Negative and Positive Symptoms of Schizophrenia in Relation to Chronicity from the Point of View of Vulnerability Theory

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INTRODUCTION

The general consensus in the Schizophrenia Bulletin Symposium on Negative Symptoms in Schizophrenia [1] was that positive symptoms occur most frequently during acute episodes only, and wax and wane with the rise and fall of the episode; and respond to neuroleptic treatment. Negative symptoms, on the other hand, relate to the so-called chronic phase of the illness, are largely irreversible, and do not respond to neuroleptics.

From the point of view of vulnerability theory, positive symptoms present no problem. However, negative symptoms present a severe challenge to the tenability of vulnerability theory because the very concepts of chronicity and irreversibility are antithetical to its assumptions. My purpose today is to present a critique of negative symptoms from the point of view of vulnerability, pointing out that the chronic features and irreversibility which negative symptoms rest on, are not indigenous elements in the natural history of schizophrenia. This may sound as sheer heresy but following Bertrand Russell - whatever is worth stating is worth overstating!

Let us first define vulnerability theory. It belongs to the general domain of diathesis - stress theory promulgated by Paul Meehl, David Rosenthal, Falconer and others. My initial formulation of this theory dates back to 1963, [2] just about the time that Paul Meehl published his seminal paper. The vulnerability theory was formulated as a compromise between the two extreme views on the etiology of schizophrenia - the biological vs. the environmental. The best contrast between these two polar extremes and their middle ground in vulnerability theory can be seen in an analysis of the various etiological models that those theories gave rise to. Elsewhere [3] it has been pointed out that the biological theory has given rise to four models: genetic, internal environment, neuropysiological and neuroanatomical. The developmental and learning theory model partake of both the biological and environmental etiologies, while the ecological model is primarily environmentally based.

Which theory is to be followed and which model should form the basis of our effort?

There is no absolute answer today. Each model has its adherents. But interdisciplinary approaches tend to incorporate some selected few of these
models, but not all. The vulnerability model proposes to make room for all of them as submodels, since it regards a person in a schizophrenic episode as essentially a vulnerable person regardless of which etiology his vulnerability springs from [3].

Vulnerability theory hypothesizes that episodes of the disorder, characterized by positive psychotic symptoms (e.g., delusions, hallucinations, thought disorder), must be "triggered" by stressful life events. When they occur, such episodes are usually time limited, arising in the wake of life stress and abating when its aftereffects dissipate. Between episodes the individual generally returns to his premorbid level of personality functioning. Viewed in this way, schizophrenia becomes an episodic illness in the same sense as depression, epilepsy or allergy. It has also been hypothesized that the pathogenic effects of life stress may be circumvented through the action of various buffering factors. For example, premorbidly learned coping skills or favorable social conditions may prevent stressful events from reaching the level of environmental challenge required to provoke an active episode of illness.

So much for theory. Now what about evidence? The theory is too young to have been subjected to full tenability tests, but here is some of the evidence in its favor. There are 3 parameters to the vulnerability model that we have extracted from vulnerability theory: 1) A measure of the vulnerability itself; 2) Triggers to elicit the vulnerability, and 3) Moderating variables to cushion the impact of the stressors.

1. We have unfortunately not yet found any quantitative measures of vulnerability but have begun to find markers for identifying the vulnerable (A review of this effort appears in a symposium conducted at the American College of Neuropsychology) [4].

2. Brown and Birley's [5] finding that in the three weeks preceding an episode 60% of schizophrenics tend to experience at least one life event stressor has been confirmed in the WHO study [6]. Why are there no similar triggering events in the remaining individuals who undergo an episode? It is likely that the stress required for triggering an episode can be generated not only by dramatic life events but also by the accumulation of minor stresses such as the hassles of everyday living.

3. Even when a triggering life event stressor impinges on a vulnerable individual, an episode need not develop if the stress can be cushioned by a moderating variable such as, a good social network, a good ecological niche or a good premorbid personality [3].

DIFFICULTIES OF THE VULNERABILITY MODEL

1. Chronicity - The problem of chronicity sticks in the craw of vulnerability theory - since according to this theory, chronicity may be an artifact produced by iatrogenic, ecogenic or nosocomial factors. Professor Ciompi [7] and I have independently arrived at a challenge to the indigenous
character of chronicity. The arguments are presented in a recent publication [3].

2. Negative symptoms. Since chronicity is associated with the presence of negative symptoms, the prior arguments should cast doubt on the indigenous character of negative symptoms. Further arguments against the indigenous nature of negative symptoms are presented elsewhere (3).

In general, negative symptoms, such as being withdrawn or lacking in energy, when inverted to their opposites - outgoing and energetic behavior - are characteristics which normals have, but schizophrenics do not have. On the other hand, positive symptoms are behaviors which schizophrenics have but normals do not have. From the point of view of measurement, negative symptoms present a dilemma - how can the absence of something be measured? Especially, is this a problem when the lower boundaries of sociability and energetic behavior expected in normals have never been established. Furthermore, the negative symptoms may have arisen not as genuine parts of the disorder but as reactions to the fact of having experienced an episode [1]. It is likely that after experiencing an episode, patients often develop an adaptive process of social disengagement that permits them to come to terms with their negative internal feelings, hostile attitudes of their environment and the general effect of being labeled as a former schizophrenic patient. Despite former normal striving for achievement, a reduction takes place in their level of aspiration, and a whole or partial withdrawal takes place from the kinds of social settings experienced as overly stressful and demanding. Demoralization sets in, making return to premorbid level of adjustment impossible, and instead a clinical poverty syndrome sets in.

In order to establish the origin of this adaptation syndrome as to whether it is due to environmental rather than to indigenous biological factors, it would become necessary to demonstrate that it is an irreversible and inevitable development in long-term schizophrenics. Targeted approaches to eliminate these negative symptoms, if successful, would lead to the conclusion that they are not as indigenous to the disorder as is thought by some of the biologically oriented workers. But even if they are irreversible, it is still possible that they are the residues of premorbid characteristics and hence not necessarily indigenous elements in the disorder.

REFERENCES


3. Zubin, J., Steinhauer, S.R., Day, R., & van Kammen,


