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Toward the Obsolescence of the Schizophrenia Hypothesis

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The disease construction of schizophrenia is no longer tenable. That construction originated during a period of rapid growth of biological science based on mechanistic principles. Crude diagnostic measures failed to differentiate absurd, unwanted conduct due to biological conditions from atypical conduct directed to solving existential or identity problems. The construction was communicated – in the absence of solid evidence – by medical practitioners by means of symbolic, rhetorical, and organizational acts. The patient came to be regarded as an object without agency or goals. In spite of enormous research funding, no biological or psychological marker has been discovered that would differentiate diagnosed schizophrenics from normals without creating unacceptable proportions of false positives and false negatives. Employing a moral category, "unwanted conduct," as a criterion, and tacitly transforming moral judgments to the medical category, schizophrenia, leads to the use of schizophrenia/nonschizophrenia as the independent variable in research designs. The failure of eight decades of research to produce a reliable marker leads to the conclusion that schizophrenia is an obsolescent hypothesis and should be abandoned.

Any effort to criticize or clarify the concept of schizophrenia must begin from the position that "schizophrenia" is a hypothetical construct. Notwithstanding the use of the term to denote a firm diagnostic entity by most textbook writers and clinical practitioners, investigators by the hundreds are still trying to establish the empirical validity of the construct. The output of published and unpublished research directed toward establishing empirical validity has been enormous, yet schizophrenia remains an unconfirmed hypothesis. A great deal of the research is directed to the task of breaking out of the circular reasoning in which "schizophrenia" appears on both sides

This essay borrows from a more extended set of arguments prepared for an international conference on schizophrenia at Clark University, Worcester, Massachusetts, June 10–11, 1990. The conference paper will be included in a book recording the proceedings. I am grateful to a number of friends and colleagues who offered suggestions to improve the essay, among them, Mary Boyle, Ralph M. Carney, David Cohen, Philip Cowan, Daniel B. Goldstine, Norman S. Greenfield, James C. Mancuso, and Frederick J. Ziegler. Requests for reprints should be sent to Theodore R. Sarbin, Ph.D., Adlai E. Stevenson College, University of California, Santa Cruz, California 95064.

of a causality equation: unwanted behaviors are taken to be symptoms of schizophrenia; schizophrenia is the cause of unwanted behaviors.

Historical accounts of psychiatry and psychology make clear that the core hypothesis – schizophrenia as a disease entity – continues to serve as an implicit guide to the construction of current versions of the schizophrenia concept. The schizophrenia construction continues to be employed in spite of the well-documented fact that it has been submitted to repeated empirical tests and has been found wanting. My thesis is that decades of research have not provided determinate findings that justify continuing the use of schizophrenia-nonschizophrenia as an independent variable. Having voiced this claim, I quickly add that my judgment of the failure of the schizophrenia hypothesis is in no way a disclaimer to the observation that some people, under some conditions, engage in conduct that others might identify as mad, insane, bizarre, foolish, irrational, psychotic, deluded, inept, unwanted, absurd, or plain crazy.

The focus of my paper is that schizophrenia is a construction put forth by nineteenth century physicians and elaborated within an epistemological context that supported the notion that unwanted conduct was caused by disease processes. Historical forces in the nineteenth century influenced doctors to regard perplexing conduct as the outcome of a subtle brain disease. The opacity of the term "schizophrenia" directed scientists and practitioners to employ a prototype when writing their own definitions or when labelling putative patients. The contemporary construction of schizophrenia is consistent with the prototype of a person with an infectious brain disease. The crude diagnostic efforts of the late nineteenth and early twentieth centuries failed to differentiate patients with organic brain disease from patients employing atypical conduct to solve their identity and extistential problems. Because so many diagnosed schizophrenics did not fit the specifications of the prototype, some authorities, notably Eugen Bleuler, suggested the employment of the plural, "the schizophrenias." This stratagem has not been productive, but has preserved schizophrenia as a sacred emblem of psychiatry when experiments have yielded indeterminate results. "The schizophrenias" and its modern equivalent "schizophrenia spectrum disorders" have also been employed to increase the size of an experimental sample in order to achieve statistical significance. Such miscellaneous categories do little more than supply Greek or Latin labels to formalize the lay concept that "people can be crazy in different ways and for different causes or reasons."

Nearly 50 years ago, when I had my first encounters with hospitalized patients, I was confronted with the official lore that schizophrenia was a disease. I did not accept, however, the official lore without reservation. Day to day interactions with inmates of a mental hospital influenced me to be tentative about adopting the prevailing doctrine. In the course of working with men

and women who had been diagnosed as schizophrenic by appropriately-qualified psychiatrists, I became aware of the multifarious actions that were interpreted as "presenting symptoms" – actions that family members or employers could not readily assimilate into their constructions of acceptable conduct.

My first patient was a middle-aged women who held the belief that agents of a foreign power were conspiring to kidnap her; the second was a man who believed that his neighbor was directing magnetic rays to the nails in his shoes so that walking was a great effort; a third was a 40 year-old man who argued with an absent opponent about metaphysical propositions; a fourth inmate behaved as if he had lost all power of speech; a fifth would not leave his room, even for meals, afraid that he would be the object of massive microbial invasions; a sixth, a seminary student, claimed to be a saint of the thirteenth century; a seventh, a retired baker, held friendly conversations in the privacy of his room with two long-dead religious figures.

Along the way, I worked with other schizophrenics whose "presenting symptoms" added to the heterogeneous array of actions, the meaning of which could only be constructed from detailed knowledge of their self-narratives. One of these cases was a young man who, for reasons that he initially kept secret, refused to eat, although he acknowledged that no one was trying to poison him nor was he bent on suicide. I spent hours with a college student who held the belief that the disembodied spirit of a convicted mass-murderer had entered his body. Another clinical experience involved trying to understand the reasons that a 20 year-old woman claimed that her recently-deceased brother was alive and visited her frequently.

In most cases, these actions were so specific to the individual's life story that it was difficult for me to accept the explanation that some brain anomaly could account for the heterogeneity. The notion of a common cause for such an assortment of human actions can be entertained only if, in Procrustean fashion, we reduce the interesting array of polymorphous actions to a small number of categories, for example, delusions, flattened affect, and hallucinations, and further, if we arbitrarily redefine the categories as "symptoms" of a still-to-be-discovered disease entity. Such a redefinition obliterates the specificity, the individuality, and the problem-solving features of each person's conduct. Further, the acceptance of the redefinition renders irrelevant the search for intentions and meanings behind perplexing interpersonal acts.

Search Strategies

In the early 1960s, I undertook seriously to question the lore that had grown up around the schizophrenia concept. I followed two strategies: the first was to determine the epistemic and social pathways from particular actions of

putative patients to diagnostic judgments by mental health professionals; the second was to determine from a search of the published experimental literature whether a stable set of referents had been discovered that would give body to the schizophrenia hypothesis.

The Search for Unique Symptoms

To implement the first strategy, I chose to delimit my search to behavior that is regarded as a "symptom" of schizophrenia by knowledgeable specialists. In an informal survey, I presented to 15 colleagues (psychiatrists, clinical psychologists, psychoanalysts) the following question: Of the many behaviors that are supposed to point to a diagnosis of schizophrenia, which would you regard as the single most significant item of behavior in establishing a diagnosis of schizophrenia? All but three of the respondents listed "hallucinations," and many added that the presence of hallucinations reflected an underlying thought disorder. The results of this informal survey were similar to those reported by Willis and Bannister (1965) who surveyed the opinions of 346 English psychiatrists. This more extensive survey made clear that "thought disorder" was considered the most important characteristic of schizophrenia.

For the next few years, I studied "hallucinations" in the laboratory, in the clinic, and in the library (Sarbin, 1967, 1972a; Sarbin and Juhasz, 1967, 1970, 1975, 1978, 1982). I was interested in the judgmental processes of diagnosticians who would classify a person's reported imaginings as "hallucination." But before addressing the diagnostic process, it was important to determine whether "hallucination" was a property exclusively of persons who were candidates for psychiatric diagnosis, persons who, in the professional vernacular, had sick minds or damaged brains. In tracing the history of the term "hallucination" from the sixteenth century to the present, it became clear that the conduct upon which the attribution "hallucination" is made is no more than the self-report of imaginings. It is important to note that imaginings (no matter how wild) that are not reported through word or deed do not become candidates for the label "hallucination."

The word "hallucination" belongs, in behavioral terms, to a family of words that includes day-dreaming, imagining, fantasy, fancy, fictions, inventions, and fabrications. Common examples of imagining include childhood imaginary companions, adult dreams of glory, imaginary interactions with celebrity figures, "the voice of conscience," and playful or romantic fantasies. The imaginings that are constructed by so-called normals appear to cover the same range of topics as the imaginings of psychiatric patients. The claim of "reality" for the imagining is not exclusive to persons identified as schizophrenic. In one experiment, for example, volunteer college students were

induced to imagine tasting salt solutions and subsequently were willing to testify in a court of law that they had tasted salt. They had only tasted distilled water (Juhasz and Sarbin, 1966).

Besides membership in a readily-understood class of behaviors, the word "hallucination" belongs to another class, the defining property of which is a pejorative value judgment. The value judgment, of course, is rendered not by the putative patient, but by another, usually a person with greater social power than the "hallucinator." To employ the concept of hallucination, then, involves two actors, the person reporting his or her imaginings and the person who is empowered to pass judgment upon such imaginings.

Since the turn of the century, psychologists have been exploring the proposal that "hallucinations" are not uncommon among the general population. That is to say, people have experiences in which they assign a high degree of credibility to imaginings, in some cases they assign the same degree of credibility as to veridical perceptions. Ethnographic studies of subcultural pockets in the United States make clear that reported imaginings (e.g., "I could feel the Holy Ghost enter my body") are not identified as hallucinations by fellow participants in the culture. In fact, such a report sometimes leads to the person being assigned honorific status in the subculture. The same imagining reported to a conventional diagnostician in a forensic or clinical context could be evaluated as meeting the official criteria for hallucination and could lead to the diagnosis of schizophrenia. Whether or not the "hallucinator" would be classified as schizophrenic would depend upon other moral judgments. If, in the eyes of the diagnostician, the person had already suffered a degradation of identity, then the reported imagining could be employed to support the schizophrenia diagnosis. Social status considerations may insidiously insert themselves into the clinician's diagnostic matrix. The frequency of schizophrenia diagnoses among persons who are poor and black supports the claim that social structural features of the diagnostic setting supply a readiness for professionals to employ pejorative interpretations of atypical conduct. For a person whose identity has not been previously degraded, the reported imagining can be assigned to other classes, such as creative imaginings, poetic language, mystical experience, even metaphor.

Social status appears to play a part in diagnosticians' categorizing of perplexing behaviors, among them, admissions of atypical beliefs. Such beliefs of a socially degraded person, sometimes shared as in popular superstitions, are more likely to be regarded as "delusions" and thus symptomatic of schizophrenia. The superstitions of persons whose social identities have not been devalued are likely to be interpreted as quaint, or accepted as harmless, empirically-empty beliefs. Whether or not a particular belief is identified as delusional has nothing to do with "truth." One can point to beliefs held by previous generations of scientists that were declared erroneous by later scien-

tists. Delusion would not be employed as a term to mark the false beliefs of respected scientists.

The conclusion to my efforts to understand hallucination and delusion was that the process of constructing imaginings and beliefs was the same for so-called schizophrenics and so-called normals. The technical and pejorative terms, hallucination and delusion, were selectively assigned by clinicians to devalued or degraded persons as symptoms of disease. In most instances, those who employed these terms were unconcerned with fathoming the meanings of such behaviors or the part such behaviors played in the patient's life story. Among the exceptions to this generalization is a study reported by Benjamin (1989). In a carefully crafted investigation she demonstrated that the auditory hallucinations of psychiatric patients were meaningful and reflected widely observed interpersonal themes. Further, the "voices" appeared to have an important adaptive function for the patients.

The Search for Research Support

The second strategy in my search was to determine to what extent, if any, the published research could be used to support the schizophrenia hypothesis. It was not unreasonable to suppose that the schizophrenia hypothesis must have some validity because so much journal space was devoted to experimental studies. In the early 1970s, I made some casual forays into the experimental literature, looking for support for the then-popular theories of schizophrenia (Sarbin, 1972b). My preliminary analysis made clear that most of the theories of schizophrenia had been initially supported on the basis of one or two experiments. When replicated by other investigators, the results of the experiments proved to be artifacts. Each theory had a short period of enthusiastic support and then a marked decline. The rise and fall of theories of schizophrenia led me to conclude that such theories have a half-life of about five years. The conclusion applied to somatic theories and psychological theories alike (Sarbin, 1972a). Cutting (1985) arrived at a similar conclusion and added: ". . . of all the proposed causes of schizophrenia, biochemical ones have the shortest life-span" (p. 138).

My preliminary excursions called for a more systematic analysis of the published literature. Professor James Mancuso joined me in a project to review every research article on schizophrenia published in the *Journal of Abnormal Psychology* for the 20-year period beginning in 1959[1] (Sarbin and Mancuso, 1980). We selected this journal because of its high standards, the average manuscript rejection rate being about 80 percent. (To avoid the criticism that

[1]Until 1964, *Journal of Abnormal and Social Psychology.*

we had introduced a bias in selecting a psychological journal, we appended to our analysis a review of selected articles from psychiatric journals.) We found 374 reports of experiments designed to illuminate the concept of schizophrenia. By any standard, the published research on schizophrenia during the 20-year period represented a prodigious effort. It is abundantly clear that in the period under review, students of deviant conduct focused on the central problem: to identify a reliable diagnostic marker, psychological or somatic, that would replace subjective (and fallible) diagnosis. The discovery of such a marker would establish the long sought-for validity for the postulated entity, schizophrenia.

In nearly all the studies, schizophrenia/nonschizophrenia was the independent variable. To accomplish their mission, investigators compared the *average* responses of "schizophrenics" on experimental tasks with the *average* responses of persons who were not so diagnosed. It is no exaggeration to say that the experimental tasks devised by creative investigators numbered in the hundreds. All were constructed for the purpose of rigorously testing miniature hypotheses, the origins of which were linked to the postulate that schizophrenia was an identifiable mental disease or disorder. The choice of these variables was influenced by the lore of schizophrenia, beliefs that could be traced to Kraepelin's and Bleuler's claims that schizophrenics were cognitively or linguistically flawed; perceptually inefficient; affectively dysfunctional; and psychophysiologically impaired. The experimental hypotheses were formulated from the expectation that whatever the task, the schizophrenics would perform poorly when compared with the performance of a control group. The range and variety of the experimental tasks suggests that the formulators of these experimental hypotheses shared the conviction that "schizophrenics" were persons who were basically flawed, that the putative disease affected all somatic and psychological systems.

Mancuso and I analyzed 374 studies on several dimensions. We drew a number of conclusions, among them, that the criteria for selecting subjects were less than satisfactory. The unreliability for psychiatric diagnosis notwithstanding, the experimenters were satisfied to accept diagnoses made by "two staff psychiatrists," "by a psychiatrist and a psychologist," "by consensus in diagnostic staff conference," etc. It is unknown to what extent the diagnosticians employed the *Diagnostic and Statistical Manual-II*, although it is likely that the lore contained in the *Manual* provided the diagnostic criteria.[2] The

[2]The constantly changing criteria for schizophrenia in the various editions of the *Diagnostic and Statistical Manual* render it well-nigh impossible to aggregate the results of research studies. Blum (1978) compared diagnostic practices in 1954, 1964, and 1974 in the same hospital. About one-third of persons diagnosed as schizophrenic in 1954 would acquire a different diagnosis 20 years later. DSM-III, DSM-III-R, and other diagnosis handbooks, each with a different set of criteria, contribute to the problem.

dependent variables were assessed with great precision, sometimes to two decimal places. In contrast, the independent variable, schizophrenia/non-schizophrenia, was assessed either by the subjective and fallible judgments of clinicians, or by a vote taken in a diagnostic staff conference.

To bring our analysis up to date, we performed the same analysis on the reports published in the *Journal of Abnormal Psychology* for the ten-year period, 1979–1988. It was in this period that DSM-III and DSM-III-R came into use, and that structured interviews were refined to increase the reliability of diagnosis. Because validity is dependent upon reliable assessments, scientists expected that these systematic aids to diagnosis would facilitate the discovery of valid markers for schizophrenia. Members of the profession were optimistic that these improvements would firmly establish the ontological status of schizophrenia. Our preliminary examination of the reports shows that the experiments reported during the period 1979–1988 followed the same pattern that we had discerned in the earlier analysis. Underlying the research hypotheses is the Kraepelinian premise that schizophrenics are basically flawed organisms (Sarbin, Mancuso, and Podczerwinski, in preparation).

About 80 percent of the studies reported that schizophrenics performed poorly when compared to control subjects. Variability in performance was the rule. Although the published studies reported mean differences between groups as statistically significant, the differences were small. In those studies where it was possible to reconstruct distributions, it was immediately clear that the performances of the schizophrenic samples and the normal samples overlapped considerably. An examination of a number of such distributions points to an unmistakable conclusion: that most schizophrenics cannot be differentiated from most normals on a wide variety of experimental tasks. If one were to employ the dependent variable as a marker for schizophrenia in a new sample, the increase in diagnostic accuracy would be infinitesimal.

That so many studies showed small mean differences has been taken to mean that the schizophrenia hypothesis has earned a modicum of credibility. The degree of credibility dissolves when we consider a number of hidden variables that could account for the observed differences. A large number of reports noted that the schizophrenic subjects were on neuroleptic medication. It is approporiate to ask whether the small mean differences could be accounted for by the drugged status of the experimental subjects and the non-drugged status of the controls. Other hidden variables are socioeconomic status and education. At least since 1855, it has been noted that the diagnosis of insanity (later dementia praecox and schizophrenia) has been employed primarily as a diagnosis for poor people (Dohrenwend, 1990). Many of the experimental tasks called for cognitive skills. The mean difference in performance on such tasks could well be related to cognitive skills, a correlate of

education and socioeconomic status. Some experimenters noted the difficulty in recruiting control subjects whose educational level matched the low levels of schizophrenic samples, in many instances, about tenth grade.

Not assessed in these studies were the effects of patienthood. At the time the hospitalized patients were recruited to be subjects, they had been the objects of legal, medical, nursing, and in some cases, police procedures, not to mention mental hospital routines and their effects on personal identity. As mentioned before, only cooperative, i.e., docile, patients were recruited. It would be instructive to investigate to what degree docility influences the subjects' approach to experimental tasks.

Any of the hidden variables could account for the small mean differences observed in experimental studies. One conclusion is paramount: the 30 years of psychological research covered in our analyses has produced no marker that would establish the validity of the schizophrenia disorder. The argument could be made that psychological variables are too crude to identify the disease process. Biochemical, neurological, and anatomical studies, some would argue (e.g., Meehl, 1989), are more likely to reveal the ultimate marker for schizophrenia. However, reported findings employing somatic dependent variables follow the same pattern as for psychological studies. Variation is the rule. For example, one variable of interest for those who would locate the seat of schizophrenia in the brain is the size of the hemispheric ventricles. Several studies employed computer tomography to measure the size of the ventricles. Homogenizing the results of measurement, they found that the schizophrenic group had larger ventricles than the controls. The degree of variation, however, was such as to preclude using the ventricular size as a diagnostic instrument (Nasrallah, Jacoby, McCalley-Whitters, and Kuperman, 1982; Weinberger, Torrey, Neophytides, and Wyatt, 1979). Another set of investigators, presumably employing a more refined method for measuring the scans, reported no differences between schizophrenics and controls (Jernigan, Zatz, Moses, and Berger, 1982a, 1982b). Another hypothesis, disarray of pyramidal cells in the hippocampus, was advanced by several researchers as a potential marker for schizophrenia. Christison, Casanova, Weinberger, Rawlings, and Kleinman (1989) conducted precise measurements on brains stored in the Yakovlev collection. They found no differences in hippocampal measurements when the brains of schizophrenics were compared to the brains of controls.

It is important to note the high degree of variability in biomedical and psychological measurements. To isolate the elusive marker, investigators must discover indicators that cluster near the mean for the experimental sample and at the same time do not overlap with the control sample or with other presumed diagnostic entities. None of the studies we reviewed met this requirement.

Schizophrenia as Disease: A Social Construction

The prevailing mechanistic framework directs practitioners to perceive crazy behavior as caused ultimately by anatomical or biochemical anomalies. An alternative framework is available, one not dependent on the notion that human beings are passive objects at the mercy of biochemical forces. The starting point in this framework is the observation that candidates for the diagnosis of schizophrenia are seldom people who seek out doctors for the relief of pain or discomfort. Rather, they are persons who undergo a prediagnostic phase in which moral judgments are made on their nonconforming or perplexing actions by family members, employers, police officers, or neighbors. In the absence of reliable tests to demonstrate that the unwanted conduct is caused by anatomical or biochemical distortions, diagnosticians unwittingly join in the moral enterprise. They confirm the initial prediagnostic judgment that the deviant behavior belongs to a class of behaviors that are unwanted. After appropriate rituals, diagnosticians can confirm the moral verdict and encode it with a proper medical term, schizophrenia.

The foregoing remarks are preliminary to my argument that schizophrenia is a social construction initially put forth as a hypothesis by medical scientists and practitioners. A social construction is an organized set of beliefs that has the potential to guide action. The construction is communicated and elaborated by means of linguistic and rhetorical symbols. The categories are vicariously received, passed on from generation to generation through symbolic action. Like any construction, the schizophrenia hypothesis serves certain purposes and not others. A pivotal purpose for schizophrenia is diagnosis – professional practice requires diagnosis before treatment can be rationally prescribed. It is important to remind ourselves that any social construction can be abandoned when alternate constructions are put forth that receive symbolic and rhetorical support from scientific and political communities.

To find the origin of the schizophrenia construction, one must refer to historical sources. Because of space limitations, a full historical account is not possible. Instead I point to some pertinent observations. Ellard (1987), an Australian psychiatrist, has contributed a provocative argument under the title "Did Schizophrenia Exist Before the Eighteenth Century?" Ellard's historical analysis begins from a skeptical posture, namely, to "reflect on the question whether or not there has ever been an entity of any kind at all that stands behind the word, 'schizophrenia', and if so, what its true nature might be" (p. 306). Citing well-known authorities, Ellard points to significant changes in the description of schizophrenia over the past 50 or 60 years. He cites the common observation that contemporary clinicians seldom encounter patients who fit the prototype advanced by Kraepelin and Bleuler. If the nosological criteria for schizophrenia changed so radically in a half-century, is it not con-

ceivable that the criteria changed significantly in the half-century before Kraepelin and Bleuler? – and in the half-century before that?[3] Ellard makes clear that schizophrenia is a construction of medical scientists that is historically-bound.

As a point of departure, Ellard takes the construction and eventual abandonment of the nineteenth century diagnosis, masturbatory psychosis. Medical orthodoxy posited a psychosis characterized by restlessness, silliness, intellectual deterioration, and inappropriate affect. The entrenched belief in the association between biological activities and crazy behavior nurtured the idea of masturbatory insanity well into the twentieth century. Although at one time professionally acceptable, it was ultimately abandoned as an empty if not counterproductive hypothesis.

Employing the vaguely-defined "thought disorder" as the criterion of schizophrenia, Ellard searched the literature for evidence of cases noted by physicians and historians. His reading of case histories and medical records led to the conclusion that insanities involving "thought disorders" were identified in the eighteenth and nineteenth centuries, but such cases were exceedingly rare in the seventeenth century. It remains for future historians to identify the social, political, and professional conditions that brought about the creation of a diagnosis centered on ambiguously-defined "thought disorder."[4]

Ellard's observation about the changing criteria for schizophrenia receives strong support from a historical analysis prepared by Boyle (in press) in which she advanced a convincing explanation for the changing symptom picture.

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[3]It appears that the rate of change in the criteria for schizophrenia is accelerating. In less than a decade, two revisions of the *Diagnostic and Statistical Manual* appeared. DSM-III was published in 1980 and DSM-III-R in 1987. A new revision, DSM-IV, is in the offing. These *Manuals* are products of consensual judgments by psychiatric experts nominated by Task Forces of the American Psychiatric Association. In the 1970s, the Present State Examination (PSE) was developed in England and implemented by a computer system for making diagnoses (Wing, Cooper, and Sartorius, 1974). The criteria in the PSE were taken from Schneider (1959) who, for example, regarded certain "hallucinations" as "first rank" symptoms. The earlier editions of the American Manual had adapted Bleuler's four "A's" as criteria (anhedonia, associations, ambivalence, and autism) and looked upon "hallucinations" as accessory, not central, phenomena. More recent editions are neo-Kraepelinian – hallucinations and delusions are categorized as psychotic phenomena. The overlap between the two systems is far from perfect, each selects different candidates for what appear to be the same diagnostic categories. The two systems were compared on an outpatient psychiatric population by van den Brink, Koeter, Ormel, Dijkstra, Giel, Sloof, and Woolfarth (1989). The two systems converged on 115 of 175 patients, yielding a kappa coefficient of .32.

[4]The origins of the antecedents to the schizophrenia diagnosis occurred about the same time as the construction of the notion of the modern nuclear family (Gubrium and Holstein, 1990). The most frequent path to the mental hospital is the complaints of family members. These observations might lead a historical researcher to take a fresh look at family communications hypotheses such as those advanced by Bateson, Jackson, Haley, and Weakland (1956), Singer and Wynne (1963), and others.

Like Ellard, Boyle cites the well-documented observation that the kind of deteriorated cases described by Kraepelin and Bleuler are rarely, if ever, seen in modern times. Kraepelin recorded somatic signs and symptoms of some of his dementia praecox patients that were consistent with his gloomy prognosis of outcome: "marked peculiarities of gait. . ., excess production of saliva, and urine; dramatic weight fluctuations; tremor; cyanosis of the hands and feet; constraint of movement and the inability, in spite of effort, to complete 'willed' acts" (cited in Boyle, in press). Kraepelin also reported brain damage as revealed microscopically at post-mortem. Bleuler noted similar phenomena, for example, he claimed to be able to diagnose a schizophrenic by his or her gait.

When Kraepelin and Bleuler were establishing the diagnoses of dementia praecox and schizophrenia, they had no way of knowing that their patient populations might have included a sizable number of persons suffering from post-encephalitic parkinsonism. It was not until 1917 that the Austrian neurologist, von Economo, identified encephalitis lethargica, popularly known as sleeping sickness. The sequelae to the infection included post-encephalitic parkinsonism, signs and symptoms very much like the signs and symptoms that Kraepelin had noted for dementia praecox. A number of encephalitis epidemics had swept through Europe culminating in the epidemic of 1916–1927. Before von Economo's identification of encephalitis lethargica, persons presenting themselves to clinics and hospitals with the symptoms of post-encephalitic parkinsonism could be tagged with any number of diagnoses, including dementia praecox. Modern-day psychiatrists and neurologists do not see crazy patients who fit Kraepelin's and Bleuler's descriptions, patients who display the features of post-encephalitic parkinsonism. The change in symptom-picture over the past 50 or 75 years, then, is the result of not including encephalitic patients in the pool of patients who come to the attention of mental health professionals.

Boyle's historical account lends credibility to the thesis that post-encephalitic parkinsonism was unwittingly employed as the prototype for dementia praecox and schizophrenia. Thus the social construction of schizophrenia as a form of disease was facilitated by erroneously sorting into a single class two types of persons: undiagnosed post-encephalitic (or other organic) patients, and persons who had engaged in various kinds of unwanted conduct to solve life problems. The latter, who presented conduct only superficially similar to brain-damaged individuals, were assimilated to the former.[5]

[5]In addition to the confounding of diagnoses, Kraepelin's construction of dementia praecox was in part developed during his tenure at the University of Dorpat (now Tartu in Estonia). The nature of his contacts with non-German speaking clinic patients influenced the development of his "degeneration" theory. He made his diagnostic judgments second-hand, so to speak. An interpreter had to translate into German patients' stories which were told in a non-literary and less inflexional form of Estonian (Berrios and Hauser, 1988).

Sustaining the Social Construction of Schizophrenia as a Disease

Two features sustain the validity of any social construction: (1) its utility in solving certain societal problems, and (2) the support it receives from authoritative sources and from the forces of concurrent ideological commitments.

The Medicalization of Deviant Conduct

The social construction of schizophrenia was elaborated in the context of the asylum movement. The history of the nineteenth century asylum movement makes clear how madness was medicalized (Sarbin, 1990). In the ferment produced by rapid strides in all branches of science and technology, madness became a fit subject for scientific work. It was in the nineteenth century that medical practitioners introduced a host of new diagnoses (Rosenberg, 1989). When called upon to deal with crazy people, in the spirit of the rapidly-advancing medical science, these practitioners formulated new diagnoses, among them, dementia praecox.

The context for this new medical activity was the asylum, soon to be renamed mental hospital. The mental hospital filled a number of societal needs, the most salient of which was social control – the maintenance of order. A cursory glance at the treatments introduced over the past 150 years demonstrates clearly the operation of a mechanistic and medical ideology to solve the control problem. Locked wards and physical restraints were supplemented with treatments that were manifestly medical. Bloodletting and emetics, relics of Galenic theory, were widely practiced and ultimately abandoned. Treatments that were consistent with the developing medical theories were invented, among them, unlimited surgery to rid the patient's body of focal infections. Scull (1987) has written a Gothic horror tale of the focal infection theory and the unwarranted surgery practiced by dentists and surgeons in their efforts to control unwanted behavior. Enthusiasm for such treatments went unchecked until it became public knowledge that the high mortality rates ensuing from treatments could not justify the small number of patients whose behavior was brought under control. The more recent history of insulin, metrazol, and electric shock therapies provide additional support to the claim that social control was the object of the therapies. Frontal lobotomy as a means of behavior control was another treatment based on the entrenched belief that unwanted conduct was somehow caused by malfunctioning frontal lobes (Valenstein, 1986). Just a short time ago, biologically-oriented psychiatrists, influenced by the same ideology, employed hemodialysis in an effort to rid patients of the presumed schizotoxin.

The most recent application of this ideology is the attempt to control

behavior through the use of neuroleptic medications, formerly called "major tranquilizers." The justification for the prescription of such medications is the dopamine hypothesis, that schizophrenic behavior is the result of an excess of dopaminergic activity. Phenothiazine medications block such activity and, in some patients, there is a diminution of unacceptable activity. It has been observed that not only unacceptable behavior is reduced, but also other activities. The behavior control brought about by the medications has its price, however. Structural and histological damage to the brain is known to follow the prolonged use of phenothiazines, among them, tardive dyskinesia which occurs in a substantial proportion of patients (Breggin, 1983; Cohen, 1989; Cohen and Cohen, 1986). Contemporary clinical practice, however, accepts the notion that dopamine blockers are the medications of choice and also the corollary notion that it would be unethical to withhold such "proven" medication from schizophrenic patients. The rationale for prescribing dopamine blockers is questioned in a recent editorial in the prestigious *New England Journal of Medicine*. "Despite a number of sugestive findings . . . there is currently no proof that either a neurotoxin or an abnormality of transmission (including a dopaminergic abnormality) is a primary feature of schizophrenia" (Mesulam, 1990, p. 843).

Clearly, the schizophrenia construction has been useful to mental health practitioners. The construction has provided a justification for diagnosis. The availablity of the diagnostic term, schizophrenia, like the availability of its superordinate, mental illness, is useful as a step in the societal process of controlling persons whose conduct is unacceptable to others. With the development of the profession of medicine and especially the discipline of psychiatry, the control of patient conduct has for the most part been accomplished by means of traditional medical procedures: surgery and medication. I have identified a few of these procedures. All had a moment in the sun and were discarded when proven to be ineffective or harmful. During the period that each of the procedures was considered professionally ethical and potentially effective, however, the sequence "first diagnosis, then treatment" gave illusory support to the construction of nonconforming conduct as a disease process. In many cases, the first step in the sequence, diagnosing, was no more than a ritual exercise because of the ignorance of the effects of available treatments and their remote outcomes.

The importance given to the development of diagnostic manuals appears to be out of proportion to their utility. The obsessive preoccupation with diagnosis is illustrated in the history of the *Diagnostic and Statistical Manual*. The *Manual* is put together by psychiatric experts guided by the need for consensus. Blashfield, Sprock, and Fuller (1990) have noted that the first *Diagnostic and Statistical Manual*, published in 1952, contained 106 categories, the second, published in 1968, contained 182, the third, published in 1980,

contained 265, and the fourth, published in 1987 (DSM-III-R) contained 292. "By linear extrapolation, the DSM-IV should be expected to contain about 350 categories. . ." (p. 18). This progression raised many questions about the underlying assumptions and purposes of such diagnostic manuals.

Intrinsic and Extrinsic Support for the Disease Construction

Despite its failure when examined by empirical methods, the social construction of schizophrenia has persisted. Its persistence is a function of the support it has received. Two classes of support can be identified: support intrinsic to the biomedical model; and support extrinsic to the model in the form of social practices and unarticulated beliefs.

Biological research has served as intrinsic support for the schizophrenia construction. I need but mention the names of hypotheses that have been subjected to laboratory and clinical testing: taraxein, CPK (creatine phosphokinase), serotonin, and dopamine, among others. The composite impact of all this research activity is that an entity exists, waiting for refined methods and high technology to identify the causal morphological, neuro-transmission, or biochemical factor. As I indicated before, countless studies have not identified the disease entity. Nevertheless, the profession and the public have interpreted the sustained research activity by responsible scientists as evidence that the schizophrenia construction is a tenable one.

Guided by the mechanistic paradigm (that behavior is *caused* by antecedent physico-chemical conditions) and operating within the medical variant of that paradigm (that the causes of atypical conduct are to be found in disease entities), research scientists employed a number of broad categories as the defining criteria of schizophrenia. Such categories as cognitive slippage, anhedonia, social withdrawal, ambivalence, thought disorder, loosening of associations, delusions, inappropriate affect, and hallucinations, among others, have been employed for classifying the observed or reported conduct of persons brought to diagnosticians by concerned relatives or by forensic or social agencies. The diagnostic process involved locating the putative patient's conduct in one or more of these broad categories, and then inferring the diagnosis of schizophrenia. Thus, immediate and remote origins of the *meanings* of an individual's atypical conduct become irrelevant to the objective of the diagnosis. A scientist interested in the *person* would have little to go on from reading research reports. Readers of these reports are frustrated if they search for connections between a particular instance of unwanted conduct – the presumed basis for the diagnosis – and some dependent variable assessed after a diagnosis has been made. No causal link can be postulated to account, for example, for a schizophrenic patient's anomalous brain scan and his specific claims to having daily conversations with St. Augustine.

Typically, journal articles provide statements of statistically significant associations between such variables and *diagnoses*, not between such variables and *conduct*. Since heterogeneous acts are lumped together into homogenized diagnoses, experimental results cannot provide information that would allow inferences about the relation between the experimental variable and specific behaviors. The conventional publication style facilitates the illusory conclusion that a cause, or partial cause, of schizophrenia has been discovered. Because distributions of the dependent variable are not usually published, the reader cannot calculate the proportions of false positives and false negatives that would be generated if the dependent variable were to be used as a diagnostic instrument. Not reporting the proportion of cases contrary to the hypothesis, like the employment of diagnoses as the independent variable, facilitates the belief that some enduring property of schizophrenia has been isolated.

The traditional method of reporting scientific data helps to support the belief that some biochemical or anatomical entity corresponding to schizophrenia exists. The common practice is to report the differences between the means of the experimental subjects and the means of the controls. If the differences between the means are statistically significant, then it is assumed that the variable under consideration is related to the dichotomy: schizophrenia-nonschizophrenia. As I mentioned in connection with the analysis of 30 years of published research, the differences may be statistically significant but so small that the variable could not be employed as a diagnostic test. There is a subtle epistemological problem here: in employing group means, the experimenter homogenizes all the subjects in the experimental group and all subjects in the control group. The measurements, whether psychological, chemical, electrical, or whatever, are lumped together. They are regarded as fungible – the assessment of the cerebral ventricles of one schizophrenic is treated as if it were the same as the assessment of the ventricles of any other schizophrenic, without regard to the form, quality, and frequency of behaviors that led to the diagnosis of schizophrenia. The epistemological assumption of the significance of mean differences has proven useful in agricultural research and in insurance studies. It is hardly tenable as a basis for diagnosis.

In addition to direct biological research, the genetic transmission hypothesis has been advanced to support the construction of schizophrenia. Highly visible scientists have reported a heritability factor for schizophrenia. Wide publicity, both within the profession and outside, has been given to studies of twins and to studies of children of schizophrenics who were reared by adoptive parents (see, for example, Gottesman and Shields, 1972; Kety, Rosenthal, Wender, and Shulsinger, 1968; Kety, Rosenthal, Wender, Shulsinger, and Jacobsen, 1975). Current textbooks cite these investigations as revealed truth, but the extensive critiques of the studies are seldom noted. That the reported

studies are riddled with methodological, statistical and interpretational errors has been repeatedly demonstrated (see especially, Abrams and Taylor, 1983; Benjamin, 1976; Kringlen and Cramer, 1989; Lewontin, Kamin, and Rose, 1984; Lidz, 1990; Lidz and Blatt, 1983; Lidz, Blatt, and Cook, 1981; Marshall, 1986; Sarbin and Mancuso, 1980). The extent of these criticisms suggests that establishing the validity of "schizophrenia" should have had logical priority over the identification of its genetic features.

My aim is not to rehash the arguments pro and con of the heredity thesis, rather to show that the wide publicity given genetic studies has served as additional support to maintain the schizophrenia construction. My thesis holds for genetic research as it does for psychological and biological research: that no firm ontological basis has been established for schizophrenia. In the absence of determinate criteria, investigators direct their efforts toward discovering intergenerational similarities – not of identifiable behavior but of *diagnosis*, a far cry from the subject matter of behavior genetics in which intergenerational similarities of *behavior* are studied.[6]

In addition to intrinsic supports, it is possible to identify a number of extrinsic supports that help explain the tenacity of the schizophrenia construction. Although constructions that are congruent with the concurrent scientific paradigm may appear self-supporting, they are in great measure sustained by forces external to the scientific enterprise.

A vast bureaucratic network at federal, state, and local levels legitimizes biochemical conceptions of deviant conduct, including schizophrenia. Federal agencies that control research grants advocate studies the aim of which is the understanding and ultimately the control of "the dread disease" schizophrenia. The National Institute of Mental Health has promoted the schizophrenia construction in many ways, including the sponsoring of its own professional journal, *Schizophrenia Bulletin*, now in its sixteenth year of publication. That the government is willing to spend precious tax dollars on such an enterprise is convincing evidence to some people that "something" is there to be studied. Some local communities have taken to the airwaves and to the press to advocate the notion that schizophrenia and other mental illness are like somatic illness and can be treated with appropriate medication.

In addition to bureaucratic advocacy, in recent decades the pharmaceutical industry has been instrumental in furthering the schizophrenia doctrine. Pharmaceutical companies support countless research enterprises in which medications are clinically tested on patients, many of whom are diagnosed as schizophrenic. The psychiatric journals are to a great extent subsidized

[6]Kety, one of the leading advocates of the genetic transmission hypothesis, wrote a critique of Rosenhan's (1973) famous study, "Being Sane in Insane Places." In the critique, he composed a rhetorical sentence that lends itself to a literal interpretation: "If schizophrenia is a myth, it is a myth with a very strong genetic component" (1974, p. 961).

by pharmaceutical advertising, such advertising being directed to physicians who are legally empowered to prescribe medications.

The implicit power of bureaucracy and the commercial goals of pharmaceutical companies would be minimal if the schizophrenia messages fell on deaf ears. A readiness to believe the schizophrenia story follows from the unwitting acceptance of an ideology – a network of historically-conditioned premises.

I use the term "ideology" in the manner in which it has been employed by political scientists. "Ideology" carries the meaning that knowledge is situationally determined – the worldview and the social status of the scientist influence the content of knowledge. An examination of ideological premises illuminates how an entrenched professional organization can become so bound to a situation that its members cannot recognize facts that would dissolve its power. An ideology has a sacred quality. A challenge to a claim based on ideological premises usually invokes passionate rather than reasoned responses. Note the heated responses to the writings of Szasz, Laing, Rosenhan, and other critics of the official schizophrenia doctrine.[7]

One strand in the texture of the schizophenia ideology is the creation of the mental hospital institution. The transformation of the asylum to a mental hospital, in the context of preserving order, paved the way for regarding inmates as objects. The hospital and its medical climate were legitimated through legislative acts and judicial rulings. The courts, usually acting on the advice of physicians, granted almost unlimited power to physicians to employ their skills and their paradigms in the interest of protecting society. Because of culturally-enscripted roles for physicians and patients, once the physician made the diagnosis, the patient became a figure in an altered social narrative. The power of physicians relative to patients created a condition in which physicians could distance themselves from patients – a necessary precondition for the draconian surgical and medical treatments mentioned previously.[8]

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[7]Hays (1984), commenting on the inclusion of a heterogeneous array of behaviors in one nosological class, addresses the matter of ideological support: "Medicine is a conservative profession. What doctors know is passed on to students. In this way they honestly associate themselves with their own body of knowledge and as responsible guarantors of its truth. It is natural for such men and women to shy away from radical formulations which threaten their hard-won data-base, introduce uncertainty, and reduce the worth of what they have learned and what they have to offer. The presentation of a conceptualization which is at variance with extant schemata may be received as an affront. . ." (p. 5).

[8]It is instructive to trace the emphasis on diagnosis to its historical roots. Craik (1959) revived the historical notion that the early Greeks recognized that different outcomes were entailed if the doctors emphasized the *disease* or the *person*. The focus on diagnosing and treating the disease is associated with a school of medical practice on the island of Cnidus. A contrary view is associated with Hippocrates of Cos. The Cosan view recognized the necessity of dealing with the whole patient, the illness in relation to biography. The doctor-patient script was a collaborative one.

The legitimate power of the physician remains as an unquestioned premise in the social construction of schizophrenia. But legitimate power is only one of the characteristics that operate as silent assumptions in physicians' enacting their roles. Physicians carry Aesculapian authority, an authority that supplements legitimate power with moral and charismatic authority (Paterson, 1966; Siegler and Osmond, 1973). Physicians are assumed to have moral power in that they are dedicated to relieving pain and curing illness. They are assumed also to have the charisma that goes with the priestly role, a derivative from the time when religious figures participated in healing activities. Aesculapian authority continues to operate as a silent premise for government bodies that allocate funds in support of research the aim of which is the control of crazy people.[9]

A parallel premise is that "certain types of people are more dangerous than other types of people" (Sarbin and Mancuso, 1980). The origins of the connection between being schizophrenic and being dangerous are obscure. Several strands in the fabric of this premise can be identified, among them, the Calvinistic equation of being poor and being damned, and the attribution "dangerous classes" to the powerless poor. "Dangerous to self or other" remains as a criterion for commitment in most jurisdictions.

The overrepresentation of poor people in the class "schizophrenics" has been repeatedly documented. In addition, Pavkov, Lewis, and Lyons (1989) have shown that being black and coming to the attention of mental health professionals is predictive of a diagnosis of schizophrenia. Recently, Landrine (1989) has concluded on the basis of research evidence that the social role of poor people is a stereotype in the epistemic structure of middle-class diagnosticians. The linguistic performances and social interactions of poor people are of the same quality as the performances of men and women diagnosed as schizophrenics, particular of the "negative type" (Andreasen, 1982), those social failures who have adopted a strategy of minimal action.

With the renewed emphasis on the Kraepelinian construction, interest in studying the relations between socio-economic status (SES) and psychiatric diagnoses has declined. This decline in interest is not due to any change in the demographics. Schizophrenia is primarily a diagnosis for poor people. The advent of neo-Kraepelinian models, especially the diathesis-stress construction, turned attention to genetics research and to the study of stress. But

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The prevailing ideology in medicine, including psychiatry, is Cnidian. The doctor-patient script diminished the role of biography in therapy. In their research, psychologists have borrowed the Cnidian point of view. They begin the research enterprise with subjects who have been "diagnosed" as schizophrenic, thus embracing – sometimes unwittingly – the disease construction. Once the diagnosis is made, the life-narrative of the patient is irrelevant.

[9]In the interest of brevity, I have omitted a discussion of several other premises that undergird the social construction of schizophrenia. These are described in Sarbin and Mancuso (1980).

SES has not figured prominently in stress research. Dohrenwend (1990), a leading epidemiologist, has noted that ". . . relations between SES or social class and psychiatric disorders have provided the most challenging cues to the role of adversity in the development of psychiatric disorders. The problem remains what it has always been: how to unlock the riddle that low SES can be either a cause or a consequence of psychopathology" (p. 45). The adversity thesis might be illuminated through an examination of the observation that the outcome of "schizophrenia" varies with economic and social conditions (Warner, 1985). Landrine's research, cited above, adds to the puzzle another dimension: lower class stereotypes held by middle class diagnosticians.

The translation from the expression of atypical, unassimilable conduct (craziness) to being dangerous is facilitated by the myth of the "wild man within" (White, 1972). The myth grew out of beliefs held by Europeans during times when unknown lands were being discovered. Because the inhabitants of exotic places engaged in conduct that differed so markedly from western norms, Europeans looked upon such people as being unsocialized, wild savages. The continually shrinking world has unearthed no wild man of Africa, Asia, or of any other place, but the myth of the "wild man within" lingers as an unspoken basis for attributing dangerousness to crazy people. The myth found expression and *a fortiori* support in Lombroso's notion of "atavism" and Freud's concept of the impulse-ridden Id.[10]

Conclusion

To recapitulate: my thesis is that schizophrenia is a social construction, generated to deal with people whose conduct was not acceptable to more powerful others. During the heyday of nineteenth century science, the construction was guided by metaphors drawn from mechanistic biology. Physicians formulated their theories and practices from constructions that grew out of developing knowledge in anatomy, chemistry and physiology. The construction has an ideological cast – its proponents were blind to the possibilities that the absurdities[11] exhibited by mental hospital patients were efforts at sense-making. Instead proponents followed the tenets of mechanistic science: that social misconduct, like rashes, fevers, aches, pains, and other somatic conditions, was caused by disease processes. Reliable and sustained empirical evidence – a cardinal requirement of mechanistic science – has not been put forth to validate the schizophrenia hypothesis. Despite the absence of

[10]A pharmaceutical advertisement in one of the psychiatric journals continues the rhetorical tradition. The product, it is claimed, will *"tame the psychotic fury."*

[11]Mancuso (1989) has offered the felicitous suggestion that we employ the descriptor "absurdity" to designate disvalued conduct.

empirical support, the schizophrenia construction continues its tenacious hold on theory and practice.

My recommendation is that we banish schizophrenia to the musty historical archives where other previously-valued scientific constructions are stored, among them, phlogiston, the luminiferous ether, the geocentric view of the universe, and closer to home, monomania, neurasthenia, masturbatory insanity, lycanthropy, demon possession, and mopishness.

I emphasize that I am not recommending formulating a new descriptive term to replace schizophrenia. It is too late for that. The referents for schizophrenia are too diverse, confounded, changing, and ambiguous (Bentall, Jackson, and Pilgrim, 1988; Carpenter and Kirkpatrick, 1988). The fact that two persons (or 200) who exhibit no absurdities in common may be tagged with the same label demonstrates the emptiness of the concept.

Abandoning the schizophrenia hypothesis, however, will not solve the societal and interpersonal problems generated when persons engage in absurd, nonconforming, perplexing conduct. The first step in solving such problems calls for critical examination of the societal and political systems that support the failing biomedical paradigm. Such examination would be instrumental in replacing the mechanistic world view with a framework that would regard persons as agents trying to solve existential and identity problems.

I have already referred to the observation that the yield from the traditional approach – derived from the mechanistic metaphors – has been disappointing. This metaphysic has guided scientists and practitioners to look upon human beings as organismic objects. From this perspective, it was assumed that the behavior of organisms could be understood, predicted, and controlled through applying the root metaphor of mechanistic science – the transmittal of forces. From this belief there flowed countless hypotheses about the internal transmittal and transformation of forces. Explanations of conduct, especially deviant conduct, focused on the transmittal of forces within the brain.

The mechanistic world view is not the only metaphysical framework. An alternate framework, contextualism, leads to a totally different approach to the understanding of deviant conduct. The root metaphor of contextualism is the historical act in all its complexity. Change, novelty, variation and contingency are the categories. Unlike mechanistic constructions in which the human being is a passive object reacting to happenings within the body, the contextualist perspective directs the scientist to perceive human beings as agents, actors, performers. Within this framework, the clinician would begin his or her study by posing questions such as "what is the person trying to do or say?" "what goals is he or she trying to reach?" "what story is he or she trying to tell?" Persons are perceived as agents trying – sometimes with poorly-developed skills – to maintain their self-narratives in the face of a

complex, unpredictable and confusing world. As agents, they may choose to incorporate into their sense-making the moral valuations imposed on their conduct by parents, siblings, employers, doctors or other power figures. I see the failure of modern research on absurd conduct as following from the perception of "schizophrenics" as without agency, as suffering from happenings in the brain, rather than as agents trying to solve existential and identity problems through the construction of atypical beliefs, unusual imaginings, and bizarre speech and gestural behavior. Were we to look upon such persons as agents we would become interested in how they arrive at constructions of the world that are so different from our own (Sarbin, 1969).

One implication of adopting a contextualist framework would be a reduction in the obsessive concern with diagnosis. Each person has his or her own story, and the expressed beliefs, the atypical imaginings, the instrumental acts of withdrawing from strain-producing situations are intentional acts designed to solve identity and existential problems. The actions designed to keep one's self-narrative consistent are not invariant or machine-like outcomes of postulated disease processes. Contingencies of many kinds enter into the person's adopting a deviant role and also – I hasten to add – of rejecting such a role. Invariance is not a feature of human social life.

Reconstructing the patient's self-narrative is central to psychosocial change efforts (Sarbin, 1986). We can revive the systematic case study (Fishman, 1990) which, although time-consuming, provides patients with a context for reconstructing their self-narratives. Respectful listening to patients' stories is the first step in granting them the status of agents, goal-directed beings. At the same time that they reconstruct their life-narratives, they are given the opportunity of recounting the conduct of significant others who have collaborated in forming the self-narratives.

Understanding the interpersonal or existential themes in the stories of troubled persons is hampered when we rely on the customary vocabulary of pathology: toxins, tumors, traumata, dysfunctional traits, or defective genes. Understanding is more likely to be facilitated if we follow the lead of poets, dramatists, and biographers, and focus on the language of social relationships.

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