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TO HONOR KRAEPELIN . . . : FROM SYMPTOMS TO PATHOLOGY IN THE DIAGNOSIS OF MENTAL ILLNESS

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> I consider hospitals only as the entrance to scientific medicine: they are the first field of observation which a physician enters; but the true sanctuary of medical science is a laboratory; only there can he see explanations of life in the normal and pathological states by means of experimental analysis.
>
> Claude Bernard (1865/1957, p. 146)

Less than two years after the publication of the 4th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV*; American Psychiatric Association, 1994), we were already gearing up for the 5th edition (e.g., Blashfield & Fuller, 1996). In trying to predict what *DSM–V* will look like, perhaps the only sure bet is that it will be longer, as the categories of mental illness proliferate beyond schizophrenia and manic-depressive illness, phobia, and obsessive–compulsive disorder to sibling relational problem (*DSM–IV* code V61.8) and nicotine-related disorder not otherwise specified (code 292.9). Those who enjoyed past controversies over the status of homosexuality, masochistic personality disorder, post-traumatic stress disorder, and periluteal phase dysphoric disorder can look forward to many similar debates in the not-too-distant future (Caplan, 1995; Kutchins & Kirk, 1997).

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However, the really interesting debate will be not whether one or another illness should be included or excluded from the nomenclature. Rather, it will be over how the nosology itself might change—that is, over the way we construe diagnosis and indeed mental illness as a whole. For psychologists, much of this more interesting debate is stimulated by the belief that the current diagnostic system is based on the medical model of psychopathology, which is viewed as a bad thing. Although it is true that the current diagnostic practice is based on the medical model, it is not necessarily true that this is a bad thing. Attitudes to the contrary are predicated on a misconception of the medical model—a misconception that is widely shared by psychologists and psychiatrists alike.

MEDICAL MODEL OF PSYCHOPATHOLOGY

The first thing that has to be understood is that, like it or not, the medical model pervades our discourse about mental illness. Consider the following:

- We speak of *symptoms* of mental illness, publicly observable manifestations of psychological abnormality; *syndromes* of mental illness, clusters of symptoms that tend to occur together; and mental *diseases*, syndromes with a known pathology.
- We speak of the *etiology*, *course*, and *prognosis* of mental disease.
- There is *diagnosis*, an activity in which a clinician assigns a classificatory label to a patient on the basis of his or her presenting symptoms.
- *Mental illness* is included in the *International Classification of Diseases* published by the World Health Organization (e.g., the 10th edition, published in 1990) of the United Nations.
- We refer to people as *mental patients* if they have mental illness, we establish *mental hospitals* to treat such illnesses (with techniques that include *therapy* and *rehabilitation*), and we teach *mental hygiene* in the hope of preventing it.

These kinds of analogies between physical and mental illness form the backbone of the medical model of psychopathology. However, there are considerable misunderstandings abroad about the nature of the medical model—including misunderstandings perpetrated by many writers of introductory textbooks in psychology. For example, the 4th edition of Gleitman's *Psychology* (1995), the book that I have used most often in teaching introductory psychology, described the medical model as follows:

> Some authors endorse the medical model, a particular version of the pathology model [which assumes that symptoms are produced by an

> underlying pathology, and that the main goal of treatment is to discover and remove this pathology], that assumes . . . that the underlying pathology is organic. Its practitioners therefore employ various forms of somatic therapy such as drugs. In addition, it takes for granted that would-be healers should be members of the medical profession. (p. 722)

Many other introductory textbooks (as well as texts in abnormal and clinical psychology) have similar passages. For the most part, these passages are intended to distinguish an ostensibly somatogenic medical model from the psychogenic models associated with cognitive and behavioral therapy, or to distinguish the profession of psychiatry, with its emphasis on drugs and other physical treatments, from clinical psychology, with its emphasis on behavioral interventions. This common association of the medical model with somatogenic theories and biological treatments reflects a deep misunderstanding, and what follows is an attempt to give an alternative perspective on this issue, based on Siegler and Osmond's (1974) sociological analysis of the medical model, *Models of Madness, Models of Medicine* (see also Shagass, 1975).[1]

According to Siegler and Osmond, the history of psychology can be traced in terms of three major models of psychopathology. The *supernatural model* prevailed before the 18th century Enlightenment. It assumes that psychology reflects the possession of the individual by demons; by implication, the proper response to psychopathology is exorcism. The *moral model*, which prevailed in the late 18th and early 19th centuries, assumes that psychopathology—or, more precisely, abnormal behavior—is deliberately adopted by the individual, much in the manner of criminal behavior; by implication, the proper response to psychopathology is confinement and other forms of punishment. The *medical model*, which began to emerge in the 19th century, assumes only that psychopathology is the product of natural causes that can be identified by the techniques of empirical science. By implication, the proper response to psychopathology is diagnosis according to a scientifically validated system and attempts at cure or rehabilitation by means of scientifically proven methods. Contrary to the popular view, the medical model does not assert that psychopathology is the product of an abnormal biological condition or that it should be treated only with drugs or surgery. Rather, the medical model is centered on particular rules regulating two primary social roles: the doctor and the patient.

The doctor (who does not have to be a physician or even hold a doctoral degree) possesses a special kind of authority called *Aesculapian* (after Aesculapius, the Greek god of medicine). Aesculapian authority is

[1]Siegler and Osmond have also promoted their perspective on the medical model in a number of journal articles on mental illness (Siegler & Osmond, 1969, 1971, 1974a; Siegler, Osmond, & Mann, 1969), alcoholism (Siegler, Osmond, & Newell, 1968), and drug addiction (Siegler & Osmond, 1968).

a combination of three other kinds of authority recognized by sociologists: *sapiential* authority, by virtue of the doctor's special knowledge and expertise; *moral* authority, by virtue of the doctor's concern for the afflicted individual; and *charismatic* authority, by virtue of the afflicted person's faith that the doctor can help. Note that doctors lack *structural* authority: They cannot enforce their prescriptions, resulting in a markedly low rate of compliance. The doctor's role is to investigate the disorder at hand, by means of procedures that might be unpleasant, intrusive, or even frightening. On the basis of this investigation the doctor makes a diagnosis, informs the afflicted person about the nature of his or her problem, absolves the patient of blame (it is critical to medical ethics that people are not blamed, and thus punished, for their illnesses), and finally creates the conditions for the afflicted person to return to health and his or her proper role in society.

The patient enacts his or her part by taking on the *sick role*: he or she must seek help from the doctor and cooperate with treatment; in return, the patient is exempt from some or all responsibilities during treatment. Note that a doctor's order has supreme authority in society—it can exempt the person from jury duty, military service, and final examinations. It has this power by virtue of our society's implicit adoption of the medical model and the sick role. However, patients cannot remain in the sick role forever; they must leave it eventually, either by recovering or dying.

A special case is when the illness is chronic and nothing more can be done to achieve a cure. Under these circumstances the role relationships change. It is the responsibility of the doctor to remove the sick role and confer the impaired role on the afflicted patient. At this point the patient must leave the hospital and active treatment. What once was an illness is transformed into a handicap, and the doctor is replaced by a rehabilitation specialist. Patients are no longer absolved from their responsibilities: They must return to some socially productive activity, do things for themselves, and cope with their handicaps as well as possible.

What has just been described is what Siegler and Osmond (1974b) referred to as the *clinical medical model*, which is one of many different versions. All versions of the medical model posit that disease is the product of natural causes and that the proper response is scientifically based treatment. However, they differ in terms of their role relationships. In the clinical medical model, the goal is to cure disease in an individual, and the role relationships are doctor and patient. In the public health medical model, the goal is to control illnesses that cannot be cured on an individual basis. Its focus is on prevention of disease in a population, rather than an individual, and in fact its prescriptions for public health may damage some individuals; moreover, the public health official may decide to permit some diseases to occur, perhaps for economic reasons. Note that the role relationships differ in the public health medical model. The doctor is replaced by the public health official, who has structural as well as sapiential au-

thority—he or she has the power of the law and the courts to enforce "doctor's orders" and to force us to fluoridate our water or be immunized against smallpox and polio. The patient is replaced by the citizen, who by his or her vote can place limits on the public health official's authority to act.

In the *scientific medical model*, there is no direct interest in intervention (prevention or cure), but there is interest in the acquisition of scientific knowledge about the nature of disease. Again, the role relationships change. The doctor is replaced by the investigator who has only sapiential authority. The investigator has no obligation to cure and prevent disease and in certain circumstances may even inflict disease (or allow it to occur) as part of a controlled experiment. The patient is replaced by the research participant who volunteers his or her services. These individuals are under no obligation to participate in research, and usually they do so only when they are compensated in some way for their services. They have rights that patients and citizens do not have: They must be protected from harm, and they must be assured that the procedures to which they are subjected are worthwhile; their only responsibility is to honor their commitment to the study.[2]

I explore the medical model in detail because it has been subject to so much misunderstanding—and also because it gives me the opportunity to unite two social sciences, psychology and sociology, at least for a moment. However, the interested reader should reflect on the implications of the medical model or models for understanding the nature, causes, treatment, and prevention of psychopathology and on the proposition that many of the abuses frequently attributed to mental health professionals—such as the confinement of mental patients in the back wards of mental hospitals, without any active treatment—actually represent violations, not expressions, of the medical model.

THE KRAEPELINIAN LEGACY

Diagnosis lies at the heart of the medical model of psychopathology: The doctor's first task is to decide whether the person has a disease and what that disease is. Everything else flows from that. A diagnostic system is, first and foremost, a classification of disease—a description of the kinds

[2]This is a good point to register my objection to the American Psychological Association's decision to substitute the term *participants* for the traditional *subjects*. In fact, there are several different participants in the social interaction known as the psychological experiment (Orne, 1962, 1973), including (but not limited to) the experimenter, the subject, and any confederates of the experimenter. The distinction is one of role, not of power: The experimenter conducts the experiment, whereas the subject provides the data (subjects might also be observers, respondents, or even informants). For more on the experimenter–subject relationship, see Danziger (1990) and Bayer and Shotter (1997).

of illnesses one is likely to find in a particular domain. However, advanced diagnostic systems go beyond description. They carry implications for underlying pathology, etiology, course, and prognosis; they tell us how likely a disease is to be cured and which cures are most likely to work; failing a cure, they tell us how successful rehabilitation is likely to be; and they tell us how we might go about preventing the disease in the first place. Thus, diagnostic systems are not only descriptive; they are also predictive and prescriptive. Diagnosis is also critical for scientific research on psychopathology—as Cattell (1940) put it, nosology precedes etiology. Uncovering the psychological deficits associated with schizophrenia requires that we be able to identify people who have the illness in the first place.

Diagnosis Before Kraepelin

Before Kraepelin, the nosology of mental illness was a mess. Isaac Ray (1838/1962) followed Esquirol and Pinel in distinguishing between insanity (including mania and dementia) and mental deficiency (including idiocy and imbecility), but otherwise denied the validity of any more specific groupings (Grob, 1991; Kendell, 1990; Shorter, 1997). It fell to Kraepelin to systematically apply the medical model to the diagnosis of psychopathology, attempting a classification of mental illnesses that went beyond presenting symptoms (Havens, 1965; Shorter, 1997). In this respect, however, Kraepelin's program largely failed. Beginning in the 5th edition (1896) of his *Textbook*, and culminating in the 7th and penultimate edition (the second edition to be translated into English), Kraepelin acknowledged that classification in terms of pathological anatomy was impossible, given the present state of medical knowledge. His second choice, classification by etiology, also was unsuccessful: Kraepelin freely admitted that most of the etiologies given in his text were speculative and tentative. In an attempt to avoid classification by symptoms, Kraepelin fell back on classification by course and prognosis: What made the manic-depressive psychoses similar, and different from the dementias, was not so much the difference between affective and cognitive symptoms, but rather that manic-depressive patients tended to improve whereas demented patients tended to deteriorate.

By focusing on the course of illness, in the absence of definitive knowledge of pathology or etiology, Kraepelin hoped to put the psychiatric nosology on a firmer scientific basis. In the final analysis, however, information about course is not particularly useful in diagnosing a patient who is in the acute stage of mental illness. Put bluntly, it is not much help to be able to say, after the disease has run its course, "Oh, so *that's* what he had!". Kraepelin appears to have anticipated this objection when he noted that

> there is a fair assumption that similar disease processes will produce identical symptom pictures, identical pathological anatomy, and an identical etiology. If, therefore, we possessed a comprehensive knowledge of any one of these three fields,—pathological anatomy, symptomatology, or etiology,—we would at once have a uniform and standard classification of mental diseases. A similar comprehensive knowledge of either of the other two fields would give not only just as uniform and standard classifications, but all of these classifications would exactly coincide. Cases of mental disease originating in the same causes must also present the same symptoms, and the same pathological findings. (Kraepelin & Diefendorf, 1904/1907, p. 117)

Accordingly, Kraepelin and Diefendorf (1904/1907) divided the mental illnesses into 15 categories, most of which remain familiar today, including dementia praecox (renamed schizophrenia), manic–depressive insanity (bipolar and unipolar affective disorder), paranoia, psychogenic neuroses, psychopathic personality, and syndromes of defective mental development (mental retardation). What Kraepelin did for the psychoses, Pierre Janet later did for the neuroses (Havens, 1966), distinguishing between hysteria (dissociative and conversion disorders) and psychasthenia (anxiety disorder, obsessive–compulsive disorder, and hypochondriasis).

The Evolution of *DSM*

Paradoxically, Kraepelin's assertion effectively justified diagnosis on the basis of symptoms—exactly the practice that he was trying to avoid. For more than a century now, that is just what the mental health professions have continued to do. True, the predecessors of the *DSM*, such as the *Statistical Manual for the Use of Institutions for the Insane* (see Grob, 1991) or the War Department Technical Bulletin, Medical 203 (see Houts, 2000) spent a great deal of time listing mental disorders with presumed or demonstrated biological foundations. For the most part, however, actual diagnoses were made on the basis of symptoms, not on the basis of pathological anatomy—not least because, as Kraepelin himself had understood, evidence about organic pathology was usually impossible to obtain, and evidence about etiology was usually hard to obtain. In distinguishing between psychosis and neurosis, between schizophrenia and manic–depressive disorder, or between phobia and obsessive–compulsive disorder, the clinician based the diagnosis exclusively on symptoms.

Similarly, while the 1st edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–I*; American Psychological Association, 1952) may have been grounded in psychoanalytic and psychosocial concepts, diagnosis was still based on lists of symptoms and signs. So too, for the 2nd edition (*DSM–II*; American Psychiatric Association, 1968). For example, the classical distinctions among simple, hebephrenic, catatonic (excited or

withdrawn), and paranoid schizophrenia were based on presenting symptoms, not on the basis of pathological anatomy; they were "functional," of unknown etiology or even course (all chronic and deteriorating).

In point of fact, the first two editions of the *DSM* gave mental health professionals precious little guidance about how diagnoses were actually to be made—which is one reason why diagnoses proved to be so unreliable (e.g., Spitzer & Fleiss, 1974; Zubin, 1967). Correcting this omission was one of the genuine contributions of what has come to be known as the *neo-Kraepelinian movement* in psychiatric diagnosis (Blashfield, 1985; Klerman, 1977), as exemplified by the work of the "St. Louis Group" centered at Washington University School of Medicine (Feighner et al., 1972; Woodruff, Goodwin, & Guze, 1974), and the Research Diagnostic Criteria (RDC) promoted by a group at the New York State Psychiatric Institute (Spitzer, Endicott, & Robins, 1975). The 3rd, 3rd revised, and 4th editions of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–III*, *DSM–III–R*, and *DSM–IV*; American Psychiatric Association, 1980, 1987, 1994, respectively) were largely the product of these groups' efforts.

Diagnosis by symptoms was codified in the Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978), geared to the RDC, and in analogous instruments geared to the *DSM*: the Structured Clinical Interview for *DSM–III–R* (SCID; Spitzer, Williams, Gibbon, & First, 1990) and Structured Clinical Interview for *DSM–IV* Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1997). The neo-Kraepelinian approach exemplified by *DSM–IV* and SCID-I has arguably made diagnosis more reliable, if not more valid. For example, clinicians can show a high rate of agreement in diagnosing multiple personality disorder (in *DSM–III*; in *DSM–IV*, renamed dissociative identity disorder), but it is difficult to believe that the "epidemic" of this diagnosis observed in the 1980s and 1990s represented a genuine increase in properly classified cases (Kihlstrom, 2001).

A more important feature of *DSM–III* and *DSM–IV*, one not often remarked on (for an exception, see Blashfield & Flanagan, 1999), was a shift in the structure of the psychiatric nosology. Before *DSM–III*, psychiatric diagnoses were, at least tacitly, construed as proper sets: summaries of instances (syndromes) that shared a set of defining features (symptoms), which in turn were singly necessary and jointly sufficient to identify an entity as an example of the category. Thus, in *DSM-II*, all the psychoses were characterized by disorders of reality testing, whereas all the neuroses were characterized by anxiety. In Bleuler's classic work (1911/1950), the "group of schizophrenias" was united by the presence of the "four As" (associative disturbance, affective disturbance, ambivalence, and autism). Similarly, Janet (1907) discussed the defining "stigmata" of hysteria. The construal of the diagnostic categories as proper sets, to the extent that anyone thought about it at all, almost certainly reflected the classical view

of categories handed down from the time of Aristotle. Indeed, much of the dissatisfaction with psychiatric diagnosis, at least among those inclined toward diagnosis in the first place, stemmed from the problems of partial and combined expression (e.g., Eysenck, 1961). Many patients did not fit into the traditional diagnostic categories, either because they did not display all the defining features of a particular syndrome, or because they displayed features characteristic of two or more contrasting syndromes. It was to handle cases such as these that such labels as *schizotypal personality disorder* and *schizoaffective disorder* were proposed.

In the 1970s, however, psychologists and other cognitive scientists began to discuss problems with the classical view of categories as proper sets and to propose other models, including the *probabilistic* or *prototype* model (for a review of these problems, and an explication of the prototype model, see Smith & Medin, 1981). According to the prototype view, categories are *fuzzy sets*, lacking sharp boundaries between them. The members of categories are united by *family resemblance* rather than a package of defining features. Just as a child may have her mother's nose and her father's eyes, so the instances of a category share a set of *characteristic features* that are only probabilistically associated with category membership. No feature is singly necessary, and no set of features is jointly sufficient, to define the category. Categories are represented by prototypes, which possess many features characteristic of the target category and few features characteristic of contrasting categories.

The prototype view solves the problems of partial and combined expression, and in fact a seminal series of studies by Cantor and her colleagues showed that mental health professionals tended to follow it, rather than the classical view, when actually assigning diagnostic labels (Cantor & Genero, 1986; Cantor, Smith, French, & Mezzich, 1980; Genero & Cantor, 1987). In a striking instance of art imitating life, *DSM–III* tacitly adopted the prototype view in proposing rules for psychiatric diagnosis. For example, *DSM–III* permits the diagnosis of schizophrenia if the patient presents any one of six symptoms during the acute phase of the illness and any two of eight symptoms during the chronic phase. Thus, to simplify somewhat (but only somewhat), two patients—one with bizarre delusions, social isolation, and markedly peculiar behavior, and the other with auditory hallucinations, marked impairment in role functioning, and blunted, flat, *or* (emphasis added) inappropriate affect—could both be diagnosed with schizophrenia. No symptom is singly necessary, and no package of symptoms is jointly sufficient, for a diagnosis of schizophrenia as opposed to something else. Although the packaging of symptoms changed somewhat, *DSM–IV* followed suit.

Other views of categorization have emerged since the prototype view, including an exemplar view and a theory-based view. Moreover, there are several versions of the prototype view, including one based on discrete

features and another based on continuous dimensions (Medin, 1989; Medin, Goldstone, & Gentner, 1993; Smith & Medin, 1981). Space does not permit elaboration of these alternatives here; it is enough to say that, except for work by Cantor and Genero on expertise in diagnosis, these models have not been applied to psychiatric diagnosis. I think we can safely predict that *DSM–V* will also be organized along probabilistic, prototypical lines—suggesting that the diagnostic categories themselves, and not just the categorization process, are organized as fuzzy sets. However, this is not enough for many psychologists, who (as exemplified by many contributors to this volume) seek to embrace another basis for diagnosis entirely.

Critiques of Categorization

This is more than a debate over whether one diagnosis or another should be included in the new nomenclature. Some colleagues, heirs of the psychodynamically and psychosocially oriented clinicians who dominated American psychiatry before the neo-Kraepelinian revolution, wish to abandon diagnosis entirely. So do contemporary anti-psychiatrists, although for quite different reasons. Classical behavior therapists also abjure diagnosis, seeking to modify individual symptoms without paying much attention to syndromes and diseases. For these groups, the best *DSM* is no *DSM* at all. Beyond these essentially ideological critiques, there appear to be essentially two (not unrelated) points of view: one that seeks only to put diagnosis on a firmer empirical basis, and another that seeks to substitute a dimensional for a categorical structure for the diagnostic nosology. Both seek to abandon the medical model of psychopathology represented by the neo-Kraepelinians who formulated *DSM–III* and *DSM–IV*.

The empirical critique is exemplified by Blashfield (1985), who has been critical of the "intuitive" (p. 116) way in which the neo-Kraepelinians did their work and who wants the diagnostic system to be placed on firmer empirical grounds. For Blashfield and others like him, a more valid set of diagnostic categories will be produced by the application of multivariate techniques, such as factor analysis and cluster analysis, which will really "carve nature at its joints," showing what really goes with what. The result may very well be a nosology organized along fuzzy-set lines, as *DSM–III* was and *DSM–IV* is. However, at least diagnosis will not depend on the intuitions of a group of professionals imbued with the traditional nomenclature. If schizophrenia or some other traditional syndrome fails to appear in one of the factors or clusters, that's the way the cookie crumbles: Schizophrenia will have to be dropped from the nomenclature. Less radically, the analysis may yield a syndrome resembling schizophrenia in important respects, but the empirically observed pattern of correlations or co-occurrences may require revision in specific diagnostic criteria.

Whereas Blashfield (1985) appears to be agnostic about whether a

new diagnostic system should be categorical or dimensional in nature, so long as it is adequately grounded in empirical data, other psychologists, viewing diagnosis from the standpoint of personality assessment, want to opt for a dimensional alternative. Exemplifying this perspective are Clark, Watson, and their colleagues (Clark, Watson, & Reynolds, 1995; Watson, Clark, & Harkness, 1994).[3] They have argued that categorical models of psychopathology are challenged by such problems as comorbidity (e.g., the possibility that a single person might satisfy criteria for both schizophrenia and affective disorder) and heterogeneity (e.g., the fact that the present system allows two people with the same diagnosis to present entirely different patterns of symptoms). Clark et al. are also bothered by the frequent provision in *DSM–IV* of a subcategory of "not otherwise specified," which really does seem to be a mechanism for assigning diagnoses that do not really fit, and by a forced separation between some Axis I diagnoses (e.g., schizophrenia) and their cognate personality disorders on Axis II (e.g., schizotypal personality disorder).

Clark and Watson's points (some of which are essentially reformulations of the problems of partial and combined expression) are well taken, and it is clear—and has been clear at least since the time of Eysenck (1961)—that a shift to a dimensional structure would go a long way toward addressing them. At the same time, such a shift is not the only possible fix. After all, heterogeneity is precisely the problem which probabilistic models of categorization are designed to address (the exemplar and theory-based models also address them), although it seems possible that such categories as schizophrenia, as defined in *DSM–III* and *DSM–IV*, may be a little too heterogeneous. Comorbidity is a problem only if diagnoses label people rather than diseases.[4] After all, dual diagnosis has been a fixture in work on alcohol and drug abuse, mental retardation, and other disorders at least since the 1980s (e.g., Penick, Nickel, Cantrell, & Powell, 1990; Woody, McClellan, & Bedrick, 1995; Zimberg, 1993). There is no a priori reason why a person cannot suffer from both schizophrenia and affective disorder, just as a person can suffer from both cancer and heart disease.[5]

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[3]Watson and Clark edited a collection of articles exploring the connection between diagnostic categories and personality dimensions, published as a special issue of the *Journal of Abnormal Psychology* (1994, Vol. 101, No. 1).

[4]Perhaps not even then. Although it may be true that adjectival nouns such as *schizophrenic* encourage stereotyping and prejudice, they are awfully convenient for linguistic expression, and I for one am sorry to see them eliminated by the forces of political correctness. There is no reason why people cannot be referred to as *schizophrenics* when discussing their schizophrenia (assuming that this is a valid disease entity), just as they might be referred to as *Irish* when discussing their ethnicity, or *males* when discussing their gender. (Is "Irish male" a case of comorbidity?)

[5]Time for a personal anecdote: When I was a clinical psychology intern in 1974, my first patient was an adolescent who was both suicidally depressed and mentally retarded (during the intake interview, he fished two bullets out of his pocket and began clicking the tip of one against the firing pin of the other). This combination of conditions created problems for disposition, because local inpatient services for mentally ill children did not want to take

There is no doubt that the diagnostic nosology should be put on a firmer empirical basis, and it may well be that a shift from a categorical to a dimensional structure will improve the reliability and validity of the enterprise. It should be noted, however, that both proposals essentially represent alternative ways of handling information about *symptoms*—subjectively experienced or publicly observable manifestations of underlying disease processes. So long as they remain focused on symptoms, proposals for revision of the psychiatric nomenclature, nosology, and diagnosis amount to rearranging the deck chairs on the *Titanic*. Instead of debating alternative ways of handling information about symptoms, we should be moving beyond symptoms to diagnosis on the basis of underlying pathology. In doing so, we would be honoring Kraepelin rather than repealing his principles, and following in the best tradition of the medical model of psychopathology, rather than abandoning it.

THE LABORATORY REVOLUTION IN MEDICINE—OR, WHATEVER HAPPENED TO FEVER?

In his reliance on symptoms, Kraepelin and other early psychiatrists (there were no clinical psychologists yet) were simply following the practice of their colleagues in other medical specialties. The categories of medical disease changed little from the time of Hippocrates and Galen to that of Pasteur and Koch (Magner, 1992; Rosenberg, 1987; Starr, 1982). Well into the 19th century, prevailing theories still ascribed disease to imbalances in the four bodily humors, and treatment emphasized palliatives that made the patient as comfortable as possible while the disease ran its course. In fact, diagnosis was not a major enterprise for physicians, who, until well into the 19th century, emphasized the individuality of the patient. All that changed, however, with what has been called the *laboratory revolution in medicine* (Cunningham & Williams, 1992; see also Berger, 1999a, 1999b, 1999c, 1999d).

Historians of medicine (e.g., Ackerknecht, 1967) commonly distinguish between three epochs in medical history. The transition from *bedside medicine*, which prevailed from the Middle Ages well into the 18th century, to *hospital medicine*, which encompassed the late 18th and early 19th centuries, has been much discussed, most famously by Foucault (1973). It was marked not just by a shift in the site where medicine was practiced, but also by the introduction of the postmortem autopsy to discover the correlations between symptoms and anatomical lesions. The findings of hos-

someone who was retarded, and the residential centers for mentally retarded children did not want to take someone who was mentally ill. However, nobody on our staff had any problem recognizing that this young man had two problems, not just one, and working to address them both.

pital medicine forced physicians to shift their theory of disease, from the prevailing humor theory to an emphasis on pathological anatomy, but pathological anatomy could only be determined after the patient had died. In hospital medicine, as in bedside medicine, diagnosis of living patients was essentially subjective, based on symptoms and signs—the patient's complaints and whatever physicians and others could observe with their unaided senses (Ogilvie & Evans, 1997). Physicians rarely even touched their patients during examination, and it was remarkably common for them to diagnose and treat illnesses at a distance, by letter, without an office or home visit.

The further transition to laboratory medicine began in the middle of the 19th century and gained momentum with advances in microbiology, biochemistry, and radiation physics (Cunningham & Williams, 1992; Magner, 1992). Measurements of body temperature, pulse rate, and blood pressure were introduced in the 18th and 19th centuries; use of the stethoscope, ophthalmoscope, and laryngoscope became common only in the 1850s. The experimental physiology of Bernard (1865/1957) and the discovery by Pasteur and Koch of the microscopic organisms responsible for cholera and anthrax paved the way for the introduction of laboratory tests to detect such diseases as tuberculosis, typhoid, and diphtheria. Physicians no longer had to wait for autopsies to determine what made their patients ill: They could rely on laboratory tests—microscopic examination of specimens, blood chemistry, and, soon, X-rays—to determine the nature of disease in living patients. By the end of the 19th century, hospital pathology laboratories were well established in France, Germany, England, and the United States.

Perhaps the signal event of the laboratory revolution in medicine was the discovery, in 1905, of the syphilis spirochete. In terms of the clinical presentation of symptoms and signs, syphilis is positively protean (Magner, 1992). It mimics a great number of other diseases, including leprosy, tuberculosis, scabies, fungal infections, and skin cancers, to such an extent that diagnosis cannot be based on symptoms alone and must be confirmed by the Wasserman test for the presence of the syphilis spirochete in the affected individual's blood. Syphilis is diagnosed by a positive Wasserman test, regardless of the patient's symptoms and signs. Similarly, the diagnosis of HIV/AIDS is not based on symptoms such as pneumonia, diarrhea, seborrhoeic dermatitis, or even Kaposi's sarcoma, but rather by laboratory tests revealing the presence of specific antibodies in the blood. We have prostate-specific antigen tests and mammograms to detect cancers of the prostate and breast long before symptoms or signs appear. Accordingly, patients can be treated before they ever present symptoms, and treatments can be focused on what the patient's problem really is.

The increasingly prominent role of the laboratory in medical diagnosis has changed the organization of medical practice. Doctors must still

learn about symptoms and signs, but increasingly the final authority over diagnosis rests with clinical pathologists and other specialists (who may not even be medical doctors) working in laboratories often far removed from the patient's bed and the practitioner's office. Symptoms and signs enable the practitioner to generate hypotheses about what ails the patient. Increasingly, however, this hypothesis is tested, treatment decisions made, and outcomes evaluated on the basis of laboratory tests. Although managed care has reduced indiscriminate testing (including tests performed by laboratories in which doctors themselves have a financial stake), the laboratory has played an increasing role in the practice of "evidence-based" medicine.

The condition of "fever" provides a case in point. Around the turn of the last century, physicians recognized dozens of different kinds of fever, running virtually from A (blackwater fever) to Z (yellow fever), depending on whatever other symptoms the patient presented besides fever and chills.[6] Physicians may still diagnose Rocky Mountain spotted fever, in a way that they no longer diagnose yellow fever, but they no longer do so solely on the basis of symptoms of fever and rash (e.g., Ogilvie & Evans, 1997). Nor do they treat fever, except as a palliative, or attempt to prevent it. Rather, observation of fever and rash often leads a physician to order one or more blood tests. Positive results (*Rickettsia rickettsii* serology and Weil-Felix reaction) confirm the diagnosis (Zaret, 1997). Public-health efforts to prevent Rocky Mountain spotted fever focus on eliminating the ticks that spread the disease. Scarlet fever, yellow fever, and paratyphoid fever (which, in the *DSM*, probably would be labeled *typhoid fever not otherwise specified*) are diagnosed, treated, and prevented similarly.

Diagnosis, treatment, and prevention based on an understanding of underlying pathology, in turn based on an understanding of normal anatomy and physiology, are the surest signs of advanced medical practice. The medical profession has evolved a rich armamentarium of diagnostic tests, including the biochemical analysis of body fluids (chiefly blood and cerebrospinal fluid), imaging techniques (including X-rays, ultrasound, and magnetic resonance imaging), endoscopy, biopsy, and genetic analysis. Clinical pathology is a recognized medical specialty, backed by specially trained laboratory technicians and specialized laboratory facilities. Testing not only informs diagnosis but is also used in the evaluation of treatment; in fact, testing is used more frequently to monitor the progress of treatment than it is to establish a diagnosis in the first place (Zaret, 1997).

[6]For the record, a computer search of *Webster's New Collegiate Dictionary* turned up the following subtypes, in addition to those already named (and ignoring such entries as *buck fever*, *cabin fever*, and *spring fever*): breakbone fever, canicola fever, cat-scratch fever, childbed fever, dengue fever, fever blister, glandular fever, hay fever, hemorrhagic fever, Lassa fever, milk fever, paratyphoid fever, parrot fever, phlebotomous fever, puerperal fever, Q fever, rabbit fever, rat-bite fever, relapsing fever, snail fever, (Rocky Mountain) spotted fever, trench fever, typhoid fever, undulant fever, and valley fever.

TAKING PSYCHOPATHOLOGY SERIOUSLY (PARDON THE DUALISM)

In medicine, the laboratory has radically altered our understanding of disease itself. Diseases are no longer categorized and diagnosed by their prominent symptoms; instead, they are categorized in terms of etiology and underlying pathology. It is the underlying pathology, not the palpable signs and symptoms, that brings unity to the symptoms associated with syphilis. Likewise, differences in pathology, not differences in skin color, distinguish between scarlet, spotted, and yellow fever. This is the direction we should be heading in formulating a new diagnostic system for mental illness.

I want to make it clear, however, that I am not suggesting that the diagnosis of mental illness be based on underlying pathologies of anatomy and physiology. Diagnosing brain lesions is a neurologist's job, and psychology is not just something to do until the biologist comes along. Rather, I am suggesting that we base diagnosis on underlying mental abnormalities —disorders of cognitive, emotional, and motivational function that underlie the abnormalities of experience, thought, and action that present themselves as palpable signs and symptoms of mental illness. As in the case of clinical medicine, diagnosing mental illness might well begin with the practitioner's evaluation of symptoms and signs, but it would end in the psychopathology laboratory with the application of objective psychological tests—not paper and pencil questionnaires, but actual laboratory procedures—interpreted in light of a comprehensive understanding of normal mental function.[7]

Of course, this is an old story. The study of psychological deficits has a long history, including early studies by C. G. Jung on word associations, David Shakow on attention, and Kurt Goldstein on concept formation (for overviews, see Hunt & Cofer, 1944; Kihlstrom & McGlynn, 1991). Historically, most attention has been devoted to psychological deficits in schizophrenia (e.g., Chapman & Chapman, 1973; Magaro, 1980; Matthysse, Spring, & Sugarman, 1979; Oltmanns & Neale, 1982), although there has also been interest in anxiety and depression (e.g., Ingram & Kendall, 1987; Ingram & Reed, 1986; Mineka & Gilboa, 1998; Mineka & Zinbarg, 1998), psychopathy (e.g., Wallace, Schmit, Vitale, & Newman, 2000), and other disorders.

Kraepelin himself contributed to this tradition. Trained at Leipzig under Wundt, in the world's first psychology laboratory, Kraepelin performed seminal studies on the timing of various mental processes (in Wundt's terms, sensation, apperception, motor reaction) using Donders's

[7]Along the same lines, Nasby and I proposed that laboratory-based assessments of personality replace traditional test methods, such as questionnaires and projective tests (Kihlstrom & Nasby, 1981; Nasby & Kihlstrom, 1986).

method of mental chronometry (Boring, 1950). Later, as a psychiatrist, he conducted research on continuous performance in dementia praecox (Hunt & Cofer, 1944). Had he not been distracted from experimental psychopathology by clinical psychiatry, we might well have expected him to report experiments on the timing of various mental processes in schizophrenia.[8]

As noted earlier, Kraepelin preferred diagnosis based on pathological anatomy or etiology to diagnosis based on symptoms. However, there are reasons for thinking that, had psychological knowledge and theory been as advanced as biological knowledge of anatomy and physiology, Kraepelin might well have promoted diagnosis based on psychopathology. After all, in both the 6th and 7th editions of his *Textbook*, Kraepelin and Diefendorf (1904/1907) preceded the description of the "Forms of Mental Diseases" (pp. 113ff) with the following chapters: "Disturbances of the Process of Perception" (pp. 3–22), "Disturbances of Mental Elaboration" (pp. 23–61), "Disturbances of the Emotions" (pp. 62–76), and "Disturbances of Volition and Action" (pp. 77–112). In this respect, Kraepelin appears to have been following Immanuel Kant's classic proposal that cognition, emotion, and motivation constitute the fundamental and irreducible faculties of mind (Hilgard, 1980). The implications are that, whatever their biological substrates might be, the psychopathology underlying the various mental illnesses reflects deficits in certain basic psychological operations. Abnormalities in these basic mental functions, not observable signs and symptoms, should form the basis of the diagnosis of psychopathology.

Cognitive neuropsychology provides a model for this sort of diagnostic testing. Neuropsychological findings are often presented as dissociations in task performance interpreted within a theoretical framework known as the *modularity of mind* (e.g., Fodor, 1983; Pinker, 1997). Suppose, for example, that one brain-injured patient has severe difficulty reading words but exhibits intact writing abilities, and another patient shows precisely the reverse pattern of deficits. This contrast, known as a *double dissociation*, may lead to the conclusion that reading and writing words are tasks that are performed by two separate cognitive modules. The modularity framework conceives the mind as a system of mental modules, or psychological faculties, analogous to the various organs of the body. In the same way that the function of the stomach is to digest food and the function of the heart is to circulate blood, each of these mental modules is specially geared to perform a particular cognitive, emotional, motivational, or behavioral task. Indeed, as Pinker (1997) noted, the linguist Noam Chomsky has often referred to these modules as *mental organs*.

The general idea in neuropsychology is that these modules, or the

[8]In fact, almost a century later Wishner and his colleagues (Wishner, Stein, & Peastrel, 1978) performed just such a study, using Sternberg's (1969) adaptation of Donders' technique.

connections among them, can be selectively damaged, resulting in particular patterns of psychological deficits. Damage to modules involved in attention or reasoning may be implicated in what we now know as schizophrenia; damage to particular modules involved in the perception, experience, or expression of emotion may be implicated in the various affective disorders. Psychopathy may involve damage to motivational modules associated with behavioral inhibition as opposed to behavioral activation. Attention deficit disorder may involve damage to a module that focuses attention, leaving modules that disengage or shift attention intact. At present, experimental psychopathologists compare the performance of various diagnostic groups, diagnosed by symptoms and signs, in an attempt to reveal underlying psychological deficits. The present proposal is, essentially, to reverse the process: to test for differential psychological deficits, in the same way that clinical pathologists now test blood for the presence of antigens and antibodies or image the body to detect lesions in various tissues and organs, and create a new nosology based on psychopathological findings. It may well be that such a testing program, systematically applied, will reveal the psychological deficit(s) uniting "the group of schizophrenias." Alternatively, it may well be that laboratory tests will revise the diagnostic system entirely, consigning the term *schizophrenia* to the dustbin of history, replacing it with a new nomenclature more closely tied to underlying psychopathology.

Laboratory testing may also unite syndromes that heretofore have been considered to be separate. Consider, for example, the vicissitudes of the diagnosis of hysteria (Kihlstrom, 1994). In the time of Pierre Janet and Morton Prince, what we now call the dissociative and conversion disorders were joined together under the rubric of *hysteria*. By contrast, *DSM–IV* lists the conversion disorders under the rubric of *somatoform disorder*, separate from the dissociative disorders. The ostensible reason for this is that the symptoms of the conversion disorders resemble those of medical illnesses (Martin, 1992, 1994). However, the underlying psychopathology in the conversion disorders is clearly dissociative (Kihlstrom, 1992; Kihlstrom, Barnhardt, & Tataryn, 1992). Just as psychogenic amnesia, fugue, and multiple personality disorder involve an impairment of conscious memory, so "hysterical" blindness and deafness involve impairments of conscious sensation and perception, and "hysterical" paralysis involves an impairment of conscious motor control. Dissociations between explicit and implicit perception, analogous to familiar dissociations between explicit and implicit memory (Schacter, 1987), are easily revealed by appropriate laboratory tests (Kihlstrom, 1992; Kihlstrom et al., 1992). In this way, evidence of shared underlying psychopathology, derived from formal laboratory tests,

warrants rearrangement of the psychiatric nosology to rejoin the conversion and dissociative disorders.[9]

Most research in experimental psychopathology has not been explicitly guided by the notion of modularity, but the pervasive developmental deficit known as *autism* provides an example of how diagnosis might be based on psychological testing, rather than on observation of symptoms and signs. In *DSM–IV*, autistic disorder is diagnosed on the basis of impairments in social interaction and communication and repetitive and stereotyped patterns of behavior, interests, and activities. These symptoms are subjectively assessed by the clinician. However, according to one prominent theory (Baron-Cohen, 1995), autism results from a specific deficit in a particular mental module, called the *theory-of-mind mechanism*. This module, one of four deemed by Baron-Cohen to be critical to social cognition (the others are an intentionality detector, an eye-direction detector, and a shared-attention mechanism), permits one to infer other people's mental states from their behavior. Each of these social-cognitive modules can be assessed by specially designed laboratory tests; for example, the theory-of-mind mechanism is assessed by a "false beliefs" task (e.g., Peterson & Siegal, 1999).

Indeed, research by Baron-Cohen and others has revealed that autistic children perform normally on assessments of intentionality and eye-direction detection; however, one subgroup of autistic children performs normally on the shared-attention mechanism but poorly on the theory-of-mind mechanism, whereas another performs poorly on both. Children with mental retardation, by contrast, show intact performance in all four domains. In other words, we appear to have two quite different forms of autism, which deserve different labels in the nomenclature. Differential diagnosis of these conditions should not be based on symptoms (both groups met the *DSM–IV* criterion for autistic disorder), but rather on the results of laboratory tests. Application of rigorous, laboratory-based diagnostic tests may indicate whether we are really experiencing an epidemic of autism (in California the number of children enrolled in autism programs more than doubled from 1987 to 1998), or whether large numbers of children are being misdiagnosed on the basis of the subjective assessment of symptoms and signs. Similar benefits might accrue from the use of laboratory tests to diagnose attention deficit disorder.

[9]Actually, it must be said that such a rearrangement would be justified solely on symptomatic grounds, even in the absence of laboratory evidence of shared underlying psychopathology. After all, the symptoms of conversion disorder do not merely resemble those of medical disorders; they specifically resemble those of neurological disorders, just as the dissociative disorders do. However, in *DSM–IV* the conversion disorders remained where they had been in *DSM–III*, classified as somatoform. A source involved in this decision told me, not for attribution, that conversion was "the jewel in the crown" of the somatoform disorders and that the committee in charge of drafting the somatoform portion of *DSM–IV* "would never give it up."

SYMPTOMS ARE NOT THE DISEASE

In modern medicine, the laboratory has supplanted symptomatic complaints, medical histories, and physical examinations as the chief way of knowing (Warner, 1992). In addition to advancing biomedical knowledge, however, the laboratory is the basis of modern medical power—the ultimate source of the practitioner's sapiential, as opposed to moral or charismatic, authority. The laboratory performs diagnostic tests, identifies diseases, and evaluates the progress of treatment. Individual practitioners disregard laboratory findings at their peril. Patients (and, for that matter, third-party payers) have more faith if doctors' diagnoses and prescriptions are based on laboratory tests. It can be this way in psychopathology, as well. In an era of evidence-based medicine, including evidence-based psychotherapy, the use of laboratory tests to diagnose mental illness and evaluate the progress of treatment will help unify (or re-unify; see Kihlstrom & Canter Kihlstrom, 1998) science and practice. By placing diagnostic concepts and practices on a firmer scientific base, a shift from symptoms to laboratory tests will also reinforce the status and autonomy of clinical psychology, as well as the profession's claim to third-party payments for services. Connecting the laboratory more closely to the living material of the clinic will make basic research and theory more interesting.

S. S. Stevens (1961) sought to honor Fechner by repealing his law. In considering Kraepelin's legacy for psychopathology, we should remember that Kraepelin himself was a psychologist as well as a psychiatrist. When it comes to the diagnosis of mental illness, we can honor Kraepelin not by repealing his principles, but by reaffirming them—by moving beyond symptoms and diagnosing mental illness in terms of underlying pathology. For Kraepelin, diagnosis by symptoms was a temporary fallback, to be used only because diagnosis by pathology and etiology was not possible. This "fallback" has dominated our thinking for more than a century, and it is time to press forward, with all deliberate speed.

REFERENCES

Ackerknecht, E. H. (1967). *Medicine at the Paris Hospital, 1794–1848*. Baltimore: Johns Hopkins University Press.

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: Author.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual for mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory of mind.* Cambridge, MA: MIT Press.

Bayer, B. M., & Shotter, J. (Eds.). (1997). *Reconstructing the psychological subject: Bodies, practices and technologies.* Thousand Oaks, CA: Sage.

Berger, D. (1999a, July). A brief history of medical diagnosis and the birth of the clinical laboratory: Part 1. Ancient times through the 19th century. *Medical Laboratory Observer*, pp. 28–40.

Berger, D. (1999b, August). A brief history of medical diagnosis and the birth of the clinical laboratory: Part 2. Laboratory science and professional certification in the 20th century. *Medical Laboratory Observer*, pp. 32–38.

Berger, D. (1999c, October). A brief history of medical diagnosis and the birth of the clinical laboratory: Part 3. Medicare, government regulation, and competency certification. *Medical Laboratory Observer*, pp. 40–44.

Berger, D. (1999d, December). A brief history of medical diagnosis and the birth of the clinical laboratory: Part 4. Fraud and abuse, managed care, and lab consolidation. *Medical Laboratory Observer*, pp. 38–42.

Bernard, C. (1957). *Introduction to the study of experimental medicine.* New York: Dover. (Original work published 1865)

Blashfield, R. K. (1985). *The classification of psychopathology: Neo-Kraepelinian and quantitative approaches.* New York: Plenum.

Blashfield, R. K., & Flanagan, E. (1999). *The DSM as a folk taxonomy.* Unpublished manuscript, Auburn University.

Blashfield, R. K., & Fuller, A. K. (1996). Predicting the *DSM–V. Journal of Nervous & Mental Disease, 184*, 4–7.

Bleuler, E. (1950). *Dementia praecox or the group of schizophrenias.* New York: International Universities Press. (Original work published 1911)

Boring, E. G. (1950). *A history of experimental psychology.* New York: Appleton-Century-Crofts.

Cantor, N., & Genero, N. (1986). Psychiatric diagnosis and natural categorization: A close analogy. In T. Millon & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the* DSM–IV (pp. 233–256). New York: Guilford Press.

Cantor, N., Smith, E. E., French, R., & Mezzich, J. (1980). Psychiatric diagnosis as prototype categorization. *Journal of Abnormal Psychology, 89*, 181–193.

Caplan, P. J. (1995). *They say you're crazy: How the world's most powerful psychiatrists decide who's normal.* Reading, MA: Addison-Wesley.

Cattell, R. B. (1940). The description of personality: I. Foundations of trait measurement. *Psychological Review, 50*, 559–594.

Chapman, L. J., & Chapman, J. P. (1973). *Disordered thought in schizophrenia.* Englewood Cliffs, NJ: Prentice-Hall.

Clark, L. A., Watson, D., & Reynolds, S. (1995). Diagnosis and classification of psychopathology: Challenges to the current system and future directions. *Annual Review of Psychology, 46,* 121–153.

Cunningham, A., & Williams, P. (Eds.). (1992). *The laboratory revolution in medicine.* Cambridge, England: Cambridge University Press.

Danziger, K. (1990). *Constructing the subject: Historical origins of psychological research.* New York: Cambridge University Press.

Endicott, J., & Spitzer, R. L. (1978). A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *American Journal of Psychiatry, 25,* 131–139.

Eysenck, H. J. (Ed.). (1961). *Handbook of abnormal psychology.* New York: Basic Books.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry, 26,* 57–63.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1997). *Structured Clinical Interview for* DSM–IV *Axis I disorders: Clinician version: Administration booklet.* Washington, DC: American Psychiatric Press.

Fodor, J. A. (1983). *The modularity of the mind.* Cambridge, MA: MIT Press.

Foucault, M. (1973). *The birth of the clinic: An archaeology of medical perception.* New York: Pantheon

Genero, N., & Cantor, N. (1987). Exemplar prototypes and clinical diagnosis: Toward a cognitive economy. *Journal of Social & Clinical Psychology, 5*(1), 59–78.

Gleitman, H. (1995). *Psychology* (4th ed.) New York: Norton.

Grob, G. N. (1991). Origins of SDM–I: A study in appearance and reality. *American Journal of Psychiatry, 148,* 421–431.

Havens, L. L. (1965). Emil Kraepelin. *Journal of Nervous & Mental Disease, 141,* 16–28.

Havens, L. L. (1966). Pierre Janet. *Journal of Nervous & Mental Disease, 143,* 383–398.

Hilgard, E. R. (1980). The trilogy of mind: Cognition, affection, and conation. *Journal for the History of the Behavioral Sciences, 16,* 107–117.

Houts, A. C. (2000). Fifty years of psychiatric nomenclature: Reflections on the 1943 War Department Technical Bulletin, Medical 203. *Journal of Clinical Psychology, 56,* 935–967.

Hunt, J. M., & Cofer, C. N. (1944). Psychological deficit. In J. M. Hunt (Ed.), *Personality and the behavior disorders* (Vol. 2, pp. 971–1032). New York: Ronald.

Ingram, R. E., & Kendall, P. C. (1987). The cognitive side of anxiety. *Cognitive Research and Therapy, 11,* 523–536.

Ingram, R. E., & Reed, M. R. (1986). Information encoding and retrieval processes in depression: Findings, issues, and future directions. In R. E. Ingam (Ed.), *Information processing approaches to clinical psychology* (pp. 131–150). Orlando, FL: Academic Press.

Janet, P. (1907). *The major symptoms of hysteria.* New York: Macmillan.

Kendell, R. E. (1990). A brief history of psychiatric classification in Britain. In N. Sartorius, A. Jablensky, D. A. Regier, J. D. Burke, & R. M. A. Hirschfeld (Eds.), *Sources and traditions of classification in psychiatry* (pp. 139–152). Toronto: Hogrefe.

Kihlstrom, J. F. (1992). Dissociative and conversion disorders. In D. J. Stein & J. Young (Eds.), *Cognitive science and clinical disorders* (pp. 247–270). San Diego, CA: Academic Press.

Kihlstrom, J. F. (1994). One hundred years of hysteria. In S. J. Lynn & J. W. Rhue (Eds.), *Dissociation: Clinical and theoretical perspectives* (pp. 365–394). New York: Guilford Press.

Kihlstrom, J. F. (2001). Dissociative disorders. In P. B. Sutker & H. E. Adams (Eds.), *Comprehensive handbook of psychopathology* (3rd ed. pp. 259–276). New York: Plenum.

Kihlstrom, J. F., Barnhardt, T. M., & Tataryn, D. J. (1992). Implicit perception. In R. F. Bornstein & T. S. Pittman (Eds.), *Perception without awareness: Cognitive, clinical, and social perspectives* (pp. 17–54). New York: Guilford Press.

Kihlstrom, J. F., & Canter Kihlstrom, L. (1998). Integrating science and practice in an environment of managed care. In D. K. Routh & R. J. De Rubeis (Eds.), *The science of clinical psychology: Accomplishments and future directions* (pp. 281–293). Washington, DC: American Psychological Association.

Kihlstrom, J. F., & McGlynn, S. M. (1991). Experimental research in clinical psychology. In M. Hersen, A. E. Kazdin, & A. S. Bellack (Eds.), *Clinical psychology handbook* (2nd ed., pp. 239–257). New York: Pergamon.

Kihlstrom, J. F., & Nasby, W. (1981). Cognitive tasks in clinical assessment: An exercise in applied psychology. In P. C. Kendall & S. D. Hollon (Eds.), *Cognitive–behavioral interventions: Assessment methods* (pp. 287–317). New York: Academic Press.

Klerman, G. L. (1977). Mental illness, the medical model and psychiatry. *Journal of Medicine & Philosophy, 2,* 220–243.

Kraepelin, E., & Diefendorf, A. R. (1907). *Clinical psychiatry; a text-book for students and physicians* (7th ed.). New York: Macmillan. (Original work published 1904)

Kutchins, H., & Kirk, S. A. (1997). *Making us crazy:* DSM: *The psychiatric Bible and the creation of mental disorders.* New York: Free Press.

Magaro, P. A. (1980). *Cognition in schizophrenia and paranoia: The integration of cognitive processes.* Hillsdale, NJ: Erlbaum.

Magner, L. N. (1992). *A history of medicine.* New York: Dekker.

Martin, R. L. (1992). Diagnostic issues for conversion disorder. *Hospital & Community Psychiatry, 43,* 771–773.

Martin, R. L. (1994). Conversion disorder, proposed autonomic arousal disorder, and pseudocyesis. In T. A. Widiger, A. J. Frances, H. A. Pincus, R. Ross, M. B. First, & W. W. Davis (Eds.), DSM–IV *sourcebook* (Vol. 2, pp. 893–914). Washington, DC: American Psychiatric Press.

Matthysse, S., Spring, B. J., & Sugarman, J. (Eds.). (1979). *Attention and information processing in schizophrenia.* Oxford, England: Oxford University Press.

Medin, D. L. (1989). Concepts and conceptual structure. *American Psychologist, 44,* 1469–1481.

Medin, D. L., Goldstone, R. L., & Gentner, D. (1993). Respects for similarity. *Psychological Review, 100,* 254–278.

Mineka, S., & Gilboa, E. (1998). Cognitive biases in anxiety and depression. In W. F. Flack & J. D. Laird (Eds.), *Emotions in psychopathology: Theory and research* (pp. 216–228). New York: Oxford University Press.

Mineka, S., & Zinbarg, R. (1998). Experimental approaches to the anxiety and mood disorders. In J. G. Adair, D. Belanger, & K. L. Dion (Eds.), *Advances in psychological science: Vol. 1. Social, personal, and cultural aspects* (pp. 429–454). Hove, England: Psychology Press/Erlbaum.

Nasby, W., & Kihlstrom, J. F. (1986). Cognitive assessment in personality and psychopathology. In R. E. Ingram (Ed.), *Information processing approaches to psychopathology and clinical psychology* (pp. 217–239). Orlando, FL: Academic Press.

Ogilvie, C., & Evans, C. C. (Eds.). (1997). *Chamberlain's symptoms and signs in clinical medicine: An introduction to medical diagnosis* (12th ed.). Oxford, England: Butterworth-Heinemann.

Oltmanns, T. F., & Neale, J. M. (1982). Psychological deficits in schizophrenia: Information processing and communication problems. In M. Shepard (Ed.), *Handbook of psychiatry* (pp. 55–61). Cambridge, England: Cambridge University Press.

Orne, M. T. (1962). On the social psychology of the psychological experiment: With particular reference to demand characteristics and their implications. *American Psychologist, 17,* 776–783.

Orne, M. T. (1973). Communication by the total experimental situation: Why it is important, how it is evaluated, and its significance for the ecological validity of findings. In P. Pliner, L. Krames, & T. Alloway (Eds.), *Communication and affect* (pp. 157–191). New York: Academic Press.

Penick, E. C., Nickel, E. J., Cantrell, P. F., & Powell, B. J. (1990). The emerging concept of dual diagnosis: An overview and implications. *Journal of Chemical Dependency Treatment, 3*(2), 1–54.

Peterson, C. C., & Siegal, M. (1999). Representing inner worlds: Theory of mind in autistic, deaf, and normal hearing children. *Psychological Science, 10*(2), 126–129.

Pinker, S. (1997). *How the mind works.* New York: Norton.

Ray, I. (1962). *A treatise on the medical jurisprudence of insanity.* Cambridge, MA: Harvard University Press. (Original work published 1838)

Rosenberg, C. E. (1987). *The care of strangers: The rise of America's hospital system.* Baltimore: Johns Hopkins University Press.

Schacter, D. L. (1987). Implicit memory: History and current status. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 13,* 501–518.

Shagass, C. (1975). The medical model in psychiatry. *Comprehensive Psychiatry, 16,* 405–413.

Shorter, E. (1997). *A history of psychiatry: From the era of the asylum to the age of Prozac.* New York: Wiley.

Siegler, M., & Osmond, H. (1968). Models of drug addiction. *International Journal of the Addictions, 3,* 3–24.

Siegler, M., & Osmond, H. (1969). The impaired model of schizophrenia. *Schizophrenia, 1,* 192–202.

Siegler, M., & Osmond, H. (1971). Goffman's model of mental illness. *British Journal of Psychiatry, 119,* 419–424.

Siegler, M., & Osmond, H. (1974a, November). Models of madness: Mental illness is not romantic. *Psychology Today,* pp. 70–78.

Siegler, M., & Osmond, H. (1974b). *Models of madness, models of medicine.* New York: Harper & Row.

Sigler, M., Osmond, H., & Mann, H. (1969). Laing's model of madness. *British Journal of Psychiatry, 115,* 947–958.

Siegler, M., Osmond, H., & Newell, S. (1968). Models of alcoholism. *Quarterly Journal of Studies on Alcohol, 29,* 571–591.

Smith, E. E., & Medin, D. L. (1981). *Categories and concepts.* Cambridge, MA: MIT Press.

Spitzer, R. L., Endicott, J., & Robins, E. (1975). *Research diagnostic criteria (RDC) for a selected group of functional disorders.* New York: New York State Psychiatric Institute.

Spitzer, R. L., & Fleiss, J. L. (1974). A re-analysis of the reliability of psychiatric diagnosis. *British Journal of Psychiatry, 125,* 341–347.

Spitzer, R. L., Williams, J. B. W., Gibbon, M., & First, M. B. (1990). *Structured Clinical Interview for* DSM–III–R. Washington, DC: American Psychiatric Press.

Starr, P. (1982). *The social transformation of American medicine: The rise of a sovereign profession and the making of a vast industry.* New York: Basic Books.

Sternberg, S. (1969). The discovery of processing stages: Extensions of Donders' method. In W. G. Koster (Ed.), *Attention and performance II* (Vol. 30, pp. 276–315). Amsterdam: North-Holland.

Stevens, S. S. (1961). To honor Fechner and repeal his law. *Science, 133,* 80–86.

Wallace, J. F., Schmitt, W. A., Vitale, J. E., & Newman, J. P. (2000). Experimental investigations of information-processing deficiencies in psychopaths: Implications for diagnosis and treatment. In C. B. Gacono (Ed.), *The clinical and forensic assessment of psychopathy: A practitioner's guide* (pp. 87–109). Mahwah, NJ: Erlbaum.

Warner, J. H. (1992). The fall and rise of professional mystery: Epistemology, authority and the emergence of laboratory medicine in nineteenth-century America. In A. Cunningham & P. Williams (Eds.), *The laboratory revolution in medicine* (pp. 140–141). Cambridge, England: Cambridge University Press.

Watson, D., Clark, L. A., & Harkness, A. R. (1994). Structures of personality and their relevance to psychopathology. *Journal of Abnormal Psychology, 101*, 18–31.

Wishner, J., Stein, M. K., & Peastrel, A. L. (1978). Information processing stages in schizophrenia. *Journal of Psychiatric Research, 14*(1, Suppl. 4), 35–45.

Woodruff, R. A., Goodwin, D. W., & Guze, S. B. (1974). *Psychiatric diagnosis*. New York: Oxford University Press.

Woody, G. E., McLellan, A. T., & Bedrick, J. (1995). Dual diagnosis. *American Psychiatric Press Review of Psychiatry, 14*, 83–104.

World Health Organization. (1990). *International classification of diseases* (10th ed.). Geneva, Switzerland: Author.

Zaret, B. L. (Ed.). (1997). *Patient's guide to medical tests*. Boston: Houghton-Mifflin.

Zimberg, S. (1993). Introduction and general concepts of dual diagnosis. In J. Solomon, S. Zimbarg, E. Shollar, & M. M. O'Neill (Eds.), *Dual diagnosis: Evaluation, treatment, training, and program development* (pp. 3–21). New York: Plenum.

Zubin, J. (1967). Classification of behavior disorders. *Annual Review of Psychology, 18*, 373–406.