Schizophrenic/Paranoid Psychoses: Determining Diagnostic Divisions

by Rue L. Cromwell and William D. Plthers

Abstract

Metadiagnostic guidelines are delineated for evaluating the utility of both existing diagnostic criteria and recently proposed revisions (Magaro 1980). Among the metadiagnostic guidelines are the demonstration of differential treatment-outcome relationships for different disorders and the applicability of the characteristics of a superordinate diagnosis to its subordinate (or subtype) diagnoses.

The authors note the conceptual development of the schizophrenia construct. Attempts to verify the existence of subtypes of schizophrenia in psychoanalytic theory, demographic traits, psychological assessment, and behavioral research are reviewed. Data accumulated from these efforts provide evidence regarding the validity of the distinction between paranoid and nonparanoid schizophrenia proposed by Magaro (1980).

The authors conclude that a number of alternate relationships between paranoid and traditional (nonparanoid) schizophrenic symptoms may be hypothesized. Only the notion that paranoid and nonparanoid subtypes are representative of differences in severity of schizophrenia may be rejected. As yet, little evidence exists for choosing one of the remaining alternatives as correct.

Integrating research from diverse fields of study is advocated in the effort to refine conceptions of psychiatric disorders. In addition to continued attempts to make improvements in psychiatric diagnosis by changing the clinical symptoms used to define disorders, refinements may be accelerated by assessing the potential utility of other sources of information as the basis for psychiatric diagnosis. Performance on tasks employed in behavioral psychopathology research may represent such an example.

The purpose of this article is to examine issues relevant to determining whether patients with paranoid symptoms should be classified as schizophrenic or considered to be a distinct diagnostic entity. Among the relevant issues are the philosophy-of-science guidelines for diagnostic classification (i.e., metadiagnostics), the historical uses of the terms schizophrenia and paranoia, the data base of laboratory and other assessment procedures not traditionally used until now in determining psychiatric diagnoses, the recent efforts to integrate knowledge about psychopathology across disciplines, and the currently used diagnostic criteria of DSM-III (American Psychiatric Association 1980). From these vantage points the relationship of paranoid and traditional (nonparanoid) schizophrenic symptoms will be discussed.

Metadiagnostics

Out of the evolution of understanding of language, and in particular the more precise language crucial to science, rules have been abstracted to identify acceptable scientific constructs. As applied specifically to diagnostic constructs, the most salient of these

---

Reprint requests should be sent to Dr. R.L. Cromwell at the Department of Psychiatry, University of Rochester, 300 Crittenden Blvd., Rochester, NY 14642.
rules holds that diagnoses should be defined by (a) historical and/or (b) currently assessable observations and should predict (c) what intervention (or absence thereof) will lead to (d) what level of outcome (prognosis). The components (a) and/or (b) constitute the clarity criteria for definition. The (c–d) or (d only) relationships to (a) and/or (b) constitute the utility or validity criteria. As applied to prevention and public health interventions, the diagnostic constructs (a and/or b) should be useful in designating (e) what prevention (or absence thereof) result in (f) what levels of incidence. Thus, useful clinical diagnoses resolve themselves into a-b-c-d relationships, and the comparable public health diagnostic classifications resolve themselves into a-b-e-f relationships (Cromwell, Strauss, and Blashfield 1975).

At the heart of this article is an often overlooked principle: an adequate subject classification construct is not necessarily an adequate diagnostic construct. For example, depression, as such, is best considered a descriptive classification if further subtyping information is necessary for treatment decisions and prediction of course. Moreover, like all constructs—scientific and nonscientific, personal and culturally shared—diagnostic constructs are artificial. They are not "naturally" derived. With new knowledge of a-b-c-d relationships, they are changed. To declare a static diagnostic system a final representation of reality is not an acceptable goal.

Within this context a prominent empirical finding has been that a-c-d relationships have usually proved more useful than b-c-d relationships. In other words, history and course of a disorder have generally proved more useful for clinical decision-making than presenting symptoms at the time of diagnosis. For example, knowledge of genetic and/or past exogenous insults is generally more useful than are the symptoms produced by them. This empirical bias has even led to the distorted notion that diagnostic constructs are valid only if they pinpoint the etiology of the disturbance.

Within the context of the emphasis upon history, the diagnostic criteria for both schizophrenia and paranoia should be recognized as entailing only currently manifest symptoms, with the exception of the required 6-months' duration criterion for schizophrenia. Only time will tell whether historical and etiological features will augment the sophistication of the symptom-based clinical classifications.

Another issue often raised in clinical diagnosis is why the observations should be classified (i.e., grouped into categories) rather than described along continua. Clearly, continua provide a greater degree of psychometric precision in predicting outcome. However, in the practical world of clinical decisions continua must be adapted to enable go/no-go decisions. One must decide on one course of treatment (surgery, drugs, psychotherapy, hospital admission) or another or none at all. Consequently, the continua implied by the questions of "how much schizophrenia" and "how much paranoia," which have been valuable in research investigation, recede in importance in the practical clinical situation.

Within these metadiagnostic guidelines how should we consider the relationship between schizophrenia and paranoia? This question must be answered separately depending upon whether they are considered to be on the same level of abstraction or in a superordinate-subordinate relationship to each other. If on the same level of abstraction, one can assert their utility as separate diagnostic categories if each, as defined, leads to different outcomes under different specified conditions of treatment and no treatment. If one considers the two constructs to be superordinate-subordinate in relation to each other, then certain a-b-c-d relationships should be applicable throughout the superordinate class (schizophrenia but not nonschizophrenia) while certain other a-b-c-d relationships should be applicable only within the subordinate class (paranoid but not nonparanoid schizophrenia). If no differential treatment-outcome relationships exist whatever, then no basis can be asserted for a differentially valid diagnosis.

**Schizophrenic and Paranoid Disorders Defined**

Since complete definitions of schizophrenic and paranoid disorders are presented elsewhere in this issue and in DSM-III (American Psychiatric Association 1980), the comments here will be "editorial" and relevant to research methods rather than inclusive. With respect to paranoia (or the paranoid subclassification of schizophrenia, if you will) the major diagnostic definition in studies reported here has involved the dual criterion of (a) unequivocal admission diagnosis of "paranoid schiz-
ophrenia" with DSM-II or DSM-III, depending upon when the study was done, and (b) delusions as the predominant symptom at the time of the study. This dual approach seems to have greater predictive power than rating scales for paranoia, such as the O'Connor-Venables scale and the Psychotic Reaction Profile. Why this is true is not clear. One possibility is that delusions early in the course of this disorder are important. Another possibility is that rating scales, even the reliable ones, deal with the extent of delusional behavior outside the context of other symptoms.

Traditionally, some delusions are classified as affective rather than paranoid. Also, some paranoia (delusional disorder) is classified as nonschizophrenic (see Winokur 1978). The b-c-d basis for affective vs. schizophrenic delusions lies primarily in differential chemotherapy results. Although a-b evidence of delusional vs. schizophrenic disorder, as based upon differential family history, is offered by Kendler and Hays (1981), no strong evidence for a-b-c-d relationships is extant.

With respect to schizophrenia the authors propose that much is lost in restricting definitional criteria to a database only from the clinical interview, behavioral observation, and traditional psychological assessment. The differences in definition between DSM-II and DSM-III are probably not important, except for ruling out temporary conditions. In each case, diagnosis is based primarily on manifest symptoms. It has been proposed elsewhere that "eight decades' focus upon symptom classification is enough" (see Cromwell, in press). Further progress in understanding the biochemical, genetic, electrophysiological, socioenvironmental, and other correlates of the disorder will probably depend upon looking to narrowly and reliably defined new procedures. As emphasized often in this article, the malfunctions in information processing, measurable within the first few milliseconds or seconds following a stimulus presentation, but not detectable through traditional clinical procedures, represent major possibilities.

**Historical Perspective**

**Kraepelin and Bleuler.** From the beginning, psychiatric diagnosticians have debated whether schizophrenia is a single disorder or a group of disorders. Kraepelin (1896, as translated by Diefendorf 1923) saw dementia praecox, the forerunner of the schizophrenia concept, as being singularly characterized by early onset and subsequent deterioration. However, he distinguished the three subcategories—paranoid, hebephrenic, and catatonic. Bleuler (1911, English translation 1950) agreed with Kraepelin about the organic basis of the singular dementia praecox but also referred to the group of schizophrenias. Emphasizing fundamental symptoms characteristic of all schizophrenia, Bleuler referred to affective, associational, ambivalent, and autistic disturbance. Delusions, hallucinations, formal thought disorder, and other features were considered secondary. These two clinician-scholars set in place the idea, lasting to the present, that if paranoid delusions occurred with other symptoms of schizophrenia, the schizophrenia diagnosis would have precedence and paranoid symptoms, if pervasive, would constitute a subgroup. At the same time paranoia and paranoid psychotic states, free of deterioration, were often recognized as separate diagnoses.

**Freud.** Freud (1911, 1914) had both direct and indirect impact upon the concepts of schizophrenia and paranoia. Perhaps because of the great focus of attention upon psychoanalysis early in the century, Bleuler's outstanding treatise of 1911 was not translated into English until 1950. Schizophrenia, still referred to as an organic condition, was also characterized by the defense mechanism of withdrawal from reality within psychoanalysis (Freud 1914). Consequently, it was judged as less amenable to psychotherapy. Paranoid symptoms were interpreted to result from the mechanism of projection and were often hypothetically associated with unsuccessfully repressed homosexual urges.

In this brief historical perspective, neither Kraepelin nor Bleuler, let alone Freud, are revealed as always attuned to the metadiagnostic principles stated here. While Kraepelin's original notion of early onset/progressive deterioration represented an a-d relationship, the early focus on dementia and primary associational deficit appears to be a search for "common denominators," indeed a step toward establishing a-b-c-d relationships. To choose as most important the stable symptoms that are most clearly evident in the advanced stages of illness, however, is problematic. By analogy, the most stable and enduring symptom of a lung or retinal infection is scar tissue. This is not necessarily
the most important symptom to enter the a-b-c-d bases of diagnostic classification.

**Projective Techniques and Other Psychological Tests.** Even before doubt was cast on the reliability of psychiatric diagnosis via clinical interview in the studies by Hunt, Wittson, and Hunt (1953), a trend to use projective techniques as a supplement to the clinical interview had begun. Whether the "projectives" were seen as merely sources of support (if they agreed with the diagnosis from clinical interview) or as crucial validation criteria (which would potentially refute the interview-based diagnosis) depended upon who was "minding the turf." Rarely were the "projectives" used as a sole informational source—and well that was, since the course of the disorder (as emphasized earlier in a-c-d relationships) was not considered.

Essentially, Rorschach's test was most greatly depended upon for assessing the imminence and/or depth of psychosis. The Thematic Apperception Test and other standard picture-story devices, while often revealing conceptual disjunctions, concrete thought processes, and patterns of unfulfilled need, were more often misleading regarding the severity of pathology. The sentence completion technique was useful in reflecting conscious behavior potential more than underlying pathology. The Rorschach, on the other hand, often revealed pathology where none could yet be detected in interview and general observation.

A basic assumption by projective diagnosticians was that "deviant behavers are deviant perceivers." Inferences about imminence or presence of psychosis were drawn from poor form quality (low F+%), content containing diseased, damaged, open, and anatomically oriented versions of human and animal body forms, and the interpretation of objects as being acted upon, as opposed to maintaining body integrity and exerting volition. A major problem was that psychopaths often displayed the same "sick" pattern of response shown by schizophrenics. Paranoid features were inferred from reports of eyes or "looking behavior" of malicious or unknown source, an overinclusive disorganized response, or strange cause-effect relationships. Beck's (1954) description of the six schizophraschics represented a landmark in the use of the Rorschach for description of schizophrenia. More recently, Singer and Wynne (1963) have emphasized dimensions of communication deviance, as opposed to form and content features, in individual and family Rorschach protocols. These are not only found in schizophrenics but also in parents (adoptive and nonadoptive) and offspring of schizophrenics.

The Minnesota Multiphasic Personality Inventory represented a sophisticated attempt to separate out the schizophrenic, the paranoid, and other dimensions of illness on a profile basis. As an early major attempt toward psychometric precision it included validity and lie scales to rule out test results of subjects who consciously created invalid impressions or had responses which were otherwise uninterpretable. It used the criterion key approach, i.e., the selection of test items not from face validity of content but from how pre-identified clinical groups had responded to the items. Standard scores based upon norms from these criterion groups made the various scales numerically comparable in means and standard deviations. The shortcomings of the technique were that (1) the preexisting Kraepelinian construct system was locked into place as a given rather than allowing at the outset the emergence of potentially more useful categories, and (2) the subsequent lack of factorial validity, orthogonality, and clear a-b-c-d interrelationships limited its application to treatment decision-making and prognosis. Consequently, schizophrenics tended to score high on all scales, not just the schizophrenia scale. While a separate paranoia (Pa) scale was included, the instrument never yielded important insights about the specific interrelationships between paranoid and nonparanoid schizophrenic phenomena.

Later, the work on projective and inventory techniques focused heavily upon b-b predictor-criterion relationships. The tests were the predictors, and the clinical syndromes were usually the criteria. Test variables were seldom used independently to develop new constructs with treatment-outcome (c-d) relationships as the criteria.

**Behavioral Psychopathology.** Beginning primarily in the 1930s came the effort to apply the research techniques of experimental psychology to the understanding of psychopathology. As these investigations began, the intent was only to introduce better measurement techniques (Zubin 1948) and laboratory experimental procedures (Diefendorf and Dodge 1908;
Huston, Shakow, and Riggs (1937). These precise behavioral laboratory measurements were not viewed as crucial diagnostic validations or as a frontier into qualitatively different data about psychopathology. Orderly descriptive psychiatry and psychoanalytic interpretations were the order of the day, and the fine-grained behavioral deficit measures were explored with the hope of enhancing the description of the already recognized clinical phenomena. Shakow (1963) and his co-workers examined a wide range of dimensions in the classic Worcester studies. They found slow motor reaction time to emerge as the most "common denominator" of schizophrenia, much in contrast to the deceptive-ly similar-looking measure of motor tapping speed. Thus, reaction time became called the "North Star" of schizophrenia research (Cancro et al. 1971). These studies usually showed that the paranoid subgroup of schizophrenics performed better than other subgroups.

Out of the reaction time research came a phenomenon called "crossover" (Rodnick and Shakow 1940), later also called "redundancy deficit" (Bellissimo and Steffy 1972, 1975). This phenomenon is a reaction time slowing under consecutive trials with equal preparatory intervals greater than 5 seconds (e.g., 7 seconds). Normals, by contrast, show improvement under these conditions. This deficit occurs primarily in process schizophrenics (e.g., Bellissimo and Steffy 1972, 1975) and also in their first degree relatives (De Amicis and Cromwell 1979). Schneider (1979), Galbraith and Steffy (1979), and others have narrowed down the stimulus and response conditions under which the deficit is revealed most clearly. Process schizophrenics are primarily nonparanoid (see demographics section below); and except for that, no particular relevance in this measure for paranoid patients has been found. The same has been true of eye tracking (Holzman, Proctor, and Hughes 1973), pursuit-rotor (Rosenbaum, Mackavey, and Grisell 1957), and other motor measures.

An early and consistent finding in behavioral psychopathology studies was that schizophrenics had greater individual and group variability. The individual variability was interpreted variously as resulting from (a) motivational variability or (b) phasing in and out of contact with reality (i.e., in and out of accurate information processing). The group variability has often been interpreted as resulting from (a) different kinds of disorders all given the term schizophrenia or (b) patients differing in stage and severity of illness. Buss and Lang (1965) and Lang and Buss (1965) interpreted this variability to result from interference in thought process. Later, it was discovered that the variability could be reduced if schizophrenic patients were subdivided along dimensions of acute vs. chronic, paranoid vs. nonparanoid, and process vs. reactive (poor vs. good premorbid adjustment). Silverman (1964a) and Venables (1964) were the first to illustrate this fact in the research literature, and their impact upon future subject selection and research design was substantial. How independent these dimensions were was unclear at the time, but the paranoid dimension was clearly set forward as a major symptomatic (nonhistorical) dimension to account for group variance.

On the level of conceptual and verbal behavior the distinction between paranoid and traditional schizophrenic behavior was more evident. Payne and coworkers (Payne, Matussek, and George 1959; Payne and Caird 1967) focused upon overinclusive vs. underinclusive conceptual behavior and found a general tendency for paranoids to be more overinclusive than other groups.

Also, clinically, paranoids, having by definition delusions as their predominant symptom, have been long known to resist giving up delusions. It is therefore not surprising that behavioral psychopathologists have found in them a rigidity or resistance in shifting concepts and cognitive sets. These findings are observed independently of the existence and content of the delusions. McCormick and Broekema (1978) brought defocused visual images gradually into focus and found paranoids, in contrast to normals and non paranoids, to maintain high confidence ratings of the accuracy of their early guesses of stimulus content in spite of the limited information. The other groups increased confidence ratings as available information increased, that is, as the picture came into focus. Groups did not differ on actual accuracy. Spaulding (1978) found paranoids to be less able to shift cognitive sets on the Wisconsin Card Sorting Test when the principle required for correct solution was changed. Similarly, Asarnow and MacCrimmon (unpublished manuscript) found paranoids, who had been searching tachistoscopic arrays for a predes-
ignated numerical digit, to have more difficulty than other schizophrenics once a different digit was designated as relevant. Keefe and Magaro (1980) found that paranoids were inferior to nonparanoids when schizophrenics were compared in creativity on an alternative uses test. This evidence of conceptual rigidity is especially interesting in light of the ample earlier evidence that paranoid schizophrenics tend to equal or excel the performance of nonparanoids on tasks in which only one cognitive set is required.

From the evidence considered until now we can speculate that the paranoid schizophrenic is either (a) more competent, (b) less affected by the psychotic disorder, and/or (c) possibly qualitatively different from other patients ordinarily defined as schizophrenic. Since no distinction has been made in treatment-outcome relationship (a-d, b-d, a-c-d, b-c-d, etc.), no conclusions can yet be drawn as to whether different diagnostic categories can be asserted.

Demographics. Certain small demographic or populational movement studies have been done which have a bearing upon the schizophrenic/paranoid issue. From attempts to constitute schizophrenic subgroups according to the Silverman-Venables criteria it was discovered that poor premorbid (process) paranoids were difficult to find. Goldstein, Held, and Cromwell (1968) reported identical but separate studies from California and Tennessee which revealed that good premorbid schizophrenic patients may be either paranoid or nonparanoid but that poor premorbid tended to be nonparanoid rather than paranoid in about a 9:1 ratio. Meanwhile, Zigler (Sanes and Zigler 1971; Zigler and Levine 1973) argued that premorbid adjustment reflected the attainment of different developmental levels in the adult patients. The good premorbid or reactive schizophrenics were developmentally more advanced. Like Johanssen et al. (1963), Zigler and his colleagues found a greater frequency of process paranoids in Veterans Administration than in State hospitals. Since veterans represent a group screened premorbidly for mental competence, this broader prevalence of paranoid features could be interpreted as in support of the developmental interpretation.

Another finding by these authors, which was not predicted or sought, was of great interest. In the lower competence (State hospital) population the poor premorbid patients, as expected, tended to withdraw "away from others" in nonparanoid fashion. By contrast, the higher competence (good premorbid) patients tended to behave "against self" (suggesting depressive or schizoaffective symptoms) or "against others" (suggesting paranoid symptoms). The correlation between the latter two tendencies was negative, which suggested that the good premorbid patients followed one path or the other but not both. Thus, the interpretation: Paranoid symptoms (delusions) are by definition the manifestation of false ideas. But, to have a false idea one must first of all have an idea. Beck’s (1964, 1967) notion that affective symptoms are closely associated with cognitive concepts (of negative self-image) also implies cognitive development as a prerequisite. So, the notion that good premorbid schizophrenics, including paranoids, have more advanced cognitive competence is not a unique one.

Strauss (1973) questioned the oft-mentioned clinical observation that schizophrenics tend to lose their paranoid symptoms as they progress into the chronic phase of illness. In his demographic study he found support not for this symptom change but instead for a selective discharge of those who displayed the paranoid symptoms. Thus, the chronic patients who were left in the hospital were typically ones who had never shown paranoid symptoms.

To summarize what new light was shed on the paranoid and traditional schizophrenic constructs, a greater measure of support was indeed cast for the notion that the paranoid patient usually has a higher level of cognitive development. Also, a symptom (i.e., paranoia) is associated with a different (better) level of outcome. While the better prognosis of good premorbid patients represents an a-d relationship, the better prognosis for those with paranoid symptoms represents a b-d relationship. What is still unclear, however, is whether the paranoid symptoms, in addition to reflecting a higher competence, might also represent less severity of disorder or, indeed, a different disorder.

Severity Differences, Cognitive Style Differences, or Different Disorders?

So long as any measure (behavioral, biochemical, electro-
physiological, or other) reveals ordinal differences between groups so that normals excel paranoids who in turn excel nonparanoids (or normals excel good premorbid who in turn excel poor premorbid), then one cannot rule out the possibility that differences result only from gradations of severity. If, on the other hand, the normals fall in the middle of the ordinal ranking and different pathological groups are at opposite extremes, then the law of parsimony would rule out a single dimension of severity to account for the results. Such appeared to be the case in instances of visual size estimation, Müller-Lyer illusion, and incidental recall, as indicated by the following studies. After Harris (1957) found good and poor premorbid schizophrenics to differ in opposite directions from normal controls in size estimation, Silverman (1964b) obtained a parallel finding for paranoid and nonparanoid schizophrenics. The good premorbid and paranoid patients underestimated size, and the poor premorbid and nonparanoids overestimated size. Since these two findings suggested that the good premorbid patients were essentially paranoid and the poor premorbid patients were essentially nonparanoid, Davis, Cromwell, and Held (1967) compared all four subgroups, respectively: good premorbid paranoids, good premorbid nonparanoids, poor premorbid paranoids, and poor premorbid nonparanoids. The earlier findings were confirmed separately and independently for both paranoid status and premorbid adjustment. Kar (1967; also see Cromwell 1975) found paranoid schizophrenics to be especially susceptible to Müller-Lyer illusion effects, as compared to normals and nonparanoids. Also, in examining incidental recall of pictured objects and words with postexposure instructions, Kar found good premorbid paranoids to be superior to normals and poor premorbid nonparanoids to be inferior to normals.

Behavioral genetic studies of adopted schizophrenics have been interpreted to indicate that slow onset, poor premorbid (called chronic or borderline, depending on severity) patients have a higher-than-expected rate of biological relatives who are also schizophrenic. By contrast, the quick-onset, good premorbid (called acute) schizophrenics have no notable evidence of schizophrenia among the biological relatives (Kety et al. 1968). Instead the relatives have a higher-than-expected incidence of affective psychosis (Strömgren, quoted by Cromwell 1978; Tsuang, quoted by Cromwell 1978). This type of design has contributed importantly to understanding schizophrenia; however, the paranoid-nonparanoid division has not been systematically studied.

Through a synthesis of the work of Neisser (1967), Schneider and Shiffrin (1977), Shiffrin and Schneider (1977), and empirical studies of schizophrenic cognition, Magaro (1980) has prepared a brilliant formulation proposing that the paranoid disorder represents a distinct information processing malfunction from the traditional (nonparanoid) schizophrenic disorder. Magaro notes the similarity of Schneider and Shiffrin's distinction between automatic and controlled cognitive processes to the characteristics of preattentive and focal attentive processes delineated by Neisser. Automatic processes do not require attentional expenditure, are overlearned, and are capable of being performed in parallel without interference. Controlled processes require attention, are performed serially, and may become automatic processes if repeated frequently.

Magaro notes that automatic processes characterize perception, whereas controlled processes are related to conception. Most individuals are capable of flexibility in using these two types of cognitive operations, enabling both "bottom-up" and "top-down" processing. Although one of the processes (either automatic or controlled) may be most appropriate for performing a specific task, individuals differ in their preferred mode of processing.

The central tenet of Magaro's hypothesis maintains that traditional (nonparanoid) schizophrenics prefer automatic processes while paranoids favor controlled processes. The traditional (nonparanoid) schizophrenic follows what might best be described as a "bottom-up" processing strategy that usually fails to advance far beyond "the bottom." Magaro suggests that rudimentary perceptual processes (at the level of sensory registers) may function normally in the schizophrenic (e.g., Spaulding et al. 1980) but that a malfunction occurs in associating the percept to the appropriate concept in semantic memory. While the schizophrenic may access schematic information, this access is slower and less precise than normal since it is not the preferred processing strategy. Hence the schizophrenic often must act on
the basis of primarily perceptual information.

Paranoids employ a "top-down" processing strategy. Delusional schemata selectively bias perceptual routines toward perceiving stimuli as confirming the delusion. Perceptions are interpreted exclusively in accord with existing schemata. (Note the similarity of this interpretation to that of the conceptual rigidity studies cited earlier.) Thus, the paranoid's problem is depicted by Magaro as being conceptual rather than perceptual.

Magaro also notes the literature indicating the presence of a left hemispheric malfunction in schizophrenia (see Gruzelier and Flor-Henry 1979). On the basis of these data, Magaro hypothesizes that automatic processing routines are lateralized more to the right hemisphere. The controlled stage of processing is more strongly lateralized to the left hemisphere. The nonparanoid schizophrenic's preference for automatic processes would be manifest in right hemisphere activation, while the paranoid's controlled processes would involve left hemisphere activation.

Comparable views about lateralization by Venables (personal communication) and by members of our laboratory, while consistent with the notion that the left is the "verbal" hemisphere (in most right-handed people), reject other traditional interpretations of brain function. Instead of the left hemisphere being "dominant" and the right hemisphere "nondominant," some evidence now suggests that the right hemisphere has a major role in earlier, perhaps preattention, processing. By contrast, the left hemisphere controls more of the verbal and conceptual activity which occur later in processing and of which we have awareness. For example, an asynchrony of activation onset occurs in the hemispheres in response to stimulation. Activation of the right hemisphere precedes that of the left. Kostandov (1978) recorded bilaterally the visual evoked response (VER) of normal subjects to a unilateral display of Russian letters or meaningless figures. When directly stimulated (i.e., recording VER from the hemisphere associated with the visual field in which the stimulus was presented), the P300 component of the VER occurred significantly faster in the right hemisphere than in the left. The hemispheric onset asynchrony was not stimulus specific. Kostandov (1978) concluded:

The efficiency of interhemispheric communication under these circumstances is evidently determined by the fact that the processing of information . . . takes place more rapidly in the right hemisphere than in the left [p. 8].

One might speculate that an activity of major importance occurs in the right hemisphere before sensory input reaches the "left hemisphere stage" of awareness and verbal representation. If viewed in this manner, the right hemisphere is more strongly implicated in the etiology of schizophrenia than previously thought. Disturbances noted in the left hemisphere may actually be a secondary reflection of malfunction lateralized and originating earlier in the right hemisphere. Or, they may reflect deviant brain activity only of paranoid schizophrenics, who, according to Magaro's formulation, have a primarily conceptual disturbance.

As the paranoia and schizophrenia concepts are reassessed on the basis of the information just presented, we can go a step beyond merely concluding that the paranoid is cognitively more advanced during morbid and probably premorbid functioning. The empirical data placing normal subjects in between the paranoid and nonparanoid preclude the interpretation that the paranoid condition merely reflects a higher level of cognitive development or a milder affliction in severity of the same schizophrenic disorder. Instead, the information-processing activities of the paranoid are distinctly different. Magaro has presented an impressive theoretical formulation to explain these cognitive differences. However, the final step to conclude that separate disorders exist is yet another matter. To draw such a conclusion with functional utility requires that treatment-outcome relationships be demonstrated as different for the two groups. In other words, although the paranoid and nonparanoid display very different cognitive styles of information processing, if the same interventions (or lack thereof) lead to the same results (or if genetic outcomes across generations lead to interchangeable diagnoses of paranoid and nonparanoid schizophrenia), then separate diagnostic classifications are not differentially valid.

Pribram. With the converging data from pharmacologic, biochemical, genetic, cognitive, and clinical studies of psychopathology, an increasing demand occurs for integrative formulations which incorporate these different levels of description. Clearly, no such
integration has been done which incorporates adequately the attentional and information-processing pathology of schizophrenia. However, the authors feel compelled to provide a glimpse of where such integrative theoretical attempts might lead. Karl Pribram’s work (e.g., 1971) provides such an example. While he has not addressed specifically the problems of schizophrenia, let alone the issue of this article, he has indeed put forth heuristic propositions which lend an integration of findings in neuropsychology, neuropsychology, cognitive psychology, and traditional stimulus-response (S-R) psychology. We do not wish to be so presumptive as to explain how Pribram’s formulation should be applied to schizophrenic and paranoid psychoses, but his work nonetheless has certain implications for abnormal behavior which cannot be avoided. A few of these implications are cited here.

One principle emphasized by Pribram is that the brain can control its own input. The brain deals with events so as to move the organism continuously from a state of uncertainty toward certainty. This is done by having a repertory of schemata (hypothesized servo-mechanisms) in the brain to operate upon the input. Two major alternatives exist to resolve the input toward the desired homeostatic condition of certainty. First, the brain has long been recognized as the locus of ability to take action (e.g., to lead the organism to look more closely; to make motor and verbal responses away from, toward, or against the environment). Second, less recognized until recent years, is the fact that the brain can control input. This would happen most frequently when the input is so intense or overwhelming that the orderly processing of information would be disrupted or have a maladaptive result. Thus, the concept of redundancy of stimulus input is proposed (Pribram and Melges 1969). If information is gated (blocked out), the organism achieves a state of higher redundancy, just as if by external action it has removed itself to an environment of greater sameness both spatially and temporally. An extreme of this is what the normal individual would call stimulus deprivation. On the other hand, the organism can have an opposite state, a lesser gating than is true for the average individual. This would be described as low redundancy. The individual would be hypersensitive, vigilant rather than "tuned out." Information would be "put in" at a fast rate and a correspondingly greater demand would be placed upon the processing mechanisms to "keep up" with the rate of input. In fact, the rate of input could potentially be so fast that the individual would have trouble making appropriate changes in the schemata (servo-mechanisms, conceptual structures) as based upon the new information available.

Pribram’s and Steffy’s redundancy concepts are different. Pribram is referring to a sameness of sensory input across space and time. Steffy is referring to a sameness of length of forewarning period during the ongoing events of the reaction time task.

Experimental predictions in accord with Pribram’s redundancy formulation were made (and cited earlier) which obtained positive results. Paranoids, being low redundancy (high-rate processing) individuals, were found to underestimate stimulus size (Davis, Cromwell, and Held 1967), have greater incidental recall of unforewarned visual images (Kar 1967; see also Cromwell 1975), and to be more sensitive to Müller-Lyer illusion effects (Kar 1967; Cromwell 1975). The nonparanoid schizophrenics, having high redundancy states, showed the opposite. Normal subjects were intermediate except for illusion effects, where both they and the nonparanoids differed from the paranoids. Thus, in addition to the continuum of perceptual-conceptual preference described by Magaro, a redundancy (or rate of input) dimension is characteristic of the paranoid-nonparanoid difference. ¹

Interpretations by Pribram of neurophysiological research suggest particular brain loci for the redundancy and size estimation phenomena. A light source leads to a patterning of neural response in the geniculate body. Frontal cortex stimulation produces a magnified pattern (size overestimation). Inferior temporal cortex stimulation leads to a constricted neural pattern (size underestimation; Spinelli and Pribram 1967). Accordingly, a hypothesis may be speculated that the nonparanoid schizophrenic has overstimulation of the frontal cortex and the paranoid schizophrenic has overstimulation of the inferior temporal cortex. Both areas of ex-

¹As cited earlier, the process-reactive distinction in schizophrenia leads to the same predictions. The process is the high redundancy and the reactive is the low redundancy subgroup of schizophrenics.
perimental stimulation show evidence of being context-sensitive.

A second principle presented by Pribram concerns still another aspect of the balance between amount of informational input and the extent of brain mechanisms available to handle the input. This principle is different from the perceptual-conceptual distinction by Magaro and also to some extent the redundancy concept. The relationships among these concepts need further empirical clarification. Pribram describes a continuum of relative balance/imbalance between input and brain mechanisms available to handle it. As input increases with respect to available brain mechanisms, the individual expresses interest, then anxiety, and then panic. To go one step further in the same direction, one could suggest then also high redundancy (gating out of input) and schizophrenic disorganization. In the opposite direction, when input decreases with respect to brain resources, boredom and then sensation-seeking behavior are the proposed result. To go one step further in this direction, sociopathy might be suggested.

A corollary to the principle of balance just described concerns the notion that unresolved conflicts can be hypothesized to “tie up” part of the brain’s capacity and thus influence the balance being postulated here. Such constriction of cognitive capacity through unresolved past conflicts would be expected not only to affect pathology but also to affect the capacity for creativity.

A third major principle emphasized by Pribram is that the brain can be categorized into areas susceptible to and not susceptible to reinforcement. The areas susceptible to reinforcement are often called the pleasure or reinforcement centers. In these centers the self-stimulation effect can occur. Reinforcement and nonreinforcement areas can be distinguished not only in this manner but also in that deep electrode stimulation will maintain electroencephalographic activation in the reinforcement areas but will habituate in the nonreinforcement areas (Glickman and Feldman 1961). If one assumes that a response (behavioral or electrophysiological) is maintained only if it is reinforced, then two different systems of response modification have been illustrated. One occurs through reinforcement and one through habituation.

It has long been observed that at any given moment a vast array of intero-, proprio-, and exteroceptive stimuli are impinging upon the individual. It has also long been observed that only a very minor portion of this input in either normal or pathological individuals reaches the stage of focal attention. Thus, the overwhelmingly greater function of the brain is to gate out stimuli so that the minor portion (which we define as “relevant”) is processed appropriately. It would appear that the habituation occurring outside the reinforcement centers may help accomplish this gating or filtering function. By contrast, the minor portion of impinging stimuli is appropriately reinforced, maintained, and made available to awareness.

Schizophrenia has been looked upon as a deficiency in stimulus filtering mechanisms. Paranoids are viewed as overinclusive and nonparanoids are viewed as underinclusive in this filtering action. Thus, the relationship of these pathological characteristics to Pribram’s constructs regarding brain activity deserves more research attention. As an example, reaction time crossover (Rodnick and Shakow 1940; also called redundancy-associated deficit: Bellissimo and Steffy 1972, 1975) might be considered. As described earlier, crossover refers to a slowing of reaction time over repeated 7-second forewarning periods. It could be speculated that this slowing in schizophrenics is a result of habituation. By contrast, since the normal individual maintains an optimal reaction time or improves across isotemporal trials, Pribram’s formulation would suggest that something is getting reinforced. Thus, the process (nonparanoid) type of schizophrenic, to whom these findings apply, would appear to have the habituation mechanism overenergized and/or the reinforcement (pleasure) centers underenergized. The latter alternative would conform to descriptions of schizophrenic behavior as anhedonic (e.g., Chapman, Chapman, and Raulin 1978).

Another principle, suggested in part by Matthysse (see Cromwell 1978) but extended by Pribram’s formulation, concerns sensory feedback from bodily movements for use as temporal cues. If one assumes that behavior is temporally or sequentially organized in subgoal and goal sequences through the aid of this feedback, then a failure in this function would appear to be characteristic of hyperkinetic children. Not only are the behaviors of such children in many cases hyperactive and erratic, but the core feature of the syndrome is distractibility. Thus, it...
has more recently and appropriately been called an attention disorder (DSM-III, American Psychiatric Association 1980). Since attention is impaired, the feedback from motor activity cannot be organized into goal-directed behavior. Since methylphenidate (Ritalin) and the amphetamines improve attention and motor behavior in some of these children, it could be speculated that these compounds facilitate the utilization of motor-produced temporal cues. Thus, the behavior becomes organized and channeled into goal-directed, nondistracted activity.

At the other extreme on this continuum, the schizophrenic may be hypothesized as one who becomes disorganized because he is unable to disattend (withdraw attention) from prior temporal cues that are no longer necessary and appropriate to meet and deal with new events (Cromwell and Drokech 1968). Thus, the schizophrenic, unlike the attention-disordered child, is overdetermined by prior temporal cues. Zahn, Rosenthal, and Shakow (1963) have described such instances in which reaction time in schizophrenics was unduly influenced by the length of preparatory intervals on prior trials (i.e., the PPI effect). A hypothesis can be speculated that the exacerbation of schizophrenic symptoms via methylphenidate or the amphetamines (as in Ritalin interviews; see Davis 1978) occurs because the drug locks the schizophrenic more than ever to prior temporal (and other) cues from which he cannot disattend.

Clearly, the extrapolation of findings across neurophysiology, neuropsychology, cognitive psychology, and S-R psychology into an integrated conception of psychopathology is hazardous. One must be prepared to propose, test, discard, reformulate, and propose again. What is more hazardous and futile, however, is to stay within only one realm of scientific description, such as cognitive information processing. In so doing one cannot likely expect to reach a full understanding of paranoid and schizophrenic psychosis. Thus, the argument is proposed that formulations such as those of Magaro must eventually be integrated with those such as proposed by Pribram. Such integrative attempts should lead toward more sophisticated theories and more testable hypotheses.

**Alternative Views**

Until now only two views about paranoia and schizophrenia have been discussed: (1) paranoia is a subclassification of schizophrenia, and (2) paranoia is a separate disorder from schizophrenia. A number of alternate views, including these, may be summarized as follows.

**Paranoia is a subclass of schizophrenia.** Such a view is essentially one of "standing pat" with the traditional Kraepelinian nomenclature that has been used for many decades. A major assumption underlying this view is that the common denominators of deficit in schizophrenia, such as slow motor reaction time, are greater in importance than the differences between the paranoid and other subgroups.

A variation of this position is that a separate paranoid psychosis may be defined as being without deterioration in formal thought process. Encapsulated delusions would emerge under certain conditions in what otherwise may be a well intact individual.

**Paranoia is schizophrenia.** This position would maintain that, since delusions are more prominent only as a matter of degree, there is no need for a subgrouping at all. Schizophrenia is a homogeneous category regardless of these individual differences. By the same token there would be no need to distinguish thought-disordered and non-thought-disordered schizophrenics (e.g., Bannister 1962).

**Schizophrenia is a subclass of paranoid conditions.** This position would maintain that delusions (and thus paranoia) are prominent in a number of conditions, including schizophrenia, amphetamine psychosis, some cases of marijuana intoxication, acute brain syndrome, chronic brain syndrome, organic alcoholism syndrome, major affective disorders, and senility.

**Paranoia is not a subcategory of schizophrenia but merely reflects the level of cognitive development among those who have been afflicted by schizophrenia.** This position has been elaborated earlier in the paper.

**Paranoia designates only the level of severity in schizophrenia in that a more severe case is less capable of delusions.** This position was referred to earlier and rejected as a sole explanation on the basis of behavioral psychopathological studies.

**Paranoia and withdrawal are alternative coping styles among schizophrenic patients.** This position can be abstracted from the parallel discussions of low vs. high redundancy, overinclusive vs. underinclusive behavior, and re-
active vs. process distinctions. It is similar to the preceding alternative except for the fact that withdrawal need not be interpreted as representing a greater level of severity.

Paranoid and schizoaffective patterns represent two pathways of reactive schizophrenia, but process schizophrenia is essentially a nonparanoid disorder. This position may be abstracted from the previous section on demographics.

Paranoia and schizophrenia are separate disorders. Following Magaro’s formulation, schizophrenia may be characterized by a preference for early (preattentive), automatic, perceptual, cognitive processes. Paranoia may involve a preference for late (focal attentive), consciously controlled, conceptual, schemata-based information processes.

Neither paranoia nor schizophrenia are constructs of adequate diagnostic utility. This view would emphasize the need for a broadened data base beyond the traditional clinical procedures (i.e., beyond merely assessing what the individual says and does). Through this broadened data base of behavioral, electrophysiological, biochemical, and genetic indices—together with application of sound metadiagnostic principles—new diagnostic constructs could potentially emerge. They would not necessarily be based centrally upon the traditional clinical features, as is now the case. Even now it is recognized that such clinical features are seen clearly and reliably only late in the stage of illness. Furthermore, they have limited utility for differential treatment and prognosis (Strauss and Carpenter 1974).

### Conclusive Comment

While competence differences exist between paranoid and traditional (nonparanoid) schizophrenia, these differences cannot be considered the sole basis for the distinction. Clearly, there are also differences in styles of cognitive functioning between paranoid and nonparanoid schizophrenics. Some of these are so marked that normal subjects fall between the two pathological groups. Whether these represent different cognitive (or personality) styles which were present before the illness and/or are independent of it has not been determined. A viable alternate hypothesis is that they do indeed represent different disorders. As more information is obtained, this question will be answered on the basis of metadiagnostic criteria. Different treatment-outcome relationships must occur before separate diagnostic constructs are validated. Until then, alternate views of the relation between paranoid and traditional schizophrenic constructs are possible, and these alternate views are not necessarily mutually exclusive. It is possible that both constructs will be discarded as the applications of new procedures gain ascendancy. On the other hand, it is also possible that advances in knowledge will reaffirm the currently held relationships and that the constructs will remain essentially as they are.

### Summary

Whether patients with paranoid symptoms should be considered schizophrenic depends upon the rules we use for the formation of scientific constructs—and in particular, diagnostic constructs. A sound diagnostic construct must be reliably defined in terms of (a) antecedent and/or (b) presently assessable observations, and must be useful (valid) in predicting that (c) given treatments or, alternatively, no treatment will lead to (d) given levels of outcome (prognosis). During the history of the concepts of schizophrenia and paranoia various interpretations have been made, but none has fully met these criteria. Studies of behavioral psychopathology (and to some extent electrophysiology, biochemistry, and genetics) have provided an expanded data base by which to reach a better understanding of the relationships between paranoid and traditional (nonparanoid) schizophrenia.

A number of alternate relationships between the two constructs are possible. There is yet no basis to choose one of these alternatives over and above the others. Only the interpretation of paranoid and nonparanoid representing solely a difference in severity may be confidently rejected. Thus, without further evidence, and the sophisticated application of metadiagnostic rules to such evidence, the relationship of paranoid to traditional (nonparanoid) schizophrenic features will remain open for investigation and debate.

### Acknowledgment

Preparation of this article was partially supported by National Institute of Mental Health Grants MH-34,114 and GM-007,098.
References


Galbraith, K., and Steffy, R.A. “Intensity of Imperative Signal Influences on Redundancy Deficit and Latency in Process Schizo-
phrenics." Presented at the Annu-
Huston, P.E.; Shakow, D.; and
Riggs, L.A. Studies of motor func-
tion in schizophrenia: Reaction
time. Journal of General Psychology,

Johannsen, W.J.; Friedman, S.H.;
Leitschuh, T.H.; and Ammons,
H.A. A study of certain schizo-
phrenic dimensions and their rela-
tionship to double alternate learn-
ing. Journal of Consulting and
Clinical Psychology, 27:375–382,
1963.

al Meeting of the Canadian Psy-
chological Association, Quebec
City, 1979.

Glickman, S.E., and Feldman,
S.M. Habituation of the arousal re-
sponse to direct stimulation of the
brain stem. Electroencephalography
and Clinical Neurophysiology,

Goldstein, M.J.; Held, J.M.; and
Cromwell, R.L. Premorbid adjust-
ment and paranoid-nonparanoid
status in schizophrenia. Psycho-

Gruzelier, J., and Flor-Henry, P.,
eds. Hemisphere Asymmetries of
Function in Psychopathology.
Amsterdam: Elsevier/North-

Harris, J.G. Size estimation of pic-
tures as a function of thematic
content for schizophrenic and nor-
mal subjects. Journal of Personality,

Holzman, P.S.; Proctor, L.R.; and
Hughes, D.W. Eye-tracking pat-
terns in schizophrenia. Science,

Hunt, W.A.; Wittson, C.L.; and
Hunt, E.B. A theoretical and prac-
tical analysis of the diagnostic
process. In: Hoch, P.H., and
Zubin, J., eds. Current Problems in
Psychiatric Diagnosis. New York:
Grune & Stratton, 1953.

Kar, B.C. “Müller-Lyer Illusion in
Schizophrenics as a Function of
Field Distraction and Exposure
Time.” M.A. thesis, George
Peabody College for Teachers,

Keeffe, J.A., and Magaro, P.A.
Creativity and schizophrenia: An
equivalence of cognitive pro-
cessing. Journal of Abnormal

Kendler, K.S., and Hays, P. Para-
noid psychosis (delusional disor-
der) and schizophrenia. Archives of
General Psychiatry, 38:547–551,
1981.

Kety, S.S.; Rosenthal, D.; Wender,
P.H.; and Schulsinger, F. The
types and prevalence of mental ill-
ness in the biological and adoptive
families of adopted schizophren-
ics. In: Rosenthal, D., and Kety,
S.S., eds. The Transmission of
Schizophrenia. London: Pergamon

Kostandov, E.A. Asymmetry of
visual perception and interhemi-
spheric interaction. Human
Physiology, 4:1–12, 1978. Translated
from Fiziologiya Cheloveka, 4:3–16,
1978.

Lang, P.H., and Buss, A.H. Psy-
chological deficit in schizophrenia:
Interference and activation. Journal
of Abnormal Psychology, 70:77–106,
1965.

Magaro, P.A. Cognition in Schi-
zophrenia and Paranoia. Hillsdale,
NJ: Lawrence Erlbaum and Associ-
ates, 1980.

McCormick, D.J., and Broekema,
V.J. Cardiac rate and size estima-
tion in schizophrenic and normal
subjects. Journal of Abnormal

Neisser, U. Cognitive Psychology.
New York: Appleton-Century-
Crofts, 1967.

Payne, R., and Caird, W.K. Reac-
tion time, distractibility, and
overinclusive thinking in psychot-
ics. Journal of Abnormal Psychology,

Payne, R.; Matussek, P.; and
George, E. An experimental study
of schizophrenic thought disorder.
Journal of Mental Science,

Pribram, K.H. Languages of the
Brain: Experimental Paradoxes and
Principles in Neuropsychology.

Pribram, K.H., and Melges, F.T.
Psychophysiological basis of emo-
tion. In: Vinken, P., and Bruyn,
G.W., eds. Handbook of Clinical
Neurology. Amsterdam: North-

Rodnick, E.H., and Shakow, D.
Set in the schizophrenic as meas-
ured by a composite reaction time
index. American Journal of Psychi-
atri, 97:214–225, 1940.

Rosenbaum, G.; Mackavey, W.R.;
and Grissel, J.L. Effects of biologi-
cal and social motivation on schiz-
ophrenic reaction time. Journal of
Abnormal and Social Psychology,

Sanes, J., and Zigler, E. Premorbid
social competence in schizophre-
nia. Journal of Abnormal Psychol-

Schneider, R. "Locus of Reaction
Time Change in Schizophrenic and
Normal Subjects." Doctoral disserta-
tion, University of Calgary, 1979.

Schneider, W., and Shiffrin, R.M.
Controlled and automatic human
information processing: Detection,
search, and attention. Psychological

Shakow, D. Segmental set. Ar-
chives of General Psychiatry, 6:1–17,
1962.


The Authors

Rue L. Cromwell, Ph.D., is Professor of Psychiatry, Psychology, and Pediatrics, Department of Psychiatry, University of Rochester School of Medicine and Dentistry, Rochester, NY. William D. Pithers, Ph.D., is a postdoctoral research fellow in the Department of Psychiatry and the Division of Genetics, University of Rochester, School of Medicine and Dentistry.