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The Advantages of Studyitng Psychological Phenomen a Rather Than Psychiatric Diagnoses

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ABSTRACT: This article argues that research efforts to understand the nature of the psychological processes un-derlying such psychological phenomena as formal thought disorder delusions, and hallucinations will be more suc-cessful if the phenomena themselves are studied directly than if diagnostic categories (e.g., schizophrenia) are studied. This point is illustrated through references to the study of cognitive mechanisms underlying symptoms of schizophrenia. However, the advantages of the symptom approach are also applicable to the study of other types of psychopathology and other types of underlying mechanisms (e.g., physiological or biochemical mechanisms).

In this article I argue that research efforts to understand the nature of the psychological processes underlying such psychological phenomena as formal thought disorder, de-lusions, and hallucinations will be more successful if the phenomena themselves are studied directly than if di-agnostic categories (e.g., schizophrenia) are studied. The article describes six advantages of studying psychological phenomena rather than psychiatric diagnoses. This issue is discussed mostly within the framework of the search for cognitive mechanisms underlying symptoms of schizophrenia, particularly symptoms of thought disorder. However, the advantages of the symptom approach are also applicable to the study of other types of psychopa-thology and other types of underlying mechanisms.

Before the main arguments are presented, a brief discussion of thought disorder is required. Because of its centrality to schizophrenia and the number of studies de-voted to it, thought disorder is treated in detail. The discussion of thought disorder presented here depends on a clear distinction between two concepts of thought disorder: The overt phenomena, or symptoms of thought disorder, and the underlying processes (Rochester & Martin, 1979). The overt phenomena of thought disorder are the overt manifestations of disordered verbal behavior (e.g., loose associations, tangentiality, neologisms, blocking). They usually fall into one of two classes: a complaint by the listener that he or she has difficulty understanding the speaker or the presence of certain items (e.g., neologisms or associative responses) in the speaker's verbal produc-tions. The term underlying process of thought disorder refers to underlying psychological mechanisms, for ex-ample, yielding to normal biases (Chapman & Chapman, 1973) or excessive vulnerability to distraction (Oltmanns, 1978).

It is important to recognize that hypotheses about underlying psychological processes of thought disorder arose from observations of the overt symptoms of thought disorder. For example, Bleuler (1911/1950) noticed that some patients exhibited illogical and bizarre connections between ideas, clang associations, blocking, and other types of verbal anomalies, and he hypothesized that an underlying deficit in association processes was the psychological mechanism responsible for these phenomena. Similarly, Chapman noticed that some patients com-plained of difficulty concentrating, and he hypothesized an underlying attentional deficit (McGhie & Chapman, 1961). Chapman and Chapman (1973) proposed that the unusual use of words and the tendency to give associative responses observed in some patients was due to what they called an excessive yielding to normal biases.

Notice that the preceding discussion of the relationship between the phenomena and processes of thought disorder made no reference to the disease of schizophrenia. It was not necessary to introduce it. However, an examination of the research literature shows that the most common experimental design adopted by investigators of thought disorder is the comparison of schizophrenic and nonschizophrenic individuals on measures of the hypothesized underlying psychological process. This approach might be called the "diagnostic category" research design. Virtually all the studies reviewed in Chapman and Chapman's (1973) classic review of the literature on cognitive processes in schizophrenia used this design, and it continues to be widely used. As a result, the study of the overt manifestations of thought disorder has been largely replaced by the study of the diagnostic category of schizophrenia.

A few investigators have recently begun to study symptoms of thought disorder directly (e.g., Harvey, 1983; Manschreck, Maher, Rucklos, Vereen, & Ader, 1981; Martin & Chapman, 1982; Miller & Chapman, 1983; Oltmanns, Ohayon, & Neale, 1978; Persons & Baron, 1985) and to recommend the symptom approach to oth-ers (Bannister, 1968; Harvey & Neale, 1983; Oltmanns & Neale, 1978). However, the overwhelming majority of studies continue to rely on the diagnostic-category design. I believe that the advantages of the study of symptoms are more compelling than is generally acknowledged. There are six important advantages of the symptom ap-proach to the study of cognitive deficit in schizophrenia: (a) avoidance of the misclassification of subjects, which can occur with the diagnostic-category design; (b) the study of important phenomena that are ignored by the diagnostic-category design; (c) facilitation of theoretical development; (d) isolation of single elements of pathology for study; (e) recognition of the continuity of clinical phe-nomena with normal phenomena; and (f) improvements in diagnostic classification.

Avoidance of the Misclassification of Subjects

The strategy of studying formal thought disorder by studying patients with a diagnosis of schizophrenia is founded on two assumptions: first, that all patients diagnosed as schizophrenic have symptoms of thought disorder, and second, that nonschizophrenics do not. Of course, few-if any-investigators would actually endorse these assumptions explicitly. However, researchers endorse them implicitly when they use the diagnostic-category design. Both assumptions are incorrect, for two reasons.

First, not all schizophrenics have symptoms of thought disorder. None of the major diagnostic systems, the Diagnostic and Statistical Manual of Mental Disorders, third edition (DSM III; American Psychiatric Association, 1980), Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1977), and Feighner's criteria (Feighner et al., 1972), require the presence of symptoms of thought disorder for a diagnosis of schizophrenia. Moreover, even those schizophrenics who are thought disordered are generally only episodically so; periods of incoherence typically alternate with periods of lucidity.

Second, patients with non schizophrenic diagnoses may have overt thought disorder. Andreasen (1979b), Gurland, Fleiss, Cooper, Kendell, and Simon (1969), Taylor and Abrams (1973), and Carlson and Goodwin (1973) showed that so-called "schizophrenic symptoms" occur frequently in manic patients. Carpenter, Strauss, and Muleh (1973) reported that even first-rank Schneiderian symptoms occurred in one fourth of their sample of manic-depressive patients. Pope and Lipinski (1978) provided an extensive review of studies that showed the presence of "schizophrenic" symptoms in nonschizophrenic patients. Others have asserted that patients diagnosed as suffering from depression (Ianzito, Cadoret, & Pugh, 1974) and aphasia, particularly posterior aphasia (Benson, 1973; Gerson, Benson, & Frazier, 1977; Lecours & Vanier-Clement, 1976), may be thought disordered.

Recent conceptions of the borderline personality disorder (Kernberg, 1975) state that these patients have overt thought disorder, although no good empirical support is yet available for this proposition.

Thus, a researcher who tests a hypothesis about the nature of the processes underlying symptoms of thought disorder by comparing schizophrenic and nonschizophrenic patients may not be comparing thought-disordered subjects with non-thought-disordered subjects. From another vantage point, the result of this misclassification is that the study does not test a hypothesis about overt thought disorder; it tests a hypothesis about the diagnostic category of schizophrenia.

Even if the investigator were lucky enough to avoid a misclassification problem (i.e., all the schizophrenic subjects were thought disordered and none of the non-schizophrenics were), another problem would arise in the interpretation of the results of the study. Any difference between the experimental and control groups might be due to any of three factors: (a) schizophrenia, (b) thought disorder, or (c) the joint effects (interaction) of schizophrenia and thought disorder; that is, the study would confound the variables of schizophrenia and thought disorder and would not allow the independent effects of the two to be examined separately.

This argument does not mean to suggest that studies that classify subjects by psychiatric diagnosis are inferior to symptom-oriented studies. A study of the diagnostic category of schizophrenia that categorizes subjects by diagnostic category obviously does not involve a misclassification of subjects. However, a study of the psychological mechanisms underlying symptoms of thought disorder that compares schizophrenic subjects with nonschizophrenic subjects does misclassify subjects.

Study of Important Phenomena Ignored by the Diagnostic-Category Design

An important disadvantage of the focus on diagnostic categories is that fascinating and important psychological phenomena are ignored. The traditional design of classifying subjects by psychiatric diagnosis cannot properly study phenomena like the following disordered verbalization, given in response to the question, Why do some people believe in God?:

Well, I figure that some of them believe that they are God because they say the word God-their own voice-so that's their voice. They said the word God, so they're God. I mean, that was their tongue that said that, know what I mean? It's no one else's tongue they don't own it. (Persons, 1979).

There are at least three reasons for this. First, as shown in the previous section, this type of verbalization is not specific to any diagnostic category. Thus, classifying subjects by diagnostic category does not indicate which subjects have symptoms of thought disorder and which do not. Second, the assignment of a psychiatric diagnosis does not give any information about the types of thought-disorder symptoms (blocking, incoherence, tangentiality, neologisms, etc.) the subject may have. These phenomena are very different, and the mechanisms underlying them may also be different. The assignment of a diagnosis of schizophrenia does not acknowledge these differences. Third, symptoms are more transient than diagnostic labels. Symptoms of thought disorder may be present at one moment or in one context, but absent at a later moment or in a different context. In contrast, the DSM-III, for example, requires a six-month period of illness for a diagnosis of schizophrenia. The importance of this point is supported by the findings of Harvey (1983), who found evidence of language deficits in the thought-disordered speech fragments of thought-disordered speakers, but not in non-thought-disordered fragments of those speakers. As a result of the emphasis on diagnostic categories, the phenomenon of thought disorder (but not theories to explain it) has virtually disappeared from the literature.

The situation is little different for other psychological phenomena associated with schizophrenia. Surprisingly little experimental work has addressed the interesting and important phenomenon of delusions. Exceptions include studies by Colby (1975), Johnson, Ross, and Mastria (1977), Cashdan (1966), and Heilbrun (Heilbrun & Bronson, 1975; Heilbrun & Heilbrun, 1977). However, most of these investigations do not examine delusions, but paranoid schizophrenia. In the same way that the study of overt thought disorder has been abandoned in favor of the study of schizophrenia, the study of delusions has been passed over in favor of the study of the subtype diagnosis of paranoid schizophrenia. However, the use of the subtype diagnosis of paranoid schizophrenia to classify research subjects in a study of delusions may also lead to a misclassification of subjects. A careful examination of DSM-III criteria reveals that a patient can receive a diagnosis of paranoid schizophrenia in the absence of delusions if he or she has "hallucinations with persecutory or grandiose content" (American Psychiatric Association, 1980, p. 191).

Another symptom of schizophrenia, hallucinations, has received somewhat more attention. The literature includes investigations of hypnotic phenomena (e.g., Spanos, Jones, & Malfara, 1982), the effects of sensory deprivation, and experimental studies of hallucinating patients (e.g., Heilbrun, 1980; Heilbrun & Blum, 1984; Mintz & Alpert, 1972).

Other symptoms of schizophrenia are only now beginning to receive serious attention. Raulin (1984), in his report of the development of a scale for measuring ambivalence, stated, "Ambivalence plays a key role in a variety of theoretical formulations of schizophrenia. . . . The phenomenon of ambivalence is widely acknowledged but seldom discussed" (p. 63). Andreasen (1982a, 1982b) recently developed scales for the assessment of what she called "negative symptoms" of schizophrenia, such as poverty of speech and poverty of content of speech. These negative symptoms are the most prominent features of chronic schizophrenic patients, yet they are only now beginning to receive serious attention from researchers.

The fact that many of these symptoms are only now receiving attention does not prove that the diagnostic-category approach suppressed their study previously. In fact, Oltmanns and Neale (1978) speculated that the recent interest in symptoms is due to the resurgence of interest in diagnosis. Perhaps part of the failure to study symptoms previously stems from the fact that scales for assessing symptoms have only recently been developed (Andreasen, 1979a; Chapman et al., 1983; Chapman, Chapman, & Raulin, 1976; Chapman, Edell, & Chapman, 1980; Eckblad & Chapman, 1983). Whatever the reason for the previous failure to study symptoms, the main point is that the diagnostic-category design, for the reasons given here, does not readily accommodate the study of symptoms.

Facilitation of Theoretical Development

The focus on the diagnostic category of schizophrenia inhibits the development of adequate theories of the psychological processes underlying the symptoms of schizophrenia in several ways. First, if they ignore symptoms, investigators risk committing themselves to theories of schizophrenia that do not account for any of the overt phenomena actually observed in schizophrenic patients. As an example, studies of the memory processes of schizophrenics are common, in spite of the generally acknowledged fact that schizophrenics do not have a memory deficit per se. Even if the proposed memory deficit might be related to other phenomena that are characteristic of schizophrenic patients (a not implausible possibility), the strands of this argument are rarely provided.

Second, the symptom approach makes it easier to formulate hypotheses about underlying mechanisms. It is not difficult to propose several interesting hypotheses to account for the fact that some individuals show loosening of associations (the symptom approach). In contrast, it is very difficult to propose a cognitive mechanism that might account for the fact that some individuals show one, several, or all of the following symptoms: unusual use of words, associative responses, anhedonia, inappropriate affect, apathy, withdrawal, delusions, hallucinations, and clang associations (the diagnostic-category approach). A frequent consequence of focusing on diagnostic categories as the object of study is that the attempt at psychological explanation is quickly abandoned, for the very good reason that an attempt to postulate a psychological mechanism that might account for unusual use of words, associative responses, anhedonia, inappropriate affect, and so forth is intimidating, if not impossible.

Third, the symptom approach allows for tighter, more elaborated explanatory links between proposed underlying mechanisms and the overt phenomena that are hypothesized to be accounted for by those mechanisms. Bannister (1968), in discussing the formulation of biochemical hypotheses, made this point when he stated, "'Schizophrenia' is an abstraction . . . involving a multiplicity of behavioral criteria, and the attempt to link it directly with a specific biochemical agent leaves, between the two, an enormous gap" (p. 185).

The work of Maher and Manschreck and their colleagues (Maher, Manschreck, & Rucklos, 1980; Manschreck, Maher, & Ader, 1981; Manschreck et al., 1981; Manschreck, Maher, Rucklos, & White, 1979) illustrates this point. They demonstrated that patients with symptoms of thought disorder often show motor abnormalities as well, and they proposed the interesting hypothesis that the psychological mechanism underlying both abnormalities might be an inability to take advantage of redundancy. The links between the symptoms and the proposed underlying mechanism are clear, plausible, and testable. Oltmanns and Neale (1978) also provided an excellent example of an elaborated, explanatory theory that attempts to account for overt symptoms of thought disorder and proposes an underlying cognitive mechanism (distractibility), possible biochemical deficits, and even a genetic basis for the symptoms.

Fourth, the symptom approach also allows investigators to study interrelationships among symptoms, a strategy that facilitates psychological theorizing. For example, the observation that delusional patients are more likely than nondelusional patients to have sensory or memory deficits (Cooper, 1976) suggests the intriguing hypothesis that delusions are the result of a cognitive attempt to account for aberrant sensory experiences (Maher, 1974). Thus, a person who is hard of hearing may conclude that an attempt is being made to conceal things from him or her. A particularly attractive feature of Maher's theory is that it accounts for several prominent features of delusions. Thus, the fact that most delusions can be classified as either paranoid or grandiose is accounted for by the proposition that individuals confronted with anomalous sensory experiences will attempt to explain why they, but not others, are having those experiences. Maher stated, "We might suggest that when the patient concludes that other people are lying, he feels victimized and persecuted; whereas when he concludes that other people really do not share these experiences, he feels superior to them and grandiosity results" (p. 104).

Following the strategy of examining relationships among symptoms, one might speculate about the relationship between delusions and formal thought disorder. Perhaps reduced perceptual abilities result in both delusions and thought disorder (certainly the two are correlated clinically). Individuals who have difficulty perceiving what is said to them may give idiosyncratic or bizarre responses. Although many studies have shown that schizophrenic individuals make unusual associations in word-association tasks (Bleuler, 1911/1950; Kent & Rosanoff, 1910; Murphy, 1923; Rattenbury, Silverstein, DeWolfe, Kaufman, & Harrow, 1983; Shakow, 1980), Moon, Mefford, Weiland, Pokorny, and Falconer (1968) showed that when the experimenter ensures that subjects correctly perceive the stimulus words, schizophrenic subjects do not make more idiosyncratic or bizarre responses than normal subjects do.

Fifth, the emphasis on diagnostic categories can make it surprisingly difficult for the researcher to be clear about the hypothesis his or her study was designed to test. Two examples illustrate this difficulty. First, consider a study entitled "Performance of Acute and Chronic Schizophrenics on an Auditory Signal-Detection Task" (this is an invented title, but, I would argue, a representative one). If one asked the investigator why he or she expected acute and chronic schizophrenic individuals to differ in performance on a signal-detection task, the investigator might answer that acute schizophrenic individuals are more likely than chronic schizophrenic individuals to experience hallucinations; therefore, acute patients will have less mental capacity available for the detection of infrequent stimuli. Notice that (at least in this made-up example) a thoughtful account of the hypothesis being tested requires a reference to symptoms (hallucinations), not to the acute-chronic distinction or the length of the patient's schizophrenic illness, which is actually the basis of the acute-chronic distinction. Of course, the nature of the psychological mechanism that accounts for the length of the schizophrenic illness is an interesting and important research question in its own right. The point here is that, at least in this invented example, psychological explanation and theorizing are blocked because the length of the schizophrenic illness, the main criterion for distinguishing between the two diagnostic subgroups, is not the phenomenon the investigator is interested in studying.

What phenomenon is the investigator studying? So far, it is not clear. He or she may be studying hallucinations. Perhaps the investigator wants to test the hypothesis that hallucinations take up mental capacity, so that patients who suffer from hallucinations have less capacity remaining for other information processing. If so, there is no need to assign psychiatric diagnoses to patients or classify patients as acute or chronic types. A direct approach to testing the hallucinations hypothesis would assess patients for hallucinations and compare patients who have hallucinations with those who do not on a measure of available mental capacity, such as the signal-detection task.

Perhaps the investigator wants to study the acute-chronic distinction in schizophrenia; he or she might want to test the hypothesis that acute patients have more hallucinations than chronic patients do. In this case, the signal-detection task is superfluous.

Another example of the way in which the focus on diagnostic categories can prevent direct testing of a promising hypothesis is illustrated in the interesting study "Temporal Lobe Epilepsy and Schizophrenia: Comparison of Reaction Time Deficits" by Greiffenstein, Milberg, Lewis, and Rosenbaum (1981). This study appears to have been prompted by Greiffenstein et al.'s observation that patients with temporal lobe epilepsy (TLE) share some common symptoms with schizophrenic patients (unusual cognitive and perceptual experiences, stereotypical motor movements). Greiffenstein et al. compared TLE patients and patients with "generalized seizures" with schizophrenics and with normal subjects on a reaction-time task using regular and irregular preparatory intervals. On this task, schizophrenics routinely showed the "crossover effect" (i.e., whereas normal subjects show faster reaction-time performance when preparatory intervals are regular than when they are irregular, schizophrenic individuals show this effect only with short preparatory intervals; when preparatory intervals are long, schizophrenics show faster reaction-time performance with irregular preparatory intervals; see Steffy & Galbraith, 1974).

The rationale for examining the performance of TLE patients on reaction-time crossover appears to be that because TLE patients have some of the same symptoms that schizophrenic patients do, they may also have the same underlying cognitive deficit (reaction-time crossover). Restated slightly, the hypothesis becomes, Reaction-time crossover accounts for certain symptoms exhibited by both schizophrenic and TLE patients. When the hypothesis is restated in this way, it is clear that a direct test of the hypothesis requires a comparison of patients with the symptoms in question and those without them. However, Greiffenstein et al. (1981) did not make such a comparison. Instead, they used the traditional diagnostic-category design, and they did not assess their subjects for the presence of the symptoms of interest.

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The use of a diagnostic-category design to test a symptom hypothesis has at least two important disadvantages. The first is the very real possibility of a Type II error. That is, because probably not all schizophrenic and TLE patients had the symptoms in question and some patients in the control groups may have had the symptoms, the diagnostic-category test of the hypothesis could yield negative results even if the hypothesis were true. (Results of the Greiffenstein et al. (1981) study were positive.) The second disadvantage is that although positive results of the study suggest a link between certain symptoms and reaction-time crossover, a direct test of the hypothesis was not carried out.

Isolation of Single Elements of Pathology for Study

Studies that compare schizophrenic groups with non-schizophrenic groups compare subjects who have many, varied, and often severe symptoms with subjects who have few or no symptoms. In contrast, the study of a single symptom allows for the study of subjects who may be quite similar except for the presence of the symptom in question. This research approach is exemplified by Chapman and Chapman and their colleagues' studies of perceptual aberration, anhedonia, and magical ideation in college-student populations (Chapman et al., 1976; Eckblad & Chapman, 1983). The study of, for example, magical ideation in college students offers a number of important advantages when compared with the study of delusions in schizophrenic patients.

A significant proportion of the delusional schizophrenic patients will be too disturbed, because of the severity of the delusions themselves or other severe symptoms, to be able to participate in any psychological study. The remaining subjects who are able to participate may represent a biased sample; certainly they will not constitute a random sample of delusional subjects, or even a random sample of delusional schizophrenic patients.

Moreover, in schizophrenic patients, the phenomena of interest may be obscured by other powerful coexistent processes, including other symptoms and the effects of medication and institutionalization. It is often very difficult, for example, to determine whether a patient who is severely thought disordered is delusional or not, because his or her attempt to relate his or her delusions may be virtually incoherent. Many of these interfering coexistent processes (e.g., medications, institutionalization) are practically and ethically very difficult to control directly. When subjects are matched on some variables, they may be systematically unmatched on others (Chapman & Chapman, 1973). Statistical control of extraneous variables in a multiple regression analysis does give the investigator some information about the effects of these variables, but only when all of them can be measured accurately and independently. The difficulty of assessing delusions in a patient who is severely thought disordered or violent illustrates this problem.

The traditional diagnostic-category design, which compares schizophrenic with nonschizophrenic or normal subjects presents the difficulty that, because schizophrenics are so severely impaired in so many areas, any difference between the two groups might be due to what Chapman and Chapman (1973) called "generalized deficit." A comparison of college students with magical ideation and those without magical ideation is less vulnerable to this problem.

Of course, the objection might be raised that magical ideation in college students is not the same phenomenon as delusions in psychiatric patients and does not have the same underlying mechanism. On the other hand, perhaps it is the same phenomenon, differing only in severity, and does have the same mechanism. Of course, this is an empirical question. A test of this hypothesis can be conducted, by comparing delusional schizophrenic individuals with college students with magical ideation on measures of the mechanism of interest, without sacrificing any of the advantages of the study of single symptoms outlined here.

Recognition of the Continuity of Clinical Phenomena With Normal Phenomena

Studies of symptoms, as opposed to studies of diagnostic categories, recognize that psychological symptoms are probably best conceived of as continuous with nonpathological phenomena, a point made earlier by Strauss (1969), Strauss, Carpenter, and Bartko (1974), and Cromwell (1975), among others. Bannister (1968) recognized this point when he criticized researchers for their tendency to "erect psychologies of schizophrenics as if they were a logically distinct species" (p. 183).

The study of symptoms offers the advantage of placing the study of schizophrenia in a larger, more general theoretical framework of psychological phenomena and processes. In contrast, the diagnostic-category design implicitly assumes that the psychological processes underlying thought disorder in schizophrenic individuals are different from those underlying thought disorder in other populations. Although this assumption may turn out to be correct, it remains an empirical question.

The tendency to focus on diagnostic categories is disproportionate to their potential for leading to an understanding of other important phenomena and psychological processes that are likely to be related, in a continuous way, to psychopathological symptoms. For example, an understanding of the psychological processes underlying delusions is likely to reveal something about other related and important psychological phenomena: attitudes, prejudices, stereotypes, cults, brainwashing, and so forth. An understanding of, for example, the process-reactive dimension in schizophrenia does not. An understanding of the process-reactive dimension may reveal something about schizophrenia. The study of delusions also does this, and much more.

Improvements in Diagnostic Classification

The study of symptoms can make important contributions to the refinement of the incomplete psychiatric classification systems currently available. In medicine, diagnostic categories are typically defined in terms of an underlying physiological mechanism. For example, the diagnostic category of diabetes is defined as a disorder caused by an underlying pathological mechanism involving the production of insulin by the pancreas. Boorse (1976) and Wishner (1969) suggested that the proper definition of mental disorder is at the level of mental processes. That is, mental disorders are properly defined in terms of underlying pathological psychological processes. Thus, the process of settling on diagnostic-category boundaries, both in medicine and in psychiatry, may be viewed as a search for underlying pathological mechanisms. How can the study of symptoms contribute to the search for underlying pathological psychological mechanisms in psychiatry?

Two suggestions are offered here. First, careful attention to the details of the symptoms themselves may be surprisingly revealing of the nature of the underlying pathological mechanisms or causes. For example, Zener (1937) showed that the salivary response given by dogs in a Pavlovian conditioning paradigm differs depending on whether it is given in response to the conditioned stimulus (CS; a tone) or the unconditioned stimulus (UCS; food): the salivary response to food is greater and contains more digestive enzymes. Similarly, the key-pecking response of pigeons who have been reinforced with grain resembles the birds' behavior when eating grain, whereas the key-pecking response of pigeons who have been reinforced with water resembles their drinking behavior. An investigator who observed closely the key-pecking response of these pigeons might be able to discern that they could be divided into two groups. He or she might even perceive the differences in learning histories of the two groups.

Analogously, Chapman, Edell, and Chapman (1980) carefully assessed a large group of psychosis-prone college students, using scales of Perceptual Aberration and Physical Anhedonia. The results of the study showed that scores on the two scales were essentially uncorrelated (-.19 for men, -.09 for women). In addition, subjects with high scores on the two scales showed different patterns of related symptoms: Students with high Perceptual Aberration scores tended to report other psychotic like experiences (unusual visual experiences, auditory hallucinations, thought transmission, or unusual beliefs), whereas those with high Physical Anhedonia scores did not report more psychotic like experiences but tended to be more socially withdrawn. Based on these findings, Chapman et al. (1980) proposed that two distinct groups of psychosis-prone subjects existed. Thus, the careful observation of the details of the symptoms themselves may contribute to the refinement of the diagnostic categorization of psychotics.

Second, if researchers can learn something about the pathological mechanism underlying a symptom or a set of symptoms, they may choose to redefine a particular diagnostic category in terms of this mechanism. This approach to the discovery of the mechanisms underlying diagnostic categories may be more profitable than the attempt to discover the mechanisms underlying the psychiatric diagnostic categories as they are currently defined. Harvey and Neale (1983) concluded that the attempt to locate the central cognitive deficit in schizophrenia has failed. It is quite possible that there is no central cognitive deficit in schizophrenia, at least as schizophrenia is currently defined. Perhaps the study of mechanisms underlying symptoms will be more profitable.

The theoretical description of the learned helplessness theory of depression (Abramson, Seligman, & Teasdale, 1978) appears to take this approach to the study of depression. (Unfortunately, most studies of the learned helplessness theory in clinically depressed populations have adopted the diagnostic-category design.) Learned helplessness theory was originally proposed to account for the motivational, cognitive, and emotional deficits that occurred in laboratory dogs when they were subjected to uncontrollable shock (Seligman, 1975). Arguing that these deficits were analogous to some of the symptoms observed in clinical depression, Seligman proposed that the laboratory phenomena might serve as an animal model of depression. The links between the experimental animal model and human depression were very carefully made on the basis of similarity of symptoms; that is, learned helplessness was proposed as a theory of a group of motivational, cognitive, and emotional phenomena, not a theory of a disease category of depression. Seligman (1978) suggested that if researchers are able to learn something about the cognitive mechanisms underlying symptoms, they may choose to redefine their diagnostic categories in terms of those mechanisms: "I suggest that learned helplessness is a mode of a to-be-identified subclass of depression. This subclass may cut across the usual ways of classifying clinical depression. . . . Its central and defining feature is its causal mechanism" (p. 169).

This strategy might contribute both to the redefinition of the diagnostic category of schizophrenia and to an understanding of its underlying cognitive mechanisms. For example, an investigator might begin by testing the hypothesis that "excessive yielding to normal biases" (Chapman & Chapman, 1973) is the cognitive mechanism underlying the symptom of loosening of associations. Suppose that investigator finds that the results support the hypothesis: Yielding to normal biases is associated with loose associations. The investigator might then want to ask the question, Is yielding to normal biases the defining characteristic of a mental disease, either schizophrenia or a new category? He or she could answer this question by comparing samples of schizophrenic and nonschizophrenic individuals, in which some members of each group show loose associations and others do not on a measure of yielding to normal biases.

This hypothetical example illustrates another way in which symptom-oriented studies can shed new light on the diagnostic categories. Symptom-oriented studies that classify subjects by both diagnosis and symptoms are particularly valuable. Investigators studying thought disorder are beginning to carry out these sorts of studies. Thus, for example, Harvey (1983) showed that manic and schizophrenic patients who were thought disordered showed cohesion and reference deficits in language performance, but schizophrenic and manic patients who were not thought disordered did not. This finding suggests that cohesion and reference deficits are characteristic of patients with thought disorder, regardless of diagnosis, and rules out cohesion and reference deficits as central deficits in schizophrenia. Similarly, Andreasen (1979a) assessed both symptoms of thought disorder and diagnostic category in a large sample of psychotic subjects. Her results suggest that symptoms of positive thought disorder are characteristic of both manic and schizophrenic patients, but that negative thought disorder is characteristic only of schizophrenic patients. Thus, the study of symptoms of schizophrenia promises to contribute to researchers' understanding of both the symptoms themselves and the diagnostic category of schizophrenia.

Conclusions

Six advantages of the study of symptoms over the study of the diagnostic category of schizophrenia have been discussed. First, the use of the diagnostic-category design in a study of thought disorder results in a misclassification of subjects. Second, the symptom approach studies important phenomena that are ignored by the diagnostic-category design. Third, the study of symptoms contributes to the development of psychological theory, particularly the development of coherent, elaborate hypotheses linking clinical phenomena to underlying mechanisms. Fourth, the symptom approach permits the isolation of single elements of pathology for study. Fifth, the symptom approach recognizes the continuity of clinical phenomena and mechanisms with normal phenomena and mechanisms. Sixth, the study of symptoms contributes to the refinement of our systems of diagnostic classification.

However, the symptom approach to the study of psychopathology cannot be viewed as a panacea for clinical researchers. One criticism of the symptom approach can be illustrated with reference to Harvey's (1983) study of language deficits in thought-disordered patients. Harvey showed that thought-disordered patients showed more deficits in cohesion and reference performance than did patients who were not thought disordered. This study might be criticized on the grounds that the mechanisms proposed to account for the symptoms of thought disorder were little more than a description of the symptoms themselves. The symptom approach is probably more vulnerable to this difficulty than is the diagnostic-category approach.

Furthermore, many of the difficulties that arise in the use of the diagnostic-category strategy appear, not surprisingly, in the symptom-oriented approach as well. The problems arising from the heterogeneity of members of the diagnostic category of schizophrenia also arise from the heterogeneity, for example, of patients labeled thought disordered. Although the term thought disorder has been used here as if it referred to a single symptom, it actually refers to a large group of quite heterogeneous symptoms. Andreasen's (1979a) scale for rating thought disorder, for example, includes ratings for 18 types of thought, language, and communication disorders, ranging from poverty of speech to pressured speech (its opposite).

Similarly, the diagnostician's difficulty in deciding whether an ambiguous case is one of manic-depressive psychosis or schizophrenia is paralleled by the symptom-oriented researcher's difficulty in deciding whether the complaint "I feel bugs crawling under my skin" is a delusion or a hallucination. The diagnostician's difficulty in deciding whether a patient is schizophrenic or simply an eccentric loner is paralleled by the symptom-oriented researcher's difficulty in deciding whether a given speech sample is tangential or simply long-winded and complex. Difficulties in obtaining reliable diagnostic judgments are paralleled by difficulties in obtaining reliable judgments of symptoms (cf. Andreasen, 1979a). Strauss (1969) has provided a more extensive discussion of some of these issues.

Thus, the study of psychological symptoms does not solve all of the problems encountered by clinical researchers and may have some weaknesses of its own. However, this article has attempted to show that the advantages of the symptom approach are more compelling than is generally recognized, even when its limitations are acknowledged.

This discussion of the advantages of the study of symptoms has been made within the framework of the search for the cognitive mechanisms underlying thought disorder and other symptoms in schizophrenia. The advantages of the symptom approach, however, extend to other research endeavors as well. Nothing in the arguments limits their applicability to symptoms of schizophrenia or to cognitive mechanisms. The advantages of the symptom approach are also applicable to the study of the mechanisms underlying other diagnostic categories and other symptoms, including depression, dyslexia, obesity, and alcoholism, and to the study of physiological, biochemical, and other mechanisms underlying psychopathology.

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