of schizophrenic disorders.

Wing and Brown (1970) suggest that schizophrenic patients may display at least three distinct kinds of handicaps. First, “premorbid handicaps,” such as low IQ, a difficult personality, and social withdrawal, which usually exist before the onset of illness. The authors note that these handicaps may well be “early manifestations of a disease process before it becomes clinically manifest.” Second, there are “primary disabilities” that are viewed as specific dysfunctions associated with schizophrenic disorders. Primary disabilities are of two kinds, negative symptoms (e.g., social withdrawal, flatness of affect, poverty of speech, lack of initiative, slowness, and underactivity) and florid, also called positive or productive, symptoms (e.g., delusions, hallucinations, incoherence of thought, overactivity, and odd behavior). These intrinsic impairments may exist together or they may appear as separate acute and clinical poverty syndromes (Wing 1978a). Finally, there are “secondary disabilities” that stem not from the illness process itself, but from the social and medical consequences of having been ill. In this category fall a wide variety of potential handicaps like institutionalism, social stigma, loss of self-confidence and self-esteem, induced dependence, and a myriad of other deviant behaviors (Lemert 1951) that may be induced by the patient’s social interaction with members of the medical profession, relatives, and the lay public.

The basic tenet of this model is that schizophrenic patients are biologically vulnerable to both understimulating and overstimulating environments. Of course, the exact nature of this underlying core defect cannot be fully specified, but Wing seems to favor some kind of genetically transmitted defect(s) in arousal and attention. Specifically, the model suggests:

[T]here are at least two fundamental processes at work. On the one hand, an under-stimulating social environment tends to increase symptoms such as social withdrawal, passivity, inertia and lack of initiative. . . . On the other hand, there is the tendency to break down, with an effusion of florid symptoms, under conditions of social over-stimulation. . . . These are general statements and the two processes sometimes vary together. Nevertheless, the sketch of a theory is discernible and depends upon the assumption that negative symptoms are a protective reaction against cognitive impairment. When the patient is allowed to withdraw, he does so, and the process can easily go too far. When he is not allowed to withdraw, but faced with what seem to be impossible demands, the underlying thinking disorder becomes clinically manifest in florid symptoms. [Wing and Brown 1970, pp. 21-22]

Let us recall that schizophrenia, as it is currently defined in most textbooks of psychiatry, involves a central or acute syndrome, as well as long-term states of impaired functioning (see Wing 1978a).

Perhaps the most important feature of this model is the provision of a common sense, clinical, and social framework for (1) explaining observed patterns of symptomatology in schizophrenic patients, and (2) predicting the effects of environmental factors on the course and outcome of the disorder. It serves as an alternative and useful corrective to theories that emphasize a “ruthlessly progressive” disease process amenable to little modification (Wing 1978b). In this role, Wing’s model proposes a relatively concrete and researchable vision of the social conditions under which we may expect to observe maximal improvement in schizophrenic patients:

The optimum environment is presumably well structured, with clear lines of behavior laid down and a neutral type of social stimulation which does not lead to emotional over-involvement, both thought disorder and withdrawal are then minimized. [Wing and Brown 1970, p. 22]

The major points of difference between this model and vulnerability theory lie in the interpretation of premorbid handicaps and negative symptoms (i.e., the clinical poverty...
syndrome). Vulnerability theory assumes that schizophrenia is an episodic rather than a chronic disorder. Between episodes, patients are expected to return to their premorbid levels of functioning, unless secondary factors unrelated to the individual's condition intervene to prevent this from occurring. For the vulnerability theorist, premorbid handicaps are independent (i.e., nonillness related) personality variables that interact with the patient's liability to episodes of schizophrenia, whereas Wing and his co-workers tend to view them as early subclinical manifestations of the patient's growing sensitivity to overstimulating and understimulating environmental situations. The importance of this distinction should become clear once we have had the opportunity to discuss the differences in the two models with regard to negative symptoms (i.e., the clinical poverty syndrome).

The model proposed by Wing and his co-workers suggests that negative symptoms (or the clinical poverty syndrome) are an "intrinsic" or "primary disability"—that is to say, a deficit in functioning that is a specific consequence of the patient's biologically grounded vulnerability to overstimulating and understimulating environments. Vulnerability theory, on the other hand, argues that only florid symptoms (or Wing's central syndrome) should be classified as primary handicaps. Negative symptoms and the clinical poverty syndrome are assimilated completely within Wing's category of secondary or external disabilities. In other words, vulnerability theory takes the position that negative symptoms are neither an inevitable (Kraepelin) nor an intrinsic (Wing) feature of schizophrenic disorders. It is proposed, instead, that the clinical poverty syndrome, as defined by Wing (1978b), is essentially an artifact or a social consequence of having been identified, labeled, and treated as schizophrenic by medical specialists, relatives, close friends, and other members of the patient's social network.

Going beyond the immediate issue of negative symptoms, the vulnerability theorist's insistence on the episodic nature of schizophrenia raises a larger question—viz., how to explain the fact that a certain proportion of schizophrenic patients at least appear to remain chronically ill. There are four possible reasons that require mention: (1) A substantial number of these patients may have been of such poor premorbid ability that their recovery from illness is difficult to discern. In contrast to affected individuals with good premorbid skills, who resume their roles in society and recover unremittingly, these "pseudo-patients" remain unable to cope adequately with their social milieu even though their episode of illness has terminated. Second, there is a group of chronic patients whose coping ability deteriorates far below its premorbid level as a consequence of factors like isolation in the hospital or community, labeling, and loss of social skills. Gruenberg (1967) has described a "social breakdown syndrome" occurring in such patients, a condition frequently mistaken for permanent psychopathology. This category also includes the vast majority of patients suffering from what has been called the clinical poverty syndrome. Third, some patients recover only briefly before falling back into an episode; their periods of emergence from the disorder are so short-lived as often to go unnoticed. A situation of this kind may be brought on by one (or a combination) of two possible situations. Either the patient lives in (or returns to) such a stressful social environment that any clinical improvements are quickly negated or the patient is so vulnerable that he may be catapulted back into an episode of illness by participation in uneventful social interaction (cf. Leff et al. 1973). Finally, there is probably a small proportion of patients—not more than 6 to 10 percent according to M. Bleuler (1978)—who may, in fact, remain chronically ill without a true remission. (For further discussion of the pros and cons of chronicity, see Zubin et al. 1985.)

The findings reported in the series of articles in this issue cannot be encompassed completely in any one of the three models presented here and thus provide a challenge for each of them. The general trend in these articles is to accept the disease model, but all of the authors are not easily fitted into the procrustean bed that this model provides. Thus, Carpenter, Heinrichs, and Alphas (this issue), especially, challenge the tenability of the disease model, and both the socioenvironmental (Wing) model and the vulnerability model (Zubin) are at loggerheads in many instances with the disease model. The resolution of these conflicts will have to wait for future research.

Future Perspectives

Permanency of PNS. With regard to positive symptoms, it is assumed that they largely wax and wane with the episode. With regard to the negative symptoms, a quandary exists. They usually are associated with so-called chronic states. The question arises as to whether they are indigenous to the schizophrenic process (the question is also raised regarding the chronic state itself). It is clear that some of these negative traits may have existed
premorbidly, and their persistence postmorbidly is due to the return to the premorbid status. But what about the traits that did not exist premorbidly? Are they part of the schizophrenic process?

One way of answering this question is to determine whether the negative symptoms are mutable. If they are not, perhaps we can accept them as part of the schizophrenic process and not an incidental effect produced by iatrogenic, nosocomial, and other ecogenic influences. If they are mutable, the likelihood is that they may have been produced as side effects. But the question remains whether the intervention that eliminated them represents an efficacious way of treating the disorder instead of eliminating a side effect. Only the presence of these side effects in nonschizophrenic patients who are subjected to the noxious niche which postepisodic schizophrenics occupy in life can finally resolve this issue.

Depending on the presumed source of the negative symptoms, the following strategies, based in part on Carpenter, Heinrichs, and Alphs (this issue), are proposed:

* If the positive symptoms are a response to the psychosis resulting from self-preoccupation or from a defensive maneuver to dampen its impact, they should generally disappear with the recovery from the episode, provided that they have not been stamped in by nosocomial and/or iatrogenic influences.

* If they are induced by drug treatment (akinesia, sedation), a change in the treatment regime should eliminate them.

* If they reflect the understimulation that Wing and Brown (1970) found to be the case in many instances, they can be eliminated by environmental manipulation.

* If they are induced by the acute psychotic episode or postepisodically, proper therapeutic intervention should be found by clinical trial and error, leading to their elimination.

* If these strategies fail to eliminate the negative symptoms, they could be regarded as genuine aspects of the schizophrenic process that give schizophrenia its chronic characteristics. However, before this conclusion is accepted, it would be of interest to determine whether these negative symptoms are not a reflection of the premorbid personality characteristics.

* Another question that needs to be tackled is whether negative symptoms are specific to schizophrenia and not found in depression and other psychiatric or nonpsychiatric statuses. For example, are they characteristic of individuals who are neglected or isolated through incarceration in prisons or poorly managed nursing homes. Only comparative studies can answer this question.

All these strategies require, in addition, a long-term followup to determine the permanancy of the negative symptoms and whether they return after being eliminated.

Construct Validity. The current technique for determining the presence of negative symptoms is based on inventories or interviews. But there is a rich psychological literature dealing with the experimental measurement of some of these symptoms, and it would seem wasteful to depend only on interviews or inventories when reliable instruments for assessing construct validity could be developed (cf. Sommers, this issue). Thus, blunted affect or flatness of affect has been measured by means of reinforcement techniques in which the degree of affective blunting is reflected in the rate of increase in affective utterances during a reinforcement period compared to the rates during operant and extinction periods (Salzinger 1984).

Comprehensibility of speech has been measured by the Cloze technique (Salzinger 1984). Poverty of speech and poverty of content can be assessed by analyzing tape recordings of the interview or by requesting the patient to tell a story. There is a scale for anhedonia (Chapman, Chapman, and Raulin 1978), and there are scales for anxiety and dysphoria. Attention has been measured by a variety of techniques—crossover and cross-modal reaction time, continuous performance task, backward masking, and span of apprehension (Zubin, in press).

The purpose of introducing the experimental techniques is to provide external criteria for validating the clinician's judgment regarding the assessment of each symptom. Since both the results of the experimental techniques and of the clinical judgments are fallible, a comparison of a group of schizophrenic patients by both methods will result in four types of groupings: a group concordant for the presence of the symptom by both methods; a group discordant for the absence of the symptom; and two discordant groups (one in which the clinician judges the symptom to be present but the experiment denies it, and vice versa). By examining the two discrepant groups, it may become possible to revise both methods until the number of discrepant individuals is reduced to a minimum. In this way the validity of the scale can be raised to the maximum.

Statistical Strategies. Among the statistical strategies that might be used are the following:
In addition to factor analyses of the currently available scales, a taxon analysis (Golden 1982) might be used. This approach clusters individuals instead of items and provides subgroups of individuals who are more homogeneously like-minded in the negative symptoms they exhibit. Perhaps the study of Kraepelinian and Bleulerian negative symptoms by Lewine and Sommers (1984) would have yielded more clarifying results if the factor analysis had been skipped and a direct clustering technique applied to the original data. The likelihood is that the items did not satisfy the prerequisites for computing correlations and consequently the factor analysis of these correlations may not have been entirely justified.

Since the scales depend on the responses of the patient, it might be of interest to see whether readiness to respond or admit presence of the negative symptoms might be a factor. It might be interesting to prepare the item list in such a form that signal detection theory methods may be applicable to separate the "criterion" component from the "sensitivity" component in the patient's response. A technique for accomplishing this has been provided by Clark et al. (1976).

Rating Problems. One of the major problems facing the scaling of negative symptoms is the problem of rating. Positive symptoms do not present as much difficulty as negative symptoms. The dramatic nature of most of the positive symptoms (delusions, hallucinations) is so striking that they do not require microscopic scrutiny for the detection of their presence. In the case of the negative symptoms, as Sommers (this issue) points out, the problem is rating something that is absent! The line of demarcation between normal and abnormal behavior is much more difficult to determine. There are no standards against which normal sociability can be assessed. Consequently, abnormal sociability (withdrawing behavior) cannot be assessed as readily as hallucinatory behavior. Questions of frequency, contingencies, duration, and intensity enter for which no norms exist. One solution is to establish operational criteria for each symptom similar to the operational criteria established for diagnostic categories. Andreasen (this issue) has begun the development of such criteria for some of the items. How to standardize such a procedure for all of the items will require the cooperation of all those concerned with the problem.

Summary

It is astonishing to see how PNS have suddenly bloomed into a thriving field of research in which outstanding researchers are engaged when the conceptual framework is so nebulous and the techniques so tenuous.

If fractionation of schizophrenia is the main purpose of PNS research, a caveat must be entered. After World War II, Stockings (1945) suggested that the then current nosology be abolished and instead a system based on response to treatment should be introduced. Thus, patients who responded to insulin treatment should be classified as dysglycemic and their characteristics noted so as to place them in a separate category from patients responding to electroconvulsive therapy who would be classified as dysoxic. The short life of this proposed system should serve as a warning to those who would use the therapies of today as a basis for classification.

Empirical research has outdistanced theory in the field of PNS, and there is a great need for a conceptual approach to harness purposefully the energies spent on empirical measurement and on honing of instruments. PNS must be linked up to the currently available concepts of the phenomenology of schizophrenia, its etiology, and its response to treatment before progress can proceed.

An urgent question that needs to be tackled is the following: Are negative symptoms indigenous elements in the psychopathology of schizophrenia, or are they artifacts of iatrogenic, ecogenetic, and nosocomial forces that impinge on the patient?

Carpenter, Heinrichs, and Alphs (this issue) have proposed a set of strategies for answering this question, to which has been added the need to determine whether the negative symptoms are a recrudescence or persistence of the premorbid personality after the episode ends.

Several authors, especially Sommers (this issue) and Andreasen (this issue), have stressed the need for what might be called research diagnostic criteria for each symptom in order to provide operational criteria for determining their ratings. To this might be added the need, as Sommers points out, of providing construct validity for the symptoms, and several techniques for providing such construct validity have been suggested.

The beauty of the Crow model is that it is specific and testable. Its shortcomings are that it is limited to a biochemical dysfunction and anatomical lesion approach. For a comprehensive attack on this problem, we need the full panoply of the environmental, genetic, neurochemical, and anatomical disciplines, as Andreasen (this issue) so aptly points out.

As for the future outlook for PNS,
it cannot help but cast light on the problem of schizophrenia research, even if the attempt to use them as a scalpel for dissecting schizophrenia becomes obsolete.

References


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